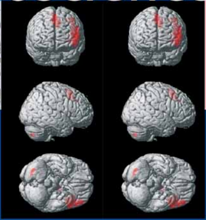
Mauro Mancia
Editor

Psychoanalysis and Neuroscience



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Cover: *left panel*, Une leçon clinique à la Salpetrière.1887, by André Brouillet
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Preface

The papers collected in this volume have the aim of integrating the contributions from two disciplines: psychoanalysis and neuroscience. Recent great developments in the latter offer the opportunity to build a bridge to psychoanalysis, providing an anatomo-functional basis for some of the functions in which the psychoanalytic theories of the mind are rooted. The functions which are specifically discussed from an interdisciplinary point of view are the following: emotions, memory, unconscious, perception, attention, wakefulness, sleep, dreams, empathy, sharing affects, intentionality, embodied simulation, and aspects of infantile mental development.

The book is divided into four sections. The first contains recent observations relating to memory in its double function (implicit and explicit) and its relation to the unconscious. This section also discusses the relationship between emotions, memory, and cognitive functions of the mind. Particular attention is paid to the right hemisphere, considered the emotional brain *par excellence*. Papers on the brain's predictive capacities and its plasticity in relation to special functional states complete this first section.

The second section is dedicated to neuropsychological findings from bioimaging investigations related to the human mind's ability to share emotional and affective experiences. Here the role of mirror neurons in empathy, intentionality, and embodied simulation is discussed.

The third section looks at the dream as a topic for dialogue between psychoanalysis, neurosciences, and cognitivism. The contributions of neurophysiology, experimental psychology, and neuropsychology are amply documented, focusing on the role of various cerebral structures and circuits (dopaminergic) in the organization of the mind's oneiric function. The importance of psychoanalysis in giving a meaning to the dream, linking it to early emotional experiences, and allowing a reconstruction of the patient's unconscious emotional and affective history is highlighted.

The fourth section considers recent observations about prenatal and neonatal life. Fetal behavior has been studied using ultrasonographic techniques from the 10th week of gestation to delivery. Electrophysiological studies after birth have made significant contributions to our understanding of the early organization of the infant mind.

In conclusion, the book hopes to give readers a wide view of the various fields of interaction where neuroscientific observations can provide a neurofunctional basis for psychoanalytic theories and clinical work.

I am especially grateful to Dr. Donatella Rizza for her support in collecting the papers and in structuring the whole book; to Mrs. Judy Baggott for her excellent translation of most of the chapters, and to Mrs. Laura Matteini for her help in revising the manuscripts.

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Introduction: How the Neurosciences Can Contribute to Psychoanalysis

MAURO MANCIA

Premise

Co-operation, not incorporation (Pugh, Chap. 1, this volume) integration, reciprocal contributions to the knowledge of the mind's functions; mutual respect for the methodological and epistemological limitations of each discipline: these are the standards that govern my thoughts on interdisciplinarity and must be accepted for reciprocal enrichment between neurosciences and psychoanalysis to be possible.

Talking of psychoanalysis to neuroscientists is no easier than talking of neurosciences to psychoanalysts. The whole of the twentieth century was characterized by deafness and sometimes vulgar argument between the proponents of the two disciplines, often marked by ignorance or limited knowledge of theoretical and clinical psychoanalysis by neuroscientists and lack of interest as well as ignorance on the part of psychoanalysts concerning the research and developments of the neurosciences. However, the great advances made in neurology, neurophysiology, neuropsychology and experimental psychology, combined with the training of a new generation of psychoanalysts who are more open than their predecessors to confrontation with other disciplines, or at all events are stimulated to consider other avenues of thought and interdisciplinary contacts–or even concerned about the "crisis" into which psychoanalysis has entered as a result of continuously focusing on the same concepts–have facilitated interactions between psychoanalysis and the neurosciences and helped to overcome reciprocal diffidence. It has, therefore, been possible to lay a common foundation on which to build a language that can promote research into the functions of the mind: an aim very dear to psychoanalysis, but which the neurosciences are now approaching with interest.

In recent years there has been a marked increase in dialogue between neurobiology and psychoanalysis. One example is the group of neuropsychoanalysts who have contributed to the study of conscious and unconscious mental activity in patients with focal neurological lesions, observed with the analytical method [1, 2]. Other studies on this subject have been published recently [3], and the European Psychoanalytical Federation has charged me to organize ad hoc groups at its annual meetings.

Thus, there are the beginnings of a shared line of thought, no longer intended to demonstrate that Freud was right or wrong, or that psychoanalytical theories are or are not compatible with the definition of science, but rather one intended to produce experimental data able to amplify basic psychoanalytical concepts and give them anatomical–functional consistency in the attempt, already theorized by Freud, of one day being able to explain the mind, with its emotions, affective aspects, passions and thoughts, in scientific terms as close as possible to those used in chemistry, physics and biology. This line of thought must be nurtured with the premise that the mind can be explained only by an ontological monism, even if the methods for studying it are epistemologically and methodologically different, and with the awareness that psychoanalysis is completely based on intersubjectivity whereas the neurosciences are based on the relationship between a subject and his or her object of interest. Finally, it must also be accepted that the logic underlying the research differs: the neurosciences are based on a *logic of explanation*, whereas psychoanalysis is characterized by a *logic of understanding*.

The areas of possible interest and integration between the various neuroscientific disciplines and psychoanalysis are numerous, and every area of research has led to a significant reciprocal enrichment:

- The study of the emotions and their role in the development of the infant mind, in the organization of the different forms of memory and in human behavior
- Research on the different memory systems, their neuroscientific correlates and their relationship with the characteristics and functions of the unconscious
- The discoveries concerning REM and non-REM sleep and their relationships with dreaming
- High-resolution ultrasound studies of the fetus, unraveling behavioral aspects and the development of the central nervous system

- Notions on the earliest communication between a neonate and its mother and the environment, as deduced from infant observation
- Research on the development of speech, starting from the effect of the maternal voice in the antenatal period, up to the early neurological organization of the sensorimotor, grammatical and semantic centers of language
- Neurobiological maturation and mental development in relation to the functions of consciousness and the unconscious
- The study of empathy and the sharing of emotional and affective states, in relation to the experience of physical pain
- Analysis of the complex question of mirror neurons, which open up important and fascinating perspectives on how the brain is able to produce "engrams" or "neural configurations" in response to its own movement or the observation of subjects performing the same movements, and in connection with the intentionality, imitation, embodied simulation and sharing of normal and pathological affective and relational states.

This book covers these varied scientific prospects with the aim of comparing objective data deriving from neuroscientific research with subjective data from psychoanalysis in the hope that this will trigger ideas and hypotheses that could give a deeper meaning to psychoanalytical work and at the same time offer possible interpretations of the neuroscientific observations. These latter, having the privilege of objectivity and reproducibility, may provide psychoanalysis with the anatomofunctional basis that can give substance to the hypotheses deriving from the intersubjective encounter between two people in the consulting room.

The aim of this book is to promote a line of theoretical and clinical thinking that gradually moves away from metapsychology to approach ever more closely a real and true psychology which is open to experimentation and scientific observation–a psychology that enables a connection between neurophysiological and mental functions as the basis for an integrated theory of the mind. This interdisciplinary work is also intended to create a common language, to allow understanding of the meaning of the terms and concepts used, an exchange of hypotheses and ideas, that can enrich both psychoanalysis and the neurosciences. These latter, in fact, like all sciences, cannot ignore the conscious and unconscious mind of the person experimenting, observing and interpreting, and the study of these functions of the mind is the main task of psychoanalysis.

Emotions and Memory

I would like to mention briefly some of the most recent contributions on mental functions that the neurosciences have investigated with experimental methods and that form the bulk of the discussion in this book. I shall start with the problem of emotions, because of the implications that these have for general psychology (especially concerning the organization of memory) and for the theory and practice of psychoanalysis.

After Darwin, who in 1872 laid the basis for a scientific treatment of the emotions in "The Expression of the Emotions in Man and Animals" [4], few scientists dealt with this theme that is so central to psychology and human and social sciences. This relative lack of interest by neuroscientists in the emotions lasted for many years but was brought to an end by the work of Ledoux [5] who, a century after Darwin, published in "The Emotional Brain" the basic ideas about how the emotions are produced in the brain, how they are expressed, how the brain perceives emotionally exciting stimuli and responds to them, how an individual learns and remembers emotions, and how the conscious feelings of humans emerge from the unconscious.

Among Ledoux's most original contributions that is of use in psychoanalysis, I mention here that related to the influence of emotions on memory processes. Ledoux re-examined the experimental data from animals and humans that had led to the discovery of the dual memory system, implicit and explicit [6, 7], the former mediated by various structures under the control of the amygdala, the latter mediated by the temporal lobe where the hippocampus plays a key role. Explicit memory is defined as conscious memory, while implicit memory is unconscious. The orchestrator of explicit memory is the hippocampus, since this is connected to the association cortex through the so-called transition cortex (perirhinal, entorhinal, parahippocampal), where the information arrives from the sensory cortices (visual, auditory, somatic, gustatory, olfactory). The hippocampus organizes "engrams" or "neural configurations" with which it selects and codes the information that it receives in order to store it, via the thalamic nuclei, in the association cortex, which is widely spread in the brain. This process explains the observations previously made by Lashley [8] that allow memory functions to be considered as holistic, that is, belonging diffusely to the neocortical layer.

The implicit memory is the more directly emotional form of memory. It is managed by the amygdala and the regions linked to it, such as the hypothalamus, the brain stem, the basal nuclei, the cerebellum and areas of association cortex. The amygdala is connected by two pathways to the hippocampus [9] and thus also influences the explicit or declarative memory. The implicit memory system is activated in conditioned emotions such as fear, which are also known to involve the cerebellum [10]. The implicit memory was also invoked by Ledoux to explain infantile amnesia. Extending work by Jacobs and Nadel [11], he stated that early trauma, even that not remembered, can have a long-lasting effect on the development of the mind, and that the amygdala matures before the hippocampus. This statement anticipated work by Siegel [12], who highlighted the different times of maturation of the two key structures in the memory systems, favoring the hypothesis that implicit memory matures earlier than explicit memory. This differential maturation has enormous implications for the development of the infant mind and for the process leading to the organization of the unconscious, as will be explained later.

There are many ways in which an emotional experience or stimulus can be memorized. The emotional stimulus can reach the amygdala through a nervous pathway and directly involve forebrain and cortical structures in order to be stored. However, via the amygdala, lateral hypothalamus, brain stem and autonomic nervous system, the same emotional stimulus can cause the release of epinephrine (adrenaline), which facilitates, through the locus caeruleus, the secretion of norepinephrine (noradrenaline). The norepinephrine returns to the amygdala itself and to the hippocampus, activating them. This coactivation of the two key structures of the memory by an emotional stimulus can facilitate learning and remembering. This process may also participate in recall in the context of the explicit memory system.

The effects of trauma and stress on the two memory systems, also considered by Ledoux, are important for both psychology and psychoanalysis, since the effects of trauma on the amygdala are different from those on the hippocampus. Recent research focused on this issue has confirmed the role of early trauma in memory functions [13]. Trauma of various types can make apparently extinct conditioned responses to fear re-emerge in mammals. This experimental finding is also of psychoanalytical interest given that it allows us to think that certain traumatic or stressful experiences in the past can re-emerge in the transference since, although apparently extinct (that is, not recallable), they are stored in the implicit memory. This apparent extinction has not cancelled the experience, which has, in fact been stored in the implicit memory and continues to influence the emotional and cognitive aspects of a person for the rest of his or her life. Ledoux [5] proposed a neurophysiological explanation of the resistance to eliminating a conditioned fear stored in the implicit memory. He hypothesized that the neuronal organization of the amygdala could be such as to create a conditioned network which, through a particular "plasticity" of neurons, produces conditions for resisting extinction. This leads to the emotional experiences being impressed indelibly in the implicit system and being therefore unable to be canceled. However, their expression is "plastic" and can be regulated and transformed by the brain through cortical control of the amygdala itself. In fact, the amygdala sends many projections to cortical areas, and thus emotional stimuli can pass directly from the amygdala to the association cortex (the right hemisphere is probably more involved in these projections) (Gainotti, Chap. II, this volume). However, the association cortex also send fibers to the amygdala, albeit to a lesser extent, thus creating a complex feedback loop between the amygdala and the association cortex [14]. It is this loop that enables the cortex to exercise a regulatory action on the amygdala. This fact lends support to the hypothesis that thought processed during the analysis, which we presume has a functional arrangement in the neocortex, can modulate and control the emotions through its action on the amygdala.

Since Ledoux, many neuroscientists have studied the emotions using more sophisticated techniques and have been able to observe in vivo which areas of the brain are excited when emotional stimuli reach them, and how these stimuli can be processed. For example, Damasio [15] considers emotion as a process capable of promoting a cascade of events: the secretion of substances that, in their turn, create an event that produces the discharge of other substances that diffuse to the various regions of the brain. For this reason, emotional stimuli produce changes in the chemical profile of the body; these changes in their turn modify the cerebral structures and functions that organize the "neural configurations" that become responsible for the emotional experience. Thus emotion is seen as a transitory change in the state of the organism and as a concrete set of "neural configurations" organized by maps of relational structures. These organizations are formed very early, influencing the development of the infant mind, and are able to condition a person's behavior and thought throughout his or her entire life.

In a subsequent study, Damasio [16] hypothesized that emotions precede feelings in that the former appear earlier in the course of evolution. Damasio suggests that the mental processes are based on maps of the body present in the brain in the form of "neural configurations" that represent the responses to events that elicit emotions and feelings. He distinguishes primary or fundamental emotions, which include fear, anger, happiness and sadness, from social emotions, which include embarrassment, guilt, jealousy, envy, etc. These latter, in particular, seem to be produced by neural configurations that regulate behavior and that are innate. An appropriate stimulus activates association and somatosensory areas of the cortex, which in their turn involve the amygdala through activation of the basal forebrain, hypothalamus and brain stem. Thus, somatosensory association areas can be considered as "switchboards" or areas that contain maps of the body and form an organ redirecting emotions and feelings that subsequently involve the anterior parts of the cingulum and the insula. The somatosensory areas that produce the highest level of representation of the state of the body are those in the right hemisphere. This fact is connected to the role of the right hemisphere in the processes of emotions and emotional memory (Gainotti, Chap. 5, this volume and [17]), as will be seen later.

The brain is endowed with systems able to create representations that simulate what other individuals feel through certain facial expressions, which can convey feelings and emotions. Adolphs et al. [18] suggest that the somatosensory cortex plays a fundamental role in these representations that can recognize facial emotions. The right somatosensory cortex, in particular, together with the amygdala and the visual cortex of the ipsilateral hemisphere, appear to be important in recovering relational information from the observation of faces. Furthermore, studies using functional imaging [19] have shown that imitating another person through visual observation produces activation of the left frontal operculum and the right parietal cortex. These experimental data in humans suggest that facial expressions of emotions involve regions of the right hemisphere that serve as visual and somatosensory representations of emotions associated with facial expressions. Neuropathological cases demonstrate that a lesion of the right somatosensory cortex can produce anosognosia and disordered awareness of one's own bodily state, often accompanied by a flattening of emotions. The right somatosensory cortex and the amygdala therefore function as two indispensable components of a neurological system that allows knowledge of the facially expressed emotions to be retrieved.

Jaak Panksepp [20] made an extremely important contribution to the discussion on early emotions when he provided evidence in favor of the hypothesis that early emotional experiences can profoundly influence the development of the brain and the personality of a child, since they cause a change in the child's mental economy. This transformation constitutes the basis of what Edelman [21] called "neural darwinism." It is now known that positive early emotions promote a healthier and more optimistic personality, while negative emotional experiences can be the cause of strong anxiety and depressive states in the subject even in adulthood. These influences can work at the level of the brain, promoting growth factors or the secretion of various molecules, including glutamate, in response to environmental stimuli. These substances can be of great relevance in modulating the "plasticity" of the central nervous system, and the responses can explain the fact that neonates and children live their emotions more intensely than do adults and are able to activate autoregulatory processes of these emotions. The primary sense of Self, which is organized right from birth, belongs more to the emotional sphere than to the cognitive one. Early trauma, such as abandonment, physical and psychological violence, abuse and stress of various types, can create the conditions for a lifelong tendency to sadness, depression, anger and resentment. A mother's contentment with her neonate and physical skin-to-skin contact convey feelings and emotions that can enhance the production of growth factors and the release of oxytocin and other substances that reduce infantile stress. The production of corticotropin-releasing factor (CRF) is important in this regard since this hormone has been shown in experimental studies of mammals to be the factor most strongly influenced by emotional stress produced by separating the neonate from its mother.

It is now certain that early environmental influences can affect neuronal plasticity [22]. The human brain stem is fully functional at birth. It responds to vital vegetative and motor functions. The limbic system (hypothalamus, amygdala, septum, cingulum and hippocampus) controls hunger and thirst and is responsible for the experience and expression of emotions including pleasure, anger, fear, joy, and desire for emotional social contact. Early environmental experiences can, therefore, influence these specific neural networks, affecting their ability to select and control behavior. For this reason early social, emotional and environmental influences can exert significant effects on the organization of the brain and on intellectual, social and emotional aspects of development.

The relevance of these observations to psychology and psychoanalysis is immediately clear if it is considered that the neonate with its limbic system, which sees to the organization of all aspects of social and emotional behavior, requires a considerable amount of social and physical stimuli, including maternal, sensory and emotional contacts, in order to develop. This "limbic need" for emotional stimulation is so pervasive that for most of the first year of life the neonate searches constantly for social contact and smiles and anyone, even complete strangers, who comes near. Of the various components of the limbic system, it is the amygdala that appears to be the organ guiding the processes underlying the affective and emotional behavior of mammals, and in particular humans. It receives multimodal sensory inputs and projects them to all levels of the central nervous system, thus consolidating the emotional memories. Its neurons show theta activity during emotional arousal and various types of oscillation during the sleep characterized by synchronous waves [23]. Indeed, Paré et al. [23] hypothesized that the synchronizing events of the amygdala can promote synaptic plasticity of the brain, facilitating interactions between the noncortical areas that participate in the deposition of information and the structure of the medial temporal lobe more directly involved in explicit or declarative memory. However, since the amygdala manages the circuit underlying the implicit memory system and has a role in influencing emotional memories, it probably participates in the organization, classification and deposition of early experiences that will be stored in this form of memory.

It is known that the emotions strongly influence memory functions. In emotive situations the amygdala modulates both the classification and the storage of the hippocampus-dependent memory, that is, the explicit memory [9]. This action is also entrusted to the stress hormones that activate adrenergic receptors in the basolateral amygdala which, in its turn, modulates the effects of these hormones on hippocampal consolidation. Bioimaging studies have suggested that the right and left amygdala are differentially involved in the process of memory of emotional stimuli in relation to the gender of the subject. The left amygdala seems to be correlated with the organization of memory in women, whereas the right amygdala is more active in the organization of memory in men. The left and right amygdala are also selectively involved in the memorization of, respectively, verbal and visual experiences. The bioimaging investigations also suggest that the relationship between the amygdala and the hippocampus can be bidirectional with regard to the classification of essentially emotional events [9].

The work by McGaugh [24] provides fascinating information on the role of the amygdala in consolidating long-term memory. This author hypothesized that the basolateral region of the amygdala has a crucial role in the process of memorizing emotional experiences. There is evidence that stress-related hormones and other neuromodulatory systems converge in regulating the activity of the noradrenergic receptors within the basolateral nucleus of the amygdala. This nucleus modulates mnemonic consolidation through its projections to other important structures of the explicit memory system such as the hippocampus, the caudate nucleus, the basal nucleus and the cerebral cortex. Alongside its involvement in the emotional aspects of the explicit memory, intuited from its capacity to activate hippocampal neurons, the amygdala participates in the consolidation of the implicit memory as emotional memory, as suggested for example by its role in consolidation of conditioned fear. These data constitute a functional basis for a reciprocal influence between implicit and explicit memory, a modulation of extreme importance in the development of the infant mind and in the processes of early learning.

Besides these observations [9], another important interaction between the amygdala and hippocampus has been described. This interaction provides subcortical limbic regulation of memory processes and the effects of early trauma and stress on the structure and function of the hippocampus. Stress increases the levels of corticosteroids, causing atrophy of the dendrites of the CA3 pyramidal cells, inhibits neurogenesis in the dentate gyrus, and hampers learning and memorization of the hippocampus-dependent explicit memory [9, 25]. These observations seem to be extremely relevant for developmental psychology. Stress caused by deprivation or separation of neonatal small mammals from their mother for 3 h each day decreases the production of new granules in the adult through a corticosteroid-dependent mechanism [13]. Such early trauma in infancy permanently hampered learning and hippocampus-dependent memory in parallel and increased the tendency to deprivation by inhibiting adult neurogenesis of the hippocampus. These observations are of particular interest for developmental psychology and for psychoanalysis, which may trace many aspects of mental disorders to disturbances and trauma that involved the neonate in an early stage of development.

The numerous contributions discussed so far on the role of the emotions in the processes of learning and memorization through their action on the amygdala and on the hippocampus–key structures in the function of the implicit and explicit memory, respectively–are complemented and integrated by the most recent findings and reflections presented in this book.

Sergerie and Armony (this volume) highlight that the essence of emotions lies in their negative effects on thought. Citing the work of other researchers [26] they report the role of the neurons of the amygdala in responding selectively to faces and in modulating attention processes through the direct projections of these neurons to cortical areas or indirectly through their effect on the cholinergic system of the "forebrain" and the noradrenergic system of the locus caeruleus [27]. The orbitofrontal cortex is considered by Sergerie and Armony to be part of a connecting system or interface that unites the system signaling a threat (the amygdala) with the attention system (the frontoparietal cortex). In this view, danger signals activate an attention system through the amygdala, with the orbitofrontal cortex acting as an interface. The amygdala is the essential part of the system that increases emotional memory. Since this increase is absent in experimental models in which the left, but not the right, amygdala is damaged, it is clearly the former that has a role in strengthening the emotional dimension of declarative memory, whether following verbal stimuli (narration) or nonverbal ones (pictures).

Some authors [24, 28] believe that the amygdala influences the deposition of emotional memories in various areas of the brain involved in episodic memory. It has been suggested that this limbic nucleus modulates emotional memory, classifies it and consolidates it through its influence on other structures such as the hippocampus, the corpus striatum and the cerebral cortex [29]. In this case the amygdala would not participate in the recall of emotional experiences, which would be the function of the hippocampus alone. This hypothesis is interesting for its distinction of the two memory systems: the explicit and the implicit. On the basis of these data it is probable that the amygdala classifies and consolidates the implicit emotional memory that does not allow recall. However, its participation in the recovery of emotional material has been hypothesized by some authors [30], while others [31] report specific responses of the amygdala in subjects looking at pictures that portray emotions. Sergerie and Armony highlight the uncertainties that still exist about the role of the amygdala in recalling memorized experiences. It is probable that it is involved in retrieving emotions deposited in the explicit memory due to its relationship with the hippocampus, while it participates in the storage of emotions in the implicit memory only in their classification and consolidation, but not their recall, which this form of memory does not, in any case, allow.

Memory and Unconscious

The emotions can be seen as being part of an adaptive process in relation to the cognitive system and the asymmetrical hemispheric functions (Gainotti, Chap. 5, this volume). It is the right hemisphere that seems to be delegated to organizing the lower level of emotions and integrating it with their higher level. Recent observations suggest that the right hemisphere is involved in the various forms of unconscious emotional memories: those produced by emotional conditioning and those represented by the various dimensions of the implicit memory. This latter function is of particular interest for psychoanalysis since it links the emotional memory to the unconscious and gives the right hemisphere the important role in the organization of this last function of the mind.

The discovery of the dual memory system [7, 32–34]–the explicit or declarative memory that is conscious, can be verbalized and recalled, and is essential to our identity and our autobiography, and the implicit memory, that is unconscious and cannot be verbalized or recalled–opened up enormous prospects for psychoanalytical theory and clinical practice. In particular, only the implicit memory develops early and is already present and active in the last weeks of gestation; this is the only form of memory that the neonate has in its first 2 years of life. The procedural and emotive–affective dimension of the implicit memory enables an infant to store its first experiences related to maternal language and the environment in which it is growing. Furthermore, the mother's relationship with the neonate's body, her talking to the baby, looking at it, and touching it–in summary, her *rêverie*–conveys feelings and emotions that will be stored in the neonate's implicit memory.

Following the line of thought of Freud [35], who considered that every event deposited in the memory structures the unconscious of the individual, we can predict a connection between the implicit memory and the unconscious function of the mind in its "nascent state." This early unconscious cannot be the result of repression, in that the structures of the explicit memory indispensable for removal (in particular, the hippocampus) are not mature before 2 years of life [12]. Thus the presymbolic and preverbal experiences that are deposited in the implicit memory are not lost even if they cannot be remembered. They form the pillars of an early nonrepressed unconscious that will condition the affective, emotional and cognitive life of the individual even in adulthood and indeed throughout his or her whole life [36, 37].

It is now possible to connect this new dimension of the unconscious functions of the mind with clinical features. This allows us to investigate thoroughly and refine our way of being with a patient in order to retrieve that nonrepressed unconscious, even without recall, through particular attention to the "musical dimension" of the transference, as it appears in intraverbal communications (Mancia, Chap. 3, this volume and [36]). Like a metaphorical bridge, this dimension of transference connects the emotions experienced during a session with the early emotions of infancy and can act as a vehicle for those feelings that cannot be communicated by narration alone. A specific focus on the reconstructive capacity of dreams will allow the analyst to recover presymbolic and preverbal experiences, through the symbolopoietic work of dreams, so that they can be verbalized and made thinkable. The neuroscientific studies related to memory thus offer the theoretical and practical analyst valuable instruments for reaching the most deeply hidden and oldest areas of the patient's personality, forgotten areas of the unconscious. These areas are nevertheless still working in the person and can resurface in the analytical relationship, forming the essence of an extensive Freudian *Nachträglichkeit*, allowing the unconscious, starting with its nonrepressed function, to be historicized.

Precisely because of the importance that memory has in the unconscious functions of the mind, it is worth referring here to the contributions of Leuzinger-Bohleber and Pfeifer (this volume) who consider (explicit) memory not as a static archive, but rather as a dynamic, interactive function, undergoing continuous "recategorization" incorporated according to the model of consciousness proposed by Edelman [21]. On the basis of the neuroscientific research and the most complex transformational processes that occur in the analytical meeting, the hypothesis I propose here is that the implicit memory, too, stimulated by the transference, can undergo dynamic interactions with the explicit memory and can be "recategorized" during the analytical process. The latter could form the basis for the transformations that follow insight and gaining of awareness, obtained through the constructive and reconstructive work on transference and through the interpretation of dreams. I find it equally interesting to think that the "persecutory and depressive anxieties without apparent cause" described in this book (Cappelli, Chap. 6, this volume), which affect many of our patients, could be explained by early preverbal unconscious traumatic experiences, never represented, never recognized and never thought, deposited in their implicit memory and therefore active parts of their nonrepressed unconscious.

Empathy, Simulation, and Shared Feelings

Neurophysiological and neuropsychological bioimaging studies are generating information that is of value to the understanding of empathy, embodied simulation, imitation, intentionality and the sharing of affective and emotional states–all very topical subjects of great interest to psychoanalysis. Empathy, in particular, has been the subject of numerous experimental and clinical studies in the last few years. In an extensive historical review and clinical analysis, Bolognini [38] described the specific characteristics of empathy in the context of the analytical relation, distinguishing it from shared feelings, fusionality and other unconscious relational modes. On a clinical level this author presented examples in which empathy, or a lack of it, had consequences for the analytical process, although the relationship that empathy has with projective identification did not emerge clearly from his work. This aspect is important in order to interpret experimental data from humans on the sharing of affective states and embodied simulation, which many researchers have been concentrating on in recent years. In particular, it has been observed in human experiments that the same affective areas of pain (anterior areas of the cingulum and of the insula) are activated not only in a subject undergoing a painful physical stimulus but also in an observer who has an affective link with the subject. The subject's distress was communicated to the observer only through a mirror, therefore through an extraverbal route, by expressions of the face and body [39]. The activation of the same affective structures of pain was also observed when the subject received verbal stimuli mimicking a painful experience [40] (Osaka, Chap 10, this volume). These experimental data suggest that the pain affect is mediated by a functional interaction between the anterior part of the cingulate cortex and the prefrontal cortex. Recent bioimaging research (Avenanti & Aglioti, Chap. 9, this volume) has demonstrated neural activity induced in the observer by the sight or imagination of pain experienced by another individual. The activation of neural pain structures (specific sensory areas, anterior cingulum and insula) overlaps widely. This effect, which involves both the affective–emotional and discriminatory pain structures of the observer and the observed, has been described as a form of empathy. The concept of empathy is thus set in relationship with "embodied simulation," which allows the mental state of an observed subject to be "replicated" in the mind of the observer. This state implies that the motor and emotional conditions of an individual can activate corresponding "representations" or neural "configurations" in the nervous system of another person who is observing that individual. The comparison-simulation of the pain of another person enables empathic experiential understanding of the pain of others.

The discovery of mirror neurons [41] (Gallese, Chap. 11, this volume) has brought important contributions to the processuality of neural systems related to a person's own sensorimotor actions and those observed in others. It has been demonstrated that an "intentional attunement" can be reached with other individuals as a result of "embodied simulation." This process allows an experiential insight into the mind of another person with whom there is a relation. The mirror neurons are the neural correlate of this complex mechanism that allows the feeling of empathy and a "shared manifold of intersubjectivity" (Gallese, Chap. 11, this volume). They may also represent the neurophysiological mechanisms of the imitation described primarily in the psychoanalytic context [43].

The extensive data from experiments on mirror neurons of the frontal and parietal cortices of the monkey allow the hypothesis that this “primary sensorimotor configurational” system may be innate, respond to specific stimuli and constitute a neurological function that is indispensable to the survival of the individual and the species. In this context, the recent functional magnetic resonance imaging (fMRI) observations [44] that autistic children lack activity of the mirror neurons in the anterior frontal gyrus (pars opercularis) are extremely interesting. Activity in this area was inversely correlated with the severity of the autistic symptoms, suggesting that dysfunction of the mirror neurons is responsible for the relational deficit observed in autism. This does, however, raise a problem that will not be easy to resolve: is it a defective system of mirror neurons, present since birth for genetic reasons, that causes the autistic syndrome, or is it a traumatic environmental disorder, connected particularly to the failure of a primary relation, that prevents the genetic expression that is essential in order for the system of mirror neurons to function at an early age and for the mental development of the child?

These results do, in any case, provide important neurophysiological correlates of mental processes that are particularly interesting for theoretical and practical psychoanalysis. Anyone who has analytical experience knows that the relation between two individuals who express various types of emotions (including pain) can lead to the passage of the most varied emotions and affects from one to the other. This passage, which has many functions (communicative, defensive, offensive, etc. [45]) was described by Klein in 1946 [46] and defined as projective identification. It consists in the fact that parts of Self dealing with feelings and emotions can be unconsciously split and projected into another person who is identified with the characteristics of these parts. The numerous experiences described above concerning the sharing of affective states or empathy, or what authors call “embodied simulation,” can be seen as the physiological expression of mental states that have strong analogies with the projective identification mentioned previously.

Dreams

In recent years dreaming has been a subject of interdisciplinary interest and a topic of dialogue between the neurosciences and psychoanalysis (Mancia, Chap. 3, this volume and [47]). As is known, dreams were of interest to psychoanalysis before any other scientific discipline took an interest in them [48], and have involved oneiromancers, theologians, poets, artists and philosophers of all times [47]. With the discovery of REM sleep, the doors to dreaming were opened to the neurosciences. Neurophysiological research focused particularly on identifying the mechanisms of sleep and the structures involved, the phases of sleep, and the neurophysiological characteristics defining them. It was psychophysiology that became interested in the mental states that occur during the different phases of sleep. Research in this field has led to a dichotomous model of sleep (REM and non-REM), in which the characteristics of a "biological frame" within which dreaming could occur were attributed only to REM sleep (Mancia, Chap. 3, this volume). On this basis, some authors [49] have denied any psychological significance to dreaming, relegating it to the sphere of biology. However, other psychophysiological research [50, 51] has demonstrated that an oneiric type of mental activity, with hallucinations, emotions and self-representations, can occur during any stage of sleep, from sleep onset to waking. On the basis of these results, Bosinelli [50] hypothesized that a single generator of dreams is activated in sleep, relatively independent of the phase of sleep. However, since dreams in the REM phase are qualitatively different from dreams in the non-REM phase [52], a double generator of dreams, corresponding to the two major phases of sleep (REM and non-REM), has also been hypothesized.

The most recent bioimaging research [53, 54] has suggested a dissociation between dreaming and states of sleep, and has indicated that it is the dopaminergic network that organizes dreaming, thus involving various structures and cerebral relays, in particular the left and right parietal regions, frontoventral areas, the occipitotemporal region and limbic areas. The dopaminergic network would be activated by frontoventral regions that preside over the motivational process. However, the afferent fibers of the parieto-occipital cortex (in particular of the right hemisphere) cause the disappearance of dreams, although sleep architecture remains completely normal [55] (Bassetti et al., Chap. 14, this volume). These observations allow one to consider that the abolition of dreaming due to central lesions that interrupt the dopaminergic circuit can be ascribed to a disconnection syndrome (Mancia, Chap. 3, this volume) [56].

It is obvious that the various neuroscientific approaches to dreaming tell us nothing of the meaning of the dreams or of their role in the economy of the mind. Psychoanalysis is the only discipline that is interested in dreams for what they reveal about the unconscious, and as a function of the mind able to symbolically transform presymbolic experiences and to create images without recollection to fill the gap of the nonrepresentations of the early unrepressed unconscious. Besides, dream can also bring to light, through recall, experiences repressed in infancy (after the age of two) and those deposited in the explicit memory in the course of life (Mancia, Chap. 3, this volume).

Dreaming therefore also has the function, which was originally described by Freud, of bringing to light repressed material, an operation that we could define as derepression. In this regard, I find the experience with voluntary repression [57] extremely interesting. Bioimaging studies show that this repression activates the dorsolateral frontal areas and bilaterally deactivates the hippocampus. These phenomena are exactly the opposite of those occurring during dreaming (in the REM phase), when there is activation of the hippocampus and deactivation of the dorsolateral frontal cortex [53]. The experience of voluntary repression would therefore confirm, at a neurophysiological level, the derepression function of dreaming.

Neuroscientific data on the oneiric activity of the brain contribute to contemporary psychoanalytical thought and in particular to the idea that there is a continuum between unconscious fantasies while awake and the fantasies (oneiric) of dreams. This idea, for which we must thank Bion [58] and Meltzer [59], is fascinating and highlights the irreplaceable role of unconscious fantasies in the mind during wakefulness and dreaming. There are, however, processual neurofunctional differences between these two mental states that neuroscientific research is now beginning to delineate. For example, the brain's processing of information depends on its functional state, intended in a global sense, at that specific moment. It is the functional state that controls the processual strategies that condition the cognitive–emotional content of memory, the storage procedures and the relation with what the subject can remember or forget with dreams (Lehmann and Koukkou, Chap. 14, this volume).

Furthermore, it has recently been demonstrated that stimuli to a cortical area of the human brain have different interhemispheric and intracortical spread depending on whether the subject is a synchronous phase of sleep or awake [60]. There is a clear relationship between these different functional states of the cerebral cortex and the different states of consciousness that characterize wakefulness and sleep. While awake, activation of a limited area of cortex spreads through cortical areas of the ispilateral and contralateral hemispheres, whereas in sleep (in which dreaming occurs) this spread of activation is inhibited and the stimulus remains confined to the area stimulated. This demonstrates a different functional processuality of the neocortical layer and highlights the difference between the two states of consciousness and, presumably, between the contents of the emotional and cognitive processes that characterize the mental states during sleep (dreams) and wakefulness (unconscious fantasies).

Consciousness and the Unconscious

An interdisciplinary dialogue between psychoanalysis and neurosciences cannot ignore consciousness and its different functional levels. Freud dealt with consciousness in relation to the unconscious in 1923 in *The Ego and the Id* [61]. The opening words of the work are: "The division of the psychical into what is conscious and what is unconscious is the fundamental premiss of psycho-analysis; [...] psychoanalysis cannot situate the essence of the psychical in consciousness, but is obliged to regard consciousness as a quality of the psychical, which may be present in addition to other qualities or may be absent" (p. 13). Nevertheless, according to Freud there is an intermediate area between the unconscious and consciousness, a psychic limbo that he called the preconscious. This corresponds to psychic material that is latent, but very close to the conscious: "We restrict the term unconscious,"–clarified Freud–"to the dynamically unconscious repressed." (p. 15).

However, in the same work, Freud, underlining the importance of the unconscious with respect to the consciousness–in the sense that it is this latter that is conditioned by the unconscious–stated that "all our knowledge is invariably bound up with consciousness. We can come to know even the *Ucs.* only by making it conscious [...] consciousness is the surface of the mental apparatus [...] All perceptions which are received from without (sense-perceptions) and from within–what we call sensations and feelings–are *Cs.* from the start" (p. 19).

Already in 1912, in "A Note on the Unconscious in Psycho-analysis" [35], Freud had used the perception–consciousness (P-C) system to indicate consciousness, in order to underline the fact that we are conscious only of those representations of which we have perceptions. Freud wrote: "Now let us call 'conscious' the conception which is present to our consciousness and of which we are aware, and let this be the only meaning of the term 'conscious.' As for latent conceptions, if we have any reason to suppose that they exist in the mind–as we had in the case of memory–let them be denoted by the term 'unconscious'" (p. 260). This description by Freud seems particularly interesting in that it connects, or rather identifies, the unconscious with the memory of latent representations.

Freud devoted much attention in *The Ego and The Id* [61] to the functions of the Ego considered to represent consciousness, without, however, forgetting that this is, in part, also the unconscious. Language (as the representation of words) assumes the role of bringing the representations of unconscious things to the conscious; nevertheless, the fact remains that the Ego behaves in an essentially passive way and is "challenged" by unknown and uncontrollable forces. The Ego is at the service of the Id: "We shall now look upon an individual as a psychical id, unknown and unconscious, upon whose surface rests the ego, developed from its nucleus the *Pcpt.* system" (p. 24). The Ego therefore appeared to Freud as a complex structure with psychic processes linked to consciousness and others to the unconscious (which behave in the way of repression).

This dual nature of the ego enables researchers of childhood to consider the ontogenesis of the human mind as characterized by the formation of consciousness running in parallel with the organization of the unconscious. At the start of life, the development of the infant mind is based on three poles: the baby's desire, connected to its preconceptions and motivations, which encourage it to relate to the maternal object; the internal genetic baggage of the baby, which can influence the baby's relation with its mother and the environment in which it grows; and, finally, the environment, in which the mother's *rêverie* has a central role in conveying feelings and emotions to the neonate through her voice, language, way of dealing with the baby's body and her sensibility (Mancia, Chap. 3, this volume).

In the earliest period of life, the baby starts to create representations that arise from its sensory experiences and from its transformative abilities linked to perceptive transmodality [62]. As a result of these capabilities, giving rise to reflexive relational functions [63], the baby is able to create representations that will form the basis of sensorimotor imitations [64]. Subsequently the first processes of projective and introjective identification begin, which will allow the baby to enrich and transform the first representations and to develop its own symbolic skills of thought organization. This pathway runs in parallel to that described by Klein, who saw the neonate move from a schizoparanoid to a depressive position, and implies a transformation process that evolves from the first affective representations, to reach processes of signification: plastic, iconic and other (in the transitional area of play), up to language development. This transformation process can be dominated by trauma, emotions, fantasies and defenses that can influence and indeed distort it profoundly. Nevertheless, this process forms the basis of the baby's consciousness and identity. At the same time, the emotions, feelings, fantasies and defenses deposited in the implicit memory will make the basic elements of the baby's early unrepressed unconscious. In fact, in this period of life the baby cannot repress painful and traumatic experiences, nor the defenses linked to them, since the structures necessary for the explicit memory (the hippocampus) indispensable for such repression are not yet mature. Rather, the baby uses unconscious modalities characterized by denial and splitting and projective identification of distressed parts of Self. It will be the mother's task to render these harmless and return them to the neonate for introjection.

The ontogenesis of consciousness is therefore accompanied by a parallel process of organization of the unconscious. Consciousness is, therefore, a complex but not autonomous function. It will be conditioned by unconscious fantasies, defenses and emotions that will influence symbolization and the whole transformation process from affective representations to signification right up to language. The experiences described here can influence the first relations of the baby and facilitate what Money-Kyrle [65] called "primary misunderstanding." The results of this misunderstanding will be to distort the unconscious affective representations, which in their turn can influence those perceptive and cognitive activities that constitute the structural elements of consciousness.

It is, therefore, clear that for psychoanalysis consciousness is connected to the world of representations. It is a sort of sensory organ of psychic qualities, linked to language, a function that enables a subject to relate to his own unconscious reality [66].

The neuroscientific approach to consciousness is different. Not being able to focus on the unconscious–given that this so far lacks a specific anatomofunctional reference–the neurosciences have concentrated on the neurophysiological basis of consciousness. Two forms of consciousness have been identified: basic consciousness (or primary consciousness, according to Edelman) and differentiated consciousness (or higher-order consciousness, according to Edelman).

The basic dimension of consciousness is equivalent to alertness and has its roots in the most ancient part of the brain, that is in brainstem systems, in particular in the ascending reticular system [67, 68] that controls the neocortical layer through the nonspecific medial and intralaminar thalamus. In this context Alfred Fessard's hypothesis is interesting. Fessard [69] suggested that the state of consciousness is due to an experienced integration occurring within the reticular formation of the brain stem. This formation is, in fact, organized as a network, integrates various sensory afferents, and organizes a feedback loop that tends to adjust its activity to an optimal working level on which the different states of consciousness, from the most alert state of wakefulness to sleep, depend; it is sensitive to circulating substances and hormones and controls the ascending pathway of the thalamocortical system. The neurons of this system are able to respond to various afferents by modifying their own activity, changing from a continuous rhythm to an oscillating rhythm like an electrophysiological equivalent of the changes in the state of consciousness [68, 70].

The cerebral cortex, with its modulating activity, remains the site of the maximum level of integration. It is conditioned by subcortical reticular and thalamic influences that can desynchronize it (as the electrophysiological equivalent of wakefulness) or synchronize it (as the electrophysiological equivalent of restricting the field of consciousness until sleep is reached).

The differentiated or higher-order consciousness has different characteristics and specific properties such as the ability to analyze, synthesize and recall experiences to the mind, and to integrate them in the structure of the Ego through a coordinated space and time system. Jasper [71] should be acknowledged for having defined the four fundamental characteristics of the consciousness related to (a) one's own identity, (b) the uniqueness of Self, (c) one's own activity, and (d) being in antithesis to the external world and with the other. There are numerous definitions of consciousness: a set of mental processes that allow a subject to be aware of his Self and his environment, his own psychological life and his own potential to integrate the present with the past, to use external and internal perceptions, or to be capable of intentionality [72].

The problems set by the neurosciences are: how can a person become aware of the space and time in which he is living, of his own body and his own identity? How can a person establish a relationship between Self and the world?

Various psychophysiological processes are essential for the organization of consciousness. *Perception* is the cornerstone of this organization, but attention, memory, ideation, critical faculty, judgment, desire, emotions and thought are also fundamental aspects of consciousness, needed by it to integrate the experiences with the sensory world. Humans are capable of self-consciousness when they are able to attribute a sense to their lived experience in which perception, imagination and reality can be integrated [73].

Over the decades, numerous neuroscientists have tried to give a neurophysiological explanation of consciousness, starting with Popper and Eccles [74], who proposed a *dualist interactionism* theory in which association areas of the cortex in the dominant hemisphere (the left) become areas of interface and interaction between the mind and the brain. However, the most interesting recent integration between neurosciences and psychoanalysis on the subject of consciousness comes from the work of various authors. Edelman [75] states that the awareness of Self is linked to activity of areas of the cerebral cortex where information from the outside and within the body is processed, selected and relayed to other cortical areas. His *theory of neuronal group selection* is based on the potential that groups of cortical neurons have of selecting certain response patterns in reaction to given stimuli, patterns that can constitute *cerebral maps.* These maps interact reciprocally through a "reentry" process, enabling the different cerebral areas to coordinate their activity in order to produce new and ever more complex functions (such as memory, symbolization and thought). Thanks to this organization into maps, the brain can carry out perceptive classification. This theory of neuronal group selection also explains a baby's ability to organize a symbolic area prior to a syntactic area and thus for this former to be the central element of the organization of higher-order consciousness.

According to Daniel Dennett [76], the brain is able to redesign itself, creating the basis for learning and memory. The functions of consciousness are thus linked to the plasticity of the brain that has allowed the development of language from *Homo habilis* to us [77]. In fact, starting in *Homo habilis,* there has been a complex organization of software that has influenced the hardware of the brain up to the point that these changes are transferred in the human genome (this is the essence of what Dennett called the "Baldwin effect").

Damasio [78] considers that the mind is a set of *representations or neural configurations* that become images and that can be manipulated to become forms of higher consciousness. All this occurs through integration of processes that involve the association areas and the lower-order sensory areas in such a way that the perceptions reaching them can be organized into concepts and categorized. The categorization of experiences in the prefrontal cortex is thought to be responsible for representations: consciousness forms part of the mind's functions and is the fruit of experiences that are processed, selected, memorized and historicized in our brain.

More recently, the thalamoreticular system has been considered as responsible for the essential properties of conscious experience, given its control of cortical modules [79]. According to these authors, in order for the conscious to emerge, different groups of neurons in the thalamocortical system must be involved in the reentry loops that are differentiated from each other over time. The reentry phenomenon is the specific mechanism that ensures the integration of information in the thalamocortical system. These groups of neurons undergo various selections during development as a result of experiences that, through various reentries, create new synapses and participate in neuronal plasticity.

A fundamental property of conscious experience is based on the capacity of the brain and its components to deal with differentiation and multiplicity while conserving its own uniqueness and coherence. Edelmann and Tononi [79] have the merit of having proposed a hypothesis that tackles, although it does not resolve, the old issue of how an objective experience, such as observations in the field of neurosciences, can explain the subjective experience that underpins the psychoanalytical relationship. This is the problem of *qualia,* intended as the specific qualities of the subjective experience. Psychoanalysis is obviously interested in *qualia* since they are subjective experiences. However, these also interest neurosciences because *qualia,* like other experiences, may be explained by the activity of groups of neurons that, through reentry processes, form an extremely complex nucleus that is the basis for subjective experience. The discrimination of neuronal activity in the neonatal, or even in the fetal, brain can derive from proprioceptive, kinesthetic and autonomous systems. These functions predominantly involve the oldest part of the brain, that is, the brain stem (in particular the pons), where cholinergic neurons develop much earlier than other systems [80]. The *qualia* that emerge from the neuronal activity of the brain stem and that can be attributed to this early discrimination, could–according to these authors–be establishing the most primitive Self.

At this point there is an extremely interesting convergence between this neuroscientific hypothesis and that emerging from studies of intrauterine life. In particular, using ultrasonography alternate *clusters* of general and partial fetal movements and of respiratory movements have been seen already from the 13th week of gestation (Piontelli, Chap. 15, this volume). The presence of other behavioral features has allowed Piontelli to hypothesize that the pontine neurons that develop early in the brain stem coordinate motor and respiratory activity by means of an alternating mechanism caused by the effect of inhibitory interneurons, and thus give rise to the start of active sleep [81]. The relevance of the neuronal activity for the *qualia* is clear as it forms the basis for a protomental organization of the fetus that could start during active sleep. The same discussion may be valid for the early mother–child interaction [37, 62, 82], in which the sensory and affective experiences that involve these brainstem structures, as foundations of the *qualia,* can constitute the first roots of a state of consciousness and of the nonrepressed unconscious.

Antenatal and Neonatal Life

Finally, I would like to make some remarks drawing on research on the origins of human mental activity starting from intrauterine life and extending into the first years of childhood.

There has been strong interest in fetal psychic life for many years among the international psychoanalytical community [83]. Particular interest has been shown in the different physiological components of the fetus: sensorimotor activity, general behavior, and integrative functions, especially related to the various states of sleep and wakefulness [81]. The early behavior of the fetus, from the 10th to the 20th week of gestation, is particularly interesting (Piontelli, Chap. 15, this volume). Early functions have been observed, such as generalized movements, respiratory movements, clonus and hiccups, as well as other movements and behaviors. It has been observed that groups of movements (generalized and respiratory) are organized in *alternate clusters*. Given that the neuronal structures of the pons are the earliest to be organized [12, 80], it can be hypothesized that these preside over the ascending and descending motor and synaptogenic functions. The alternation of the *clusters* provides interesting evidence in favor of the hypothesis that maturation of some inhibitory interneuronal circuits in the brain stem act as switches that regulate motor and respiratory organization. Early activity of the pons is, therefore, an important issue for psychoanalysis, in the sense that it can represent the basis of an early start of active sleep in the fetus analogous (in some aspects) to REM sleep in the mature brain [84].

At birth and with the start of its first relationships, the neonate has an early and intense capacity to learn, based above all on the emotions and feelings that trigger these relationships. Already during gestation, the fetus perceives some maternal biological rhythms such as heart rate and breathing frequency. Furthermore, it hears the *maternal voice* and its intonations that convey specific affective and emotional states [85]. These experiences can be memorized [86]. On this basis, the fetus starts its first relationship with its mother, which will develop at birth with progressive development of language. A mother’s voice is able to influence the heart and sucking rate of the neonate [87]. The neonate’s sensitivity to maternal language and its surrounding environment allows it to learn its prosody very early and, starting from the 6th month of life, to represent sequential intonations relative to vowels and consonants of the same language.

Recent research [88] has shown that the perception of language and grammatical knowledge develop in the neonate within the first month of life. The linguistic elements that are organized earliest are the auditory–phonological ones, followed by lexical–semantic features. The former are linked to a process involving the superior posterior temporal gyrus (Brodman's area 22), while the latter involve the temporoparietal regions (angular and supramarginal gyri, Brodman's areas 39 and 40) of the left hemisphere. Grammatical and syntactic processes play a critical role in the selective integration of lexicosemantic information and are located in the inferior frontal gyrus of the left hemisphere, which includes the opercular and triangular areas (areas 44 and 45) and the frontal lateral premotor areas (areas 6, 8 and 9) on the left. These areas constitute what is defined as the "grammatical center" of language [88]. This center is activated in response to any language a baby is exposed to, confirming the universal nature of the grammatical processes already proposed by Chomsky [89]. However, it should be remembered that the baby responds with generalized motor activity to adult language (heterosynchronic) and to its own lallation (autosynchronic) [90]. This means that all the sensorimotor areas participate in the beginning of language development, and only subsequently do the various grammatical, syntactic and semantic centers of language become organized. This is the period in which the linguistic functions become concentrated in the different areas of the left hemisphere.

Besides voice and speech, visual contact is extremely important in the early relationship of a baby with its mother. Already during the first year of life, the neonate quickly learns that the gaze of others contains significant information [91]. This information is essentially of an affective nature and produces intense emotions in the neonate. The body is another element in the relationship between a baby and its mother. The way in which the mother holds a baby, touches it, looks at it and speaks to it, the level of her *rêverie* are important aspects in the relationship since they transmit feelings and emotions that the neonate will deposit and store in its implicit memory. These deposits could be defined, with a metaphor taken from genetics, as the *psychological DNA* that will characterize the individual's personality for the rest of his life.

Lehtonen's group (Chap. 16, this volume) has made a significant contribution to the understanding of these first neonatal experiences, by expanding psychophysiological and neurophysiological knowledge of the events accompanying breastfeeding and the early organization of the conscious and unconscious mind of the neonate. In the first period of life, the neonate's interaction with its mother is based on a communicative play involving the body. This bodily interaction between the neonate and mother reaches its peak during breastfeeding, which produces an intense exchange during sucking and other skin-to-skin contacts that satisfy the vital instinctive needs of the baby. The “Body-Ego,” which can be considered as a primordial *matrix-like structure,* is organized on this interaction.

During breastfeeding there is a variation in the amplitude of EEG activity in the posterior cortex of the right hemisphere of the neonate. This phenomenon is particularly marked in hungry neonates, whereas it is not present during peaceful sucking. The intense need for food together with the sensory stimulus of breastfeeding can, therefore, modify the cortical activity of the neonate. This change is no longer present at 3 months of age. However, at 6 months of life theta activity appears on the EEG during breastfeeding. This reinforcing effect of theta waves shows that the connections between the subcortical structures and the cortical areas have matured, such as to create the basis for an “organized neural network” that enables the cortex to record sensory events associated with breastfeeding and activates subcortical centers that regulate the feeding behavior of the baby.

Theta activity has been seen at 9 months of life [92] not only when the neonate is at the breast, but also when it is observing pleasant images; as a result, this activity has been defined “hedonic theta” activity. It has been hypothesized that theta rhythm can act as an electrical equivalent of *early affective experiences* (pleasant and unpleasant). It is interesting to remember that theta activity is also present during dreaming in the REM phase in adults. The interest of these observations lies in the fact that the mother–baby interaction, with its neurophysiological components, can be connected to those processes that lead to the formation of the implicit memory in the first years of life.

The neonate’s relationship with its body, derived from the interaction with the mother, is essential to the formation of the image of the body and its functions in mental space [93]. The “Body-Ego” should, therefore, be considered as an organization of the first *protomental image of Self.* Sucking at the breast is an important element in the early mental organization of the Body-Ego.

Psychoanalytical interest in these varied and complex happenings in antenatal and perinatal life is triggered by the fact that the human mind is organized very early and that various types of trauma affecting a fetus can influence the development of its brain and its early conscious and unconscious mental functions, thus conditioning the way in which the neonate can relate to its mother and the environment in which it grows once it is born. All this has enormous importance for the practice and theory of psychoanalysis [94]. The feelings and the emotions that the maternal voice has conveyed to the fetus and the neonate, like the attention and care given to its body, can reappear in transference and be represented symbolically in the dreams of our patients. This adds value to the research on fetal and neonatal life, from *infant observation* [95–98] to studies on bonding [99], reflex functions [100], formation of the earliest Self [62] and the organization of conscious and unconscious functions of the human mind.

References

1. Kaplan-Solms K, Solms M (2000) Clinical studies in neuro-psychoanalysis. International Universities Press, London
2. Solms M, Turnbull O (2002) The brain and the inner world. Other Press, New York
3. Scalzone F (2005) Notes for a dialogue between psychoanalysis and neuroscience. Int J Psychoanal 86:1405–1423
4. Darwin C (1965) The expression of the emotions in man and animals. University of Chicago Press, Chicago (original publication 1872)
5. LeDoux JE (1996) The emotional brain. Simon and Schuster, New York
6. Squire LR (1994) Declarative and nondeclarative memory: multiple brain system supporting learning and memory. In: Schacter DL, Tulvin E (eds) The memory systems. MIT Press, Cambridge MA, pp 203–232
7. Schacter DL (1996) Searching for memory. The brain, the mind, and the past. Basic Books, New York
8. Lashley K (1950) The problem of serial order in behavior. In: Jeffers LA (ed) Cerebral mechanisms in behavior. Wiley, New York
9. Phelps EA (2004) Human emotion and memory: interactions of the amygdala and hippocampal complex. Curr Opin Neurobiol 14:198–202
10. Sacchetti B, Scelfo B, et al (2004) Long-term synaptic changes induced in the cerebellar cortex by fear conditioning. Neuron 42:973–982
11. Jacobs WJ, Nadel L (1985) Stress-induced recovery of fears and phobias. Psychol Rev 92: 512–531
12. Siegel SJ (1999) The developing mind: toward a neurobiology of interpersonal experience. Guilford, New York
13. Karten YJC, Olariu A, Cameron NA (2005) Stress in early life inhibits neurogenesis in adulthood. Trends Neurosci 28:171–172
14. LeDoux JE (2000) Emotion circuits in the brain. Annu Rev Neurosci 23:155–184
15. Damasio AR (2000) The feeling of what happens: body and emotion in the making of consciousness. Harcourt Brace, New York
16. Damasio AR (2003) Looking for Spinoza. Joy, sorrow, and the feeling brain. Harcourt, New York
17. Gainotti G (2001) Components and levels of emotion disrupted in patients with unilateral brain damage. In: Gainotti G (ed) Emotional behavior and its disorders. Boller F, Grafman J (eds) Handbook of neuropsychology, vol 5, 2nd edn. Elsevier, Amsterdam, pp 161–180
18. Adolphs R, Damasio H, et al (2000) A role for somatosensory cortices in the visual recognition of emotion as revealed by three-dimensional lesion mapping. J Neurosci 20:2683–2690

19. Iacoboni M, Woods RP, et al (1999) Cortical mechanisms of human imitation. Science 286:2526–2528
20. Panksepp J (2001) The long-term psychobiological consequences of infant emotions: prescriptions for the twenty-first century. Neuropsychoanalysis 2:149–178
21. Edelman GM (1987) Neural darwinism: the theory of neuronal group selection. Basic Books, New York
22. Joseph R (1996) Neuropsychiatry, neuropsychology and clinical neuroscience. Williams and Wilkins, Baltimore
23. Paré D, Collins DR, Pelletier G (2002) Amygdala oscillations and the consolidation of emotional memories. Trends Cogn Sci 7:306–314
24. McGaugh JL (2002) Memory consolidation and the amygdala: a systems perspective. Trends Neurosci 9:456–461
25. McEwen BS, Sapolsky RM (1995) Stress and cognitive function. Curr Opin Neurobiol 5:205–216
26. Leonard CM, Rolls ET, et al (1985) Neurons in the amygdala of the monkey with responses selective for faces. Behav Brain Res 15:159–176
27. Holland PC, Gallagher M (1999) Amygdala circuitry in attentional and representational processes. Trends Cogn Sci 3:65–73
28. Cahill L, Weinberger N, et al (1999) Is the amygdala a locus of "conditioned fear"? Some questions and caveats. Neuron 23:227–228
29. Packard M, Teather L (1998) Amygdala modulation of multiple memory systems: hippocampus and caudate-putamen. Neurobiol Learn Mem 69:163–203
30. Dolan RJ, Lane R, et al (2000) Dissociable temporal lobe activations during emotional episodic memory retrieval. Neuroimage 11:203–209
31. Sharot T, Delgado MR, Phelps EA (2004) How emotion enhances the feeling of remembering. Nat Neurosci 7:1376–1380
32. Warrington EK, Weiskrantz L (1974) The effect of prior learning on subsequent retention in amnesic patients. Neuropsychologia 12:419–428
33. Mancia M (2000) Sulle molte dimensioni della memoria: neuroscienze e psicoanalisi a confronto. Psiche 2:181–192
34. Semenza C (2001) Types of memory in psychoanalysis. Congress on "Neuroscientific and Psychoanalytic Perspectives on Memory," New York, 20–22 April 2001
35. Freud S (1912) A note on the unconscious in psycho-analysis. SE 12
36. Mancia M (2003) Dream actors in the theatre of memory: their role in the psychoanalytic process. Int J Psychoanal 84: 945–952
37. Mancia M (2004a) Sentire le parole. Archivi sonori della memoria implicita e musicalità del transfert. Bollati Boringhieri, Torino (Feeling the words: resonant archives of the implicit memory and musicality of the transference. Hove: Routledge, in press)
38. Bolognini S (2002) L'empatia psicoanalitica. Bollati Boringhieri, Torino
39. Singer T, Seymour B, et al (2004) Empathy for pain involves the affective but not sensory components of pain. Science 303:1157–1164
40. Osaka N, Osaka M, et al (2004) A word expressing affective pain activates the anterior cingulate cortex in the human brain: An fMRI study. Behav Brain Res 153: 123–127
41. Rizzolatti G, Craighero L (2004) The mirror neuron system. Ann Rev Neurosci 27:169–192
42. Gallese V (2001) The "shared manifold" hypothesis: from mirror neurons to empathy. J Conscious Stud 8(5–7):33–50
43. Gaddini E (1969) On imitation. Int J Psychoanal 50:475–484
44. Dapretto M, Davies MS, Pfeifer JH (2005) Understanding emotions in others: mirror neuron dysfunction in children with autism spectrum disorders. Nature Neurosci,

online publication 4 December 2005; print publication 2006, 9:28–30
45. Rosenfeld HA (1987) Impasse and interpretation. Tavistock, London New York
46. Klein M (1975) Notes on some schizoid mechanisms. In: Envy and gratitude and other works (1946–1963). Hogarth Press, London, 1946, pp 1–24
47. Mancia M (2004b) Il sogno e la sua storia. Marsilio, Venezia
48. Freud S (1900) The interpretation of dreams. SE 4–5
49. Hobson JA, Pace-Schott EF, Stickgold R (2003) Dreaming and the brain: toward a cognitive neuroscience of conscious states. In: Pace-Schott EF, Solms M, Blagrove M, Harnad S (Eds) Sleep and dreaming. Scientific advances and reconsiderations. Cambridge University Press, Cambridge, pp 1–50
50. Bosinelli M, Cicogna P (eds) (1984) Psychology of dreaming. Clueb, Bologna
51. Foulkes D (1985) Dreaming: a cognitive-psychological approach. Erlbaum, Hillsdale NJ
52. Nielsen TA (2003) A review of mentation in REM and NREM sleep: “Covert” REM sleep as a possible reconciliation of two opposing models. In: Pace-Schott EF, Solms M, et al (eds) Sleep and dreaming. Scientific advances and reconsiderations. Cambridge University Press, Cambridge, pp 59–74
53. Maquet P, Péters J-M, et al (1996) Functional neuroanatomy of human rapid-eye-movement sleep and dreaming. Nature 383:163–166
54. Solms M (2003) Dreaming and REM sleep are controlled by different brain mechanisms. In: Pace-Schott EF, Solms M, et al (eds) Sleep and dreaming. Scientific advances and reconsiderations. Cambridge University Press, Cambridge, pp 51–58
55. Bischof M, Bassetti CL (2004) Total dream loss: a distinct neuropsychological dysfunction after bilateral PCA stroke. Ann Neurol 56: 583–586
56. Geshwind N (1965) Disconnection syndromes in animals and man. Brain 88:237–274 and 585–644
57. Anderson M Ochsner KN et al (2004) Neural systems underlying the suppression of unwanted memories. Science 303:232–237
58. Bion WR (1962) Learning from experiences. Heinemann, London
59. Meltzer D (1984) Dream-life. A re-examination of the psycho-analytical theory and technique. Clunie Press, Perthshire
60. Massimini M, Ferrarelli F et al (2005) Breakdown of cortical effective connectivity during sleep. Science 309:2228–2232
61. Freud S (1923) The ego and the id. SE 19
62. Stern DN (1985) The interpersonal world of the infant. Basic Books, New York
63. Fonagy P, Target M (1997) Attachment and reflective function: their role in self-organization. Dev Psychopathol 9: 679–700
64. Mancia M (1996) Imitazione, rappresentazione, identificazione: loro ruolo nello sviluppo e nel transfert. Riv Psicoanal 42: 225–247
65. Money-Kyrle R (1978) The collected papers of Roger Money-Kyrle. Roland Harris Educational Trust/Clunie Press, Perthshire
66. Semi AA (2003) La coscienza in psicoanalisi. Cortina, Milano
67. Moruzzi G (1972) The sleep-waking cycle. Ergeb Physiol 64:1–165
68. Moruzzi G, Magoun HW (1949) Brain stem reticular formation and activation of the EEG. Electroencephalogr Clin Neurophysiol 1:455–473
69. Fessard A (1954) Mechanisms of nervous integration and conscious experience. In: Delafresnaye JF (ed) Brain mechanisms and conscioussnes. Charles Thomas, Springfield IL
70. Mancia M (1982) Some physiological properties of the ascending brain stem reticular system. Arch Ital Biol 120:72–94

71. Jasper K (1964) Psicopatologia generale. Il Pensiero Scientifico, Roma
72. Mancia M (1998) Coscienza sogno memoria. Borla, Roma
73. Ey H (1954) Etudes psychiatriques. Desclée de Brower, Paris
74. Popper K Eccles JC (1977) The self and its brain. Springer, New York
75. Edelman GM (1992). Bright air, brilliant fire. On the matter of the mind. Basic Books, New York
76. Dennet DC (1991) Consciousness explained. Little Brown, Boston
77. Tobias PV (1988) Olduvai gorge, vols 4A and 4B: The skulls, endocast and teeth of Homo habilis. Cambridge University Press, Cambridge
78. Damasio AR (1994) Descartes' error: emotion, reason and the human brain. Groaaet/Putnam, New York
79. Edelman GM, Tononi G (2000). Un universo di coscienza. Come la materia diventa immaginazione. Einaudi, Torino
80. Joseph R (1999) Environmental influences on neural plasticity, the limbic system, emotional development and attachment: a review. Child Psychiatry Hum Dev 29:189–208
81. Mancia M (1981) On the beginning of mental life in the foetus. Int J Psychoanal 62:351–357
82. Mancia M (1994) From Oedipus to dream. Karnac Books, London
83. Rascovski A (1977) El psiquismo fetal. Paidos, Buenos Aires
84. Mancia M (1976) Sommeil, rêve et instincts: une approche interdisciplinaire. Rev Fr Psychanal 1:31–58
85. Kolata G (1984) Studying learning in the womb. Science 225:302–303
86. De Casper AJ, Fifer WP (1980) Of human bonding: newborns prefer their mothers' voices. Science 208:1174–1176
87. Mehler J, Bertoncini J, et al (1978) Infant recognition of mother's voice. Perception 7:491–497
88. Sakai KL (2005) Language acquisition and brain development. Science 310:815–819
89. Chomsky N (2002) On nature and language. Cambridge University Press, Cambridge
90. Condon WS, Sander LW (1974) Neonate movement is synchronized with adult speech: interactional participation and language acquisition. Science 183:99–101
91. Farroni T, Gergely C, et al (2002) Eye contact detection in humans from birth. Proc Natl Acad Sci USA 99:9602–9605
92. Maulsby RL (1971) An illustration of emotionally evoked theta rhythm in infancy. Hedonic hypersynchrony. Electroencephalogr Clin Neurophysiol 31:157–65
93. Gaddini E (1981) Notes on the mind-body question. Int J Psychoanal 68:315–329
94. Bergeret J, Soulé M, et al (2006) Anthologie du foetus. Abords Pluridisciplinaires. Dunod, Paris
95. Bick E (1968) The experience of the skin in early object-relations. Int J Psychoanal 45:558–566
96. Harris M (1975) Some notes on maternal containment in "good enough" mothering. J Child Psychother 4:35–51
97. Vallino D, Macciò M (2004) Essere neonati. Borla, Roma
98. Piontelli A (1986) Backwords in time: a study in infant observation by the method of Esther Bick. Clunie Press, Perthshire
99. Bowlby J (1969) Attachment and loss. Hogarth Press, London
100. Fonagy P (1999) Memory and therapeutic action. Int J Psychoanal 80:215–223

PART 1
Memories and Emotions

Chapter 1

Cooperation not Incorporation: Psychoanalysis and Neuroscience

GILBERT PUGH

In this essay I support the view that psychoanalysis and neuroscience[1] are two quite distinct disciplines which increasingly have more to offer each other in collaboration, but I strenuously reject the views that either neuroscientific advances will render psychoanalysis superfluous, or that such advances will not make further major contributions to mental health, particularly in the field of psychoactive medication.

As an introduction to the topic I provide four snapshot views about this interrelationship by well-known analysts and neuroscientists, and then argue that Freud's psychoanalysis did *not* evolve as a consequence of his failure to substantiate his theories scientifically. This carries the implication that the way forward for psychoanalysis is not necessarily the imposition onto it of more and more "hard science," but the sharing of new information which aims at the clarification of mind–body issues that seem to require more than one approach for fuller understanding.

Following this introduction, I shall turn to a discussion of memory, a topic that pre-occupied Freud early in this career and where joint appraisal by neuroscientists and analysts has been so fruitful in recent years. I offer a caveat about the dangers of transcribing ideas from neuroscience to psychoanalysis beyond their usefulness. I end with two clinical vignettes which illustrate some of the ideas contained in this essay.

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[1] In this essay, the term "neuroscience" refers to all neurological sciences.

Some Views on the Collaboration between Neuroscience and Psychoanalysis

Professor Jaak Panksepp (behavioral neuroscientist); Professor Mark Solms (neuroscientist and psychoanalyst) [1]. Both agree that neuroscience and psychoanalysis in collaboration are not in the business of proving Freud right or wrong but of simply finishing the job that he began more than a hundred years ago.

Arnold Goldberg (psychoanalyst) [2]: "There is no gap between neuroscience and psychoanalysis. They are separate worlds".

Mortimer Ostow (neurologist, psychiatrist and psychoanalyst) [3]: "It may be that psychoanalysis will survive only as a component of neuropsychoanalysis and as a component of general psychiatry . . . But I don't think it [psychoanal] will remain as a separate discipline."

Andre Green (psychoanalyst) [4]: "The relation between neurology and psychical activity is not direct. The only suggestion I have for expanding the debate between psychoanalysis and neuropsychology is the creation of discussion groups wherein psychoanalysts report a series of sessions and ask scientists for their interpretation . . . . One must admit that a Shakespearean quotation can be more enlightening for a psychoanalyst than a ton of scientific literature!"

Science and beyond...

As we think we live. This is why the assemblage of philosophical ideas is more than a specialist study. It moulds our type of civilization.

A.N. Whitehead [5]

Freud's letters to Fliess during the 1890s reveal a young neurologist struggling to develop a clinical theory of the neuroses and psychoses with the central theme that symptoms resulted from the repression of unpleasant sexual affects whether based on actual events or on phantasies. He gradually lost confidence in his "seduction theory," not because he ever denied that childhood seduction was emotionally damaging but because the evidence for it often derived from fallible memory. Freud put it his way: ". . . one cannot distinguish between truth and fiction that has been cathected with affect" [6].

He tried another approach "with a determined effort to examine what shape the theory of mental functioning takes if one introduces quantitative considerations, a sort of economics of nerve forces" [6]. This refers of course to his *Project for a Scientific Psychology* [7], which he abandoned after much frenetic effort with the words to Fliess: "to me it appears to have been a kind of madness" [6]. However, as Kanzer observed, "the project is a set of neurologically clad psychological propositions drawn from clinical observation" [8]. And Mancia [8a] pointed out that: "the language of the *Project* is only apparently physiological. Substantially it is a metaphorical language...". Solms and Saling endorse this view, pointing out that Freud retained many of the ideas in the *Project* but "none of these was based on the neurophysiology or neuroanatomy of the day" [9]. Of greater significance for the future of psychoanalysis was that, by 1893, Freud had rejected first Meynert's and then Charcot's views on the cortical localization of psychological pathology and had adopted John Hughlings Jackson's approach, which emphasized that complex mental processes are best understood by not trying to isolate them in specific areas in the brain. This view, which Freud had championed in *On Aphasia,* enabled him to regard the biological aspects of mental functioning and the psychological as separate but interrelated [9–11]. However, almost 40 years later he was, if anything, more pessimistic about understanding this interrelationship:

> Everything that lies between [the brain and our acts of consciousness] is unknown to us . . . . If it [knowledge of this interrelationship] existed it would at the most afford an exact localisation of the processes of consciousness and would give us no help towards understanding them [12].

Fifty-five years later the philosopher Chalmers [13] called this the "hard problem." Toulmin [14], quoted by Ulrike May [15], has pointed out that great investigators often have a "vision" early on in their lives and then devote the rest of their lives to its verification. In Freud's case it is this vision which dominates the 1890s. He is driven to understand psychic functioning using any intellectual discipline that serves this purpose, but underscored by the conviction that unconscious processes and human sexuality and its repression were essential ingredients in this endeavor. He writes to Fliess in January 1896:

> I see, via the detour of medical practice, you are reaching your first ideal of understanding human beings as a physiologist just as I most secretly nourish the hope of arriving, via these same paths, at my initial goal of philosophy. For that is what I wanted originally, when it was not yet at all clear to me to what end I was in the world (p. 159 in [6]).

Three months later he repeats the same *cri de coeur:*

If both of us are still granted a few more years for quiet work, we shall certainly leave behind something that can justify our existence. Knowing this, I feel strong in the face of all daily cares and worries. As a young man I knew no longing other than for philosophical knowledge, and now I am about to fulfil it as I move from medicine to psychology. (p 180 in [6])

The impact of these two quotations is not fully felt until it is realized that Freud uses his own idiosyncratic version of the terms "psychology" and "philosophy." Ulrike May [15] explains that psychology for Freud at this time "means an overarching explanatory theory forming a more abstract frame of reference in which the clinical findings can be accommodated and whereby they can be systematically linked to each other. As we know, this part of the theory was called metapsychology from February 1896 on."

Philosophy on the other hand was for Freud a global overview of medicine, neurology, and psychology embracing brain/mind problems, and the hard problem of consciousness. It is certainly true that not enough "hard science" was available to Freud at the turn of the century for him to attempt to quantify his psychological findings to his own satisfaction–and indeed he described his own attempt to do so as madness–but it is questionable whether Freud ever really wanted his psychoanalysis to become what we now call neuroscience. Several modern commentators have written as if it were axiomatic that Freud would have liked his metapsychology to be neuroscience and that therefore his *Project* was a failure.

Freud was a conquistador. He was interested in neurology and hormones, psychology and psychosis, and he wanted to see how it all fitted together to make a philosophy. "Philosophical theories and systems," he wrote, "have been the work of a small number of men of striking individuality. In no other science does the personality of the scientific worker play anything like so large a part as in philosophy" [16].

In the 1890s Freud first read Theodor Lipps, a German philosopher in Munich, who held the view that the "psychical is in itself unconscious and that the unconscious is the truly psychical" (p. 286 in [17]). This was an unusual view for a philosopher to take at that time, the more usual view being that for something psychical to be unconscious is self-contradictory: what is unconscious cannot be mental–and indeed Freud was a little nonplussed when he discovered how close Lipps's views were to his own. "I found the substance of my insights stated quite clearly in Lipps, perhaps rather more so than I would like." He adds: "The seeker often finds more than he wished to find" (p. 62, note 5, in [6]).

But the philosopher that Freud found most remarkable was Schopenhauer. He wrote about him: “ not only did he assert the dominance of the emotions and the supreme importance of sexuality but he was even aware of the mechanisms of repression” [18]. Freud claimed to have read Schopenhauer very late in his life [18]. However, there are three references to him in bibliography A in *The Interpretation of Dreams* [19], so it is not possible that he was being disingenuous, merely an innocent victim of priming [20]!

Nietzsche was another philosopher who interested Freud in the 1890s, and Freud tells Fliess that he bought his works in February 1900 (p. 398 in [6]). Freud wrote of Nietzsche that he was a “philosopher whose guesses and intuitions often agree in the most astonishing way with the laborious findings of psychoanalysis, was for a long time avoided by me on that very account; I was less concerned with the question of priority than with keeping my mind unembarrassed” [18].

The Interpretation of Dreams

Freud’s struggle to complete *The Interpretation of Dreams*–it took more than 4 years–reflects the struggle that neuroscientists and psychoanalysts have today, not so much because they disagree on the facts but more because there is a gulf between the poet, the artist and philosopher, and the empirical scientist and mathematician. The latter expect to find “the truth”–or a version of it; the former know there is never an answer to the question “Is it true or is it false?” An example of this might be a most ingenious study carried out by Fabiani et al. [21] that collected event-related potentials in an attempt to distinguish true from false or constructed memories. They found that true memories left a sensory signature whilst false ones did not. Here was some evidence of an engram or memory trace, but the type of memory tested was semantic, declarative memory and therefore had no autobiographical content and no emotional content. This “sensory signature” has an in vitro quality about it. It could be useful in testing the integrity of a neuronal pathway but at present is of very little interest to psychoanalysts because it doesn’t take into account the perceptual and recall distortions of “real life” memory.

Freud tried to be both philosopher and scientist, and how he suffered! He had little trouble with the “art” and the dreams; it was the last chapter, Chapter 7, the *Psychology,* which tormented him. By June 1898 his distress was obvious, "with the continuation of the dream [book] something is amiss . . . For it is wretchedly difficult to set out the new psychology in so far as it pertains to the dream . . . So I am stuck at the *relationship of the two systems of thinking* (my italics); I must deal with them in earnest. For a while I again shall be of no use to anyone. The tension of uncertainty makes for an infamously unpleasant state, which one feels almost physically" (p. 315 in [6]).

By May 1899 he had decided to publish, though doubts remained as to whether his reach had exceeded his grasp. In confirmation, he writes the well-known funny story to Fliess about the husband and wife trying to decide whether to kill a cock or a hen for their holiday celebrations. If either dies the other will pine. After the rabbi suggests that the hen should be killed, he is told that the cock will pine. The rabbi's reply: "So let him pine! (p. 353). Exactly right or not, Freud would publish.

He had made his decision, but even after he had sent Fliess the first proof page to acknowledge Fliess's "share in the dream [book]," he writes: "A strange feeling, in the case of such a child of sorrow! I [still] have great difficulties with it; I can not manage more than two hours a day without calling on Friend Marsala for help. 'He' deludes me into thinking that things are not really so bleak as they appear to be when sober" (p. 359 in [6]).

Fliess as organic advisor could come up with nothing to help him; it was, however, Fliess's gift of a case of fine Marsala that consoled him!

The first copy of *The Interpretation of Dreams* was in Fliess's hands on 27 October 1899. In his famous Chapter 7, Freud makes it clear that he is using models, analogies, and metaphors to illustrate his mental apparatus. "I shall entirely disregard the fact that the mental apparatus with which we are here concerned is also known in the form of an anatomical preparation . . ." (p. 536 in [19]) and yet he uses what appear to be neurological terms in his descriptions. A closer look reveals that he is using the vernacular version of the term and not the precise neurological meaning. The term "innervation," which means the nerve supply to an area of the body, is here used by Freud to mean the transmission of energy. Similarly, his *apparatus* has a sensory and motor end, but by this Freud means that it has direction. The term *reflex* (p. 538) is not here used in the sense of an involuntary action but indicates that the *apparatus* is reversible, and the reference to "degrees of conductive resistance" (p. 539) hints at important ideas from the *Project*. It is as if the philosopher is disassociating himself from *science* but asking the reader to keep half an eye open for some correspondence between the two.

It is the author's view that the presentation of memory in this chapter is no more than a schematic version of memory which serves the purpose of illustrating the processes of progression and regression in dreams but does not represent either a psychoanalytical theory of memory or even the extent and variety of Freud's ideas at this time. For example, in Freud's 1899 paper "Screen Memories," he closes with the statement: "It may indeed be questioned whether we have any memories at all *from* our childhood: memories *relating to* our childhood may be all that we possess" [22]. An extraordinary statement that anticipates one of the core complexities of memory theory 80 years later.

Anatomy, physiology, neurology, and psychology–somehow there was no comfortable or overall fit: Freud's vision transcended them all. His was the vision of the poet, the artist, and the philosopher. In a moving passage in a letter to Fliess while he was struggling with his Chapter 7, he wrote: "Somewhere inside me there is a feeling for form, an appreciation of beauty as a kind of perfection: and the tortuous sentences of my dream book, with the parading of indirect phrases and squinting at ideas deeply offended one of my ideals" (p. 374 in [6]).

Freud's vision was to search for the truth about mental functioning, but he wanted to position his insights into the right category of realization. At all costs he was trying to avoid the category mistake of trying to locate within the brain events and processes that belonged to the mind.

By 1924, disillusioned by the lack of interest in psychoanalysis by philosophy, medicine, and by the "cultural hypocrisy" [23] of society, Freud had lowered his sights somewhat: "So it comes about that psychoanalysis derives nothing but disadvantages from its middle position between medicine and philosophy" (p. 217 in [23]).

Memory

I want now to turn to the topic of memory, and more particularly to nonconsciously processed memory, a field in which cooperation between the neurosciences and psychoanalysis had stimulated so many theories and ideas particularly in the "nonverbal" realms of psychoanalysis [24–26].

Though it can not be said that the neurosciences "discovered" unconscious memory, the new typology offered empirical validation of what had been long known phenomenologically by psychoanalysis, namely that memorial processes had a large part to play in understanding highly charged transference reactions and other experiential events outside the transference [26].

By the 1980s there was a proliferation in the discovery of memory systems, and in particular Cohen and Squire [27] had differentiated procedural memory from declarative memory, thus providing the first formal description of a nonconsciously processed memory system. In 1984 Peter Graf and Daniel Shacter realized that to avoid confusion in the nomenclature, a new typology was required. They came up with the term "explicit memory" for consciously processed declarative memory–which had been subdivided into "episodic" and "semantic" by Tulving in 1972–and "implicit memory" for all forms of nonconsciously processed memory including procedural, emotional, and priming. Priming operates "invisibly" in our minds by influencing our memory as a result of prior exposure to events, ideas, and perceptions. Schacter writes interestingly about priming and plagiarism (pp. 167–168 in [20]).

Nonconsciously Processed Memory

I want to examine briefly what psychoanalysis has made of the nonconsciously processed aspects of the new memory typology.

Psychoanalysis has tended to lump together that version of the new memory typography which regards implicit memory as being the same as, and confined to, procedural memory, rather than regarding procedural memory as a category of implicit memory along with priming and emotional memory. For example, Ross [28] confirms my impression when he writes: "When all is said and done, for the time being, I will tend to stick to our current psychoanalytical convention and use the term 'procedural' to refer to implicit memory even though some cognitive scientists will see this as overinclusive." Other authors, for example Fonagy [29], Levin [30], and Target [31], refer to "procedural or implicit memory" as if they were interchangeable. Fonagy goes some way towards explaining why he regards them this way in a footnote (footnote 3, p. 216) where he writes: "from the therapeutic perspective, awareness of an active and passive mode of remembering should suffice." Though the terminology in psychoanalysis is much less important than the ideas conveyed, I regard this interchangeability as a confusion of species with genus.

Procedural Memory

Procedural memory is for motor, perceptual, and cognitive skills and habits [27]. Sometimes called "skill and habit" memory, procedural memory is typified by the acquisition of a motor skill, such as playing the piano, which, after many repetitions, becomes automatic. Once a skill has become a routine or a habit it can be downloaded to other brain systems including the basal ganglia, the motor cortex, and the cerebellum, where it is processed unconsciously (pp. 187–188 in [20]). A skill that has become "second nature" no longer requires diligent cortical monitoring. So here we have a memory process, once declarative–originally you have to learn consciously and remember that *Every Good Boy Deserves Fruit*–that eventually becomes automatic. This might be compared to our split perceptual system [32] which enables us to react to danger immediately, bypassing the cortex, thereby eliminating consciousness from the evasive action, or, if the danger is less threatening, the situation is thought out and a plan of action instigated in conscious awareness. Procedural memory and both aspects of our split perceptual system would be employed by a pilot landing a stricken aircraft.

In my view, the *mise en scène* and nature of procedural memory hardly fit it for the roles that have been ascribed to it by many psychoanalysts. It has become overloaded with significance that it does not warrant. The over-working of procedural memory has restricted our awareness for the other far more important implicit memory processes such as priming, which subliminally has considerable impact on our thoughts and behavior, and of course–and most important of all for psychoanalysis–emotional memory. It is certainly true that procedural memory and knowledge are nonconsciously processed, but it is not conscious in a very different sense from the way that mental contents that have been repressed are unconscious, or in a very different sense from the way that material that is not available to consciousness because of the neurological immaturity of parts of the brain (the hippocampus in particular) is not conscious. As procedural memory was the first unconsciously processed memory to be described by neuroscience, this feature of manifest "simple" nonconsciousness made it an attractive arena for theorizing. Psychoanalysis was excited too about the concept, because hitherto "unconscious" had meant Freud's dynamic, repressed unconscious, and here was a nonconscious mental process that was not repressed. Its formation from explicit activity is as devoid of affect as is the nonconscious memory that results, and perhaps, like semantic memory, its durability has something to do with its somewhat mechanical if not alexithymic nature! There is no autobiographical content and "it does not involve representations of an individual's internal states" [33]. It is understandable why the term "procedure" lends itself to improvisation and particularly to a way of consolidating early relationships and experiences which become habits, the "way we see things," which then become part of our character and influence the way we relate to others as adults. I do not disagree with the content of this sequence, but I do not believe that procedural memory "shoulders" this process. It is too one-dimensional both as memory and knowledge: it short-circuits the complexities of the interactions between all the memory systems, implicit and explicit, which bring versions of the past via many different neuronal pathways into the consulting room. Freud himself believed that a person's "character" is based on the memory traces of our impressions (p. 539 in [19]) but not, in my view, as Grigsby and Hartlaub [34] theorize, mediated by procedural memory. Inevitably procedural processes play some part in consolidating repetitive learning where coordinating and motor components are involved, but these would more frequently occur in older children and adults. Ryle [35] makes the point that there is a crucial difference between the procedures developed to manage physical skills and those concerned with human relationships, and that this difference is the presence of another person and another mind. I do not know whether neuroscience can accommodate that crucial difference; I don't think psychoanalysis can.

The work of Stern et al. on "implicit relational knowing" [26] and their conclusions that "the process of rendering repressed knowledge conscious is quite different from that of rendering implicit knowing conscious" is no less valid because they regard implicit and procedural knowledge synonymously, but I do think it is curious that in the intimate settings of mother with child and therapist with patient, the vehicle hypothesized to carry the subtle affective nuances of the "proto-conversation" (p. 55 in [36]) between mother and child and the affective immediacy of the "now" moments between patient and therapist should be a memory process that is without affect! In my view, the "something more" than interpretation would be greatly enhanced in their theory of implicit relational learning if implicit in this context meant unconscious emotional knowledge, supported by priming; and the habituation, now theorized as procedures, involved all memory systems, simple associative learning and classical conditioning.

Emotional Memory

Emotional memory is the conditioned learning of emotional responses to a situation and is mediated by the amygdala. The emotional memory representations are thought to be stored separately from the factual details of the events [37].

For more than 20 years, there has been a suspicion that the brain structures that support implicit memory are in place before the systems needed for explicit memory [38]. This is based on the fact that the human hippocampus, necessary for processing explicit memory, is immature at birth and for the first 2 years of life, while the amygdala and basal ganglia, necessary for processing implicit emotional memory, are well developed at birth. From this the conclusion has been drawn that implicit memory is impaired in the early years of childhood. Weiskrantz [39] suggested that implicit memory may be encoded and retained from infancy, in contrast to explicit memory, which does not become durable until 3 or 4 years of age.

Though Shacter [20] was moved to write that the symptoms of the hysteric patients studied by Freud and Breuer "are plagued by implicit memories of events they cannot remember explicitly," both neuroscience and psychoanalysis preferred to make what they could of the undeniable nonconsciousness of procedural memory rather than pursuing the more speculative route that suggests that emotional memory is represented much earlier than explicit memory. From the neuroscientific point of view this is understandable as a reliable research project to demonstrate this proposition faces enormous difficulties, not least because the orbitofrontal control system which plays an essential role in the regulation of emotion matures in the middle of the 2nd year, at which time the average child has a productive vocabulary of less than 70 words [40].

Freud and Unconsciously Processed Memory

From Freud's pre-analytic writings, it is clear that he was only too aware of his patients' powerful feelings directed towards him, but also of their propensity to act on those feelings instead of reporting them.

As is well known, Freud's early thoughts about transference were that it was an "obstacle" to be overcome and a powerful form of resistance

[41]; however, by 1905 he was able to write: "Transference, what seems ordained to be the greatest obstacle of psychoanalysis, becomes its most powerful ally, if its presence can be detected each time and explained to the patient" [42]. Nobody nowadays would disagree with his assessment of this "powerful ally", transference; however, it is only in the era of the new memory typology, with its confirmation of the importance and significance of an unconscious process that is not repressed, that acting out can be reevaluated in much the same way that Freud reevaluated transference. It is my view that Freud actually wrote about what we now call implicit memory knowledge without grasping that, like transference, acting out was not an obstacle, if understood by the analyst, but another "powerful ally" for communicating emotional memory that had no words. Freud's comments in "Remembering, Repeating and Working Through" indicate that acting out is regarded as "yielding to the compulsion to repeat, which now replaces the impulsion to remember" [43]. He continues: "The part played by resistance, too, is easily recognized. The greater the resistance the more extensively will acting out (repetition) replace remembering." He writes that he would prefer his patients to remember in "the old manner" (p. 153), a rueful reference to the fact that memories recalled under hypnosis were less stormy! "Doing something" appeared to be the only access to memory that the patient had. We now know that expressing feelings, sometimes through action, is the only way that a patient can express some part of a memory whose explicit component had not registered because of neurological immaturity or, as in a child of two-and-a-half or three-and-a-half, only in a fragmentary way. So the affective component is struggling for expression in the absence of any temporal or contextual signature from the hippocampus that might have indicated that the experience belonged to the past and not to the here and now. The archaeology of the mind knows nothing of carbon dating! Because Freud was unaware of unconscious material that was not repressed, he assumed that acting out was a way of remembering when what was forgotten had been repressed (p. 150 in [43]).

Fifty years later, acting out was still no ally. Greenson writing in 1974 described acting out under the heading of *Transference Resistances* and ends this section of his book by pointing out that when the reenactment is ego-syntonic "it is then always more difficult to enlist the patient's reasonable ego, to establish a working alliance and to uncover or re-construct the underlying memories" [44].

Acting out as described by both authors is more like a battle ground! Here is Freud again: "If . . . the transference becomes hostile or unduly intense and therefore in need of repression, remembering at once gives way to acting out . . . The patient brings out of the armoury of the past the weapons with which he defends himself against the progress of the treatment–weapons which we must wrest from him one by one" (p. 151 in [43]).

Although Freud ascribes acting out to the repression of memory in this paper [43], what is most intriguing is that he makes connections (pp. 148, 149) between screen memories, childhood amnesia, and internal psychical processes; he then writes: "In these processes it particularly often happens that something is 'remembered' which could never have been 'forgotten' because . . . it was never conscious." Immediately following these remarks, and we will return to them shortly, he associates to "one special class of experiences of the utmost importance for which no memory can, as a rule, be recovered. These are experiences which occurred in very early childhood and were subsequently understood and interpreted. One gains knowledge of them through dreams."

It is my view that Freud is here trying to understand the difference between the forgetting of what we would now call declarative events and nondeclarative events. Hence he writes that the forgetting of impressions, scenes, and experiences "nearly always reduces itself to shutting them off" (p. 148). However, with internal processes of reference, emotional impulses, and thought connections, "in these processes it particularly often happens that something is 'remembered' which could never have been 'forgotten' because . . . it was never conscious." This then is a description of nonrepressed memory. It becomes even clearer if we reverse the direction of the "psychical apparatus" (p. 536–542 in [19]) from "regression" to "progression": we are now considering *remembering*. Freud's passage now reads: "It particularly often happens that something is 'forgotten' which could never have been 'remembered' because it was never conscious." This then is a very clear description of what the author understands by implicit memory, that is to say emotional memory with priming. It might be said, too, that priming and Freud's screen memories have common ingredients!

The essential point here is that Freud identifies two different processes for remembering and forgetting internal and external "events." Internal and external acts "must," he writes, "in their relation to forgetting and remembering, be considered separately" [43]. Furthermore, he writes about the internal acts that it makes "no difference whatever whether such a thought connection was conscious and then forgotten or whether it never managed to become conscious at all. The conviction which the patient obtains in the course of his analysis is quite independent of this kind of memory" (p. 149 in [43]). In modern parlance Freud is saying that declarative "events" are forgotten by pushing them out of the mind (Freud says "shutting them off")–suppressing, not repressing, them–whereas nondeclarative "events" are "constructed" and it matters not to the patient whether they were once conscious or not.

Though Freud never produced a complete theory of memory, in this remarkable paper we see how neuroscientific ideas have brought to life the careful and truthful observations of a pioneer thinker.

Repression, the Unconscious Mind, and Unconsciousness

From a phenomenological and descriptive point of view, it is my view that Freud's Id energies derive from phylogenetic biological mechanisms, whose contents therefore have not been acquired by the individual. They do not contribute to consciousness as there is no neocortical involvement. Cautiously Freud writes something similar in the last paragraph of *The unconscious* [45]: "If inherited mental formations exist in the human being–something analogous to instinct in animals–these constitute the nucleus of the unconscious." Repression maintains the unconscious state and is here defined as the rendering unconscious of any sexual or aggressive conflict-laden feeling, impulse as idea. The author draws a clear distinction between the homeostatic motivational systems [46] (for example, sleep or nutrition), other motivational systems which include attachment interactions [47, 48], and the sexual and aggressive instincts described by Freud. It remains to be seen how Panksepp's affect-generating "emotional command systems" [49], which range from seeking systems, through rage, fear, panic, lust, and care systems, to those that elaborate play, relate to Freud's instincts. Panksepp regards these as regulatory systems but not drives. All these motivational systems are innately based, and it is reasonable to suppose that some are phylogenetically older than others. It is not proposed here that instinctual life is a simple dichotomy between sexuality and aggression, but that these two drives have a unique importance in understanding human behavior to the extent that with them has evolved their own controller–repression. Freud was always perplexed by the degree of unpleasure that derived from sexuality which then demanded repression while equally or more severe unpleasure from other aspects of life did not.

In my opinion there must be an independent source for the release of unpleasure in sexual life: once that source is present, it can activate sensations of disgust, lend force to morality, and so on . . . . As long as there is no correct theory of the sexual process, the question of the origin of the unpleasure operating in repression remains unanswered. (p. 164 in [6])

One of the most important reasons why Freud introduced his second topography was that he realized that no single agency could be described as "the unconscious," since not only the Id but parts of the Ego and Superego in the second topography could be described as unconscious. And as Laplanche and Pontalis [50] point out (p. 474), the term "unconscious" is used more as an adjective within the second topography. It could be said that this amounts to awareness that, though there appears to be an agency, "the unconscious," the mind also utilizes unconscious processing in many different ways and for different "purposes." For example, the suppression of memory (it is sometimes called forgetting!) is probably the most commonly employed mechanism for removing from consciousness an idea or an event which may cause discomfort or inconvenience. It does not involve the dynamic unconscious and could be described as preconscious. For example, it allows us a "now" experience when we take the same bus on the same route every day. We *could* remember yesterday's bus journey if we tried. Similarly the reason why we did not post that letter with the cheque in it was, on reflection, because we thought the parking fine unjust.

Neuroscientists are on the whole much happier with the adjectival version of unconscious! For example, Kandel [51], following a discussion on implicit memory (he appears to be confining himself to procedural memory), writes that these findings "bear no resemblance to Freud's unconscious. It is not related to instinctual strivings or to sexual conflicts, and the information never enters consciousness … Where, if it exists at all, is the other unconscious?" Using this formulation, where indeed! There is absolutely no resemblance between the nonconsciousness of reflexive procedural memory and Freud's dynamic unconscious: to suggest otherwise is like saying that the silent transport of neural impulses reveals another "unconscious."

The new memory typology revealed to psychoanalysis another set of phenomena in which unconscious and nonconscious processes participate. Before our awareness of implicit memory, other known events involving unconscious or nonconscious mental processes or events were: fainting (for psychological reasons), sleeping and dreaming, dissociative states including somnambulism and fugue states, suppression of memory (nonconsciously processed), posthypnotic states, and, of course, repression. It is not surprising then, that following the description of three further "scientifically based" examples of unconscious or nonconscious mental activity–priming, emotional memory, and procedural memory–both disciplines began to question whether Freud's unconscious should be reassessed in the light of these discoveries. For the reasons I have stated, I think psychoanalytical theorists were intrigued by the nonconsciousness of procedural memory without examining whether this was a truly "mental" process or nothing more than a reflexive, physiological one. Not-being-conscious was confused with unconsciousness. The "wrong" type of implicit memory, the "mindless memory," was chosen by some psychoanalysts, supported by neuroscience, to challenge the relevance and importance of Freud's unconscious and its jailer–repression. It seems perfectly reasonable, though contrary to the thesis of this essay, to advance the idea that object relations patterns form procedural memories which are unconscious because they are procedural and not because they are repressed [52], but why does this then mean that "the removal of repression is no longer to be considered a key to therapeutic action?" (p. 218 in [29]). Because other types of unconscious processing have been described, does analytic work on repression lose significance? Cannot a door have more than one lock? I entirely agree with Fonagy [29] that one of Freud's central ideas, that lifting repression and overcoming resistance would reveal the patient's past, has turned out to be flawed, because we now know not only that perception is not immaculate, but that much recall is a mere improvisation on actuality! The important point, however, is that repression has for so long been regarded as a nuclear theme in psychoanalysis that it is hard for psychoanalysis not to regard the repression–unconscious equation as the only manifestation of unconscious or nonconscious processing. My understanding of the essence of Freud's paper "Remembering, Repeating and Working Through" is that because Freud was consciously "unaware" of the significance of what we now call implicit memory, he employed repression as the process whereby memories were concealed. We now suspect that the declarative element in these memories was absent or distorted because they could not be securely registered in the mind due to functional immaturity in the brain. So acting out became for Freud the only window on the memory that was composed of feelings without words.

Looking for truthful memory behind the door marked "repression" is looking in the wrong place; however, squinting above, below, and behind this door for scraps of evidence about that phylogenetic alien that we call the dynamically repressed unconscious remains a vital part of helping our patients to learn about their own minds.

Of increasing importance is the attempt to look behind that other door marked "implicit" memory which is not repressed but clues to which are just elusive: access, as has been described, is restricted by the nature of the "plumbing." It would seem likely that the dream process, as well as "servicing" the royal road, the preconscious routes and unconscious manifestations of neurological damage, also offers glimpses into implicit emotional memory [53].

I have implied that the process of gaining some access to some aspects of the repressed unconscious and implicit memory is a circuitous process; in fact, gaining access directly to either is impossible. Freud makes it quite clear that the dynamic unconscious is only accessible to consciousness when, through analysis, it has become preconscious. So the preconscious is not repressed, but it is not conscious either. Freud is emphatic when he writes in *The Ego and the Id:* "We see, however, that we have two kinds of unconscious–the one which is latent but capable of becoming conscious ["the suppressed"], and the one which is repressed and which is not, in itself and without more ado, capable of becoming conscious" [54].

The new memory typology from neuroscience has had far-reaching consequences for psychoanalysis. In addition to the dynamic repressed unconscious and the unconscious suppressed content, we are now aware of implicit memory comprising nonconscious and unconscious emotional memory, unconscious priming and nonconsciously processed procedural memory. Because different pathways and systems are involved, it is not surprising that implicit memory and the repressed unconscious require different techniques for their elucidation.

The "flavor" of a clinical session may suddenly change and the routine of association followed by interpretation may be interrupted by a change of gear from the patient when affect takes over from cognition. The therapist must be alert to the sudden, often subtle and silent variation in the patient's affective experience during a session. So as not to miss the fleeting nature of a small concealed eruption of emotion, I sit behind but to the side of my patients from where I can see their face more clearly. To miss such a moment would, I think, be described by Stern et al. as a "failed now moment" [26]. Stern et al. differentiate their "something more" from a transference interpretation by describing the first as a "mutative relationship" and the interpretation as "mutative information" (p. 903 in [26]).

I entirely agree with them that the foundations of this "shared implicit relationship" have their roots in the earliest relationships, and that implicit knowledge and memory (in my view emotional memory and priming, not procedural) is not repressed because it does not belong to the unconscious instinctual system that employs repression to maintain the unconsciousness. It belongs to the implicit emotional memory system that has initially no explicit counterpart.

My small area of dissent from Stern et al.'s point of view concerns their description of a "moment of meeting" (p. 917 in [26]). To paraphrase their five points: It is the opposite of business as usual; I am not a modern representative of anybody; the experience is now; we don't need to talk about it; and, lastly, a technical response is inadequate and the "analyst must respond with something that is experienced as specific to the relationship with the patient and that is expressive of her own experience . . . and carries her signature" (p. 917). I disagree with only this last point because at that moment the analyst can be experienced as a facilitator, genuinely empathic, "on my side" and so on. However, the experience for the patient is an emotion coming from they "know not where," and the analyst's signature at this point could be experienced as an intrusion. A grunt of acknowledgment or a hyperattentive silence could serve the situation better when a patient feels free to experience being alone with their feelings in the presence of a trusted other whose existence and support is not in question [55]. The safety of the therapist's total involvement permits this "memory in feeling" [56].

It is often not easy to judge when an interpretation, transference or otherwise, is most helpful or whether "now" is definitely not a business-as-usual experience for the patient and a different sort of listening would be more useful. The degree of affect in a patient's utterances is not necessarily a good guide because the patient may be experiencing strong feelings towards the therapist which originate from a time when declarative memory was fully functioning and the autobiographical memory accompanied the feelings. A transference interpretation may then be the only appropriate intervention. However, it is often the patient who makes the situation clear. An interpretation, however accurate, will not be heard by the patient if he is in the grip of powerful implicit emotional memory where words from the therapist are as irrelevant as they once were. The interpretation, if heard at all, will be experienced as a background noise or even as intrusive. For the patient, his feelings are not at that moment about the relationship in the room. Concentrated attunement is required from the therapist at such times. Sometimes comments such as: "I'm into something else right now" or "I'm somewhere else" can give the therapist a verbal clue. Practically speaking, it is often much more difficult!

It is suggested that “It is the emotional availability of the caregiver in intimacy which seems to be the most central growth-promoting feature of the early rearing experience” [57], and where this availability appears not to have been there, it is likely that there will be a need in the patient for more “emotional memory” communications; “repressed contents” communications including transference interpretations will seem to be more helpful where the patient’s mental life is predominantly “depressive” rather than “paranoid-schizoid.”

The moving account by Margaret Little, as *patient,* where she describes her “implicit relational knowing” experiences with Winnicott and how she later ordered these experiences in her mind as *author* when her anxieties had become more depressive, illustrate how feelings without words can be metabolized though empathy. “It is a very remarkable thing that the unconscious of one human being can react upon that of another without passing through the conscious” [58]. This surely refers to an implicit, non-repressed unconscious, sensed but not described by Freud [53]. Margaret Little writes:

Literally, through many long hours he held my two hands clasped between his, almost like an umbilical cord, while I lay, often hidden beneath the blanket silent, inert, withdrawn, in panic, rage or tears, asleep and sometimes dreaming. Sometimes he would become drowsy, fall asleep and wake with a jerk, to which I would react with anger, terrified and feeling as if I had been hit…[a “failed now moment” [26]; author’s comment]. He must have suffered much boredom and exhaustion in these hours, and sometimes even pain in his hands” [59].

Margaret Little’s own implicit awareness later enabled her to reciprocate Winnicott’s empathy when she diagnosed and assisted him when, unknown to him, he had a heart attack during a session with her. Truly, a shared implicit relationship. (M. Little, personal communication).

Memory Objects

The philosopher Chalmers [13], when considering mind/brain issues and more particularly consciousness, suggested dividing the latter into two aspects: the “hard problem” and the “easy problem.” The easy problem for neuroscience involves constructing experiments that identify those physiological processes in the brain that relate to consciousness. The hard problem is to explain how do these processes produce our sensory experiences?

However, for psychoanalysis there really are no easy problems; the "ghost in the machine" makes sure of that! Psychoanalysis has to contend with unconscious processes that obfuscate; defenses which deflect, deny, and confuse; a psyche that is a past master at self-deception; and a perception/memory system that distorts, invents, and falsifies, but none the less carries forward into the present a version of the past!

In this last part of the essay, I want to discuss briefly how experiences from the past are processed and remembered. Ways of relating to others, satisfactory and unsatisfactory experiences, loss and illness, all influence and shape each present moment as it comes. The experiences of birth influence the child's earliest relationships with its primary carer. This in turn influences how the child negotiates the oedipal situation and so forth. The sum of all these experiences influences how, when we are old or ill, we contemplate our own death.

Klein's internal object relations theory provides psychoanalysis with a largely intrapsychic system of interacting unconscious experiences as a contribution towards understanding the past in the present, but it contains such seemingly obscure ideas, inimical to neuroscience (e.g., its attempt to differentiate a representation from a concrete experience), that neuroscience has largely ignored the topic. It may also have been the case that neuroscientists subsumed the topic under the general heading of memory. I was surprised that Kandel [60] suggested that Klein's internal object relations theory was limited because it was constructed and based on *outdated* conceptualizations of memory, because there is no evidence that she regarded her theory as having anything to do with a memorial system, although others–notably Marjorie Brierley [61]–wondered whether there was a common basis. It can certainly be said that Melanie Klein's observations on "memories in feeling," which she described as the "very vivid revival of early internal and external situations," is one of the first descriptions of implicit emotional memory [62] and anticipated the new memory typology by more than two decades!

Both Klein and Fairbairn stressed the dynamic nature of internal objects. Fairbairn writes: "Under certain conditions internalized objects may acquire a dynamic independence . . . It is doubtless in this direction that we must look for an explanation of the fundamental animism of human beings" [63]. It was this animism and dynamism of internal objects which suggested to me a link between internal object relationships and Melanie Klein's "memories in feeling" which seemed such a clear account of implicit emotional memory [64].

In 2002, following work carried out by the Hampstead Index Project and reported by J. and A.-M. Sandler [65], a distinction was drawn between internalizations that modify the self-representation, which they called "identifications," and internalizations that elevate object representations to the special status of "introjects." Using this distinction, I proposed that the "introjects" involve both self and object experiential representations and form part of the explicit memory system. Bringing attention to bear opens the possibility of reflection and modification. "How would my father have tackled this problem?" indicates something of the nature of these introjects.

On the other hand, the "identifications" are part of character, part of the self, and bringing attention to bear in no way opens up a debate as they do not involve subjective experience. They can be regarded as part of the implicit emotional memory system. The terminology is much less important than the idea that aspects of the ego and all internal objects including the superego are composed of memorial material, some of which is accessible to consciousness and some of which is not. I termed the "identifications" "implicit memory objects" and the "introjections" "explicit memory objects" because it seemed to me that what Klein was describing in terms of a subjective model of the inner world had much in common with the more dynamic aspects of the new memory model, particularly implicit emotional memory.

However, memory systems as we now know them are probably insufficient to account for how events, feelings, relationships, and attitudes are carried forward from the past to inform the present. Because it seems certain that memory systems are interactive and have multiple distributions in the brain, it is likely that both declarative and nondeclarative memory systems are involved. Consciously and unconsciously processed memory can be seen to interact. For example: How often have we heard a friend espousing a view on something with absolutely no awareness that it was "lifted" from a conversation we had had together a few weeks before (and it probably contradicts his original view!)? This example of nonconsciously processed priming is an important way that children pick up information and also how some information can be gleaned about implicit memory that is otherwise not conscious. I do not agree with the general statement "that all forms of memory can be retrieved, including . . . implicit, by the priming effects of the contents of dreams, transference, mental slips etc" [30]. However, priming unconsciously influences all types of memory except perhaps semantic memory, and of course emotional memory profoundly influences episodic memory.

Classical conditioning processes, which are involved in all forms of knowledge acquisition including emotion, must be differentiated from procedural processes. It could be said that the outcome of conditioning is the formation of a procedure, but this has nothing to do with the process involved in the formation of procedural memory. Davis writes that "simple classical conditioning is not a low level or reflex process. It is a primary means through which the world is represented. These representations are highly complex and precise" [66]. By contrast, however, the procedural process and the resultant memory is low level and akin to a reflex, and all that it has in common with classical conditioning is its nonconscious status. Classical conditioning would come into the category of "identifications" in the memory object classification and is of course nondeclarative.

Two Clinical Vignettes

In this first vignette, I will describe aspects of three sessions which illustrate the emergence of feeling memories following a head injury.

Peter is a talented middle-aged married man in one of the caring professions. His father was a gentle, scholarly man and his mother a kind, self-effacing woman who suffered from bouts of severe depression which required admission to mental hospital at irregular intervals during Peter's childhood; this meant that he was required to stay with relatives for some weeks at a time.

The week before the three sessions I shall briefly describe, Peter had a sudden, violent, but not life-threatening accident in which he struck his head and body on a concrete post and suffered transient unconsciousness. After a week's convalescence, he returned to therapy in a sorry state, with stitches in his head and a cut and bruised face. He lay down very slowly and with great difficulty. He recounted details of the accident and clearly was still quite shocked. He told me he wanted me to know what it was like to feel he was dying. Near the end of the session he attempted to sit up but said it was impossible. He tried several more times but each time he sank back in a very distressed state saying he could not get up. I fetched him a glass of water and he made it clear that he did not want me to help him up. I moved the armchair closer to the couch, reassured him that there was no hurry, and told him I was leaving the room for a few minutes. Three or four minutes later he had managed to sit up with the use of the chair. When I returned, he asked me to leave again because he said my absence made it easier for him to get up. Five minutes later he called a farewell and left. My medical knowledge reassured me that he had not had a cardiovascular episode; however, I felt quite painfully while I was outside the room that I had been neglectful of him, but I also sensed that at that moment my presence was symbolically too close to a desperately needed object.

In our next session he again slowly and painfully lay down. He told me that he had felt very reassured that I had left the room last session because it meant that I had not regarded him as a medical emergency. He spoke further about his accident and how he had thought he was dying. Then he said to me with great feeling: "There is one area where you have not understood me, you haven't realized that I want to look for what is actually true. Bottle feeding isn't real milk. My mother seems to be saying: 'Drink it up: it doesn't matter that there isn't a real breast[2].' She would say, 'No such thing as can't, worse things at sea and such like.' I was longing for you to hold my hand last session. I don't want to be told my thoughts are at fault."

I told him that he was telling me what if it felt like to be "fobbed off," not to be allowed to have feelings of self pity or helplessness or need, and what it felt like to be told that "others had it worse" and that "you aren't really feeling what you're feeling." I also told him that the terrifying fear he had had after his accident that he was dying was probably an echo of a terror from childhood.

However, what I did not tell him at the time, and which I now believe in restudying the material, was that he was also struggling to disentangle the difference between his experience of me now, as a representative of his autobiographical "internal mother," and the reemergence of actual "bits" of feeling memory from the past, possibly from amygdala circuits, as a result of his head injury [67].

As the end of the session approached, once more he struggled to sit up and sank back again onto the pillow. He tried several more times and with the help of the chair eventually sat upright. He then said hesitantly: "I think you ought to leave me." I replied that I would if that was what he really wanted. Slowly and painfully he inched his legs onto the floor, smiled, and said: "You're a cunning b . . . aren't you? You were absolutely right not to leave. I don't feel embarrassed any more by this incident."

In the following session he spoke more about his accident and his various medical advisors, but near the end of the session he struggled to the

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[2] He is here illustrating his need for authenticity. He was in fact only breast fed, but on a rigid 4-hourly regime.

sitting position and said a little desperately: "I'm so sorry I'm putting on this show." I said straightaway: "This is not a performance, it *is* a "show"; you are showing me in the only way you can what it is like to have powerful feelings that never have had and never will have words."

Space does not allow for a full discussion of all the important material in the sessions following Peter's accident, so I will confine it to a few comments on aspects that are directly relevant to this essay. When Peter found that he was unable to move from the couch, it came as no surprise to me because I realized we had been there before and that I had been "primed" in the sense that there had been clues in his material from past sessions which indicated his need for close attachment to me; for example, he would often make jokes as he was getting off the couch; he would write out his cheque at the end of a session always borrowing my pen; if he had been angry in a session, he would always "make up" before leaving. His overinclusive accounts of happenings in his life as a defense against feelings came to be known to us both as "my seminars." Though I tried to understand his feelings using transference interpretations, it always seemed that he wanted and needed "something more" [26]. Two sessions before his accident he realized halfway through a session that he had been using his "seminar" technique to talk his way away from his feelings. He directed his frustration and amused vexation at himself and struck his forehead with the palm of his hand: "Ahrr, there I go again, I feel so frustrated, I feel I want to be hysterical . . ." I particularly noticed his unusual phrase.

I suspected that most of the transference references were for the years from 5 onwards when his autobiographical memory was firmly established and deference for others and concealing painful feelings had become egosyntonic. Intrapsychically he had to find a way of getting at these earlier feelings which had no autobiographical content . . . As with repressed contents so with implicit memory, it cannot directly be brought into consciousness.

In the sessions that followed he did much useful self-interpretation. He identified the tears in his eyes following the rendition of some French religious music with words that spoke of the relief of suffering, through the supporting hands of God. One morning he asked if he could bring his bicycle into my hall and he almost preempted my interpretation in the session with a clarity of understanding to which I had nothing to add.

There were many more sensations and phantasies in the sessions after his accident and they revealed a complex bridging system between implicit and explicit memory, the concrete experiences of Klein's internal objects, and the representations of others.

Two sessions after his head injury he wondered aloud whether this had *entirely* been an accident; many sessions later, he said that in retrospect, the experience had had its useful side.

The last point I wish to examine from Peter's first session after his accident is my feeling when I was out of the consulting room for a few minutes. I felt that I was abandoning him; I felt neglectful, guilty, and coerced. I worked out that I was processing a powerful projective identification, and that as a concrete version of his mother I was being punished for not being more attentive. However, a few days after the incident, I read Cimino and Correale's paper "Projective identification and consciousness alteration" [68], in which they suggest that projective identification, as well as being a defense mechanism, also has a powerful communication role. They propose that aspects of the patient's early unconscious experience can be transmitted to the analyst where it exerts strong emotional pressure, provoking emotional upheaval, a sense of coercion, and even altered consciousness. They suggest that in these situations nondeclarative memory is here exercising its influence, and that where the infant has not had the experience of having its projections "metabolized" and "returned" by the primary object, the patient may sometimes show this type of projective identification.

I believe that in Peter's case his autobiographical memory was nonexistent at the level we are discussing, but his unconscious emotional memory with later priming ("worse things at sea," etc.) found discharge by projective identification in the way suggested by the authors and in the way experienced by me.

The second vignette is a brief description of those "devices" that a middle-aged male patient used to combat what he called "a shy penis," by which he meant an inability to urinate in public.

His first device was to remember himself aged about 11, a fine athlete, captain of football and cricket and much admired by his peers. Contemplating these times enabled him to micturate in public.

His second device was to say out loud his father's Christian name. His father had been a powerful and at times harsh figure in my patient's life, but was now very old and frail. Naming his father allowed him to urinate.

The third device puzzled him more. Since his brother, who was two-and-a-half years younger than he, had died about 10 years before, he found that saying his name out loud enabled him to urinate. He was devoted to his brother and was aware of no negative feelings towards him. He was heartbroken when he died. His younger brother had been chronically ill as a baby which had necessitated his mother's constant attention. My patient had "heard" that at about the age of two-and-a-half, as a consequence of his brother's illness, he had been sent away for a while, and the extent of his distress at this separation was legendary in the family. My patient had no conscious memory of this.

Of the first device, conscious and unconscious anxiety from the past and present inhibits the conscious relaxation of the urethral sphincter; bringing attention to bear on a conscious memory of feeling "cock of the walk" overrides the anxiety and allows the conscious relaxation of the sphincter.

Of the second device, the process is the same in that he brings his father's Christian name to consciousness but there are no conscious feelings of triumph or revenge, though my patient believes that these are active unconsciously.

Of the third device, his brother's name is uttered, and from then on until the point where the sphincter is consciously relaxed, the whole process involves implicit memory with no conscious recall. What I surmise–and my patient felt that this was correct–was that despite his sincere and profound grief, the fact of his brother's death released some implicitly stored feelings from childhood rivalry and anger with his brother which are unconsciously expressed as triumph after his death and which facilitate his ability to urinate in public.

The "conjuring" up of sexual phantasies in order to achieve orgasm no doubt uses similar neural pathways.

These vignettes illustrate the complexity of the interactions between implicit and explicit memory and the rest of the nervous system and point to the need for further research, by neuroscience and psychoanalysis, to discover more about how the past is represented in the present. Until then a theoretical model involving dynamic internal objects, animated by all types of memory, often perceived as being concrete and happening "now," is essential to avoid the reductionist view that implicit memory amounts to an assortment of physiological procedures.

We now know unequivocally that there is more than one source of unconscious psychic material. Freud spent a lifetime describing the first, the repressed dynamic unconscious, and had inspirational glimpses at others. Psychoanalysis must not be seduced away from its work with Freud's unconscious, but at the same time must not ignore the necessary refinements and adjustments to technique which will become necessary as we learn more about implicit memory and direct emotional experiences that are not transferred.

References

1. Solms M (2004) Freud returns. Sci Am, May 58–62
2. Goldberg A (2004) Ongoing discussions on David Milrod's "The concept of the Self and the self-representation. Neuropsychoanalysis 6:91–92
3. Oliver Turnbull (2004) Founders of neuro-psychoanalysis. Subject: Mortimer Ostow. Neuropsychoanalysis 6:209–215
4. Green A (2001) Advice to Psychoanalysts: "Cognitive psychology is good for you". Neuropsychoanalysis 3:16–19
5. Whitehead AN (1967) Adventures of ideas [first published 1933]. Prentice-Hall, U.S.A.
6. Masson JM (ed) (1985) The complete letters of Sigmund Freud to Wilhelm Fliess 1887–1904. Belknap Press, London
7. Freud S (1895a) Project for a scientific psychology. SE 1
8. Kanzer M (1973) Two prevalent misconceptions about Freud's Project. Ann Psychoanal 1:88–103

8a. Mancia M (1983) Archaeology of Freudian thought and the history of neurophysiology. Int Rev Psychoanal 10:185–191

9. Solms M, Saling M (1986) On psychoanalysis and neuroscience:Freud's attitude to the localisation tradition. Int J Psychoanal 67:397–416
10. Stewart S (1969) Psychoanalysis: the first 10 years 1888–1898. George Allen and Unwin, London
11. Kaplan-Solms K, Solms M (2000) Clinical studies in neuro-psychoanalysis. Karnac, London
12. Freud S (1940) An outline of psycho-analysis. SE 23, pp 144–145
13. Chalmers DJ (1995) Facing up to consciousness. J Conscious Stud 2:200–219
14. Toulmin S (1978) Psychoanalysis, physics and the mind-body problem. Ann Psychoanal 6:315–351
15. May U (1999) Freud's early clinical theory (1894–1896). Int J Psychoanal 80:769–779
16. Freud S (1913) The claims of psychoanalysis to scientific interest. SE 13, pp 165–190
17. Freud S (1940) Some elementary lessons in psycoanalysis. SE 23, pp. 281–286
18. Freud S (1927) An autobiographical study. SE 20,pp. 7–70
19. Freud S (1900) The interpretation of dreams. SE 4 and 5
20. Schacter DL (1996) Searching for memory. Basic Books, New York
21. Fabiani M, Stadler GA, Wessels PM (2000) True but not false memories produce a sensory signature in human lateralized brain potentials. J Cogn Neurosci 2:941–949
22. Freud S (1950) Screen memories. SE 3
23. Freud S (1950) Resistances to psychoanalysis. SE 19
24. Jacobs TJ (1994) Non verbal communications: some reflections on their role in the psychoanalytical process and psychoanalytic education. J Am Psychoanal Assoc 42:741–762
25. Shore AN (1994) Affect regulation and the origin of the self: the neurobiology of emotional development. Erlbaum, Mahwah NJ
26. Stern D, Sander L, Nahum JP et al (1998) Non-interpretative mechanisms in psychoanalytic therapy. Int J Psychoanal 79:903–921
27. Cohen N, Squire L (1980) Preserved learning and retention of pattern-analyzing skill in amnesia: dissociation of knowing how and knowing that. Science 210:207–209
28. Ross JM (2003) Preconscious, defence analysis memory and structural change. Int J Psychoanal 84:59–76

29. Fonagy P (1999) Memory and therapeutic action. Int J Psychoanal 80:215–223
30. Levin M (2004) Letter. Int J Psychoanal 85:1276
31. Target M (1998) Book review essay. The recovered memories controversy. Int J Psychoanal 79:1015–1028
32. Le Doux J (1995) Emotion: clues from the brain. Annu Rev Psychol 46:209–235
33. Shacter DL, Tulving E (1994) What are the memory systems of 1994? In: Shacter DL, Tulving E (eds) Memory systems. MIT Press, Cambridge MA, pp 197–214
34. Grisby J, Hartlaub GH (1994) Procedural learning and the development and stability of character. Percept Mot Skills 79:355–70
35. Ryle A (2003) Something more than the "something more than interpretation is needed." Int J Psychoanal 84:109–116
36. Trevarthen C (2003) Neuroscience and intrinsic psychodynamics: current knowledge and potential for therapy. In: Corrigall J, Wilkinson H (eds) Revolutionary connections. Karnac, London
37. Le Doux J (1994) Emotion, memory and the brain. Sci Am June 270:32–39
38. Shacter DL, Moscovitch M (1984) Infants, amnesics and dissociable memory systems. In: Moscovitch M (ed) Infant memory. Plenum, New York, pp 173–216
39. Weiskrantz L (1997) Memories of abuse, or abuse of memories? In: Sandler J, Fonagy P (eds) Recovered memories of abuse: true or false? Karnac, London pp 3–21
40. Shore AN (2003) The seventh annual John Bowlby memorial lecture. Minds in the making: attachment, the self-organizing brain and developmentally-oriented psychoanalytical psychotherapy. In: Corrigall J, Wilkinson H (eds) Revolutionary connections. Karnac, London
41. Freud S (1895) Psychotherapy of hysteria. SE 2, pp 255–305
42. Freud S (1905) Postscript. SE 7, pp 112–122
43. Freud S (1915) Remembering, repeating and working through. SE 12, pp 147–156
44. Greenson RR (1974) The technique and practice of psychoanalysis, vol 1. Hogarth Press, London, pp 247–268
45. Freud S (1915) The unconscious. SE 14, pp 161–204
46. Edelman G (1989) The remembered present: a biological theory of consciousness. Basic Books, New York
47. Gilbert P (1989) Human nature and suffering. Erlbaum, London
48. Migone P, Liotti G (1998) Psychoanalysis and cognitive-evolutionary psychology: an attempt at integration. Int J Psychoanal 79:1071–1095
49. Panksepp J (1998) Affective neuroscience: the foundations of human and animal emotions. Oxford University Press, Oxford
50. Laplanche J, Pontalis J-B (1980) The language of psychoanalysis. Hogarth Press, London
51. Kandel ER (1998) A new intellectual framework for psychiatry. Am J Psychiatry 155:457–469
52. Clyman R B (1991) The procedural organization of emotions: a contribution from cognitive science to the psychoanalytical theory of therapeutic action. J Am Psychoanal Assoc 39(Suppl):349–382
53. Mancia M (2003) Dream actors in the theatre of memory: their role in the psychoanalytic process. Int J Psychoanal 84:945–952
54. Freud S (1923) The Ego and the id. SE 19, pp 12–66
55. Winnicott DW (1958) The capacity to be alone. In: The maturational process and the facilitating environment. Hogarth Press, London, p. 33
56. Klein M (1961) Narrative of a child analysis. In: The writings of Melanie Klein, vol 4. Hogarth Press, London

57. Emde RN (1988) Development terminable and interminable. Innate and motivational factors from infancy. Int J Psychoanal 69:23–42
58. Freud S (1915) The unconscious. SE 14, p 195
59. Little M (1985) Winnicott working in areas where psychotic anxieties predominate. Free Associations 3:9–42
60. Kandel ER (1999) Biology and the future of psychoanalysis: a new intellectual framework for psychiatry revisited. Am J Psychiatry 156:505–524
61. Brierely M (1991) Marks from minutes of third discussion of scientific controversies. In: King P, Steiner R (eds). The Freud-Klein controversies 1941–45. Routledge, London, pp 400–401
62. Klein M (1957) Envy and gratitude. In: The writings of Melanie Klein, vol 3. Hogarth Press, London, pp 176–235
63. Fairbairn WRD (1952) Psychoanalytical studies of the personality. Tavistock/Routledge, London, p 132
64. Pugh G (2002) Freud's problem: cognitive neuroscience and psychoanalysis working together on memory. Int J Psychoanal 83:1375–1394
65. Sandler J, Sandler A-M (1998) Internal objects revisited. Karnac, London, pp 127–128
66. Davis JT (2001) Revising psychoanalytic interpretations of the past. Int J Psychoanal 82:449–462
67. Jacobs WJ, Nadel L (1985) Stress induced recovery of fears and phobias. Psychol Rev 92:512–531
68. Cimino C, Correale A (2005) Projective identification and consciousness alteration: a bridge between psychoanalysis and neuroscience. Int J Psychoanal 86:51–60

Recollecting the Past in the Present: Memory in the Dialogue Between Psychoanalysis and Cognitive Science

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Introduction

Psychoanalysis and the Neurosciences/Cognitive Sciences in Dialogue

In the last 20 or 30 years a vision of Sigmund Freud has been seeming to become reality: It is well known that Freud never gave up his hope that some day developments in the neurosciences might contribute to a "scientific foundation" of psychoanalysis in terms of the natural sciences. One reason why Freud himself did not continue his own attempts for such a neuroscientific foundation of psychoanalysis, his *Outline of psychoanalysis* [1], was his confrontation with the obvious limitations of the methodologies of the neurosciences of his time [2]. He then consistently defined psychoanalysis as a "pure psychology of the unconscious."

Recent developments in the neurosciences, e.g., the fascinating possibilities for studying the living brain by neuroimaging techniques (MEG, magnetence phalogram; ERP, event related potential; PET, positron emission tomography; fMRI, functional magnetic resonance imaging), as well as studies with the so-called neuroanatomical method developed by Kaplan-Solms and Solms [2], have initiated a boom and intensified dialogue between psychoanalysis and the neurosciences during the last 20 years or so.[1] 1999 saw the publication of the first volume of the international journal *Neuro-Psychoanalysis*, in which leading psychoanalysts and

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[1] A large number of studies using neuro-imaging are going on at the moment and seem to discover a large variety of different memory systems in the brain [3–6].

neuroscientists present their studies of emotion and affect, memory, sleeping and dreaming, conflict and trauma, conscious and unconscious problem solving, etc. In 2000 the International Society for Neuropsychoanalysis was founded, which organizes international conferences every year. In many different countries interdisciplinary groups of researchers have started to work systematically with patients who have suffered brain lesions which can be precisely localized in the brain. The joint aims of these research groups are the development of specific psychoanalytic treatment techniques which will enable us to help groups of patients (e.g., those suffering from a neglect syndrome after a stroke) therapeutically in the future [7, 8]. Another common aim is the intention to study the old topic of European philosophy in a new way: to investigate the relationship between brain and mind by systematic and critical reflections on the clinical psychoanalytical findings in these groups of patients worldwide [9, 10].

As the different contributions to this volume illustrate, the dialogue between psychoanalysis and the neurosciences is of outstanding importance thanks to an increasing number of fascinating empirical and experimental studies in the areas of psychotherapy research, developmental and dream research, and many others, as well as to studies in the field of so-called *basic science*. However, just one critical introductory remark: after our experiences in a joint effort between 20 psychoanalysts and neuroscientists studying memory, dreams, and cognitive and affective problem solving from the perspectives of these two disciplines in a joint research project from 1992 to 1998 (supported by the Köhler Foundation, Darmstadt, Germany), it seems important to us to *critically reflect on the epistemological dimensions of this dialogue* [11, 12]. We see the dialogue between these disciplines as fascinating, innovative, and interesting–but also challenging and complicated for both sides. We often do not speak the same language, and apply different concepts even when we are using analogous terms. Furthermore, we often feel identified with divergent traditions in science and in philosophy of science. We need a great deal of tolerance and a lot of staying power to really achieve an intensive exchange of ideas of the kind that will enable us to reach new frontiers in our own thinking: to crack apart our former understanding and conceptualizations and resist the idealizing tendency to expect "solutions" for unsolved problems in our own discipline from the other (foreign) discipline–which is, like a blank screen, capable of attracting projections and projective identifications. To take new findings from the other discipline seriously means to undergo a period of uncertainty and of unease: it is always painful to leave “certainties” and false beliefs developed in your own field. To go through such a period of uncertainty and unease is, however–as shown by our actual interdisciplinary experiences–essential and unavoidable: it seems to be a prerequisite for a productive and constructive dialogue that goes beyond rediscovering already established disciplinary knowledge. Comparing models that have been developed in the two disciplines to explain their own specific data, collected by specific (and very different) research methods, involves encountering complex and sophisticated problems of philosophy of science and epistemology. To mention just two examples: the well-known danger of the eliminative reductionism of psychological processes onto neurobiological processes, or the consequences of unreflecting transferral of concepts, methods, and interpretations from one scientific discipline onto another one have to be prevented.

Another interesting aspect is elaborated on in a recent book by Michael Hagner [13], who discusses the enormous influence of neuroimaging techniques on current science and societies. The fantasized possibility “to have a direct view into the living and working brain” carries enormous seductive and fascinating power. It may, for example, mobilize the fantasy of gaining new, direct diagnostic capacities:

You may then differentiate between unstructured, chaotic forms of thinking and mathematical problem solving . . . between memories of earliest experiences in childhood, the last fight with your spouse, or conflicts with your parents, between erotic dreams and most exciting love affairs. In the twentieth century such discoveries were more or less reserved to the field of psychoanalysis. Probably none of the ‘X-ray examinations’ of the brain will ever be capable of extracting the biographical details, intimacies, and covered-over psychological levels that psychoanalysis has done. But the point is different: psychoanalysis, without any doubt, has had a great influence and has changed many things enormously, but it has not become a standardized method for bio-psycho-politics. This is probably not due mainly to the fact that the assumptions of psychoanalysis are mistaken, or that the unconscious and the drives are not attractive to such social engineering. The real problem is that psychoanalysis is too complicated, too unwieldy, too difficult to practice, and needs too much time . . .

The alteration [from psychoanalysis to the application of neuroimaging techniques] could lead to the danger that the variety and relevance of mental life will be evaluated according to their ability to be visualized . . . The prize for such a development is that the investigation of the deeper connections, correlations, explanations, calculations, and narratives–in other words the historic, scientific, textual linear thinking–will be displaced by a new, visualizing, "superficial" kind of thought. In respect to the sciences of human beings, this means that the "deep digging" for which psychoanalysis stood might be replaced by the superficial insights of neuroimaging pictures. In this case, understanding human beings would turn into an "externalization of materialized forms of representation." I don't mean that the subject will be eliminated, but another anthropology could turn into reality which–in a double sense of the word–would produce structures of the surface [13].

Taking Hagner's analyses and warnings seriously, I gained a new appreciation of a long tradition that has been trying to bridge between psychoanalysis and the neurosciences and other scientific disciplines, which seems to be not very well known. This is the dialogue between psychoanalysis and so-called cognitive science. In this dialogue it has always been very clear that careful reflection on epistemological and methodological problems is essential to any careful and fruitful comparison of models developed in the different fields. Again and again researchers have to realize that mental processes will never be directly observable. "Precisely because mental phenomena are not directly observable and therefore, from the purist standpoint of natural sciences, do not even exist, it is fundamentally impossible to regard them as *explananda* and to look for an explanation for them in the sense intended in the natural sciences" [14]. To summarize this epistemological finding in a simplified way: One can never observe mental processes directly, "objectively." Only subjects can describe mental processes–the mind! One can also never directly compare data collected in different fields by different research methods: there is no such things as "looking directly into the functioning of the brain"! All we have are explanations, interpretations, in other words "models" which try to explain the collected data in a particular field of observation as adequately and productively as possible–models which can then be tested, validated, and modified by further experiments or empirical studies in the neurosciences on the one hand or by further clinical observations in the psychoanalytic situation in psychoanalysis on the other hand. For this reason, getting involved in a dialogue between psychoanalysis and the neurosciences means starting an exchange on models based on very different kinds of data, research instruments, and so on.

Developing and comparing models of the mind is the specific research topic of cognitive science, which was developed as a separate scientific discipline in the 1960s and 1970s in various centers around the world.

Cognitive science was at that time defined as an interdisciplinary approach to studying the mind and includes artificial intelligence, psychology, neurosciences, linguistics, and philosophy. More recently, biology and engineering have also been included. Given the novel focus on embodiment, as we will discuss below, cognitive science could be viewed as the scientific discipline that tries to unravel the mechanisms underlying intelligent behavior. This includes, but is not restricted to, the study of the mind. Cognitive science specializes in integrating knowledge from different disciplines into theoretical models, and in discussing critically the fundamental problems, e.g., epistemological and methodological, connected with this attempt [7]. This is one reason for the introduction of this research tradition into this volume devoted to the dialogue between psychoanalysis and the neurosciences.

Just a few more remarks on some characteristics of this scientific approach: In contrast to traditional empirical science that uses an analytic methodology, cognitive science employs a synthetic approach. It can be characterized as "understanding by building." In other words, in order to understand some aspects of a natural system–e.g., memory–an artifact is constructed that mimics the natural system. The standard "artifact" is a computer model, which is now used widely in virtually all the sciences. In psychoanalytic research, computer simulations have been used since the 1960s to test the internal consistency and the terminological and logical precision of complex theories. For example, Colby and Gilbert [15] used computer simulation to validate parts of the models explaining neuroses. Colby [16] studied the determinants of paranoia [17]. Wegmann [18] developed a computer simulation model of the counter will. Clippinger [19] simulated the cognitive processes of a patient at the beginning of a psychoanalytic session. The research group of Ulrich Moser in Zurich undertook a whole series of such studies simulating defense and dream mechanisms [20].

Other researchers have used computer simulation to develop models of different types of memory phenomena [21]. One problem with most computer simulations of memory is that there is no direct interaction with the environment: the input and output of these models are typically feature vectors predefined by the designer of the model. This contrasts strongly with reality: humans interacting with their environment are exposed not to "feature vectors," but to continuously changing sensory stimulation. This is why many cognitive scientists have turned to using autonomous robots in the last years, because they have to interact with the real world without human intervention. In other words, if the robot is to display appropriate behavior, it must always include the complete sensory–motor loops. One of the central advantages of using robots is that the sensory stimulation from different modalities (visual, optic, auditory, proprioceptive) can easily be recorded and analyzed. Moreover, the developmental processes which are typically encoded in an artificial neural network–a model of a biological neural system–can be monitored precisely. Thus, in contrast to living systems, changes in the neural network can be observed and traced over extensive periods of time. This is especially important within the context of a developmental framework, which is why this research methodology is increasingly being used in developmental psychology. In other words, the internal changes in an organism during the interaction with the environment can be studied in detail by this procedure. Metaphorically speaking, we can look into the brains of the robots as they are performing their task and therefore learn how experiences influence the (artificial) neural network.[2] For this reason, experiments with these mobile systems can offer important insights in the field of basic science.

Over the last 20 years the authors of this chapter have published a series of papers trying to apply new concepts developed in this field, which is now called "embodied cognitive science," to central issues of modern psychoanalysis. We think that psychoanalysis has much in common with cognitive science: it also deals with (unconscious) mechanisms underlying adaptive or maladaptive functionings of the mind and tries to integrate complex (clinical–empirical) findings in theoretical models. Just to mention one of many impressive examples: for years Joseph and Anne-Marie Sandler [22] worked at integrating new insights from memory research into their conceptualizations of a "present unconscious" and "past unconscious," thus modifying and differentiating one of the central issues in psychoanalysis: unconscious functioning. For us, the analogy in research questions and aims of psychoanalysis and cognitive science is one reason why this interdisciplinary dialogue promises to be fruitful [17, 23–27].

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[2] Of course, compared with psychoanalytic processes, empirical research using robots as a methodological tool deals with far less complex research question. Nevertheless we think that, in terms of "basic science," this research field can inspire our thinking on fundamental research questions, such as the functioning of memory. "Looking into the brain of a robot" allows changes in the neural network to be observed; it does not solve the epistemological problem of how to study mental processes by the methods of the natural sciences.

In this chapter we will focus on just one aspect of this research direction, in which the dialogue with modern neurosciences is particularly important: *interdisciplinary memory research*[3].

Besides, remembering–as one central function of memory–is still a central issue in clinical psychoanalysis. Most contemporary psychoanalysts probably share the common ground that inappropriate, psychopathological thoughts, emotions, and behaviors of patients are determined by unconscious fantasies and conflicts which were once adaptive in an unknown past situation. In other words, they believe that their patients are guided by their implicit memories of earlier pathogenic or traumatic[4] object-relations and experiences which are–unconsciously–repeated in their current feelings and symptoms. Sandler and Sandler [22] speak of dynamic templates of early experiences. But how do these "templates" function? How does memory work? Can patients remember "historical truth" or are memories mainly "constructed" narratives containing the truth about history which the patient can stand emotionally at the moment, but which, perhaps, do not have much to do with biographical facts [31–42]? Is it justified for Fonagy and Target [43] to conclude that " . . . whether there is historical truth and historical reality is not our business as psychoanalysts and psychotherapists." Why then is the remembering of a specific biographical situation often connected with a therapeutic progress, especially with severely traumatized patients, as analysts in many clinical papers claim [28, 44–50]? And assuming this proves to be true: in what specific transference situations do new memories occur? And why? In other words: *how we can recollect the past of our patients in the presence of transference in the here and now of the psychoanalytic situation?* And what kind of relevance do such memories have for the therapeutic process? All these questions are under intense debate within the psychoanalytic community at present [51–59]. One aim of our paper is thus to attempt to offer some integrational suggestions on these

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[3] This paper is a modified and shortened version of our paper "Remembering a depressed primary object?" [27]

[4] We cannot summarize the ongoing debate on trauma here (see e.g. Bohleber [28]). As used here, "trauma" designates events leading to the organism's being flooded by sensory stimulation that cannot be regulated. We find Terr's differentiation helpful [29, 30]: this distinguishes between two types of trauma, one referring to a single experience of abuse or maltreatment (type I) and the other to repeated exposure to extremely distressing events (type II). The case example (Mr. X) in the present paper underwent type II trauma in his early childhood (see below).

questions, which are central to contemporary psychoanalysis, based on the current discourse on memory in cognitive science. Of course we can only give a short impression of the direction and some of the results of this theoretical debate within the limits of this chapter.

A challenging side of interdisciplinary dialogue in practice is that each partner has to dive into the fascinating, but often strange and confusing world of the other scientific discipline. As is well known, every science has developed its own knowledge base, terminology, and research methodology–and also its specific ways and styles of argumentation and communication. This applies as much to psychoanalysis as to cognitive science or the neurosciences. We hope that our interdisciplinary readers will enjoy and tolerate the journey to unknown scientific worlds and finally share our own experience that the "triangular" perspective from a foreign discipline on one's own practice and theories is an innovative and worthwhile enterprise.

In the hope of softening some troubles of such an interdisciplinary journey, we have chosen to discuss some of the insights into memory in embodied cognitive science not exclusively theoretically, but in the context of a clinical case study, a short sequence of a psychoanalysis with a severely ill psychosomatic patient[5] who has been in psychoanalysis with me (M.L.-B.) for several years. The case material is intended to illustrate only one point: how can we understand the pathogenic past of our patients in the present of transference? Our main arguments in some theoretical points in this illustration are:

- Memory is a theoretical construct explaining current behavior by reference to events that have happened in the past. This implies a clear separation between describing behavior in terms of memory (e.g., remembering the past in the "here and now" of transference) and underlying brain mechanisms subtending this behavior (a frame-of-reference problem as part of the epistemological reflections mentioned above in the dialogue between psychoanalysis and the neurosciences).
- Memory is not to be conceived of as stored structures (the computer metaphor, or, in psychoanalysis, "stored" representations, etc.) but as a function of the whole organism, as a complex, dynamic, recategorizing, and interactive process which is always "embodied", i.e., based on

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[5] In the above-mentioned paper [27] we illustrated our memory concepts by a sequence from a psychoanalysis of a borderline patient suffering from psychogenic frigidity and sterility.

actual sensory–motor experiences, and manifests itself in the behavior of the organism.

- Memory is not an abstract cognitive function but is embodied, and as such is a property of the entire organism. It is important to realize that embodiment does not simply mean "nonverbal": it implies that there is a coupling between the sensory and the motor processes which has significant implications for neural information processing.
- Memory always has a subjective and an objective side. The subjective side is given by the individual's history (developmental perspective), the objective side by the neural patterns generated by the sensory–motor interactions with the environment. This implies that both "narrative" (subjective) and "historical" (objective) truth have to be taken into account in achieving stable psychic change.

We start our chapter with a short presentation of the clinical background, "Hypotheses Concerning the Biographical Roots of Psychosomatic Symptoms of a Young Man." Then we focus on "Recollecting the Past in the Present: Remembering in the Transference" and discuss classical and recent, biologically oriented, embodied models of memory processes. Finally, in the section entitled "Conceptualization of Early Memory Processes," we will illustrate what these models may add to a genuine psychoanalytic understanding. We hope to show that clinical psychoanalytical theorizing of early memory can be integrated with empirical and conceptual findings and summarize what, in our view, we can learn from this endeavor (4)

Hypotheses Concerning the Biographical Roots of Psychosomatic Symptoms of a Young Man

[Case history by Marianne Leuzinger-Bohleber]

A 30-year-old computer science student (Mr. X) was looking for psychotherapeutic help in a desperate life situation. Because of heavy psychosomatic symptoms (eating and sleeping disorder, migraine, attacks of dizziness, skin irritation) he had not been able to pursue his studies for the past 5 years. He was living completely isolated and seemed increasingly to be developing paranoid fantasies. The only relationship he maintained at that stage was to his brother, who is 3 years younger than him. However, this relationship was crumbling, chiefly because he had insulted and even physically attacked his brother in outbursts of rage that neither he nor his brother was able to understand.

Mr. X arrived with the explicit desire to start a psychoanalysis. He had read about it and considered the method to be the one right for him. I had my doubts concerning this indication because I considered Mr. X to be a borderline patient and asked myself whether high frequent treatment would be the right treatment decision for him. During the first year of psychoanalysis Mr. X controlled the distance between us by means of marked intellectualization and an almost complete repulsion of emotions during the sessions. It was almost impossible to obtain new analytical insights: the analysis seemed mostly to cover conscious processes. Still, the patient arrived on time for the appointments and insisted vigorously on replacing any appointments that I had to cancel. He seemed to existentially need the holding function of psychoanalysis. He changed his behavior–although this did not appear to be connected to any insights gained in the psychoanalysis. He attended lectures and courses again and was more able to concentrate. Also, the psychosomatic symptoms remitted somewhat, which led Mr. X to say before we entered the summer break: "*The treatment does me good . . .*"

The sequence I would like briefly to describe now took place after this first long summer break. Mr. X arrived obviously distraught at our first appointment. He began straight away to heavily insult me and seemed to become absolutely beside himself with rage and anger because I had dared to disappear, to go on vacation for 4 weeks. This was irresponsible, selfish, and showed that I was not at all interested in my job and in my analysands. "I doubt whether you have had proper training as an analyst at all . Maybe you are just a 'run-of-the-mill' analyst" . . . I was surprised by the violence of his anger and despair, and during the session failed to reach him emotionally or by means of an interpretation of the experiences he had had due to the separation etc. Although it was possible to address his severe reaction to the separation and to prevent another outburst of rage, Mr. X instead fell into a long silence, which for me was of an even more frightening quality than his insults.

Some extremely difficult weeks followed. Mr. X seemed to only be able to choose between two states of mind on the couch: either heavy insults, anger, and attacks, or else silence and retreat. As to the content, I noticed that his attacks were mostly aimed at my analytical function. Mr. X insulted me not only as stupid, restricted, and unable to understand him even in the widest sense, but also as incompetent and not professionally trained. As a consequence of his attacks and extreme silence, I found myself confronted with a severe feeling of impotence, inadequacy, and even depressing self-doubts. However, the most difficult thing was to bear the physical reaction: his attacks during the sessions finally caused an inner tension to that extent that I began to feel sick and from time to time even suffered from stomach cramps–psychosomatic reactions which are unusual for me during psychoanalytical sessions. I then sought a better understanding of what had happened in the psychoanalytical session by means of a supervision session with an experienced colleague.

We presumed that the enormity of the attack and the silence indicated a traumatization suffered in a very early stage of Mr. X's development, probably during his first year of life, in a phase of development in which physical and affective states of mind can not yet be either enclosed or symbolized. Had he suffered from an early traumatization, perhaps caused by separation from the primary object which I felt in my depressive countertransference feelings? The discussion with my colleague had, for me, mediated a certain distance and enabled me to increasingly reflect critically on my fierce reactions and countertransference fantasies. Not long after, Mr. X, following a session that had included heavy outbursts of rage, arrived a bit calmer to the next appointment. I carefully communicated my supposition that the long summer break could have led to an intense reactivation of unbearable feelings of dependence and desolation, which he could have tried to cope with by means of extreme aggressive attacks. I asked, following an intuitive idea, if he had, after sessions like the one before, felt any physical reaction. He told me that he had "felt sick throughout his whole body," that he had not been able to eat, and that he had suffered from heavy stomach cramps. I was surprised by the analogy to my own psychosomatic symptoms during and after such sessions. I told him that the total quality of these states of mind led me to assume a reactivation of very early experiences, " which could have been preserved in the body" and which "might try to become accessible to our analytical comprehension by this means which both of us find unbearable. Do you know, by chance, whether you suffered from a severe illness or an eating disorder during your first year of life, or whether you and your mother were separated?" Mr. X answered in the negative, but called his mother and found that 6 weeks after giving birth she had had the impression that she did not have enough milk. She abruptly stopped breast-feeding her baby and used baby food. Her baby reacted with a strong allergy, with a painful, itching skin irritation over his whole body. The mother told the analysand that she had not been able to touch the infant, that he always screamed, and that it had been almost impossible to calm him. She almost fell into despair, but after 3 months she had, in her words, "everything under control," and gave a different formula food to the child. As a consequence, the symptoms disappeared. "And you've been shouting at me since the summer holidays that I did everything wrong, that I gave you 'the wrong analytical nutrition,' that I had changed completely in the holidays and that I refused to give you the right 'analytical nutrition' which could make everything turn out well again . . . The analytical sessions no longer do you any good as they used to before the holidays–they are just horrible now. Every contact with me seems to be unbearable . . ."–Mr. X began to cry for the first time in the psychoanalysis.

During the following weeks we were able to successively understand the reactivation of the early traumatization: the trauma laboriously found its way into his images, visualizations, and finally his language: it had become conscious and in the following years became successively better understood as one of the unconscious sources of the patient's severe psychosomatic suffering.

Recollecting the Past in the Present: Remembering in the "Transference"

The question if and how such early traumatizations can be remembered and then be understood in the transference have been debated at length and in great detail within the psychoanalytic community during the last few years–an interesting discourse that we can do no more than mention here. The following section describes some of our own efforts to contribute to this discussion.

Models of Memory in "Classical Cognitive Science"

Fifteen years ago we discussed these questions in a paper in the International Review of Psychoanalysis, analyzing three key scenes of a psychoanalysis [25]. In these analyses we tried to start an interdisciplinary dialogue between "classical cognitive science" and psychoanalysis on questions relating to memory processes. In 1983 we had begun to refer to the recent approaches to memory–which were much discussed at the time and were expounded by Schank among others in his book *Dynamic Memory* [60]–to explain memory processes during psychoanalyses and seemed to shed new light on core psychoanalytic concepts, such as repetition compulsion, the rule of abstinence, and the working through of central conflicts in transference. Among other things, we showed that the focus concept of psychoanalysis can be described very precisely by a memory structure that resembled Schank's TOP, the "thematic organization point" [60]. We showed that the psychoanalytical concept of a focus, as illustrated by the "triangle of insight" (Fig. 1) connecting analogous structures of an *current conflict* with those discovered in the *transference* and *biographical information*, corresponds in detail to Schank's TOP. We illustrated this hypothesis by analyzing some information from the psychoanalysis of a severely depressed woman. We found analogous components in the *current conflict situation* (feeling exploited by her husband), the *transference* (being convinced that her analyst would "only" be interested in pursuing her own goals, e.g., earning money), and a *traumatic experience of early childhood* (being exploited by her mother as "protection" against Russian soldiers during a frightening night-time escape during World War II). We discussed how in all these three "key scenes" we found the components of the TOP–a finding which seemed to deepen our clinical and theoretical understanding of the psychodynamics of the analysand and her memories evoked in psychoanalysis.

We could now apply this memory concept in an matter analogous to the above case. In the *current conflict* with his brother Mr. X. experienced a sudden, unexpected change: he lost his good relationship to him from

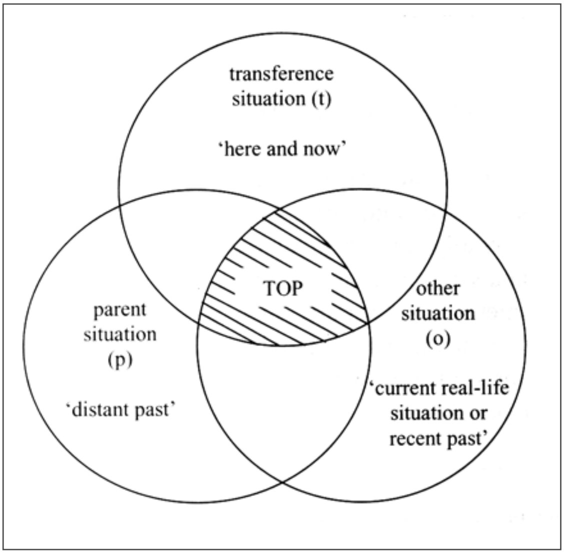

Fig. 1. Triangle of insight

one day to the next and felt enormous rage and furious impulses towards him. In the *transference* we observed similar reactions: after the summer break the analyst was no longer experienced as a "good object" but an unempathetic, cold one responsible for painful and unbearable feelings of rage, disappointment, and despair in the patient and refused to help him find a way out of his present unbearable inner and physical state. Inquiring of his mother, Mr. X obtained the *biographical information* about the allergic milk reaction in the seventh week of his life which exposed him for 3 months to unbearable bodily states, and the "refusal" to offer a solution, to get him out of the painful situation. In all the three topics we find the same (cognitive) components of the TOP.

According to Schank, a TOP includes the following components: a goal configuration, expectations of plans and outcomes, actual plans and outcomes, and explanations of discrepancies. TOPs are abstract memory structures that are stored and are usually unconsciously recalled by so-called demons. "Demons" are programs that are continuously on the alert for an event to take place; in this case the event is the occurrence of a situation that is structurally similar to an earlier one. Although Schank talks about "dynamic memory," this concept presupposes a "static" notion of memory. The dynamic aspect of memory in Schank's concept is the establishment of new references to other memory structures. For example, if there is a new failed expectation, a reference is established from the failure point to a representation of the situation from which the (failed) expectation was generated. However, this notion still implies a kind of storehouse in which memory structures–knowledge–are kept, like records stored on a disk in a computer. Long-term memory is understood by analogy to Aristotle's famous notion of memory as a wax tablet on which experiences are written. If the demons recognize a certain pattern in current information as being similar to a memory structure stored in the long-term memory, this structure will be transferred to the short-term memory, where it then is accessible to conscious remembering.

Schank thus formulates a "classical" definition of memory which is still very popular, and a significant number of psychologists, psychoanalysts, memory researchers, and nonscientists also maintain this view. If one asked a layperson what memory was, more than likely his answer would be something like "a place in the brain where information is stored." In everyday language, we often describe mental processes as objects in an actual physical space. For example, we speak about storing something in the memory, or searching through our memory, or of holding ideas in our minds; like physical objects, memories may be lost, hard to find, and so forth. Memory as a stored structure is also found in many textbooks today [61]. Baddeley [21] uses the following definition: "Human memory is a system for storing and retrieving information, information that is, of course, acquired through our senses." In essence memory is viewed as information that is stored and later retrieved.

Models of Memory in Embodied Cognitive Science: Memory as a Dynamic and Constructive Process of the Whole Organism

The classical conceptualization of memory leads to many theoretical problems, as has been discussed extensively in the cognitive science literature [5, 27, 63–69][6] and in the psychoanalytic literature [22, 38, 41, 57, 70–72]. It cannot, for example, be used to give a plausible explanation of how knowledge can be applied repeatedly to new situations, i.e., how learning processes occur that require new situational analysis, how problem solutions can be transferred from one domain to another, and how new categories are established. Just one example: it is no problem for us to recognize the Jupiter symphony by Mozart even if a neighbor is practicing parts of it on the piano. This process of remembering cannot be based on simple pattern matching because the current pattern (piano music) is different from the earlier one (orchestral music). It would therefore not be helpful to just store a pattern somewhere in the brain. Recognition is rather a constructive inner process relating a past experience (listening to the symphony) to the present situation (hearing Mozart played on the piano).

For this reason (and many cognitive scientists today agree) it is necessary to conceptualize memory in a fundamentally different way; in other words, a change of paradigm seems indispensable [5, 65–67, 73]–a topic that we want to discuss now.[7] Most of these alternative conceptual-

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[6] It should be mentioned that Ashby [62], one of the fathers of cybernetics, long ago questioned the above- described memory theories of "classical cognitive science," but his criticisms have remained unheard in the scientific literature.

[7] Although our analyses are still plausible to us today, from our present point of view we would say that we committed a so-called "category error" then (see also p. 79 on the frame-of-reference problem). We only became aware of this because of the recent discussions of memory in the context of embodied cognitive science [5]. We had not realized that though we had given a detailed descriptive analysis of memory feats with the help of the TOPs, we had nevertheless said nothing about the underlying memory processes.

izations capitalize on the notion of embodiment which means–as we will discuss below–that memory can only be understood in the interaction of an organism with its environment. It turns out that if memory is conceptualized according to this new paradigm, some of the fundamental problems in understanding memory can be resolved.

For a long time I found it difficult to understand thoroughly the fundamental differences between the concepts of memory in classical and in embodied cognitive science, which has been intensively influenced by the neurobiological brain research of the last years. We therefore want to discuss three of the central topics quite extensively:

- memory as a theoretical construct
- the frame-of-reference problem
- the developmental perspective
- illustrating them briefly with psychoanalytic discourses and our case material.

Memory as a Theoretical Construct

As we will illustrate, the ideas developed in this section naturally connect to clinically relevant concepts, although the mode of argumentation might at first seem unfamiliar or strange for readers unacquainted with this research tradition (see Introduction). Cognitive scientists often use metaphors or short stories to explain their ideas. For instance, Ashby wrote this "story" discussing memory as a theoretical construct: "Suppose I am in a friend's house and, as a car goes past outside, his dog rushes to a corner of the room and cringes. To me the behavior is causeless and inexplicable. Then my friend says: 'He was run over by a car six months ago.' The behavior is now accounted for by reference to an event of six months ago" [62]. In other words, the explanation is not given in terms of the current internal state of the dog, but by reference to an event in the past. *Memory, then, is a theoretical construct that connects the state of the individual in the past and the influence the event had on the individual to the behavior in the current situation.* This theoretical notion of memory is to be clearly distinguished from the mechanisms mediating these processes. In this sense, memory is not something sitting somewhere in a box inside the head of the dog, but is a theoretical construct and is attributed to the complete organism. A similar idea is also reflected in the so-called “ecological perspective” on memory, where the function of memory within natural contexts is investigated [74].

This notion of memory is very natural to psychoanalytic thinking. The analyst observes in a specific context inappropriate behavior that he or she does not understand. The goal then is to find analogies to behaviors in earlier situations (events that have happened in the past) that were perhaps adaptive at the time, and by invoking the concept of memory might explain the person’s current behavior.[8]

In our case history we described how the analyst did not understand the sudden extreme change in the behavior of Mr. X after the summer break. She understood that his rage and attacks on her had to do with the separation, but she had no idea why the affects were of such intensity, or why the anger and disappointment obviously had such a total and archaic quality connected to obviously psychosomatic reactions in the patient as well as in her own countertransference feelings. Only the information on the sudden change in his bodily state when his mother changed his food in the 7th week of his life made it possible to understand that the extreme reaction in the presence of the psychoanalytical relationship had been brought about by “memories” of preverbal early experiences.

From a theoretical perspective it is important that Mr. X’s memories were not “retrieved from a box inside his head” but were products of a theoretical construct connecting the observed state of Mr. X in the analytical situation with his probable experiences in early childhood.

Again, in order to connect present with past information, nothing needs to be said about the internal neural mechanisms that mediate this transfer. These considerations represent an instance of the notorious frame-of-reference problem, which will be discussed next.

The Frame-of-Reference Problem

The frame-of-reference problem states that in explaining memory functions we must make a clear distinction between observable behavior and the internal brain mechanisms that, in the interaction with the real world,

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[8] Similar theoretical considerations underlie the postulate of unconscious fantasies and conflicts that determine current behaviour in typically unknown ways.

lead to a particular behavior. This implies that behavior cannot be reduced to internal processes, nor to brain processes for that matter. Doing so would be to commit a category error (to use a philosophical term). This seems trivial, but then it is even more surprising that there is great confusion in the literature about this issue [27, 39, 65].

Applied to memory, it implies that a clear distinction must be made between the theoretical construct and underlying mechanisms responsible for mediating between the past and the present. Ashby's concept of memory is neutral to the mechanisms by which it is implemented in the organism. In biological organisms the mechanisms are to be found at the level of neural plasticity, whereas in artificial systems such as robots or computers they are situated at the level of switching circuits implemented in silicon. Another example would be immune systems, which can also be described by invoking the concept of memory in the interaction of the organism with environment [39, 68]. In all these cases, it makes sense to use the concept of memory.

We can only describe on an observable level when and in what interactional context Mr. X was able to "remember" his unbearable bodily state from his first weeks of life. Our observations are exclusively based on the psychoanalytical situation (analysis of Mr. X's behavior, feelings, and verbalizations, the analyst's countertransference reactions after the summer break, etc.)–we never had the possibility of "looking straight into Mr. X's brain," and thus cannot know what neural and neurophysiological processes had been activated when Mr. X was able to remember the childhood experiences. This differentiation seems simple. Nevertheless, you can often find a confusion between the level of description of memory processes and underlying brain mechanisms in the literature. We ourselves made this category error in the above-mentioned 1986 paper [25].

Some authors [67, 75] have tried to ferret out the mechanisms underlying memory in biological systems. It is important to emphasize that these mechanisms should not be seen as "being" the memory, but rather as implementing those processes which, as the organism interacts with the environment, lead to behavior that we try to explain by invoking the theoretical notion of memory.

Thus a clear distinction must be made between description of behavior in terms of "memory" and the brain mechanisms subtending this behavior (see also focus box in [27]).

The Developmental Perspective

A third important aspect of the methodology of embodied cognitive science is the developmental perspective. When conceptualizing memory processes, embodied cognitive scientists do not primarily attempt to directly model the internal processes of memory. The goal of their modeling is to define the developmental and learning processes and to explain the current behavior as resulting from these processes as the individual matures and interacts with the real world. The advantage of this perspective for modeling memory processes is that fewer assumptions have to be made about internal representations. Moreover, in this way, we are forced to work out the underlying mechanisms that eventually–during development–lead to the observed behavior. Much of the work in embodied cognitive science is based on a developmental perspective. For instance, constructing robots means implementing their "memory" as self-learning systems. This means that the robots change their knowledge and "memory" automatically by interacting with the environment. As already mentioned, unlike when analyzing living organisms, the researcher is able to "look into the robot's brain" and observe the continuous changing of its neural network interacting with its environment. In other words, the researcher can study with great precision the influence of the developmental or learning history of the robot on his internal representation (neural network). According to the findings of this kind of research, memory has therefore to be understood as a product of developmental processes in constant change (see below).

This perspective is compatible with psychoanalytic theorizing, where the developmental view is one of the underlying principles.

As described above (see p. 71), the analyst tried to understand the present behavior of Mr. X in connection with his biography, his idiosyncratic development. The "objective" information from Mr. X´s mother, that her baby son had suffered from an unbearable bodily state and that she had not been able to comfort him for 3 months (partly also because she had suffered from severe postnatal depression), seemed to have many analogies to the analyst's countertransference feelings of helplessness, total insufficiency, identification with the painful psychosomatic symptoms of the analysand, and finally depression. The developmental perspective has thus, of course, additional relevance for psychoanalytic treatments, but it is beyond the scope of this paper to also describe Mr. X's processes of change during psychoanalysis itself in detail.

From our theoretical perspective it is relevant that memories are not seen as one-to-one retrievals of certain historical events but as a continuous process of change of the whole organism in interaction with its environment; or, to put it provocatively: each process of recalling changes the memory, although at the same time this process is not an arbitrary construction but a complex approach towards the historical truth of earlier developmental experiences (see below).

Let us take another example to further illustrate the view of an embodied memory taking account of all three of the basic theoretical topics just mentioned:

We observe the 6-month-old infant Peter taking apples and newspapers from a table. Peter puts everything in his mouth–apples and newspaper. After a while he only grasps the apples, leaving the newspapers aside. From our perspective as observers of this scene we suppose that Peter has learnt from experience and "remembers" that apples taste better then newspapers. Therefore he now prefers to taste apples. Analyzing the infant's behavior, we (the observers) postulate that Peter selects apples by reference to his former experiences: according to the observer he has functioning memory at his disposal. We have defined memory from an "outside" perspective–observing the infant's behavior–and not by looking into his brain (memory as a theoretical construct and frame-of-reference problem). It is an attribution to the infant as a whole (we evaluate his whole behavior), not to one part of the infant, say its brain. This means that we do not have to postulate any kind of internal representation in order to describe his behavior (or his memory).

Another important observation is that the infant has developed categories: he can now differentiate between apples and newspapers (for further discussion see [27]).

Neural Implementation of Memory as a Dynamic Constructive Process

Working out the biological basis of categorization in the real world is precisely one of the main goals of Edelman [66, 68]. Edelman's ideas are of interest not only because of his focus on processes of self-organization which provide the organism with the required adaptive potential, but because he considers memory from an embodied perspective. The notion of "memory as recategorization" is based on processes of sensory–motor coordination which in a very direct sense anchor memory, or its manifestation in behavior, in the interaction of an individual with the environment.

We want to pick up two central ideas of Edelman here, sensory–motor coordination and value systems. The general framework proposed by Edelman suggests that the results of motor activity are an integral part of categorization. “While sensation and perhaps certain aspects of perception can proceed without a contribution of the motor apparatus, perceptual categorization depends upon the interplay between local cortical sensory maps and local motor maps. The strongest consequence of this assumption is that categorization cannot be a property of one small portion of the nervous system” [66]. Thus, categorization involves not only the brain but also the sensory–motor apparatus, a key implication of the principle~~s~~ of sensory–motor coordination. The essential mechanism of categorization in Edelman’s framework is a parallel sampling of the environment by multiple sensory maps within the same modality and between different modalities. This sampling is a process of sensory–motor coordination in which various maps pick up different, but temporally correlated, signals from the environment. These correlations play a fundamental role in categorization (see below). Edelman illustrates the principle of sensory–motor coordination as shown in Fig. 2.

Thelen and Smith [76] point out that “this perfect temporal association of multimodal information is perhaps the only perceptual invariant that spans all ages, contexts and modalities. We believe, with Edelman, that this correlation is the primary link between the mind and the world.” This is a central point in embodied memory theory: sensory–motor coordination structures the high-dimensional sensory space by inducing regularities. The temporal correlation of signals in the neural maps related to the different sensory modalities, generated by the interaction with an object, is the most basic example of such regularities. Which of these patterns of correlations are chosen or selected in the process of categorization is modulated by a value system.[9] Value systems are basic evolutionary adaptations that define broad behavioral goals of an organism. For example, if an organism (like Peter) succeeds in grasping an object or sticking it into its mouth, a value signal is generated that enables the

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[9] There is an extended debate in the literature about the nature of value systems that we cannot go into here. Although value systems are related to motivation and emotion, the way the term is used here, they are viewed as elementary, directly related to biological processes of adaptation. In essence, value systems have evolved to guide the processes of self-organized adaptation and category formation; they determine very broadly what is good for the organism.

Fig. 2. Principle of sensory–motor coordination. *Top panel:* Primary repertoire. The first interconnection of nerve cells is the result of developmentally controlled selection. Briefly, Edelman takes this to mean a selection process among the multiplicity of neurons competing topobiologically with one another. Nerve cells send out axons which are attracted by a so-called nerve growth factor, which the nerve cells need to survive. This factor is produced by sensory epithelial cells and other kinds of cells. It brings about a close connection with nerve tissue and is absorbed by the axons and dendrites. Thus neurons compete for this connection with the "sources" according to Darwin's principle of the survival of the fittest. In the unsuccessful cells, programmed cell death results. This means that cell wandering and cell death turn an initially immense surplus of nerve cells into tissue which looks rather like the top line of the figure. Edelman calls this network the "primary repertoire". It is the matrix of nerve tissue and of the brain. It is formed on the basis of a chemical process, genetically controlled yet subject to various influences, i.e., it is a product of heredity and environment – a fascinating thesis for us psychoanalysts. So far no truly functioning circuit has been formed, but a network exists which can be further extended. *Middle panel:* Secondary repertoire. This further extension is based on the nerve cells' ability to be electrically active, a selection controlled by experience, generally leaving the anatomy unaltered. Synaptic connections are selectively strengthened or weakened by the experience (behavior) of synaptic connections in the existing anatomy. This mechanism, which underlies memory and a series of other functions, leads selectively to a multitude of circuit elements (with strengthened synapses) in the anatomical network. The multitude of these elements is what constitutes the secondary repertoire. For psychoanalysis it is significant to note that the mechanisms which lead to the primary and secondary repertoire sometimes mix. *Bottom panel:* Neural cards. The so-called cards develop from functional circuits. They consist of several ten thousands neurons working functionally in one direction. Thus every system of perception–the visual apparatus, the sensual surface of the skin, etc.–has many such cards, which are stimulated by qualitatively different impressions: color, touch, direction, warmth, etc. These cards are interconnected by parallel and reciprocal fibers which ensure the renewed and repeated entry, flow, and exchange of signals. If stimuli select the groups of neurons of one card, stimulation of the cards connected to it follows simultaneously. By the reentry of the impulses a strenghtening or weakening of the synapses in a neural group of each card follows: the connections between the cards themselves are also modified. Thus new, selective qualities develop. (Figure reproduced with permission from [66].)

association of the activation in the neural maps corresponding to the different sensory and proprioceptive modalities. *In this way the organism is capable of generating categories on its own as it interacts with the environment.* To take our example again: Peter learns to differentiate between apples and newspapers by picking up apples and newspapers and sticking them into his mouth. The two sequences of events lead to different activations in the different neural maps (visual, optic, auditory, and proprioceptive) which are then, via the modulation of the value system, associated with each other. If a new situation (e.g., grasping a piece of soap) affords this, a new category has to be developed ("soap"–compared with "newspaper"–compared with "apples"). Because there is no limit to the patterns of sensory stimulation, new perceptual categories can be formed

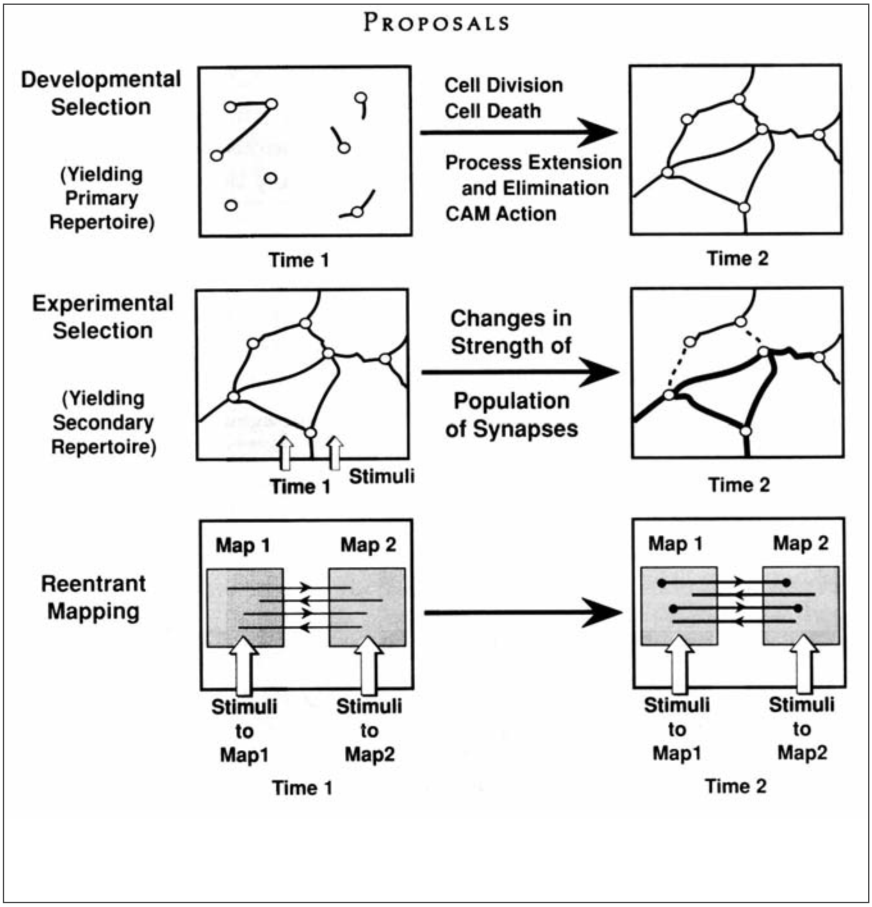

Fig. 2. See description on page 22

if they have distinct behavioral consequences. These associations between the different neural maps are the biases that shape the behavior of the organism in a novel situation. In this sense, they can be viewed as the neural basis of categorization and thus of memory. It is interesting to note that with each sensory–motor coordination these associations are changed. Edelman speaks of a never-ending process of recategorization[10] which allows the organism to adapt constantly to new situations by applying knowledge gained in past experiences.

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[10] The process of recategorization can be compared to the continuous process of assimilation and accommodation already described by Piaget.

This view leads to a definition of memory that sharply contrasts with the traditional view of memory as stored structures: " . . . memory is the enhanced ability to categorize or generalize associatively, not the storage of features or attributes of objects as a list" [66]. *Memory is defined as the ability of the whole organism to recategorize, a capacity which always stems from sensory–motor coordination processes.* A relation notion of memory is put forward by Clancey's definition [64]: *"Human memory is a capability to organize neurological processes into a configuration which relates perceptions to movements similar to how they have been coordinated in the past."* A detailed discussion of related memory concepts can be found in Pfeifer and Scheier [5].

In summary, Edelman's notion of memory is based on processes of sensory–motor coordination, i.e., it includes sensory and motor processes. The motor processes, but also the kind of sensory stimulation to be processed by the neural system, depend on the specifics of the sensors (their physical characteristics and shape and their position in the organism, e.g., "apple" and "newspaper" in the example above) *and* the motor system. As mentioned earlier, the buzzword used in the modern cognitive science literature is "embodiment." It is important to emphasize that categorization, and thus memory, is not only a matter of internal processing of sensory signals, but of sensory and motor processes. It is hard to overstress this fundamental point: perception is no longer viewed as an event of mapping a sensory stimulation (e.g., a stimulation of the retina) onto some kind of internal representation, but as an act that involves the senses as well as the motor system. Although this point was made over a hundred years ago by the American philosopher and psychologist John Dewey [77], it has not really been taken seriously. The implications for our understanding of memory are enormous, especially in clinical contexts. All of a sudden, memory is no longer a box where conscious or unconscious memories are stored, from which they can or cannot be retrieved, but a characteristic of a biological physical organism that interacts with the real world. Manifestations of memory can always be traced back to sensory–motor interactions, and as a consequence sensory–motor processes play an essential role in memory.[11]

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[11] It is important to note that sensory-motor coordination does not simply mean non-verbal: It implies that there is a coupling between the sensory and the motor processes, i.e. the two mutually influence one another. Biologically, this coupling is implemented via the neural maps which are embedded in the sensory-motor systems of the organism (see our description above).

We finally want to underline that from this perspective it is entirely obvious that the concepts of sensory–motor coordination, categorization, learning, memory, and perception are tightly intertwined and cannot be separated clearly from one another.[12] This is because the underlying mechanisms responsible for these phenomena are largely shared.

Conceptualization of Early Memory Processes: A Preliminary Comparison Between Current Biologically Oriented Embodied Memory Research and Clinical Psychoanalytical Research

Let us illustrate how this theoretical thinking can enhance our understanding of memory processes, e.g., "*recollecting the past in the present*"(in other words: remembering early experiences in a psychoanalytic situation), looking at our case material again. It can, in our view, be plausibly integrated with our psychoanalytical theorizing.

Summarized briefly using our psychoanalytic concepts: the memory of infantile object relations with a depressed, unempathetic, and helpless mother (who doubts whether she will be able to feed her baby, takes 3 months to find out that her baby is suffering from a severe allergy, etc.) were evoked in the transference situation. The sudden change in bodily state due to the allergic reaction was "remembered" by Mr. X unconsciously after the summer break. Relevant transference patterns from the traumatic early object relations and central unconscious fantasies were suddenly unfolded–a process (determined by projections and projective identifications, etc.) which the analyst did not yet consciously understand, but to which she responded by intensive countertransference feelings of insufficiency, depression, and strange psychosomatic reactions. After supervision she was able to regain interest in the psychodynamics

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[12] From this theoretical perspective it is therefore problematic to postulate that the different memory systems are separated from each other in an absolute way; the postulate of different memory systems is mostly based on findings made by the so-called neuroanatomic research method (e.g., the finding that patients with lesions in the hippocampus lose their procedural memory) [2, 4, 10]. However, as we have discussed elsewhere in detail, the conclusion that these findings can be interpreted as localizing the different memory systems in a particular region of the brain may be too simplistic [27].

of her analysand, in other words to regain an intermediate space, as Winnicott calls it, which is a prerequisite for visualizations and verbalizations of preverbal sensory–motor experiences, and also for fulfilling therapeutic functions such as "containing" and "holding." This could be one reason why she ceased to react analogously to a helpless, depressed object, but was able empathetically and actively to communicate to her patient her hypotheses concerning possible tragedies during the first year of life, motivating him to ask his mother for information about his early experiences. These early traumata in the object relation with his primary object, and the sudden, unexpected change in his existential unbearable bodily feelings and states, probably had a determining influence on the early development of his self and object representations, his lack of symbolization and psychological integration of his archaic aggressive and libidinal impulses, and therefore on the development of psychosomatic symptoms (in the sense of "body language" and indicators of "recollections" of his early painful bodily experiences).

But how had these early traumatic experiences during the first weeks of life been kept in Mr. X's memory? Does interdisciplinary research help us answer this question? Is it this even the right question in the first place?

As we have tried to show, current memory research indicates that memory processes are not to be conceptualized by analogy to the computer model of storing and retrieving information in a static and unchangeable sense. Memory always consists of new and constructive processes in the "here and now" of a current interactional situation (system–environment–interaction) which is indispensable for constituting memories. At the same time, this constitution of memories is not arbitrary, because the way the current system–environment interaction is structured and the way the sensory–motor patterns are interpreted are determined by the individual's history. Memories are constructed by analogy to previous situations with similar sensory–motor patterns. Although this physical stimulation is always subject to interpretation depending on the individual's history, the sensory stimulation itself is still "objective" and not arbitrary. This is a consequence of embodiment: sensory–motor states are, at least theoretically, measurable physical processes; the sensory–motor coordination is determined by the way the neural maps are integrated in the organism, which is, again, objective. In this sense memories result on the one hand from constructive processes but on the other hand are influenced by the "historical truth," which means that the processes that came historically first–i.e., those constituted to deal with the original (traumatic) situation–constrain the recategorization of the new, analogous situation. In this sense recategorizations in later interactional situations are related to the original trauma. Metaphorically we therefore could postulate that memory is always based on new and idiosyncratic narratives taking place in current interactional situations but at the same time contains traces of the "historical truth." As we mentioned briefly above, the "objective" biographical information (allergic reaction in the first weeks of life, etc.) proved helpful in the psychoanalytic sessions in that they at last made possible the recognition of these traces of the "historical trauma" in Mr. X's current behavior (e.g., seeing the analogies between his current psychosomatic and emotional reactions and those of a baby interacting with a depressive, helpless mother offering him the "wrong food"; see, e.g., [79]). Thus, we think that for stable therapeutic change in our patients, both approaches are indispensable: understanding the idiosyncratic ways of unconscious functioning [22, 48, 80–82], and attempting to understand the highly individual, biographical (historical) truth in the sense of understanding the "specific, undeniable reality of trauma" [28, 31, 83, 84].

To repeat our thesis once again: experiences and memories have an objective and a subjective aspect. The objective one is given by the patterns of sensory stimulation in a particular sensory–motor interaction, which is, in principle, physically measurable. The subjective aspect refers to how the individual experiences associated with these patterns, which are the result of an interpretation–a constructive process–determined by the individual's history The sensory stimulation to which the organism is "objectively" exposed is not a passively undergone physical stimulation, but rather is generated as the organism interacts with its environment. The patterns of sensory stimulation resulting from this interaction are structured and contain correlations which can be easily interpreted by neural mechanisms. The types of interactions in turn are a result of developmental processes. Experiences of extreme pain and bodily unease and loneliness, such as Mr. X had gone through as a baby in his first 3 months, have a determining influence because the developmental processes strongly depend on the appropriateness, richness, and structuredness of the sensory stimulation. Thus, it is the sensory–motor coupling which provides the basis from which the developmental processes can be bootstrapped.

In the intensive interaction of Mr. X with the analyst, these deficiencies in categorizing and in the inner object relations of the analysand became observable and thus opened a way to gradually understand their roots in detail;, in other words, *the unconscious recollections of past experiences* *with the primary object became observable in the transference relationship to the analyst.* In other words: in the psychoanalytic setting, the schema of “being with others” (with a depressed, helpless, uncaring “dead” mother) was not “evoked” like a stored knowledge structure from early mother–child interactions (in terms of the TOP discussed above), but actively “constructed,” unconsciously interpreting the sensory–motor stimuli that resulted from Mr. X’s seeing the analyst after the break and perceiving “radical”–according to his unconscious anxieties–change in the object (the analyst had a different hairstyle, tanned skin from sunny holidays, etc.)–a situation which had similarities to the original stimuli in the early mother–child interaction (“sudden change in the object”) and thus “triggered” analogous unconscious interpretations and fantasies (“the object has withdrawn, will not feed me well any more, will not be able to empathize with my bodily state, understand what is wrong with it, stop offering the “catastrophic food,” help to eliminate the unbearable feelings and impulses, etc.).

These (unconsciously) ongoing memory processes in the psychoanalytic situation enabled, as we tried to show, observations of transference and countertransference reactions which had been central to our growing understanding of unconscious procedures and fantasies (implicit memories). The (unconscious) perception of certain sensory–motor states and processes “triggered” the sensory–motor reactions (stomach pains, extreme bodily tension during and after the sessions, feeling sick) and the (unconscious) fantasies of the analyst in the analytic situation and finally enabled the analyst (with the help of supervision) to reflect on these countertransference reactions (extreme bodily reactions due to a very early trauma?). In other words: the psychoanalytic interaction was indispensable for the discovery of the unconscious determinants of Mr. X’s symptoms and psychopathology: to discover the past of the analysand in the presence of the psychoanalytic situation.

We have also attempted to illustrate how embodied memory research might explain the remembering of traumatic childhood experiences, namely as occurring in a new interaction with a “meaningful other” (i.e., in transference to the analyst). A situative, constructive understanding of interactions is for a prerequisite of remembering! Remembering is dependent on a dialogue in the inner and outside reality with an object, an interactive process, an integrative, “embodied” experience between two persons. Ms. X would not have been able to remember alone for herself lying in her bed at home (see also results from recent trauma research [51, 52, 84–87]).

It therefore seems to us that the clinical research of psychoanalysis in the last 20 years or so, which has postulated more and more radically that only a working through of traumatic experiences in early object relations in transference led to a structural change within our patients, receives interdisciplinary support from current biologically oriented memory research. It seems that these findings of the neurosciences and embodied cognitive science are "externally coherent" [88] with psychoanalytic insights. According to these findings we can summarize as follows:

- Remembering is not the activation of static stored information, but a highly dynamic process of recategorizations in the here and now of transference.
- Remembering is dependent on the system–environment interaction (inner or real dialogue with objects) and thus on embodiment. It is sensory–motor, and not only internal (in the sense of purely mental), because even during internal processing we have sensory–motor stimulations [89].
- Remembering is therefore dependent on the construction of "narrative truths" in actual or actualized object relationships. At the same time it can be seen as a constructive, creative process of approaching "historical truth." Edelman [67], Fuster [75], Pfeifer and Scheier [5], and others argue convincingly that social experiences are anchored in embodied structures–that means biology.
- Memory always has a subjective and an objective side. The subjective side is constituted by the constant interpretations of ones own history; the objective side by the neural patterns generated by the sensory–motor interactions with the environment.
- Remembering is a precondition for a process of recategorization of unconscious traumatic experiences and therefore for a structural change of behavior.

References

1. Freud S (1940) An outline of psycho-analysis. SE 23, pp 144–207
2. Kaplan-Solms K, Solms M (2000) Clinical studies in neuro-psychoanalysis. Karnac Books, London
3. Schacter DL (1989) Memory. In: Posner MI (ed) Foundations of cognitive science. MIT Press, Cambridge, pp 683–725
4. Tulving E (1985) How many memory systems are there? Am Psychol 60:385–398
5. Pfeifer R, Scheier Ch (1999) Understanding intelligence. MIT Press, Cambridge, MA

6. Meares R (2000) Intimacy and alienation. Memory, trauma and personal being. Routledge, London
7. Leuzinger-Bohleber M, Dreher AU, Canestri J (eds) (2003) Pluralism and unity? Methods of research in psychoanalysis. International Psychoanalytical Association, London
8. Röckerath K (2004) Neglekt und Anosognosie: Psychoanalytische Arbeit mit rechtshirngeschädigten Patienten. In: Leuzinger-Bohleber M et al (eds) Psychoanalyse als Profession und Wissenschaft. Kohlhammer, Suttgart, pp 310–330
9. Sacks O (1985) The man who mistook his wife for a hat. Summit Books, New York
10. Damasio A (1999) The feeling of what happens. Body and emotion in the making of consciousness. Harcourt, Brace, New York
11. Koukkou M, Leuzinger-Bohleber M, Mertens W (eds) (1998) Erinnerung von Wirklichkeiten. Psychoanalyse und Neurowissenschaften im Dialog, vol 1: Bestandsaufnahme. Internationale Psychoanalyse, Stuttgart
12. Leuzinger-Bohleber M, Mertens W, Koukkou M (eds) (1998) Erinnerung von Wirklichkeiten. Psychoanalyse und Neurowissenschaften im Dialog, vol 2: Folgerungen für die psychoanalytische Praxis. Internationale Psychoanalyse, Stuttgart
13. Hagner M (1996) Der Geist bei der Arbeit. Überlegungen zur Visualisierung cerebraler Prozesse. In: Borck C (ed) Anatomien medizinischen Wissens. Fischer, Frankfurt, pp 259–286
14. Welding SO (2003) Kritische Glosse: Das Problem der naturwissenschaftlichen Erforschung des Mentalen. Psyche Z Psychoanal 57:658–666
15. Colby KM, Gilbert JP (1964) Programming a computer model of neurosis. J Math Psychol A:405–417
16. Colby KM (1975) Artifical paranoia. Computer simulation of paranoid processes. Pergamon Press, Elmsford
17. Colby KM, Stoller RJ (1988) Cognitive science and psychoanalysis. Erlbaum, Hillsdale, NJ
18. Wegmann C (1977) A computer simulation of Freud's counterwill theory. Behav Sci 22:218–233
19. Clippinger JH (1977) Meaning and discourse. A computer model of psychoanalytic speech and cognition. Johns Hopkins University Press, Baltimore
20. Moser U, Zeppelin IV (1996) Der geträumte. Traum. Wie Träume entstehen und sich verändern. Kohlhammer, Stuttgart
21. Baddeley A (1997) Human memory (rev ed). Psychology Press, Hove
22. Sandler J, Sandler AM (1997) A psychoanalytic theory of repression and the unconscious. In: Sandler J, Fonagy P (eds) Recovered memories of abuse. True or false? Karnac Books, London, pp 173–181
23. Bucci W (1997) Psychoanalyse und cognitive science. Guilford Press, New York
24. Moser U, Zeppelin I v (eds) (1996) Cognitive-affective processes. New ways of psychoanalytic modeling. Springer, Berlin Heidelberg New York
25. Pfeifer R, Leuzinger-Bohleber M (1986) Application of cognitive science methods to psychoanalysis: a case study and some theory. Int Rev Psychoanal 13:221–240
26. Pfeifer R, Leuzinger-Bohleber M (1992) A dynamic view of emotion with an application to the classification of emotional disorders. In: Leuzinger-Bohleber M, Schneider H, Pfeifer R (eds) "Two butterflies on my head ..." Psychoanalysis in the interdisciplinary scientific dialogue. Springer, New York Berline Heidelberg, pp 215–245
27. Leuzinger-Bohleber M, Pfeifer R (2002) Remembering a depressive primary object.

Memory in the dialogue between psychoanalysis and cognitive science. Int J Psychoanal 83:3–33
28. Bohleber W (2000) Die Entwicklung der Traumatheorie in der Psychoanalyse. Psyche Z Psychoanal 54:797–840
29. Terr LC (1991) Childhood traumas. An outline and overview. Am J Psychiatry 148:10–20
30. Terr LC (1994) Unchained memories. True stories of traumatic memories, lost and found. Basic Books, New York
31. Bohleber W (2005) Vergangenes im Hier-und-Jetzt oder: Wozu noch lebensgeschichtliche Erinnerung im psychoanalytischen Prozess? Psyche Z Psychoanal 59(Suppl):2–10
32. Cooper A (1989) Will neurobiology influence psychoanalysis? In: Cooper A, Kernberg OF, Person ES (eds) Psychoanalysis toward the second century. Yale University Press, New Haven, CT, pp 202–219
33. Fonagy P (1999) Memory and therapeutic action (guest editorial). Int J Psychoanal 80:215–223
34. Ganaway G-K (1989) Historical versus narrative truth. Clarifying the role of exogeneous trauma in the etiology of MPD and its variants. Dissociation 2:201–205
35. Hartke R (2005) The basic traumatic situation in the analytical relationship. Int J Psychoanal 86:267–290
36. Herzog JM (2005) Los Degradados. Out, down, dead. Transmitted and inflicted trauma as encountered in the analysis of a 6-year-old girl. Int J Psychoanal 86:291–310
37. Howard GS (1991) Culture tales. A narrative approach to thinking, cross-cultural psychology, and psychotherapy. Am Psychol 46:187–197
38. Köhler L (1998) Einführung in die Entstehung des Gedächtnisses. In: Koukkou M, Leuzinger-Bohleber M, Mertens W (eds) Erinnerte Wirklichkeiten. Zum Dialog zwischen Psychoanalyse und Neurowissenschaften. Internationale Psychoanalyse, Stuttgart, pp 131–223
39. Leuzinger-Bohleber M, Pfeifer R (1998) Erinnern in der Übertragung–Vergangenheit in der Gegenwart? Psychoanalyse und Embodied Cognitive Science. Ein interdisziplinärer Dialog zum Gedächtnis. Psyche Z Psychoanal 52:884–919
40. Spence DP (1982) Narrative truth and historical truth. Meaning and interpretation in psychoanalysis. Norton, New York
41. Thomä H, Kächele H (1985) Lehrbuch der psychoanalytischen Therapie. Springer, New York Berlin Heidelberg
42. Vinar MN (2005) The specifity of torture as trauma. Int J Psychoanal 86:311–333
43. Fonagy P, Target M (1997) Perspectives on the recovered memories debate. In: Sandler J, Fonagy P (eds) Recovered memories of abuse. True or false? Karnac Books, London, pp 183–217
44. Bokanowski T (2005) Variations on the conept of traumatism. Int J Psychoanal 86:251–265
45. Kogan I (1998) Der stumme Schrei der Kinder. Die zweite Generation der Holocaust-Opfer. Fischer, Frankfurt
46. Krystal H (1988) Intergration and self-healing. Analytic Press, Hillsdale, NJ
47. Laub D (2000) Eros oder Thanatos? Der Kampf um die Erzählbarkeit des Traumas. Psyche Z Psychoanal 54:860–895
48. Laub D (2005) Der Genozid in Ruanda–Das Kaleidoskop der Diskurse aus psychoanalytischer Perspektive. Psyche Z Psychoanal 59(Suppl):106–124
49. Varvin S (2000) Die gegenwärtige Vergangenheit. Extreme Traumatisierung und Psychotherapie. Psyche Z Psychoanal 54:895–931

50. Varvin S (2005) Conceptual research and trauma. Paper given at the IPA Congress 2005, Rio de Janeiro
51. Brenneis B (1994) Belief and suggestion in the recovery of memories of childhood sexual abuse. J Am Psychoanal Assoc 42:1027–1053
52. Brooks-Brenneis C (1996) Memory systems and the psychoanalytic retrieval of memories of trauma. J Am Psychoanal Assoc 44:1165–1187
53. Brooks-Brenneis C (1997) Recovered memories of trauma. Transferring the present to the past. International Universities Press, New York
54. Loftus EF (1993) The reality of repressed memories. Am Psychol 48:518–537
55. Morton J (1997) Cognitive perspectives on recovered memories. In: Sandler J, Fonagy P (eds) Recovered memories of abuse. True or false? Karnac Books, London, pp 39–64
56. Sandler J, Fonagy P (eds) (1997) Recovered memories of abuse. True or false? Karnac Books, London
57. Shevrin H (2001) A psychoanalytic and cognitive exploration of a memory of childhood sexual abuse. Paper given at the Sigmund Freud Institute, 2 April 2001
58. Sinason V (1997) Remembering in therapy. In: Sandler J, Fonagy P (eds) (1997) Recovered memories of abuse. True or false? Karnac Books, London, pp 81–100
59. Sugarman A (ed) (1994) Victims of abuse. International Universities Press, New York
60. Schank DC (1982) Dynamic memory. A theory of reminding and learning in computers and people. Cambridge University Press, Cambridge
61. Ashcraft MH (1994) Human memory and cognition (2nd edn). Harper Collins, New York
62. Ashby W (1956) An introduction to cybernetics. Chapman and Hall, London
63. Bursen HA (1980) Dismantling the memory machine. A philosophical investigation of machine theories of memory. Reidel, Dodrecht
64. Clancey WJ (1993) The biology of consciousness. Comparative review of Israel Rosenfield, The Strange, Familiar, and Forgotten and Gerald M Edelman, Bright Air, Brilliant Fire. Artificial Intelligence 60:313–356
65. Clancey WJ (1997) Situated cognition. On human knowledge and computer representations. Cambridge University Press, Cambridge
66. Edelman GM (1987) Neural Darwinism: the theory of neural group selection. Basic Books, New York
67. Edelman GM (1989) The remembered present. A biological theory of consciousness. Basic Books, New York
68. Edelman GM (1992) Bright air, brilliant fire. On the matter of the mind. Basic Books, New York
69. Rosenfield I (1992) The strange, familiar, and forgotten. An anatomy of consciousness. Knopf, New York
70. Hirshberg LM (1989) Remembering. Reproduction or construction. Psychoanal Contemp Thought 12:342- 381
71. Leuschner W, Hau S, Fischmann T (1998) Couch im Labor–Experimentelle Erforschung unbewusster Prozesse. Psyche Z Psychoanal 52:824–850
72. Kurthen M (1998) Intentionalität und Sprachlichkeit in Psychoanalyse und Kognitionswissenschaft. Psyche Z Psychoanal 52:850–884
73. Glenberg AM (1997) What memory is for. Behav Brain Sci 20:1–56
74. Neisser U (1988) The ecological approach to perception and memory. New Trends Exp Clin Psychiatr 4:153–166
75. Fuster JM (1997) Network memory. Trends Neurosci 20:451–459

76. Thelen E, Smith L (1994) A dynamic system approach to the development of cognition and action. MIT Press, Cambridge, MA
77. Dewey J (1896) The reflex arc concept in psychology. Psychol Rev 3:357–370. Reprinted in: Mc Dermott JJ (1981) (ed) The philosophy of John Dewey. University of Chicago Press, Chicago
78. Edelman GM (1987) Neural Darwinism. The theory of neural group selection. Basic Books, New York
79. Stern D (1995) The motherhood constellation. Basic Books, New York
80. Bollas C (1992) Being a character. Psychoanalysis and self experience. Hill & Wang, New York
81. Kohon G (1999) The Greening of psychoanalysis. André Green in dialogue with Gregorio Kohon. In: Kohon G (ed) The dead mother. The work of André Green. Routledge, London, pp 10–58
82. Hinshelwood RD (1991) A dictionary of Kleinian thought (2nd edn). Free Association Books, London
83. Fischer G, Riedesser P (1998) Lehrbuch der Psychotraumatologie. Reinhardt, München
84. van der Kolk B, McFarlane AC, Weisaeth L (eds) (1996) Traumatic stress. The effects of overwhelming experience on mind, body, and society. Guilford Press, New York
85. Kihlstrom JF (1994) Hypnosis, delayed recall. and the principles of memory. Int J Clin Exp Hypnosis 42:337–345
86. Leuzinger-Bohleber M, Pfeifer R, Röckerath K (1998) Wo bleibt das Gedächtnis? Psychoanalyse und Embodied Cognitive Science im Dialog. In: Koukkou M, Leuzinger-Bohleber M, Mertens W (eds) Erinnerung von Wirklichkeiten. Psychoanalyse und Neurowissenschaften im Dialog, vol 1.Internationale Psychoanalyse, Stuttgart, pp 517–589
87. Person ES, Klar H (1994) Establishing trauma. The difficulty distinguishing between memories and fantasies. J Am Psychoanal Assoc 42:1055–1081
88. Strenger C (1991) Between hermeneutics and science. An essay on the epistemiology of psychoanalysis. International Universities Press, New York
89. Gray CM, Singer W (1989) Stimulus-specific neuronal oscillations in orientation columns of cat visual cortex. Proc Natl Acad Sci USA 86:1698–1702

Chapter 3

Implicit Memory and Unrepressed Unconscious: How They Surface in the Transference and in the Dream

Mauro Mancia

Memory and the Neurosciences

Psychologists and neuroscientists have always been fascinated by the memory, but it is now of particular importance for psychoanalysts too because it is essential to an individual's identity and for the organization of his conscious and unconscious mind. Recent years have seen the growth of interdisciplinary studies on the memory, a field shared by psychoanalysis and the neurosciences.

The *short-term memory,* also called the *operative memory,* holds information that is only needed for a few minutes. The *long-term memory* is where we file information for life. The two are, of course, related. According to Atkinson and Shiffrin [1], information has anyway to pass through the short-term–operative–memory, where it is selected for eventual storage in the long-term memory. The two are therefore "in line." Other models, however, view them more as "in parallel," long-term information being filed directly without necessarily passing through the operative memory [2].

The long-term memory comprises an *explicit* or *declarative memory* and an *implicit, nondeclarative one* [3,4]. The explicit memory can be recalled consciously, and rendered verbally. It may be selective and episodic, dealing with the person's autobiographic experiences as regards certain specific events, or else it may be semantic, dealing with facts and knowledge, giving meaning to the oldest recollections. The explicit memory thus permits a "reconstruction" of one's own life, through recollection.

The *implicit memory* is the archive for unconscious experiences that cannot be remembered or described verbally. It deals with various aspects of learning, such as (a) *priming* (meaning a person's ability to identify an object by sight or by sound as a result of having been exposed to it earlier, even only subliminally); (b) the *procedural memory,* which holds sensorimotor and cognitive experiences, such as the movements needed for certain sports or to play a musical instrument, and for a thousand other little things we do automatically every day without actually being conscious of them; (c), the *emotive and affective memory*, which stores emotions related to affective experiences marking a newborn child's first encounters with its surroundings, especially with its mother. This implicit memory may include the late phases of pregnancy, when the relation between the fetus and its mother is particularly close, and her rhythms–heart and breathing–and her voice in particular combine in a model of constancy, rhythm and musicality around which the child assembles its first representations, at birth [5–8].

I shall return later to the implicit memory and its relations with the unrepressed unconscious that is organized in early childhood.

Knowledge from Neuropsychology

This century offers numerous clinical examples of disorders of the short- and long-term memory. Studies in humans with brain damage have provided much useful information on the memory.

A typical memory problem arises with Alzheimer's disease, when patients are no longer able to store new information, but persist in harking back to experience dating from before the onset of the illness. These patients also show abnormalities of the semantic memory which gives meaning to recent events on the basis of past experiences. Bioimaging techniques indicate that patients with Alzheimer's disease suffer a loss of function of the hippocampal neurons, bilaterally, of the cingulate cortex and the basal frontal areas. This implies that medial temporal lobe (MTL) structures–particularly the hippocampus–and frontal areas are needed to select and store information in the long-term memory.

Another cause of memory loss in man is Korsakov's syndrome; patients with this syndrome too can dig out recollections from before the onset of their disease but are no longer able to select and process recent experience and transfer it to the long-term memory. Once again, bioimaging findings point to alterations to the hippocampus and dorsal medial nucleus of the thalamus, by which information reaches the prefrontal cortex. A case that confirms these clinical observations was the patient HM, who had undergone bilateral removal of the hippocampus and temporal lobe cortex, for therapeutic reasons; his memory could no longer store new experiences, but he was perfectly able to recollect early events [9].

Alexander Lurija [10], in his fascinating book, describes the case of Lieutenant Zasetskij, who had suffered a lesion to the parieto-occipital region of the left hemisphere, where the angular and supramarginal gyri are found (Brodmann's areas 39 and 40). Zasetskij lived in a state of "mental aphasia": he had difficulty reading because he immediately forgot the first letter of each word, and if he made it as far as a whole phrase he forgot the first word as soon as he read it. This meant he could never grasp the meaning of what was written (this is semantic amnesia).

Recent electrophysiological and neuropsychological findings confirm that the hippocampus and MTL are fundamental to the memory. The operative memory basically needs the prefrontal cortex where specific neurons [11] organize "memory fields." These provide a good example of how the memory operates in "compartments" as each neuron is selectively activated for a specific type of information–for instance, a face or an object with a typical shape–and is functionally linked to other associative areas, particularly the posterior parietal cortex [12]. Inferior temporal cortex neurons are also involved in the short-term memory [13].

The MTL is the site *par excellence* of the explicit memory. It stores information in the rhinal cortex, comprising the inter-rhinal and the perirhinal, which is believed to be responsible for recognizing objects from their shapes, and memorizing them. The hippocampus is essential to humans for selecting and coding information, transferring it to the dorsomedial nucleus of the thalamus and through this to the prefrontal cortex. The hippocampus is involved in locating the object in space, and the amygdala formulates the emotional response evoked by the object [14]. Bilateral lesions to the hippocampus mean that information cannot be coded, and no information can be added that was not already present before the onset of the amnesia [15]. In patients with lesions to the right occipital lobe the explicit memory is intact but they have a disorder of the implicit memory for words. This confirms the theory that separate systems process these two types of memory, and that the memory system in the right occipital cortex is responsible for the visual implicit memory for words [16].

These clinical examples indicate that the long-term memory (explicit or declarative) uses structures in the MTL, comprising the rhinal, perirhinal, and para-hippocampal cortex and the hippocampus itself. Depending on the nature of the sensory experiences, information can be "filed" in different associative areas of the cortex. All these associative areas are potential archives of information. This was the rationale for old experiments in rats that showed the memory persisted even after extensive cortical lesions [17].

This has meant that the memory was considered a *holistic* phenomenon involving the whole associative neocortex in both hemispheres. The more specific process of selection, adaptation, and elaboration of information, ready for filing in the memory, involves the hippocampus and MTL in particular. As we shall have occasion to repeat, the amygdala controls the emotions in this process [18].

Neuropsychological research has not yet completely clarified the functional circuits governing the implicit memory. The amygdala, with its control of emotions [19–21], governs the circuit involving the cerebellum (particularly for the experience of fear), the basal ganglia as regards the procedural memory, and the cortical temporo-parieto-occipital associative areas of the right hemisphere, considered totally in charge of emotions [22]. Experiences of early infancy–including traumas–can only be filed in this memory, as it is the only one available so early in life. However, as life proceeds, strongly stressful or traumatic experiences that cause loss of hippocampal neurons [23] alter the circuits of the explicit memory, so that they have to be filed in the implicit memory, where they form the building blocks of a *late unrepressed unconscious*.

The question of the organization and recuperation of the implicit memory has been tackled experimentally by Stickgold et al. [24]. Patients with bilateral lesions to the temporal lobe and hippocampus were asked to play a simple computer game calling for spatial organization in order to test their ability to memorize things. After a few trial runs normal subjects had no problems remembering the game but the patients with hippocampal lesions remembered nothing about it all. *However, they reported dreaming about it.*

These findings are particularly interesting because they show it is possible to memorize something learned outside the hippocampus through circuits that enable the information to be stored directly in the neocortex. In these cases the hippocampal lesion had abolished the declarative memory, but an “unconscious”, nondeclarative memory could still surface in dreams in the early stage of sleep (a form of implicit memory). These researchers do not mention what specific cortical areas might be involved. We might speculate, remembering that Sperry [25] described commissurotomized subjects whose left hand obeyed the visual command received from the right hemisphere, but without being aware of it or being able to speak about it. From this and subsequent observations in primates

[26], we are led to suggest that storage of information that does not reach the threshold of consciousness may involve the posterior (parieto-temporo-occipital) cortical areas, particularly in the right hemisphere.

The Contribution of Molecular Biology

Current biological research has added much important information to our understanding of the molecular mechanisms underlying information storage. Molecular biology works on the assumption that memory operates through biochemical-molecular and structural events which still fall within the framework of the neuronal conjunction points that Sherrington defined in 1906 as *synapses.*

Early electrophysiological investigations of learning showed, for example, that if one applies high-frequency electrical impulses ("tetanization") to a dorsal root of the spinal cord (which governs sensitivity) and records the reflex response of the ventral root, which governs movements and is connected to the dorsal root through only one synapse, the normal response is potentiated, and remains so for some time (*post-tetanic potentiation*). This simple experiment suggested that the membrane structure had undergone stable plastic changes at this synaptic junction in the spinal cord, explaining why the potentiation persisted [27]. Much earlier experiments [28] on the persistent postural and motor asymmetries induced by cerebellar ablation preceding spinal cut lead to the same conclusion. Another worthwhile contribution to understanding the biological memory was provided by long-term potentiation (LTP) experiments, which involved repeated stimuli to central structures (the hippocampus was studied widely), potentiating their responses for a long time as if they had "remembered" the stimulus [29]. When stimulated, therefore, the synapses can undergo permanent plastic and structural changes: hypertrophy and the creation of new synapses after repeated stimuli, atrophy and reduction in their number when stimuli are lacking.

The neurochemical research of Stephen Rose [30] added enormously to our understanding of the biological mechanisms of the memory. Working with chicks, he showed there was a genetic memory operated through chromosomal DNA. This memory led the chicks to behave innately in a particular way in response to certain stimuli. The learning produced by these stimuli (such as a moving figure, as in the well-known example of Lorenz's duck) lasts throughout life and is essential to the survival of the species. Rose subsequently showed that biochemical changes occur in the brain during this learning process, involving ribonucleic acid

(RNA) which is needed for protein synthesis. This is obviously important for the formation of new proteins and new synapses, which then build up new networks and circuits for long-term consolidation of incoming information. There is ample proof today that long-term memory even of a specific movement–as in the examples mentioned earlier, of sport or playing an instrument–involves a rise in RNA levels at the synapses of the neurons involved, hence an increase in protein synthesis that can facilitate synaptic hypertrophy, the organization of new synapses, and therefore new nerve circuits.

About ten years ago, Kandel et al. [31], studying learning and memory in a marine mollusc, *Aplysia californica*, found that repeated stimulation of the mechanoceptors of the siphon caused retraction of the gills. This elementary reflex, involving a chain of only two or three neurons, is easily recorded and can undergo a *habit,* defined as a basic form of learning that persists with time but the response becomes progressively weaker, finally no longer appearing after repeated stimulation. Kandel did in fact show, recording from the neurons involved in the reflex, that with repeated stimulation the amplitude of the excitatory postsynaptic potentials of the neurons that move the gills diminished gradually, finally disappearing. This is caused by inactivation of the Ca^{2+} channels of the presynaptic terminations, leading to a reduction in transmitter release, hence a weaker postsynaptic response. This chain of events is due to a change in the ion channels resulting from a change in gene expression, which in turn causes a lasting change in protein synthesis in the presynaptic membrane. Unlike habit, *sensitization* of the reflex–an increase that is another form of learning that can be memorized–involves presynaptic facilitation of the axo-axonic synapses that use serotonin as neurotransmitter. This facilitation persists with time, and is due to an increase in Ca^{2+} entry resulting from a change in presynaptic membrane protein synthesis which increases the number of synaptic contacts and makes them stronger.

Kandel [32] showed that in mammals *dopamine,* whose output rises with attention, facilitates the fixation of the proteins expressed by genes on specific synapses responsible for long-term memory of certain types of experience. This suggests that in man too–also a mammal–dopamine, which controls the pleasure and sexuality pathways [33], might also be involved in the attention processes governing the persistence of information, through the plasticity of the synapses implicated in memorization.

These new findings confirm an earlier proposal put forward by Kandel [34] that environmental stimuli–such as speech, with the attention, emotions, and affects it sets in motion–may stably modify the expression of gene proteins and their fixation in the synapses, creating a situation of neuronal and synaptic plasticity as the organic basis for remembering an experience. These observations must be extrapolated cautiously to the human brain, but their main interest lies in the fact that speech itself–a basic tool of psychoanalysis–may act in the synapses by working through the genes, becoming responsible for plastic modifications that might form an anatomo-functional basis for personality changes in the person under analysis.

Memory and Psychoanalysis

The concept of memory runs through the whole of Freud's work, starting from his *Project for a Scientific Psychology* in 1895 [35], where he offered a model of the relation between mind and brain, and–using language that might appear to be neurophysiological but is in fact metaphorical–outlines the new psychology he was busy discovering at that time [36]. Freud starts out from the idea that memory is one of the basic characteristics of the nervous system, meaning the *faculty of undergoing a permanent change as a result of repetition of an event.* He proposes that on the one hand the neurons preserve traces of the energy that has flowed through them while, on the other, their original receptive conditions remain unchanged, so that each time they can establish a fresh approach to reality. Freud deals with the complexity of the problem by maintaining there are two classes of neurons, the φ-permeable ones whose function is perceptive, and the Ψ-impermeable ones, responsible for memory.

In Freud's "hydrodynamic" concept, which has something of Bernoulli's much earlier model [37], nervous energy is visualized as a fluid that cuts a passage as it flows through a medium offering some degree of resistance; thus the next time through the fluid tends to take the same path, and the neurons are permanently altered by the flow of excitation. Memory, as Freud wrote in 1895 [35], is made up of the facilitations between φ and Ψ neurons, or–better–of the differences in facilitations between these neurons as memory itself requires a selection and distinction between one nerve conduction pathway and another. The various degrees of change in neuronal permeability form the basis for this selection. In the hydraulic metaphor of the *Project,* Freud suggests that, as a river bed grows wider and deeper as more water flows through it more often, so the memory depends on the entity of the impression and on the frequency with which this impression is repeated. Consequently, the facilitation depends on the energy passing through the neuron during excitation and on the number of times the process is repeated. In *Neurosis and Psychosis* [38], Freud comes back to this point, speaking about a memory of previous perceptions which, as an interior world, make up the Ego itself.

In his *Interpretation of Dreams* the concept of memory is substantially the same. Imagining the psychic apparatus as composed of systems laid out together in a constant spatial orientation, Freud [39] writes: "A trace is left in our psychic apparatus of the perceptions which impinge upon it. This we may describe as a 'memory trace', and to the function relating to it we give the name of 'memory'" (p. 538). He goes back to the idea of the *Project*, writing: "We shall suppose that a system in the very front of the apparatus receives the perceptual stimuli but retains no trace of them and thus has no memory, while behind it there lies a second system which transforms the momentary excitation of the first system into permanent traces" (p. 538). The underlying idea still involves the two systems of neurons, φ and Ψ. But in *The Interpretation of Dreams* he sets out a more advanced concept of the memory, according to which its job is to collect and connect all our experiences and perceptions.

However, while he was working on his theory of dreams, Freud's curiosity dwelt on the amnesia of early childhood, his intuition almost leading him to the implicit memory. Actually, though, he introduced another concept, the *screen memory*, which involves repressing certain facts or shifting them onto contiguous facts. He maintained [40] that screen memories are tendentious falsifications of the memory, whose aim is repression and substitution of perturbing experiences, rather like the relation between the manifest content of a dream and its latent content.

Concentrating on centering his theory of the mind around the Oedipus complex, Freud overlooked the importance of the early, pre-oedipal, preverbal, and presymbolic experiences filed in the implicit memory, in which they could not be repressed. This explains why the references in "Remembering, repeating and working-through" [41] are aimed at the explicit memory, that can be brought to light through the patient's free associations, although in the same work Freud writes: "For a particular kind of important situation, that arose in distant infancy [. . .] it is not generally possible to produce a recollection. One becomes conscious of it through dreams" (p. 355). Freud was not in a position to elaborate on this magnificent intuition as he had no way of knowing about the implicit memory. In fact, he (wrongly) believed this type of memory was the expression of (original) repression.

Freud comes back to the problem of the memory in his "Note upon the 'mystic writing-pad' [42], where he stresses the analogies between the writing-pad and our memory, because our psychic apparatus can offer us both of the writing-pad's services if it is divided between the two systems and described in the *Project* [34]. Later, in *Civilization and its Discontents* [43], Freud uses a historical–archeological metaphor in relation to the memory, stating that what we have experienced cannot be erased. Analytical work by definition looks back to the past as it survives in the present, through the transference which enables us to bring affective situations from the past back to the present.

Freud actually talks about the past "surviving" in the present, but surviving does not necessarily mean remembering. It can survive in a *recollectable* or in a *nonrecollectable* form. This is a vital point regarding the unrepressed unconscious that I propose here, as there is no access to the recollection.

The metaphor Freud [44] uses to compare the analyst's work with that of an archeologist who brings to light all the things that time has buried seems incomplete today, and calls for some explanation. The analyst is an archeologist and historian at the same time. As a historian *sui generis* he has to rely on hidden documents that he will never be able to consult directly, though he may bring them to the surface indirectly using the special tools of his trade. To do this the analyst/historian has to rely on the patient's narration, but at the same time must focus on the modalities the patient uses for communication–the tone, rhythms, and timing of his language–and particularly on the "musical" aspects of his voice and the "reconstructive" dimension of the dream; the aim is to dig out not the recollection itself but the emotions aroused by the oldest, significant relational experiences that form part of the early unrepressed unconscious material.

Here we can perhaps turn for help to a philosophical reflection on time and memory proposed by Paul Ricoeur [45]. This offers a view of the analyst as a historian working to help the patient acquire a *historical consciouness of his own unconscious.* Starting out from Koselleck [46], Ricoeur suggests that the historical conscience may be the result of dialogue between two poles, one the "space of experience" made up of everything we have inherited from the past, and the other the "horizon of expectation" where we line up our forecasts, projects, and hopes for the future.

In analysis, the historical conscience of the unconscious can be considered the expression of a dialogue taking place in the present, in the *hic et nunc* of the relation, between the distant past–starting from the earliest unconscious, unrepressed, preverbal and presymbolic experiences–and the person's plans for the future, resulting from the transformation achieved during analysis.

Memory and Unconscious

This brings us to a central point in this discussion: the relation between the memory systems discussed above and the unconscious, starting from Freud's visions in 1915 [47, 48] up to the present day. The unconscious, basically as a function of mind, is the storehouse of the memory of affective representations linked to an individual's experiences, fantasies, and defenses from the first days of life. The functions of the memory and the unconscious therefore cannot exist separately. They become organized in parallel during our lifetime. This is in line with Freud's statement in 1912 that, "As for latent conceptions, if we have any reason to suppose that they exist in the mind–as we had in the case of memory–let them be denoted by the term 'unconscious'" (p. 260 in [49]).

The discovery of the implicit memory alongside the explicit, declarative one raises the question of a dual memory system with different brain circuits and functions. This then suggests the idea of a dual unconscious system operating in a person from birth; this would contain in particular the infant's experiences immediately before birth, particularly its mother's rhythms and voice, especially the tone of the voice, which the fetus memorizes, and intersubject relations in the infant's first 2 years, which have to be relegated to the implicit memory, since the amygdala, which governs our emotions and plays a role in the implicit memory, matures very early, while the hippocampus, essential for the explicit memory, takes at least two years [50]. The child's emotional and affective experiences, fantasies, and defenses in its first 2 years have therefore necessarily to be "filed" in the implicit memory. These are the "building blocks" of the early unconscious which cannot be repressed as the explicit memory, whose structures are essential for repression, matures so much later.

The early relations that form the basis of the unrepressed unconscious basically involve the intonation of the mother's voice and how her language is organized. The fetus memorizes her voice [51], with its rhythm and prosody, and uses them at birth to form a “wrapping” of sensations comparable to the skin experience mentioned by Bick [52] and Anzieu [53]. This voice forms the *imprinting*, marking relations with the child’s mother, and creates an area of affective interaction where early introjections and projections between the two can occur. The “musical dimension” of the mother’s speech insinuates itself into this early relation between the infant and the affective environment in which it grows, marking the child throughout its life.

Besides language, the body is another object of affective exchange (introjective and projective) between the mother and child. The mother’s way of containing, touching, watching, and speaking to the child communicates affects and emotions essential to the development of its personality. Trauma of various kinds, repeated over time, can give rise to emotions, fantasies, and defenses that are stored in the newborn’s implicit memory and subsequently disturb the organization of its unrepressed early unconscious, affecting its attachment system [54], its capacity for reflection [55], and its early intersubject relations [56], which form the foundation for organizing a solid Self [57].

The concept of unrepressed unconscious outlined here is quite different from the one Freud described in 1923 [58], where part of the Ego is unconscious, derived from the Id under the effect of outside reality through the perception-consciousness system (P-C). It is also different from the past unconscious of the Sandlers [59], which is not so early and is therefore repressed; it does, however, bear some resemblance to the unrepressed unconscious described by Matte Blanco [60], which I discussed recently [61]. As I saw it, it is the result of storage in the implicit memory of experiences, fantasies, and defenses dating from the presymbolic and preverbal stage of development, that therefore cannot be recollected even though they carry on influencing a person’s affective, emotional, cognitive, and sexual life even as an adult.

Recent neuropsychological findings on the organization of the memory offer an opportunity to speculate that there may be cortical and subcortical synaptic circuits housing unconscious mental functions. The possibility of identifying the explicit and implicit memories as respectively the repressed and unrepressed unconscious offers exciting prospects for integrating the neurosciences and psychoanalysis, possibly establishing the anatomo-functional location of the different forms of unconscious. This implies one assumption: that the experiences, life events, emotions, fantasies, and defenses around which the person’s unconscious psychic reality has been organized, from birth and throughout life, are in fact stored in the nerve structures used by the memory (implicit and explicit).

Along these lines we can speculate that the *repressed unconscious* is located in the same structures as the explicit, autobiographic memory. This is to some extent upheld by Anderson et al. [62], who showed that purposely forgetting mental experiences, which they compare to *Freudian repression,* is accompanied by increased activity in the dorsolateral prefrontal areas and a parallel reduction in hippocampal activity[1]. This is the opposite of the "de-repression" in dreams (during REM sleep), when hippocampal activity increases and there is disactivation of the dorsolateral prefrontal cortex [64].

The organization of the *unrepressed unconscious,* however, with the implicit memory, would be facilitated by activation of the amygdala which, as we said before, governs emotions [19–21]; this would be in the posterior associative cortical areas (temporo-parieto-occipital) of the right hemisphere, the basal ganglia, and the cerebellum. There is experimental and clinical proof of this: implicit experiences have an emotional component implicating the amygdala [21] and for certain emotions such as fear, the cerebellum too [65]. The right hemisphere, specifically its temporo-parieto-occipital areas, is considered the "hemisphere of the emotions" [22] and the site of the implicit memory, particularly for information linked to speech [16]. These areas are more active during REM sleep–hence during dreams–than the corresponding areas in the left hemisphere [66, 67]. They correspond to the angular and supramarginal gyri (Brodmann's areas 39 and 40) where there is a maximum of sensory integration (somatic, auditory, visual), and which play a part in more sophisticated processes related to symbolic, gnosic, and praxic function [68–71]. In addition, in commissurotomized patients, these areas govern geometric-spatial, artistic, and musical functions [25], and if they are injured, even if only partially, the architecture of sleep remains the same but dreaming may stop [72–74].

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[1] An objection to this work by Anderson et al. [62] is that Freudian repression is unconscious, whereas repression is conscious. However, the authors themselves suggest that Freud allowed either conscious or unconscious repression, and that it was mainly Anna Freud who set limits to the unconscious process [63].

How to Bring the Unrepressed Unconscious to the Surface in Analysis

Probably the most interesting question from the clinical viewpoint is: how in the analytical relationship can we gain access to the early unrepressed unconscious, which cannot be recalled? The discovery of the implicit memory and the unrepressed unconscious leads us to pay close attention to certain aspects of the transference and the elements of dreams that lend themselves to psychic "configuration" [75], to narrow the gap created by lack of figurative forms in the unrepressed early unconscious.

The extra- and infraverbal component of the transference must be exploited: the patient's general behavior in the setting, his facial expressions and posture, even his movements, reflect the influence of the procedural dimension of the implicit memory on the early unrepressed unconscious [76]. The infraverbal components regard the "signifying" functions [77] of the intonation of the voice and the prosody of language, as these hark back to the child's early relations with its mother, which the patient can relive with the analyst in the transference.

In analysis, where speech is essential, it is through the voice that words create sounds and carry affects. In this sense the voice is an "experience" of oneself while one is speaking [78], but at the same time an "expression" of the self in relation to the other person. It sets up a "transference current" recalling a sensory dimension linked to the mother's voice [79]. Combined with these elements of communication are the rhythm, tone, timbre of the voice, the musical turn of the phrase, and the syntax and pace of the language. In analysis all this adds up to the "musical dimension" of the transference [80–83]. Knoblauch [84] also described this as the "musical edge of therapeutic dialogue," meaning a "shared musical performance" by the analytical pair; the author even suggested there were some similarities with jazz!

The musical dimension of analysis refers to music as a language *sui generis* whose symbolic structure parallels that of our emotional and affective world [85–87]. More than the content of the narration [88], it is this modality that expresses the metaphor in the transference of affective, emotional, and cognitive experiences–some possibly traumatic–that have defined the implicit model of the patient's mind. This model has its roots in the mother's language, especially the emotional tone of her voice, which the child learns long before he understands its actual meaning. The mother's voice and language have therefore privileged access to the child's feelings [89], creating a metaphorical area of exchange for extremely early processes of projection and introjection.

Similarly, the two people involved in analysis use their voices to communicate their affects, and to facilitate–or impede–their affective investment in each other [90–92]. The semantic component of the patient's (or therapist's) speech is markedly influenced by the emotional significance originating in his early unconscious history, and is based on the tones and prosody heard before he could grasp the actual meaning. This rhythm and musical dimension can only belong to the unrepressed unconscious, in view of its origin in the person's earliest emotional experiences. It can easily be split and identified projectively by the analyst, and stings his countertransference skin more than any semantic content of the narration.

An analyst listening sensitively will grasp, in the here and now of the session, the unrepressed unconscious meaning of this particular mode of transference–especially the age-old quality of the split-off, projected affects–and put them into words, giving them a symbolic sense and reconstructively tying them to the past.

There is a continuous exchange of emotions and feelings between the patient and analyst. Normally it is the patient who projects his affective states onto the analyst, who cannot help being involved, but then has to be able to work through them. At this stage it is worth mentioning some important recent neuropsychological observations of activation of the brain areas for pain (anterior part of the cingulate cortex and insula) in an observer affectively linked to the person showing extraverbal signs of pain [93] or speaking about it [94]. Smells that disgust someone can activate the same structures in the observer (the anterior part of the insula and to a lesser extent the cingulate cortex) [95]. Experience with "mirror neurons" [96, 97] also provides neurophysiological proof of an exchange of feelings and sensations between individuals who are somehow related, as the physiological basis of a process we can call projective identification. These are some of the points where neurosciences and psychoanalysis meet, referring to potential functional neurological changes in people who are somehow connected, as a basis for the transference and countertransference.

The dream, that living metaphor of our private theatre, offers a privileged representation of the fantasies, affects, and defenses manifested in the transference, and an opportunity for reconstruction linked to the preverbal and presymbolic events of the implicit model of the patient's mind. The dream offers internal figures–or objects–that are related among themselves (its intrapsychic dimension) and to outside objects (its intersubjective dimension). The dream can create images or make a mental figurability, closing the gap created by the absence of representation, and symbolically configuring experiences that were originally presymbolic. Their interpretation will facilitate the process of reconstruction necessary for the mind to become able to "mentalize" and render thinkable–though obviously still not recollectable–experiences that originally could not be represented or even thought. The dream therefore works in the memory, drawing on repressed experiences stored in the explicit memory, activating their recollection, but also recuperating possibly traumatic events from the implicit memory, where they cannot be remembered. In this case the dream helps reconstruct a person's earliest history, giving an historical dimension to his unconscious.

This means that a critical component of the therapeutic action of psychoanalysis today involves transforming symbolically and rendering verbalizable the implicit, early unconscious structures in the patient's mind. The experience there is loaded with emotion and rooted in the affective tone of the primary relations, condensed in the language and tone and accents of the voice more than the autobiographic recollections of events that happened after the preverbal stage. Making the implicit structures of the patient's mind thinkable, and bringing to the surface the unconscious ways in which they work, means enabling him to configure what was not representable in his unrepressed unconscious and to recover those parts of the self that were denied or split off and projected when his mind was still in its early stages of development.

Experiences filed in the explicit and implicit memory may come to light in the transference, influencing each other, like in the normal process of development of a child's mind [50]. Just as work on the implicit memory can make it easier for fantasies and recollections to surface from the explicit memory, reconstruction through the autobiographic memory can help the patient's earliest experiences, with their related fantasies and defenses, to emerge from the implicit memory in the transference and in dreams. Through the transference and his dreams the patient, with the analyst's help, will be able to reconstruct his own life-story and, by rewriting his affective history, will give his unconscious an historical meaning starting from the earliest emotions of his life.

A Tranche of Analysis

"I'm glad I've been able to rewrite my personal history and tell myself the whole story." This was Mrs. R's opening remark one day in her third year of thrice-weekly analysis with me, which she had started after 10 years in the hands of another analyst.

Mrs. R. is a sophisticated 50-year-old lady from an upper-middle-class intellectual family. She teaches, under contract, at a Milanese university and came to me after her earlier analysis, dissatisfied at how that first approach had gone, and dominated by a profound anxiety that made her relational life particularly problematic. In addition to frequent bursts of anger at work Mrs. R. said she had suffered from ulcerative colitis for years, and was always afraid it would flare up. She complained that she was completely unable to set up a stable and sexually satisfying affective relationship and, in fact, in the 7 or so years since her divorce she had never had a new partner.

In analysis it immediately became clear that her internal family was an emotional breakdown; her mother was depressed, the paternal grandmother wanted to command, and her father had been absent, more interested in his own life than the family. The whole situation had caused her repeated trauma in childhood. Her mother was a cool, detached intellectual, whose husband repeatedly walked out on her. He was successful and wealthy, traveled frequently, and often got tied up in numerous other relationships.

One particular feature of her mother, which became important in the transference, was her language. Although she was of German Jewish origin, she would only speak English at home, creating a sort of phony communication that certainly did not correspond to her original culture. Then she had added another falsity: she had renounced her Jewish origins and been baptized.

Mrs. R's father, constantly tied up with his frequent trips and life abroad, was authoritarian when at home and had little patience for his small daughter's insistent complaints, peevishness, arguments, and provocations.

When she was two a baby sister was born, taking her mother's already cool, scant attention from her even more. This was an extremely important early infantile trauma that surfaced insistently in the transference in the shape of unrestrainable anger, resentment, dissatisfaction, often sadness, insomnia, and fierce anxiety that she should not cope with, originating from this old fear of being ousted and abandoned.

Another noteworthy point that came to light in the transference was that she could not stand any sort of argument or disagreement; she could not put up with being contradicted, or not being listened to and understood, or being pushed aside by some comment or gesture that she had not been able to foresee and control. Disagreement, arguments, and conflicts with me also fanned the flames of her worry about being set aside and not being listened to. Like a reflex, this automatically fueled her feelings of resentment and uncontrollable anger.

Mrs. R. was an intrusive patient, highly critical and provocative, insolent, complaining, argumentative, and often not easy to like. She kept a tight rein on her vocal expression and language, and the content of her associations, but tended to make arid, envious comments on my tone of voice and language, my movements, even how I dressed, sarcastically belittling whatever I said to her. She often provoked me with some know-all comment or interpretation, or humiliated me for whole sessions, saying she found my interpretations a concentrate of banality, repetition, and boredom, that could never have any possible effect on her. My efforts to convince her that the banality, repetitiousness, and boredom that she felt came from me might well be the result of her own attempts to get rid of these unpleasant feelings and emotions dating back to her infancy only evoked a sarcastic and skeptical response.

For the first two years of analysis I used all my patience to contain her provocations and aggression, careful not to be tempted into that continuously argumentative atmosphere that her behavior tended to attract. It took me some months to grasp the infantile nature of this negative modality of projectively identifying her own worst parts in me, particularly the boredom and obsessive repetitivity she brought to the session, whining, however, as if it came from me, not her.

For many months she did not dream, indicating that she was not willing to engage herself emotionally with me; she practically boasted that she had obliged her previous analyst therapist to work without dreams, and she could not understand why I should be interested in them, as she was not. At the end of this first period of analysis, however, despite her angry provocations and her apparent lack of interest in or even refusal of my interpretations, Mrs. R. started to dream, and was astounded at the work this opened up. To begin with her dreams were strongly persecutory: terrorists were stopping her traveling; or she identified with the pupils at a local high school who had flooded it. However, she seemed to show some awareness in these dreams that the violent, persecutory presences might make things difficult for me and jeopardize our travel together.

By working on her dreams, we managed to get deeper into her psychic reality, and started to understand the origins of her violent intolerance of what she felt was my lack of attention and interest, my distance, or my poor memory of whatever she told me. We started to understand that this intolerance, that fueled her anger, resentment and persecutory tendencies, were linked to early childhood memories. Many could be traced to when her sister was born, when no-one in the family–particularly her mother–seemed to take any notice of her and what she wanted any more. She had felt put aside, completely forgotten by the whole family. This built up intense anger, that made her spiteful, provocative, willful, whining–unbearable! The anger and tantrums were also one way of attracting attention, so she no longer felt like a poor, worthless, powerless, forgotten child.

As a reaction to her "spiteful outbursts" her father also became intolerant and more authoritative, sometimes shutting her up in the dark cellar for a whole day. This "black hole" terrorized her, but also stood for an affront to her narcissism, for her frustration and violent separation from her parents, besides the anguished loneliness of the dark.

This old process from her distant past was exactly what marked her transference, which was often dominated by a persecutory feeling about any disagreement or argument; she felt this as a conflict that could create a gap between us, when she went back to being the small child left on her own, considered the dunce of the family.

Her fear of argument led Mrs. R. to become critical of whatever I said, her sarcasm being unconsciously intended to stop me talking, especially to stop my interpretations which she felt were distant and different from what she expected. Added to this was her envy, which led her to criticize me. She would interrupt me while I was speaking, and take a very stiff pose, gesticulating with her hands like an obstinate teacher.

On those occasions she would tell me arrogantly that what she needed was an "individual consultant" more than an analyst, highlighting her intolerance of the asymmetry in our relationship, and the fact that I was available to other patients as well.

As I said before, whereas in her earlier analysis Mrs. R. had never dreamed, after an initial dreamless period with me she started to dream quite often. This brought up evidence of her rigid, intolerant internal mother, abandoned and depressed, with the falsity of her dual linguistic and religious identity, her inability to contain this, and her dislike of her child's sensory experiences and sensitivity. The evidence showed an absent internal father who flashed his money around but was unable to understand his little daughter's wishes and feelings; this father was intolerant of her provocations and naughtiness, and ever ready to shut her up for hours in the black hole of the dark cellar.

Her own part as a small child, on her own, not contained in any way, never affectionately cuddled against a warm body, left to her own devices once her little sister arrived, had no choice but to develop a sort of motor stiffness–evident in her behavior and posture even now in the sessions. She could only express her anger and resentment by acting grumpily and being naughty, provoking her parents to get their interest, while at the same time testing their ability to tolerate her insistent, exasperating demands. These outbursts sometimes became real tantrums, threatening the stability of the whole family; she could not control her anger, which just grew steadily while her thought processes remained paralyzed, leaving her totally unable to manage it.

At work she also had frequent outbursts of temper when she shouted angrily for several minutes before suddenly cooling down. These tantrums arose when an employee did not listen to her closely, or did not remember what she had ordered; sometimes they were triggered by her own disappointment when something she had expected did not materialize, or someone contradicted her on questions related to her work.

Mrs. R's analysis finally came out of the woods towards the end of a long period when she had been particularly provocative to me, looking for arguments all the time by belittling whatever I said. She humiliated me by reducing to banalities all my attempts to understand the reasons for this annoying negative transference.

One day, Mrs. R. walked into my office with a tense frown and worried expression, definitely distressed about something. The lines on her face seemed particularly heavy and she held her whole body stiffly, like in armor, ready for battle. As soon as she was on the couch she told me that analysis was not doing any good; she had been worried about her intestine–on account of an attack of ulcerative colitis–and her gums were very painful. Then, with a sly knowing look, she suggested that her gum problems might be psychosomatic.

I was surprised by her self-diagnosis, though it did sound very much like just another trick to test me once more and see if I would come out with some banal remark that she could criticize, to slight me. I replied that I did not agree, and that the sore gums might be due to something else–like not really wanting treatment (metaphorically using her unwillingness to go to the dentist to represent her unwillingness to accept therapy from me). Mrs. R. only grasped my actual words, which she interpreted as insulting and out of keeping, but which automatically made her feel like the little girl of her youth, ignored, contradicted and not listened to.

Her reaction was unexpected: she arched up stiffly on the couch, clenching her fists like a newborn baby in a fit of rage, and, waving her arms around, she set into me, shouting and insulting me. Her attack was extremely violent: I didn't understand anything about her; I was exploiting her financially without giving her anything in return; the money she gave me was not worth what she was getting, and so forth. But what struck me most was the tone, pitch, and volume of her voice. Mrs. R. was screeching so loudly that the sound carried well beyond the door of my office, right through the whole house, even down into the courtyard! Her shrieks were so shrill that they penetrated my ears with the violence of a projective identification that enabled her to evacuate her anger and made me feel invaded and paralyzed by her violence. At the height of her angry display, gesticulating furiously with her hands, she shouted: "You must be out of your mind"!

Despite the difficulty of the situation I took advantage of what seemed to be a brief pause in Mrs. R's tantrum to tell her, in as calm a voice as I could muster, that her temper and her shrieking made me think of an angry little girl who was so furious that she could not think straight, and this in fact sent her "out of her mind." This could have happened to her when she was little and no-one listened to her or understood her; her mother constantly contradicted or ignored, her attention taken by the baby sister; it could have happened when she was sent out because of her angry demands, with no-one taking any notice, or perhaps even sending her to that black hole in the cellar.

Mrs. R's relaxed unexpectedly on the couch, her arms alongside her body, her hands loose; after a few seconds of silence she asked me, in quite a different voice from before–no longer argumentative, but almost affectionate–"Are you perhaps telling me off ?" I replied that I was not telling her off at all but was trying to contain her anxiety and anger, to restrict this attack of uncontrolled emotions, to help her understand them by calling up the image of the little girl who had re-lived here with me–because I had contradicted her–the anger that so many years ago used to send her "out of her mind".

The session ended here. From this time on, however, Mrs. R's transference changed dramatically, to the point of convincing me that my basically reconstructive intervention had served her as a source of worthwhile insight and certainly brought about a change. Her anger at work and with me in the transference certainly did not go away overnight, as it were, but she could now link it, as if by a bridge, to her childish part, so intolerant of not being listened to, of not having her parents' or my full attention, of being contradicted, and so willing to throw a tantrum at the first sign of any discord that made her feel like a silly child, to be ignored.

Mrs. R. behaves less arrogantly in our sessions now, with less self-control and less tendency to "rationalize things." When we work on her dreams she agrees now to identify herself with that child who suffered because of the distraction, inadequacy, absences, and forgetfulness of her mother, identified with me in the transference.

One weekend she *dreams that she is at home with her mother and realizes there are no family portraits on the walls any more*. There are only the patches left where a robber has stolen them. When she has finished describing this dream Mrs. R. says nothing for a while. I propose the image of a young girl for whom the separation from the mother/analyst is the equivalent of a thief removing the affective traces of familiar presences, leaving empty spaces on the walls of her inner house. Mrs. R. replies emotionally, with the voice of a little girl: "When my sister was born she stole all my mother's affection, all her attention. In that dream I am together with my mother–maybe you?–looking through all the painful voids left inside me".

I tell her that her mother's absence and the affective void she feels again in our separations have left the young girl with no containment and no stimuli linked to her sensory awareness. This helps explain her difficulties in managing her body serenely and living comfortably with her sexuality.

The next day Mrs. R. describes another *dream, in which I come into her bedroom and sit at the foot of her bed, and we start to take about sensuality. Then I ask some questions and she realizes that this meeting is unscheduled*. When she has finished telling me about the dream, Mrs. R. says: "My mother was very fussy about timetables. At seven each evening I had to go to bed, with no questions asked".

I pick up the thread of our session the previous day, when we had talked about a young girl who had never been held and cuddled, and never been properly contained and lovingly stimulated by her mother, who was cold, depressed, and distant, and fussy about keeping to times. Now, finally, in her dream the analyst had become a tender and affectionate mother who "outside the scheduled times" allowed her to talk about her sensuality and possibly even her sexuality. After a short silence, Mrs. R. replied, in a warm voice with a hint of irony: "But in the dream I only let you come into my bedroom, certainly not into my bed!"

In the next few months we still had a few tantrums, when Mrs. R. shrieked uncontrollably at an employee who had disappointed her, who had not paid enough attention, had not remembered exactly what she had been told, or had let her down somehow. I was able to link these attacks to her early unconscious when as a child she was distressed because she felt her mother did not pay her enough attention and betrayed her confidence, disappointing her expectations.

Unexpectedly, Mrs. R. remembers a fragment of a dream: *she was in her house in Italy with her mother and her sister . . . a house where there was only suffering.* Then she adds: "But my father let me down too when he made me come and live in Italy, taking me away from the town where I was born".

In addition to the lack of attention and let-downs of her expectations, and the lack of containment by her mother, it became evident during analysis that her mother had been highly anxious and depressed on account of her husband's lack of understanding and frequent absences. Sometimes this had the effect of projections onto her infant girl of her own depressive anxieties and unhappiness; it was as if in the patient's infancy the conditions had been created for an inversion of the process of projective identification which, instead of going from the child to the mother–as would be natural–went the other way, from the mother to the child. The child, whose thought processes were not up to the task of managing these emotions, found herself obliged to set up extreme defenses such as anger, resentment, tantrums, and shrieking, so as to lob back to her mother the anxieties that the child herself was not equipped to work through. The same happened with her father, who responded to her provocations by shutting her away in the "black hole".

This was in fact confirmed in a session at about that time. Mrs. R. admitted that her childish provocations must have been very stormy, but all the same they needed to be contained and worked through, and certainly not acted out by her parents–particularly her father–by shutting her up in the cellar. This led her to recall a painful session with her previous analyst who had threatened to stop analysis on account of her continued provocation. She acknowledges that with me she had never felt threatened with the "black hole" and in fact felt I had set a limit to her anger, so she felt contained and had been able to work through it without being intimidated.

During the session Mrs. R. recalls two short fragments of *dreams. In one was her aunt B, who was adopting a small girl. In another there were guests at her house, but far more than she had expected.* Aunt B was the person who had introduced her mother and father. I suggest that this aunt seemed somehow to represent those two parents, and in the dream she also represented me, in the role of a good adoptive parent who takes care of the child. Her immediate reply was: "But the little girl was also happy to be adopted . . . I really don't understand this second dream . . . ."

I then suggest that all the things we have discovered in the last few sessions–the personages, emotions, affects, anger, and understanding that have gone through our "analytical home"–are in fact a bit more than what she expected at the start of this experience with me. Mrs. R. finds this an interesting hypothesis. She admits that she has now understood many things, despite her problems accepting my method of analysis, and despite her arguments because I contradicted her, and the anger she had lived in analysis.

We therefore come to the end of this *tranche*, which coincides with some of the patient's fantasies of terminating her analysis with me at some not too distant time in the future. The sentence I quoted at the very beginning–"I'm glad I've been able to rewrite my personal history and tell myself the whole story"–came up in a recent session, when Mrs. R. told me about this dream*: I am with Stefano, a friend of mine, who asks me if I'd go with him to take some old papers to the land registry. I hesitate a moment, then decide I'll go.* We ask ourselves: "Are these perhaps the official papers of an old house that has been restored by analysis?"

References

1. Atkinson RC, Shiffrin, RM (1971) Il controllo della memoria a breve termine. Le Scienze 39:76–84
2. Vallard G (1983) La neuropsicologia della memoria a breve termine. Le Scienze 31(184):34–55
3. Squire LR (1994) Declarative and nondeclarative memory: multiple brain system supporting learning and memory. In: Schacter DL, Tulvin E (eds) The memory systems. MIT Press, Cambridge, MA, pp 203–232
4. Schacter DL (1996) Searching for memory. The brain, the mind, and the past. Basic Books, New York
5. Mancia M (1981) On the beginning of mental life in the foetus. Int J Psychoanal 62:351–357
6. Mancia M (1989) Vita prenatale e organizzazione della mente. In: Ammaniti M (ed) La nascita del Sé. Laterza, Roma–Bari, pp 129–137
7. Imbasciati A (1998) Nascita e costruzione della mente. UTET, Torino
8. Cheour N, Marynova O, Näätänen R et al (2002) Speech sounds learned by sleeping newborns. Nature 415:599–600

9. Gazzaniga MS (ed) (2000) The new cognitive neurosciences. Chapter VI: Memory. MIT Press, Cambridge MA, pp 727–840
10. Lurija AR (1973) Un mondo perduto e ritrovato. Editori Riuniti, Rome
11. Fuster JM (1997) The prefrontal cortex. Anatomy, physiology, and neuropsychology of the frontal lobe. Lippincott-Raven, Philadelphia
12. Fuster JM (1997) The prefrontal cortex. Anatomy, physiology, and neuropsychology of the frontal lobe. Lippincott-Raven, Philadelphia
13. Miller EK, Li L, Desimone R (1993) Activity of neurons in the anterior inferior temporal cortex during a short term memory task. J Neurosci 13:1460–1478
14. Murray EA (2000) Memory for objects in nonhuman primates. In: Gazzaniga MS (ed) The new cognitive neurosciences. MIT Press, Cambridge, MA, pp 753–763
15. Cipolotti L, Shallice T, Chan D et al (2001) Long-term retrograde amnesia: the crucial role of the hippocampus. Neuropsychologia 39:151–172
16. Gabrieli JDE, Fleishman DA, Keane MM et al (1995) Double dissociation between memory systems underlying explicit and implicit memory in the human brain. Psychol Sci 6:76–82
17. Pribram KH (1969) La neurofisiologia della memoria. Le Scienze 2:28–39
18. Mishkin M (1978) Memory in monkeys severely impaired by combined but not separate removal of amygdala and hippocampus. Nature 273:297–298
19. Damasio A (1999) The feeling of what happens. Harcourt Brace, New York
20. Ledoux J (2000) Emotion circuits in the brain. Annu Rev Neurosci 23:155–184
21. Bennet MR, Hacker PMS (2005) Emotion and cortical-subcortical function: conceptual developments. Prog Neurobiol 75:29–52
22. Gainotti G (2001) Components and levels of emotion disrupted in patients with unilateral brain damage. In: Gainotti G (ed) Emotional behavior and its disorders. Handbook of neuropsychology, vol 5, 2nd edn. Elsevier, Amsterdam, pp 161–181
23. McEwen BB, Sapolsky RM (1995) Stress and cognitive function. Curr Opin Neurobiol 5:205–216
24. Stickgold R, Malia A, Maguire D et al (2000) Replaying the game: hypnagogic images in normals and amnesics. Science 290:350
25. Sperry RW (1974) Lateral specialization in the surgically separated hemispheres. In: Smith FD, Worden FG (eds) The neurosciences. Third Study Program. MIT Press, Cambridge, MA, pp 5–19
26. Schacter DL, Curran T (2000) Memory without remembering and remembering without memory: implicit and false memories. In: Gazzaniga MS (ed) The new cognitive neurosciences. MIT Press, Cambridge, MA, pp 829–840
27. Bliss TVP, Lömo T (1973) Long-lasting potentiation of synaptic transmission in the dentate area of the anaesthetized rabbit following stimulation of the perforant path. J Physiol 232:331–356
28. Di Giorgio AM (1929) Persistenza, nell'animale spinale, di asimmetrie posturali e motorie di origine cerebellare. Arch Fisiol 27:519–542
29. Kornhuber HH (1973) Neural control of input into long term memory: limbic system and amnesic syndrome in man. In: Zippel HP (ed) Memory and transfer of information. Plenum Press, New York, pp 1–22
30. Rose S (1993) The making to memory: from molecules to mind. Bantum Press, London
31. Kandel ER, Schwartz JH, Jessel TM (1994) Principi di Neuroscienze. Ambrosiana, Milano
32. Kandel ER (2001) The molecular biology of memory storage: a dialogue between genes and synapses. Science 294:1030–1038

33. Gessa GL, Tagliamonte A (1974) Possible role of brain serotonin and dopamine in controlling male sexual behavior. Adv Biochem Psychopharmacol 11:217–228
34. Kandel ER (1999) Biology and the future of psychoanalysis: a new intellectual framework for psychiatry revisited. Am J Psychiatry 156:505–524
35. Freud S (1895) Project for a scientific psychology. SE 1
36. Mancia M (1983) Archaeology of freudian thought and the history of neurophysiology. Int J Psychoanal 10:185–192
37. Bernouilli J (1742) Opera omnia. Tomus II. Marci-Michaelis Bousquet, Lausanne and Geneva
38. Freud S (1923) Neurosis and psychosis. SE 19
39. Freud S (1900) The interpretation of dreams. SE 4–5
40. Freud S (1899) Screen memories. SE 2
41. Freud S (1914) Remembering, repeating and working-through (further recommendation on the technique of psycho-analysis, II). SE 12
42. Freud S (1924) Note upon the "mystic writing-pad". SE 19
43. Freud S (1930) Civilization and its discontents. SE 21
44. Freud S (1937) Constructions in analysis. SE 23
45. Ricoeur P (1998) Das Rätsel der Vergangenheit. Erinnern–Vergessen-Verzeihen. Wallstein, Göttingen
46. Koselleck R (1979) Vergangene Zukunft. Zur Semantik geschichtlicher Zeiten. Suhrkamp, Frankfurt am Main
47. Freud S (1915) Repression. SE 14
48. Freud S (1915) The unconscious SE 14
49. Freud S (1912) A note on the unconscious in psycho-analysis. SE 12
50. Siegel SJ (1999) La mente relazionale. Neurobiologia dell'esperienza interpersonale. Cortina, Milano
51. De Casper AJ, Fifer WP (1980) Of human bonding: newborns prefer their mothers' voices. Science 208:1174–1176
52. Bick E (1968) The experience of the skin in early object - relations. Int J Psychoanal 49:484–486
53. Anzieu D (1987) Le moi-peau. Dunod, Paris
54. Bowlby J (1969) Attachment and loss. Hogarth Press, London
55. Fonagy P, Target M (2001) Attaccamento e funzione riflessiva. Cortina, Milano
56. Stern DN (2004) The present moment in psychotherapy and everyday life. Norton, New York
57. Stern DN (1985) The interpersonal world of the infant. Basic Books, New York
58. Freud S (1923) The ego and the id. SE 19
59. Sandler J, Sandler AM (1987) The past unconscious, the present unconscious and the vicissitudes of guilt. Int J Psychoanal 68:331–341
60. Matte Blanco I (1975) The unconscious as infinite sets. An essay in bi-logic. Duckworth, London
61. Mancia M (2004) L'inconscio non rimosso nella psicoanalisi attuale e nel pensiero di Matte-Blanco. 4th Conference of the International Bi-logical Group on "Emotion as infinite experience," Rome, 17–19 September
62. Anderson MC, Ochsner KN, Kuhl B et al (2004) Neural systems underlying the suppression of unwanted memories. Science 303:232–237
63. Erdelyi MH (2001) Defense processes can be conscious or unconscious. Am Psychol 56:761–762
64. Braun AR, Balkin TJ, Wesensten NJ et al (1998) Dissociated pattern of activity in visual cortices and their projections during human rapid eye movement sleep. Science 279:91–95

65. Sacchetti B, Scelfo B, Tempia F, Strata P (2004) Long-term synaptic changes induced in the cerebellar cortex by fear conditioning. Neuron 42:973–982
66. Antrobus J (1983) REM and NREM sleep reports: comparison of world frequencies by cognitive classes. Psychophysiology 5:562–568
67. Bertini M, Violani C (1984) Cerebral hemisphere, REM sleep and dreaming. In: Bosinelli M, Cicogna P (eds) Psychology of dreaming. Clueb, Bologna, pp 131–135
68. Geschwind N (1965) Disconnection syndromes in animals and man. Brain 88:237–274, 585–644
69. Critchley M (1966) The parietal lobe. Hafner, London
70. Bisiach E, Denes F, De Renzi E et al (1978) Neuropsicologia clinica. Franco Angeli, Milano
71. Hyvarinen J (1982) The parietal cortex of monkey and man. Springer, Berlin
72. Solms M (1995) New findings on the neurological organization of dreaming: implications for psychoanalysis. Psychoanal Q 44:43–67
73. Solms M (2003) Dreaming and REM sleep are controlled by different brain mechanisms. In: Pace-Schott EF, Solms M, Blagrove M, Harnad S (eds) Sleep and dreaming. Scientific advances and reconsiderations. Cambridge University Press, Cambridge, pp 51–58
74. Bischof M, Bassetti CL (2004) Total dream loss: a distinct neuropsychological dysfunction after bilateral PCA stroke. Ann Neurol 56:583–586
75. Botella C and S (2001) La figurabilité psychique. Colloquium Champs Psychanalytiques, Delachaux et Niestlé, Lausanne
76. Clyman RB (1991) The procedural organization of emotions: a contribution from cognitive science to the psychoanalytic theory of therapeutic action. J Am Psychoanal Assoc 39 (Suppl):349–382
77. Saussure F de (1916) Cours de linguistique générale. Édition critique préparée par Tullio De Mauro. Payot, Paris 1972
78. Ogden JH (2001) Conversations at the frontier of dreaming. Jason Aronson, Northvale NJ
79. Godfrind J (1993) Les deux courants du transfert. Press Universitaire de France, Paris
80. Mancia M (2003) Dream actors in the theatre of memory: their role in the psychoanalytic process. Int J Psychoanal 84:945–952
81. Mancia M (2003) Implicit memory and unrepressed unconscious: their role in creativity and transference. Israel Psychoanal J 3:331–349
82. Mancia M (2003) Il sonno della memoria genera mostri. Riv Psicoanal 4:691–708
83. Mancia M (2004) Sentire le parole. Archivi sonori della memoria implicita e musicalità del transfert. Bollati Boringhieri, Torino
84. Knoblauch SH (2000) The musical edge of therapeutic dialogue. Analytic Press, Hillsdale NJ
85. Langer S (1942) Philosophy in a new key. Harvard University Press, Cambridge, MA
86. Ognibene A (1999) La spinta di Eros dalle rappresentazioni al linguaggio. In: Bimbi A (ed) Eros e Psiche. Cerro, Tirrenia (Pisa), pp 27–37
87. Di Benedetto A (2000) Prima della parola. L'ascolto psicoanalitico del non detto attraverso le forme dell'arte. Angeli, Milano
88. Ferro A (2002) In the analyst's consulting room. Routledge Brunner, London New York 2002
89. Amati Mehler J, Argentieri S, Canestri J (1993) The Babel of the unconscious. Mother tongue and foreign languages in the psychoanalytic dimension. International Universities Press, Madison, CT
90. Rizzuto AM (2004) Language and affects in the analytic practice. International

Psychoanalytical Association, 43rd Congress, Panel P01, New Orleans
91. De León de Bernardi B (2004) Affects and words in interpretative modes of transference. International Psychoanalytical Association, 43rd Congress, Panel "Language and affects in the analytic practice," New Orleans
92. Etchegoyen L, Amati Mehler J (2004) Language and affects in the analytic practice. Int J Psychoanal 85:1479–1483
93. Singer T, Seymour B, O'Doherty J et al (2004) Empathy for pain involves the affective but not sensory components of pain. Science 303:1157–1164
94. Osaka N, Osaka M, Morishita M et al (2004) A word expressing affective pain activates the anterior cingulate cortex in the human brain: an fMRI study. Behav Brain Res 153:123–127
95. Wicker B, Keysers C, Plailly J et al (2003) Both of us disgusted in my insula: the common neural basis of seeing and feeling disgust. Neuron 40:655–664
96. Gallese V (2001) The 'shared manifold' hypothesis. From mirror neurons to empathy. J Consciousness Studies 8:33–50
97. Gallese V (2003) The manifold nature of interpersonal relations:the quest for a common mechanism. Phil Trans R Soc Lond 358:517–528

Chapter 4

Interactions Between Emotion and Cognition: A Neurobiological Perspective

Karine Sergerie and Jorge L. Armony

Throughout human history, cognition and emotion have been considered two separate, usually opposing, mental processes. We already find evidence of this presumed dichotomy in the Bible, where we learn that "a tranquil mind gives life to the flesh, but passion makes the bones rot" (Proverbs 14: 30). But it was really with the ancient Greek philosophers that cognition would firmly impose its long-lasting dominance over the emotions. As Aristotle wrote in *Politics* [1], "It is clear that the rule of the soul over the body, and of the mind and the rational element over the passionate, is natural and expedient; whereas the equality of the two or the rule of the inferior is always hurtful." In fact, Aristotle *defined* emotion as "that which leads one's condition to become so transformed that his judgment is affected" [2]. That is, the very essence of emotion rested on its negative effect on reasoning. Thus, emotions were to be avoided, if one wanted to think clearly, "for desire is a wild beast, and passion perverts the minds of rulers" [1].

And so the rule of reason over emotion was established, for centuries to come (reaching its extreme, perhaps, with the Stoics, who regarded emotions as conceptual errors and therefore something to be avoided at all costs), until the Scottish skeptic philosopher David Hume turned things around, by famously declaring in his *Treatise of Human Nature* [3] that "reason is, and ought only to be, the slave of the passions, and can never pretend to any other office than to serve and obey them." Hume suggested (using a strikingly similar argument to the one proposed by Herb Simon two centuries later [4]) that without motivational and emotional drives, we would not be able to start nor finish anything. This idea was later followed up by Nietzsche, who stated that, in contrast to the traditional Aristotelian view on the control of emotions by reason, "the will to overcome an emotion is ultimately only the will of another emotion" [5]. Or, more bluntly expressed by Dostoevsky, "reason is only reason, and it only satisfies man's rational requirements. Desire, on the other hand, is the manifestation of life itself–of all of life–and it encompasses everything from reason down to scratching oneself" [6].

Unfortunately, the emancipation of emotion from its cognitive master seemed a more Herculean task to accomplish in the scientific domain. In fact, it seemed to get even worse in this century, when emotions were explicitly left out of any "serious" attempt to study the mind in the Cognitive Science movement that began to dominate psychology in the 1950s [7]. The issue of cognitive–emotional interactions, though, was not completely ignored. There were heated debates about the nature of the interaction (see, for example [8–12]). However, much of that debate revolved around semantic issues about what the terms "cognition" and "emotion" mean. In contrast, early cognitive pioneers had already recognized that information processing does not occur in an emotional and motivational vacuum. For example, Herbert Simon wrote in 1967 that "since in actual human behavior motive and emotion are major influences on the course of cognitive behavior, a general theory of thinking and problem solving must incorporate such influences" [4]. Two decades later, Marvin Minsky stated "the question is not whether intelligent machines can have any emotions, but whether machines can be intelligent *without* any emotions" [13]. This view of emotion as another form of information processing led to the possibility of studying emotions in a systematic, "scientific" way, much like the study of other aspects of brain function, such as memory or sensory processing. This approach, particularly followed by researchers within the field of neuroscience [7], led to significant progress in the understanding of the neural systems underlying emotional processing, especially fear. Once emotional processes were on an equal footing with cognitive ones, it became possible to begin exploring their interactions, not as master and slave, but as equally important partners in governing our mental processes.

In this chapter we present recent experiments by our group and others aimed at investigating the neural mechanisms which underlie the interactions between emotional processing and cognition, specifically attention and memory. We begin by briefly describing the neural systems underlying emotion. We focus on the fear system, as this is the one emotion that has been most studied, both in experimental animals and in humans.

Neural Circuitry of Emotion

Brown and Schaffer [14] were the first to report that large temporal lesions transformed fierce monkeys into tame animals. Later, Heinrich Kluver and Paul Bucy [15] found that bilateral removal of the temporal lobe in rhesus monkeys caused a dramatic effect on the animals' behavior. Emotional changes were particularly striking. Specifically, the monkeys exhibited oral tendencies, hypersexuality, and a loss of emotional reactivity (e.g., absence of arousal, little emotion in their facial expressions and vocalizations). They also showed a decrease in fear and were generally calm and able to approach objects, animals, or humans, which they would usually consider to be threatening. Kluver and Bucy called this pattern of behavior *psychic blindness*; because although the animals did not exhibit any significant sensory or motor deficits, they seemed to be severely impaired in the processing of emotional stimuli. Later, Weiskrantz [16] reported that lesions restricted to the amygdala complex resulted in a similar pattern of behavior as that encountered in the Kluver–Bucy syndrome. Consistent with a general function in fear, it emerged that the amygdala was also involved in aversive learning such as conditioned emotional responses in monkeys [16] and rats [17]. Together, these results suggested an involvement of the amygdala in emotionally motivated behavior, particularly in forming associations between stimuli and negative reinforcement (for a review, see [18]).

What is the Amygdala?

The term "amygdalar nucleus" was first used by the anatomist Karl Friedrich Burdach to identify an almond-shaped cell mass of gray matter deep within the human medial temporal lobe [19] (Fig. 1). In 1923, J.B. Johnston described, in a landmark paper [20], the amygdalar region in numerous mammalian species, nonmammalian vertebrates, and human embryos. He identified, on the basis of their relative locations within the amygdaloid complex, six main nuclei: central (CE), medial (ME), cortical (CO), basal (B), accessory basal (AB), and lateral (LA) nuclei. The regions that Burdach called "amygdala" referred to the three latter nuclei, which are now collectively termed the basolateral amygdala complex. In differ-

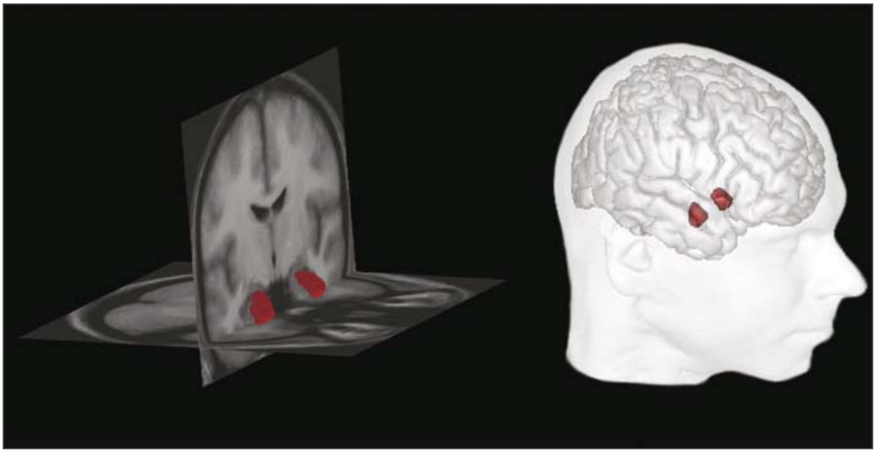

Fig. 1. Three-dimensional reconstruction of a human amygdala obtained with magnetic resonance imaging, showing its location in the medial temporal lobes

ent classification schemes, AB is also called the basomedial nucleus and B the basolateral nucleus [21]. While there is still no consensus on its exact borders, most researchers now agree that the amygdaloid complex is composed of approximately 13 different nuclei and cortical areas, each of them typically further subdivided into two or more subregions, with different and unique molecular, cytoarchitectonic, chemoarchitectonic, and connectivity characteristics [22]. The amygdala appears to have been highly conserved throughout evolution. The essential structure and organization of the amygdala is similar between rats, monkeys, and humans [23]. In fact, it has been argued that even birds and reptiles have amygdala-like structures which subserve similar functions as in mammals, especially in terms of fear-related behavior [7].

Although there have been numerous studies investigating the anatomic and functional aspects of amygdaloid connectivity, especially in rodents, much remains unknown. Nonetheless, recent tract-tracing studies have revealed the organizational principles of the intrinsic amygdalar connections [24]. Briefly, the intrinsic circuitry is organized in a unidirectional lateral-to-medial flow of information. Specifically, the lateral nucleus receives sensory information from the neocortex and projects to the basal, accessory basal, and periamygdaloid complex, which in turn project to the medial and central nucleus [22]. In terms of its afferent and efferent connections, the amygdala is, as indicated in Fig. 2, interconnect-

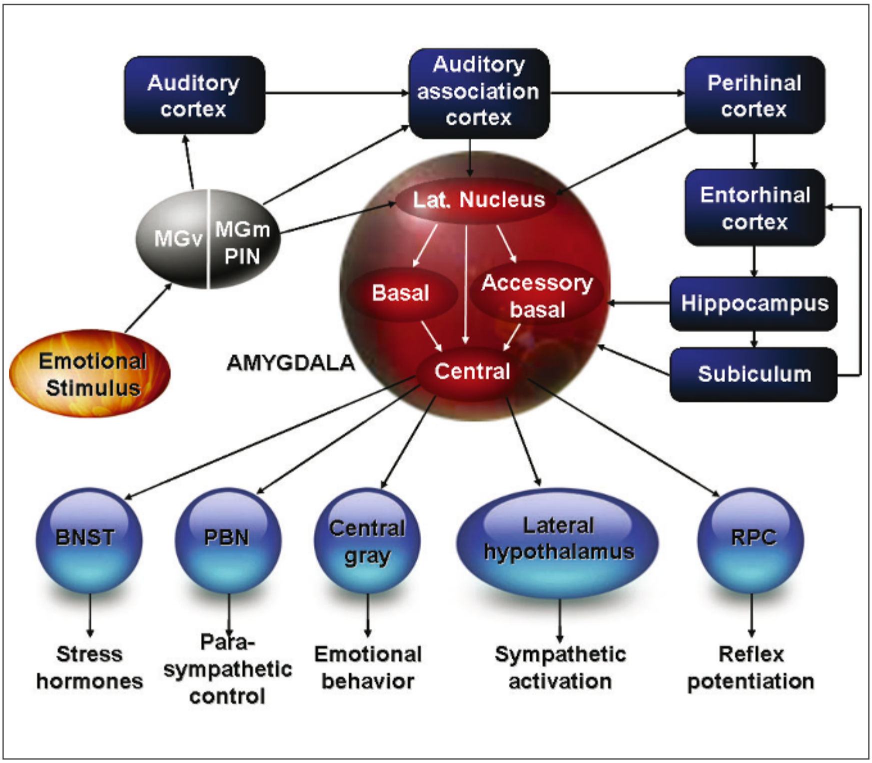

Fig. 2. Neural circuit involved in the processing of auditory emotional stimuli. *BNST,* bed nucleus of the stria terminalis; *PBN,* parabrachial nucleus; *RPC,* nucleus reticularis pontis caudalis

ed with a multitude of brain regions. It receives sensory information at different stages of processing from the thalamus, neocortex, and hippocampus through its lateral and basolateral nuclei. These nuclei then send information to the central nucleus, which in turn projects to several hypothalamic and brainstem target areas that mediate specific signs of fear and anxiety [25]. For example, projections to the central gray are involved in freezing responses, those to the lateral hypothalamus are involved in sympathetic autonomic responses, and projections to the reticular region are involved in the potentiation of startle responses [26]. In addition, the amygdala also has extensive feedback projections to cortical structures, including primary sensory areas and frontal regions, particularly the orbitofrontal cortex [27, 28].

Fear Conditioning

Much of what is known about the role of the amygdala in emotional processing comes from studies using Pavlovian (or classical aversive) conditioning. Also known as fear conditioning, it is a simple form of associative learning that supports the acquisition of emotional information. Simply stated, fear conditioning is a procedure by which a subject (human or animal) initially shows weak or no responses to a conditioned stimulus (CS; e.g., a tone), but a measurable unconditioned response (UR; e.g., freezing) to an unconditioned stimulus (US; e.g., mild electric shock). After repeated pairings of the CS with the US, an association between the US and the CS is formed, and the individual shows the conditioned response (CR) to the CS alone. Conditioned fear stimuli trigger many of the same behaviors that innate fear stimuli do. For example, rats will freeze in response to either cats or to sounds that have been paired with shock [29]. The fear response can be quantified by different behavioral responses such as startle [30, 31], freezing [17], changes in heart rate and in blood pressure [32], conditioned defensive burying [33], and vocalizations [34, 35]. Hence, fear conditioning associates evolutionarily shaped ways of responding to danger with new stimuli that have become warning signals, allowing the organism to protect itself in advance or even to avoid the impending danger altogether. Interestingly, these conditioned fear responses can be disrupted by a variety of amygdala manipulations, including conventional and neurotoxic lesions, as well as the intra-amygdaloid infusion of drugs such as *N*-methyl-D-aspartate (NMDA) antagonists [26, 30, 31]. Through such studies, neural circuits and essential components for various aspects of fear conditioning have been identified.

An acoustic CS is transmitted from cochlear receptors through the brainstem to the auditory thalamus, specifically the medial part of the geniculate body (MGm) and the associated posterior intralaminar nucleus (PIN), which relay CS information to the amygdala through two different pathways [36, 37]. One is a direct monosynaptic projection, and the other is an indirect pathway by the auditory cortex. It is believed that the auditory thalamus provides faster but less detailed information to the amygdala [38, 39], whereas cortical inputs appear to be involved in the conditioning to a more complex auditory stimuli pattern [40, 41]. Nonetheless, fear conditioning to a simple auditory stimulus can be supported by either of these pathways. In fact, it has been shown that the direct thalamo-amygdala pathway can support more complex forms of conditioning than was previously thought [42]. The two pathways seem to be phylogenetically developed to allow the organism to react rapidly to dangerous situations where a quick response is obviously more important than a detailed and precise identification of the threat. Indeed, the thalamo-amygdala pathway has been proposed to facilitate processing of the conditioned stimulus within the lateral amygdala and its afferent structures by increasing the transmission speed, and thus preparing for the later-arriving thalamo-cortical-amygdala input by priming neurons in this network [43, 44]. The direct thalamo-amygdala pathway may also play a role in the detection of threat signals occurring outside attention or awareness, as described below.

Although fear conditioning has been studied in most detail in the rat, using auditory stimuli (CS) and electric shock (US) [36], the main findings can be generalized to other modalities, as well as to other species, including cats, monkeys, rabbits and, perhaps most importantly, humans. For example, human lesion [45, 46] and functional neuroimaging [47–52] studies have confirmed the involvement of the above-mentioned structures in aversive fear-conditioning studies.

Interactions Between Emotion and Attention

Selective attention is a powerful mechanism that allows for the selection of goal-relevant sensory stimuli for the guidance of behavior, by allocating the limited pool of processing resources to the task-relevant (i.e., attended) stimuli, at the expense of the competing (unattended) ones [53–57]. Thus, stimuli that are unattended tend to be perceived less accurately and may even escape awareness altogether [58–60]. Single-cell recordings in monkeys (e.g. [28, 60, 86]) and human neuroimaging studies [61–63] have shown that top-down selective attention results in a suppression of neural responses to unattended stimuli. These attention effects appear to be strongest in higher-order sensory and association cortical areas [64–68]. In contrast, attention modulation of sensory processing in primary sensory cortical areas and in the sensory areas of the thalamus is thought to be much weaker [66, 68–70].

Results from studies on brain-damaged patients exhibiting attentional deficits, and functional neuroimaging studies in healthy individuals, have revealed a distributed neural network of higher order regions in the parietal and frontal cortex, which appears to be involved in the control of selective attention, by exerting top-down modulation in sensory areas (for a review, see [69]). These regions include the superior parietal lobule, the frontal eye fields, and the anterior cingulate cortex. Interestingly, largely overlapping networks appear to be involved in different types of selective attention, such as spatial-based attention, object-based attention, feature-based attention, as well as working memory.

A consequence of this attentional filtering is that a threat signal–such as one indicating the presence of a predator–arising outside of the focus of attention would have a reduced cortical representation. As a result, the threat would be ignored, leading to catastrophic consequences. Fortunately, this does not seem to be the case, as emotionally relevant stimuli can still be processed outside the focus of attention [71–74]. In order for this to occur, there must be a mechanism that monitors the environment continuously and detects significant stimuli, such as those that predict impending danger, independently of the current focus of attention.

Based on the experimental evidence described above, it has been suggested [7] that the direct thalamo-amygdala pathway may be less subject to top-down attentional modulation than the cortico-amygdala pathway. Thus, the subcortical inputs to the amygdala may provide the attention-independent parallel channel necessary to detect danger in parts of the environment that are outside the focus of attention. Consistent with this hypothesis, recent studies in patients have shown that the thalamus plays an important role in interrupting attentional control to allow for the detection of task-irrelevant but important stimuli [75]. In addition, a recent neuroimaging study has shown an increased coupling between amygdala and thalamic activity during unconscious processing of fearful faces, compared with neutral faces [76].

Recently, researchers have begun to investigate the effects of attention on the neural processing of fear-related stimuli using functional neuroimaging. In the first of such studies [77], subjects were presented with visual stimuli consisting of four pictures–two faces and two houses–arranged in vertical and horizontal pairs, in a non-predictive fashion. The faces could have either a neutral or a fearful expression. Subjects were instructed to pay attention to only two pictures at a time, either the vertical or the horizontal pair, and perform a similarity judgment. Thus, this was a 2x2 factorial design with respect to the face stimuli (attended vs. unattended and neutral vs. fearful). Consistent with previous studies, the fusiform cortex (a region involved in the processing of faces [61]) showed both a main effect of attention and emotional expression, whereas significant amygdala activation was observed in the main effect of expression. However, whereas fusiform responses to fearful faces were modulated by attention, those of the amygdala were not. That is, the amygdala responded equally to all fearful faces, regardless of whether they were being attended to or not. Thus, this finding supports the hypothesis that detection of emotionally salient stimuli by the fear system, in particular the amygdala, is independent of top-down selective attention and that its responses are, at least to some extent, independent of cortical inputs. This conclusion was supported by another study [78] using a slightly different paradigm in which houses and faces were overlapped in the same location, thus extending the findings to object-based selective attention. In contrast, another study [79] reported a significant interaction between responses to fearful faces and attention in the amygdala. The authors argued that one possible reason for the discrepancy between their own and previous studies could lie in the difference in attentional demands between tasks. If the processing load of the attended stimuli is low, attention may “spill over” to the processing of the task-irrelevant stimuli [80]. However, because of the important differences in the paradigms used in these studies, this explanation remains a speculative hypothesis which needs to be further tested.

It is important to keep in mind that all the studies mentioned here used faces with emotional expressions as stimuli. Faces have an intrinsic, probably evolutionarily determined, affective value. In this sense, they are special stimuli, with a privileged representation in the brain. Indeed, several brain regions, including the amygdala, contain cells that selectively respond to faces [81]. Also, faces are more difficult to ignore, as they intrude more readily into awareness when initially unattended [58, 82]. Thus, it is yet to be determined whether the observations reported above are due to the special nature of these ecologically relevant stimuli, or whether they are a property of the fear system itself.

Modulation of Attention by Emotion

Once a potentially dangerous stimulus is detected in the environment, the fear system should have the capacity to interrupt ongoing processing and redirect attentional resources to the location where this signal appeared, in order to achieve the immediate, high-priority goal of survival in the face of an unexpected threat. There is mounting evidence suggesting that the amygdala may indeed have the capacity to modulate attentional processing in the cortex (see [83]). This can be done by direct feedback projections from the amygdala to cortical areas [23] or, indirectly, by exerting influence on the various modulatory systems of the brain, such as the basal forebrain cholinergic system [84–86], and the noradrenergic system originating in the locus coeruleus [16, 87].

To investigate the neural systems involved in the modulation of spatial attention by fear-related stimuli, we conducted an event-related fMRI study combining fear conditioning and spatial attention paradigms [88]. Subjects performed a covert spatial orienting task, consisting in the detection of a peripheral target [89] preceded by a brief presentation of two face stimuli, side by side. Critically, one of these two faces had been paired with an aversive US, and hence was the CS+, while the other face was a CS- (i.e., never paired with the US). The face stimuli appeared in all possible combinations, in a pseudo-random order. Our hypothesis was that, when the CS+ and CS- were presented together, the subjects' attention would be drawn towards the CS+ (the threat signal) and therefore they would be faster to respond to targets appearing in that hemifield (congruent trials) than when target and CS+ appeared on opposite sides (incongruent trials). This hypothesis was confirmed behaviorally through a significant difference in reaction times between congruent and incongruent conditions.

Furthermore, an analysis of the fMRI data revealed that the trials in which attention was captured by the CS+ were associated with the activation of a network of brain regions in frontal and parietal cortices that has been consistently implicated in spatial attention. These findings are in agreement with the hypothesis that threat-related stimuli can automatically capture attention by engaging the same areas involved in voluntary top-down selective attention. In addition, we also observed activation in lateral orbitofrontal cortex. This finding is particularly intriguing, as this region, which is not thought to be involved in normal spatial attention tasks [69], could play a role in the link between the threat detection system (amygdala) and the attention system (fronto-parietal network). Because the face stimuli used in this experiment were presented very briefly (50 ms) and they were irrelevant to the task (i.e., non-predictive of target location), it is likely that this capture of attention by the CS+ occurred in an automatic, bottom-up, fashion. This hypothesis is consistent with preliminary behavioral studies [90, 91] suggesting that emotional facial expressions (anger or fear) can capture attention even when they are presented below the level of awareness.

In summary, a growing body of evidence, though not entirely devoid of controversy, suggests that emotional stimuli, particularly those that signal potential threats, can be detected by the amygdala even if they arise in parts of the environment that are not in the current focus of voluntary attention. In addition, detection of these danger signals leads to an automatic shift of attention towards them. This capture of attention by emotion appears to take place through the modulation of the fronto-parietal attention network by the amygdala, with the lateral orbitofrontal cortex providing the interface between the fear and attention systems.

Interactions Between Memory and Emotion

A large number of studies have consistently shown that emotionally arousing, particularly negative, stimuli–such as stories [92, 93], pictures [94], and words [95]–are better remembered than similar material without emotional value. Based on the fear-conditioning animal literature, it has been assumed that the amygdala plays a role in the enhancement of memory by emotion. Lesion studies with patients with unilateral temporal lobectomy (surgical treatment for intractable epilepsy) or selective bilateral lesions of the amygdala (Urbach–Wiethe disease or herpes simplex encephalitis) have largely confirmed this hypothesis. One of the first studies was performed by Markowitsch and colleagues with two patients, known as B.P. and C.P., with Urbach–Wiethe disease [96], presenting bilateral mineralization of the amygdala and deficits in emotional memory (B.P. less impaired than C.P.). Specifically, B.P. failed to show any memory enhancement for emotional words, whereas C.P. had a superior memory for neutral pictures as compared with emotional pictures in a recognition test; that is, the opposite of the pattern generally observed in healthy controls.

Three other studies with patients with selective bilateral amygdala damage [97–99] have also provided interesting results and refined our understanding of the amygdala's implication in emotional memory. Two of these used a task and set of stimuli developed by Heuer and Reisberg [92] in healthy individuals, in which subjects were shown 12 slides along with a narration. Slides and narration were divided into three parts: (1) neutral material, (2) emotionally arousing material, and (3) neutral material. A surprise memory test (multiple-choice questionnaire) was conducted 24 h later. All studies reported impairments in emotional memory enhancement in subjects with bilateral amygdala damage. Interestingly, when Adolphs and colleagues [97] asked subjects to rate their emotional reactions to the story, they found that the rating was similar for all subjects (controls and patients).

Adolphs and colleagues also used this task in a study of patients with unilateral amygdala damage following temporal lobectomy [100]. Each slide was rated in terms of emotional valence, arousal, unusualness, and complexity. No group differences in slide ratings were observed. As expected, normal controls and, additionally, brain-damaged controls showed an enhanced memory for the emotional part of the story (phase 2). Interestingly, the two patients with right amygdala damage also showed this pattern. Only the left amygdala patients failed to show this enhanced memory for the emotional slides, similar to the pattern observed in subjects with bilateral amygdala damage. These results suggest a specific involvement of the left amygdala in declarative memory for emotional material, at least when verbal material is used.

In another study, Phelps and colleagues [99] assessed the consequences of unilateral amygdala damage in the emotional memory for words while also recording skin conductance responses (SCRs). They administered a surprise recall test 1 min after encoding and found that negative and positive words were better remembered by all groups, with no differences between the patient and the control groups. Because of the observed greater SCR responses for neutral words compared to the emotion word category, the authors argued that the emotional words used (negative and positive) were probably not arousing enough to produce any group differences in emotional memory performance. They then performed a follow-up study to specifically evaluate the influence of arousal on memory [51]. Subjects were presented with a list of words (arousing and neutral). These arousing words elicited increased SCRs and were rated as more arousing than neutral words by all groups. Two free-recall tests were administered, immediately after and 1 h after encoding. Memory was assessed in terms of “forgetting rates” (immediate vs. delayed free recall). Only the control group showed an increased recall at the delayed test for the arousing words. The two other groups (right and left temporal lobectomy) presented decreased memory for the arousing words in the delayed condition.

Buchanan and colleagues [95] investigated the separate contributions of the left and right amygdala to memory for verbal and nonverbal emotional material. They tested patients with unilateral amygdala damage following temporal lobectomy, brain-damaged controls, and normal controls. Subjects were shown pictures (pleasant, unpleasant, and neutral) accompanied by a one-sentence verbal narrative description, and SCRs were recorded. In a free-recall test administered 24 h later, subjects with left amygdala damage did not present the usual enhancement of memory for emotional material. Most importantly, they were significantly more impaired on memory for emotional narratives as compared with memory for emotional pictures. The performance of the right amygdala damage group was, however, more similar to the control groups: they showed the normal pattern of better memory for emotional verbal and nonverbal stimuli despite an overall decreased memory performance.

Finally, a recent study [101] examined recognition memory in individuals with left hippocampal sclerosis and amygdala damage due to epilepsy. They performed an implicit encoding (living/nonliving decision) and a surprise recognition phase (remember/know/new decision) 90 min later. They found that the severity of left hippocampal damage predicted memory performance for both neutral and negative words, whereas the extent of sclerosis on the left amygdala predicted performance only for the negative items.

Neuroimaging Studies

Cahill and colleagues [93] were among the first to investigate the role of the human amygdala in emotional memory using functional neuroimaging techniques. In a simple paradigm, male subjects were scanned with positron emission tomography (PET) while watching emotionally arousing and neutral films. Three weeks later a surprise free-recall test was administered where subjects were asked to produce all the information they could remember for each film. Consistent with previous studies, subjects recalled significantly more information from the emotional film clips than from the neutral videos. In addition, a positive correlation was observed between the activity in the right amygdala during encoding and the number of emotional videos recalled, whereas no such results were observed with neutral film clips. The authors interpreted these findings as supporting the role of the amygdala in the formation of long-term memory of emotional material. Interestingly, a follow-up study with only females found a similar correlation, but within the left amygdala, thus suggesting a potential influence of gender in the lateralization of emotional memory [102]. However, no clear consensus regarding the lateralization and/or gender difference in amygdala responses to emotional stimuli has been obtained on the basis of imaging studies, as different studies, some even employing the same experimental conditions, have reported left-side, right-side, or bilateral activation (see [103, 104]).

These results for negative stimuli have been extended by two PET studies in which a correlation between bilateral amygdala activity (during encoding) and memory performance was found for both unpleasant and pleasant material [94, 105]. Hamman and colleagues additionally tested the specificity of the involvement of the amygdala in memory by adding a stimulus category of interesting and memorable pictures. Findings revealed that although there was an enhancement of memory for these interesting pictures (in both the free-recall test 10 min after and the delayed recall 4 weeks after, as well as in the delayed recognition), this enhancement was not correlated with amygdala activity. In fact, amygdala activity was specifically related to memory performance only for aversive and pleasant material and only for the delayed recognition. These findings therefore provide further support for a specific involvement of the amygdala in the facilitation of memory by emotion.

The development of event-related fMRI techniques has enabled researchers to extend the use of the subsequent memory paradigm by allowing them to directly compare the to-be-remembered and to-be-forgotten stimuli during the encoding phase [106–108]. This paradigm is particularly well suited for the identification of areas specifically involved in successful memory without the potential confounds related to differences in stimulus perceptual characteristics, task demands, or attentional load [109]. Using this approach, Canli and colleagues [106] showed in an event-related fMRI study that left amygdala activity correlated with increased memory performance only for the stimuli rated as the most emotionally arousing. Moreover, Erk and colleagues [110] found, using a subsequent memory effect paradigm with fMRI, that right amygdala activation predicted successful memory retrieval only for neutral words that had been presented in a negative context (i.e., following a unpleasant picture).

In light of these findings, it appears quite clear that there is a general agreement on the critical role of the amygdala in the encoding of emotional stimuli, which results in an enhancement of the memory performance for this material. However, it remains controversial whether it is also involved in the retrieval process of emotional material [111, 112] or not [108, 113]. These conflicting results have contributed to a more general discussion regarding the locus of emotional memory storage. Specifically, one view, mainly put forward by McGaugh and Cahill [114, 115], proposes that amygdala activity in response to emotional material influences the long-term storage of this information in other brain regions traditionally involved in episodic memory. In contrast, other researchers have argued that the amygdala is a storage site, albeit not necessarily the only one, of emotional memories [7, 116]. These two positions, and the neuroimaging evidence supporting them, are summarized below.

Emotional Memory Modulation Hypothesis

Some evidence, derived from animal research, points towards a modulatory role of the amygdala, rather than a storage site, in memory for emotional stimuli [115]. Based on this, McGaugh proposed a model in which the amygdala is a critical component of a neurobiological system that modulates the memory formation of emotional material via its influence on other brain structures, such as the hippocampus, cortex, and striatum [117]. Thus, this view posits that the amygdala plays a role in the modulation during encoding and consolidation of memories, but it is not the site of memory storage and, moreover, it may even not be necessary for the retrieval of these memories.

In terms of human neuroimaging studies, support for this model comes from Taylor and colleagues [113], who conducted a PET experiment in which they scanned both the encoding and recognition of emotionally unpleasant and neutral pictures. Consistent with its hypothesized role in emotional memory encoding, a left amygdala activation was observed during the encoding of the unpleasant pictures (compared with neutral ones). In contrast, the visual recognition memory process did not activate the amygdala but, rather, recruited regions such as right middle prefrontal and superior anterior cingulate cortices, which were not specific to the unpleasant stimuli. Thus, they did not find any evidence to support the notion that enhanced memory for emotional material was subserved by a specific neural network, different from that involved in the retrieval of neutral stimuli.

Other support for this view came from an fMRI study of memory recognition of unpleasant and neutral words by Tabert and colleagues [118]. During encoding, blocks of unpleasant and neutral words were presented and subjects were instructed to perform an emotional decision task. In the recognition phase, they were asked to perform an old/new judgment. Right amygdala activation for unpleasant compared to neutral words was observed during the encoding but not during the recognition session. The authors interpreted these findings as providing support for the memory modulation view.

Finally, Strange and Dolan [108] reported that whereas amygdala activation was observed during encoding of correctly remembered emotional words compared to neutral ones (using the subsequent memory paradigm), only hippocampal activation was detected during the retrieval. In addition, the administration of the β-adrenergic antagonist propranolol at encoding abolished the enhanced amygdala encoding and hippocampal retrieval effects, despite propranolol no longer being present at retrieval. The authors interpreted these findings as lending support to the modulatory role of the amygdala on consolidation in other episodic memory structures (in this case, the hippocampus) and they proposed that this modulation of amygdala–hippocampal interactions is associated with β-adrenergic-dependent system.

Site of Emotional Memory Storage Hypothesis

This alternative view is also derived from animal studies, specifically those involving the fear conditioning paradigm. It proposes that the observed plasticity in the amygdala during fear conditioning [39, 119] represents the mnesic trace of the acquired emotional information and thus demonstrates the necessary involvement of the amygdala in the storage and retrieval of emotional memories [36, 116, 120]. Further evidence comes from lesion and temporary inactivation studies [121] which show that an intact functioning amygdala at both learning and retrieval is essential for the formation and the expression of fear-related memories. It is important to point out that this hypothesis of the amygdala being a site of emotional memory storage does not rule out the possibility of an additional modulatory role of this structure in memory formation occurring elsewhere in the brain, possibly encoding the non-emotional aspects of the event [116].

One of the first functional neuroimaging studies providing evidence for a role of the amygdala in the retrieval of emotional material was conducted by Dolan and colleagues [122]. In a PET study, subjects performed an emotional decision on emotionally pleasant, unpleasant, and neutral pictures prior to the scan, followed by a memory (target detection) and a judgment (indoor/outdoor decision) task during scanning. The principal aim was to control for emotional responses to the pictures while examining amygdala activity during the recognition phase. They found increased left amygdala responses during the retrieval of emotional pictures (in the task-by-emotion interaction). They also observed that this left amygdala retrieval-related activation was independent of material valence (negative or positive).

Maratos and colleagues [123] tested subjects in an fMRI study in which neutral words were presented in a positive, negative, or neutral context, and later presented alone with other words not seen before. Only this recognition (old/new judgment) phase was scanned. Left amygdala activation was observed for words previously presented in a negative context compared with those presented in a neutral context. This finding was seen as reflecting an involvement of the amygdala in the retrieval of material that had been associated with a negative context. Similar findings regarding the amygdala and the retrieval of emotional context were obtained by Smith et al. [124] using pictures instead of words (in this study, activation from both negative and positive conditions were collapsed together).

In addition, Kensinger et al. [125] assessed the effects of emotion on the neural processes engaged in the retrieval of accurate compared with distorted memories. Prior to the scan, participants were shown names of neutral (e.g., horse) and emotional (e.g., spider) objects and were asked to form a mental image of the object named. In addition, they were shown photographs of half of those objects. During the scan, they viewed object names and were instructed to indicate whether or not they had seen the corresponding photograph during encoding. Activity in the amygdala/periamygdaloid cortex and orbitofrontal cortex was specific to the accurate retrieval of emotional items, suggesting that emotional salience modulates, through the engagement of limbic regions, accurate memory retrieval processes.

Further support for an amygdala role in accurate emotional memory retrieval comes from a study of Sharot et al. [111] using the remember/know paradigm. This paradigm allowed them to determine whether successful retrieval of previously learned material was accompanied by a recollection of details or was simply based on a sense familiarity. They observed an amygdala response specific to emotional remembered pictures. Intriguingly, participants reported an increased subjective sense of remembering emotional photographs without any actual differences in accuracy between emotional and neutral stimuli. The authors proposed that a "remember" judgment for emotional photographs may be based on the feeling of arousal and heightened perceptual fluency that does not necessarily result in higher accuracy.

Using a similar remember/know paradigm, Dolcos et al. [112]. investigated the contribution of the amygdala to retrieval processes, specifically with long retention intervals. They measured neural activity during the retrieval of emotional and neutral pictures 1 year after encoding. Enhanced memory for emotional pictures was found only for recollection-based responses. Successful retrieval of emotional pictures elicited greater activity in the amygdala, and this emotion effect was greater for recollection than for familiarity, similar to Sharot's findings.

In summary, there is growing support for a role of the amygdala in the successful retrieval of emotional memories, although inconsistencies do exist. This lack of agreement could be partly explained by differences in experimental paradigms, stimulus type, task, or statistical analyses (specifically, contrast used). For example, most early studies [113, 118, 122] used a block design in which stimuli from each emotional category were presented together, thus potentially introducing confounding effects due to stimulus habituation and/or expectation. In addition, most of these studies compared emotional vs. neutral stimuli, for all old and new items, regardless of whether these had been remembered or not. In the more recent event-related studies, however, old–new and correct–incorrect stimuli were separated. In several studies, only the correct responses were included in the analysis, and comparisons were then done between emotional and neutral stimuli [123, 124], hits and correct rejections [108], or remember and know responses [111]. Only a few recent studies have performed the most appropriate contrast, namely the direct comparison between remembered and forgotten old stimuli [112, 126].

Based on this evidence, and given the anatomical and functional complexity of the human amygdala [127], the new challenge for researchers is now to explore whether some degree of functional specialization within the amygdala could exist for the different stages of memory involving emotional stimuli. We started to address this issue [128] by conducting an event-related fMRI study of memory for faces with different emotional expressions (fearful, happy, and neutral), in which both encoding and recognition were scanned. There was a significant effect of expression on memory performance, as fearful faces were better remembered than happy or neutral ones. In terms of neural activity, we found significant amygdala activation associated with both successful encoding and retrieval of fearful faces. Critically, a direct comparison between both stages revealed that the anterior aspect (possibly the lateral nucleus) of the right amygdala was activated during emotional memory formation, whereas the retrieval of those memories appeared to rely on a more dorso-caudal region (possibly the central nucleus) of the left amygdala. These findings suggest that different aspects of emotional memory are mediated by different amygdala nuclei and, therefore, distinct mechanisms, as it is the case with other emotional learning and memory processes, such as fear conditioning.

Thus, the commonly held belief that emotional memories are somewhat special, distinct from other types of memory, seems to be supported by experimental data. Not only are they more vivid, better recalled, and probably longer lasting, but they also engage specialized neural systems, particularly the amygdala. Yet, as recent studies suggest, the involvement of the amygdala, and its regional specificity, in emotional memory may be more complex than previously thought. Future studies, combining lesion and recording techniques, in both experimental animals and humans, should be able to shed light on this important question.

References

1. Aristotle (350 BCE/1985) Politics. Chicago University Press, Chicago
2. Aristotle (350 BCE/1984) Rhetoric. Modern Library, New York
3. Hume D (1739/1928) A treatise of human nature. Clarendon Press, Oxford, UK
4. Simon HA (1967) Motivational and emotional controls of cognition. Psychol Rev 74:29–39
5. Nietzsche F (1886/1990) Beyond good and evil. Penguin, New York
6. Dostoevsky FM (1864/1961) Notes from the underground. Signet, New York
7. LeDoux JE (1996) The emotional brain. Simon and Schuster, New York
8. Lazarus RS (1984) Thoughts on the relation between emotion and cognition. In: Scherer KR, Ekman P (eds) Approaches to emotions. Lawrence Erlbaum Associates, Hillsdale, N.J., pp 247–258
9. Lazarus RS (1991) Cognition and motivation in emotion. Am Psychol 46:352–367
10. Leventhal H, Scherer K (1987) The relationship of emotion to cognition: a functional approach to a semantic controversy. Cognition and Emotion 1:3–28
11. Zajonc R (1980) Feeling and thinking: preferences need no inferences. Am Psychol 35:151–175
12. Zajonc RB (1984) On the primacy of affect. Am Psychol 39:117–123
13. Minsky M (1985) The society of mind. Simon and Schuster, New York
14. Brown S, Schafer EA (1888) An investigation into the functions of the occipital and temporal lobes of the monkey’s brain. Philos Trans R Soc London B 179:303–327
15. Kluver H, Bucy PC (1939) Preliminary analysis of functions of the temporal lobes in monkeys. Arch Neurol Psychiatry 42:979–1000

16. Weiskrantz L (1956) Behavioral changes associated with ablation of the amygdaloid complex in monkeys. J Comp Physiol Psychol 49:381–391
17. Blanchard DC, Blanchard RJ (1972) Innate and conditioned reactions to threat in rats with amygdaloid lesions. J Comp Physiol Psychol 81:281–290
18. Baxter MG, Murray EA (2002) The amygdala and reward. Nat Rev Neurosci 3:563–573
19. Swanson LW (2003) The amygdala and its place in the cerebral hemisphere. Ann N Y Acad Sci 985:174–184
20. Johnston JB (1923) Further contributions to the study of evolution of the forebrain. J Comp Neurol 35:337–481
21. McDonald AJ (2003) Is there an amygdala and how far does it extend? An anatomical perspective. Ann N Y Acad Sci 985:1–21
22. Pitkanen A, Savander V, LeDoux JE (1997) Organization of intra-amygdaloid circuitries in the rat: an emerging framework for understanding functions of the amygdala. Trends Neurosci 20:517–523
23. Amaral DG, Price JL, Pitkanen A, Carmichael ST (1992) Anatomical organization of the primate amygdaloid complex. In: Aggleton JP (ed) The amygdala: neurobiological aspects of emotion, memory, and mental dysfunction. Wiley-Liss, New York
24. Pitkanen A, Amaral DG (1998) Organization of the intrinsic connections of the monkey amygdaloid complex: projections originating in the lateral nucleus. J Comp Neurol 398:431–458
25. Pitkanen A, Amaral DG (1991) Demonstration of projections from the lateral nucleus to the basal nucleus of the amygdala: a PHA-L study in the monkey. Exp Brain Res 83:465–470
26. LeDoux JE (1995) Emotion: clues from the brain. Annu Rev Psychol 46:209–235
27. Carmichael ST, Price JL (1995) Limbic connections of the orbital and medial prefrontal cortex in macaque monkeys. J Comp Neurol 363:615–641
28. Krettek JE, Price JL (1977) Projections from the amygdaloid complex to the cerebral cortex and thalamus in the rat and cat. J Comp Neurol 172:687–722
29. Fendt M, Fanselow MS (1999) The neuroanatomical and neurochemical basis of conditioned fear. Neurosci Biobehav Rev 23:743–760
30. Campeau S, Davis M (1995) Involvement of the central nucleus and basolateral complex of the amygdala in fear conditioning measured with fear-potentiated startle in rats trained concurrently with auditory and visual conditioned stimuli. J Neurosci 15:2301–2311
31. Davis M (1992) The role of the amygdala in fear-potentiated startle: implications for animal models of anxiety. Trends Pharmacol Sci 13:35–41
32. LeDoux JE, Sakaguchi A, Reis DJ (1984) Subcortical efferent projections of the medial geniculate nucleus mediate emotional responses conditioned to acoustic stimuli. J Neurosci 4:683–698
33. Treit D, Pinel JP, Fibiger HC (1981) Conditioned defensive burying: a new paradigm for the study of anxiolytic agents. Pharmacol Biochem Behav 15:619–626
34. Miczek KA, Weerts EM, Vivian JA, Barros HM (1995) Aggression, anxiety and vocalizations in animals: GABAA and 5-HT anxiolytics. Psychopharmacology (Berlin) 121:38–56
35. Borszcz GS (1995) Pavlovian conditional vocalizations of the rat: a model system for analyzing the fear of pain. Behav Neurosci 109:648–662
36. LeDoux J (2000) Emotion circuits in the brain. Annu Rev Neurosci 23:155–184
37. LeDoux J (2003) The emotional brain, fear, and the amygdala. Cell Mol Neurobiol 23:727–738

38. Armony JL, LeDoux JE (2000) How danger is encoded: towards a systems, cellular, and computational understanding of cognitive-emotional interactions. In: Gazzaniga MS (ed) The new cognitive neurosciences, 2nd edn. MIT Press, Cambridge, Mass., pp 1067–1079
39. Quirk GJ, Repa C, LeDoux JE (1995) Fear conditioning enhances short-latency auditory responses of lateral amygdala neurons: parallel recordings in the freely behaving rat. Neuron 15:1029–1039
40. McCabe PM, Schneiderman N, Jarrell TW, Gentile CG, Teich AH, Winters RW, Liskowsky DR (1992) Central pathways involved in differential classical conditioning of heart rate responses. In: Gormezano I, Wasserman EA (eds) Learning and memory: the behavioral and biological substrates. Erlbaum, Hillsdale, N.J., pp 321–346
41. Jarrell TW, Gentile CG, Romanski LM, McCabe PM, Schneiderman N (1987) Involvement of cortical and thalamic auditory regions in retention of differential bradycardiac conditioning to acoustic conditioned stimuli in rabbits. Brain Res 412:285–294
42. Armony JL, Servan-Schreiber D, Cohen JD, Le Doux JE (1997) Computational modeling of emotion: explorations through the anatomy and physiology of fear. Trends Cogn Sci 1:28–34
43. Romanski LM, LeDoux JE (1992) Equipotentiality of thalamo-amygdala and thalamo-cortico-amygdala circuits in auditory fear conditioning. J Neurosci 12:4501–4509
44. Rogan MT, LeDoux JE (1996) Emotion: systems, cells, synaptic plasticity. Cell 85:469–475
45. LaBar KS, LeDoux JE, Spencer DD, Phelps EA (1995) Impaired fear conditioning following unilateral temporal lobectomy in humans. J Neurosci 15:6846–6855
46. Bechara A, Tranel D, Damasio H, Adolphs R, Rockland C, Damasio AR (1995) Double dissociation of conditioning and declarative knowledge relative to the amygdala and hippocampus in humans. Science 269:1115–1118
47. Buchel C, Dolan RJ, Armony JL, Friston KJ (1999) Amygdala-hippocampal involvement in human aversive trace conditioning revealed through event-related functional magnetic resonance imaging. J Neurosci 19:10869–10876
48. Cheng DT, Knight DC, Smith CN, Stein EA, Helmstetter FJ (2003) Functional MRI of human amygdala activity during Pavlovian fear conditioning: stimulus processing versus response expression. Behav Neurosci 117:3–10
49. Jensen J, McIntosh AR, Crawley AP, Mikulis DJ, Remington G, Kapur S (2003) Direct activation of the ventral striatum in anticipation of aversive stimuli. Neuron 40:1251–1257
50. Knight DC, Smith CN, Cheng DT, Stein EA, Helmstetter FJ (2004) Amygdala and hippocampal activity during acquisition and extinction of human fear conditioning. Cogn Affect Behav Neurosci 4:317–325
51. LaBar KS, Phelps EA (1998) Arousal-mediated memory consolidation: role of the medial temporal lobe in humans. Psychol Sci 9:490–493
52. Morris JS, DeGelder B, Weiskrantz L, Dolan RJ (2001) Differential extrageniculostriate and amygdala responses to presentation of emotional faces in a cortically blind field. Brain 124:1241–1252
53. Desimone R, Schein SJ, Moran J, Ungerleider LG (1985) Contour, color and shape analysis beyond the striate cortex. Vision Res 25:441–452
54. Duncan J, Humphreys G, Ward R (1997) Competitive brain activity in visual attention. Curr Opin Neurobiol 7:255–261

55. Kahneman D, Treisman A (1984) Changing views of attention and automaticity. In: Parasuraman R, Davies DR (eds) Varieties of attention. Academic Press, Orlando, Fla., pp 29–61
56. Posner MI, Petersen SE (1990) The attention system of the human brain. Annu Rev Neurosci 13:25–42
57. Posner MI, Dehaene S (1994) Attentional networks. Trends Neurosci 17:75–79
58. Mack A, Rock I (1998) Inattentional blindness. MIT Press, Cambridge, Mass.
59. Neisser U, Becklen P (1975) Selective looking: attending to visually superimposed events. Cogn Psychol 7:480–494
60. Rock I, Gutman D (1981) The effect of inattention on form perception. J Exp Psychol Hum Percept Perform 7:275–285
61. Kanwisher N, Wojciulik E (2000) Visual attention: insights from brain imaging. Nat Rev Neurosci 1:91–100
62. Kastner S, De Weerd P, Desimone R, Ungerleider LG (1998) Mechanisms of directed attention in the human extrastriate cortex as revealed by functional MRI. Science 282:108–111
63. Wojciulik E, Kanwisher N, Driver J (1998) Covert visual attention modulates face-specific activity in the human fusiform gyrus: fMRI study. J Neurophysiol 79:1574–1578
64. Connor CE, Gallant JL, Preddie DC, Van Essen DC (1996) Responses in area V4 depend on the spatial relationship between stimulus and attention. J Neurophysiol 75:1306–1308
65. Connor CE, Preddie DC, Gallant JL, Van Essen DC (1997) Spatial attention effects in macaque area V4. J Neurosci 17:3201–3214
66. Luck SJ, Chelazzi L, Hillyard SA, Desimone R (1997) Neural mechanisms of spatial selective attention in areas V1, V2, and V4 of macaque visual cortex. J Neurophysiol 77:24–42
67. McAdams CJ, Maunsell JH (1999) Effects of attention on orientation-tuning functions of single neurons in macaque cortical area V4. J Neurosci 19:431–441
68. Sinclair RJ, Burton H (1993) Neuronal activity in the second somatosensory cortex of monkeys (*Macaca mulatta*) during active touch of gratings. J Neurophysiol 70:331–350
69. Kastner S, Ungerleider LG (2000) Mechanisms of visual attention in the human cortex. Annu Rev Neurosci 23:315–341
70. Maunsell JH (1995) The brain's visual world: representation of visual targets in cerebral cortex. Science 270:764–769
71. Globisch J, Hamm AO, Esteves F, Ohman A (1999) Fear appears fast: temporal course of startle reflex potentiation in animal fearful subjects. Psychophysiology 36:66–75
72. Lang PJ, Bradley MM, Cuthbert BN (1998) Emotion and motivation: measuring affective perception. J Clin Neurophysiol 15:397–408
73. Öhman A, Esteves F, Soares JJF (1995) Preparedness and preattentive associative learning: electrodermal conditioning to masked stimuli. J Psychophysiol 9:99–108
74. Wells A, Matthews G (1994) Attention and emotion: a clinical perspective. Lawrence Erlbaum, Hove, UK
75. Michael GA, Boucart M, Degreef JF, Godefroy O (2001) The thalamus interrupts top-down attentional control for permitting exploratory shiftings to sensory signals. Neuroreport 12:2041–2048
76. Morris JS, Ohman A, Dolan RJ (1999) A subcortical pathway to the right amygdala mediating "unseen" fear. Proc Natl Acad Sci USA 96:1680–1685
77. Vuilleumier P, Armony J, Driver J, Dolan R (2001) Effects of attention and emotion

on face processing in the human brain: an event-related fMRI study. Neuron 30:829–841

78. Anderson AK, Christoff K, Panitz D, De Rosa E, Gabrieli JDE (2003) Neural correlates of the automatic processing of threat facial signals. J Neurosci 23:5627–5633
79. Pessoa L, McKenna M, Gutierrez E, Ungerleider LG (2002) Neural processing of emotional faces requires attention. Proc Natl Acad Sci USA 99:11458–11463
80. Lavie N (1995) Perceptual load as a necessary condition for selective attention. J Exp Psychol Hum Percept Perform 21:451–468
81. Leonard CM, Rolls ET, Wilson FA, Baylis GC (1985) Neurons in the amygdala of the monkey with responses selective for faces. Behav Brain Res 15:159–176
82. Ro T, Russell C, Lavie N (2001) Changing faces: a detection advantage in the flicker paradigm. Psychol Sci 12:94–99
83. Holland PC, Gallagher M (1999) Amygdala circuitry in attentional and representational processes. Trends Cogn Sci 3:65–73
84. Carnes KM, Fuller TA, Price JL (1990) Sources of presumptive glutamatergic/aspartatergic afferents to the magnocellular basal forebrain in the rat. J Comp Neurol 302:824–852
85. Grove EA (1988) Efferent connections of the substantia innominata in the rat. J Comp Neurol 277:347–364
86. Price JL, Amaral DG (1981) An autoradiographic study of the projections of the central nucleus of the monkey amygdala. J Neurosci 1:1242–1259
87. Aston-Jones G, Rajkowski J, Kubiak P, Valentino RJ, Shipley MT (1996) Role of the locus coeruleus in emotional activation. Prog Brain Res 107:379–402
88. Armony JL, Dolan RJ (2002) Modulation of spatial attention by fear-conditioned stimuli: an event-related fMRI study. Neuropsychologia 40:817–826
89. Posner MI (1980) Orienting of attention. Q J Exp Psychol 32:3–25
90. Armony JL, Dolan RJ (2001) Modulation of auditory neural responses by a visual context in human fear conditioning. Neuroreport 12:3407–3411
91. Mogg K, Millar N, Bradley BP (2000) Biases in eye movements to threatening facial expressions in generalized anxiety disorder and depressive disorder. J Abnorm Psychol 109:695–704
92. Heuer F, Reisberg D (1990) Vivid memories of emotional events: the accuracy of remembered minutiae. Mem Cognit 18:496–506
93. Cahill L, Haier RJ, Fallon J, Alkire MT, Tang C, Keator D, Wu J, McGaugh JL (1996) Amygdala activity at encoding correlated with long-term, free recall of emotional information. Proc Natl Acad Sci USA 93:8016–8021
94. Hamann S, Ely T, Grafton S, Kilts C (1999) Amygdala activity related to enhanced memory for pleasant and aversive stimuli. Nat Neurosci 2:289–293
95. Buchanan TW, Denburg NL, Tranel D, Adolphs R (2001) Verbal and nonverbal emotional memory following unilateral amygdala damage. Learn Mem 8:326–335
96. Markowitsch HJ, Calabrese P, Wurker M, Durwen HF, Kessler J, Babinsky R, Brechtelsbauer D, Heuser L, Gehlen W (1994) The amygdala's contribution to memory: a study on two patients with Urbach-Wiethe disease. Neuroreport 5:1349–1352
97. Adolphs R, Cahill L, Schul R, Babinsky R (1997) Impaired declarative memory for emotional material following bilateral amygdala damage in humans. Learn Mem 4:291–300
98. Cahill L, Babinsky R, Markowitsch H, McGaugh J (1995) The amygdala and emotional memory. Nature 377:295–296
99. Phelps EA, LaBar KS, Spencer DD (1997) Memory for emotional words following unilateral temporal lobectomy. Brain Cognit 35:85–109

100. Adolphs R, Tranel D, Denburg N (2000) Impaired emotional declarative memory following unilateral amygdala damage. Learn Mem 7:180–186
101. Richardson MP, Strange BA, Dolan RJ (2004) Encoding of emotional memories depends on amygdala and hippocampus and their interactions. Nat Neurosci 7:278–285
102. Cahill L, Haier R, White N, Fallon J, Kilpatrick L, Lawrence C, Potkin S, Alkire M (2001) Sex-related difference in amygdala activity during emotionally influenced memory storage. Neurobiol Learn Mem 75:1–9
103. Wager T, Phan K, Liberzon I, Taylor S (2003) Valence, gender, and lateralization of functional brain anatomy in emotion: a meta-analysis of findings from neuroimaging. Neuroimage 19:513–531
104. Zald DH (2003) The human amygdala and the emotional evaluation of sensory stimuli. Brain Res Rev 41:88–123
105. Canli T, Zhao Z, Desmond JE, Glover GH, Gabrieli JD (1999) fMRI identifies a network of structures correlated with retention of positive and negative emotional memory. Psychobiology 27:441–452
106. Canli T, Zhao Z, Brewer J, Gabrieli JDE, Cahill L (2000) Event-related activation in the human amygdala associates with later memory for individual emotional experience. J Neurosci 20:RC99
107. Canli T, Desmond JE, Zhao Z, Gabrieli JDE (2002) Sex differences in the neural basis of emotional memories. Proc Natl Acad Sci USA 99:10789–10794
108. Strange BA, Dolan RJ (2004) β-Adrenergic modulation of emotional memory-evoked human amygdala and hippocampal responses. Proc Natl Acad Sci USA 101:11454–11458
109. Buckner RL, Kelley WM, Petersen SE (1999) Frontal cortex contributes to human memory formation. Nat Neurosci 2:311–314
110. Erk S, Kiefer M, Grothe J, Wunderlich AP, Spitzer M, Walter H (2003) Emotional context modulates subsequent memory effect. Neuroimage 18:439–447
111. Sharot T, Delgado MR, Phelps EA (2004) How emotion enhances the feeling of remembering. Nat Neurosci 7:1376–1380
112. Dolcos F, LaBar KS, Cabeza R (2005) Remembering one year later: role of the amygdala and the medial temporal lobe memory system in retrieving emotional memories. Proc Natl Acad Sci USA 102:2626–2631
113. Taylor S, Liberzon I, Fig L, Decker L, Minoshima S, Koeppe R (1998) The effect of emotional content on visual recognition memory: a PET activation study. Neuroimage 8:188–197
114. Cahill L, Weinberger N, Roozendaal B, McGaugh J (1999) Is the amygdala a locus of "conditioned fear"? Some questions and caveats. Neuron 23:227–228
115. McGaugh J (2002) Memory consolidation and the amygdala: a systems perspective. Trends Neurosci 25:456
116. Fanselow M, LeDoux J (1999) Why we think plasticity underlying Pavlovian fear conditioning occurs in the basolateral amygdala. Neuron 23:229–232
117. Packard M, Teather L (1998) Amygdala modulation of multiple memory systems: hippocampus and caudate-putamen. Neurobiol Learn Mem 69:163–203
118. Tabert M, Borod J, Tang C, Lange G, Wei T, Johnson R, Nusbaum A, Buchsbaum M (2001) Differential amygdala activation during emotional decision and recognition memory tasks using unpleasant words: an fMRI study. Neuropsychologia 39:556–573
119. Quirk GJ, Armony JL, LeDoux JE (1997) Fear conditioning enhances different temporal components of tone-evoked spike trains in auditory cortex and lateral amygdala. Neuron 19:613–624

120. Maren S (2003) The amygdala, synaptic plasticity, and fear memory. Ann N Y Acad Sci 985:106–113
121. Fanselow MS, Gale GD (2003) The amygdala, fear, and memory. Ann N Y Acad Sci 985:125–134
122. Dolan RJ, Lane R, Chua P, Fletcher P (2000) Dissociable temporal lobe activations during emotional episodic memory retrieval. Neuroimage 11:203–209
123. Maratos E, Dolan R, Morris J, Henson R, Rugg M (2001) Neural activity associated with episodic memory for emotional context. Neuropsychologia 39:910–920
124. Smith APR, Henson RNA, Dolan RJ, Rugg MD (2004) fMRI correlates of the episodic retrieval of emotional contexts. Neuroimage 22:868–878
125. Kensinger EA, Schacter DL (2005) Retrieving accurate and distorted memories: neuroimaging evidence for effects of emotion. Neuroimage 27:167–177
126. Sergerie K, Lepage M, Armony JL (2005) A face to remember: emotional expression modulates prefrontal activity during memory formation. Neuroimage 24:580–585
127. De Olmos JS (2004) Amygdala. In: Paxinos G, Mai J (eds) The human nervous system, 2nd edn. Elsevier Academic, San Diego, Calif., pp 739–868
128. Sergerie K, Lepage M, Armony JL (2006) A process-specific lateralization of the human amygdala in emotional memory. J Cogn Neurosci (in press)

Chapter 5

Unconscious Emotional Memories and the Right Hemisphere

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Introduction

The problem of the possible relationships between hemispheric asymmetries and aspects of the psychoanalytic model of mind has been repeatedly raised during the past century. In a rather arbitrary manner, we could even say that this problem has passed through three distinct stages. In the first stage (roughly covering the first part of the twentieth century), psychoanalytically oriented authors, although acknowledging that some clinical phenomena could suggest a special link between aspects of psychoanalytical theory and the right hemisphere, substantially rejected the specificity of these relationships. In the second period, influenced by the influential work on split-brain patients of Sperry and co-workers [1], the interest that the right hemisphere could have for psychoanalysis has been explicitly acknowledged, but this has been made focusing attention more on cognitive than on emotional features and mapping onto the right and left hemispheres, in a rather global manner, two main constructs of the psychoanalytical theory, namely those of primary and secondary process [2, 3]. Finally, in the most recent period, Kandell's paper [4], stressing the need for an intimate relationship between psychoanalysis and cognitive neuroscience, has suggested a new way of investigating the links between hemispheric asymmetries and the psychoanalytic model of mind. This new approach aims to take into account in a more analytical way the interest that some constructs, developed in the field of cognitive and affective neuroscience, could have for some components of the psychoanalytical model of mind. In this chapter I briefly consider some of the main aspects of the first two periods, and then analyze in more detail a model which tries to integrate some theoretical constructs of the neurosciences relevant for psychoanalysis (such as those of emotions, of unconscious processing, and of implicit memory) with aspects of the functional organization of the right hemisphere.

First Encounters of Psychoanalysis with the "Right Hemisphere Problem"

We can say that psychoanalysis first met the "right hemisphere problem" when influential members of the Psychoanalytical Society, such as Ferenczi [5] and Fenichel [6], or psychoanalytically oriented authors, such as Weinstein and Kahn [7] became aware of the lateralization to the left side of the body of unconscious, emotionally-laden clinical phenomena, such as conversion symptoms and anosognosia for hemiplegia.

Lateralization of Conversion Symptoms

The lateralization of conversion symptoms will be considered first for two main reasons: (a) the importance that the study of hysteric symptoms had played in the development of Freud's first models of mind; (b) the fact that, from the chronological point of view, the prevalent lateralization to the left side of the body of conversion symptoms was the first clinical observation suggesting a crucial role of the right hemisphere in unconscious emotional phenomena. As a matter of fact, several important French authors, such as Briquet [8], Pitres [9] and Gilles de la Tourette [10] had already noticed by the second half of the nineteenth century that in hysteric patients conversion symptoms occur more frequently on the left than on the right side of the body. Developing these observations, Ferenczi [5] stated that "in general the anaesthetic stigma occurs more frequently on the left" side of the body and Fenichel [6] reported that hysterical motor disturbances appear more frequently on the left side of the body. In more recent times, this prevalence of conversion symptoms on the left half of the body has been confirmed in well-controlled conditions by several authors [11–14] and can, therefore, be considered as a well-established fact. This asymmetry of conversion symptoms, however, was not considered by psychoanalytically oriented authors to be a consequence of a special link between right hemisphere and emotional-unconscious phenomena, but as a consequence of handedness and of the correlative different level of activity of the two sides of the body. Indeed, Ferenczi [5] said that "the left part of the body is *a priori* more accessible to unconscious impulses than the right, which, in consequence of the more powerful attention-excitation of this more active and more skilful half of the body, is better protected against influences from the unconscious." However, this explanation has been more recently criticized by Stern [11], who, in a very well-controlled study has demonstrated that both right- and left-handed hysteric patients show a higher proportion of left-sided conversion symptoms. This finding shows that asymmetries in conversion symptoms are related to the different organization of the right and left hemispheres and not to the different levels of activity on the two sides of the body.

Lateralization of Anosognosia for Hemiplegia

From the historical point of view, the problem of the relationships between right hemisphere, emotions, and unconscious processes has been raised by the Babinski's [15] observation of patients with left-sided hemiplegia who ignored or seemed to ignore their striking motor defect. Babinski named this condition "anosognosia" and stressed two features of anosognosic patients that have been subsequently developed in the context of different interpretations of anosognosia. The first feature, later highlighted by psychodynamically oriented authors, was that some of these patients had been, for many years, very afraid of the condition that they now apparently ignored. The second, which anticipated contemporary views about a crucial role of the right hemisphere in emotional processes, was that anosognosia for hemiplegia had been observed only in patients with right-brain lesions. The mismatch observed by Babinski [15] between previous anxious expectancies and present lack of concern suggests that defensive mechanisms may force these patients to deny a condition that they are unable to accept. This interpretation has been fully developed by Weinstein and Kahn [7], who, in a large and influential series of papers, considered anosognosia for left-sided hemiplegia as an instance of a general attitude of denial toward illness. This attitude was characterized by the following features:

1. Anosognosia never occurs as an isolated entity, but is always part of a generalized behavioral disturbance in which the patient denies whatever he feels is very dangerous for him.
2. The anosognosic person symbolically relates to his body or to the environment in terms of his need to be well.
3. Various symbolic forms (such as verbal denial, confabulation, mini-

mization of the disability, or inattention to the paralyzed limbs) can be used to express this need.

4. Personality factors account both for the presence of denial and for the symbolic form in which denial is expressed.

Weinstein and Kahn's work was very important, since it gave a convincing psychodynamic interpretation of several patterns of behavior shown by brain-damaged patients. However, being based on a very general psychodynamic mechanism, it failed to acknowledge the specificity of the links between right hemisphere and anosognosia for hemiplegia, just as this specificity had not been considered by the Ferenczi's [5] interpretation of the left-side prevalence of conversion symptoms. In their first encounters with the "right hemisphere problem," psychoanalytically oriented authors have, therefore, failed to acknowledge the relations between emotions and brain laterality which were just beginning to emerge at that time.

Psychoanalysis and the Model of Split-Brain Patients

A renewed interest in cerebral asymmetries (and in particular the functional organization of the right hemisphere) in relation to psychoanalysis was stimulated in the second half of the last century by the results of studies conducted by Sperry and his colleagues (including Bogen, Gazzaniga, Levy, Nebes, and Trevarthen, among others) on patients submitted to a surgical disconnection of the two sides of the brain for the relief of intractable epilepsy. The results of these studies, which have extensively documented the different specialization and style of information processing typical of the right and left hemispheres, have influenced psychoanalytically oriented authors in two main ways. On one hand, they have allowed the direct study of split-brain patients by means of psychoanalytic concepts and techniques (e.g., [16]), documenting an impoverishment of dreams, fantasies, and symbolization. On the other hand, they have suggested that the duality of the brain may be reflected in a duality of mind, consistent with the distinction, drawn by Freud in his *Project for a Scientific Psychology* [17], between the holistic perception of a gestalt and the logical, sequential thinking bound to language. In particular, Bogen [18] quoting Fenichel's [6] claim that primary process thinking "is carried out more through pictorial, concrete images, whereas the secondary process is based more on words," stressed the correlation of the two hemispheres with primary and secondary processes. This proposal was very influential and several authors (in particular [2, 3, 19]) tried to give a more detailed and critical interpretation of the correspondence between primary and secondary processes and the cognitive specializations of the right and left hemisphere. This attempt, however, was not conclusive for two main reasons: the first was that mapping the constructs of primary and secondary process respectively on the right and left hemisphere was probably too global. The development of scientific models requires an explicit, analytical definition and a detailed articulation of constructs to be submitted to empirical testing, and this was not the case for primary and secondary processes. The second reason was that the hemispheric asymmetries on which the correspondence between primary and secondary processes and functional organization of the right and left hemisphere was based were mainly cognitive, but this ignored a series of data, gathered during that period from brain-damaged patients and from subjects submitted to a mono-hemispheric pharmacological inactivation, which suggested that the difference between right and left hemispheres also extends to the emotional domain. The present approach to the relationships between hemispheric asymmetries and psychoanalysis is mostly based on the development of this last line of research.

Clinical Data Raising the Problem of Hemispheric Asymmetries in Emotional Processing

The first data which raised the problem of hemispheric asymmetries in emotional representation were gathered by authors who observed different emotional behavior in patients submitted to pharmacological inactivation of the right or left hemisphere [20–21]. These authors reported that injection of sodium amobarbital into the left carotid artery produces a "depressive-catastrophic reaction," whereas inactivation of the right hemisphere is followed by a "euphoric-maniacal reaction." These emotional manifestations were interpreted as resulting from disruption of the neural mechanisms underpinning opposite aspects of mood and located respectively in the right and left hemisphere. According to this interpretation, "depressive-catastrophic reactions" of left-brain-damaged patients should be due to inactivation of a center for "positive emotions" located in the left hemisphere and "euphoric-maniacal reactions" of right-brain-damaged patients to disruption of a center for "negative emotions" located in the right hemisphere.

Some years later, I was able to partly confirm these observations by analyzing the patterns of emotional behavior shown by right and left brain-damaged patients during a neuropsychological examination [22, 23]. A "catastrophic reaction" was, indeed, typically observed in (aphasic) left-brain-damaged patients, whereas an "indifference reaction" was usually found in patients with right hemisphere damage. However, a more careful analysis of the clinical context and of the qualitative aspects of these different emotional reactions led me to refute the equivalence proposed by previous authors between "catastrophic reaction" and biological depression and between "indifference reaction" and euphoric-maniacal state. Catastrophic reactions were, indeed, usually observed in a context of severe Broca's aphasia and were triggered by repeated frustrating attempts at verbal expression. They were, therefore, considered as a dramatic, but psychologically appropriate, form of reaction to a catastrophic event, rather than as a form of biological depression. On the other hand, right-brain-damaged patients with an "indifference reaction," were apathetic and unduly jocular rather than excited or euphoric. Furthermore, they often showed paradoxical behaviors, such as a tendency to deny or minimize their disabilities, sometimes coexisting with exaggerated expressions of hatred toward the paralyzed limbs. Overall, these patterns of behavior suggested an abnormal or inappropriate reaction to a dramatic event, rather than pointing to a mood shift toward the manic pole. To explain the contrast between the different emotional reaction of left- and right-brain-damaged patients, I advanced the hypothesis of a dominance of the right hemisphere for emotional functions, analogous to the dominance of the left hemisphere for language processing. According to this interpretation, the emotional reaction of a patient with a left brain damage is often dramatic but appropriate, since the right hemisphere, which plays a critical role in emotional functions, is intact. This reaction can, on the other hand, be inappropriate in a right-brain-damaged patient if the lesion disrupts structures crucially involved in emotional processing.

Thus, the first clinical observations that have raised the problem of hemispheric asymmetries in emotional representation have also prompted the two models of emotional lateralization which are still considered to be the most important in this area and which can be labeled as the "different hemispheric specialization" and the "general right hemisphere dominance" hypotheses. To be sure, slightly different versions of each of these models have been proposed by various authors. Thus, the hypothesis of a different hemispheric specialization for positive and negative emotions has been restricted to the level of emotional expression by Davidson [24, 25], who, in the most recent version of this model, assumes that only the generation (and not the comprehension) of positive and negative emotions may be lateralized to the anterior parts of the left and of the right hemisphere, respectively. In any case, a critical analysis of these new models requires a more theoretically motivated approach to the emotional system, its organization and its relationships with the cognitive system. An illustration of the theoretical grounds necessary to understand the recent developments of studies on hemispheric asymmetries in emotional representation will therefore be discussed next.

The Nature of Emotions and Relationships Between Emotional and Cognitive Systems

From the biological viewpoint, emotion can be considered as a multicomponent adaptive system, whose functional architecture has evolved during phylogenetic evolution, ranging from very primitive, hard-wired survival-related behavioral schemata to much more complex and learned social patterns, highly integrated with the cognitive system. The general adaptive nature of both the emotional and the cognitive system has been stressed by Oatley and Johnson-Laird [26]. These authors have claimed that, to face a partially unpredictable environment and to select the most appropriate response patterns, the organism disposes of two operative systems: (1) *the emotional system*, considered as an emergency system, able to interrupt the ongoing action to rapidly select a new operative scheme; and (2) *the cognitive system*, considered as a more complex and evolved system, but needing much more time to carry out its work.

Both structural similarities and functional differences exist between emotional and cognitive system. The structural similarity stems from the fact that both systems base their activity on the integrated work of components that must: (a) scan the external milieu; (b) analyze the ongoing information to compute its significance; (c) select the most appropriate response patterns; (d) put all these pieces of information into appropriate memory systems. The functional differences (which concern the manner in which each system deals with sensory information and selects specific action schemata) result, however, from the different logic employed by the two systems.

Main Components of the Emotional System

A different kind of *analysis of sensory data* is performed by the emotional and the cognitive system; since an exhaustive analysis of highly processed information is accomplished by the cognitive system, whereas a quick computation of poorly processed sensory data is sufficient to decide whether an external situation has an emotional (pleasant or dangerous) meaning for an individual.

Equally different are the *action schemata* activated by the processes of emotional and of cognitive evaluation of the external data. The action schemata activated by the process of emotional computation are immediately selected from a small number of innate operative patterns which typically include expressive-communicative components and an important recruitment of the autonomic nervous system. On the other hand, actions selected by the cognitive system consist of controlled strategic plans, which usually do not include a communicative-expressive component and do not require a concomitant strong activation of the autonomic nervous system.

Also very different are the *learning mechanisms* used by the emotional and the cognitive system, since emotional learning is basically based on mechanisms of conditioned and unconscious learning, whereas the cognitive system uses conscious and controlled mechanisms to store new information in declarative memory.

Hierarchical Organization of the Emotional System

The description of emotions as forming an emergency system, independent from and parallel to the cognitive system, refers above all to the simplest forms of emotional behavior and to the earliest stages of the emotional development. As a matter of fact, emotional and cognitive systems become more and more interconnected during both phylogenetic and ontogenetic development and the complexity of emotions evolves accordingly. All these factors have prompted the construction of hierarchically organized developmental models of emotions aiming to explain how complex emotions can be formed, starting from the simplest ones, and how the highest (cognitively oriented) components of this structure keep under control the lower and more primitive parts of the emotional system. In particular, Leventhal [27, 28] has put forward a developmental model which proposes that human emotions may be based on three functional levels: (a) the sensorimotor level, (b) the schematic level, and (c) the conceptual level.

The *sensorimotor level* consists of a set of innate expressive-motor programs, which are triggered automatically by a certain number of stimuli and include components of motor and autonomic activation, as well as the corresponding subjective emotional feelings.

During individual development, these basic programs are linked, by mechanisms of conditioned learning, to situations of the individual experience, building the "emotional schemata," which are the units of the second *schematic level* of emotional processing. These emotional schemata correspond to spontaneous emotions and are accompanied by the emotional feelings which give to these schemata the hallmarks of a true emotion.

The last stage of this model is the *conceptual level*, which is based on mechanisms of conscious declarative memory and does not store instances of concrete emotional experiences, but abstract notions about emotions and about the social rules dealing with their expression. The activation of these representations is, therefore, not accompanied by the corresponding emotional feelings.

Models of Emotional Lateralization Based on the Componential and Hierarchical Organization of Emotions

Earlier in this chapter I stated that two general models of emotional lateralization (the "different hemispheric specialization" and the "right hemisphere dominance" hypothesis), are today considered as the most important models in this area, although the great majority of studies conducted both on normal subjects and with brain-damaged patients have supported the "right hemispheric dominance" hypothesis (see [29–30] for reviews). I am therefore focusing my attention on this second hypothesis, taking into account: (a) studies which have documented a right-hemisphere superiority for specific components of emotions; (b) studies which have tried to link hemispheric asymmetries to the hierarchical structure of emotions.

Studies Showing a Right Hemisphere Superiority for Specific Components of Emotions

Several authors have documented a crucial role of the right hemisphere for specific components of emotions, such as:

- The communicative aspects of emotions through different expressive and receptive modalities
- The vegetative/autonomic response
- The subjective experience of emotions
- The process of unconscious emotional conditioning

Each of these lines of research will be examined separately in the following paragraphs.

The Right Hemisphere and Communicative Aspects of Emotions

The comprehension and expression of emotions through the facial or vocal channels of emotional communication have been extensively investigated both in brain-damaged patients (BDP) and in normal subjects (see [31, 32] for reviews). Studies conducted in BDP have consistently shown that patients with right-sided lesions are often impaired in recognizing emotions expressed through tone of voice and/or facial expression and in the capacity to express emotions with the prosodic contours of speech or through expressive facial movements [33, 34]. On the other hand, investigations conducted in normal subjects (see [35] for review) have allowed a better testing of the "hemispheric specialization" hypothesis and have tended to give more support to the "right hemisphere dominance" than to the "different hemispheric specialization" hypothesis. They have, indeed, usually confirmed the general dominance of the right hemisphere for communication of both positive and negative emotions. Results of these investigations have led Ross [36, 37] to hypothesize that the functional specialization of the right hemisphere may not primarily concern emotional processing, but nonverbal communication, and that the emotional disturbances usually observed in these patients only reflect their inability to comprehend and produce emotional signals. This interpretation, however, is at variance both with the previously mentioned qualitative aspects of the "indifference reactions" of right-brain-damaged patients and also with the results of studies concerning the vegetative/autonomic response to emotional stimuli and the subjective experience of emotions of patients with right-sided lesions, which we will summarize in the following paragraphs.

The Right Hemisphere and Vegetative Components of the Emotional Response

Three lines of research have examined the problem of hemispheric asymmetries for vegetative functions. These lines of inquiry have separately investigated:

- The psycho-physiological correlates of emotional activation in right and left BDP
- The psycho-physiological correlates of selective emotional stimulations of the right and left hemisphere in normal subjects
- Hemispheric asymmetries in sympathetic and parasympathetic control of the heart (see [29] and [38] for reviews). Investigations conducted in unilateral BDP have consistently shown that an important reduction of autonomic activation is observed after presentation of emotional stimuli to patients with right, but not left, brain lesions [39–41]. In line with these data, studies conducted in normal subjects have shown that the largest psycho-physiological responses are obtained after presentation of emotional material to the right hemisphere [42, 43].

Taken together, these data are quite consistent with the model of a right-hemisphere dominance for vegetative functions, and in particular for those sympathetic activities which are intimately linked with the "emergency functions" of the emotional system.

The Right Hemisphere and the Subjective Experience of Emotions

Data pointing to a crucial role of the right hemisphere in the subjective experience of emotions have been obtained, among others, by Mammucari et al. [44] in unilateral BDP, and by Wittling and Roschmann [45] in normal subjects. Mammucari et al. [44] noted that during the presentation of a very unpleasant film, normal subjects and left-BDP tended to look away from the screen, being unable to tolerate these very crude scenes, whereas right-BDP, being more emotionally indifferent, did not feel the need to look away. Wittling and Roschmann [45], on the other hand, using a lateralized presentation of emotional films to normal subjects, have shown that the subjective emotional experience was more intense when the film was presented to the right, rather than to the left, hemisphere.

The Right Hemisphere and the Process of Emotional Conditioning

Some authors (e.g., [46, 47]) have suggested, on theoretical grounds, a greater involvement of the right hemisphere in Pavlovian conditioning to emotional stimuli. This suggestion has been confirmed by Johnsen and Hugdahl [48, 49], who have shown that after lateralized visual presentation of a facial emotional expression paired with a shock unconditioned stimulus, a significantly greater resistance to extinction was observed when the conditioned emotional face was presented to the right as compared to the left hemisphere.

Models Linking Hemispheric Asymmetries to the Hierarchical Organization of Emotions

The above data have clearly shown a right-hemisphere dominance for several components or aspects of emotions. Some authors have, however, suggested that the brain lateralization of emotions might concern not only some components, but also the hierarchical structure of emotions. Indeed, Lamendella [50], Buck [51], Rinn [52], and Gainotti et al. [53] have suggested that the two sides of the brain could play a complementary role in emotional behavior. More precisely, these authors argued that the right hemisphere might be more involved in the automatic expressive and autonomic components of emotions (corresponding to Leventhal's "schematic level") and the left hemisphere in functions of control and of modulation of the spontaneous emotional expression (corresponding to Leventhal's "conceptual level"). Two independent lines of evidence support the hypothesis that the left hemisphere may play a critical role in functions of emotional control. The first consists of the observation that left-BDP often show an excess of emotional reactivity which concerns both the expressive motor ([23, 54–56]) and the vegetative components of the emotional response [39, 41]. This observation suggests that, at least in a subgroup of left-brain-damaged patients, the defect in cortical control may provoke an accentuation of the expressive behavioral reaction and an increased vegetative response to emotionally laden stimuli. The second line of evidence concerns the fact that, according to some authors (e.g., [57, 58]), in normal subjects the greater expressivity of the left half face, usually observed for negative emotions, becomes much less evident for smiling or other positive emotions. Now, Etcoff [59] has rightly pointed out that smiling differs from the other facial emotional displays because it constitutes the facial "emotional" expression more often intentionally used for social communication. The greater asymmetry between the right and left half face in the expression of negative emotions could, therefore be due either to a greater intentional use by the left hemisphere of the right half face expressive apparatus for communicative purposes, or to the greater inhibition exerted by the left hemisphere on this half face in the overt expression of socially noncommunicable negative emotions [51, 52].

The model assuming a major role of the right hemisphere in the "schematic" and of the left hemisphere in the "conceptual" level of emotional processing is consistent with other general models of functional lateralization of the human brain, which concern: (a) the prevalent involvement of the left hemisphere in general cognitive and control functions, which is usually acknowledged by most authors; and (b) the greater role of the right hemisphere in other kinds of automatic activities. As for the leading role of the left hemisphere in cognitive and control functions, I will briefly quote here only the classical views of Luria [60] and some recent studies by Gazzaniga et al. [61, 62] on this subject. According to Luria [60], the development of language in the left hemisphere has greatly increased not only the conceptual skills, but also the control capacities of this hemisphere, because of the regulatory role played by language on various aspects of human behavior. These statements are consistent with results obtained by Gazzaniga et al. [61, 62] studying various abilities of the isolated right and left hemisphere in split-brain patients. This author has, in fact, shown that the left hemisphere is surprisingly superior to the right hemisphere not only on linguistic tasks, but also on nonverbal problem-solving tasks requiring organization and control functions.

The hypothesis that the right hemisphere may be critically involved in other kinds of automatic activities is consistent with the interpretation that Gainotti [63, 64] has recently offered of the unilateral neglect syndrome typical of right hemisphere lesions. After having reviewed the evidence suggesting that the neglect syndrome is due to a selective disruption of the automatic components of the spatial orienting of attention, Gainotti [63, 64] proposes that automatic and controlled components of the spatial orienting mechanisms may be subserved respectively by the right and left hemispheres. Automatic functioning and intentional control could therefore be a main feature of the right and the left hemisphere, respectively, both in orienting attention and in emotional responding.

Relationships Between the Automatic–Controlled and the Unconscious–Conscious Dichotomies

For more than a century, in spite of the Freud's discoveries and in part conditioned by the influence of the behavioristic model of mind, academic psychology has ignored or even denied the possibility that "consciousness" may be a respectable object of scientific inquiry. For this reason, experimental data showing that psychological processes can be unconsciously mediated has met with considerable resistance. Even data gathered in the early 1950s by the *New Look on Perception*, which claimed to have demonstrated unconscious influences on perception (e.g., [65]) were submitted to severe critical scrutiny. To overcome some of the methodological objections raised to these studies, Lazarus and McCleary [66] proposed a conditioning paradigm which has been subsequently extensively developed and which consisted in demonstrating a skin conductance response (SCR) to consciously nonrecognized conditioned words. In more recent years this method has been extensively used by Ohman and co-workers (see Ohman and Wiens [67] for review) for basic research and to study possible hemispheric asymmetries in emotional processing. These authors used backward masking (namely briefly presented conditioned target pictures followed by long-lasting masking stimuli) to unconsciously activate human emotion. Marcel [68] has, indeed, shown that even though the target is blocked from conscious access, it is still processed to a considerable depth, and this process may automatically activate a given response, before the mask disrupts more elaborate processing. With this methodology, some authors were able to demonstrate that nonrecognizable visual emotional stimuli can elicit both autonomic [69] and electromyographic [70] facial emotional responses. In another section of this chapter we will see that the same authors have also documented, with a functional neuroimaging experiment [71] based on the same methodology, a crucial role of the right hemisphere in this conditioned unconscious form of emotional learning.

Coming back from the methods to the theoretical models, we can say that the debate on unconscious processes took a new turn in the 1960s and 1970s, with the end of behaviorism and the advent of a strong, scientifically based cognitive psychology, since information processing models necessitated unconscious processing stages [72]. In particular, a basic distinction was introduced in research on attention, between automatic vs. conscious [73] or automatic vs. controlled [74] information-processing, since automatic processes are described as nondemanding and controlled processes as heavily demanding from the viewpoint of the cognitive resources. This distinction between automatic and controlled processes obviously captures important analogies with the distinction between unconscious and conscious mental processes. Therefore, the model assuming that automatic functioning and intentional control may characterize the right and left hemisphere, respectively, implicitly assumes that the right hemisphere may be more involved in nonconscious and the left hemisphere in consciously mediated psychological phenomena.

Unconscious Emotional Experiences and the Right Hemisphere

If the level of emotional processing represented in the right hemisphere is mainly the "schematic" one, then an emotional stimulation only involving the right hemisphere should provoke an unconscious emotional experience, activating the level where emotional schemata are automatically and unconsciously aroused. This prediction is confirmed by data obtained some years ago, studying in a split-brain patient [75] and in normal subjects [76] the cognitive evaluation and the autonomic response to subliminar and above threshold presentation of emotional and nonemotional stimuli, briefly lateralized to the right and left visual fields. Both studies showed: (a) that only the right hemisphere is able to produce an appropriate and selective autonomic response to the presentation of emotional material, and (b) that in the right hemisphere the generation of the appropriate autonomic response can be dissociated from the conscious, cognitive evaluation of the elicited stimulus. Results consistent with the same prediction have been more recently obtained by Morris et al. [71], who used positron emission tomography to study the mechanism of an unconscious form of emotional learning in which an aversively conditioned masked emotional face elicited an unconscious emotional response. The masked presentation of the conditioned emotional face elicited a significant neural response in the right, but not the left, amydgala, whereas the unmasked presentation of the same stimulus enhanced neural activity in the left, but not the right, amygdala. The authors conclude that the right amygdala plays a major role in unconscious and the left in conscious forms of emotional learning. In a further paper, the same authors [77] have tried to clarify the mechanisms through which this unconscious emotional learning could be mediated. To do this, they have taken into account, on one hand, the crucial role of the amygdala in classical emotional conditioning [78, 79] and, on the other hand, the existence of a cortical and a subcortical route [80–84] through which perceptual stimuli might reach the amygdala (see Gainotti [85] for discussion). The covariance of right and left amygdala response with activity in other important subcortical structures during presentation of masked and non-masked conditioned emotional faces was considered separately. An increased connectivity between right amygdala, pulvinar, and superior colliculus was found during unconscious (masked) presentation of conditioned emotional stimuli. However, no masking-dependent changes in connectivity were observed among the same subcortical structures and the left amygdala. Morris et al. [77] concluded that emotionally laden stimuli can de detected, processed, and learned without conscious awareness by a right hemisphere subcortical pathway, mediating unconscious emotional learning.

Emotional conditioning, however, is not the only component of emotional memory that could be mainly linked to the right hemisphere. The long-term memory system, whose structure is schematically reported in Fig. 1, is indeed a complex system, which consists both of conscious (explicit/declarative) and of nonconscious (implicit/nondeclarative) subsystems.

Within the unconscious implicit subsystems, both emotional conditioning and preverbal implicit memories (which could be included in the "procedural subsystem," since they are a sensory-motor manifestation of neuroplasticity) could have a preferential link to the right hemisphere.

Spontaneous emotions are, indeed, accompanied by bodily changes, whose importance has been rightly stressed by Damasio [86], and produce motor habits and postures that can be considered as part of the "preverbal implicit memory system," whose importance as a nonrepressed unconscious nucleus of the self has been recently proposed by Mancia [87]. This preverbal implicit memory system could also be preferentially linked to the right hemisphere, because, according to Damasio [86], body-related information elicited by positive and negative emotional experiences is linked to a few critical brain structures: amygdala, ventro-medial frontal areas, and right somatosensory cortices. The right somatosensory cortices could, therefore, mainly subserve the implicit memory of postural and somatic changes linked to the earliest emotional experiences. Consistent with this hypothesis are data reported by Adolphs et al. [88], which show that damage to the right somatosensory cortex impairs per-

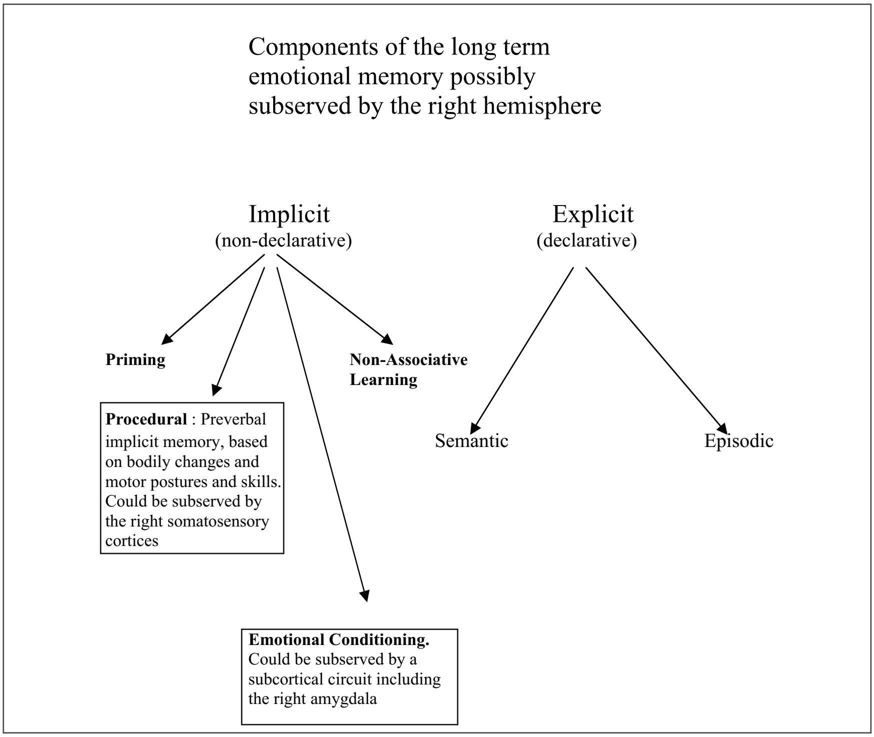

Fig. 1. Schematic illustration of the long-term memory system and of its components which could be preferentially linked to the right hemisphere

formance on "theory of mind" [89] tasks, namely on tasks requiring the capacity to form a representation of other people's mental states, probably via a process of internal simulation of other people's emotional status.

Concluding Remarks and Proposed Distinctions Among Unconscious Emotional Phenomena

Taken together, the theoretical models and empirical data reviewed in this chapter strongly suggest that the right hemisphere may be crucially involved in those unconscious emotional memories which must be reactivated and reworked during the psychoanalytical treatment. This statement concerns both the memory of events automatically eliciting (through a conditioning process) a specific emotional experience and those more stable postural and motor habits, which have been formed by repeated exposures to emotional situations during the earliest emotional experiences. The former could be mediated by a pathway linking subcortical sensory structures to the right amygdala, whereas the latter could be linked to the right somatosensory cortices and store those implicit memories, which are considered by Mancia [87] as the nonrepressed unconscious nucleus of the Self. These conclusions are obviously speculative and can be criticized from two different viewpoints. The first consists in claiming that the reported data and the suggested functional properties of the right hemisphere point more to a sort of "unawareness" than to a properly "unconscious" form of experience. The second consists, on the contrary, in assuming that the role of the right hemisphere in unconscious emotional processes may not be limited to the "procedural unconscious" (i.e., to the episodic or implicit somatic emotional memories that are not accessible to consciousness, but are not repressed) but may also concern the "dynamic unconscious". In our opinion, both these objections are highly problematic, since the distinction between "unawareness" and "unconscious experiences" is an ambiguous and controversial one, and since empirical results supporting the hypothesis of a crucial role of the right hemisphere in active defensive mechanisms are much less convincing than those concerning the generation and retrieval of nonrepressed unconscious emotional memories. In any case, to clarify this very complex issue, I would suggest some distinctive features which could help in distinguishing three kinds of right-hemisphere-related unconscious emotional phenomena, that could be labeled respectively "disease unawareness," "dynamic unconscious," and "procedural unconscious".

The term *disease unawareness* should be reserved for those pathological phenomena which are due to a lack of automatic orienting of attention towards the defective parts of the body, but that can easily be corrected if attention is properly directed to them. In patients with a right-hemisphere lesion, anosognosia for the left-sided hemiplegia could be a typical example of disease unawareness, due to a lack of automatic orienting of attention towards the defective parts of the body. The possibility that in these patients anosognosia for the left-sided hemiplegia may be markedly improved by reorienting attention towards the neglected side of space has been empirically demonstrated by Cappa et al. [90]. These authors have shown that some anosognosic patients become transiently aware of the previously ignored left-sided hemiplegia by strongly reducing neglect of the left half space with a caloric vestibular stimulation, which counterbalances the right-sided orienting bias.

The term *dynamic unconscious* should, on the other hand, be reserved for those pathological phenomena where the symptom has a symbolic value and cannot be acknowledged or improved by attentional or cognitive factors. Under this heading could probably be included conversion phenomena for the left side of the body and those instances of anosognosia for the left-sided hemiplegia that attracted the attention of Weinstein and Kahn [7]. As a matter of fact, in the previously mentioned study on the effects of a forced re-orienting of attention on nonspatial disorders of right-brain-damaged patients, Cappa et al. [90] noticed that some anosognosic patients still deny their left-sided hemiplegia, in spite of now having their attention equally distributed on both sides of personal and extrapersonal space.

Finally, the term *procedural unconscious* should be reserved for all those nonpathological conditions (such as emotional conditioning or emotional response to nonconsciously perceived stimuli), which are mainly subserved by the right hemisphere, because of the automatic processing of information typical of this hemisphere and of its crucial involvement in the "schematic level" of emotional processing.

The tentative nature of these suggested distinctions is clearly worth stressing and, in any case, further investigations are certainly needed to check whether these distinctions can help in clarifying the complex issue of the relationships between right hemisphere and unconscious emotional processes.

References

1. Sperry RW, Gazzaniga M, Bogen J (1969) Interhemispheric relationships: the neocortical commissures: syndromes of hemispheric disconnection. In: Vinken PJ, Bruyn GW (eds) Handbook of clinical neurology. North Holland Publishing, Amsterdam
2. Galin D (1974) Implications for psychiatry of left and right cerebral specialization: a neurophysiological context for unconscious processes. Arch Gen Psychiatry 31:572–583
3. McLaughlin JT (1978) Primary and secondary process in the context of cerebral hemispheric specialization. Psychoanal Q 47:237–266
4. Kandell ER (1999) Biology and the future of psychoanalysis: a new intellectual framework for psychiatry revisited. Am J Psychiatry 156:505–524
5. Ferenczi S (1926) An attempted explanation of some hysterical stigmata. In: Ferenczi S (ed) Further contributions to the theory and techniques of psychoanalysis. Hogarth Press, London, pp 110–117

6. Fenichel O (1945) The psychoanalytic theory of neurosis. Norton, New York
7. Weinstein EA, Kahn RL (1955) Denial of illness: symbolic and physiological aspects. Charles C Thomas, Springfield, Ill.
8. Briquet P (1859) Traité clinique et therapeutique de l'hysterie. Ballière, Paris
9. Pitres A (1891) Lecons cliniques sur l'hysterie et l'hypnotisme. Doin, Paris
10. Gilles de la Tourette G (1895) Traite clinique et therapeutique de l'hysterie. Plon Nourrit et Cie, Paris
11. Stern D (1977) Handedness and the lateral distribution of conversion reactions. J Nerv Ment Dis 164:122–128
12. Galin D, Diamond R, Braff D (1977) Lateralization of conversion symptoms: more frequent on the left. Am J Psychiatry 134:578–580
13. Ley RG (1980) An archival examination of an asymmetry of hysterical conversion symptoms. J Clin Neuropsychol 2:61–70
14 Axelrod S, Noonon M, Atancio B (1980) On the laterality of psychogenic somatic symptoms. J Nerv Ment Dis 168:517–525
15. Babinski J (1914) Contribution à l'étude des troubles mentaux dans l'hémiplégie organique cérébrale (anosognosie). Rev Neurol 27:845–848
16. Hoppe KD (1977) Split brains and psychoanalysis. Psychoanal Q 46:220–244
17. Freud S (1895) Project for a scientific psychology. In: Origins of psychoanalysis: letters to Wilhelm Fliess, drafts and notes, 1887–1902. Basic Books, New York
18. Bogen J (1969) The other side of the brain. II. An appositional mind. Bull Los Angeles Neurol Soc 34:135–162
19. Stone MH (1977) Dreams, free association and the non-dominant hemisphere: an integration of psychoanalytical, neurophysiological and historical data. J Am Acad Psychoanal 5:225–284
20. Terzian H, Cecotto S (1958) Su un nuovo metodo per la determinazione e lo studio della dominanza emisferica. G Psichiatr Neuropatol 87:889–924
21. Perria L, Rosadini G, Rossi GF (1961) Determination of side of cerebral dominance with amobarbital. Arch Neurol 4:173–181
22. Gainotti G (1969) Réactions "catastrophiques" et manifestations d'indifferénce au cours des atteintes cérébrales. Neuropsychologia 7:195–204
23. Gainotti G (1972) Emotional behavior and hemispheric side of lesion. Cortex 8:41–55
24. Davidson RJ (1984) Affect, cognition and hemispheric specialization. In: Izard CE, Kagan J, Zajonc R (eds) Emotion, cognition, and behavior. Cambridge University Press, New York, pp 320–365
25. Davidson RJ (1993) Parsing affective space: perspectives from neuropsychology and psychophysiology. Neuropsychology 7:464–475
26. Oatley K, Johnson-Laird P (1987) Toward a cognitive theory of emotions. Cogn Emot 1:29–50
27. Leventhal H (1974) Emotions: a basic problem for social psychology: classic and contemporary integrations. McNally, Chicago
28. Leventhal H (1987) A perceptual motor theory of emotion. In: Berkowitz L (ed) Advances in experimental social psychology, vol 17. Academic Press, New York, pp 117–182
29. Gainotti G (2001) Components and levels of emotion disrupted in patients with unilateral brain damage. In: Gainotti G (ed) Emotional behavior and its disorders: Handbook of neuropsychology, vol 5, 2nd edn (Boller F, Grafman J, series eds). Elsevier, Amsterdam, pp 161–180
30. Gainotti G (2003) Emotional disorders in relation to unilateral brain damage. In:

Feinberg TE, Farah M (eds) Behavioral neurology and neuropsychology. McGraw-Hill, New York, pp 725–735

31. Borod J, Tabert MH, Santschi C, Strauss EH (2000) Neuropsychological assessment of emotional processing. In: Borod J (ed) The neuropsychology of emotions. Oxford University Press, New York, pp 80–105
32. Gainotti G (2000) Neuropsychological theories of emotions. In: Borod J (ed) The neuropsychology of emotions. Oxford University Press, New York, pp 214–238
33. Blonder LX, Bowers D, Heilman KM (1991) The role of the right hemisphere in emotional communication. Brain 114:1115–1127
34. Borod JC (1993) Cerebral mechanisms underlying facial, prosodic, and lexical emotional expression: a review of neuropsychological studies and methodological issues. Neuropsychology 7:445–463
35. Borod JC, Zgaljardic D, Tabert MH, Koff A (2001) Asymmetries of emotional perception and expression in normal adults. In: Gainotti G (ed) Emotional behavior and its disorders: Handbook of neuropsychology, vol 5, 2nd edn (Boller F, Grafman J, series eds). Elsevier, Amsterdam, pp 181–205
36. Ross ED (1981) The prosodias: functional-anatomical organization of the affective components of language in the right hemisphere. Arch Neurol 38:561–569
37. Ross ED (1984). Right hemisphere's role in language, affective behavior and emotion. Trends Neurosci 7:342–346
38. Wittling W (2001) Brain asymmetry in the control of the stress response. In: Gainotti G (ed) Emotional behavior and its disorders: Handbook of neuropsychology, vol 5, 2nd edn (Boller F, Grafman J, series eds). Elsevier, Amsterdam, pp 207–232
39. Heilman KM, Schwartz HD, Watson RT (1978) Hypoarousal in patients with neglect and emotional indifference. Neurology 28:229–232
40. Morrow L, Vrtunski PB, Kim Y, Boller F (1981) Arousal responses to emotional stimuli and laterality of lesion. Neuropsychologia 19:65–71
41. Meadows ME, Kaplan RF (1994) Dissociation of autonomic and subjective responses to emotional slides in right hemisphere damaged patients. Neuropsychologia 32:847–856
42. Wittling W (1990) Psychophysiological correlates of human brain asymmetry: blood pressure changes during lateralized presentation of an emotionally laden film. Neuropsychologia 28:457–470
43. Wittling W (1995) Brain asymmetry in the control of autonomic-physiologic activity. In: Davidson RJ, Hugdahl K (eds) Brain asymmetry. MIT Press, Cambridge, Mass., pp 305–357
44. Mammucari AC, Caltagirone C, Ekman P et al (1988). Spontaneous facial expression of emotions in brain-damaged patients. Cortex 24:521–533
45. Wittling W, Roschmann R (1993) Emotion-related hemisphere asymmetry: subjective emotional responses to laterally presented films. Cortex 29:431–448
46. Hugdahl K (1987) Pavlovian conditioning and hemispheric asymmetry. In: Davey GLC (ed) Cognitive processes and Pavlovian conditioning in humans. Wiley and Sons, Chichester, UK, pp 147–182
47. Hugdahl K, Brobeck CG (1986) Hemispheric asymmetry and human electrodermal conditioning: the dichotic extinction paradigm. Psychophysiology 23:491–499
48. Johnsen BH, Hugdahl K (1991) Hemispheric asymmetry in conditioning to facial emotional expressions. Psychophysiology 28:154–162
49. Johnsen BH, Hugdahl K (1993) Right hemisphere representation of autonomic conditioning to facial emotional expressions. Psychophysiology 30:274–278

50. Lamendella JT (1977) The limbic system in human communication. In: Whitaker H, Whitaker HA (eds) Studies in neurolinguistics. Academic Press, New York, pp 157–222
51. Buck R (1984) The communication of emotion. Guilford Press, New York
52. Rinn WE (1984) The neuropsychology of facial expression: a review of the neurological and psychological mechanisms for producing facial expression. Psychol Bull 95:52–77
53. Gainotti G, Caltagirone C, Zoccolotti PL (1993). Left/right and cortical subcortical dichotomies in the neuropsychological study of human emotions. Cogn Emot 7:71–93
54. Gainotti G (1983) Laterality of affect: the emotional behavior of right and left brain-damaged patients. In: Myslobodsky MD (ed) Hemisyndromes: psychobiology, neurology, psychiatry. Academic Press, New York, pp 175–192
55. Buck R, Duffy RJ (1980) Nonverbal communication of affect in brain-damaged patients. Cortex 16:351–362
56. House A, Dennis M, Molyneux A et al (1989) Emotionalism after stroke. Br Med J 298:991–994
57. Sackeim HA, Gur RC (1978) Lateral asymmetry in intensity of emotional expression. Neuropsychologia 16:473–481
58. Schwartz GE, Ahern GL, Brown SL (1979). Lateralized facial muscle response to positive and negative emotional stimuli. Psychophysiology 16:561–571
59. Etcoff N (1986) The neuropsychology of emotional expression. In*:* Goldstein G, Tarter RE (eds) Advances in clinical neuropsychology. Plenum Press, New York, pp 127–179
60. Luria AR (1966) Higher cortical functions in man. Basic Books, New York
61. Gazzaniga MS (1995) Principles of human brain organization derived from split-brain studies. Neuron 14:217–228
62. Gazzaniga MS, Smylie CS (1990) Hemispheric mechanisms controlling voluntary and spontaneous facial expressions. J Cogn Neurosci 2:239–245
63. Gainotti G (1993) The role of spontaneous eye movements in orienting attention and in unilateral neglect. In: Robertson IH, Marshall JC (eds) Unilateral neglect: clinical and experimental studies. Erlbaum, Hove, UK, pp 107–122
64. Gainotti G (1993) Lateralization of brain mechanisms underlying automatic and controlled forms of spatial orienting of attention. Neurosci Biobehav Rev 20:617–622
65. McGinnies E (1949) Emotionality and perceptual defense. Psychol Rev 56:244–251
66. Lazarus RS, McCleary RA (1951) Autonomic discrimination without awareness: a study of subception. Psychol Rev 58:113–122
67. Ohman A, Wiens S (2001) To think and to feel: nonconscious emotional activation and consciousness. In: Kaszniak A (ed) Emotions, qualia and consciousness. World Scientific, Singapore, pp 235–246
68. Marcel A (1983) Conscious and unconscious perception: an approach to the relation between phenomenal experience and perceptual processes. Cogn Psychol 15:238–300
69. Ohman A, Soares JJF (1994) Unconscious anxiety: phobic responses to masked stimuli. J Abnorm Psychol 103:231–240
70. Dimberg UM, Thunberg M, Elmehed K (2000) Unconscious facial reactions to emotional facial expressions. Psychol Sci 11:86–89
71. Morris JS, Ohman A, Dolan RJ (1998) Conscious and unconscious emotional learning in the human amygdala. Nature 393:467–470

72. Erderlyi MH (1974) A new look at the New Look: perceptual defense and vigilance. Psychol Rev 81:1–25
73. Posner MI (1978) Chronometric explorations of mind. Erlbaum, Hillsdale, N.J.
74. Shiffrin RM, Schneider W (1977) Controlled and automatic human information processing. II. Perceptual learning, automatic attending and a general theory. Psychol Rev 84:127–190
75. Làdavas E, Cimatti D, Del Pesce M, Tuozzi G (1993). Emotional evaluation with and without conscious stimulus identifications: evidence from a split-brain patient. Cogn Emot 7:95–114
76. Spence S, Shapiro D, Zaidel E (1996) The role of the right hemisphere in the physiological and cognitive components of emotional processing. Psychophysiology 33:112–122
77. Morris JS, Ohman A, Dolan RJ (1999) A subcortical pathway to the right amygdala mediating 'unseen' fear. Proc Natl Acad Sci USA 96:1680–1685
78. LeDoux JE (1986) Cognitive-emotional interactions in the brain. Cogn Emot 3:267–289
79. LeDoux JE (1996) The emotional brain. Simon and Schuster, New York
80. Papez JW (1937) A proposed mechanism of emotion. Arch Neurol Psychiatry 79:217–224
81. Russchen FT (1982) Amygdalopetal projections in the cat. II. Subcortical afferent connections: a study with retrograde tracing techniques. J Comp Neurol 207:157–176
82. LeDoux JE, Sakagachi A, Reis DJ (1984) Subcortical efferent projections of the medial geniculate nucleus mediate emotional responses conditioned by acoustic stimuli. J Neurosci 4:683–689
83. LeDoux JE, Ruggiero DA, Reis DJ (1985) Projections to the subcortical forebrain from anatomically defined regions of the medial geniculate body in the rat. J Comp Neurol 242:182–213
84. LeDoux JE, Sakaguchi A, Iwata J, Reis DJ (1986). Interruption of projections from the medial geniculate body to an archi-neostriatal field disrupts the classical conditioning of emotional responses to acoustic stimuli in the rat. Neuroscience 17:615–627
85. Gainotti G (2001) Emotions as a biologically adaptive system: an introduction. In: Gainotti G (ed) Emotional behavior and its disorders: Handbook of neuropsychology, vol 5, 2nd edn (Boller F, Grafman J, series eds). Elsevier, Amsterdam, pp 1–15
86. Damasio AR (1994) Descartes' error: emotion, research and the human brain. Avon, New York
87. Mancia M (2003) Dream actors in the theatre of memory: their role in the psychoanalytic process. Int J Psychoanal 84:945–952
88. Adolphs R, Damasio H, Tranel D, Cooper G, Damasio A (2000) A role for somatosensory cortices in the visual recognition of emotion as revealed by three-dimensional lesion mapping. J Neurosci 20:2683–2690
89. Frith CD (1992) The cognitive neuropsychology of schizophrenia. Erlbaum, Hove, UK
90. Cappa S, Sterzi R, Vallar G, Bisiach E (1987) Remission of hemineglect and anosognosia during vestibular stimulation. Neuropsychologia 25:775–782

Chapter 6

Psychoanalysis and Neurosciences: Anxiety in Perspective

Luigi Cappelli

Clinical Forms of Anxiety

Among the forms of pathological anxiety, the DSM-IV-TR [1] distinguishes, in addition to generalized anxiety, phobias, panic attacks, post-traumatic stress disorder (PTSD), and obsessive-compulsive disorder (OCD). There are some theories that since OCD involves structural and functional organic deficits, it is questionable whether it can be classified as an anxiety or depressive disorder [2]. The ICD-10 [3] in fact classifies this disorder separately from the other manifestations often associated with it.

Pathological and Normal Anxiety

Pathological anxiety is a weighty social problem. It is estimated that about 25% of the general population suffers, or has suffered, anxiety manifestations that significantly affect their mental equilibrium [4]. This is in fact the most widely encountered problem in mental pathology. If we then add that physiological anxiety can take a wide range of forms, there is no question that it is present throughout mental life. Both pathological and physiological anxiety, therefore, merit all the attention they can get from clinicians and researchers.

A distinction between normal and abnormal anxiety is a useful basis for defining anxiety as such, since normal anxiety plays a role in adaptation, while the pathological form tends to lead to progressive dysfunction.

Taking fear as a stimulus that usefully activates an organism's resources in response to a definite danger, then the nearer anxiety comes to fear, in its intensity and specificity, the more we can consider it useful for survival. Even an undifferentiated state of activation can help avoid potential danger, so a certain degree of anxiety–in the sense of a form of fear with no immediate reason–can be useful for survival too: in the natural state it prepares the organism for reaction to a possible threat. The boundary between normal and abnormal is not quite as clear as it might seem at first glance, therefore, but too strong or constant anxiety is, of course, harmful because it causes neurological damage and may progress to a state of depression.

Genetics

Neoteny in the higher mammals makes their offspring highly dependent on interaction with others of their species while they grow up, in order to learn to balance fear and anxiety according to a cost–benefit ratio. However, humans appear to be genetically predisposed to anxiety, which is probably a trait selected as giving a survival advantage. About 70% of the normal population have a genetic profile that penalizes serotonin transport, so they develop relatively high levels of anxiety. The other 30% of healthy individuals enjoy a genetic pattern that facilitates serotonin transfer, raising their levels of serenity [5]. These findings have led to the conclusion that in most people anxiety is probably a beneficial stimulus that boosts mental resources, in normal conditions, while the unworried–although subjectively enviable–are more likely to come to harm.

Temperament and Environment

Some types of anxiety, particularly panic attacks, can be traced back to the sufferer's separation from the parents while still an infant, which predisposes them to pathological anxiety. Kendler et al. [6], in a study of a series of 1018 twin-pairs, showed that separation from parents, or their death, was correlated with the onset of panic attacks in the children [7]. Gabbard also notes that Kagan et al. [8] described a type of child that showed fear and behavioral inhibition when faced with the unknown.

These infants tend to ask their parents repeatedly for reassurance, and throw temper tantrums if their demands are not satisfied. The anger aggravates the problem, because it is likely to produce negative reactions in the parents, and a sense of guilt in the children, who fear they may damage the bond with these significant figures in their lives. This therefore develops into a condition of hostile, fearful dependence which is the ideal terrain for pathological anxiety to flourish on later [9, 10].

This sort of anxious attachment has been associated with difficulties in managing the normal oscillations between attachment and separation, and overt manifestations of acute anxiety [11].

Emotions and Anxiety

Fear, joy, sadness, anger, surprise, and disgust are all basic emotions, widely considered to be innate. On the topic of primary emotions, Guido Gainotti [12] agrees with the distinction made by Oatley and Johnson-Laird [13] between the emotional system and the cognitive system. The former is defined as a primitive emergency system that processes a restricted number of signals and promptly evokes unconscious, automatic responses; the latter is considered a more developed and complex system of adaptation, which can deal with the signals in more detail and with more awareness, but which takes longer to work out its responses.

To explain the progression from elementary to more complex emotional forms, Gainotti cites Leventhal [14, 15] who proposed three ontogenic levels in emotional development, which can be ranked as follows. The first is the sensory-motor level, where a series of innate neuromotor programs, automatically activated by stimuli, induce neurovegetative and motor responses. During development, these basic programs become intertwined with individual learning, giving the second level, the "schematic level." This consists of emotional schemes corresponding to the typical spontaneous emotions. The third level of organization is the "conceptual level," where we have the conscious mechanisms of the declarative memory, conceptual abstraction and awareness of social roles in emotional expression, even if the individual does not experience the emotion itself.

Patients with right hemisphere brain damage suffer impairment in their expression and understanding of emotions, indicating that this hemisphere plays a major part in generating emotions. Gainotti suggests that in addition to the communication aspect, the right hemisphere is specialized in producing neurovegetative responses in the subjective experience of emotions, and the emotional conditioning related to positive or negative emotions.

On the topic of specialization of the hemispheres, and with reference to Leventhal, the left-hand hemisphere appears to deal with the conceptual level of the elaboration of emotions, and the right-hand hemisphere with the schematic level. This means that the basic aspects of emotions are processed mainly on the right, and the finer aspects, such as vanity or shame, call for the functions of the left hemisphere.

Morris et al. [16], using PET, showed experimentally that the right-hand amygdala was functionally activated when a subject was faced with masked adverse stimuli, but was not aware of them. Only the left amygdala was functionally activated when the subject was aware of the stimulus. In line with these studies, two response pathways are described, mediated by the amygdala, to emotional and anxiety-producing stimuli; one is rapid, subcortical and unconscious, and the other is slower, cortical and conscious [17–19].

It seems clear, therefore, that the right hemisphere has a role in processing unconscious elementary emotions, whether good or bad; this helps explain the pattern of affective indifference and denial of hemiplegia in patients with right-brain lesions. Patients with lesions to the left, on the other hand, show anxiety and depression about the neurological damage, which can be considered a normal reaction of alarm and despair at the functional loss, triggered by the intact right hemisphere.

Anxiety can therefore be considered a normal extension of fear, a "negative" emotion that elicits immediate reactions or responses based on external and internal stimuli, linked together.

Pleasure–Unpleasure and the Primary Ego

On the question of the functional location of the pleasure–unpleasure system, the periaqueductal gray (PAG) appears to play a major role; it has two sectors, in the brainstem–a ventral sector that processes pleasant sensations, and a dorsal one that generates unpleasant ones [20]. The PAG, together with other structures (hypothalamus, tegmentum, parabrachial nuclei, raphe nuclei, nucleus of the locus coeruleus, and reticular formation), elaborate visceral states. The PAG also processes somatosensory sensations of pain, and painful emotions. Close to this area, at the dorsal tegmentum and the tectum of the trunk, there is a restricted area that receives sensory data from the musculoskeletal apparatus, thus from inside and outside the body. These sensations may be pleasant or unpleasant, and a dense network of connections to the motor cortex permits the automatic responses of moving nearer or further away, body expressions, and mimicking emotions. Jaak Panksepp [21] therefore identified this region as the site of an elementary, primary Ego, which he called a Simple Ego-like Life Form, or SELF.

The Amygdalas

The amygdalas are the basic functional brain centers for identifying potentially dangerous stimuli [4]. It is quite possible that the amygdala deals specifically with fear, while the bed nucleus of the stria terminalis (BNST), a nearby structure, deals with the processes of anxiety [22, 23].

Le Doux [4] provided a description of the mechanism of activation of stress, and I shall refer to this at various points in this paper. Le Doux takes an approach intended to overcome the problem of scientific reliability, inevitable when trying to define emotional phenomena, by focusing on cognitive, emotional, and motivational aspects, which can be described in objective terms on the basis of the neurochemical processes underlying them.

In response to anxiogenic stimuli, the central nucleus of the amygdala solicits, either directly or indirectly through the BNST, the hypothalamic paraventricular nucleus (PVN) and the locus coeruleus. This latter is essential for the production of noradrenaline, which raises blood pressure, accelerates heart rate, and–with the nucleus accumbens–activates a motor reaction; the PVN then produces corticotropin-releasing factor (CRF), which stimulates the pituitary (PIT) to release adrenocorticotropic hormone (ACTH), which is carried in the bloodstream to the adrenals. Cortisol, a hormone secreted by the capsules, then returns to the brain in the bloodstream, regulating pituitary stimulation. The body thus finds the sympathetic system activated and can prepare itself–or at least predispose itself–to attack or to flee.

Cortisol activates the hippocampus to record explicit memories that are particularly significant for survival, but when there is too much stimulation, or for too long a duration, glucose depletion occurs in the hippocampal cells. These cells then become vulnerable to subsequent stimuli and may suffer atrophy or even die. This influences hippocampal neurogenesis, which is inhibited. These events help explain the memory defects in patients with PTSD or depression. Cortisol also causes damage to the prefrontal cortex, impeding decision-making processes.

The amygdala is part of a system of reciprocal regulation with the adrenals, as it is sensitive to the adrenaline and noradrenaline they produce, and governs the acquisition of explicit memories, generated in conditions of emotional activation. When the emotional stimulus is adequate, the memory is potentiated but it may have difficulty dealing with excessive stimuli, and the hippocampus finds it hard to establish the right context for an event that is emotionally important. If we add that these feedbacks also influence the processes of attention, we can see how important these neurobiological aspects are in describing automatic brain operations related to emotional states, in an area of unconscious functioning. Hippocampal impairment may lead to difficulty in putting fear and normal anxiety into their appropriate context, paving the way for dysfunction and pathological symptoms.

On the functional synergy between the amygdala, hippocampus, and cerebellum [24], it is worth remembering that the cerebellum and pons must be intact for the memory to consolidate itself, and to put anxiety-arousing situations into context, though we still do not know much about how these regions work. In reversible inactivation experiments, Attwell [25] reached conclusions similar to those of Sacchetti et al. [26] on the role of the cerebellar cortex in recording memory traces.

These findings underscore the importance of knowledge of brain processes and their implications, in seeking possible solutions to problems. In new or critical situations, conscious attention is aroused and the chain of associative memory is activated to find the best answers.

Attachment

Regina Pally [27] recalls observations by Hofer [28, 29] on newborn and breast-feeding rat pups, on the topic of attachment. Contact with the mother stimulates physiological functions such as heart rate, temperature, and activity level. Separation from the mother causes separation anxiety, and the animals emit a “distress cry,” using the same laryngeal muscles as human babies use in similar situations. If the mother is absent for a long time, despair sets in, with associated dysfunctions of biological regulation, such as bradycardia and hypoactivity. Hofer noted the importance of attachment bonds in biological regulation throughout life: for instance, the death of a spouse can increase the survivor's vulnerability to cardiovascular and other diseases, probably because of some weakening of the immune-endocrine system. Infusion of benzodiazepine receptor agonists into the amygdala reduces the distress cry due to separation, and antagonists increase it, even if the mother is present [30].

Kalin et al. [31], investigating reunion behavior in primates, found that when they were given naltrexone, an opioid antagonist, reunion manifestations were boosted, whereas morphine, an opioid agonist, had the opposite effect. Kalin suggested that reunion stimulated the production of endogenous endorphins.

Amphetamine and cocaine induce excitement and anxiety, arousing expectations and satisfaction-seeking, while the opiates induce feelings of satiety and weaken attachment. Lactate injected into the locus coeruleus, which produces 60% of the brain's noradrenaline, can induce a panic attack in predisposed subjects [7]. A benzodiazepine or selective serotonin re-uptake inhibitor (SSRI) injected into the amygdala reduces the manifestations of anxiety. The benzodiazepines and imidazopyridine act promptly on free anxiety, promoting GABAergic inhibitory action. Tricyclic antidepressants, anti-MAO agents, and SSRI relieve the symptoms of other forms of anxiety, increasing the availability of monoamines and serotonin [4].

Attachment dysfunction can give rise to confusional states, anxiety, and depression [32–35]; traumatic experiences of separation, especially from the mother, or massive exposure to evasive, contradictory, or disorganized parental models, can cause terror, ambivalence and dissociation in the children.

Paranoid and Depressive Anxieties

The broad variety of clinical psychoanalysis situations includes a range of forms of pathogenic anxiety, reviewed by Gabbard [7]. Freud, Melanie Klein, Winnicott, and Kohut have all dealt with the topic, and this is clearly not the place to attempt a summary. However, while supporters of the "conflict theory," including Freud and Klein, have worked to define the intrapsychic facets of anxiety, the "deficit theory," upheld by thinkers like

Winnicott and Kohut, focuses more on the relational aspect of worry and anxiety [36]. As Mitchell and Black point out [37], Sullivan considered maternal anxiety a fundamental reference point in the child's development, forming a "good me," "bad me," or "non-me" depending on the mother's reactions.

Freud [38] saw anxiety mainly in relation to weakening of repression, under the impact of instincts. The ego emits an anxiety signal as libidinal and destructive impulses emerge from the id, incompatible with the dictates of the super-ego.

Melanie Klein [39] laid particular stress on the early, terrifying anxiety resulting as the constitutional destructive potential, the death instinct, emerges in the child's mind. This anxiety sets up unconscious processes of affirmation of a perfect, omnipotent identity, with denial of the internal destructive impulse, splitting off of the dangerous aspects and projection of these components onto family members, objects of the outside world, or the person's own organs. The targets of this projective identification become dangerously destructive in the mind of its author, who is not aware of the process, but who "expels" them from his mind, and they subsequently set in motion persecutory or hypochondriac anxieties. According to Klein, however, these identification processes do not change their "containers," except in the mind of the person making the identification.

Klein always considered the relation between subject and object fundamental to development and instinctual vicissitudes, though mainly in the person's internal world. Only Bion [40] believes the "container" of the identification is strongly influenced emotionally, and responds on the same level of exchange, or by processing the events so as to return them to the sender in better shape.

Klein [41, 42] maintains that during good development, gratifying experiences at the breast and with the mother figure as a whole, by fostering the primacy of libidinal aspects, enable the child to re-introject the "expelled" destructiveness in a way that facilitates its integration. However, this process of taking possession once more involves guilt feelings and depressive suffering, as the child becomes "conscious" of his own destructive impulses.

A stage of persecutory anxiety is then followed by a phase of depressive anxiety, linked to the fear of being overwhelmed, along with one's significant figures, by the destructive facets of the impulse. The encounter with the risk of losing a loved object arouses the instinct to repair the damage produced in the imagination, so as to rebuild self-confidence and the bond, as a sign of love and care. If something arises to stop this progression, for instance, excessive aggression causing intolerable frustration, the child risks regressing to the paranoid-schizoid position, or may present manic-depressive symptoms, where denial of the destruction alternates with despair for the loss of the bond. Failures during development are fixation points from which the child can subsequently regress when faced with fresh hurdles.

Once past the paranoid-schizoid and depressive phases, a person can find themselves later in a paranoid-schizoid or depressive position in any situation that revives memories of the past circumstances. This permits further work on mourning for one's own ideal identity and that of the idealized love objects, allowing re-affirmation of love of oneself and one's objects, though the gratification is always accompanied by the frustration of aspirations toward omnipotence.

Briefly, therefore, Freud and Klein defined mainly the vicissitudes of the internal, fantasy world, and Winnicott and Kohut spent more time on actual relations and the external reality of mothering. Today we can try to combine these two approaches in a unified vision that takes account of clinical experience related to the theories of conflict and deficit. A proposal put forward by Otto Kernberg [43] contains material derived from Kleinian theory regarding the world of internal relations, against a solid background of relational reality. Stephen Mitchell [44] also aims to overcome the traditional dichotomy.

Here I want to look at two major forms of anxiety: paranoid and depressive. Klein gave good descriptions of the internal aspects of mental conditions strongly linked to survival: fear of predators, of violent attack from outside, and of being abandoned by caring figures. These events can certainly cause the death of immature creatures, not just in a child's imagination. Such events actually happen in the outside world, though they can be imaginary too, and can trigger internal fear, anger and defenses very similar to those Klein describes as purely internal phenomena.

There is no point in digressing into a full exploration of the stages of development set out by Klein, but Balint [45] and then Bleger [46] and Ogden [47] all proposed changes to Klein's theory. Balint and Bleger defined stages of development prior to the Kleinian paranoid-schizoid phase: Balint went for primary love and Bleger for the "glischro-carica" position or agglutinate nucleus, a residue in the mind of early experiences of fragmentation and nonintegration. Ogden's strategy, on the other hand, flanks the contiguous-autistic position with the classic Kleinian paranoid-schizoid and depressive positions in a view that alternates the three conditions, or sometimes runs them simultaneously.

These are fascinating evolutionary themes but here I shall simply stress the importance, in clinical theories, of an overall view of the external and internal worlds, considering that they are inseparable in phylogenesis and ontogenesis, entwined functionally for the acquisition of the knowledge essential to survival.

According to Mancia [48–50], fear and anxiety stored in the emotional and affective implicit memory can to some extent become independent of external references that might trigger them. These memories, however, exert a constant pressure on a person's conscious psychic life and on the declarative memory, even if they are not formulated verbally and cannot easily be called up voluntarily. As Mancia points out, in psychoanalysis these nonthinkable, nonsymbolic emotional levels tend to surface in nonverbal form, in the tones and rhythms of the voice, and the structure of the language, more than the content of what the patient is saying. Dreams and the transference are other gateways through which they can irrupt in analysis. Mancia considers this level of memory as part of an unrepressed unconscious that may contain traumatic experiences. These presymbolic features, manifested in the "musicality" of the patient's language in the transference and through the figurative and symbolopoietic aspects of the dream, can thus be defined in explicit, conscious terms.

Paranoid and depressive anxieties may be powerful signals of unconscious affects in the psychoanalytical relation, sending the transference in a certain direction. The fear of being attacked or abandoned by the analyst, once dependence has set in, will influence the style of the relationship, particularly the negative side of the transference. If these processes can be shared and worked through together it will be much easier to integrate them and overcome the problem.

There may well be anxieties dating even farther back than those described by Melanie Klein: Gabbard [7] speaks of "disintegration anxiety" to describe this very early terror of losing one's own integrity if the fundamental relation is threatened by internal or external vicissitudes or if the line between the self and the object becomes blurred, creating a risk of merging with the other significant figure.

Fear, Anger, Panic

Neuroscientists tend to distinguish two separate paths for processing anxiety. Within certain limits they do correspond to the two lines set out by psychoanalysis: paranoid and depressive. Two functional pathways have been identified. The one for anxiety seems to be based on the central and lateral nuclei of the amygdala, while panic or separation anxiety seems to come from the anterior cingulate gyrus which, in turn, has dense thalamic and hypothalamic connections (as well as with the BNST and the ventral-tegmental area). All these areas are essential for sexual and maternal behavior in the lower mammals [20].

This system is also greatly influenced by endogenous opioids–as also by oxytocin and prolactin, whose role in facilitating mothering is well known. As an aside, in some autistic children the opioid system is hyperactive, and this might be related to their reduced need for affective exchange.

Separation is painful because of the lowered levels of cerebral opioids, which normally stimulate the infant, who depends on its carer figure close by to boost its subjective well-being, and help ensure survival. The child reacts to separation first by seeking then by withdrawing; this is rather like the way a prey “freezes” to escape predators, and bears some resemblance to true depressive withdrawal.

The fear system is also connected with flight responses, through the short subcortical pathway, and with attack through the medial nucleus of the amygdala. The attack reaction is based on anger (anger–rage), a very heated aggressivity accompanied by activation of the sympathetic autonomous system. It is stimulated by frustration of biologically important aims. The “cold” types of aggressivity, such as predatory aggression and forms related to the dominant role, seem to use different neuronal pathways, shared with seeking processes.

Recollections and Forecasts

The normal form of fear can thus develop into survival anxiety, in the face of the danger of attack from outside, or of a separation, or of bodily malfunctioning. The emotive brain develops before the cognitive one, and fears learned and archived early in the procedural memory tend to persist over the years, even if development of the cortical functions inhibits their manifestation to outsiders [4]. We also know that our brain tends to anticipate events, in order to be ready to assess and respond to a danger; it does this by drawing on an outline of previous experience as applicable to current perception of the situation, without waiting to specifically assess the details. At the same time it works out possible outcomes, on the basis of what it already knows might happen. This is the "closed system" theory, offered by Llinas and Pare [51] and authoritatively upheld by Edelman and Tononi [52]. The brain, though always in a state of "osmosis" with the outside world, tends unconsciously to forecast events on the basis of previous happenings.

These findings seem closely compatible with the idea of an inner world and with the transference in psychoanalysis. The inner world is not only made up of sensory perceptions from outside. We know that the processes of perception, especially the top-down (descending) ones, are heavily influenced by early experiences stored in the implicit memory, which therefore cannot be formulated in a declarative or explicit form.

Drawing on this knowledge of brain function, it becomes clear that the mind is quite capable of suffering persecutory and depressive anxiety that is detached from the appropriate context and can become generalized, superimposing itself on subsequent perceptions and altering them. Hypothetically, therefore, unfavorable or inadequate mothering might lead to both forms.

Clinical experience shows that when the search for satisfaction of basic needs is frustrated, a secondary form of aggression develops; this in turn may be inhibited because the carer figure(s) may penalize its expression. This aggressivity can fuel persecutory and depressive anxiety if the expected danger comes from the outside world; likewise, if the threat appears to come from inside the body, the anxiety may take a hypochondriac form.

The mechanism Klein described as projective identification can be viewed as a clinical manifestation of a process that has now been confirmed in other areas of investigation. The expectation of danger may be independent of the appropriate context, boosted by emotions triggered in the subject under the influence of repressed, unconscious recollections, and by the unrepressed, unconscious memory of early experience.

Physiological fear and anxiety are fundamental feelings on which, if genetic and environmental conditions are unfavorable, pathological symptoms can develop, taking either a paranoid or a depressive pattern (or disintegrative or hypochondriac). Phobias and obsessions are attempts to control these anxieties, by reinforcing the inhibitory processes in the frontal cortex that normally act as filters to early appetitive urges.

In psychoanalytic therapy, it is conceivable that the supportive-expressive aspects of the exchange are in fact new declarative formulations of context, using the frontal cortex and hippocampus, for implicit emotional experiences from childhood. These early events are mainly processed by the amygdala, and only later by the basal nuclei and pons/cerebellum, which need longer to mature. This rearrangement fosters the growth of new synapses, with different functional organization, mainly a much greater degree of integration between affects and knowledge formulated in symbols.

Affectionless Control and Anxiety

The caring environment can induce anxiety and depression. The Australian researcher Gordon Parker described a model of parental caring which involved emotional coldness and a tendency to control the children, termed "affectionless control," which he maintained could induce depression in the infants [53–55]. He also devised a questionnaire, the "parental bonding instrument" (PBI), for administration to the children of these parents, asking them to describe their parents' attitude to them, on the basis of their recollections of care. He found a significant relation between "affectionless control" and depression among the children, and maintained that this was not a subjective interpretation by the children of their parents' behavior, but reflected the parents' actual characteristics.

With Pietro San Martini, I have employed this method to investigate more than a thousand subjects. The PBI has been fleshed out with a series of additional items, suggested by the British researchers Gilbert et al. [56] to add predictive power to the original questionnaire. The findings with this "reinforced" version confirmed Parker's results: affectionless control can cause depression in the children [57].

We subsequently extended the study to manifestations of anxiety, and found this too was significantly correlated with the parental attitude (Cappelli L, San Martini P: Stile di accudimento genitoriale, ansia e depressione nei figli - in prep.). Thus a lack of affection combined with severe control can frustrate the process of attachment, which requires empathy and confidence, resulting in anxiety and depression.

These findings confirm clinical observations on the role of significant relations in producing psychopathological symptoms; neuroscientific clarification helps us understand how and through which pathways the symptoms arise. This marks the start of an integrated concept of neurology, psychology and the various disciplines involving care of the mind.

Clinical Considerations

This seems an appropriate moment to outline some summary guidelines for clinical work, on the basis of points brought to light by developments in neuroscience.

We have seen that it is essential during development and for adult mental life to have implicit, presymbolic, preverbal memories in the brain. These memories influence feelings, valuations and thoughts, even if they cannot be brought to the surface immediately, set in context and defined so they are clearly accessible to their "host" and communicable as a narrative to other people. These memories, which tend to be stable, form the basis for our forecasts of what is likely to happen in future situations and relations.

Using a process of "pattern matching" [27] we run through our current perceptive information, assigning to it the characteristics of similar situations we have encountered in the past. The present is instantly compared, automatically, with the past on the basis of compatibilities with what we already know. If there are substantial similarities, the memory immediately displays a series of emotions and associations we have already experienced, and offers any solutions available. If the situation involves something we know nothing about, or differs markedly from what we can recall from the memory, our attention and awareness are aroused, and our ability to connect, and we look more closely at the perceptive data, widening our references to experience in the light of this awareness and, if necessary, formulating a new type of experience to include greater adaptability and complexity in the evaluation.

This process, with its greater detail, is more fluid and distinctly divided, therefore slower, and is hard to set in motion in conditions of anxiety. But our memory can still offer the speedy, automatic, well-trodden pathway that tends to take priority in states of alarm.

This takes us back to clarify some basic points in psychoanalytical theory and clinical work: the importance of childhood memories in the adult's mental life; the fact that these memories are beyond our awareness, unconscious but not repressed, and can be brought to light in the transference and in dreams [48–50]; the basic role of therapy for emotionally re-living in the transference experience that cannot be recalled but that, in the right conditions of development and with the right relationship, can be worked through and integrated, in the light of the consciousness and verbalization of the intense, often confused, emotional states they arouse.

We also have confirmation of dysfunctional processes arising out of experiences that were so painful and paralyzing that they could not be thoroughly assimilated, giving rise to failure to integrate properly, dissociation, and splitting. This is a particularly interesting point because there has been much discussion of the best ways of helping patients with these pathologies to integrate their disturbing experiences through psychotherapy. There appears to be widespread agreement that a well-divided, organic relation with the therapist, including therapeutic exchange of symbolic and particularly presymbolic aspects, is a good way of emotionally breaking down the calcifications of the memory, helping the reconstruction that enables the psychic components to live together more serenely, particularly the cooperation–and synergy–between conscious and unconscious functions.

With specific reference to anxiety, we can speculate that some clinical forms of neurosis or psychosis, not traceable to recognizable current causes, and thus not reactive, are in fact closely bound up with the unconscious memory. This is where traces of experience remain, not repressed and not remembered, but acted out in symptomatic manifestations, obscuring the higher functions of thought.

It is clear that psychotherapy today cannot do without the emotional participation of the therapist in the interaction with the patient. The state referred to variously as identification, empathy, sharing, syntony, tuning in, mirroring, or resonance holds top rank in this therapeutic process. This does complicate the therapist's task, but at the same time it raises thorny methodological problems in research, especially when negative emotions are involved–as so often in pathological processes–from which not only the patient but the therapist too tends to keep their distance.

I am personally convinced from long clinical experience that psychoanalysis is still a basic reference point in the therapy of these disorders, by sensitively working at length through the pathological themes, using sophisticated exploration strategies to make contact with and overcome painful vicissitudes as part of a deep, rich, mature relationship.

References

1. DSM- IV-TR (2000) Diagnostic and statistical manual of mental disorders, 4th edn (text rev). American Psychiatric Association, Washington, DC
2. Blundo C (2004) Neuropsichiatria, i disturbi del comportamento tra neurologia e psichiatria. Masson, Milan, Italy

3. DSM- IV-TR (1995) Diagnostic and statistical manual of mental disorders, 4th edn (international version with ICD-10 codes). American Psychiatric Association, Washington, DC
4. Le Doux J (2002) Synaptic self: how our brains become who we are. Penguin Putnam, New York
5. Lesch KP, Bengel D, Heils A et al (1996) Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. Science 274:1527–1531
6. Kendler KS, Neale MC, Kessler RC et al (1992) Childhood parental loss and phobias in women: the interrelationship of agoraphobia, social phobia, situational phobia, and simple phobia. Arch Gen Psychiatry 49:273–281
7. Gabbard GO (2000) Psychodynamic psychiatry in clinical practice, 3rd edn. American Psychiatric Press, Washington, DC
8. Kagan J, Reznick JS, Snidman N (1988) Biological bases of childhood shyness. Science 240:167–171
9. Busch FN, Cooper AM, Klerman GL et al (1991) Neuropsysiological cognitive-behavioral, and psychoanalytic approaches to panic disorder: toward an integration. Psychoanal Inquiry 11:316–332
10. Milrod BL, Busch FN, Cooper AM et al (1997) Manual of panic-focused psychodynamic psychotherapy. American Psychiatric Press, Washington, DC
11. Manassis K, Bradley S, Goldberg S et al (1994) Attachment in mothers with anxiety disorders and their children. J Am Acad Child Adolesc Psychiatry 33:1106–1113
12. Gainotti G (2005) Emotions, unconscious processes, and the right hemisphere. Neuro-Psychoanalysis 7:71–81
13. Oatley K, Johnson-Laird P (1987) Toward a cognitive theory of emotions. Cogn Emot 1:29–50
14. Leventhal H (1979) A perceptual motor processing model of emotion. In: Pliner P, Blankenstein K, Spiegel IM (eds) Perception of emotion in self and others, vol 5. Plenum Press, New York, pp 1–46
15. Leventhal H (1987) A perceptual motor theory of emotion. In: Berkowitz L (ed) Advances in experimental social psychology, vol 17. Academic Press, New York, pp 117–182
16. Morris JS, Ohman A, Dolan RG (1998) Conscious and unconscious emotional learning in the human amygdala. Nature 393:467–470
17. Le Doux JE, Sakaguchi A, Reis DJ (1984) Subcortical efferent projections of the medial geniculate nucleus mediate emotional responses conditioned to acoustic stimuli. J Neurosi 4:683–698
18. Le Doux JE, Ruggiero DA, Reis DJ (1985) Projections to the subcortical forebrain from anatomically defined regions of the medial geniculate body in the rat. J Comp Neurol 242:182–213
19. Le Doux JE (1986) Sensory systems and emotion. Integr Psychiatry 4:237–248
20. Solms M, Turnbull O (2002) The brain and the inner world: an introduction to the neuroscience of subjective experience. Other Press, New York
21. Panksepp J (1985) Mood changes. In: Vinken P, Bruyn P, Klawans H, Frederiks J (eds) Handbook of clinical neurology, vol 45. Elsevier, Amsterdam, pp 271–285
22. Davis M, Walker DL, Lee Y (1997) Amygdala and bed nucleus of the stria terminalis: differential roles in fear and anxiety measured with the acoustic startle reflex. Philos Trans R Soc London B 352:1675–1687
23. Davis M, Lee Y (1998) Fear and anxiety: possible roles of the amygdala and bed nucleus of the stria terminalis. Cognition and Emotion 12:277–305

24. Sacchetti B, Ambrogi Lorenzini C, Baldi E et al (1999) Auditory thalamus, dorsal hippocampus, basolateral amygdala, and perirhinal cortex role in the consolidation of conditioned freezing to context and to acoustic conditioned stimulus in the rat. J Neurosci 19:9570–9578
25. Attwell PJ, Cooke SF, Yeo CH (2002) Cerebellar function in consolidation of a motor memory. Neuron 34:1011–1020
26. Sacchetti B, Baldi E, Ambrogi Lorenzini C, Bucherelli C (2002) Cerebellar role in fear-conditioning consolidation. Proc Natl Acad Sci USA 99:8406–8411
27. Pally R (2000) The mind–brain relationship. H. Karnac Books, London
28. Hofer MA (1995) Hidden regulators: implications for a new understanding of attachment, separation and loss. In: Goldberg S, Muir R, Kerr J (eds) Attachment theory: social, developmental, and clinical perspectives. Analytic Press, Hillsdale, N.J., pp 203–230
29. Hofer MA (1996) On the nature and consequences of early loss. Psychosom Med 58:570–581
30. Davis M (1992) The role of the amygdala in fear and anxiety. Annu Rev Neurosci 15:353–375
31. Kalin NH, Shelton SE, Lynn DE (1995) Opiate systems in mother and infant primates coordinate intimate contact during reunion. Psychoneuroendocrinology 20:735–742
32. Bowlby J (1969) Attachment and loss, vol I: Attachment. Basic Books, New York
33. Bowlby J (1973) Attachment and loss, vol II: Separation: anxiety and anger. Basic Books, New York
34. Bowlby J (1980) Attachment and loss, vol III: Loss: sadness and depression. Basic Books, New York
35. Siegel DJ (1999) The developing mind. Guilford Press, New York
36. Arrigoni Scortecci M (1988) La tecnica del trattamento degli stati psicotici. In: Semi AA (ed) Trattato di psicoanalisi. Cortina, Milan, Italy, pp 617–674
37. Mitchell SA, Black MJ (1995) Freud and beyond: a history of modern psychoanalytic thought. Basic Books, New York
38. Freud S (1926) Hemmung, Symptom und Angst. Internationaler Psychoanalytischer Verlag, Leipzig–Wien–Zurich
39. Klein M (1948) On the theory of anxiety and guilt. In: Contributions to psychoanalysis 1921–1945. Hogarth Press, London
40. Bion WR (1967) Second thoughts: selected papers on psychoanalysis. Heinemann, London
41. Klein M (1935) A contribution to the psychogenesis of manic-depressive states. In: Contributions to psycho-analysis 1921–1945. Hogarth Press, London
42. Klein M (1940) Mourning and its relation to manic-depressive states. In: Contributions to psycho-analysis 1921–1945. Hogarth Press, London
43. Kernberg O (1984) Severe personality disorders. Yale University Press, New Haven, Conn.
44. Mitchell S (1988) Relational concepts in psychoanalysis: an integration. Harvard University Press, Cambridge, Mass.
45. Balint M (1953) Primary love and psychoanalytic technique. Liveright Publishing, New York
46. Bleger J (1967) Simbiosis y ambeguedad, estudio psicoanalitico. Paidos, Buenos Aires, Argentina
47. Ogden TH (1989) The primitive edge of experience. Jason Aronson, Northvale, NJ
48. Mancia M (2003) Dream actors in the theatre of memory: their role in the psychoa-

nalytic process. Int J Psychoanal 84:945–952
49. Mancia M (2004) Coscienza e inconscio, sogno e memoria: possibili contaminazioni tra neuroscienze e psicoanalisi. Psiche 1:75–90
50. Mancia M (2004) Sentire le parole. Bollati Boringhieri Editore, Turin, Italy
51. Llinas RR, Pare D (1996) The brain as a closed system modulated by the senses. In: Llinas RR, Churchland P (eds) The mind–brain continuum. MIT Press, Cambridge, Mass, pp 1–18
52. Edelman GM, Tononi G (2000) A universe of consciousness: how matter becomes imagination. Basic Books, New York
53. Parker G, Tupling H, Brown LB (1979) A parental bonding instrument. Br J Med Psychol 52:1–10
54. Parker G, Hadzi-Pavlovic D (1992) Parental representations of melancholic and non-melancholic depressives: examining for specificity to depressive type and for evidence of additive effects. Psychol Med 22:657–665
55. Parker G, Hadzi-Pavlovic D, Greenwald S, Weissman M (1995) Low parental care as a risk factor to lifetime depression in a community sample. J Affect Disord 60:47–51
56. Gilbert P, Allan S, Gross K (1996) Parental representations: shame, interpersonal problems and vulnerability to psychopathology. Clin Psychol Psychother 3:23–34
57. Cappelli L, San Martini P (2004) Stile di accudimento genitoriale misurato dal Parental Bonding Instrument in un campione non clinico. Rass Psicol 3:31–57

Chapter 7

The Predicting Brain: Psychoanalysis and Repeating the Past in the Present

Regina Pally

Introduction

“Beauty is in the eye of the beholder” and, according to neuroscience, … so is everything else! In other words, reality is subjective. This is because the brain constructs our experience of events, people, objects, as well as our emotional and behavioral responses to them [1–6]. From a subjective perspective, as events occur, we perceive them and then react to them. From the perspective of the brain, even before events happen, the brain nonconsciously makes a prediction about what is most likely to occur, and starts to construct the perceptions, behaviors, emotions, and physiologic responses that best fit with what is predicted. Predictions evolved as short cuts, to enhance adaptive functioning [4, 7]. Predictive mechanisms prepare us ahead of time, so that we are able to respond more smoothly, efficiently and rapidly once an event does occur. This makes sense from the evolutionary standpoint. In the competition for scare resources, animals “prepared” and able to react more quickly are more likely to survive and pass on their genes to their progeny.

Prediction “seems to be the ultimate and most general of all global functions of the brain” [8]. Predictions are generated continually, automatically, and nonconsciously at every level of brain function, including perception, emotion, behavior, and social relationships [9–15]. To formulate predictions, the brain relies on the individual’s prior experience and generalized knowledge about the world, to anticipate forthcoming events and prepare for them [14, 16–19]. In a sense, we predict the future and then live the future we expect.

Conscious predictions do not alter the outcome. It does not rain because the weatherman predicts it. The stock market does not rise or fall because a stock analyst predicts it will. Nonconscious predictions, by contrast, do alter the outcome. They lead to biases in how we experience the world and all of our social relationships. Since nonconscious predictions are not always correct, an oversight system monitors them to see whether expectations have occurred or have failed to occur. Consciousness may have evolved to enhance the brain's oversight system [20].

Prediction and Psychoanalysis

I am not a neuroscientist. I am a psychiatrist and psychoanalyst interested in how neuroscience can enhance the understanding and treatment of psychological problems [21–24]. Psychoanalysis is a powerful tool in the treatment of long-standing maladaptive thought patterns, belief systems, as well as emotional and behavioral responses within interpersonal situations. Psychoanalysis has benefited from the infusion of ideas from other disciplines, regarding such diverse topics as child development, attachment, inborn personality traits, genetic vulnerabilities, the use of medication, and neuroscience. In this chapter my main aim is to examine the neuroscience paradigm of *prediction* and to demonstrate its relevance to the practice of psychoanalysis.

For healthy cognitive and emotional development, as well as adaptive brain development, it is important for a child to receive love, affection, and emotional responsiveness from its parents [25–29]. It need not be perfect, but some children receive far less than the optimal. In these cases, the way in which a child experiences and reacts to early caretakers can be quite adaptive in dealing with a highly painful situation. One of the central tenets of psychoanalysis is the idea that people often unconsciously repeat in their adult life these same painful relationships. However, what may have been adaptive as a child, when repeated in adult relationships, is often quite maladaptive.

Freud was the first to *systematically* describe the phenomena of unconscious repetition, and connected it to his view of drives and "signal anxiety" [30]. Within psychoanalytic treatment, the repetition of past relationships is studied in the relationship the patient has with the analyst and is called "the transference relationship." Contemporary models of psychoanalysis retain the idea of repetition and transference but have modified Freud's conceptualization. They focus more on the whole nature of the individual's early relationships with caretakers, including the painful emotions, behaviors, conflicts, as well as the defenses and other strategies the child has developed to cope with problematic relationships [31].

Relevant to these contemporary views, neuroscience considers a child's tie to its mother (i.e., attachment) as a biological need like hunger or thirst, which organizes the infant's behavior with the caretaker, to enhance security and survival [27]. Kandel links "signal anxiety" with the neuroscience paradigm of "conditioning," a form of predictive learning, and argues that prediction plays an important role in the child's ability to maintain a safe attachment [32]. Additionally, Kandel points out that repeated experiences, in which the parent responds to the infant's need for closeness and safety, "become encoded in procedural memory as expectations of how to feel secure" [32]. Neuroscience maintains that learning is more deeply encoded the younger a person is and the closer a situation is related to survival. Therefore what a young child learns about how relationships *tend to go* with parents can leave an enduring impact on the child's expectation of how relationships will tend to go with people *in general*.

A few brief clinical vignettes illustrate the kinds of repetition I am referring to. Julian's father was harsh and critical, his mother kind and loving but burdened by the care of his younger handicapped sister. Fearful that if he burdened her further, she would totally collapse, and have even less to give him, he became overly self-sufficient. Additionally, to protect his mother from his father's criticisms of her, Julian took it as his responsibility to keep his mother happy. As an adult, he is unable to express his needs or emotions with women, and feels excessively guilty when women are upset. As a child, Patricia felt rejected and left out of the family. Her parents were too preoccupied with their own lives to have enough time for her. As a child she did not complain, afraid of incurring further rejection by her parents. She feels left out in her adult life, with colleagues and friends, and tends to isolate herself in response. Roger's mother gave him a lot of attention, but was perfectionist and critical. He could never do anything right. To cope, he tuned out feelings of emotional hurt and anger. When his wife disappoints him, he doesn't say anything. He denies feeling hurt or angry, but his behaviors of overeating and giving her the "silent treatment" indicate otherwise. Therapy helps patients like these to consciously make the connection between past and present and to consciously reflect on developing more adaptive ways of relating to people in current life.

The neuroscience of prediction is relevant to psychoanalysis in many ways. It suggests that *repetition* is not simply a psychoanalytic construct, but is fundamental to adaptive brain functioning. Predictive mechanisms help explain how the past continues to have such a powerful influence over present functioning in all realms of human functioning. The prediction paradigm helps to explain how conscious reflection can promote therapeutic change, and why, even with effort and conscious awareness, repetitions are so difficult to change [33]. Finally the neuroscience of prediction suggests that with some patients, analysts may need to become more *active* in the process, drawing the patient's attention to the nature of the repetition and encouraging the patient to "problem-solve," so to speak, in order to develop ways of inhibiting maladaptive repetitive tendencies and to shift to a more adaptive responses. To some, this is controversial, because it dilutes the psychoanalytic process. I would argue the opposite. It is the very process of the psychoanalytic treatment that makes it possible for this kind of maladaptive repetition to be studied in depth, so that adaptive strategies for change can occur. While it is true that for some individuals change occurs quite naturally from the analyst's interpretations and the gaining of insight, for others, the maladaptive patterns are too deeply entrenched. In these cases, along with more traditional analytic methods, a more active stance, which encourages focused conscious attention and effortful attempts to develop and try out different response patterns, is necessary.

A corollary aim of this chapter is to counteract some of the *over*emphasis, within the fields of medicine and psychiatry, on genetic explanations and psycho-pharmacologic treatments for psychological difficulties. Fortunately, within neuroscience a growing body of research evidence indicates that BOTH genes and environment, including the environment of early relationships play important roles in psychological functioning [34–39], The prediction paradigm adds additional mechanisms to the nature–nurture interface, and helps explain why *psychoanalytic/interpersonal* forms of treatment may be essential for treating certain kinds of psychological problems.

Why Predictions are so Heavily Weighted Toward the Past

During a person's interactions with the world, the brain learns "what tends to go with what." Certain sensory events tend to go together (e.g., a particular movement of the lips is associated with a particular spoken word, and a cup usually has a rim and a handle). Certain events tend to follow one another (e.g., when brushing your teeth, take off the cap, squeeze the tube, put paste on the brush, brush and rinse). Certain consequences tend to follow from one's behavior (e.g., kick a ball, it moves; cry, mother comes to comfort you). This learning is stored in memory, to be used in the future, when similar experiences are encountered. When a person encounters something in their *current* life, the brain relies on this memory of *past* learning to more quickly figure out what is happening and how to respond to it. From a current stimulus, the brain "predicts" what other stimuli are *most likely* also present. From a current event, the brain predicts what event is most likely to follow. From a current motor action, the brain predicts the most likely consequence of that action. Prediction has such an impact because at a nonconscious level, predictions *set in motion* the perceptual processes and responses to what is predicted. Therefore, we are biased to hear what we expect to hear, see what we expect to see, in the physical world, as well as the interpersonal world.

The system is designed such that when a particular result seems plausible for the situation, the person may not even notice a prediction error. As a result, an incorrectly predicted world may nevertheless be experienced as the actual world. This helps explain why people who have come to expect to be regularly misunderstood, criticized, disappointed, abused, or abandoned, seem to continually experience this in relationships with the people in their current life. In the case of Julian, he intellectually recognizes that his therapist has always treated him with the utmost care and kindness. Nevertheless he experiences her as someone not to be trusted with his inner most vulnerable feelings and he remains suspicious and on guard. Making things even worse is the fact that others do indeed become angry and impatient when he stubbornly holds onto this kind of experience, despite the other's well-intentioned efforts. Gabbard (personal communication) suggests this might be one of the underlying mechanisms of *projective identification.*

Relying so heavily on past experience for determining what is happening now, or what will happen next, and formulating a response, may seem like an odd way to organize a brain. However most lives are more-or-less the same from day to day and people repeat familiar patterns over and over again. Every day when we wake up, we still have the same name, and the same appearance. We still live at the same address, speak the same language, and go to the same office, where people continue to treat us the same way as they did yesterday. Rather than figuring out anew every day who we are and what is happening to us, relying on prediction infused with past learning to figure things out is efficient, and reduces the overall burden on the brain. The brain uses nonconscious predictive processes for handling situations that have become routine and familiar. In this way there is more "brainpower" available for dealing with situations that are very complicated, or novel and unfamiliar.

Prediction and the Integrated Brain

The brain is both modular and integrated [14]. Various modules include: visual perception, auditory perception, motor behavior, memory, emotion, and social functioning. These exist in hierarchical fashion. Modular processes become integrated as information "moves up" the hierarchy. The pinnacle of the hierarchy is the prefrontal cortex, where lower activity becomes maximally integrated. The prefrontal cortex makes possible the integration of modular processes *over time*, which is essential for voluntary behavioral coordination, voluntary attention control, and voluntary decision-making, as well as all uniquely human activities, such as language, culture, and abstract thinking.

The "perception action cycle" illustrates how predictive mechanisms function to integrate the brain's modular activity over time [14]. An example is looking at a cup of coffee. When seeing the cup of coffee, the visual sensory system sends predictive signals to motor areas to prepare the behavior most likely to be used with the cup (e.g., lifting it up). As the decision to lift is initiated, predictive signals are sent to somatosensory areas to activate the most likely sensations that will occur as a result of lifting (e.g., a certain arm position, certain weight of the cup, aroma of the coffee). In cyclic fashion, as these sensations are perceived, they send predictive signals to initiate the type of action most likely to be performed next (e.g., drinking from the cup).

In the following sections, predictions are discussed as they operate in relation to the basic processes of perception, behavior, and emotion, and to "higher" processes such as interpersonal interactions involving empathy, intuition, understanding the minds of others, and psychotherapeutic treatment.

Perception and Prediction

Although it *seems* to us as if we simply "take in" what is "out there," the brain actually *constructs* our perceptions. Even the words on this page are a construction of the brain. All that the brain receives are "bits and pieces" of individual sensory cues (e.g., lines, angles, color frequencies, sound frequencies, textures, shapes, directions of movement) through the external sense organs. Individual external cues eventually become integrated with each other and with sensory cues from the internal body, as well as with emotional factors and memory as information *moves up* the processing hierarchy. The brain compares "patterns of incoming data" with "patterns already stored in memory." Sensation becomes *perception* when a match occurs between what is coming in and what is already stored in memory.

Prediction leads to perceptual biases that are relevant to the psychoanalytic concept of repetition. The brain relies on memory as a source of prediction of what is most likely to be happening now, in the present. For efficiency's sake, the brain "searches" in memory only for a *good enough* match. In this way a person can experience as identical two events which may be only similar. Additionally, memory stores send signals *back down* to primary sensory cortex to increase activity for the kinds of sensory inputs already in memory [17]. This enhancement of primary sensory cortex, by memory stores, increases the likelihood that a person's past experience will shape how they experience something in the present, and may contribute to *repetition* phenomena.

Research on *imagery* supports this view of perceptual construction. A subject in an fMRI[1] scanner is asked to form a visual image of an object. The same primary visual area "lights up" as when the subject *actually sees* the object. Similarly, when the subject is asked to rotate the imagined object in space, it takes as long to rotate it *in imagery* as it does to rotate the *actual* object. Researchers theorize that when a subject is asked to imagine an object, information stored in memory sends signals that activate the primary sensory cortex. This is also why even imagined events can seem so vivid.

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[1] Many experiments are done with subjects in an fMRI neuroimaging scanner, which directly measures changes in blood flow but which is an indirect measure of changes in glucose metabolism, and reflects changing levels of neural activity. The fMRI scanner can thus 'detect' what part of the brain is functionally active during a particular cognitive task (e.g., perception, memory, behavior, speaking, thinking).

Predictions enhance the speed and efficiency of perception. Subjects more quickly perceive an object they expect, and are slower to perceive an object they do not expect. Subjects in an fMRI scanner are told that the sound of a tone will be followed by the appearance of a particular object. As soon as the subject hears the tone, increased activity appears in primary visual cortex in *anticipation*, before the appearance of the object. Predictive activity *from past learning stored in memory* enables the brain to take in just a few sensory details and "predict the rest." If an object, such as a cup, is partly occluded by another object, such as a milk carton, we have no trouble in concluding that a whole cup is there. In fact, the whole cup is represented in neural activity in the brain, as the predictive signals actually "fill in" for the missing visual inputs.

An optical illusion, the Kaniza effect, which demonstrates "illusory shapes," occurs as a result of predictions, in a way similar to imagery or "fill-in" phenomena. In Fig. 1, there are three real "pac-man" shapes, three real black-lined angles, and one *illusory* shape, a "white triangle." The illu-

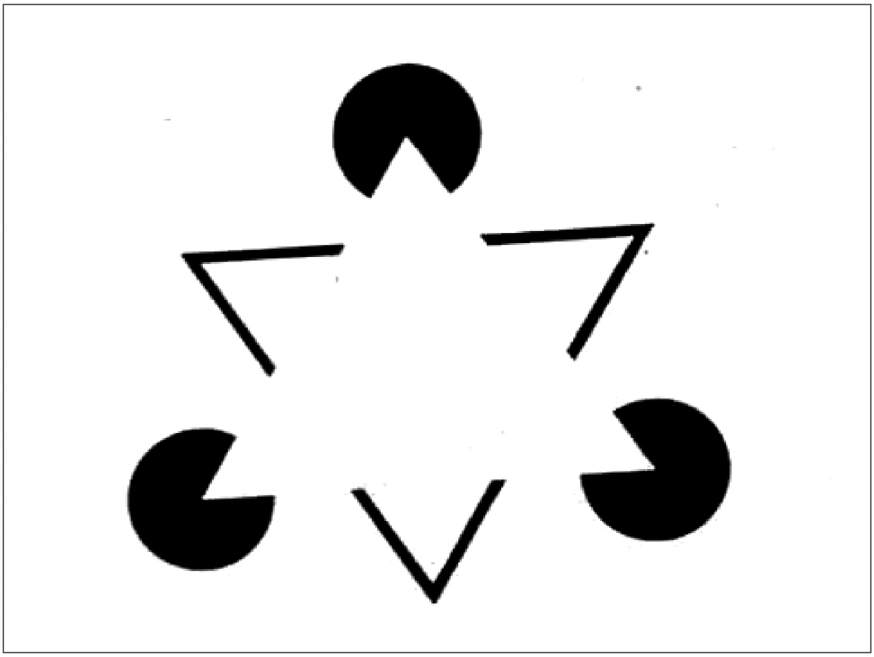

Fig. 1. The Kaniza effect: Does the white triangle really exist? Answer: It exists in the brain, not in the world. Neuroimaging studies indicate that actual activity in visual cortex represents the illusory figure

sory figure occurs as a result of the brain's predictive mechanisms. The angles and the pac man shapes are actual sensory inputs. The brain predicts that for this configuration to occur, "most likely" a white triangle overlays them. As a result of the prediction, neuroimaging reveals enhanced activity in primary visual cortex, corresponding to the perception of the white triangle. The white triangle does not exist out there in the world, but it does exist in the brain.

Our perceptual experience of our own body movement is more closely tied to predictive mechanisms than to actual action. In one set of experiments, subjects are asked to trace either a circle or an oval on a computer screen, using the cursor [40]. The cursor is controlled by a handle underneath the table, out of sight of the subject. Initially, when the subject traces a circle on the screen, the handle guiding the cursor also moves in a circular path; when tracing the oval, the handle moves in an oval path. Subjects accurately report the correct path of their hand as it moves the handle. After a few such repetitions, and unbeknownst to the subject, the "gain" on the handle is switched. Now when the subject traces a circle, the handle actually moves in an oval path. However, this time, the subject reports that their hand moved in a circular path (i.e., the same as the circle on the screen, but not the actual path the handle traverses).

Pain is a type of perception processed by the "somatosensory" cortex, a brain area responsible for sensations from the body viscera, including the skin, muscles, joints, and all internal organs. When a subject actually experiences a painful stimulus (e.g., a mild shock on the arm), certain brain areas are active on fMRI. These include the somatosensory area, as well as the anterior cingulate cortex and the thalamus. When subjects are told to *expect* this same sensation, the same brain regions "light up" in anticipation of the pain, *before* any stimulus is given. Similarly, the placebo effect in pain relief treatment produces analgesia by altering expectations [41]. While being scanned, subjects are told that a cream, which is rubbed onto their arm, is a powerful analgesic and that they will not experience any pain from the shock. On fMRI, the "pain" regions do not show activity. Instead, activity is increased in the prefrontal cortex. The *placebo effect* occurs because the prefrontal cortex anticipates *no pain.* The increased activity inhibits the activity in the anterior cingulate and somatosensory area, such that the person no longer perceives the pain when the shock is delivered.

Predictions, Learning, Emotion, and Behavior

All mammals need to learn how to provide for their biologic *survival* needs, including hunger, thirst, mating, avoidance of danger, and a safe connection to mother [27, 42, 43]. All primates also need to learn how to obtain affection and maintain an emotionally responsive tie with the mother [29]. The brain has a number of different systems for such learning, all of which involve a link between what is learned and the emotions, behaviors, and even body physiology which may be associated with that learning. At the most basic level, the individual has to learn what brings benefit (reward) and how to obtain it; what brings harm (danger) and how to avoid it. A danger, such as a predator, is associated with the emotion of fear, a "flight" response, and the body physiology to support the behavior, such as increased heart rate, blood pressure, and muscle tone. A reward, such as food, involves the positive sense of pleasure, the behavior of eating, and the body physiology of increased salivation and gut motility for digesting the food.

The brain comes equipped with a certain number of innate or inborn responses to natural dangers and rewards that promote survival, such as predators and food. In a process known as *classical conditioned learning*, or *Pavlovian learning*, an association is made between a natural danger or reward with "survival value" and an otherwise neutral stimulus, which in itself does not have survival value. All mammals share the same brain circuitry for conditioning, so animals serve as good models for how conditioning operates in humans. In a typical conditioned learning paradigm, an auditory tone is played just before the animal is given a shock. Once conditioned learning occurs, the previously neutral stimulus (i.e., the tone) takes on survival value. On the very next occasion, the animal will jump with fear when the tone is played, even if no shock is given. The tone stimulus is now said to predict the appearance of the shock. This prediction sets in motion the *emotion* and *behavior* toward the previously neutral stimulus that the animal previously used in relation to the shock. A similar mechanism occurs with rewards. A blue light flashes just before a sip of juice reward is given. After several repetitions, the light predicts food and the animal will salivate and run to the place in the cage where it receives the juice. Any behavior used to avoid the danger, or obtain the reward, becomes reinforced.

Babies, right from birth, show evidence of conditioned learning [44]. A baby just a few days old recognizes the smell of its mother's milk. By 6 weeks of age, a baby's innate response of sucking and turning its head as soon as it smells its mother's milk is evident. In a conditioned learning paradigm, the mother wears a certain perfume while she is caring for the child. Within a matter of days, the baby exhibits a *conditioned response* to the perfume. The baby sucks and turns its head, when it smells its mother's perfume, even if it is presented on a cotton pad, in the absence of nursing or care-giving.

Conditioned learning involves a number of subcortical structures, such as the amygdala and nucleus accumbens, and can operate without conscious awareness. The more frequently a conditioning situation occurs, the more closely associated to survival the need is, the earlier in an offspring's life the conditioning occurs, and the more irregular the reinforcement schedule, the more powerful and enduring the conditioned response will be and the more difficult to unlearn. Unlearning a previously learned conditioned response requires consciousness and the *learning* of a new type of response and the growth of new brain connections in the prefrontal cortex, which serve to inhibit the original, conditioned learning stored in subcortical sites [45]. The old response is only inhibited. It does not disappear. Under stress it can re-emerge [46].

Typically, cortical and subcortical regions work together. The relative importance of cortical versus subcortical involvement depends on the situation. Very familiar situations are often handled without conscious attention, while new ones tend to require conscious attention. Voluntary choice favors cortical control, whereas when an automatic speedy response is indicated, subcortical control is favored. For this reason a person begins to run out of the way of a fast approaching car, *before* they are consciously aware of the car's approach and well before they determine whether it is a Porsche or a Ferrari.

Nonconscious processing is less precise than conscious processing and therefore conditioned responses are more likely to generalize to other contexts. For example, Martha, who was raped in the parking lot after work by a man with a beard, has no conscious recollection of the beard, having focused all conscious attention on the knife he held in her back. For months, not only does she avoid parking lots, as would be expected, but she is fearful of men with beards. Only when she reads the police report describing his appearance does she understand her fear.

Traumatic childhood events, such as neglect or physical or sexual abuse, can be particularly powerful sources of conditioning. The brains of children who have been abused show increased activity in the amygdala, and seem to constantly "predict" danger, in the form of heightened vigilance and alertness to danger, increased startle reaction even to benign events, and a greater tendency to interpret the actions and facial expressions of others as dangerous. These predictive mechanisms may result in excessive avoidance of intimacy, leading the person to feel as emotionally deprived as they did in childhood. The heightened vigilant and suspicious nature of these already traumatized individuals can be abrasive and difficult for others to deal with. This may lead to more conflict, negative affect, and rejection in social situations, again further reinforcing the person's lack of trust in personal relationships. Even *without* overt trauma, high degrees of infant distress can occur with failures of emotional responsiveness [25]. A child with, for example, a hostile or withdrawn parent may learn through conditioning to use compliance or cheerfulness to avoid the misattuned parental response.

A patient may be completely unaware that conditioning has occurred, because conditioning can operate under subcortical control and entirely outside awareness. This explains why a child who suppresses conscious feelings of fear associated with abuse may nevertheless exhibit *conditioned fear* responses to otherwise neutral events associated with the abuse. For example, when she was a young child, Rosalie was abused by the housekeeper. After the death of her mother, Rosalie developed a phobia about the color yellow, which in treatment was "discovered" to be the color of the housekeeper's bedspread. Responsiveness failures can be so frequent that conditioned responses to them operate outside of conscious awareness. The fact that a patient is unable to explain their reasons for being afraid of something, or behaving in a certain way, may not be the result of unconscious psychological defenses or "resistance", as is typically considered. Rather it is due to the *normal* way that the brain processes conditioned responses. Even with insight into these reactions, the tenacity of early conditioning makes it very difficult to change.

Much of human functioning goes beyond immediate survival needs, such as the learning of moral guidelines, and social appropriateness, or skills such as reading or keyboarding. In these situations there is no innate response. In a process known as *operant conditioning*, the child learns through trial and error to develop an appropriate response. A child who wants to play while mother is cooking dinner will try different approaches to get mother to respond. The one that succeeds will be reinforced. Since each mother is different, each child's learning will be different. Some children will learn to be affectionate. Others learn to whine or bother their siblings. As this learning occurs, the brain builds up a record, and eventually voluntarily and selectively applies this learning to other entirely new situations, never previously encountered. Learning not to bother mommy as a 3 year old, helps the child know to not bother the teacher, even on the first day of preschool.

Much of our life involves making adaptive decisions on how to respond, by evaluating the costs and benefits of the available options, a process that has been termed "neuro-economics" [47]. With respect to meeting the human needs for love, attention, approval, and understanding, either through *classical* or *operant* conditioning, the brain's endorphin and dopamine systems are both involved [27, 29]. The brain's dopamine circuit serves as the individual's history, of the relative reward value of any particular response, and promotes the motivation or wanting of a reward. Choosing what behavior is most adaptive relies heavily on the dopamine system to predict the probability and cost of a particular response leading to a desired reward. Endorphin is released when the reward is obtained and underlies the *liking* of a reward. Although initially there are some conscious and voluntary aspects to this type of behavioral learning, as discussed in more detail below, in repeated situations, such learning shifts to an automatic, nonconscious system of control. For human children, affection and attention (positive or negative) serve as "social rewards" and activate dopamine and endorphin. Therefore a child can become deeply attached to, seek out, and love even those people who mistreat them, if they also provide some type of attention or affection. The powerful effect of the dopamine and endorphin systems can make these maladaptive social response styles quite enduring.

What neurobiology adds to psychoanalytic theory is that events and their emotional significance may not be available to memory and consciousness, not because of neurotic defenses, such as repression or disavowal, but because of the immaturity of the child's brain at the time of the event. Additionally, the enduring nature of these responses may reflect not only powerful psychological defenses, but also the powerful neural circuits that are formed in the small and dependent child's brain, which are more resistant to change, despite years of excellent psychotherapeutic work. When such a patient is perpetually anxious in intimate situations or perpetually finds him or herself drawn to people who emotionally deprive them, I find it useful to explain some of the neurobiology of learning in the context of parent–child relationships. This can help to reduce some of the bad feelings a patient has about themself, since they often feel childish, stupid, and ashamed of the tenacity of their reactions and their inability to change.

Prediction and Habitual or Routine Behavior

Prefrontal cortex and basal ganglia circuits typically work in tandem in relation to behavior [9, 48]. Prefrontal cortex is generally involved in consciously acquiring *new* motor skills. When a new behavior is repeated over and over, it becomes "automatic" or habitual, and activity shifts to subcortical structures such as the cerebellum and basal ganglia, where it is stored as *procedural memory* of the behavior [9, 49–52]. Most of the time, we are not aware of the nature of the knowledge and do not even remember when or how we have learned the skill. Procedural memory is what we use when we learn how to play a musical instrument, how to dance, how to play a sport, or how to speak native language. Grigsby [53], who studies personality, and Beebe [25], who studies mother–infant interaction, suggest that this memory is also the basis for learning social *interaction* patterns. Basal ganglia can activate these kinds of behaviors nonconsciously, leaving the prefrontal cortex and consciousness system free to process new information.

When however, the *old* behavior needs to be modified, consciousness and prefrontal cortex are again engaged, both to learn the new behavioral response and to signal the basal ganglia to inhibit the old habitual motor program. The shift to the new behavior takes time and is not smooth, as the old habit keeps intruding into the newly learned behavior [54]. Once a new behavior has been repeated often enough, it too becomes habitual, and once again activity shifts to basal ganglia control. In learning a new tennis serve, conscious effort is necessary for the player to change *how* the ball is thrown and *how* the ball is hit with the racket. Nevertheless, the old serve keeps intruding. However, with practice, eventually the new behavior shifts into being activated automatically and unconsciously. When learning to drive, consciousness is required for each and every detail of driving. But once a driver is familiar with driving, it can be quite automatic. If the traffic is moving along in the "expected" or "predictable" fashion, a driver can be deep in thought about plans for dinner and *not be conscious* of driving, which is why the driver may miss their exit. When a car unexpectedly swerves ahead, the driver *immediately* stops thinking of dinner and shifts into consciousness of driving.

Most behaviors are not *single* actions, but comprise sequential steps of behavior or "routines" aimed at achieving a goal (e.g., to drink a cup of coffee, you reach out your hand, clasp the handle, lift the cup up towards your mouth, lean your head back, draw the liquid into your mouth, and swallow). Nonconscious predictions operate to select and activate the best-adapted behavioral routine for the situation, and also to link the individual steps within the routine. This is why people operate on *automatic pilot* more than is generally realized.

In certain situations consciousness enhances behavioral effectiveness, such as when I trim my rose bushes and want to avoid getting stuck by a thorn. But many behaviors operate better and more smoothly *without* conscious awareness, such as when a pianist memorizes a piece and plays it "by heart." Even though a Beethoven piano sonata is long and complicated, a skilled pianist plays it fluently and effortlessly, without consciously paying attention to each finger movement. Predictive mechanisms make for *smoother* behavioral sequences by linking steps together in the basal ganglia [9]. The novice must consciously think of each step. But with practice, a preceding movement serves as a *prediction* for the next step. Eventually, in a process known as "chunking," greater *automaticity* and *smoothness* are achieved. A series of steps is treated as a single unit or chunk, and one chunk automatically activates the next chunk.

Mothers and babies engage in repeated patterns of interaction involving the physical care of the baby as well as nonverbal emotional and communicative exchanges. Repeated interactions with caretakers can be stored in the basal ganglia as the procedural memory of these exchanges [28, 29]. In interacting with another person, a person's brain predicts how the interaction will tend to go. Processes such as emotion and behavior become organized around these expectations. The basal ganglia can activate the repeated patterns of interaction automatically and nonconsciously, as the habitual or routine ways in which we tend to relate to others [25, 33]. Grigsby [53] argues that certain kinds of personality traits ought to be considered as the procedural memory of early interactions with parents and other important figures. For example, Nora grew up in a family in which she felt she had to be compliant in order to keep her parents' love and attention. As she matured, automatic compliance became a part of her personality structure, despite the fact that other people are quite willing to love her, even if she disagrees or argues. These habitual ways of relating can be quite deeply engrained. Psychotherapy is aimed at modifying these patterns with the use of conscious reflection and effort, which at the neurobiological level can inhibit and override basal ganglia automatic responses.

Prediction, Empathy and Understanding the Mind of Others

As human beings we are automatically able to “read the mind” of another person, even without their telling us. This capacity is called “theory of mind” [55, 56]. Using current nonverbal gestures, context, and past experience, the brain makes a prediction as to the other person’s feelings, motivations, beliefs, and intentions [57]. This allows us to better anticipate how *they* will interact with *us* and be able to make a plan for how we will react to them [56, 58]. We readily determine from someone’s facial expression and posture whether they are happy, or angry, and whether they intend to lift their hands to greet us or to hit us, as we plan what our appropriate response should be. During conversation, as we listen, we plan our reply before the other person has even finished speaking.

Theory of mind has a developmental trajectory. A baby as young as 9 months old will point to an object when it *intends* for mother to look at it, and will look where mother points, because the baby recognizes what its mother intends [59]. When very young children “read another’s mind” it is very egocentric [55, 56]. If a child wants to go out for a walk, when you put on your tennis shoes, the child will assume *you* want to go out for a walk. Not until the child is about 5–6 years old do they recognize that others may have mental agendas that differ from their own. All forms of psychoanalytic therapy rest on the maturation of this capacity. Some individuals completely lack theory of mind ability, most notably people who suffer from autism or Asperger’s syndrome. It is proposed that patients with borderline personality disorder have impairments of theory of mind, which underlie their difficulty in affect regulation and in interpersonal relationships [60].

One brain system that contributes to theory of mind ability involves “action-observation” and is referred to as the *mirror neuron system* [61]. When we observe another individual performing a purposeful or *goal-directed* action, we immediately “know” their intention[2]. If an apple is sitting on the table, and a person reaches out to pick it up, an observer auto-

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[2] Some behavior is random, but most of it is considered goal-directed, i.e., purposeful or intentional.

matically assumes they intend to eat the apple. Neuroimaging studies indicate that while *observing another individual perform a goal-directed action*, mirror neurons in the *premotor* area of the observer's brain[3] become active, in the same way they would if the observer were performing that action. The premotor neurons in the observer's brain are linked to regions that generate the intentions behind goal-directed behaviors. It is presumed that we know what someone else intends, because the brain *predicts* that they have the same intention we would have if we were performing that same action [62].

Empathy, or the ability to feel the feelings of others, also relies on the mirror neuron system and predictions [63]. When we observe the nonverbal behaviors associated with human emotion, such as a person's facial expression, posture, or head position, mirror neurons in one's own brain become active, in the same way they would if we were performing those same nonverbal emotional behaviors. We know what *they* feel, because our brain predicts that *they* feel the way we would feel if we were moving our face and body in the same way. We essentially read the minds of others by predicting that others are like ourselves [59].

Consciousness: Monitoring and Correcting Prediction Errors

Predictive mechanisms provide speed and efficiency, but leave room for error, because predictions are not always correct. A person who grew up being criticized all the time, will tend to "hear" criticism in the comments of others, even when no criticism occurs. In order to function adaptively in social relationships, they will need a way of correcting this misperception. Fortunately the brain has systems for monitoring and correcting prediction errors.

A number of experiments illustrate monitoring and correction of prediction errors. In one experiment, subjects are given goggles to wear, with

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[3] The premotor area is part of the prefrontal cortex and is considered the region in which motor actions are prepared. The primary motor area, also in the prefrontal cortex, gives the signal to muscles to perform that particular action.

inverted prism lens, which make the world appear upside down [6]. Since light seems to be coming from the opposite direction, the brain predicts the scene has been inverted and constructs the perception accordingly. Over a period of time other inputs come into the brain (e.g., proprioceptive inputs from limb and joint position, tactile sensations from the soles of the feet), which indicate the world has not turned upside down. This causes the perception to “flip.” Subjects again “see” the world right-side-up, despite the fact that they are still wearing the inverted prisms.

Another experiment uses the Stroop test while subjects are in an fMRI scanner [20]. In a typical Stroop test, color words (e.g., red, green, blue) are written either in their own color ink or in another color ink. Green for example, may be written in green ink, or it might be written in either blue or red ink. In the test, these words appear on a computer screen and subjects are asked to either name the word itself, or the color ink the word is written in. As soon as a person sees a word, the brain predicts the most likely thing would be to read the word and prepares this response. If the command instead is “name the ink color,” the pre-planned predicted action must be inhibited, and the correct action initiated instead. Because of the automatic and nonconscious nature of these predictive mechanisms and the biases they cause, many errors in response occur. A subject’s correct response correlates with the scan showing activity in their anterior cingulate cortex (ACC). An incorrect response, which is either noticed by the subject or pointed out by the experimenter, also correlates with ACC activity. When the person makes an error, however, but does not notice it and does not have it pointed out by the experimenter, this correlates with a *lack* of activity in the ACC during that trial. Neuroscientists conclude that the ACC and prefrontal cortex work in tandem. The ACC helps detect prediction errors and then signals the prefrontal cortex to inhibit the inappropriate response and to select the appropriate response instead.

The reason that conscious awareness enhances monitoring and correction is that—unlike nonconscious processing, which is automatic, involuntary, and rather rough and approximate—the consciousness system involves choice, voluntary control, and can make more fine-grained perceptual distinctions and more finely tuned motor responses. The consciousness system, however, is rather limited and easily overwhelmed. We can be consciously aware of only one, or at most very few, items at a time. The nonconscious system, by contrast, can process a large number of items simultaneously. Therefore, the brain is designed to function without the intervention of consciousness most of the time [20]. As long as things go as predicted, nonconscious processes suffice. We function by rote in habitual ways, when brushing our teeth, driving, or jumping out of the way to avoid being hit by a car, as well as behaving compliantly, defensively, or suspiciously in social situations. When something new or unexpected happens, some degree of conscious awareness and effort is required for inhibiting the automatic predicted response and selecting another, more appropriate, response [20, 64].

Implications for Psychoanalysis: Concluding Remarks

Psychoanalysis emphasizes that painful and conflicted aspects of childhood can be unconsciously repeated in current relationships, in ways that are maladaptive for current functioning. Psychoanalytic treatment involves examining this unconscious repetition within the transference relationship. Psychoanalysts are often criticized for being too preoccupied with a patient's childhood. These skeptics have difficulty in accepting that an emotionally distressing problem with a parent that happened "so long ago," in childhood, can still affect the person as an adult. Yet by contrast these same skeptics often have no difficulty accepting the many *everyday* ways in which childhood relationships with parents can lead to permanent changes in the way a person lives their life. Parents teach children how to tie their shoes, brush their teeth, cultural values, morals, and social rules such as waiting one's turn to speak, or saying please and thank you. Such teaching often lasts a person's lifetime.

The neuroscience of prediction indicates that, in a similar way, what is learned with parents *emotionally*, in terms of being rejected, disappointed, shamed, or being made to feel inadequate, can also influence the individual for a lifetime. As a result of how the brain is organized, we predict from our experiences in early childhood (with important care-givers) what to expect from people in general. This biases us to experience current relationships as similar to past ones. It is not that current relationships are in fact identical to past ones, but that we subjectively experience them in this way.

The link between past and present is often difficult to recognize. For one thing, the brain disconnects repeated learning, from the specifics of person, place and time in which it was learned. I ride a bicycle, but have no memory recall of who taught me. Secondly, as a coping device, a child often disconnects conscious awareness from the painful emotions they experienced when little and vulnerable. For example, Jane reports that her mother frequently threatened to "leave" when she and her two siblings acted up. Her father loved her but berated and devalued her mother. Jane denies having any emotional feelings about either of these. At the same time, however, she despises her dependency needs, avoids commitment with men, and goes into "attack mode" at any indication that a man is critical of her. In the course of treatment as she emotionally connects to the childhood emotions, she is better able to realize how they in fact, do affect her in her current relationships. In the course of treatment Jane is finally able to marry a loving man and have children. However, she remains *susceptible* to her defense of not feeling painful emotions. She still has the tendency to go on "attack" with hostile criticism of her husband, rather than reveal feelings of hurt or vulnerability. In the treatment I continually point out the difference between what she expects and how her husband actually feels and treats her (He adores her, saying "she is my dream woman.") I help her to feel less ashamed about vulnerability, and to express hurt feelings without resorting to maladaptive "attack" defenses.

In the case of Julian, despite the fact that he recognizes that his therapist, over 2 years of treatment, has always treated him with the utmost care and kindness, remains suspicious and on guard. He remains reluctant to share his innermost vulnerable feelings, for fear of how the therapist will react. Martha, who was raped in the parking lot after work by a man with a beard, has no conscious recollection of the beard, having focused all conscious attention on the knife he held in her back. For months, not only does she avoid parking lots, as would be expected, but she is fearful of men with beards, even her kindly husband. Only when she reads the police report describing her attacker's appearance does she understand her fear. Nora grew up in a family in which she felt she had to be compliant in order to keep her parents' love and attention. As she matured, automatic compliance became a part of her personality structure, despite the fact that other people are quite willing to love her, even if she disagrees or argues. Patricia, whose parents were too preoccupied with their own lives to pay much attention to her, grew up expecting people to neglect her and not want to be with her. Even now, as an adult, when she goes out with friends, she experiences that people leave her out of the conversation, avoid her, or seem impatient to get away. It is not that her friends do this in fact, but this is her subjective perception of what is happening and she reacts accordingly. In this case, she always tries to please others, conforms to opinions, doesn't join in to the conversation much, and tends to leave social events early, to avoid the feeling of them leaving her. Unfortunately not only do her perceptions confirm her predicted expectations, but her own automatic behavior of isolating herself serves to reinforce her assumptions of being left out and unwanted by others. A type of *projective identification* can occur, when others do become angry and impatient, as people like Patricia stubbornly hold onto their *old* and *threatened* experience, despite the other's current well-intentioned efforts.

In the treatment situation, whether it is psychoanalysis or psychoanalytically oriented psychotherapy, initially it is the analyst or therapist who draws the patient's attention to how the patient *tends to* feel and behave with people, and inquires as to any automatic assumptions and beliefs which may underlie these. Eventually, particularly in psychoanalysis, greater emphasis is given to examining the nature and quality of the transference relationship. In this way maladaptive patterns of relating can be revealed and evaluated in terms of whether they may be derived from similar patterns developed during early relationships. For the patient to change, they must be aware that the therapist does not currently react in the expected way. For example, the therapist is interested in the patient's stories and feelings, not disinterested as expected. The therapist is supportive and nonjudgmental of the patient, not critical or humiliating as expected. For example with Roger, after many sessions in which he describes his interactions with his wife, his children and his colleagues, I point out that there seems to be no difference for him between what he anticipates regarding how a person feels toward him, and what he believes they actually feel. This helps Roger to notice the ways in which people feel differently from what he expects.

Recognizing the difference between past and present, while providing insight, is often only the first step. It is generally not sufficient for change to occur. The patient must also be able to inhibit the old response and shift to a new one. The patient becomes consciously aware of their "maladaptive tendency" and then must voluntarily choose to respond differently. For many people this second step takes a lot of conscious effort and time. For example Ella's mother tended to belittle her and compete with her for who was smarter and more talented. Similarly Ella tends to experience people, including me, as either criticizing her, devaluing her, or jealously trying to be "one up" on her. In response she is constantly on the defensive. Within the treatment it requires that she make a lot of effort to notice that I am quite sensitive and concerned about her feelings, and that I admire her a great deal. It took quite a while for her to modify her defensive behavior. However even after a great deal of change occurred, she still sometimes would first act defensively and then afterwards say, "I know you were being supportive, but sometimes I just can't stop myself from sniping at you." Often she complains, "It takes too much work! Sometimes it is just easier to either withdraw or get angry."

There are many cases in which patients feel ashamed that "events that happened so long ago" can still affect them. For this reason I often explain to the patient that to a large extent, repeating the past is embedded in the very nature of how the brain operates. This can also help patients deal better with the idea that a full "cure" may not be possible. The old tendencies may still continue to emerge, because that is how the brain tends to work. The patient may still have the old feelings of hurt or humiliation, the old tendencies to want to shut down or strike back, like Ella. But much can still be done to counteract these tendencies, and help the patient function more adaptively. The patient can use the "consciousness" system, to pay attention, and make voluntary efforts to regulate emotion, inhibit maladaptive behaviors, and select more adaptive ones. The therapist who recognizes the biology of the brain will be in a better position to empathize with the lifelong challenge the patient must face in this struggle, and will be less likely to feel discouraged themselves as to their therapeutic effectiveness [65].

There are so many ways that patients suffer. They don't cope well with life. They don't like themselves. They have feelings such as emptiness, despair, shame, and guilt. They are self-defeating in their jobs and their relationships. The neuroscience of prediction adds to current psychoanalytic theory and technique. Perhaps most controversial is the implication that the analyst should be more active in helping the patient to focus their attention on the nature of the problematic repetitive pattern and to encourage them to "problem-solve," so to speak, in order to develop ways of consciously inhibiting old patterns and voluntarily initiating new more adaptive ones. By understanding the biology of predictive brain mechanisms, analysts may be less "resistant" to the use of active approaches to treating long-standing maladaptive interpersonal response styles.

The neuroscience of prediction suggests changes for the society at large as well. The lifelong consequences of prediction indicate just why it is so important to provide services to help children and families. The tenacious nature of maladaptive repetitions in social relationships, and the role that interpersonal forms of treatment specifically can play, may encourage greater emphasis on long-term, non-pharmacologic forms of treatment.

References

1. Clark A (1997) Being there. MIT Press, Cambridge, Mass.
2. Crick F (1994) The astonishing hypothesis: the scientific search for the soul. Macmillan, New York
3. Edelman GM (1992) Bright air, brilliant fire. Basic Books, New York
4. Edelman GM (1989) The remembered present. Basic Books, New York
5. Edelman GM, Tononi G (2000) A universe of consciousness: how matter becomes imagination. Basic Books, New York
6. Llinas R, Pare D (1996) The brain as a closed system modulated by the senses. In: Llinas R, Churchland PS (eds) The mind–brain continuum. MIT Press, Cambridge, Mass, pp 1–18
7. Freeman WJ (2000) How brains make up their minds. Columbia University Press, New York
8. Llinas R 1988) 'Mindness' as a function of state of the brain. In: Blakemore C, Greenfield S (eds) Mindwaves. Blackwell, Oxford, pp 339–358
9. Graybiel A (1998) The basal ganglia and chunking of action repertoires. Neurobiol Learn Mem 70:119–136
10. Llinas R (2001) I of the vortex. MIT Press, Cambridge, Mass
11. Miller GF (2000) The mating mind. Doubleday, New York
12. Engel AK, Fries P, Singer W (2001) Dynamic predictions: oscillations and synchrony in top-down processing. Nat Rev Neurosci 2:704–716
13. Freeman WJ (2003) Neurodynamic models of brain in psychiatry. Neuropsychopharmacology (suppl 1):S54–S63
14. Fuster JM (2003) Cortex and mind. Oxford University Press, New York
15. Schultz W (2000) Multiple reward signals in the brain. Nat Rev Neurosci 1:199–207
16. Desimone R, Duncan J (1995) Neural mechanisms of selective visual attention. Annu Rev Neurosci 18:193–222
17. Grossberg S (1999) The link between brain learning, attention and consciousness. Conscious Cogn 8:1–44
18. Miller EK, Cohen JD (2001) An integrative theory of prefrontal cortex function. Annu Rev Neurosci 24:167–202
19. Schall JD (2001) Neural basis of deciding, choosing and acting. Nat Rev Neurosci 2:33–42
20. Posner MI, Rothbart MK (1998) Attention, self-regulation and consciousness. Philos Trans R Soc London B 353:1915–1927
21. Pally R (2005) Non-conscious prediction and a role for consciousness in correcting prediction errors. Cortex 5:617–738
22. Pally R (2001) A primary role for nonverbal communication in psychoanalysis. Psychoanal Inquiry 21:71–93
23. Pally R (2002) The neurobiology of borderline personality disorder: the synergy of "nature and nurture". J Clin Psychiatry 8:133–142
24. Pally R (2000) The mind–brain relationship. London, Karnac Books
25. Beebe B, Knoblauch S, Rustin J, Sorter D (2005) Forms of intersubjectivity in infant research and adult treatment. Other Press, New York
26. Davidson RJ, Jackson DC, Kalin NH (2000) Emotion, plasticity, context and regulation: perspectives from affective neuroscience. Psychol Bull 126(6):890–909
27. Panksepp J (1998) Affective neuroscience. Oxford University Press, Oxford, UK
28. Greenspan S, Shanker S (2004) The first idea: how symbols, language, and intelli-

gence evolved from our primate ancestors to modern humans. Da Capo Press, Cambridge, Mass

29. Depue RA, Morrone-Strupinsky JV (2005) A neurobehavioral model of affiliative bonding: implications for conceptualizing a human trait of affiliation. Behav Brain Sci 28:313–350
30. Solms M (1996) What is consciousness? Charles Fischer memorial lecture to the New York Psychoanalytic Society. J Am Psychoanal Assoc 45:681–778
31. Gabbard GO, Weston D (2003) Rethinking therapeutic action. Int J Psychoanal 84:823–844
32. Kandel ER (1999) Biology and the future of psychoanalysis: a new intellectual framework for psychiatry revisited. Am J Psychiatry 156:505–524
33. Pally R (2005) A neuroscience perspective on forms of intersubjectivity in infant research and adult treament. In: Beebe B, Knoblauch S, Rustin J, Sorter D (eds) Forms of intersubjectivity in infant research and adult treatment. Other Press, New York, pp 191–241
34. Meaney MJ (2001) Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. Annu Rev Neurosci 24:1161–1192
35. Suomi SJ (1999) Attachment in rhesus monkeys. In: Cassidy J, Shaver PR (eds) Handbook of attachment: theory, research, and clinical application. Guilford Press, New York, pp 181–197
36. Suomi SJ (2003) Gene–environment interactions and the neurobiology of social conflict. Ann N Y Acad Sci 1008:132–139
37. Reiss D, Neiderhiser JM (2000) The interplay of genetic influences and social processes in developmental theory: specific mechanisms are coming into view. Dev Psychopathol 12:357–374
38. Caspi A, Sugden K, Moffitt TE et al (2003) Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. Science 301:291–293
39. Kaufman J, Yang B, Douglas-Palumberi H, Houshyar S, Lipschitz D, Krystal J, Gelernter J (2004) Social supports and serotonin transporter gene moderate depression in maltreated children. Proc Natl Acad Sci USA 101:17325–17326
40. Schwartz A (1992) Not art but science: applications of neurobiology, experimental psychology and ethology to psychoanalytic technique. I. Neuroscientifically guided approaches to interpretive "what's" and "when's". Psychoanal Inquiry 12:445–474
41. Wager T, Rilling J, Smith E, Sokolik A, Casey K, Davidson R, Kosslyn S, Rose R, Cohen J (2004) Placebo-induced changes in FMRI in the anticipation and experience of pain. Science 303:1162–1167
42. LeDoux J (1996) The emotional brain. Simon and Schuster, New York
43. Bowlby J (1969) Attachment and loss, vol 1. Basic Books, New York
44. Leon M (1992) The neurobiology of filial learning. Annu Rev Psychol 43:377–398
45. Quirk GJ, Likhtik E, Pelletier JG, Pare D (2003) Stimulation of medial prefrontal cortex decreases the responsiveness of central amygdala output neurons. J Neurosci 23:8800–8807
46. Nadel L, Land C (2000) Memory traces revisited. Nat Rev Neurosci 1:209–212
47. Sugrue LP, Corrado GS, Newsome WT (2005) Choosing the greater of two goods: neural currencies for valuation and decision making. Nat Rev Neurosci 6:363–375
48. Berns GS, McClure SM, Pagnoni G, Montague PR (2001) Predictability modulates human brain response to reward. J Neurosci 21:2793–2798
49. Knowlton BJ, Mangels JA, Squire LR (1996) A neostriatal habit learning system in humans. Science 273:1399–1402

50. Raichle ME (1998) The neural correlates of consciousness: an analysis of cognitive skill learning. Philos Trans R Soc London B 353:1889–1901
51. Hikosaka O, Miyashita K, Miyachi S, Sakai K, Lu X (1998) Differential roles of the frontal cortex, basal ganglia, and the cerebellum in visuomotor sequence learning. Neurobiol Learn Mem 70:137–149
52. Schumann JH, Crowell SE, Jones NE, Lee N, Schuchert SA, Wood LA (2004) The neurobiology of learning: perspectives from second language acquisition. Lawrence Erlbaum, Mahwah, NJ
53. Grigsby J (1994) Procedural learning and the development and stability of character. Percept Mot Skills 79:355–370
54. Wise SP, Murray EA, Gerfen CR (1996) The frontal cortex-basal ganglia system in primates. Crit Rev Neurobiol 10:317–356
55. Baron-Cohen S (1993) From attention-goal psychology to belief-desire psychology: the development of a theory of mind and its dysfunction. In: Baron-Cohen S, Tager-Flusberg H, Cohen JD (eds) Understanding other minds. Oxford University Press, Oxford
56. Frith CD, Frith U (1999) Interacting minds: a biological basis. Science 186:1692–1695
57. Bischoff-Grethe A, Proper AM, Mao H, Daniels KA, Berns GS (2000) Conscious and unconscious processing of nonverbal predictability in Wernicke's area. J Neurosci 20:1975–1981
58. Ruby P, Decety J (2001) Effect of subjective perspective taking during simulation of action: a PET investigation of agency. Nat Neurosci 5:546–550
59. Tomasello M (1999) The cultural origins of human cognition. Harvard University Press, Cambridge, Mass
60. Fonagy P, Gergely G, Jurist EL, Target M (2002) Affect regulation, mentalization, and the development of the self. Other Press, New York
61. Rizzolatti G, Arbib M (1998) Language within our grasp. Trends Neurosci 21:188–194
62. Iacoboni M, Molnar-Szakacs I, Gallese V et al (2005) Grasping the intentions of others with one's own mirror neuron system. PLoS Biol 3(3):e79
63. Carr L, Iacoboni M, Dubeau M, Mazziotta JD, Lenzi GL (2003) Neural mechanisms of empathy in humans: a relay from neural systems for imitation to limbic areas. Proc Natl Acad Sci USA 100:5497–5502
64. Fernandez-Duque D, Baird J, Posner M (2000) Executive attention and metacognitive regulation. Conscious Cogn 9:288–307
65. Gurvits IG, Koenigsberg HW, Siever LJ (2000) Neurotransmitter dysfunction in patients with borderline personality disorder. Psychiatr Clin N Am 23(1):27–40

Chapter 8

The Brain's Experience-Dependent Plasticity, State-Dependent Recall, and Creation of Subjectivity of Mental Functions

DIETRICH LEHMANN AND MARTHA KOUKKOU

Introduction

The international conference on "Neurosciences and Psychoanalysis: memory, emotions and dreams" held in Genoa in 2004 focused on three core themes in theory and research in both psychoanalysis and human brain sciences: memory, emotion, and dreaming. We participated in this conference by presenting the basic concepts of an integrative model of the brain functions that create biography (and thus individual thoughts, emotions, plans, and dreams), leading to an individual subjective viewpoint during all states of development and of consciousness. Using these concepts we discuss (1) psychosocially manifested developmental changes as products of the brain's learning and memory functions that create biography, i.e., autobiographical memory via experience-dependent cortical plasticity; and (2) the role of the brain's state-dependent but memory-driven retrieval processes in forming the individual's momentary thoughts, emotions, and actions, and their conscious perception, as well as the dream's content and the possibility of it being remembered during wakefulness.

The Model of the Brain Functions that Create Biography

The model integrates well-established findings in neurosciences and psychophysiology, including the findings of electroencephalogram (EEG) studies on learning, memory, and retrieval during development, and during wakefulness and sleep. In particular, it examines the adaptive brain functions in the neocortex that associate the meaning and significance of experiences with the psychosocially recognizable dimensions of behavior [1–4]. This model was originally used to investigate dreaming, its functional significance, and its use in psychoanalysis [5].

The model follows basic principles of dynamic systems theory as used to study the behavior of complex, living systems and as used in human neurosciences to study brain–behavior relationships. The basic principles of dynamic systems theory that are of prime importance for our considerations are:

- Postnatal survival and biopsychosocial development presupposes (and emerges out of) the continuous and dynamic interaction of individuals with their internal realities (their organs, including the brain) as well as with the external physical and social environment. This interaction occurs continuously both during wakefulness and during sleep.
- A given degree of cooperative interactions, the dialogue between the social environment (the caretakers) and the developing individual is the prerequisite for healthy biopsychosocial development.

The Functions

The functions within the organism that enable newborn infants to embark on postnatal life as a dynamic interaction between the external realities in which they are situated and the internal realities with which they were born are the functions of the central, peripheral, and autonomic nervous system. These functions matured during gestation. They are studied by neurophysiologists and psychophysiologists as the continuously recycling memory-driven information-processing operations of perception, evaluation, and response.

We discuss these functions of the nervous system as the operations of the human cycle of communication.

At birth, infants have all the operations of the cycle of communication at their disposal, but no knowledge about the realities into which and with which they were born. The operations of the cycle of communication predispose the newborn to perceive human-specific signals (e.g., smells, sounds, phonemes) and to evaluate them in relation to their significance for the organism's basic needs (innate knowledge about what is supporting psychobiological well-functioning and, thus, well-being and what disturbs well-being) and, as result of this evaluation, to initiate a complex response pattern manifested by the functions of the central, peripheral, and autonomic nervous system.

Some aspects of this complex response pattern of the child have been interpreted–in our opinion erroneously–as aggressive, not only by psychoanalysts but also by neuroscientists. We cannot cover this topic in this chapter (cf. [2, 6]).

With this complex response pattern, infants (1) may keep or restore the well-functioning, and thus well-being, by restoring the homeostatic values to within their functional levels; (2) are able to communicate these effects to the caretakers, and invite them to care for the well-being, i.e., for the needed degree of cooperativity; and (3) can initiate the molecular mechanisms of learning and memory, the brain functions that create biography. These functions translate the effects of the human-specific experiences as well as the effects of the caregivers' behavior on innate knowledge about what supports well-being and what disturbs well-being into autobiographical memory. These learning and memory functions have been studied as the experience-dependent neuronal plasticity of the neocortex, as the neuronal mechanisms that extract personal meaning out of the interaction of individuals with their environments and translate them into the neural architectures of the cortico-cortical connectivity, into the mnemonic networks that represent the autobiographical memory, the myriad of idiosyncratic associations of personal experiences, events, objects, names, actions, thoughts, emotions, decisions and so on, which characterize the individual's cognitive-emotional and behavioral styles [7–11].

The basic conclusion of the integrated evidence is that the brain functions that initiate and coordinate the continuous interactions of humans with their physical and social environment create individual memory, i.e., neuronal, mnemonic networks which represent the biography. The biography's contents shape, with the memory-driven, consciously and nonconsciously functioning, information-processing operations, 1) all aspects of the individual's behavior (thoughts, emotions, decisions, and actions) and also 2) the individual's interaction style with others and with the physical world.

The data integrated in our model show that the internal realities for human beings consist of the functional state of the organs, including the brain, and of the progressively acquired, and at each moment accessible contents of the autobiographical memory. Within this context, the human brain is understood as a self-organizing system that creates the individual's behavior (cognitions, emotions, and actions) on the basis of its biography (in other words, on the basis of acquired knowledge).

The functions of the brain are:

1. To assimilate and use information coming from the internal and external (physical and social) realities in order to acquire knowledge, i.e., symbolic representations about the characteristics of the realities in which the individual is born and lives, and about the outcome of the interactions with them.
2. To update and enlarge this knowledge continuously.
3. To use this knowledge with memory-driven, conscious and nonconscious information-processing operations in order to generate (to create) new, "private" knowledge in the form of thoughts, fantasies, emotions, actions, decisions, coping and problem-solving strategies, and herewith to organize and coordinate the individual's behavior within his/her own realities.

Autobiographical memory contents are multimodal and multicoded, which means they are coded in acquired verbal and nonverbal codes, as well as in acquired emotional codes. Emotional codes represent the quality of the interactions between a developing individual and the environment, mainly the social environment. This is of basic importance for the dialogue between psychoanalysis and brain research, and is one of the basic proposals of the brain model we are working with.

The Biopsychosocial Development of Individuals

We next present a short survey of the well-established correlations between psychosocially manifested behavioral development and systematic changes of (1) cortical neuroanatomy as manifested in the experience-dependent increase of cortico-cortical connections, and (2) cortical functioning as manifested in systematic changes of EEG amplitude, wave frequency, coherence, and dimensional complexity. We then discuss the functional significance of such parallel developmental changes for understanding changes in accessibility to autobiographic memory contents from early to later developmental stages (childhood amnesia), as well as from sleep to wakefulness and the opposite (dream recall).

Developmental Changes in Cortical Neuroanatomy

Research during the past decades has shown that humans are born with most of the brain neurons they will ever have (more than 100 billion). The mass of the brain at birth is, however, only one-fourth of that of the adult brain. During development, in addition to other changes, a tremendous increase in cortico-cortical connections occurs. Each neuron interacts via synapses with 10 000–15 000 other neurons; the number of synaptic contacts in the cerebral cortex of an adult human is estimated to be between 10^{14} and 10^{16}.

These neuroanatomically manifested developmental changes resulting in the increase in cortico-cortical connectivity go hand in hand with the behavioral changes that characterize the child's psychosocial development. They are considered to be the products of the experience-dependent cortical plasticity and to represent individual memories [10–13].

Individual memories, in the words of Fuster [7], are "deposited and represented in the neocortex. This does not imply that memory function is restricted in this part of the brain–far from it. We now know that the deposition of memories essentially requires the intervention of certain limbic and subcortical structures. Furthermore, normal behavior is probably determined to a large extent by neural changes that have occurred in noncortical structures as a result of individual experiences. However, what we commonly understand as memory–that is the aggregate of personal experiences of events, objects, names, actions, and knowledge of all sorts, whether or not accessible to consciousness–is represented in the neocortex, particularly within what we commonly call the cortex of association" (p. x in [7]).

"A memory is basically a network of neocortical neurons and the connections that link them and is formed by experiences. That network is the result of a concurrent activation of neuronal assemblies that represent diverse aspects of the internal and external environment and of motor actions. What most critically defines a network (i.e., a memory representation) is the ensemble of connections that has formed it. Relationship is the essence and, in this sense, all memory is associative . . . association . . . understood as an attribute of all memories, at the root of their genesis as well as their evocation" (p. 2 in [7]).

Developmental Changes in Cortical Functioning

The neuroanatomically manifested developmental changes described above are accompanied by changes in cortical functioning as manifested in the EEG. More than 60 years of work using EEG have demonstrated a systematic increase in EEG wave frequency, in EEG coherence between regions, between hemispheres, and between anterior and posterior areas, and in dimensional complexity during wakefulness, from birth to adulthood [14–16].

What is considered to be the functional significance of these well-established parallel developmental changes in EEG, and of cortico-cortical connectivity, during human psychosocial development?

It is more or less generally accepted that the developmental EEG changes during wakefulness reflect the developmental increase in cortico-cortical synaptic connectivity, and imply that with age, the complexity of the mnemonic networks (i.e., the number of involved neuronal populations and their quantity) increases; in other words, the amount of autobiographical knowledge increases [7, 8, 12, 17–20].

Thus, developmental changes in the brain's functional state as manifested in the EEG during wakefulness reflect the level of attained complexity of the neuronal representations of the autobiographical memory contents. However, this developmental increase in individual knowledge is linked with the phenomenon of childhood amnesia (i.e., the finding that it is very difficult for people to remember events that occurred before they were 4 years old).

Childhood amnesia, together with dreaming and dream recall, are of basic importance in psychoanalytic theory and practice. In order to discuss the nature and functional significance of these phenomena within the framework of our working model, we now introduce its central concept. This is the concept of multifactorially defined and EEG-manifested functional states of the brain. It implies memory-driven information-processing operations with EEG-state-dependent accessibility of knowledge. More precisely, the multifactorially defined and EEG-manifested functional states of the brain reflect (1) *in the ontogenetic domain*, the level of attained complexity of the neuronal networks (the memory representations) of the individual knowledge, of the autobiographical memory contents; and (2) *in the short-term temporal domain*, the level of complexity of the mnemonic networks that are accessible to the memory-driven information processing operations and thus can be used for the organization of behavior at each moment in time. Memory representations of higher complexity (accompanied by faster EEG wave frequencies during wakefulness) can be activated during EEG states of slower frequencies (during sleep EEG), but not the opposite. This is the asymmetric accessibility of acquired knowledge. In other words, the electrically manifested functional states of the brain define which autobiographical knowledge at which level of complexity is activated, and thus can be used by the memory-driven information-processing operations for the organization of behavior at a given moment in time.

The above stated central concept of our model was developed on the basis of research findings that show close relations between cognitive-emotional and action styles with EEG macrostates during development and during wakefulness and sleep, as well as, more specifically, with EEG microstates during adult wakefulness. In the following sections, recent data on EEG microstates and their relation to subjective conscious and nonconscious experience are briefly discussed, and childhood amnesia is addressed, as well as dreams, in particular the autobiography-reflecting cognitive and emotional contents of dreams, and the fragility of their recall.

EEG Microstates and their Relation to Subjectivity

The correspondences between mental functions and EEG measurements in terms of EEG wave frequencies, coherences, and dimensionality concern time epochs in the range of seconds to minutes. Clearly, a successful interaction between an individual and the surrounding world needs to occur within fractions of a second–the time range during which perceptions, thoughts, and emotions are incorporated in the brain. Brain activity in this subsecond time range can be assessed by analyzing electrical activity. Such studies have revealed close correspondences between the objective EEG measurements and personal experience. Using multichannel EEG data, for each moment in time, a potential distribution map (potential landscape map) can be constructed on the scalp. It has been shown that these landscapes change quasi-stepwise, at intervals of 10 ms to about 100 ms. Different maps must have been produced by different neuronal generating processes. Accordingly, this observation of quasi-stable epochs of brain electric potential maps indicates that brain work occurs in brief temporal packages, on the average lasting below 100 ms, in "microstates," which are the putative "atoms of thought and emotion."

The following sample studies illustrate that different classes (defined by map landscapes) of brain electric microstates are associated with different types or classes of subjective experience that occur spontaneously or that are driven by input information.

During spontaneous mental activity (mentation) in a day-dreaming condition, 13 healthy subjects reported "what just went through your mind" when prompted at random intervals (about 30 reports per subject) while their EEG was recorded [21]. Independent raters classified the reports as mentation of visual imagery or abstract thought. Examples are: "I saw our lunch on that sunny beach with the blue ocean," or "I was worrying about the meaning of 'theory,' the concept." The EEG data were parsed into successive microstates: on the average, across subjects, the last microstate before the report prompt showed significantly different landscape maps for visual imagery than for abstract thought. A follow-up analysis confirmed that there was no significant difference of map landscape between the two mentation classes in the last-but-one microstates.

In another study, on input-driven experience, 25 subjects silently read nouns from a computer screen [22]. The nouns were words of high or low imaginability (e.g., "dog" versus "belief"). Subjects repeated the last word aloud if (rare) a question mark followed. Hence, subjects were not aware that visualizable versus abstract mentation was being studied, but attended to the memory task. The EEG map sequences during word presentation (450 ms) were averaged separately for visualizable and abstract words (event-related potential ERP map series). Microstate segmentation of the ERP map series determined seven microstates during the 450 ms of word display. Microstate number 5 (which lasted from 286 ms to 354 ms after word display onset) showed, across subjects, significantly different map landscapes for the two word classes.

In both studies, the microstate maps that incorporated abstract mentation versus those that incorporated visual imagery showed a clockwise rotation of the axis of the electric field map, and also the electric gravity center of the relevant microstates was more anterior and left for abstract mentation, and more posterior and right for visual imagery. In a post-hoc localization analysis, the scalp map data were recomputed to intracortical distributions of source electric strength (current density) using low-resolution electromagnetic tomography (LORETA). Across subjects (voxel-by-voxel *t*-tests), in both experiments independently [23], the results showed stronger activity for visual imagery microstates in a cluster of right posterior voxels (Brodmann areas 20, 36, and 37), and stronger activity for abstract thought microstates in a cluster of left anterior voxels (Brodmann areas 38 and 47).

In a study on automatic processing of emotional information [24], 21 healthy subjects read words silently from a computer screen, nouns with emotionally pleasant or unpleasant connotations (such as "peace" versus "death"), while their EEG was recorded. Again, subjects repeated the last word aloud if it was followed by a question mark. During the 450 ms of word display, 14 brain electric microstates were identified in the ERP maps. Three of these 14 microstates showed different brain electric maps for pleasant and unpleasant words (96–122 ms, 184–202 ms, and 248–274 ms after word onset). The brain electric activity was then localized using LORETA functional imaging tomography. In all three relevant microstates, positive-emotion words produced more anterior localized activity, while negative-emotion words produced more posterior localized activity. However, activity differed in other respects between the microstates: e.g., in two it was predominantly left-lateralized, but in one it was predominantly right-lateralized, for negative as well as positive emotion.

In the microstate studies discussed above, the subjects did not have to distinguish visual imaging versus abstract mentation, or positive-versus negative-emotion words. The subjects consciously attended to the recall tasks. Thus, the mentation type-specific activation of the brain areas during a given microstate occurred automatically without any attempt to imagine or formulate a conscious thought or to judge or mimic an emotion. In addition, identical brain areas were activated when the mentation was driven by exterior events (displayed words) or internal events (spontaneous ideas).

In summary: In packages of fractions of seconds, very different patterns of brain electric activity incorporate abstract or imagery-type mentation, or emotional positive or negative evaluation of information, both conscious and nonconscious. The very rapidly changing brain electric activity configurations that follow each other in packages, the brain electric microstates, apparently incorporate identifiable, subjective, consciously retrievable experience. These space-defined, subsecond microstates are embedded into the longer-lasting EEG macrostates that are defined by wave frequency, coherence, and dimensionality.

Childhood Amnesia, Dreams, Dream Content, and the Fragility of Dream Recall

"Childhood amnesia" refers to the observation that juveniles and adults have difficulty in recalling events and experiences that happened before the age of about 4 years, although very often their behavior indicates that they do have some knowledge of these experiences.

Dreaming refers to any image, thought, or emotion attributed by the individual to the condition of sleep, before awakening. This sleep condition consists of various sleep stages that are manifested by repeated changes of the brain's functional state as recorded in the EEG; they are characterized in terms of EEG macrostates by systematic slowing of EEG wave frequency from wakefulness to NREM stages and subsequent appearance of faster wave frequencies during REM stages.

We have seen that psychosocial development goes parallel with EEG changes from slower to faster wave frequencies. They reflect the increasing complexity and quantity of the autobiographical memory contents. We now see that sleep shows EEG changes from faster to slower EEG wave frequencies. Thus, there are reverse relationships between developmental EEG changes during wakefulness and vigilance changes from wakefulness to sleep. What is the functional significance of these EEG findings for childhood amnesia and for dream content and dream recall within the framework of our model?

Childhood Amnesia

Within the concept of multifactorially defined and EEG-manifested brain functional states with state-dependent and asymmetrical accessibility of acquired knowledge, the phenomenon of childhood amnesia is explained as follows. Mnemonic representations of knowledge of events and experiences acquired during earlier stages of development are represented by less complex mnemonic networks; when these become incorporated by repetitions during further development into more complex mnemonic networks (reflected in EEG-manifested later stages of development) they cannot be activated and thus recalled in their original context during later stages of development. For example, once one has learned to read fluently one recognizes (i.e., reads) whole words and not the individual letters.

Dreams

The flow of information to the brain from the internal and external realities and the dynamic and selective interaction with these realities are not interrupted by the EEG changes from waking to sleep. During all sleep stages (NREM and REM) the sleeping individual (1) perceives incoming information, (2) evaluates the significance of this information for the momentary psychobiological priorities, i.e., for sleep necessity, and (3) selects and executes all aspects of sleep behavior.

However, many dimensions of behavior during sleep, especially thoughts and emotions, are different from behavior during wakefulness. Empirical dream research has shown clearly that dreams, in spite of the way they are (bizarre, unrealistic, fantastic, discontinuous), reflect the dreamer's own knowledge and personal views about the world in which she/he lives, and about him/herself [25–27]. Dreams often include memories from the dreamer's childhood. Thus, the sources of the dream's design have to be found in the dreamer's biography. These characteristics of dreams have been integrated into the framework of our and related models to imply that humans during sleep have access to mnemonic networks of lesser complexity, to earlier memories.

Within the framework of the model, these dream characteristics are explained both by the active elimination and change of the incoming external information by the preparations for going to sleep and by the changes in the level of accessibility of the mnemonic networks during sleep, induced by the changes in the brain's EEG-manifested functional states from waking to sleeping.

Based on the similarities of EEG patterns between developmental states of the awake brain with the states of the adult sleeping brain, we propose that sleep stages–and this mainly after the age at which children can report dreams–imply functional regressions of the level of complexity of the accessible mnemonic networks to the level of complexity of earlier stages of development.

Considering dream generation, this functional regression during sleep means that knowledge acquired during earlier developmental stages, and thus represented by networks of lower complexity, again becomes available to the operations of the cycle of communication for the organization of the interactions with the realities during sleep. This corresponds to a "primary process"-like analysis of realities, and within the framework of the model supports the maintenance of sleep so that the functions of sleep have time to occur [1, 25].

On the other hand, this functional regression permits, due to the asymmetric way of functioning of state-dependent recall, the comparison of the incoming information with the mnemonic contents of earlier and actual developmental stages, and therefore its evaluation in the light of biographically defined previous and recent memory content [4, 28, 29].

Thus, the data summarized in the concept of EEG-manifested functional brain states with state-dependent asymmetrical accessibility of acquired knowledge suggest that reported dreams are the aspects of the sleep-stage-dependent (accessible knowledge-dependent) interactions of the individual with his/her internal and external realities during sleep that are recallable in wakefulness. Awakenings out of REM stages (EEG states close to wakefulness) are very often accompanied by dream recall. Thus, dreams originate from the interpretation of the incoming information during sleep in the light of the contents of the sleep-stage-dependent portions of accessible knowledge as synthesized to the dream's thoughts, emotions, and fantasies by the synergetic functions of the neocortex that use accessible knowledge to create subjectivity. Dreams portray the individual's current biopsychosocial priorities in wakefulness as interpreted in the light of the sleep-stage-dependent accessible knowledge.

Accordingly, within the framework of the model, dreaming is a continuous process, like mentation during wakefulness, in all stages of development. It originates from the same memory-driven information-processing operations (brain functions) from which all other subjectively perceived aspects of human existence originate in all levels of development and vigilance. Dreaming reflects the results of the active and selective interaction of the individual during sleep with his/her internal realities (e.g., sleep need, experiences during the day, sleep-state-dependent activated knowledge) and external realities (e.g., noise from the environment, darkness, new sleep surroundings). Consequently, the dream's thoughts, images, and emotions might reflect an "effort of problem solving," as has been proposed for work with dreams in psychoanalysis, inasmuch as there might be a "problem" in the individual's current realities and/or in the content of the accessible autobiographic memory content. Otherwise, dreams reflect the continuous interaction of the sleeper with the environment and the herewith associatively activated thoughts, emotions, and fantasies during sleep. However, since during sleep, due to the asymmetry of the retrieval processes, a wider spectrum of the individual's knowledge is accessible (less complex mnemonic networks of earlier developmental stages incorporated in mnemonic networks in the level of current complexity), a wider spectrum of individual "solutions" is available for tentative application to new contextual or problem material. This is reflected in dream content. Thus, working with dreams in psychotherapy helps to gain insight into a wider spectrum of the individual's emotions, thoughts, and cognitive-emotional coping strategies.

References

1. Koukkou M, Lehmann D (1993) A model of dreaming and of its functional significance: the state-shift hypothesis. In: Moffitt A, Kramer M, Hoffmann R (eds) The functions of dreaming. State University of New York Press, Albany, NY, pp 51–118
2. Koukkou M, Lehmann D (1996) Models of human brain functions and dysfunctional elements in human history: there is a close relation. In: Razis D (ed) The human predicament: an international dialogue on the meaning of human behavior. Prometheus Books, Amherst, NY, pp 269–280
3. Koukkou M, Lehmann D (2002) Memory, adaptive orienting and psychosomatics: a brain model. Int Congr Ser ICS 1241C:305–311
4. Lehmann D, Koukkou M (1990) Brain states of visual imagery and dream generation. In: Kunzendorf RG, Sheikh AA (eds) The psychophysiology of mental imagery: theory, research and application. Baywood, Amityville, NY, pp 109–131
5. Koukkou M, Lehmann D (1983) Dreaming: the functional state-shift hypothesis, a neuropsychophysiological model. Br J Psychiatry 142:221–231
6. Koukkou M, Lehmann D (2002) Human brain synaptic plasticity as key for understanding wise and erroneous decisions. In: Razis DV (ed) The human predicament, II. S&P Advertising, Athens, pp 103–135
7. Fuster JM (1995) Memory in the cerebral cortex. MIT Press, Cambridge, MA
8. Fuster JM (2001) The prefrontal cortex–an update: time is of the essence. Neuron 30:319–333
9. O'Leary DDM (1996) Areal specialization of the developing neocortex: differentiation, developmental, plasticity and genetic specification. In: Magnusson D (ed) The lifespan development of individuals: behavior, neurobiological, and psychosocial perspective. Cambridge University Press, Cambridge, UK, pp 23–37
10. Buonamano DV, Merzenich MM (1998) Cortical plasticity: from synapses to maps. Annu Rev Neurosci 21:149–186
11. Merzenich MM, deCharms RC (1998) Neural representations, experience, and change. In: Llinas R, Churchland PS (eds) The mind–brain continuum: secondary processes. MIT Press, Cambridge, MA, pp 61–91
12. Huttenlocher PR (1994) Synaptogenesis in human cerebral cortex. In: Dawson G, Fischer KW (eds) Human behavior and the developing brain. Guilford Press, New York, pp 137–152
13. Dityatev A, Schachner M (2003) Extracellular matrix molecules and synaptic plasticity. Nat Rev Neurosci 4:456–468
14. Harmony T, Marosi E, Diaz de Leon AE et al (1990) Effect of sex, psychosocial disadvantages, and biological risk factors on EEG maturation. Electroenceph Clin Neurophysiol 75:482–491
15. Thatcher RW (1994) Cyclic cortical reorganisation: origins of human cognitive development. In: Dawson G, Fischer KW (eds) Human behavior and the developing brain. Guilford Press, New York, pp 232–266
16. Anokhin AP, Birbaumer N, Lutzenberger W et al (1996) Age increases brain complexity. Electroenceph Clin Neurophysiol 99:63–68

17. Bell MA, Fox NA (1992) The relations between frontal brain electrical activity and cognitive development during infancy. Child Dev 63:1142–1163
18. Arbib MA, Érdi P (2000) Précis of Neural organization: structure, function, and dynamics. Behav Brain Sci 23:513–533 and Discussion 533–571
19. Johnson MH (2003) Development of human brain functions. Biol Psychiatry 54:1312–1316
20. Koukkou M, Federspiel A, Bräker E et al (2000) An EEG approach to the neurodevelopmental hypothesis of schizophrenia studying schizophrenics, normal controls and adolescents. J Psychiatr Res 34:57–73
21. Lehmann D, Strik WK, Henggeler B et al (1998) Brain electric microstates and momentary conscious mind states as building blocks of spontaneous thinking. I. Visual imagery and abstract thoughts. Int J Psychophysiol 29:1–11
22. Koenig T, Kochi K, Lehmann D (1998) Event-related electric microstates of the brain differ between words with visual and abstract meaning. Electroencephalogr Clin Neurophysiol 106:535–546
23. Lehmann D, Koenig T, Henggeler B et al (2004) Brain areas activated during electric microstates of mental imagery versus abstract thinking. Klin Neurophysiol 35:169
24. Gianotti LRR, Faber PL, Pascual-Marqui RD et al (2004) Processing of positive versus negative emotion is incorporated in anterior versus posterior brain areas: an ERP microstate LORETA study. Eur Arch Psychiatr Clin Neurosci 254 Suppl 1:17
25. Moffitt A, Kramer M, Hoffmann R (eds) (1993) The functions of dreaming. State University of New York Press, New York
26. Strauch I, Meier B (1996) In search of dreams: results of experimental dream research. State University of New York Press, New York
27. Mancia M (2004) The dream between neuroscience and psychoanalysis. Arch Ital Biol 142:525–531
28. Lehmann D, Koukkou M (2000) All brain work–including recall–is state-dependent. Behav Brain Sci 23:964–965
29. Mancia M (2003) Dream actors in the theatre of memory: their role in the psychoanalytic process. Int J Psychoanal 84:945–952

PART 2
The Shared Emotions

Chapter 9

The Sensorimotor Side of Empathy for Pain

Alessio Avenanti and Salvatore Maria Aglioti

Introduction

This chapter revolves around the phenomena and neural mechanisms underlying the human capability to empathize with the actions, emotions, and feelings of other individuals. Special attention is paid to the neural activity induced by observation and imagination of others' pain. It will be shown that representing others' pain brings about the activation of neural structures largely overlapping with those activated during the experience of pain on oneself and that neural structures involved in both emotional and sensorimotor processing may be recruited during empathy for pain.

Pain is an unpleasant subjective, sensory, and emotional experience associated with actual or potential tissue damage [1– 4]; and has a protective function related to the implementation of escape reactions. Thus, it is closely linked to the motor system [2, 5]. Sensory-discriminative components (e.g., evaluation of locus, duration, and intensity of a noxious stimulus) and affective-motivational components (e.g., unpleasantness of the noxious stimulus) contribute to the experience of pain [1–3]. Sensory and emotional components are represented in separate nodes of a complex neural network referred to as the "pain matrix" [6–9]. While the neural processing of pain perception has been widely studied, much less is known about the neural underpinnings of empathy for pain.

The term "empathy" is the English translation of the German word *Einfühlung*. This term was introduced into the psychology of aesthetics by Lipps [10] to indicate the experiences of individuals while contemplating pieces of artwork. The extension of this concept to the domain of intersubjectivity supported the notion that empathy is inherently linked to an inner imitation process [10].

Empathy plays a fundamental social role insofar as it allows the interindividual sharing of experiences, beliefs, aims, and inner states. It is widely held that empathy plays an important role in psychoanalysis and psychoanalytic therapy [11–13]. For instance, in *Jokes and Their Relation to the Unconscious* [14], Freud used this concept (influenced by the work of Lipps, which he profoundly admired) to designate the process of putting oneself into another's position, either consciously or unconsciously. According to Kohut [15, 16], empathy allows the therapist to understand what is going on inside the patient's mental life in an "experience-near" way. This would imply that the therapist had to place himself/herself into the mental life of the patient through a process of "vicarious introspection" [15]. A basic aspect of empathy in the psychoanalytic tradition appears thus to be related to the experiential understanding of others' mental states. This view of empathy seems tightly related to the accounts of empathy developed in the philosophy of mind, psychology, and neuroscience, and based on the notion of "simulation." According to simulation theories (ST) we understand others' behavior and mental states by putting ourselves in the "mental shoes" of others and thus covertly replicating their inner states in our own mind [17–27]. According to the neuroscientific formulation of this notion, empathy implies that perceptual, motor, or emotional states of a given individual activate the corresponding neural representations in another individual who observes that state [24–27].

Neural Representation of Physical Pain

Pain is a complex and enigmatic feeling that typically signals actual or potential tissue damage. The experience of pain can be described along two main phenomenological axes: (i) the sensory-discriminative dimension, comprising spatial, temporal, and intensity properties of pain; and (ii) the affective-motivational dimension, related to the unpleasantness of the stimulus, as well as the behavioral and autonomic reactions it evokes [1–3].

Consistent with the multidimensional view of pain, neuroimaging techniques (such as positron emission tomography, PET; and functional magnetic resonance, fMRI) demonstrate that a complex neural network, referred to as the "pain matrix," is involved in the experience of pain [6–9]. Sensory and affective components of pain are mapped in two major separate nodes of the pain matrix, namely the sensorimotor and the affective node. The sensorimotor node of the pain matrix includes the primary (S1) and secondary (S2) somatosensory cortices (and also sensorimotor structures, such as cerebellum, premotor, and motor areas). Animal studies demonstrate that somatosensory cortices receive noxious and innocuous somatosensory input from the somatosensory thalamus [2–30] and contain nociceptive neurons that code key features of the sensory-discriminative dimension of stimulus processing such as spatial, temporal, and intensive aspects of innocuous and noxious somatosensory stimuli [30–34]. Accordingly, neuroimaging studies in humans indicate that S1 and S2 process sensory features of pain and display a somatotopical organization [6, 8, 35–39]. Lesions to these areas may induce deficits of pain sensation in brain-damaged patients [40, 41]. For example, Ploner et al. [41] observed that a patient who had suffered a stroke that encompassed S1 and S2 did not experience a painful sensation when a hot laser stimulus was applied to the affected arm, indicating that intact somatosensory cortices are necessary for the normal experience of pain sensation. However, the patient reported an ill-localized and ill-defined unpleasant feeling in the absence of a clear pain sensation, suggesting that pain affect was present in the absence of pain sensation.

The affective node of the pain matrix includes at least the anterior cingulate cortex (ACC) and the insular cortex (IC) [42–50], which are phylogenetically old regions and are considered to be components of the classical limbic system [51] and of MacLean's "visceral brain" [52].

In primates, ACC receives input from medial thalamic nuclei that contain nociceptive neurons, including nucleus parafascicularis and the ventrocaudal part of nucleus medialis dorsalis [53, 54]. Direct pain input to the ACC is further suggested by the observations that painful stimuli evoke potentials over the human anterior cingulate gyrus and that single nociceptive neurons are present in the ACC of humans [55, 56], monkeys [57], and rabbits [58]. Neuroimaging studies have emphasized the role of the ACC in the perceived unpleasantness of physical pain [6–8]. Rainville et al. [49] used hypnotic suggestion to modulate the perception of unpleasantness during noxious stimulations. When the experimental subjects were influenced to perceive the noxious stimulations as highly unpleasant there was a concomitant increase in the activity in the ACC compared with when the subjects were influenced to perceive the same stimulation as less unpleasant [49]. However, the activity in the somatosensory areas was unaltered. In a similar vein, studies further indicate that increasing levels of ACC activity correspond with increasing levels of self-reported pain unpleasantness and distress [59, 60]. Thus, individuals who are dispositionally pain-sensitive show more ACC activity and report greater levels of perceived distress to painful stimulation [61].

The IC also receives direct thalamocortical nociceptive input in the primate [54], and has been implicated in autonomic regulation [62, 63]. The implication of IC in the subjective experience of pain is consistent with a function of the IC in higher-order processes relevant to homeostatic regulation [54, 64] and awareness of internal bodily processes [65]. Lesions to the IC may produce a clinical condition called "asymbolia for pain," or "Schilder–Stengel syndrome," in which patients show deficits in the affective-motivational component of pain but preserve their sensory discrimination. Such patients perceive painful stimuli but do not display the appropriate emotional responses to painful stimulation [66]. Neuroimaging studies indicate that the affective dimension of pain is mainly encoded in the anterior sector of the IC (anterior insula, AI) [6–8].

Beyond Nociception

Various physical painful experiences, ranging from being pricked with a pin to feeling an aching phantom pain [67], are represented in the different nodes of the pain matrix. However, pain does not have only a physical dimension related to tissue injury. Pain is also conceived of as a universal human experience that is commonly generalized to psychic suffering of any sort [54].

Interestingly, numerous languages characterize "social pain" (the pain resulting from social injury, e.g., in cases where social relationships are threatened, damaged, or lost) using words typically reserved for describing physical pain ("broken heart," "broken bones"). Animal lesion and human neuroimaging studies indicate that the neural circuitry and the computational processes underlying physical and social pain largely overlap [68, 69]. Interestingly, a recent fMRI study in humans demonstrates that the same sectors of the ACC that are involved in the perception of painful stimuli are also activated during the experience of social loss [70].[1]

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[1] In this fMRI study of social exclusion, participants were scanned while playing a virtual ball-tossing game from which they were ultimately excluded. Paralleling results from physical pain studies, the anterior cingulate cortex (ACC) was more active during exclusion than during inclusion, and correlated positively with self-reported distress [70].

Another indication of pain-related neural activity in the absence of physical noxious stimulations comes from a recent fMRI study in which Japanese participants listened to Japanese pain-evoking onomatopoeic words and nonsense syllables [71]. Listening to these sounds induced an increase in the fMRI BOLD signal in ACC, suggesting an activation of affective pain representation (see also Chapter 10 in this volume).

Although pain has been described as an essentially private subjective experience by some philosophers [72, 73], neuroscience studies support the view that pain processing has a fundamental social dimension that may extend to basic levels of neural processing.

Studies on empathy for pain further support and expand this view. In the following paragraphs we show that both affective and sensorimotor representations of the pain supposedly felt by a model are mapped in the observer's neural circuitry dedicated to processing the pain felt by oneself. We also discuss the important role of the motor system in the personal experience of pain and in some aspects of social cognition. These notions may allow us to construct a neuroscientifically based concept of empathy.

Pain and the Motor System

Pain is closely linked to action systems that can be considered as the division of the pain matrix (part of the sensorimotor node) involved in the implementation of appropriate reactions to actual or potential noxious stimuli. Nociceptive stimuli can elicit a series of defensive or reactive responses, such as withdrawal reflexes, avoidance behaviors, and emotional-motor reactions [2, 74, 75]. Moreover, chronic pain affects motor control by limiting and impairing not only actual movements [76] but also their covert mental simulation [77, 78]. Furthermore, electrical [79–81] or magnetic stimulation [82] of the primary motor cortex (M1) in patients suffering from intractable chronic pain (e.g., phantom pain) can attenuate their symptoms; in addition, pain severity in amputees increases with the shrinkage of the limb representation in M1 [67, 83]. Although the physiological basis of these phenomena is largely unknown, they hint at bidirectional influences between pain and motor systems; thus, specific activity in the motor systems influences activity in nociceptive systems and is influenced by it. Accordingly, some neuroimaging studies have shown that changes of metabolic activity in M1 and other motor-related structures can be induced by the delivery [8, 50, 84, 85] or even anticipation [86, 87] of painful stimuli. More reliable results have been observed by means of a neurophysiological assessment, such as transcranial magnetic stimulation (TMS).[2] TMS studies in humans show that a strong reduction in the excitability of corticospinal motor systems occurs in association with different types of nociceptive stimulation [88–91]. This motor inhibition is likely to represent the electrophysiological correlate of a defensive, withdrawal reflex.

All these studies demonstrate the important link between pain and motor systems. In the section entitled "The sensorimotor side of empathy for pain" we will show that this link may also occur at a social level. But first we will focus on the role of the motor system in social cognition and empathy.

Motor System and Mirror Neurons

Recent research in nonhuman and human primates has pointed at the role of motor systems in higher-order cognitive processes [92–94]. Particularly relevant to the present discussion is the discovery in the mon-

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[2] Transcranial magnetic stimulation (TMS) is a powerful, noninvasive neurophysiological technique based on Faraday's principles of electromagnetic induction. A brief pulse of current flowing through a coil of wire generates a magnetic field. If the magnitude of this magnetic field changes over time, then it will induce a secondary current in any nearby conductor. For brain stimulation, a pulse is produced in a coil held over a subject's head. As a brief pulse of current is passed through it, a magnetic field is generated that passes through the subject's scalp and skull with negligible attenuation. This time-varying magnetic field induces an electric current in the subject's brain, causing depolarization of cellular membranes and thereby neuronal activation. In many experiments, single pulses of TMS are applied over the motor cortex. The stimulation of the motor cortex is able to transsynaptically activate the corticospinal system and to produce a response in controlateral extremity muscles, the motor-evoked potential (MEP), which can be recorded by means of electrodes. The amplitude of MEPs is used as a measure of corticospinal excitability. The amplitude of these potentials is modulated by the behavioral context. Thus, the modulation of the amplitude of MEPs can be used to assess the central effects of various experimental manipulations. This approach has been used in basic neuroscience to study the effect of actual nociceptive stimulation on corticospinal excitability [5], and in cognitive neuroscience to study the modulation of the motor system during the observation of painful events delivered to others [133] or during the observation of actions performed by others [98, 99].

key premotor and parietal cortices of a particular population of visuomotor bimodal cells called "mirror neurons." The most remarkable functional characteristic of these neurons is the increase in their firing rate both when the monkey performs an action, and when he observes a similar action made by another human or monkey agent [95–97].

Evidence in support of a motor mirror system (MMS) in humans comes from a single-pulse TMS study showing that the mere observation of a given movement brings about a specific increase in amplitude of MEPs recorded from the muscles that would be recruited during actual execution of the observed movements [98, 99].

The link between perception and execution of actions is further supported by behavioral studies showing that execution of a given action is positively or negatively modulated by observation of the same or a different action [100, 101]. Importantly, neuroimaging and neurophysiological studies in humans indicate that frontoparietal structures known to be involved in action execution become active during action observation [102–111]. Moreover, the observation of actions made with different effectors activates different regions of premotor and parietal areas, thus suggesting that the MMS may be organized according to somatotopic rules [112]. These studies suggest that humans have a MMS similar to that originally discovered in monkeys. When we observe an action performed by others, our motor system becomes active *as if* we were executing the very same action. This covert mimicking can be conceived as an inner simulation of the action. It has been proposed that simulating others' actions may be crucial for action understanding [21– 23, 25, 26, 93, 113–118]. Indeed these inner motor simulations lead to shared states between self and others and may allow us to directly understand the meaning of others' action without any explicit reflective mediation [23, 26]. Thus, MMS might constitute a basic system for coding and understanding observed actions which can be of fundamental importance, not only for motor learning and imitation but also for other social aspects of cognition, such as an understanding of others' intentions and beliefs [97, 119–121].

From Mirror Neurons to Empathy

There is a rapidly growing neuroscientific literature supporting the idea that we understand other people's behavior and thoughts, in part, by putting ourselves in the "mental shoes" of others [21–26]. Indeed, several authors have proposed that the simulative processes originally discovered and described in the domain of actions could constitute a basic characteristic of our social brain and of our ability to understand and empathize with others [23–26].

Empathy is the ability to have a direct experiential understanding of others' feelings and inner states [22, 23, 26]. Empathy is deeply grounded in the experience of our living body [122], and it is this experience that enables us to directly recognize others as *persons* like us [23, 123]. As a body-owner, we can easily grasp, through a process of sharing, the meaning of actions, sensations, or emotions displayed by others [23].

Current neuroscientific models of empathy postulate that a given motor, perceptual, or emotional state of an individual activates corresponding representations and neural processes in another individual observing that state [22–24, 27]. Thus, a basic mechanism in the empathic experience may involve the transformation of third-person visual information about others into first-person, bodily representations [27, 124, 125].

This feature may be a basic attribute of sophisticated forms of empathy and may be neurally implemented through several types of mirror simulative mechanisms [22, 23]. In keeping with this notion, a number of brain systems with mirror properties have also been recently described in the domain of emotion [126–129] and sensory processing [50, 125, 130–134]. Thus, the MMS may be only one of the systems of our social brain that allow us to have a direct experiential knowledge about others.

Evidence supporting the idea of mirror activity in a purely sensory domain has been provided by two recent studies on "empathy for touch" [130, 131]. Blakemore et al. [131], for example, found common S1 activations with a topographic organization during the personal experience of touching stimuli delivered to the face or the neck and the observation of such stimulations in others.

According to ST, there is evidence to suggest that perception of emotion automatically activates mechanisms that are responsible for the generation of emotion [25]. For instance, viewing facial expressions triggers expressions on one's own face (as measured by electromyography), even in the absence of conscious recognition of the stimulus [135, 136]. Moreover, fMRI studies indicate that similar networks of motor and emotional brain areas are activated by the perception of emotional expressions and the overt imitation of similar emotions [126, 128, 137]. Lesion studies indicate that right frontoparietal cortex is necessary for the correct recognition of emotions from prosody [25, 138].

Somatosensory-related structures are also crucial for emotion recognition [139–142]. These areas may become active in tasks involving facial expression judgment [141]. Importantly, lesions to somatosensory areas in brain-damaged patients [25, 139], or interference with the activity of these structures obtained by means of magnetic brain stimulation [140], impairs facial emotion recognition. According to ST, during the recognition of another's emotion, specific sensorimotor structures could provide a somatic description of the experience derived from actually feeling the same emotion. This may help us to learn about others' emotional states [25, 140, 142].

Some direct evidence of a sharing of emotional representations between self and others comes from studies on the emotion of disgust. Calder et al. [143] reported the case of the patient N.K., with left IC and putamen damage, who was selectively impaired in detecting social signals of disgust from facial expressions, nonverbal sounds, and emotional prosody. Interestingly, this perceptual deficit for disgust expressions was mirrored by an equivalent deficit in the phenomenological experience of the same emotion. Indeed, patient N.K. was less disgusted than controls by disgust-provoking scenes. The involvement of insula in the recognition of disgust has also been supported in healthy subjects using fMRI [144]. Consistent with these findings is the fMRI study showing that viewing another person's facial emotional reactions to unpleasant odorants activates sectors of the AI and ACC that are also activated when the subject himself inhales the same unpleasant odorants [127].

Empathic Mirroring of Others' Pain

As mentioned in the Introduction, various painful personal experiences are represented in a complex neural network referred to as the "pain matrix" [6, 7, 8, 9]. Affectively distressing components (such as unpleasantness) and sensory components (such as localization and intensity) of painful stimuli are encoded in the affective and sensorimotor node of the pain matrix, respectively. The presence of distinct sensory and affective components makes pain a particularly interesting model for testing simulative theories of empathy based on the notion of shared neural representations. Undeniably, the empathic sharing of pain representations may hold a special status in the domain of empathic processes. On the one hand, sharing affective representations of pain (distress, unpleasantness) may represent most directly a predicate for sophisticated forms of empathy, i.e., helping or altruistic behavior and ethical and moral reasoning [75, 122, 125]. On the other hand, sharing sensorimotor representations of pain may imply that others' pain is mapped onto one's own body. This may be crucial for social learning of protective behaviors and defense reactions to potentially damaging situations [133, 145].

In this section we present findings that support the notion of shared neural representations between self and others in the domain of pain processing. According to current neuroscientific theories of empathy [22- 27], the basic simulative mirror mechanisms described in the domain of action, touch, and emotion may also be at the root of our ability to understand and empathize with the pain of others. The possibility that the human ability to recognize the pain of others is grounded in a mirror-like pain system is suggested by the anecdotal report of a patient suffering from an unusual form of allodynia (a pathological condition in which nonnoxious touching stimuli are perceived as painful) [145]. This patient apparently experienced observed pain as pain in himself. For example, when the patient's wife experienced a sudden minor injury (e.g., knocking her hand against a table), he would become very agitated, claiming that it hurt him to witness such accidents. If she merely commented that she had knocked her fingers, there was no such reaction. Although the report was anecdotal and no information about the neural circuitry involved in this type of phenomena was provided, the results have been attributed to an abnormal "mirror pain" system [145]. More direct evidence of "pain mirror neurons" comes from neurophysiology data on pain-related processing in human neurosurgical patients [55]. Using microelectrodes, Hutchison et al. [55] found several nociceptive neurons in the ACC, including cells that discharged preferentially to mechanical noxious stimuli. In that study it was noticed that a neuron responded selectively to the anticipation and delivery of noxious mechanical stimulation (pinching, pinpricks) applied to the patient's hand. Interestingly, this cell also responded during the observation of the experimenter receiving pinpricks in the hand [55].

Recent fMRI studies show that only affective components of the pain matrix are crucial for empathy for pain, thus suggesting that only emotional representations of pain are shared between self and others [50, 125, 132, 134]. In a first fMRI study by Singer et al. [50], empathy for pain was induced by means of arbitrary visual cues signaling an impending painful stimulus to the participant's romantic partner. Empathy for pain brought about an increase of fMRI signal in AI and ACC cortices, which are part of the affective division of the pain matrix. Importantly, neural activity correlated with the subjects' emotional empathy traits scores.

Neural activity in the affective pain network was also reported in fMRI studies involving unknown human models, where subjects observed pictures [132] or movies [125] in which potentially painful stimuli were delivered to hands or other human body parts, or movies depicting facial expressions of pain [134].

Despite some activations in structures that may be involved in somatic processing, such as the thalamus, brainstem, parietal cortex, and cerebellum, found in studies when participants imaged others' pain [50], watched facial pain-related behavior [134], or observed potentially painful situations [132], the authors concluded that only the affective division of the pain matrix is crucial for empathy for pain.

The Sensorimotor Side of Empathy for Pain

The ability to understand and to experience indirectly the pain of others may be fundamental to social cohesion [24, 50, 75]. Previous fMRI studies on empathy for pain indicate that perceiving pain in others mainly involves cerebral regions known to play an important role in the affective experience of pain [50, 125, 132, 134]. This "affective resonance" may be at the base of complex forms of empathy [50], e.g., emotional concern, piety, and altruistic behavior.[3]

Do we share with others only emotional representations of pain? From a developmental and evolutionary perspective, having a detailed repre-

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[3] The study of Singer et al. [50] may reflect more directly the activity of such sophisticated forms of empathy. In this study neural activity in ACC and AI correlated positively with two personality questionnaires assessing the subjects' emotional empathy trait (e.g., the tendency to experience feelings of concern and piety in response to others in distress, and the tendency to desire to help others). In the other fMRI studies involving unknown human models [125, 132, 134], activity in the affective division of the pain matrix may imply a more simple mechanism of mirroring others' unpleasant emotional state. For instance, in the study by Jackson et al. [132], neural activity in ACC correlated with the level of the pain ascribed to the model, but not with the same personality questionnaire adopted by Singer et al. [50]. Interestingly, both high and basic levels of empathic emotional mechanism are mapped in the same emotional neural structures (ACC and AI).

sentation of the source and nature of others' pain may be crucial for survival. Some authors have speculated that, during infancy, avoidance of noxious stimuli may be facilitated by early recognition of others' pain [145]. We posit that mirror mechanisms that map detailed sensory representation of others' pain (e.g., locus and intensity of a noxious stimulus) onto one's own body may be fundamental for the social learning of escape or avoidance reactions to noxious stimuli.

Why has previous fMRI on empathy for pain failed to find specific somatic activations? The simplest explanation[4] is that previous studies may have adopted nonbiological relevant visual stimuli for evoking pain body-mapping, such as static pictures of potentially painful situations [132], very superficial injections in the hands [125], or stimuli in which the body was not directly shown [50, 134].

One may speculate that body mapping of others' pain may occur especially when the visual scene is of functional relevance for the individual (e.g., when the stimuli are shocking or very intense). Thus, in a recent study we presented subjects with "flesh and bone" painful stimulations shown on the body of a human model [133]. We used TMS[5] to record changes in corticospinal motor representations of the hand muscles of individuals observing needles penetrating the hands or feet of a human model or noncorporeal objects (Fig. 1a). Videos depicting static hands and feet and Q-tips touching the same body parts were used as control stimuli. We found a reduction in amplitude of MEPs that was specific to the hand muscle that subjects observed being deeply pricked (Fig. 1b). No inhibition of the muscles of the hand was found during observation of harmless touching stimuli or needles in feet or noncorporeal objects. Importantly, this motor inhibition was clearly related to the observer's subjective empathetic rating of the sensory, but not affective, qualities of the pain ascribed to the model. In other words, the largest motor inhibition was found in the participants who evaluated as most intense the model's pain [133] (Fig. 1c).

We interpreted the observational pain-related motor inhibition as reflecting the activity of a simulative mirror mechanism that extracts

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[4] For an alternative hypothesis see Singer and Frith [149].

[5] As reported above, previous TMS studies indicate that actual nociceptive stimulations bring about a decrease in excitability of the corticospinal motor system [5, 88, 89, 90, 91]. That is, pain reduces the amplitude of the MEP induced by TMS. This motor inhibition may represent the corticospinal correlate of a defensive withdrawal reaction.

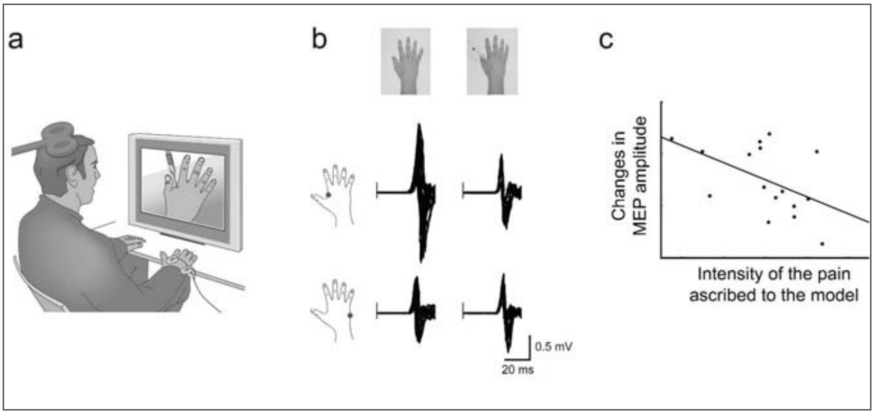

Fig. 1. a A magnetic pulse was delivered over the left primary motor cortex during the observation of different types of video-clips. Motor-evoked potentials (*MEPs*) induced by transcranial magnetic stimulation (*TMS*) were recorded from the first dorsal interosseus (*FDI*, in the region of the index finger) and the abductor digiti minimi (in the region of the little finger) of the observer's right hand. **b** Examples of MEPs recorded from the FDI (*top*) and the ADM (*bottom*) muscles during the observation of the model's static hand (*right*) and of a needle deeply penetrating the FDI region of the model's hand. Note the specific reduction of amplitude of MEPs recorded from FDI muscle during the observation of a syringe penetrating the model's FDI region. **c** Amplitude changes of MEPs recorded from the FDI during the observation of pain with respect to the static hand correlated with the intensity of the pain ascribed to the model

basic sensory qualities of the model's painful experience (location and intensity of the noxious stimulus) and maps them onto the observer's motor system according to topographical rules [133].[6] This hypothesis was strongly supported by the muscle specificity and by the link between MEP inhibition and the intensity of the pain attributed to the model.

Motor responses to one's own pain allow freezing or escape reactions, and ultimately survival. The observational pain-related motor inhibition indicates that similar motor responses occur as a result of observation of "flesh and bone" painful events in others [133].

It is worth noting that neuroimaging studies indicate that anticipation

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[6] Alternative interpretations, such as the shift of attention to the target body part, or the predictive motor imitation of the model's behaviour, were not likely based on the proprieties of the neurophysiological results (inhibition rather than facilitation, and muscle-specificity). A discussion of these alternative hypotheses is provided by Avenanti et al. [133].

of painful stimuli being administered to one's own body increases the hemodynamic signal in several regions of the pain matrix [46, 87, 146, 147]. These activations triggered by pain anticipation may also include somatotopical organized spots in the primary sensorimotor cortices (M1, S1) [86, 148]. According to shared representation models [24, 27], it is possible that the simulative mirror responses triggered by the observation of "flesh and bone" painful stimulations in others may reflect anticipation of pain in oneself [132, 133, 149]. The selective embodiment of others' pain in the observer's corticospinal system, sensitively more than emotionally denoted, may thus be crucial for the social learning of reactions to painful stimuli in that it may help the observer's corticospinal system to implement specific escape or freezing reactions before painful stimuli are actually experienced [133].

By means of neurophysiological techniques such as somatosensory-evoked potentials (SEPs) and laser-evoked potentials (LEPs) (that allow direct testing of activity within S1 and S2, respectively), we recently found support to the idea of pain sensorimotor representation sharing [150, 151]. In those studies, we found that some brain potentials evoked by somatosensory [151] and nociceptive stimulations [150] that originated from somatosensory cortices were selectively modulated by the observation of "flesh and bone" painful stimulations in others. In keeping with our TMS study, such potentials were highly linked with sensory, but not affective, components of pain. Preliminary fMRI data from our laboratories indicate that premotor and multisensory parietal structures may participate in such a mapping of sensory components of others' pain.

Conclusions

In humans, empathy for pain may rely not only on affective-motivational [50, 125, 132, 134] but also on fine-grained somatic representations [133]. This supports the notion that empathy is based on different types of sensory, motor, and emotional simulative mechanisms [22–24, 27].

It may be possible to think of at least two forms of empathy linked to one another in an evolutionary and developmental perspective. A comparatively simple form of empathy, based on somatic resonance, may be primarily concerned with mapping external stimuli onto one's own body [133]. This mapping may be important for learning of reactions to pain [133, 145]. A more complex form of empathy, based on affective resonance, may deal with emotional sharing [50, 125, 132, 134] and with the evaluation of social bonds and interpersonal relations [50].

All in all, studies on empathy for pain indicate that the affective and sensorimotor divisions of the pain matrix are important nodes in the complex neural network recruited not only during the personal experience of pain [5–9, 50, 84–91] but also during empathy for others' pain [50, 125, 132–134].

A direct matching of specific sensory aspects of others' pain occurs in sensorimotor structures of the pain matrix [133], whereas emotional components of others' painful experiences (along with other-oriented compassion feelings) are coded in the affective division of the network [50, 125, 132, 134]. Such a sensorimotor and affective mapping of others' pain components may allow us to have a direct experiential empathic understanding of others' painful experiences that does not necessitate any explicit reflective mediation. In order to understand others, we use our internal body representations.

Hence, empathy for pain may take different forms in different nodes of the complex neural network that represent sensations, feelings, and emotions linked to the experience of pain. Philosophers have emphasized that our bodily sensations are intrinsically private [72, 73]. However, cognitive neuroscience suggests that, at least in humans, the social dimension of pain extends even to the very basic, sensorimotor levels of neural processing.

References

1. Fernandez E, Turk DC (1992) Sensory and affective components of pain: separation and synthesis. Psychol Bull 112:205–217
2. Melzack R, Casey KL (1968) Sensory, motivational and central control determinants of pain: a new conceptual model. In: Kenshalo DR (ed) The skin senses. Thomas, Springfield, IL, USA, pp 423–443
3. Price DD, Harkins SW, Baker C (1987) Sensory-affective relationships among different types of clinical and experimental pain. Pain 28:297–308
4. International Association for the Study of Pain Task Force on Taxonomy (1994) Classification of chronic pain: descriptions of chronic pain syndromes and definitions of pain terms. IASP Press, Seattle, WA, USA
5. Farina S, Tinazzi M, Le Pera D, Valeriani M (2003) Pain-related modulation of the human motor cortex. Neurol Res 25:130–142
6. Ingvar M (1999) Pain and functional imaging. Philos Trans R Soc Lond Biol 354:1347–1358
7. Rainville P (2002) Brain mechanisms of pain affect and pain modulation. Curr Opin

Neurobiol 12:195–204

8. Peyron R, Laurent B, Garcia-Larrea L (2000) Functional imaging of brain responses to pain: a review and meta-analysis. Neurophysiol Clin 30:263–288
9. Derbyshire SWG (2000) Exploring the pain neuromatrix. Curr Rev Pain 6:467–477
10. Lipps T (1903) Einfühlung, innere nachahmung und organenempfindung. Arch Gesamte Psychol 1:465–519
11. Beres D, Arlow JA (1974) Fantasy and identification in empathy. Psychoanal Q 43:26–50
12. Basch MF (1983) Empathic understanding: a review of the concept and some theoretical considerations. J Am Psychoanal Assoc 31:101–126
13. Bohart A, Greenberg LS (1997) Empathy reconsidered. American Psychological Association, Washington, DC
14. Freud S (1905) Jokes and their relation to the unconscious (SE 8). Hogarth, London
15. Kohut H (1959) Introspection, empathy, and psychoanalysis: an examination of the relationship between mode of observation and theory. In: Ornstein PH (ed) The search for the self, vol 1. International University Press, New York, pp 205–232
16. Kohut H (1984) How does analysis cure? University of Chicago Press, Chicago
17. Goldman AI (1992) In defense of the simulation theory. Mind Lang 7:104–119
18. Gordon RM (1986) Folk psychology as simulation. Mind Lang 1:158–171
19. Gordon RM (1992) The simulation theory: objections and misconceptions. Mind Lang 7:11–34
20. Carruthers P, Smith PK (1996) Theories of theories of mind. Cambridge University Press, Cambridge, UK
21. Gallese V, Goldman A (1998) Mirror neurons and the simulation theory of mindreading. Trends Cogn Sci 12:493–501
22. Gallese V (2001) The ‘shared manifold’ hypothesis: from mirror neurons to empathy. J Consc Studies 8:33–50
23. Gallese V (2003) The manifold nature of interpersonal relations: the quest for a common mechanism. Philos Trans R Soc Lond Biol 358:517–528
24. Preston SD, de Waal FBM (2002) Empathy: its ultimate and proximate bases. Behav Brain Sci 25:1–71
25. Adolphs R (2003) Cognitive neuroscience of human social behaviour. Nat Rev Neurosci 4:165–178
26. Gallese V, Keyser C, Rizzolatti G (2004) A unifying view of the basis of social cognition. Trends Cogn Sci 8:396–403
27. Decety J, Jackson PL (2004) The functional architecture of human empathy. Behav Cogn Neurosci Rev 3:71–100
28. Friedman DP, Murray EA (1986) Thalamic connectivity of the second somatosensory area and neighboring somatosensory fields of the lateral sulcus of the macaque. J Comp Neurol 252:348–374
29. Rausell E, Jones EG (1991) Chemically distinct compartments of the thalamic VPM nucleus in monkeys relay principal and spinal trigeminal pathways to different layers of the somatosensory cortex. J Neurosci 11:226–237
30. Shi T, Apkarian AV (1995) Morphology of thalamocortical neurons projecting to the primary somatosensory cortex and their relationship to spinothalamic terminals in the squirrel monkey. J Comp Neurol 361:1–24
31. Kenshalo DR Jr, Isensee O (1983) Responses of primate SI cortical neurons to noxious stimuli. J Neurophysiol 50:1479–1496
32. Chudler EH, Anton F, Dubner R, Kenshalo DR Jr (1990) Responses of nociceptive SI neurons in monkeys and pain sensation in humans elicited by noxious thermal

stimulation: effect of interstimulus interval. J Neurophysiol 63:559–569

33. Craig AD, Bushnell MC, Zhang ET, Blomqvist A (1994) A thalamic nucleus specific for pain and temperature sensation. Nature 372:770–773
34. Dong WK, Chudler EH, Sugiyama K et al (1994) Somatosensory, multisensory, and task-related neurons in cortical area 7b (PF) of unanesthetized monkeys. J Neurophysiol 72:542–564
35. Andersson JL, Lilja A, Hartvig P et al (1997) Somatotopic organization along the central sulcus, for pain localization in humans, as revealed by positron emission tomography. Exp Brain Res 117:192–199
36. Porro CA, Cettolo V, Francescato MP, Baraldi P (1998) Temporal and intensity coding of pain in human cortex. J Neurophysiol 80:3312–3320
37. Bushnell MC, Duncan GH, Hofbauer RK et al (1999) Pain perception: is there a role for primary somatosensory cortex? Proc Natl Acad Sci USA 96:7705–7709
38. Hofbauer RK, Rainville P, Duncan GH, Bushnell MC (2001) Cortical representation of the sensory dimension of pain. J Neurophysiol 86:402–411
39. Bingel U, Lorenz J, Glauche V et al (2004) Somatotopic organization of human somatosensory cortices for pain: a single trial fMRI study. Neuroimage 23:224–232
40. Greenspan JD, Lee RR, Lenz FA (1999) Pain sensitivity alterations as a function of lesion location in the parasylvian cortex. Pain 81:273–282
41. Ploner M, Freund HJ, Schnitzler A (1999) Pain affect without pain sensation in a patient with a postcentral lesion. Pain 81:211–214
42. Coghill RC, Talbot JD, Evans AC et al (1994) Distributed processing of pain and vibration by the human brain. J Neurosci 14:4095–4108
43. Craig AD, Reiman EM, Evans AC, Bushnell MC (1996) Functional imaging of an illusion of pain. Nature 384:258–260
44. Davis KD, Taylor SJ, Crawley AP et al (1997) Functional MRI of pain- and attention-related activations in the human cingulate cortex. J Neurophysiol 77:3370–3380
45. Jones AK, Brown WD, Friston KJ et al (1991) Cortical and subcortical localization of response to pain in man using positron emission tomography. Proc R Soc Lond B Biol Sci 244:39–44
46. Ploghaus A, Tracey I, Gati JS et al (1999) Dissociating pain from its anticipation in the human brain. Science 284:1979–1981
47. Talbot JD, Marrett S, Evans AC et al (1991) Multiple representations of pain in human cerebral cortex. Science 251:1355–1358
48. Vogt BA, Derbyshire S, Jones AK (1996) Pain processing in four regions of human cingulate cortex localized with co-registered PET and MR imaging. Eur J Neurosci 8:1461–1473
49. Rainville P, Duncan GH, Price DD et al (1997) Pain affect encoded in human anterior cingulate but not somatosensory cortex. Science 277:968–971
50. Singer T, Seymour B, O'Doherty J et al (2004) Empathy for pain involves the affective but not sensory components of pain. Science 303:1157–1162
51. Papez JW (1937) A proposed mechanism of emotion. Arch Neurol Psychiatry 38:725–743
52. MacLean PD (1949) Psychosomatic disease and the "visceral brain": recent developments bearing on the Papez theory of emotion. Psychosom Med 11:338–353
53. Apkarian AV, Shi T (1998) Thalamocortical connections of the cingulate and insula in relation to nociceptive inputs to the cortex. In: Ayrapetyan SN, Apkarian AV (eds) Pain mechanisms and management. IOS, Washington, DC, pp 212–220
54. Craig AD (2003) Pain mechanisms: labeled lines versus convergence in central processing. Annu Rev Neurosci 26:1–30

55. Hutchison WD, Davis KD, Lozano AM et al (1999) Pain-related neurons in the human cingulate cortex. Nat Neurosci 2:403–405
56. Lenz FA, Rios M, Zirh A et al (1998) Painful stimuli evoke potentials recorded over the human anterior cingulate gyrus. J Neurophysiol 79:2231–2234
57. Koyama T, Tanaka YZ, Mikami A (1998) Nociceptive neurons in the macaque anterior cingulate activate during anticipation of pain. Neuroreport 9:2663–2667
58. Sikes RW, Vogt BA (1992) Nociceptive neurons in area 24 of rabbit cingulate cortex. J Neurophysiol 68:1720–1732
59. Tolle TR, Kaufmann T, Siessmeier T et al (1999) Region-specific encoding of sensory and affective components of pain in the human brain: a positron emission tomography correlation analysis. Ann Neurol 45:40–47
60. Lieberman MD, Jarcho JM, Berman S et al. (2004) The neural correlates of placebo effects: a disruption account. Neuroimage 22:447–455
61. Coghill RC, McHaffie JG, Yen YF (2003) Neural correlates of interindividual differences in the subjective experience of pain. Proc Natl Acad Sci USA 100:8538–8542
62. Augustine JR (1985) The insular lobe in primates including humans. Neurol Res 7:2–10
63. Augustine JR (1996) Circuitry and functional aspects of the insular lobe in primates including humans. Brain Res Brain Res Rev 22:229–244
64. Craig AD, Chen K, Bandy D, Reiman EM (2000) Thermosensory activation of insular cortex. Nat Neurosci 3:184–190
65. Critchley HD, Wiens S, Rotshtein P et al (2004) Neural systems supporting interoceptive awareness. Nat Neurosci 7:189–195
66. Berthier M, Starkstein S, Leiguarda R (1988) Asymbolia for pain: a sensory-limbic disconnection syndrome. Ann Neurol 24:41–49
67. Flor H (2002) Phantom-limb pain: characteristics, causes, and treatment. Lancet Neurol 1:182–189
68. Panksepp J (1998) Affective neuroscience: the foundations of human and animal emotions. Oxford University Press, New York
69. Eisenberger NI, Lieberman MD (2004) Why rejection hurts: a common neural alarm system for physical and social pain. Trends Cogn Sci 8:294–300
70. Eisenberger NI, Lieberman MD, Williams KD (2003) Does rejection hurt? An fMRI study of social exclusion. Science 302:290–292
71. Osaka N, Osaka M, Morishita M et al (2004) A word expressing affective pain activates the anterior cingulate cortex in the human brain: an fMRI study. Behav Brain Res 153:123–127
72. Descartes R (1972) Traité de l’homme. In: Hall TS (trans) Treatise of man, Harvard University Press, Cambridge, MA, USA
73. Wittgenstein L (1963) Philosophical Investigations. Blackwell, Oxford, UK
74. Inghilleri M, Cruccu G, Argenta M, Polidori L, Manfredi M (1997) Silent period in upper limb muscles after noxious cutaneous stimulation in man. Electroencephalogr Clin Neurophysiol 105:109–115
75. Williams AC (2002) Facial expression of pain: an evolutionary account. Behav Brain Sci 25:439–488
76. Lundt JP, Stholer CS, Widmer CG (1993) The relationship between pain and muscle activity in fibromyalgia and similar conditions. In: Vaeroy H, Merskey H (eds) Progress in fibromyalgia and myofascial pain. Elsevier, Amsterdam, pp 311–327
77. Schwoebel J, Friedman R, Duda N, Coslett HB (2001) Pain and the body schema: evidence for peripheral effects on mental representations of movement. Brain 124:2098–2104

78. Schwoebel J, Coslett HB, Bradt J et al (2002) Pain and the body schema: effects of pain severity on mental representation of movement. Neurology 59:775–777
79. Tsubokawa T, Katayama Y, Yamamoto T et al (1991) Chronic motor cortex stimulation for the treatment of central pain. Acta Neurochir 52:137–139
80. Saitoh Y, Shibata M, Sanada Y, Mashimo T (1999) Motor cortex stimulation for phantom limb pain. Lancet 353, 212
81. Drouot X, Nguyen J-P, Peschanski M, Lefaucheur J-P (2002) The antalgic efficacy of chronic motor cortex stimulation is related to sensory changes in the painful zone. Brain 125:1660–1664
82. Lefaucheur JP, Drouot X, Nguyen JP (2001) Interventional neurophysiology for pain control: duration of pain relief following repetitive transcranial magnetic stimulation of the motor cortex. Neurophysiol Clin 31:247–252
83. Flor H, Elbert T, Knecht S et al (1995) Phantom-limb pain as a perceptual correlate of cortical reorganization following arm amputation. Nature 375:482–484
84. Casey KL, Minoshima S, Morrow TJ, Koeppe RA (1996) Comparison of human cerebral activation pattern during cutaneous warmth, heat pain, and deep cold pain. J Neurophysiol 76:571–81
85. Wager TD, Rilling JK, Smith EE et al (2004) Placebo-induced changes in fMRI in the anticipation and experience of pain. Science 303:1162–1167
86. Porro CA, Cettolo V, Francescato MP, Baraldi P (2003) Functional activity mapping of the mesial hemispheric wall during anticipation of pain. Neuroimage 19:1738–1747
87. Ploghaus A, Becerra L, Borras C, Borsook D (2003) Neural circuitry underlying pain modulation: expectation, hypnosis, placebo. Trends Cogn Sci 7:197–200
88. Farina S, Valeriani M, Rosso T et al (2001) Transient inhibition of the human motor cortex by capsaicin-induced pain: a study with transcranial magnetic stimulation. Neurosci Lett 314:97–101
89. Le Pera D, Graven-Nielsen T, Valeriani M et al (2001) Inhibition of motor system excitability at cortical and spinal level by tonic muscle pain. Clin Neurophysiol 112:1633–1641
90. Svensson P, Miles TS, McKay D, Ridding MC (2003) Suppression of motor evoked potentials in a hand muscle following prolonged painful stimulation. Eur J Pain 7:55–62
91. Urban PP, Solinski M, Best C et al (2004) Different short-term modulation of cortical motor output to distal and proximal upper-limb muscles during painful sensory nerve stimulation. Muscle Nerve 29:663–669
92. Georgopoulos AP (2000) Neural aspects of cognitive motor control. Curr Opin Neurobiol 10:238–241
93. Jeannerod M (1997) The cognitive neuroscience of action. Blackwell, Oxford, UK
94. Kosslyn SM, Ganis G, Thompson WL (2001) Neural foundation of imagery. Nat Rev Neurosci 2:635–642
95. Di Pellegrino G, Fadiga L, Fogassi L et al (1992) Understanding motor events: a neurophysiological study. Exp Brain Res 91:176–180
96. Gallese V, Fadiga L, Fogassi L, Rizzolatti G (1996) Action recognition in the premotor cortex. Brain 119:593–609
97. Fogassi L, Ferrari PF, Gesierich B et al (2005) Parietal lobe: from action organization to intention understanding. Science 308:662–667
98. Fadiga L, Fogassi L, Pavesi G, Rizzolatti G (1995) Motor facilitation during action observation: a magnetic stimulation study. J Neurophysiol 73:2608–2611
99. Fadiga L, Craighero L, Olivier E (2005) Human motor cortex excitability during the

perception of others' action. Curr Opin Neurobiol 15:213–218

100. Brass M, Bekkering H, Prinz W (2001) Movement observation affects movement execution in a simple response task. Acta Psychol 106:3–22
101. Craighero L, Bello A, Fadiga L, Rizzolatti G (2002) Hand action preparation influences the responses to hand pictures. Neuropsychologia 40:492–502
102. Grafton ST, Arbib MA, Fadiga L, Rizzolatti G (1996) Localization of grasp representation in humans by positron emission tomography. 2. Observation compared with imagination. Exp Brain Res 112:103–111
103. Rizzolatti G, Fadiga L, Matelli M et al (1996) Localization of grasp representation in humans by PET. 1. Observation versus execution. Exp Brain Res 111:246–252
104. Grèzes J, Costes N, Decety J (1998) Top-down effect of strategy on the perception of human biological motion: a PET investigation. Cogn Neuropsychol 15:553–582
105. Grèzes J, Costes N, Decety J (1999) The effects of learning and intention on the neural network involved in the perception of meaningless actions. Brain 122:1875–1887
106. Hari R, Forss N, Avikainen S et al (1998) Activation of human primary motor cortex during action observation: a neuromagnetic study. Proc Natl Acad Sci USA 95:15061–15065
107. Iacoboni M, Woods RP, Brass M et al (1999) Cortical mechanisms of human imitation. Science 286:2526–2528
108. Cochin S, Barthelemy C, Roux S, Martineau J (1999) Observation and execution of movement: similarities demonstrated by quantified electroencephalography. Eur J Neurosci 11:1839–1842
109. Grèzes J, Decety J (2001) Functional anatomy of execution, mental simulation, observation, and verb generation of actions: a meta-analysis. Hum Brain Mapp 12:1–19
110. Calvo-Merino B, Glaser DE, Grèzes J et al (2005) Action observation and acquired motor skills: an fMRI study with expert dancers. Cereb Cortex 15:1243–1249
111. Costantini M, Galati G, Ferretti A et al (2005) Neural systems underlying observation of humanly impossible movements: an fMRI study. Cereb Cortex 15:1761–1767
112. Buccino G, Binkofski F, Fink GR et al (2001) Action observation activates premotor and parietal areas in a somatotopic manner: an fMRI study. Eur J Neurosci 13:400–404
113. Jeannerod M (1994) The representing brain: neural correlates of motor intention and imagery. Behav Brain Sci 17:187–245
114. Jeannerod M (2001) Neural simulation of action: a unifying mechanism for motor cognition. Neuroimage 14:S103–S109
115. Decety J, Grèzes J (1999) Neural mechanisms subserving the perception of human actions. Trends Cogn Sci 3:172–178
116. Rizzolatti G, Fadiga L, Gallese V, Fogassi L (1996) Premotor cortex and the recognition of motor actions. Cogn Brain Res 3:131–141
117. Rizzolatti G, Fogassi L, Gallese V (2001) Neurophysiological mechanisms underlying the understanding and imitation of action. Nat Rev Neurosci 2:661–670
118. Rizzolatti G, Craighero L (2004) The mirror-neuron system. Annu Rev Neurosci 27:169–192
119. Blakemore SJ, Decety J (2001) From the perception of action to the understanding of intention. Nat Rev Neurosci 2:561–567
120. Grèzes J, Frith CD, Passingham RE (2004) Inferring false beliefs from the actions of oneself and others: an fMRI study. Neuroimage 21:744–750
121. Iacoboni M, Molnar-Szakacs I, Gallese V et al (2005) Grasping the intentions of others with one's own mirror neuron system. PLoS Biol 3:529–535

122. Thomson E (2001) Empathy and consciousness. J Consc Stud 8:1–32
123. Meltzoff AN, Decety J (2003) What imitation tells us about social cognition: a rapprochement between developmental psychology and cognitive neuroscience. Philos Trans R Soc Lond B Biol Sci 358:491–500
124. Decety J, Sommerville JA (2003) Shared representations between self and other: a social cognitive neuroscience view. Trends Cogn Sci 7:527–533
125. Morrison I, Lloyd D, di Pellegrino G, Roberts N (2004) Vicarious responses to pain in anterior cingulate cortex: is empathy a multisensory issue? Cogn Affect Behav Neurosci 4:270–278
126. Carr L, Iacoboni M, Dubeau MC et al (2003) Neural mechanisms of empathy in humans: a relay from neural systems for imitation to limbic areas. Proc Natl Acad Sci USA 100:5497–5502
127. Wicker B, Keysers C, Plailly J et al (2003) Both of us disgusted in *my* insula: the common neural basis of seeing and feeling disgust. Neuron 40:655–664
128. Leslie KR, Johnson-Frey SH, Grafton ST (2004) Functional imaging of face and hand imitation: towards a motor theory of empathy. Neuroimage 21:601–607
129. Hennenlotter A, Schroeder U, Erhard P et al (2005) A common neural basis for receptive and expressive communication of pleasant facial affect. Neuroimage 26:581–591
130. Keysers C, Wicker B, Gazzola V et al (2004) A touching sight: SII/PV activation during the observation and experience of touch. Neuron 42:335–346
131. Blakemore SJ, Bristow D, Bird G et al , Frith C, Ward J (2005) Somatosensory activations during the observation of touch and a case of vision–touch synaesthesia. Brain 128:1571–1583
132. Jackson PL, Meltzoff AN, Decety J (2005) How do we perceive the pain of others? A window into the neural processes involved in empathy. Neuroimage 24:771–779
133. Avenanti A, Bueti D, Galati G, Aglioti SM (2005) Transcranial magnetic stimulation highlights the sensorimotor side of empathy for pain. Nat Neurosci 8:955–960
134. Botvinick M, Jha AP, Bylsma LM et al , Fabian SA, Solomon PE, Prkachin KM (2005) Viewing facial expressions of pain engages cortical areas involved in the direct experience of pain. Neuroimage 25:312–319
135. Dimberg U, Thunberg M, Elmehed K (2000) Unconscious facial reactions to emotional facial expressions. Psychol Sci 11:86–89
136. Wallbott HG (1991) Recognition of emotion from facial expression via imitation? Some indirect evidence for an old theory. Br J Soc Psychol 30:207–219
137. Ekman P, Davidson RJ (1993) Voluntary smiling changes regional brain activity. Psychol Sci 4:342–345
138. Adolphs R, Damasio H, Tranel D (2002) Neural systems for recognition of emotional prosody: a 3-D lesion study. Emotion 2:23–51
139. Adolphs R, Damasio H, Tranel D et al (2000) A role for the somatosensory cortices in the visual recognition of emotion as revealed by three-dimensional lesion mapping. J Neurosci 20:2683–2690
140. Pourtois G, Sander D, Andres M et al (2004) Dissociable roles of the human somatosensory and superior temporal cortices for processing social face signals. Eur J Neurosci 20:3507–3515
141. Winston JS, O'Doherty J, Dolan RJ (2003) Common and distinct neural responses during direct and incidental processing of multiple facial emotions. Neuroimage 20:84–97
142. Adolphs R (2002) Neural systems for recognizing emotion. Curr Opin Neurobiol 12:169–177

143. Calder AJ, Keane J, Manes F et al (2000) Impaired recognition and experience of disgust following brain injury. Nat Neurosci 3:1077–1078
144. Phillips ML, Young AW, Senior C et al (1997) A specific neural substrate for perceiving facial expressions of disgust. Nature 389:495–498
145. Bradshaw JL, Mattingley JB (2001) Allodynia: a sensory analogue of motor mirror neurons in a hyperaesthetic patient reporting instantaneous discomfort to another's perceived sudden minor injury? J Neurol Neurosurg Psychiatry 70:135–136
146. Peyron R, Garcia-Larrea L, Gregoire MC et al (1999) Haemodynamic brain responses to acute pain in humans: sensory and attentional networks. Brain 122:1765–1780
147. Sawamoto N, Honda M, Okada T et al (2000) Expectation of pain enhances responses to nonpainful somatosensory stimulation in the anterior cingulate cortex and parietal operculum/posterior insula: an event-related functional magnetic imaging study. J Neurosci 20:7438–7444
148. Porro CA, Baraldi P, Pagnoni G et al (2002) Does anticipation of pain affect cortical nociceptive systems? J Neurosci 22:3206–3214
149. Singer T, Frith C (2005) The painful side of empathy. Nat Neurosci 8:845–846
150. Valeriani M, Betti V, Le Pera D et al , Restuccia D, De Armas L, Miliucci R, Avenanti A, Aglioti SM (2006) The pain of a model in the pain system of an onlooker: a laser-evoked potentials study of empathy for pain. J Neurosci [submitted]
151. Bufalari I, Aprile T, Di Russo F et al , Avenanti A, Aglioti SM (2006) Empathy for pain in the somatosensory cortex. J Cogn Neurosci [submitted]

Chapter 10

Human Anterior Cingulate Cortex and Affective Pain Induced by Mimic Words: A Functional Magnetic Resonance Imaging Study

Naoyuki Osaka

Introduction

Cassirer [1] proposed that the development of language proceeded through three stages, which he termed the mimic, the analogical, and the symbolic. As we pass from stage to stage, we find a shift in the basic relationship between speech sound and meaning, a shift from an intrinsic and nonarbitrary relationship to an increasingly extrinsic and arbitrary relationship. The mimic stage corresponds to the onomatopoeic use of speech—the representation through speech of some acoustical event in nature by means of direct imitation. In this stage the relationship between sound and meaning is essentially intrinsic (e.g., imitation of animal sounds, like "cockadoodle"). Interestingly enough, recent findings on "mirror (mimic) neurons" in the human inferior frontal gyrus (IFG) strongly suggest that the mimic stage of our language system may originate from IFG. Recent neuroimaging studies concluded that verbal working memory's executive function (ventrolateral part; which plays a role in producing phonetic rehearsal) is located in the left IFG in normal adults [2, 3].

Conscious Experience and Sound Symbolism

Analogies among the subjective senses are not just abstract symbols but vital phenomena of our mental life. For example, speech sounds, both individually and in combination, sometimes serve in and by themselves to evoke meaning and sensory image—as if the sounds that constitute a word form part of the semantic and sensory content [4]. Although sound symbolism differs from simple processes of onomatopoesis, it plays a crucial role in the generation of onomatopoeia-words. In onomatopoesis, consonants and vowels of speech actually mimic some naturally occurring nonspeech sound, i.e., words like "buzz," "crackle," and meow." Speech sounds can convey sensory meanings, whether visual, tactile, gustatory, olfactory, or even pain sensation. Marks [4] argues that sound not only serves as a medium for conveying meanings, but the medium itself can also serve to express meanings. These expressed meanings represent the values of suprasensory attributes such as perceptual size, brightness, and implied pain. The vowel sound /a/ appears to refer to a larger object than does the vowel /i/. Children as well as adults, and native speakers of Japanese as well as native speakers of English, agreed that "mal" suggests a larger table than does "nil" [5]. Sapir [5] made nonsense syllables including different vowels and asked subjects about the size of the objects they subjectively refer to. Newman [6] replicated Sapir's experiments and confirmed the results. We can find a similar relationship between sound and implied pain in this intrinsic mimic stage.

In the present study, we took sound symbolism as an object of functional magnetic resonance imaging (fMRI) study for the first time. Thus, words have the potential to selectively stimulate specific brain areas. Using positron emission tomography (PET), Martin et al. [7] showed that generation of color words selectively activated a brain area in the ventral temporal lobe anterior to the area involved in the subjective perception of color, while generation of action words activated a region in the middle temporal gyrus anterior to the area involved in the perception of motion in humans. Osaka et al. [2] used fMRI to show that laughter modules using onomatopoeia words highly suggestive of laughter, heard by the ear, significantly activated both the extrastriate visual cortex near the inferior occipital gyrus and the premotor/supplementary motor area in the superior frontal gyrus. However, the effect of an onomatopoeia-based word expressing pain on the pain-related brain region has not as yet been explored. An emotion-based onomatopoeia (mimic) word containing a salient affective pain component plays a unique role in human communication. Onomatopoeia words could essentially be defined as sound symbolism used to describe human emotions and mental states.

Mimic Words

The role of onomatopoeia in the Japanese language is a very critical because Japanese has a very limited number of verbs (but, as previously noted, these mimic words may be effective both for native speakers of Japanese as well as for native speakers of English). One role of onomatopoeia words is to fill in the gap and provide a means for concise expression when a sufficiently descriptive verb does not exist. These words make the language very vivid and instantly conjure up images in the mind of a native Japanese speaker, thus producing a strong synaesthetic effect. Japanese is uniquely rich in this type of expression, which is frequently used in daily conversation, magazines, and newspapers, especially for headlines, because of its brevity and power to project vivid imagery [8]. A rough English equivalent would be, for example, "butterflies in the stomach." The expressions are classified into categories of different sensory and emotional expressions, such as laughter, pain, and other more cerebral states. Osaka [9] suggested onomatopoeia as a unique language for expressing "sensory qualia" in human consciousness and used multidimensional scaling based on rated subjective intensity to classify the six top pain-inducing onomatopoeia words into Euclidian space. The uniqueness of this type of expression frequently represents a peculiar Japanese way of expressing feelings and/or mentality [8]. Although there is a considerable amount of knowledge about the neural representation of subjective pain, little is known about higher cognitive brain function with regard to pain in connection with language function.

Cognitive Pain

It is known that subjective pain information involves multiple ascending pathways projecting to several brainstem and cortical regions including the limbic system [10]; however, little is known about higher cognitive aspects of unpleasantness due to affective pain. Some mimic words involve very salient pain components. Sounds of pain-related onomatopoeia can convey a subjective quality of the unpleasantness of affective pain [4].

Recent findings from neuroimaging studies on the subjective unpleasantness of pain suggest that information about implied pain is not processed in a unified fashion in any single area of the cortex. Rather, a highly cognitive sense of the unpleasantness of pain seems to be processed and generated over distributed cortical areas in which information about specific features, such as a stabbing and/or throbbing subjective pain, is processed in both the anterior cingulate cortex (ACC) of the brain, which mediates attention-related pain perception, and the lateral prefrontal cortex, which mediates the generation of unpleasant subjective pain [11]. How these features are linked to create the intrinsic unpleasantness of implied pain representations is investigated here using mimic words that are strongly characterized by implied pain. We presented the subject's ear with mimic words that are highly suggestive of stabbing, throbbing, and splitting pain to investigate whether these words activate, not the auditory cortex, but the cortex related to the subjective unpleasantness of pain perception.

We hypothesized that a language-generated affective pain realization task would be excellent for testing the higher-order pain-sensitive region involving affective pain, because pain-associated mimic words have specific sounds indicating strong implied affective pain and would demand extensive processing devoted to affective pain representation. Such neural substrates devoted to the unpleasantness of pain during the generation of mental pain images have not yet been explored. We used fMRI to measure brain activity associated with subjective pain realization through the generation of a mental image of the affective pain described by the word and compared fMRI activations between words that implied pain and control words (nonsense syllables).

Brain and Implied Pain

We employed 20 healthy college students or graduates aged 20–27 years (11 females and 9 males) as the subjects. Subjects were recruited as paid volunteers from the Psychology Department of Kyoto University. Subjects with chronic pain were excluded. Informed consent in accordance with the protocol approved by the ATR Brain Imaging Institute Review Board was obtained from all subjects for this fMRI experiment.

We obtained behavioral indices of Pain and Control conditions: Six Japanese pain-evoking mimic words were selected from the top six high frequency words (mean judgment for evoking affective pain was 92%) for generating unpleasantness due to affective pain; mimic words were "*zuki-zuki*" for throbbing pain with a pulsing sensation, "*ghan-ghan*" for splitting headache as if being continuously struck, "*kiri-kiri*" for stabbing pain with a feeling of being drilled into with something sharp, "*chiku-chiku*" for an intermittent pain akin to being struck by thorns, "*hiri-hiri*" for a lingering feeling of pain, "*zukin-zukin*" for continuous throbbing pain. These six words were selected based on previous scaling studies (n=290) using a multidimensional scaling and a method of magnitude estimation for affective pain [9]. In the psychophysical magnitude estimation task, observers were asked to assign numbers (from 1 to 100) according to the strength of their psychological impression (average evaluated value=80) [12]. We employed six nonsense syllables having no pain-related association value selected from a standard non-association table [13], which are the same as those we used as the control condition in a previous mimic words study [2]: They are "*rhini-rhini*," "*heyu-heyu*," "*sonu-sonu*," "*mena-mena*, "*runi-runi*," and "*nuhe-nuhe*," each of which consisted of a repeated series of syllables that have no meaning. We confirmed that these six nonsense syllables functioned as a valid Control condition in the previous study.

We employed a block design and introduced two conditions: Pain and Control conditions. Each subject performed four sessions: In each session all conditions were tested. In the Pain block, six onomatopoeic words were presented. Each word was presented for 2-s followed by 3-s interstimulus interval. In the Control block, six nonsense syllables, i.e., having no pain-related association, with a syllable length similar to that of the respective pain-associated words, were presented in the same time sequence as words in the Pain block. During the tasks, subjects were instructed to form unpleasant mental images of affective pain corresponding to each pain-related word stimulus, while keeping their eyes closed throughout the entire condition. In the Control condition, the subjects were just required to listen to the nonsense syllables. The order of word presentation was counterbalanced across sessions and subjects in order to avoid possible habituation of repeated word/nonword presentations. Our previous study showed that this procedure is valid for approximately four sessions [2]. During the session, subjects were asked to listen to words presented to both ears through an air-driven earphone system.

Whole brain imaging data were acquired on a 1.5 Tesla whole-body MRI scanner (Marconi Magnex Eclipse) using a head coil. Head motion was minimized with a forehead strap. For functional imaging, we used a gradient-echo echo-planar imaging sequence with the following parameters: TR=3 s, TE=55 ms, flip angle 90°, FOV=22x22 cm, and pixel matrix 64x64. Sixteen contiguous, 6-mm thick slices with a 1.2-mm gap were obtained on the axial plane for each subject. After collection of the functional images, T1-weighted images (154 slices with no gap) using a conventional spin echo pulse sequence (TR=12 ms; TE=5 ms; flip angle 8°, FOV=22x22 cm, and pixel matrix 256x256) were collected for anatomical co-registration at the same locations as the functional images.

After image construction, functional images were analyzed using SPM99 (Wellcome Department of Cognitive Neurology, London). Five initial images were discarded from the analysis to eliminate nonequilibrium effects of magnetization. All functional images were realigned to correct for head movement. We selected images with less than 1 mm movement within runs when the images were corrected. The functional images were normalized and spatially smoothed with an isotropic Gaussian filter (6-mm full width-half maximum). Low frequency noise and differences in global signal were removed. Data were modeled using a box-car function. Single subject data were analyzed with a fixed-effects model while group data were analyzed using a random effects model.

Table 1 summarizes the coordinates for significant activation relative to the control (uncorrected, $P<0.001$), the peak Z scores and the number of activated voxels. Figures 1, 2, and 3 each show activated brain areas on axial, sagittal, and coronal planes of standard glass brain images, rendered brain images, and two axial planes ($z=10$ and $z=46$) in Pain>Control conditions, respectively. An analysis of the Control>Pain condition did not show any significant activation area. We applied an uncorrected criterion because we focused on two specific regions of interest, that is, the PFC and ACC, where increased activation has been reported for similar tasks [14].

Table 1. The coordinates for significant activation relative to the control (uncorrected, $P<0.001$), the peak Z scores and the number of activated voxels. Adapted from Osaka et al. [17], reprinted with permission from Elsevier BV

Brain region	Brodmann area	Pain-Control Coordinates x	y	z	Z score	voxel
L anterior cingulate cortex	32	0	18	46	5.76	829
		-8	16	44	4.86	
L inferior frontal gyrus	45/44	-54	18	10	5.11	2304
		-54	14	2	4.89	
		-42	8	52	4.83	
L thalamus		-12	-12	10	4.50	106
L superior parietal lobule	7	-16	-66	50	4.36	30
L putamen		-18	4	14	4.22	30
R cerebellum		34	-74	-48	3.97	108
		22	-76	-50	3.66	
		18	-80	-40	3.46	
R anterior cingulate cortex	32	10	26	28	3.92	46
R putamen		14	-10	14	3.74	22
L inferior parietal lobule	40	-62	-28	30	3.65	47
		-58	-36	36	3.49	
R putamen		24	10	-6	3.58	12
R inferior frontal gyrus	47	48	14	-4	3.49	23

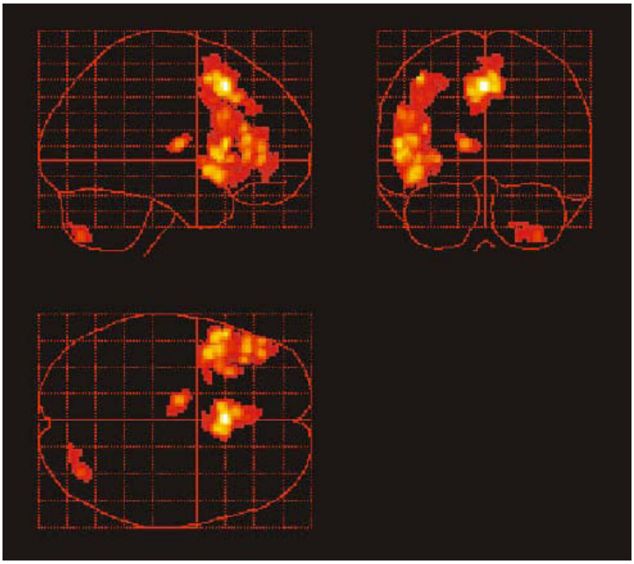

Fig. 1. Brain areas activated after hearing pain-evoking mimic words shown on axial, sagittal, and coronal planes of standard glass brain images. The figures show Pain>Control condition across 20 subjects (random effect model). Adapted from Osaka et al. [17], reprinted with permission from Elsevier BV

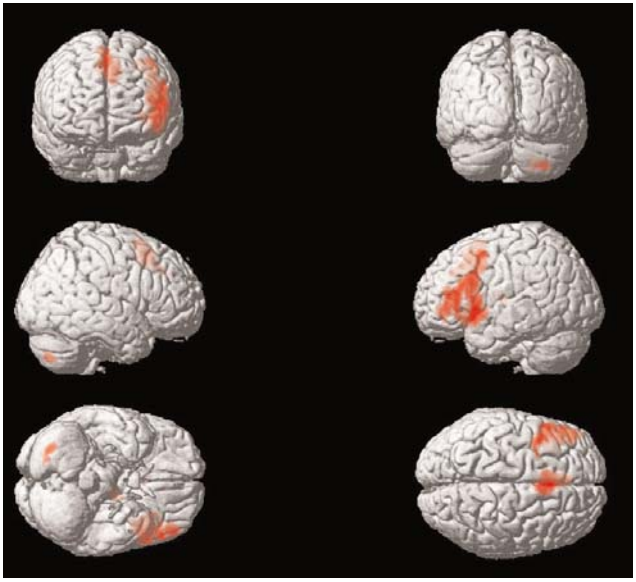

Fig. 2. Brain areas activated after hearing pain-evoking mimic words shown on rendered brain images. The figures show Pain>Control condition across 20 subjects (random effect model). *Right panel* shows front, right, and bottom view (from top to bottom), respectively. *Left panel* shows back, left, and top view (from top to bottom), respectively

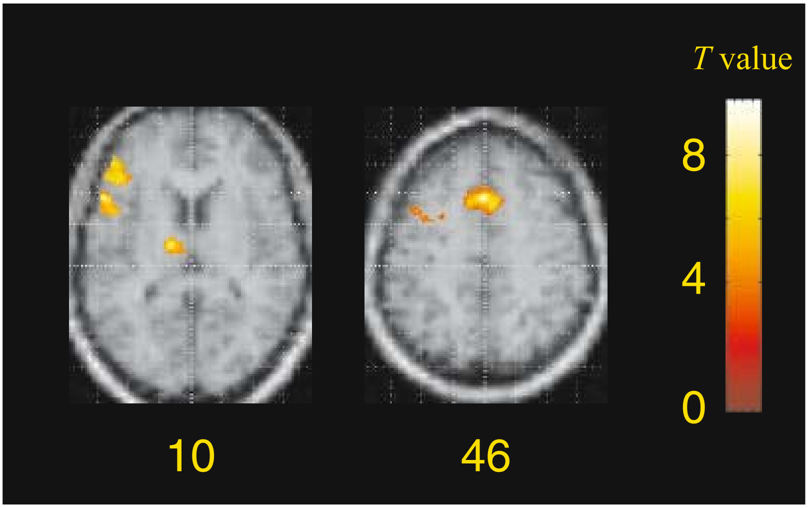

Fig. 3. Brain areas activated after hearing pain-evoking mimic words shown on two axial planes. *Right* and *left panels* show the anterior cingulate cortex (ACC) and inferior frontal gyrus (IFG) (z=10 and z=46), respectively. Adapted from Osaka et al. [17], reprinted with permission from Elsevier BV

ACC and Implied Pain

Extensive cortical areas including the left inferior frontal gyrus (BA45), ACC (BA32), left auditory area, and right superior temporal gyrus are activated in the Pain condition, while only the primary auditory cortex is activated in the Control. As shown in Figs 1, 2, and 3, we subtracted the Control from Pain and obtained significant values in the ACC (BA32: x=0, y=18, and z=46 as Talairach coordinates with a peak score of Z=5.76) and left inferior frontal gyrus (BA45: x=-54, y=18, and z=10 with Z=5.11), while there were no significant activations seen for the reverse contrast (Pain activation subtracted from Control activation). Interestingly, as shown in Table 1 and Fig. 2, both the ACC and left inferior frontal gyrus appeared closely related to imaginary pain-specific images, while the extrastriate visual cortex and superior frontal gyrus appeared related to imaginary laughter-face specific images, as we previously reported using a similar onomatopoeia-induced paradigm [2].

The most interesting result obtained was activation in the ACC (BA32). The ACC is an anterior part of the limbic system that includes specific processing modules for sensory and nociceptive information. These include a dorsal cognitive division and a rostal-ventral affective division and the former maintain strong reciprocal interconnections with the lateral prefrontal cortex [15, 16]. The role of ACC has not yet been investigated in detail; however, recent fMRI studies have shown that the cognitive area is activated under cognitive conflicts, such as the Stroop color test [15], and when performing high-load dual-task during a working memory task in which central executive attentional control is required [2, 17]. The ACC is part of a circuit involved in a form of attention switching that serves to control both cognitive and emotional processing involved in the perception of both pain and implied pain.

Activation of the affective division has been reported when subjects anticipate pain and control attention [18, 19]. Using intracranial somatosensory event-related potential recording, Kropotov et al. [20] demonstrated the critical involvement of the ACC in the control of pain with hypnotically suggested analgesia. Using PET, Talbot et al. [21] found that painful heat causes significant activation of the contralateral ACC as well as somatosensory cortex. These findings based on physical stimuli indicate that the normal processing of painful stimuli in humans is not distributed over large areas of the cortex, but is relatively restricted to the ACC, PFC, insula, and somatosensory cortices.

As Figs 1, 2, and 3 clearly showed, onomatopoeia for which subjective pain could very easily be imaged significantly activated the cognitive division of ACC, as found for cognitive conflict and attentional control situations [15]. The reason that not the affective but the cognitive division of ACC was activated could be explained by the use of implied-pain induced by mimic words which are highly symbolic and cognitive in nature. An implied unpleasantness evoked by word-induced pain is likely to redirect capacity-limited attention to unpleasantness. Using PET, Rainville et al. [16] demonstrated "significant changes in pain-evoked activity within ACC while primary somatosensory cortex activation was unaltered." They argued that the ACC encodes pain affect. The other major prefrontal activation area (left inferior frontal gyrus, BA45) has been called the ventrolateral prefrontal cortex (VLPFC), close to Broca's area (BA44), in which the word's internal phonological loop (word rehearsal mechanism) is involved in the verbal working memory module [2]. Observation of co-activation of the VLPFC and ACC suggests a functional connectivity between the VLPFC and ACC: Increased activation in the VLPFC in the present findings most likely represents the attention-driven semantic retrieval processes in which the implied pain information recognized from the long-term episodic memory system is derived from one's previous pain experiences in the past. Thus, ACC in connection with VLPFC is likely to serve in generating imaginary pain closely regulated by the attention-controlling system. This controlling system was preferentially engaged while the subjects were required to imagine unpleasant pain based on the pain-evoking word.

Interestingly, the presentation of mimic words provoking positive laughter, instead of negative pain as here in this study, have been confirmed to activate the supplementary motor area (SMA)/ACC and thereafter the striatal reward complex involving the putamen, caudate, and nucleus accumbens [22]. The SMA apparently extended contiguously to the dorsal part of the ACC. Furthermore, both SMA and ACC appear to receive rich dopamine input via ascending mesocortical projections from the ventral striatum [18], therefore, the striatal self-rewarding system having a dopamine-enriched structure may be interconnected to the dorsal ACC/SMA, suggesting that the striatal regions play a critical role in dopaminergic reward associated with implied laughter. This suggests an interesting interpretation of why we laugh, and why we scowl during pain-inducing situations. We could not observe any activation of the striatal system during generation of word-induced negative pain. Cognitive pain is likely to inhibit the dopaminergic reward system.

In brief, we would conclude that a mimic word highly suggestive of affective pain, heard by the ear, significantly activated ACC, while nonsense words that did not imply affective pain under the same task did not activate this area in humans. This suggests that the ACC may be a critical brain region involved in perceiving affective pain evoked by a mimic word that implies affective pain. We show that pain affect sustained by pain unpleasantness may depend on ACC–prefrontal (limbic–prefrontal) cortical interactions that modify the cognitive evaluation of emotions associated with word-induced pain.

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References

1. Cassirer E (1953) The philosophy of symbolic forms. Vol.1. Language. Yale University Press, New Haven, CT
2. Osaka N, Osaka M, Kondo H et al (2003) An emotion-based facial expression word activates laughter module in the human brain: a functional magnetic resonance imaging study. Neurosci Lett 340:127–130
3. Osaka M, Osaka N, Kondo H et al (2003) The neural basis of individual differences in working memory capacity: an fMRI study. NeuroImage 18:789–797
4. Marks LE (1978) The unity of the senses. Academic Press, New York
5. Sapir EA (1929) A study of phonetic symbolism. J Exp Psychol 1:225–239
6. Newman SS (1933) Further experiments in phonetic symbolism. Am J Psychol 45:53–75
7. Martin A, Haxby JV, Lalonde FM et al (1995) Discrete cortical regions associated with knowledge of color and knowledge of action. Science 270:102–110
8. Chang AC (1990) A thesaurus of Japanese mimesis and onomatopoeia: usage by categories. Taishukan, Tokyo
9. Osaka N (1990) Multidimensional analysis of onomatopoeia. Stud Phonol 24:25–33
10. Price DD (2000) Psychological and neural mechanisms of the affective dimension of pain. Science 288:17691–1772
11. Hardcastle VG (1999) The myth of pain. MIT Press, Cambridge, MA
12. Adair ER, Stevens J, Marks LE (1968) Thermally induced pain, the dol scale, and the psychophysical power law. Am J Psychol 81:147–164
13. Umemoto T, Morikawa Y, Ibuki M (1955) The non-association values and meaning-

fulness of 1892 Japanese two-letter syllables and word. Jpn J Psychol 26:148–155
14. Vogt BA, Finch DM, Olson CR (1992) Functional heterogeneity in cingulate cortex: the anterior executive and posterior evaluative regions. Cereb Cortex 2:435–443
15. Bush G., Luu P, Posner MI (2000) Cognitive and emotional influences in anterior cingulate cortex. Trends Cogn Sci 4:215–222
16. Rainville P, Duncan GH, Price DD et al (1997) Pain affect encoded in human anterior cingulate but not somatosensory cortex. Science 277:968–971
17. Osaka N, Osaka M, Morishita M et al (2004) A word expressing affective pain activates the anterior cingulate cortex in the human brain: an FMRI study. Behav Brain Res 153:123–127
18. MacDonald AW III, Cohen JD, Stenger VA, Carter CS (2000) Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. Science 288:1835–1838
19. Sawamoto N, Honda M, Okada T et al (2000) Expectation of pain enhances response to nonpainful somatosensory stimulation in the anterior cingulated cortex and parietal operculum/posterior insula: an event-related functional magnetic resonance imaging study. J Neurosci 20:7438–744
20. Kropotov JD, Crawford HJ, Polyakov YI (1977) Somatosensory event-related potential changes to painful stimuli during hypnotic analgesia: anterior cingulated cortex and anterior temporal cortex intracranial recordings. Int J Psychophysiol 27:1–8
21. Talbot JD, Marrett S, Evana AC et al (1991) Multiple representation of pain in human cerebral cortex. Science 251:1355–1358
22. Osaka N, Osaka M (2005) Striatal reward areas activated by implicit laughter induced by mimic words in humans: an fMRI study. NeuroReport 16:1621–1624
23. Mobbs D, Greicius MD, Abel-Azim E et al (2003) Humor modulates the mesolimbic reward centers. Neuron 40:1041–1048

Chapter 11

Intentional Attunement: Embodied Simulation and Its Role in Social Cognition

VITTORIO GALLESE

Introduction

> *A path leads from identification by way of imitation to empathy, that is to the comprehension of the mechanism by which we are enabled to take up any attitude at all towards another mental life.*
>
> S. Freud [1]

Psychoanalysis has always identified the body as the source of the energies feeding psychological representations. Interestingly, some recent developments in cognitive neuroscience have emphasized the role of the acting body and of sensorimotor systems in constituting the way our minds represent reality, and in particular our intersubjective reality, by shaping our cognitive schemas.

Our social mental skills enable us to successfully retrieve the mental contents of others. Sometimes we misrepresent them, hence misunderstanding others. Most of the time, though, we are pretty good at understanding what is the goal of others' behavior, why the goal was set, and on the basis of what previous elements it was set as such. We do it effortlessly and continuously during our daily social interactions. How do we do this? The dominant approach in cognitive science is to put most effort in clarifying the formal rules structuring a solipsistic mind. Much less investigated is what triggers the sense of social identity that we experience with the multiplicity of "other selves" populating our social world. Is the solipsistic type of analysis, inspired by folk psychology, the best explanatory approach? In particular, is it doing full justice to the phenomenal aspects of our social intentional relations? My answer to both questions is no.

As human beings, we do not only mentally entertain an "objective" account of the behaviors constituting the social world in which we live. In addition to phenomenally experiencing the external, objective nature of an observed action, and viewing it as something displayed and acted by an external biological object with distinct qualities, we also experience its goal-directedness or intentional character, similar to when we experience ourselves as the willful conscious agents of our ongoing behavior. From a first-person perspective, our dynamic social environment appears to be populated by volitional agents capable of entertaining, like us, an agentive intentional relation to the world. We experience ourselves as being *intentionally attuned* to other individuals. This "intentional attunement" allows us to experience others as *directed* to certain target states or objects, similarly to how we experience ourselves when doing so.

The same dual perspective is at work when witnessing the emotions and sensations experienced by others. We can provide an "objective" description of these emotions or sensations. When explicitly asked to recognize, discriminate, parameterize, or categorize the emotions or sensations displayed by others, we exert our cognitive operations by adopting a third-person perspective, aimed exactly at *objectifying* the content of our perceptions. The overall goal of these cognitive operations is the deliberate categorization of an external state of affairs.

However, when we are involved on-line with social transactions, we experience a totally different attitude toward the objects of our social perceptions. There is actually a shift of the object of our intentional relation. We are no longer directed to the content of a perception in order to categorize it. We are just *attuned to the intentional relation displayed by someone else.* Unlike Mr. Spock, the famous character of the Star Trek saga, our social mental skills are not confined to a declarative third-person perspective. We are not alienated from the actions, emotions, and sensations of others, because we entertain a much richer and affectively nuanced perspective of what other individuals do, experience, and feel. What makes this possible is the fact that *we own* those same actions, emotions, and sensations.

To naturalize social intentionality we should therefore follow a different route. The alternative strategy I suggest here is a bottom-up characterization of the nondeclarative and nonpropositional contents of social cognition. It consists in investigating the neural basis of our capacity to be attuned to the intentions of others. By means of *intentional attunement,* "the other" is much more than a different representational system; it becomes a *person,* like us. The advantage of this epistemological approach is that it generates predictions about the intrinsic functional nature of our social cognitive operations that cut across, and neither necessarily depend on, nor are subordinate to any specific cognitive mind ontology, folk psychology included.

Neuroscientific research has started to unveil the neural mechanisms that mediate between the multilevel personal experiential knowledge we hold of our lived body and the implicit certainties we simultaneously hold about others. Such personal body-related experiential knowledge enables our intentional attunement with others, which in turn constitutes a *shared manifold of intersubjectivity.* This we-centric space allows us to directly experience the meaning of the actions performed by others, and to decode the emotions and sensations they experience. An implicit form of "experiential understanding" is achieved by modeling the behavior of other individuals as intentional experiences on the basis of the equivalence between what the others do and feel and what we do and feel. This modeling mechanism is embodied simulation. Mirror neurons are probably the neural correlate of this mechanism.

This chapter will be structured as follows. After clarifying the sense and the use made here of the notion of embodied simulation, recent neuroscientific results are presented on how the execution of actions and their observation when executed by others are neurally mapped. The second part focuses on the first- and third-person experience of emotions and sensations, and their neural underpinnings. I show that the same neural circuits involved in the first-person experience of emotions and sensations are also active when witnessing the same emotions and sensations of others. It will be concluded that the functional mechanism underlying the double activation pattern of those neural circuits is embodied simulation.

In the last part of the chapter I discuss the possible relevance of my approach to psychopathology, with a particular emphasis on schizophrenia and autism.

Embodied Simulation

The notion of simulation is at present employed in many different domains, often with different, not necessarily overlapping meanings. Simulation is a functional process possessing certain content, typically focusing on possible states of its target object. For example, an authoritative view on motor control characterizes simulation as the mechanism employed by forward models to predict the sensory consequences of impending actions. According to this view, the predicted consequences are the simulated ones.

In philosophy of mind, on the other hand, the notion of simulation has been used by the proponents of the Simulation Theory of mind reading to characterize the pretend state adopted by the attributer in order to understand others' behavior [2–12].

The *Oxford English Dictionary* provides three different definitions of "simulation":

1. The action or practice of simulating, with intent to deceive; false pretence, deceitful profession.
2. A false assumption or display, a surface resemblance or imitation, of something.
3. The technique of imitating the behavior of some situation or process (whether economic, military, mechanical, etc.) by means of a suitably analogous situation or apparatus, especially for the purpose of study or personnel training.

The first two definitions convey the idea of simulation as something fake, something supposedly aimed to deceive, by *pretending to be similar* to what really differs in many respects. The third definition conveys a different meaning; namely, it characterizes simulation as a process intended to produce a better understanding of a given situation or state of affairs, by modeling it.

The third definition of simulation appears to be closer than the previous ones to the etymology of the word. The verb "to simulate" comes from the Latin *simulare*, which in turn derives from *similis*, "like," "similar to." The third definition of simulation, incidentally, also defines the prevalent epistemic approach of the Graeco-Roman classical western world: knowledge is conceived of as a process in which the knower *assimilates* what he/she is supposed to know (cf. the Latin expression *similia similibus, or* the Greek verb *homologhêin*).

I shall use the term *embodied simulation* in a way that is close to the third definition given above, that is, as a mandatory, nonpropositional functional mechanism, whose function is the modeling of objects, agents, and events in the world. Embodied simulation, as conceived of in the present chapter, is therefore not necessarily the result of a willed and conscious cognitive effort, aimed at interpreting the intentions hidden in the overt behavior of others, but rather a basic functional mechanism of our brain. However, because it also generates representational content, this functional mechanism plays a major role in our epistemic approach to the world. It represents the outcome of a possible action, emotion, or sensation one could take or experience, and serves to attribute this outcome to another organism as a real goal-state it tries to bring about, or as a real emotion or sensation it is experiencing.

Successful perception requires the capacity to predict upcoming sensory events. Similarly, successful action requires the capacity to predict the expected consequences of action. As suggested by an impressive and coherent amount of neuroscientific data (for reviews, see [13, 14]), both types of prediction seem to depend on the results of nonconscious and automatically driven neural states, functionally describable as simulation processes. According to the use I will make of this notion in the present chapter, embodied simulation is not conceived of as being confined to the domain of motor control, but rather as a more general and basic endowment of our brain. It is mental because it has content, but it is sensorimotor because its function is realized by the sensorimotor system. I call it "embodied"–not only because it is neurally realized, but also because it uses a preexisting model of the body in the brain, and therefore involves a nonpropositional form of self–other representation.

In this context, embodied simulation in social cognition can also be seen as an exaptation. It is possible that there has never been any "special design" for the function I describe here. It might be an extended functionality later coopted from a distinct original adaptational functionality, namely, sensorimotor integration for body control purposes.

Understanding Action

Our social world is inhabited by a multiplicity of acting individuals. Much of our social competence depends on our capacity to understand the meaning of the actions we witness. These actions basically pertain to two broad categories. The first is the category of transitive, object-related actions, like grasping a coffee mug, picking up a phone, biting an apple, or kicking a football. The second category of social actions is that of intransitive, expressive, or deictic actions, like blowing kisses, uttering words, or pointing to a person or location in space. What makes our perception of both types of action different from our perception of the inanimate world is the fact that there is something shared between the first- and third-person perspective of the former events; the observer and the observed are both human beings endowed with a similar brain–body system, making them act and perceive alike [15].

The discovery of mirror neurons has triggered new perspectives on the neural mechanisms at the basis of action understanding. I deal first with object-related actions.

Understanding Object-Related Actions

About 10 years ago we discovered in the macaque monkey brain a class of premotor neurons that discharge not only when the monkey executes goal-related hand actions such as grasping objects, but also when the monkey observes other individuals (monkeys or humans) executing similar actions. We called them "mirror neurons" [13, 15–21]. Neurons with similar properties were later discovered in a sector of the posterior parietal cortex reciprocally connected with area F5 (PF/PFG mirror neurons, see [22–23]).

The observation of an object-related action leads to activation of the same neural network as is active during its actual execution. Action observation causes in the observer the automatic simulated reenactment of the same action. We proposed that this mechanism could be at the basis of a direct form of action understanding [16–19, 22].

The relationship between action understanding and action simulation is even more evident in light of the results of two more recent studies carried out in our laboratory. In the first series of experiments, Umiltà et al. [24] tested F5 mirror neurons in two conditions. In the first condition the monkey could see the entire action (e.g., a hand grasping action); in the second condition, the same action was presented, but its final critical part, that is, the hand–object interaction, was hidden. Therefore, in the hidden condition the monkey only "knew" that the target object was present behind the occluder. The results showed that more than half of the recorded neurons also responded in the hidden condition [24].

These results seem to suggest that predictions–or, to use a mentalistic term, inferences–about the goals of the behavior of others appear to be mediated by the activity of motor neurons coding the goal of the same actions in the observer's brain. Out of sight is not "out of mind" just because, by simulating the action, the gap can be filled.

Some transitive actions are characteristically accompanied by a specific sound. Often this particular sound enables us to understand what is going on even without any visual information about the action producing the sound. The perceived sound has the capacity to make an invisible action inferred, and therefore present and understood.

We showed that a particular class of F5 mirror neurons, "audiovisual mirror neurons," discharge not only when the monkey executes or observes a particular type of noisy action (e.g., breaking a peanut), but also when it just hears the sound produced by the action [25].

These audiovisual mirror neurons not only respond to the sound of actions, but also discriminate between the sounds of different actions. The actions whose sounds maximally trigger the neurons' discharge when heard are also those that produce the strongest response when observed or executed. The activation of the premotor neural network that normally controls the execution of action A by sensory information related to the same action A, be it visual or auditory, can be characterized as simulating action A.

The multimodally driven simulation of action goals underpinned by the activation of neurons situated in the ventral premotor cortex of the monkey instantiates properties that are strikingly similar to the symbolic properties characteristic of human thought. The similarity with conceptual content is quite appealing: the same conceptual content ("the goal of action A") results from a multiplicity of states subsuming it: sounds, observed, and executed actions. These states, in turn, are subsumed by differently triggered patterns of activations within a population of "audiovisual mirror neurons."

The *action simulation* embodied by audiovisual mirror neurons is indeed reminiscent of the use of predicates. The verb "to break" is used to convey a meaning that can be used in different contexts: "seeing someone breaking a peanut," "hearing someone breaking a peanut," "breaking a peanut." The predicate, like the responses in audiovisual mirror neurons, does not change depending on the context to which it applies, nor depending on the subject/agent performing the action. All that changes is the context the predicate refers to [14, 26].

The general picture conveyed by these results is that the sensorimotor integration supported by the premotor-parietal F5-PF/PFG mirror matching system instantiates simulations of transitive actions utilized not only to generate and control goal-related behaviors, but also to map the goals and purposes of others' actions, by means of their simulation. This account does not entail an explicit declarative format. It is meaningful, implicit, and direct.

What is the import of these data for our understanding of *human* social cognition? Several studies using different experimental methodologies and techniques have demonstrated in humans as well the existence of a similar mirror system, matching action perception and execution [27–34]. In particular, it is interesting to note that brain imaging experiments in humans have shown that during action observation there is a strong activation of premotor and parietal areas, the likely human homologue of the monkey areas in which mirror neurons were originally described (for reviews, see [35, 36]).

Understanding Intransitive Actions

The macaque monkey ventral premotor area F5 also contains neurons related to mouth actions. These neurons largely overlap with hand-related neurons, but in the most lateral part of F5, mouth-related neurons tend to be prevalent. We recently explored the most lateral part of area F5 where we described a population of mirror neurons mostly related to the execution/observation of mouth-related actions [37]. The majority of these neurons discharge when the monkey executes and observes transitive object-related ingestive actions, such as grasping, biting, or licking. However, a small percentage of mouth-related mirror neurons discharge during the observation of intransitive, communicative facial actions performed by the experimenter in front of the monkey ("communicative mirror neurons"[37]). These actions are lip-smacking, and lip or tongue protrusion. A behavioral study showed that the observing monkeys correctly decoded these and other communicative gestures performed by the experimenter in front of them, because they elicited congruent expressive reactions [37]. It is therefore plausible to propose that communicative mirror neurons might constitute a further instantiation of a simulation-based social heuristic.

A recent brain imaging study, in which human participants observed mouth actions performed by humans, monkeys, and dogs [38], further corroborates this hypothesis. The observed mouth actions could be either transitive, object-directed actions, like a human, a monkey, or a dog biting a piece of food, or intransitive communicative actions, like human silent speech, monkey lip-smacking, and dog barking. The results showed that the observation of all biting actions led to the activation of the mirror circuit, encompassing the posterior parietal and ventral premotor cortex [38].

Interestingly, the observation of communicative mouth actions led to the activation of different cortical foci according to the different observed species. The observation of human silent speech activated the pars opercularis of the left inferior frontal gyrus, the premotor sector of Broca's region. The observation of monkey lip-smacking activated a smaller part of the same region bilaterally. Finally, the observation of the barking dog activated only extrastriate visual areas. Actions belonging to the motor repertoire of the observer (e.g., biting and speech reading) or very closely related to it (e.g., monkey's lip-smacking) are mapped on the observer's motor system. Actions that do not belong to this repertoire (e.g., barking) are mapped and henceforth categorized on the basis of their visual properties.

The involvement of the motor system during observation of communicative mouth actions is also testified by the results of a transcranial magnetic stimulation (TMS) study by Watkins et al. [39], in which they showed that the observation of silent speech-related lip movements enhanced the size of the motor-evoked potential in lip muscles. This effect was lateralized to the left hemisphere. Consistent with the brain imaging data of Buccino et al. [38], the results of Watkins et al. [39] show that the observation of communicative, speech-related mouth actions facilitates the excitability of the motor system involved in the production of the same actions. Again, we have evidence that embodied simulation mediates the decoding of social meaningful actions.

Experiencing the Actions of Others as Embodied Action Simulation

When a given action is planned, its expected motor consequences are forecast. This means that when we are going to execute a given action we can also predict its consequences. This prediction is the computational result of the action model. Through a process of "equivalence" between what is acted and what is perceived, given its shared and overlapping subpersonal neural mapping, this information can also be used to predict the consequences of actions performed by others. This equivalence–underpinned by the activity of mirror neurons–is made possible by the fact that both predictions (of our actions and of others' actions) are simulation (modeling) processes. The same functional logic that presides over self-modeling is employed also to model the behavior of others: to perceive an action is equivalent to internally simulating it. This enables the observer to use her/his own resources to penetrate the world of the other by means of an implicit, automatic, and unconscious process of motor simulation.

This simulation process establishes a direct link between agent and observer, in that both are mapped in a neutral fashion. The agent parameter is specified, but not its specific filler, which is indeterminate. Mirror neurons constitutively map an agentive relation; the mere observation of an object not acted upon does not evoke any response. It is just the agentive relational specification to trigger mirror neurons' response. The fact that a *specific agent* is not mapped does not entail that an agentive relation is not mapped, but simply that the agent parameter can be either oneself or the other.

As we have seen, in humans as in monkeys, action observation constitutes a form of embodied action simulation. This kind of simulation, however, is different from the simulation processes occurring during visual and motor imagery. Action observation *automatically triggers action simulation*, while in mental imagery the simulation process is triggered by a deliberate act; one purposely decides to imagine observing something or doing something. An empirical validation of this difference comes from brain imaging experiments carried out on healthy human participants. Comparing the motor centers activated by action observation with those activated during voluntary mental motor imagery, it emerges that only the latter leads to the activation of pre-SMA (the area just rostral to the supplementary motor area] and of the primary motor cortex [40].

That said, it appears nonetheless that both mental imagery and action observation are kinds of simulation. The main difference is what triggers the simulation process: an internal event, in the case of mental imagery, and an external event, in the case of action observation. This difference leads to slightly different patterns of brain activation. However, both conditions share a common mechanism: the simulation of actions by means of the activation of parietal premotor cortical networks. I submit that this process of automatic simulation also constitutes a basic level of experiential understanding, a level that does not entail the explicit use of any theory or propositional representation.

Mirror Neurons and the Understanding of Intentions

When an individual starts a movement aimed to attain a goal, such as picking up a pen, he/she has clear in mind what he/she is going to do, for example writing a note on a piece of paper. In this simple sequence of motor acts, the final goal of the whole action is present in the agents' mind and is somehow reflected in each motor act of the sequence. The action *intention*, therefore, is set before the beginning of the movements. This also means that when we are going to execute a given action, we can also predict its consequences.

Monkeys may exploit the mirror neuron system to optimize their social interactions. At least, the evidence we have collected so far seems to suggest that the mirror neuron system for actions is sophisticated enough to enable its exploitation for social purposes. Recent results by Cisek and Kalaska [41] show that neurons in the dorsal premotor cortex of the macaque monkey can covertly simulate observed behaviors of others, like a cursor moved to a target on a computer screen, even when the relation between the observed sensory event and the unseen motor behavior producing it is learned through stimulus–response associations. My hypothesis is that monkeys might entertain a rudimentary form of "teleological stance," a likely precursor of a full-blown intentional stance. This hypothesis extends to the phylogenetic domain, the ontogenetic scenario proposed by Gergely and Csibra [42] for human infants. New experiments are being designed in my lab to test this hypothesis.

But monkeys certainly do not entertain full-blown mentalization. Thus, what makes humans different[1]? At present we can only make hypotheses about the relevant neural mechanisms underpinning the mentalizing abilities of humans that are still poorly understood from a functional point of view. In particular, we do not have a clear neuroscientific model of how humans can understand the intentions promoting the actions of others they observe. A given action can be originated by very different intentions. Suppose one sees someone else grasping a cup. Mirror neurons for grasping will most likely be activated in the observer's brain. A simple motor equivalence between the observed action and its motor representation in the observer's brain, however, can only tell us *what* the action is (it's a grasp) and not *why* the action occurred. This may lead to an argument against the relevance of mirror neurons for social cognition, and in particular, for determining the intentions of others. We should ask ourselves: what is an action intention? More specifically, what

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[1] The easiest answer would be, of course, the presence of language. This answer, though, is question-begging in that it only transposes the human cognitive endowment to be explained. Furthermore, it implies a perfect overlap between language and our mentalizing abilities. A discussion of these debated issues is beyond the scope and space limits of this article.

does it mean to determine the intention of the action of someone else? I propose a deflationary answer. Determining why action A (grasping the cup) was executed, that is, determining its intention, can be equivalent to detecting the goal of the as yet unexecuted and impending subsequent action B (say, drink from the cup).

We empirically addressed these issues in a recently published functional magnetic resonance imaging (fMRI) study [43]. Subjects watched three kinds of stimuli: grasping hand actions without a context, context only (a scene containing objects), and grasping hand actions embedded in contexts. In the latter condition the context suggested the intention associated with the grasping action (either drinking or cleaning up). Actions embedded in contexts, compared with the other two conditions, yielded a significant signal increase in the posterior part of the inferior frontal gyrus and the adjacent sector of the ventral premotor cortex where hand actions are represented. Thus, premotor mirror areas–areas active during the execution and the observation of an action–previously thought to be involved only in action recognition are actually also involved in understanding the "why" of action, that is, the intention promoting it. Detecting the intention of action A is equivalent to predicting its distal goal, that is, the goal of the subsequent action B. Furthermore, this study shows that being or not being explicitly instructed to determine the intention of the observed actions of others makes no difference in terms of the activation of the premotor mirror areas. This means that–at least for simple actions such as those employed in this study–the ascription of intentions occurs by default and it is underpinned by the mandatory activation of an embodied simulation mechanism.

The neurophysiological mechanism at the basis of the relationship between intention detection and action prediction was recently unveiled. Fogassi et al. [23] described a class of parietal mirror neurons whose discharge during the observation of an act (e.g., grasping an object) is conditioned by the type of not yet observed subsequent act (e.g., bringing the object to the mouth) specifying the overall action intention. This study shows that the inferior parietal lobe of the monkey contains mirror neurons discharging in association with the execution/observation of motor acts (grasping) only when they are embedded in a specific action aimed at different goals. It must be emphasized that the neurons discharge before the monkey observes the experimenter starting the second motor act (bringing the object to the mouth or placing it in the cup). Individual motor acts are dependent on each other as they participate in the overarching distal goal of an action, thus forming prewired intentional chains, in which each motor act is facilitated by the previous one. This suggests that in addition to recognizing the goal of the observed motor act, mirror neurons allow the observing monkey to predict the agent's next action, and hence its overall intention. It is possible to interpret this mechanism as the neural correlate of the dawning of more sophisticated intention understanding abilities, such as those characterizing our species.

The results of another recently published fMRI study emphasize the role of the premotor cortex in the coding of sequential events. Schubotz and von Cramon [44] contrasted the observation of biological hand actions with that of abstract motion (movements of geometric shapes). In both conditions 50% of the stimuli failed to attain the normally predictable end state. The task of participants was to indicate whether the action was performed in a goal-directed manner ("yes" button) or not ("no" button). After abstract motion observation, participants had to indicate whether the object sequence was regular until the end of presentation ("yes" button) or not ("no" button). Results showed that both conditions elicited significant activation within the ventral premotor cortex. In addition, the prediction of biological actions also activated BA 44/45, which is also part of the mirror neuron system. Schubotz and von Cramon [44] concluded that their findings point to a basic premotor contribution to the representation or processing of sequentially structured events, supplemented by different sets of areas in the context of either biological or nonbiological cues.

The mechanism of intention understanding just described appears to be rather simple: depending on which motor chain is activated, the observer is going to activate the motor schema of what, most likely, the agent is going to do. How can this be accomplished? The statistical frequency of action sequences as they are habitually performed or observed in the social environment can determine preferential paths of inferences/predictions. It can be hypothesized that this may be equivalent to chaining different populations of mirror neurons coding not only the observed motor act, but also those that in a given context would normally follow. Ascribing intentions would therefore consist in predicting a forthcoming new goal. If this is true, it follows that one important difference between humans and monkeys could be the level of recursivity attained by the mirror neuron system in our species. A similar proposal has been recently put forward in relation to the faculty of language [45, 46]. According to my perspective, and in sharp contrast with what mainstream cognitive science would maintain, action prediction and the ascription of intentions do not belong to different cognitive realms. They are related phenomena, determined by embodied simulation mechanisms underpinned by the activation of chains of logically related mirror neurons.

The Body of Emotions

Let us now turn to emotions. Emotions constitute one of the earliest ways available to the individual to acquire knowledge about its own state, thus enabling her/him to reorganize this knowledge on the basis of the outcome of the relations entertained with others. This points to a strong interaction between emotion and action. We dislike things that we seldom touch, look at, or smell. We do not "translate" these things into motor schemas suitable for interacting with them, which are probably "tagged" with positive affective-hedonic values, but rather into aversive motor schemas, probably "tagged" with negative affective-hedonic connotations.

The coordinated activity of sensorimotor and affective neural systems results in the simplification and automatization of the behavioral responses that living organisms are supposed to produce in order to survive. The close coupling between affect and sensorimotor integration appears to be one of the most powerful drives leading the developing individual to the achievement of progressively more "distal" and abstract goals [47, 48].

Such a coupling between emotion and action is indeed highlighted by a study of Adolphs et al. [49], in which over 100 brain-damaged patients were reviewed. This study showed that the patients who suffered damage to the sensorimotor cortices were also those who scored worst when asked to rate or name facial emotions displayed by human faces. As emphasized by Adolphs [50, 51], the integrity of the sensorimotor system appears to be critical for the recognition of emotions displayed by others, because the sensorimotor system appears to support the reconstruction of what it would feel like to be in a particular emotion, by means of simulation of the related body state.

Before addressing the role of embodied simulation in the understanding of emotions, it is necessary to clarify what exactly we are referring to when we speak of emotions. There are many different ways to experience an emotion. "Emotion" is a word that designates and refers to a multidimensional aspect of our life. To experience an emotion can be described as subjectively experiencing "inner body states" of varied intensity and amplitude that can surface, with a variety of degrees of explicitness, as ostensive behaviors, often localized to specific body parts, like the face.

Under both first- and third-person perspectives of emotion experience, a complex state of the organism is accompanied by variable degrees of awareness and meta-awareness, variously indicated as "appraisal." It is a common experience to be asked by people we know questions like "Why are you so angry at me?" without realizing until the very moment at which the question was asked that we were indeed expressing the emotion of anger. We can be in a given emotional state, and express it ostensively with our body, without fully experiencing its content as the content of a particular emotion. Lambie and Marcel [52] have distinguished two levels of emotion appraisal: a first-order phenomenal state, which they call "first-order emotion experience," and conscious second-order awareness. Both states can be either self-directed (first-person perspective) or world-directed (third-person perspective). The content of the first-order phenomenal state is physical, centered on one's body state. The content of second-order conscious awareness can be either propositional or nonpropositional.

It should be emphasized that it is indeed possible to witness the expression of a given emotional state displayed by someone else without explicitly relying on the propositional description of that state. It is precisely this unmediated, direct form of emotion understanding that I will be addressing here. More specifically, I will characterize the neural underpinnings of a simulation-based type of basic social emotion understanding.

A recent empirical support for a tight link between embodied simulation and our perception of the emotions of others as displayed by their facial expressions comes from an fMRI study on healthy participants by Carr et al. [53]. This study shows that both observation and imitation of the facial expression of emotions activate the same restricted group of brain structures, including the ventral premotor cortex, the insula, and the amygdala. These data show that both the perception and the production of emotion-related facial expressions impinge upon common neural structures whose function could be characterized as that of a neural mirror matching mechanism. However, one might argue that pretence–the purposive enactment of the overt body expression of an emotion–does not bestow the characteristic phenomenal awareness of that emotion. Imitating the expression of emotions does not necessarily produce the first-person experience of the emotion one is imitating.

In a recently published fMRI study carried out on healthy human participants, we specifically addressed the issue whether the first- and third-person experience of a particular emotion are mapped by a shared neural representation. To that purpose, we scanned the brain activity of healthy participants during the phenomenal experience of disgust, by having them inhaling disgusting odorants, and during the observation of the same emotion as displayed by video clips of other individuals dynamically expressing it with their facial expression. The results of this study showed that witnessing the facial expression of disgust of others activates the left anterior insula at a location overlapping the one activated by the first-person subjective experience of disgust [54].

The anterior sector of the insula receives rich connections from olfactory and gustatory structures and from the anterior sectors of the ventral bank of the superior temporal sulcus, where cells have been found in the monkey to respond to the sight of faces. The anterior insula thus appears to link gustatory, olfactory, and visual stimuli with visceral sensations and the related autonomic and visceromotor responses (see also [35]). Penfield and Faulk [55] electrically stimulated the anterior insula in humans undergoing neurosurgery. During the stimulation the patients reported feeling nauseous and sick. More recently, Krolak-Salmon et al. [56], using shorter and weaker stimulation parameters, evoked unpleasant sensations in the throat and mouth. These findings support the link between the anterior visceromotor insula and the experience of disgust or related aversive visceral sensations and visceromotor reactions.

A few clinical cases also show that when the anterior insula is damaged, both the subjective experience of disgust and the capacity to recognize this emotion in others are seriously impaired. Calder et al. [57] reported the case of the patient N.K., who after lesions of the left insula and neighboring structures was selectively impaired in recognizing disgust in the facial expressions of others. This incapacity to perceive disgust extended to the auditory modality: he did not recognize the emotional valence of sounds typical for disgust such as retching, while easily recognizing that of sounds characteristic of other emotions such as laughter. His recognition of the facial expression of other emotions, including that of fear, was normal. What is most interesting for our discussion is the fact that N.K.'s multimodal perceptual deficit for disgust was mirrored by an equivalent deficit in his first-person experience of the same emotion. He reported having a reduced sensation of disgust, ranking almost two standard deviations below the normal score in a questionnaire measuring the emotional experience of disgust. His experience of other emotions, though, was fairly normal.

A similar pattern of deficits was reported by Adolphs et al. [58]. They described patient B., who following bilateral damage to the insula showed substantial deficits in recognizing the facial expression of disgust, while preserving his recognition of other facial expressions. B.'s incapacity to experience disgust is evident from the fact that he ingests food indiscriminately, including inedible items, and fails to feel disgust when presented with stimuli representing disgusting food items.

Experiencing disgust and witnessing the same emotion in the facial expression of someone else both activate the same neural structure, the anterior insula. Damage to this structure impairs the capacity both to experience disgust and to recognize it in others. This suggests, at least for the emotion of disgust, that the first- and third-person experience of a given emotion are underpinned by the activity of a shared neural substrate. When I see a given facial expression, and this perception leads me to understand that expression as characterized by a particular affective state, I do not achieve this type of understanding through an argument by analogy. The other's emotion is constituted and experienced, and hence directly understood by means of an embodied simulation producing a shared body state. It is the body state shared by the observer and the observed to enable direct understanding. A similar simulation-based mechanism has been proposed by Goldman and Sripada [59] as "unmediated resonance."

Being "In Touch"

In the second posthumous book of his *Ideas* [60], Husserl points out that the lived body (*Leib*) is the constitutive foundation of any perception, the perception of others included. Were we adopting a similar perspective to frame social cognition, we could say that the self-modeling functional architecture of the living body is the scaffold for the modeling of the intentional relations of other individuals. The multimodal dynamic model of our body as of a goal-seeking organism brings about the basic representational architecture for the mapping of intentional relations. The empirical evidence so far reviewed on action and emotion perception seems to support this line of thought. Let us focus now on tactile sensations as the target of our social perception.

Touch has privileged status in making possible the social attribution of living personhood to others. "We'll be in touch" is a common phrase in everyday language which metaphorically refers to the wish to continue being related, being in contact with someone else. Such examples show how the tactile dimension is intimately related to the interpersonal dimension. In a recent fMRI experiment we have shown that the first-person subjective experience of being touched on one's body activates the same neural networks as are activated by observing someone else's body being touched [61]. Within SII-PV, a multimodal cortical region, perhaps exceeding the limits of the traditional unimodal second somatosensory area, there is a localized neural network activated similarly by the self-experienced sensation of being touched and by the perception of an external tactile relation.

Such an activation, obtained during the perception of another's body being touched, could perhaps be more parsimoniously interpreted as the outcome of the prediction of a body impact on the observer's own body. However, in sharp contrast with what this interpretation would predict, the manipulation of the perspective (subjective vs objective) under which the observed tactile stimulation was presented to participants did not modify the degree of activation of the same region within SII-PV. Thus, visual stimuli activate SII/PV in a way that is unaffected by how easily they can be integrated into our body schema.

In a second experiment, we replaced the legs of the actors in the movies by inanimate objects: rolls of paper towels and binders. Results indicated that even seeing an object being touched produced a significantly larger activation of SII/PV than did seeing the object being only approached [61]. The touching of two surfaces in the outside world is something in principle very abstract, if only visually mapped. Mapping it onto what we feel when one of the surfaces being touched is our own body, fills this abstract visual event with a very personal meaning: what it feels like to be touched. These results were recently confirmed and extended by another fMRI study [62].

It appears therefore that the critical stimulus for SII/PV activation is the perception of touch; be it the touch of an object, another human being, or our own legs. This double pattern of activation of the same brain region seems to suggest that both our capacity to recognize and implicitly understand the tactile experience of others, and a more abstract notion of touch (as in the case of object touch) could be mediated by embodied simulation.

It is interesting to note that Husserl [60] wrote that everything we see, we simultaneously also see as a tactile object, as something which is directly related to the alive body, but not by virtue of its visibility. The "tactile lived body," in particular, provides the constitutive foundation of our cognitive and epistemic self-referentiality. The perspectival spatial location of our body provides the essential foundation for our determination of reality.

The Shared Manifold and Social Identification

The establishment of self/other identity is a driving force for the cognitive development of more articulated and sophisticated forms of intersubjective relations. I have proposed that the mirror matching system could be involved in enabling the constitution of this identity [13, 15, 20–21]. I think that the notion of social identification should accommodate and account for all different aspects of expressive behavior enabling us to establish a meaningful link between others and ourselves. This "enlarged" notion of social identification opens up the possibility of unifying under the same account multiple aspects and possible levels of description of intersubjective relations.

As we have seen, when we enter into relation with others there is a multiplicity of states that we share with them. We share emotions, our body schema, our being subject to somatic sensations such as touch, pain, etc. A comprehensive account of the richness of content we share with others should rest upon a conceptual tool capable of being applied to all these different levels of description, while simultaneously providing their functional and subpersonal characterization.

I introduced this conceptual tool as the *shared manifold* of intersubjectivity [13, 15, 19]. I posit that it is by means of this shared manifold that we directly experience other human beings as similar to us. It is just because of this shared manifold that intersubjective communication, social imitation, and mind reading become possible [13, 21]. The shared manifold can be operationalized at three different levels: a phenomenological level, a functional level and a subpersonal level.

The phenomenological level is the one responsible for the sense of similarity, of being individuals within a larger social community of persons like us that we experience whenever we are confronted with other human beings. It could also be defined as the *empathic* level, provided that empathy is characterized in the "enlarged" way I am advocating here. Actions, emotions and sensations experienced by others become *experientially* meaningful to us because we can *share* them with others.

The functional level can be characterized in terms of *as if* modes of interaction enabling models of self/other to be created. The same functional logic is at work during both self-modeling and the understanding of others' behavior. Both are models of interaction, which map their referents on identical relational functional nodes. All modes of interaction share a relational character. At the functional level of description, the relational logic of operation produces the self/other identity by enabling the system to detect coherence, regularity and predictability independently of their situated source. This view is similar to that proposed by Damasio and Adolphs on the understanding of emotions [51, 63, 64]. There is, however, an important difference between the "as if" view proposed here and that of Damasio, as far as the underlying neural mechanism is concerned. According to the present proposal, a crucial factor in both first- and third-person experiential understanding of social behavior is the activation of the cortical sensorimotor or visceromotor centers whose outcome, when activating downstream centers, determines a specific "behavior," be it an action or an emotional state. When only the cortical centers, decoupled from their peripheral effects, are active, the observed actions, emotions or sensations are "simulated" and thereby understood.

The subpersonal level is instantiated as the level of activity of a series of mirror matching neural circuits. The activity of these neural circuits is, in turn, tightly coupled with multilevel changes within body states. We have seen that mirror neurons instantiate a multimodal intentional shared space. My hypothesis is that analogous neural networks are at work to generate multimodal emotional and sensitive shared spaces [13, 15, 19–21, 35, 65]. These are the shared spaces that allow us to directly experience and understand the emotions and the sensations we take others to experience.

It should be clarified that the shared manifold of intersubjectivity does not entail that we experience others as we experience ourselves. The shared manifold simply enables and bootstraps mutual intelligibility. In contrast with emotional contagion, empathy entails the capacity to experience what others experience while still attributing these experiences to others and not to the self. Obviously, self/other identity is not all there is in empathy. As noted by Husserl [66], if this were the case, others could no longer be experienced as such (see also [67]). On the contrary, it is the alterity of the other that grounds the objective character of reality. The quality of our erlebnis of the "external world" and its content are constrained by the presence of other subjects that are intelligible, while preserving their character of alterity: an alterity that is present also at the subpersonal level, instantiated by the different neural networks–and/or their different levels of activation–coming into play when I act with respect to when others act, or when I experience an emotion or a sensation with respect when others do the same.

The Intentional Attunement Hypothesis

Our seemingly effortless capacity to conceive of the acting bodies inhabiting our social world as *goal-oriented persons* like us depends on the constitution of a "we-centric" shared meaningful space. I have proposed that this shared manifold space [13, 15, 19–21] can be characterized at the functional level as embodied simulation, a specific mechanism, probably constituting a basic functional feature by means of which our brain/body system models its interactions with the world.

The mirror neuron systems and the other nonmotor mirroring neural clusters in our brain represent *one particular* subpersonal instantiation of embodied simulation. With this mechanism we do not just "see" an action, an emotion or a sensation. Side by side with the sensory description of the observed social stimuli, internal representations of the body states associated with these actions, emotions and sensations are evoked in the observer, "as if" he/she were doing a similar action or experiencing a similar emotion or sensation. Social cognition is not *only* explicitly reasoning about the contents of someone else's mind. Our brains, and those of other primates, appear to have developed a basic functional mechanism, embodied simulation, which gives us an experiential insight into other minds.

The shareability of the phenomenal content of the intentional relations of others, by means of the shared neural underpinnings, produces *intentional attunement*. Intentional attunement, in turn, by collapsing the others' intentions into the observer's ones, produces the peculiar quality of familiarity we entertain with other individuals. This is what "being empathic" is about. By means of a shared neural state realized in two different bodies that nevertheless obey the same functional rules, the "objectual other" becomes "another self." Furthermore, the mirror neuron system for actions in humans appears to be suitable for the detection of the intentions promoting the behavior of others. Thus, as previously hypothesized [68], the mirror neuron system could be at the basis of basic forms of mind reading.

This of course does not account for all of our social cognitive skills. Our most sophisticated mind-reading abilities probably require the activation of large regions of our brain, certainly larger than a putative domain-specific "theory of mind module." The same actions performed by others in different contexts can lead the observer to radically different interpretations. Thus, social stimuli are also understood on the basis of the explicit cognitive elaboration of their contextual aspects and of previous information.

The point is that these two mechanisms are not mutually exclusive. Embodied simulation is experience-based, while the second mechanism is a cognitive description of an external state of affairs. Embodied simulation is the scaffold for the propositional, more cognitively sophisticated mind-reading abilities. When the first of these mechanisms is not present or is malfunctioning, as perhaps in schizophrenia and autism (see the next two sections below), the second one can provide only a pale, detached account of the social experiences of others. It is an empirical issue to determine how much of social cognition–language included–can be explained by embodied simulation and its neural underpinnings.

This proposal also opens interesting new perspectives for the study of the neural underpinnings of psychopathological states and psychotherapeutic relations.

Psychopathological Implications of Intentional Attunement: Schizophrenia

All of our social transactions depend on mutual understanding. Simultaneously, however, interpersonal intelligibility is accompanied by the capacity of to establish clear-cut boundaries, carving out a "self" from the "outside world." Being "oneself" is experienced as being similar to other selves, while simultaneously experiencing its unique character.

In schizophrenia, self and other are no longer mutually interrelated, but tend to diverge and crystallize into segregated, incomprehensible and impenetrable realms. In spite of this lack of interpersonal relatedness, the self can experience dramatic loss of its boundaries, as epitomized by Schneiderian positive symptoms such as thought insertion, auditory hallucinations, and delusion of action control. Social and personal identities are both disrupted. The problem of psychopathology is therefore to reconcile all these different psychotic articulations within a coherent explanatory frame.

Schizophrenia, as pointed out by Terenius [69], has so far been an elusive target for research. Furthermore, the current DSM-IV-inspired operational diagnostic criteria provide a much clearer picture of what schizophrenia *is not* than of what *it is*. A possible reason accounting for this elusiveness could be the fact that a comprehensive account of schizophrenia–but the same could be said of all psychoses–would imply a global understanding of the human mind. Any serious attempt to understand cognition, emotions and language that is devoid of a "global perspective" is doomed to failure. This challenging enterprise requires an integrative approach. I believe the same to hold true for schizophrenia. From that it follows that a global approach to schizophrenia cannot but incorporate the same multiple levels of explanation that we adopt when trying to build a coherent account of cognition, language and affective behavior.

However, this is by no means a new idea. In his seminal monograph *La Schizophrénie* [70], Minkowski wrote that we cannot fully understand schizophrenia unless we are able to frame it within a thorough account of the structure of subjectivity. Autism, the incapacity to be attuned with the world, according to Minkowski [70], constitutes the basic clinical essence of schizophrenia. Minkowski developed an original intuition of his mentor Bleuler, who wrote that schizophrenics cut themselves off from any contact with the external world [71]. The core problem of schizophrenics is, accordingly, their lack of "vital contact with reality" (p. 98 in [70]), viewed as an incapacity to "resonate with the world," to establish meaningful bonds with other individuals. The contact with reality is loosened or completely lost not only with respect to the transactions with the social world, but also from the first-person perspectival point of view.

Schizophrenia as "lack of resonance," as an empathic disorder, has been a constant theme in the reflections of phenomenologically inspired psychiatry. Blankenburg [72] characterizes the autistic dimension of schizophrenia as a global crisis of "common sense," incapacity to prereflexively grasp the meaning of the world, a world that looks terribly unfamiliar and strange to the schizophrenic's eyes. Parnas and Bovet [73] have argued that schizophrenic autism derives from a transformation of the structure of subjectivity in its tripartite dimensions: self-awareness, intentionality and intersubjectivity. A lack of attunement would be at the origin of the incapacity of schizophrenics to draw a coherent and meaningful picture of their social world. In a more recent paper Parnas et al. (p. 133 in [74]) argue that in schizophrenics " . . . experience is more observed than lived," most likely because of the incapacity to attain a " . . . nonreflective, tacit sensibility, procuring a background texture or organization to the field of experience." This is exactly the same level of prereflexive, nonpropositional experiential understanding of the world of interpersonal relations which I have been characterizing throughout this chapter under the title "shared manifold of intersubjectivity."

The "defective attunement" hypothesis of phenomenological psychiatry is highly consonant with the picture I have presented here. A disruption of the multilevel simulation processes characterizing the shared manifold might be a possible cause of "defective intentional attunement" in schizophrenic patients. The ineffable nature of schizophrenics' estrangement is just a negative sign of their core problem. There are no words or propositions available to describe what healthy individuals directly and preverbally experience. If the mechanisms enabling us to constitute the implicit certainties we normally entertain about the world do not function properly, we are left with a need to purposively attribute sense to a world that looks totally strange.

Psychopathological Implications of Intentional Attunement: Autism

A similar proposal can be made for the autistic syndrome in children. The autistic syndrome is a severe and chronic developmental disorder characterized by social and communicative deficits and by a reduced interest in the environment, towards which restricted and often stereotyped initiatives are taken [75]. To be an autistic child means, with variable degrees of severity, to be incapable of establishing meaningful social communications and bonds, to establish visual contact with the world of others, to share attention with the others, and to be incapable of imitating others' behavior or of understanding others' intentions, emotions and sensations.

I would like to briefly focus on some of the early symptoms. Towards the end of the first year of life, autistic children experience difficulties or even impossibility in orientating themselves on the basis of cues provided by others. They are incapable of sharing attention with others, incapable of reacting in a congruent fashion to others' emotions. They are also highly impaired in their ability to recognize human faces or display imitative behaviors. All of these early manifestations of autism share a common root: the cognitive skills required to establish meaningful bonds with others are missing or seriously impaired.

My hypothesis is that these deficits are to be ascribed to a deficit or malfunctioning of "intentional attunement." If it is true–as held throughout this chapter–that at the basis of our social competence is first of all the capacity to constitute an implicit and directly shared interpersonal meaningful space, enabling us to establish a link with the multiple intentional relations instantiated by others, then it follows that a disruption of this shared manifold should be the core problem of the autistic mind. Incapacity to develop a full and comprehensive intentional attunement with others implies as a consequence the development of an incomplete or malfunctioning shared manifold.

The lack of full-blown intentional attunement will produce various and diversified cognitive and executive deficits, all sharing the same functional origin: a lack or malfunctioning of embodied simulation routines, probably underpinned by impairments in connectivity and/or functioning of the mirror neuron system. If my hypothesis is correct, the posited intentional attunement deficit should become manifest at the various levels of social cognition it normally underpins. A series of experimental data seems to suggest this to be the case.

A recent study investigating postural adjustments in autistic children has shown that, unlike healthy individuals, they use motor strategies basically relying on feed-back information, rather than on feed-forward modes of control. This disturbance of executive control strategies prevents autistic children from adopting anticipatory postural adjustments [76]. Given the functional characterization of forward models as simulation-based, it is difficult not to interpret these data as evidence of a simulation deficit. Such postural deficits are not intrinsically social; however, they stem from a disruption within the executive control domain of a functional mechanism–simulation–that I proposed to be at the root of the constitution of the shared experiential interpersonal space.

Two recent studies employing different techniques such as electroencephalography and TMS show that individuals with autistic spectrum disorder (ASD) might be suffering an action simulation deficit induced by a dysfunction of their mirror system for action. The study by Oberman et al. [77] showed that individuals with ASD, unlike healthy controls, did not show mu frequency suppression over the sensorimotor cortex during action observation. The study by Theoret et al. [78] showed that, again unlike healthy controls, individuals with ASD did not show TMS-induced hand muscle facilitation during hand action observation.

A further indication of simulation deficits in the autistic syndrome is exemplified by imitation deficits. Autistic children have problems in both symbolic and nonsymbolic imitative behaviors, in imitating the use of objects, in imitating facial gestures and in vocal imitation [79]. These deficits characterize both high- and low-functioning forms of autism. Furthermore, imitation deficits are apparent not only in comparison with the performances of healthy subjects, but also with those of mentally disabled nonautistic subjects. According to my hypothesis, imitation deficits in autism are caused by the inability to establish a motor equivalence between demonstrator and imitator, most likely due to malfunctioning of the mirror neuron system, or because of disrupted emotional/affective regulation of the same system. Imitation deficits thus can be characterized as further examples of a disrupted shared manifold.

Let me now briefly turn to emotional/affective deficits. Several studies have reported the severe problems autistic children experience in the facial expression of emotions and the understanding of the same in others [80–83]. Furthermore, Hobson and Lee [84] reported that autistic children score much worse than healthy controls in reproducing the affective qualities of observed actions. All these deficits can be framed as affective attunement deficits, hence as further instantiations of a lacunate shared manifold.

My hypothesis for interpreting the autistic syndrome as an intentional attunement deficit is quite divergent from many of the mainstream ideas concerning the origin of this developmental disorder. One of the most credited theories on autism, despite its varying–and not always congruent–articulations, posits that autism is caused by a deficit in a specific mind module, the "theory of mind" module, selected in the course of evolution to build theories about the mind of others [85–87]. One of the problems of this theory is that it is hard to reconcile it with what we learn from the reports of some high-functioning autistic individuals. What they claim is that in order to understand how they supposedly should feel in given social contexts, and what others supposedly feel and think in those same contexts, they have to rely on theorizing. What these reports seem to suggest is that theorizing about the minds of others is not quite the basic deficit, but the only compensating strategy available in the absence of more elementary and basic cognitive skills enabling a direct experiential take on the world of others. Furthermore, a recent study carried out on a patient who suffered a focal bilateral lesion of the anterior cingulate cortex, previously identified as the candidate site for the theory of mind module, showed no evidence of mind-reading deficits [88].

The shared manifold of intersubjectivity and the intentional attunement it generates constitute a general hypothesis on social cognition [20–21, 35] that can be empirically tested at multiple levels in both healthy and psychotic individuals. Furthermore, this proposal and the approaches it generates have the merit of revealing the possibility of establishing more insightful therapeutic bonds with psychotic patients.

Conclusions

The main point of the present chapter is that cognitive neuroscience has discovered some of the neural mechanisms mediating between the multilevel personal knowledge we hold of our lived body and the *implicit certainties* we simultaneously hold about others. Such personal body-related knowledge enables us to understand directly the actions performed by others, and the intentions promoting them, and to decode the emotions and sensations experienced by others. Our seemingly effortless capacity to conceive of the acting bodies inhabiting our social world as *goal-oriented persons* like us depends on the constitution of a shared meaningful interpersonal space. I propose that this shared we-centric space can be characterized at the functional level as embodied simulation, a specific mechanism, probably constituting a basic functional feature by means of which our brain/body system models its interactions with the world. Embodied simulation constitutes a crucial functional mechanism in social cognition, and it can be neurobiologically characterized. The mirror neurons matching systems represent a subpersonal instantiation of embodied simulation.

The neuroscientific evidence reviewed here suggests that social cognition is tractable at the neural level of description. This level is implicit, though, when the organism is confronting the intentional behavior of others; it produces a specific phenomenal state of "intentional attunement." This phenomenal state generates a peculiar quality of familiarity with other individuals, produced by the collapse of the others' intentions into the observer's ones. This seems to be what being empathic is about.

The sharp distinction classically drawn between the first- and third-person perspective of acting and experiencing emotions and sensations appears to be much more blurred at the level of the subpersonal mechanisms mapping it. The gap between the two perspectives is bridged by the way the intentional relation is functionally mapped at the neural body level. Any intentional relation can be mapped as a relation holding between a subject and an object. The mirror neural circuits described in this chapter map the different intentional relations in a compressed and indeterminate fashion, which is neutral as to the specific quality or identity of the agentive/subjective parameter. By means of a shared functional state realized in two different bodies that nevertheless obey the same functional rules, the "objectual other" becomes "another self."

The shareability of the phenomenal content of intentional relations as mediated by sensorimotor multimodally integrated neural circuits, has interesting consequences–both from a theoretical and empirical point of view–on the debate on how semantics is mapped in the brain. The picture conveyed by the neuroscientific data reviewed here suggests the necessity of cutting across the widely endorsed dichotomy between distinct semantic and pragmatic cognitive domains. Social meaning is primarily the object of practical concern and not of theoretical judgement [89]. It relies on noninferential mechanisms which do not require the explicit use of rationality. As proposed by Gordon [5], the implicit recognition of conspecifics as intentional agents is a case of procedural rather than declarative knowledge.

Furthermore, if embodied simulation and its neural counterpart–the mirror neuron circuits–do indeed constitute an automatic, nonpropositional mechanism for the attribution of social meaning, the sharp dichotomy of a semantic/pragmatic division of labor between brain areas receiving the ventral/dorsal visual streams [90–92] should also be questioned.

Social cognition is not only thinking about the contents of someone else's mind. The automatic translation of the folk psychology-inspired "boxology"–endorsed by functionalism and by some quarters in cognitive science–into newly formed brain modules specifically dedicated to mind-reading abilities should be carefully scrutinized. Looking for the brain location of intentions "as such" might not be the best epistemic strategy for revealing what social cognition really is. A more promising and potentially fruitful strategy lies in the integration of multiple approaches (from genetics and molecular biology all the way up to neurophysiology, brain imaging and cognitive psychology) to the study of the role played in social cognition by the sensorimotor systems of primate brains.

Our brains, and those of other primates, appear to have developed a basic functional mechanism, embodied simulation, which gives us an experiential insight of other minds. This mechanism may provide the first unifying perspective of the neural basis of important aspects of social cognition.

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References

1. Freud S (1921) Group psychology and the analysis of the ego. SE 18, pp 67–143
2. Gordon R (1986) Folk psychology as simulation. Mind Lang 1:158–171
3. Gordon R (1995). Simulation without introspection or inference from me to you. In: Davies M, Stone T (eds) Mental simulation Blackwell, Oxford, pp 53–67
4. Gordon R (1996) 'Radical' Simulationism. In: Carruthers P, Smith P (eds) Theories of Theories of Mind. Cambridge University Press, Cambridge, pp 11–21
5. Gordon R (2005) Intentional Agents Like Myself. In: Hurley S, Chater N (eds) Perspectives on Imitation: From Cognitive Neuroscience to Social Science, vol 2. MIT Press, Cambridge, MA, pp 95–106
6. Goldman A (1989) Interpretation psychologized. Mind Lang 4:161–185
7. Goldman A (1992) In defense of the simulation theory. Mind Lang 7:104–119
8. Goldman A (1992) Empathy, mind, and morals: Presidential address. Sixty-Sixth Annual Pacific Division Meeting of the American Philosophical Association in Portland, Oregon, March 27, pp 17–41
9. Goldman A (1993) The psychology of folk psychology. Behav Brain Sci 16:15–28
10. Goldman A (1993) Philosophical applications of cognitive science. Boulder, Colo., Westview Press
11. Goldman A (2000) The mentalizing folk. In: Sperber D (ed) Metarepresentation. London, Oxford University Press
12. Goldman A (2005) Imitation, Mind Reading and Simulation. In: Hurley S, Chater N (eds) Perspectives on Imitation: From Cognitive Neuroscience to Social Science, vol 2. Cambridge, MA: MIT Press, pp 79–93
13. Gallese V (2003) The manifold nature of interpersonal relations: the quest for a common mechanism. Phil Trans R Soc London B, 358:517–528
14. Gallese V, Lakoff G (2005) The brain's concepts: The Role of the Sensory–motor System in Reason and Language. Cognit Neuropsychol 22:455–479
15. Gallese V (2001) The "Shared Manifold" Hypothesis: from mirror neurons to empathy. J Conscious Stud 8(5–7):33–50
16. Gallese V, Fadiga L, Fogassi L, Rizzolatti G (1996) Action recognition in the premotor cortex. Brain 119:593–609
17. Rizzolatti G, Fadiga L, Gallese V, Fogassi L (1996a) Premotor cortex and the recognition of motor actions. Cog Brain Res 3:131–141
18. Gallese V (2000) The acting subject: towards the neural basis of social cognition. In: Metzinger T (ed) Neural Correlates of Consciousness. Empirical and Conceptual Questions. MIT Press, Cambridge, MA, pp 325–333
19. Gallese V (2003) The roots of empathy: the shared manifold hypothesis and the neural basis of intersubjectivity. Psychopathology 36(4):171–180

20. Gallese V (2005) "Being like me": self-other identity, mirror neurons and empathy. In: Hurley S, Chater N (eds) Perspectives on Imitation: From Cognitive Neuroscience to Social Science, vol 1. Cambridge, MA: MIT Press., pp. 101–118
21. Gallese V (2005b) Embodied simulation: from neurons to phenomenal experience. Phenomenology and the Cognitive Sciences 4:23–48
22. Gallese V, Fogassi L, Fadiga L, Rizzolatti G (2002b) Action Representation and the inferior parietal lobule. In: Prinz W, Hommel B (eds) Attention and Performance XIX. Oxford University Press, Oxford, pp 247–266
23. Fogassi L, Ferrari PF, Gesierich B et al (2005) Parietal lobe: From action organization to intention understanding. Science 302:662–667
24. Umiltà MA, Kohler E, Gallese V et al (2001) "I know what you are doing": a neurophysiological study. Neuron 32:91–101
25. Kohler E, Keysers C, Umiltà MA et al (2002) Hearing sounds, understanding actions: Action representation in mirror neurons. Science 297:846–848
26. Gallese V (2003) A neuroscientific grasp of concepts: From control to representation. Phil. Trans. R Soc. London, B., 358:1231–1240
27. Fadiga L, Fogassi L, Pavesi G, Rizzolatti G (1995) Motor facilitation during action observation: a magnetic stimulation study. J Neurophysiol 73:2608–2611
28. Grafton ST, Arbib MA, Fadiga L, Rizzolatti G (1996) Localization of grasp representations in humans by PET: 2. Observation compared with imagination. Exp Brain Res 112:103–111
29. Rizzolatti G, Fadiga L, Matelli M et al (1996b) Localization of grasp representations in humans by PET: 1. Observation versus execution. Exp. Brain Res 111:246–252
30. Decety J, Grezes J, Costes N et al (1997) Brain activity during observation of actions. Influence of action content and subject's strategy. Brain 120:1763–1777
31. Hari R, Forss N, Avikainen S et al (1998) Activation of human primary motor cortex during action observation: a neuromagnetic study. Proc Natl Acad Sci USA 95, 15061–15065
32. Cochin S, Barthelemy C, Lejeune B et al (1998) Perception of motion and qEEG activity in human adults. Electroencephalogr Clin Neurophysiol 107:287–295
33. Iacoboni M, Woods RP, Brass M et al (1999) Cortical mechanisms of human imitation. Science 286:2526–2528
34. Buccino G, Binkofski F, Fink GR et al (2001) Action observation activates premotor and parietal areas in a somatotopic manner: an fMRI study. Eur J Neurosci 13:400–404
35. Gallese V, Keysers C, Rizzolatti G (2004) A unifying view of the basis of social cognition. Trends Cognitive Sci 8:396–403
36. Rizzolatti G, Craighero L (2004) The mirror neuron system. Ann Rev Neurosci 27:169–192
37. Ferrari PF, Gallese V, Rizzolatti G, Fogassi L (2003) Mirror neurons responding to the observation of ingestive and communicative mouth actions in the monkey ventral premotor cortex. Eur J Neurosci 17:1703–1714
38. Buccino G, Lui F, Canessa N et al (2004) Neural circuits involved in the recognition of actions performed by nonconspecifics: An fMRI study. J Cogn Neurosci 16:114–126
39. Watkins KE, Strafella AP, Paus T (2003) Seeing and hearing speech excites the motor system involved in speech production. Neuropsychologia 41:989–894
40. Ehrsson HH, Geyer S, Naito E (2003) Imagery of voluntary movement of fingers, toes, and tongue activates corresponding body-part-specific motor representations. J Neurophysiol 90:3304–3316

41. Cisek P, Kalaska J (2004) Neural correlates of mental rehearsal in dorsal premotor cortex. Nature 431:993–996
42. Gergely G, Csibra G (2003) Teleological reasoning in infancy: the naive theory of rational action. Trends Cogn Sci 7:287–292
43. Iacoboni M, Molnar-Szakacs I, Gallese V et al (2005) Grasping the intentions of others with one's owns mirror neuron system. PLoS Biol 3:529–535
44. Schubotz RI, von Cramon DY (2004) Sequences of abstract nonbiological stimuli share ventral premotor cortex with action observation and imagery. J Neurosci 24:5467–5474
45. Hauser MD, Chomsky N, Fitch WT (2002) The faculty of language: what is it, who has it, and how did it evolve? Science 298:1569–1579
46. Hauser MD, Fitch WT (2004) Computational constraints on syntactic processing in a non human primate. Science 303:377–380
47. Gallese V, Metzinger T (2003) Motor ontology: The representational reality of goals, actions, and selves. Philos Psychol 13:365–388
48. Metzinger T, Gallese V (2003) The emergence of a shared action ontology: building blocks for a theory. Conscious Cogn 12:549–571
49. Adolphs R, Damasio H, Tranel D et al (2000) A role for somatosensory cortices in the visual recognition of emotion as revealed by three-dimensional lesion mapping. J Neurosci 20:2683–2690
50. Adolphs R (2002) Neural systems for recognizing emotion. Curr Opin Neurobiol 12: 169–177
51. Adolphs R (2003) Cognitive neuroscience of human social behaviour. Nat Rev Neurosci 4:165–178
52. Lambie JA, Marcel AJ (2002) Consciousness and the varieties of emotion experience: A theoretical framework. Psychol Rev 109:219–259
53. Carr L, Iacoboni M, Dubeau MC et al (2003) Neural mechanisms of empathy in humans: a relay from neural systems for imitation to limbic areas. Proc Natl Acad Sci USA 100: 5497–5502
54. Wicker B, Keysers C, Plailly J et al (2003) Both of us disgusted in my insula: the common neural basis of seeing and feeling disgust. Neuron 40:655–664
55. Penfield W, Faulk ME (1955) The insula: further observations on its function. Brain 78:445–470
56. Krolak-Salmon P, Henaff MA, Isnard J et al (2003) An attention modulated response to disgust in human ventral anterior insula. Ann Neurol 53:446–453
57. Calder AJ, Keane J, Manes F et al (2000) Impaired recognition and experience of disgust following brain injury. Nat Neurosci 3:1077–1078
58. Adolphs R, Tranel D, Damasio AR (2003) Dissociable neural systems for recognizing emotions. Brain Cogn 52:61–69
59. Goldman A, Sripada CS (2005) Simulationist models of face-based emotion recognition. Cognition 94:193–213
60. Husserl E (1989) Ideas pertaining to a pure phenomenology and to a phenomenological philosophy, 2nd book: Studies in the phenomenology of constitution. Kluwer Academic, Dordrecht
61. Keysers C, Wickers B, Gazzola V et al (2004) A touching sight: SII/PV activation during the observation and experience of touch. Neuron 42:1–20
62. Blakemore SJ, Bristow D, Bird G et al (2005) Somatosensory activations during the observation of touch and a case of vision–touch synaesthesia. Brain 128: 1571–1583
63. Damasio AR (1994) Descartes' error. Putnam, New York
64. Damasio AR (1999) The feeling of what happens: body and emotion in the making

of consciousness. Harcourt Brace, New York

65. Goldman A, Gallese V (2000) Reply to Schulkin. Trends Cogn Sci 4:255–256
66. Husserl E (1977) Cartesian meditations. Kluwer Academic, Dordrecht
67. Zahavi D (2001) Beyond empathy. Phenomenological approaches to intersubjectivity. J Consc Stud 8:151–167
68. Gallese V, Goldman A (1998) Mirror neurons and the simulation theory of mind-reading. Trends Cogn Sci 12:493–501
69. Terenius L (2000) Schizophrenia: pathophysiological mechanisms–a synthesis. Brain Res Rev 31:401–404
70. Minkowski E (1927) La Schizophrénie. Psychopathologie des schizoïdes et des schizophrènes. Payot, Paris
71. Bleuler E (1911) Dementia praecox oder Gruppe der Schizophrenien. In: Aschaffenburg G (ed) Handbuch der Psychiatrie. Deuticke, Leipzig
72. Blankenburg W (1971) Der Verlust der natürlichen Selbstverständlichkeit. Ein Beitrag zur Psychopathologie symptomarmer Schizophrenien. Enke, Stuttgart
73. Parnas J, Bovet P (1991) Autism in schizophrenia revisited. Compr Psychiatry 32:7–21
74. Parnas J, Bovet P, Zahavi D (2002) Schizophrenic autism: clinical phenomenology and pathogenetic implications. World Psychiatry 1/3:131–136
75. Dawson G, Webb S, Schellenberg GD et al (2002) Defining the broader phenotype of autism: genetic, brain, and behavioral perspectives. Dev Psychopathol 14:581–611
76. Schmitz C, Martineau J, Barthélemy C, Assaiante C (2003) Motor control and children with autism: deficit of anticipatory function? Neurosci Lett 348:17–20
77. Oberman LM, Hubbard EH, McCleery JP et al (2005) EEG evidence for mirror neuron dysfunction in autism spectrum disorders. Cogn Brain Res 24:190–198
78. Theoret H. Halligan E, Kobayashi M et al (2005) Impaired motor facilitation during action observation in individuals with autism spectrum disorder. Curr Biology 15:84–85
79. Rogers S (1999). An examination of the imitation deficit in autism. In: Nadel J, Butterworth G (eds) Imitation in infancy. Cambridge University Press, Cambrige, pp 254-279
80. Snow ME, Hertzig ME, Shapiro T (1988) Expression of emotion in young autistic children. Annual Progress in Child Psychiatry & Child Development, 514–522
81. Yirmiya N, Kasari C, Sigman M, Mundy P (1989) Facial expressions of affect in autistic, mentally retarded and normal children. J Child Psychol Psychiatr 30:725–735
82. Hobson RP, Ouston J, Lee A (1988) Emotion recognition in autism: coordinating faces and voices. Psychol Med 18:911–923
83. Hobson RP, Ouston J, Lee A (1989). Naming emotion in faces and voices: Abilities and disabilities in autism and mental retardation. Br J Dev Psychol 7:237–250
84. Hobson RP, Lee A (1999). Imitation and identification in autism. J Child Psychol Psychiatr 40:649–659
85. Baron-Cohen S, Leslie AM, Frith U (1985). Does the autistic child have a “theory of mind”? Cognition 21:37–46
86. Baron-Cohen S (1988) Social and pragmatic deficits in autism: cognitive or affective? JAutism Dev Disord 18:379–402
87. Baron-Cohen S (1995) Mindblindness. An Essay on Autism and Theory of Mind. MIT Press, Cambridge, MA
88. Bird CM, Castelli C, Malik O et al (2004) The impact of extensive medial frontal lobe damage on ‘theory of mind’ and cognition. Brain 127:914–928
89. Millikan RG (2004) Varieties of meaning. Boston, MA: MIT Press, in press

90. Goodale MA, Milner AD (1992) Separate visual pathways for perception and action. Trends Neurosci 15:20–25
91. Milner D, Goodale MA (1995) The visual Brain in Action. Oxford University Press, Oxford
92. Jacob P, Jeannerod M (2003) Ways of seeing–the scope and limits of visual cognition. Oxford University Press

PART 3
The Dream

Chapter 12

The Dream in the Dialogue Between Psychoanalysis and Neuroscience

Mauro Mancia

Psychoanalysis Discovers the Dream

Dreams in Freud's Time

Freud opened the doors of science to the dream in 1900 [1]. His "discovery" grew from a model of the mind based on the concept of psychic energy behind man's desire. This has its roots in infancy and must be satisfied. Freud's thinking thus upturned the whole of the old order of the origin of dreams long held by the Greek "oneiromanciers" and the Medieval and Renaissance dream diviners. In place of the idea that dreams are sent to man by the gods or through supernatural forces, Freud offered the alternative that they come from man himself–from his unconscious mind. Not from on high, then, but from down below, on earth! By suggesting that dreams originated naturally "inside" man, Freud gave them an anthropological dimension, which opened the way to scientific investigation.

In 1895 [2], a period of great changes in Freud's scientific thinking, he shifted away from the exact sciences approach to venture onto the sands of the universe of the mind. This move paralleled a shift in personal identification: from Breuer, strict guardian of the scientific method, to Fliess, whose approach was less tightly bound to scientific rigor. The *Project for a Scientific Psychology* bears witness to this, as does the mind–brain relation model Freud set up as the foundation for his concept of the dream–to which he returned, modifying it, in Chapter 7 of the *Traumdeutung* (*The Interpretation of Dreams*).

Figure 1 summarizes Freud's mind/brain model (elaborated between 1895 and 1900) relative to the organization of the memory and the unconscious, and the role of the latter in the production of dreams. There is the

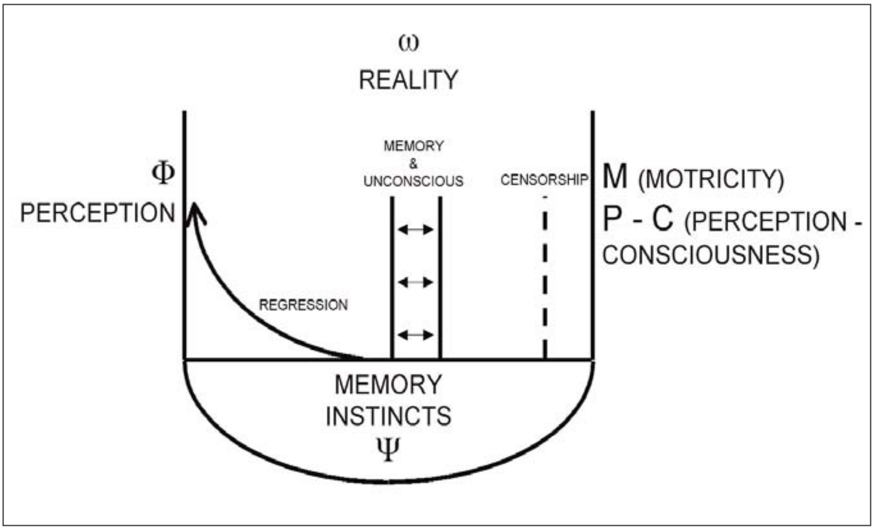

Fig. 1. Drawing representing the synthesis of the two models for the brain/mind relationship proposed by Freud in the *Project for a Scientific Psychology* (1895) and *The Interpretation of Dreams* (1900). For explanation, see the text

φ system, responsible for perception; the *ψ* system, containing memory and instincts; and the *ω* system, representing reality. On the right-hand axis Freud puts movement (*M*) and the perception–consciousness system (*P–C*). Outside stimuli reaching the gateway of perception (*φ*) run along the abscissa, reaching movement according to the reflex scheme drawn up by Sherrington [3]; at the same time, however, they are deposited in the memory, where they integrate themselves with the world of instincts to create the unconscious psychic system (*ψ*). In the waking state, reality (*ω*) guides progress along this path. In sleep, however, with the inhibition of movements and loss of contact with reality, the psychic energy built up in the *ψ* system cannot proceed towards *M*, and is obliged to regress towards the gateway of perception (*φ*) which, pushed from inside as it were, sets up a perception with no external object, i.e., a hallucination. Through this regressive path the hallucination can satisfy the repressed desire. On the basis of this model Freud formulated his definition of the *dream as a hallucinatory satisfaction of a desire repressed in infancy.*

In Chapter 7 of *The Interpretation of Dreams* Freud elaborates the model further with ordinates for the memory, the unconscious, and the censorship. Dreams not only provide hallucinatory satisfaction of a repressed desire: they enable the unconscious to pick its way through censorship to reach the P–C system. But to manifest itself to the conscious mind, the unconscious has to undergo the distortions and transformations imposed by the censorship. This is what causes the *rhetoric* of dreams, meaning the difference between its manifest and its latent contents. The censorship executes the main work of dreaming, preventing the unconscious from gaining direct access to the conscious mind. Censorship creates a dream as the primary event, but also makes us forget it. The primary event cannot be known as such, but we can gain access to the secondary event through narration which transforms the representations of the dream into a system with linguistic significance. Censorship, however, always has the main job in the work of dreams: condensation, displacement, symbolization, and dramatization.

Condensation involves space and time, helping to give the dream its typical feelings of estrangement and unreality, of being bizarre, absurd and "alien." Displacement is what arranges the content of the dream around features that are not its main lines, so the essential aspects of the dream come to look as though they are not really valuable. Displacement also involves the affects, but its most interesting aspect regards the language of the dream and its narration. This is linked to an associative syntagmatic chain by which the dream can generate a linguistic "swap-over," enabling one proposition to take the place of another. This swap benefits condensation too, and the whole work of the dream which, as a secondary process, is based on linguistic transformation of the primary representations. Freud had grasped this subtle aspect of dreams, where language becomes the crossroads where the meanings of the various representations meld and multiply.

Symbolization has particular weight in the dream, alongside displacement: one object is seen in place of another, acquiring a totally personal meaning linked to the dreamer's own affective history and his relational context. Symbolization has a natural part in the dream's metaphoric operations, making its language poietic. Closely related to symbolization is the dream's dramatization of the emotional and affective contents. The dream is thus like a *private theater* where the individual personages and parts in the *pièce* are united by a range of logical and affective relations. The mind stages this play in its dreams by transforming the individual representations, each with its own symbolic significance. Freud believed the dream, as a primary scene, was the mind's true nourishment, something it really needed. He maintained that the dream could stage more complex scenes than the theater, as several events could be presented at the same time. In this sense it was comparable to an artist depicting lots of people together in one painting, relating to each other (the *intrapsychic dimension* of the dream) but also to the person enjoying the work of art (the *intersubjective dimension*). Freud [1] introduces another concept fundamental to psychoanalytic theory into the dream: identification, in the sense that one can be hidden, by identification, behind another person.

The work of the dream is permeated with affectivity and builds a bridge to the events of infancy, making it possible to transcribe the contents of the memory from the child's earliest affective experiences. Freud defines this emotional recovery of memories *Nachträglichkeit*–a "rewriting of memory." The concept stretches to the implicit memory too and its specific transcription of experiences forming the structure of the unrepressed unconscious [4, 5]. We shall come back to these points later on. Freud's concept of *Nachträglichkeit* embodies what he considered the aim of work on dreams: to transcribe from a patient's childhood the history of traumatic events that might explain a neurosis. Thus, Freud proposes using the dream to rewrite the patient's true life story, as faithfully as possible. This meant he could base his dream-reconstruction work on the historical, autobiographic memory, and the events from the past irrupting in the analytical relation, even if in *Screen Memories* [6] he cast doubts on whether we have precise recollections of our infancy, or more likely "recollections" constructed on what we remember of it. He comes close here to the more recent idea of the implicit memory, from which we cannot recall events and which therefore cannot hide anything repressed. This is another point we shall come back to.

In 1937, in *Constructions in Analysis* [7], Freud goes back to the past surviving into the present as the basis for the analytical relation and the work of (re)construction of things forgotten, starting from the traces they have left behind. In the 1930s, then, he shifted back to his original definition of the dream–made 30 years earlier in 1895–1900–as the hallucinatory satisfaction of a desire repressed in infancy–though with some exceptions. His theory is still valid as a whole, but two conditions need reconsidering: dreams referring to traumatic experiences, and those evoking painful recollections of infancy. He thus admits that his theory may need modification, to permit the dream to be seen as an *attempt* at fulfilling a repressed desire. His theory is safe but cannot stand up to the evidence produced by advances in psychoanalytical thought.

Dreams Since Freud

The 1930s were an important time for psychoanalysis, as its paradigm shifted. Melanie Klein [8] burst onto the international psychoanalytical scene, radically transforming Freud's instinctual energy model, replacing it with a relational model. This metapsychological change of the mind wrought far-reaching changes in the concept of the dream and how it could be worked on in analysis. The importance of affective relations, linked primarily to primary experiences, suggested that it was not repression that triggered our mind to work in dreams, but more likely a dynamic relation between affective representations (internal objects) that have precipitated and layered themselves in the unconscious in early infancy but manifest themselves through the primary processes of splitting and projective and introjective identification.

Although not explicitly, the Kleinian unconscious that manifests itself in dreams is therefore no longer linked to repression but to the splitting and projective identification that Klein introduced into clinical work, and is one of the most important features of current psychoanalysis. Klein is also credited with giving the dream a major part in the economy of the mind, where it represents the various stages of development that can surface in the transference.

In line with Klein's metapsychological model, the interpretation of dreams takes on a *theological* dimension: it links up with the internal objects–the internal representations of parental figures that are "sacred" to the individual–becoming the gods and devils of his mental universe [9]. It is interesting to see that Umberto Eco [10] maintains that behind every strategy of the symbolic world there is a theology legitimizing it. This encouraged me to look for matches and similarities between dreams and religion, taking this as an internal process reflecting an individual's level of religious belief that enables him in dreams to *re-ligare*–to take a Latin root that means to *re-bind*–or link up in a complex relationship all the items with the greatest affective and emotional significance that have been layered down over the years in his inner world [9].

Thus the dream becomes an internal theater [11] where man's mind is represented by people (internal objects) relating among themselves (the *intrapsychic dimension*) who give rise to a meaning that is then carried out into the outside world and external relations (the *intersubjective dimension*) [12]. The theory of internal objects has had considerable heuristic utility, bringing values into the psychoanalytical dimension of the mind, making man responsible for the state of his internal and external objects, giving new meaning–basically relational–to the concept of unconscious fantasy, hence to the deepest significance of dreams. This shift at the top constituted an important movement in psychoanalytical thought, replacing Freud's instinctual energy model based on desire and its repression with a relational model based on more complex modalities in the organization of the personality and the mind's unconscious functions. From this starting point, work on dreams implies finding out the state of the patients' internal objects, their conflicts and defenses.

The theoretical model of the mind has thus been transformed and enriched, and the dream has acquired a new purpose, studied by the last generation of analysts [13, 14]: it has become a basic tool of knowledge. The proposed model can be considered *epistemological,* and is based on an elegant statement by Money-Kyrle [15], who said that if man is his representation of the world, and this is identified with his knowledge dimension, the dream–representing man's inner world–is itself a source of knowledge. In 1962 Bion [13] formulated a task for the dream, executed by the *alpha* function: this was to transform sensory, emotive, and emotional experiences reaching the mind during waking hours in the form of *beta* elements into thoughts in the dream. This turned the dream into a tool the mind uses to work over sensory experiences and convert them to thought. This permitted a valuable continuity of mental functions in the passage from waking (dominated by fantasies) to sleep (dominated by dreams). Bion overturned Freud's relation between dreams and unconscious, maintaining that censorship and personal resistances in the dream were not the product of the unconscious, but tools dreams use to create and distinguish the conscious from the unconscious.

Bion introduced an extremely interesting theoretical-clinical concept of the dream, which he called the *contact barrier,* a sort of semipermeable membrane that allowed an exchange between conscious and unconscious but also differentiated their functions in waking and sleep. The contact barrier is the expression of the utmost *plasticity* of the mind in the transference[1]. In fact, in analysis the dream sets in motion a process where the barrier continually shifts and changes, enabling the analyst and patient to grasp the various dimensions of the dream–intrapsychic and interpersonal–and interpret them differently. It is often thanks to this plasticity of the barrier that we can detect, in the dream before anywhere else, the patient's resistances and defenses, significant changes in his transference "temperature," and his relation with the analyst. The dream, placed in its right context in the analytical relation, through the transference, defines its

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[1] I have borrowed the term *plasticity* here from biology, where it indicates the synapses' ability to adapt.

cognoscitive dimension. We call this dimension *epistemological* in that it enables the mind to grow on the basis of the knowledge it has gained of itself and its own internal and external objects. But since this knowledge is governed by the strength and value of its own representations, we can conclude that the mind's cognoscitive function is governed by its *theology* (and the affects binding these "sacred" objects).

This epistemological aspect of the dream raises the question of man's *need* to dream, since dreaming is essential for knowledge of oneself and of one's objects, and the mind needs these to mobilize itself and grow. One of the purposes of the dream might therefore be to produce knowledge through the world of representations. Freud [1] had guessed this when he compared dreams to a newspaper under a dictatorship: it has to come out every night but, as it is not allowed to tell the truth directly, the truth must be masked between the lines. We can use the same metaphor, except that for us the dream that comes out every night has to tell the truth even if it is distorted, to show the dreamer's affective state; this is connected–as if by a bridge–to his early experiences, but at the same time reflects his relationship with the analyst as regards the dreamer's mind and his objects, and the analyst's mind with his own personal and countertransferential affects. Today, therefore, we can consider the dream a real experience that represents the dreamer's inner world in the immediate present, and therefore expresses the whole of the transference.

The dream, however, has an active and irreplaceable role in the *dramatization* of the relationship, building a line of thought that both the analyst and his patient can share. This involves bringing "on stage" the affects in play in the relation at that particular, fleeting moment. From this viewpoint the transference is a *total relational situation* [16], shifted from the past to the present, but at the same time it projects the dynamics between internal objects into the analytical relationship. It enables the patient to represent through a dream and communicate through its narration the current state of his internal objects, in relation to the analyst, and its connections with his own primary affective life. The analyst can use the dream to recognize the splits, identifications, denials, idealizations, fears and defenses, aggressions and seductions activated by the patient, and can employ them in work on the dream. This work involves various parts: interpretation, decoding the manifest and translating it to the latent, exploring, hypotheses, second thoughts, re-working, moves and waiting like in chess: all with the aim of gaining knowledge–and making the patient gain knowledge–of the internal objects and their dynamics in relation to the patient's earliest experiences, the defenses set up in response to the pressures of the transference, and action towards the outside world. This complex work on dreams is possible thanks to the relational context that has its part in the organization of the dream.

Along this line, which is where psychoanalysis is going right now, we can view the dream as an event drawing on the mental life of both the patient and the analyst; not only does the analyst have to interpret the patient's symbols, but the process of symbolization itself is the result of the meeting of the two people, working to create a new, shared meaning for the dream [12, 17]. Although it is the patient who dreams–though occasionally the analyst does too!–there is more than just one truth to be uncovered, exclusive to the patient: there is a truth belonging to both of them that enables them to achieve an affective harmony–or dissonance–like that bonding the mother and child [18]. The patient must be reached wherever–and to whatever extent–he can bear it. Every dream carries a truth that must be grasped by both people involved on its two levels: *intrapsychic,* where dynamically related parts of the self are identified with objects in the dream, and *intersubjective,* where parts of the self are identified with the object–the analyst in the transference.

Taken in the right context and viewed in the here and now of the analytical setting, the dream is a most valuable aid for grasping a given moment in the transference, selecting the emerging affects, and assembling the pieces of the relational mosaic into a *construction* on which to attempt an interpretation. At the same time, however, dreaming makes it possible to bring early experiences to the surface and give them new meaning to "up-date" them to the present. Thus the dream can be seen as the most credible and reliable tool in what Freud called *Nachträglichkeit,* referring to the reassigning of significance to some past experience, even if it was preverbal and presymbolic, by rewriting the memory (with or without the actual recollection). This is the true work of *reconstruction* on dreams [5, 19].

The Dream in Psychoanalysis Today

The discovery of the implicit memory and the unrepressed early unconscious [4, 5, 20] and their connections with the experiences of early infancy–sometimes traumatic–has given the dream another, particularly valuable, dimension in theory and clinical practice. Here we must look at the child's earliest relations with its mother and the environment where it grows up [21]. From late in pregnancy the fetus is in direct contact with its mother's rhythms–her heartbeat and breathing–and her voice [22], storing them in its memory. They serve as important relational references at birth [23]. The newborn is highly sensitive to its mother's tone of voice and, gradually, also to the prosodic structure of her language [24].

After the voice, the child's body is an important area of interaction with the mother. How she contains, touches, watches, and speaks to the baby, and her capacity for *rêverie,* are basic vehicles of affects in this early relation. This presymbolic and preverbal experience, and the affects, fantasies, and emotions they evoke in the newborn, are all filed in the implicit memory, where they help build up the main structures of an unrepressed, unconscious nucleus of the self. They cannot be repressed because the structures of the explicit memory, needed for repression, do not mature until a child is about 2 years old [25]. We must therefore imagine that the child's unconscious does not allow recollections, but nevertheless influences his whole affective, emotional, and cognitive life [4, 5].

The dream is so important because, besides the various modalities of transference that rely on the patient's voice and language (which I call the "musical dimension" of the transference), it renders symbolic experiences that were originally presymbolic and preverbal, so they can be expressed verbally and thought about. To this extent the dream, through its representations, can create psychic figurability [26], helping fill the representation gap of the unrepressed unconscious.

This new function of the dream not only opens up the transference in the immediate present, helping with the work of *construction* in analysis, but also allows a *reconstruction* that enables the patient to transcribe his life story, as it relates to his identity, and to recuperate the "history" of his unconscious from his earliest significant preverbal and presymbolic experiences, which he could never recollect[2]. This involves some broadening of Freud's *Nachträglichkeit,* which he conceived as a "transcription" of the memory of events in the distant past connected to the patient's life story, hence to his explicit memory. This therefore involved reconstruction of an event that had really taken place, brought to the surface through the recollection, using the dream, which is the best possible means of de-repression.

We are now in a position to extend this concept of *Nachträglichkeit* to the implicit memory too, hence to a process of transcription of unrepressed emotional experiences that cannot be recalled but which have marked the infant from its very first encounters with its mother. In this

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[2] For my definition of the work of *construction* and *reconstruction*, see Mancia [27].

light, *Nachträglichkeit* includes the early events that Freud [28] wrongly believed were subject to primary (originary) repression, but that today we consider as laying the groundwork, together with fantasies and defenses, for an early, unrepressed unconscious that can influence the infant's mind and subsequent processes of repression.

To sum up, therefore, psychoanalysis currently sees the dream not only as a window open onto the transference [29], or as a real experience that represents the dreamer's inner world in the immediate present (thus expressing the transference as a whole [16,19]), but also as a symbolic transformation of a presymbolic happening that the patient can re-live emotionally in analysis, even without the actual recollection. In addition, the dream creates images, mentally compensating for the lack of figurative coverage, filling the gaps in the dreamer's affective and emotional history linked to his earliest experiences, filed away in the implicit memory (see Chapter 3, this volume).

The Neurosciences Contribute to Dreams

At the beginning of the last century, when Freud had already published his *Interpretation of Dreams,* European psychology seemed more interested in sleep than in dreaming. At that time Henry Pieron [30] defined sleep as a physiological state that was periodically necessary, its cyclic pattern relatively independent of outside conditions, during which there was a complete break in the complex sensory and motor relations between the subject and his environment. This past century has in fact seen great steps forward in understanding the neurophysiology of sleep, which Pavlov [31] pioneeringly proposed was an *active* form of cortical inhibition, though later it was again considered a *passive* event induced by either sensory deafferentation [32] or functional disactivation of the ascending reticular system [33, 34]. However, there was evidence that it was an *active* state produced by sleep-inducing caudal reticular systems [35, 36], or by preoptic basal forebrain structures [37].

It was only in 1953 that neurophysiologists started taking any real interest in the mental aspects of sleep. This was when Aserinsky and Kleitman [38] observed a paradoxical phase, which we now know as REM sleep, opening the gates of dream to physiologists and experimental psychologists. These researchers noted that children sleeping deeply presented eye movements visible under their eyelids, with twitching of the limbs, and a desynchronized EEG tracing like during waking. This phase therefore became known as paradoxical, usually referred to by the abbreviation for *rapid eye movements–REM*.

Neurophysiological research in the 1960s [39] clarified the electrophysiological parameters and structures responsible for REM sleep, distinguishing it from the non-REM phases: EEG desynchronization, atony of postural muscles, rapid eye movements, monophasic waves in the visual system, known as ponto-geniculo-occipital (PGO) waves, and neurovegetative turbulence, involving respiratory and cardiac arrhythmias and changes in blood pressure. The endocrine system too showed significant changes during REM sleep. The cholinergic reticular organization of the pons was found to be responsible for the ascending and descending events of this important phase, and is therefore the *deus ex machina* of REM sleep.

Around the same time Kleitman's group [40, 41] studied mental activity during sleep in healthy volunteers, waking them during REM or non-REM phases. When they were woken at the end of an REM episode they often reported an experience that it seemed logical to classify as a dream, with rich perceptive–hallucinatory detail and strong emotional participation of the dreamer, with self-representations. When they were woken outside the REM phases their mental activity consisted only of fragments of reality and thought, not organized like in a dream. The physiological basis was thus laid for a dichotomic REM/non-REM model as the electrical equivalent of different types of mental activity. Subsequently the duration of each REM episode was correlated with the amount of "dream material" narrated [42], the amount of eye movements with the content of the dream [43], and the specific direction of the eye movements with the spatial organization of events in the dream [41, 44].

These psychophysiological investigations led to the idea that REM sleep was the neurophysiological basis, or "neurobiological frame" within which dreams could be organized. The EEG desynchronization itself reflects neocortical activation that is a basis for perceptive and cognitive activity and for activation of the memory, all events common to dreaming. The eye movements were interpreted as the motor equivalent of hallucinatory activity that would be enough to create a dream-space. The monophase PGO waves recorded in the visual system were considered to be the electrical reflection of a form of decoding and reading information arising within the nervous system, which the dreamer then lived as visual hallucinations [45].

Psychophysiological research during REM sleep subsequently showed that the geometric–spatial and emotional aspects of the dream are mainly organized in the right hemisphere [46, 47], while the left hemisphere is involved more in its narration. More sophisticated observations, taking account of the experimental setting, the dreamers' expectations, and how they narrated their dreams on waking, showed psychophysiologists that there was complex mental activity not only in the REM phase, but also while falling asleep and in non-REM sleep. The different results reflect methodological differences but also how the dream is interpreted. Starting out from an extremely broad definition like that given by Fromm [48], according to which any mental activity during sleep can be considered a dream, we may progress to more restrictive ones such as that proposed by Bosinelli and Franzini [49], who describe a dream as a mental experience involving estrangement and unreality, vivid perception, personal participation of the dreamer but inability to examine reality, and loss of voluntary control of the thought process. If you ask a young sleep volunteer to tell you everything that passed through his mind just before waking, a high percentage of those woken in either REM or non-REM sleep describe an experience that we hardly hesitate to call a dream. While falling asleep, too, one goes through a hallucinatory state with the same sort of mental images as in other sleep phases. Thought becomes fragmented as voluntary control of reality is gradually lost. This allows bizarre, regressive contents to intrude, laying the foundations for dreams. These contents grow into the visual and auditory images that build up the hypnagogic hallucinations amply described in the literature [50].

An analysis of the mental activities that go on as one falls asleep, however, finds differences from those in other sleep phases: the spatial structuring of the dream-like state is different, and so is the level of the dreamer's personal participation. Antrobus [51] suggested that the number of words used to narrate the dream(s) might be the most efficient basis for analyzing the psychological differences in the two phases, REM and non-REM sleep. Another might be the number of representations of the dreamer himself in REM sleep compared to the falling asleep stage [52].

Current psychophysiological findings confirm that the human mind produces dream-like experiences in all phases of sleep, the characteristics and contents in the REM or non-REM phases depending more on the length of the "story," and hence the number of words needed to narrate it, than on the quality of the happenings. Antrobus [53] too agreed that people dream in all phases of sleep, though only REM sleep offers the conditions of cortical activation that guarantee enough recovery of memory for long narrations of the scenarios. The rapid cortical activity found in REM sleep (40 Hz) [54] suggests there may be more room in this phase for cognitive activity. Descriptions from volunteers who have been woken up also present a higher degree of linguistic organization after REM sleep than after the other phases.

It does appear, therefore, that the lower level of cortical activation in non-REM sleep leads to dreams with less capacity for elaborating on material stored in the memory, and the dreamer has less capacity for narrating them. This research shows up differences in cognitive functions during sleep: people are more able to remember and describe the experiences of REM sleep; they use more words to tell the dreams, whose content tends to be stranger and more bizarre. Cognitivists such as Antrobus [51], Foulkes [55], and Cavallero [56] consequently consider REM sleep as the phase most likely to produce dreams, for several reasons: (a) the cerebral cortex is strongly activated, like in waking, so it can recuperate memories more easily; (b) the associative cortex participates in the symbolic organization of the experiences typical of dreams; (c) the activated brain is capable of generating "multimedia" happenings, like during waking. However, since people dream in REM and non-REM sleep–though to different extents–Bosinelli's group suggested that the brain may have a *single system* for dream production active, in different measure, in all phases, from falling asleep to waking up. This theory, however, has its opponents, who insist that there are *qualitative* differences, in the level of bizarreness and the more emotional aspects, between dreams in the various phases of sleep; they suggest that dreams are generated by *two mechanisms* that operate separately in the brain in REM or non-REM sleep. This brings us back to the old REM/non-REM dichotomy, though in a blander form. There is also a third idea, according to which "undetected" REM-like processes are active in non-REM sleep, producing dreams [57].

It is clear in any event that our mind does not rest, even during sleep. The mental activity changes, basically because the dreamer's ego has a different relation with reality. Perceptive reality is replaced with hallucinatory reality, with self-representations and strong emotional participation in the dream events. Although there is no substantial difference between mental events during the stages of falling asleep, REM and non-REM sleep, psychophysiological research confirms that dream-activity in REM sleep is more lively, with greater reference to the contents of the memory, more bizarre imagery, and a longer narration than in the other biological frames of sleep.

In view of the greater role of REM sleep in dream organization, Hobson and McCarley [58] and Hobson et al. [59] proposed what they called an *activation-synthesis* hypothesis as a brain mechanism for producing dreams. They suggested that in REM sleep the brain is actually a generator of the dream state; the motor is the pons, which stimulates the brain from inside, producing information that is projected to the forebrain and limbic system, which then elaborate it to recover recollections stored in the explicit memory, so as to build the plot of the dream and ensure the dreamer's emotional participation.

This theory implies that: (a) the primary energy responsible for the dream is physiological, produced internally by the pontine generator, determined genotypically rather than psychologically; (b) elaboration of the information from the pons (*deus ex machina* of the dream) and its synthesis is done by the forebrain, aimed at organizing the perception in the form of a hallucination, self-representation, the emotions these elicit, recovery of experiences from the memory, and the cognitive elaboration typical of dreams; (c) the brain in REM sleep can be compared to a sophisticated computer looking for key words, to integrate phenotypic data obtained through experience with genotypic stimuli; (d) the content of the dream that makes its way through our consciousness is neither unconscious nor deformed by censorship, as Freud would have had us believe, but is a *chaotic process* of self-activation starting from the pons, where the content of the dream originates, with all its bizarreness, and the other features Freud called the work of dreams.

Hobson et al. [60] have now suggested that (a) visual hallucinations are due to self-activation of the visual part of the brain, due to output from the pons which also activates the parietal cortex needed for spatial organization of the dream; (b) the emotions in a dream are due to activation of the amygdala which involves other limbic and paralimbic structures; (c) oneiric delusion, loss of self-awareness, and the illogical experiences of a dream are due to aminergic de-modulation and inhibition of the dorsolateral frontal cortex. This led Hobson [61] to radicalize this concept, stating that the mind during dreaming is simply the self-activated brain.

Bioimaging has supplied interesting information on the activation of various brain areas during REM sleep. On the assumption that this was the phase when people dreamed most, some researchers directly correlated activation and inhibition in certain brain areas with the production of dreams. This resulted in a significant neuropsychological contribution that has cast light on the brain areas and structures involved in dream organization. In humans, positron emission tomography (PET) shows the following areas activated during REM sleep: the pontine tegmentum, the amygdaloid nuclei on both sides, the left thalamus, the cingulate cortex, and the right parietal operculum, this last region being important for spatial construction; limbic activation might be the neurophysiological substrate for the emotional components of the dream [62]. Earlier, Braun et al. [63] had helped make a structural distinction between the mechanisms of waking and of REM sleep, confirming the activation in REM sleep of limbic and paralimbic areas, including the insula, the cingulate cortex, and the cortex of the medial temporal lobe. Later, the same group [64] observed increased activity during REM sleep in the hippocampus and parahippocampal gyrus, and the extrastriate cortex. The dorsolateral prefrontal cortex, striatum, and orbital cortex are all de-activated during this phase of sleep.

In a study to demonstrate the importance of REM sleep in the process of memorization, Maquet et al. [62] noted that waking experiences influenced specific brain areas during subsequent sleep. In particular, the brain areas activated during a waking-hour task were significantly more active during REM sleep, indicating that memory traces are processed during this phase in man. However, Huber et al. [65] maintained that specific areas of the cerebral cortex were also able to memorize a sensorimotor experience from the daytime waking hours during that night's synchronous sleep. This suggests that, while dreaming in REM or other phases of sleep, a person can recover events from his waking hours from his memory. This is what Freud [1] called daytime residues. These results are particularly interesting when compared with more recent findings by Anderson et al. [66]. They found that *voluntarily forgetting* mental experiences, a process comparable to Freudian repression,[3] is accompanied by increased activity in the dorsolateral prefrontal areas and a parallel reduction in hippocampal activity. This pattern is the opposite of that in dreaming (in REM sleep), where hippocampal activity increases and dorsolateral frontal activity diminishes [64]. This recent picture holds out hope that neuropsychology using bioimaging will provide some satisfactory explanation for repression [28], which sets up the dynamic unconscious [68], and of the opposite process, "return of the repressed" (or derepressed) which, as lost material is dug out of the memory, permits dreams to surface.

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[3] One objection might be that Freudian repression is unconscious whereas repressing by "forgetting" is conscious. Anderson et al. [66] might therefore be referring more to this conscious model than to true repression. However, in a footnote to their paper, Anderson et al. observe that some authors [67] maintain that the strict distinction between unconscious and conscious repression was in fact a distortion of Freud's theory, upheld by Anna Freud. Freud himself used the term to indicate either an unconscious or a conscious process.

Neuropsychological investigations using bioimaging on patients with brain lesions [69] found that dreams and REM sleep develop in separate parts of the brain: dream organizers are not regulated only by the pons, since patients with extensive lesions to this region still dream. However, lesions to the forebrain and corresponding associative cortices prevent dreams. People with lesions to the temporo-occipital associative cortex still dream, though with some loss of the hallucinatory component, while patients with damage to the limbic structures cannot distinguish between dreams and reality, and may live a virtually continuous dream-like existence. Solms [70] suggested that there may be a dissociation between dreams and various brain activation states. People do in fact dream when dopaminergic circuits in the ventromedial anterior brain are activated, which might explain the genital reactions in males and females during REM sleep [45].

More recently, in a synthesis of various clinical experiments, Solms [71] stated that: (a) dreams and REM sleep can be dissociated, as dreaming is linked to forebrain mechanisms that do not govern REM sleep; (b) the forebrain structures responsible for dreaming are the anterior and lateral hypothalamic areas, the amygdaloid complex, the subventral striatal areas, and the cortical areas of the occipitofrontal limbic system, the anterior cingulum and the insula; (c) the primary visual areas and the dorsolateral prefrontal cortex are de-activated during dreaming in REM sleep; (d) dream imagery is not due to chaotic activation of the pons, but to forebrain organization that builds up the cognitive process. This mechanism is generated by a dopaminergic circuit that can be activated in any phase of sleep. Bischof and Bassetti [72] described the total abolition of dreaming in a 73-year-old woman with focal lesions to the temporo-occipital areas, more severe in the right hemisphere. These findings suggest that disruption of the dopaminergic circuit involving temporo-occipital areas, particularly in the right hemisphere, may abolish dreaming; this would be an expression of a disconnection syndrome [73].

A Synthesis in the Functional Significance of Dreams

The neurosciences have for many years focused their investigations more on the functional significance of sleep than dreams. Sleep is in fact essential for normal biological vital functions, and long periods of sleep deprivation can lead to organic loss of condition with severe degeneration of organs such as the heart and kidneys, and even death in animals such as the monkey [74]. Sleep, from the evolutionary viewpoint, is certainly part of an individual's adaptation to his environment. An example is the dolphin, whose synchronous sleep alternates between the two hemispheres, and who has no REM sleep, for reasons of adaptation and survival [75].

It is generally believed that non-REM sleep is needed for metabolic activity, homeostasis, and thermoregulation, while REM sleep–which interests us more here in relation to mental activity–is required to elaborate and memorize information the brain has received during waking hours. During development, starting with the fetus, REM sleep acts as an endogenous stimulus to synaptogenesis and maturation, particularly of the neocortex. It is also necessary for restoration of the catecholaminergic system. This is interesting, since the catecholamines help regulate mood. Both REM and non-REM sleep can stimulate the synaptic structures that are inadequately activated during waking [76]. A structural hypothesis suggests that REM sleep may function as an information "processor," particularly for the right-hand hemisphere, which is considered the seat of the emotions [77] and is more involved than the left hemisphere in dream organization [47]. In addition, REM sleep is believed to permit recurrent, periodic genetic re-transcription [78].

Psychoanalytical assumptions, based on theoretical and clinical considerations, point to the dream as an essential tool for bringing past events up from the memory and activating unconscious functions of the mind. This implies the simultaneous action of two major memory systems to recuperate repressed and unrepressed experiences stored in the memory. For the *repressed unconscious* this would mean de-activation of the dorsolateral prefrontal areas and activation of the hippocampus, to recollect experiences from the explicit memory. For the *unrepressed unconscious,* it might mean activation of the amygdala and parieto-temporo-occipital areas–particularly in the right hemisphere–which help reach back to presymbolic and preverbal experiences, with their strong emotional component, in the implicit memory, rendering them symbolizable so that they can be brought up to the level of thought and expressed verbally, even without being remembered.

From the strictly psychological and psychoanalytical viewpoints, it is not acceptable to reduce the dream merely to a chaotic phenomenon with a straightforward physiological explanation. Dreams are produced through activation and de-activation of specific brain areas responsible for the memory (explicit and implicit), forebrain organization of a dopaminergic circuit, and the involvement of the whole of the cerebral cortex. They are in any case a mental function that is of interest mainly to psychology and psychoanalysis even though their roots are obviously neurophysiological. The dream can be described as a pictorial representation in the mind, made possible by a specific physiological organization of the brain and achieved through organized internal activation, which is not a chaotic process. This is an obligatory mental event, teeming with significance derived from the dreamer's affective and emotional history, stored in the implicit and explicit memory and rooted respectively in the unrepressed and repressed unconscious.

These considerations combine in the idea that the dream can only be a product of the brain operating independently and autonomously while it is disconnected from perceptive reality. But within the neurobiological framework, which is the same for all humans, the contents and forms of dreams, their thoughts, perceptions, and emotions, are different for every individual. This permits a specific type of bridge-function to link a person's earliest experiences connected to the implicit and explicit memory to the current affects and emotions activated in the transference. In analysis, these memories form part of the transference, brought to light in the *here and now* of the analytical relation. They permit the work of construction and reconstruction on the patient's affective and emotional history, from infancy.

The question of the functional significance of dreams definitely belongs more to psychoanalysis than neuroscience. The dreams psychoanalysts deal with are different from those neuroscientists investigate: neurophysiology's main focus is on the mechanisms that produce the various physiological phases of sleep, whereas psychophysiology concentrates more on understanding the specific mental activities arising in the sleep phases the neurophysiologists have identified. Neuropsychology employs bioimaging techniques to investigate the brain structures and areas needed to produce dreams. This has considerably helped psychoanalysis too, indicating which parts of the brain are activated or de-activated during REM sleep, and furthering understanding of the implicit and explicit memory systems. Only psychoanalysis, however, is really interested in the significance of the dream, in relation to the intrapsychic and intersubjective context. Like a microscope focused on psychic reality, the dream reveals the dynamics between internal and relational objects, that link the subject to reality and the world. It is a tool for achieving adequate knowledge of the unconscious human mind, making it possible to trace the dreamer's emotional history from his earliest infancy and create a historical context for his unconscious. The dream is thus indispensable for the mind's "economy" as it permits an objective formulation of subjective experiences and emotions, dramatizing them in each person's private theater, re-working and recuperating them from the earliest stages of the mind's development.

References

1. Freud S (1900) The interpretation of dreams. SE 4–5
2. Freud S (1895) Project for a scientific psychology. SE 1
3. Sherrington C (1952) The integrative action of the nervous system. Cambridge University Press, Cambridge (originally published 1906)
4. Mancia M (2003) Dream actors in the theatre of memory: their role in the psychoanalytic process. Int J Psychoanal 84:945–952
5. Mancia M (2004) Sentire le parole. Archivi sonori della memoria implicita e musicalità del transfert. Bollati Boringhieri, Torino
6. Freud S (1899) Screen memories. SE 2
7. Freud S (1937) Constructions in analysis. SE 23
8. Klein M (1932) The psycho-analysis of children. Hogarth Press, London
9. Mancia M (1987) Il sogno come religione della mente. Laterza, Rome–Bari
10. Eco U (1981) Voce simbolo. In: Enciclopedia, vol 12. Einaudi, Torino, pp 877–915
11. Resnik S (1982) Psicosi, gruppi, istituzioni. Quaderni di psicoterapia di gruppo 2. Borla, Rome
12. Meltzer D (1984) La vita onirica. Una revisione della teoria e della pratica psicoanalitica. Borla, Rome 1989
13. Bion WR (1962) Learning from experiences. Heinemann, London
14. Money-Kyrle R (1978) The collected papers of Roger Money-Kyrle. Duckworth, London
15. Money-Kyrle R (1961) Man's picture of his worlds. Duckworth, London
16. Joseph B (1985) Transference: the total situation. Int J Psychoanal 66:447–450
17. Nissim Somigliano L, Robutti A (eds) (1992) L'esperienza condivisa. Saggi sulla relazione psicoanalitica. Cortina, Milan
18. Stern DN (1985) The interpersonal world of the infant. Basic Books, New York
19. Mancia M (2004) Il sogno e la sua storia. Marsilio, Venice
20. Mancia M (2003) Implicit memory and unrepressed unconscious: their role in creativity and transference. Israel Psychoanal J 3:331–349
21. Vallino D, Macciò M (2004) Essere neonati. Borla, Rome
22. Mancia M (1981) On the beginning of mental life in the foetus. Int J Psychoanal 62:351–357
23. De Casper AJ, Fifer WP (1980) Of human bonding: newborns prefer their mothers' voices. Science 208:1174–1176
24. Mehler J, Bertoncini J, Barriere M, Jassik-Gerschenfeld D (1978) Infant recognition of mother's voice. Perception 7:491–497
25. Siegel SJ (1999) La mente relazionale. Neurobiologia dell'esperienza interpersonale. Cortina, Milan

26. Botella C, Botella S (2001) La figurabilité psychique. Colloquium Champs Psychanalytiques, Delachaux et Niestlé, Lausanne
27. Mancia M (1993) In the gaze of Narcissus. Karnac Books, London
29. Mancia M (2000) Il sogno: una finestra aperta sul transfert. Riv Psicoanal 46:225–268
30. Pieron H (1913) Le problème physiologique du sommeil. Masson, Paris
31. Pavlov I (1915) Donnée sur la physiologie du sommeil. C R Soc Biol Paris 79:1079–1984, 1916
32. Bremer F (1935) Cerveau isolée et physiologie du sommeil. C R Soc Biol Paris 18:1235–1241
33. Moruzzi G, Magoun HW (1949) Brain stem reticular formation and activation of the EEG. Electroencephalogr Clin Neurophysiol 1:455–473
34. Moruzzi G (1972) The sleep-waking cycle. Ergeb Physiol 64:1–165
35. Batini C, Moruzzi G, Palestini M et al (1959) Effects of complete pontine transection on the sleep-wakefulness rhythm: the midpontine pretrigeminal preparation. Arch Ital Biol 96:1–12
36. Cordeau JP, Mancia M (1959) Evidence of an electroencephalographic synchronization mechanism originating in the lower brain stem. Electroencephalogr Clin Neurophysiol 11:551–564
37. Sterman MB, Clemente CD (1962) Forebrain inhibitory mechanisms: cortical synchronization induced by basal forebrain stimulation. Exp Neurol 6:91–102
38. Aserinsky E, Kleitman N (1953) Regularly occurring periods of eye motility, and concomitant phenomena during sleep. Science 118:273–274
39. Jouvet M (1962) Recherches sur les structure nerveuses et les mécanismes responsables des différentes phases du sômmeil physiologique. Arch Ital Biol 100:125–206
40. Dement WC, Kleitman N (1957) The relation of the eye movements during sleep to dream activity: an objective method for the study of dreaming. Exp Psychol 53:339–346
41. Dement WC (1965) Does rapid eye movements sleep have a function? In: Jouvet M (ed) Aspects anatomo-fonctionnels de la physiologie du sômmeil. A Symposium. CNRS, Paris, pp 567–604
42. Wolpert EA, Trosman H (1958) Studies in psychophysiology of dreams. I. Experimental evocation of sequential dream episodes. Am Med Assoc Arch Neurol Psychiatr 79:603–606
43. Dement WC, Wolpert EA (1958) Interrelation in the manifest content of dreams occurring on the same night. J Nerv Ment Dis 126:568–578
44. Molinari S, Foulkes D (1969) Tonic and phasic events during sleep: psychological correlates and implications. Percept Mot Skills 29:343–368
45. Mancia M (1996) Sonno e sogno. Laterza, Rome-Bari
46. Antrobus J, Ehrlichman H, Wiener M, Sollma H (1983) The REM report and the EEG: cognitive processes associated with cerebral hemispheres. In: Koella W (ed) Sleep 1982. Karger, Basel, pp 49–51
47. Bertini M, Violani C (1984) Cerebral hemisphere, REM sleep and dreaming. In: Bosinelli M, Cicogna P (eds) Psychology of dreaming. Clueb, Bologna, pp 131–135
48. Fromm E (1951) The forgotten language. Holt, New York
49. Bosinelli M, Franzini C (1986) Psicofisiologia del sonno. In: Stegagno L (ed) Problemi di psicofisiologia. Boringhieri, Torino
50. Bosinelli M (1991) Il processo di addormentamento. In: Bosinelli M, Cicogna P (eds) Sogni: figli d'un cervello ozioso. Bollati Boringhieri, Torino, pp 249–270

51. Antrobus J (1983) REM and NREM sleep reports: comparison of world frequencies by cognitive classes. Psychophysiology 5:562–568
52. Bosinelli M, Cicogna P, Molinari S (1974) The tonic-phasic model and the feeling of self-participation in different stages of sleep. Int J Psychol 1:35–65
53. Antrobus J (1986) Dreaming: cortical activation and perceptual thresholds. J Mind Behav 7:193–212
54. Llinas R, Ribary U (1993) Coherent 40Hz oscillation characterizes dream state in humans. Proc Natl Acad Sci USA 90:2078–2081
55. Foulkes D (1985) Dreaming: a cognitive-psychological approach. Erlbaum, Hillsdale, NJ
56. Cavallero C (1991) Recenti sviluppi dello studio sperimentale sul sogno: l'approccio cognitivista. In: Bosinelli M, Cicogna P (eds) Sogni: figli d'un cervello ozioso. Bollati Boringhieri, Torino, pp 354–71
57. Nielsen TA (2003) A review of mentation in REM and NREM sleep: "Covert" REM sleep as a possible reconciliation of two opposing models. In: Pace-Schott EF et al (eds) Sleep and dreaming. Scientific advances and reconsiderations. Cambridge University Press, Cambridge, pp 59–74
58. Hobson JA, McCarley RW (1977) The brain as a dream generator: an activation synthesis hypothesis of the dream process. Am J Psychiatr 134:1335–48
59. Hobson JA et al (1998) The neuropsychology of REM sleep dreaming. Neuroreport 9:R1-R14
60. Hobson JA, Pace-Schott EF, Stickgold R (2003) Dreaming and the brain: toward a cognitive neuroscience of conscious states. In: Pace-Schott EF et al (eds) Sleep and dreaming. Scientific advances and reconsiderations. Cambridge University Press, Cambridge, pp 1–50
61. Hobson JA (2002) Dreaming. Oxford University Press, Oxford
62. Maquet P et al (2000) Experience-dependent changes in cerebral activation during human REM sleep. Nature Neurosci 3:831–836
63. Braun AR, Balkin TJ, Wesensten NJ et al (1997) Regional cerebral blood flow through the sleep-wake .cycle. Brain 120:1173–97
64. Braun AR, Balkin TJ, Wesensten NJ et al (1998) Dissociated pattern of activity in visual cortices and their projections during human rapid eye movement sleep. Science 279:91–95
65. Huber R, Ghilardi MF, Massimini M, Tononi G (2004) Local sleep and learning. Nature 430:27–28
66. Anderson MC, Ochsner KN, Kuhl B et al (2004) Neural systems underlying the suppression of unwanted memories. Science 303:232–237
67. Erdelyi MH (2001) Defense processes can be conscious or unconscious. Am Psychol 56:761–762
68. Freud S (1915) The unconscious. SE 14
69. Solms M (1995) New findings on the neurological organization of dreaming: implications for psychoanalysis. Psychoanal Q 44:43–67
70. Solms M (1999) Dreaming and REM sleep are controlled by different brain mechanisms. Annual Meeting of the American Academy for the Advancement of Science, Anaheim, CA, 25 March 1999
71. Solms M (2003) Dreaming and REM sleep are controlled by different brain mechanisms. In: Pace-Schott EF et al (eds) Sleep and dreaming. Scientific advances and reconsiderations. Cambridge University Press, Cambridge, pp 51–58
72. Bischof M, Bassetti CL (2004) Total dream loss: a distinct neuropsychological dysfunction after bilateral PCA stroke. Ann Neurol 56:583–586

73. Geschwind N (1965) Disconnection syndromes in animals and man. Brain 88:237–274, 585–644
74. Mancia M, Desiraju T, Chhina GS (1968) The monkey split brain-stem: effects on the sleep-wakefulness cycle. Electroencephalogr Clin Neurophysiol 24:409–416
75. Mukhametov LM (1995) Sleep in marine mammals. In Borbely A, Valatx JL (eds) Sleep mechanisms. Springer, Berlin heidelberg New York, pp 227–238
76. Krueger JM, Obal F Jr (1993) A neuronal group theory of sleep function. J Sleep Res 2:63–69
77. Gainotti G (2001) Components and levels of emotion disrupted in patients with unilateral brain damage. In: Gainotti G (ed) Emotional behavior and its disorders. In: Boller F, Grafman J (eds) Handbook of neuropsychology, vol 5, 2nd edn. Elsevier, Amsterdam, pp 161–18
78. Jouvet M (1967) Neurophysiology of the states of sleep. Physiol Rev 47:117–177

Chapter 13

Repression: A Cognitive Neuroscience Approach

Michael C. Anderson

Over a century ago, Freud proposed that memories can be forgotten by pushing them into the unconscious, a process called repression. The existence of repression has remained controversial for more than a century, in part because of its strong coupling with trauma and the ethical and practical difficulties of studying this process in controlled laboratory experiments. In the popular media, skeptics of repressed memories have cast repression as a “myth,” a bit of clinical folklore with no bearing on reality, and a process for which no scientifically valid psychological mechanism exists. In contrast to this, repression, or at least behavior resembling it, is often reported by psychoanalysts and other mental health professionals who interact regularly with psychological patients. The distance between these perspectives on repression is striking, reflecting an enduring disconnection between those who claim to witness the phenomenon or its effects, and those who insist on rigorous specification and evaluation of this construct. Psychology, as a field, has two ways in which it might respond to this persisting conflict: to continue with dissociated traditions and viewpoints, leaving matters unresolved, or to reconcile these perspectives by specifying the mechanisms of repression, relating it to the growing body of research on the cognitive and neural mechanisms of memory, and by making it empirically testable. But is a science of repression even possible?

In this chapter, I review a program of research that I have been pursuing now for over a decade that may reconcile these dissociated views. A central strategy I have pursued has been to consider how repression might be understood in terms of mechanisms that are widely studied in cognitive psychology and cognitive neuroscience. To this end, I have focused on a well-specified question: what are the mechanisms by which human beings willfully control awareness of unwanted memories, when confronted with reminders to them? Although my colleagues and I view this question through the lens of cognitive psychology, the situation bears a strong resemblance to repression. Thus, a better understanding of memory control may provide both the theoretical and empirical grounding necessary to make repression a scientifically tractable problem. The resulting theory may not be identical to Freudian repression, but it clearly speaks to the situations characterized by Freud. First, I describe our general theoretical perspective, and the behavioral and neurobiological evidence that supports it. I then discuss how these constructs relate to Freudian repression.

Executive Control and the Mechanisms of Retrieval

The current perspective about memory control begins with the indisputable observation that actions, once started, can usually be stopped. This fact was impressed upon me one evening while opening the kitchen window. As the window slid along its track, it pushed a cactus off the sill. My hand darted to catch the cactus. Mere centimeters from it, I stopped my hand from clutching the cactus's needle-dense body. This timely save was made possible by my ability to stop physical action–an ability so pervasive that it goes unnoticed in daily life.

The preceding case is a classic example of a situation in which we need to stop a strong habitual response, a situation widely regarded as requiring executive control. This is sometimes referred to as response override, and is illustrated in Fig. 1. In response override, one must stop a prepotent response to a stimulus, either because the response needs to be withheld, or because a less common response is more appropriate. The capacity to stop or redirect action in this way is crucial. Without it, we would lose the flexibility to adapt behavior according to changes in goals, or to changes in the environment. We would be slaves to habit or reflex.

But how do we keep from being automatically controlled by the habitual action? One widely discussed answer is that inhibition is used to suppress the habitual response. On this view, the appearance of a stimulus activates a representation of that stimulus in memory. Activation then spreads to associated responses in proportion to how strongly associated

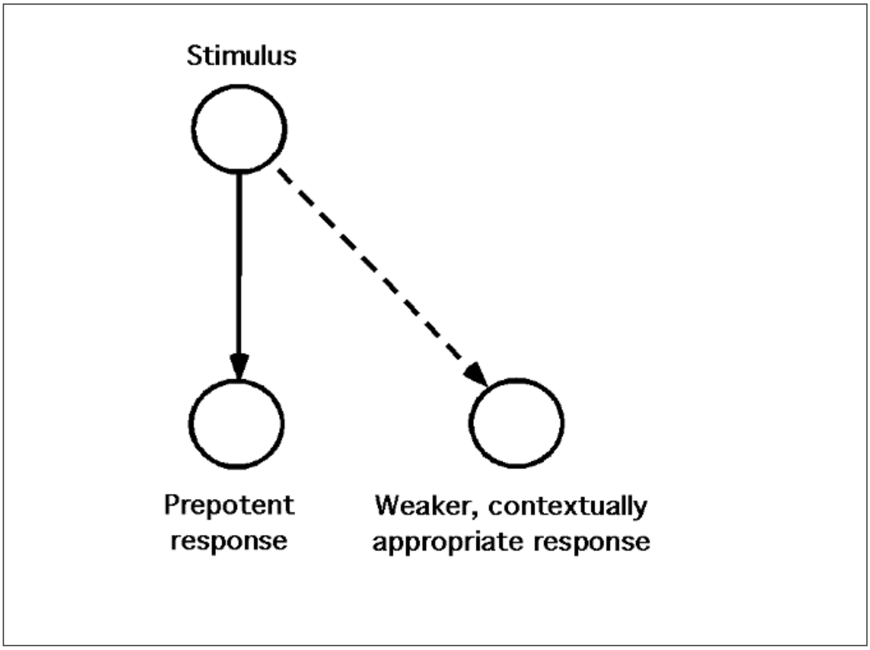

Fig. 1. A typical response override situation. *Circles* correspond to representations in memory, with *lines* representing associations between these representations. The stimulus is linked to two responses, one of which is stronger (prepotent), and the other of which is weaker (*dotted line*). Response override must occur when the organism needs to either (a) emit the weaker, but more contextually appropriate response, despite the stronger association with the prepotent response, or (b) stop any response from occurring. Inhibitory control is thought to suppress activation of the prepotent response to permit response override. The response override situation characterizes many paradigms in work on executive function, including the stroop and go/no-go tasks

they are to the stimulus. When a response becomes sufficiently activated, it will be emitted. If there are multiple responses, the one that achieves threshold most quickly will generally be emitted. However, if a weaker response is more appropriate, inhibition can be recruited to suppress the stronger ones. Inhibition is thought to reduce the activation level for a given response, preventing it from achieving threshold. By doing so, weaker but more appropriate responses can be expressed, enabling flexible, context-sensitive behavior. This is known as inhibitory control.

Given the importance of inhibitory control in managing overt behavior, one might ask whether internal actions might also be influenced by such mechanisms. Parallels exist between the control of action and the control of memory. Just as a stimulus may spread activation to a prepotent motor response, a retrieval cue may activate a strongly associated item in memory, leading it to be retrieved. The retrieval of associated memories is not always desirable; sometimes we may wish to retrieve a memory associated with the cue; at other times we may wish to avoid retrieval altogether, either because the memory is unpleasant or because we wish to maintain focus on the cue concept. Although we often retrieve things that we do not intend, we can control this tendency; we can recollect the event we are seeking despite interference from stronger competitors, and we can stop ourselves from thinking about unwanted memories. Given these parallels between motor behavior and retrieval, response override mechanisms may be recruited to control unwanted memories. If so, we should find evidence for inhibitory control in memory situations likely to involve response override.

In my view, strong evidence for these parallels exists. In support of this, I briefly review evidence for inhibition in memory selection and in memory stopping. Memory selection is required when our goal is to recall an event or fact from memory in the face of interference from related traces that become activated by cues guiding retrieval. The need to stop retrieval arises when we confront a cue and wish to prevent an associated memory from entering awareness. In both cases, attempts to limit the influence of distracting memories have been found to impair later retention, highlighting an important link between forgetting and the control of retrieval.

Selective Retrieval and Retrieval-Induced Forgetting

The need to select a weaker response to a stimulus in the face of interference from a prepotent competitor has a parallel in memory in the situation of selective retrieval. Here, the aim is to recall a target memory when given one or more cues. Typically, a cue will be associated with other memories as well–and some may be more associated to the cue than the target item. It is well known that when multiple traces are associated with the same cue, they compete for access to consciousness [1, 2]. This form of competition presents a problem of control because the cue cannot be relied upon to access the target–in fact, the presence of a strong competitor could perpetually divert us from that target. If inhibitory control is recruited to override prepotent responses, they might also be used to override prepotent memories. To the extent that inhibition persists, situations demanding the selective retrieval of a target should induce lasting memory impairment on competitors. Thus, the act of remembering should cause the forgetting of related memories.

Over the last decade, I have explored this prediction with a procedure we refer to as the retrieval practice paradigm [3]. In the typical study, subjects encode lists of category–exemplar pairs (e.g., *fruit–orange*; *drinks–scotch*; *fruit–banana*). They then perform retrieval practice on half of the exemplars from half of the categories by completing cued stem recall tests (e.g., *fruit–orange*). Each practiced item is tested several times to increase the effect of retrieval practice on related items. After a delay, subjects are tested on all studied exemplars. Performance on this category cued recall test can be measured for three item types: practiced items (e.g., *orange*), unpracticed items from the practiced categories (e.g., *banana*), and unpracticed baseline items from unpracticed categories (e.g., *scotch*). Figure 2 illustrates typical findings. As can be seen, recall of the practiced exemplars was improved relative to performance on baseline items. More importantly, recall of the unpracticed exemplars from the

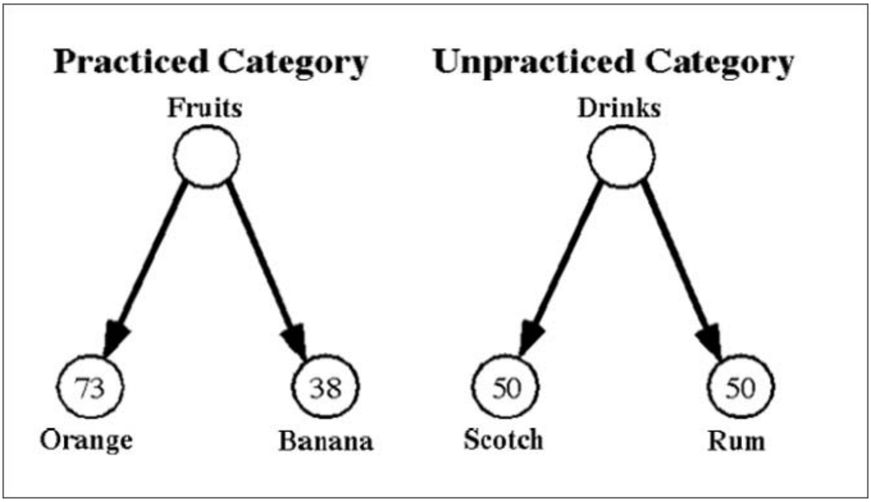

Fig. 2. A typical within-category retrieval-induced forgetting study, as done by Anderson et al. [3]. The example illustrates two items from each of two categories that subjects have studied (six items are usually studied in eight categories), for illustration. In this example, subjects have performed retrieval practice on *fruits–orange*, but not on *fruits–banana* (unpracticed competitor) or any members from the *drinks* category (an unpracticed baseline category). As shown here, practice typically facilitates recall of the practiced item, and impairs recall of the unpracticed competitor, relative to performance in baseline categories

practiced categories (e.g., *banana*) was worse than for the items from baseline categories (e.g., *drinks*). Thus, remembering some items during retrieval practice led subjects to forget related items on a delayed test. We refer to this finding as *retrieval-induced forgetting* [3] to highlight the central role that retrieval plays in generating the effect. Retrieval-induced forgetting is consistent with the view that inhibitory control is recruited to combat interference during retrieval, with inhibition manifesting as recall impairment for competitors on the final test.

Although the basic finding of retrieval-induced forgetting is compatible with inhibition, other mechanisms can explain this effect as well, including McGeoch's classical response competition theory of interference [4]. According to this theory, the likelihood of recalling a target should decrease either when a new response becomes associated with the cue used to retrieve it or when an existing alternative response is strengthened. In either case, the target will suffer increased competition from the alternative response. These competitive dynamics have become formalized in several memory theories that posit relative strength rules of retrieval (e.g., [5, 6]).In these models, the probability of recalling a target is determined by that item's association with a cue, relative to the strengths of association of all items related to that cue. When an alternative response is strengthened, say by retrieval practice, the relative strength of all nonpracticed items declines. Later, when the subject tries to recall the target, the strengthened competitor will have a retrieval advantage, leading it to intrude so persistently that subjects abandon efforts to recall the unpracticed exemplars (see also [7]). This approach does not require inhibition; rather, practiced items become so strongly linked to the practice cue that they block other exemplars. This account is plausible, given the strengthening that practiced items enjoy (however, see section below on "Properties of Retrieval-Induced Forgetting"). Other mechanisms may also contribute to retrieval-induced forgetting. For example, retrieval practice may damage the association linking the category to the affected exemplar, or it may alter the meaning of the practiced category cue (e.g., by biasing *fruit* towards *citrus fruits*) so that the category label is no longer a functional cue for retrieving the unpracticed competitor. All of these mechanisms have been proposed as theories of interference (for a review of noninhibitory sources of impairment, see [8]). Although it might seem difficult to distinguish these alternatives, focused empirical research has yielded evidence for properties of retrieval-induced forgetting that favor the inhibition view. We briefly review these next.

Properties of Retrieval-Induced Forgetting

Work on retrieval-induced forgetting has revealed properties that uniquely support the inhibitory control hypothesis and that suggest that alternative strength-based models may not be correct (see [9] for a review). First, several findings demonstrate that strengthening practiced items does little to impair the recall of related competitors provided that all sources of retrieval-induced forgetting are eliminated from the strengthening process and from the measurement of impairment. For instance, retrieval-induced forgetting appears to be *recall-specific:* retrieval practice impairs the delayed recall of competing items, but the same number of repeated study exposures does not (provided that output interference is controlled on the final test), even though the two practice procedures strengthen practiced items to the same degree. The fact that strengthening can occur with little associated impairment suggests that retrieval practice, not strengthening, is responsible for the effect. Consistent with this, when retrieval practice is performed, the amount of impairment often has no relationship to the amount of strengthening observed on practiced items–that is, retrieval-induced forgetting appears to be *strength-independent*. Impairment does appear to be *interference-dependent*, however: whether retrieval practice impairs a related item depends on whether the item causes interference during retrieval practice. So, for instance, high-frequency exemplars of categories suffer retrieval-induced forgetting whereas low-frequency exemplars do not; similarly, the dominant meanings of homographs suffer significant retrieval-induced forgetting, whereas the subordinate meanings do not. Interference-dependent impairment is exactly what one would expect if inhibitory control is recruited to override retrieval of distracting competitors. Finally, retrieval-induced forgetting exhibits a crucial theoretical property that is difficult for traditional associative interference accounts to explain: *cue independence*. Cue independence refers to the tendency for retrieval-induced forgetting to generalize to novel cues other than those used to perform retrieval practice. Not only does retrieval practice on *fruit–orange* impair the later recall of *banana* when it is tested with the cue *fruit*, but also when it is tested with a novel, independent cue such as *monkey–banana*. These findings argue against interpretations of retrieval-induced forgetting such as associative blocking or cue change, which posit mechanisms that are specific to the cues guiding retrieval practice. They are consistent, however, with the idea that competing memories themselves are suppressed by an inhibitory process in order to retrieve the retrieval practice target.

Taken together, the foregoing properties indicate that the impairment underlying retrieval-induced forgetting is unlikely to be produced by traditional associative interference mechanisms. Rather, it is likely to reflect the action of an inhibitory control process acting to override unwanted retrievals of competitors in memory, helping to achieve selective memory retrieval. This supports the view that selective memory retrieval may be regarded as a special case of response override arising in long-term memory retrieval.

Stopping Memory Retrieval

Response override is also involved when we need to stop a response. In retrieval, the ability to stop could prove useful in preventing a memory from entering consciousness. We sometimes confront reminders of things that we would prefer not to think about: the sight of a car may remind us of an accident we had, or of a former significant other who drove that type of car; or the sight of the World Trade Center in a movie may lead us to stop the natural progression from cues to memories. Other times, we may wish to focus on a thought without letting our mind wander. Can inhibitory mechanisms be engaged to serve these goals? Anderson and Green [10] looked at this issue by examining how stopping retrieval affected the memories that were to be retrieved. To study this, they developed a procedure modeled after the widely used go/no-go task, which has been used to measure the ability to stop a prepotent motor response and to study its neural basis in humans [11-13]. In one version of this task, letters are presented one at a time and subjects press a button as quickly as possible whenever they see a letter, *except* when the letter is an X. When they see an X, they are supposed to avoid pressing the button. The majority of trials require a button press, so that when an X occurs, subjects have difficulty withholding their response. The ability to withhold the response is taken as a measure of inhibitory control.

To explore whether people can stop retrieval, Anderson and Green [10] adapted the go/no-go task to create the *think/no-think paradigm*. In this procedure, subjects studied pairs of weakly related words (e.g., *flag–sword*, *ordeal–roach*) and were then trained to provide the second word (e.g., *roach*; hereinafter referred to as the response word) whenever they were given the first word as a cue (e.g., *ordeal*). Subjects then entered the think/no-think phase, which required them to exert control over retrieval. For most of the trials, the task was the same as it had been during training–to recall and say aloud the associated word as quickly as possible at the sight of its cue. For certain cues, however, subjects were admonished to avoid thinking of the response. It was emphasized that it was not enough to avoid *saying* the response word–it was crucial to prevent the memory from entering *conscious awareness* at all. Thus, subjects had to override not only a vocal response, but also the cognitive act of retrieval. Could subjects recruit inhibitory control mechanisms to stop the memory from entering consciousness?

Of course, Anderson and Green could not directly measure whether subjects controlled consciousness. However, if inhibitory control was recruited, later recall of the excluded memory should be impaired. To examine this, immediately after the think/no-think phase, subjects were given the cues for all of the pairs, and asked to recall the response for each. As predicted, forgetting occurred: response words that subjects excluded from awareness were impaired compared to baseline pairs they had studied initially but had not seen during the think/no-think phase. The more often subjects tried to stop retrieval, the worse recall became (see Fig. 3a). Interestingly, avoided words were harder to recall even though subjects had seen as many as *16 reminders* (i.e., cues) during the think/no-think phase. Normally, reminders facilitate memory, much as it did for the items to which subjects continued to respond (Fig. 3a). Anderson and Green [10]

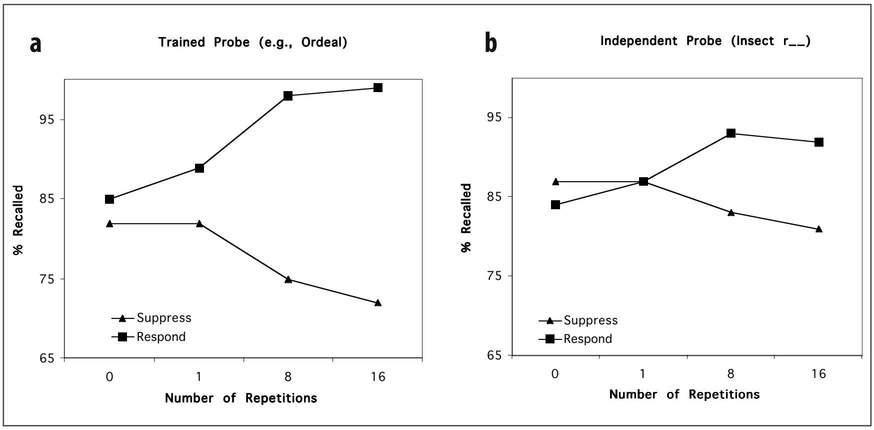

Fig. 3. Final recall performance in experiment 1 of Anderson & Green (2001) using the think/no-think procedure. The plot represents the percentage of items that subjects recalled on the final recall test as a function of the number of times that suppressed the item (*Suppress*) or tried to recall it (*Respond*). The *lefthand panel* represents final recall performance when tested with the originally trained retrieval cue (i.e., the "trained probe"), whereas the *righthand panel* represents final recall performance when tested with a novel, independent, extra-list category cue

further established that this impairment was cue-independent, echoing the results of Anderson and Spellman [14] forgetting occurred regardless of whether subjects were tested with the studied cue word (e.g., *ordeal*) or with a novel cue never studied in the experiment (e.g., *insect r____* for *roach*; Fig. 3b). This argues that the forgetting is not caused solely by associative interference; rather, impairment reflects suppression of the excluded memory itself. In a control experiment, subjects were merely asked to avoid saying the response out loud and all mention of preventing it from entering awareness was eliminated. No inhibition was observed, indicating that the recall deficits were not merely due to suppression of the vocal response for avoided words. These results isolate forgetting in the think/no-think paradigm to processes directed at keeping the unwanted memory out of awareness and demonstrate that this cognitive act has persisting consequences for the avoided memories.

The impaired memory observed by Anderson and Green [10] suggests that inhibitory control mechanisms may be recruited to regulate awareness of intrusive memories. In particular, whenever the environment is such that it presents unavoidable reminders of something that we would prefer not to think about, people may resort to controlling their memories instead to resolve this conflict. The end result may be impaired memory for the things that people avoid thinking about. This suggests that the think/no-think paradigm of Anderson and Green [10] may provide a useful laboratory model of the repression proposed by Freud [15]. If so, results from this paradigm and other related paradigms such as the directed forgetting procedure may have implications for understanding clinical phenomena relating to motivated forgetting [10, 16-20] (see [21] for a review of directed forgetting).

Neural Systems Underlying Voluntary Memory Suppression

The above work on selective retrieval and retrieval stopping indicates functional parallels between controlling retrieval and overriding prepotent responses. This suggests the intriguing possibility that the ability to control unwanted memories may in part rest on neural systems essential for controlling overt behavior. More direct evidence for this relationship might be gained if more were known about the anatomical systems that support memory control. Recently, I have used neuroimaging to identify the neural systems underlying this ability. If memory control and response override are related, one might expect stopping memory retrieval to recruit neural systems known to be involved in overriding prepotent responses to control structures involved in memory.

Research on the neural basis of executive control and declarative memory indicates that at least two brain regions may play important roles in the neurobiological basis of memory control: the hippocampus and the lateral prefrontal cortex. The hippocampus is essential for declarative memory formation [22], and increased hippocampal activation is associated with the subjective experience of consciously recollecting a recent event [23]. Memory suppression requires people to stop retrieval to prevent conscious recollection. The lateral prefrontal cortex is involved in overriding prepotent motor responses [24-26], switching task set, and combating interference in a range of cognitive tasks [27-29]. We hypothesized, therefore, that people suppress consciousness of unwanted memories by recruiting lateral prefrontal cortex to disengage the hippocampal processing that supports recollection.

Anderson et al. [30] recently examined this frontohippocampal hypothesis using the think/no-think procedure. Subjects first learned pairs of words. They then underwent trials in which they were given the first member of the word pair and asked (if the word appeared in green) to think of its corresponding response (respond condition) or (if the word appeared in red) to suppress awareness of the response (suppression condition). Subjects performed this task while being scanned in an event-related functional magnetic imaging design. After this phase was over, scanning ended, and memory was tested for all of the word pairs the subjects had studied. Replicating prior work, the subjects recalled significantly fewer suppression words (i.e., responses to red hint words) than baseline items (i.e., responses to pairs that were learned in the initial study phase, but which did not appear during the think/no-think phase), showing that suppression has occurred.

To examine the neural systems underlying suppression, we contrasted activation during suppression and respond trials of the think/no-think phase. In both trial types, subjects had been presented for 4 seconds with a cue word that had previously been associated with a response word; the only difference was that the cues presented in red had directed subjects to suppress consciousness of the unwanted memory, whereas words presented in green had directed subjects to recall and maintain the corresponding response. Several remarkable findings were obtained. First, a network of regions was more active during suppression than during active retrieval, including bilateral dorsolateral and ventrolateral prefrontal cortex and anterior cingulate cortex. This network overlaps strongly with networks involved in motor response suppression tasks (such as go/no-go), even though no motor responses were required. These findings support the possibility that neural systems involved in overriding prepotent responses are recruited to control retrieval. More generally, they indicate that regulating consciousness of unwanted memories is an active process, and is not accomplished by simply failing to engage retrieval mechanisms.

Interestingly, Anderson et al. [30] also found that suppressing awareness of a memory significantly reduced activation in the hippocampus bilaterally, relative to retrieval. Because activation in the hippocampus has been linked to conscious recollection, this finding suggests that subjects can control awareness of past experiences by strategically disengaging activation in the hippocampal memory system that might otherwise support conscious recollection.

One might wonder to what extent the suppression regions observed in the overall analysis are functionally involved in suppressing unwanted memories. More compelling support for this role could be obtained if it could be shown that these regions predicted later memory suppression effects. We evaluated this by using regression to examine which brain regions predicted individual differences in memory inhibition. Crucially, activation in dorsolateral prefrontal cortex and lateral premotor cortex–regions often observed in go/no-go task performance–predicted subsequent memory impairment for suppression items (Fig. 4a). The more the activation in these regions, the more suppression subjects showed (Fig. 4b). Anderson et al. further showed that activation in the hippocampus predicted later memory failures for suppressed items, and that these variations were correlated with activation in the dorsolateral prefrontal cortex. These findings suggest that dorsolateral prefrontal cortex interacted with mediotemporal lobe structures to attenuate activity, reducing recollection and disrupting retention.

This work provides a strong indication that some of the neural systems involved in overriding prepotent responses may also be recruited to terminate internal actions such as retrieval. Such systems appear to be targeted at mediotemporal regions that support declarative memory rather than motor representations. Because ideas and memories are brought into consciousness by retrieval, the capacity to stop retrieval provides a specific cognitive and neurobiological foundation for how human beings regulate consciousness of unwanted memories. This work, and related work with retrieval-induced forgetting and directed forgetting [16, 17] further

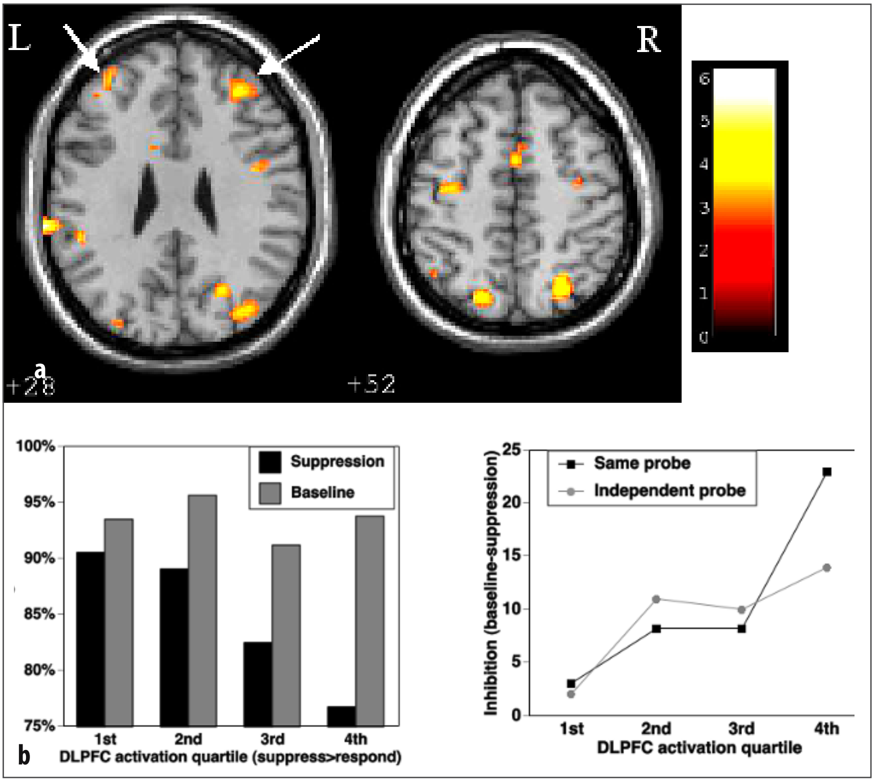

Fig. 4. Relationship of suppression-related activations to memory inhibition. **a** Regions for which activation during suppression trials predicted differences in below-baseline inhibition ($n = 24$). *White arrows* highlight dorsolateral prefrontal cortex (DLPFC) regions from our regression analysis that also predict hippocampal activity for suppression items. **b** Memory inhibition effects for four subject groups matched for counterbalancing manipulations, differing in degree of activation in right DLPFC. Note that increasing activation in DLPFC predicts reduced suppression performance, but leaves baseline performance unaffected. **c** Memory inhibition effects (baseline–suppression) in four DLPFC groups, separated out by test type

supplies a model for how motivated forgetting occurs that may be applicable to understanding the adaptation of memory in the face of traumatic experiences.

Relations to Freudian Repression

The framework and paradigm developed in the foregoing experiments may provide a viable laboratory model of repression. The correspondences between the situation studied in these experiments and the characterization of repression by Freud are striking. Consider, for example, the following brief definition of repression, offered by Freud: “The essence of repression lies simply in the function of rejecting and keeping something out of consciousness” (p. 147 in [31]). Clearly, if this is what constitutes the essence of repression, as Freud indicates, then research done with the think/no-think procedure directly addresses what Freud had in mind. In our procedure, subjects confront reminders to an experience that they are instructed to reject from consciousness for the duration of each suppression trial. From the many experiments that we have done with this procedure, this cognitive act clearly reduces the accessibility of the excluded memory. Thus, within the confines of a carefully designed laboratory procedure, this work establishes a close connection between efforts to regulate consciousness of unwanted memories and their later accessibility. It is now no longer possible to say that there is no mechanism that could possibly support repression.

One might raise several objections to the think/no-think procedure as a model of Freudian repression, however. In the final section, I raise several key objections one might have. The first concerns the distinction between suppression and repression, and whether this work is better characterized as suppression. The second concerns whether the impairment produced by the think/no-think procedure is missing some core feature, proposed by Freud, that renders this irrelevant as a model of repression.

On the Distinction Between Suppression and Repression

One might object to characterizing work on memory inhibition as evidence for repression on the grounds that this work better fits Freud’s idea of suppression than repression. According to this argument, Freud intended a strong distinction between suppression, which was a conscious, intentional process, and repression, which was an unconscious, unintentional process. In this view, the fact that we instructed our subjects to intentionally exclude unwanted memories from awareness renders the think/no-think paradigm a way of studying intentional suppression, but has no bearing on the unconscious repression process. The unconscious repression process, it is asserted, is capable of excluding unwanted mental contents quickly and without any conscious intention or awareness on the part of the subject that the excluded thought ever occurred, or even that the act of repression ever occurred. If one accepts that Freud intended this strong distinction, work with the think/no-think procedure would not be considered repression. Indeed, it is conventional wisdom amongst psychoanalysts that Freud intended this distinction, a wisdom reflected in textbook treatments of repression, which usually characterize it as an unconscious psychological defense process.

Not all scholars believe, however, that Freud intended repression to be an exclusively or even primarily unconscious process. Erdelyi, for example, reports a scholarly analysis of Freud's writings that dismantles this view–a view that he contends is a historical distortion of Freud's theory [32-34]. Although many in the psychoanalytic field (and certainly all those seeking to criticize it) presume that Freud intended repression to be unconscious, Erdelyi argues that it was Freud's daughter Anna who imposed this requirement. By contrast, Erdelyi persuasively shows that Sigmund Freud wrote about repression in terms that allowed for it to be an active, intentional process of exactly the sort characterized in the work reviewed here. Consider this quotation, which is one of several offered by Erdelyi [34] in support of this argument: "It was a question of things which the patient wished to forget, and therefore intentionally repressed from his conscious thought and inhibited and suppressed" (p. 10 in [35]). Clearly, Freud uses the term repression here in a manner consistent with the intentional suppression studied in the current work. One might worry that this is merely an isolated quote, a lapse in speech not in agreement with an otherwise consistent distinction between repression and suppression, but Erdelyi argues that this is not the case [35]:

"Although Freud's half century of psychological writing on repression is not without some ambiguities and even contradictions, the overwhelming textual evidence is that Freud used repression and suppression interchangeably, from his earliest writings (e.g., 1893, as we have seen), to his last (e.g., *An Outline of Psychoanalysis*, 1940)" [36, 37].

Erdelyi argues that it was Anna Freud [38]who, in an effort to tidy up her father's "messy work," introduced the claim that repression was unconscious and suppression was its conscious counterpart. Erdelyi argues that this forced distinction is actually deeply inconsistent with Freud's general insistence on the "continuity of mental life." According to Erdelyi's reading of Freud, complex mental processes could be conscious or unconscious, and did not become something else by virtue of crossing a hypothetical threshold of consciousness. He offers a quotation from Freud to make his point: "It would be unjustifiable and inexpedient to make a break in the unity of mental life for the sake of propping up a definition" (p. 286 in [39]).

Thus, according to Erdelyi, the modern day truism that repression is fundamentally unconscious reflects a serious distortion of Freud's thinking about this concept. This distortion has been perpetuated on the one hand, by psychoanalysts trained according to Anna Freud's interpretation of psychoanalytic theory, and on the other hand, by skeptics of repression who wish to marginalize the concept by insisting on its most extreme, least intuitive (to the lay public) form as the only legitimate definition of repression.

If Erdelyi's scholarship and historical analysis are correct, this suggests that the psychoanalytic field may need to reconsider the distinction between repression and suppression, at least to the extent that it is intended to reflect Freud's views. Moreover, Erdelyi's analysis suggests that work with the think/no-think procedure fits Freud's ideas of repression well. This certainly seems true from Freud's own statements. If so, then work with the think/no-think procedure provides a tractable way to scientifically evaluate the existence and properties of repression, laying the groundwork for a theoretical synthesis of this idea with well-established constructs within the field of cognitive psychology and cognitive neuroscience.

The Object of This Work Lacks a Key Feature of "Repression"

One might object to calling the object of the current work "repression" because it is lacking some additional quality viewed as essential to match Freud's concept. For instance, the current experiments used neutral, nonemotional stimuli, and the materials were simple word pairs. One might argue that repression is entirely about psychological defense and is, by definition, tied to personal trauma or to psychological discomfort; thus, any research that does not include these critical ingredients, though it may be interesting, has little to do with Freudian repression. This view endorses the idea that repression is a special-purpose mechanism dedicated to helping the individual cope with psychological conflict, anxiety, or pain. Alternatively, one might insist that repressed contents continue to influence behavior after they are banished into the unconscious, or that the repressed contents should be recoverable over time. I consider these objections in turn.

Repression as a Specialized Defense Mechanism

Freud clearly emphasized the role of repression in reducing psychological conflict and anxiety, and thus also emphasized the emotional content of the memories being repressed. Based on this, one might argue that because our experiments do not present psychologically threatening material, the effects cannot reflect true repression. It is important, however, to distinguish the mechanism underlying repression from the use to which that mechanism is put: psychological defense. I have argued [10] that the mechanism underlying repression, by virtue of acting on memorial representations in the human brain, must be considered a cognitive process, and, as such, might reflect general mechanisms used in a range of circumstances. In particular, repression may reflect the action of executive control processes, directed at declarative memories. If so, one can decouple the study of the cognitive mechanism (executive control over memory retrieval) from the particular psychological uses to which that process is put. One might wish to control unwanted memories for a variety of reasons: to enhance concentration, update outdated knowledge, minimize embarrassment, reduce anger, control anxiety, focus a retrieval process, or even to deceive oneself or others. Even though each of these circumstances has a different intention, the same computational mechanism can be engaged; to propose a distinct mechanism for each would certainly not be parsimonious. If one separates the mechanism from the use to which it is put, repression would be construed as the use of executive control to control unwanted memories, for the purpose of reducing conflict, anxiety, or psychological pain.

This definition of repression has significant advantages. First, one might argue that the failure of the field to reach consensus on repression for over a century derives in part from inappropriately equating psychological defense with the underlying cognitive mechanism that implements that defense. By requiring that repression be a process unique to trauma or unbearable psychological conflict, one limits the potential to investigate its properties scientifically. One cannot ethically induce trauma or unbearable psychological conflict in the laboratory, and if one instead studies patients coping with these circumstances, one is limited to studying memories, feelings, and thoughts that are, of course, extremely difficult to objectively validate (who knows what a person actually experienced, and whether a repressed memory is accurate?). By acknowledging that the mechanism underlying repression is a general process engaged on behalf of psychological defense, we can study repression in the laboratory using procedures that do not require the induction of trauma or psychological conflict. If this mechanism can be established in a bland emotional context of this kind, one may imagine that when human beings have powerful motives for engaging memory control, it would be quite effective. Moreover, this approach allows repression to be integrated with a wealth of cognitive and neurobiological knowledge about the mechanisms of memory and emotion. Nevertheless, it remains important to progress from studying neutral stimuli and bland motivational contexts to studying stimuli and motives more akin to those likely to require repression outside of the laboratory.

Fortunately, the current findings have been generalized to emotional materials. Several investigators have manipulated the valence of the memories to be suppressed. In work from my laboratory, we took the small, but well-controlled step of manipulating whether the response word to be suppressed was negative or neutral, the stimulus word being always neutral (e.g., phone–bell, twine–rape), and with all other verbal attributes of the response words being held constant (e.g., length, number of syllables, concreteness, frequency). We found significant and nearly identical amounts of inhibition for both classes of stimuli, suggesting that subjects were as able to suppress negatively valenced material as they were neutral material. Other investigators have performed similar manipulations with word pairs and found greater memory inhibition for negative than for neutral response words [40.]

Perhaps the most interesting, however, is a recent demonstration using pairs composed of faces and scenes. Depue, Banich, & Curran [41] manipulated whether a face was paired with a negative scene (e.g., a picture of a car crash) or a neutral scene. One might think that vivid depictions of emotionally aversive scenes might provide a stronger manipulation of valence than presentations of negative words, and thus provide an important test of whether negative experiences can truly be suppressed. Depue et al. [41] found impaired retention of suppressed material, with greater impairment for negative scenes than for neutral scenes. These findings indicate that the mechanisms studied by Anderson and Green [10] provide a viable model of how people suppress memories with affective content. Clearly, additional mechanisms will be involved in regulating accessibility of more intensely traumatic experiences, such as extinction, reappraisal, and other affect regulation processes [42, 43].

Unconscious Influence and Recoverability as Necessary Features of Repression

Even if intentional suppression can be considered repression, and even if this process can be profitably studied outside the context of psychological defense, one might insist that memories that are truly repressed must (a) exert an unconscious influence on behavior, and (b) be recoverable. Freud discussed the "return of the repressed," suggesting that he viewed unconscious influences as important to the phenomenon. Because unconscious influences and recovery have not yet been demonstrated for memory inhibition in the think/no-think procedure, one might question whether this phenomenon is truly repression.

Although it is true that unconscious influences and memory recovery have not been demonstrated for suppressed items in the think/no-think paradigm, these are not good reasons to conclude that this phenomenon is unrelated to repression. They are, at worst, reasons to defer judgment. Empirical work must be conducted to evaluate these possibilities, and this work is quite tractable with the methods we have developed. In fact, memory inhibition is very likely to exhibit these attributes. First, the fact that these effects are linked to modulation of hippocampal activation indicates that suppression is affecting declarative memory for the suppressed events. If the effect primarily occurs in declarative memory, we might see preserved implicit memory for nondeclarative aspects of the suppressed experiences, including perceptual priming, and even affective learning, given a suitable adaptation of the think/no-think procedure. Second, other related inhibition effects (e.g., retrieval-induced forgetting) have been found in some studies to dissipate over time, allowing for memory recovery [44]. These implicit memory and recovery effects are similar to those observed in other related phenomena such as retroactive interference and directed forgetting [45-47]; see also [34] for discussion of related findings). If these phenomena are truly related, memory recovery may be possible with the think/no-think procedure. Thus, if the proper experiments are conducted, the current phenomena may have most of the core characteristics of repression envisioned by Freud.

Conclusions

In this chapter, I have reviewed our efforts to understand repression in terms of processes that are widely studied in cognitive neuroscience today. These efforts have led to procedures and findings that strongly confirm the existence of a process of active forgetting with several of the key characteristics envisioned by Freud. By conceptualizing repression in terms of attention and executive control, we have begun to demystify how such a process might function. Although one might wish to reserve judgment about whether the process under study here is the same as the one proposed by Freud, one point bears emphasis: whatever one may decide, one cannot deny the relevance of the problems addressed in work on memory control to situations confronted by people dealing with unpleasant experiences. Very often in life, we confront reminders of things that we would rather not think about, and when we do, we often take action to stop the unwanted material from entering awareness. Skill at this process is essential to coping in the aftermath of traumatic experiences [48]. The current work addresses this functional situation directly, and, as such, can illuminate the mechanisms involved in exercising control over memory. Whether this work satisfies Freud's original definition of repression ultimately is not the most important question to ask; the most important questions are whether this work can establish things that go beyond or that even correct Freud's initial assertions through careful, systematic study, and whether these new insights can help people. Nevertheless, we believe that this work addresses what Freud had in mind. By conceptualizing the mechanism that implements repression as executive control, we may, after decades of uncertainty, gain traction on an unnecessarily controversial proposal about the human mind.

References

1. Postman L (1971) Transfer, interference, and forgetting. In: Kling JW, Riggs LA(eds)Woodworth and Schlosberg's: Experimental Psychology, 3rd ed. Holt, Rinehart & Winston, New York, pp 1019-1132
2. Anderson MC, Neely JH (1996). Interference and inhibition in memory retrieval. In: Bjork EL, Bjork RA (eds) Memory. Handbook of perception and cognition, 2nd ed. Academic Press, San Diego, CA, pp 237-313
3. Anderson MC, Bjork EL., Bjork RA (1994). Remembering can cause forgetting: Retrieval dynamics in long-term memory. J Exp Psychol Learn Mem Cogn 20:1063-1087

4. McGeoch JA (1942) The psychology of human learning: an introduction. Longmans, New York
5. Raaijmakers JW, Shiffrin RM. (1981). Search of associative memory. PsycholRev 88:93-134
6. Anderson JR (1983) The architecture of cognition. Harvard University Press, Cambridge, MA
7. Rundus D (1973) Negative effects of using list items as retrieval cues. Journal of Verbal Learning and Verbal Behavior 12:43-50
8. Anderson MC, Bjork RA (1994) Mechanisms of inhibition in long-term memory: A new taxonomy. In: Dagenbach DT, Carr T (eds) Inhibitory processes in attention, memory and language. Academic Press, San Diego, pp 265-326
9. Anderson MC (2003) Rethinking interference theory: Executive control and the mechanisms of forgetting. J Mem Lang 49:415-445
10. Anderson MC, Green C (2001) Suppressing unwanted memories by executive control. Nature 410:131-134
11. Casey BJ, Trainor RJ, Orendi JL et al (1997) A developmental functional MRI study of prefrontal activation during performance of a Go-No-Go task. J Cogn Neurosci 9:835-847
12. Caravan H, Ross TJ, Stein EA (1999) Right hemispheric dominance of inhibitory control: An event-related functional MRI study. Proc Natl Acad Sci USA 96:8301-8306
13. Sakagam M, Niki H (1994) Spatial selectivity of go/no-go neurons in the monkey prefrontal cortex. Exp Brain Res 100:165-169
14. Anderson MC , Spellman BA (1995) On the status of inhibitory mechanisms in cognition: Memory retrieval as a model case. Psychol Rev 102:68-100
15. Freud S (1966). A case of successful treatment by hypnotism: with some remarks on the origin of hysterical symptoms through counterwill. In: Strachey J (ed) The Standard Edition of the Complete Psychological Works of Sigmund Freud. Hogarth, pp 117-128
16. Anderson MC (2001) Active forgetting: Evidence for functional inhibition as a source of memory failure. Journal of Aggression, Maltreatment, & Trauma, 4:185-210.
17. Bjork EL, Bjork RA, Anderson MC (1998) Varieties of goal-directed forgetting. In: Golding JM, MacLeod MC (eds) Intentional forgetting: Interdisciplinary approaches. Lawrence Erlbaum Associates, Mahwah, NJ, pp 103-137
18. Conway MA, Harries K, Noyes J et al (2000) The disruption and dissolution of directed forgetting: inhibitory control of memory. J Mem Lang 43:429-430
19 Deprince AP, Freyd JJ (2001) Memory and dissociative tendencies: The roles of attentional context and word meaning in a directed forgetting task. J Trauma Dissociation 2:67-82
20. Myers LB, Brewin CR, Power MJ (1998) Repressive coping and the directed forgetting of emotional material. J Abnorm Psychol 107:141-148
21. Golding JM, MacCleod CM (eds) (1998) Intentional Forgetting: Interdisciplinary Approaches. Lawrence Erlbaum, Mahwah, NJ
22. Squire LR(1992) Memory and the hippocampus: A synthesis from findings with rats, monkeys, and humans. Psychol Rev 99:195-231
23. Eldridge LL, Knowlton BJ, Furmanski CS et al (2000) Remembering episodes: A selective role for the hippocampus during retrieval. Nat Neurosci 3:1149-1152
24. Garavan H, Ross TJ, Murphy K et al (2002) Dissociable executive functions in the dynamic control of behavior: Inhibition, error detection, and correction. Neuroimage 17:1820-1830

25. Menon V, Adleman NE, White CD et al (2001) Error-related brain activation during a Go/NoGo response inhibition task. Hum Brain Mapp 12:131-143
26. Aron AR, Fletcher PC, Bullmore ET et al (2003) Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. Nat Neurosci 6:115-117
27. Knight RT, Staines WR, Swick D, Chao LL (1999)Prefrontal cortex regulates inhibition and excitation in distributed neural networks. Acta Psychol (Amst) 101:159-178
28. Shimamura AP (2000) The role of the prefrontal cortex in dynamic filtering. Psychobiology (Austin, Tex) 28:207-218
29. Aron AR, Robbins TW, Poldrack RA (2004) Inhibition and the right inferior frontal cortex. Trends Cogn Sci 8:170-178
30. Anderson MC, Ochsner K, Kuhl B et al (2004) Neural systems underlying the suppression of unwanted memories. Science 303:232-235
31. Freud S (1915) Repression (C M Baines Trans). In: Rieff P (ed) Freud: General psychological theory. Collier, New York, 1963, pp 104-115
32. Erdelyi MH (1990) Repression, reconstruction, and defense: History and integration of the psychoanalytic and experimental frameworks. In: Singer (ed) Repression and dissociation: Implications for personality theory, psychopathology, and health. Chicago, Chicago University Press, pp 1-31
33. Erdelyi MH (2001) Defense processes can be conscious or unconscious. Am Psychol 56:761-762
34. Erdelyi MH (2006) The unified theory of repression. Behav Brain Sci (in press)
35. Breuer J, Freud S (1895) Studies on hysteria. In: Strachey J (ed and trans) The standard edition of the complete psychological works of Sigmund Freud, vol 2. Hogarth Press, London, 1955
36. Freud S (1892-1893) A case of successful treatment by hypnotism: with some remarks on the origin of hysterical symptoms through “counter-will.” In: Strachey J (ed)The standard edition of the complete psychological works of Sigmund Freud,vol 1. Hogarth Press, London, 1966
37. Freud S (1940) An outline of psycho-analysis. In: Strachey J (ed) The standard edition of the complete psychological works of Sigmund Freud, vol.23. Hogarth Press, London, 1964
38. Freud A (1936) The ego and the mechanisms of defense (C M Baines Trans). International Universities Press, New York
39. Freud S (1938) Some elementary lessons in psycho-analysis. In: Strachey J (ed) The standard edition of the complete psychological works of Sigmund Freud, vol 23. Hogarth Press, London, 1964
40. Joorman J, Hertel PT, Brozovich F, Gotlib IH (2005) Remembering the good, forgetting the bad: intentional forgetting of emotional material in depression. J Abnorm Psychol 114:640-648
41. Depue BE, Banich MT, Curran T (2006) Suppression of emotional and non-emotional content in memory: Effects of repetition on cognitive control. Psychol Sci (in press)
42. Phelps EA, Ledoux JE (2005) Contributions of the amygdala to emotion processing: from animal models to human behavior. Neuron 48:175–187
43. Ochsner KN, Bunge SA, Gross JJ, Gabrieli JD (2002) Rethinking feelings: an FMRI study of the cognitive regulation of emotion. J Cogn Neurosci 14:1215–1229
44. MacLeod MD, Macrae CN (2001) Gone but not forgotten: The transient nature of retrieval-induced forgetting. Psychol Sci 12:148–152
45. Bjork EL, Bjork RA (2003) Intentional forgetting can increase, not decrease, residual influences of to-be-forgotten information. J Exp Psychol Learn Mem Cogn 29:524-531

46. Goernert PN, Larson ME (1994) The initiation and release of retrieval inhibition. J Gen Psychol 121:61-66
47. Wheeler MA (1995) Improvement in recall over time without repeated testing: Sspontaneous recovery revisited. J Exp Psychol Learn Mem Cogn 21:173–184
48. Dougall AL, Craig KJ, Baum A (1999) Assessment of characteristics of intrusive thoughts and their impact on distress among victims of traumatic events. Psychosom Medi 61:38-48

Dreaming: A Neurological View*

Claudio L. Bassetti, Mathias Bischof, and Philipp Valko

Introduction

Dream Theories before 1953

In ancient culture dreams were often considered to be of supernatural origin, allowing direct communication with the gods and capable of providing hints of the future (prophetic and premonitory dreams). Among Egyptians, Greeks and Romans, dream interpreters, working in such places as the oracles of Delphi, were valued, powerful figures. The Egyptians (the oldest known book about dreams dates back to the twelfth dynasty, 2000 BC), when suffering from disease or infertility, used to go to these so-called incubation sites, e.g., the Temple in Memphis, the Temple of Thoth at Khimunu or the Temple of Hathor at Sinai, in order to get meaningful and, hopefully, helpful dreams. This concept of prophetic dreams reappears in the Bible, for example in the Book of Genesis, where Joseph interpreted correctly the dreams of the Pharaoh.

Natural theories of dreaming were less common. Hippocrates and particularly Aristotle (third to fourth century BC) recognized psychological and physiological elements of dreaming. Aristotle differentiated between dreaming and dream recall, and asked “whether the truth is that sleepers always dream but do not always remember their dreams.” Plato anticipated some of Freud’s ideas by recognizing that various desires, among them sexuality, frequently become incorporated in dreams. Lucretius (first cen-

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tury BC) observed the twitching leg movements of a sleeping dog and linked them to a presumed ongoing dream about a chase. In the same century, Petronius Arbiter (author of the *Satyricon*) had to interpret Nero's frightful dreams; he comforted him by explaining that dreams were not of divine origin, but simply the product of ourselves. The same opinion was also held by Cicero. In the second century Artemidorus gave a detailed description and a first classification of dreams in his *Oneirocriticon*, supporting the idea of a premonitory value of dreaming. Galen wrote in his *Diagnosis from Dreams* that dreams can have a premonitory character ("somnia") but also reflect natural processes, changes in the body "humors" ("insomnia").

Natural and supernatural theories of dreaming were often discussed in combination in the Middle Ages. Augustine recognized the existence of corporal, intellectual and spiritual sources of dreams. He and other Christian thinkers, from Tertullian and Isidore of Seville (author of *De tentamentis somniorum*) onwards, condemned the practice of dream interpretation because of the difficulty in differentiating between internal and external, as well as between corporeal and noncorporeal contents and sources of dreams (*revelatio divina* versus *illusio demonica*) [1].

Gerolamo Cardano (1501–1576), while referring to Artemidorus' theories, further elaborated lay and scientific views of dreaming. He described what are now called lucid dreams ("*rendersi conto di stare sognando indica forza mentale,*" "*vegliare nel sonno.*") [2]. Haller's opus *Elementa physiologie* (1769) contained 36 pages devoted to sleep and dreaming. He believed that dreams were due to stimulation interrupting quiet sleep, and that sleep without dreams was therefore more refreshing [3].

The first experimental works on dreaming began in France in the second half of the nineteenth century. Alfred Maury (1817–1892) systematically recorded his dreams after being awakened by his assistant. He found that the presentation of specific sensory, visual or acoustic stimuli during sleep tended to be incorporated into his dreams. He was also among the first, in 1848, to describe sleep onset (hypnagogic) hallucinations [4]. Hervey de Saint-Denis (1822–1892) filled a dream journal of 22 volumes, soon becoming a "lucid dreamer." Both Maury and Saint-Denis were convinced that dreams arise from external stimuli leading to a partial awakening during sleep. Frederick Greenwood recorded hypnagogic visions, with a particular focus on faces, throughout his life. He stated that these faces, unlike hypnagogic hallucinations, were never seen except when the eyelids were closed. Hermann von Helmholtz (1821–1894) speculated that the perception of movements during dreams is related to activation of the motor cortex during sleep. A similar concept was also put forward by his pupil Wilhelm Wundt (1832–1920), the founder of experimental psychology, who recognized in dreams the coexistence of excitation ("autoactivation" of sensory and, particularly, visual cortices) and inhibition (of "volition" and "attention") of cortical brain areas. Maria Manasseina (1843–1903), one of the first Russian woman doctors, wrote in her monograph *Sleep* that during sleep the brain is in an active state. She regarded dreams as evidence of an ongoing psychic life of sleep generated by the brain [5]. Mary Calkins (1863–1930) performed the first quantitative analysis of dream contents, pointing to the preponderance of visual experiences [6].

G. Heermann in 1838 and later Joseph Jastrow in 1888 collected the dream reports of blind subjects, finding a reduction of dream frequency in comparison with sighted people, and a complete absence of visual dreams in subjects who had lost their sight before the age of 5 years. The dreams of the latter were mainly filled with auditory and motor stimuli, but also contained tactile, taste and other sensory images [7, 8].

Johannes Müller (1801–1858) and Jan Purkyne or Purkinje (1787–1869) were among the first to study the peculiar mental imagery during sleep onset, which are most marked in youth. Weir Mitchell (1829–1914) remarked that he had been able from his childhood to summon visions before falling asleep, but that, once present, they could not be controlled, and changed and disappeared of themselves.

The first all-night observations of sleeping humans were made by the French physician Nicholas Vaschide (1874–1907), who noted periodic motor activation in association with dreaming. He also described alternating periods of vasoconstriction and vasodilatation throughout the night and changing periods with good and poor dream recall at sleep interruption [9]. At the end of the nineteenth century several observations documented the dream-influencing effects of various drugs, in particular of the mescal button, which produced visions similar to hypnagogic hallucinations [10]. William Gowers (1845–1915) studied the visual sensations which occur at the onset of epileptic fits or during migraine aura, attributing them to spontaneous discharges in the visual cortex.

Lasègue was the first to describe the small animals and fantastic shapes appearing as visual hallucinations or in the dreams of alcoholics with delirium tremens [11]. De Sanctis differentiated between microzooscopic and macrozooscopic visions, the former predominating in the dreams and hallucinations of alcoholics and the latter in hysterical cases. Manasseina believed that dreams were the result of the same processes as produce hallucinations.

As early as in 1868, Griesinger observed the twitching of the eyelids during sleep both in animals and in humans and postulated an association with dreaming. De Sanctis in 1899 and de Lisi in the 1930s also described the typical limb twitches of rapid eye movement (REM) sleep. In 1944 sleep-related periodic penile erections were described by Ohlmeyer et al. [12]. Following the first EEG recordings by Hans Berger, several authors including Loomis et al. noted in the 1930s that sleep is associated with a slowing of the electroencephalographic activity [13]. In 1953 Hess, Koella and Akert described the activated EEG of REM sleep in the cat, giving, however, the wrong interpretation of this observation in the absence of data on eye movements and muscle tone [14].

REM Sleep and Its Control by the Brainstem: The "REM–Dream Equation"

In search of an objective measure of the process of falling asleep, and motivated by an article on blinking in the wake–sleep transition [15], in 1953 Kleitman and Aserinsky described the presence of rapid eye movements (REMs) in deep sleep [16, 17]. They observed that REMs appear approximately 90 min after sleep onset, are associated with desynchronized cortical low-voltage fast EEG activity and a significant increase and instability in heart and respiratory rates, and occur at regular intervals throughout the whole night. To test the hypothesis that REM sleep may be linked with dreaming, ten probands were awakened during periods with and without REMs. Vivid dreams were reported in 74% when awakened during periods with REMs, but in only 17% when awakened in the absence of REMs.

The neurosurgeon and neurophysiologist Jouvet discovered muscle atonia of REM sleep in 1959 [18]. The combination of REMs, activated EEG and muscle atonia suggested the term "paradoxical sleep" as a synonym of REM sleep. Jouvet also demonstrated that the generation of REM sleep depends on an intact pontine tegmentum [19] and that REM atonia is due to an inhibition of motor centers in the medulla oblongata. Cats with lesions around the locus pericoeruleus exhibit a variety of complex behaviors during REM sleep, including motor patterns suggesting attack, defense and exploration [20].

Another co-worker of Kleitman, Dement, elaborated further on the relationship between REM sleep and dreaming. He was convinced that dreams only occur during REM sleep, and suggested a direct connection between dream content, REMs and motor activities during REM sleep [21, 22]. In line with Freud’s theory, he also suggested, on the basis of a REM sleep deprivation study (which later was strongly criticized), that REM sleep was essential for psychological health [23].

In the 1970s, following the pioneer work of Jouvet, Hobson et al. further elucidated the brainstem-related neurophysiological and neurochemical machinery of REM sleep. They described cholinergic “REM-on” neurons and monoaminergic “REM-off” neurons (producing norepinephrine and serotonin) in the pontine tegmentum, and REM sleep as the result of their reciprocal interaction (Fig. 1) [24]. These authors developed a new dream theory called the “activation-synthesis hypothesis,” according to which dreams are the result of random/nonspecific activation provided by the brainstem, and subsequent, more or less coherent interpretation (synthesis) by the forebrain [25].

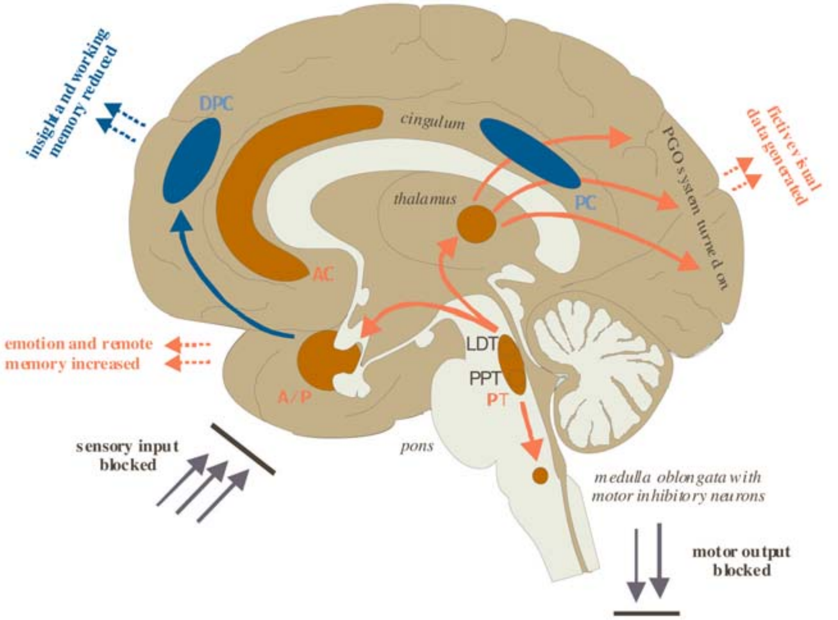

Fig. 1. Subcortical and cortical brain activation/deactivation during REM-sleep based on positron emission tomography (PET) and neurophysiological data (red, activated; blue, deactivated). Brain areas: *PT*, pontine tegmentum; *A/P*, amygdala and parahippocampal cortex; *AC*, anterior cingulum; *PC*, posterior cingulum; *DPC*, dorsolateral prefrontal cortex. Nuclei: *LDT*, laterodorsal tegmental nuclei; *PPT*, pedunculopontine nuclei

NREM Sleep and Dreaming

The awaking studies of Aserinsky and Kleitman had already shown that subjects can report dreams or dream-like experiences even when awakened from non-REM (NREM) sleep. This observation was confirmed by several other authors and consequently challenged the so-called REM–dream equation [26–28]. The persistence of dream experiences despite complete pharmacological REM sleep suppression gives further support for the existence of a dream-like mentation in NREM sleep [29]. On the other hand, Aserinsky and Kleitman found that so-called non-dreamers had REMs just like those of subjects with frequent dream recall.

Dream reports from patients awakened from REM sleep are more frequent, more real, more emotional and more bizarre, and their content is more often visual and/or motor [30, 31].

Brain Metabolism/Activation During Sleep and Dreaming

Neuroimaging studies using positron emission tomography (PET), single-photon emission computed tomography (SPECT) and functional magnetic resonance imaging (fMRI) [32–35] have shown, in line with neurophysiological observations, different patterns of brain activation during the different states of being (wakefulness, NREM sleep, REM sleep).

REM sleep is characterized by sustained neuronal activity, high cerebral energy requirements and high cerebral blood flow. Compared to wakefulness, in most studies a deactivation of the dorsolateral prefrontal cortex and associative parietal areas during both NREM and REM sleep, and an activation of pontine tegmentum, thalamus, limbic/paralimbic structures (including the amygdala) and temporo-occipital areas (other than primary visual cortex) during REM sleep were reported. Medial prefrontal cortex, however, remained as active during REM sleep as during wakefulness, whereas it is significantly decreased during NREM sleep [36, 37].

These profiles of activation and deactivation are thought to explain the characteristics of mental activity during wakefulness, sleep onset, and NREM and REM sleep. Deactivation of the prefrontal cortex may explain the lack of insight and the decrease in volitional control typical of oneiric experiences in general. Activation of the limbic/paralimbic structures (including the amygdala and the anterior cingulum and orbitofrontal cortex) and temporo-occipital cortices, on the other hand, could explain the strong emotional and visual character of REM sleep dreaming and the frequent translation of new and old memories into dreams. The medial prefrontal cortex, which remains activated during REM sleep, is involved in the ability to attribute intentions, thoughts and feelings to oneself and others, which may illustrate the preserved "mind representation" during dreaming. The sleep-associated functional connectivity (coherent neuronal firing) of neurons within the mesiotemporal lobe may underlie the variable ability—in normal subjects and possibly also in patients—to recall dream experiences [38].

The exact profile of brain activation (and deactivation) underlying the different mental activities across states of being (thinking, day dreaming, sleep onset hallucinations, NREM and REM dreams, sleep end hallucinations) remains essentially unknown at this point. Furthermore, little is known about the brain metabolism during NREM sleep dreaming.

Dreaming in Neurological Patients

The frequency and extent of dreaming changes in neurological patients has rarely been assessed in a systematic fashion. The available data in the literature suggest, nevertheless, a relatively high frequency of altered dreaming in neurological disorders.

The brain mechanisms involved in dream changes are, with the exception of REM sleep behavior (see below), essentially unknown. Neurochemical[1] and/or structural changes in the above-mentioned thalamolimbic–temporo-occipital "dreaming circuits" can generally be postulated. In many instances clinical symptomatology can be understood as a "dissociated state" arising from the loss of the physiological boundary between the different states of being (wakefulness, NREM and REM sleep).

The exact pathophysiology is expected to vary according to the specific form of altered dreaming. In patients with narcolepsy and Parkinson's disease, visual hallucinations may represent, at least on some occasions, the intrusion of REM sleep mentation into wakefulness [39]. In "out of

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[1] Cholinergic and monaminergic (e.g., dopaminergic, serotoninergic, noradrenergic) transmission, which is known to regulate sleep-wake functions, is probably also implicated in the modulation of dream experiences.

body experiences" a dysfunction of body- and self-processing at the temporoparietal junction has been suggested [40]. In addition to brain dysfunction per se, other factors such as age, personality, mood, current physical and psychic status, stress, fever, medications (e.g., antidepressants, β-blockers, acetylcholinesterase inhibitors), alcohol and presence of sleep disorders may influence dreaming in neurological patients [41].

Decreased Dreaming/Cessation of Dreaming

In 1883 Charcot described a patient who lost the ability to recall the visual mental images of his dreams in association with deficit in revisualization during wakefulness ("visual irreminiscence") [42]. In 1887 Wilbrand presented a case of global cessation of dreaming in association with prosopagnosia (inability to recognize familiar faces) after infarction in the occipitotemporal region (Fig. 2) [43]. The terms "Charcot–Wilbrand syndrome" (CWS) and "anoneira" were used to describe cessation of dreaming as a consequence of focal brain damage. Grünstein noted that

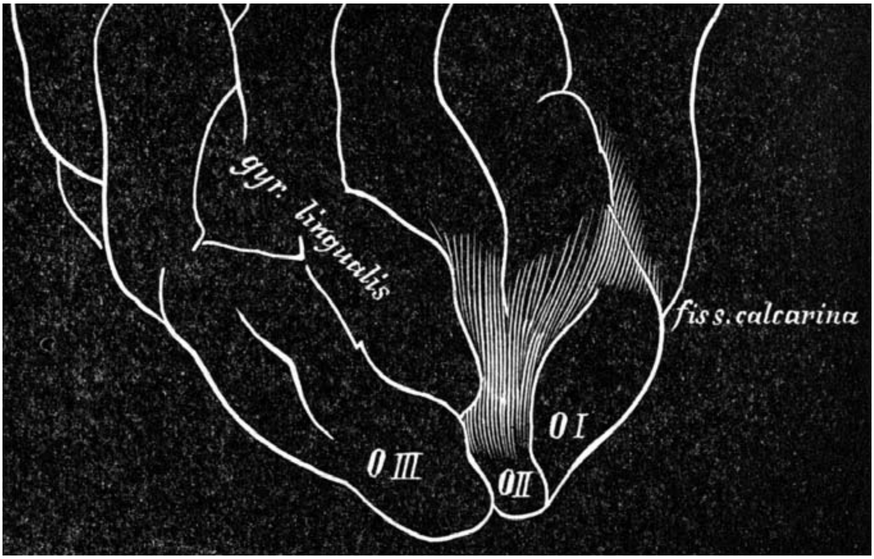


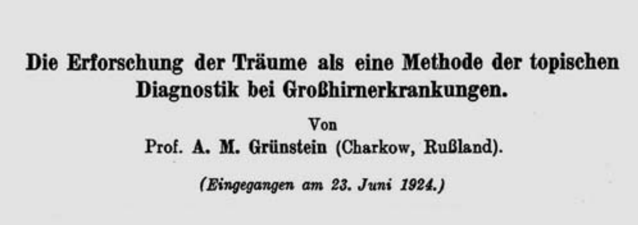
Fig. 2. The brain autopsy of the patient described by Wilbrand in 1887 with loss of visual dreaming following a cerebral infarction in association with prosopagnosia. This precise drawing shows the infarcted area in the right deep occipital lobe, just beside the inferior lingual gyrus

CWS is associated with visual agnosia and an irreminiscence but not with cortical blindness, correctly suggesting a role of the extrastriate visual cortices in normal dreaming (Fig. 3) [44]. Later studies confirmed the strong association (in 91% of cases reviewed by Solms) between CWS and irreminiscence [45].

A decrease in and even (transient) cessation of dreaming is frequently seen as a nonspecific symptom of acute neurological disorders, particularly of those affecting the central nervous system.

CWS has been reported following focal lesions (e.g., stroke, traumatic brain injury) in the inferior parietal, medial temporo-occipital or bifrontal brain areas [45–49]. In a study of 53 patients with acute unilateral brain lesions (stroke, neoplasia), those with posterior lesions showed a frequent loss of dream recall while those with anterior lesions rarely did so (61–75% vs 11–15%, respectively). The cessation of dream recall was associated with defective performance in tests for visuoperceptive functions. There were no right–left differences, whereas aphasic patients were more commonly non-recallers than patients without aphasia (83% vs 33%, respectively) [48].

Dementia and amnesic syndromes can lead to a reduction in frequency and verbal expression of dreams [50, 51]. It is noteworthy that, even in the presence of profound declarative amnesia, patients with extensive bilateral medial temporal lobe damage report at sleep onset, like normal controls, intrusive, stereotypical, visual images following prolonged playing of the computer game Tetris [52].



Die Erforschung der Träume als eine Methode der topischen Diagnostik bei Großhirnerkrankungen.

Von
Prof. **A. M. Grünstein** (Charkow, Rußland).

(Eingegangen am 23. Juni 1924.)

Fig. 3. Grünstein's publication from the year 1924 with the title "Die Erforschung der Träume als eine Methode der topischen Diagnostik bei Grosshirnerkrankungen" (The study of dreaming as a method for topical diagnosis in cerebral diseases)

Increased Dreaming

Increased dreaming is seen with anterior limbic damage [45] and, rarely, as an epileptic phenomenon in temporal lobe seizures (epileptic dreams, see below). Often, but not always, increased dreaming is accompanied by the appearance of dream-like experiences during wakefulness and by difficulty in differentiating between dreams and reality.

Changes in Dream Contents/Accompaniments

Dreams with a strong anxiety component (terrifying dreams) can be seen as REM-related parasomnia (nightmare) and in the course of focal, neurodegenerative brain diseases (e.g., Parkinson's disease) and, rarely, as epileptic equivalent.

Dreams with increased vividness, violent or aggressive contents and increased or uncontrolled motor activity (enacted dreams) suggest the presence of a REM sleep behavior disorder (RBD). The diagnosis is proven by the polysomnographic demonstration of a loss of physiologic atonia and increased motor phasic activity during REM sleep. RBD can be observed in the course of neurodegenerative disorders, brainstem lesions, narcolepsy, drug or alcohol abuse, and in the so-called parasomnia overlap syndrome.

A cessation of visual dream imagery ("nonvisual dreaming") has been reported following medial occipitotemporal lesions [45]. Patients with unilateral medial temporo-occipital lesions have been reported to have less expressive activity and distorted settings in their dreams [53].

Dreams with unaltered perception of the self as a critical observer of the oneiric experience and a unique capability of influencing their contents (lucid dreams) are occasionally reported by patients with narcolepsy.

Dream-Like Experiences During Wakefulness (Hallucinations)

Visual hallucinations during wakefulness, at sleep onset (hypnagogic hallucinations) and on awakening (hypnopompic hallucinations) can occur in normal subjects as well as in patients with migraine, epilepsy, midbrain/thalamic lesions (peduncular hallucinosis), lesions causing visual loss (Charles Bonnet syndrome), sleep paralysis, narcolepsy, and

Parkinson's syndromes [54]. Visual hallucinations can sometimes be highly complex and colorful ("movie-like"). This is well illustrated by the following episode (audiotaped) reported by a 48-year-old man with peduncular hallucinosis due to tegmental midbrain stroke that had appeared after coronary arteriography (Fig. 4):

I suddenly lost the feeling of my own body weight. In front of me I saw—on the right side of the wall—a parade of young and old people that I could not recognize. There was a little girl in front that attracted my attention. She was wearing a very bright dress with red-orange flowers on it like in a painting by Gauguin. I was ready to leave for an adventure. .. when suddenly the voice of the cardiologist woke me up.

Patients are often aware that these hallucinatory phenomena are not real. They may be indifferent to, amused or disturbed by them. In some patients, however, difficulties in differentiating dreaming from hallucinations may be reported. The occurrence of both hallucinations and increased dreaming is also possible [55]. The existence of such a continuum had already been suggested by Lasègue ("le délire alcoolique n'est pas un délire, mais un rêve" [11].

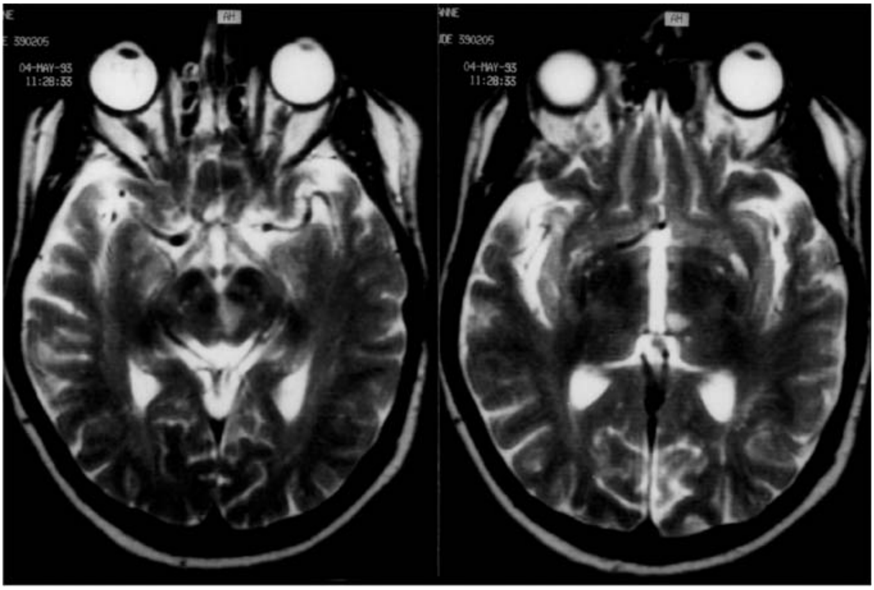


Fig. 4. Brain magnetic resonance image (MRI) of a 48-year-old man with peduncular hallucinosis due to a unilateral subthalamic–mesencephalic stroke that appeared after coronary arteriography (see text)

Simple and complex acoustic hallucinations can complicate peripheral or central hearing loss. Somesthetic hallucinations are reported following amputation of an extremity or profound sensory loss (phantom limb syndrome). Polymodal hallucinations are typical of delirium tremens, but can also be seen with focal brain damage, as reported by a 49-year-old patient with tegmental pontine stroke (and history of alcohol abuse):

I suddenly saw the face of a madonna, like on a church window, on my left side, lasting for about one minute . .. I found in front of me a large white snow ball . .. on my right side the face of a madonna . .. I heard for hours in my sleep school songs in my left ear and a bird singing.

Hallucinations not related to a false perception (so-called psychic hallucinations after Baillarger) include the sense of a presence of persons (often relatives standing in the room, sitting on the edge of the bed). This type of hallucination ("sensed presence") is particularly common in patients with narcolepsy, sleep paralysis and Parkinson's disease [56, 57]. Less commonly, patients may project or feel their own body image and/or their self in the external space ("out of body" and autoscopic experiences) [40].

Unaltered Dreaming

Dreaming can remain unaltered despite dorsolateral prefrontal lesions, right hemispherectomy [58], split-brain procedures [59] and brainstem lesions [45], with and without REM sleep changes [45].

Dream Changes in Specific Neurological Disorders

Sleep Disorders

Narcolepsy

Narcolepsy is a chronic debilitating disorder characterized by excessive daytime sleepiness, cataplexy, hallucinations and sleep paralysis [56]. Narcolepsy is believed to be caused by an autoimmune destruction of hypothalamic hypocretin-producing neurons [60].

Hallucinations, dreams, nightmares, arousal disorders, and RBD are more frequent in narcoleptics than in the normal population (Fig. 5). In two recent systematic studies nightmares (54%) and life-long hallucina-

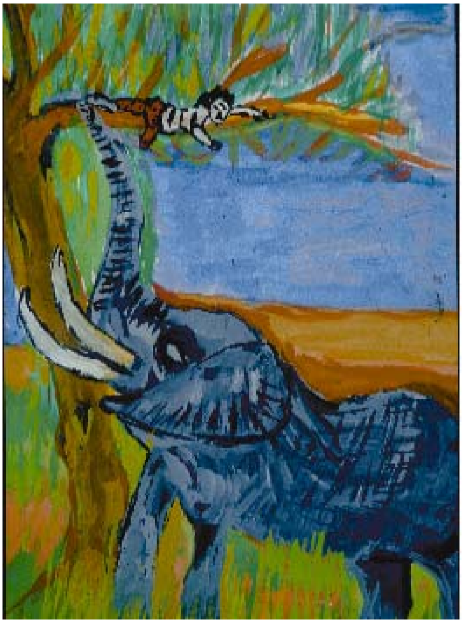


Fig. 5. Painting by a 56-year-old man with narcolepsy, depicting frightening and vivid visual hallucinations. (Reproduced with permission.)

tions (81%) were found to be more common than in normal controls (5% and 37%, respectively) [56, 61]. Not infrequently, patients may experience a variety of hallucinations and dream-like experiences, as is well illustrated by the following report of a 19-year-old patient:

During the day I often had the feeling as if somebody was holding my left leg when passing a dark passage ... At sleep onset I often saw a dark, silent, black shadow on the right side of my bed, which vanished immediately the light was turned on ... While sleeping I often had strange dreams. I would see myself as acting in the dream action, and at the same time I would feel myself as separated from it, "floating" over the scene and realizing the illusionary nature of the dream.

In narcoleptics, hallucinations and dreaming may accompany or follow attacks of cataplexy and sleep paralysis. Dreams can occur at sleep onset during daytime (napping) and night-time, as well as on awakening (Rosenthal's syndrome). Typical of narcoleptic dreams is the presence of elements of the normal awake mentation in form of volitional control of mentation and some awareness of the environment. Fantastic and absurd dreams with religious, mysterious or sexual components are possible. Hallucinations are often visual, less commonly somatosensory, vestibular, gustatory/olfactory or vegetative. The hallucinations of the presence of someone nearby (people, intruders, animals, "sensed presence"), of a pressure on the chest with breathing difficulties ("incubus/succubus") and of floating/flying sensations with out-of-body experiences are typically associated with sleep paralysis [62].

Lee et al. compared polysomnographic findings and dream reports between 24 patients with insomnia and 16 patients with narcolepsy, both diseases associated with disturbing dreams [63]. Compared to insomniacs, narcoleptics had more frightening, recurrent dreams and shorter REM segments. No correlation was found between dream content and polysomnographic findings such as REM segment length or REM density.

Dahmen et al. stressed the occasional difficulty in differentiating between narcoleptic and schizophrenic hallucinations [61]. They investigated different aspects of hallucinations between 148 patients with narcolepsy, 21 patients with exacerbated acute schizophrenia and 128 healthy controls. Auditory hallucinations were twice as frequent in the schizophrenic group compared to narcoleptics, whereas 83% of narcoleptic patients reported visual hallucinations compared to only 29% among schizophrenics.

In a study of 15 narcoleptics and 9 controls, Fosse et al. found emotions to be more common and more intense in narcoleptic sleep onset REM periods than in night-time REM of either narcoleptic or normal subjects, with anxiety/fear exhibiting the strongest increase, followed by joy/elation. Comparing night-time REM in narcoleptic and normal subjects, narcoleptics were also found to have more intense feelings of anxiety/fear and of joy/elation but to have a less frequent experience of surprise and anger [64].

Sturzenegger and Bassetti collected the dream reports of 57 narcoleptics with cataplexy and compared them with those from patients with nonnarcoleptic hypersomnia and from normal controls [56]. The dreams of narcoleptics more often included such contents as flying, being chased, crawling in a tube, monsters and the presence of persons in the patient's room ("sensed presence"). Moreover, dream reports from narcoleptics contained more negative emotions such as sorrow, anger, fear, guilt, disgust and disdain. No link was found between emotions in the dreams and emotions triggering cataplectic attacks.

The possibility that emotions in the dreams of narcoleptics may precipitate symptoms on awakening was suggested in 1928 by Wilson. One of his patients had a terrifying dream about a murder and immediately thereafter woke up with an unusually pronounced sleep paralysis [65].

Attarian et al. reported a case of RBD in a narcoleptic patient arising from cataplexy and wakeful dreaming, probably facilitated by his use of tricyclic antidepressants to treat cataplexy [66].

Sleep paralysis (SP) frequently occurs also in nonnarcoleptic subjects. Its prevalence is estimated to be up to 25–40% [62]. Sturzenegger and Bassetti found a prevalence of SP of 49% among narcoleptics, 18% among hypersomnolent patients and 5% among healthy controls [56]. SP is almost always accompanied by some hallucinatory activity. Among 387 subjects with SP only 1.6% did not report the experience of hallucinations of any kind [67]. In narcolepsy, SP-related hallucinations may be particularly terrifying and lead to extreme anxiety with fear of dying, especially at the beginning of the disease when the patient has not yet become accustomed to them. Hypnopompic hallucinations, in particular, may be perceived as extremely realistic.

Idiopathic Hypersomnia

Idiopathic hypersomnia (IH) is a rare condition characterized by excessive daytime sleepiness, prolonged unrefreshing sleep and difficulty waking up ("sleep drunkenness"). The frequency of hypnagogic hallucinations, sleep paralysis and habitual dreaming in patients with IH is estimated at around 40%, a proportion similar to that found in narcoleptic patients without cataplexy (monosymptomatic narcolepsy) [68]. However, the dreams and hallucinations of IH patients are less vivid, less bizarre, less frightening and less emotional than those of narcoleptics. Bassetti et al. reported a 22-year-old female patient with IH who had frequent hallucinations both at sleep onset and on awakening, but without sleep paralysis, without sleep onset REM periods and without cataplexy [69].

Kleine-Levin syndrome

Kleine–Levin syndrome (KLS) is a rare disorder of uncertain etiology characterized by recurrent episodes of hypersomnia lasting for days or weeks, compulsive eating behavior and various psychopathological abnormalities such as hypersexuality, irritability or apathy. Visual or auditory hallucinations, delusions and derealization have been reported by 14–24% of patients with KLS, whereas cataplexy and sleep paralysis were never mentioned as co-morbid symptoms [70]. Characteristically, most patients with KLS report that during an attack they perceive everything happening around them with a feeling of unreality, "with the feeling of being almost in a dream" or "with a nightmarish sense of the surroundings" [70]. Interestingly, this dream-like perception is often followed by amnesia of the events that occurred during the attack. Merriam, for example, described a case of postencephalitic KLS in a young male patient, who saw ghosts and famous TV actors and believed himself to be pursued by armed attackers [71]. The frequency of hallucinations and delusions in 18 patients with secondary KLS was even higher (44%) [70].

Sleep Terror and Sleepwalking (NREM Parasomnias/Arousal Disorders)

Sleep terror (pavor nocturnus) and sleepwalking are parasomnias of the the first part of the night. These parasomnias are so-called disorders of arousal because they represent a sudden but incomplete arousal from slow-wave sleep (stages 3 and 4 of NREM sleep) [72]. Arousal disorders have for a long time erroneously been regarded as behavioral expressions of ongoing dreams [73, 74]. In fact, oneiric activity is usually absent or consists in brief, fragmentary imagery [75, 76]. The possibility to induce sleep terrors by forced awakenings during slow-wave sleep also argues against the old belief that sleep terrors might be triggered by some fearful dream.

Particularly in adults, oneiric experience in conjunction with sleepwalking may be reported and amnesia for the episodes can be incomplete [77]. The presence of oneiric experiences in sleepwalking has also been suggested by the fight-or-flight imitating behavior occasionally observable in sleepwalkers [78].

In sleep terrors subjects abruptly sit up, producing a loud scream in association with a facial expression of extreme fear. The attacks are accompanied by a sudden and pronounced increase in heart and respiratory rate and muscle tone. Most characteristically, the extremely worried parents are usually unable to arouse and comfort their child. The combination of features of arousal disorders with REM sleep behavior disorder has been called "overlap parasomnia."

Sleep terrors and sleepwalking are more common in children but may be present in up to a few per cent of the adult population. They are not associated with specific physical findings. Developmental, genetic, psychological, and organic factors can contribute to their occurrence. About one-third of individuals with frequent nightmares have a positive family history, and an association with the HLA haplotype DQB1*05 has been suggested. Sleepwalking can be triggered by fever, stress, sleep deprivation, acute brain disorder (stroke, trauma) and treatment/withdrawal from drugs.

Nightmares/Post-traumatic Stress Disorder

Nightmares are parasomnias of the middle of the night or early morning when REM sleep is more common. Nightmares are typically dreams of physical danger (e.g., being chased) or psychological threat (e.g., being teased). Typical contents include monsters, ghosts, animals, and bad individuals. Vocalizations, movement and autonomic symptoms are typically minimal. When awakened, subjects are rapidly oriented, can be calmed and usually recall the details of the dream. These features distinguish nightmares from sleep terrors (see above) and REM sleep behavior disorder (see below).

Nightmares are more common in children and are not associated with specific physical findings. About 10% of individuals with frequent nightmares have family history of nightmares. Nightmares are more common in persons with mental retardation, chronic alcoholism, depression, and central nervous system disease, as well as in association with fever and treatment/withdrawal from drugs. Nightmares may result from a severe traumatic event and indicate post-traumatic stress disorder.

Nocturnal Panic Attacks

Nocturnal panic attacks are to be differentiated from nightmares. In contrast to the latter, they typically arise from NREM sleep, are accompanied by marked autonomic activation with tachycardia and arterial hypertension, and detailed dream recall is usually lacking. Patients with nocturnal panic attacks often show also diurnal panic attacks, but occasionally diurnal attacks may be absent or develop only some years later [79].

Epic Dreaming and Complex Nocturnal Visual Hallucinations

Epic dreaming was described by Schenck and Mahowald in 1995 and represents a dreaming disorder characterized by endless and exhausting dreams associated with morning tiredness and chronic daytime fatigue [80]. The condition shows a strong female predominance (85%). The dream content typically consists in relentless, mostly banal physical activity reoccurring nightly in 90% of affected patients. A remarkable feature is the poor affective tone of the dreams and the absence of emotional arousals. Polysomnographic recordings do not reveal any particular abnormalities, and the underlying disturbance of the disorder remains unknown.

Silber et al. have recently reported a series of 12 patients (11 of whom were females) with vivid, silent, often distorted images of people and animals occurring after waking, disappearing with increased light. Idiopathic hypersomnia, beta-blocker use, dementia with Lewy bodies, macular degeneration and anxiety were associated factors [81].

REM Sleep Behavior Disorder

This disorder was formally first described in 1986 [82], although single reports had described features of this entity already before. RBD presents with dramatic, often violent, motor behaviors occurring during sleep in association with vivid, frightening dreams. Since RBD occurs during REM sleep, it typically appears in the second half of night-time sleep, when REM sleep predominates. Injuries and assaults on bed partners have been reported to occur in up to 32% and 64% of patients, respectively (Fig. 6) [83]. In a study of 49 patients with polysomnography-confirmed RBD, Fantini et al. found dreams in these patients to be characterized by an elevated proportion of aggressive contents, despite normal levels of daytime aggressiveness [84].

Polysomnography demonstrates excessive muscle tone during REM sleep (e.g., in the chin EMG), which may represent the first sign of RBD [85], an enhanced phasic muscle activity with corresponding excessive or abnormal motor behavior and vocalizations. Patients with an idiopathic RBD have an increase in slow-wave sleep and delta power and an increased frequency of periodic limb movements during all stages of sleep [86, 87]. The differential diagnosis of RBD includes sleep-related seizures, confusional arousals, sleepwalking, sleep terrors, nightmares, panic attacks and arousals related to obstructive sleep apnea.

The pathophysiology of RBD corresponds, as first shown experimentally in the cat by Jouvet [19], to a dysfunction of pontomedullary REM mechanisms of atonia. Approximately 60% of RBDs are idiopathic. Idiopathic RBD usually presents in the sixth or seventh decade of life and predominates in males. In these patients cognitive (visuospatial, constructive) disturbances, slowing of occipital EEG activity and decreased striatal dopaminergic availability during wakefulness have been reported [88–91]. A significant proportion of patients with idiopathic RBD later develop a parkinsonian disorder [92]. In about 40% of cases RBD is due to narcolepsy or neurodegenerative disorders such as Parkinson's disease, multisystem atrophy, Lewy body disease and, less commonly, brainstem dysfunction of other origin (e.g., multiple sclerosis, stroke, limbic

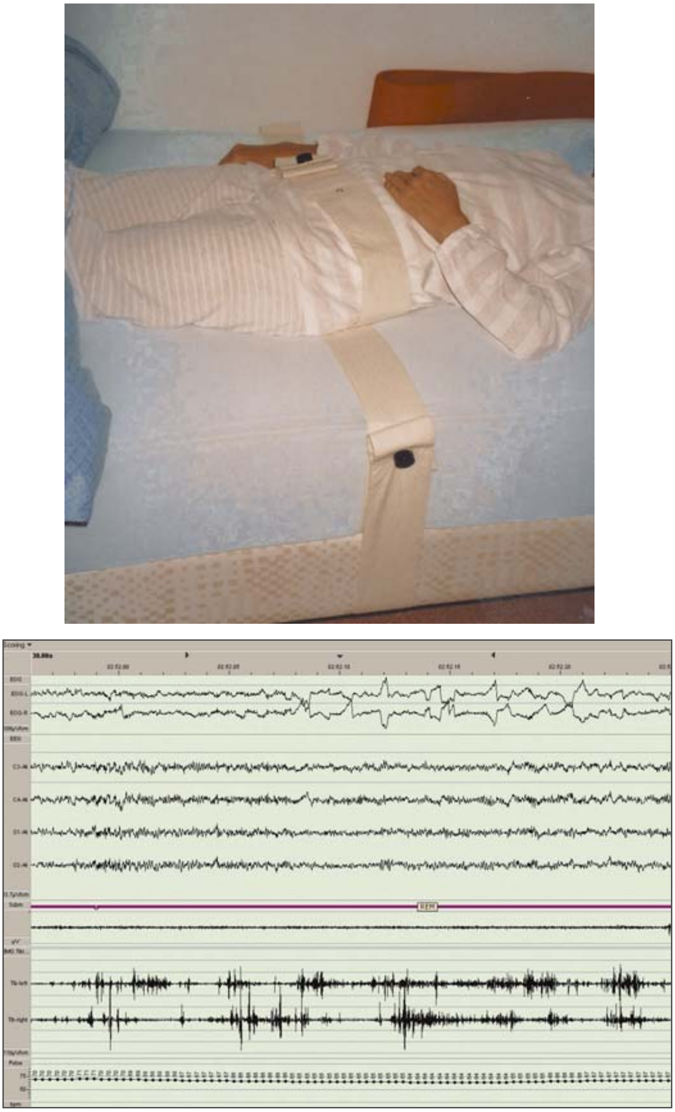


Fig. 6. Picture (*above*) of a 65-year-old woman with an idiopathic REM sleep behavior disorder, who restrained herself for years with ropes in order to prevent herself from falling out of the bed. Polysomnography (*below*) shows the characteristic finding of enhanced phasic muscle activity during a stage of REM sleep

encephalitis). According to the theory of Braak, the emergence of the common feature of alpha-synucleinopathies, the Lewy bodies, follows a defined and predictable path in the CNS which starts in the lower brain stem and finally reaches the cerebral cortex [93]. Because the early stages of this condition do not seem to have any obvious symptoms, it is impossible for clinicians to identify these patients at present. RBD together with olfactory disturbances might represent such early symptoms [94]. An acute/transient form of RBD can be seen in patients discontinuing the consumption of alcohol and REM-suppressing medications (e.g., antidepressants). Most cases of RBD respond to small doses of clonazepam, which is usually effective in controlling both motor behavior and dream changes. This supports the hypothesis of a common neuronal generator for motor and dream pattern. Melatonin, dopaminergic drugs and donepezil are other therapeutic options.

Insomnia

Insomnia is the most common sleep-related complaint and affects up to 47% of patients attending general practices [95]. A substantial number of investigations have suggested that in healthy individuals a significant correlation exists between the frequency of dream recall, frequency of nocturnal awakenings and low sleep quality [96–98]. Schredl et al. have recently confirmed that insomniacs' dream recall frequency is elevated and dream reports are longer [99]. Dreams of insomniacs are characterized by more negative emotions, frequent reports on health themes, depression and aggression and a reduced degree of explicit visual perception.

If on the one hand insomnia can increase dream recall frequency, there might on the other hand exist a subcategory of insomnia that is explicitly produced by dreaming. This condition is called "dream interruption insomnia" [100]. Patients complain about repetitive nocturnal awakenings and typically mention frequent episodes of nightmares prior to the onset of their insomnia.

Sleep-Disordered Breathing

Schredl et al. found an elevated dream recall frequency in patients with sleep-disordered breathing (SDB), but without any correlation with the frequency of nocturnal awakenings [101]. A strong negative correlation was found between the frequency of respiratory events and dream bizarreness, suggesting that apnea-induced arousals may interrupt the (REM) dream process.

Gross and Lavie reported that dreams of patients with SDB were completely devoid of any feelings of suffocation and did not contain peculiar signs of fear or anxiety [102]. Patients with SDB may, however, present with sudden and anxious nocturnal awakenings and recall dreams. In such cases, differentiation from nightmares and nocturnal panic attacks may be difficult and require polysomnographic evaluation. In addition, Iranzo and Santamaria have recently reported the occurrence of agitated, violent dreams with abnormal motor behaviors mimicking RBD in patients with severe SDB [103].

Restless Legs Syndrome

No increased dream frequency was found in patients with restless legs syndrome compared to healthy controls [104].

Stroke and Other Focal Brain Diseases

Following the seminal papers by Charcot and Wilbrand on cessation of dreaming [42, 43], a few authors reported on dream characteristics of patients with stroke [44, 46, 48, 105–107]. More recently, Hobson has given a first-person account of "abnormal visual percepts" in association with total insomnia and dream suppression following a lateral medullary stroke [108].

The most extensive work on dreaming in patients with focal brain damage has been performed by Solms [45]. Out of the 332 patients with cerebral lesions included in his systematic analysis, 83 received a diagnosis of cerebrovascular disease and 79 one of neoplasm. The four main changes in dreaming were the following: (1) global cessation of dreaming (35% of cases), usually with parietal or frontal lesions, and persistence over 1 year, particularly with right-sided lesions; (2) cessation of visual dreaming (1%), usually with medial occipitotemporal lesions; (3) dream–reality confusion (5%), usually with anterior limbic lesions; (4) recurrent nightmares (8%), without a specific topographic association.

An example of increased dreaming and nightmares is given in the following report of a 68-year-old woman seen in our department with severe right-sided hemispheric stroke in the middle cerebral artery distribution:

I saw little Appenzeller dogs entering my room through the window, at the same time my mother-in-law was sitting on my bedside. I tried to touch her with my right hand ... I saw a dark snake ... and then suddenly a bouquet of flowers of different colors which was offered to me by a chimpanzee ...

Solms' work is of paramount value but has some limitations. First, no awakenings from REM sleep were undertaken. The possibility that Charcot–Wilbrand syndrome (CWS) may in some cases be due to amnesia therefore cannot be ruled out. Second, the anatomical data are based mainly on CT scans, which do not always allow—particularly in the posterior fossa—determination of the exact extent of the underlying lesion. Third, the patients suffered from a variety of diseases and were examined at different intervals following disease onset, factors which can significantly influence oneiric experiences.

In a personal observation of CWS we tried to be aware of these limitations [49]. This previously healthy 73-year-old woman was hospitalized because of the sudden visual loss and weakness of the left side of her body. Brain magnetic resonance imaging showed an acute ischemic infarction of both deep occipital lobes (including the lingual gyrus on the right side) and the right posterolateral thalamus (Fig. 7). Clinical examination revealed a left homonymous hemianopsia, a right inferior quadrantanopsia, achromatopsia and bilateral decrease of visual acuity. The third night after stroke, the patient reported an unusual, very vivid and colorful dream:

An unknown person gives me a huge piece of cotton with a drawing on it of many dwarfs dressed in bright colors ... Later the same person shows a second ... larger piece of cotton with hundreds of tiny men on it. I try to find the first group of dwarfs but I don't succeed. I am upset. At this point the nurse wakes me up.

Following this unusual dream, the patient–who used to recall dreams three to four times per week before the stroke–stopped dreaming for months. A detailed neuropsychological examination was unrevealing. In particular, the patient could imagine well-known surroundings and places, was able to recognize familiar faces and displayed normal topographic orientation. Sleep studies were also essentially unremarkable with, especially, normal REM amounts, REM density and REM latency. Dream experiences were also denied after four awakenings from REM sleep undertaken during one polysomnographic recording. This report is interesting because it demonstrates the following three points: (1) the possible association of initial vivid dreaming with CWS (we postulated in this patient a release phenomenon of the partially damaged visual areas as responsible for the episode of vivid dreaming); (2) the existence of CWS as a distinct neuropsychological dysfunction, in the absence of other cognitive deficits (and in particular of a so-called irreminiscence, see above); (3) the independency of dream and REM sleep generators.

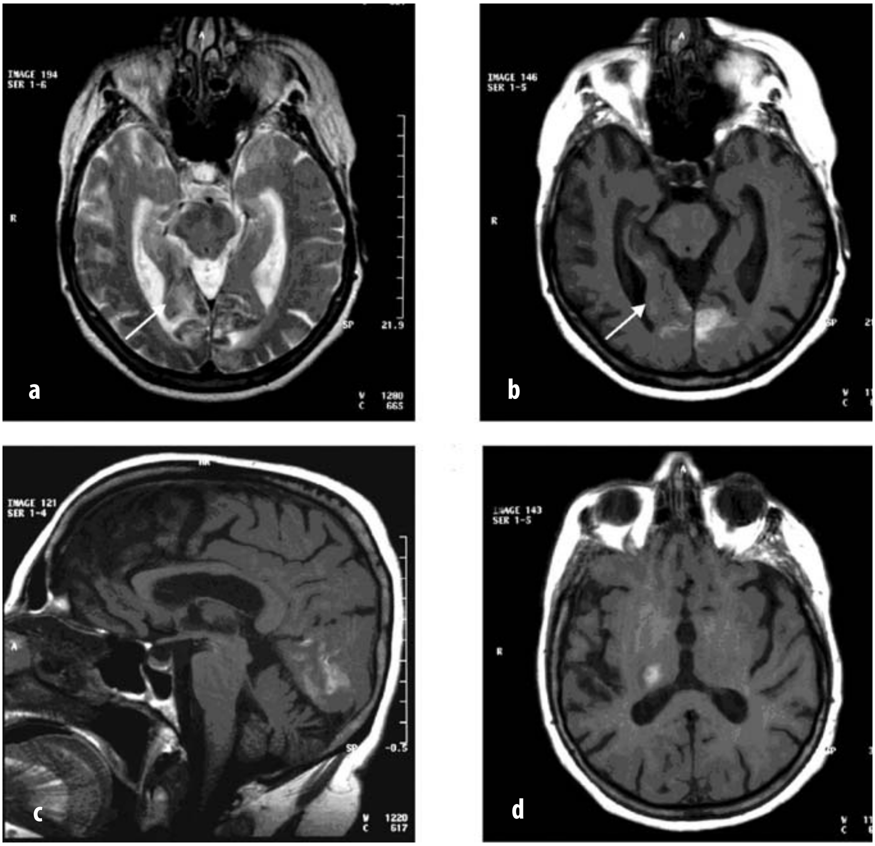


Fig. 7a-d. Brain MRI of a 73-year-old woman with total dream loss, performed one day after stroke onset (see text). **a** T_2 and **b–d** native T_1 images. The images show a subacute infarction of both occipital lobes and the right posterolateral thalamus (**d**) with slight bleeding in the infarcted area. On the right side the inferior lingual gyrus is involved (*arrows*)

Traumatic Brain Injury

Traumatic brain injury may lead to visual hallucinations, anxiety dreams and post-traumatic stress disorder [109–111]. Also one systematic study found overall only small differences in dreaming between the pre- and post-traumatic periods [112].

Depending on the presence and topography of the underlying brain damage, traumatic brain injury has been shown to cause dream changes (see also above) [45, 48]. It is noteworthy that out of the 361 patients included in Solms's systematic analysis, 108 carried the diagnosis of head trauma. In particular, lesions located in the parieto-occipital or temporal areas, independent of which side is affected, are frequently associated with decreased dream recall in the acute phase of brain damage and subsequent gradual recovery [113]. Loss of dreaming following traumatic brain injury has not been found to be correlated with an analogous reduction or loss of REM sleep [109]. The recovery phase of traumatic brain injury is sometimes complicated by the appearance of hallucinations, which are thought to reflect intrusions of REM sleep into wakefulness [114]. A few cases of post-traumatic narcolepsy and Kleine–Levin syndrome with the appearance of hallucinations and alterations of dreaming have been reported [115–117].

Parkinson's Syndromes

Changes in dream frequency and contents and the occurrence of nightmares, hallucinations and RBD (see above) are frequently seen in patients with Parkinson's syndromes. A correlation with the presence of other sleep–wake disturbances, cognitive changes, axial motor symptoms and medication (dopaminergic drugs, anticholinergics) has been recognized [55, 118, 119].

Visual hallucinations are reported by about 25–50% of patients with Parkinson's syndromes and predict Lewy body pathology with involvement of the parieto-occipital and limbic cortices [57, 119]. In Parkinson's disease (PD) hallucinations typically occur in the second half of the disease course, whereas they can occur early in the course of dementia with Lewy bodies (DLB). They usually arise at the end of the day without trigger or voluntary effort and persist for seconds or minutes, and insight is usually preserved. Visual hallucinations in PD have been linked with a higher risk of institutionalization [120]. Reduction of dopaminergic drugs and prescription of atypical neuroleptics (e.g., clozapine, quetiapine) and acetylcholinesterase inhibitors may improve hallucinations in PD [121, 122].

REM sleep behavior disorder is seen in about 25–50% of patients with PD and in more than 50% of patients with DLB and multisystem atrophy (MSA) [83, 85, 123–126]. In non-synucleinopathies (progressive supranuclear palsy, corticobasal degeneration) RBD is conversely rare. RBD can precede the onset of these conditions by years to decades and often exhibits a fluctuating course after its onset.

Dementia

Visual hallucinations are common in Alzheimer's disease (about 20% of cases) and dementia with Lewy bodies (>50% of cases) [119, 127, 128].

They correlate with cognitive decline and worse outcome (institutionalization, death) [129].

Alzheimer's disease can also lead to a reduction in the frequency and contents of dreaming [50, 51]. A few patients are observed to show improved language skills during sleep than wakefulness [130]. Treatment with acetylcholinesterase inhibitors may induce vivid dreams [41].

A demented syndrome with hypersomnia, enacted dreams and loss of slow wave sleep has recently been reported [131].

Epilepsy

Epileptic hallucinations are usually brief, simple, stereotyped and fragmentary associated with other seizure manifestations such as altered awareness, motor activity and automatisms [54]. Less commonly, epileptic hallucinations are complex ("dream-like") or, in seizures of temporal or parietal origin, consist of autoscopic experiences [132].

Recurrent epileptic nightmares have been reported in temporal lobe epilepsy as well as a post-stroke event [133, 134]. Scheffer et al. described a patient with autosomal dominant nocturnal frontal lobe epilepsy, who suffered from recurrent nightmares [135]. Epstein reported a patient with recurrent partial seizures in the form of painful dreaming of dying [136]. Occasionally, patients report similar contents in dreams and daytime epileptic seizures [137]. In 1954 Penfield and Jasper described in their book *Epilepsy and the Functional Anatomy of the Brain* the report of dream-like experiences in a patient following cortical (temporo-parietal) stimulation [138].

Patients with complex partial seizures have a higher frequency of dream recall than those with generalized seizures [139]. In a sleep laboratory study the frequency of dream recall was lower in patients with complex partial seizures after awakening from stage 2 NREM sleep than REM sleep, regardless of the side of epileptic focus, while the length and structural organization of dreams did not significantly differ in REM and NREM sleep [140]. Vercueil reported a 74-year-old woman who suffered from temporal lobe epilepsy due to a left dysplastic amygdala and who dreamed about a typical seizure accompanied by intense emotionality [141].

Migraine and Cluster Headache

Migraine aura is well recognized as a cause of simple visual hallucinations; complex dream-like visual hallucinations are less common. They are seen more frequently in migraine coma and in familial hemiplegic migraine. Rarely migraine-associated hallucinations may be of purely auditory nature, possibly reflecting a dysfunction of the temporal lobe due to ischemia [142, 143]. Olfactory, gustatory and other more complex hallucinations such as temporal–visual distortions have all been reported [144, 145]. Podoll and Robinson described the unusual case of auditory–visual synesthesia during migraine attacks: the visual hallucinations consisted in a colored geometric figure induced by the sound of the alarm clock, which then disappeared with the alarm [146]. A few cases of recurrent lilliputian hallucinations during migraine attacks have been reported, although this type of hallucinations is usually associated with mesencephalic and/or thalamic lesions (peduncular hallucinosis) [147].

Levitan et al. analyzed the content of 23 dreams that immediately preceded a migraine attack, finding a significant predominance of powerful negative emotions [148].

Recurrent dreams and nightmares have been reported as aura equivalent [149].

A few patients with cluster headache may report a visual aura, often characterized by black-and-white flashing lights preceding the attacks. One patient with cluster headache has been described to have olfactory hallucinations (“bad citrus fruits smell”) preceding the attack by 3–4 minutes [150].

Creutzfeldt–Jakob Disease

Fatal familial insomnia (FFI) is a hereditary, occasionally sporadic prion disease with prominent neurodegenerative changes affecting mainly the anteroventral and mediodorsal thalamic nuclei; the disease was described in 1986 [151]. The clinical features of FFI consist in progressive insomnia, sympathetic overactivation (perspiration, tachycardia, hypertension, evening pyrexia) and motor disturbances (dysarthria, myoclonus, disturbed coordination). Patients typically show peculiar episodes of vivid dream enactment that may spontaneously intrude into wakefulness, during which they are in a stupor-like state (“oneiric stuporous state”) and perform complex jerk-like movements, corresponding to the dream content reported afterwards [152, 153]. Polysomnography reveals profound sleep EEG changes with progressive disappearance of the hallmarks of NREM sleep including sleep spindles, K-complexes and slow-wave sleep [154]. Recently, Landolt et al. described similar sleep–wake disturbances in seven patients with sporadic Creutzfeldt–Jakob disease (sCJD); three patients also showed dream–reality confusion and had visual and/or acoustic hallucinations [155].

Encephalitis and Other CNS Infections

Dream-like hallucinations can be observed in patients with herpes encephalitis and other CNS infections involving the mesiotemporal areas (personal observation).

Delirium Tremens and Other Withdrawal Syndromes

Delirium tremens complicates sudden alcohol withdrawal after chronic alcohol abuse and carries a mortality of 5–15% [156]. The main symptoms are agitation, tremor, increased autonomic activity and an acute confusional state with polymodal hallucinations and dream enactment.

Polysomnography in acute alcohol withdrawal syndrome may show a markedly reduced sleep efficiency, reduction or absence of NREM sleep and long periods of wakefulness alternating with abruptly emerging periods of REM sleep without atonia observed by Plazzi et al. [157]. The patient repeatedly presented with intense enacting dreams with violent fighting behavior, jerks and talking throughout the night and also during daytime episodes of drowsiness. Whenever awakened he reported vivid dream content.

Acute withdrawal from barbiturate and benzodiazepine can also be complicated by visual hallucinations and vivid dreaming.

Autoimmune Disorders

Morvan's Fibrillary Chorea

This is a rare disorder which has been linked with autoantibodies against voltage-gated potassium channels [158]. Severe agrypnia usually lasts only a few weeks in about 90% of cases, but occasionally may persist and lead to death; it is associated in most cases with intense anxiety, delirium and often remarkable hallucinatory behavior with enacted dreams [159]. In addition, patients exhibit symptoms of autonomic hypertension and peculiar motor disturbances with agitation, cramps, myoclonic jerks and fasciculations ("fibrillary chorea").

Limbic Encephalitis

Limbic encephalitis is a rare disorder which has been linked with autoantibodies against voltage-gated potassium channels and with the presence of REM sleep behavior disorder [160, 161].

Guillain–Barré Syndrome

In a series of 139 patients with Guillain–Barré syndrome (GBS), mental status changes were found in 31% of patients, including vivid dreams (19%), illusions (30%, including an illusory body tilt), hallucinations (60%, mainly visual) and delusions (70%, mostly paranoid). They appeared in a median of 9 days after disease onset and lasted for a median of 8 days. Autonomic dysfunction, assisted ventilation and high cerebrospinal fluid (CSF) protein levels were significant risk factors for abnormal mental status in patients with GBS, whereas CSF hypocretin-1 levels were normal [162]. The origin of these disturbances remains speculative at this point.

Others

Patient with motor deficits, acquired blindness or aphasia not infrequently dream of moving, seeing and speaking normally (personal observation). Intraindividual differences in the latency of "incorporation" of daytime deficits in oneiric experiences are present.

Conversely, changes in dream frequency and contents have also been observed in patients with disorders of the peripheral nervous system such as myasthenia gravis [163]. Patients with phantom limbs often experience similar but painless sensations in their dreams [164].

Effects of Alcohol and Drugs

Alcohol consumption before bedtime leads to a shortened sleep latency, increased NREM sleep and reduced REM sleep during the first hours of sleep. Since alcohol has a relatively short half-life, symptoms related to alcohol withdrawal occur in the second half of the night, such as shallow disrupted sleep, sympathetic arousals and increased REM sleep with more vivid dreams and nightmares. Similar symptoms may appear during the weeks after alcohol withdrawal in alcoholic patients. Hershon found that 29% of a group of 100 alcoholics reported that they started again drinking in an attempt to alleviate their sleep and dream disturbances [165].

Numerous classes of drugs have been associated with nightmares, including sedative/hypnotics, beta-blockers, amphetamines and dopamine agonists [166]. Suppression of REM sleep with subsequent increased intensity of the REM episodes is thought to be the pharmacological mechanism responsible for nightmares induced by beta-blockers [167]. Dopamine receptor stimulation may be another common mechanism involved in the generation of hallucinations and nightmares, accounting for the effects of amphetamines and dopamine agonists such as levodopa, pergolide and cabergoline.

Conclusions

Dreaming changes in patients with neurological disorders have been poorly studied, although their frequency is not negligible (e.g., in Parkinson's syndromes, stroke and narcolepsy). Despite the little attention that neurologists typically pay to dream reports, their assessment can be of diagnostic interest (e.g., REM sleep behavior disorder, visual hallucinations), have prognostic implications (e.g., visual hallucinations in Parkinson's disease) and be a source of major distress for patients and their bed partners (e.g., nightmares, RBD). In addition, specific and effective management of "anxiety dreams" and disturbing "dream-like experiences" relies upon a knowledge of their phenomenological and etiological differential diagnosis (Tables 1 and 2). Finally, systematic studies of dream experiences are expected to offer unique insights into the brain, mind and emotions of neurological patients.

Table 1. Differential diagnosis of anxiety/violent dreams

Nightmares
Rapid and full arousal from REM sleep; minimal concomitant autonomic symptoms (tachycardia, palpitations, dyspnea, sweating); good recall of oneiric activity
Episodes occur in the second part of the night

Sleep terrors (pavor nocturnus), sleepwalking
Rapid but incomplete arousal from deep NREM sleep; variable, occasionally strong concomitant autonomic symptoms; usually poor/partial recall of oneiric activity
Single or few episodes (usually 1–2) occurring in the first part of the night

continue ⟶

Table 1 *continue*

Nocturnal panic attacks
Rapid and complete arousal from light NREM sleep; variable, occasionally strong concomitant autonomic symptoms; usually no recall of oneiric activity

REM sleep behavior disorder
Excessive motor activity during REM sleep without arousal; typically no concomitant autonomic symptoms; recall of violent oneiric activity (the day after)
Episodes occur in the second part of the night

Seizures
Variable arousal from NREM or REM sleep; variable concomitant motor and autonomic symptoms; usually no recall of oneiric activity
Typically several (often >5) attacks per night

Arousals secondary to sleep-disordered breathing
Variable arousal from all stages of sleep; no or minimal motor, but conversely often strong concomitant autonomic symptoms; usually no recall of oneiric activity

Post-traumatic stress disorder

Epic dreaming

Nocturnal visual hallucinations

Table 2. Neurological causes of dream-like hallucinations

Peripheral hearing loss
Peripheral visual loss (Charles Bonnet syndrome)
Hemispheric/brainstem stroke (e.g., peduncular hallucinosis)
Other focal brain lesions (e.g., herpes encephalitis)
Parkinson’s disease
Dementia with Lewy bodies
Alzheimer’s dementia
Migraine
Narcolepsy
Epilepsy
Metabolic/toxic encephalopathy
Withdrawal syndromes (e.g., Korsakoff’s syndrome)

References

1. Kruger SE (1992) Dreaming in the Middle Ages. Cambridge: Cambridge University Press
2. Cardano G (1989) Sul sonno e sul sognare. Supertascibili Marsilio

3. Von Haller A (1996) irst lines of physiology. 1786. Translated from the Latin. Reprinted New York: Johnson Reprint Company
4. Maury A (1848) Les hallucinations hypnagogiques ou les erreurs des sens dans l'état intermédiaire entre la veille el le sommeil. Ann Med Psychol 11:26–40
5. De Manacéïne M, Manasseina M (1897) Sleep: its physiology, pathology, hygiene, and psychology. London, Walter Scott Ltd
6. Calkins MW (1893) Statistics of dreams. Am J Psychol 5:311–343
7. Heermann G (1838) Beobachtungen und Betrachtungen über die Träume der Blinden, ein Beitrag zur Physiologie und Psychologie der Sinne. Monatsschr Med 3:116–180
8. Jastrow J (1888) Dreams of the blind. New Princeton Review 5:18–34
9. Vaschide N (1920) Le sommeil et les rêves. Paris, Ernest Flammarion
10. Mitchell SW (1896) Remarks on the effects of Anhelonium lewinii (the mescal button). Br Med J 1625–1629
11. Lasègue C (1881) Délire alcoolique n'est pas un délire, mais un rève. Arch Gen Med 88:513–536
12. Ohlmeyer P, Brilmayer H, Hüllstrung H (1944) Periodische Vorgänge im Schlaf. Pflügers Arch 248:559–560
13. Loomis AL, Harvey EN, Hobart G (1935) Potential rhythms of the cerebral cortex during sleep. Science 81:597–598
14. Hess R, Koella WP, Akert K (1953) Cortical and subcortical recordings in natural and artificially induced sleep in cats. Electroencephalogr Clin Neurophysiol Suppl 5:75–90
15. Lawson RW (1950) Blinking and sleep. Nature 165:81–82
16. Aserinsky E, Kleitman N (1953) Regularly occurring periods of eye motility, and concomitant phenomena, during sleep. Science 118:273–274
17. Aserinsky E, Kleitman N (1955) Two types of ocular motility occurring in sleep. J Appl Physiol 8:11–18
18. Jouvet M, Michel F (1959) Corrélations éléctromyographiques du sommeil chez le chat decortiqué et mésencéphalique chronique. C R Séances Soc Biol Fil 153:422–425
19. Jouvet M, Michel F (1960) Release of the "paradoxal phase" of sleep by stimulation of the brain stem in the intact and chronic mesencephalic cat. C R Soc Biol 154:636–641
20. Jouvet M, Delorme F (1965) Locus coeruleus et sommeil paradoxal. C R Soc Biol 159:895–899
21. Dement W, Kleitman N (1957) The relation of eye movements during sleep to dream activity: an objective method for the study of dreaming. J Exp Psychol 53:339–346
22. Dement W, Wolpert EA (1968) The relation of eye movements, body motility, and external stimuli to dream content. J Exp Psychol 55:543–553
23. Dement W (1960) The effect of dream deprivation. Science 131:1705–1707
24. Hobson JA, McCarley RW, Wyzinski PW (1975) Sleep cycle oscillation: reciprocal discharge by two brainstem neuronal groups. Science 189:55–58
25. Hobson JA, McCarley RW (1977) The brain as a dream state generator: an activation-synthesis hypothesis of the dream process. Am J Psychiatry 134:1335–1348
26. Foulkes D (1962) Dream reports from different stages of sleep. J Abnorm Soc Psychol 65:14–25
27. Rechtschaffen A, Verdone P, Wheaton J (1963) Reports of mental activity during sleep. Can Psychiatr Assoc J 257:409–414
28. Cavallero C, Cicogna P, Natale V et al (1992) Slow wave sleep dreaming. Sleep

15:562–566
29. Landolt HP, de Boer LP (2001) Effect of chronic phenelzine treatment on REM sleep: report of three patients. Neuropsychopharmacology 25:S63–S67
30. Antrobus JS (1983) REM and NREM sleep reports. Psychophysiology 20:562–568
31. Antrobus JS (1991) Dreaming: cognitive processes during cortical activation and high afferent thresholds. Psychol Rev 98:96–121
32. Braun AR, Balkin TJ, Wesensten NJ et al (1997) Regional cerebral blood flow throughout the sleep-wake cycle. An H2(15)O PET study. Brainb 120:1173–1197
33. Maquet P, Peters J, Aerts J et al (1996) Functional neuroanatomy of human rapid-eye-movement sleep and dreaming. Nature 383:163–616
34. Braun AR, Balkin TJ, Wesensten NJ et al (1998) Dissociated pattern of activity in visual cortices and their projections during human rapid eye movement sleep. Science 279:91–95
35. Lövblad KO, Remonda I, Heid O et al (1999) Silent functional magnetic resonance imaging demonstrates focal activation in rapid eye movement sleep. Neurology 53:2193–2195
36. Maquet P, Ruby P, Maudoux A et al (2005) Human cognition during REM sleep and the activity profile within frontal and parietal cortices. A reappraisal of functional neuroimaging data. Prog Brain Res 150:219–227
37. Maquet P, Degueldre C, Delfiore G et al (1997) Functional neuroanatomy of human slow wave sleep. J Neurosci 17(8):2807–2812
38. Fell J, Fernandez G, Lutz MT et al (2006) Rhinal-hippocampal connectivity determines memory formation during sleep. Brain 129:108–114
39. Arnulf I, Bonnet AM, Damier P et al (2000) Hallucinations, REM sleep, and Parkinson’s disease: a medical hypothesis. Neurology 55:281–288
40. Blanke O, Landis T, Spinelli L, seeck M (2004) Out-of-body experience and autoscopy of neurological origin. Brain 127:243–258
41. Singer M, Romero B, Koenig E et al (2005) Nightmares in patients with Alzheimer’s disease caused by donepezil. Therapeutic effect depends on the time of intake. Nervenarzt 76:1127–1129
42. Charcot JM (1883) Un cas de suppression brusque et isolée de la vision mentale des signes et des objets (formes et couleurs). Progr Med 2:568–571
43. Wilbrand H (1887) Ein Fall von Seelenblindheit und Hemianopsie mit Sectionsbefund. Dtsch Z Nervenheilkd 2:361–387
44. Grünstein AM (1924) Die Erforschung der Träume als eine Methode der topischen Diagnostik bei Grosshirnerkrankungen. Zeit Ges Neurol Psychiatr 93:416–420
45. Solms M (1997) The neuropsychology of dreams. Mahwah, NJ, Erlbaum
46. Humphrey M, Zangwill O (1951) Cessation of dreaming after brain injury. JNNP 14:322–325
47. Cathala HP, Laffont F, Siksou M et al (1983) Sleep and dreams in patients with parietal and frontal lobe lesions. Rev Neurol (Paris) 139:497–508
48. Murri I, Arena R, Siciliano G et al (1984) Dream recall in patients with focal cerebral lesions. Arch Neurol 41:183–185
49. Bischof M, Bassetti CL (2004) Total dream loss: a distinct neuropsychological dysfunction after bilateral PCA stroke. Ann Neurol 56:583–586
50. Greenberg R (1968) Cerebral cortex lesions: the dream process and sleep spindles. Arch Gen Psychiatry 18:203–209
51. Cathala HP, Laffont F, Esnault S et al (1988) Sleep and dreams in Korsakoff’s amnesia due to alcoholism. Neurophysiol Clin 18:173–186
52. Stickgold R, Malia A, Maguire D et al (2000) Replaying the game: hypnagogic images in normals and amnesics. Science 90:350–353

53. Peralta R, Gomes A, Costa A et al (2005 Dream recall and dream content in posterior cerebral artery (PCA) stroke patients. Sleep Med S207–S208
54. Manford M, Andermann F (1998) Complex visual hallucinations. Clinical and neurobiological insights. Brain 121:1819–1840
55. Goetz CG, Wuu J, Curgian LM, Leurgans S (2005) Hallucinations and sleep disorders in PD: six-year prospective longitudinal study. Neurology 64:81–86
56. Sturzenegger C, Bassetti C (2004) The clinical spectrum of narcolepsy with cataplexy: a reappraisal. J Sleep Res 13:395–406
57. Fenelon G, Mahieux F, Huon R, Ziegler M (2000) Hallucinations in Parkinson's disease: prevalence, phenomenology and risk factors. Brain 123:733–745
58. McCormick L, Nielsen T, Ptito M et al (1997) REM sleep dream mentation in right hemispherectomized patients. Neuropsychologia 35:695–701
59. Greenwood P, Wilson DH, Gazzaniga MS (1977) Dream report following commissurotomy. Cortex 13:311–316
60. Bassetti C (2005) Narcolepsy: selective hypocretin (orexin) neuronal loss and multiple signaling deficiencies. Neurology 65:1152–1153
61. Dahmen M, Kasten N, Mittag K, Muller MJ (2002) Narcoleptic and schizophrenic hallucinations. Implications for differential diagnosis and pathophysiology. Eur J Health Econ 3 Suppl 2:S94–S98
62. Cheyne JA, Rueffer SD, Newby-Clark IR (1999) Hypnagogic and hypnopompic hallucinations during sleep paralysis: neurological and cultural construction of the nightmare. Consc Cogn 8:319–337
63. Lee JH, Bliwise DL, Lebret-Bories E et al (1993) Dream-disturbed sleep in insomnia and narcolepsy. J Nerv Ment Dis 181:320–324
64. Fosse R, Stickgold R, Hobson JA (2002) Emotional experience during rapid-eye-movement sleep in narcolepsy. Sleep 25:724–732
65. Wilson SAK (1928) The narcolepsies. Brain 51:63–109
66. Attarian HP, Schenck CH, Mahowald MW (2000) Presumed REM sleep behavior disorder arising from cataplexy and wakeful dreaming. Sleep Med 1:131–133
67. Spanos NP, DuBreuil C, McNulty SA et al (1995) The frequency and correlates of sleep paralysis in a university sample. J Res Personality 24:285–305
68. Bassetti C, Aldrich MS (1997) Idiopathic hypersomnia. A series of 42 patients. Brain 120:1423–1435
69. Bassetti CL, Pelayo R, Guilleminault C (2005) Idiopathic hypersomnia. In: Kryger MH, Roth T, Dement WC (eds) Principles and practice of sleep medicine, 4th edn. WB Sanders, Philadelphia
70. Arnulf I, Zeitzer JM, File J et al (2005) Kleine-Levin syndrome: a systematic review of 186 cases in the literature. Brain 128:2763–2776
71. Merriam AE (1986) Kleine-Levin syndrome following acute viral encephalitis. Biol Psychiatry 21:1301–1304
72. Broughton RJ (1968) Sleep disorders: disorders of arousal? Enuresis, somnambulism, and nightmares occur in confusional states of arousal, not in "dreaming sleep." Science 159:1070–1078
73. Roger H (1932) Les troubles du sommeil–hypersomnies, insomnies, parasomnies. Masson, Paris
74. Seashore CE (1916) The frequency of dreams. Sci Monthly 2:467–474
75. Gastaut H, Broughton RJ (1965) A clinical and polygraphic study of episodic phenomena during sleep. Biol Psychiatry 7:197–221
76. Jacobson A, Kales A, Lehmann D, Zweizig JR (1965) Somnambulism: all-night electroencephalographic studies. Science 148:975–977

77. Bassetti C, Vadilonga D, Tafti M, Hess CW (2002) Adult sleepwalking: a study of 74 patients. Neurology 58[Suppl 3]:509–510
78. Schenck CH, Mahowald MW (1995) A polysomnographically documented case of adult somnambulism with long-distance automobile driving and frequent nocturnal violence: parasomnia with continuing danger as a noninsane automatism? Sleep 18:765–772
79. Rosenfeld DS, Furman Y (1994) Pure sleep panic: two case reports and a review of the literature. Sleep 17:462–465
80. Schenck CH, Mahowald MW (1995) A disorder of epic dreaming with daytime fatigue, usually without polysomnographic abnormalities, that predominantly affects women. Sleep Res 24:137
81. Silber MH, Hansen MR, Girish M (2005) Complex nocturnal visual hallucinations. Sleep Med 6:363–366
82. Schenck CH, Bundlie SR, Ettinger MG, Mahowald MW (1986) Chronic behavioral disorders of human REM sleep: a new category of parasomnia. Sleep 9:293–308
83. Olsen EJ, Boeve BF, Silber MH (2000) Rapid eye movement sleep behaviour disorder: demographic, clinical and laboratory findings in 93 cases. Brain 123:331–339
84. Fantini ML, Corona A, Clerici S, Ferini-Strambi L (2005) Aggressive dream content without daytime aggressiveness in REM sleep behavior disorder. Neurology 65:1010–1015
85. Gagnon JF, Bedard MA, Fantini ML et al (2002) REM sleep behavior disorder and REM sleep without atonia in Parkinson's disease. Neurology 59:585–589
86. Massicotte-Marquez J, Carrier J, Decary A et al (2005) Slow-wave sleep and delta power in rapid eye movement sleep behavior disorder. Ann Neurol57:277–282
87. Fantini ML, Michaud M, Gosselin N et al (2005) Periodic leg movements in REM sleep behavior disorder and related autonomic and EEG activation. Neurology 59:1889–1894
88. Ferini-Strambi L, Di Gioia MR, Castronovo V et al (2004) Neuropsychological assessment in idiopathic REM sleep behavior disorder (RBD): does the idiopathic form of RBD really exist? Neurology 62:41–45
89. Fantini ML, Gagnon JF, Petit D et al (2003) Slowing of electroencephalogram in rapid eye movement sleep behavior disorder. Ann Neurol 53:774–780
90. Eisensehr I, Linke R, Noachtar S et al (2000) Reduced striatal dopamine transporters in idiopathic rapid eye movement sleep behaviour disorder. Comparison with Parkinson's disease and controls. Brain 123:1155–1160
91. Eisensehr I, Linke R, Tatsch K et al (2003) Increased muscle activity during rapid eye movement sleep correlates with decrease of striatal presynaptic dopamine transporters. IPT and IBZM SPECT imaging in subclinical and clinically manifest idiopathic REM sleep behavior disorder, Parkinson's disease, and controls. Sleep 26:507–512
92. Schenck CH, Bundlie ST, Mahowald MW (1996) Delayed emergence of a parkinsonian disorder in 38% of 29 older men initially diagnosed with idiopathic rapid eye movement sleep behaviour disorder. Neurology 46:388–393
93. Braak H, Del Tredici K, Rub U et al (2003) Staging of brain pathology related to sporadic Parkinson's disease. Neurobiol Aging 24(2):197–211
94. Stiasny-Kolster K, Doerr Y, Moller JC et al (2005) Combination of "idiopathic" REM sleep behaviour disorder and olfactory dysfunction as possible indicator for alpha-synucleinopathy demonstrated by dopamine transporter FP-CIT-SPECT. Brain 128:126–137
95. Hohagen F, Rink K, Kappler C et al (1993) Prevalence and treatment of insomnia in

general practice. A longitudinal study. Eur Arch Psychiatry Clin Neurosci 242:329–336

96. Cory TL, Ormiston DW. Predicting the frequency of dream recall. J Abnorm Psychol 1975;84:261–266.
97. Halliday G (1988) Relationship of spontaneous awakenings to dreams and nightmares. Assoc Stud Dreams Newslett 5:4
98. Schredl M, Montasser A (1996/97) Dream recall: state or trait variable? Part II: state factors, investigations, and final conclusions. Imagination Cogn Pers 16:231–261
99. Schredl M, Schafer G, Weber B, Heuser I (1998) Dreaming and insomnia: dream recall and dream content of patients with insomnia. J Sleep Res 7:191–198
100. Greenberg R (1967) Dream interruption insomnia. J Nerv Ment Dis 144:18–21
101. Schredl M, Kraft-Schneider B, Kröger H, Heuser I (1999) Dream content of patients with sleep apnea. Somnologie 3:319–323
102. Gross M, Lavie P (1994) Dreams in sleep apnea patients. Dreaming 4:195–204
103. Iranzo A, Santamaria J (2005) Severe obstructive sleep apnea/hypopnea mimicking REM sleep behavior disorder. Sleep 28:203–206
104. Schredl M (2001) Dream recall frequency and sleep quality of patients with restless legs syndrome. Eur J Neurol 8:185–189
105. Adler A (1959) Course and outcome of visual agnosia. J Nerv Ment Dis 111:41–51
106. Gloning K, Sternbach I (1953) The dreams in case of cerebral focal lesions. Wien Z Nervenheilkd Grenzgeb 6:302–329
107. Greenberg R (1996) Cerebral cortex lesions: the dream process and sleep spindles. Cortex 2:357–366
108. Hobson JA (2002) Sleep and dream suppression following a lateral medullary infarct: a first-person account. Conscious Cogn 11:377–390
109. Prigatano GP, Stahl ML, Orr WC, Zeiner HK (1982) Sleep and dreaming disturbances in closed head injury patients. J Neurol Neurosurg Psychiatry 45:78–80
110. Askenasy JJ, Gruskiewicz J, Braun J, Hackett P (1986) Repetitive visual images in severe war head injuries. Resuscitation 13:191–201
111. Glaesser J, Neuner F, Lutgehetmann R et al (2004) Posttraumatic stress disorder in patients with traumatic brain injury. BMC Psychiatry 4:5
112. Benyakar M, Tadir M, Groswasser Z, Stern MJ (1988) Dreams in head-injured patients. Brain Inj 2:351–356
113. Murri L, Mancino M, Massetani R et al (1989) Effect of acute and chronic brain damage on dreaming. Res Comm Psychol Psychiatry Behav 14:121–142
114. Mahowald MW, Woods SR, Schenck CH (1998) Sleeping dreams, waking hallucinations, and the central nervous system. Dreaming 8:89–102
115. Maccario M, Ruggles KM, Meriwether MW (1987) Posttraumatic narcolepsy. Military Med 152:370–371
116. Pelin Z, Öztürk L, bozluolcay M (2004) Posttraumatic Kleine-Levin syndrome: a case report. Eur Psychiatry 19:521–522
117. Will RG, Young JP, Thomas DJ (1988) Kleine-Levin syndrome: report of two cases with onset of symptoms precipitated by head trauma. Br J Psychiatr 152:410–412
118. Levy G, Tang MX, Cote LJ et al (2000) Motor impairment in PD: relationship to incident dementia and age. Neurology 55:539–544
119. Williams DR, Lees AJBassettiVisual hallucinations in the diagnosis of idiopathic Parkinson's disease: a retrospective autopsy study. Lancet Neurol 4:605–610
120. Goetz CG, Leurgans S, Pappert EJ et al (2001) Prospective longitudinal assessment of hallucinations in Parkinson's disease. Neurology 57:2078–2082
121. The French Clozapine Parkinson Study Group (1999) Clozapine in drug-induced

psychosis in Parkinson’s disease. Lancet 353:2041–2042

122. Emre M, Aarsland D, Albanese A et al (2004) Rivastigmine for dementia associated with Parkinson’s disease. N Engl J Med 351:2509–2518
123. Comella CL, Nardine TM, Diederich NJ, Stebbins GT (1998) Sleep-related violence, injury, and REM sleep behavior disorder in Parkinson’s disease. Neurology 51:526–529
124. Plazzi G, Cortelli P, Montagna P et al (1998) REM sleep behaviour disorder differentiates pure autonomic failure from multiple system atrophy with autonomic failure. J Neurol Neurosurg Psychiatry 64:683–685
125. Boeve BF, Silber MH, Ferman TJ et al (1998) REM sleep behavior disorder and degenerative dementia: an association likely reflecting Lewy body disease. Neurology 51:363–370
126. Scaglione C, Vignatelli L, Plazzi G et al (2005) REM sleep behaviour disorder in Parkinson’s disease: a questionnaire-based study. Neurol Sci 25:316–321
127. Cummings JL, Kaufer D (1996) Neuropsychiatric aspects of Alzheimer’s disease: the cholinergic hypothesis revisited. Neurology 47:876–883
128. Ropacki SA, Jeste DV (2005) Epidemiology of and risk factors for psychosis of Alzheimer’s disease: a review of 55 studies published from 1990 to 2003. Am J Psychiatry162:2022–2030
129. Scarmeas N, Brandt J, Albert M et al (2005) Delusions and hallucinations are associated with worse outcome in Alzheimer disease. Arch Neurol 62:1601–1608
130. Fisher CM (1988) Neurologic fragments. Clinical observations in demented patients. Neurology 8:1868–1873
131. Cibula JE, Eisenschenk S, Gold M et al (2002) Progressive dementia and hypersomnolence with dream-enacting behavior: oneiric dementia. Arch Neurol 59:630–634
132. Maillard L, Vignal JP, Gavaret M et al (2004) Semiologic and electrophysiologic correlations in temporal lobe seizure subtypes. Epilepsia 45:1590–1599
133. Silvestri R, Bromfield E (2004) Recurrent nightmares and disorders of arousal in temporal lobe epilepsy. Brain Res Bull 63:369–376
134. Boller F, Wright DG, Cavalieri R, Mitsumoto H (1975) Paroxysmal “nightmares.” Sequel of a stroke responsive to diphenylhydantoin. Neurology 5:1026–1028
135. Scheffer IE, Bhatia KP, Lopes-Cendes I et al (1995) Autosomal dominant nocturnal frontal lobe epilepsy. A distinctive clinical disorder. Brain 118:61–73
136. Epstein AW (1979) Effect of certain cerebral hemispheric diseases on dreaming. Biol Psychiatry 14:77–93
137. Reami DO, Silva DF, Albuquerque M, Campos CJ (1991) Dreams and epilepsy. Epilepsia 32:51–53
138. Penfield WG, Jasper H (1954) Epilepsy and the functional anatomy of the human brain. Churchill, London
139. Bonanni E, Cipolli C, Iudice A et al (2002) Dream recall frequency in epilepsy patients with partial and generalized seizures: a dream diary study. Epilepsia 43:889–895
140. Cipolli C, Bonanni E, Maestri M et al (2004) Dream experience during REM and NREM sleep of patients with complex partial seizures. Brain Res Bull 63:407–413
141. Vercueil L (2005) Dreaming of seizures. Case report. Epilepsy Behav 7:127–128
142. Rubin D, McAbee GN, Feldmann-Winter LB (2002) Auditory hallucinations associated with migraine. Headache 42:646–648
143. Baloh RW (1997) Neurotology of migraine. Headache 37:615–621
144. Morrison DP (1990) Abnormal perceptual experiences in migraine. Cephalalgia 10:273–277

145. Blau JN, Solomon F (1985) Smell and other sensory disturbances in migraine. J Neurol 232:275–276
146. Podoll K, Robinson D (2002) Auditory-visual synaesthesia in a patient with basilar migraine. J Neurol 249:476–477
147. Podoll K, Robinson D (2001) Recurrent Lilliputian hallucinations as visual aura symptom in migraine. Cephalalgia 21:990–992
148. Levitan H (1984) Dreams which culminate in migraine headaches. Psychother Psychosom 41:161–166
149. Podoll K, Topper R, Robinson D, Sass H (2000) Recurrent dreams as migraine aura symptoms. Fortschr Neurol Psychiatr 68:145–149
150. Silberstein SD, Niknam R, Rozen TD, Young WB (2000) Cluster headache with aura. Neurology 54:219–221
151. Lugaresi E, Medori R, Montagna P et al (1986) Fatal familial insomnia and dysautonomia with selective degeneration of thalamic nuclei. N Engl J Med 315:997–1003
152. Tinuper P, Montagna P, Medori R et al (1989) The thalamus participates in the regulation of the sleep-waking cycle. A clinico-pathological study in fatal familial thalamic degeneration. Electroencephalogr Clin Neurophysiol 73:117–123
153. Montagna P (2005) Fatal familial insomnia: a model disease in sleep physiopathology. Sleep Med Rev 9:339–353
154. Ferrillo F, Plazzi G, Nobili L et al (2001) Absence of sleep EEG markers in fatal familial insomnia healthy carriers: a spectral analysis study. Clin Neurophysiol 112:1888–1892
155. Landolt HP, Glatzel M, Blatter T et al (2006) Sleep-wake disturbances in sporadic Creutzfeldt-Jakob disease. Neurology (in press)
156. Erwin WE, Williams DB, Speir WA (1998) Delirium tremens. South Med J 91:425–432
157. Plazzi G, Montagna P, Meletti S, Lugaresi E (2002) Polysomnographic study of sleeplessness and oneiricisms in the alcohol withdrawal syndrome. Sleep Med 3:279–282
158. Liguori R, Vincent A, Clover L et al (2001) Morvan's syndrome: peripheral and central nervous system and cardiac involvement with antibodies to voltage-gated potassium channels. Brain 124:2417–2426
159. Provini F, Lombardi C, Lugaresi E (2005) Insomnia in neurological diseases. Semin Neurol 25:81–89
160. Lee EK, Maselli RA, Ellis WG, Agius MA (1998) Morvan's fibrillary chorea: a paraneoplastic manifestation of thymoma. J Neurol Neurosurg Psychiatry 65:857–862
161. Iranzo A, Graus F, Clover L et al (2006) Rapid eye movement sleep behavior disorder and potassium channel antibody-associated limbic encephalitis. Ann Neurol 59:178–181
162. Cochen V, Arnulf I, Demeret S et al (2005) Vivid dreams, hallucinations, psychosis and REM sleep in Guillain-Barre syndrome. Brain 128:2535–2545
163. Happe S, Klosch G, Zeitlhofer J (2004) Perception of dreams and subjective sleep quality in patients with myasthenia gravis. Neuropsychobiology 50:21–27
164. Frank B, Lorenzoni E (1989) Experiences of phantom limb sensations in dreams. Psychopathology 22:182–187
165. Hershon HI (1977) Alcohol withdrawal symptoms and drinking behavior. J Stud Alcohol 38:953–971
166. Hershon HI (1977) Alcohol withdrawal symptoms and drinking behavior. J Stud Alcohol 38:953–971
167. Koella WP (1985) CNS-related (side-) effects of b-blockers with special reference to mechanisms of action. Eur J Clin Pharmacol 28[Suppl]:55–63

Part 4
The Fetus and the Newborn

Chapter 15

On the Onset of Human Fetal Behavior

Alessandra Piontelli

Introduction

Psychoanalysts in their daily practice can find themselves confronted with adults who have seemingly regressed to a fetal stage. In these circumstances, the analyst is often treated as a nurturing “container” having to fulfil any need and function of the patient, including thinking and verbal communication. These exceedingly passive patients simply expect the analyst to “do it all,” as a highly idealized uterus would. Some of the most regressed patients seem to live as if they are still in an unborn state, enclosed inside a “mental womb” which renders them almost totally impervious to life in the outside world. The negative facets of this kind of extreme regression can be manifested in claustrophobic feelings: the patient feels trapped inside a container which has turned into a persecuting object robbing him or her of any ego functioning. Many other features typify this kind of “fetal transference.” However, even less regressed patients can report dreams and fantasies that seem clearly related to intrauterine life. All this inevitably elicits in the analysts a retrospective interest in the actual facts characterizing fetal life, but at the same time makes him or her realize how distant these patients are from the concrete reality of the intrauterine past and how this past is inextricably colored by all sorts of feelings and events pertaining to later stages alone [1].

Child analysts, especially when working with very young children (i.e., children between the ages of two and three), meet daily with vivid fantasies of life inside the womb and of birth. Young children are still close in time to the actual event of their birth and for them their intrauterine past is not yet a remote past. Presumably the seeming memories they present the analyst with are relatively less distorted and colored by the many accretions of later life. Additionally, child analysts have the chance to make firsthand acquaintance with the child's parents, thus gaining a fairly direct impression of them and of what the child's early object relations might have been like. Furthermore, parents are usually keen to describe their children's actual pre- and perinatal history [1]. However, when trying to reconstruct the intrauterine past of these children through their current manifestations in the transference, dealing as they are with reconstructed and not actual facts, child analysis largely suffers from the same limitations as adult analysis.

Infant research is close to pre-natal life. However the neonate is not a fetus. Even the premature infant, though chronologically "true to its fetality" [2], has been catapulted into a totally different environment and has to adapt to it. In the term infant, and especially in the premature infant, some features pertaining to the fetal stage may still be observed, but others will have been replaced, modified or erased.

The investigation of human fetal behavior has understandably been guided principally by clinical considerations. Researchers mainly concentrated on the later stages of pregnancy, searching for potential pointers of fetal compromise and/or well-being. For this reason, relatively little attention has been paid to the previable fetus. The present work, by describing the onset of human fetal behavior during the first half of pregnancy, may appeal to psychoanalysts, child analysts and infant researchers, including neuroscientists, who want to deepen their understanding of the actual behavior of the fetus, its onset and evolution up to mid-pregnancy. By starting from the onset, later facts can be clarified and a "natural history" of our development be defined.

Complex technological advances have revolutionized the medical approach to gestation. Fetal medicine is now an autonomous and rapidly expanding field and the fetus has thus become a patient in its own right. With the help of ultrasound, studies of human fetal behavior have begun to emerge from the mist of anecdotal evidence. A wealth of data on fetal competencies is now available and rapidly accumulating, but for obvious reasons any experimental approach is difficult and most often downright impossible.

The visibility of the fetus, coupled with its inaccessibility to experimental research, has often favored in researchers from other fields–including psychoanalysts–a tendency to use it as a kind of blackboard onto which to project the wildest hypotheses. Fetal motions are often interpreted as hyperactivity disorders or anxiety-driven states. Moreover, continuities between pre- and postnatal life, imagined to be displayed in every possible behavior, complex feelings, "object relations," defenses, hypothetical genetic programs and many more have all been postulated and believed to be true [3].

In this chapter a few general considerations that may dispel some of the many myths encumbering fetal life will be discussed first. The main behavioral events characterizing early pregnancy and their evolution with advancing gestational age will then be reported in some detail and a tentative interpretation of these facts will be put forward. Some hypotheses of a more speculative nature linking early behavior with the possible onset of sleep will be discussed next. Finally, data derived from the intrauterine behavior of twin fetuses will be presented in order to illustrate how they can further our knowledge of prenatal life. Since twins are doubly exposed to a host of myths, a distinction between myths and facts will again be considered.

Myths

The image of the fetus which most readily comes to the public's mind is that of the one in early first trimester. Visualization on an ultrasound scan of the fetal body in its entirety is only possible up to 19–20 weeks. Beyond this gestational age, only parts of the fetal body can be observed at any one time. A third trimester fetus merely visible in segments is clearly less glamorous and less prone to being illustrated in nonspecialist publications. As will be described, early fetuses move a lot and this lively activity is also more appealing for a nonspecialist audience, which would find relatively long stretches of immobility tedious to watch.

Growth and development are extremely concentrated and accelerated in fetal life. We often talk about days and weeks rather than months and years, as in postnatal life. What is appropriate to ascribe to a certain gestational age may not be equally pertinent to an earlier or later phase. The tendency to view all developmental phenomena as a continuous process in which earlier events are regarded as being connected by a succession of cause and effect relationships with later events is very pervasive. Implicit in this view is the idea that the sole purpose of development is the evolution into a mature organism. Fetuses inhabit a profoundly different environment from the neonate and even more so from the adult. Furthermore, fetuses change rapidly during their development. As Oppenheim says, "On the way to becoming an adult, the developing organism must also be adapted to the special ecological conditions it encounters at each step in the progression; that is, it must possess ontogenetic adaptations" [4]. Such adaptations may entail the formation of structures and processes tailored for functioning at a given stage, but unneeded and unsuited for the requirements of later ones. Structures and functions used adaptively for a given developmental stage may be lost and discarded or reorganized and incorporated into later traits. This does not mean that "ontogenetic adaptations" are just imperfect, immature, inadequate or embryonic manifestations of a fully developed feature. Ontogenetic adaptations are perfectly functional and suitable for a given, even if very brief, stage.

Despite all these rapid developmental changes, most people refer to "the fetus" in general, quite independent of gestational age. By referring to "the fetus" in general, they either mean the early, active first trimester one or the mature fetus approaching birth.

The lively activity of the early fetus is all too often interpreted as representing wakefulness, which is then considered identical to intentionality and consciousness. In fact, an early active fetus is not awake at all. Wakefulness is acquired late during prenatal life [5]. Brief spells of waking have been noted only towards the end of pregnancy. Additionally, although wakefulness is a precondition for consciousness, the two do not necessarily coincide [6]. Malformed fetuses born without the cerebrum, cerebellum and flat bones of the skull–so-called anencephali–can be intermittently awake and exhibit several behavioral patterns of healthy newborns [7].

Ultrasound has uncovered what was once withheld from view by nature during the 9 months of pregnancy, opening up a visual dimension which usually started only after the delivery. Such premature unveiling has, amongst other things, triggered the habit of applying the phenomenon of "meaning attribution" to the physiologically concealed motions of the fetus. Meaning attribution is a fundamental mechanism in postnatal life which helps us to care for our newborns and to deal with the complex nuances of social interactions in general [8, 9]. Attributing the meaning associated with neonatal or even adult manifestations to the behavior of the fetus, and especially of the early fetus, is not a functional or an appropriate phenomenon.

At the other end of the spectrum we find those who assume the fetus (or even the neonate) to lack any sense of boundaries with their environment. The existence of a "fusional" stage has been postulated [10].

Some bodily sense probably starts in the early prenatal stages. Fetuses frequently toss and turn and by doing so continually touch the uterus.

When at rest, the fetal body usually lies against some part of the uterus, thus granting further bodily contact with this external surface. Additionally, fetuses often touch the umbilical cord and other components of their intrauterine environment, which provide a wealth of contacts with extrinsic elements and an abundance of sensory–motor feedback. Self-touching is also regular. Fetuses frequently cover and stroke their face with their hands, touch their legs, feet, genitals and more. Some bodily sense or protoself [11] could have begun to emerge by then. "Bodily sense" does not mean any complex form of awareness or even perceiving. Sensing–the receiving of information about the external environment–and perceiving–a more complex operation involving the interpretation of the sensations to give them meaning–may not coincide in the fetus [12]. An early fetus is possibly quite different from a mature one in this respect. An active early fetus may just sense what a mature one approaching birth could by then perceive.

Even simple organisms are capable of quite complex motions and of withdrawing from or approaching various kind of stimuli and be conditioned by these. Amongst others the work of Kandell et al. on the mollusk called *Aplysia* has demonstrated precisely that [13].

So-called spinal cats–animals whose nervous systems have been transected immediately above the spinal cord–can walk (if supported) on a treadmill with a close to normal stepping pattern. If their limbs come into contact with an obstacle, they exhibit a "stumble corrective reaction," allowing their limbs to bypass the obstacle [14]. Early fetuses can almost be regarded as "spinal" creatures, as they function chiefly at the level of the spinal cord and the brainstem. Their motor patterns are generated within these structures and modulated by sensory inputs.

Mere bodily sense may possibly be present from very early in pregnancy. This would make it possible for the organism to adjust to changing internal and environmental conditions. Later in gestation, bodily self could possibly evolve into the dawning of a "core self" or "perceptual and motor self" [11]. These are just hypotheses as it is not possible to take a "quantum leap" from observable behavior alone.

Facts

The main fetal behavioral manifestations characterizing the first half of pregnancy and their variations with advancing pregnancy will be described first. These data are drawn from ad hoc studies of 30 healthy fetuses observed weekly between 10 and 20 weeks' gestation with ultrasonographic sessions lasting 1 h each. All observations were recorded on videotape and subsequently analyzed offline by independent researchers.

Emergence of Behavioral Phenomena

Some form of diffuse bodily motion, so-called "just discernible movements," begins from 7.5 weeks [15]; however, clearly identifiable behavioral phenomena emerge at around 10 weeks. Between 10 and 13 weeks, three behavioral events are prevalent: *startles* (sudden spontaneous jerks of the entire fetal body lasting 1 s each); *general movements* (outbreaks of diffuse whole body activity); and *hiccups* (jerky contractions of the diaphragm lasting less than 1 s and generally following each other in regular succession).

The following other behavioral events begin to surface sporadically during the 10–13 weeks' span, but become conspicuous thereafter: *partial movements* (less spread, relatively "targeted," patterned motions involving segments of the body only); *fetal breathing movements* (noncontinuous, irregular and so-called "paradoxical" breathing movements involving simultaneous downward movements of the diaphragm, inward movements of the thorax and outward movements of the abdomen); *shallow fetal breathing* (a form of superficial and regular "breathing" characterized by low synergistic outward excursions of the thorax and the abdomen); and *swallowing* (rhythmical bursts of regular jaw opening and closing at a rate of 1 per second accompanied by displacement of the tongue and larynx) [15].

Developmental Trends

As shown in Fig.1a, startles are the prevalent behavioral phenomenon up to 13 weeks. Their rate then falls considerably, with a first drop between 13 and 14 weeks and an even more dramatic fall at 17 weeks. Subsequently startles continue on an even slope. General movements are especially conspicuous up to 13 weeks, decrease steadily, but not as dramatically until 15 weeks, then also continue on an even slope. Partial movements are infrequent between 10 to 12 weeks, then rapidly outnumber general movements from 13 weeks, continue to increase till 14 weeks and subsequently stabilize.

As shown in Fig.1b, fetal breathing movements surface at 10 weeks and continue to increase sharply and progressively thereafter. Hiccups outnumbers fetal breathing movements until 12 weeks, continues to increase till 13 weeks and subsequently continues to be observed at low frequency. Swallowing is extremely sporadic before 12 weeks. Subsequently, swallowing increases similarly to fetal breathing movements, albeit less markedly [16]. However, bouts of swallowing cannot be visualized at all times as a turning away of the fetal head can conceal their true occurrence. Thus the incidence of swallowing is always likely to be underestimated.

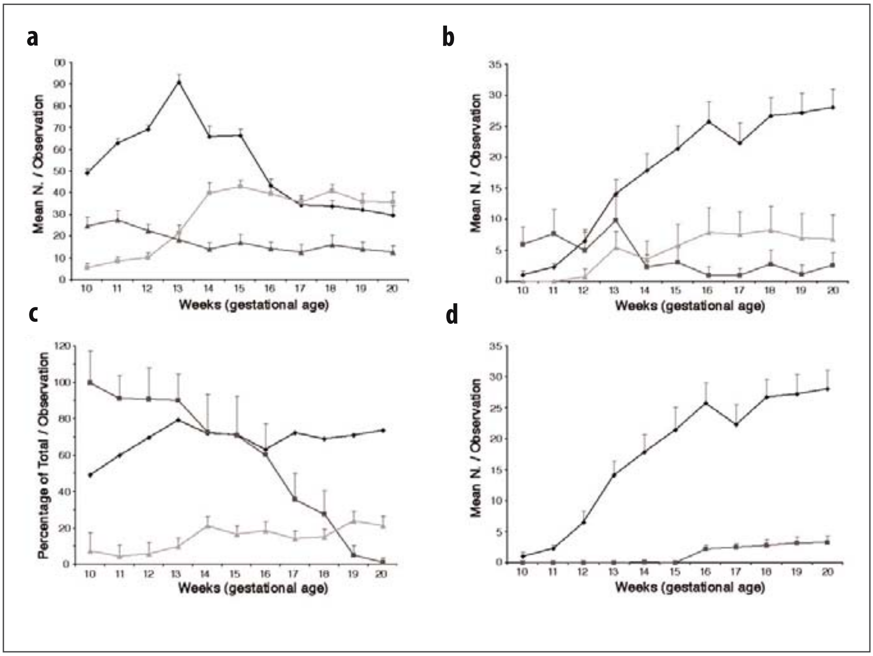


Fig. 1. a Comparison between the developmental trends of startles (◆), partial movements (■) and general movements (■) in the 10–20 weeks period of gestation. Each point represents the mean ± standard deviation (plotted as a vertical bar over each point) of the number of the above-mentioned behavioral events calculated for 30 cases over 1 h observation time per week. **b** Comparison between the developmental trends of fetal breathing movements (◆), hiccups (■) and swallowing (▲) in the 10–20 weeks period of gestation. **c** Comparison between the developmental trends of fetal breathing movements (◆, same as shown in **b**) and shallow fetal breathing movements (■) in the 10 to 20 week interval of GA. **d** Percentage of general movements (■) and partial movements (▲) activated by startles. An event was classified as activated if it followed a startle within 2 s. Startles were considered nonactivators (startle-nonactivators) whenever they fired without eliciting any event with 2 s. Startle-nonactivators (◆) are plotted as a percentage of the total number of startles per observation.

Figure 1c shows a comparison between the developmental trend of fetal breathing movements and shallow fetal breathing movements. Shallow fetal breathing begins to be noticed at 12 weeks, increases until 16 weeks and maintains a steady trend thereafter. Whilst fetal breathing movements display a steep increase, the frequency of shallow fetal breathing movements within the hour continues to be low up to mid-pregnancy.

Temporal Sequencing of Behavioral Events

Analysis of the temporal sequence of the behavioral phenomena described above reveals several interesting features. Figure 2a is a graphic representation of how observational sessions were analyzed in this way. All behavioral events were identified and their duration was counted in seconds. The exact temporal sequencing (including phases of complete rest) was then charted. A behavioral phenomenon was considered to be activated by another phenomenon when its beginning regularly followed the appearance of the latter within a 1- to 2-s interval [17].

As shown in Fig. 1d, up to 13 weeks' gestation general movements show an absolute dependence on startles for their commencement. Dependence on startles for activation continues to be substantial until 16 weeks. Subsequently, general movements acquire progressive autonomy. Dependence is not reciprocal: startles can fire without eliciting any general movements. Occasional partial movements are initiated by startles, and increasingly so after 13 weeks. However, partial movements never show absolute or even substantial dependence on startles for activation [17].

With advancing gestation, fetal breathing movements change from emerging as isolated episodes to sequential ones. The duration of the episodes increases, but so does the length of "apneic pauses" between the episodes, defined as periods of complete inactivity between two successive bursts of fetal breathing. Up to mid-pregnancy fetal breathing movements are independent and nonsynchronous with bouts of swallowing.

From this type of analysis, the existence of behavioral aggregates also readily emerges (Fig. 2a). General movements appear to have a mutually exclusive relationship with fetal breathing movements, shallow fetal breathing movements and swallowing. In other words, when fetuses "breathe" and/or swallow they do not perform any general movements. The aggregates are denominated cluster A (general movements present) and cluster B (fetal breathing movements, shallow fetal breathing movements and swallowing present).

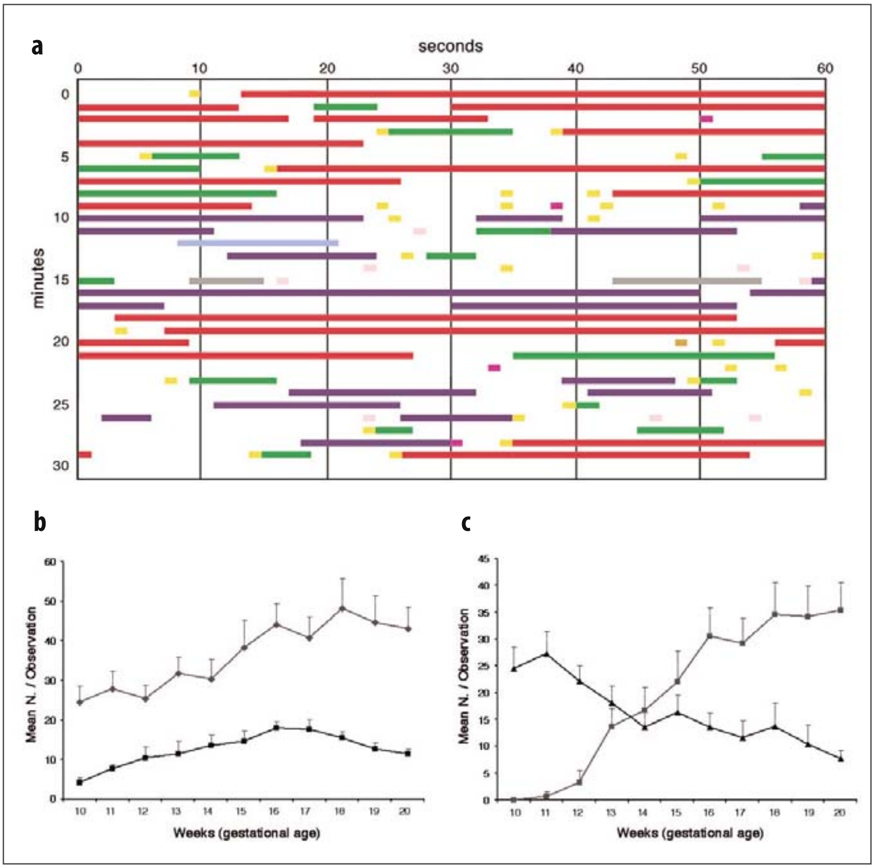


Fig. 2. **a** Example of how an observational session was graphically represented. The plot corresponding to the first 30 min of a typical observation, recorded at 15 weeks of gestation, is shown. The vertical axis represents the 30 min of the observation, with the progression of time plotted from top to bottom The horizontal axis represents the 60 s within each minute, with time increasing from left to right. Each fetal movement is represented by a bar in a specific color. The length of each bar represents the duration of each fetal movement in seconds. General movements are represented in *red*, partial movements in *green*, startles in *yellow*, fetal breathing movements in *navy blue*, shallow fetal breathing movements in *light blue*, swallowing in *gray*, hiccups in *purple*. The absence of any colored bar indicates a period of complete rest of the observed fetus. **b** Clustering of behavioral phenomena. The number of times each single behavioral episode within cluster A (general movements present) and within cluster B (fetal breathing movements, shallow fetal breathing movements and swallowing present) is directly followed by an episode belonging to the same (◆) or to the other (■) cluster. Each point represents the mean ± standard deviation (plotted as a vertical bar over each point) of the number of the above-mentioned events calculated for 30 cases over 1 h observation time per week. **c** Comparison of the total number of behavioral phenomena belonging to cluster A (▲) and to cluster B (■) during gestation. Each point represents the mean ± standard deviation (plotted as a vertical bar over each point) of the number of the above-mentioned events calculated for 30 cases over 1 h observation time per week

This hypothesis was statistically validated in the following way. A cluster was considered such when composed of at least two successive episodes of events belonging to the same aggregate. The mean number of the sum of episodes belonging to cluster A and B was calculated and plotted (Fig. 2b) against the mean number of tail events marking the transition from one cluster to the next (A to B and B to A). Behavioral events within the clusters substantially outnumbered those that marked the transition between clusters. These data strongly suggest that the temporal distribution of such behavioral phenomena is far from random. Figure 2c illustrates the trends of cluster A and cluster B with increasing gestation. The events constituting cluster A progressively decreased, whilst an opposite, increasing trend resulted for those events constituting cluster B. These opposite tendencies were in line with the developmental trends of the individual behavioral components of these clusters: general movements (cluster A) progressively decrease, whilst fetal breathing movements, shallow fetal breathing movements and swallowing increase with advancing gestation.

Rest

Rest is also a behavioral phenomenon. From 10 weeks, two distinct types of motionlessness could be observed. All behavioral phenomena were interspersed by brief (1–60 s) periods of rest. The fetus stopped whatever activity it was doing and then quickly resumed the same activity or started a different one. Parallel to these short spells of inactivity, longer phases of complete rest exceeding the duration of 2 min were also noticed.

Rest also underwent developmental changes with increasing gestation. The global quota of rest, comprising both short and longer spells, was particularly elevated (over 75% of the observation time) until 11 weeks. From 12 to 17 weeks the total quiet time diminished, and continued on a plateau ranging from 69% to 63% of the observation time. At 18 weeks a crossover point between rest and activity was reached. Subsequently rest continued to decrease slightly until it represented 48% of the observation time at 20 weeks.

Phases of absolute rest underwent an opposite trend. At 10–11 weeks these were on average 3 min. Between 12 and 17 weeks the average increased to 4 min and from 18 weeks to 11 min. With increasing gestational age fetuses were able to sustain more movement, but were also plunged into longer spells of absolute rest.

Possible Significance of Early Fetal Behavior

The views put forwards in this section are all of an hypothetical nature, to be tested with sound methods properly pertaining to physiology and neurology. Fetal behavior can only give a macroscopic indication of what fetuses do or do not perform. Nevertheless, a thorough knowledge of behavioral functioning is important for and understanding of physiology, and physiology in turn is invariably a prerequisite for comprehending pathology.

Startles

Up to 13 weeks, spontaneous startles are the predominant behavior of the human fetus. Subsequently they decrease dramatically, but can still be found in the neonate and occasionally in the adult in the initial phases of sleep [18]. Startles have generally been overlooked as a mere epiphenomenon of the immature nervous system. Furthermore, they are often confounded with the startle responses that can be found even in lower organisms and help these move away from potentially noxious stimuli. The classic studies of Kandell et al. demonstrating startled and withdrawal responses in *Aplysia* are an example of the latter [12]. In the human fetus, startles are endogenously generated [19]. In addition they serve a relevant temporary function.

Startles appear to be temporary ontogenetic adaptations setting general movements in motion during early pregnancy. As noted previously, general movements are totally dependent on startles for activation from 10 to 13 weeks. A consideration of the evolution and changes of the fetal body as well as the manner in which general movements are performed at various stages may help to elucidate the reasons for which startles are required.

Fetal bodily proportions change rapidly. During the 10–13 weeks span, the head initially constitutes almost half of the fetus, then its growth slows down, and by 13 weeks it makes up roughly one-third of the fetal body. The muscles of the thorax are more developed than those of the limbs and the limbs are short. By the end of the 13th week, the upper limbs have almost reached their final length, but the lower ones are still short [20]. At this stage, general movements are achieved through a rotation and displacement of the thorax, followed by a partial rotation of the head and finally by a passive displacement of the limbs. Startles, by causing a pronounced upward lift of the fetal body with a subsequent uneven drop, promote displacement of the thorax, thus allowing the setting in motion of general movements. Between 14 and 16 weeks, the dependence of general movements on startles decreases, but is still significant. Around 14 weeks, pelvic and shoulder joints acquire independence of movement from trunk muscles, trunk and neck muscles are no longer closely tied, and the skeleton, so far only cartilaginous, begins to harden and solidify [20]. The limbs are, however, still too weak and short to upturn independently the greatly increased fetal body. Although startles progressively lose their power to cause a pronounced lift of the fetal body, dependence on this "propulsive" mode is still significant. By 17 weeks, the fetal body has acquired sufficient strength, behavioral independence and adequate proportions to perform general movements autonomously. Dependence on startles becomes absent by 20 weeks. By then, startles no longer act as "propulsors," but, as in the neonate, merely cause a pronounced extension or flexion of the limbs, often associated with head extension. However, startles, albeit greatly decreased in number, do continue to occur and may have other unknown functions. For instance, they can appear within phases of general movements. It is interesting to note that startles occasionally also act as "perturbers." From 10 weeks' gestational age, phases in which fetuses are reactive to startles alternate with cycles of nonreactivity, characterized by relative quiescence and absence of general movements. The epochs of nonreactivity to the abrupt and massive stimulation presented by startles could be regarded as strong "attractors" towards which fetuses spontaneously shift following a cycle in which general movements are present. Only towards the beginning or end of such cycles of rest characterized by refractoriness at mid-cycle do startles become increasingly effective as "perturbers" precipitating the shift to another state [21, 22].

General Movements

General movements are especially pronounced up to 13 weeks, when they are outnumbered by partial movements. However, they continue to be present in the late fetus and in the neonate during active sleep. This sleep-associated behavior then diminishes progressively in frequency and intensity over the first 6 months of postnatal life [23]. This continuity does not necessarily mean that general movements may fulfil the same functions at early and later stages. General movements could have both adaptive and preparatory functions. Within the 10–12 weeks span, they are the almost sole form of motion displayed by the fetus. Two main hypotheses on the function of general movements during early prenatal life have been put forward: (1) that frequent changes of position may prevent adhesion and local stasis of blood in the early fetus, whose skin is very thin, or (2) that such motions may be essential for muscular and skeletal development. Both hypotheses could be true. General movements are not stereotyped; while performing them, fetuses constantly adapt to external and internal environmental conditions. General movements could also be regarded as massive storms of sensory-motor stimulation to the nascent brain, thus fostering its development.

Partial Movements

Partial movements appear to be different from general movements, in the sense of being segmental and of seeming more "goal directed." For instance, fetuses display a wealth of hand-to-face contacts; touch the uterine wall with their feet; begin to move their legs in alternation, showing stepping movements by 15–16 weeks; rotate their heads or stretch their spines. Partial movements, too, provide the fetus with an abundance of sensory-motor stimulation. As such, they could also foster the development of the embryonic brain. However, their relatively "goal-directed" nature may also indicate a more long-term anticipatory function. Unlike general movements, one could see in partial movements the first tentative signs of much later voluntary motions. Volition and true goal-directedness are absent in the early fetus; however, it could be preparing for these through partial movements.

Hiccups

Hiccups is still a largely unexplained phenomenon. In the neonate hiccups tend to appear mainly after meals, when the full stomach may act as an irritant on the diaphragm wall and on the phrenic nerve in particular. However, hiccups can also surface when the abdomen is not distended. In the early fetus, hiccups is a marked phenomenon. Until the fetus begins to swallow consistently (not before 14 weeks), its stomach is hardly distended. Some authors have put forward the hypothesis that hiccups may be a precursor of fetal breathing [24, 25]. It is worth noting that hiccups starts decreasing just when fetal breathing increases, reaching a crossover point with it at 12 weeks. One could postulate that hiccups, by causing repeated contractions of the diaphragm, may smooth the progress of the subsequent diaphragmatic motions necessary to fetal breathing. However, although the coincidence is of interest, an answer as to the function or functions of hiccups during prenatal life requires animal experimentation.

Fetal Breathing Movements

Although aerial respiration only begins at birth, it is currently widely recognized that breathing has a long preparatory history during the 9 months preceding parturition. Several authors purposely omit the idiomatic expression "first breath" and propose talking about a shift from periodic respiration to the establishment of continuous respiration when describing the changes that occur at birth. In addition to having preparatory functions essential for the shift to continuous aerial respiration at birth, animal studies have shown that fetal breathing movements also play a fundamental role in the anatomical and functional development of the lungs [26, 27].

Up to mid-pregnancy, breathing can be considered an "either/or" event. Fetuses either "breathe" or move, swallow and hiccup. During the first half of pregnancy breathing and swallowing are not conjugated. This physiological phenomenon acquires pathological significance in the premature infant that is having to cope with the different requirements of extrauterine life. The fine coupling of these activities, necessary for feeding after birth, is not yet established by mid-gestation.

Growth is associated with an increasing capacity of the fetus to sustain prolonged episodes of breathing. Moreover, bouts of breathing become less fragmented. Parallel to this, the intervals between bouts also increase in duration: long apneic intervals become a consistent feature of fetal breathing around mid-gestation. It is established that breathing movements are the first movements to disappear in compromised and growth-restricted fetuses, where energy and oxygen consumption have to be spared for more delicate areas [28]. A concomitant increase in blood flow to the brain, heart and adrenals is usually observed in such cases and is taken as a clinical indicator of fetal compromise [28]. The increased spacing between episodes of breathing could also acquire pathological significance in the premature infant and occasionally also in the neonate. In only too many ways a premature infant is a fetus born at an untimely age. Under particular circumstances the premature infant, but also the neonate, could revert to a fetal form of "breathing" with long apneic phases separating breaths. Apneic phases and the suppression of breathing during hypoxia are functional during the fetal stage and represent an efficient sparing method. This same sparing could have dire consequences if applied at a later, inappropriate stage. Hypoxia of whatever origin could expose the premature infant and some vulnerable neonates to the danger of reverting to a fetal form of breathing with long apneic spells which in turn would increase hypoxia and cause the cessation of breathing. Hypoxia and obstruction of the upper airways (with consequent hypoxia) have been postulated to be at the basis of some cases of sudden infant death syndrome.

Swallowing

During early prenatal life, swallowing is both preparatory and functional. It is preparatory in the sense that the neonate is no longer passively dependent as the fetus was on maternal nutrients reaching it through the placenta and the umbilical cord, but must be capable of actively ingesting food. It is functional in the sense that fetal swallowing is one of the main regulators of amniotic fluid volume, together with urine flow and absorption of fluids into the fetal blood across the fetal surface of the placenta (so-called intramembranous absorption) [28].

Shallow Fetal Breathing Movements

Fetal breathing movements start at 10 weeks while shallow fetal breathing movements emerge at 16 weeks. Six weeks is a considerable time lag in the context of the extremely accelerated fetal mode of development. In addition, shallow fetal breathing movements continue up to mid-pregnancy to be a fairly sporadic occurrence compared to the ever-increasing conspicuousness of fetal breathing movements. Therefore, it is possible that fetal breathing movements represent an earlier and more “vitally” important form of breathing during development. Due to their shallow and sporadic nature, shallow fetal breathing movements are unlikely to play a critical role in lung development.

Rest

The very high quota of rest observed during the 10–11 weeks period could be accounted for by the scarcity of behavioral phenomena present at this stage. Startles, general movements and hiccups are the almost exclusive forms of motility displayed by the fetus within this time span.

Apart from shallow fetal breathing, all other behavioral phenomena emerge from 12 weeks. However, amongst others, energetic metabolic factors could possibly be relevant, especially in this and the subsequent 12–17 weeks period. All fetal motions involve an expenditure of energy. The young fetus is rapidly growing, particularly in length, and growth necessitates nonexcessive metabolic consumption. Interestingly, fetuses begin to "put on weight" particularly after 16–17 weeks [20]. By then, their considerably increased bodily mass could possibly sustain more "strenuous exercise" without a parallel consumption of weight.

Periods of absolute rest fuse into longer phases just when general movements decrease, and consequently behavioral events pertaining to cluster B outnumber those contained within cluster A. Breathing, swallowing and partial movements clearly involve minimal consumption of energy as compared to the turbulent motions of general movements. Fetuses on the whole move more, but their movements are "light," thus favoring weight gain.

Onset of Sleep

The full-term infant displays clearly ascertainable, well-developed phases of sleep which have been variously named. In this chapter reference will be made to active and quiet sleep [29, 30]. In the mature fetus, active sleep is accompanied by the surfacing of somatic activity. Prior to birth, active sleep is largely predominant.

Well-defined phases of sleep have been identified ultrasonographically in the human fetus from 34 to 36 weeks' gestation [29, 30]. Parameters used to distinguish these phases cannot be applied to earlier stages of pregnancy. Eye movements are absent or extremely sporadic and fetal heart rate patterns do not vary with motion. Prior to 34–36 weeks, authors talk about rest–activity cycles. "Activity cycles" are taken to comprise any kind of behavioral activity. "Rest cycles" are regarded as totally devoid of these [31]. However, sleep is considered a primary "instinct" [32–34], and its well-organized phases cannot be postulated to begin to operate without a previous preparatory history. The data discussed earlier in this chapter indicate that so-called rest–activity cycles are not disorganized. Various behavioral phenomena display organization, albeit of a changing nature, from very early prenatal life. Mutually incompatible behavioral clusters are formed, and cycles of rest seem to have an inbuilt regulator.

Behavioral manifestations and their variations can give us important indications about the neural substrate presiding over them. Neurophysiological data too can elucidate the link between various behavioral displays. Studies on the ontogenesis of the brain have mainly focused on the formation of the cortex and the diencephalon. However, the vital structures of the brainstem, specifically the bulb and the pons, have been found to organize first [35, 36].

From 10 weeks' gestation, fetuses show signs of brainstem functioning. Startles are known to originate within the brainstem and can be considered the expression of a massive depolarization of reticular neurons, which in turn control spinal motoneurons [37, 38]. This could explain the initial dependence of general movements on startles for activation.

Between 10 and 12 weeks, fetuses show other signs of brainstem functioning. Fetal breathing movements and other nonspinal activities such as swallowing begin to emerge. Breathing in particular is widely recognized to be inextricably linked with the same brainstem reticular structures as control sleep [39, 40].

Between 13 and 15 weeks, rest–activity cycles show analogies with active sleep of the mature fetus [29, 30]. During active sleep, general movements surface when fetal breathing and swallowing are not present, as in the clustering observed in the early fetus. With advancing gestation, this primordial type of active sleep takes on a progressively more complex somatosensory integration. However, given the analogies between early clusters and later ones observed within active sleep, initial sleep organization could be postulated to have started by 13 weeks.

On the other hand, quiet sleep is barely developed during late pregnancy and its quota always remains low [41]. Thalamic structures which regulate the synchronizing processes of quiet sleep mature late. In particular, the reticular nucleus of thalamus, on which synchronization depends, is not yet fully developed at birth. This accounts for the low percentage of quiet/synchronous sleep even in the term infant.

During quiet sleep the mature fetus displays no movement. Breathing is regular, superficial and emerges in isolated bouts. By analogy with what has been observed in the mature fetus, the surfacing of shallow fetal breathing around 16 weeks could be interpreted as the first sign heralding quiet sleep. Shallow fetal breathing, the only behavioral manifestation possibly indicating the functioning of quiet sleep, is very sparse during the first half of pregnancy [42, 43]. One could thus postulate that up to mid-gestation quiet sleep may fulfil a less crucial role than active sleep.

Like the reticular nucleus of thalamus, which is exclusively inhibitory in nature, other inhibitory subcortical structures are not mature during the first half of pregnancy. This explains the intense bodily activity which accompanies active sleep. The clustering of cycles of rest into phases of longer duration could represent the indication of an initial functioning of inhibitory structures.

Besides descending neuronal projections, the brainstem also controls ascending ones. These in turn keep the forebrain and cortical structures under synaptogenic and maturational control [44, 45] (Fig. 3).

The intense activity of the early fetus could be viewed as an important sensory–motor stimulation to the brain. Fetal motions are not stereotyped, and fetuses constantly adapt to the changing requirements of the internal and external environment. Such motions could represent a vital "awakening" to the environment, and active sleep could be regarded as an essential component of prenatal "learning" and development.

The first episodes of wakefulness do not appear in the fetus earlier than 36 weeks. Wakefulness is a late addition, which presumably arises in order to meet the different requirements of the physical and social postnatal environment. Nonetheless, fetuses do not emerge into this world as

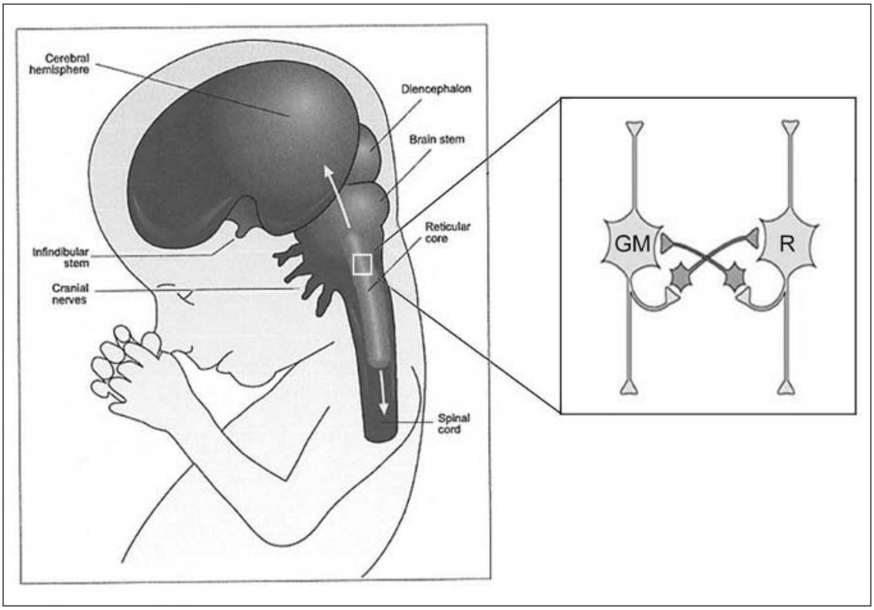


Fig. 3. The fetal brain at 15 weeks' gestation. Ascending and descending projections originating in the brainstem are represented with *arrows*. The insert shows a reciprocal inhibitory circuit between neurons controlling general movements (GM) and those involved in "respiration" (R) which could explain the clustering observed

tabulae rasae unable to cope with it. Unlike in postnatal life, fetuses were able to master their environment during active sleep. Around 3–4 months the neonate is awake for long enough periods to consistently interact with its environment and starts learning from it. Sufficient waking time is also necessary for quiet/synchronous sleep to develop within phases of rest and to become the physiological function that allows experiences to be processed.

All the parameters described can be considered as an indirect proof that active sleep is beginning to undergo organization and represents the predominant and paramount physiological phase during early pregnancy. It also represents the embryonic inception of sleep.

Twin Fetuses and What They Can Tell Us About Early Prenatal Life

In addition to all the mythology encumbering singleton fetuses, twin fetuses are subject to specific myths. On the other hand, the behavior of the twin fetus can illuminate some interesting features of prenatal life. "Twin myths" will be dispelled first, before recounting "twin facts."

Myths

Open to Mutual Communication

The "mystic" union so often attributed to twins in postnatal life is frequently believed to date back to prenatal life. Intrapair stimulation, which is indeed limited to twins, is taken to mean various types of communication. Twin fetuses are considered as particularly interactive companions, relating to each other in all sorts of complex and sophisticated ways. Even kissing and fighting have been "scientifically" described to occur in utero [46]. Apart from any other consideration, it is difficult to imagine how elaborate behavioral and emotional patterns such as kisses could be carried out across the membranes which in 99% of all twin pregnancies separate the two different amniotic sacs in which twin fetuses are contained [47]. These activities, like all truly and complex emotional patterns, clearly only belong to later stages of postnatal life.

Although fetuses are rapidly and progressively preparing to enter into a social world, this prelude is geared towards the different stimuli and the more "mature" human beings that they will encounter in the extrauterine environment. Fetuses are simply not equipped for complex social interactions with other fetuses. Human communication is foreign and nonfunctional to the usually solitary 9 months of gestation, and especially so to the first half of gestation. Intrapair stimulation, which is a feature of twin pregnancies, seems to be considered relevant solely to support the belief that twins are highly communicative partners. As a consequence, all fetuses are regarded as potentially open to communication.

The premature infant is in many ways simply a fetus removed from its natural environment. Though certainly not numb to various forms of stimulation, including human contact, it clearly does not enjoy an intense and lively "social" life. Most of the time, premature infants lie inert and hypotonic, crushed by gravity, dormant and barely responsive to all the commotion surrounding them. Even more so, previable twin fetuses incapable of living autonomously outside the uterus can hardly be assumed to be fit for communication with each other at all [3].

Maternal Emotions and Their Impact on the Twin Fetus

The impact of maternal emotions on the fetus is a "hot" topic amongst psychologists and psychoanalysts, who concentrate especially on "negative" emotions such as anxiety and stress. The research on the impact of maternal emotions on the fetus is fraught with enormous and, for the moment, probably insoluble methodological problems. To mention but a few: our knowledge of the physical substrata of emotions is still primitive, our understanding of placental crossing is also limited, and our comprehension of the mechanisms regulating fetal behavior is virtually nil. Yet, twins have been thought of as a possible "ideal model" to evaluate the impact of maternal emotions on the fetus [46]. According to this model, since both twins are equally and systematically affected by the same maternal emotion or by the same byproduct of an emotion, this proves per se that behavioral alterations are caused by the emotion. The underlying assumption is that twins live in and share exactly the same intrauterine environment. Even assuming that maternal "stressors," of whatever origin and kind, can reach the fetus, twins fetuses would only receive the same quality, but never the same quantity, of such substances.

Pregnancy-induced hypertension is a frequent complication in twin pregnancies [48]. However, this condition is not proven to have any causal relation to maternal anxiety. The opposite may well be true. Furthermore, pregnancy-induced hypertension does not impinge on both twins in the same way. Twin fetuses have different umbilical cords, an unequal distribution of placental mass; they float in different amounts of amniotic fluid and have differing blood flows. Their environment, including potential maternal "stressors," can hardly be regarded as "equal."

Uterine contractility is particularly elevated in twin gestations [48]. Uterine contractions have been also postulated to be the result of maternal anxiety and stress. According to this hypothesis, uterine contractions, by applying pressure to the amniotic sac, would produce increased tension in the amniotic fluid. This pressure would be perceived as disruptive by the fetus, which would then start to move more. However, even assuming that this "increased pressure" hypothesis were true, the vast majority of twins are contained in separate amniotic sacs; also, the amniotic fluid is unequally distributed between the two. Additionally, pressure on sacs which are different from each other may well act differently on each twin. Basically, lively fetal motions are confounded once more with anxiety-driven hyperactive states after birth [3].

Twin gestations, due to the various complications and many discomforts associated with them, are anxiety-provoking pregnancies. Already overanxious prospective mothers of twins should be spared the additional burden of having to try and suppress their emotions, which may often be uncontrollable anyway and whose harmfulness is in doubt.

Bereavement in the Twin Fetus

Anecdotal evidence from children and adults reporting a sense of loss which they can only explain in terms of a wish for a reunion with a dead co-twin fetus is accumulating. As a consequence, therapy groups for "prenatal mourners" are proliferating. However, it is impossible to ascertain how much of this longing and loss is derived from real reminiscences of sensations felt in utero and how much is the result of later constructions belonging to postnatal life alone. The event of fetal loss, and especially of a late fetal loss, always has a deep impact on parents [49]. It is not hard to imagine how this could reverberate in only too many ways on the surviving twin. The "missed" twin fetus can become an easy container into which one can project all that has been missed in life and the inevitable dissatisfactions connected with it.

Much has been made about the so-called "vanishing twin" phenomenon [50], the not infrequent finding of another gestational sac during a first trimester scan. It is hard to imagine that an early fetus could "miss" something that it has never felt. As will be explained, intrapair stimulation usually only starts when such a "vanishing twin" would have already ceased to exist.

Only when intrapair stimulation has become a consistent component of the intrauterine environment could one postulate that a surviving twin might feel some kind of sense of "loss." Again, one should not forget that the majority of twins live in separate environments. What a twin fetus might "miss" would clearly not be a whole, distinct person, but just the stimulation, however strong, arising from the other twin fetus. Although intrapair stimulation is an important factor in the intrauterine life of twins, the evidence is too scanty and unconvincing, for the time being at least, to allow us to assess whether it will remain forever embedded in the unconscious of a surviving twin [51].

Facts

The information expounded under the next two subheadings derives from two different studies focusing on the first half of pregnancy. One compared the behavior of dizygotic and monozygotic twins. The other centered on the beginning and evolution of evoked behavior in twin fetuses.

Behavioral Individuality Begins to Be Shaped In Utero

Monozygotic (or so-called identical) twins share all their genes while genetically speaking dizygotic twins are like ordinary siblings. Dizygotic twins always have separate placentas and amniotic sacs. Monozygotic twins can have different types of placentas according to when the division initiating the twinning phenomenon takes place. The majority (70%) of monozygotic twins share the same placenta and inhabit two different amniotic sacs and are thus called monochorionic–diamniotic. Diagnostic refinements allow the determination of placental type early in pregnancy with a high degree of accuracy. It is thus possible to compare the behavior of two populations of twins: monochorionic–monozygotic and opposite-sex dizygotic twins in utero [52]. The levels of spontaneous and reactive activity can be calculated and compared in the two groups.

Although monozygotic twins initially show greater intrapair similarities in levels of spontaneous and reactive activity, they are not behaviorally identical from the start. Furthermore, their behavioral differences show a tendency to increase with advancing gestation. By halfway through the pregnancy they reach the same degree of behavioral individuality as shown by dizygotic twins. However, when one analyzes individual movements, the larger behavioral differences initially found in dizygotic twins in macroscopic features such as activity and reactivity levels are no longer noted. Examined in detail, the two groups tend to merge together and become behaviorally indistinguishable. Some fetuses move their arms more, some their legs, some their heads, some cover or rub their faces almost continuously, others frequently touch their thighs. Each fetus, regardless of its zygosity, has its own fairly distinctive way of acting and reacting.

As in life after birth, monozygotic twins may be considered behaviorally alike at a macroscopic level of analysis, but can never be considered identical upon detailed examination during pregnancy. Individual propensities can be discerned in the variety of repeated fetal movement patterns, of preferred positions and activities. Although we can speak only of tendencies and inclinations–and clearly not of well-determined characteristics–nevertheless, individuality and uniqueness are shaped during gestation and are evident by the time the twins are born. All twins, including monozygotic twins, emerge from the troubled time of pregnancy as unique individuals with fairly distinct inclinations and behavioral manifestations.

Twin Fetuses as an "Experiment in Nature"

Twins can be used as a perfect "experiment in nature" to demonstrate the functioning in utero of two sensory modalities which would otherwise be impossible to verify in the fetus within its solitary natural environment. Responses to touch and pressure originating from the other twin are elicited by intrapair stimulation. This proves per se that so-called tactile and proprioceptive sensitivity (that is, touch and muscular/body sense) are operative in utero [53]. These sensory modalities had only been investigated by Minkowski, Hooker and Humphrey [54-56] in the exteriorized and preagonal singleton fetus. Before the 11th week, intrapair stimulation is a fairly exceptional event. From 11–13 weeks, intrapair stimulation becomes a progressively more frequent event in monochorionic twin pregnancies. Presumably the nearness brought about by this placental sharing and the thinness of the membranes dividing the two amniotic sacs favors earlier contact.

From 13 weeks onwards, intrapair stimulation begins to be noted in dichorionic pregnancies as well. The separate placentas of dichorionic twins can be implanted in different and quite distant sites. The membranes dividing the amniotic sacs also are considerably thicker, making earlier contact of a sufficient strength fairly improbable.

With advancing gestational age, twins grow rapidly in size, with a corresponding increase in the vigor of their movements. This results in a greater likelihood of contact between them. By 15 weeks, intrapair stimulation is a constant and increasing feature of all twin gestations. Reactive movements come to correspond to almost one-third of all motions. Therefore, intrapair stimulation appears to be an important, consistent and unique determinant in the intrauterine behavior of all twins from late in the first trimester. It is also an active and important part of the intrauterine environment of the twin fetus.

The same variety of movements observed in spontaneous motions is also found in the reactive responses due to intrapair stimulation. However, evoked movements change with advancing gestation. General movements are observed as the only evoked responses up to 11 weeks. From then on a growing complexity and variability of reactions is noted. Partial movements become the most prominent response.

Twin fetuses may constitute an important "experiment in nature" in another respect. From the outset of intrapair stimulation, twin fetuses are not always responsive to it. Periods can be noted when they appear to be impervious to any, even intense, stimulation from the co-twin. During these periods they are clearly stimulated, but their body is passively displaced within the amniotic fluid and they show no response. As with startles, intrapair stimulation can act as a "perturber." Cycles of rest are characterized by refractoriness at mid-cycle. Only in periods of "instability," towards the beginning or the end of cycles of rest, does intrapair stimulation become increasingly effective as a "perturber" precipitating the shift to another state. In other words, a careful analysis of the perturbation represented by intrapair stimulation in twins could also increase our knowledge of the onset of sleep.

Conclusions

Fetal behavioral research is very time-consuming. Obstetricians are generally reluctant to engage in it, possibly due to lack of interest, or more simply because they are constantly busy dealing with urgent clinical matters. Patient, prolonged, detailed and repeated observations are more germane to the frame of mind of infant researchers and psychoanalysts alike. Having trained as a neurologist, a psychoanalyst and an infant researcher has certainly helped me in my studies of fetal behavior.

Research on fetal behavior is still patchy. A comparison with what is known about the neonate makes this all the more obvious. In essence, the so-called "developmental milestones" of fetal behavior have yet to be established. These pointers could then form the basis for a complete neurological examination in utero. Only when physiological functioning has been properly characterized will it be possible to accurately differentiate deviations from the norm.

However, in order achieve such a significant goal several steps would need to be taken. First of all, one would have to observe large samples. Secondly, a prolonged behavioral, psychological and neurological postnatal follow-up of all the subjects under observation would have to be carried out. Thirdly, the observation of recognizable pathological cases such as anencephali, fetuses suffering from various malformations of the brain or from known genetic defects affecting the central nervous system would add greatly to our knowledge of variations from the norm. Undoubtedly this endeavor could only be achieved by a multicenter joint effort. Hopefully, this work might stimulate an interest in pursuing the investigation of the origins of our behavior in other researchers who, like the neuroscientist, would be willing patiently to tease out pathology from physiology and thus piece together the puzzle of how we were once able to function in the intrauterine world.

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References

1. Piontelli A (1992) From fetus to child. Routledge, London
2. Gesell A (1945) The embryology of behavior. MacKeith Press, London
3. Piontelli A (2002) Twins: from fetus to child. Routledge, London

4. Oppenheim RW (1984) Ontogenetic adaptations in neural development. In: Prechtl HFR (ed) Continuity of neural functions from prenatal to postnatal life. MacKeith Press, London, pp 16–30
5. Parkes MJ (1991) Sleep and wakefulness do they occur in utero? In: Hanson MA (ed) Fetal and neonatal brainstem. Cambridge University Press, Cambridge UK, pp 230–256
6. LeDoux J (1966) The emotional brain. Simon and Schuster, New York
7. Peiper A (1963) Cerebral functions in infancy and childhood. Consultants Bureau, New York
8. Trevarthen CB (1979) Communication and cooperativity in early infancy:a description of primary intersubjectivity. In: Bullowa M (ed) Before speech. Cambridge University Press, Cambridge UK, pp 64–78
9. Stern DN (1985) The interpersonal world of the human infant. Basic Books, New York
10. Malher MS, Pine F, Bergman A (1975) The psychological birth of the human infant. Hutchinson, London
11. Damasio AR (1999) The feeling of what happens: body and emotion in the making of consciousness. Harcourt Brace, New York
12. Hepper PG (1992) Fetal psychology, an embryonic science. In: Nijhuis JG (ed) Fetal behavior, developmental and perinatal aspects. Oxford University Press, Oxford, pp 129–156
13. Kandell ER et al (1983) Classical conditioning and sensitization share aspects of the same molecular cascade in the Aplysia. Quant Biol 48:821–830
14. Grillner S, Shik ML (1973) On the descending control of the lumbosacral spinal cord. Acta Phsysiol Scand 87:320–333
15. Vries JIP de, Visser GHA, Prechtl HFR (1982) The emergence of fetal behavior: I qualitative aspects. Early Hum Dev 7:301–322
16. Vries JIP de, Visser GHA, Prechtl HFR (1984) The emergence of fetal behavior: II quantitative aspects. Early Hum Dev 16:85–103
17. Piontelli A et al (2001) Startles: activators during early prenatal life. In: Einspieler C, Wolff P, Prechtl HFR (eds) Acts of the International Symposium on Time and Timing in Developmental Neurology. University of Graz, Graz, Austria, pp 11–12
18. Wolff PH (1966) The causes, controls and organization of behaviour in the neonate. Psychol Issues 5:1–105
19. Prechtl HFR (1984) Continuity and change in early neural development. In: Prechtl HFR (ed) Continuity of neural functions from prenatal to postnatal life. MacKeith Press, London, pp 1-15
20. Moore KL (1998) The developing human. Clinically oriented embryology. Saunders, Philadelphia
21. Kelso JAS, Scholz JP, Schoner G. (1986) Non-equilibrium phase transitions in coordinated biological motion: critical fluctuations. Phys Lett 118:279–284
22. Thelen E, Smith LB (1994) A dynamic system approach to the development of cognition and action. MIT Press, Cambridge MA
23. Parmelee AH, Stern E (1972) Development of states in infants. In: Clemente CD, Purpura DP, Mayer FR (eds) Sleep and the maturing nervous system. Academic Press, New York, pp 85–98
24. Hofer MA (1988) On the nature and function of prenatal behavior. In: Smootherman WP, Robinson S (eds) Behavior of the fetus. Telford Press, Caldwell, NJ, pp 45–59
25. Pillai M, James D (1990) Hiccups and breathing in human fetuses. Arch Dis Child 65:1072–1075

26. Rigatto A, Moore M, Cates D (1986) Fetal breathing and behavior measured through a double-wall Plexiglas window in the sheep. J Appl Physiol 61:160–164
27. Harding R (1994) Fetal breathing: relation to postnatal breathing and lung development. In: Hanson MA, Spencer JAD, Rodeck CH (eds) Fetus and neonate: physiology and clinical applications, vol 2: breathing. Cambridge University Press, Cambridge UK, pp 63–84
28. Manning FA (1995) Fetal medicine: principles and practice. Appleton and Lange, Norwalk NJ
29. Nijhuis JG, Prechtl HFR, Martin CB (1982) Are there behavioral states in the human fetus? Early Hum Dev 6:177–195
30. Arduini D et al (1986) The development of fetal behavioural states: a longitudinal study. Prenat Diagn 6:117–124
31. Robertson SS et al (1982) Human fetal movement:spontaneous oscillations near one cycle per minute. Science 218:1327–1330
32. Tinbergen N (1951) The study of instinct. Oxford University Press, Oxford
33. Moruzzi G (1969) Sleep and instinctive behaviour. Arch Ital Biol 107:175–216
34. Mancia M (1975) Sleep and instinct: an interdisciplinary approach. In: Levin P, Koella WP (eds) Sleep. Karger, Basel, pp 5–13
35. Purpura DP (1974) Introductory remarks on sleep ontogenesis. In: Petre-Quadrens O, Schlag JD (eds) Basic sleep mechanisms. Academic Press, New York, pp 150–174
36. Spreafico R (1994) Some developmental aspects of telecephalic structures involved in motor control. In: Fedrizzi E, Avanzini G, Crenna P (eds) Motor development in children. John Libbey, London pp 5–66
37. Alajouanine TH, Gastaut H (1955) La syncinésie-sursaut et l'épilepsie-sursaut a déclanchement sensorial ou sensitive inopiné. Rev Neurol 93:29–41
38. Llinàs RR, Terzuolo CA (1964) Mechanisms of supraspinal actions upon spinal cord activities. J Neurophysiol 27:579–591
39. Jouvet M (1962) Recherches sur les structures nerveuses et les mécanismes responsables des différentes phases du sommeil physiologique. Arch Ital Biol 100:125–206
40. Lydic R, Baghdoyan AH (2002) Neurochemical evidence for cholinergic modulation of sleep and breathing. In:C arley D, Radulovacki M (eds) Sleep related breathing disorders. Marcel Dekker, New York, pp 57–91
41. Dreyfus-Brisac C (1968) Sleep ontogenesis in early human prematurity from 24 to 27 weeks of conceptional age. Dev Psychobiol 1:162–169
42. Mancia M (1980) Neurofisiologia e vita mentale. Zanichelli, Bologna
43. Peterson BW (1980) Participation of pontomedullary reticular neurons in specific motor activity. In: Hobson JH, Brazier MBA (eds) The reticular formation revisited. Raven Press, New York, pp 171–192
44. Rakic P (1988) Specification of cerebral cortical areas. Science 241:170–176
45. Marin-Padilla M (1995) Prenatal development of fibrous (white matter), protoplasmatic (grey matter), and layer I astrocytes in the human cerebral cortex: a Golgi study. J Comp Neurol 357:554–572
46. Arabin B, Gembruch U, van Eyck J (1995) Intrauterine behavior. In: Keith LG, et al (eds) Multiple pregnancy: epidemiology, gestation and perinatal outcome. Parthenon, New York pp 331–349
47. Baldwin VJ (1994) Pathology of multiple pregnancy. Springer, New York
48. Kochenour NK (1992) Obstetric management in multiple gestation. In: Fanaroff AA, Martin RJ (eds) Neonatal-perinatal medicine. Mosby Year Book, St Louis MO, pp 225–229
49. Piontelli A (2000) Is there something wrong? The impact of technology in pre-

gnancy. In: Raphael-Leff J (ed) Perinatal loss and breakdown. IPA Publications, London, pp 39–52
50. Landy H et al (1986) The vanishing twin. Ultrasonographic assessment of fetal disappearance in the first trimester. Am J Obstet Gynecol 155:14–19
51. Piontelli A (1999) Twins in utero. In: Sandbank A. (ed) Twin and triplet psychology. Routledge, London, pp 7–18
52. Piontelli A et al (1999) Differences and similarities in the intrauterine behaviour of monozygotic and dizygotic twins. Twin Res 2:264–273
53. Piontelli A et al (1997) Patterns of evoked behaviour in twin pregnancies during the first 22 weeks of gestation. Early Hum Dev 50:39–46
54. Minkowski M (1928) Neurobiologische Studien am menschlichen Fetus. In: Handbuch der biologischen Arbeitsmethoden. ABT, Teil 5B, pp 511–618
55. Hooker D (1952) The prenatal origin of behaviour. University of Kansas Press, Lawrence KS
56. Humphrey T (1970) Function of the nervous system during prenatal life. In: Stave U (ed) Perinatal physiology. Plenum Press, New York, pp 651–683

Chapter 16

In Search of the Early Mental Organization of the Infant: Contributions from the Neurophysiology of Nursing

Johannes Lehtonen

The way the psyche unfolds itself from the biological organism at the beginning of life is bound to be a multifaceted and multidimensional phenomenon. Confirmed knowledge in this area of early human development is still sparse. Several different conceptual and empirical approaches have been applied to study the development of the early mind, comprising the span from the fetal stage to the successive postnatal developmental steps. Work such as that by Piontelli [1] has cast preliminary light on the impact of sensory experiences in utero. In the contributions by René Spitz [2,3], Donald Winnicott [4], Margaret Mahler and her study group [5], Stern [6], Tyson and Tyson [7] and Fonagy et al. [8], theoretical and empirical approaches have been combined. Retrospective derivation from adult or child clinical psychoanalytical material has been the method used by several psychoanalytical investigators, the most influential authors among them being Melanie Klein [9] and Wilfred Bion [10].

Although the metapsychological approach has been criticized during the last 20 or 30 years, the framework it provides for binding together the diverse aspects of psychoanalytical work and theory still has an important function. Freud's contribution to early infant mental development was limited and was a part of his general metapsychology. In *The Ego and the Id* [11] he stated that the basis of all ego development is the body ego and characterized its origin as psychic projection of the body surface. Due to the condensed nature of this statement, however, it has been problematic to find its meaning and the relevant clinical and developmental phenomena behind it.

During the postnatal weeks and months of life, the mother–infant interaction is based on bodily interaction which is complemented by their communicative interplay. The bodily interaction between the infant and mother culminates in breastfeeding, which brings about intense body surface exchanges in sucking and other skin contact and simultaneously provides the infant with the satisfaction of its vital instinctual needs. In a natural way, such early experiences form a developmental frame for defining how the body ego is formed and how it begins to function as the basis of later ego development.

The sensory experiences of neonatal caring and nursing have a coenesthetic character according to Spitz [2], i.e. the contributions of sensory experiences from inside the organism and outside from the external sense organs cannot be fully differentiated. Skills based on practicing of the sensory systems have not yet been acquired, since the volitional motor behavior has not matured and is not at the disposal of the infant. The infant responds by means of reflexes and in a mode of total organismic reactions even though the sense organs are already able to register and differentiate incoming stimuli such as the mother's voice from non-mother voices at 1 or 2 days after birth [12], and an interaction exists between the instinctual activity such as sucking of the infant and the mother's speech, which reinforces the former [13].

The primitive bodily affects of satisfaction arising from infant care and nursing merge with the pertinent sensory processes and result in instinctually intense all-encompassing experiences that are able to change the psychophysiological state of the infant. It can be assumed that, in its nascent form, the body ego is characterized by an undifferentiated matrix-like structure which connects the various sources of stimuli, external and internal, and the responses following them into a network that regulates the whole state of the organism. In this chapter, aspects of infant neurophysiology and their relation to metapsychology are brought together to illuminate the hypothesis of the body ego as a primordial matrix-like network.

The Impact of Birth

When intrauterine life comes to the end at delivery, fundamental psychophysiological changes are brought about. The continuous biological tube-feeding through the umbilical cord, which has enabled passive survival of the infant in utero, comes to an end. The infant is required to make active efforts to survive by initiating breathing and sucking. Otherwise, the life of the helpless mammalian human offspring would not continue. Therefore, the satisfactions provided by feeding and other care implicit in feeding not only have hedonistic meaning, but they are of vital significance. The satisfactions repel the imminent death that would be the fate of the infant if care remained absent. Caring for the infant's basic needs therefore pertains not only to the satisfaction of hunger and libidinal oral drives, but also to the neutralization of imminent death processes that potentially endanger the infant's life if adequate care is not provided.

In the first half of the last century, Isakower [14] and Lewin [15] reported on hypnagogic hallucinations and dream screen phenomena. The observations were made from children in a state of just falling asleep, especially when somatically ill and with a fever, and from adult regressive patients with severe psychopathology. Isakower and Lewin concluded that the hypnagogic hallucinations and the dream screen phenomena sometimes discernible behind adult dream images both represent sensory memories stemming from infantile breastfeeding. These observations lent the first clinical support to the notion of Freud that the early experiences of care involve a principle of psychical projection, i.e. sensory memory-formation by body surface stimulation.

The structural elements included especially in Lewin's [15] formulation of the nature of the dream screen have passed with little notice, even though they seem to have implications for Freud's definition of early body ego development. In previous communications it has been suggested that the dream screen represents the first proto-psychological ego organization, i.e. the emergent body ego [16, 17], in spite of Lewin's own opinion that structural effects were not yet involved. Lewin [15] pointed out, however, that both libidinal wish-fulfillment and longing for a death-related nirvana principle are included in the dream screen phenomena simultaneously in the satisfaction of the oral instinct and the reduction of the effects of excitatory stimuli on the infant (stimulus shield). In this way, the principles involved in the formation of the dream screen imply a fusion of the libidinal and death instincts and suggest that structural elements are created as the dream screen begins to take shape, a view also taken by Spitz [2]. Besides the fusion of instincts, a fusion between self and object also takes place in the mouth–nipple contact and in the reciprocal skin stimulation of the infant and mother. Nursing thus provides a unique and all-encompassing experience of satisfaction that favors conditions for the transformation of id needs into proto-psychological body ego functions of the infant.

Neurophysiology of Nursing

Experimental studies performed by the interdisciplinary research team at the Department of Psychiatry, Kuopio University Hospital, have provided results suggesting that nursing is paralleled by concomitant neurophysiological mechanisms [18, 19]. In the newborn period, nursing of the infant is connected with a change in the amplitude level of the posterior cortical areas of the infant which is more apparent in the right posterior cortical area. This has been observed when a hungry infant's electroencephalogram (EEG) has been quantitatively recorded before nursing, during nursing (breast or bottle) and immediately after nursing has been brought to a satisfactory end while the infant is still awake and calm.

The amplitude change in especially the right posterior areas was only a little higher during nursing than during bottle-feeding, but in contrast to nutritional nursing, no significant amplitude changes in the EEG were detected in connection with pacifier sucking. Instinctual satisfaction together with the pertinent sensory stimulation involved in nursing procedures may thus change the activity of the newborn infant's cortex and leave traces in its neurophysiological functioning, especially with regard to the repetition of the feeding procedures hundreds of times during the first weeks and months of life.

At the age of 3 months, using the same study protocol, significant amplitude changes during feeding could no longer be observed. However, when the same infants were followed up to the age of 6 months, the EEG displayed a new type of significant increase in activity, i.e. theta rhythm during feeding (Fig. 1) [19].

Organized rhythmic EEG activity in the waking state is not possible before the age of 3–4 months and its appearance is a sign of a new maturational level in the neurophysiology of the infant brain. In this respect the increase in rhythmic theta activity during nursing suggests that nursing in a 6-month-old infant is able to activate the neural network that connects subcortical and cortical sources of activity. While it is clear that nursing in the newborn period also involves subcortical brain structures concerned with the regulation of appetite, sucking and satisfaction of hunger, the reinforcement of the rhythmic activity at the age of 6 months explicitly shows that the connectivity between subcortical and cortical areas has matured enough to create a basis for an *organized neural network* that allows the cortical registration of nursing-related sensory events together with the activation of subcortical centers regulating feeding behavior.

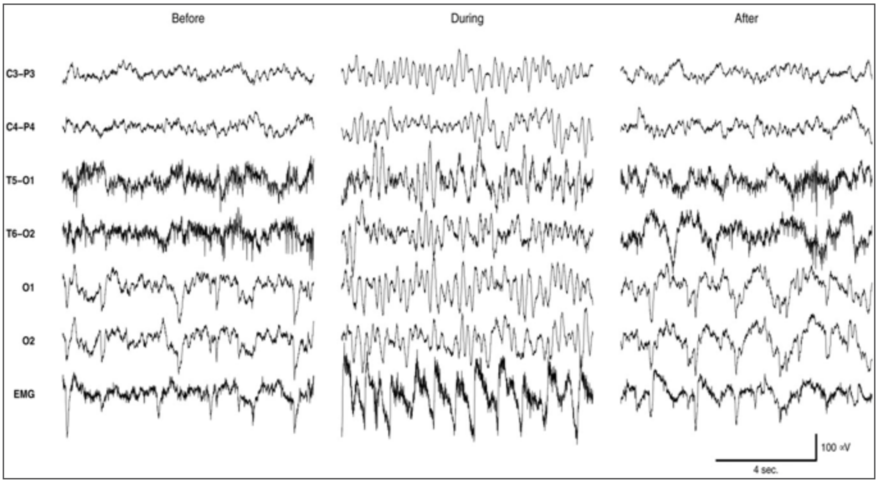

Fig. 1. Electroencephalogram (EEG) of a female 6-month-old infant from six different channels reflecting brain activity from corresponding cortical areas in both hemispheres before, during and after feeding. Waveforms from the top: *C3–P3* left centroparietal, *C4–P4* right centroparietal, *T5–O1* left temporooccipital, *T6–O2* right temporo-occipital, *O1* left occipital, and *O2* right occipital. Feeding is associated with a marked increase of rhythmic 3- to 5-Hz theta activity in the EEG channels. EMG channel (*lowermost waveform*) shows the sucking activity

The nursing experience of the infant also involves activation of the autonomic nervous system. Monitoring the heart rate of the infant during nursing demonstrated that sucking is connected with a regular increase in the heart rate. Its acceleration signifies that cardiovascular work is required by the infant while sucking and that satisfaction is not gained passively [18]. The frequency of the sucking movements is between 1 and 2 Hz (60–120 sucks/minute), i.e. almost at the rate of the heart beats, and they occur in 5- to 10-s bursts (Fig. 2).

Neurophysiology of Nursing Integrated with Psychoanalysis

The neurophysiology of nursing as related to incipient mental organization of the infant thus consists of an entity that entails three major elements: (1) the role of regulation of subcortical centers, (2) the capability to perform active oral muscular work (first reflexively and later volitionally) and (3) an incipient mental image of the nursing procedures at the

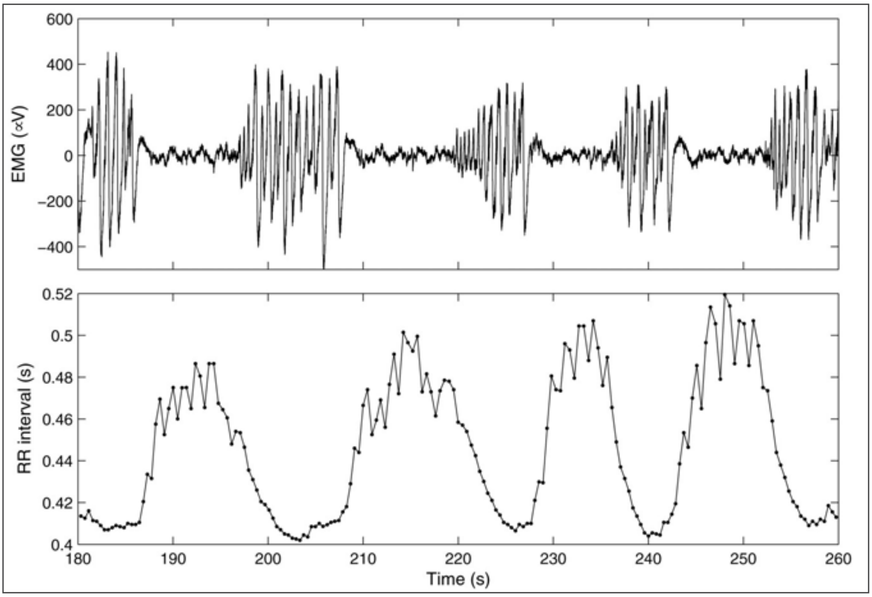

Fig. 2. Surface electromyogram measured from under the jaw shows the rhythmic sucking activity of a newborn (*upper panel*). Each muscular activity burst is accompanied by an immediate response in heart rate. Heart rate increase is visualized by shortening of the distance between consecutive R peaks of the electrocardiogram, i.e., negative deflection of the waveform (*lower panel*)

cortical level. It seems plausible that the formation of neurophysiological connectivity between the different subcortical nuclei and cortical areas begins right after birth. However, due to the low level and undifferentiated character of the EEG activity of the newborn, the findings obtained so far from newborn infants need to be taken as preliminary and require further confirmation.

Once the organization of brain-stem, striatal and cortical activity has been established as a functioning unity, however, the ensuing proto-mental ego most likely begins to function as a template for more mature interactions between the developing subject and his/her object. The age of around 2 to 3 months is a landmark in the developmental process from a poorly organized neonatal stage to the first signs of purposeful behavior and sensory-motor coordination, which coincides with the appearance of a social smile and the waning of a disposition to colic-type cry signals [20, 21]. This age has been regarded as a threshold to new developmental organization by Spitz [2, 3], Mahler et al. [5], Stern [6], Gaddini [22] and Greenspan [23] and the mechanisms mediating the promoting effect on the development of infant-mother interaction have been studied extensively by Fonagy et al. [8].

The behavioral changes appearing at this age are concomitant with signs of a new neurophysiological organization which is shown in emergent rhythmic EEG activity that is not yet present in the newborn EEG [24]. The ongoing formation of an organized neural model for processing of stimuli provided by the mother has also been confirmed by means of auditory cortical responses when event-related potentials (ERP) of 4-month-old infants have been recorded in response to the mother's voice and compared to the response to voices of female strangers [25].

Other investigators such as Maulsby [26], Paul et al. [27] and Futagi et al. [28] have made similar observations of the neurophysiological effects of nursing behavior and provided results that support these findings. Maulsby [26] recognized rhythmic theta activity in a 9-month-old infant while the child was being bottle-fed, and also while she was watching pleasant pictures, and termed the rhythm "hedonic theta activity." Futagi et al. [28] were also able to record rhythmic theta activity in connection with handling and gazing of the infant, and also when the infant was crying.

It is plausible that the rhythmic theta activity is connected not only to pleasant, but also to unpleasant affective stimulation, i.e. it may function as a neurophysiological template for early affective experience including both pleasure and unpleasure, a principle that was already suggested by Walter [29] following his classical studies on older children.

Theta rhythm is also activated in the brain during dreaming [30]. Its generation is dependent on the hippocampus and its mapping functions of the bodily, environmental and temporal space [31, 32]. It can be assumed that theta activity during dreaming could be capable of supporting the transformation of a neurophysiological map of bodily functions into a mental space, since spatial and mental imagery are assumed to originate in the same sensory and associative cortical areas, especially in the right hemisphere [33, 34].

Freud's [35] view of the role of the unrepressed unconscious, i.e. the role of systemic, but not yet dynamically repressed unconscious, may thus be linked to neurophysiological phenomena. The infantile origin of the unrepressed unconscious has been suggested by Mancia [36, 37] to be related to the emerging capacity of the infant to form nonverbal implicit memories laid down from the interactions between the infant and the mother, a view that is consonant with the findings and interpretations of the impact of nursing presented in this chapter. The instinctual components of caring for the infant's vital needs and the sensory processes providing the basis of memory and cognitive functions should not be seen as separately working developmental lines. They should rather be regarded as reciprocal processes that interact and influence each other and together create the basis of the first mental images representing the bodily self of the infant and, respectively, the nascent image of the caring mother-person.

The Double Roots of the Body Ego

From a neurobiological perspective the neural connectivity that structures the brain is determined on the one hand by internal genetic rules for the architectural design of the brain, and on the other hand by actual experiences of satisfaction or reward mechanisms [38]. In interactional and psychological terms, the early mental matrix arises from two sources, those provided by the infant's own organism and those contributed by the mother. The emergent organization of the experiential world is instigated by their interaction and gradually leads to a proto-mental formation *within the infant* as the traces of the neurophysiological processes related to feeding are repeated and consolidated. Winnicott's statement that there is no such a thing as a baby, but in the beginning, there is a baby–mother unity, thus finds interesting support from the neurophysiological effects of nursing.

A proto-mental matrix also appears to bear a relation to the idea of a grid presented by Bion [10] to describe the proto-psychological transformation that takes place by means of nursing satisfactions and results in a process changing somatic beta elements into a representation-capable alpha element organization. The age between 2 and 3 months also seems to coincide with the emergence of primary identification of the infant with the mother [39], which gives an object-relational shape to the incipient mental organization and forms, according to Salonen [40], an object-relational precondition for organized self-experience.

The double roots of the early mental organization—in the infant and in the mother—also have clinical implications. Boyer [41] has concluded from the treatment of severely regressed adult patients that revitalization in the transference of the dream screen plays a therapeutic role. Several other authors have made similar suggestions that the activation of the dream screen or its equivalent matrix may represent a turning point in clinical treatment and may be capable of revitalizing the self–object relation and promoting clinical integration in the psychotherapy or analysis of adult patients [17, 42–45]

The first object of the infant acquires the nature of a thing cathexis, which is characteristic of the stage preceding the capacity for verbal symbolic communication. The nonverbal thing cathexis is the first and true object cathexis of the infant according to Freud pp. 201–202 in [35]). Ferrari [46] presents the same developmental phenomenon in only slightly different words when he suggests that the original psychological meaning of the body is to function as a concrete original object of the mind. A concordant principle is also implied in the work of Gaddini [22], who has given impressive descriptions of the relation of the infant to its body and the development of this relation as a derivate of infant–mother interaction, both when successful and when maladaptive.

Hägglund and Piha [47] have regarded body experiences as essential to the formation of the inner space of the body image, i.e. for the creation of a mental image of body functions. A parallel line of thinking is also presented by the neurologist Antonio Damasio [48–50]. In his view, the image created in the cortex represents the functions of vital homeostatic brain processes and is central to the arising self-experience. He regards the formative process of the self-image as taking place in reciprocal neurophysiological activations between the different levels of brain functioning.

The Intermediating and Active Role of the Nascent Mental Images

In the chain of neurophysiological events from sensory input to motor output, the nascent mental images acquire the function of an in-between variable between the endpoints of the sensory-motor chain. Nascent mental images, and later mental images proper, provide the infant with new enriched means to respond behaviorally to the demands stemming from internal and external sources. The early mental images thus serve a nuclear intermediating function in the perceptual-motor cycle that corresponds to the functions Freud attributed to the mind in his schematic model in *The Interpretation of Dreams* (p. 541 in [51]).

It is important to recognize that the body ego as an organization of the first (proto) mental image of the self is not just a passive structure, but an active protagonist in doing its job of achieving the vital satisfactions. The increase in heart rate during sucking reveals the correlation between the sucking work and physiological effort. Clinical observations of infants incapable of sucking suggest that sucking is an important element in the early mental organization of the body ego. Infants who have not been able to suck during the neonatal period because of esophageal atresia have been found to show retarded mental development. This was first discovered by Spitz [2] in the Monica case and later by Dowling [52], who has given an account of a series of seven infants with esophageal atresia. If surgical correction was not performed early enough, motor, mental and social retardation developed, suggesting that sucking activity is a stimulus for brain development and is likely to reinforce the intentionality of the infant in his/her relation to the mother-object. A related point has also been raised by Gaddini [22], who has suggested that compensation of object loss by pathological incorporative acts such as rumination is followed by retardation of mental development.

Conclusions

Clinical psychoanalytic work centers on metaphoric and symbolic aspects of the mind within fully developed psychoanalytical object relations. The meaning of the body and the earliest layers of the personality is often insufficiently integrated into this work due to the preverbal and unconscious nature of early mental layers. Recently, however, a number of psychoanalytical papers [36, 37, 53–57] and books [33, 58–61] have been published on the role of the body and brain in psychoanalysis. Increasing understanding of the nature of the psychophysiological and neuroscientific aspects of the organization of the basic mental layers has several implications for developmental research and also for clinical work, i.e. how to listen, conceptualize and respond to the early layers of the human personality in the clinical setting [17, 33, 36, 37, 62–65].

The developmental and clinical integration of the meaning of the body in psychoanalysis can be related to principles in brain functions that are connected to instinctual satisfactions and infant–mother interactions. The neurophysiological functions activated by mother–infant interactions may support the development of the first organization of the mind into a body ego that binds the various elements of the infant's responses into a primordial mental matrix.

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References

1. Piontelli A (1988) Prenatal life and birth as reflected in the analysis of a 2-year-old psychiatric girl. Int Rev Psycho-Anal 15:73–81
2. Spitz RA (1957) No and yes. International Universities Press, New York
3. Spitz RA (1959) A genetic field theory of ego formation. International Universities Press, New York
4. Winnicott DW (1987) Through paediatrics to psycho-analysis. Hogarth Press, London
5. Mahler M, Pine F, Bergman A (1975) The psychological birth of the human infant. Basic Books, New York
6. Stern DN (1985) The interpersonal world of the infant. A view from psychoanalysis and developmental psychology. Raven Press, New York, pp 93–104
7. Tyson P, Tyson RL (1990) Psychoanalytic theories of development. An integration. Yale University Press, New Haven
8. Fonagy P, Gergely G, Jurist EL, Target M (2004) Affect regulation, mentalization, and the development of the self. Karnac, London
9. Klein M (1975) Love, guilt and reparation and other works. Hogarth Press, London
10. Bion WR (1963) Elements of psycho-analysis. Karnac, London
11. Freud S (1923). The Ego and the id. SE 19:1–59, pp 26–27
12. Fifer WP, Moon CM (1994) The role of mother's voice in the organization of brain function in the newborn. Acta Paediatr 397(Suppl):86–93
13. Mehler J, Bertoncini J, Barrière M, Jassik-Gerschenfeld D (1978) Infant recognition of mother's voice. Perception 7:491–497
14. Isakower O (1938) A contribution to the patho-physiology of phenomena associated with falling asleep. Int J Psychoanal 19:331–345
15. Lewin B (1946) Sleep, the mouth, and the dream screen. Psychoanal Q 15:119–134
16. Lehtonen J (1991) The body ego from the point of view of psychophysical fusion. Psychother Psychosom 56:30–35
17. Lehtonen J (1997) On the origins of the body ego and its implications to psychotic vulnerability. In: Volkan V and Akhtar S (ed) The seed of madness: constitution, maternal environment, and fantasy in the organization of the psychotic core. International Universities Press, New York, pp 19–57
18. Lehtonen J, Könönen M, Partanen J et al (1998) The effect of nursing on the brain activity of the newborn. J Pediatr 132:646–651
19. Lehtonen J, Könönen M, Partanen J, Saarikoski S (2002). The effects of feeding on the electroencephalogram in 3- and 6-month-old infants. Psychophysiology 39:73–79

20. Soltis J (2004) The signal functions of early infant crying. Behav Brain Sci 27:443–490
21. Lehtonen J (2004) From a undifferentiated cry towards a modulated signal. Behav Brain Sci 27:467
22. Gaddini E (1987) Notes on the mind-body question. Int J Psychoanal 68:315–329
23. Greenspan SI (1992) Infancy and early childhood. International Universities Press, Madison, CT
24. Scher MS (1989) Pediatric electroencephalography and evoked potentials. In: Swaiman KF (ed) Pediatric neurology: principles and practice, vol 1. Mosby, St Louis, pp 67–103
25. Purhonen M, Kilpeläinen-Lees R, Valkonen-Korhonen M et al (2005) Four-month-old infants process own mother’s voice faster than unfamiliar voices—electrical signs of sensitization in infant brain. Brain Res Cogn Brain Res 24:627–633
26. Maulsby RL (1971) An illustration of emotionally evoked theta rhythm in infancy. Hedonic hypersynchrony. Electroencephalogr Clin Neurophysiol 31:157–165
27. Paul K, Dittrichová J, Paposek H (1996) Infant feeding behaviour: development in patterns and motivation. Dev Psychobiol 29:563–576
28. Futagi Y, Ishihara T, Tsuda K et al (1998) Theta rhythms associated with sucking, crying, gazing, and handling in infants. Electroencephalogr Clin Neurophysiol 106:392–399
29. Walter WG (1968) The living brain. Penguin Books, Harmondsworth, pp 171–199
30. Winson J (2002) The meaning of dreams. Sci Am 12:54–61
31. Bland BH, Oddie SD (2001) Theta band oscillation and synchrony in the hippocampal formation and associated structures: the case for its role in sensorimotor integration. Behav Brain Res 127:119-36
32. Yamaguchi Y, Aota Y, Sato N et al (2004) Synchronization of neural oscillations as possible mechanism underlying episodic memory: a study of theta rhythm in the hippocampus. J Integr Neurosci 3:143–157
33. Solms M, Turnbull O (2002) The brain and the inner world. Karnac, London New York
34. Schore AN (2003) The seventh annual John Bowlby memorial lecture. In: Corrigall J, Wilkinson H (eds) Revolutionary connections. Psychotherapy and neuroscience. Karnac, London New York, pp 7–51
35. Freud S (1915). The unconscious. SE 14:159–215
36. Mancia M (2003) Dream actors in the theatre of memory: their role in the psychoanalytic process. Int J Psychoanal 84:945–952
37. Mancia M (2006) Implicit memory and early unrepressed unconscious: their role in the therapeutic process. (How the neuroscience can contribute to psychoanalysis.) Int J Psychoanal (in press)
38. Kandel E (1998) A new intellectual framework for psychiatry. Am J Psychiatry 155:457–469
39. Hoffer W (1966) Infant observations and concepts related to infancy. In: Hoffer W, Early development and education of the child. The International Psycho-Analytical Library. Hogarth Press, London, pp 75–90
40. Salonen S (1989) The restitution of primary identification in psychoanalysis. Scand Psychoanal Rev 12:102–115
41. Boyer LB (1960) A hypothesis regarding the time of appearance of dream screen. Int J Psychoanal 41:114–22
42. Rycroft C (1951) A contribution to the study of the dream screen. Int J Psychoanal 32:178–1884

43. Volkan VD (1975) Cosmic laughter: a study of primitive splitting. In: Giovannini PL (ed) Tactics and techniques of psychoanalytic psychotherapy, vol 2. Jason Aronson, New York, pp 425–440
44. Quinodoz JM (1999) ‘Dreams that turn over the page’. Integration dreams with paradoxical regressive content. Int J Psychoanal 80:225–238
45. Saarinen PI, Lehtonen J, Joensuu M et al (2005) An outcome of psychodynamic psychotherapy: a case study of the change in serotonin transporter binding and the activation of the dream screen. Am J Psychother 59:61–73
46. Ferrari AB (2004) From the eclipse of the body, the dawn of thought. Free Association Books, London
47. Hägglund T-B, Piha H (1980) The inner space of the body image. Psychoanal Q 49:256–283
48. Damasio AR (1996) Descartes’ error. Emotion, reason and the human brain. Putnam Books, New York
49. Damasio AR (2000) The feeling of what happens. Body, emotion and the making of consciousness. Vintage, London
50. Damasio AR (2003) Looking for Spinoza: joy, sorrow and the feeling brain. Harcourt, New York
51. Freud S (1900) The interpretation of dreams. SE 5:336–625
52. Dowling S (1977) Seven infants with esophageal atresia: a developmental study. Psychoanal Study Child 32:215–256
53. Lehtonen J (1980) The relationship between neurophysiology and psychoanalysis. Perspect Biol Med 23:415–423
54. Lombardi R (2002) Primitive mental states and the body: a personal view of Armando B. Ferrari’s concrete original object. Int J Psychoanal 83: 363–381
55. Chetrit-Vatine V (2004) Primal seduction, matricial space and asymmetry in the psychoanalytic encounter. Int J Psychoanal 85:841–856
56. Paniagua C, Hartmann E (2004) What has happened to the body in psychoanalysis? Int J Psychoanal 85:973–976
57. Mancia M (1999) Psychoanalysis and neuroscience: a topical debate on dreams. Int J Psychoanal 80:1205–1213
58. Reiser MF (1990) Memory in mind and brain. What dream imagery reveals. Basic Books, New York
59. Levin FM (1991) Mapping the mind. Analytic Press, Hillsdale, NJ, London
60. Cozolino L (2002) The neuroscience of psychotherapy. Building and rebuilding the human brain. Norton, New York London
61. Corrigall J, Wilkinson H (2003) Revolutionary connections. Psychotherapy and neuroscience. Karnac, London, New York
62. Lewin B (1955) Dream psychology and the analytic situation. Psychoanal Q 24:169–199
63. Isakower O (1992) The analyzing instrument. J Clin Psychoanalysis 2:209–215
64. Scalzone F, Zontini G (2001) The dream’s navel between chaos and thought. Int J Psychoanal 82:263–282
65. Quinodoz D (2003) Words that touch. Int J Psychoanal 84:1469–1485

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