The Interpretation of Dreams and the Neurosciences.

Prof.Dr. Mark Solms

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Shortly after Freud's death, the study of dreaming from the perspective of neuroscience began in earnest. Initially, these studies yielded results which were hard to reconcile with the psychological conclusions set out in this book. The first major breakthrough came in 1953, when Aserinsky and Kleitman discovered a physiological state which occurs periodically (in 90 minute cycles) throughout sleep, and occupies approximately 25% of our sleeping hours. This state is characterized, among other things, by heightened brain activation, bursts of rapid eye movement (REM), increased breathing and heart-rate, genital engorgement, and paralysis of bodily movement. It consists, in short, in a paradoxical physiological condition in which one is simultaneously highly aroused and yet fast asleep. Not surprisingly, Aserinsky and Kleitman suspected that this REM state (as it came to be known) was the external manifestation of the subjective dream state. That suspicion was soon confirmed experimentally, by Aserinsky and Kleitman (1955) and Dement and Kleitman (1957a, 1957b). It is know generally accepted that if someone is awakened from REM sleep and asked whether or not they have been dreaming, they will report that they were dreaming in as many as 95% of such awakenings. Non-REM sleep, by contrast, yields dream reports at a rate of only 5-10% of awakenings.

These early discoveries generated great excitement in the neuroscientific field; for the first time it appeared to have in its grasp an objective, physical manifestation of dreaming - the most subjective of all mental states. All that remained to be done, it seemed, was to lay bare the brain mechanisms which produced this physiological state; then we would have discovered nothing less than how the brain produces dreams. Since the REM state can be demonstrated in almost all mammals, this research could also be conducted in subhuman species (which has important methodological implications, for brain mechanisms can be manipulated in animal experiments in ways that they cannot in human research.)

A sequence of studies followed, in quick succession, in which different parts of the brain were systematically removed (in cats) in order to isolate the precise structures that produced REM sleep. On this basis, Jouvet was able to report in 1962 that REM (and therefore dreaming) was produced by a small region of cells in a part of the brain stem known as the pons. This part of the nervous system is situated at a level only slightly above the spinal cord, near the nape of the neck. The higher levels of the brain, such as the cerebral hemispheres themselves which fill out the great hollow of the human skull, did not appear to play any causal role whatever in the generation of dreaming. REM sleep occurs with monotonous regularity, throughout sleep, so long as the pons is intact - even if the great cerebral hemispheres are removed completely.

Neuroscientific research into the mechanism of REM sleep continued along

these lines, using a wide variety of methods, and by 1975 a detailed picture of the anatomy and physiology of `dreaming sleep' had emerged. This picture, which is embodied in the reciprocal interaction and activation-synthesis models of McCarley and Hobson (1975, 1977), has dominated the field ever since - or at least, as we shall see, until very recently. These authoritative models proposed that REM sleep and dreaming were literally `switched on' by a small group of cells situated deep within the pons, which excrete a chemical called acetylcholine. This chemical activates the higher parts of the brain, which are thereby prompted to generate (meaningless) conscious images. These meaningless images are nothing more than the higher brain making `the best of a bad job ... from the noisy signals sent up from the brain stem' (Hobson & McCarley, 1977, p. 1347). After a few minutes of REM activity, the cholinergic activation arising from the brainstem is counteracted by another group of cells, also situated in the pons, which excrete two other chemicals: noradrenaline and serotonin. These chemicals `switch off' the cholinergic activation (and thereby, according to the theory, the conscious experience of dreaming).

Thus all the complex mental processes that Freud elucidated in this book were swept aside and replaced by a simple oscillatory mechanism by means of which consciousness is automatically switched on and off at approximately 90 minute intervals throughout sleep by reciprocally interacting chemicals which are excreted in an elementary part of the brain that has nothing to do with complex mental functions. Thus, even the most basic claims of Freud's theory no longer seemed tenable:

> The primary motivating force of dreaming is not psychological but physiological since the time of occurrence and duration of dreaming sleep are quite constant suggesting a preprogrammed, neurally determined genesis. In fact, the neural mechanisms involved can now be precisely specified ... If we assume that the physiological substrate of consciousness is in the forebrain, these facts [i.e. that REM is automatically generated by brainstem mechanisms] completely eliminate any possible contribution of ideas (or their neural substrate) to the primary driving force of the dream process. (Hobson & McCarley, 1977, pp. 1346, 1338)

On this basis, it seemed justifiable to conclude that the causal mechanisms underlying dreaming were `motivationally neutral' (McCarley & Hobson, 1977, p. 1219), and that dream imagery was nothing more than `the best possible fit of intrinsically inchoate data produced by the auto-activated brain-mind' (Hobson, 1988, p. 204). The credibility of Freud's theory was, in short, severely strained by the first wave of data about dreaming that was obtained from `anatomical preparations' (Freud, 1900a, p. 000) - and the neuroscientific world (indeed the scientific world as a whole) reverted to the pre-psychoanalytic view that `dreams are froth' (Freud, 1900a, p. 133).

However, alongside the observations just reviewed, which provided an increasingly precise and detailed picture of the neurology of REM sleep, a second body of evidence gradually began to accumulate, which led some neuroscientists to recognize that perhaps REM sleep was not the physiological equivalent of dreaming after all (Solms in press).

The notion that dreaming is merely `an epiphenomenon of REM sleep' (Hobson et al, 1998, p. R12) rested almost exclusively on the observation that arousal from the REM state yielded dream reports on 70-95% of awakenings, whereas non-REM awakenings yielded such reports in only 5-10% of attempts. Considering the vagaries of subjective memory (and especially memory for dreams), this is as close to a perfect correlation as one could reasonably expect. However, the sharp division between REM (`dreaming') sleep and non-REM (`non-dreaming') sleep began to fray when it was discovered that reports of complex mentation could, in fact, be elicited in as many as 50% of awakenings from non-REM sleep. This became apparent when Foulkes awakened subjects from non-REM sleep and asked them `what was passing through your mind?' rather than `have you been dreaming?' (Foulkes, 1962). The resultant non-REM dream reports were more `thought-like' (less vivid) than the REM dream reports, but this distinction held only for the statistical average. The fact remained that at least 5-10% of non-REM dream reports were `indistinguishable by any criterion from those obtained from post-REM awakenings' (Hobson, 1988, p. 143). These findings `do not support a dichotomic distinction between REM and NREM mentation, rather they suggest the hypothesis of the existence of continuous dream processing characterized by a variability within and between sleep stages' (Cavellero et al, 1992, p. 563).

The non-REM dream reports could not be explained away as misremembered REM dreams, for it soon became apparent that dream reports could regularly be obtained even before the dreamer had entered the first REM phase. In fact, we now know that dream reports are obtainable from as many as 50-70% of awakenings during the sleep onset phase - that is, in the first few minutes after falling asleep (Foulkes & Vogel, 1965; Foulkes et al, 1966; Vogel et al, 1972). This is a far higher rate than at any other point during the non-REM cycle, and almost as high as the REM rate. Similarly, it was recently discovered that non-REM dreams appear with increasing length and frequency towards the end of sleep, during the rising morning phase of the diurnal rhythm (Kondo et al, 1989). In other words, non-REM dreams do not appear randomly during the sleep cycle; dreaming is generated during non-REM sleep by specific non-REM mechanisms.

The only reliable difference between REM dream reports, sleep-onset reports, and certain other classes of non-REM dream report is that the REM reports are longer. In all other respects, the non-REM and REM dreams appear to be identical. This demonstrates conclusively that fully fledged dreams can occur independently of the unique physiological state of REM sleep. Therefore, whatever the explanation may be for the strong correlation that exists between dreaming and REM sleep, it is no longer accepted that dreaming is caused exclusively by the REM state.

The presumed isomorphism between REM sleep and dreaming was further undermined by the emergence, very recently, of new and unexpected evidence regarding the brain mechanisms of dreaming. As already noted, the hypothesis that dreaming is merely an epiphenomenon of REM sleep rested on the high correlation between REM awakening and dream reports. But this does not imply that REM and dreaming share a unitary brain mechanism. In the light of the discovery that dreams regularly occur independently of REM sleep, it is certainly possible that the REM state and dreaming are controlled by independent brain mechanisms. The two mechanisms could well be situated in different parts of the

brain, with the REM mechanism frequently triggering the dream mechanism. A two-stage causation of REM dreaming implies that the dream mechanism could also be stimulated into action by triggers other than the REM mechanism, which would explain why dreaming so frequently occurs outside of REM sleep.

This hypothesis, that two separate mechanisms - one for REM and one for dreaming - exist in the brain, can easily tested by a standard neurological research method known as clinico-anatomical correlation. This is the classical method for testing such hypotheses: the parts of the brain that obliterate REM sleep are removed and the investigator observes whether or not dreaming still occurs; then the parts of the brain that obliterate dreaming are removed and the investigator observes whether or not REM still occurs. If the two effects dissociate, then they are caused by different brain mechanisms. If they are affected simultaneously by damage to a single brain structure, then they are served by a unitary mechanism.

It is known that destruction of parts of the pons (and nowhere else) leads to a cessation of REM sleep in lower mammals (Jones, 1979), but such experiments cannot, of course, be performed on humans - the only species which is in a position to tell us whether or not destruction of those parts of the brain leads simultaneously to a cessation of dreaming. Fortunately (for science) the relevant brain structures are occasionally destroyed in human cases by naturally occurring damage, due to spontaneous illness or traumatic injury to the brain. Twenty-six such cases have been reported in the neurological literature, with damage to the pons which resulted in a total or near-total loss of REM sleep.[1] Surprisingly, the elimination of REM in these cases was accompanied by reported loss of dreaming in only one of the 26 patients (Feldman, 1971). In the other 25 cases, the investigators either could not establish this correlation or they did not consider it. By contrast, in all the other cases ever published in the neuroscientific literature in which damage to the brain did result in a reported loss of dreaming (a total of 110 patients), a completely different part of the brain was damaged and the pons was spared completely.[2] Moreover, it has been proven that REM sleep is completely preserved in these cases, despite their loss of dreaming.[3] This dissociation between cessation of REM and cessation of dreaming seriously undermines the doctrine that the REM state is the physiological equivalent of the dream state.

The parts of the brain that are crucial for dreaming and those that are crucial for REM sleep are widely separated, both anatomically and functionally.

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[1] Adey et al, 1968; Chase et al, 1968; Cummings & Greenberg, 1977; Feldman, 1971; Lavie et al, 1984; Markand & Dyken, 1976; Osorio & Daroff, 1980 (for bibliographic details see Solms, 1997).

[2] Basso, Bisiach & Luzzatti, 1980; Boyle & Nielsen, 1954; Epstein, 1979; Epstein & Simmons, 1983; Ettlinger, Warrington & Zangwill, 1957; Farah, Levine & Calviano, 1988; Farrell, 1969; Gloning & Sternbach, 1953; Grünstein, 1924; Habib & Sirigu, 1987; Humphrey & Zangwill, 1951; Lyman, Kwan & Chao, 1938; Michel & Sieroff, 1981; Moss, 1972; Müller, 1892; Neal, 1988; Nielsen, 1955; Peña-Casanova et al, 1985; Piehler, 1950; Ritchie, 1959; Solms, 1997; Wapner, Judd & Gardner, 1978; Wilbrand, 1887, 1892 (for bibliographic details see Solms, 1997).

[3] Benson & Greenberg, 1969; Brown, 1972; Cathala et al, 1983; Efron, 1968; Jus et al, 1973; Kerr, Foulkes & Jurkovic, 1978; Michel & Sieroff, 1981; Murri et al, 1985 (for bibliographic details see Solms, 1997).

The parts of the brain that are crucial for REM are in the pons, which is located in the brainstem, near the nape of the neck. The parts of the brain that are crucial for dreaming, by contrast, are situated exclusively in the higher parts of the brain, in two specific locations within the cerebral hemispheres themselves.

The first of these locations is in the deep white matter of the frontal lobes of the brain, just above the eyes (Solms, 1997). This part of the frontal lobes contains a large fibre-pathway which transmits a chemical called dopamine from the middle of the brain to the higher parts of the brain. Damage to this pathway renders dreaming impossible, but it leaves the REM cycle completely unaffected (Jus et al, 1973). This suggests that dreaming is generated by a different mechanism than the one that generates REM sleep - a conclusion which is strongly supported by the observation that chemical stimulation of this dopamine pathway (with drugs like L-DOPA) leads to a massive increase in the frequency and vividness of dreams without it having any effect on the frequency and intensity of REM sleep (Klawans et al, 1978; Scharf et al, 1978; Hartmann et al, 1980; Nausieda et al, 1982). Likewise, excessively frequent and vivid dreaming which is caused by dopamine stimulants can be stopped by drugs (like anti-psychotics) which block the transmission of dopamine in this pathway (Sacks, 1985, 1990, 1991). In short, dreaming can be switched `on' and `off' by a neurochemical pathway which has nothing to do with the REM oscillator in the pons.

What, then, is the function of this higher brain pathway which is so crucial for the generation of dreams? Its main function is to `instigate goal-seeking behaviors and an organism's appetitive interactions with the world' (Panksepp, 1985, p. 273), that is, to motivate the subject to seek out and engage with external objects which can satisfy its inner biological needs. These are precisely the functions that Freud attributed to the `libidinal drive' - the primary instigator of dreams - in his (1900a) theory. Accordingly, it is of considerable interest to note that damage to this pathway causes cessation of dreaming in conjunction with a massive reduction in motivated behaviour (Solms, 1997). In view of the close association between dreams and certain forms of insanity, it is also interesting to note that surgical damage to this pathway (which was the primary target of the prefrontal leucotomies of the 1950s and 60s) leads to a reduction in some symptoms of psychotic illness, together with a cessation of dreaming (Frank, 1946, 1950; Partridge, 1953; Schindler, 1953). Whatever it is that prevented leucotomized patients from maintaining their psychiatric symptoms also prevented them from generating dreams.

In short, the current neuroscientific evidence gives us every reason to take seriously the radical hypothesis - first set out in this book 100 years ago - to the effect that dreams are motivated phenomena, driven by our wishes. Although it is true that the (cholinergic) mechanism which generates the REM state is `motivationally neutral', this cannot be said of the (dopaminergic) mechanism which generates the dream state. In fact, the latter mechanism is the appetitive (i.e. libidinal) `command system' of the brain (Panksepp, 1985, 1998).

As stated, it now appears that REM only causes dreaming via the intermediary of this motivational mechanism. Moreover, REM is just one of the many different triggers which are capable of activating this mechanism. A variety of other triggers, which act independently of REM, have exactly the same effect. Sleep-onset dreams and late morning dreams are two examples of this kind. Dreams induced by L-DOPA (and various stimulant drugs) are further examples.

Of special interest in this regard is the fact that recurring, stereotyped nightmares can be induced by seizures which occur during sleep.[4] We know from the work of Penfield[5] exactly where in the brain these seizures begin, namely, in the temporal limbic system - which subserves emotional and memory functions, is situated in the higher forebrain, and is richly interconnected with the frontal lobe dopamine pathway discussed above. Moreover, we know that such seizures usually occur during non-REM sleep (Janz, 1974; Kellaway & Frost, 1983). The fact that nightmares can be `switched on' by mechanisms in the higher parts of the brain which have nothing to do with the pons and nothing to do with REM sleep is further evidence that dreaming and REM are generated by separate and independent brain mechanisms.

It is surely no accident that what all of these different mechanisms capable of triggering dreams have in common is the fact that they create a state of arousal during sleep. This lends support to another of the cardinal hypotheses that Freud put forward in this book, namely the hypothesis that dreams are a response to something which disturbs sleep.[6] But it appears that the arousal stimuli enumerated above only trigger dreaming if and when they activate the final common motivational pathway within the frontal lobes of the brain, for it is only when this pathway is removed (rather than the arousal triggers themselves, including REM) that dreaming becomes impossible. This relationship between the various arousal triggers and the dream-onset mechanism itself is reminiscent of Freud's famous analogy: dreaming only occurs if the stimulus which acts as the `entrepreneur' of the dream attracts the support of a `capitalist' - an unconscious libidinal urge, which alone has the power to generate dreaming (1900a, p. 561).

Thus, Freud's major inferences from psychological evidence regarding both the causes and the function of dreaming are at least compatible with, and even indirectly supported by, current neuroscientific knowledge. Does the same apply to the mechanism of dreaming?

Our current neuroscientific understanding of the mechanism of dreaming revolves centrally around the concept of regression. The prevailing view is that imagery of all kinds (including dream imagery) is generated by `projecting information backward in the system' (Kosslyn, 1994, p. 75). Accordingly, dreaming is conceptualized as `internally generated images which are fed backwards into the cortex as if they were coming from the outside' (Zeki, 1993, p. 326). This conception of dream imagery is based on wide-ranging neurophysiological and neuropsychological research into numerous aspects of visual processing. However

[4] De Sanctis, 1896; Thomayer, 1897; Clarke, 1915; Kardiner, 1932; Naville & Brantmay, 1935; Rodin et al, 1955; Ostow, 1954; Epstein & Ervin, 1956; Snyder, 1958; Epstein, 1964; Epstein & Hill, 1966; Epstein, 1967; Boller et al, 1975; Epstein, 1979; Epstein & Freeman, 1981; Solms, 1997 (for bibliographic details see Solms, 1997).

[5] Penfield was able to artificially generate the recurring nightmare scenes by directly stimulating the seizure focus in the temporal lobe (Penfield, 1938; Penfield & Erickson, 1941; Penfield & Rasmussen, 1955).

[6] Solms (1995, 1997) provides limited empirical evidence to support the hypothesis that dreams protect sleep: patients who lose the ability to dream due to brain damage report more disturbed sleep than brain damaged patients with intact dreaming.

the regressive nature of dream processing has recently been demonstrated directly in clinical neurological cases (Solms, 1997).

In order to illustrate this point it is necessary to remind the reader that loss of dreaming due to neurological damage is associated with damage in two brain locations. The first of these is the white fibre pathway of the frontal lobes that we have considered already. The second location is a portion of the grey cortex at the back of the brain (just behind and above the ears) called the occipito-temporo-parietal junction. This part of the brain performs the highest levels of processing of perceptual information, and it is essential for:

> the conversion of concrete perception into abstract thinking, which always proceeds in the form of internal schemes, and for the memorizing of organized experience or, in other words, not only for the perception of information but also for its storage' (Luria, 1973, p. 74).

The fact that dreaming ceases completely with damage to this part of the brain suggests that these functions (the conversion of concrete perceptions into abstract thoughts and memories), like the motivational functions performed by the frontal lobe pathway discussed previously, are fundamental to the whole process of dreaming. However, if the theory that dream imagery is generated by a process which reverses the normal sequence of events in perceptual processing is correct, then we may expect that in dreams abstract thoughts and memories are converted into concrete perceptions. This is exactly what Freud had in mind when he wrote, on p. 543 of this book, that `in regression the fabric of the dream-thoughts is resolved into its raw material.' This inference is supported empirically by the observation that dreaming as a whole stops completely with damage at the highest level of the perceptual systems (in the region of the occipito-temporo-junction), whereas only specific aspects of dream imagery are affected by damage at lower levels of the visual system, closer to the perceptual periphery (in the region of the occipital lobe).[7] This implies that the contribution of the higher levels precedes that of the lower levels. When there is damage at the higher levels, dreaming is blocked completely, whereas damage at the lower levels merely subtracts something from the terminal stage of the dream process. This is the opposite of what happens in waking perception, which is obliterated entirely by damage at the lowest levels of the system. In other words, dreaming reverses the normal sequence of perceptual events.

The available neuroscientific evidence, therefore, is compatible with Freud's conception of where and how the dream process is initiated (i.e., by an arousing stimulus which activates the emotional and motivational systems), and of where and how it terminates (i.e. by abstract thinking in the memory systems which is projected backwards in the form of concrete images onto the perceptual systems).

In fact, it is now possible to actually see where this neural activity is distributed in the dreaming brain. Modern neuroradiological methods produce pictures of the pattern of metabolic activity in the living brain while it is actually

[7] Charcot, 1883; Adler, 1944, 1950; Brain, 1950, 1954; Macrae & Trolle, 1956; Tzavaras, 1967; Kerr et al, 1978; Botez et al, 1985; Sacks & Wasserman, 1987; Solms, 1997 (for bibliographic details see Solms, 1997).

performing a particular function, and in the case of dreaming these images clearly show how the brain's energic `cathexis' (as Freud called it) is concentrated within the anatomical areas discussed above - namely, the (frontal and limbic) parts of the brain concerned with arousal, emotion, memory and motivation, on the one hand, and the parts (at the back of the brain) concerned with abstract thinking and visual perception, on the other hand.[8]

These radiological pictures also reveal something about what happens in between the initial and terminal ends of the dream process. The most striking feature of the dreaming brain in this respect is the fact that a region of the brain known as the dorsolateral frontal convexity is completely inactive during dreams. This is striking, because this part of the brain which is inactive during dreams is one of the most active of all brain areas during waking mental activity. If one compares the pictures of the waking brain with those of the dreaming brain, one literally sees the truth of Fechner's (1889) assertion to the effect that `the scene of action of dreams is different from that of waking ideational life' (cf. Freud, 1900a, p. 536). Whereas in waking ideational life the `scene of action' is concentrated in the dorsolateral region at the front of the brain - `the upper end of the motor system - the gateway from thought to action' (Solms, 1997, p. 223) - in dreams it is concentrated in the occipito-temporo-parietal region at the back of the brain - on the memory and perceptual systems. In short, in dreams the `scene' shifts from the motor end of the apparatus to the perceptual end.[9]

This reflects the fact that whereas in waking life the normal course of mental events is directed toward action, in dreams this path is unavailable. The `gateway' to the motor systems (the dorsolateral frontal convexity of the brain) is blocked in dreams (Braun et al, 1997, 1998; Solms, 1997), as are the motor output channels (the alpha motor neurons of the spinal cord; Pompeiano, 1979). Thus both the intention to act and the ability to act are blocked during sleep, and it seems reasonable to infer (as did Freud) that this block is the immediate cause of the dream process assuming a regressive path, away from the motor systems of the brain, toward the perceptual systems (Solms, 1997).

Finally, due to relative inactivation during sleep of crucial parts of the reflective systems in the frontal parts of the limbic brain, the imagined dream scene is uncritically accepted, and the dreamer mistakes the internally generated scene for a real perception. Damage to these reflective systems (which evidently are not entirely inactive during sleep) results in a curious state of almost constant dreaming during sleep and an inability to distinguish between thoughts and real events during waking life.[10] This provides further evidence of a continuous thought process occurring during sleep, which is converted into dreaming under various physiological conditions, of which REM sleep is just one among many.

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[8] Braun et al, 1997, 1998; Franck et al, 1987; Franzini, 1992; Heiss et al, 1985; Hong et al, 1995; Madsen, 1993; Madsen & Vorstrup, 1991; Madsen et al, 1991a, 1991b; Maquet et al, 1990, 1996 (for bibliographic details see Braun et al, 1997).

[9] It is of utmost interest to note that the major inhibitory systems of the forebrain are concentrated at its motor end, just as they were in Freud's (1900a) diagrammatic representation of the mental apparatus.

[10] Whitty & Lewin, 1957; Lugaresi et al, 1986; Gallassi et al, 1992; Morris et al, 1992; Sacks, 1995; Solms, 1997 (for bibliographic details see Solms, 1997).

The picture of the dreaming brain which emerges from recent neuroscientific research may therefore be summarized as follows: the process of dreaming is initiated by an arousal stimulus. If this stimulus is sufficiently intense or persistent to activate the motivational mechanisms of the brain (or if it attracts the interest of these mechanisms for some other reason), the dream process proper begins. The functioning of the motivational systems of the brain is normally channelled toward goal-directed action, but access to the motor systems is blocked during sleep. The purposive action which would be the normal outcome of motivated interest is thereby rendered impossible during sleep. As a result (and quite possibly in order to protect sleep) the process of activation assumes a regressive course. This appears to involve a two-stage process. First, the higher parts of the perceptual systems (which serve memory and abstract thinking) are activated; then the lower parts (which serve concrete imagery) are activated. As a result of this regressive process, the dreamer does not actually engage in motivated activity during sleep, but rather imagines himself to be doing so. Due to inactivation during sleep of the reflective systems in the frontal part of the limbic brain, the imagined scene is uncritically accepted, and the dreamer mistakes it for a real perception.

There is a great deal about the dreaming brain that we still do not understand. It is also evident that we have not yet discovered the neurological correlates of some crucial components of the `dream-work' as Freud understood it. The function of `censorship' is the most glaring example of this kind. However, we are beginning to understand something about the neurological correlates of that function, and we know at least that the structures which are most likely to be implicated (Solms, 1998) are indeed highly active during dreaming sleep (Braun et al, 1997, 1998).

Hopefully it is apparent to the reader from this brief overview that the picture of the dreaming brain which has begun to emerge from the most recent neuroscientific researches is broadly compatible with the psychological theory that Freud advanced in this book. In fact, aspects of Freud's account of the dreaming mind are so consistent with the currently available neuroscientific data that I personally think we would be well advised to use Freud's model as a guide for the next phase of our neuroscientific investigations. Unlike the research effort of the past few decades, the next stage in our search for the brain mechanisms of dreaming (if it is to succeed) must take as its starting point the new perspective we have gained on the role of REM sleep. REM sleep, which has hitherto diverted our attention away from the neuropsychological mechanisms of dreaming, should simply be added to the various `somatic sources' of dreams that Freud discussed in chapters 1 and 5 of this book. The major focus of our future research efforts should then be directed toward elucidating the brain correlates of the mechanisms that Freud discussed in his 6th and 7th chapters - the mechanisms of the dream-work proper.

> We shall feel no surprise at the over-estimation of the part played in forming dreams by stimuli which do not arise from mental life. Not only are they easy to discover and even open to experimental confirmation; but the somatic view of the origin of dreams is completely in line with the prevailing trend of thought in psychiatry to-day. It is true that the dominance of the brain over the organism

> is asserted with apparent confidence. Nevertheless, anything that might indicate that mental life is in any way independent of demonstrable organic changes or that its manifestations are in any way spontaneous alarms the modern psychiatrist, as though a recognition of such things would inevitably bring back the days of the Philosophy of Nature, and the metaphysical view of the nature of mind. The suspicions of the psychiatrists have put the mind, as it were, under tutelage, and they now insist that none of its impulses shall be allowed to suggest that it has any means of its own. This behaviour of theirs only shows how little trust they really have in the validity of a causal connection between the somatic and the mental. Even when investigation shows the primary exciting cause of a phenomenon is psychical, deeper research will one day trace the path further and discover an organic basis for the mental event. But if at the moment we cannot see beyond the mental, that is no reason for denying its existence. (Freud 1900a, pp. 41-2)

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Prof.Dr.med. Mark Solms
Academic Department of Neurosurgery
St. Bartholomew's &
Royal London School of Medicine
London E1 1BB
Großbritannien