Out-of-the-Body Experiences

Implications for a Theory of Psychosis

Charles McCreery, DPhil
Formerly Lecturer in Experimental Psychology
Magdalen College, Oxford

Oxford Forum

Chapter 8

A theory of psychosis – I

Introduction

In the preceding two chapters I argued that out-of-the body experiences are a phenomenon of sleep, and of Stage 1 sleep in particular. I suggested that in the majority of cases the experience supervened on a state of low arousal, as do most occurrences of sleep, but that in a significant minority of cases it supervened on sleep triggered by hyperarousal, in the manner documented by Oswald (1962).

In the present chapter I shall show how this model can be extended to provide us with an explanatory model of psychosis. I shall argue that many of the phenomena of psychosis, including those I list immediately below, either consist of the brief intrusion of sleep processes into waking life, or represent a secondary response to such intrusions, in an attempt to make sense of them.

A key observation in the context of this theory will be the apparently paradoxical finding of Stevens and Darbyshire (1958) that catatonic patients can be aroused from their stupor by the administration of sedatives. I will be suggesting that catatonic patients are indeed, as Stevens and Darbyshire suggest, in a state of hyperarousal, and that this state of hyperarousal can lead to such phenomena as hallucinations and delusions due to brief episodes of Stage 1 sleep intruding into their waking life.

I will begin by enumerating the phenomenological similarities between Stage 1 sleep and psychosis, such similarities being what we might expect if psychosis is associated with hyperarousal leading to brief episodes of Stage 1 sleep.

Phenomenological similarities between psychosis and dreaming

The similarities between the respective phenomenologies of dreams and psychoses have often been remarked upon. The following list of historical quotations was compiled by La Barre (1975):

> **Aristotle:** '[...] the faculty by which [...] we are subject to illusion when affected by disease, is identical with that which produces illusory effects in sleep.'[1]
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> **Kant:** 'The lunatic is a wakeful dreamer.'
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> **Schopenhauer:** 'A dream is a short-lasting psychosis, and a psychosis is a long-lasting dream.'
>
> **Freud:** 'A dream, then, is a psychosis.'
>
> **Jung:** 'Let the dreamer walk about and act like one awakened and we have the clinical picture of dementia praecox.'

Among the phenomenological features common to both dreams and psychosis I would include the following:

(1) Autism.[2] The dreamer and the psychotic are both preoccupied with internal rather than external events. In the most severe cases of schizophrenia Eugen Bleuler (1911) wrote:

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[1] It is interesting to note that elsewhere in the work from which this quotation is taken Aristotle may be referring to the possibility of lucid dreaming: 'Sometimes, too, opinion says [to dreamers] just as to those who are awake, that the object seen is an illusion [...]' Aristotle, *On Dreams*, translated by J.I. Beare, Part 1.

[2] The word 'autism' is used here in the sense introduced by Eugen Bleuler (1911), and refers to a symptom or phenomenon and not to a diagnostic label. The latter usage, as in the phrase 'autistic children', has this in common with Bleuler's usage, that individuals labelled as 'autistic' may display the phenomenon of autism, along with a range of other possible symptoms. Jaspers (1963, p.328) characterises autistic thinking in Bleuler's sense as 'self-encapsulation in an isolated world'.

> [... the subjects] have no more contact with the outside world [and] live in a world of their own [...] This detachment from reality, together with relative and absolute predominance of the inner life we term autism.

It might be objected that the dreamer *thinks* that he or she is grappling with external events during the dream; but the same could often be said of the psychotic, since one of the most characteristic features of psychosis is the experience of internal events (thoughts and experiences) as being externally imposed.

(2) Loss of autonomy in relation to mental content. The dreamer experiences the dream-world as largely autonomous and beyond his control. The psychotic may experience thought insertion and other forms of subjective loss of control of his or her mental content.

(3) Flattened or inappropriate affect. This was regarded by Bleuler (1911) as one of the two cardinal symptoms of schizophrenia, along with disorder of thought. In dreams we may commit murder with very little emotional disturbance; alternatively, we may experience 'nightmarishness' or horror before anything appears in the dream environment which could rationalise it.

(4) Disorders of meaning. Psychotics can suffer from an excess of meaning, as in paranoia, ideas of reference[3] or what Jaspers (1963) called the 'primary delusional experience' (discussed further below). Alternatively, they may suffer from a dearth of meaning, as in depersonalisation, derealisation, or various forms of 'existential anxiety' (Cutting 1985). Dreams may likewise present us

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[3] Many interesting examples of the overinvestment of external stimuli with personal meaning can be found in Strindberg's autobiographical work *Inferno*. The following is an example: 'One morning [...] I entered the Luxembourg Gardens [...] There on the ground I found two dry twigs, broken off by the wind. They were shaped like the Greek letters for P and y. I picked them up and it struck me that these two letters P-y must be an abbreviation of the Popoffsky. Now I was sure it was he who was persecuting me, and that the Powers wanted to open my eyes to my danger. I became very agitated, notwithstanding this indication that the Unseen was benignly disposed towards me.' (Strindberg 1962, pp.68-69)

with a general feeling of heightened significance, or specific meanings which seem odd from outside the dream, such as elements which in retrospect seem emotionally loaded in ways in which they would not be in waking life.

(5) Delusional beliefs. Jaspers describes delusions as almost the defining condition of madness. He writes: 'Since time immemorial delusion has been taken as the basic characteristic of madness. To be mad was to be deluded [...]' (Jaspers 1963) In dreams our beliefs, such as the idea that one has just been made Chancellor of the Exchequer, may be unjustified, not merely in relation to waking life, but in relation to the hallucinatory events of the dream.

(6) Disorders of thought and language. Oswald (1962) and Mavromatis (1987) give many examples of analogies between the dereistic thinking of dreams, or the hypnagogic state, and that of psychosis. Mavromatis in fact comments that 'practically all of the schizophrenic thought disturbances are encountered in hypnagogia' (p.161).

(7) Lack of insight. Again, this has been held by many to be the defining condition of psychosis, marking it off from other mental disorders such as neurosis. It is certainly a defining condition of dreams, excepting the special case of lucid dreams (Green 1968a; Green and McCreery 1994).

It may be worth expanding on (5) in view of the centrality of delusion as a condition of psychosis. Jaspers (1963) stresses the incorrigibility of delusional beliefs in psychosis, and how this is not related to the general level of intelligence of the subject. In dreams the linkage between beliefs and evidence can be broken in a similar way. There can be a dissociation between beliefs and the 'sense data' which might justify them within the context of the dream. For example, we identify people in our dreams without concern for the fact that they look quite unlike the people they are supposed to be. It is only on waking that we are surprised that the incongruity did not strike us at the time. In the dream our conviction that the person in question stands before us is somehow

self-validating; it scarcely stands in need of any justification from the evidence of our ‘senses’.

It is also possible to see a parallel between the delusory interpretation of external stimuli in psychosis and the incorporation of external stimuli into dreams, as in the classic case of Maury's guillotine dream:

> I was slightly indisposed and was lying in my room; my mother was near my bed. I am dreaming of the Terror. I am present at scenes of massacre; I appear before the Revolutionary Tribunal; I see Robespierre, Marat, Fouquier-Tinville, all the most villainous figures of this terrible epoch; I argue with them; at last, after many events which I remember only vaguely, I am judged, condemned to death, taken in a cart, amidst an enormous crowd, to the Square of the Revolution; I ascend the scaffold; the executioner binds me to the fatal board, he pushes it, the knife falls; I feel my head being severed from my body; I awake seized by the most violent terror, and I feel on my neck the rod of my bed which had become suddenly detached and had fallen on my neck as would the knife of the guillotine. (Maury 1861, pp.133-134; quoted in Mavromatis 1987, p.24.)

In a case such as this the dream rationalises the stimulus in a manner which to the outside observer may seem fanciful. Similarly, the delusory belief of the psychotic may crystallize round a kernel of fact, but one which the psychotic interprets in a highly idiosyncratic way.

False awakenings

To the above phenomenological similarities I would add the following observation which I believe has not been made before: namely that there is a remarkable similarity between the phenomenology of Jaspers’ ‘primary delusory experience’ and the phenomenon which Green (1968a) has termed a ‘Type 2 false awakening’. In view of the relative unfamiliarity of this concept,

it is worth repeating here Green's characterisation of this type of experience, quoted in Chapter 2:

> In this type of false awakening the subject appears to wake up in a realistic manner, but to an atmosphere of suspense. These experiences vary in respect of the length of time which elapses before the subject becomes aware that something unusual is happening. His surroundings may appear normal, and he may gradually become aware of something uncanny in the atmosphere, and perhaps of unwonted sounds and movements. Or he may 'awake' immediately to a 'stressed' and 'stormy' atmosphere. In either case, the end result would appear to be characterized by feelings of suspense, excitement or apprehension. (Green 1968, p.121)

The following is a first-hand account of this phenomenon provided by a subject studied by myself, 'Subject E':

> This consists of waking up, apparently in the usual way, then realising that something is 'wrong'; the atmosphere grows tense and eerie and hallucinatory effects appear. The first few times this happened I was very bewildered for, in spite of the odd effects, it was unlike a dream and, moreover, I seemed to be exactly where I would expect to be at that time of night. The thought that I must be awake and 'seeing things' made me panic and struggle to get free. When, exhausted, I just lay still for a while, everything would suddenly 'click' back to normal. Even in retrospect I was uncertain whether I had been awake or dreaming. Gradually I lost my fear of these experiences, realising that I need only relax in order to awake. I started trying little experiments, such as moving an object during the false awakening and checking its position subsequently, and concluded that my apparent movements and speech did not actually occur, and that the experience was a type of dream. (McCreery 1973, p.118)

It is interesting to compare these descriptions with Jaspers' characterisation of the primary delusory experience:

> Patients feel uncanny and that there is something suspicious afoot. Everything gets a *new meaning*. The environment is somehow different –

> not to a gross degree – perception is unaltered in itself but there is some change which envelops everything with a subtle, pervasive and strangely uncertain light. A living-room which was formerly felt as neutral or friendly now becomes dominated by some indefinable atmosphere. Something seems in the air which the patient cannot account for, a distrustful, uncomfortable, uncanny tension invades him […] (Jaspers 1963, p.98)

The experience is characterized by Jaspers as 'primary' because it precedes any specific hallucinatory perception or delusional belief. It is said to be 'delusory' for the related reason, that there is no specific content in the patient's experience at the time which can be said to rationalize or justify his/her feeling of heightened meaning or significance.

Reed (1972) characterises the primary delusory experience in very similar terms to Jaspers:

> The experience is basically the uneasy awareness of a *change in significance*. Everything seems to be different, changed and disordered. Patients suffering from this primary delusional experience naturally find it difficult to describe. They feel a sense of frightening uncertainty, an awareness of a sinister 'atmosphere', an apprehension of disintegration. (Reed 1972, p.153)

Cutting (1985, p.319) proposes an alternative term for the primary delusional experience, namely *delusional mood*. This he characterizes as 'an uncanny and sometimes terrifying atmosphere in which aspects of the outside world are invested with new meaning.'

It is interesting to note that in both types of experience – the Type 2 false awakening and the primary delusory experience – the feeling of uncanniness or altered significance can be present *before* any 'perceptual' experience (i.e. hallucination proper) occurs to rationalise it.

The remarkable similarity between the two phenomena of the primary delusory experience and the Type 2 false awakening in normal subjects strengthens the case for there being a close relationship between the two realms of sleep and active psychosis. I suggest that the specific manifestation of psychosis, the primary delusory experience, resembles the sleep phenomenon of the false awakening because active psychosis involves the intrusion of episodes of Stage 1 sleep into waking life.[4]

As already mentioned, subjects who experience the Type 2 false awakenings seem to be relatively rare, and there appears to be as yet no quantitative data with regard to which stage in the sleep cycle such experiences may be associated. However, it seems clear that in at least some recorded cases the experience was followed by waking, as in the case reported by Oliver Fox which was quoted above in Chapter 1. The following is another case of a Type 2 false awakening which seems to have been followed by a true awakening; in this case the correspondent is Subject E, quoted earlier in this section.

> I awoke to the realisation that the bedside radio was still on. Someone passed the door on the way downstairs. I turned the music low: strange, I thought, the radio being on this time of night – what was the time? I reached out of bed to look at the clock (about six feet away) but as I did so an eerie feeling came over me, and I hesitated; yet everything looked perfectly natural so I went ahead, against a mounting tension of the atmosphere, and picked up the clock – whereupon it suddenly changed in my hands! Hastily I put it down; its black dial had turned white and the hands moved to the 9 and 10. I recognised this was the false awakening. Pausing a moment to ponder the significance of the position of the hands (for I knew this could not represent the actual time) I dived back under the blankets. Monsters were pressing in on me, I called for help but could not

[4] 'Stage 1', paradoxically, can refer to the hypnopomic (ascending), as well as the hypnagogic (descemding) stage of sleep. In other words, it can refer to the state of emerging from sleep, particularly in the morning, as well as the stage of slipping into sleep at the start of the nocturnal cycle.

> wake up – not until I had seized hold of the monsters and fought them, and flung them on the floor. (Quoted in McCreery 1973, p.119)

In cases such as this, it seems at least possible that the EEG of the subject would have shown the characteristics of emergent Stage 1 sleep.

Three criteria of sleep

My argument in this chapter will be that when the psychotic person appears like one asleep it is because he or she is asleep. As a preliminary I will distinguish three separate criteria of sleep, behavioural, verbal, and electrophysiological, and establish that I shall be regarding the third of these as primary in the present discussion.

The behavioural criterion is the one most likely to be used by an external observer in everyday life when trying to judge whether or not another person is asleep. An observer is likely to judge by such cues as the eyes being closed, the observed person snoring, being unresponsive to verbal questions, etc. In most contexts such cues are a reliable indicator, but not always; for example, there is always the possibility of deception on the part of the observed person. There is also the fact that people can show electrophysiological signs of sleep when displaying none of the usual behavioural signs.

The criterion of verbal report by the observed person may also be used in everyday life. We may meaningfully ask someone, ‘Were you asleep?’, when they have just emerged from a state in which they displayed some or all of the behavioural signs listed above. Again, however, as we shall show below, the observed person’s response is not always to be relied upon; in particular, people are liable under certain circumstances, not simply to dissimulate, but to be mistaken in their retrospective judgements about their own state, believing they

have not been asleep when the third, electrophysiological, criterion, clearly indicates that they have.

For the purposes of the present discussion, the third, electrophysiological criterion will be taken as the necessary and sufficient condition of sleep being ascribed to a subject. In particular, the presence of recognised criteria of sleep in the electroencephalogram (EEG) will be regarded as the primary criterion.

There are at least two advantages of an electrophysiological criterion of sleep in the present context. In the first place, the EEG is relatively immune from conscious control, except perhaps in the relatively restricted area of learned control of the alpha rhythm[5], and is therefore relatively immune from the possibility of deceptive manipulation. In the second place, there is experimental evidence, to be discussed further in the next chapter, to show that electrophysiological data can confirm behavioural evidence of sleep when verbal reports, given in good faith, deny it (Oswald 1962).

A proposed mechanism for the link between dreams and psychosis

In view of the phenomenological similarities between psychosis and dreaming, a number of attempts have been made in the past to propose some underlying mechanism common to the two phenomena. Feinberg (1970), for example, proposed the idea that the visual hallucinations of drug-withdrawal delirium represent the intrusion into waking consciousness of processes normally associated with rapid eye movement sleep. Hypotheses of this kind seem to have made little headway, however.

One reason for the lack of success of the sleep hypothesis seems to be empirical: studies of actively hallucinating schizophrenic patients did not appear to show the features, such as rapid eye movements, which are specific to

[5] See Glossary for a definition.

REM sleep and which might therefore have been expected to appear. As Robbins (1988) puts it, 'The physiological correlates of dreaming and hallucinations appear to be different.'

I suggest that the relative failure of the psychosis-as-dream hypothesis hitherto, at least on the electrophysiological level, is due to the fact that theorists and researchers have been looking for sleep in the wrong place; or to put it another way, they have been looking for the wrong kind of sleep. They have been assuming that the sleep processes in psychosis must be those of REM (rapid eye movement) sleep.

I shall be proposing instead, that the sleep processes of psychosis arise out of a state of hyperarousal, and one associated with Stage 1, not REM sleep.

Hyperarousal during sleep

One reason why we do not immediately think of Figure 3.2 in Chapter 3 as the normal model for the relationship between arousal and sleep is perhaps because for most of us sleep is indeed a state of low arousal. However, for some people, and in certain circumstances, sleep can be a state of high arousal, higher even than waking life. Nightmares, which seem to occur predominantly in conjunction with REM sleep (Parkes 1985, pp.211-212), are the obvious examples of this. But there is also the phenomenon of the night terror, which is associated with slow wave sleep (*ibid*.).

One might question whether the REM phase of sleep should be characterised as a state of low arousal, even in the absence of nightmares, granted the similarity of the EEG to that of aroused rather than relaxed wakefulness. REM sleep seems to be a paradoxical state which shows some analogy to that of catatonia in schizophrenics, which, as we shall see below, is regarded by Stevens and Darbyshire (1958) as a state of cortical hyperarousal, despite its

muscular passivity. In REM sleep, as in catatonia, the behavioural inertness due to lowered muscle tone is apparently accompanied by cortical arousal, at least in comparison with other phases of sleep.

It is also interesting to note that nightmares seem to be particularly prevalent in people with a predisposition to psychotic breakdown. For example, Hartmann *et al* (1981), in a study of 38 subjects who reported frequent nightmares (at least once a week), found a strikingly raised incidence of psychopathology, both among the subjects themselves and their relatives. Four of the subjects met the DSM-III criteria for schizophrenia, nine for borderline personality and six for schizotypal personality;[6] and 22 of them had a close relative who had been institutionalized for mental illness or had had a serious 'nervous breakdown'. The authors go so far as to suggest that a child's continuing to have frequent nightmares at the age of, say, 10 to 12, i.e. several years beyond the age at which such experiences tend to diminish, might have useful predictive value as a marker of risk for schizophrenia.

Various studies have also suggested that the dreams of schizophrenics are particularly liable to include aggressive or threatening elements. Robbins (1988), after reviewing these studies, summarised them thus:

> [...] The dreams of schizophrenics are fraught with anxiety-provoking situations. We all have these experiences occasionally in dreams, but in the schizophrenic they seem to be running rampant. It is as if the control mechanisms that protect us from being overwhelmed by anxiety have gone out of kilter. One has a sense of floodgates that do not work. (Robbins 1988, p.64.)

The present hypothesis is that psychosis-proneness consists in a proneness to states of hyperarousal. I think that the raised incidence of full-blown nightmares

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[6] For a discussion of the theoretical construct of *schizotypy*, and its variant forms, see McCreery and Claridge (2002).

in a psychosis-prone group, and the heightened arousal which seems to characterise the dreams of schizophrenics, are observations consistent with this view. In these two observations we see the tendency to hyperarousal expressing itself in nocturnal sleep, rather than in the daytime sleep episodes which I am suggesting underlie the phenomenon of active psychosis.

Hyperarousal during catatonia

The equation of mania with a state of hyperarousal is almost tautological. However, I believe a similar case can be made for a number of the manifestations of schizophrenia also. Many researchers have believed stress to play an important role in triggering relapse in chronic schizophrenics. What is also highly significant for the present model is the fact that a state of hyperarousal apparently underlies the superficially 'negative' syndrome of catatonia. Stevens and Darbyshire (1958) found that their catatonic subjects became more active, behaviorally, with the administration of amobarbital, a sedative, as if their nervous systems were so over-aroused that they had 'seized up' behaviourally, and the effect of the sedative was initially to release this inhibition, before finally sending them to sleep in the normal way. They write:

> We [...] propose that the term catatonic 'stupor' is a misnomer due to confusion of a psychic state with a behavioural manifestation. The psychic state in catatonic schizophrenia can be described as one of great excitement (i.e., hyperalertness), whether the behavioral manifestation is one of overactivity or underactivity. The inhibition of activity apparently does not alter the inner seething excitement.

I suggest that the psychotic phenomena associated with catatonia in such patients are indeed the phenomena of sleep, but entered from the upper end of the arousal continuum rather than the lower, as represented in Fig. 3.2 in Chapter 3.

Stage 1 versus REM sleep

To return to the question of what kind of sleep is implicated in the mechanism just proposed: as mentioned above, it has previously been assumed that it is REM sleep processes we should be looking for in connection with psychosis. This was presumably because of the historical equation of REM sleep with dreaming, following the discovery of rapid eye movements, and their apparent correlation with dream reports, by Aserinsky and Kleitman (1955). However, subsequent research has suggested that this equation is too simplistic. Mentation, albeit of a somewhat different kind, can be reported following other phases of sleep, and there seems to be no good reason to confine the term 'dream' to the sorts of dramatic, narrative mental events which seem to be predominantly, though not exclusively, associated with REM sleep. I suggest that it is reasonable to expect descending Stage 1 sleep, to be associated with psychotic processes, for the following reasons.

Logic: First, there is the logical reason: human beings characteristically do not plunge straight into REM phases of sleep; they go through the complete cycle of Stages 1 to 4 before reaching the first REM phase, after perhaps 45 minutes (Empson 1989). In other words it is Stage 1 which is usually contiguous with the waking state at the start of a sleep episode in normal people. It would therefore seem natural to look first for descending Stage 1 processes in psychotics if we are looking for sleep processes to explain their symptoms.

The paralysis argument: Apart from rapid eye movements, REM sleep is characterised by lowered muscle tone, amounting to virtual paralysis. There seems little evidence for any corresponding phenomenon in connection with psychosis, with the possible exception of the relatively rare condition of catatonia. Paralysis is occasionally reported in connection with various types of

hallucinatory episode in the sane, including false awakenings, OBEs and apparitional experiences; however, its occurrence is rare and the majority of such cases do not display it (Green and McCreery 1994). It therefore seems reasonable to assume, both in the case of these transient hallucinatory episodes of the sane and in the case of the more chronic episodes of psychosis, that it is some stage other than REM that is primarily implicated in the mechanisms underlying the phenomena.

Phenomenology: The phenomenology of Stage 1 sleep seems to offer many parallels to that of psychosis. These have been extensively reviewed by Oswald (1962), and more recently by Mavromatis (1987).

Oswald particularly highlights the 'dereistic thinking' of the hypnagogic state, and quotes examples of hypnagogic mentation of his own which he considers closely parallel psychotic thinking.

Stage 1 is also a fertile ground for the occurrence of autonomous imagery and hallucinations of various kinds, even in normal people, including auditory images apparently superimposed on the external world. Schacter (1976), for example, cites the case of an experimental subject who, 'thinking that he had been solicited, unhooked himself from the biofeedback equipment and ran into the hallway, only to realize that he had experienced an auditory hypnagogic image'. Leaning (1925) quotes two cases in which the subject heard him- or herself being commented on in the third person, very much in the manner often suffered by schizophrenics. In the first of these cases the subject, a professional man, wrote of his hypnagogic experience:

> There was quite a company of people about me, young women I believe, who looked towards me and passed on. One of them spoke. I heard the voice distinctly, soft and clear. It said '*he isn't asleep.'* That is all [...]

In the second case a woman had a hypnagogic vision after waking in the night; in it the bows of a ship appeared ‘on the left hand side of the bed’, twice. The subject added:

> I said it was not so strange, as a friend had left a few days before for a long voyage, and others were arriving, when quite clearly and *authoritatively* a voice spoke on my right a little behind my pillows: ‘There’s no occasion to warn her. We’ve got one ship off already.’

Psychosis as a disorder of arousal

As may already be apparent, it is the implication of the present model that the fundamental disorder in psychosis is one of arousal. All the other symptoms are held to be the direct result of arousal reaching dysfunctional heights.

As mentioned above, the idea of mania as a dysfunctional extreme of arousal is almost tautological. Less obvious perhaps is how the depressive phase of manic-depressive psychosis may be so regarded. However, two points may be relevant here. One is the clinical notion of ‘agitated depression’, which implies that even ostensibly depressive phases may mask a state of high arousal. The second is the idea that depression results from chronic over-arousal due to stress.

What of schizophrenia, the commoner form of psychosis, and the archetypal form of ‘madness’ to most lay people? What evidence is there for the significance of extremes of arousal in this syndrome? We have already alluded to Stevens and Darbyshire’s view that one of the manifestations of schizophrenia, catatonia, is to be viewed as a state of hyperarousal, despite the behavioural evidence to the contrary. To this I would add recent evidence from the study of schizotypy[7] using non-clinical subjects.

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[7] See Glossary for an analysis of this term.

A questionnaire has been developed by the Chapman laboratory which aims to measure Hypomania in normal subjects (Eckblad and Chapman 1986). It is possible to see this scale as a questionnaire measure of tendency to transient disorders of arousal. Many of the items are designed to tap episodes of what might be called hyperarousal (e.g. 'I often get into excited moods where it's almost impossible to stop talking', keyed True). Others are designed to tap abnormal *lability* of arousal (e.g. 'I seem to be a person whose mood goes up and down easily', keyed True). I have administered this scale, along with others measuring different aspects of schizotypy, to a population of 450 non-clinical subjects reporting at least one out-of-the-body experience and found them to score significantly higher on Hypomania than a group of 214 controls (McCreery and Claridge 1995a). This effect held good across two different methods of subject recruitment, and was mirrored in a within-group comparison of people reporting different numbers of OBEs: it was found that the more experiences they reported the higher they scored on the Hypomania scale.

What is also highly significant for the present theory is that this Hypomania scale is highly correlated in normal subjects with scales measuring various forms of perceptual aberration, such as disorders of the body image (Chapman, Chapman and Raulin 1978) and auditory hallucinations (Launay and Slade 1981). In my own data these correlations were of the order of 0.6. It should be noted that there is no question of cross-contamination between these two sorts of scale, since the 'arousal' scale (Hypomania) does not contain any items concerned with perceptual anomalies, and *vice versa*.

It may also be noted that Slade and Bentall (Slade 1976; Slade and Bentall 1988) have invoked arousal as one of a number of key factors in the aetiology of auditory hallucinations, adducing experimental evidence from clinical subjects in support of this view. They suggest that a state of high internal

arousal, when it interacts with the individual's current level of hallucinatory disposition, can be a crucial factor in triggering such hallucinatory episodes. In particular, they suggest that 'minimal stress [...] may trigger hallucinations in highly predisposed individuals while severe stress would be necessary to trigger a comparable experience in a mildly predisposed person' (Slade and Bentall 1988).

Finally, one might add that the term 'major tranquillisers', which has been applied collectively to the most widely used current pharmacological treatments for schizophrenia, itself carries the implication that hyperarousal is a condition of at least the active phase of the disorder.

Electrophysiological evidence for the model

Because previous attempts to link dreams and psychosis have postulated REM sleep as the mediating mechanism, there have so far been no direct tests of the present hypothesis that descending Stage 1 could be the relevant type of sleep. At this stage, therefore, we have to look for indirect or circumstantial indications of the correctness or otherwise of the hypothesis. However, I believe a number of such indications can be found in the literature.

The EEG

The first observation I would adduce is a curious fact about the alpha rhythm which is so familiar that it almost escapes remark, namely the fact that it responds in a similar way to both an increase and a decrease in arousal: by disappearing, or at least by being masked by other sorts of activity. As Oswald (1962) puts it:

> The alpha rhythm is a feature of a certain level of cerebral vigilance.[8] It gets faster and disappears with increase of cerebral vigilance, and gets slower and disappears with fall of cerebral vigilance. The disappearance of alpha rhythm from the EEG of a person in whom it is normally present requires us always to ask the question, 'Is the individual now very alert or is he drowsy? (Oswald, p.35)

I suggest that this observation is at least consistent with the model of the relationship between sleep and arousal represented in Figure 3.2 in Chapter 3.

Furthermore, a relative dearth of tonic alpha and a relative abundance of beta[9] seem to be characteristic of the EEG of schizophrenics as a group. Flor-Henry (1979), for example, comments: 'Since Berger in 1937 noted the predominance of beta waves in "many mental disturbances" a very large number of studies have consistently found an excess of EEG power in the fast frequencies in the 20 to 50 Hertz band.'

A tonic EEG shifted towards the upper end of the spectrum is what one would expect, on the present model, in people prone to psychosis. It would be compatible with their being habitually in a state of relatively high tonic arousal such that any additional stress factor was liable to precipitate them into that domain of hyperarousal in which sleep is liable to supervene as a provoked reaction.

Another interesting observation in the present context is the fact that some studies have reported a higher than average incidence of delta activity[10] in the waking EEGs of schizophrenics (see for example, Sponheim, Clementz, Iacono

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[8] 'Vigilance' in this context refers to the ability of the brain to respond to a signal detection task. 'Cerebral' refers to the brain as a whole, in contrast to 'cortical', which refers only to the outer layer of the brain, the cortex.

[9] See Glossary for a definition.

[10] See Glossary for a definition.

and Beiser 1994). Although visible delta waves are most characteristic of sleep Stages 3 and 4, spectral analysis suggests that activity in the delta band may be going on all the time, even in the waking state, but masked by faster rhythms, such as alpha, since the power spectrum of the waking EEG will normally show considerable power in the 0-3 Hertz band, albeit less than in the sleeping state. Moreover, short bursts of delta waves visible to the naked eye can be seen even in Stage 1 sleep in certain subjects.

I suggest that the raised incidence of delta waves in the waking EEGs of schizophrenics is also compatible with the idea that sleep processes are 'nearer the surface' in such subjects, and therefore more liable to break into consciousness under the trigger of extreme stress.

The GSR

One of the most consistent findings concerning the functioning of schizophrenics on various physiological measures is such subjects' inconsistency. That is to say, they tend to show both greater variance as a group when compared with controls and greater within-subject variance when tested on a number of different occasions. This phenomenon could be explained by reference to the idea that, unlike normal subjects, schizophrenics are liable to a change of state (from waking to sleeping and *vice versa*) even during daylight hours. Testing them as a group would randomly pick up some subjects who were temporarily in the grip of sleep processes and others who were temporarily free of them. Likewise testing the subject repeatedly might find him or her in the grip of these processes on some days but not on others, depending perhaps on his or her prevailing level of stress at the time.

We may illustrate this idea by reference to the galvanic skin response or GSR. A number of studies, such as that of Gruzelier and Venables (1972), have

found a bimodal distribution among groups of schizophrenics on measures of responsiveness and habituation of the orienting response. That is to say, subjects either did not respond to a standard tone at all in the normal way, or failed to habituate as normal subjects usually do. This pattern of either hypo- or hyper-responsiveness appears to map in an interesting way onto the distinction between negative and positive syndromes in the disorder. Dawson, Schell and Filion (1990, p.319), for example, write: '[...] non-responders and responders have been reported to show different symptomatology, with responders generally displaying symptoms such as excitement, anxiety, manic behaviour, belligerence, and inappropriate mannerisms, whereas non-responders tend to show symptoms such as emotional withdrawal and conceptual disorganisation.'

It is interesting to compare this dichotomy between hypo- and hyper-responders among schizophrenics with the progress of the GSR as an individual subject enters the sleep state. According to Oswald, the GSR may first become less apparent (i.e. skin conductance level become less labile) as the subject falls asleep, but may then reappear as deeper stages of sleep are reached. He writes: 'The interesting thing about the human GSR was that, having disappeared as the individual fell asleep, we found it often returned as cortical vigilance[11] fell even lower [...] not only did the GSRs return during medium or deep sleep in eight of our 19 subjects but they disappeared again as cortical vigilance rose [...]'(Oswald 1962, p.34).

I suggest that this progression may map onto the distinction between the two sorts of responding (or lack of it) in schizophrenic groups.

Claridge (personal communication) has found that the same schizophrenic subject, if tested on more than one occasion, may show a hyporesponsive GSR on one occasion and a hyperresponsive one on another. Such a phenomenon

[11] 'Vigilance' refers to the brain's ability to respond to external stimuli. 'Cortical' refers more specifically to the outer layer of the brain.

would be consistent with the present model, according to which the difference from one occasion to another could be ascribed to a change of state on the part of the subject from one occasion to the next. It is proposed that the hyporesponsive occasions would correspond to those on which the subject was liable to experience moments of the postulated sleep state at the time of testing.

Smooth pursuit eye movements

Although the suggestion in this book is that descending Stage 1 rather than rapid eye movement (REM) sleep is the basis of psychotic symptomatology, I would nevertheless like to suggest that one eye-movement phenomenon in psychotics may indeed be a further indication that sleep mechanisms are involved, namely the relative weakness of schizophrenics at tasks involving smooth pursuit eye tracking movements. Claridge (1994) has reviewed the many studies of this phenomenon and concludes that it is one of the most eligible candidates for being a marker for the disorder. Although rapid eye movements are not characteristic of descending Stage 1 sleep in most subjects, slow rolling eye movements are, as we have seen in the passage from Parkes (1985) quoted in Chapter 3. I would suggest that whatever form of disinhibition is responsible for this phenomenon at nocturnal sleep onset may also be responsible for the poor performance of schizophrenics at smooth pursuit eye movement tasks.

In the concluding chapter I will address some possible objections to the theory proposed above.