

## CESSATION OF DREAMING AFTER BRAIN INJURY

BY

M. E. HUMPHREY and O. L. ZANGWILL

*From the Department of Neurology, Radcliffe Infirmary, Oxford, and the Institute of Experimental Psychology, University of Oxford*

Loss of visual imagery in dreams was first described by Charcot (1883) in an early case report of visual agnosia. The patient, a linguist and classical scholar of some eminence, sustained a sudden and virtually complete abolition of visual memory following a cerebral vascular catastrophe. Although formerly an excellent visualizer, the patient was quite unable to imagine the appearance of his wife and children, or to summon up a mental image of his childhood home. This defect extended to all spheres of visual memory, remote no less than recent, and affected the visualization of colour as markedly as that of form. It is also said to have deprived the patient of visual experience in his dreams. In view of the patient's well-preserved intelligence and general memory, the defect was regarded by Charcot as a circumscribed visual amnesia, and held by him as wholly responsible for the agnosic state.

Although it is not now customary to attribute visual agnosia, wholly at least, to defects of visual imagery, it is a matter for regret that so few investigators have studied the fate of dream imagery in agnosic syndromes. Russell Brain (1941), it is true, comments that one of his patients, a boy of 15 with persistent agnosia and kindred defects, continued to experience visual imagery in his dreams. On the other hand, the patient reported by Adler (1944, 1950) is said to have ceased to experience visual imagery in dreams after the injury which gave rise to a severe agnosic syndrome. In this case, however, unlike Russell Brain's, visual imagery in the waking state appears to have been much depressed. More recently Russell Brain (1950) has referred to a patient who presented a circumscribed loss of visual imagery in the absence of visual agnosia. In this case, too, visual dreaming ceased entirely though the patient continued to experience non-visual dreams. It is therefore probable that loss of visual imagery in dreams is associated with loss of visualization rather than with loss of visual recognition. The whole problem of the relation between visual agnosia, loss of

visual imagery, and changes in dream activity is obviously in need of further elucidation.

In the course of an investigation of psychological changes associated with posterior parietal lesions, three patients have been encountered in which *cessation of dreaming* was spontaneously reported as an after-effect of brain injury. This loss appeared to be permanent in two cases, and temporary in one. All patients reported marked defects of visualization in the waking state together with some degree of visual-spatial loss. The patients were of good previous intelligence level, and two had received a university education. Although studies of imagery are inevitably lacking in objective control, there was no obvious reason to question the reliability and good faith of the patients' testimony. Despite the small number of cases so far studied, it is felt that the whole question of dream activity in its neurological aspects is of sufficient interest to justify a brief communication.

### Case Reports

**Case 1 (No. 9693).**—An officer, aged 26, right-handed, sustained a wound of the right posterior parietal region of the brain in August, 1944 (Fig. 1). The fragment of metal seen in Fig. 1 was removed at operation two days after wounding. His principal disabilities at first were complete left homonymous hemianopia, left hemianesthesia and mild pyramidal tract signs. One month later, neurological findings (apart from hemianopia) were minimal, but fairly marked topographical loss and defects of visual recognition were in evidence. Some defects of visualization and spatial judgment were shown in psychometric tests. On re-examination six years after injury, the hemianopia was still present and the patient reported moderate residual topographical impairment and some disturbance of visual memory. He stated that he had formerly been a good visualizer, but at present his visual images are "very dim" and hard to evoke. He then spontaneously remarked that he seemed to have "lost the habit" of dreaming, and that he had "almost no dreams nowadays". In general, the patient's account of his disabilities led us to conclude that all thought processes of a predominantly visual character (including dreaming) were markedly depressed.

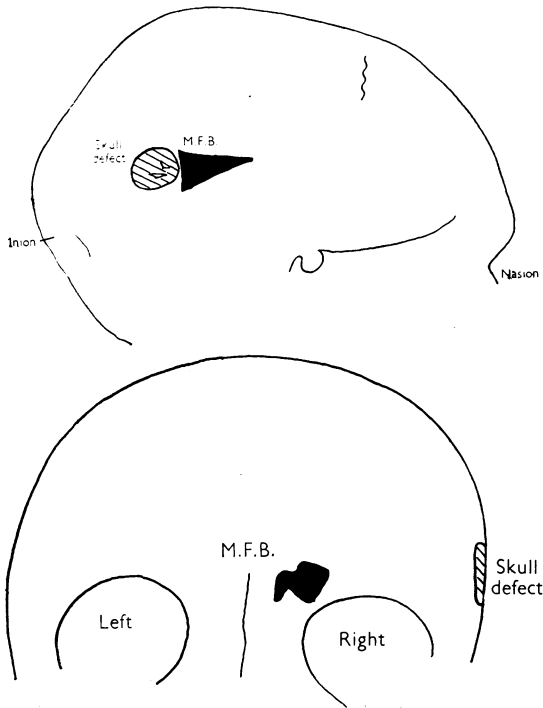


FIG. 1.—Tracing of skull radiographs of Case 1 to show position of skull defect and metal fragment.

There was no suggestion of a general memory loss for recent events which might have provoked amnesia for dream experiences.

**Case 2 (No. 9963).**—A corporal, aged 21, right-handed, sustained a parasagittal posterior parietal mortar wound in September, 1944, which involved the parieto-occipital areas of both sides, predominantly the left (Fig. 2). There was retrograde amnesia for several hours, and the duration of post-traumatic amnesia was about a week. Principal neurological disabilities after remission of the post-traumatic confusional state were bilateral visual field defect (complete left homonymous hemianopia and visual disorientation in right half fields), impairment of joint sense in the right limbs, and dysphasia. There was also mild dyspraxia and topographical loss. On re-examination six years after injury, the patient presented a left homonymous hemianopia and mild residual dysphasic and dyspraxic signs. There was also some topographical and related visual memory loss. He reported that his visualization was still very defective. Before the injury, he had regarded his capacity for visualization as moderately good. Now, however: “. . . if someone says: ‘Can you visualize what your home is like?’ ‘Well, I can do that, but I can’t visualize a lot of things, such as faces sometimes or places I’ve been to and tried to recall.’” He then added spontaneously: “And I never dream”. On further inquiry, he replied that he used to dream “fairly often” before his injury, but that he *had not had a single dream in the*

*last six years.* It also transpired that he now experienced no hypnagogic imagery preceding the onset of sleep. “Lying awake at night trying to go to sleep, I think only in words, never in pictures.” As in Case 1, it appeared unlikely that the apparent loss of dreaming could be explained on the basis of a recent memory defect.

**Case 3 (No. 8846).**—An officer, aged 32, left-handed, sustained a mortar wound of the right posterior parietal area in June, 1944 (Fig. 3). The retrograde amnesia was a few seconds; the post-traumatic amnesia was about one hour. One week after injury, the principal neurological findings were: left homonymous lower quadrantic hemianopia, severe left hemiplegia and cortical sensory loss, and moderate dysphasia. One month after injury, speech and motor function showed considerable improvement, but there was evidence of topographical loss, dressing dyspraxia, confusion of right and left and visual-constructive disability. Psychometric testing at this stage showed some intellectual retardation and marked impairment on all tests commonly held to demand visualization (e.g. “cube-counting”). On discharge, six months after injury, residual disabilities were slight motor weakness of left arm and leg, mild left-sided sensory signs, slowness of speech and reading, and some topographical loss. The visual fields were normal. Despite these handicaps, the patient was able to return to an active and responsible position in civilian life.

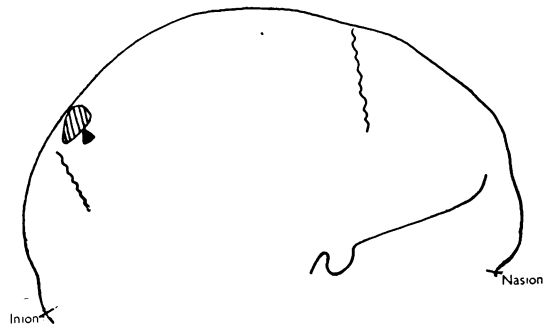


FIG. 2.—Tracing of skull radiograph of Case 2 to show site of wound.

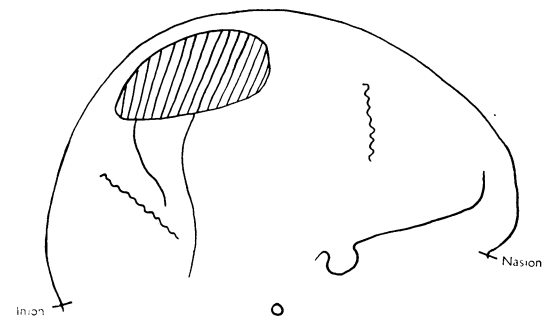


FIG. 3.—Tracing of skull radiograph of Case 3 to show skull defect after operation.

In a progress report five years after discharge, the patient stated :

"An interesting factor is that I have been dreaming again—a thing which I have not done since my injury in 1944."

When interviewed one year later, he confirmed this statement, and added that he now thought that he dreamed as frequently as before his injury. His dreams, moreover, were predominantly visual. He also thought that waking visualization had greatly improved during the last year or so. In this case, then, we may note the restitution of dreaming five years after an injury which caused pronounced defects in higher visual functions.

#### Comment

The above three cases are of interest in presenting marked disturbances of visualization and visual memory as after-effects of brain injury. Case 1 claimed to have been a strong visualizer before his injury, and reported that his visual images had become dim and difficult to evoke. Case 2 reported similar difficulties, and stated that he no longer experienced visual phantasy in the hypnagogic state preceding sleep. Case 3 reported that during the five years in which he experienced no dreams, his capacity for waking visual imagination and recall was much depressed. In general, it may be safely concluded that visual thinking was affected in all three cases. If we regard dreaming as a predominantly visual thought-process, it is not altogether surprising to note its loss in these patients. Case 1 stated that he had "lost the habit" of dreaming almost completely. Case 2 claimed that he had completely ceased to dream since his injury. Case 3 spontaneously reported re-appearance of dreaming after five years of allegedly dreamless sleep. On present evidence, then, it would appear that dreaming is much depressed in patients with impaired visualization due to injury to the occipito-parietal region of the brain.\*

It cannot yet be stated with confidence whether cessation of dreaming occurs only in association with occipito-parietal lesions, or whether it is also found with lesions involving other areas of the cortex. In preliminary inquiries, we have been unable to establish consistent changes in the frequency or character of dreaming in several patients with lesions of the frontal lobes. On the other hand, reduction of dreaming has been reported in a number of patients following *pre-frontal leucotomy* (Partridge, 1950; Slater, personal communication, 1951). Partridge comments that "a large number of patients observed that post-

operatively they slept—as one of them phrased it—'dead', and several remarked that their sleep had become unusually dreamless. This latter tendency, however, disappeared, as did the undue heaviness of sleep, between six months and a year after operation, if not before". Whereas it is evident that Partridge is disposed to attribute post-operative reduction in dreaming to increased depth of sleep, one may point out that it could equally well be due to amnesia on awakening, or to some more generalized depression of cerebral function. In view of the locus of the surgical lesion, however, it is *prima facie* unlikely that the change is due to the implication of higher visual mechanisms, as has been postulated in the present cases. At the same time, the possibility that loss of dreaming in the latter depends, in part at least, on some more general depression of cerebral activity cannot be excluded. A more detailed study of dreaming in cases with cerebral involvement, both diffuse and circumscribed, will obviously be necessary before final conclusions can be drawn.

It is in order to inquire briefly whether data obtained from experiments on cortical stimulation in man are relevant to the present discussion. As is well known Penfield and Erickson (1941) have reported dreamlike states following stimulation of points on the temporal lobe in cases of "psychical" epilepsy. In one of their cases, the subjective phenomena experienced in attacks, and reduplicated by stimulation, had actually formed part of a recurrent nightmare. In a discussion of these findings, Penfield and Erickson observe that "in dreams under normal circumstances there may be a sort of patterned neuronal activity within the temporal cortex of one or both sides. One may be said to dream with his temporal lobes".

In a more recent contribution Penfield and Rasmussen (1950) lay stress on the undeniable similarity between mental states induced by stimulation and ordinary dreams, and regard both as due to the activity of neuronal patterns located in the temporal area. Suggestive though these findings are, it must none the less be pointed out that no case is known to the present writers in which *abolition* of dreaming has been reported to follow temporal lobectomy. Further, it appears most unlikely that dream-like states could be provoked by stimulation in non-epileptic subjects or in cases such as our own, in which higher visual activity had already been depressed by a circumscribed cerebral lesion. The significance of Penfield's findings for any theory of the cerebral localization of dream processes must therefore be evaluated with caution.

In conclusion, we may enquire whether there is any parallel to be drawn between the cessation of

\* It is probable that in such cases dreaming is affected much more frequently than is commonly supposed. We are indebted to Prof. R. C. Oldfield for the information that a patient with right occipito-parietal brain abscess, operated upon by Sir Hugh Cairns in 1938, has entirely ceased to dream in the 13 years which have elapsed since operation.

dreaming in cases such as those reported here and the disturbances of language observed in cases of aphasia. From the psychological point of view, there is some reason to regard dreaming as an expressive function, albeit at an inferior (hyponoic) level. As Dalbiez (1941) puts it: "Language expresses thought vocally, the dream expresses it psychically. We may say that the dream is a natural and individual psychic language". On this view it may be argued that, just as the aphasic is unable to express his thought in propositional form, so the agnostic patient may fail to express his ideation at the lower level of phantasy and dream. While not denying that the trend and content of any given dream, or indeed of any given proposition, cannot be interpreted without reference to psychological factors, we would like at the same time to suggest that visual thinking, dreaming and imagination are liable to organic dissolution in a manner directly comparable to the dissolution of symbolic thought in aphasia. But whereas aphasia is specifically associated with lesions of the dominant hemisphere, disorders of visual imagination appear liable to follow lesions on either side, and to be especially marked in cases with involvement of the occipito-parietal region. Their more complete elucidation remains a task for future inquiry.

#### Summary

Three cases are briefly reported in which cessation of dreaming was spontaneously described as an after-effect of occipito-parietal brain injury. The locus of the lesion was right-sided in two cases (one of which was a left-handed man) and bilateral, though predominantly left-sided, in one. This loss appeared to be permanent in two cases and temporary in one. Depression of dreaming was associated with impaired visual imagination and memory in the waking state and with residual

topographical loss. All three patients were of good pre-traumatic intelligence, and reasonable confidence could be placed in their testimony.

Kindred observations from the literature of visual agnosia are mentioned. It is tentatively suggested that dreaming is likely to be affected only in those agnostic states in which there is appreciable impairment of visual imagery.

Reduction of dreaming as an early sequel of prefrontal leucotomy and the dreamlike states which may be induced in certain cases of epilepsy by temporal lobe stimulation are briefly discussed.

It is tentatively suggested that depression of dreaming and visual imagery in consequence of brain injury may be regarded as a dissolution in some respects analogous to aphasia.

We wish to thank Dr. W. Ritchie Russell for his generous encouragement and kind permission to quote extracts from case records at the Oxford Head Injuries Bureau and to interview patients admitted under his care. We have also to express our gratitude to Dr. W. Russell Brain for his helpful comments on this paper.

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