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Episodic Depersonalization

Observations on 7 Patients

By K. DAVISON

The constellation of symptoms which is termed depersonalization is often as perplexing to the doctor as to the patient. It is more easily diagnosed than defined, and much of the controversy over its nosological position can be attributed to this vagueness of outline. Saperstein (1949) reviewed the historical development of the concept and concluded that the essential feature is the experience of a sense of strangeness or unreality to the individual of his personality, his body or the external environment. Other symptoms of cerebral dysfunction, such as perceptual distortion of the body image, déjà vu experiences, olfactory and auditory hallucinations, metamorphopsia, autoscopy and a disordered time sense, are frequently, but not necessarily, associated (Antoni, 1946; Roth, 1959). The difficulty of expressing unusual subjective events in words gives rise to descriptions remarkable for their rich and often bizarre metaphor (e.g. Lewis, 1934; Bockner, 1949; Roth, 1959). Saperstein (1949) and Bird (1958) emphasize the "as if" qualification of these accounts, which distinguishes them from the delusional experiences of depression and schizophrenia.

Depersonalization has been reported in association with a wide variety of organic brain lesions (Ackner, 1954), but the commonest physical correlates are temporal lobe epilepsy (Williams, 1956) and tumour (Bingley, 1958). It has also been experienced by normal people, spontaneously (Roberts, 1960; Dixon, 1963), after prolonged sleep deprivation (Bliss *et al.*, 1959) and under the influence of hallucinogenic drugs (Guttman and Maclay, 1936); nor are children immune (Salfield, 1958). However, the majority of patients with depersonalization of sufficient intensity to cause complaint do not have any demonstrable cerebral lesion or physical precipitant. On this group psychiatric opinion is divided between those who recognize a specific "depersonalization syndrome" (Shorvon *et al.*, 1946) and those who regard depersonalization as always a symptom of some other psychiatric state, such as depression, hysteria, or schizophrenia (Mayer-Gross, 1935; Ackner, 1954). Roth's observation that depersonalization most frequently occurs in a setting of stress-precipitated anxiety led him to formulate the phobic anxiety-depersonalization syndrome (Roth, 1959, 1960). However, the cases reported below indicate that depersonalization of severe degree can occur as an almost isolated symptom, spontaneously or on very slight provocation, and it would seem reasonable to award such cases the appellation "depersonalization syndrome".

Case Summaries

Case No. 1. National Hospital No. 29018

Miss E.H., born 1923, experienced her first depersonalization episode in 1945 and thereafter at intervals of 1–2 years. Her most recent attack in 1962 was her tenth. The episodes begin and end abruptly—"something goes click in my head"—and, if untreated, last for three months. No physical or psychological precipitant has been discerned except that one episode in 1960 developed immediately she recovered consciousness after having a general anaesthetic (thiopentone and nitrous oxide) for a minor operation. During each episode she feels strange and unreal—"like in a dream"—her movements feel automatic and she has difficulty in grasping what is being said to her. Although at times looking depressed and tearful, she maintains that her emotions are numb and she experiences neither pleasure nor sadness. Poor concentration compels her to relinquish her job as a typist during the episodes. Her sleep is not affected, there is no diurnal variation in her symptoms nor does she experience guilt, suicidal ruminations or anxiety.

Between episodes she displays no psychiatric abnormality other than a markedly obsessional personality; and she copes well with her secretarial work and is now engaged to be married. Her father is also extremely obsessional; at the age of 19 years he experienced a

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single episode, identical to those of the patient, which lasted for three months but did not recur. Her mother and elder sister are normal.

The first four episodes were untreated and each lasted approximately three months. In 1951, one month after the commencement of an attack, an intravenous injection of 20 mg. methylamphetamine hydrochloride (methedrine) produced complete relief within one minute and the remission lasted for a year. In the next episode the effect of intravenous methedrine, though immediate and dramatic, was less persistent, but remission occurred after 4 weekly injections. Subsequent episodes were found to respond to 2 or 3 electroconvulsive treatments (E.C.T.). Her last attack, in 1962, was relieved within one minute of injecting 10 mg. methedrine intravenously, but the remission lasted only 3 days. After 2 E.C.T. it was completely relieved, and she has so far remained well for over a year. Oral amphetamine, barbiturates, and a variety of anti-depressant drugs have failed to relieve the symptoms.

Case No. 2. National Hospital No. 21184

Mrs. J.R., born 1929, between 1944 and 1950 experienced episodes of "dreaminess" which had an abrupt onset and termination and lasted 1 to 3 weeks. These occurred every 3 months for the first 2 years and subsequently once a year but no precipitating factors were detected. During the attacks she felt unreal, "like in a dream" and the outside world appeared flat—"lacking the third dimension, like looking at a picture". Her own voice seemed far away and she felt that other people could not grasp what she said. At times she felt sleepy during the day but this was not an invariable feature. She appeared dull, but not obviously depressed or anxious, and her nocturnal sleep was unaffected. Between attacks she felt quite normal. Her last such episode was in September, 1950, and they have not so far recurred. However, in November, 1950, she developed a neurotic depression related to marital difficulties, which was relieved by oral barbiturates and dextro-amphetamine. Further brief periods of anxiety and depression occurred in 1953 and 1955 in association with the illness of her children; then in 1959, after the sudden illness of her mother, she developed a moderately severe anxiety state and reactive depression which responded to iproniazid therapy. She is now symptom-free.

She has always been of meticulous personality, especially concerned with cleanliness and tidiness, and given to worrying that unlikely dangers may overtake her children. However, she is well able to cope with her job as resident caretaker of an office block. After some initial discord she is now happily married and has two daughters. She is an only child and there is no relevant family history.

Case No. 3. National Hospital No. 90327

Mr. H.L., born 1933, developed classical migraine in 1951 and thereafter had attacks every 6–8 weeks. His first episode of depersonalization occurred in November, 1959, whilst having dinner at home with his wife and two friends. He was feeling perfectly normal when suddenly he felt unreal, and everything that was being said seemed an exact replica of something that he had seen and heard before. This lasted for an hour and was accompanied by a sensation of fear; then it ended abruptly, leaving him exhausted but otherwise normal. During the next 18 months, he experienced short attacks of two to three minutes duration in which he felt unreal; felt that he was two people, one of whom was a spectator of his actions. At this time his migraine attacks were much less frequent. He then had a symptom-free period of 5 months without treatment, which was terminated by a severe episode similar to the initial one but lasting two hours. After this he was treated with sodium amytal 1 gr. t.d.s. and for the past year has had only a few episodes of two to three minutes' duration.

He has always been of anxious disposition and describes himself as "super-super careful". He worries about unlikely disasters, has a set routine for the day, and spends fifteen minutes checking the doors and windows of his home before going to bed. However, he sleeps well and is not subject to any phobic anxiety symptoms or depression. No specific sources of stress could be identified. He is happily married, with a son of 4 years; his parents and elder brother are normal, but a cousin is subject to migraine.

Case No. 4. National Hospital No. 94697

Mr. B.C., born 1938, was well until one evening at the age of 17 years, when he drank two pints of beer for the first time. The following morning he felt "as though in a continuous daydream, everything in view seems as important as everything else—I can't focus my attention, nothing seems real, I look past myself in the mirror". This attack lasted for 1 week, then suddenly lifted, leaving him feeling perfectly normal. During the next five years he suffered 12 similar episodes, each lasting from one to two weeks and each following the consumption of alcohol, as little as half a pint of beer being sufficient to precipitate an attack the next morning. Anxiety and depression were conspicuously absent during these episodes; in fact he complained that his emotions were numb. In May, 1961, he decided to stop drinking alcohol, and for the next year he had only two spontaneous attacks of two days' duration. Epanutin therapy was given the credit for this remission, but in April, 1962, whilst still taking epanutin he drank some wine whilst visiting the Continent and the next morning awoke in another attack which persisted for ten days. Since then he has drunk no alcohol and has remained symptom-free without treatment.

He is unmarried and works with his father as a painter and decorator. His parents and younger sister are psychiatrically normal. His mother describes him as an overconscientious, methodical type, but normally cheerful, energetic and friendly.

Case No. 5. National Hospital No. 49367

Mr. C.D., born 1902, experienced his first episode of depersonalization at the age of 16 years. It began abruptly whilst he was visiting a fairground, and lasted six weeks, then ended equally abruptly, leaving him feeling

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quite normal again. The next attack occurred at the age of 19 years whilst in hospital, one week after a mastoidectomy operation, and persisted for three months before again lifting quite suddenly. Further attacks occurred without obvious precipitant at the ages of 24, 30, 38, 51, 59, 60 and 61 years, usually lasting three months.

The sensations he experiences are identical for each attack, though their intensity varies. He complains of feeling in a trance, detached from his surroundings which appear unreal, and other people seem odd and unnatural. The most distressing symptom is a feeling that his head has entirely disappeared, leaving only his eyes protruding on stalks. He feels he is going mad, and ruminates on the possibility of having damaged his brain. During the attacks he is restless, unable to concentrate and unable to work and appears depressed, but there is no diurnal variation in mood and his sleep is unaffected. Anxiety symptoms are absent, and there is no suggestion of phobic aversions of any kind. Between attacks he is psychiatrically normal apart from his unusual personality.

He has always been excessively meticulous and has the reputation of being the most conscientious worker in his firm. He regularly works late in the evening without extra pay, so that everything will be in order for the next day, and he won a national competition for having the cleanest and tidiest machine shop. Throughout his life he has checked lights and doors several times before going to bed. His work record is excellent, and he has held his present job as a maintenance engineer for 27 years. His father, sister and his three children are all said to have very similar personalities.

He has been in a mental hospital on two occasions during an attack, when a diagnosis of depression with hypochondriacal and obsessional features was made. On the first occasion he received modified insulin therapy, and on the second four E.C.T., both without effect at the time but with spontaneous remission occurring later. He had further E.C.T. as an out-patient in 1961 (5) and 1962 (7). He showed some improvement, but in 1961 the duration of the episode was not significantly reduced. In 1962 he felt worse after his 7th E.C.T., but I.V. methylamphetamine 15 mg. relieved all his curious sensations within one minute. This remission lasted for five days; then the symptoms recurred, though less intensely. After a further two injections at weekly intervals he experienced a remission for four weeks. He was then given four E.C.T. with little change, but a further four I.V. injections of 15 mg. methylamphetamine, each of which produced immediate relief of progressively longer duration, led to complete remission which has so far lasted for six months, though the total duration of illness was again three months. No significant benefit has been noted from phenelzine (Nardil), chlordiazepoxide (Librium), imipramine (Tofranil) or amitriptyline (Tryptizol).

Case No. 6. National Hospital No. 42470

Mr. F.J., born 1917, has experienced many episodes in which he feels unreal, his movements are automatic and he loses the ability to feel any emotion. He describes the sensation as "like being in another world", "time has no meaning", "as though I am dead". In some of the attacks he experiences an unpleasant smell for approximately five minutes, which reminds him of an occasion when his car radiator boiled dry. At other times his fingers feel greatly enlarged.

These attacks begin and end abruptly and last from three hours to three months, but most are of three to four days' duration and occur practically every week. He recalls having brief feelings of unreality of one to two minutes' duration at the age of 13 years but the first major episode began abruptly at 1.50 p.m. on 6 May, 1931, when he caught sight of a newspaper headline, "Does pipe smoking cause cancer?" He recalled that he had smoked a cigarette the previous day; at once the attack began and persisted for four days, when it abruptly lifted. Subsequently episodes have occurred spontaneously, but they occasionally follow some minor irritation, e.g. slight criticism of his work. This type of event does not, however, invariably precipitate an attack. During the episodes, he is distressed and agitated and occasionally contemplates suicide, but the moment the episode ceases he feels quite well. Phobic anxiety does not occur.

Between attacks he is a cheerful, talkative, gregarious man, but inclined to worry over trifles. He has always been extremely fastidious over cleanliness and tidiness, a meticulous worker and checker of switches and locks. He suffered from migraine until the age of 14 years. He runs his own tailoring business and is happily married, with a daughter of 16 years and a son of 12 years, both of whom are well. He has four older siblings. An elder brother experienced a single attack of depersonalization at the age of 23 years after reading about the dire consequences of masturbation. This lasted 6 months but has not recurred, although he is also of obsessional personality. His mother, who died at 82 years, is said to have been extremely nervous.

Various treatments, including two courses of 6 E.C.T., anticonvulsants, antidepressants and tranquillizers, have failed to influence the attacks. However, intravenous methedrine 10 mg. usually terminates an attack within 30 seconds (*vide infra*). The severity and frequency of attacks have abated during treatment with trifluoperazine (Stelazine).

Case No. 7. National Hospital No. A11984

Mr. K.A., born 1924, experienced his first episode at the age of 12 years when in hospital three days after an appendicectomy of which he had been very frightened. It began abruptly—"in a split second"—and ended abruptly the next day but returned after two days' relief and then persisted unchanged for six months. During the next year he experienced attacks every three to four weeks, each lasting one to two weeks and beginning and ending abruptly. He could not attend school for the whole eighteen month period. Ever since then he has been subject to identical episodes every three to four months, lasting one to seven days, during which he stays in bed. He is not aware of any specific precipitants, but notices that they seem more frequent when he is under any strain at work. At times he has experienced a sense of unusual well-being for a few hours before an attack

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develops. He describes his sensations during the episodes as being in a dream, detached from reality, as though half his mind is asleep. Colours lose their lustre, the future doesn't seem to exist. During the attacks he often experiences intense déjà vu sensations for several minutes, and he has also frequently seen a "scene like a film" incorporating himself passing before his eyes in vivid detail accompanied by a pleasant smell which he does not recognize. Although during the episodes he is morose and irritable, he has no guilt feelings or diurnal variation, and insomnia is usually initial in type.

Between episodes he is shy and rather solitary, mildly claustrophobic and with a particular dread of hospitals or of losing consciousness. He is scrupulously tidy and checks locks and switches several times at night. He remarks "I'm so fussy that it hurts". Nevertheless he works quite well as a professional golfer and is happily married, with four children. His mother is domineering, meticulous and excessively fastidious, and this made his childhood rather unhappy, but his father and an elder sister are quite placid and easy-going.

The symptoms have been found to disappear completely within 15 seconds of an intravenous injection of 15 mg. methedrine, but usually return after about 30 minutes. Various tranquillizing and antidepressant drugs have failed to influence the symptoms, but his most recent episode terminated abruptly whilst he was taking amitriptyline hydrochloride (Tryptizol) and trifluoperazine (Stelazine) though its total duration was similar to previous untreated episodes. A single E.C.T. during an episode aggravated his symptoms so much that he refused to have any more. When trifluoperazine was withdrawn he immediately suffered a relapse but recovered again when it was reinstituted.

Investigations

In each case physical examination, routine blood count and urine examination, and radiographs of skull and chest were normal, and the W.R. was negative.

Psychological Examination

The patients were tested by Dr. E. Warrington and Miss E. M. Sprayson, using the Wechsler Adult Intelligence and Memory Scales, Weigl's Sorting Test, the Babcock Sentence and Binet Figures, and tests for dysphasia and dyspraxia. The patients' I.Q.s were within the range 104/117 and memory and learning were normal. No evidence of focal cerebral dysfunction was detected in any patient.

Otoneurological Examination

Tests of cochlear and vestibular function were performed by Dr. C. S. Hallpike and Dr. M. R. Dix. Although Case 5 had slight conductive deafness of the left ear attributable to an old mastoidectomy and Case 6 showed a marked bilateral reduction of the caloric responses which was thought to be due to an old vestibular neuronitis, no changes characteristic of a focal cerebral lesion were found (Carmichael *et al.*, 1954, 1961).

Electroencephalographic Examination

In all cases except No. 1 an EEG was obtained during at least one depersonalization episode and also during a remission. EEG activation by a combined hyperventilation-hypoxia technique (Preswick and Reivich, 1962) was also attempted and a thiopentone-sphenoidal lead tracing was recorded during an episode in Cases 6 and 7. The results are summarized in Table I. It can be seen that between episodes there is often a slight excess of slower rhythms and during an attack this tends to increase and the alpha rhythm becomes slower. However, no significant EEG change was observed in Cases 5 and 6 when episodes of depersonalization were terminated by intravenous methedrine whilst an EEG was continuously recorded. No definite focal or epileptic activity was evoked, although Case 2 showed asymmetrical theta activity during an attack.

Treatment

The evaluation of any form of therapy in a condition which is prone to sudden spontaneous remission presents difficulties. No really satisfactory method of treatment for depersonalization has yet been found.

Psychotherapy is rarely effective (Schilder, 1939) and though tranquillizing and antidepressant drugs may relieve associated anxiety or depression, the depersonalization often remains unchanged. Shorvon *et al.* (1946) found that continuous narcosis, modified insulin therapy, benzedrine, epanutin and vasodilator drugs were ineffective. However, claims for the efficacy of repeated intravenous injections of thiopentone in the phobic anxiety-depersonalization syndrome have been made (King and Little, 1959).

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TABLE I

Electro-encephalographic Observations on Cases 2-7

Case No.	Resting EEG		Response to Combined Hypoxia-Hyperventilation Activation	Other EEG Investigations
	During Attack	Between Attacks		
2 ..	Normally responsive alpha rhythm at 9 c/s. Symmetrical excess of theta rhythm. Photic responses symmetrical.	Normally responsive alpha rhythm of higher voltage at 11 c/s. Marked reduction in amount of theta rhythm. Photic responses symmetrical.	Delta rhythm appeared at lower threshold than normal. No epileptic or focal abnormality evoked.	
3 ..	Alpha rhythm 10 c/s, slightly larger on the right, normally responsive. Irregular theta rhythm in right posterior temporal region.	Alpha rhythm 10 c/s, normally responsive. Bilateral theta rhythm in temporal regions only on over-breathing. Photic responses symmetrical.	High threshold. No epileptic or focal abnormality evoked.	
4 ..	Alpha rhythm symmetrical and normally responsive at 8 c/s. Little scattered theta rhythm. Photic responses symmetrical.	Alpha rhythm normally responsive at 9 c/s. Few random theta waves. Photic responses symmetrical.	Low threshold. No epileptic or focal abnormality evoked.	
5 ..	Normally responsive alpha rhythm at 9 c/s. Occasional bilateral theta rhythm only. Photic responses symmetrical.	Normally responsive alpha rhythm at 11 c/s. No theta rhythm. Photic responses symmetrical.	High threshold. No epileptic or focal abnormality evoked.	No significant EEG change during relief of an attack by I.V. methedrine.
6 ..	Normally responsive alpha rhythm at 9 c/s. Paroxysms of bilateral theta rhythm on overbreathing. Photic responses symmetrical.	Normally responsive alpha rhythm at 10 c/s. Occasional random theta rhythm, increased on overbreathing. Photic responses symmetrical.	Ditto	Ditto I.V. Thiopentone with sphenoidal leads showed no abnormality during an attack.
7 ..	Normally responsive alpha rhythm at 9 c/s. No change on overbreathing. Photic responses symmetrical.	Normally responsive alpha rhythm at 12 c/s. Low voltage theta rhythm in both temporal regions on overbreathing. Photic responses symmetrical.	Ditto	Ditto

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There are conflicting reports on the value of electroconvulsive therapy (E.C.T.) in the relief of depersonalization. Ackner and Grant (1960) found that E.C.T. was effective when clear-cut features of endogenous depression were also present, whereas Shorvon *et al.* (1946) and Sargant and Slater (1963) maintain that E.C.T. is rarely effective and may even precipitate the condition. The fact that Ackner and Grant found the combination of depersonalization with endogenous depression to be not uncommon, in contradistinction to Roth (1959) who regards it as extremely rare, suggests that differing diagnostic criteria are being applied. The alleged response to a prolonged course of E.C.T. (Bockner, 1949) could well be due to spontaneous remission. However, the experience of Case 1 (*vide supra*) indicates that E.C.T. occasionally brings dramatic relief and is, therefore, worth trying in an intractable case.

Shorvon (1953) noted that depersonalization is often relieved by intravenous methedrine (methylamphetamine hydrochloride) and this observation was confirmed in Cases 1, 5, 6, and 7. In these, 10–20 mg. methedrine intravenously usually produced complete abolition of all the symptoms in less than one minute without the occurrence of emotional abreaction. Furthermore, the remissions frequently lasted for days, weeks, or even months, long outlasting the pharmacological effects of the drug. Too frequent administration of methedrine may produce habituation or addiction; so it is not a satisfactory treatment for recurrent episodes at brief intervals. However, considerable theoretical interest attaches to its often dramatic effect on depersonalization, which provides a clue to the possible nature of the cerebral processes involved (*vide infra*).

Finally pre-frontal leucotomy has relieved intractable depersonalization (Shorvon, 1947; Sargant and Slater, 1963, p. 122).

Differential Diagnosis

The patients reported here were diagnosed at various times as suffering from schizophrenia (Case 1), endogenous depression (Cases 2, 5, 6, and 7) and temporal lobe epilepsy (Cases 3, 4, and 6), and these are the commonest diagnostic labels for this type of case.

Schizophrenia

The view that depersonalization is often a prodromal symptom of schizophrenia has been expressed by many authors (see Ackner, 1954). The often bizarre nature of the symptoms may suggest this diagnosis, but insight is usually fully retained (Bird, 1958) and it has been claimed that the vague feelings of change experienced by early schizophrenics can be distinguished as qualitatively different (Saperstein, 1949). However, Sedman and Kenna (1963) have recently reported the occurrence of depersonalization in 6 out of 54 schizophrenic patients, but always associated with a depressive mood change and a previous insecure personality. The situation is further complicated by the use of the term depersonalization to refer to passivity feelings and ideas of reference (Langfeldt, 1960) which are indubitable symptoms of schizophrenia. Of the patients reported here, Case 1 had originally been given this diagnosis but neither she nor the other patients showed any definite features of schizophrenia. Depersonalization alone is not a sufficient basis for diagnosing schizophrenia, and in the author's view if the non-delusional criterion for the diagnosis of depersonalization (Störring, 1933; Bird, 1958) is rigorously applied its occurrence in schizophrenia will prove to be comparatively infrequent.

Endogenous Depression

The patients reported here experienced a cyclical illness in which they appeared to be unhappy, had anorexia, poor concentration, and often insomnia, so there is some justification for lumping them together with endogenous depression. Yet there are significant phenomenological differences which suggest that this would be over-simplifying the position. Typical depressive features such as early morning waking, diurnal variation, guilt feelings and a family history of depressive illness were absent. Perceptual distortion of the body image and olfactory hallucinations are, to say the least, unusual symptoms for a straightforward depression. Moreover a depressive illness should respond more consistently to E.C.T., and endogenous depression is usually aggravated rather than relieved by intravenous methedrine

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(Roberts, 1959). Although there have been many reports of depersonalization occurring in the setting of a depressive illness (see Ackner, 1954), Roth (1959) found the association of the two conditions to be rare, and Saperstein (1949) comments that poorly defined delusions in depression are often erroneously called depersonalization. The circular argument that depersonalization is a depressive symptom and therefore its occurrence signifies a depressive illness has little to commend it.

Temporal Lobe Epilepsy

Depersonalization and its associated phenomena often occur as an aura or an ictal event in patients with temporal lobe epilepsy (Williams, 1956). However, it has long been known that it is not the occurrence but rather the paroxysmal quality which is diagnostic of epilepsy (Hughlings Jackson, 1888; Antoni, 1946). Even this dictum is not entirely reliable, since two of the patients reported above have attacks of 2–3 minutes duration, beginning and ending abruptly. In a recent investigation comparing the clinical features of thirty phobic anxiety-depersonalization syndrome patients with thirty temporal lobe epileptics, Harper and Roth (1962) found that depersonalization, déjà vu experiences, metamorphopsia, hallucinations, paroxysmal fear and even loss of consciousness occurred in both groups with equal frequency. The non-epileptic group was, however, distinguished by a commoner family history of neurosis, the occurrence of childhood phobias and migraine, and a more gradual termination of the attacks. Depersonalization lasting minutes or hours was found only in the non-epileptic group, the EEGs of which were either normal or showed only mild non-specific abnormalities.

Of the cases reported here, only Case 7 showed any phobic anxiety features and then only of mild degree. Case 6 alone gave a definite family history of neurosis, although depersonalization had occurred in the relatives of Cases 1 and 6. Cases 3 and 6 had suffered from migraine, but only Case 7 had been subject to phobias in childhood. In all cases the episodes usually terminated abruptly. However, all the patients were of obsessional anxious personality and in Cases 6 and 7 the initial episodes were closely related to psychological stress though subsequent episodes were largely autonomous. If the view of Antoni (1946) is correct that depersonalization without anxiety and anxiety without depersonalization are at opposite ends of a spectrum with varying combinations between, then these cases are obviously generically related to the phobic anxiety-depersonalization syndrome of Roth (Roth, 1959).

The normal or non-specifically abnormal EEGs of these patients do not rule out epilepsy, but the persistent absence of epileptic activity even during the attacks certainly argues against it. The absence of loss of consciousness over a period of years, the duration of the episodes, the lack of response to anticonvulsants and response to such measures as intravenous methedrine or E.C.T. are further distinguishing features. No evidence of focal cerebral dysfunction was obtained from the otoneurological and psychological investigations. The episodes experienced by these patients would seem to be based on a disturbance of cerebral function of a subtler nature than the usual epileptic disturbance, although there are many phenomenological similarities.

The Mechanism of Depersonalization

An association with migraine has been noted by several observers (Hughlings Jackson, 1888; Shorvon *et al.*, 1946; Harper and Roth, 1962), and Todd (1955) regards depersonalization as often a migraine equivalent. Cases 3 and 6 show this association, but no other migrainous features occur during their attacks, and in Case 6 subcutaneous ergotamine tartrate 0·5 g. and the inhalation of amyl nitrite failed to influence the symptoms. But this is not conclusive evidence against a vascular mechanism, hence the question arises whether methedrine exerts its action in these cases via an effect on the circulation. Himwich (1957) adduces evidence that the central stimulant effect of amphetamine derivatives is secondary to their peripheral vasoconstrictor properties, but other workers maintain that they have a direct effect on receptors in the brain-stem reticular system

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(Rothballer, 1957; Bradley and Elkes, 1957). In Case 6 the intravenous injection of 30 mg. mephentermine sulphate (Mephine, Wyeth), which has similar vascular effects to methedrine but produces little cerebral stimulation, did not ameliorate his symptoms. On the other hand, in the same patient it was found that on three occasions whilst he was taking by mouth 150 mg. chlorpromazine daily, intravenous methedrine had no effect, though it returned when chlorpromazine was stopped. Since chlorpromazine has been shown to block adrenaline-induced EEG arousal (Unna and Martin, 1957) and to raise the threshold for EEG and behavioural arousal to stimulation of the reticular formation (Takaori and Deneau, 1962) it seems likely that in this patient methedrine was acting directly on the reticular formation.

The precipitation of depersonalization by alcohol (Case 4), general anaesthesia (Case 1, also see Roth, 1959), and sleep deprivation (Bliss *et al.*, 1959), and its relief in some instances by a drug with an action on the reticular formation, suggest that its basis is a disturbance of cerebral arousal mechanisms (Roth and Harper, 1962). The minor EEG changes observed during the attacks are compatible with this view, though clearly the ordinary EEG is not a very sensitive indicator of slight changes in cerebral arousal.

The occurrence of depersonalization in a high proportion of the normal population (Dixon, 1963; Roberts, 1960) supports the view of Mayer-Gross (1935) that it is a pre-formed functional response of the brain. As such, it has presumably subserved a useful biological function during our phylogenetic history. Roth and Harper (1962) make out a plausible case for the hypothesis that it protects against the disruptive effects on behaviour of excessive emotion, particularly anxiety. As in the cases reported here, the obsessional personality is prone to develop this response with extreme facility and intensity in response to physiological or psychological stress, or even spontaneously (Shorvon *et al.*, 1946; Roth, 1959; Sedman and Reed, 1963). This must surely reflect a peculiarity of the cerebral organization on which this type of personality is based. The occurrence of severe depersonalization in other members of the family of Cases 1 and 6 suggests a genetic basis for this exaggeration of a probably normal response.

Conclusion

The cases reported here support the view of Shorvon *et al.* (1946) that there exists a primary idiopathic depersonalization which usually starts suddenly in adolescence and often recurs over a period of years. As in other investigations, no evidence of a structural cerebral lesion has been detected, but a functional disturbance of cerebral arousal mechanisms is suspected. This syndrome is of considerable interest from the viewpoint of the brain-mind relationship and it would well repay more intensive pathophysiological study.

Summary

The case histories of seven patients with recurrent episodes of depersonalization are presented. Psychological, otoneurological and electro-physiological studies failed to demonstrate any organic cerebral lesion, though minor EEG changes were detected during attacks. Evidence is put forward in support of the hypothesis that a disturbance of cerebral arousal mechanisms may be concerned. The differential diagnosis and treatment of depersonalization are discussed, and it is concluded that the depersonalization syndrome is a distinct entity of which these cases are examples.

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