A peculiar type of post-concussive 'blackout'

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The purpose of this paper is to call attention to an unusual form of post-concussive 'blackout' and to present two case reports which illustrate the syndrome. One report includes a description of a witnessed attack and this encourages us to publish these case reports. One of the reasons why so little is written and known about post-concussive 'blackouts' is that they are seldom seen by a doctor who can examine the patient at the time of the attack.

Although blackouts of one sort or another are common after head injury, the type we describe is rare. We have not found any previous description of such cases, though little attempt is made to differentiate types of attack in the descriptions of post-concussive blackouts in the literature.

The attacks we describe occur in people of good previous personality of any age and either sex. They are characterized by severe headache preceding the blackout, complete and sudden loss of consciousness with falling and injury to the patient, absence of physical signs and E.E.G. changes, and the persistence of attacks for years without response to any form of treatment.

CASE REPORTS

CASE 1  A 51-year-old, married, white housewife was first seen on 13 October 1959 complaining of recurrent blackouts with headache, following a head injury on 2 June 1959.

While working as a sales clerk in a grocery store, she slipped on a patch of jam from a broken jar and struck her head, and remembers falling to the floor striking her head. She was unconscious after the accident until she awoke in a community hospital 30 minutes later and recovered normal consciousness at once without confusion (post-traumatic amnesia 30 minutes). There was no retrograde amnesia. Physical examination at that time was normal except for bruising of the face. She was described as worried and anxious. Skull and chest radiographs, E.E.G., and lumbar puncture showed no abnormality. She remained in hospital three weeks and during the first week 'blackened out' twice in bed without warning. The attacks were not described in detail.

There was no previous history of serious illness or accidents, and she had not visited a doctor for nine years. She was a happily married woman with 10 children, and there was no family history of migraine, seizures, or fainting attacks.

She was first examined by one of us (L.D.O.) on 10 October complaining of recurrent 'blackouts' and headaches since the accident. The headache was temporal and occipital in distribution, of dull aching character, often present on waking in the morning and unaccompanied by nausea or visual symptoms. Although she was seldom free of headache, it became much worse for several hours before each blackout.

Blackouts occurred suddenly two or three times a week and she was forewarned by exacerbation of the headache. She would suddenly fall unconscious and on various occasions had cut her scalp, scraped her knees, broken her dentures, and acquired a black eye. In one attack, while sitting at supper with the family, she fell forward with her head on the table. The family stated that she was limp and unconscious for three minutes without frothing, tongue biting, enuresis, or convulsion. She was tired and cold after the attack, but not confused. The family had noted tension and irritability, poor concentration and sleep disorder since the accident, in this previously placid person.

Physical examination showed a pleasant, well-nourished white woman with a scar on the left forehead from a recent fall. The temperature and pulse rate were normal. The blood pressure was 140/80 mm. Hg. No abnormality was found on general or neurological examination and the mental state was normal. There were no intracranial bruises. An E.E.G. was normal.

The diagnosis of post-concussive syndrome with an unusual seizure disorder was made and she was treated with dilantin and phenobarbitol.

Over the next eight months she was seen at monthly intervals and the blackouts gradually decreased in number to four or five per month, without other changes in her condition. The following drugs in full dosage were tried without effect: Dilantin, phenobarbitol, Equanil, Ergomar, ephedrine sulphate, Mysoline, Darvon, and Mesantoin. A high-calorie diet for six weeks, with 50 g. of glucose at times of exacerbation of headache had no effect.

On 4 July 1960 the patient was admitted to the Massachusetts Memorial Hospitals for full investigation. Physical examination showed no abnormality on admission.
Routine blood examination, urine analysis, and stool examination were normal. The blood Hinton test was negative. Fasting blood sugar and two-hour post-prandial blood sugar levels were 81 mg % and 117 mg % respectively. The blood urea nitrogen was 8 mg %. Lumbar puncture gave clear fluid under normal pressure containing 0 cells, protein 17 mg %, and sugar 54 mg %. The E.E. G., skull, and chest radiographs were normal. A cardiologist found no abnormality of the cardiovascular system and normal E.E.G. A pneumoencephalogram was normal.

The patient was discharged unchanged.

Follow-up examinations at monthly intervals revealed a decrease in the number of blackouts to two or three a month over the next year. They remained unchanged in character and unaffected by treatment.

In September 1961, for the first time a blackout was followed by a complete right-sided hemisensory loss which persisted for 48 hours. Her local doctor found no other physical signs. This right-sided sensory loss also followed two or three subsequent blackouts, clearing within 24 hours each time.

On 14 January 1963, during a blackout, she fell and struck her head on a concrete step with severe bruising, and was re-admitted to the Massachusetts Memorial Hospitals.

Physical examination on admission was negative except for severe bruising of the forehead. On the second hospital day a blackout was witnessed by one of us (M.F.) during a physical examination.

The attack The patient had left her bed 15 seconds previously and was standing at the time. Without warning she became limp and unresponsive and would have fallen without support. She was returned to bed. The blood pressure was 200/70 mm. Hg. The pulse rate was regular at 110 per minute. Respiratory rate was 16 per minute, regular and deep. There was no response to deep pain or pinprick stimulus. The pupils were equal and reacted to light: there was no resistance to eye opening. Both plantar reflexes were down-going. The trunk and extremities showed no temperature change, and there was no pallor or sweating. After two minutes of unconsciousness the patient said, 'Oh, my God', opened her eyes, held her forehead in both hands, and complained of severe headache. After four to five minutes she regained full consciousness and complained of extreme tiredness. On questioning she was unaware of the attack except by inference from the severity of her headache. She was confused at first and thought she had left her bed to go to the bathroom, but rapidly remembered that a physical examination was in progress, and she soon recognized the examiner. Throughout the attack the pulse and respiratory rates did not change and the blood pressure remained constant. Unfortunately an E.E.G. could not be obtained at the time but early the next day it was normal.

Routine investigations, including serum phosphorus and calcium estimations, were all normal, except for a mild iron-deficiency anaemia. An electroencephalogram taken on the tilt table with carotid artery compression resulted in no change of pulse rate or blood pressure. However, the patient became unresponsive for a few seconds and on awakening showed a transient weakness of the right arm and leg. No definite change in the electro-

encephalogram accompanied this episode, but the record was marred by artefact and difficult to interpret.

For this reason a left carotid arteriogram was made on 11 March 1963 by Dr. Edward Spatz and proved normal.

From the date of discharge onwards blackouts occurred at the rate of one per month through the first four months of 1964. There have been none since April 1964 to the present date. The legal case was concluded, and a cash settlement made in March 1964.

CASE 2 A 55-year-old, married, white, male, real estate broker was admitted to the Massachusetts Memorial Hospitals on 3 December 1962 under the care of Dr. Charles Kane, complaining of blackouts preceded by severe headache for the previous two years.

His illness began at midnight one day in November 1960 when he was assaulted on the street, stabbed in the chest wall, and knocked unconscious for several minutes. There was no retrograde amnesia. He was confused on recovering consciousness and drove home at night without headlights. The following day he noticed pain and swelling over the left parietal area of the skull, and he was confused until 1.30 p.m. Thus, the period of post-traumatic amnesia lasted 13½ hours. Three days later he began to have blackouts, all similar in character, occurring once or twice a week. His attacks began with a sensation of warmth in the epigastrium and excessive sweating followed by a sharp pain over the left side of the head lasting for one minute. In some attacks, he was confused during the minute preceding the blackout. He then fell unconscious for several minutes, without convulsion, tongue biting or enuresis, but often bruising himself in the attack. He was confused and drowsy after the attack.

In the two years before admission, the patient had been admitted to other Boston hospitals on three occasions for diagnostic study. Physical examination, skull radiographs, lumbar punctures, and electroencephalograms with sleep study and carotid compression on a tilt table had all been normal. Bilateral carotid arteriograms and two pneumoencephalograms had been normal.

He had been treated with Dilantin, Mysoline, Mesantoin, Librium, Dexamyl, Ritalin, Tofranil, and Bellergal in full dosage without effect.

His previous medical history included concussive injuries 26 years and seven years before admission, each without sequelae. There was a history of excessive drinking many years previously, with one withdrawal seizure 22 years before. For 17 years he had been practically a total abstainer, except for a short period following the death of a daughter 12 years ago. He had suffered from jaundice, pneumonia with empyema, and influenza in childhood. There was an old history of duodenal ulcer.

The family history was negative for epilepsy, migraine, fainting attacks, and psychiatric illness.

Physical examination revealed a well-developed, well-nourished, alert, right-handed white man in no distress. The pulse rate, temperature, and respiratory rate were all normal. The blood pressure was 130/70 mm Hg. Apart from an amблиopic left eye present since childhood and inability to elicit the abdominal reflexes, the physical and neurological examination was normal. The mental state was normal.
Routine blood investigation, urine analysis, fasting blood sugar, blood urea nitrogen, blood Hinton test, total serum protein, and albumin-globulin ratio, skull and chest radiographs, electrocardiogram, and sleep electroencephalogram were all normal. A radiograph of the cervical spine showed minimal hypertrophic osteo-arthritis change. Upper gastro-intestinal radiographs showed a small hiatus hernia and scarring of the duodenal bulb. Bilateral brachial arteriograms showed normal filling of subclavian, vertebral, and right internal and common carotid arteries. The left carotid arteries had been normally filled on previous carotid arteriography.

The patient had no blackouts during his six days in hospital and was discharged unchanged.

Follow-up examinations to the present date show that the blackouts persist twice weekly, and that no medication has influenced them.

**REVIEW OF THE LITERATURE**

In the best known series (Russell, 1932; Ascroft, 1941; Denny-Brown, 1944; Penfield and Shaver, 1945; Quadfasel and Walker, 1947; Rowbotham, 1949; Walker, Caveness, Barrow and Allen, 1954; Wertheimer, 1956; Dereux and Dereux, 1958; Gurdjian and Webster, 1958; Jennett and Lewin, 1960; Lennox and Lennox, 1960; Paillas, Courson, Naquet, and Paillas, 1962) of open and closed head injuries in the literature we have been unable to find any reference to the type of blackout we describe, and we believe this is the first detailed report of such cases.

Gastaut and Gastaut (1957) are two of the few physicians who have discussed the more unusual types of post-concussive blackout following closed head injury. However, they confine themselves to attacks of syncope occurring within hours or days of a head injury and ceasing within a year. Such attacks do not resemble those in the two previous case reports.

Walker and Jablon (1959), in a follow-up study of 932 cases of head injury in World War II, described 12 cases with what they called 'borderline' attacks. The attacks were characterized by dizziness, lightheadedness, and a faint or vague feeling without loss of consciousness, and the electroencephalogram was normal. They considered that the attacks fell into the group described by Gowers as vaso-vagal attacks. In a second group of the 12 cases there were those whose dizzy spell led to a short period of unconsciousness called a 'blackout' but without falling or injury. We do not feel that these cases are comparable with our own.

Hagenmuller (1960), in a statistical study of 2,941 cases of head injury, found 470 cases of post-traumatic epilepsy. Amongst these, 65 patients suffered from 'falling attacks' without other stigmata of grand mal. He comments that in this group of 65 cases there are many with the symptoms of a post-concussive syndrome and unexplained blackouts. He feels that in some of these cases symptoms which appear psychiatric may be masking true post-traumatic epilepsy. In the absence of detailed description of these cases it is hard to assess whether the attacks, but the group may well include cases such as ours.

Jennett (1962) discussed 282 cases of closed head injury, including 13 cases with so-called 'akinetic' general attacks. These patients suffered attacks of unconsciousness lasting five to 30 minutes in which there was no convulsion and the patient was inert and limp. Although no further details of the attack are given and there is no mention of headache in relation to the 'blackouts', it seems possible that this group may include cases like those we describe. There are insufficient data to confirm this idea.

Walker and Jablon (1961), in another follow-up study of 739 cases with head wounds from World War II, found 20 cases which showed '... epigastric feelings, staring or blank spells, associated with definite loss of consciousness for as long as a minute so that 10 of them fell down but did not convulse'. They consider that these 20 cases properly belong to the convulsive group, although the type of epilepsy is not clear either from the case histories or the electroencephalograms, which were abnormal in four cases.

Symonds (1942), discussing unusual types of seizure following contusion, mentions varieties of dizziness which are less easily placed: 'I would draw attention in particular to one which is often described as a "blackout". The onset is sudden, there is dizziness of vision and a sense of insecurity of balance which may result in falling, without any description of true vertigo. Consciousness is often momentarily disturbed and may be lost. The main features of these attacks are syncopal rather than epileptic. Nevertheless in some cases after repetition there is a transition into epilepsy.' He then gives a case report illustrating this type of blackout. The main features of our own cases suggest epilepsy rather than syncope.

Matthews (1963), in a chapter discussing blackouts under the subheading, 'The undiagnosable blackout', describes a case which closely resembles our case 1, though without preceding concussion. 'A woman of 46 has suffered for 10 years from attacks of unconsciousness in which she falls, having from one to six attacks a month. There is no warning, no convulsion or incontinence, and she is unconscious for about five minutes. There do not appear to be any precipitating causes. Investigation has virtually ruled out any of the causes described in this chapter, and anticonvulsants in great variety have never influenced the frequency of the attacks.' This is the
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The nearest description we have found in the literature to our own case reports. Unfortunately, as in our cases, the cause remains unexplained.

The association of headache with blackout in our cases led us to review the literature of the association of epilepsy with migraine. Many authors (Liveing, 1873; Gowers, 1907; Vining, 1922; Bramwell, 1926; Ely, 1930; Bickerstaff, 1961b; Lees and Watkins, 1963) describe cases in which there is a clear and direct transition from a migraine attack to an epileptic fit. However, most of these authors are agreed that this is a rare occurrence and too much significance must not be attached to the few cases in which it occurs. The modern view is that the association of migraine with epilepsy may well be coincidental. Nevertheless, it certainly occurs, and this is of importance in considering later the possible causes of the attacks we are describing.

In brief then, a study of the literature reveals groups of cases in many of the large series of head injuries, which are hard to classify in the absence of detailed descriptions. Most authors regard these blackouts as atypical convulsive disorders, but no mention is made of associated headache or the response of such cases to treatment.

DISCUSSION

In this paper we have attempted to isolate one particular form of post-concussive blackout and consider its peculiar features in detail. These are: severe headache or head pain before an attack, unconsciousness without convulsion, persistence of attacks for many years, lack of response to anticonvulsants or other drugs, and absence of E.E.G. abnormality. We must emphasize that there is no report of an E.E.G. taken during an attack.

The whole subject of these odd forms of post-concussive blackout is a neglected one and merits further study. Although we cannot be sure of the cause of the type we describe, certain possibilities deserve discussion. These are syncope or fainting attacks, vasovagal attacks (Gowers), cerebrovascular insufficiency attacks, hypoglycaemic attacks, cardiac attacks, functional or hysterical attacks, post-traumatic epileptic attacks, and epileptic attacks preceded by vascular change.

We feel certain that the attacks are not simple faints. Headache is unusual before fainting attacks, and the more common symptoms of dry mouth, blurred vision, sweating, and lightheadedness which precede a faint are not seen in these attacks. The attacks are too sudden for faints and they result in falling with injury. Some of them occurred in bed or when the patient was sitting and they were associated with confusion. Finally the attack we witnessed had none of the features of a faint, the patient's colour and blood pressure remaining normal.

Vasovagal attacks as described by Gowers (1907) were prolonged attacks attributed to disorder of the medullary centres. The attacks are never really brief. They seldom last less than ten minutes, and more often continue for half an hour or more. 'The symptoms comprehend subjective gastric, respiratory, and cardiac discomfort, sometimes cardiac pain and even a sense of impending death.' Such attacks are quite unlike those we describe.

The whole subject of cerebrovascular insufficiency is poorly understood. Basically it implies disease of cerebral or extra-cerebral arteries with periods when the blood pressure is inadequate to maintain cerebral blood flow. During the attack witnessed by us, our patient, while still unconscious, showed no fall of blood pressure, no localizing signs, and the pulse rate was rapid and the skin colour normal. Arteriography was normal in both patients and the rest blood pressure was also normal. There would seem to be no serious basis for postulating that cerebrovascular insufficiency caused the attacks.

Hypoglycaemia, as a cause, can be dismissed. Neither patient ever showed an abnormally low blood sugar level, nor were the attacks related in any way to meals. Case 1 was given a high calorie diet for six weeks and advised to take sugar at the onset of headache without any effect whatsoever on the attacks.

It is well known that various types of cardiac disease may cause sudden blackouts. This cause was carefully eliminated in our patients. Cardiologists found no evidence of heart disease in either patient and both showed normal electrocardiograms.

Of major appeal to insurance company lawyers is the explanation of such attacks on a functional or hysterical basis. Certainly many of the symptoms of the 'post-concussive syndrome' closely resemble those found in patients of hysterical personality.

'Pressure type' headaches, dizziness, insomnia, faints, poor concentration, irritability, and altered personality recur again and again in the post-concussive syndrome in persons whose personalities before the accident, though differing widely in type, show no evidence of hysterical illness. Indeed it is this very repetition of the same group of symptoms in case after case of concussion regardless of age, sex, and previous personality, which convinces us that the post-concussive syndrome has an organic basis rather than a psychological one. This is not to deny that secondary neurotic symptoms may become superimposed later. However, it is known that brain injury or disease in a person of normal personality may manifest itself by the appearance of florid hysterical symptoms long before any organic signs
of the underlying brain change have become apparent. Knowing that brain tumour, multiple sclerosis, or the onset of arteriosclerosis may cause a reversion from a normal to a more immature or hysterical personality, it is not unreasonable to suppose that concussion can do the same. What we do not believe is that patients in middle age and previously normal will show hysterical symptoms for the first time, unless some organic change has occurred in the brain. Sargant and Slater (1963) have summed the matter up very clearly: ' . . . interference with bodily well being makes a man more, not less, liable to be disturbed by psychogenic stimuli. In the war neuroses, the psychological causes of anxiety and hysteria, till then relatively inoperative, began to have a greater effect when the man had lost severely in weight. The change in physique had altered his disposition and his ability to adapt. Head injuries illustrate the same phenomenon. Psychiatrists and neurologists are very apt to discover in the headaches of the post-concussive the operation of psychological factors, and to conclude, therefore, that the proper treatment is psychotherapeutic. The change that has escaped notice is that the psychogenic stimuli which now produce symptoms would not have been effective before the injury. The real disability is not a hysterical headache, but an increased susceptibility to hysteria: not the faints, attacks of giddiness, or ill temper which are so clearly due to the circumstances of the moment, but the raised autonomic liability.'

While we would accept this hypothesis as a reasonable explanation of many of the symptoms, including some faints and blackouts, which occur as part of the post-concussive syndrome, we cannot accept it for the type of blackout we are discussing. The sudden onset of these blackouts, the lack of a precipitating cause, the injuries and severe bruising which accompany the falls, and the severely painful headache which precedes the attack, make them unlike the type of blackout which represents a retrogression to a more infantile or hysterical personality after head injury. In our experience such blackouts seldom result in true unconsciousness or injury, have a precipitating psychogenic stimulus and respond eventually to psychiatric help of one sort or another. Moreover in the attack witnessed by us there were no features of any type of hysterical attack with which we are familiar. There was no precipitating cause, the patient was unconscious, the eyes could be opened without resistance, and the attack was followed by confusion. Certainly neither patient showed a hysterical personality before the injury. But it must be admitted that case 1 after the injury did develop a hemisensory loss following some of her blackouts which would certainly be called functional or hysterical. And though the attacks diminished in frequency steadily over the years in case 1, it is a remarkable fact that they ceased entirely one month after the legal settlement. However, for the reasons given, we cannot easily accept the idea that these blackouts are functional, though we certainly cannot unequivocally disprove it.

This brings us to the last two possibilities. Are these attacks an unusual form of post-traumatic epilepsy, or some form of vascular headache which leads on to an epileptic seizure? In favour of epilepsy is the suddenness of the attacks with falling, the injuries sustained by the patient, and the transient confusion which follows them. It is well known that certain types of epileptic attack are not convulsive, and the association of headache with epileptic seizure is also well known. The objections to a diagnosis of post-traumatic epilepsy are first that the E.E.G. remains normal (though unfortunately there are no reports of any taken during a seizure), and secondly that the attacks are stubbornly resistant to all forms of anticonvulsant therapy. If these are post-traumatic epileptic attacks then they represent a form of short, non-convulsive, 'akinetic' attack unresponsive to the usual forms of treatment.

The remaining possibility is that concussion in some way alters the autonomic control of blood vessels leading to vascular headache and subsequent blackout. Certainly head injury may lead to more frequent vascular headaches or 'migraine' in susceptible people, i.e., those of migrainous personality. The difficulty comes in relating the preceding headache to the blackout. The modern view of the relation of epilepsy to migraine is that it is coincidental and that there is no evidence that the vascular changes of migraine result in epileptic seizure except in predisposed persons. Bickerstaff (1961a) has described basilar artery migraine in which unconsciousness may result from the attack, but there are well-marked preceding symptoms of basilar artery insufficiency in those cases, and nothing of this kind occurs in the type of blackouts we are describing. Finally no form of migraine therapy has been of value in our cases.

To sum up, we feel that the probable cause of the attacks is one of the last two possibilities we have discussed. Either the attacks arise from post-traumatic autonomic change of some sort, or they are an unusual form of post-traumatic epilepsy.

Finally we wish to stress the need for further investigation of the post-concussive blackout. We require more information about the electroencephalogram during or immediately following attacks. The medico-legal aspects of these cases demand greater attention than they have received in the past.
SUMMARY

Two cases with an unusual type of post-concussive blackout are described, with a report of a witnessed attack in one case. This type of blackout is preceded by severe headache, refractory to all forms of treatment, persistent for many years, and seen in patients without physical signs or E.E.G. changes.

The literature of the post-concussive blackout is reviewed and its possible causes discussed. The conclusion is reached that the attacks are probably epileptic and may possibly be precipitated by a change in the autonomic control of cerebral blood vessels.

The need for further investigation and the medicolegal importance of the subject is stressed.

We wish to thank Dr. Charles Kane for permission to report case 2.

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