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TRANSIENT GLOBAL AMNESIA

A report of eleven cases, including five of amnesia at the seaside.

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THOUGH described by Bender in 1956 under the title of "Syndrome of Isolated Confusion with Amnesia" the launching of "Transient Global Amnesia" was in the paper of Fisher and Adams in 1964. Three cases were described by Evans in 1966. In the same year the late Lord Brain described a series in a paper to the Association of British Neurologists. It was evident from the discussion that most neurologists had seen examples of the condition and that it was not rare. It therefore deserves to be in the diagnostic repertory of every physician. The cases reported in this paper illustrate well the clinical features.

Case I

Was a 61 year old farmer whose illness occurred on a cold, windy November afternoon, in 1967. His doctor's letter reads: "When this man was working on the farm on Saturday, he suddenly lost his memory—did not remember what he had done —did not recognise his own tractor and when I saw him half an hour later had memory loss for about 2 years back. He did recognise his family, know his age, date of birth, date and time of year. He was unable to tell me any events in the past week and was continually asking questions as to what had happened" . . . Seen 2 days later the period of memory loss had reduced to 4-5 hours of the Saturday evening. Examination was normal. Blood pressure was 140/80.

When seen a few days later by Dr. L. J. Hurwitz there were no neurological findings. X-rays of the chest showed calcification of the aorta and left ventricular enlargement. The E.E.G. was normal. E.C.G. showed evidence of left ventricular stress. Followed up 7 weeks later he was free of symptoms.

Case II

Was an example of "amnesia by the seaside". It was August, 1967, at Arklow. A 45 year old man on a caravan holiday had been taking a daily mid-morning swim with his family for a week. On the final day (Saturday) he went in before breakfast. Sea temperature was 14°C. in the area (air temperature 11°). He came out of the water looking bewildered and said: "Where am I?, What are we doing here?, What day is it?". He recognised all his family, but not the caravan owner. He kept asking the same questions. He was put to bed. Next day he had improved but did not recover fully until the evening. The disturbance lasted 36 hours. He was referred to a psychiatrist.

When seen 2 weeks later and again after 7 months he had no memory at all of the Saturday and there were gaps in his memory for some events of the week prior to the attack. For example, he could not remember several car trips made during this time. Examination was entirely normal. E.E.G. was normal.

Case III

Was a case of amnesia following a bath. A 43 year old road maker came home by lorry from Limerick to Dublin on a cold (—1°C.) snowy night in February, 1963. He had supper followed by a bath. It was not clear whether or not he washed his hair. He emerged from the bathroom ofter 20-30 minutes looking dazed. His eyes were staring. He looked as if in a trance and kept feeling his head. He asked his wife who she was. He did not seem to know he was in his own house. He kept saying, "What am I doing here? I can't remember". He could not remember where he worked though he had been in the same job for 8 years.

He was brought immediately to hospital where no abnormality was found except a blood pressure of 160/110. Seen next day, he had no complaints and examination

was normal except that he could not remember the bath or his visit to the hospital on the previous night and had patchy memory for his stay in Limerick in the previous week.

His wife wrote in 1967 to say that he had had no further trouble. He had ultimately recovered fully his memory for the visit to Limerick.

At the time of the episode no firm diagnosis was made. It was thought that the attack might have been hysterical.

Case IV

Was a further case of amnesia at the seaside.

A man aged 60 years went for a swim in the sea in July, on the east coast of Scotland. He emerged in a state of confusion, which lasted until the following day. He remained in normal health for 20 years. He then began to have occasional days on which he was confused. He was not hypertensive. Finally, at the age of 80 he collapsed and had some sort of seizure with frothing at the mouth. He recovered consciousness but was very weak and died within a few days of pneumonia.

Case V

In 1960 a man aged 52 went for a swim in the sea at Barna, Co. Galway. He emerged from the water in a confused state and his wife had to drive to the hotel. He had no grasp of what had happened and kept asking repeatedly of his wife "Tell me what happened right from the beginning". At dinner that night his wife had to do the ordering from the menu. Next day, he was normal. He subsequently could remember going into the water and that he became extremely cold and then there was a blank until he was sitting fully dressed in the car.

Seven years later during an ordinary afternoon at work in his office, he became somewhat confused. He intended driving his secretary home. He had difficulty find- his own car. He took the wrong route. His secretary managed to get him to take her to tea and during this time he improved. The whole attack lasted 4 hours. Afterwards, he had amnesia for some of the events during this time.

Neurological examination (Dr. J. B. Lyons) was normal. Blood pressure was normal. E.E.G. was normal.

Case VI

A 67 year old man began to make strange remarks to his wife during a visit to a cemetery, on November 11th, 1967. It was not unusually cold (approx. 10°C). He was confused about the purpose of their visit to the cemetery. He became normal after a few hours. Subsequently, when seen by Dr. Harold Millar he remembered the beginning of the visit to the cemetery but could not remember anything of events during a period of 2½ hours. There were no abnormal signs. Blood pressure was normal. There was a history of intermittent claudication. E.E.G. was normal.

Case VII

A 57 years old man was referred to Dr. L. J. Hurwitz, in May, 1968, because of an episode of amnesia lasting from 5.30 p.m. to 9 p.m. He was working at stocktaking in his shop. During this time he apparently looked well but his behaviour was unusual and did not answer questions properly. There was a history of a sudden brief episode of loss of consciousness some years previously. No conclusion had been reached concerning this. Examination showed no abnormality. Blood pressure was normal.

Case VIII

A man aged 64 years was referred to Dr. L. J. Hurwitz, in March, 1968. Five weeks before the consultation he had wakened at 3.30 a.m. His wife was awake at the same time and made reference to him about events of the previous evening. He replied that he could not remember anything about this. She questioned him further and found that he had no memory of what had happened on the previous day. During this, he looked perfectly well and his behaviour was normal. His memory appeared to recover fully after one hour. He had been in good health apart from a few attacks of non-specific dizziness. Neurological examination was normal. He was obese. Blood pressure was 110/70. E.E.G. and E.C.G. showed no definite abnormalities. He has remained normal since.

Case IX

A 59 years old woman saw Dr. L. J. Hurwitz, on July 15th, 1968. She remembered breakfast time on 24/6/'68 and felt her normal self. After that she had impaired memory until 5.30 p.m. of that day. She was said to have gone to the seaside at 11 a.m. to have a swim. Coming out of the water she said to a stranger, "I don't know who I am or where I am". When asked if she had a husband she said she did not know where he was (he had been dead for a year). She was upset and crying. From 5.30 p.m. she became normal but at 7.00 p.m. she noticed double vision which persisted until the following day.

Seven years previously coming out from bathing in the sea she had had loss of memory for an hour.

Case X

A man aged 68 in May, 1968, after emerging from Church suddenly asked his wife where they were. He could not remember which church they had been to or who the clergyman was. He recovered within a few days, but 4 weeks later when seen by Dr. Hurwitz amnesia for 2 days had persisted. There were no neurological abnormalities. Blood pressure was 180/80.

Discussion

The clinical picture then is that of a middle aged or elderly person who suddenly develops a disorder of memory which lasts for a period of hours or a day or two. During this time the registration and recall of current events are impaired and afterwards there is a partial, rarely complete, amnesia for events during the confused period. There is an associated retrograde amnesia which may be for a few minutes or as far back as 10 years. With recovery, this retrograde amnesia shrinks and usually disappears. During the attacks, the subject appears healthy. He is usually distressed, but sometimes may not realise that anything is wrong. There is no loss of personal identity and complex functions like driving a car may be performed correctly. Recovery is complete. Recurrences are unusual.

Site of Lesion

This pattern of memory disturbance has been the subject of much interest for a number of years and has been reviewed by many authors, notably Whitty and Zangwill (1966) and Rose and Symonds (1960). It has been linked with many pathologies including electrical stimulation of the posterior part of the middle temporal gyrus on the right or left side (Bickford, *et al.*), epilepsy (Whitty and Zangwill), bilateral temporal lobectomy (Scoville and Milner, 1957), unilateral temporal lobectomy (Walker, 1957), head injury (Whitty and Zangwill, 1966), tumours (Williams and Pennybacker, 1954), encephalitis (Rose and Symonds, 1960) and vascular lesions (Victor, *et al.*, 1961; Whitty and Zangwill, 1966). The hippocampus, fornix and mamillary bodies are the areas most frequently demonstrated to be damaged. Where infarction has been found it has been usually in the area of supply of the posterior cerebral artery or its posterior choroidal branch. In the patients of this series there have been no features which are contrary to the view that the lesion is in the temporal lobe of the brain or its connections.

Patho-Physiology of the Lesion

Epilepsy in the patients under discussion seems very unlikely in view of the age of onset, the low tendency to recurrence, the absence at any time of any attacks clearly recognisable as epilepsy and the absence of E.E.G. abnormalities. Transient ischaemia seems most likely. If this were a correct view, then one would expect a wide spectrum of possibilities. Infarction might produce permanent memory defects. While not present in this series or those of Fisher and Adams, this has been recorded by Victor, *et al.* (1961), Glees and Griffith (1952) and Mabille and Pitres (1913). Transient ischaemia on an atherosclerotic basis might be isolated as in most of the cases. It might be related sometimes to embolism. Migraine might be operative. Moersch (1924) and Nielsen (1958) have described transitory memory defects in this condition. Nielsen suggested that the posterior cerebral

artery is at fault in such patients. Diffuse vasconstriction might result in temporal lobe ischaemia, the temporal lobe being a border area between the areas of irrigation of the three major cerebral arteries (Romanul and Abramovitch, 1964). Swimming in cold water may induce intense and sometimes uncontrollable hyperventilation (Keating, *et al.*, 1969). The hypocapnia of hyperventilation is a very powerful direct vasoconstrictor stimulus to cerebral blood vessels (Sokoloff, 1960). Cerebral blood flow falls with hypocapnia (Kety, 1948) and cerebral damage may result (Allen and Morris, 1962). Impairment of consciousness and amnesia have been recorded during the standard E.E.G. recording technique of hyperventilation for 3 minutes. Hypothermia has been found to result in decreased cerbral blood flow in anaesthetized dogs (Rosomoff and Holaday, 1954) but this seems unlikely to be a factor in the patients described who were in the water for restricted periods of time. Other responses to sudden cold such as a large acute rise in blood pressure (Ichihashi, 1936) producting hypertensive encephalopathy or hypersensitivity and collapse (Horton and Roth, 1937) also seem unlikely. Hyperventilation may have been the triggering factor in two of Fisher and Adams patients where attacks occurred during sexual intercourse.

Attacks during or after a hot bath could possibly also be triggered by hypocapnia. Hyperventilation may also occur here, corresponding to the dog panting to lose heat by evaporation. Hypotension could be an additional factor in these circumstances. A combination of these is seen in Case XI

Case XI

A Dublin school was invaded by a variant of the "fainting lark" in 1970. While leaning over and touching his toes the boy breathed in and out as rapidly and as deeply as possible ten times and then expired fully. At this point he straightened up and was seized around the chest by another boy who prevented inspiration. Fainting ensued with rapid recovery on release.

A boy aged 13 who had done this a number of times repeated it at 8.40 one evening with an assistant who was bigger and stronger than on previous occasions. On recovery from his collapse he was confused and violent and spent a disturbed night during which he did not recognise his headmaster or friends. In the morning he was normal. He could not remember events for 2 hours before the incident and had patchy memory loss for the night's events.

It might be considered that patients in whom spontaneous attacks occur would be likely to show more evidence of atherosclerosis and be older than those whose attacks were provoked by particular circumstances such as swimming or bathing or sexual intercourse. However, adding together the data from Fisher's and Adam's paper and that of Evans and the present series there was no noteworthy difference. Nine of 17 adult patients with spontaneous attacks showed evidence of vascular disease such as hypertension, cardiomegaly, auricular fibrillation or claudication and 6 of 13 adults with provoked attacks. For isolated spontaneous attacks (2 patients where migraine might have been a factor being excluded) the mean age was 64 (range 35-80). For provoked attacks the mean age was 57 (range 43-70). It cannot be said that the difference is of significance. There was no obvious difference in incidence of spontaneous attacks between summer and winter.

Wider recognition of this condition should allow closer study of cases in the acute phase. In this series of patients and the others recorded in the literature no attempt has been made to induce attacks by reproducing precipitating circumstances. As evidence accumulates that the condition is a

benign one with a very good prognosis this would seem to be not unethical in very selected patients.

When the episode has run its course to recovery, diagnosis will be obvious in some cases but in others epilepsy, hysteria, stroke and cerebral tumour may need consideration. Diagnosis may be impossible if the subject has been away from home and there are no witnesses to the details. One possible case occuring in a businessman on a visit to London and another in a clergyman who disappeared for 3 days have not been included because of lack of documentation. Unacceptable behaviour on the beach or in a shop could bring a person into legal and social suspicion and disrepute.

Summary

Eleven patients with transient global amnesia are described. There were 13 episodes. Five of these began during swimming, one during a bath. One occurred following voluntary hyperventilation. Pathophysiology is discussed.

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