THE KORSAKOV SYNDROME IN SPONTANEOUS SUBARACHNOID HAEMORRHAGE.

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THE amnestic-confabulatory symptom-complex to which the eponymous title of Korsakov's syndrome is usually given was probably first described by the Swedish physician Magnus Huss (1807–90), who spent the greater part of his lifetime in the study of alcoholism. There is, however, no doubt that Korsakov's (1890) paper on the subject gave an excellent description of the syndrome, and stressed the fact that whereas it often developed in patients with alcoholism and polyneuritis, numerous other metabolic and neurological disorders could be complicated by the characteristic mental changes. Indeed, in Korsakov's (1890) own series of cases alcoholics were in the minority. One of the neurological conditions in which the syndrome has been described significantly often is spontaneous subarachnoid haemorrhage. The first reports of the association were probably those of Flatau (1918 and 1921), and it was discussed in some detail by Goldflam (1923) and Herman (1925 and 1926). Each of these authors described the mental symptoms exhaustively but gave no actual case-histories; the first fully documented cases in the literature were the two reported by Hall (1929). Single cases have since been described by Cubitt (1930), Popow (1930) and Kulkow (1935), and in 1939 Tarachow gave an extensive review of the literature and reported an additional 3 cases. It is remarkable that since 1939 the association has received little attention, although it has been mentioned by Sands (1941) and Meadows (1951).

The purpose of this paper is to describe 6 cases of subarachnoid haemorrhage, from a series of 312 cases recently reviewed elsewhere (Walton, 1952), in which the classical features of the Korsakov syndrome developed. Other mental symptoms noted in the remaining cases will not be described, as they have been discussed previously (Walton, 1952).

CASE HISTORIES.

Case 59.

A housewife aged 66 was admitted to the Royal Victoria Infirmary under the care of Professor F. J. Nattrass on 29 November, 1947. For 20 years she had been troubled by occasional vague headaches, and 5 years before admission she had experienced an attack in which her left arm and leg became useless for about 20 minutes, but then recovered rapidly. She had also had recurrent anxiety symptoms with hypochondriasis for some years. Five days before admission, when setting the table for a meal, she suddenly clutched her head and fell on the floor, groaning and complaining of intense pain all over the head. She was taken to bed, where she vomited once and then lapsed into coma. Next morning she could be roused

and answered questions in monosyllables, though she was extremely drowsy and resented interference. She remained in this state until her admission to hospital 4 days later.

On examination on admission she was drowsy and irritable; she resented interference and refused to give a history. There was marked neck stiffness and Kernig's sign was positive. The pulse rate was 80 per minute, blood-pressure 130/80 mm. Hg, temperature 100·8° F. The optic fundi and cranial nerves were normal but there was a very slight left-sided hemiparesis. There were no other abnormal signs in the central nervous system.

Lumbar puncture was performed 6 hours after admission and revealed a deeply blood-stained fluid under high pressure; on centrifuging, the supernatant fluid was yellow. The urine contained no abnormal constituents.

The patient's state of awareness gradually improved and she became amenable and co-operative and less irritable. However, 4 days after admission it was noted that she was euphoric and disorientated for time and place; she had lost the power of recall of remote events and of retention of recent impressions. The following day she began to confabulate and described extraordinary activities in which she had been engaged, though in fact she had never left the hospital bed. Physically, improvement continued, but the mental state remained abnormal for over a month; in the second week in January, 1948, it slowly began to improve, and on 18 January she was physically well and mentally normal and was discharged from hospital.

She has remained reasonably well since the illness and now shows (July, 1952) no evidence of dementia, although she is a chronic hypochondriac and a burden to her family. She has also had two brief attacks of loss of consciousness, one in 1949 and one in 1951, which were probably epileptic. There are no abnormal signs in the central nervous system.

Case 127.

A barman aged 43 was admitted to the Royal Victoria Infirmary under the care of Professor F. J. Nattrass on 7 January, 1947. He had been perfectly well until the day of admission. While standing behind the bar in a public-house he developed a sudden intense occipital headache which rapidly became generalized; the room seemed to spin round him and he fell sideways into a chair but did not lose consciousness. Up to the time of admission 4½ hours later he vomited repeatedly. He gave no previous history of any significant illness, but he had regularly consumed large quantities of alcohol (5–6 pints of beer a day) for many years.

On examination he was drowsy, extremely irritable and resented interference, though he could answer questions and repeatedly complained of headache. There was no neck stiffness and Kernig's sign was negative. The pulse was 60 per minute, blood-pressure 150/85 mm. Hg, temperature 98° F. The optic fundi were normal and there were no abnormal physical signs in the central nervous system. A lumbar puncture 5 hours after the onset gave a deeply blood-stained fluid under increased pressure, and on centrifuging the supernatant fluid was very faintly yellow. The urine contained no abnormal constituents. He was treated with injections of morphine and paraldehyde, but almost immediately after admission it was noticed that, in addition to being drowsy, incontinent and confused, he had no memory of the onset of his illness; he was completely disorientated for place and time and began to confabulate wildly. This abnormal mental state continued; on 6 April he suddenly became unconscious and remained so for 20 minutes. On coming round he complained of severe headache and was found to have developed a left-sided hemiparesis, and a fresh subhyaloid haemorrhage was noticed in the right optic fundus. The C.S.F. contained fresh blood. Following this recurrence he improved physically but remained confused, confabulating and euphoric and was eventually transferred to Preston Hospital, North Shields, on 15 April, 1947, with his mental symptoms unchanged and still showing signs of a left hemiparesis.

After some months his mental symptoms began to abate and he was discharged home in July, 1947, still mildly confused and disorientated but no longer confabulating. He says that he remained mentally "queer" until the end of 1947, when the mental symptoms gradually got better though he has been abnormally cheerful ever since. By this time the weakness of the left arm and leg had also cleared completely. He has since worked regularly as a labourer, as a commissionaire,

and now as a cellarman. He had a vagotomy for duodenal ulcer in 1949. He is occasionally forgetful and the euphoria has persisted.

For the past 2 years he has had occasional " frightful " headaches lasting only 5 to 10 minutes and usually confined to one or other side of the forehead ; these headaches are preceded by jagged lines like " battlements " to one or other side of his field of vision, and on one occasion he seemed to go momentarily blind. When last seen on 6 October, 1951, he was cheerful and euphoric but perfectly rational ; his memory was good, there was no evidence of dementia, and no abnormal physical signs were found on examination of the central nervous system.

Case 164.

A caretaker aged 62 was admitted to the Royal Victoria Infirmary under the care of Dr. A. G. Ogilvie on 8 May, 1944. He had been quite well until two weeks before admission and there was no history of previous psychiatric disturbance ; while working in his garden he suddenly developed a severe occipital headache and began to vomit repeatedly. He became extremely drowsy but did not lose consciousness ; the headache spread from the occiput to become generalized, and was made worse by movement. The patient was admitted to a fever hospital as a suspected case of meningitis, but on admission lumbar puncture revealed that he was suffering from subarachnoid haemorrhage, since the fluid was uniformly blood-stained and on centrifuging the supernatant fluid was yellow. The following day the patient was noted to be confused, irrational and disorientated, and he had apparently lost his memory ; however, he was no longer drowsy, and his headache had abated. Over the next two weeks he improved physically, becoming free from headache and neck stiffness, but although his mental state fluctuated from day to day, at no time was he completely orientated with regard to time and place and there were periods when he confabulated wildly. In view of the mental change he was transferred to this hospital.

On admission he was mentally slow, confused and disorientated ; he had no idea of the date or of the name of the hospital and showed retrograde amnesia for several days before the ictus, although recall of remote events was fair. However, recent impressions could not be retained. He covered up these deficiencies by relating a number of fantastic activities in which he said he had been engaged during the past weeks. There was no neck stiffness, and no abnormal signs were found in the central nervous system. His temperature was 100·4° F., blood-pressure 150/90 mm. Hg. Lumbar puncture 14 days after the ictus revealed a faint homogeneous blood-staining of the fluid ; on standing, the supernatant fluid was bright yellow. The urine contained no albumen, but a trace of sugar was present. The patient slowly improved spontaneously ; after 5 days the confabulation ceased and the period of amnesia began to recede ; 10 days after admission, though somewhat slow mentally, he was perfectly rational and orientated and could remember details of the onset of his illness. He was discharged from hospital on 19 May, 1944, and continued to improve ; however, two weeks later a catastrophic recurrence of bleeding occurred at home and he died within 8 hours. No autopsy was performed.

Case 187.

A housewife aged 35 was admitted to the Royal Victoria Infirmary under the care of Dr. A. G. Ogilvie on 19 January, 1944. Apart from occasional anxiety symptoms she had been well until three weeks before admission, when she developed a sudden severe headache followed by some stiffness of the neck. After a week's rest in bed she was so much better that she got up and resumed normal activity. Seven days before admission slight headache returned in the occipital region ; it later became generalized and she returned to bed. Since then she had continued to complain of headache and her relatives had noted that she had become mentally abnormal.

On examination on admission, although she appeared bright and answered questions well at times she occasionally confabulated preposterously. There was no neck stiffness and Kernig's sign was negative ; the optic fundi were normal and there were no abnormal signs in the central nervous system. The pulse was 80 per minute, the blood-pressure 140/90 mm. Hg, temperature 99·8° F. A lumbar

puncture on the day of admission revealed a clear xanthochromic fluid under a pressure of 80 mm. of C.S.F.; the fluid contained 200 lymphocytes per c.mm. Urine examination revealed no abnormality. Lumbar puncture produced slight improvement in the patient's headache but she remained mentally abnormal, and two days after admission, in addition to retrograde amnesia, confusion, disorientation with regard to time and place and confabulation, she was noted to be suspicious, evasive and paranoid and she believed that someone in the hospital was influencing her with electricity. At times she was reasonably co-operative, but on other occasions refused to allow herself to be touched and would not take medicine.

There was no improvement in her mental state, and she was discharged to Relton Hospital on 10 February, 1944. She remained there for a month but then began to improve mentally, and over the course of a week her abnormal behaviour and mentality resolved and she gradually became clear and rational, though somewhat euphoric. She was discharged on 16 March, 1944.

For about three months she did very little, but since then has done all her own housework, including heavy washing, and has been completely free from headache or other significant symptoms. She has remained mentally normal and has had no recurrence of confusion or disorientation. She is very nervous in traffic but has always been so; from time to time she has been worried at the thought of her illness returning, but for the last two years such thoughts have not troubled her and she is now, at the age of 62, living an active normal life. When last examined (April, 1952) she showed no evidence of mental abnormality and there were no abnormal signs in the central nervous system.

Case 204.

A housewife aged 28 was admitted to the Royal Victoria Infirmary under the care of Dr. F. J. Nattrass on 13 March, 1940. She had been perfectly well until the morning of admission, and had no personal or family history of psychiatric abnormality. While getting dressed she bent down to put on her slippers and "everything suddenly went black." Shortly afterwards her husband found her lying unconscious on the bedroom floor, but within two hours she regained her senses and complained of headache and pain in the back of the neck. For 12 hours she was drowsy and confused and vomited repeatedly, but then became perfectly lucid and rational.

On examination on admission, 16 hours after the ictus, she was dazed and restless but rational and co-operative. There was moderate neck stiffness and Kernig's sign was negative. The optic fundi were normal, and there were no abnormal signs in the central nervous system. The pulse was 60 per minute, blood-pressure 125/85 mm. Hg, temperature 98° F., rising gradually to a peak of 100° F. 10 days after admission. A lumbar puncture 18 hours after the ictus revealed deeply blood-stained fluid under increased pressure, and on standing the supernatant fluid was yellow. The patient was treated with morphine injections for her headache but showed little improvement; the day after admission she was drowsy once more and remained so for a considerable period. For a week she was incontinent of urine and faeces. On 25 March, although there had been no evidence of further bleeding, she still complained of headache and neck stiffness and bilateral papilloedema was noted. It was felt that communicating hydrocephalus had developed and daily lumbar punctures were performed. This produced striking improvement, and on 31 March she was free from headache and perfectly alert and rational. On 6 April she was feeling very well and the papilloedema had subsided completely. She was discharged from hospital on 8 April with instructions to rest in bed for a further fortnight.

After three weeks in bed she gradually resumed normal activity and was symptom-free until August, 1945. She then began to have severe frontal headaches which were paroxysmal in nature, occurred every few days and lasted a few hours before passing off. On 13 October, 1945, she came into the house after shopping and was going upstairs when she suddenly screamed that she had severe pain in the head. She seemed to improve for a moment or two, but then the headache became violent and she fell unconscious.

On admission to hospital 6 hours later she was deeply comatose; there was severe neck stiffness and Kernig's sign was positive. All tendon reflexes were diminished, but there were no other abnormal signs in the central nervous system.

The pulse was 76 per minute, blood pressure 130/80 mm. Hg, temperature 99·0° F. A lumbar puncture eight hours after the onset revealed deeply blood-stained fluid under high pressure and on centrifuging the supernatant fluid was yellow. The urine contained no albumin or sugar. Within 24 hours she regained consciousness but discovered that her left eye was completely blind. There were, however, no abnormal signs in the optic fundi. The left pupil was small and fixed but there was no ptosis, and objectively the ocular movements were normal. She improved steadily at first, but on 26 October she was noticed to be confused and disorientated; she had lost all memory of the onset of her illness and she began to confabulate, telling improbable stories. This abnormal mental state persisted unchanged for a week, but suddenly disappeared overnight between 2 and 3 November, and on the latter date she was perfectly rational and co-operative. Improvement continued thereafter, although the left eye remained completely blind; on 21 November the left optic disc was noted to be pale. The patient was discharged from hospital on 23 November. After three weeks at home she resumed her household duties; she felt well in herself and was perfectly normal mentally. Soon after discharge, however, she began to have severe neuralgic pains in the left side of the face; in 1946 she was admitted to the Newcastle General Hospital under Dr. G. F. Rowbotham for surgical treatment; unfortunately compression of the left common artery produced aphasia and hemiparesis, so the artery was not tied. From that time progressive deterioration of vision in the right eye occurred, and in February, 1951, she became completely blind. Intracranial exploration was then attempted but an inoperable suprasellar aneurysm was discovered; the patient died 6 days later from a respiratory infection.

At autopsy an aneurysm 3·9 cm. in diameter was found, arising from the left internal carotid artery; it filled the suprasellar region and was compressing both optic nerves and the optic chiasm. The aneurysm was filled with firm blood-clot. Though basal structures were generally compressed there was no evidence of selective damage to the hypothalamus.

Case 213.

A miner aged 62 was admitted to the Royal Victoria Infirmary under the care of Dr. Horsley Drummond on 9 September, 1940. For several years he had been a heavy drinker, having drunk 7 to 8 pints of beer per day. On the afternoon of admission he was drinking in a public-house; after consuming just over two pints of beer he suddenly put his hand to his head and collapsed on the floor. He was comatose for 10 minutes but then came round; he became able to answer questions, but it was noted that he was confused and disorientated and had lost all memory of where he was or of what he had been doing. Furthermore he was euphoric and very talkative, he insisted that he was in China, and began to describe his recent activities in that country.

On examination on admission 6 hours later he was a plethoric man, with the complexion of the chronic beer-drinker; he was still disorientated, amnesic and confabulating, but there were brief periods when he would answer questions about his present condition quite rationally. However, retention of recent impressions was grossly impaired. He made no complaint of headache, but disliked being moved; there was marked neck stiffness and Kernig's sign was positive. There were no other abnormal signs in the central nervous system. The pulse was 80 per minute, blood-pressure 190/90 mm. Hg, temperature 99·6° F. Lumbar puncture 8 hours after the ictus revealed a deeply blood-stained fluid under a pressure of 240 mm. C.S.F.; on centrifuging, the supernatant fluid was faintly yellow. The urine contained no abnormal constituents.

The patient's mental state remained much the same for 36 hours after admission but then improved suddenly; overnight confabulation ceased, the patient became orientated and his memory returned, and 2 days after admission his mental state was entirely normal. Now, for the first time, he complained of mild generalized headache. His condition continued to improve, and he was discharged from hospital free from headache and other symptoms on 21 October, 1940.

He returned to work two months after admission and persisted in his alcoholic habits; on 30 November, 1948, a recurrence of subarachnoid haemorrhage occurred and gave rise to a right hemiplegia and dementia. Dementia with incontinence of

urine and faeces persisted and the patient was admitted to the geriatric unit, Newcastle General Hospital, where he died from a further attack of subarachnoid haemorrhage on 22 August, 1949. No autopsy was performed.

Discussion.

1. *Incidence.*

Goldflam (1923) remarked that features of Korsakov's syndrome were noted in practically all of his 13 cases of subarachnoid haemorrhage. While it is true that variable degrees of confusion are a common feature of this complaint, Goldflam's (1923) suggestion certainly gave an exaggerated idea of the incidence of the fully-developed Korsakov syndrome in such cases. Tarachow (1939) recognized 3 cases in a series of 105 patients with subarachnoid haemorrhage, an incidence of 3 per cent., but the absence of any mention of the syndrome in the large series of cases of subarachnoid bleeding reported by Magee (1943), Wolf, Goodell and Wolff (1945) and Ask-Upmark and Ingvar (1950) suggested that it might be even less common. Indeed, Richardson and Hyland (1941) and Meadows (1951) gave the impression that it was a very rare complication. Information derived from the Newcastle series does not confirm this view, as the 6 cases here described were noted in a series of 312 cases, an incidence of 2 per cent. Furthermore, several other patients showed some of the classical features of the syndrome, but the recorded information was not sufficiently complete for them to be included here. A paranoid reaction like that which occurred in case 187 was noted in another case, but this cannot be considered a typical feature of the Korsakov syndrome; similar mental changes occurred in the case described by Silverman (1949).

A considerable number of other patients showed variable degrees of confusion and more careful observation of their mental state might have revealed some of the characteristic features. It is probable, therefore, that the amnestic-confabulatory syndrome occurs more often in subarachnoid haemorrhage than is generally realized, and failure to recognize its less distinctive features may have led to a mistaken impression of its rarity. Bleuler (1951) has pointed out that mental changes of this type, if sought carefully, are present in some degree in a considerable proportion of patients with many types of organic cerebral disease.

Tarachow's (1939) 3 cases were all female and aged 15, 33 and 59 years respectively. Three cases of the present series were male, aged respectively 43, 62 and 62 years, and 3 female, aged 66, 55 and 34 years respectively. It has been shown elsewhere (Walton, 1952) that subarachnoid haemorrhage occurs equally in males and females, and is most common between the ages of 40 and 60 years. Although the number of cases reported is insufficient for a statistical analysis, there is no reason to suggest that the age and sex incidence of cases complicated by the Korsakov syndrome is at all unusual.

In Tarachow's (1939) 3 cases the syndrome developed 5 days, 2 months and 4 weeks respectively after onset of haemorrhage; in the 6 Newcastle cases the respective intervals were 9 days, 6 hours, 24 hours, 2 weeks, 13 days and 10 minutes before the mental changes were noted. Although it is therefore true to say that in many cases there is an appreciable latent period after the ictus,

this is by no means invariable, and mental changes may sometimes be evident almost immediately.

2. *Prognosis.*

The mental abnormality persisted for 5 weeks, 11 months, 3 weeks, 10 weeks, one week and 36 hours respectively in the cases reported here. Recovery took place in all, although one patient has been euphoric ever since, and another was rather slow mentally for a few weeks before he died of a recurrence of bleeding. Recovery was slow in 4 cases, but in 2 the mental state suddenly became normal overnight; a sudden change of this type has been mentioned by Tarachow (1939). The bearing which these facts may have upon the pathogenesis will be discussed shortly.

3. *Pathogenesis.*

The aetiology of Korsakov's syndrome has aroused considerable speculation. Firstly, there seems to be little doubt that it may result from a variety of metabolic disturbances. Korsakov (1890) described its occurrence in cases of intrauterine infection, puerperal septicaemia, faecal obstruction, typhus, tuberculosis, diabetes, jaundice, Hodgkin's disease, and in poisoning with ergot, arsenic, carbon disulphide and carbon monoxide as well as in alcoholism with polyneuritis. Other conditions since implicated in the aetiology include intractable vomiting (Vermelin and Louijot, 1936), toxaemia of pregnancy (Ely, 1922; Brocklehurst, 1934), pernicious anaemia (Pickett, 1904; Barrett, 1913; Parfitt, 1934) and diabetes mellitus (Sittig, 1912; Klemperer and Weissman, 1930), while it has also occurred post-operatively (Kleist, 1916). It may be argued that deficiency of the vitamins of the B complex, due either to inadequate intake or impaired absorption or utilization (as in heavy metal poisoning), may be the common aetiological factor in this group.

No such deficiency can be adduced as a rule, however, to explain the development of the syndrome in a variety of neurological disorders, including encephalitis lethargica (Climenko, 1920; Kirby and Davis, 1921) and other forms of encephalitis (Davis, 1929), cerebrospinal meningitis (Aronov, 1928), neurosyphilis (Roemheld, 1906), head injury (Meyer, 1904), tumours of the third ventricle, including colloid cysts (Meyer, 1899; Foerster and Gagel, 1933; Kelly, 1951), and tumours of the cerebral hemispheres (Bleuler, 1951). However, the fact that 2 of the 3 chronic alcoholic patients in the 312 Newcastle cases (Walton, 1952) developed the syndrome suggests that even when it is due to a neurological disturbance, alcoholism may be a predisposing factor.

The mechanism by which these varied neurological conditions may all give rise to a similar psychiatric syndrome is still obscure. An early suggestion was that it resulted from damage to the cerebral cortex, but this explanation would not fit all the known facts, and in Cubitt's (1930) case, as in many others, the cortex was normal at autopsy. There is certainly no evidence that a localized cortical lesion, say in the frontal lobe, can be the cause; the absence of clinical features of focal cerebral damage in the 6 Newcastle cases may be added to the already abundant evidence which confirms this suggestion. Froin (1904) felt

that toxic absorption was responsible, and Carmichael and Stern (1931) also blamed the haemolysed blood in the subarachnoid space. It is now clear that this cannot be the responsible factor, since although there is often a latent period before the mental changes appear, sometimes, as in Case 213 of the present series, they may develop immediately after the ictus, before haemolysis can have begun. Furthermore, this hypothesis does not aid in the explanation of the association with other neurological disorders in which no bleeding occurs. It also seems unlikely that psychological factors are important in the aetiology; certainly none of the Newcastle cases had any personal or family history of psychiatric illness.

A popular view is that hydrocephalus is responsible for the association (Tarachow, 1939; Kelly, 1951). Tarachow (1939) explains the latent period which is often evident in cases of subarachnoid haemorrhage by suggesting that the hydrocephalus may be of the communicating variety due to a plastic meningitis as a result of irritation of the meninges by the effused blood. It may be suggested that in the cases where the syndrome appears soon after the ictus an acute hydrocephalus, due to severe bleeding, is responsible. Although this hypothesis has the virtue that it can be adduced to explain the appearance of the syndrome in other cerebral disorders in addition to subarachnoid haemorrhage, it is remarkable that papilloedema was noted in none of the Newcastle cases when the syndrome was at its height. It has been shown elsewhere (Walton, 1952) that papilloedema in subarachnoid haemorrhage may occur early due to severe bleeding, within a few days due to intracerebral haemorrhage, or late due to communicating hydrocephalus. Case 204 showed clear evidence of communicating hydrocephalus in a previous attack of subarachnoid haemorrhage but remained mentally normal; in a second attack of bleeding 5 years later Korsakov's syndrome developed, though there was no evidence of any undue degree of hydrocephalus. Nor was there any evidence of mental changes of this type in the cases of communicating hydrocephalus following subarachnoid bleeding described by Krayenbuhl and Luthy (1948) and Pluvinage (1949), and increased pressure could not have been responsible in cases of neurosyphilis (Roemheld, 1906) or encephalitis lethargica (Kirby and Davis, 1921). Hence it seems unlikely that hydrocephalus *per se* is responsible for the mental syndrome, although the cause may lie in the effects which it and other cerebral lesions have upon the brain.

There now appears to be two main schools of thought on the aetiology of the syndrome. Gamper (1928), Meyer (1944) and others have suggested that it usually results from a lesion in or near the hypothalamus, and particularly in the mammillary bodies; certainly Meyer's (1944) case supports this suggestion. Bleuler (1951) on the other hand, has claimed that it may result from any diffuse and chronic cerebral process; it was present in some degree in 38 per cent. of a series of 600 cases of cerebral tumour seen in his clinic and the situation of the tumour was immaterial. However, as Roth (1952) has pointed out, it is possible to suggest that pressure upon the diencephalon was a factor common to all of Bleuler's (1951) cases. Unfortunately the absence of histological evidence in the Newcastle cases does not allow a firm opinion to be given in favour of one opinion or the other. The only case to come to autopsy

had shown the manifestations of the Korsakov syndrome 7 years previously and at autopsy the entire hypothalamic area was distorted and compressed by an aneurysm, although there was no evidence of recent mental disturbance. Certain clinical pointers do exist, however, which may be of some value in deciding in favour of one of the two hypotheses. It has been shown elsewhere (Walton, 1952) that signs developing as a result of hypothalamic irritation are a relatively common feature of cases of subarachnoid haemorrhage; these include high fever, hyperpiesis, albuminuria and glycosuria and haematemesis. Any one or a combination of these features could be present in an individual case, and usually indicated that bleeding had been severe. If the Korsakov syndrome were invariably due to a hypothalamic lesion it is difficult to understand why it is not more common in conscious patients showing some of the above features; further, apart from slight glycosuria in one, none of the 6 cases reported here showed any other evidence of hypothalamic irritation. It is also difficult, on this basis, to explain the latent period between the ictus and the mental changes in patients who were showing physical improvement, with no evidence of hydrocephalus or other neurological signs. In addition, if the hypothesis is correct, why do some cases recover suddenly, and why is recovery from the syndrome invariable in cases of subarachnoid haemorrhage?

4. *Conclusions.*

The available evidence suggests that the Korsakov syndrome in cases of subarachnoid haemorrhage is usually a reaction to diffuse cerebral damage. This supports Bleuler's (1951) view on the aetiology of the syndrome, and there is no positive evidence to suggest that a lesion in the hypothalamic region is responsible. Even if this view is accepted it still cannot be explained why the syndrome is relatively so uncommon; probably it is particularly likely to occur in an alcoholic patient who develops subarachnoid haemorrhage, but this is not a very frequent event. It is likely that minimal evidence of the typical mental changes will be found more often in cases of subarachnoid bleeding if looked for carefully.

Summary.

Six cases of Korsakov's syndrome developing as a result of subarachnoid haemorrhage are reported with reference to the relevant literature. They were noted in a survey of 312 cases of spontaneous subarachnoid bleeding, an incidence of 2 per cent.; it is probable that the syndrome occurred in less overt form in many more cases.

Three cases were male, 3 female, and the patients were of ages at which subarachnoid haemorrhage commonly occurs. Two patients gave a history of alcoholism. In one case the mental abnormality was noted immediately after the ictus, in 2 others it appeared within 24 hours, and in the others it was delayed for between 9 and 14 days. Mental changes usually persisted for a few weeks, although in one case they lasted only 36 hours and in another 11 months. In all cases the patients recovered mentally, although one patient has been euphoric ever since. Recovery was gradual in 4 cases, abrupt in 2.

The pathogenesis of the mental changes is discussed; it is concluded that in these cases there was no positive evidence of a hypothalamic lesion. It seems more likely that the syndrome developed as a reaction to diffuse cerebral damage. If may be particularly likely to occur in alcoholic patients who develop subarachnoid haemorrhage, and will probably be recognized in an increasing proportion of cases of subarachnoid haemorrhage if minimal mental changes are sought with care.

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