OBSERVATIONS ON THE CEREBROSPINAL FLUID IN CLOSED HEAD INJURIES

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These observations are based on the study of 85 patients who were admitted to an E.M.S. head injury centre within a few days of an injury from which no open wound of the skull had resulted. Most of the patients were involved in motor-cycle or lorry accidents, but some were air-raid casualties who had been thrown to the ground or hit by flying masonry or metal fragments. In all these cases a lumbar puncture was performed shortly after they were admitted, and in some of them repeated punctures were carried out.

Lumbar Puncture Pressure

The cases have been divided into three groups, of low, normal, and high pressure, the normal pressure being taken as 90 to 180 mm. of water. In 42 the pressure was within normal limits, in 28 it was above 180 mm., and in 15 it was below 90 mm. An attempt has been made to correlate the pressure with features of the clinical state.

TABLE I

State of Consciousness	Lumbar Puncture Pressure		
	0-90 mm.	90-180 mm.	Over 180 mm.
Full consciousness	11	27	9
Drowsy	2	8	11
Restless stupor	1	5	6
Coma	1	2	2
Total	15	42	28

These findings were recorded at the time of the first lumbar puncture; in many cases this was done within twenty-four to forty-eight hours of injury, but in others, owing to delay in admission to a head injury centre, it was not carried out for seven to ten days. It will be seen from Table I that the pressure of the cerebrospinal fluid in the lumbar sac bears little relation to the patient's state of consciousness: deep coma can be present with a normal or low pressure as well as with a high one. It is, however, true that drowsiness is a frequent symptom in patients with a high pressure, though it is not confined to them.

TABLE II

Condition	Lumbar Puncture Pressure		
	0-90 mm.	90-180 mm.	Over 180 mm.
No headache	11	24	1
Moderate headache	2	8	10
Severe headache	0	3	9
Stupor or coma	2	7	8

In the acute stages of a head injury the lumbar puncture pressure bears a closer relation to the degree of headache present than to the state of consciousness. It will be seen from Table II that only one patient with a high pressure

was free of pain. He was a soldier admitted six days after a motor-cycle accident from which he had been unconscious for five days, and though his lumbar puncture pressure was 300 mm. he showed no symptoms. In all the other patients with high pressure who were conscious headache was complained of, and in 9 of them it was severe and not necessarily accompanied by blood in the C.S.F. In the low-pressure group bad headache was not a feature, but 3 of the patients with normal pressures had severe pain.

The same relation does not appear to hold in patients with late post-traumatic headache, in whom the lumbar puncture pressure is usually within normal limits but occasionally low.

Pulse Rate

As a slow pulse rate may be found soon after a closed head injury with any degree of C.S.F. pressure it cannot be taken as a reliable index of a raised intracranial pressure. Further, the pulse rate may remain slow when the pressure is rapidly reduced. The presence of a slow pulse rate with a normal C.S.F. pressure in head injuries has been remarked on by Denny-Brown (1941), and has been ascribed by him to medullary contusion. This explanation would be compatible with the persistence of a slow rate in spite of rapid reduction of intracranial pressure.

Reduction of Increased Pressure

Patients with a raised C.S.F. pressure who have had only small quantities of blood in the fluid have been treated by dehydration by means of hypertonic enemata of 6 oz. of 50% magnesium sulphate administered twice daily. In most cases this method has succeeded in reducing the pressure and relieving symptoms. In a few patients severe headache has continued and the pressure has remained high in spite of this treatment, and to them 40 c.cm. of five-times-concentrated serum has been given intravenously. This treatment has usually resulted in a fall of pressure and often a dramatic relief of headache. An account of this group of cases is in preparation.

As a rule, in uncomplicated cases the clinical condition improves as the C.S.F. pressure falls, but in one case of closed injury in which the clinical condition remained stationary, though the C.S.F. pressure fell, an extradural haematoma was found at necropsy.

This patient was a soldier, aged 20, who was admitted five days after an injury of which no details were available. He had been admitted to another hospital, where he was found to be in a semi-comatose state with right-sided spasticity, but no pupillary or reflex changes. When seen by me he was in a restless stupor, and had a small sutured scalp wound in the left frontal region; there was slight weakness of the right side of

the face and right arm and leg, all the tendon jerks and the abdominal reflexes were absent, the plantars were flexor, and the fundi and pupils were normal. Radiographs of the skull showed no fracture, and lumbar puncture pressure was 260 mm., the fluid containing 3 cells and 50 mg. of protein per 100 c.cm. As his condition remained stationary a left parietal trephine hole was made, but no haematoma was found. The patient's clinical condition remained much the same as on admission, but the lumbar puncture pressure thirteen days after the injury was 70 mm. and twenty-one days after was 10 mm., the fluid still being normal. He died on the twenty-third day, and at the post-mortem examination a large extradural haematoma containing solid blood clot was found low down in the left temporal region; this had arisen from a torn anterior branch of the middle meningeal artery.

Similarly, in penetrating injuries a space-occupying lesion needing operation may be present though the C.S.F. pressure is falling, as in the following case.

A soldier with a penetrating wound of the left parietal region was admitted in coma with a right hemiparesis on the day on which he was wounded by a bomb fragment. The small external wound had been sutured before admission. His lumbar puncture pressure was 310 mm. and the fluid was deeply blood-stained. In the course of the next twelve days there was gradual improvement and he passed into a restless stupor, the lumbar puncture pressure gradually falling to 140 mm. On the twelfth day the parietal lobe was explored and an intracerebral haematoma was evacuated from it. This was followed by rapid improvement in his condition.

These cases emphasize that in spite of a progressively falling C.S.F. pressure there may still be present a compressive lesion which needs operation, and this is especially likely if the clinical condition does not improve fairly rapidly as the pressure falls.

Blood in the C.S.F.

Blood in moderate quantity may be present after what appear to be comparatively slight injuries which are followed by only a few seconds' post-traumatic amnesia or even no loss of consciousness (cf. McKissock and Browncombe, 1941). The presence of blood in any considerable quantity is accompanied by symptoms, especially headache, neck rigidity, and a positive Kernig sign; but Ritchie Russell's (1932) suggestion that a patient with over 100,000 red cells per c.mm. in the C.S.F. is stuporous or comatose has been found to be not always true.

A man aged 46 had fallen off a ladder on the day previous to admission, striking the back of his head on the ground; he had been unconscious for about three hours. When seen thirty hours after the accident he was drowsy but conscious and co-operative, and there was marked neck rigidity. Lumbar puncture gave a pressure of 270 mm., and the C.S.F. resembled pure blood, except that there was no tendency to clot. Cell counts showed over four million red blood cells per c.mm. and a protein content of 2 grammes %. On the following day, when the count was still over 100,000 red cells per c.mm., the patient was conscious but drowsy.

It is difficult to account for the remarkably high red cell count in this case unless there was gravitation of blood to the lumbar theca.

It is probable that the state of consciousness is not related directly to the quantity of blood in the C.S.F.: it depends rather on the site of origin of that blood. If it comes from cortical lacerations it is likely that there is extensive cerebral damage which will be accompanied by coma or stupor; if it comes from an injured venous sinus there may be profuse bleeding with little cerebral damage, and in these cases unconsciousness may not be present. This latter type of injury probably occurred in the above case, as there was never any further evidence of cerebral damage after the first loss of consciousness, and there was a long crack in the occipital bone.

In cases in which there is blood in the C.S.F. it is important to culture the fluid and to do a differential white cell count on it, as it is possible to ascribe to subarachnoid bleeding the fever and meningeal signs which are usually present, when in reality there is an infective meningitis as well as a chemical one from the presence of blood. This is illustrated by the following case.

This patient, a soldier, was admitted to hospital four days after a motor-cycle accident in France; details of his condition in the first four days were lacking. On admission he was drowsy, had a severe headache, presented meningeal signs, and had a left lower facial weakness. The C.S.F. was under a pressure of 240 mm. and was heavily blood-stained; haemoglobin amounted to 16%, and the supernatant fluid was yellow. During the next three days he became more deeply unconscious, and as he was thought to be bleeding a right subtemporal exploration was performed and a burr-hole made in the left subtemporal region, but no haematoma was found. At post-mortem examination two days later there was seen to be a fracture of the anterior fossa with a purulent meningitis spreading from the posterior wall of the frontal sinus, which was involved in the fracture.

If a culture and a differential white cell count had been made on this man's fluid it is probable that the purulent meningitis would have been recognized; as it was, the meningeal signs were ascribed to subarachnoid bleeding.

Disappearance of Blood from the C.S.F.

Red blood cells usually disappear from the fluid in four to five days; even in the case with over four million cells in the fluid no red cells were found on the fifth day. Occasionally they may persist for considerably longer periods after a closed injury, and it may be assumed that in these cases subarachnoid bleeding has continued for some days. For example, a soldier was admitted eight days after a motor accident in a restless confused state; the C.S.F. contained 100,000 red cells and was under a pressure of 310 mm. It was not until fourteen days after the injury that this man's fluid became free of red cells.

As the red cells disappear it is common for an excess of lymphocytes to persist in the C.S.F., 10 to 20 cells per c.mm. being a common finding. This increase of lymphocytes may last for three to four weeks after severe bleeding: it is usually found even when the yellow coloration of the fluid has disappeared. The presence of more than 2 or 3 polymorphs per c.mm. is unusual after a closed injury unless there is infection.

Protein Content of C.S.F.

The protein content of the C.S.F. shortly after an injury seems to depend on the amount of blood the fluid contains, and it usually falls as the blood disappears, but in the majority of cases in which there has been much bleeding the fall in the protein content is slower than the disappearance of the red cells. Occasionally there is a small rise of 20 to 40 mg. of protein per 100 c.cm. as the red cell count is falling. It seems possible that this is due to corpuscular protein set free by haemolysis, as in the ordinary protein estimation the amount present in the corpuscles is not included, because they are centrifuged off before doing the estimation.

In a few cases the C.S.F. protein remained higher than normal for several weeks after an injury. This does not seem to be related to the severity of the injury, as we have seen 100 to 120 mg. of protein per 100 c.cm. three to four weeks after comparatively minor injuries which were followed by only half an hour to one hour of post-traumatic amnesia. The significance of this continued raised protein content of the C.S.F. is at present undetermined, as the cases do not differ clinically from those in which the protein content has fallen fairly rapidly to normal.

Summary

The pressure and constitution of the C.S.F. have been studied in relation to the clinical condition in 85 cases of recent closed head injury.

The C.S.F. pressure bears little relation to the state of consciousness, but in conscious patients a raised pressure is usually accompanied by headache. The C.S.F. pressure may fall steadily even when a space-occupying lesion is present.

The degree of consciousness is not related directly to the amount of blood present in the C.S.F.; it depends rather on the site of origin of the blood.

The importance of a differential white cell count and a culture on a blood-stained C.S.F. is emphasized. If these are not done the presence of subarachnoid bleeding may mask an infective meningitis.

A lymphocytosis in the C.S.F. may be present for three to four weeks after a severe head injury.

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"NIGHT-BLINDNESS"—A PSYCHO-PHYSIOLOGICAL STUDY

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Night-blindness has been defined as a disturbance of light perception resulting in a difficulty and occasionally an inability to adapt the faculty of vision to very faint illumination (Jeghers, 1937). Figures as to its incidence in the average population under peacetime conditions are not available. Minor degrees of it are fairly common (Maitra and Harris, 1937; Jeans and Zentmire, 1934, 1936; Schuck and Miller, 1938). It is said to be commoner in the under-nourished poorer classes than in the well-fed well-to-do classes (Jeghers). During the war of 1914-18 numerous cases of night-blindness were reported in the armies of the various belligerent countries:

French Army: Bourdier (1916), Truc (1916), Bussy (1916-17), Frenkel (1916-17), Aubaret (1917), Landolt (1917). Belgian Army: Weekers (1916), Danis (1918). Rumanian Army: Savo Goiu (1922). Serbian Army: Néchitch (1918). British Army: Smith (1921), Derby (1921). American Army: De Schweinitz (1921). German Army: Augstein (1915), Best (1915), Braunschweig (1915), Feilchenfeld (1915), Paul (1915), Wessely (1915), Wietfeldt (1915), Zade (1915), Birch-Hirschfeld (1916), Elschmig (1916), Gelenczér (1916), Junius (1916), Löhlein (1916), Rauch (1916), Uhthoff (1916), Wolffberg (1916), Hübener (1917), Koeppe (1917), Wohlfarth (1917), Hillemanns (1918), Hölscher (1918), Meyer (1918), Sattler (1918), Schieck (1918), Wiese (1918), Jess (1921).

Towards the end of that war cases of night-blindness were seen in increasing numbers in the German Army, and

the problem is again presenting itself there (Fillat, 1940; Hofe and Glees, 1940). Its significance for both the combatant and the non-combatant arms of the Services is obvious. It impairs the efficiency of the soldier: sufferers from this malady are unfit for night guards, night driving, and night fighting. In the darkness they are likely to inflict serious injury on themselves or to damage Army property.

Apart from congenital and hereditary night-blindness various views on the aetiology of this disorder have been put forward. It has been attributed to: (1) diseases of the eye (advanced myopia, choroidoretinitis, retinal detachment, optic neuritis, glaucoma, etc.); (2) overexposure to strong light and glare; (3) defective nutrition; (4) toxæmia (diseases of the liver, alcoholism); and (5) great fatigue. The most commonly accepted and, indeed, widely advertised view is that it is due to vitamin A deficiency (Jeghers, 1937; Sweet and K'ang, Frandsen, 1935). This view is regarded as so well established that dark-adaptation tests have been used as a criterion and a quantitative indicator of vitamin A deficiency. Many authors state that patients with night-blindness are often found to be nervous, irritable, and depressed (Duke-Elder, 1932; Elschmig, 1916; Park, 1935; Paul, 1915; Weekers, 1916; etc.). A few writers believe that every night-blind patient is a malingerer (Derby, 1921; Feilchenfeld, 1915; Smith, 1921), a view which has been emphatically repudiated by others (Fillat, 1940). Most authors have recognized and separated a group of cases labelled as nervous or hysterical night-blindness. The neurotic implications of night-blindness have been discussed by Culpin (1940), but so far as is known to us no attempt at a systematic study of the personalities concerned has as yet been undertaken. Everyday experience shows that anxious individuals object more than others to going out in the black-out. Coming out into the street, they cling like frightened children to others and ask for their support. These considerations and the idea that anxiety may be related in some way to dark adaptation prompted us to undertake the following study.

Material: Method of Examination

Biographical studies were made of 52 soldiers complaining of night-blindness who either were sent to us without selection by the eye department or were referred direct to us because of psychological disorders. Their ages at the time of examination were from 19 to 50; 39 of them were under 30 (9 had suffered from night-blindness since childhood, 16 for several years; 13 noted difficulty in seeing in the dark for the first time when the black-out started, and 14 in the course of the war). Four were Regulars, 5 Territorials, 12 volunteers, and 31 conscripts; 43 were privates, 8 N.C.O.s, and 1 an officer. The patients, so far as was possible, were examined ophthalmologically by Major Scott, eye specialist at a military hospital, and were tested for their dark adaptation by Dr. Semeonoff of the psychological department, University of Edinburgh.

Findings

Symptomatology.—All the patients examined suffered from severe night-blindness and stated that they were unable to find their way about in the dark without help from others; they often gave a dramatic description of how, with their arms stretched out in front of them, they groped their way in the dark or clung to their wives, mothers, or comrades. Several of them, unable to see in the dark, had met with serious accidents or had damaged Army or Air Force property—e.g., vehicles.

In association with their night-blindness they often complained of other eye symptoms, such as photophobia, blurring of sight, black spots or floating blobs in front of the eyes, aching or beating sensation in the eyeballs, quivering