SOME OBSERVATIONS ON THE CEREBROSPINAL FLUID IN CLOSED HEAD INJURIES

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SINCE the introduction of lumbar puncture as a therapeutic and diagnostic procedure in acute head injuries by Quincke in 1904, much has been written on the subject, particularly in regard to the indications and contra-indications for the procedure. Nevertheless, whilst the alteration in pressure, cytology and chemistry of the cerebrospinal fluid occurring in the acute state following a head injury has already been frequently discussed (usually in terms of clinical impressions) in the literature, there still seems to be room for further factual studies of the resultant changes on the lines notably adopted by Ritchie Russell (1932); particularly as these changes naturally have an important bearing on the treatment of acute head injuries, on which there are not a few conflicting views at the present time.

This investigation was undertaken, therefore, with a view to assessing the actual changes in cerebrospinal fluid pressure, the frequency and the degree of subarachnoid bleeding demonstrable by early lumbar puncture in acute head injuries, as well as the relation of these changes to such factors as severity of injury and the resultant alteration in the mental state.

Method

300 cases with head injuries on which lumbar punctures had been performed at a relatively early stage were reviewed for this communication, the procedure adopted being substantially the same in each case. Lumbar puncture was performed under local anæsthesia, the patient being placed in the usual lateral position, the head being level with the lumbar spine. After adequate relaxation of the patient had been obtained, the initial cerebrospinal fluid pressure was carefully measured with a manometer, before cerebrospinal fluid was collected for examination.

These cases were selected in that open injuries with dural penetration were excluded, as also were cases complicated by extradural hæmatomas, subdural hæmatomas (when diagnosed and operated on) or by infective meningitis.

Results

The Cerebrospinal Fluid Pressure.—Table I was constructed from the pressure readings obtained on

the occasion of the initial lumbar puncture in the first week after injury in 235 cases. These cases were grouped in accordance with the Post Traumatic Amnesia in the belief that the latter is as accurate a guide to the severity of a head injury as any other single factor, and the height of the Initial Pressure. It will be noticed that 27 of the 235 cases had an initial cerebrospinal fluid pressure of 250 mm. or over, of these only 11 gave a reading of 300 mm. or more. The highest pressure recorded in this series was 370 mm. In two-thirds of the cases the cerebrospinal fluid pressure was normal.

TABLE I.—THE INITIAL PRESSURE WITHIN 7 DAYS OF INJURY—235 CASES

Post-Traumatic Amnesia	Pressure			Total
	0-180 mm.	180-250 mm.	250 mm. and over	
Under 24 hours	% 70	% 24	% 6	Cases 179
1-7 days ..	56	25	19	32
Over 7 days ..	29	29	42	24
Total	150 cases	58 cases	27 cases	235 cases

For the purpose of this review the upper limit of normality was taken to be a pressure of 180 mm. of water. This figure seems to be accepted by most authorities, though some would put the limit at a lower level such as 150 mm. Merritt and Fremont-Smith (1937) after investigation of the lumbar cerebrospinal fluid pressure in 1033 patients having a normal blood pressure and showing no evidence of an inflammatory or expanding lesion in the brain concluded that the normal pressure ranged between 70 and 180 mm. of water.

Attention is drawn to the percentage distribution of the cases in the three amnesia classes. There is a significant increase in the proportion of cases with

raised pressure in the more severe injuries, though it should be noted that even when that injury was severe (post-traumatic amnesia over 7 days) in 29 per cent. of the 24 cases the initial pressure was not above normal.

TABLE II.—THE MEAN CEREBROSPINAL FLUID PRESSURE AT INITIAL PUNCTURE IN 300 CASES

Post-traumatic amnesia	0-7 days after injury	7-21 days after injury
Under 24 hrs. (222 cases)	159 mm. (184 cases)	150 mm. (38 cases)
1-7 days (42 cases)	191 mm. (31 cases)	133 mm. (11 cases)
Over 7 days (36 cases)	225 mm. (22 cases)	144 mm. (14 cases)

This table represents an attempt at demonstrating the natural rate of return of the cerebrospinal fluid pressure to normal levels after a closed injury to the head. The pressure at initial puncture in 300 cases was utilized, the mean pressures being plotted after the cases had been grouped in their respective amnesia classes, and in accordance with the interval between injury and initial puncture. It will be seen that though the mean initial pressure within one week of injury rises in proportion to the severity of that injury, in all three amnesia classes it falls within normal limits after the first seven days. In other words the cerebrospinal fluid pressure will return to normal levels within one week of injury, that is to say below a pressure of 180 mm. in the average case without repeated lumbar punctures.

TABLE III.—THE MEAN RED CELL COUNT IN THE CEREBROSPINAL FLUID AT INITIAL LUMBAR PUNCTURE—120 CASES.

Days after injury..	0-2 days	3-6 days	7-15 days
No. of cases ..	70	35	15
Mean C.S.F. red cell count	65,600	9,600	600

Blood in the Cerebrospinal Fluid Following Closed Head Injuries.—Initial puncture revealed the presence of more than 100 red cells per cu. mm. in 120 of the 300 cases under consideration. A count of less than 100 was disregarded in order to allow an ample margin for the possibility of contamination of the fluid with red cells at the time of withdrawal by the lumbar route. This table demonstrates the mean red cell count at initial puncture in relation to the interval between puncture and injury, and appears to indicate that clearance of red cells from the cerebro-spinal fluid by natural means proceeds very rapidly. On the other hand, in cases submitted to repeated lumbar puncture, of 39 with blood in the cerebrospinal fluid at initial puncture, 32 were free from blood at the end of the first week after injury; but of these, in only 5 cases was the result achieved by the fourth day after injury. Thus it

would seem that repeated punctures do not appear to speed up the natural rate of clearance of blood from the cerebrospinal fluid to an appreciable degree.

The occurrence of further delayed subarachnoid hæmorrhages in the first and second weeks following injury and occasionally at an even later date is an interesting complication which is difficult to explain. In several of the cases under review such hæmorrhages were noted, and were accompanied by symptoms such as neck rigidity, headache, and even by a marked rise in the cerebrospinal fluid pressure. Minor delayed hæmorrhages unaccompanied by clinical manifestations might well occur with greater frequency, and these may only be recognized if repeated punctures be performed. When the results of repeated puncture were analyzed it was found that in no less than 16 out of 42 cases secondary increments in the red cell count in the fluid took place, usually within one week of injury, but how frequently these increments are attributable to further hæmorrhages cannot be assessed, as it should be emphasized that too much importance must not be attached to the actual red cell content of the lumbar fluid. Apart from the factor of sedimentation of the red cells into the lumbar sac which may well occur if the head be at a higher level than the lumbar spine, a discrepancy may possibly result from partial occlusion of the Incisura Tentorii with blood clot, preventing to a certain extent the escape of blood from the cortical subarachnoid spaces into the spinal theca; a mechanism which has been invoked to explain the rare case of clear blood-free cerebrospinal fluid obtained on early puncture of a proven case of spontaneous subarachnoid hæmorrhage from a ruptured aneurism (Strauss, Globus, and Ginsburg, 1932). These and other factors might with advantage be born in mind when one is tempted to consider a moderate increase in red cells in the lumbar fluid as indicating the occurrence of a fresh subarachnoid hæmorrhage. It should be noted too, that in the opinion of many authorities it is unlikely that repeated punctures cause a recrudescence of subarachnoid hæmorrhage (McKissock and Brownscome, 1941).

When the frequency with which blood is found in the cerebrospinal fluid at initial puncture is considered in relation to the post traumatic amnesia (Table IV) it is seen that clear blood-free cerebrospinal fluid is but rarely found in cases with a post-traumatic amnesia of over 24 hours, and of 53 such cases only 4 were free from blood.

In 105 cases punctured for the first time within 7 days of injury the cerebrospinal fluid red cell count was compared with the initial lumbar cerebrospinal fluid pressure (see Table V). If these figures are significant it would seem that there is little relation between the two factors except when considerable bleeding has taken place into the cerebrospinal fluid spaces. When this occurs raised pressures are to be expected in the majority of cases, though it would seem that the association is by no means the rule.

TABLE IV.—THE FREQUENCY WITH WHICH BLOOD IS FOUND IN THE CEREBROSPINAL FLUID AT INITIAL PUNCTURE WITHIN 7 DAYS OF INJURY—240 CASES

Post-traumatic amnesia	Blood in C.S.F. (Over 100 cells per cu. mm.)	No blood in C.S.F.
Under 24 hours	73 cases	114 cases
1-7 days	28 cases	2 cases
Over 7 days ..	21 cases	2 cases

TABLE V.—THE CEREBROSPINAL FLUID PRESSURE AT INITIAL PUNCTURE WITHIN 7 DAYS OF INJURY IN 105 CASES WITH BLOOD IN THE FLUID.

Red cells in the C.S.F. (per cu. mm.)	Pressure			Total
	0-180 mm.	180-250 mm.	250 mm. and over	
100-1,000 ..	22	6	8	Cases 36
1,000-10,000 ..	16	5	3	24
10,000-100,000 ..	16	11	5	32
Over 100,000 ..	4	3	6	13

The Mental State in Relation to the Cerebrospinal Fluid Pressure and Red Cell Count.—In 192 cases with closed head injuries the cerebrospinal fluid pressure as obtained at initial puncture was tabulated with the mental state of the patient at that time. To simplify the data so tabulated all cases who were comatose, stuporose, or who showed definite drowsiness were classed as being "not alert." The results are shown in Table VI.

TABLE VI.—THE MENTAL STATE IN RELATION TO THE CEREBROSPINAL FLUID PRESSURE AT INITIAL PUNCTURE IN 192 CASES

Pressure	Alert	Not alert	Total
0-180 mm. ..	98	27	125 cases
180-250 mm. ..	27	19	46 cases
250 mm. and over	5	16	21 cases

From this table it will be seen that only 5 out of 21 cases with a pressure of 250 mm. and over were alert at the time of puncture; of these in only one was the pressure over 300 mm., in contrast to 8 cases with a similar pressure who were drowsy, stuporose or comatose. These figures must not, of course, be taken to imply a direct relationship between the mental state and cerebrospinal fluid pressure; rather they suggest that other more important factors are concerned, and it will be noticed that even with normal pressures 27 out of 125 cases were other than alert at the time of puncture.

TABLE VII.—THE MENTAL STATE IN RELATION TO THE RED CELL COUNT IN THE CEREBROSPINAL FLUID AT INITIAL PUNCTURE WITHIN 7 DAYS OF INJURY IN 105 CASES

Red cells in the C.S.F. (per cu. mm.)	Total	Alert	Percentage of total cases Alert
	Cases		%
100-1,000 ..	36	25	68
1,000-10,000 ..	24	13	54
10,000-100,000 ..	32	8	25
Over 100,000 ..	13	1	8

When the mental state at the time of puncture is considered in relation to the red cell count in the cerebrospinal fluid, there appears to be quite an impressive increase in the proportion of the non-alert cases *pari passu* with an increasing cerebrospinal fluid red cell count. In Table VII this apparent relationship is shown in 105 cases punctured within seven days of injury. Thus, when the red cell count exceeded 100,000 red cells per cu. mm. of fluid, only one case was alert at the time of puncture (in this the red cell count was 236,000 cells per cu. mm.), as opposed to 12 which were not alert. In three of the latter the count was over a million cells per cu. mm., and all were unconscious at the time of puncture. This apparent relationship will be discussed later.

Discussion

It is of interest, especially in view of the importance which has hitherto been attached to the reduction of intracranial pressure by dehydration and repeated puncture, to note how frequently the cerebrospinal fluid pressure was found in this review to be normal or only moderately raised at initial puncture in the first week after injury. Though pressures of 300 mm. and over were recorded in such circumstances in 11 of 235 cases, and of 250 mm. and over in 27 of the cases, in nearly two-thirds the cerebrospinal fluid pressure was not raised above a normal limit of 180 mm. of water. Even when the injury was severe (resulting in a post traumatic amnesia of over 7 days) in 29 per cent. of the 24 cases the initial pressure was not above normal. It should be stressed, however, that the above cases were selected in that the factor of compression of the brain by extradural or subdural bleeding had been deliberately eliminated to a large extent, and it can be assumed that in such cases surgical measures are required to relieve the cerebral compression, which frequently gives rise to very high cerebrospinal fluid pressures.

It seems unlikely, therefore, in the absence of cerebral compression by bleeding that a raised initial cerebrospinal fluid pressure plays an important rôle in the production of symptoms and signs in acute head injuries, especially in view of the rapid return to normal levels which has been shown

in this review to occur in the average case untreated by lumbar puncture.

In consideration of the above data it will at once be apparent that no light is thrown on the mechanism by which a raised intracranial pressure is produced in acute head injuries in the absence of cerebral compression by extradural or subdural hæmorrhage. Approaching the problem from the theoretical aspect, two such mechanisms can be postulated—(1) alteration in the rate of production or absorption of cerebrospinal fluid; (2) alteration in brain bulk, whether by increase in the volume of brain tissue or by engorgement of the brain with blood. Let us consider the first theoretical mechanism. An increased production of cerebrospinal fluid may result either from local changes at the site of formation—the choroid plexuses—or from vascular factors resulting in a change in the rate of blood flow or intracapillary pressure in the plexuses. Rand and Courville (1931) from observations on the histological appearance of the choroid plexuses and the ependyma of brains from fatal head injuries suggested that an increased formation of cerebrospinal fluid takes place in such cases, and by analogy in non-fatal cases also. Other authorities, however, believe the changes they describe to be secondary to an increased intraventricular pressure. Furthermore, the normal channels of absorption may well be impeded when the cortical subarachnoid spaces are filled with blood.

Only a few years ago, when generalized traumatic cerebral œdema was thought to be common, the resultant increase in brain bulk was considered to be a primary factor in the production of an increased intracranial pressure, but recently grave doubts as to the existence of generalized cerebral œdema in such cases have arisen. Greenfield (1942) considers that œdema following injury to the brain is limited to the neighbourhood of the bruised area of cortex alone.

It would seem then, that this problem is as yet unsolved, and it may well be that the solution will not be found until we have at our disposal safe and reliable methods of investigation of the rate of formation and absorption of cerebrospinal fluid in acute head injuries.

As has already been stated, there appears to be a superficial relationship between the amount of blood in the cerebrospinal fluid and the mental state in those cases in which bleeding into the subarachnoid spaces has occurred, although there are notable exceptions. Turner (1941) reported a case in which the patient, though drowsy, was conscious, whilst at lumbar puncture over 4,000,000 red cells per cu. mm. were found in the cerebrospinal fluid. The latter resembled pure blood, except that it showed no tendency to clot. Thus such a relationship cannot be upheld if the source of hæmorrhage be not also taken into consideration. Bleeding from a torn venous sinus with penetration of blood through the arachnoid into the subarachnoid space, whilst being massive, might well occur in the absence of conspicuous alteration in the mental state.

Furthermore, it is possible, as Ritchie Russell (1932) suggested, that a large quantity of blood in the subarachnoid space may of itself cause loss of consciousness, and he quoted Bagley (1929) as showing in dogs that the subarachnoid injection of blood from the animal itself might produce loss of consciousness, whilst it is well known that in cases of spontaneous subarachnoid hæmorrhage in which the cerebrospinal fluid is heavily loaded with blood loss of consciousness may occur, though the fluid itself is found not infrequently to be under a normal pressure.

Nevertheless, blood in the cerebrospinal fluid in acute head injuries most commonly results, in all probability, from cortical laceration and contusion, and it has been postulated that such cortical damage accounts for the coincident alteration in mental state. Jefferson (1942), however, rejects the belief that unconsciousness is necessarily due either to cortical injury or to compression, and favours the view that it is due to interference with the functions of the mid-brain, whether arising from primary neuronal damage at the moment of injury or later from the formation of a tentorial pressure cone.

All that has been shown here is that the frequency of bleeding into the cerebrospinal fluid is directly related to the severity of the head injury, and that there appears to be a relationship between the intensity of such hæmorrhage and the frequency with which full consciousness is impaired. But though the intensity of cortical hæmorrhage can thus be assumed to be roughly related to the severity of the injury, the mechanism of direct and immediate neuronal damage to the brain stem is such that it likewise must be related to the same factor. Thus the present observations do not in any way conflict with Jefferson's views as to the causation of traumatic loss of consciousness.

Summary

1. 300 cases of selected acute closed head injuries in which lumbar punctures had been performed at an early stage following injury were reviewed.
2. The lumbar cerebrospinal fluid pressure at initial puncture within one week of injury was analyzed in 235 cases. Whilst the pressure was normal in two-thirds of the cases, there appeared to be a significant increase in the proportion of cases with a raised pressure in the more severe injuries.
3. An attempt was made to ascertain the natural rate of return of the cerebrospinal fluid pressure to normal levels after an acute injury. It was concluded that in the average case untreated by lumbar punctures this was achieved within one week of injury.
4. In 120 of the 300 cases under consideration blood was found in the cerebrospinal fluid in significant quantities at initial puncture, but it was shown that subsequent clearance of blood proceeded very rapidly by natural means alone, being largely

completed within one week of injury. Repeated punctures did not appear to speed up this rate of clearance to an appreciable degree.

5. The occurrence of delayed subarachnoid hæmorrhage especially in relation to repeated punctures was briefly discussed.

6. It was shown that whereas in mild or moderately severe head injuries the cerebrospinal fluid was frequently free from blood at initial puncture, when the injury was severe (post traumatic amnesia of over 24 hours) only 4 cases out of 53 were free from blood.

7. When bleeding into the cerebrospinal fluid had taken place, there appeared to be little correlation between the cerebrospinal fluid pressure and the red cell count at initial puncture.

8. The mental state at initial puncture was considered in relation to the cerebrospinal fluid pressure and red cell count; it was shown that there appeared to be a fairly close relationship between the mental state and the latter factor.

9. The importance of a raised cerebrospinal fluid pressure in such selected cases, the mechanisms which might give rise to a raised pressure, and the relation of traumatic subarachnoid hæmorrhage

to the mental state in acute head injuries were briefly discussed.

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