POSTOPERATIVE INTRACRANIAL HYPOTENSION

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In contrast to the increased intracranial tension which often follows brain operations, the occurrence of intracranial hypotension is rare, but this condition is also of practical importance, because if unrecognized and wrongly treated it may cause an otherwise avoidable fatality.

The syndrome of low intracranial pressure was first described in detail by Leriche (1920, 1935) and Leriche and Emery (1922) in patients who sustained open or closed head injury. Leriche observed it in 12 out of 75 consecutive cases of cranio-cerebral injury. The symptoms, which may appear either immediately or late after the trauma, consist of severe headache, nausea and vomiting, brady- or tachycardia, tachypnoea, and in more severe cases, of pyrexia up to 39°C. The skin of the face and the neck is flushed. The more or less somnolent patients lie curled up, and resent all external stimuli. Stiffness of the neck and Kernig’s sign, noted in some cases, may suggest inflammatory irritation of the meninges. In severe forms of the syndrome epileptic seizures occur. Lumbar puncture shows the cerebrospinal fluid pressure to be far below 100 mm. of water (in the sitting-up position), or so low as to be manometrically unmeasurable. The withdrawal of even a few drops of fluid is apt to aggravate the cephalgia whereas intravenous infusion of 250 ml. of normal saline or 20-40 ml. of distilled water is promptly followed by relief of headache and disappearance of the somnolence. In some of the patients who were operated upon Leriche found the dura mater to be retracted; in others he found an empty cerebral subarachnoid space with a dry and shrunken brain. After intravenous administration of normal saline solution the previously collapsed meninges became tense, the subarachnoid space filled with cerebrospinal fluid, and the volume of the brain increased.

The development of symptoms of intracranial hypotension may be preceded by the appearance of those of increased tension, the clinical differentiation between the two often being possible only by measuring the cerebrospinal fluid pressure. According to Leriche, the clinical picture results from hypotension of the cerebrospinal fluid, due either to loss of large quantities of the fluid or cessation of its production by the choroid plexus caused by vasoconstriction of its blood vessels and ischaemia.

A similar symptom complex, appearing even after mild cranio-cerebral injury, was also observed by Henschen (1927 and 1930), who emphasized the difficulty of clinical differentiation between intracranial hypotension, commotio cerebri and subdural haematoma, the last being often diagnosed where an exploratory operation revealed simply a dry and shrunken brain.

From the clinic of Clovis Vincent experience of intracranial hypotension after neurosurgical operation and cranio-cerebral injury was reported by Mahoudeau (1936) and Krebs, Puech, and Brunhes (1937), respectively. The former published case histories of 17 patients, chiefly children, who were operated on for posterior cranial fossa tumour. Since the syndrome of decreased intracranial pressure does not as a rule appear immediately but usually some time after the intervention, it is due not to operative trauma, but to insufficient secretion of the cerebrospinal fluid by the choroid plexus, the activity of which has been decreased during the development of the hydrocephalus, usually of high grade. The brains of the patients with intracranial hypotension who died showed marked venous congestion and multiple foci of subependymal haemorrhage in the ventricular system. Krebs and others (1937) pointed out that clinically the condition of decreased intracranial pressure is often difficult to diagnose, as it resembles the syndrome of brain oedema or intracranial haemorrhage. Indeed, they quote Puech and Askensay as having observed a similar syndrome in a patient with encephalitis and subarachnoid haemorrhage. In cases of lowered intracranial pressure they advise the insufflation of air or the injection of normal saline solution into the collapsed ventricular system.

Chorobski and Kunicki (1938a) reported case histories of six patients, who, following operation
for various cerebral lesions, developed a syndrome similar to that described by Leriche in patients suffering from cranio-cerebral injury.

In considering the signs and symptoms of intracranial hypotension we quoted Leriche (1935) who stressed the point that “indeed, there is nothing in the whole syndrome which one could consider as pathognomonic, yet its symptoms form a striking clinical entity.”

In the same year (1938) the intracranial hypotension following trauma to the head was the subject of a clinical demonstration by Zenker and Hardt and of a paper by Sprockhoff. According to the latter, the state of decreased intracranial pressure may be due to dehydration of the brain following its injury, impaired production of the cerebrospinal fluid, or leakage of the fluid into the extracranial tissues through a basilar skull fracture.

In 1940 (a and b) Sprockhoff reported the experience in the clinic of Tonnis with intracranial hypotension following neurosurgical operations. Of nine cases, two were patients in whom a suboccipital craniectomy was carried out and seven were operated upon for various supratentorial lesions. The defect in the skull in seven patients was no larger than the usual subtemporal bone defect left after an osteoplastic craniotomy, while in two it was “very large”. The syndrome of decreased intracranial pressure developed 10 hours to several months after the operation, and consisted of more or less severe headache, anxiety, excitation changing gradually into apathy, stupor, and coma. During the last condition breathing was of the Cheyne-Stokes type. Rigidity of the neck, nausea, mild hyperthermia and bradypnoea, normal blood pressure and pulse rate were the other elements of the syndrome. There was marked pulling in of the skin overlying the bone defect, and the volume of the cerebrospinal fluid, which, as a rule, was normal in composition, was zero in four patients, 15 ml. in one, 20 ml. in two, 40 ml. in one, 70 ml. in one, and 100 ml. in one (obtained from the ventricle, 10 hours after operation for internal hydrocephalus).

The symptoms are likely to appear suddenly, often following diagnostic lumbar puncture, or slowly. Their intensity is mild or very severe, with coma. They are of short duration or may last for as long as one month, as in one of Sprockhoff’s (1940a) patients. There is in these cases a great lability in the intracranial pressure, hypotension rapidly and without any apparent cause changing into hypertension, and vice-versa. It is necessary, therefore, to be cautious in filling the subarachnoid space with physiological solutions, as the injection of too much saline may easily cause a rise of intracranial pressure.

Wolff (1942) in his monograph on the clinical significance of low cerebrospinal fluid pressure states that the condition may develop spontaneously, or follow lumbar puncture, cranio-cerebral injury, subdural haematoma, and brain operations. He considers that it is due to diminished formation of the cerebrospinal fluid by the choroid plexus, caused by alteration in its venous circulation. The cerebrospinal fluid hypotension may be accompanied by headache, accentuated by an upright position, and abating when the head is put down, vertigo, nausea and vomiting (intensified by movements of the head), tinnitus, rigidity of the neck, and, in more severe cases, disturbance of consciousness. The cerebrospinal fluid often contains some blood and the amount of protein is increased. The symptoms usually disappear after the patient is given tablets of ephetonin dl-phenylaminopropanol HCl and 1 to 2 litres of 0.5% saline intravenously.

At the thirteenth annual meeting of the Harvey Cushing Society, in May, 1944, List spoke on the clinical significance of intracranial hypotension. He also stated that the more severe and persistent forms of this condition were due to a combination of cerebrospinal fluid loss, its diminished production, and a reduced brain volume. This is likely to be observed after evacuation of large chronic subdural haematomata, surgical operations for hydrocephalus and cerebral tumours. The clinical syndrome associated with this type of hypotension may be frankly alarming and resemble a state of increased intracranial pressure. In the subsequent discussion Sachs related having seen several cases of hypotension similar to those described. One of his patients who had sustained a cranio-cerebral injury at first had been doing well, but excessive dehydration caused the appearance of symptoms suggesting meningitis. The pressure of cerebrospinal fluid was about 5 mm. of water. Within 24 hours after giving 25 ml. of distilled water, the patient brightened, the meningeal symptoms cleared, and the cerebrospinal fluid pressure came up to 50 mm. Within another few days the cerebrospinal fluid pressure was normal. In two cases of subdural haematoma there was a fatal issue because the brain had lost its capacity to expand. To replace lost cerebrospinal fluid in cases of hydrocephalus Sachs uses the artificial cerebrospinal fluid developed by Hartman.

Case Reports

Since our first report on intracranial hypotension, published in 1938, we have seen several cases of a similar syndrome. Unfortunately, we are not sure about their exact number as many of our case histories were lost during the upheaval of the last war. All we know is that since 1945 we have certainly observed another
six patients with intracranial hypotension, but of these the case history of one has been lost also. It concerned a woman, aged 50 years, with a huge meningioma in the right temporal lobe, whose condition of decreased intracranial pressure lasted for one week, during which time she was quite aware of what was going on around her, but could not talk, move her extremities, or react to the stimuli we were applying to rouse her, and which she later resented strongly. Two other patients with intracranial hypotension had an astrocytoma of the left and right frontal lobes, respectively. The fourth had a left parietal meningioma, the fifth a right parietal posthaemorrhagic cyst, and the sixth a post-traumatic extra- and subdural haematoma. The syndrome noticed in all of them was more or less the same as that already described, and the treatment was in all cases more or less uniform.

The following three case histories illustrate the type of the condition we are here discussing. The first two have already been reported by Chorobski and Kunicki (1938a), and concern uncomplicated intracranial hypotension following, in the one, exposure of the brain and incision into the cortex, and, in the second, excision of a meningo-cortical scar. The third case is that of a patient recently observed whose postoperative recovery was first complicated by repeated blood clot formation and then by two episodes of decreased intracranial pressure.

Case 1.—R. S., aged 32 years (No. 225/37), was referred by Dr. Z. Kuligowski on October 23, 1937. For 18 months she had suffered from bouts of headache and vomiting, and lately had become somnolent and somewhat “hazy in her thoughts”. Objectively, there was incipient papilloedema on both sides, a right homonymous hemianopia and a hypaesthesia over the right side of the body. Amnesic aphasia, acalculia and agraphia led us to diagnose a neoplasm in the left parietal lobe, and consequently the patient was operated upon on October 26, 1937. No attempt was made to extirpate the tumour which had an infiltrative character and was located deeply beneath the angular gyrus. The patient withstood the operation, consisting of osteoplastic craniotomy, incision into the cortex and large subtemporal decompression, very well. During the following night she became restless, and the next morning she was lying motionless, with her eyes closed, reacted to questions only by opening the eyes, but swallowed fluids properly. Her temperature was 36°9 C., respiration 22, pulse 84. Eight hours later she ceased to react to questions almost completely, but when very strongly pinched she did flex her extremities. Her temperature was 37° C., pulse 80, respiration 24, and the blood pressure, which before the operation was 135/75 mm. Hg, was now 140/80 mm. Hg. Lumbar puncture revealed a colourless cerebrospinal fluid, coming out drop by drop so that it was impossible to measure its pressure. The patient was given 10 ml. of distilled water intravenously, changed into the horizontal position, and her condition began to improve; that is, she opened her eyes, murmured something when spoken to and drank tea with avidity. She continued to improve and the next morning (October 28, 1937) she was fully conscious, tried to answer questions, and was smiling contentedly. Her temperature was 36°8° C., pulse 82, respiration 22, blood pressure 140/80 mm. Hg. Seven days after the operation she left the neurosurgical unit for another department with her condition progressively improving.

Case 2.—M. R., aged 27 years (No. 229/37), was referred on November 3, 1937, by Dr. L. Hiszhalt-Zeldowicz. Eighteen months previously he was struck on the head, and was unconscious for about one week. He had at first a complete and then a partial motor aphasia with paresis of the right upper extremity. For the past year he had suffered from epileptic fits, which consisted of paraesthesiae of the right hand which might develop into grand mal seizures. Since the injury he had complained of headache and vertigo. Objectively, he had two small bone defects in the left frontal and parietal regions, weakness of the lower right face, paresis of both extremities on the right side, and hypeaesthesia of the whole right side of the body, particularly marked over the right hand. The puncture of the ventricles, carried out in the half-sitting position before the introduction of air, revealed a marked hypo-tension of the cerebrospinal fluid, which had to be drawn out with a syringe. The anterior horn of the left lateral ventricle was definitely enlarged, but there was no pulling of it towards the frontal bone defect or the expected scar.

Osteoplastic craniotomy performed on November 8, 1937, in the left frontal region, revealed a large meningo-cortical scar, running from the middle of the motor cortex down to the region of Broca’s centre. Immediately after the excision of the cicatrix with an electrocautery, the cortical veins became enormously engorged and the brain swollen. Because of this unexpected complication the bone flap could not be properly replaced and had to be taken off. The postoperative course was as follows.

During the next 12 hours the patient had five generalized epileptic fits. The first two days after the operation he lay with his eyes closed, which he opened to command but otherwise did not react to questions or mild external stimuli. His rate of respiration was 32, pulse 120-150, temperature 37-6°-38-5° C.; blood pressure, which before the operation was 145/90 mm. Hg, was then 110/70 mm. Hg. The lumbar puncture, carried out at two different levels on the second postoperative day, showed the cerebrospinal fluid pressure to be very low, while the skin overlying the huge skull defect, which for several hours after the intervention was very tense, was now definitely pulled in. Immediately this was noted the patient received intravenously 20 ml. of distilled water and was put into the horizontal position. Within the next few hours he became animated, opened his eyes immediately when asked to do so, tried to speak, and ate a hearty meal. The skin of his face and neck was still red. His temperature was 39° C., pulse 144, respiration 44. The next day (November 11, 1937) he was still better, kept his eyes constantly open, and spoke a few words. Nine days later (November 21, 1937) he once again became somnolent, and was crying and complaining of severe headache.
the bone defect was markedly pulled in. A few hours after he had been changed into the horizontal position and received 20 ml. of distilled water he again felt well, and there was no pulling in of the skin flap. On November 25, 1937, that is two weeks after the operation, the patient left us almost completely recovered.

Case 3.—D. K., aged 35 years (No. 203/49), was referred by Dr. S. Wlodarczyk on September 16, 1949. One month before she had suddenly noticed that several times a day first her right index and middle fingers and then the whole hand would become numb. At the same time she began to complain of severe frontal headache and vertigo, and vomited frequently. While talking she had some difficulty in finding proper expressions. On admission we found some weakness of the lower portion of the face on the right and a slight paresis and hypaesthesia of the right upper extremity. Otherwise, no abnormalities were noticed on general or neurological examination. The laboratory findings were essentially negative. The fields of vision were normal, but the radiographs of her skull showed an area of erosion in the left postcentral region and markedly dilated vascular channels for the meningeal blood vessels. A left postcentral meningioma was diagnosed.

The operation performed under local anaesthesia on September 23, 1949, revealed the tumour, the size of a very large plum, invading the overlying bone. It was reddish brown in colour, of rather soft consistency, and showed multiple foci of old haemorrhage. It was removed in toto. There was also an old thinly encapsulated subdural haematoma reaching from the base of the skull up to the superior longitudinal sinus which was also taken out. Because of the suspicion at the time of operation that we were dealing with sarcoma, the small defect in the dura mater produced by invasion of the tumour was left uncovered, and since the slightly swollen brain showed a tendency to prolapse, the dura mater was not sutured. The bone flap, from which there was at the time of closure no more bleeding than it is usual in similar cases, was replaced and the galea and the skin sutured in layers.

When the patient left the operating room her blood pressure was 120/90 mm. Hg, pulse 80, and respiration 17. She was perfectly conscious and was talking without much difficulty. During the operation she was given 1,500 ml. of normal saline intravenously. In spite of her good postoperative condition, however, she was given 450 ml. of whole blood. One hour later she complained of numbness in her right hand and still later she became slightly more apathetic. About ten hours after the operation she ceased to talk, became incontinent, did not swallow fluids or react to external stimuli. Her temperature was 37.8° C., pulse 84, respiration 24, blood pressure 120/70 mm. Hg. The cerebrospinal fluid was colourless but under increased pressure, so about 30 ml. was withdrawn and the patient received 30 ml. of 15% salt solution intravenously. When no improvement was noticed, exploration of the operative field was carried out 12 hours after the initial operation, and a large blood clot, closely attached to the bone flap, removed. No bleeding was seen from the dura mater or from the cavity in the brain, which was not entirely filled in, resulting from removal of the tumour. The whole brain was rather dry and sunken.

After the removal of the haematoma the condition of the patient definitely improved. She regained consciousness, but was rather restless. However, this improvement lasted only for some nine hours, and then the state of coma returned. We decided to carry out a second exploration, but before that she was given another 450 ml. of whole blood, although her blood pressure was 110/70 mm. Hg, pulse 93, respiration 25, and the temperature 37.4° C.

The third operation was performed 24 hours after the first, and once again, as at the second operation, we found a large blood clot firmly attached to the bone flap. After each removal of the haematoma and before the wound was closed, we sealed all the possible sources of bleeding with the utmost care, and yet the patient developed a third blood clot, but this time attached only to the bone flap. Six hours after the third operation, and an improvement which lasted for two hours, she was operated upon for the fourth time after having received a third transfusion of 450 ml. of whole blood. This time there was some oedema of the brain and we had to remove the bone flap which was the source of her uncontrollable bleeding (Fig. 1).

On leaving the operating room, the patient had a blood pressure of 110/70 mm. Hg, temperature 37.9° C., pulse 110, and respiration 24. She again recovered from her serious condition, but less promptly and markedly than after the second operation. The next morning, September 25, 1949, she was conscious although somnolent. We cut, therefore, some three skin and galea stitches and inspected the surface of the brain: there were a few small blood clots and some fluid, and the brain was neither shrunken nor swollen.

From then on the patient began to improve slowly but steadily until the next day when again she ceased to swallow fluids, or respond to questions, and only reacted to very strong external stimuli. Her blood pressure was...
at that time 120/90 mm. Hg, temperature 36.8° C., pulse 68, respiration 20. The skin flap overlying the skull defect was definitely pulled in and her face was flushed. When she was put into the horizontal position and received 1 litre of normal saline solution subcutaneously, her condition almost immediately improved; she became animated, started to drink and asked for the bed pan. The skin flap was distinctly elevated. However, one hour later, while still improving, she went into status epilepticus, which consisted of clonic seizures of the right angle of the mouth and the right hand. It lasted for almost four hours, being finally broken down by 0.6 g. of sodium luminal given hypodermically, 20 ml. of 10% sodium bromide injected intravenously, and 20 ml. of air introduced into the lumbar subarachnoid space. When, at last, the right-sided clonic movements ceased, the left angle of the mouth began to move and the clonic seizures continued for two hours.

After this rather stormy episode the patient again felt well, and the next day, September 27, 1949, ate a hearty meal. Six hours later, the clonic movements of the left angle of the mouth returned, but only for a few minutes. Thereafter her condition was satisfactory until the afternoon of the next day, September 28, 1949, when she gradually became somnolent, ceased to react to questions, responding only to pinching and pricking. Her skin flap was once again pulled in. Because of our experience that withdrawal of even a few drops of the cerebrospinal fluid may aggravate the condition of decreased intracranial pressure, no lumbar puncture was made. The blood pressure was at that moment 90/60 mm. Hg, pulse 90, respiration 16, and the temperature 37° C. (Fig. 2). Some 20 minutes after she had been put into the horizontal position the patient was much better. Her blood pressure rose to 100/70 mm. Hg.

From then on she recovered slowly, yet refused to lie in her bed with the head up. She told us that every time she was in the semicomatous condition she was perfectly aware of what we did to her, but could not react to it. She left us on October 10, 1949, with only a slight motor aphasia, paresis, and hypesthesia of the right hand.

Microscopical examination showed the tumour to be a meningioma with a few mitotic figures.

The bone flap was replaced on April 3, 1950, and when seen some two months later, the patient was entirely well, and practically without any signs and symptoms.

Discussion

It is, no doubt, interesting to note that both Sprockhoff's (1940a and b) and List's (1944) independent description of the signs and symptoms of intracranial hypotension appearing after neurosurgical procedures, correspond closely with the one reported (1938a and b) by Chorobski and Kunicki. Indeed, no difficulties are encountered in recognizing this syndrome, although its physiopathology is still debatable.

In all the three reports the greatly reduced pressure of the cerebrospinal fluid due to its diminished volume and the disturbance of consciousness are considered the pathognomonic signs of the syndrome. However, it is not easy to answer the question whether the decreased production of the cerebrospinal fluid represents only one of the causative factors or whether it causes the appearance of all the other signs and symptoms.
Loss of cerebrospinal fluid after lumbar puncture or spinal anaesthesia may cause a more or less persistent intracranial hypotension (Jacobaeus and Frumerie, 1923; Solomon, 1924; Alpers, 1925; Nelson, 1930; Pickering, 1939 and 1948; Kunkle, Ray, and Wolff, 1943; Thorsen, 1947), the low intracranial pressure being by many considered to be due to leakage of the fluid through the perforated spinal theca. It is also assumed that withdrawal of the cerebrospinal fluid column supporting, as it does, the cerebrum, causes shifting of the nauraxis and tension on the meninges and cerebral blood vessels and thus headache and other signs and symptoms. Yet, it seems that there must be in cases of intracranial hypotension some other mechanism which causes the insufficiency of compensatory cerebrospinal fluid formation.

We cannot explain our postoperative cases of decreased intracranial pressure by loss of the cerebrospinal fluid alone, inasmuch as, contrary to the experience of Mahoudeau (1936), Sprockhoff (1940a and b), and List (1944), we have never seen the syndrome occur after removal of tumours closing the fourth ventricle, nor Stookey's and Torkildsen's operations, surgical procedures which, naturally, would favour the development of the symptomatic complex of intracranial hypotension. Actually, the ventricular system was opened in only one of our 12 cases and in only a second was one of the lateral ventricles found to be dilated. Therefore, in only two cases could there be any question of a more or less marked loss of the cerebrospinal fluid. In none, including the two cases just mentioned, did we observe after the operation the fluid to accumulate beneath the skin flap which, of course, would denote leakage of the cerebrospinal fluid into the extracranial tissues.

We believe that the diminished volume of the cerebrospinal fluid and its low pressure does not therefore represent the causative factor, but only the most important objective sign of postoperative intracranial hypotension, and is not due to leakage of the fluid, but to its disturbed formation by the choroid plexus. How this disturbance is caused is another question not easy to answer. From the evidence now available, it seems that the cerebrospinal fluid is not a dialysate produced by filtration, but a product of secretion by the choroid plexus, as already postulated by Flexner (1934). If the formation of cerebrospinal fluid conformed to the physico-chemical laws of filtration, its insufficient production in our cases would have to be caused by a decrease in intracranial venous pressure, hindering its formation and facilitating absorption, or an increase in osmotic blood pressure, favouring absorption. Whether there was in our patients any change in the protein and crystalloid constituents of the blood and consequently in osmotic pressure following loss of blood, plasma, or fluid, has not been determined. We cannot be quite certain, therefore, whether the decreased volume of the cerebrospinal fluid should not be referred to alteration in the chemical composition of the blood.

One of the most characteristic features of the syndrome of intracranial hypotension is the normal or nearly normal behaviour of the systemic arterial blood pressure, both before the appearance and during the duration of hypotension. None of our patients had the systemic blood pressure markedly altered for more than a matter of minutes, so we have no reason to assume that the eventual decrease in choroidal venous pressure was induced reflexly or passively by this extracerebral circulatory factor. If the systemic blood pressure were markedly lower it could decrease the cerebral and choroidal blood flow, but as any changes in the systemic arterial blood pressure were unimportant, the cerebral vascular reflexes were certainly adequate to deal with them.

Only three of our 12 patients showed an increase in the rate of respiration, this being observed not before the development, but during the state of intracranial hypotension. Over-ventilation, by lowering the arterial carbon dioxide tension, depresses the cerebral blood flow (Gibbs, Gibbs, and Lennox, 1935) and thus hinders cerebrospinal fluid formation. Increased respiratory excursions facilitate the venous return to the thorax, and reducing intracranial venous pressure hinders the production and favours the absorption of the cerebrospinal fluid. But, as the respiration in the great majority of our patients was practically normal, the reason for the disturbed cerebrospinal fluid formation must be looked for in other factors.

In our cases, as the complication was postoperative, we should a priori suspect the surgical trauma to be the causative agent. The exposure of an area of the brain at operation to relatively low temperature made more or less anoxaemic and a depot of injured tissue metabolites, surely gives rise to afferent nerve impulses and absorption into the blood circulation of toxic agents, both of which factors may cause a decrease in the cerebral blood flow and thus modify the various activities of the brain. Indeed, we believe that the course of events leading to postoperative intracranial hypotension may well be like this. Due to unknown circumstances, some individuals react even to simple operative procedures by reversible circulatory changes, which presumably are only quantitatively different from those described by Prados, Strowger, and Feindel (1945) in animals with the brain simply
exposed. The alteration in the activity of the central nervous system due to ischaemia and anoxaemia, developing more or less rapidly, takes place both in the close vicinity of the operative field and in remote parts of the cerebral hemispheres, affecting as a rule the hypothalamus, hence, exacerbation of the symptoms existing before, or caused by the operation, such as the epileptic seizures starting in our Case 3 from the hemisphere opposite to that operated upon, the development of disturbances of consciousness, and of cerebrospinal fluid production.

The alteration of consciousness, characterized by a lethargic-like state, is distinctly different from the coma or even semi-coma of marked intracranial hypertension. The condition of one of our patients who “slept” motionless for one week, during which time she swallowed fluids quite properly, and afterwards complained about all the prickings and pinchings with which we tried to rouse her but to which she was unable to react by motion, characterizes the disturbance of consciousness in patients with low intracranial pressure. We feel that this condition, which seems to resemble very much the state of “arrested consciousness” of patients with tumours affecting the posterior part of the hypothalamus (Bailey, Buchanan, and Bucy, 1939), results from involvement of the diencephalon. This would be in harmony with the now universally accepted view on the localization of sleep, both normal and pathological, in this part of the brain (Hess, 1932), as well as with the thesis put forward by Penfield (1938) on the diencephalic location of the highest level of integration within the central nervous system essential to consciousness.

Nothing is known about any possible relationship between the activity of the hypothalamic sympathetic and parasympathetic centres and the function of the choroid plexus. However, once we accept the now prevailing view that the choroid plexus secretes the cerebrospinal fluid, there is perhaps an excuse for postulating a centre controlling its activity and located in the diencephalon. If this is the case, then the insufficient secretion of the cerebrospinal fluid by the choroid plexus, the glandular activity of which is affected by ischaemia and anoxaemia of the diencephalon, would represent the negative sign of hypothalamic anoxaemia, as lacrimation, salivation, and perspiration in cases of diencephalic epilepsy (Penfield, 1929) represent the reverse. However, even if one could not accept the view that the choroid plexus secretes the cerebrospinal fluid, i.e. that it is formed by the specific tissue activity of the gland, controlled by the secretory nerves, there still would be a possibility of assuming a controlling influence of the diencephalon on the cerebrospinal fluid formation, the filtration of which would then be regulated in the same way as the filtration of urine. Prados and others (1945) found some indirect evidence that damage of the nerve centres of the hypothalamus was probably depressing the activity of the anterior lobe of the pituitary. It would be of great interest to know whether our hypothesis of ischaemia and anoxaemia of the diencephalon in cases of postoperative intracranial hypotension alters the function of its posterior lobe, and thus affects cerebrospinal fluid formation. The deficit of the cerebrospinal fluid in cases of intracranial hypotension, that is, loss of a buffer and reservoir necessary to regulate the contents of the cranium, is followed by displacement of the brain towards the posterior cranial fossa and tension on blood vessels of the base of the brain, including the anterior and posterior choroidal arteries. The temporary closure of these basilar blood vessels, due to stretch, adds to the difficulties not only of cerebrospinal fluid secretion but also of oxygenation of the cerebral tissue, already impaired by the after-effects of operative trauma. Hence, the aggravation of the patient’s condition in the sitting-up position, and the striking improvement, particularly in the state of consciousness, on lowering the head horizontally, that is, by the reshifting of the brain and elevating the intracranial arterial and venous pressures.

The reformation of the cerebrospinal fluid starts most promptly after intravenous injection of distilled water or hypotonic salt solution infused at room temperature. It seems however that the physical mechanism by which the injected solutions act upon the choroid plexus may not be the only one in promoting cerebrospinal fluid formation. Indeed, we feel that the influence of intravenously administered solutions on cerebrospinal fluid formation may be partly of a nervous nature, the infused fluid breaking off the inactivity of the choroid plexus through reflex stimulation of its secretory centre, similarly as the swallowing of hot food may stimulate lacrimation.

Summary

Surgical trauma to the brain is followed in some 0.8% of patients operated upon for various

*Puech and others, speaking on intracranial hypotension at the French-English neurological meeting in Paris in May, 1948, the proceedings of which were published quite recently in Revue Neurologique (1948, 80, 447-480), state that they had observed the condition, following neurosurgical operation, in 10-5% of their patients. This high incidence is truly surprising. However, it is rather difficult to be sure whether the syndrome of intracranial hypotension means to them the [Continued in next page]
intracranial lesions by a decrease in intracranial pressure caused chiefly by cessation or insufficient formation of the cerebrospinal fluid.

The diminished volume of cerebrospinal fluid represents the pathognomonic sign of a syndrome consisting of semicoma or a lethargic-like state, pulling-in of the skin overlying any existing skull defect, flushing of the face and the neck, aggravation of symptoms existing before the operation or caused by it, and normal or nearly normal blood pressure, pulse, respiration, and temperature.

The whole clinical picture of postoperative intracranial hypotension probably results from ischaemia and anaesthesia of the brain, particularly the diencephalon, the disturbance of the cerebral blood flow being most likely caused by exposure of the brain to lowered temperature, afferent nerve impulses originating at the site of operation, and absorption of injured and anoxaemic tissue metabolites, all of which may well act upon the cerebral blood vessels.

It looks as if both the disturbance of consciousness and interrupted cerebrospinal fluid production were caused by ischaemia and anaesthesia of the hypothalamus, which seems to be the seat of nervous integration essential to consciousness, and may be a "centre" controlling the cerebrospinal fluid secretion.

Displacement of the brain towards the foramen magnum and tension on the basilar blood vessels in the sitting-up position, due to a deficit of the cerebrospinal fluid normally supporting the intracranial contents, add to the difficulties of cerebral blood circulation, hence the marked improvement when the patient is put in a horizontal position. This change of posture renews the brain with its basilar blood vessels and increases the intracranial arterial and venous pressures.

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same thing as to us, since—for them—it may be "predominant", or "associated" with serous meningitis, dilatation of the ventricles, oedema of the brain, or "other intracranial lesions". For Puech and others the physiopathology of intracranial hypotension is easy to be conceived. This does not seem to be the view of Ritchie Russell (1948), who, discussing the various papers concerning the condition of decreased intracranial pressure, made the stimulating suggestion that biochemical studies concerning water metabolism, particularly in connexion with dysfunction of the hypothalamus, might perhaps elucidate the actual mechanism of intracranial hypotension.

Stimulation of the choroid plexus by the intravenous infusion of 0.5% salt solution is another therapeutic measure which often is useful even in a serious situation.

Unrecognized intracranial hypotension if wrongly treated may end fatally.

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