CASE VI. Male, four years of age. Entered the hospital January 8th. There was no history of a previous illness. The present illness began with headache eleven days before the patient's entrance into the hospital. There was no vomiting. The temperature was not taken. In the afternoon of the same day the child complained of pain in the left arm. At 4 o'clock in the afternoon he was said to have become unconscious and to have remained so until he came to the hospital. This was probably an error in observation because the parents stated that three days later the head was somewhat retracted and he cried when handled, which would not have been the case had he been unconscious. It is probable that there was more or less stupor. The retraction lasted for two or three days, and there was some tremor of the hands at this time. There was no recurrence of acute symptoms after the first four or five days.

A physical examination made January 8th is as follows: "The child is fairly well developed and nourished. The cheeks are flushed. The speech is thick and difficult to understand, but the mental condition is not impaired. There is no retraction of the head, or hyperesthesia. The pupils are equal and react to light. There is no paralysis. The heart and lungs are not abnormal. The abdomen is not retracted. There are evidences of rachitis in the ribs, epiphyses and shape of the head. There is no albumin in the urine. The temperature is normal."

Lumbar puncture was performed on January 11th and a very slightly cloudy fluid was withdrawn. The fluid contained very few leucocytes and very little fibrin. No organisms were found. Cultures on blood serum remained sterile. The patient was in good condition. The speech was still thick. He sat up in bed much of the time.

On January 16th the speech was much clearer. His appetite was good. On the 20th he walked about the ward and appeared perfectly well. His speech was normal for a child of that age. During his stay in the hospital the temperature remained between 98° and 99° F. The pulse varied from 100 to 120. The respirations for the first four days were slightly increased in frequency and afterwards were normal.

Diagnosis.—The diagnosis of epidemic cerebrospinal meningitis is based upon certain symptoms which are common to all the types of the disease with the exception of certain comparatively rare fondroyant cases. These cases prove fatal in a few hours and the symptoms may resemble those of septicemia or toxemia more than those of meningitis. The diagnostic symptoms are a sudden and often severe onset characterized by headache, vomiting and fever; soon followed by pain and tenderness in the neck and spine; usually more or less retraction and rigidity of the head; general hyperesthesia; mental disturbance varying from apathy to unconsciousness; the occurrence in many cases of an herpetic efflorescence about the mouth or face; a moderate and irregular type of temperature in striking contrast to the severity of the other symptoms. A positive diagnosis can be made by means of lumbar puncture.

Lumbar Puncture.—The fluid obtained is more or less turbid. In severe acute cases, a puriform deposit frequently settles to the bottom of the test-tube in a short time. Microscopic examination of cover-glass preparations made from the sediment shows numerous polymorphonuclear leucocytes, "pus corpuscles," occasional smaller mononuclear lymphoid cells and fibrin. Groups of the diplococcus intracellularis are found in varying numbers in the protoplasm of some of the leucocytes. The cover-glass preparations should be stained with some preparation of methylene-blue, preferably Löffler's, which stains both the nuclei and the organisms. To differentiate the diplococcus intra-

cellularis from other organisms a second cover-glass preparation should be stained by Gram's method. (By this method the intracellular diplococcus becomes decolorized.)

Cultures should be made on blood serum at the time of puncture by allowing one or more cubic centimetres of the spinal fluid to drop into the culture-tube. Care must be taken to prevent the fluid from running down the side of the test-tube at its upper part in order to avoid contamination by wetting the portion of the tube which comes in contact with the cotton. One may fail to obtain a growth of the organism if the puncture is not performed during a period of the disease in which active symptoms are present. After obtaining the fluid in the serum-tube allow it to flow over the surface of the serum and remain in contact with it for a minute or two. In a number of cases in which the organisms were present in the cells and in which active symptoms were also present the writer has failed to obtain cultures by the use of a small platinum loop filled with fluid, because the quantity of fluid was too small.

The macroscopic appearances of the fluid are not to be relied upon in all cases. A very slight admixture of blood will render the fluid turbid; and if sufficient blood is present, a web of fibrin forms in the fluid after standing. Hence it is necessary to make a microscopic examination in all cases. If blood, or bloody fluid appears at the end of the needle it is well to allow the fluid to escape until a clear drop appears. It is rarely necessary to allow more than a cubic centimetre to escape in this way.

Inoculation experiments are, as a rule, unsuccessful. The organism is not pathogenic for guinea-pigs and rabbits. Goats are susceptible to it.

(To be continued.)

INCREASE OF INTRADURAL PRESSURE IN HEAD INJURIES.¹

BY W. N. BULLARD, M.D.

SEVERAL years ago I drew attention to the fact that an apparent increase of the intracranial pressure occurred in severe injuries of the head, and that the existence of this pressure and of the symptoms to which it gave rise were among the important factors in the determination of the condition in a given case and of the advisability of operation.

Indeed, the determination of the advisability of operation in cases of head injury in which no external signs exist depends very largely on the apparent degree of the intracranial pressure. Further observation and investigation of these cases has only served to confirm the views previously expressed.

I hope to-day to enter more into detail in regard to this condition. The first questions which confront us are, Does this apparent increase in intracranial pressure actually exist? and, if so, where is it situated and what is its cause?

That it does exist is, I believe, fully proved by clinical observation. Certain it is that in most cases of operation (trephining or in any way opening the cranium) that we find a marked tension of the dura, with pulsation of the brain absent or much diminished, and that on cutting or opening the dura this tension is

¹ Read before the Boston Society for Medical Improvement, December 13, 1897.

diminished while the pulsation is proportionally increased.

Such increase of tension existed in many cases which I have personally observed, and may be considered the usual condition found on trephining for head injury.

Formerly increase of intradural pressure after injury to the head was supposed to be due to pressure from a blood-clot. In some cases this is practically the case—the blood-clot, most frequently from the middle meningeal artery, is removed and the cerebral pressure ceases; but there are numerous cases in which the intracranial pressure occurs in connection with head injury in which no blood-clot exists. I wish specially to emphasize this point, as I have seen cases in which the surgeon trephined the cranium on both sides to find the blood-clot, which could not be found and, in my opinion, did not exist. It is not my province to enter into the methods of distinguishing between intracranial pressure due to blood-clot and that form due to other causes. In certain cases they probably cannot be distinguished; but in many cases hemorrhage can be diagnosticated. Intracranial pressure accompanying head injury does not, however, necessarily imply hemorrhage or blood-clot. It often exists without blood-clot.

Can now the intracranial pressure which we see on trephining after a head injury be due to any other cause than the injury?

This question has long seemed to me important. We know but little by direct observation of the intradural pressure in the adult under normal conditions. In infants before the closure of the anterior fontanelle we are often able to form some idea of the condition, but in many cases the thickened dura prevents any accurate knowledge of the degree of pressure beneath. We have, however, definite clinical evidence, or what is generally supposed to be such, of increase of intracranial pressure after head injuries.

Moreover, the amount of intracranial pressure which we directly observe after trephining is greater than we should expect to occur without the production of symptoms.

Is it possible, however, that any temporary or accidental causes may be the active factor in these cases rather than the injury? I have long felt that the influence of ether could not be eliminated; and it was not until I obtained definite, direct observations that I was able to be certain that it did not play an important part.

Last September a male adult was brought into the City Hospital unconscious, with hemorrhage from the right ear and a scalp wound nearly vertical in direction, three and a half inches long above the right mastoid. At the bottom of this wound the bone was exposed, but no fracture was detected. There was an ecchymosis around the right eye.

When seen by me, five hours after admission, he was deeply unconscious. The left pupil was larger than the right. All the limbs were in a condition of total flaccidity, but there was no facial paresis. Knee-jerks slightly increased. There was thought to have been a gradual increase of swelling about the right eye and some protrusion of the eyeball.

Operation was advised, and was performed at once without ether. The incision was lengthened downwards and a fissure was found running antero-posteriorly. On following this forward about an inch, a comminuted fracture with loose pieces of bone ap-

peared. The opening made by the removal of these loose pieces was enlarged by the rongeur. The brain beneath was bright red, not covered by dura, protruded through the opening, and did not pulsate except on pressure. No large clot of blood anywhere.

The patient died later, and the autopsy showed (1) a blood-clot of considerable size over the external surface of the left hemisphere posterior to the frontal lobe and thickest in lower posterior parietal region, (2) destruction of brain substance at site of bone removed (right lower parietal region), (3) that the longitudinal fissure found extended backwards across the right occipital bone passing directly through the torcular Herophili into the left occipital, and anteriorly it ran into the right middle fossa and thence apparently across the sella turcica, and a crack appeared in the left middle fossa. There was an effusion of blood in the right orbit, though no visible crack in the anterior fossa or orbital plate was detected on examination.

In this case, although no ether was given, there was marked tension of the brain, as evidenced by its protrusion through the opening in the cranium. I do not think that this was in any way dependent on the clot over the other hemisphere. On the other hand, such condition is apt to exist in certain cases of contusion. In this case there was some question of alcohol. While it is hardly likely that alcohol played any essential part in the increased brain pressure its influence cannot be wholly excluded.

In the following case the effect of alcohol is excluded. A boy, nine years old, was brought unconscious into the Boston City Hospital with tonic spasm of the left upper extremity. He had an immense antero-posterior extravasation over the whole length of the sagittal suture, and ecchymosis of the right eyelids. There was dilatation of the right pupil; knee-jerks slight. Lower extremities negative.

Operation was performed at once without ether. The sagittal suture was separated throughout its whole length and the longitudinal sinus laid open. There was a transverse fracture of the right frontal bone and a piece of loose bone there was removed. The surgeon trephined over about the middle of the right ascending frontal convolution. The dura was blue and very tense. On opening it the brain protruded perhaps as much as two inches. The protruding portion of the brain was contused and disintegrated. There were no large clots beneath the dura. Bubbles of air came through the trephine hole, suggesting fracture of the ethmoid.

In both these cases in which no ether was used there was contusion and disintegration of the brain substance; and in such cases we are apt to have—as in the last described—a marked brain pressure and protrusion when the opportunity exists.

It seems, however, fair to assume that the ether is not the cause of the increase of intradural pressure in the ordinary case of head injury, as it certainly was not in these two cases. Alcohol as a cause can also be excluded in a considerable number of cases. We are therefore compelled to assume that the increase in intradural pressure is due to the injury directly, as the clinical symptoms imply.

The pathological condition which exists in these cases is apparently a swelling of the brain itself, due in part perhaps to a filling and dilatation of its blood-vessels, in part to an edema of the cerebral tissue resulting therefrom, and in part also in some cases to an

excess of fluid between the pia and the dura. This latter, which has been referred to by Dr. Walton in his interesting paper read before the American Neurological Association at its last meeting, occurs in certain cases but not in all those in which intradural pressure exists.

The following cases illustrate this form of intradural pressure.

Male, thirty years old, was struck a glancing blow on the head one morning with a hammer. This occurred seventy miles from Boston. He came to Boston by train, ate a good dinner and a moderate supper, and after supper came to the hospital as an out-patient. Was seen about 9.30 P. M. There was a depression about three inches across, just to the left of the sagittal suture and immediately posterior to the coronal suture. No symptoms.

Operation immediate. Depression found in region described, with several radiating fissures. Trephined, and depressed fragments raised. Longitudinal sinus not injured. Dura did not pulsate. On puncture of dura considerable clear fluid escaped. Recovered without symptoms.

Male, eleven years old, was injured by an electric car. A large portion of the left scalp was torn back, and two fissures were plainly visible in the cranium, one running vertically through the left parietal bone just posterior to the vertical plane which passes through the anterior edge of the pinna; the other running horizontally forwards about two inches above the base of the cranium. The bone posterior to the vertical fissure was depressed, and ran under the edge of the anterior fragment. The bone above the horizontal fissure was slightly depressed. Blood oozed through both fissures; there was no hemorrhage from the ear. Patient was unconscious. No paralysis. No pressure symptoms. No decided difference in size of pupils.

Operation under ether. Trephined just posterior to vertical fissure. No clot. Dura bluish, tense, does not pulsate. On incision of dura, no clot; considerable cerebro-spinal fluid escaped.

I wish in passing to draw attention to the fact that there were no clinical symptoms of intradural pressure in this case in spite of the increase in the subdural or arachnoid fluid.

I am personally convinced, however, that the excess of subdural fluid is not alone the cause of the pressure symptoms in cases of head injuries.

(1) The increase in the amount of this fluid is, in many cases, not very great or even not apparent.

(2) It is shown by the tension and protrusion of the brain after the dura has been cut that the intradural pressure, as it still exists, was not in large part due to the fluid which has escaped.

(3) I have seen cases in which the pressure of the subdural fluid was very much greater than any shown in the ordinary cases of head injury with pressure symptoms—so great that when the dura was cut the fluid spurted, and, yet, there were clinically no general pressure symptoms. The following cases illustrate these points:

A female, age fifty-two, fell from an electric car. Was admitted to the hospital shortly after. Conscious; pupils equal; no paralysis anywhere. There was a depression of the cranium in the right temporal region. The surgeon trephined in this region, the operation being done under ether. No extradural clot. The dura was tense and scarcely pulsated. On cutting the

dura the brain bulged. No excess of cerebro-spinal fluid noted. Recovered well and quickly.

This is the ordinary condition, in my experience, in those cases of intradural pressure due to head injury in which no clot exists and there is no serious brain injury.

A male, eleven years old, fell down the subway and received a fracture of the frontal bone, with depression of a portion thereof. Was operated upon under ether on the fourth day after the accident. He then had an acute pachymeningitis. The dura was incised; no excess of subdural fluid noted, no subdural clot.

If, then, the cause of the increase of intradural pressure in head injuries in which no clot exists is not due wholly, or even in large part, to the excess of subdural fluid, to what may we consider it due? There is no doubt that it is, at least in part, the result of the congestion of the intracranial blood-vessels, either directly or indirectly. This is apparent, not only from direct observation and also from the fact that in no other way can we account for the rapid changes of the intradural pressure which are so often observed, but also from those cases (and they are not rare) in which after head injury intradural pressure does not occur. These are an extremely interesting class of cases, and I hope at some future time to be able to consider them in detail. I will only say here that I feel certain that in these cases the absence of increase of intradural pressure is (with rare exceptions to be noted later) due to hemorrhage, whether with or without clot formation. The relief of the congestion of the intracranial vessels causes the bulging brain to sink and the pulsation to reappear.

The opposite is the case when, as I have seen, on tying an intracranial artery or stopping a hemorrhage, the brain which has been sunken and pulsating feebly, at once or shortly begins to swell and the pulsations to become more strong.

Cases might easily be reported illustrating these points, but at the present moment this seems hardly advisable.

SUMMARY.

(1) An abnormal increase in the intradural pressure often occurs as an accompaniment or result of severe head injuries, where no large clot exists.

(2) This intradural pressure may be in part due to an excess in the amount of subdural fluid. This, however, is, as a rule, not the chief element in the intradural pressure which is principally due to the bulging of the brain itself.

(3) The cause of this intradural pressure is apparently a congestion or filling of the intracranial blood-vessels and the results thereof.

A WARNING TO OBSTETRICIANS. — Don't count your chickens before they are hatched. How Dr. Brickett lost his fee and ten dollars besides. When he was living in China, before the war, he was called to attend a case of confinement in Palermo. After being at the bedside a few moments a child was born. It looked rather small and the doctor remarked, "Mrs. Blank, if you have another one I won't charge any fee." In about twenty minutes another child was born (No. 2). Then the doctor said, "Mrs. Blank, if you have another I will make you a present of ten dollars." In less than half an hour another child was born (No. 3).