THE INDICATIONS FOR OPERATION IN THE TREATMENT OF INJURIES INVOLVING THE BRAIN.

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The indications for operation in the treatment of injuries involving the brain are three:

First, a compounded fracture of the skull with injury to the brain.

Second, a depressed fracture of the skull involving either both tables or the inner table alone.

Third, and most important of all, an increase in the intracranial pressure.

Exclusive of the more or less specialized forms of fracture of the skull—the compound and the depressed varieties, I do not consider fracture per se an indication for operative interference.

Not infrequently the first thought and all of the attention of a physician who sees a patient who has received an injury to his head is devoted to determining whether or not there is present a fracture of the bony covering of the brain.

I wish to show that this is, except in the case of a compound or depressed fracture, a mistake that may cost the patient his life, or at least decrease his chances of recovery, and furthermore, that having obtained this information, the physician is no better off from the point of view of locating the injury than if he had made no effort in this direction at all.
Patients who have received an injury to the brain do not develop symptoms and do not die from the fracture which may or may not be present in the bones of the head. They do develop symptoms, and they do die as a result of the increase in the intracranial pressure which accompanies the fracture, or which not infrequently is present in the absence of any fracture.

**Compound Fracture.**

The indications for operation in compounded fracture of the skull are clear. Just as in a compounded fracture of any other bone we have at the very least a potentially infected wound, and steps must be taken to prevent this infection from spreading to surrounding clean tissues—in this case, of course, primarily the brain and meninges. Briefly, this may be best accomplished in the majority of cases by the process known as debridement, developed and used in the medical services of the allied armies during the war.

**Depressed Fracture.**

In a depressed fracture of the skull it is important to remember that there may be a depression of the inner table of the skull alone, without a break in the contour of the outer table. Consequently, it is at once apparent that in these cases the X-ray examination of the skull becomes of greatest importance. I believe that more information may be obtained with less effort if stereoscopic plates in both anterior-posterior, and lateral directions are taken at once if possible. Of course, many times, with excited or restless patients this is impossible, but I believe that this should be the aim of the roentgenologist. These depressions should be
either raised or, if that is impossible, removed.

This, then, briefly, covers the cases of injury to the skull and its contents in which the fracture in itself has any bearing on the operative treatment of the case.

**INCREASED INTRACRANIAL PRESSURE.**

Increased intracranial pressure as an indication for operation in the treatment of injuries of the brain is of paramount importance. The determination of its presence or absence should be the first information to be ascertained in any such case. Neglect of this all important information in the diagnosis, and a cloudy or mistaken conception of its relation to the future treatment and the present condition of these cases, I believe to be the one great factor that as late as 1916 kept the death rate in this class of case as high as 50 per cent.¹

To appreciate the importance of a pathological increase in intracranial pressure, it is necessary to have a knowledge of some of the more important points of the anatomy and physiology of the cerebrospinal fluid, its origin, distribution, and ultimate destination.

**PRODUCTION AND DISTRIBUTION OF THE CEREBROSPINAL FLUID.**

Magendie,² whose extensive researches in this subject were published between 1825 and 1842, was the first to appreciate the great physiological importance of this fluid. Through his investigations it was shown that not only are the ventricles and subarachnoid space normally filled with fluid, but that the fluid passes freely from one to the other of these divisions. Since that time many workers have added to our information in this field through chemical, intra-vitam staining,
and experimental methods culminating finally in a series of experiments conducted recently by Dandy at Johns Hopkins, and published in 1919 and previous years. It is not necessary to repeat here a detailed description of this work, but certain points should be emphasized. The cerebrospinal fluid is secreted by the choroid plexus practically in its entirety. This plexus lies in the two lateral, the third and the fourth ventricles. From the lateral ventricles the fluid passes by way of the foramina of Monro into the third ventricle and receives its quota from the choroid plexus there. The fluid then passes through the aqueduct of Sylvius into the fourth ventricle and receives its quota from this portion of the plexus. From the fourth ventricle the fluid passes by way of the foramina of Magendie and Luschka to the cisterna magna, which in turn communicates with the entire cerebral and spinal subarachnoid space. Approximately one-half of the total quantity of cerebrospinal fluid is contained within the spinal subarachnoid space. The ventricles contain from 20 to 30 c.c.4

ABSORPTION OF THE CEREBROSPINAL FLUID.

This then is the method of production and distribution of the cerebrospinal fluid. We must now consider its absorption. In connection with this it is well to recall that the subarachnoid space lies beneath the arachnoid and above the pia, that this space closely invests every part of the central nervous system, and, owing to its relation to the pia, comes into intimate contact with the surface capillaries and many of the larger vessels of the entire brain and cord. From this great space the cerebrospinal fluid passes into the venous circulation either by way of a filtration process via the arachnoidal villi, or
by diffusion into the capillaries of the subarachnoid space. The investigations of Key and Retzius, Renier and Schnitzler, Sicard and Cestan, Frazier and Peet, Mott, Dandy, and Blackfan, and others have definitely shown that the cerebrospinal fluid reaches the venous circulation, and that the lymphatic absorption, if any, is so slow and of such a small amount that it can be disregarded. The only point in question at this time is just what the channels are that the fluid passes through after leaving the subarachnoid space, and before reaching the venous circulation.

In brief then it can be said that the cerebrospinal fluid is secreted by the choroid plexus in the ventricles; passes from them by way of the foramina of Magendie and Luschka into the cisterna magna and thence is distributed over the entire surface of the central nervous system within the subarachnoid space, and in intimate relation with the pial vessels whence it is absorbed into the venous circulation.

INTRACRANIAL PRESSURE.

Normally the secretion of the cerebrospinal fluid is constant. Necessarily, therefore, the absorption must also be constant. As a corollary to these two facts the secretion must be carried on at a sufficient rate to maintain pressure enough to promote absorption. This secretory pressure depends on two variable factors—first, the rate of secretion, and second, the rate of absorption. With a variation in either the rate of secretion or the rate of absorption, there will be a direct and resultant variation in the pressure. Normally this pressure, to which we can now give the name of intracranial pressure, varies with and is equal to the pressure in the venous sinuses.
It depends upon the amount of cerebrospinal fluid present, and also upon the systemic blood pressure and respiration. Interference with the arterial circulation of the brain causes only a transitory lowering of the intracranial pressure, at once followed by a return to normal as would be expected. On the other hand, equally as would be expected, interference with the venous circulation, by impeding the absorption of the cerebrospinal fluid causes an immediate and more or less constant increase in the intracranial pressure. In view of the distribution of the cerebrospinal fluid it is apparent that any increase of intracranial pressure will have an effect upon the entire central nervous system, and more especially upon the cerebrum and medulla. That this must be so is clear when we recall that not only is this pressure exerted upon the more superficial aspect of the cerebrum and medulla through the subarachnoid space and cisterna magna, but also is exerted in a similar manner and to an equal extent upon the innermost surfaces of the cerebrum and medulla by way of the ventricles. The cerebrum and medulla, therefore, may be said to be subjected to a process of squeezing whenever the intracranial pressure is raised. The amount of squeezing and consequently the amount of damage to the delicate nervous tissue of the brain depends upon the amount of the increase in the intracranial pressure.

Here, then, is a possible source of injury to the brain and medulla the extent of which depends only upon the height to which the intracranial pressure rises. That this may cause the death of the individual subjected to this rise is obvious, especially when we take into consideration the fact that regulatory centres, upon the
normal functioning of which the continuance of life depends, are located in the medulla. Furthermore, it is evident that any lesion which interferes with the normal cerebral venous circulation will give rise to this increase of pressure. Enumeration of the possible lesions that may cause this shows on the one hand how unimportant a relation a linear fracture of the bony coverings of the brain bears to this serious condition, and on the other hand how important and what a direct effect upon life such conditions as asphyxia, reflex disturbances of vasomotor control leading to acute congestion and oedema of the meninges, effusion of blood into the ventricles or between the meninges, or in the substance of the cord or brain, contusion and laceration of the brain by contra-coup or direct violence, hemorrhage into the subarachnoid space, and other similar conditions may have. Treatment must be directed toward relieving this pressure. This may be done in one of two ways: first, by withdrawing some of the cerebrospinal fluid, and second, by allowing the brain more room to expand, or in other words by a decompressive operation upon the skull.

MEASUREMENT OF CEREBROSPINAL PRESSURE.

The ability to diagnose correctly this important and dangerous condition depends upon our ability to measure, first, the normal cerebrospinal pressure, and second, an increase in cerebrospinal pressure.

The normal cerebrospinal pressure has now been measured many times, and a variety of manometers have been devised for this purpose. The pressures recorded in the case reports below were measured by the mercury manometer devised by L. H. Landon while at Frazier’s clinic.
in Philadelphia. Briefly, it consists of a U tube containing mercury, against one arm of which is set a millimeter scale for convenience in measuring the rise of the mercury columns. With the adult patient lying quietly on his left side with the entire spinal column horizontal and the top of the columns of mercury on a level with the needle, which is inserted as for an ordinary lumbar puncture, the normal pressure varies between 6 and 10 mm., and in the large majority of cases is in the immediate neighborhood of 8 mm.$^{11, 20}$ New-born babies tend to have a somewhat lower reading than adults, but otherwise show no great variation. Any reading above 10 mm. Hg. is abnormal and is evidence of an increase in intracranial pressure.

**OTHER SIGNS OF INCREASED CEREBROSPINAL FLUID PRESSURE.**

Cases which are suffering from an injury to the brain, and in whom can now be demonstrated, by means of the spinal manometer, a definite rise in intracranial tension or pressure, also show certain more or less constant changes in the eye grounds, blood pressure, pulse rate and volume, temperature, respiratory rate, and reflex activities. These changes were previously considered to be indications which controlled absolutely the treatment of the case at any given time. They are still considered so in many clinics, and by some neurological surgeons are considered of more importance than the measurement by manometer of the intracranial pressure. I believe this to be a mistake, and within the last two months have seen one case with a rising pulse, normal blood pressure, and essentially normal reflexes, who showed at lumbar puncture a pressure of 30 mm. Hg., and at operation a large
middle meningeal clot, and have known of another case in which a demonstrated pressure of 40 mm. Hg. was ignored and autopsy revealed a large subdural hemorrhage.

**EYE GROUNDS.**

Of all of these concomitant signs of pressure that of the eye grounds is the most constant and by far the most valuable. A small rise of pressure above the normal may dilate the veins of the retina, and cloud the nasal margin of the disc. A further rise will produce the classical choking of the disc.\(^{12}\)

**PULSE, BLOOD PRESSURE, ETC.**

The changes in pulse, blood pressure, respiration and temperature, are themselves due to the rise in intracranial pressure, and indicate nothing more than successive stages in the increase in this pressure.\(^{13}\) They are not directly connected with the injury and have no bearing upon the diagnosis, treatment, or prognosis of the case other than that indicating further pressure.

Given a case which has been struck sufficiently hard upon the head to produce symptoms, the least severe injury to the contents of the skull that could occur would be an irritation of the meninges. Injuries of greater magnitude would, of course, include laceration and contusion of the brain of varying extent, and massive intracranial hemorrhages. In connection with this,\(^{14}\) Kolmer, writing in Frazier's "Surgery of the Spine and Spinal Cord," states that "as the result of acute inflammatory changes in the meninges, with great increase in the volume of fluid, this regulating mechanism (controlling the intracranial pressure and volume) is disturbed." In other words, more cerebrospinal fluid is se-
creted than is absorbed, and as a result the intracranial pressure rises. With an extensive injury to the brain surface itself, or with a large intra or extra-dural hemorrhage, we have also in addition to the meningeal irritation an actual blocking of a certain portion of the subarachnoid space. That is, a blocking of a part of the absorptive area. As a result of this we have an actual excess of cerebrospinal fluid. The brain being non-compressible and this fluid having no place to go stays within the residual subarachnoid space under a greater pressure. This pressure in its turn causes a more or less local cerebral anemia and resulting cerebral venous stasis. This condition is spoken of by Sharpe15, 16 as the First Stage of Compression. The symptoms are headache, drowsiness, stupor, or possibly marked excitement. The retinal veins may be engorged. The manometric cerebrospinal fluid pressure is from 12 to 14 mm. Hg. Repeated lumbar puncture with withdrawal of sufficient cerebrospinal fluid to lower the pressure to normal will frequently cure patients in this condition.

The following will serve to illustrate the type of case referred to in the preceding paragraph:

J. A. W.—Male, 40, white, married; working as freight conductor; Seen May 10, 1921, in consultation with Dr. A. P. Lowell, Fitchburg, Mass. Diagnosis: Multiple Contusions and Abrasions, Contusion and Oedema of the Brain—probably contra-coup—with Subarachnoid Hemorrhage. Severe Concussion. Treatment: Cerebrospinal fluid drainage by lumbar puncture. Discharged: Cured. The patient was found unconscious on the floor of a caboose at 2.30 A. M., May 8, 1921. No history was available. Carried at once to Burbank Hospital, Fitchburg.
On admission he had two generalized convulsions and was in considerable surgical shock with pulse about 120. Was also noisy, irrational, and very restless. May 9, 1921, condition improved. No further convulsions and patient quieter. His pulse dropped to 72, and he recognized his wife. This A. M. (May 10, 1921) pulse still dropping—around 44. Blood pressure 120. Seen at 3 P. M. when he was conscious and rational, but somewhat stuporous. Had a complete amnesia dating from at least one week previous to the accident and continuing to the present. Recognized his wife and sister, but did not know the day of week nor the month. Examination showed lacerated wounds of scalp and outer canthus of left eye and multiple contusions and abrasions over the body. Examination of the heart, lungs, abdomen, and bony skeleton is normal everywhere. Urine negative. Neurological examination shows no changes in either the somatic or splanchnic motor or sensory systems. The cranial nerves are all normal except for complete deafness in the left ear. Eye grounds are normal. There is no ataxia, adiadokokinesis nor astereognosis. The speech is thick, but otherwise not changed. Mentally he is dull and apathetic, but answers questions correctly except as above. There is a complete amnesia dating to one week previous to the accident and extending up to the present. X-rays of the skull show no fracture and no evidence of any intracranial pressure. Lumbar puncture showed a pressure of 14 mm. Hg. Sufficient fluid was withdrawn to reduce this pressure to 7 mm. Hg. without symptoms. The cerebrospinal fluid was colored and mixed with blood which evidently came from a subarachnoid hemorrhage higher up. Last seen on June 3, 1921, having been out of the hospital
since May 29. On this former date had no symp­toms of any sort and is to go back to work in a day or so.

G. W.—Male, 18, white, single; working as a lineman for an electrical power company. Seen May 16, 1921, in consultation with Dr. H. W. Ellam of Gardner, Mass. Diagnosis: Contusions of Neck, Shoulders, and Back—Lacerated Wound of Posterior Part of Scalp—Fracture of the Right Occipital and Parietal Bones—Sub­arachnoid Hemorrhage probably due to contu­sion of Cerebellum—Moderate Intracranial Hyp­ertension. Treatment: Cerebrospinal fluid drainage by lumbar puncture. Discharged: Cured. May 15, 1921, while at work was struck on back of head, neck and shoulders by an ‘‘insu­lator’’ said to have weighed about fifty pounds. Taken at once to the hospital following the in­jury. No loss of consciousness or memory at any time. Said to have been very talkative and excited immediately following the accident. On entering hospital temperature was 101 and pulse 120. A few hours after entrance became stupor­ous and vomited repeatedly, being unable to take any food. Today is somewhat better, but still unable to take food and complaining of nausea and dizziness on sitting up. Temperature now normal and pulse 72. Physical examination of heart, lungs, abdomen, and bony skeleton normal except for contusion of shoulders and back, and a lacerated wound of posterior portion of scalp. Blood pressure 130/70. Urine negative. Neuro­logical examination shows no changes in either the somatic or splanchnic motor or sensory sys­tems. Cranial nerves are all normal except for a moderate nystagmus of both eyes with the quick component toward the right, on extreme
rotation of the eyes to the right. *Eye grounds* normal. There is a slight past-pointing toward the right with the right hand in the finger to finger and finger to nose tests. The left is normal. There is no *adiadokokenesis* nor *aste­rognosis*. Mentally he is alert and rational. No changes in his speech. *X-rays* of skull show a linear fracture of both tables of the skull about 1\(\frac{1}{2}\) inches long running at right angles to and crossing the occipito-parietal suture at the junction of the lower and middle thirds on the right. *Lumbar Puncture* showed a pressure of 14 mm. Hg. About 5 cc. of cerebrospinal fluid was withdrawn which lowered the pressure to 6 mm. Hg. without producing any symptoms. This fluid was colored by blood from a subarachnoid hemorrhage higher up. When last heard from in November, 1921, this patient had been back at work and entirely without symptoms since early in June.

It is to be noted that in neither of these cases were there any reflex changes, nor other objective symptoms of any characteristic degree. One suffered from a severe concussion, and the other did not. One had a fracture of the skull, and the other did not. In both, however, the cerebrospinal fluid pressure was raised to an equal degree. There was a subarachnoid hemorrhage, and withdrawal of enough cerebrospinal fluid to reduce the pressure to normal effected a prompt cure in both cases.

The next stage in increasing intracranial pressure is spoken of by Sharp\(^{17,18}\) as the Ideal Operative Stage. In this stage the rising intracranial pressure causes a more extensive and complete cerebral anæmia. If the pressure is due to an extradural lesion, such for example as a middle meningeal hemorrhage, or a de-
pressed fracture, the anæmia is more localized and we have symptoms due to the impairment of the particular part of the cortex underlying the lesion. Owing to the falx and the tentorium the majority of the pressure exerted by the extradural lesions is in this stage localized to one hemisphere, and the symptoms are not, therefore, as grave, due to the fact that the medulla is protected. With an intradural lesion, however, the protection afforded by the falx and tentorium is of not so much avail, and consequently the medulla is included in the more or less generalized cerebral anæmia. The first effect of this anæmia on the medulla is said to stimulate the vagal nucleus, and does in any event produce a slowed pulse, and also a slight rise of the peripheral blood pressure due to stimulation of the vasomotor centres. The cerebral venous stasis is more pronounced, and the patient presents symptoms similar to those noted above, only more severe. Manometric readings of cerebrospinal fluid pressure are from 18 to 30 mm. Hg. Treatment consists of subtemporal decompression. Neither at this nor at any subsequent stage of increasing intracranial pressure should more than 1 cc. of cerebrospinal fluid be withdrawn by lumbar puncture for examination. To break this rule subjects the patient to the danger of a herniation of the medulla through the foramen magnum with resulting death.\textsuperscript{19, 21}

A. S.—Male, 39, white, married, proprietor of a grain store. Seen April 30, 1921, in consultation with Dr. Thomas Donovan, Fitchburg, Mass. Diagnosis: Linear Fracture of Left Temporal Bone—Rupture of Left Middle Meningeal Artery with Extradural Hemorrhage—Medullary Anæmia with marked Intracranial Hypertension.
Treatment: Left Subtemporal Decompression. Discharged: Relieved. April 26, 1921, while moving a 250 pound bag of grain, a pile of similar bags fell over on top of him burying him beneath them. Taken at once to the hospital unconscious. On arrival there became conscious again, and then for a period became unconscious again following which he again became conscious but very stuporous. Speech has been thick at all times, and the stupor has been gradually increasing. Pulse has been constantly around 44, and temperature slightly elevated. Has complained bitterly of a severe frontal headache. April 30, 1921, patient became a little clearer mentally though still complaining of his headache, and still showing the same alteration in his speech. The pulse is now irregular, varying every few hours from 72 to 40. Temperature has risen to 100°. Respirations are normal in rate and character. Physical examination of heart, lungs, abdomen and bony skeleton is all normal. Urine normal. Neurological examination shows no changes in the somatic or splanchnic motor or sensory systems except for an ankle clonus on the right. Cranial nerves all normal. Eye grounds could not be successfully examined on account of the patient's restless condition. Apparently no ataxia. Diadokokinesis and stereognosis not tested. Mentally very stuporous and dull and difficult to arouse. Answers questions and obeys directions with only a moderate amount of success. Speech is very thick and slow and at times the patient is at a loss for a word that he obviously knows and wishes to use but cannot. At these times he will substitute some other word sometimes synonymous and sometimes not. No attempt was made to have the patient read or write. He could not repeat
a test sentence and evidently did not understand its meaning. Remembers nothing relating to the accident previous to his first night in the hospital. X-rays: thought previous to operation to be negative, but after the fracture had been located at operation it was possible, with the aid of a magnifying glass, to make out a line of fracture previously missed about 1/2 inch in length in the squamous portion of the left temporal bone. Lumbar Puncture showed a pressure of 30 mm. Hg. No fluid was withdrawn. Operation: Typical left subtemporal decompression through a muscle-splitting incision. Bony opening 3 inches in diameter extending from the level of the floor of the middle fossa of the skull. There was a large extra-dural clot evidently from a rupture of the middle meningeal artery although no bleeding point was found. As much of this clot as could be was removed. The dura and arachnoid were then opened and a moderate amount of cerebrospinal fluid under pressure was allowed to escape. Following this the pulse rose from 72 to 120, and the brain which had not been pulsating began to pulsate. The cortex was flattened and injected, but otherwise normal. Dura was closed with a rubber tissue drain between it and the brain on the floor of the middle fossa and the rest of the wound sutured with drainage through the upper end to the dura. The brain was pulsating normally with no sign of herniation at the end of the operation. May 5, 1921, patient now normal in every way. The decompression is entirely beneath the temporal muscle and well protected. All drainage is out. Last heard about in August, 1921, at which time he was back at work free from symptoms.

Should the intracranial pressure still rise, we
have a stage in which the symptoms are those of compression of the medulla.\textsuperscript{22, 23} The anemia of the medulla becoming more profound, this lack of blood acts as a stimulus to the vasomotor centre which in its turn raises the general arterial blood pressure, and slows the pulse, thus forcing more blood into the medulla and temporarily overcoming the anemia until the intracranial pressure rises a little further and the process is repeated. In addition to this we have a gradual slowing of the respirations with longer and longer periods of apnea, finally becoming the typical Cheyne-Stokes type of respiration, probably resulting from anemia of the respiratory centre. It is at this stage that we find the typical choked disc due to an oedema of the optic nerve. The symptoms of this stage then are rising blood pressure with dropping pulse of a full bounding character, and Cheyne-Stokes respiration. The patient is unconscious, and often all reflex activity is abolished. Manometric readings are always above 20 mm. Hg., and may reach 40 or even higher. Subtemporal decompression is imperative at this stage, and even in spite of this operation the patient may die. No patient should be allowed to reach this extreme stage of compression, and were the cerebrospinal pressure measured when the patient is first seen—provided he has recovered from the initial shock of the injury—no patient would reach this stage.

N. P.—White, male, 50, married, born in Greece; works in restaurant. Seen May 24, 1921, in consultation with Dr. R. F. Burns, Fitchburg, Mass. Diagnosis: Laceration and Contusion of Right Parietal and Temporal Lobes of Cerebrum,—Medullary Compression with extreme Intracranial Hypertension,—Fracture of
Right Occipital and Parietal Bones extending into the petrous portion of the right Temporal Bone. **Treatment:** Right Subtemporal Decompression. **Discharged:** Relieved. Patient speaks no English, having been in this country only a few months and history was incomplete and more or less inaccurate. It is said that on May 19, 1921, patient fell down stairs striking his head against a barrel of cement. Was conscious following this, and able to ride to the hospital in an automobile. Was somewhat dazed and stuporous on admission, but this has cleared gradually up to May 23 on which day he tried to get out of bed, and appeared to be fairly bright. May 24 is not so well, and is again somewhat irrational and stuporous. The temperature has been always more or less elevated up to 101. Pulse has recently been gradually but continuously dropping. May 24 pulse was 65 and respirations were taking on the Cheyne-Stokes character. Physical examination of heart, lungs, abdomen, and bony skeleton is all normal. **Blood Pressure** is 190/100. Urine shows a massive coagulation of albumin with fine and coarse granular casts but no blood. Neurological examination showed the following: **Somatic Motor**—both arms and the right side of the abdomen and right leg are normal in every way. The left abdominal reflexes and both cremaster reflexes are all absent. There is a flaccid paresis of most of the muscles of the left upper and lower leg involving especially the extensors of the thigh, the flexors and tibialis groups of the lower leg. The knee and ankle jerks are present and normal. Stroking the sole of the foot, squeezing of the calf or pressure over the inner border of the tibia elicits reflex motions affecting the toes of the right foot which fan with a plantar flexion of the great
toe. Attempts at voluntary motion of the paralyzed leg show as corresponding motions in the other leg. **Somatic Sensory:** there is a diminution, and very probably a complete anesthesia, of the entire left lower leg from the knee down. There are no other sensory changes. **Splanchnic, Sensory and Motor Systems are normal. Cranial Nerves:** examination of these nerves was unsatisfactory and incomplete owing to the patient's inability to co-operate, but the following definite changes were made out: The eye-grounds showed a marked dilatation of the retinal veins, but no hemorrhage, and a definite choking of both discs. The first, eighth, eleventh, and the muscular portion of the fifth could not be examined. The other cranial nerves were all normal. **Diadokokinesis and Stereognosis** could not be tested. There was no bleeding from the nose, mouth, or ears, and no hemorrhage into either orbit. **Mentally** is very dull and frightened, and objects to having his right eye forcibly opened. **X-ray** shows a horizontal line of fracture, about 3 inches long running forward from the right occipital bone through the parietal, to end in the petrous portion of the temporal bone on this side. This is a linear fracture involving both tables, and is everywhere widely separated. **Lumbar puncture** showed a pressure of 28 mm. Hg. No fluid was withdrawn. **Operation:** Typical Right Subtemporal Decompression through a muscle splitting incision. Bony opening included all of the temporal bone beneath the muscle. Dura was not pulsating, dark blue in color, and very tense. Beneath the dura the brain was found moderately contused and lacerated and covered with old clotted blood. The cortex was flattened and injected, and the pial veins were enlarged in many instances to the
size of a normal external jugular vein. There was practically no cerebrospinal fluid in the subarachnoid space exposed at operation. Attempts to puncture the lateral ventricle through the temporal lobe and the third ventricle through the corpus callosum were not successful. There was a moderate herniation of the brain, but no splitting of the cortex. Wound was closed in layers with rubber tissue drainage to the floor of the middle fossa between the dura and the brain. The patient left the table in some shock, but soon rallied, and had an uneventful convalescence. October 11, 1921, comes for examination. Complains of slight dizziness on stooping, but otherwise O. K. Examination shows a distinct muscular weakness of the entire left side of body with a diminution of pain and tactile sensation also in this area. There is no paresis or paralysis nor anesthesia, however, and patient walks about all right. There is marked adiadokokinesis of the left arm. The cranial nerves are all normal, and the eye grounds show nothing but a slight blurring of the nasal sides of the discs. The decompression pulsates normally, is not adherent to the skin, and lies entirely beneath the temporal muscle. Fills but does not bulge on stooping. To do light work in a week. Blood Pressure 199/125. Last heard about in December, 1921, at which time he was back at work with no complaints.

M. A.—Female, white, 4½ years. Seen July 18, 1921. Diagnosis: Extensive fracture of Occiput—Extreme Compression of the Medulla verging on Oedema, with extreme Intracranial Hypertension—Laceration and Contusion of the Brain. Treatment: Right Subtemporal Decompression. Discharged: Dead. Patient said to have
been struck by automobile while playing on the street, on July 16, 1921. Carried home unconscious, where she remained until admitted to the hospital July 18, 1921. Vomited considerably during this time. Seen on day of injury by a local doctor (unknown) who is said to have given no treatment, but to have endeavored to have X-ray plates made of skull. He was not successful in this until the afternoon of July 18, following which the roentgenologist, recognizing the gravity of the case sent the child to the hospital, where she was seen late the evening of the 18th. On admission the pulse was 110, temperature 102 4/10, and the blood pressure 110/70. Some hours previous to being seen, 10 cc. of cerebrospinal fluid had been removed by lumbar puncture, and this fluid was colored with blood from a subarachnoid hemorrhage. 

Physical examination of heart, lungs, abdomen, and bony skeleton is normal. There are multiple contusions over the body. The pulse is around 110, temperature elevated, and respirations distinctly slowed and beginning to show a Cheyne-Stokes character. The entire scalp is very boggy. Blood Pressure, 110/70. Urine normal. Neurological examination as follows: Somatic Motor System: all arm reflexes, abdominal reflexes, and ankle jerks are absent on both sides. The knee jerks are present and equal. The left arm shows a complete flaccid paralysis. Somatic Sensory system could not be tested on account of patient's condition. Splanchnic Motor and Sensory systems: normal except that the left pupil is widely dilated and fixed. The right reacts promptly to light. Cranial nerves: The examination was incomplete on account of patient's condition. There was a definite paralysis of the right sixth. Both facial nerves appeared to be normal. There was a
marked choking of the left disc with dilated retinal veins, but no hemorrhages. The right disc could not be seen. Mentally the patient was unconscious and irrational, very restless, tossing all over the bed, and requiring constant watching, and restraint to keep her from throwing herself out of the bed on to the floor. She was noisy and cried and moaned constantly. Lumbar puncture showed a cerebrospinal fluid pressure of 40 mm. Hg. No fluid was withdrawn. X-ray not seen, but reported to have shown a linear fracture of the occipital bones, extending from above one ear and running around the back of the head to terminate beneath the opposite ear. Operation: Typical Right Subtemporal Decompression under local anesthesia. Muscle splitting incision and bony opening which included the entire squamous part of the temporal bone. On exposure of the dura it was found to be not pulsating, dark colored, and extremely tense. On wide incision considerable dark colored old fluid blood and clots escaped. The brain still did not begin to pulsate although there was no herniation and a successful attempt was made to enter the lateral ventricle with a blunt needle. Considerable bloody cerebrospinal fluid was drained off in this way, slowly, following which the brain volume diminished, and the pulsations returned. Previous to this the cortex was flattened, injected, and the pial veins much enlarged. The portion of the brain exposed at operation had not been lacerated, and there was practically no cerebrospinal fluid in this portion of the subarachnoid space. Following the drainage of the ventricle the patient's pulse rose rapidly, became of poor quality and before the scalp could be entirely closed she died.

The first of these two cases exemplifies a se-
vere type of brain injury treated late in the disease. The patient is again a useful member of society, and at present shows only a relatively small amount of permanent damage considering the severity of the injury, and the complicating diseases from which he suffered.

The second case is inserted as a contrast to the first, and because it exemplifies practically all of the mistakes in treatment that could possibly have been made in this type of injury. First: About sixty hours were allowed to elapse before a diagnosis was made, not to mention the fact that during this time no treatment had been instituted. This time was used to obtain relatively useless X-ray plates, which when obtained added nothing to the indications for or against treatment. Second: In a case obviously suffering from a high intracranial pressure 10 cc. of cerebrospinal fluid was withdrawn by lumbar puncture previous to any cranial decompression. This subjected the patient to the danger of immediate death from herniation of the medulla in the foramen magnum, and the fact that this complication did not arise is due to pure chance. Third: The operative procedure was badly planned. The operation of choice would without question have been a suboccipital decompression, and this was ruled out only because of the patient’s poor general condition. As a substitute a subtemporal decompression was performed. This I believe now to have been bad judgment. While a much less formidable procedure than a suboccipital operation, nevertheless, in view of the fact that the child was verging on, if not actually suffering from, an oedema of the medulla, and consequently even from the most favorable viewpoint only in a condition to stand the minimum of surgical interference, even a subtemporal de-
compression should have been recognized as being of too formidable a nature. Either the case should have been classified at once as a case of medullary oedema, and allowed to die without operative interference of any kind, or granting, as was thought at the time, that the patient was suffering only from an extreme degree of medullary compression, which had not as yet developed into an oedema of this portion of the brain, operative interference should have been limited to a simple ventricular puncture and drainage of the excess cerebrospinal fluid followed later on by either a subtemporal or suboccipital decompression, depending upon the amount of improvement following the ventricular puncture.

I wish to stress particularly, however, that this patient was allowed to go sixty hours without treatment, and without a diagnosis, and that in consequence she passed through at least two stages of intracranial hypertension, during either of which she would have been a favorable subject for such operative interference as would have offered at the worst an even chance of recovery.

The final stage of increasing intracranial pressure is that in which there is an oedema of the medulla itself. In this stage the regulatory centres of the medulla have become fatigued and the vasomotor stimulation will no longer raise the general arterial pressure sufficiently to force enough blood to the medulla to overcome the anaemia there. As a result the blood pressure drops rapidly while at the same time the pulse and temperature rise, the pulse becoming very weak and irregular, and the temperature reaching 105 and 106—the so-called brain temperature. The respiration also rises to 40 or beyond.
—oedema of the lungs develops, and the patient dies. In this stage nothing that can be done, no matter what it is, will save the patient. Consequently these patients should not be operated nor touched in any way; it merely hastens the end.

It can be seen from the above that the signs and symptoms—pulse, blood pressure, respiratory rate and mental state commonly used as indications governing diagnosis, treatment, and prognosis—all depend for their variations upon the basic factor of an increase in intracranial tension or pressure. If the intracranial pressure is measured, these subsidiary manifestations are not needed, and should not be regarded in any other light than as confirmatory evidence.

REFLEXES.

With regard to reflexes and their presence and absence, it is my opinion that they serve only as a guide to the point of maximum injury to the brain, and give us no indication whatsoever in regard to the patient's condition, and most certainly none in regard to the method of treatment.

BLOOD IN CEREBROSPINAL FLUID.

I have frequently heard it said that blood in the cerebrospinal fluid obtained by lumbar puncture means everything from a fracture of the base of the skull to a middle meningeal hemorrhage. I have further heard this referred to as an absolute indication, and also as an absolute contraindication to operation. Blood in the cerebrospinal fluid as obtained by lumbar puncture, providing it is not due to trauma from the puncture itself, means one thing and only one thing—namely—hemorrhage into the subarachnoid space. It has no bearing whatsoever
on the line of treatment to be adopted. As a matter of fact and experience, blood in cerebrospinal fluid obtained by lumbar puncture usually means a laceration of the brain providing the injury has been to the head.

OTHER SYMPTOMS.

Bleeding from the nose, mouth, and ears is often considered important from the point of view of treatment. This is not so. In the first place extra-cranial lacerations of these cavities must be ruled out. Having done that—and it is surprising how often this simple complication to a head injury will occur—bleeding from the mouth is practically always due to bleeding from the posterior part of the nose, the blood having dropped into the back of the mouth instead of running out through the nostrils. Bleeding from the nose means usually a fracture through the cribriform plate, and indicates nothing except that the prognosis should be extremely guarded on account of the liability to meningitis. This is due to the extension of infection through this fracture from the septic cavities of the nose and mouth. Scrupulous care should be taken to prevent this if possible. Bleeding from the ear may mean a fracture of the petrous portion of the temporal bone, and may carry with it injury to the seventh and eighth nerves on this side, possibilities that should be carefully looked into at the time the patient is first seen. Otherwise this symptom indicates nothing in the line of treatment, and directly affects the prognosis only in so far as to offer the possibility of a basal meningitis arising from neglect to keep the ear clean.

SURGICAL SHOCK.

Any patient who has received an injury to the
head severe enough to produce symptoms is in a state of surgical shock immediately following the injury. The condition and appearance of these patients in no way varies from that seen in a patient suffering from surgical shock following other severe injuries. Until these patients recover from this condition of shock, it is of no importance what other complications they may be suffering from. They must have immediate and careful treatment for their shock. This includes absolute quiet and rest together with warmth and fluids. If they are to have absolute quiet and are to be kept warm, that means that an extensive neurological examination and a lumbar puncture cannot be done. Neither of these examinations should be carried out at this stage. If the patient will not recover from the surgical shock it is of no importance to treat his lacerated brain. On the other hand, if and when he does recover from his shock, then there will be plenty of time to treat his lacerated brain without exposing him to unjustifiable risks. Patients who have been subjected to severe head injuries usually recover from their shock in from two to eight hours, depending on the extent of the injury, or else they have been so severely injured that they do not recover at all.

Careful observation of this rule in treating trauma to the head will save many patients who are now killed by too precipitate operating while they are still in a badly shocked condition.

An increase in intracranial pressure, then, is of paramount importance as an indication for operation in injuries involving the brain. From the foregoing we learn that there is a normal intracranial pressure of from 6 to 10 mm. Hg., depending upon the existence of a normal relationship between the secretion and absorption of
the cerebrospinal fluid. The cerebrospinal fluid is secreted by the choroid plexus, and distributed for absorption throughout the spinal and cerebral subarachnoid space. From this space it is chiefly—in fact almost wholly—absorbed into the venous circulation. A rise in intracranial pressure occurs when either the rate of secretion is increased, or the rate of absorption interfered with. A rise in the pressure of the cerebral venous circulation interferes with the rate of absorption of the cerebrospinal fluid. This rise may be produced in a variety of ways varying from a more or less temporary congestion of the meninges, to large extra or intra-dural blood clots, and subarachnoid, subpial, or intra-cortical hemorrhages. Depending upon the location and extent of these hemorrhages, localizing symptoms, such as reflex changes, paralyses, etc., may be produced. Depending on the amount of increase of the cerebrospinal pressure systemic symptoms involving the pulse and respiratory rates, blood pressure, changes in the eye grounds, and mental state may be produced. All of these objective symptoms are inconstant and variable, and the determination of their degree cannot be carried out with any accuracy. The degree of rise of intracranial pressure, on the other hand, can be measured with absolute accuracy, and the amount of the increase above the normal gives a constant and invariable indication as to what operative procedure must be undertaken to correct the intracranial pathology. Intracranial pressure as measured by the Landon Spinal Manometer, reading above 10 and below 16 mm. Hg. calls for repeated lumbar puncture with drainage of sufficient cerebrospinal fluid to reduce the pressure to normal. Readings above 16 mm. Hg. call for decompression, preferably subtemporal. No in-
individual suffering from an injury to the brain should be either treated or examined while in a state of surgical shock, and no such patient who can be demonstrated to have reached the final stage of intracranial hypertension—namely oedema of the medulla—should be operated upon, as the mortality in this latter class of case is 100% regardless of the treatment instituted. It is dangerous and often fatal to postpone treatment on a patient suffering from intracranial hypertension until X-rays of the skull are available. They can give no pertinent information that is not already at hand, and have no bearing on the operative indications of the case.

CONCLUSIONS.

1. The indications for operation in injuries involving the brain are three: compound fracture of the skull, depressed fracture of the skull, and a rise in the intracranial cerebrospinal fluid pressure.

2. The intracranial cerebrospinal fluid pressure depends on the relation between the secretory powers of the choroid plexus and the absorptive powers of the cerebral venous circulation.

3. Intracranial hypertension may and often does cause death in the absence of any injury to the bony coverings of the skull.

4. All cases that have received or that are suspected of having received an injury to the brain, no matter how slight, should, as soon as they have recovered from their surgical shock, have the pressure of their cerebrospinal fluid measured, and the treatment should be based primarily upon this finding alone.
REFERENCES.

5. Ibid., 144 et seq.
6. Ibid., 147.
7. Ibid., 150.
8. Ibid., 150.
9. Ibid., 150.
10. Ibid., 151.
12. Ibid., 54 et seq.
15. William Sharpe: The Diagnosis and Treatment of Brain Injuries: 50, 1926.
19. Ibid., 195.
21. Ibid., 173.