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Head injuries with special reference to the pressure syndrome

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SENIOR THESIS
UNIVERSITY OF NEBRASKA
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HEAD INJURIES
with
SPECIAL REFERENCE
to
THE PRESSURE SYNDROME

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INTRODUCTION

In 1933 the New England Journal of Medicine published an editorial stating that, "The diagnosis, prognosis and treatment of cranial injury, after head trauma are in an unsettled state." The editorial further stated that, "The more modern devices of treatment, such as the use of intravenous hypertonic solutions, repeated lumbar puncture and even subtemporal decompression of the brain are all methods not as yet proved to be either essential or even valuable in the treatment of this condition." Taken by itself such a sweeping condemnation of the present methods of treatment of brain injuries amply justifies critical examination. In addition it should not be forgotten that "cranial injury after head trauma" is no longer a problem reserved for solution by the specialist, be he neurologist or neurosurgeon. Thanks to the automobile every practicing physician is having these cases deposited daily, as it were, on his very doorstep and in the main has learned to care for them very efficiently, partly through personal experience and partly through perusal of the mass of literature that has collected on this subject in the last fifteen years. This experience has taught him that lumbar puncture judiciously used may be a life saving measure, that hypertonic solution given intravenously are very often a prompt and efficient method of meeting a serious surgical emergency. Also that subtemporal decompression used as a panacea in treating fractured skulls as advocated in the years from 1910 to 1920 up to as late as 1933 kills more than it cures. If such treatment by dehydration and lumbar puncture is not effective, and if diagnosis

and prognosis of this condition rests on such a shaky foundation, the physician should be so informed, however, if this situation is non-existent, that fact should be made apparent also for no academic question is involved. The correct answer affects the well being and lives of many hundreds of people, and the most efficient treatment should be a matter of common knowledge.

HISTORICAL

Surgery is the oldest branch of the healing art, for injuries must have been common events in the life of our remote ancestors. Evidence of ancient skulls shows that trephining was practiced in prehistoric times.

With the dawn of civilization came history (4), written records of events. Such record is the Edwin Smith Papyrus and Egyptian Medical Treatise of the seventeenth century before Christ. This papyrus contains the most important body of medical knowledge which has survived to us from ancient Egypt. The treatise is a part of an ancient book of surgery and external medicine which began its discussion with the top of the head and proceeded downward in orderly progression to the lower members. Forty-eight cases are described of which the first ten are head injuries. The prognosis of these cases is interesting and was always one of these three; 1. An ailment I will treat. (Favorable) 2. An ailment I will contend with. (Doubtful) 3. An ailment I will not treat. (Unfavorable)

The first group (cases one to ten) deal with wounds in the head of which the first seven are knife or sword wounds, penetrating to the bone. They vary in character according as the skull itself escapes injury or suffers contusion, a gash, or a fracture. In all of these cases the surgeon is instructed in the examination, "You should probe the wound." If he finds only a contusion of the skull or no injury the verdict is favorable, gash in the skull, doubtful, a fracture, unfavorable. The examination in one case notes a feeble pulse and a fever as a symptom. That skull injuries were sometimes healed there can be no doubt since healed skulls have been found.

Case eight deals with a "fracture of the skull under the skin." When examination has demonstrated the presence of the fracture, the surgeon is unconditionally charged to operate, to open at the point of contusion and to elevate the depression outward. It may be for the modern surgeon to decide whether this could be done without trephining. If trephining was involved as would seem possible, we have here, the earliest reference in literature to this famous operation. Although an operation known to have been practiced by peoples in a primitive state of culture, trephining has not yet been unmistakably identified on surviving ancient Egyptian skulls. The attitude of the physician, as gleaned from these records, is distinctly that of cooperation with nature.

Hippocrates (500B.C) in the section of his writings on, "injuries of the head" recommended trephining in order to "slacken the tightness of the head." (1) In other words, to decompress in cases of inflammation and to allow escape of blood or matter, and to remove foreign bodies. He believed that in contusions, the internal structure of the brain is extensively injured, and that irritation with hypertrophy, are the consequences. He advocated instrumental interference, in order (as stated above) to give more room to the brain and relieve from its state of compression. This, no doubt, was the rationale of his practice also in simple fractures, not attended with depression. His object in perforating the skull was to remove tension.

Apropos at this time, should be a few words on the constitutional treatment by Hippocrates, no mention of which was made in his treatise - that of venesection in the treatment of injuries of

the head. - Now certainly it does not appear that Hippocrates regarded bleeding as necessarily forming a portion of the system of treatment in injuries of the bones of the head anymore than in those of other bones. But, although these were his views, it can be as little doubted by anyone who is acquainted with his general views of practice, that he bled whenever abstraction of blood was indicated. We know, for example, that in pains of the back part of the head he opened the temporal vessels and that in all inflammation and fevers he extracted blood freely. And that Hippocrates enforced the depletory system of treatment in injuries of the head, when pain and fever intervened is quite obvious from its having been the system pursued in such cases by all subsequent authorities who looked up to him as their great guide in practice. Further proof of above is that Ambrose Pare', who was a great advocate for depletion in treatment of skull fractures, goes to great pains to show that he has Hippocrates on his side in support of this practice.

In 30 B. C. Celsus recommended trephining for extra dural hematoma. (23)

LaFranchi, of Milan in 1296 A. D., in his *Chirurgia Magna* was the first man to describe concussion of the brain and his chapter on the symptoms of fracture of the skull is accounted a classic. Depressed fragments and irritation of the dura are his only indications for trephining.

Paracelsus (1483 to 1541) native of Switzerland, knew of paralysis and disturbance of speech after head injuries.

Wilhelm Fabry of Germany (1560-1624) in his "Century of Surgical Cases", showed that head injuries may cause insanity.

Hendrick von Roonhuysen (1663) of Amsterdam, treated wounds of the head without trephining.

Jean Petit, (1674-1750), French Surgeon argued that if an intracranial effusion could be diagnosed, operation should be done to remove the cause of convulsions.

Percival Pott (1714-1788), (23) of London wrote a lengthy treatise on head injuries (1760) with case reports. In his treatment of cranial injuries he recommended, "phlebotomy and an open belly." This appears to be the first record of using pergartion in the treatment of head injuries, one which is popular today, but which was undoubtedly not looked upon by Pott as efficacious is dehydration. Pott was also, apparently the first man to say that symptoms arising in head injury are not due to fractured skull, but to injury to the brain. That, of course, was in the pre-antiseptic period. His method of attack included making mutiple trephines along the line of fracture. In one case he did twenty-six trephines.

The following is a list of inferences from a careful study of all principal authorities who have written on head injuries from Hippocrates down to the present time:

1. All the serious injuries of the skull may be arranged conveniently under contusions, simple and depressed fractures.
2. Hippocrates recommended skull perforation in cases of simple fractures and contusions, whenever he thought these would be followed by serious consequences.
3. Hippocrates operated in these cases during the first three days, before onset of serious symptoms. Celsus

rejected this idea, not operating until onset of symptoms.

4. The objects which Hippocrates had in view by perforating the skull, would appear to have been to slacken the tightness of the skull and procure evacuation.
5. The object for which Celsus opened the skull would appear to have been solely to remove irritation.
6. All the ancient authorities looked upon contusions and simple fractures as being formidable injuries, which produced brain congestion with inflammation.
7. In modern times, at least within the last one hundred years, the trephine has never been applied in the above cases; it is done to relieve symptoms not present.
8. Much contradiction of the above principles was in evidence by French surgeons during the eighteenth century.
9. Hippocrates did not operate in cases of depressed fracture.
10. Celsus removed all spicules at once, raised or removed the depressed pieces.
11. Pott trephined. Mr. Abernethy cautioned not to interfere until necessary.
12. In 1886 the operation of trephining was condemned by most workers in the field.

So concludes the historical sketch.

PATHOLOGY AND PATHOLOGICAL PHYSIOLOGY

Despite the safeguards offered by the construction of the skull the brain suffers all degrees of injury at every age. (13) Following a blow the brain is set in motion and strikes the sides of the skull. Perhaps the cisternae offer a degree of protection at the base of the brain but this is very doubtful. Indeed it is open to serious doubt whether the cisternae are not more harmful than serviceable in cranial injury, for they offer a large space into which bleeding can occur, and they certainly offer a tremendous risk to life when a cerebrospinal fistula results. Certainly, elsewhere, a great bulk of the brain is afforded no protection whatever by the fluid.

The brain is very sensitive to trauma and the white matter far more so than the cortical grey matter. This fact is in evidence when operating on the brain, the white matter tolerating far less trauma. This fact also explains the high proportion of severe injuries below an intact or nearly intact surface of the brain.

The end products of trauma to the brain are precisely the same as those of trauma elsewhere, namely edema, hemorrhage and destruction of tissue. And just as following trauma elsewhere the relative proportion of edema and blood varies tremendously. Moreover all tissue becoming necrotic through injury acts as a foreign body and demands more blood and edema in surrounding areas to promote absorption and repair.

This total new volume of blood and edema obviously requires intracranial space, and since the volume of the cranial chamber is fixed, compensation is possible only from within, i.e., through reduction in the amount of the normal intracranial content

of blood, cerebrospinal fluid and tissue fluids. If the injury is not severe an adequate adjustment can be made and life preserved if the quantity of edema and hemorrhage is too great for space compensation, death results from intracranial pressure. The ultimate cause of death is anemia of the life-controlling centers which are located in the medulla. There are many cases in which death is also due to the direct injury of this important part of the brain and not to generalized increased intracranial pressure.

It is impossible to traumatize the brain without producing transudation of fluid. This effect may be local or diffuse. The edema may remain without the brain tissue, and if so, the white matter is always more effected than the cortex because it is less resistant. When edema occurs an increased volume of the brain results. The fluid may also find its way from the brain tissue through ruptures in the cortex or meninges and accumulate in the subdural space. In severe injuries an enormous quantity of fluid may form in this otherwise potential space. The fluid is sometimes slightly tinged with blood. There is much reason to believe that much of this fluid may arise from the primary injury of the vessels on the surface of the brain.

In a study of 1494 cases of head trauma Munro (40) states that excepting in pure concussions, the primary reaction of the brain to injury is like that of soft tissues anywhere in the body. There is a congestion and edema and a rise in the pressure of the cerebral venous circulation. This is accompanied by a concomitant rise in intracranial pressure. If this process is continued for

long there is anoxeima and tissue damage resulting. Munro contends that increased intracranial pressure does not aid in prevention of intracranial bleeding.

Injuries of the brain and intracranial hemorrhage are the two main complications in head injuries, according to Rawlings. (41) Important are the cortical veins, thin walled, and elastic to allow participation in normal variations of intracranial pressure. When ruptured, the blood from these veins flows by gravity to the medullary centers. Hemorrhage has an effect on the flow of spinal fluid, continues Rawlings. The spinal fluid is absorbed at a constant rate into the venous system. The pressure of the fluid is just above that of the venous blood; also the specific gravity of spinal fluid is less than that of blood.

The delicate arrangement is upset in head injuries, namely

1. There is a loss of absorbable area.
2. The venous engorgement and rise of venous pressure in the cranium upsets the ratio between secretion and absorption.
3. The blood effused into the pia-arachnoid space impedes the flow of fluid. There is a resultant damming back of the spinal fluid, a definite and generalized brain edema and tendency to dilatation of ventricular spaces.

Apropos in this place is a word on the physiology of the spinal fluid. The source is in the choroid plexuses of the lateral and third ventricles. From these cavities circulation takes place downward to the cisterna, then up over the surface of the midbrain thence upward through the cleft in the tentorium cerebelli spreading out over the cerebral hemisphere in the subarachnoid spaces. The

fluid is returned to the venous circulation through the arachnoid villi. The average amount of fluid varies from 120 to 150 cc.

The normal circulation of spinal fluid may be interfered with by a number of factors causing obstruction to the flow in any part of its course. A common and potent factor in intracranial pressure is traumatic edema. This always follows injury. Swelling appears after trauma and reaches a maximum in twenty-four to forty-eight hours, according to Jackson. (27) This swelling subsides gradually and disappears in from three to seven days in favorable cases. During this time intracranial tension is raised and paths of fluid are obstructed. Swelling is more pronounced in the vicinity of the areas of contusion. Edema is like that elsewhere in the body following injury, but the brain being encased in a non-expanding dura mater under a rigid, bony vault swelling can proceed only to a limited degree and then only by encroachment on ventricles and subarachnoid spaces. Then pressure on the vessels must occur and anemia of the brain follows. The spinal fluid is formed still and the dilated ventricles pressing out in all directions against an already swollen cortex adds to the pressure of the brain against the dura. There is now stasis of spinal fluid circulation. Unless this vicious cycle is broken at a point below the tentorium early enough to relieve the medulla, death must occur. Normal spinal fluid pressure is from 7-9 in adults. In serious cases it may rise to 60 mm. HG. Subtemporal decompression does not give adequate relief to the cerebrum or medulla.

In another article by this same author (26) he states that

the most frequent change in the brain found in persons dying from fracture of the skull is traumatic edema. Here the convolutions are found flattened suggesting pressure of the brain against the dura and skull and the disappearance of the subarachnoid water-bed. The cerebral vessels are empty and flattened suggesting an anemia of the cortex. The peripheral ends of the sulci were closed obliterating the normal cisterns of cerebral spinal fluid. The fluid in the meninges was lessened and the arachnoid almost dry when the dura was first removed.

Cannon, (8) the famous Harvard physiologist, states, "The secondary increase in pressure is due mainly to three factors: deprivation of normal nutrition in injured parts, passage of water into these parts with consequent swelling and and the rigid enclosure of the brain, causing the swelling in one region to effect markedly near regions." The thromboses, extravasations, and hemorrhages which accompany contusion, impair the blood supply of the injured region, especially since the nutrient arteries of the brain are terminal.

Brain tissue deprived of blood undergoes chemical changes resulting in greater internal osmotic pressure and the passage of water in to the tissues. Brains placed in normal salt solution increase continuously in weight even to 33%. The swelling which the tissue undergoes must cause it to compress neighboring regions, and thus further impair the circulation so that new regions are involved. Thus is formed a vicious cycle. The main force effective in causing swelling is probably osmotic pressure, which, in brain tissue, may

attain a degree so much greater than blood pressure that the blood would really be prevented from entering the cranium.

According to some authors the increased intracranial pressure resulting from an injury is effective in compressing bleeding vessels in the skull. Munro (40) says this is a pernicious teaching and untrue. He states it has been proven that cerebral venous and capillary pressures are approximately equal to intracranial cerebrospinal fluid pressure, and that they rise and fall with it. Thus high intracranial pressure can not cause hemostasis without causing anoxemia and necrosis of the brain. He says that lowering intracranial pressure by lumbar puncture decreases venous bleeding because venous pressure drops with lowered cerebrospinal fluid pressure.

SIGNS AND SYMPTOMS

How can we know from clinical observations the exact state of intracranial pressure? The following observations give the desired information according to Dandy. (13) 1. The state of consciousness. This is the most important observation. We know if a patient is unconscious that he has intracranial pressure beyond the limit of compensation. If the patients consciousness is returning, we know that cerebral pressure is being reduced and the patient is improving. 2. The pulse and respiration are taken every ten to fifteen minutes and charted in curves. A slow pulse and slow respiration are indications of intracranial compensation. A temperature below 101.5 or 102 F. is another valuable indication of a patients safety. A broken compensation or an impending break in compensation is shown by vacillations in the pulse rate and the respiration rate also. One of the most important and most delicate indices of intracranial pressure is the temperature. Blood pressure records are of little value.

Two other observations are of utmost importance: 1. Restlessness 2. Involuntary micturition and defecation. Restlessness means that the patient is either coming out of coma or going into it. Incontinence of urine or feces occuring when the patient is seemingly conscious means a break in cerebral compensation.

Cases of increased intracranial tension show a gradual fall in respiratory rate and rising temperature readings. In the early stages, Fay (16), says that the pulse rate too will fall rapidly with respiratory rate. Later the pulse may rapidly rise to 160, and over, in the final stages of severe concussion. It is, therefore,

necessary to regard carefully the respiratory rate as an index of intracranial tension. The fall may be gradual to zero, or Cheyne-Stokes respiration may supervene.

If the cases seen sometime following the injury, the pulse may have passed over its original depressed rate to a second phase with rapid pulse which simulates so closely that seen in shock. In this stage, however, two things will help to differentiate the condition from that of true shock. The respiration will be found to be below normal and the temperature rapidly ascending with the pulse. As is well known in shock, the reverse is found and the respiration that of air hunger and above the normal rate; the temperature well below the normal line and the pulse which is rapidly rising.

Dilated and fixed pupils in my experience, (Fleming) (20) are invariable a sign denoting a fatal outcome. Dilation of one pupil, in the majority of cases, is on the side of maximum damage to and pressure on the brain. The response of the patient to firm palpation about the head will frequently localize the fracture even though the patient be in deep stupor. Cervical rigidity, soon after the accident, suggests injury to the neck or a considerable amount of blood in the spinal fluid. Increasing weakness, impaired sensation, changed or pathological reflexes point to the sight of major injury to the brain. As a rule, neurological findings are a far more reliable indication of localization than visible injury or roentgen evidence of fracture.

Browder and Myers (7) have presented a paper to show that the relationship of the systemic blood pressure, the pulse rate and

the cerebrospinal fluid pressure following a severe injury of the head, should not be accepted as the basis for diagnosis, prognosis, nor treatment in an individual so injured. They point out that Kocher, the German (1901) on the basis of his animal studies, subdivided the progressive manifestation of brain compression into four stages, namely:

1. The stage of accomodation.
2. The stage of early manifest symptoms.
3. The stage of advance manifest symptoms.
4. The stage of medullary collapse.

These ideals were accepted and applied unqualifiedly in clinical medicine and surgery as the basis for diagnosis, prognosis and treatment. These authors state that Cushing several years later was the first man to bring up serious doubts as to the ideas of Kocher. Eyster (1901) stated that intracranial pressure of less than the mean blood pressure (90-110 mm. HG.) has little or no effect upon blood pressure, pulse rate, or respiration. Trotter (1927) and Howe (1929) expressed the same thought.

Following these experimenters, the authors continued, "The majority of writers and teachers have concurred on the following Points:

1. The essential clinically important effect of many intracranial lesions is the production of increased intracranial pressure.
2. Increasing intracranial pressure produces its general and vital effects through the medullary centers.
3. The symptoms of increasing intracranial pressure are:
 - a. Rise in blood pressure

- b. Slowing of pulse.
- c. Slowing of respiratory rate.
- d. Rise in temperature
- e. Headache.
- f. Vomiting.
- g. Stupor, coma.
- h. Papilledema.

4. Death results from over stimulation of medullary centers under the influence of steadily increasing intracranial tension."

Many neglect, the authors go on to say, the point that the experimenters have repeatedly emphasized, namely, that the rise of intracranial pressure must reach the level of the systemic blood pressure before medullary compression symptoms arise.

In many of our experiences, they continue, with traumatic cases they were surprised to note how frequently and how completely they were misled by placing reliance on the forgoing signs.

After reviewing the different opinions of many authorities on the subject, the authors decide that not all of these views can at once be correct since many are openly contradictory. They find the only point of agreement is that it is the intracranial pressure which brings the patients into trouble and that, this being so, it is the item which merits prognostic and therapeutic attention.

The conclusion drawn by these men are that they feel quite strongly that the surgeon who regards his traumatic brain case through the orthodox schema, which has taken a permanent place in

the literature, will be misled more times than properly guided by it. It is dangerous because it gives him a sense of false security in handling the case. They are unable to suggest any "Pattern" as a substitute for the one negate. They urge formal abandonment of the Kocher scheme and suggest extensive study of each case as a whole on its own merits.

GENERAL TREATMENT

Each year about 125,000 injuries involving head and spine occur in these United States. The number is increasing year by year because of the increased speed of transportation and mass production in industries. For hundreds of years Medicine and Surgery have tried to develop the standardized treatment for injuries of the head and have so far been unsuccessful. So much confusion exists today in regard to proper methods of treating cerebro trauma, fractures of the skull and intracranial pressure that the physician is frequently in doubt as to which course to pursue. Classifications, warnings, indications, contra-indications, surgery and symptomatic treatment are advocated on all sides.

It has been necessary to abandon many of the older fixed ideas regarding cerebral trauma. The recent advances and wider understanding involved by neurosurgery have made this possible. The vast majority of cases that die from head injuries succumb to intracranial pressure or hemorrhage. A few may develop complications such as meningitis, pulmonary edema, pyelitis, etc., but the matter of greatest concern should be the prompt treatment and control of intracranial pressure and hemorrhage and the early recognition of the symptoms that require specific means of relief.

Dandy (12) says the first and paramount concern with acute head injury is preservation of life, the second preservation of function. The immediate cause of death is intracranial pressure. The effects of injuries are immediate and late. Extradural hematoma is the only lesion which may be treated in the acute stage. In all other cases the cause is ignored and the effect (the pressure) is treated. Nature attempts to combat swelling by removing spinal

fluid. The doctor should know when nature cannot compensate further by the study of the signs and symptoms. Conservative treatment is best. Rest in bed is essential for treatment. Seventy per cent of patients recover in spite of any treatment. If left alone the remaining thirty per cent will die. Dandy says that twenty per cent can not be saved by any means. The patient should be strictly left alone until one knows that nature is unable to cope with the situation. There is no panacea for head injuries.

In a study of 1,494 cases Munro (40) concludes that the immediate prognosis of cerebral injury is good. In his series of 1,494 cases the mortality was 17%. He goes on to say that the only choice in treatment has been between the extreme of watchful waiting with rest in bed, and of operative interference. This operative interference was usually in the form of a subtemporal decompression. More recently, according to the same author, with the use of hypertonic and hypotonic solutions intravenously and saturated solutions per rectum, in association freer use of lumbar drainage checked by manometric readings the pendulum has gradually swung away from these earlier methods. Of course in the rush of enthusiasm tending the adoption of these new methods there have been many instances of misapplication. The failures he says should not be blamed on the method but on there improper use.

Rawlings (41) gives some useful points to remember. 1. As a general rule it is wise to avoid an expression of opinion as to the probable outcome of any given case until a careful clinical investigation has been carried out. 2. There is a good deal of glamour about a "fracture of the skull." We are constantly hearing that

some person died from it. This is far from the truth. Death is nearly always due to laceration of the brain or intracranial hemorrhage.

There seems to be considerable argument as to whether morphine should be used or not. Munro (40) says he is definitely against the use of morphine when there is any indication of increased intracranial pressure because it only aids in embarrassing an already depressed respiratory center. According to Kennedy and Wortis (33) this drug along with glyceryl trinitrate, hypotonic solutions and the assumption of the horizontal position all cause heightened pressure within the skull. This is also true as regards coughing, straining, struggling, and ether and anesthesias. On the other side of this argument we find King (34) recommending morphine along with paraldehyde, phenobarbital, and nembutal for sedation.

Such an authority as W. W. Babcock (2) cannot possibly be ignored in connection with our subject. His routine treatment of fractured skull says "record patients condition as to conscious, coma, spasm, or flaccidity; noting pupils, pulse, respiration, temperature, and blood pressure. If the systolic pressure is below 60 mm. of HG., the patient should be treated for shock, by rest, quiet, inclination of head of the bed. Open wounds are filled with dichloramine-T, packed with gauze, the surrounding skin painted with iodine, and an occlusive dressing applied making sure that hemorrhage is arrested.

No case admitted to the hospital should be considered hopeless until every measure has failed to maintain or prolong life. Fay (18) says it is astounding to find that cases which would have

been considered hopeless five years ago have recovered, while even cases that appeared to be expiring at the time of admission may respond sufficiently to permit the satisfactory management of the hemorrhage and pressure and their ultimate recovery.

From the time of admission the patients temperature, pulse, and respiration are taken every fifteen minutes, blood pressure every half hour and upon every patient admitted there should be done a diagnostic lumbar puncture, which affords the opportunity of determining with a mercury manometer the exact intracranial pressure of the cerebrospinal fluid and whether or not bloody spinal fluid is present. The entire course of treatment is determined by these findings and their relation to one another.

Shock is the first consideration above all else and this is determined by (a) subnormal temperature (b) diastolic pressure below 60 and (c) pulse usually above 120. Extremities are cold and wet. Treatment of shock consists of warm, dry clothing, heat applied to the body surfaces, atropin and pituitrin.

DEHYDRATION TREATMENT

ENTERAL

In the American Journal of Physiology of October 1920, Foley and Putnam (21) reported the results of their experiments on cats in which they showed that the introduction of hypertonic solutions into the gastro-intestinal tract had the effect of reducing cerebrospinal fluid pressure and diminishing the bulk of the brain. They stated that this route of administration was more convenient, and by their use the disturbances of circulation and respiration common with intravenous infusions was avoided.

They further proved that saturated solutions of magnesium sulphate which is not absorbed from the gastro-intestinal tract produced the same results as when absorbable salts were used.

To the above men cannot be given the credit of being the first to show the value of hypertonic salt solutions by bowel for reduction of intracranial pressure. This honor rightly belongs to Weed and McKibben in 1919. The above men merely confirmed the work of the two later workers. First clinical application of these findings were made by Haden, Cushing, Foley, and Sachs in 1920.

Shortly after the introduction of the above method of dehydration it was adopted by the neurological service as a means of reducing intracranial pressure and volume, following the observation by Temple Fay of Philadelphia that infants showed a marked retraction of fontanelles after its use, because of the dehydration that it produces through out the cerebral system. (17)

In cases of traumatic injuries to the brain in which fracture of the skull or concussion is associated with marked stupor,

respiratory and cardiac depression, due to intracranial pressure and "medullary edema," dehydration by Magnesium Sulphate is followed by marked relief of these symptoms.

Fay says that the old adage, "If pulse pressure crosses pulse rate that is the time to decompress," has been supplanted by the more modern method which may be substituted in this statement, "When the pulse pressure crosses the pulse rate that is the time to dehydrate."

The use of Mag. Sulphate in proper doses by mouth or rectum effects a rapid withdrawal of fluid from the body. (18) It is evident that Mag. Sulphate will be powerless to produce the desired result if the patient is permitted fluids by mouth, vein, or otherwise as the fluid thus given rapidly replaces that claimed by the Mag. Sulphate. The physician frequently desires to deplete blood volume by the use of hypertonic solution realizing that the circulatory system will turn to tissue fluids to replenish its supply. This will not only temporarily prevent the further elaboration of cerebrospinal fluid but actually withdraw from this reservoir and materially reduce intracranial pressure. In order to perfect this process there should be no available added fluid, so that the circulatory system must turn to the interstitial compartment for its replenishment of volume. This method is ideal in cases in which no bloody fluid is found. Mag. Sulphate of course should never be given until all signs of shock are past.

Hypertonic salt solution has been found to be indispensable for treatment of cases in which cerebrospinal pressure is increased, due to acute or chronic lesions within the cranial cavity.

T. Fay (16) had an opportunity to observe and compare the relative values and merits of Mag. Sulphate and sodium chloride by bowel for relief of intracranial tension. He says in his clinic he has come to rely on Mag. Sulphate in preference to Sodium Chloride.

In order to establish the relative values of these salts it was necessary to understand their characteristic actions in the gastro-intestinal tract experiments on dogs proved that Mag. Sulphate was almost twice as efficient as sodium chloride under identical circumstances.

The use of sodium chloride by bowel clinically was found to be attended with great discomfort, such as thirst, nausea, and the effects were transitory and not so complete as those obtained from the use of Mag. Sulphate. It would appear, he states, that an explanation lies in the fact that sodium chloride is dialyzable when in the intestinal tract, and that its influence is first exerted on the vascular bed about the intestinal walls with the extraction of fluid from the circulation. Hypertonic sodium chloride, being hypertonic to the Cl. content of the blood, is rapidly absorbed and enters the blood system, which in turn becomes hypertonic, and therefore extracts fluids from the tissue spaces, and especially ventricular system.

In the mean time the sodium chloride of the blood stream has entered the tissue spaces, including the ventricular system. It is apparent that as soon as the blood sodium chloride falls due to elimination via the kidneys, below that of tissue sodium chloride content, the reverse processes will become manifest and fluids from the blood stream then add water to the tissue bound sodium chloride

and cause a secondary wave of tissue edema with an increase in intracranial pressure.

Mag. Sulphate, on the other hand, is non-dialyzable and, not being absorbed into the blood stream exerts its entire effect on the vascular bed about the intestinal wall; hence the direct extraction of fluid from the circulation and the secondary withdrawal of ventricular or tissue bound fluids brings about dehydration, for the circulation calls on all the available body fluids to compensate for the depletion in blood volume by way of the intestinal tract. This, Fay believes, is the reason for the exception results obtained from the use of Mag. Sulphate.

Great care must be taken in the treatment of acute traumatic cases to distinguish between cases which show symptoms of intracranial pressure and those with increased tension plus profound shock. In the treatment for concussion and intracranial tension, dehydration is necessary to relieve the cerebral congestion, and Mag. Sulphate may be used to deplete blood volume and combat increased fluid tension of the blood. If, however, following head injuries, as is so frequently the case the patient has lost large quantities of blood, or sustained injuries which have produced a profound state of shock, then dehydration is contra-indicated and, in fact, the use of an agent to deplete an already impoverished and failing circulatory system is but to hasten the final termination of the case.

The criterion by which these two conditions may be distinguished lies mainly with the respiratory and temperature curves.

In checking conditions following injuries with loss of blood and shock, fluid volumes, must be maintained and increased rather than depleted. In this type of case the dilemma as to which phase to treat actively, that of intracranial tension or that of shock, usually solves itself in that the shock condition of the patient prohibits progressive edema of the brain, and the case must be carefully watched so that fluid may be added, or withdrawn as the symptoms of one or the other become serious.

Fay (16) recommends the use of one and one half ounces of crystals of Mag. Sulphate by mouth, every fourth hour, if necessary, or if the patients condition makes it impossible to give the drug by mouth because of vomiting, stupor or delirium, then the administration by rectum of six ounces of fifty per cent solution, every four hours. He says the beneficial use of salt in cranial injuries has been well established in his clinic.

The author warns against over dehydration by active use of hypertonic salt solution. He says cases with signs of over dehydration can be saved by forcing fluid. He continues that the respiratory rate seems to be the most reliable index of intracranial pressure disturbances. He has seen no cases of Mag. Sulphate poisoning. Since inaugurating the use of Mag. Sulphate for dehydration, he states it has become unnecessary to subject patients to the added operative risks of decompression and the consequent disfigurement.

In the opinion of Earl (15) the most efficient drug for reducing intracranial pressure is Mag. Sulphate as a retention enema. Two to four ounces of a saturated solution given slowly by gravity

through a soft No. 10 French catheter is advisable. The enema should be repeated every three hours for four doses. Then wait twelve hours. Mag. Sulphate can be given by mouth but often causes vomiting according to this author.

Munro (40) after a study of 1,494 cases of head trauma concludes that all that any hypertonic solution can do, when given intravenously or by bowel, is to reduce brain volume. The intravenous method acts more rapidly but its effect is more transient. Of course these methods of dehydration reduce intracranial pressure by shrinking the brain but still there exists an acute hydrocephalus due to mechanical blockade of absorptive channels. Therefore complete treatment calls for more than these procedures. Furthermore, dehydration may be dangerous unless its effects are measured. Also those who have used intravenous therapy recognize the devastating effect of the secondary wave of cerebro edema which follows therapeutic brain shrinkage. When properly limited to cases of edema or as a preliminary emergency treatment in cases of contusion and laceration both 50% glucose by vein and saturated Mag. Sulphate by rectum (3 doses) will prove to be of real value and will often be actually life saving measures. Concomitant manometric readings are essential.

Ralwings (41) recommends use of rectal infusions of hypertonic solution of not over ten to twenty per cent.

"Serious injury of the chest or abdomen with internal hemorrhage having been eliminated by examination, 60 Grams of Mag. Sulphate in 120 cc. of water are introduced by rectum, and the patient watched until the temperature reaches normal and the systolic pressure

is 80 or above." In all cases the Mag. Sulphate enema is repeated every three hours to reduce the intraduro pressure, and to prevent secondary cerebro edema. (Babcock) (2)

Before proceeding to the discussion of the intravenous method of dehydration, a few words concerning some very applicable and less widely known procedures directed toward the same end should not be amiss. Such methods include restriction of fluids, posture in bed, and the use of certain specific drugs. On the subject of fluid restriction Fay (18) has some definite advise. He recommends a strict fluid limititation to 30 ounces total intake per day. This includes coffee, tea, milk, soup, fruit juices or liquids in any form, and has found to be approximately sufficient to meet necessary requirements. An exact intake and output record is maintained upon the patient until the second week. Thus it is possible to know at all times the actual fluid status of the patient. The exception to this fluid restriction is the demonstrated presence of blood in the cerebrospinal fluid. It is necessary to allow enough additional fluid so as to permit daily spinal fluid drainage and favor washing out of red blood cells. About 30 ounces is ample for this purpose. Stupor or unconsciousness should not be a matter of days, but of hours, when proper fluid balances are maintained.

Earl (15) syas restriction of fluids is essential while acute symptoms are present and for a time after they have subsided, but in most extreme cases they should not be reduced below 500 cc. in twenty-four hours.

Kennedy and Wortis (33) suggest two measures which apparen-

tly have been overlooked by most authors on the subject of head injury. These are attention to posture in bed and the use of Caffeine Sodibenzoate for reduction of increased intracranial pressure. This drug injected hypodermically or intravenously in doses of $7\frac{1}{2}$ grs. (0.5 Gm.) reduces intracranial pressure for a period of 45 minutes according to these authors. It may be repeated every two or three hours, and may well be inter spaced between rectal taps or intravenous injections of hypertonic solutions. The pressure - reducing effect of caffeine has been measured by final manometer; within three minutes of injection, spinal fluid pressure drops from 20-50 mm. in the tube. There is no change in the systemic blood pressure during these events.

As to posture in bed the latter authors in this article (32) state that it was considered good practice up to the year 1931 in the management increased intracranial pressure, especially when the pressure is due to skull injury, to keep the patient horizontal with the head as the same level as the heart. This has been proven to be based not on sound physiology. Accordingly, these authors state that for many years at Bellvue Hospital they have placed such patients in Goetsch beds with the head and trunk elevated at an angle of from 15 to 60 degrees from the horizontal position of the rest of the body.

DEHYDRATION TREATMENT

1. INTRAVENOUS NaCl.

In 1919, Weed and McKibben (45) after a series of experiments on cats, first called attention to the fact that it was possible to decrease the pressure of the cerebrospinal fluid by the intravenous injection of hypertonic solutions.

Haden (24) was the first to publish his clinical application of the intravenous method of brain dehydration. He administered a concentrated hypertonic solution to two patients with meningitis and obtained some improvement in their condition, which he attributed to a lowering of the intracranial tension.

In the same year Cushing and Foley (10) followed the work of Haden with a demonstration of the possibility of decreasing the brain volume by the intravenous injection of hypertonic solutions.

Sachs and Belcher (42) were able to ameliorate the increased intracranial tension occasioned by a brain tumor through the intravenous injection of a concentrated solution of sodium chloride.

The following will help to explain the physiology of intravenous dehydration. The immediate result of introducing any hypertonic solution intravenously is an increase in the fluid volume of the blood greater than the volume of the solution injected. (16) The hydremia is due to withdrawal of water from the tissues into the blood, a response to the increased osmotic pressure of the latter. The volume of fluid taken up by the blood is said to be directly proportional to the increase in molar concentration resulting from the

injection, but it is apparent that the rate at which the injected substance diffuses into the tissues must be taken into consideration since the movement of fluid into the blood will cease as soon as osmotic equilibrium is established. The osmotic effect on such substances as NaCl. and glucose, which pass through the capillary wall with ease, is greatly curtailed by diffusion. The effect which hypertonic solutions given intravenously have upon the cerebrospinal pressure is of course only one part of their physiological action.

Rawlings (41) in his book, "Head Injuries," states that intravenous hypertonic solutions do lower intracranial pressure within fifteen minutes to thirty-four hours. He says that such dehydration can only be harmful if the brain is damaged and if intracranial bleeding is taking place. He contends that indiscriminate use of hypertonic saline can only do harm.

"If intracranial tension increases, as indicated by respiratory depression and increased pulse pressure, 100 cc. of a fifteen per cent solution of NaCl. are given by vein at the rate of 10 cc. every five minutes." These are the words of Babcock. (2)

Intravenous therapy is not recommended by King. (34)

According to Browder (5) Foley and Putnam (21) (1920), found by comparing results obtained from intravenous and gastrointestinal route of administration, that the extent of fall of cerebrospinal pressure is approximately equal with both methods.

Foley (1921) (22), calls attention to the fact that there may be medullary depressant effects during or immediately following intravenous hypertonic solutions, which are not observed when the

gastro-intestinal route was used.

Browder (5) states that since 1919 the methods of treating increased intracranial pressure following trauma, began to change from time honored subtemporal decompression or repeated spinal drainage to intravenous and rectal hypertonic solutions.

Observations of spinal fluid pressure over periods of from 5 to 8 hours continuously after intravenous hypertonic NaCl. were made by Mills and Hurwitz (37) on a number of patients. They summarize their results as follows:

1. The reduction of cerebrospinal pressure with single doses of hypertonic salt and dextrose is transient.
2. A secondary rise occurs which reaches a point much higher than the original level.
3. High concentration of NaCl. are definitely toxic.
4. In view of these facts, it is probable that recurrent pressure symptoms and the occasional deaths reported are due to secondary rise in spinal fluid pressure.

A point often overlooked is brought up for consideration by Stultz and Stricker (44). It is a matter of fact that the pressure of the cerebrospinal fluid is lowered in some cases of cranial trauma. In these cases the evacuation of cerebrospinal fluid by lumbar puncture leads to an exafferation of the nervous accidents while on the contrary the raising of its pressure has at once a favorable influence and transforms the condition of the patient. Hypotension of the cerebrospinal fluid therefore seems to some extent at least, to play a prominent part in the symptomatology of cranial traumatism, and the

actual existence of a well individualized syndrome of acute hypotension of the cerebrospinal fluid seems to be established beyond a doubt.

M. Leriche of France (1920-1922) was the first to point out these hether to unknown facts. Following the experiments of Weed and McKibben in America, he tried intravenous injection of distilled water by which he obtained a very quick modification of the hypotension of cerebrospinal fluid.

According to these authors hypotension of cerebrospinal fluid is met with rather often in cases of skull fracture. Theoretically, every time the sub-arachnoid spaces are opened the equilibrium of the fluid is broken and the pressure may decrease more or less quickly.

As to the symptoms, the acute hypotension of the cerebrospinal fluid as rather well defined clinical features. Sometime there is lasting frontal cephalalgia. The headache can be accompanied with nausea, vomiting and giddiness. There is noted some time semi-coma but more frequently torpor and mental stupor. The differential diagnosis of hypotension and hypertension will not therefore meet with insuperable difficulties. Of course the exact diagnosis depends upon lumbar puncture with pressure determination.

From the therapeutic point of view, the authors do nothing but fight against the fluid hypotension. The rational treatment they say, is the intravenous injection of from 30 to 40 cc. of distilled water. This procedure may be repeated twice or more since it seemed that the pressure becomes stabilized only after 2 or 3 injections.

2. HYPERTONIC GLUCOSE

Following the introduction of intravenous hypertonic glucose for the purpose of reducing intracranial tension, the pendulum swung and very often happens, to excess in the use of this measure in head injuries. It was not long however, until certain investigators began to question the advisability of such routine use of this method of dehydration. Authorities today are divided on the subject. Some continue its use in every case, some condemn it unqualifiedly and still others think it is useful in selected cases only. Other men more recently would substitute the use of sucrose.

As examples of adherents to the feasibility of using hypertonic glucose intravenously as a dehydrating agent may be named Kennedy and Wortis (33). These men have had a wide experience in the treatment of hundreds of cases of head injuries and much value should be placed upon their opinion in this matter. They recommend intravenous injection of 50% glucose. 100 cc. of this solution may be thus given two or three times in twenty-four hours. They state that this not only reduces intracranial tension but it introduces a readily available food. They stress its value in combating acidosis caused by prolonged vomiting.

Keegan (30) of the University of Nebraska Clinic states, "The most physiological hypertonic solution for intravenous use is glucose, chemically pure and buffered. Such a solution can be obtained from drug houses, 100 cc. ampoules of 50% strength. Toxic reactions are usually avoided by the use of buffered solution. This

— should be given twice to reduce pressure symptoms."

Browder (5) recommends 75 to 100 cc. of 50% glucose as the best method of dehydration. He states however that this measure does not give universally a good result and sometimes is detrimental. He warns to use care about reducing intracranial pressure after head injury by changing osmotic pressure of the blood.

In an article in the A. M. A. Journal published in March 1933 a series of authors including Jackson, Kutsunai, Leader, and Joseph (28) the following conclusions are reached: that the use of hypertonic dextrose solution in acute states of intracranial hypertension due to injury of the brain demand results in immediate increase of pressure in half of the cases they studied, in slight temporary reduction of pressure in half of the cases, and in secondary increase in all of the cases after from 15 to 30 minutes. This they contend is due to pressure on the venous flow in the sinuses of the brain. According to them hypertonic dextrose solution not only does not appreciably lower intracranial pressure as measured directly by the spinal manometer but in many cases the pressure is actually increased. The use of these solutions, say the authors leads to the false sense of security in the treatment of acute cranial injuries, allowing prolonged pressure on the cortex of the brain and leaving to atrophy and fibrosis of the cortical nerve cells.

In the same Journal, Masserman (36) writing in June 1934 states that recent observations on animals and men indicate that initial decrease in tension of cerebrospinal fluid induced by hypertonic solutions of salt or dextrose in the blood stream and gastro-

intestinal tract is superceded within from one to three hours by a persistent rise in sub-arachnoid pressure and an exacerbation of the initial symptoms of intracranial hypertension. The author studied the problem further and gives results of his experiments; "When more than 75 grams of dextrose was given in 20 to 50% solution the initial rise of cerebrospinal pressure was followed by a fall in pressure of from 26 to 162 mm. of water, the curve of pressure reaching the lowest point from 50 to 170 minutes after the injection. In the ensuing 50 to 120 minutes the pressure then recovered to the basic level after which the upward trend continued until levels of tension of from 8 to 148 mm. of water above normal were reached and in most cases maintained throughout the period of observation (4 hours). Also in the majority of cases the reactive intracranial hypertension had subsided within 8 hours after administration.

'The ensuing diminution in sub-arachnoid pressure in cases in which the dextrose has been administered in effective concentration may be attributed both to a resorption of spinal fluid and a diminution in volume in nervous tissue. Conversely the rise of pressure to normal and above subsequent to the period of depression would theoretically be due to, 1. An increased rate of dialysis of cerebrospinal fluid from the blood which had meanwhile been rendered relatively hypotonic to the cerebrospinal fluid through loss of dextrose or salt into the tissues. 2. The deposition of dextrose in the nervous tissues with ensuing compensatory intercellular and intracellular edema. The possibility of the latter through effect is of prime clinical importance may well account for the recurrence and

exacerbation of symptoms of increased intracranial pressure after transient initial improvement in patients suffering from intracranial hypertension treated by intravenous hypertonic solution."

3. INTRAVENOUS SUCROSE

For the reduction of cerebrospinal pressure and intracranial tension, a substance is desired which: 1. Is non-toxic when given intravenously in high concentrations. 2. Which produces rapid reduction in the pressure of the cerebrospinal pressure. 3. Maintains the period of low pressure for several hours, and finally, 4. Does not cause a secondary rise in pressure. To be effective in rapidly drawing fluid from the tissues as indicated by 2, the substance must increase the osmotic pressure of the blood markedly. Conditions 3 and 4 require that it should not leave the blood, or at least that it should not diffuse to a significant degree into the brain or spinal fluid.

As in the case of other hypertonic solutions given intravenously, 50% sucrose lowers the spinal fluid pressure because it increases the osmotic pressure of the blood; but a combination of circumstances enables it to have a more prolonged effect than either NaCl. or glucose. (Published in the American Journal of Physiology for May 1935 is the article from which much of the material on this subject was derived.) (6) It probably remains in blood for a longer time after its injection since its molecules are larger and less diffusible. It is not broken down to any significant extent or utilized (Folin, Trimble, and Newman, 1927) as is glucose, nor is it stored in the tissues as is NaCl.; sucrose is essentially a non-toxic foreign body once it has entered the blood stream. The reduction of spinal fluid volume produced by its osmotic effect is prolonged by a powerful diuretic action which results in rapid elimination from the

body of the sucrose, together with the fluid withdrawn from the tissues.

Mag. Sulphate (6) when given by mouth, or rectum draws water in to the guts from the blood, which in turn drains fluid from the tissues including the brain (Fay, 1924). The fall in cerebrospinal pressure caused by Mag. Sulphate therefore actually depends upon realizing the large decrease in the fluid volume of the blood. Whereas this may be of little consequence in the presence of a normal blood volume, it imperils the circulation when the blood is already low. Sucrose on the contrary, produces a temporary state of hydremic plethora during the period when the cerebrospinal pressure is falling. Diuresis removes the excess water from the blood stream in from 2 to 3 hours. The final effect upon the plasma volume must of course be determined by the amount of available body water and by the amount of sucrose which is given.

According to Keith, Wakefield and Power (31) showed by experiments on humans that 97 to 98% of sucrose injected intravenously is excreted via the kidneys in sight of 24 hours.

Masserman (36) produced in 1935 an instructive article on the superiority of intravenous hypertonic sucrose. He presents for consideration the following points:

1. Fifty per cent solution of sucrose satisfactory for clinical use may be easily procured (Lilly) or prepared. Domino granulated sugar on the open market is dissolved in twice its weight of freshly distilled water. The solution is immediately filtered, placed in cotton

stoppered 1,000 cc. flasks, sterilized in a boiling water bath for three hours and cooled.

2. When 300 to 500 cc. of this solution is administered intravenously the initial rise of cerebrospinal fluid pressure (probably co-incident to the initial increase in blood volume and venous pressure) is of lesser degree and duration than is the case with the dextrose or electrolytic solutions. Subsequently the desired diminution in intracranial tension (probably resulting from the diuresis and tissue dehydration) is greater and more prolonged.
3. Corresponding to the relative impermeability of the blood-cerebrospinal fluid barrier to sucrose, there is only a slight and transient intracranial hypertension as the final hydro-dynamic effect of its intravenous administration. In contrast to this, dextrose and most of the physiologic salts pass readily from the blood into the central nervous system and cerebrospinal fluid and thereby induce, as their terminal osmotic effect, a marked increase in spinal fluid pressure and possibly also an edema of the central nervous tissues.
4. A 50% solution of sucrose when administered intravenously does not cause an overt phlebitis in the injected vein, and induces only a mild reaction if allowed to escape in small amounts into subcutaneous tissues.
5. Comparatively large amounts of sucrose in the circula-

tion are apparently not toxic, and cause no serious disturbances in the chemistry or cytology of the blood.

6. Repeated injections of sucrose can be given when necessary without cumulative adverse effects.

LUMBAR PUNCTURE

About 1840 Magendie discovered in the roof of the fourth ventricle, the minute opening that bears his name and pointed out the connection between the cerebrospinal fluid in the ventricular system and that in the subarachnoid spaces of the brain and cord. By this epocal discovery he led the way to a correct understanding of the circulation of the cerebrospinal fluid and to problems associated with increased intracranial tension. (26) About 1885 Corning an American, tapped for the first time this cerebrospinal water bed by inserting a needle between the last two spinous processes of the dorsal vertebrae. In 1891 Wynter drained the spinal system for tuberculous meningitis. It is to Quinke, however, that we owe our greatest knowledge of lumbar puncture and intracranial tension. In 1891 he published his studies on the diagnostic and therapeutic application of lumbar puncture and his work still stands, as a perfect elucidation of the pathogenesis of lesions that effect the cerebrospinal fluid system. He made it a rule always to measure the pressure of the cerebrospinal fluid by connecting the lumbar puncture needle with a fine glass pipette in which the fluid was allowed to rise.

Weed has shown how the cerebrospinal fluid finds its way from the foramina of Luschka and Magendie through the subarachnoid spaces over the cortex of the brain to be absorbed mainly by the arachnoidal villi along the sinuses, especially the longitudinal sinus. It has been found by Jackson that any swelling or edema of the cortex of the brain due to injury arrests the circulation of

the cerebrospinal fluid and raises intracranial tension ultimately endangering the medulla and floor of the fourth ventricle.

Jackson (26) states that there has been too much over-emphasis on the symptoms and signs of increased intracranial tension, as increased blood pressure, slow pulse, vomiting, eye ground changes, stertorous respirations. He says that they are delayed evidences of intracranial tension that come on slowly and indirectly by pressure on the medulla and are influenced by other factors such as shock and hemorrhage and result often in death. He argues that it is better to estimate the pressure early and directly before these dangerous symptoms arise. He advocates the use of the mercury manometer while doing the puncture and drainage. He says the procedure is not dangerous in the acute traumatic cases; says this is an error that has crept into our calculations from results with cases of chronic tension, especially in tumor cases of the base of the brain.

Schoenbeck (1916) in a recent review of deaths following lumbar drainage collected 71 cases and in 67 found by autopsy there was tumor or chronic disease of the brain. Gumbrecht collected 17 cases of sudden death following lumbar drainage and autopsy showed them all to be caused by pressure of tumor. Many of these were punctured in the sitting position and as much as 15 to 60 cc. of fluid was withdrawn at one time. The accidents are due to pressure on the medulla by the walls of the foramen magnum and hemorrhage from tumors by sudden release of tension on their vessels. He points out that he and his assistants did 300 punctures without untoward symptoms. He says that his patients were all in the horizontal

position and usually not more than 10 to 20 cc. was slowly removed at one time. He points out that Sharpe reports 1,100 punctures in three years without a death. He goes on to say that in chronic cases there is considerable molding of the brain by long continued pressure.

Jackson (26) advocates first the treatment of shock followed by routine lumbar puncture. If blood is present in the fluid and pressure is high he recommends withdrawal of 10 to 20 cc. of cerebrospinal fluid determining the fall of pressure by special stop clock arrangement of needle attached to mercury manometer, stopping the removal of fluid when pressure has been reduced one half or to normal. If pressure reading is high and no blood is present in the fluid, suspect middle meningeal hemorrhage without dural rupture. His technique is as follows: Have patient on right on right or left side on horizontal plain and on an unyielding surface, with spine flexed - if necessary in a restless patient give a preliminary hypo of morphine or gas anesthesia. Surgically cleanse the area of the fourth lumbar interspace and insert needle in midline. Withdraw stilet and as soon as cerebrospinal fluid appears connect the needle with the mercury manometer held by an assistant at the level of the spine and take a reading. Then open the side arm and collect cerebrospinal fluid in sterile test tubes. Shut off the fluid when 10 to 20 cc. is collected or when pressure reaches normal, withdraw the spinal needle and seal with collodion.

Jackson (26) concludes that the relief of increased pressure on the brain and re-establishment of the normal path of absorption of cerebrospinal fluid can be obtained by repeated lumbar drainage

(at intervals of 6 to 24 hours or longer depending on the height of pressure and rapidity with which pressure rises after withdrawal of fluid.) That lumbar drainage is superior to subtemporal decompression in relieving pressure below the tentorium cerebelli and has the further advantage that it leaves no mutilating skull defect, Jackson has no doubt.

In March 1935 issue of the A. M. A. Journal appears an article by Sharpe (43) concerning the present topic. This man says that the use of lumbar puncture is an important aide in the diagnosis of lesions of the central nervous system has become much more frequent in the past ten years: no longer is the test considered an "operation" deferred until the increasing severity of the patients condition demands an accurate diagnosis and then used only as a last resort. Today in many hospitals lumbar puncture is used only for diagnosis, and the use of repeated spinal drainage in acute traumatic cranial lesions, is considered dangerous and radical.

As a diagnostic aide there can be little criticism of its possible danger, except in subtentorial expanding lesions, and even in these latter there is no danger of serious complications if the lumbar puncture is properly performed (43). Diagnostic and by all means therapeutic lumbar punctures should always be performed with the mercurial manometric attachment if possible. In this way the spinal fluid pressure is accurately registered, and under no circumstances should an amount of cerebrospinal fluid be withdrawn to lower the pressure more than one half of the initial reading. Two cc. of fluid is ample for diagnostic purposes, but, it is possible

- to withdraw slowly 20 cc. and more for therapeusis. Therapeutic cases, the free escape of fluid from the puncture needle, when no manometer is being used to determine pressure, is attended by the well known danger of medullary complications due to direct pressure on the foramen magnum collar, and also intracranial vascular alterations to the degree of spontaneous rupture of the arterioles. Such lumbar punctures, done without the use of the manometer, are not properly performed and therefore the resulting complications are not valid criticisms of the test itself. Cases of these latter complications are rare (3 in 20 years in one clinic) in the better clinics.

The only accurate method of determining the presence of increased intracranial pressure, according to Sharpe (43) is by lumbar puncture. However, no prolonged examinations such as x-rays and lumbar punctures should be done until the patient has recovered from the initial shock of the injury, as such examinations merely prolong the shock.

Sharpe states that if the diagnostic lumbar puncture reveals clear fluid under a normal pressure (6-8 mm. of Hg.), or if the pressure is only slightly increases to 10 mm. or 12, or if the fluid is merely blood tinged, a favorable prognosis may be given, if there are no other serious bodily injuries. On the other hand if the cerebrospinal fluid pressure is increased to more than twice normal (14-16 mm.) and especially if it is bloody, an amount of fluid should be slowly withdrawn to lower the initial pressure reading by one half. That is, if the initial pressure was 20 mm. In this manner, there is no danger of medullary herniation or increased intracranial hemorrhage. The latter complication, so often referred to in the liter-

ature, does not occur if the above directions are followed. Depending on the height of the increased intracranial pressure and the degree of percentage of blood in the cerebrospinal fluid, the frequency of the repeated lumbar punctures for spinal drainage will vary from one to three daily, and in severe cases showing both high pressure and blood percentage as many as four spinal drainages each day gives best results.

In the treatment of acute head injuries, when the diagnostic puncture discloses clear or merely blood tinged fluid under normal or only slightly increased pressure, the routine expectant palliative method, aided by dehydration, is usually entirely satisfactory according to Sharpe. If, during convalescence, symptoms and signs of intracranial complications develop, lumbar puncture is perhaps the best means of conclusive proof of the condition. Repeated punctures during the course of treatment are invaluable.

The relationship of fracture of the skull to the presence or absence of bloody spinal fluid is not says Fay (18) the determining factor of life or death. Many cases with extremely bloody spinal fluid and evidence of cerebral contusion are to be found without demonstrable evidence of a fracture by means of the Roentgen ray. Other cases of fractured skull exists without bloody spinal fluid, presumably therefore without gross cerebral contusion. Fay contends that if the spinal fluid is found to be bloody, complete drainage of all fluid obtainable is necessary. It has been shown by Weed and Bagley that the red blood cells produce intense reaction in the subarachnoid spaces and they tend to stop up normal pathways and filters for elimination of cerebrospinal fluid. Thus they temporarily pro-

duce obstruction of the cerebrospinal fluid circulation, chiefly the cortical subarachnoid spaces and Pacchionian bodies. The physician is faced with a ten day period before the red blood cells in the cerebrospinal fluid are entirely hemolyzed. This means for at least the first seven days following trauma, cerebrospinal fluid elaborated will not find proper means of escape and intracranial pressure will probably ensue if fluid intake is not controlled. (48) Every effort should therefore be made to remove the red blood cells by drainage. Sufficient fluid should be given by mouth in the twenty-four hour period to permit re-accumulation of cerebrospinal fluid for subsequent drainage.

Lumbar drainage must be resorted to as often as a rise of pulse pressure and respiratory changes indicate. If clear fluid is obtained at the lumbar puncture and the pressure reading at the time carefully taken, the problem becomes one of management of cerebrospinal pressure with or without drainage. Prompt reduction of intracranial pressure may be accomplished by spinal drainage when symptoms require it.

It is at once evident that this means of "decompression" is far more effective and desirable than the operative methods used heretofore and gives better results without leaving a permanent cranial defect. Decompressions have been entirely abandoned by many men. Fay (18) therefore, considers spinal puncture of extreme importance not only as a diagnostic aid to determine the presence of clear or bloody spinal fluid, but also to determine intracranial pressure and establish the method subsequent treatment of the patient.

Fleming (20) in a recent article (1935) contends that spinal

puncture is of more diagnostic than therapeutic value. The danger, of medullary herniation into the foramen magnum, resulting from lumbar puncture, in patients with head injuries, probably has been exaggerated. In his opinion a greater hazard is the possibility of increasing intracranial hemorrhage. It is advisable, says he, to delay lumbar puncture from 4 to 6 hours after injury, if possible, and to avoid altering pressure relations until free bleeding has stopped.

The opinion of Prof. Babcock on the subject of lumbar puncture must rightly be included in a discourse of this type. "If the spinal pressure in a horizontal position is above the normal of 10 mm. of Hg., if there is free blood in the fluid and no clinical evidence of extra duro pressure, enough fluid is permitted to slowly escape to reduce the pressure to normal or, if the pressure is above 25 to 15 mm Hg. With high pressures the spinal tap is repeated every eight hours or until the pressure drops to normal or the symptoms improve.

Keegan (30) of the University of Nebraska says that spinal punctures do not relieve brain edema since the ventricles and sub-arachnoid spaces are compressed by the swollen brain and there is little fluid to drain off. The failure to relieve traumatic brain edema by dehydration prolongs the symptoms and favors development of chronic post-traumatic headaches. He believes less total harm will be done by carefully performed diagnostic and therapeutic spinal punctures in head injuries, than to allow low brain pressure symptoms to continue to long a time without diagnosis and therapeutic spinal drainage.

Concerning lumbar puncture as an aid to treatment, Rawlings (41) says that this procedure is useful in diagnosis but useless in treatment of acute head injuries, especially when there is intracranial bleeding.

Earl (15) says lumbar drainage is indicated when gross blood is present in the spinal fluid. This diminishes the irritation and reduces the adhesions between the pia and arachnoid. He thoughtfully advocates novocain for punctures.

According to Munro (40) decompression by lumbar puncture is suitable for all three types of uncomplicated brain injuries. The edema is relieved because with the reduction of intracranial pressure to normal, the venous congestion is corrected and the re-activated circulation allows the partially asphyxiated cells to recover as much as possible. The excess intra and extra cellular fluid is then absorbed and brain volume is returned to normal. In contusions and lacerations the excess unabsorbed fluid is mechanically removed together with a small amount of free blood, and the meningocytes, if the drainage be repeated enough, are aided in the uncorking of the absorptive channels in the arachnoidal villi.

Cases with lumbar puncture have a mortality 9% less than similar cases without lumbar puncture. In over 1,000 punctures in acute brain injuries Munro (40) has seen not one death attributed to this procedure. In another article by the same author (18) he recommends punctures for treatment every twenty-four hours. In a series of cases reviewed by him amounts of spinal fluid varying from 1 cc. to 35 cc. were removed, the average being 9.5 cc. He concludes that mortality is lowest following treatment by lumbar

puncture than by any other method. He states that average intracranial pressure is nearly two and one half times higher in fatal than in non-fatal cases.

Crutchfield (9) contends that increased intracranial pressure as determined by spinal puncture is frequently found in patients not showing evidence of progressive cerebral impairment. This in itself is not indication for burrhole exploration or subtemporal decompression unless the pressure persists for four to five days. A prolonged high degree of intracranial pressure no doubt impairs cerebral function. In such cases, Crutchfield believes that carefully performed daily spinal punctures are beneficial not only in bringing about symptomatic improvement but also in improving cerebral circulation and thereby lessening cerebral edema. Cerebrospinal fluid, says he, should be drawn off slowly and its pressure should not be reduced below the normal level.

Dandy (13) an authority on the subject of head injuries, is numbered among the opposition in regard to lumbar punctures as a means of treatment. He says: "Why perform lumbar punctures on very ill patients with injuries of the brain? Why not get the necessary facts by the old fashioned methods of observation? There would be no harm of course, in doing a lumbar puncture to get additional information if it were possible, if in so doing nothing harmful resulted, but you are doing harm when you do a lumbar puncture.

'You can't do a lumbar puncture on a normal individual without producing a headache, and if you withdraw much fluid it may persist for several days. The headache is due to cerebral injury. The injury probably occurs because the brain is thrust against the

sides of the skull. If a lumbar is done in the presence of increased intracranial pressure, which obtains in cerebral injuries the resultant trauma will be proportionately greater. It is true that the patient may improve immediately after you do the puncture, but it is only a transient. If an extra dural hemorrhage is present the release of pressure is the worst possible thing that can happen to induce further bleeding.

'All intracranial pressure results from hemorrhage and edema. Lumbar puncture doesn't remove either this edema or blood. It removes the fluid from the ventricles and this nature is able to do and does in her manner of space compensation, the fluid being absorbed by the blood.'

As a final word on the present subject some impressions from an article by Masserman and Schaller (35) should be in order. After an investigation of intracranial hydrodynamic in human cadavers the authors discovered that they could not produce descent of the cerebellum or medulla into the foramen magnum by hydrodynamic means alone, e.g., by sudden release of high intracranial fluid pressure through rapid lumbar or cisternal tap. They were therefore unable to confirm the generally accepted importance of the mechanical factor in the ethiology of cerebellar herniation and bulbar compression in cases in which a block about the foramen magnum had not been artificially effected.

After a consideration of the literature these authors point to the possibility that rapid removal of cerebrospinal fluid by lumbar tap in cases of intracranial hypertension may cause a secondary and greater increase in intracranial pressure, and possibly also

edema of the central nervous system. They find after experimenting on animals that rapid decompression of intracranial hypertension may cause cerebral cellular damage and edema. They point out that the experiments, if clinically applicable, may explain the necessity of gradual decompression when cerebrospinal fluid is withdrawn by rachicentesis in cases of cranial hypertension.

Moch (38) writing in March 1936 says it requires the closest observation to detect the difference between the signs and symptoms due to cerebral edema and other causes of a milder form of increased intracranial pressure and the signs and symptoms of a threatened medullary compression due to more severe causes of increased pressure. Says he a lumbar puncture done early before medullary compression has developed is often extremely spectacular in its results. Patients will frequently come out of a deep coma a few moments later and even converse with the doctor or nurse. In many of these cases, the puncture is only required once if ordinary dehydration is utilized.

SURGICAL TREATMENT

According to Mock (38) the definite indications for operative intervention in skull fractures are the following:

- A. Definitely depressed fractures.
- B. The compound depressed skull fracture.
- C. Middle meningeal hemorrhages. Often these extra dural hemorrhages are contracoup to the site of injury.
- D. Subdural hematoma. True blood clots are not always at operation when this diagnosis is made.
- E. Subtemporal decompression. As a rule, this operation is now limited to those few cases with persistent symptoms, growing worse, not responding to lumbar puncture and with no definite focal signs. This operation is not indicated in more than one or two per cent of the cases.

Fleming (20) says cranial operations should be reserved for cases in which free fluid can be removed, as in meningeal hemorrhages, subdural collections of blood or cerebrospinal fluid and intracranial clots. Operation on a brain hence from edema, accomplishes little good and often results in material damage. Cushing's subtemporal decompression is best suited to the majority of cases in which operation is indicated. This approach allows an adequate exposure of the middle meningeal artery and, on the right side uncovers a fairly silent part of the brain. The closure of the incision offers a satisfactory covering of muscle and fascia. Large decompressions are seldom necessary as the drainage of fluid is all that is required. The exposure of motor and speech centers must be avoided.

Unnecessary contusion may cause scars and traction on the brain resulting in convulsive states.

In the management of patients showing evidence of progressive cerebral impairment with a definitely elevated spinal fluid pressure, Crutchfield (9) feels that a unilateral or bilateral burr exploration should be made in the skull for the purpose of a more accurate determination of the cause for the increased intracranial pressure. The operation can be done under local anesthesia with practically no danger. The site chosen for exploration is high in the temporal region since it is in this location that localized hemorrhages and clots are more in evidence. The neurological examination usually gives some indication of the side involved. Should the exploration give negative findings the other side should likewise be explored. In case of a middle meningeal hemorrhage the opening in the skull can be enlarged sufficiently for the removal of clots and access to the bleeding vessel. In the absence of an extradural hemorrhage a small opening should be made in the dura for the purpose of detecting a subdural hematoma or hydroma. Either lesion can, in many instances be satisfactorily drained through this small exploratory opening. When organized subdural clots or a high degree of cerebral edema is found it sometimes becomes necessary to enlarge the bony and dural openings to permit evacuation of the clots or to decompress the swollen brain.

Kennedy and Wortis (32) speak from a wide experience. They name the decompressing surgical operations as suboccipital decompressive craniotomy and subtemporal decompressive craniotomy. These major

operations they say should not be used until all other procedures have proved futile and incapable of arresting the progressive signs of steadily increasing pressure within the skull. In their 1,000 cases, 37 were operated. Of these 14 lived and 23 died.

Davis (14) says, "It should be noted that a subtemporal decompression has not been described as a method of therapy in a group of skull injuries to which by far the largest number of patients belong. No matter how carefully performed and in the hands of a surgeon experienced in handling nerve tissue some edema will follow this operation. This adds to that intracranial pressure which already exists. It has been proved that the mortality rate following the treatment of skull fractures in a large charity hospital has been lowered tremendously since the abandonment of decompression operations for the relief of traumatic edema."

Subtemporal decompression, according to Munro (40) is by its very nature only efficient in mild cases in which it is not urgently needed. In serious cases, says he, the decompressive effect is shortly lost because the dural and bony opening is soon lightly plugged by an edematous cortex.

An extradural hemorrhage is a rather common result of head trauma says Dandy (11). These hemorrhages can in most cases be removed without damage to the patient. The most common signs and symptoms are; a primary loss of consciousness followed by a lucid interval, then another lapse into unconsciousness. Also motor weakness and fits on the opposite side of the body.

Babcock (2) says, "Cranial decompression is performed only

for evidence of cortical pressure as shown by hemiparesis or monoplegia, Jacksonian convulsions, motor or sensory aphasia. The decompression is made on the side of the injury, on the side of the dilated pupil, opposite to the side of the paralysis, usually first over the anterior branch of the middle meningeal artery. Decompression is not made for basilar injury. It is harmful for the pressure of blood, serum or bone upon the cortex."

HOW CAN THE HIGH MORTALITY RATE BE REDUCED?

A. In the first twenty-four hours. (38)

45 % of deaths occur in the 1st twenty-four hours.

1. Greater knowledge concerning early management by rank and file of profession.
 - a. Laity education - prevention and first aid.
 2. Treat shock first - everything else can wait.
 - a. Don't suture lacerations at once.
 - b. Don't reduce fractures at once.
 - c. Exception - a severe life threatening hemorrhage.
 - d. Least possible moving of patient chief essential.
 3. Every serious head injury is an emergency case.
 - a. Requires immediate attention of attending physician.
 - a. Early skillful examination - blood and urine.
 4. Don't overlook less obvious, often more serious associated injuries
 5. X-Ray every head injury - but don't X-Ray:
 - a. In presence of shock.
 - b. In presence of delirium or deep coma.
 6. Immediate operation of skull fracture seldom indicated.
 7. Treat every head injury as serious until proven otherwise.
 8. Morphine lulls the surgeon, as well as the patient, to sleep.
- B. After twenty-four hours.
- 40 % of deaths occur from the 2nd to the 7th day.
1. Greater knowledge concerning general management.
 - a. Dehydration started early (after shock) and persisted

in will reduce mortalities.

- b. Examine frequently - certain signs and symptoms, interpreted correctly, require life saving special treatment.
2. The earlier lumbar puncture is performed, when indicated, the lower the death rate.
 - a. Not indicated in presence of grave shock.
 - b. Increased intracranial pressure - persisting after early dehydration requires lumbar puncture.
 3. repeating lumbar punctures when operation is indicated increases fatalities.
 4. Eight percent of cases require operation
 - a. Operative rate of over 12% or below 6% increases mortality rate.
 - b. The longer operation can be safely postponed the lower operative death rate.
 - c. Subtemporal decompressions seldom indicated. High mortality.

In order to impress the importance of avoiding the transportation of patients with head injuries Mock (38) coined a slogan as follows: "A live skull fracture in a farm house is better than a dead one in a hospital."

CONCLUSION

The laity see much glamour in a "fracture of the skull", but to the physician the skull fracture is not as of great importance as the injury to the contents of the skull. The importance of knowing how to treat this condition is emphasized by the increasing number of such accidents every year. Doctors everywhere should know exactly what to do for these cases and the public can do their part with knowledge of proper first aid treatment.

Every case is a "law unto itself" and therefore real standardization of treatment would not be advisable. There are however certain principles which apply to every case. These have been set down above and are well outlined in the portion adopted from Mock. The definite value of these principles has been proven by thousands of cases. The general trend in the management of head injuries is toward more conservative treatment.

The movement on foot for prevention of automobile accidents in the United States should prove effective in reducing the number and severity of head injuries. American physicians can be depended upon to do their part in any movement of this sort. And so, onward to the Millennium!

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