

THE TREATMENT OF ACUTE AND CHRONIC CASES OF CEREBRAL TRAUMA, BY METHODS OF DEHYDRATION

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THE treatment of acute cerebral trauma by means of "dehydration," had its origin in 1921 on the neurosurgical service of Dr. Charles H. Frazier. The encouragement and guidance extended to the author by his former Professor and Chief made possible the development of the present clinical program of "dehydration" and a better understanding of the principles involved.

The application of the principles of "dehydration" now extend into many neurosurgical problems, complicated by increased intracranial pressure, as well as acute and chronic "hydration states" arising throughout the general field of medicine and surgery.

It is a sincere pleasure at this time to dedicate to Doctor Frazier the author's observations on methods of "dehydration" employed in cerebral trauma during the past twelve years.

Introduction.—The term "dehydration" has been applied to clinical measures designed to subtract water from the body tissue reservoirs, the principal objective in cerebral problems being depletion of the large cerebrospinal fluid reservoir.

The first deliberate attempt to evolve a clinical program of "dehydration" followed observations on the rise and fall of the fontanel in infants, on the pædiatric service at the University of Pennsylvania Hospital, early in 1921. These clinical observations, as they apparently concerned intracranial volume relationships, were correlated with the then recent experiments of Weed and McKibben,¹ 1919–1921, showing a reduction of intracranial pressure following the intravenous and ingested introduction of hypertonic saline solutions.

The first opportunity to apply these observations clinically arose in September, 1921. A patient (I. R. S.), on the neurosurgical service of Dr. Charles H. Frazier, manifested signs of respiratory failure and cerebral œdema some hours following operation for a parietal lobe tumor of the brain. Large doses of magnesium sulphate were administered to the unconscious patient by a stomach-tube, in an attempt to reduce the cerebral œdema and pressure, when signs of a failing respiratory centre had developed. Following the administration of the hypertonic solution, the respirations rose promptly from a rate of eight per minute to twelve. The dose was repeated in four hours with continued improvement, and the return of the patient to consciousness (Fig. 1A).

At this time (1921), subtemporal decompression, ventricular puncture and spinal drainage were the accepted methods at the disposal of the surgeon

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for relief of pressure in such desperate situations. The occasional use of concentrated solutions of sodium chloride, by vein, as advocated by Cushing and Foley,^{2, 3} 1919-1920, produced temporary manifestations of benefit, although frequently secondary phases of pressure ensued which made the use of intravenous sodium chloride a measure of last resort.

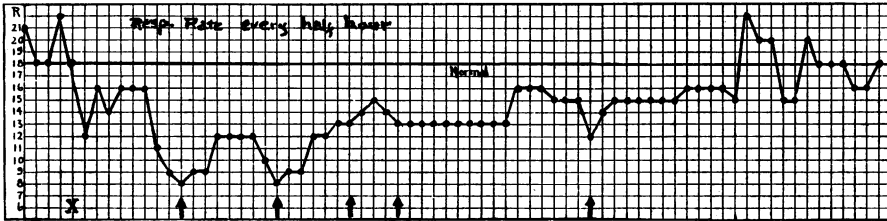


FIG. 1A.—(I. R. S.).—Rapid post-operative fall in respiratory rate due to medullary pressure, relieved by magnesium sulphate, with eventual recovery: R, respiration; X, exploratory craniotomy; arrows, magnesium sulphate. (Courtesy J. A. M. A.)

The beneficial response, due to “dehydration,” by means of the introduction of a saline purge, such as magnesium sulphate, into the gastro-intestinal tract, was observed further when applied to cases suffering from acute cerebral injury due to trauma (Fig. 1B).

A routine method of treatment soon arose, designed to meet the needs of acute cerebral pressure problems, but the principles involved concerned those of chronic pressure as well, so that its importance became evident in the

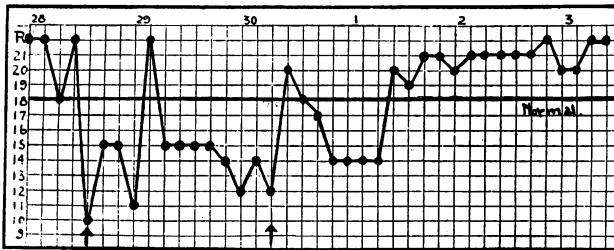


FIG. 1B.—(H. S.).—Respiration after concussion resulting from blow on head; medullary depression relieved by magnesium sulphate with rapid return of respirations to normal: R, respiration; arrows, magnesium sulphate. (Courtesy J. A. M. A.)

management of brain tumors, communicating hydrocephalus and other hydrated states such as epilepsy, eclampsia, and the various chronic post-traumatic-pressure syndromes.

The profound importance which the subject of cerebral trauma holds in our social and economic world today can be judged by a recent comparison of the losses in the American Expeditionary Force during the World War: “killed in action, 36,694; died of wounds, 13,691 or a total of 50,385.”⁴

According to a report from the Surgeon General’s office, “In 1928, 100,762 individuals in this country met violent deaths; and in 1929, 103,942 individuals died in the United States from violent causes.”

A recent announcement by the National Safety Council (February 1,

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1934), states that 33,000 individuals met violent deaths by motor-vehicle accidents, alone, in 1933.

No figures are available as to the proportion of these cases which received complicating cerebral injuries. The majority of deaths, due to violent causes, involve directly or indirectly the central nervous system.

Swift⁵ states that there are approximately 112,000 cases of skull fracture annually in the United States, with an average mortality of 25 per cent. In his analysis, patients treated in private hospitals showed a mortality of 24.8 per cent., whereas the mortality in the county hospital was 35.5 per cent.

Wortis and Foster Kennedy⁶ report a mortality of 37.8 per cent. in one thousand cases of cerebral injury admitted to the Bellevue Hospital between the years 1926 to 1929.

Mock, Morrow and Shannon,⁷ in a wide survey covering 3,278 cases of cerebral injury, present the following table of comparative mortality rates:

| | Cases | Deaths | Per Cent. |
|------------------------------|-------|--------|-----------|
| Hospital A..... | 1,173 | 464 | 39.5 |
| Hospital B..... | 190 | 59 | 31. |
| Hospital C..... | 32 | 13 | 40.6 |
| Hospital D..... | 34 | 11 | 32.4 |
| Hospital E..... | 114 | 37 | 32.4 |
| Hospital F..... | 191 | 57 | 29.8 |
| Collected cases..... | 800 | 209 | 26. |
| Doctor Fay's reports..... | 224 | 41 | 18.3 |
| Doctor Wright's reports..... | 349 | 73 | 21.8 |
| Mock's cases..... | 171 | 33 | 19.2 |

These authors add, "At least three of the surgeons who have been concentrating their efforts on the management of skull fractures (Fay, Wright and Mock), have shown that the mortality rate can be reduced to around 18 to 21 per cent."

This is obviously a challenge to those who have made no effort to understand or cope with the problem of cerebral trauma, excepting to adopt palliative measures leading to a high mortality.

The importance of the problem in peace-time military circles is further indicated by the fact that during the ten years between 1922 and 1932, twenty-six officers and 398 enlisted men in the Army died of injuries received in automobile and motor-cycle accidents. This "class of accidents," according to Major G. F. Lull, M. C., U. S. Army,⁸ "leads all others as a cause of death in the Army."

No figures are available as to the economic loss, in terms of individuals incapacitated by severe cerebral trauma, who have subsequently survived. The facts regarding the eventual institutionalization of the cerebral trauma patient (whether arising from outside accidental causes, or secondary to birth injury), are not available, but the chronic, mentally deteriorated, as well as the mentally retarded and post-traumatic deteriorated groups, constitute a large proportion of our institutionalized patients today.

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The enormous financial burden imposed upon the people of the United States, to care for and maintain these victims of cerebral injury during the various subsequent periods of life, is beyond estimation.

It is obvious that the subject of cerebral trauma, its proper consideration and management, constitutes one of the outstanding problems before the medical profession today.

The material presented in this paper has been divided into consecutive yearly groups to indicate what general improvement in mortality may have arisen from the measures of "dehydration" as they became elaborated, and as a means of comparison between this recent method and other methods currently employed during the development of the program which has now become an established method of treatment for such conditions.

In spite of the wide variety of factors which have contributed toward the success or failure in the management of cerebral trauma, it seems evident during recent years that a general trend has occurred away from the former concepts of "surgical decompression" and toward the more conservative methods of "physiological decompression."

In order to definitely arrive at a comparative basis for such a study, all accident cases suffering from unconsciousness complicating injury, admitted to the hospital, have been included in this series. This is in order that deaths occurring within the accident ward, a few minutes or several hours after admission, may find their rightful place in the total mortality figures.

In the author's opinion, patients who have survived the three-hour period, after admission to the hospital, offer the possibility of analysis of treatment. The effect of treatment instituted immediately in the accident ward requires at least two to three hours to become adequate and effectual. On the other hand, patients who have received minor injuries without sufficient symptoms to warrant their final admission to the hospital service have been entirely excluded from this analysis.

It is therefore apparent in this series, which includes all major cases admitted to the hospital, irrespective of the survival period, that a clearer idea of the merits of treatment can be obtained in those patients who have survived at least the third hour after admission, and the introduction of treatment.

The hopeless group, with almost an "immediate" mortality (died within three hours) may serve to indicate the general character of cases received by the hospital services analyzed. The total mortality indicates the basis of prognosis justifiable from the moment of accident, whereas the hospital mortality after the third hour represents a fair comparative basis for treatments now employed.

MATERIAL ANALYZED.—The following tables contain cases analyzed on three hospital services, covering a period of fifteen years, including twelve years of collateral growth of the method of "dehydration."

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A control series from 1918 to 1921, in one hospital, serves to indicate the results obtained by former methods of treatment.

A parallel series of cases in the same institution observed from 1921 to 1925 serves to indicate the results obtained on those treated by measures of "dehydration" as contrasted to those treated by other current methods until the principal of "dehydration" became adopted (Table I, Series A).

SERIES A

TABLE I

| Date | Total Cases | Deaths | Total Mortality | Died within Three Hours | Immediate Mortality | Died after Three Hours | Final Mortality | |
|-------|----------------------|--------|-----------------|-------------------------|---------------------|------------------------|-----------------|------|
| 1918 | | 94 | 18 | 19.6 | 4 | 4.2 | 14 | 15.5 |
| 1919 | | 44 | 12 | 27.2 | 3 | 6.3 | 9 | 21.9 |
| 1920 | | 70 | 15 | 21.4 | 3 | 4.2 | 12 | 17.9 |
| 1921 | (Dehydration series) | 14 | 1 | 7.1 | | | 1 | 7.1 |
| | (Control series) | 31 | 7 | 22.6 | | | 7 | 22.6 |
| 1922 | (Dehydration series) | 28 | 2 | 7.1 | 1 | 3.6 | 1 | 3.6 |
| | (Control series) | 19 | 6 | 31.6 | | | 6 | 31.6 |
| 1923 | (Dehydration series) | 21 | 3 | 14.3 | | | 3 | 14.3 |
| | (Control series) | 11 | 3 | 27.3 | | | 3 | 27.3 |
| 1924 | (Dehydration series) | 20 | 4 | 20 | | | 4 | 20 |
| | (Control series) | 33 | 5 | 15.10 | 2 | 6.6 | 3 | 9.6 |
| 1925 | (Dehydration series) | 60 | 6 | 10 | | | 6 | 10 |
| 1926 | (Dehydration series) | 15 | 2 | 13.3 | | | 2 | 13.3 |
| Total | | 460 | 84 | | | | | |
| | (Dehydration series) | 158 | 18 | 11.3 | 1 | | 17 | 10.8 |
| | (Control series) | 302 | 66 | 21.8 | 12 | 2.7 | 54 | 18.6 |

In another institution, rotating services provided an opportunity to contrast results between the old and the new methods (Table II, Series B).

The series of cases treated from 1929 to 1934, at Temple University Hospital, on a full program of "dehydration" is contrasted with the results obtained three years prior to the establishment of the author's neurosurgical department, although the method had already been adopted in a modified form by his predecessors.

From 1929 to the present time, the full measures of "dehydration" and fluid limitation have been carried out on the neurosurgical service of Temple University Hospital with a total mortality of 18.2 per cent. and a final mortality (after the third hour) of 13.8 per cent. In contrast to these figures

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the total mortality for the control series in the same institution for three years prior to the establishment of "dehydration," was 25.6 per cent. with a final mortality of 19.4 per cent.

The striking fact revealed by this study of 1,032 cases is that the *total*

SERIES B

TABLE II

| Date | Total Cases | Deaths | | Total Mortality | Died within Three Hours | Immediate Mortality | Died after Three Hours | Final Mortality |
|----------------|----------------------|--------|---|-----------------|-------------------------|---------------------|------------------------|-----------------|
| 1925 | (Dehydration series) | 22 | 5 | 22.7 | 1 | 4.5 | 4 | 19 |
| | (Control series) | 9 | 4 | 44.4 | 0 | 0 | 4 | 44.4 |
| 1926 | (Dehydration series) | 13 | 1 | 7.6 | | | 1 | 7.6 |
| Total Series B | | 44 | | | | | | |
| | (Dehydration series) | 35 | 6 | 17.1 | 1 | 2.8 | 5 | 14.2 |
| | (Control series) | 9 | 4 | 44.4 | 0 | 0 | 4 | 44.4 |

mortality of the "dehydration" series (556 cases) has been definitely below the *final* mortality of the control series (476 cases) even after the number that perished in the first three hours had been deducted.

SERIES C

TABLE III

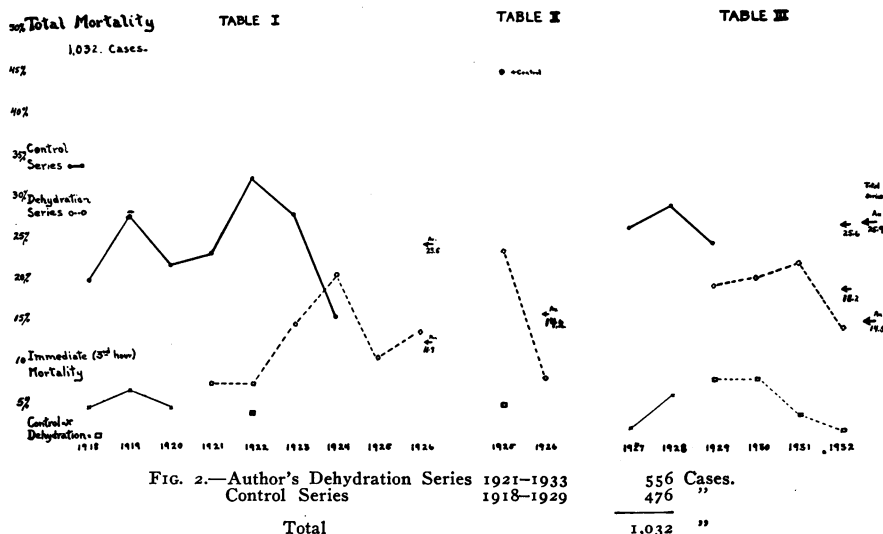
| Date | Total Cases | Deaths | | Total Mortality | Died within Three Hours | Immediate Mortality | Died after Three Hours | Final Mortality |
|-------|----------------------|--------|-----|-----------------|-------------------------|---------------------|------------------------|-----------------|
| 1927 | (Control series) | 55 | 14 | 25.45 | 1 | 1.81 | 13 | 23.63 |
| 1928 | (Control series) | 59 | 13 | 28.1 | 3 | 5.08 | 10 | 16.94 |
| 1929 | (Dehydration series) | 27 | 5 | 18.52 | 2 | 7.4 | 3 | 12 |
| | (Control series) | 51 | 12 | 23.52 | 3 | 5.88 | 9 | 17.64 |
| 1930 | (Dehydration series) | 118 | 23 | 19.49 | 9 | 7.6 | 14 | 12.8 |
| 1931 | (Dehydration series) | 123 | 26 | 21.14 | 4 | 3.2 | 22 | 17.88 |
| 1932 | (Dehydration series) | 95 | 13 | 13.71 | 1 | 1.05 | 12 | 12.7 |
| Total | | 528 | 106 | | 23 | | 83 | |
| | (Dehydration series) | 363 | 67 | 18.4 | 16 | 4.4 | 51 | 14.6 |
| | (Control series) | 165 | 39 | 23.6 | 7 | 4.2 | 32 | 20.2 |

The average yearly total mortality for the series (Fig. 2) shows a difference of more than 11 per cent. between the control group (25.9 per cent.) and those placed on "dehydration" (14.5 per cent.).

As both the control and "dehydration" series show an immediate mortality which is almost identical (4-5 per cent.) for the three-hour group, it seems justifiable to attribute the definite decrease in mortality found in the "dehydration" series to the management and methods of treatment employed.

Surgical decompression was performed as a matter of last resort in eleven cases out of 363. Nine of these patients died (operative mortality 81.8 per cent.) in spite of "dehydration" and decompression. These deaths are included in the final and total mortality figures.

Surgical intervention in this recent series was responsible for less than 0.5 per cent. of the reduction in final mortality, so that this procedure may well be relegated to the specific relief of subdural and epidural hæmorrhage, débridement for compounded wounds, and trephine for drainage of "wet



brain." Subtemporal decompression undertaken in cases showing bloody cerebrospinal fluid has, in the author's experience, been invariably fatal. The operator usually adds insult to injury when the dura is opened and the congested and swollen brain is allowed to rapidly herniate into the wound. Even in a well-organized neurosurgical clinic equipped to handle such emergencies, the conviction prevails that surgical decompression is justified in acute cerebral injury only as a matter of last resort and that, where physiological methods of decompression fail, operative decompression fails likewise.

GENERAL SURGICAL CONSIDERATIONS.—Frazier,⁹ in 1930, pointed out his abandonment of surgical decompression in favor of the more conservative methods of treatment. The practice of subtemporal decompression for relief of increased intracranial pressure, by permitting an herniation of the brain, has been too widely practiced in the past although the results obtained do not justify its use. This type of radical procedure is not to be confused

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SERIES D

TABLE IV

| Date | Total Cases | Deaths | Per Cent. Total Mortality |
|--------------|-------------------------------|--------|---------------------------|
| 1931 1933 | (Doctor Genkins' series)* 55 | 8 | 14.5 |
| 1932 1933 | (Doctor Delaney's series)† 26 | 3 | 11.5 |
| 1934 | (Doctor Mock's series) 171 | 33 | 19.2 |
| Total | 252 | 44 | Average mortality 15.0 |

NOTE.—Table IV, Series D, includes the reported results of the most recent application of the broad principles of dehydration, in their ultimate refinement, when placed under the direct care and observation of those well trained to manage cerebral trauma, from the moment of admission to the final period of discharge.

* Genkins, M. H.: Report of 55 Cases of Cerebral Trauma, Treated by Dehydration. Survey Submitted November 16, 1933.

† Delaney, W. E.: Management of Head Injuries. *Am. Jour. of Surg.*, vol. 21, p. 3, September, 1933.

with the definitely indicated exploratory craniotomy for a localized or pre-determined subdural hæmorrhage, elevation of a deeply depressed bone fragment or control of an accessible bleeding point within the cranial cavity. Such "explorations" may or may not become "decompressions," depending upon the judgment of the operator and the conditions encountered at the time of operation.

As a general rule it may be stated that all cases showing signs of subarachnoid hæmorrhage (bloody spinal fluid) should be spared any surgical intervention as long as possible. If the patient shows definite signs of a subdural hæmorrhage or large depressed fracture, operation after the fifth to seventh day is much to be preferred to early intervention. So long as the patient's general condition can be maintained with safety, the focal problem may be considered as one would a brain tumor; the most favorable results are obtained when the general cerebral pressure, congestion, and œdema have subsided. The bone may then be elevated or the clot removed without difficulty or great risk.

The desirability of delaying all surgical intervention until the acute reaction of the brain has subsided must depend on careful observation of the patient and palliative measures directed toward relief of intracranial pressure in the interval. Here, "dehydration" has been of great benefit.

The one clear-cut indication for prompt surgery after cranial trauma is the presence of a compounded fracture of the skull, where foreign bodies, fragments of bone, hair or dirt may have entered the brain substance. Here the first principle is cleanliness and protection of the brain structures against infection. Débridement and proper repair of the wound should be done as promptly as possible, instituting appropriate drainage if necessary.

FUNDAMENTAL CONSIDERATIONS UNDERLYING THE PRINCIPLES OF DEHYDRATION.—In order to clinically apply the principles of “dehydration” to the many varieties of symptom-complexes which arise following injury to the brain, it is necessary to clearly establish the basis for the manifestations encountered.

The age of the individual, the physical characteristics as to whether they are of the “hydrated” (plump, fat, water-logged) type, or are thin, emaciated, and without surplus water storage, become important factors in modification of treatment.

The appropriate management of a case of cerebral trauma requires a broad concept of the mechanisms involved, so that the most important of these components will be discussed in detail as they may apply to the problem under consideration.

THE CEREBROSPINAL FLUID RESERVOIR.—The physiological function of cerebrospinal fluid has never been clearly established. The presence of from 120 to 130 cubic centimetres (adult) of this clear filtrate of blood-plasma in the subarachnoid and ventricular spaces of the brain plays an important part in the regulation of intracranial pressure by the volume and space which it occupies within the fixed limits of the cranial cavity.

From a physical standpoint it can hardly be considered a “water-bed” or protective cushion for the important nerve-centres. Certain areas of the brain are not included in its field of distribution (Fig. 3), namely, the occipital and temporal lobes, the under-surface of the frontal lobes and the dorso-lateral aspects of the cerebellum. The laws of hydraulics indicate that this “fluid cast” may become a potent factor in the destruction rather than protection of the areas adjacent to its pressure application. The fact that “pressure exerted at any point in a closed fluid system is equally distributed to all points in that system” applies so far as the limits of the craniovertebral cavity is concerned, and this fluid mass serves to register pressure changes uniformly in the cranial or spinal portion of the cavity, if freely communicating.

This fact of recognized pressure transmission is utilized in the procedure of lumbar puncture by establishing existing *intracranial* pressure from the distant lumbar sac. It also serves to establish when the fluid system is interrupted (blocked by spinal tumor or foraminal hernia, as evidenced by the Queckenstedt test and other observations), based upon the ready transmission of pressure by the spinal fluid “cast” from point to point.

The same property also conveys upon the free spinal fluid cast dangerous and destructive possibilities. Spinal compression or concussion may be instantly transmitted to the brain surfaces. Changes in intra-abdominal and intrathoracic pressure may be relayed to the fluid cast by means of the large paravertebral venous sinuses as well as the cerebral dural sinuses, so that coughing, straining, the exertion of labor, and visceral compression, variously affect intracranial pressure. Cerebral function and activity are frequently impaired by prolonged applications of such transmitted disturbances.

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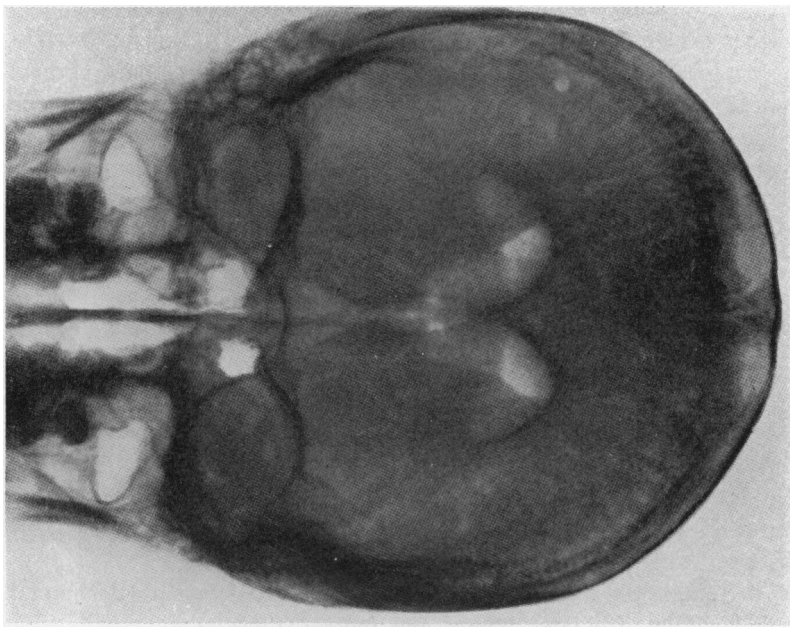


Fig. 3A.—Anterior-posterior view showing normal air pathways. Note the presence of air at the vertex in the pachionian area, the diamond-shaped third ventricle, the aqueduct and upper portion of the fourth ventricle. Both lateral ventricles are normally outlined. Air may be seen just above the corpus callosum. The longitudinal sinus is well shown and the falx seen separating the two hemispheres. (Courtesy, *Am. J. Psych.*, vol. 8, p. 5, March, 1929.)



Fig. 3B.—Lateral view, normal encephalogram. Note the fine air markings following the sulci over the cortex, the slight increase of air at the vertex and normal ventricle. The descending horn is not seen. The cisterna chiasmatis and cisterna pontis are clearly defined. The third ventricle may be vaguely seen and the aqueduct of Sylvius noted. The air markings over the frontal lobe are normal. (Courtesy, *Am. J. Psych.*, vol. 8, p. 5, March, 1929.)

One cannot justifiably ascribe to this fluid the purpose alone of a physically protecting "shock-absorbing" mechanism. Phylogenetically, this fluid system arose at the level of the lower fish and has been carefully retained in the central nervous system plan since the appearance of an outer protecting tube, wall, or bony cavity developed to surround the important nerve-centres.

A closer study of this mechanism seems to indicate a far more important rôle such a volume of fluid may play within the closed confines of the cranio-vertebral cavity.

When the craniovertebral cavity became a fixed and unyielding boundary for the housing of the brain organ, then some easily mobile and variable *volume* component had to evolve to expand or contract, depending upon the needs of the enclosed organ, in order to permit the high and low tides of cerebral circulation and the possibilities of swellings, tumors, and compensable accidents that might arise in the life cycle of the species. Certain it is that those forms of life which could not adjust the physical needs of the brain organ to such changes perished quickly without the possibility of reproducing their kind.

Spinal fluid arises as a dialysate from blood-plasma (Fremont-Smith¹⁰, ¹¹) and closely simulates lymph and œdema fluid in its protein content. It is readily restored into the venous channels and large sinuses of the dura. Here according to Weed¹² the pacchionian structures function as large filter beds. Fluid may enter directly into the small veins of the cortex (Dandy¹³, Kubie¹⁴) or be more rapidly passed through the larger areas offered by the pacchionian bodies when emergency readjustment of fluid volume is required. During periods of surgical shock and loss of blood volume the choroid plexus is said to draw freely upon the fluid collection in the ventricles, thus reversing its usual function.

The important fact remains, however, that spinal fluid volume and pressure may be rapidly diminished by withdrawal of this fluid into blood volume, thus not only assisting in the adjustment of volumes in the cranial cavity, but serving also as a source of emergency supply of physiological saline solution when the total blood volume of the body requires temporary replacement (Swift⁵).

In this sense, the spinal fluid reservoir responds, as other interstitial collections of fluid do, to assist in the balance of blood volume, so that adequate blood-pressures may be maintained throughout the body. This body of fluid situated in the most highly protected portions of the individual serves as a "safety vault" or emergency storehouse for water when hæmorrhage, "surgical" shock, and rapid loss of fluid from the skin or bowels require prompt replacement to maintain adequate vascular volume.

This fact is responsible for the fall in the fontanel of the infant when diarrhœa or dehydration occurs. It is the basis for the observations of Weed and McKibben who noted the fall in spinal pressure occurring when intestinal hydragogues were used in animals. It is the basis of purgation and

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sweating where so-called toxic substances ("water-intoxication" of Rown-tree) are eliminated from the body with clearing of the patients' sensorium and improvement in mental and neurological states. It is the age-old practice of medicine—purgation, bleeding and sweating—all of these measures serve to deplete the spinal fluid reservoir, provide space for a return of better cerebral circulation to the brain, allowing better cerebral function in a crisis.

The effectiveness of the use of hypertonic solutions by bowel or vein depends first upon an adequate subtraction of fluid from blood volume so that appropriate withdrawal of fluid from the body tissue reservoirs will be required. These indirect "dehydrating" methods fail if rapid elimination of water from the body is not obtained, or restriction of liquid intake practiced. Blood volume will not deplete its natural storehouses if other sources of fluid are easily obtainable.

As the principles of "dehydration" are based primarily upon this fundamental concept, it is important that the objective in treatment should constantly be the regulation and control of this compensatory cerebrospinal fluid volume by whatever clinical means now in our possession.

It is obvious that as swelling, hæmorrhage or tumor (clot) occurs within the craniovertebral cavity, the space occupied by these abnormal intruders must be surrendered by some other component. Naturally, we desire to retain the nervous structures and the brain organ intact if possible. We do not wish to sacrifice this brain volume (herniation, decompression, lobectomy) unless forced to do so. The volume of blood within the arteries, capillary bed, and veins must be maintained if cerebral cellular activity is to continue. The brain requires constantly large quantities of oxygen, proper nutrition and elimination of carbon dioxide, if its function is to endure. Where damage or injury has occurred even additional amounts of blood are needed to repair the wounded tissue. The same principles are involved in the repair and retention of tissue viability here as in other body tissues. If pressure and anæmia persist or reach the subvital level, degeneration and softening occur. A decubitus on the back undergoes the same cycle. Clinically we employ heat, massage, and stimulants to encourage a return of local circulation to the skin. The full integrity of circulation must be encouraged if we are to promote repair and prevent tissue death. The first step in the treatment of a decubitus is the removal of the offending cause of pressure so as to permit a return of blood to the anæmic area. In the brain the same principle must be promptly undertaken and constantly maintained as the craniovertebral bony walls not only limit our accessibility to the brain organ, but serve as a relentless barrier against relief from swelling and pressure within. Almost every other organ in the body finds some additional space for swelling, so as to permit continuation of its blood supply without sacrificing its remaining function. The limits of adaptability of an organ or member are determined by the character and integrity of its confining surfaces.

As brain cells experimentally show swelling and chromolysis after ninety seconds of complete anoxæmia, and degenerate completely after four minutes of such a state, it is obvious that cerebral circulatory volume must be maintained at all costs, and to the full need of the organ.

In view of the fact that fluids are incompressible, and that the cranio-vertebral contents can be divided roughly into: (a) red fluid (arteriovenous blood), (b) intracellular fluid (cells contain from 68–86 per cent. of water), and (c) clear fluid (spinal fluid and interstitial fluid), the volumes of these three components must vary reciprocally if the container does not expand or is not “decompressed” (Fig. 4). Furthermore, the entire brain organ

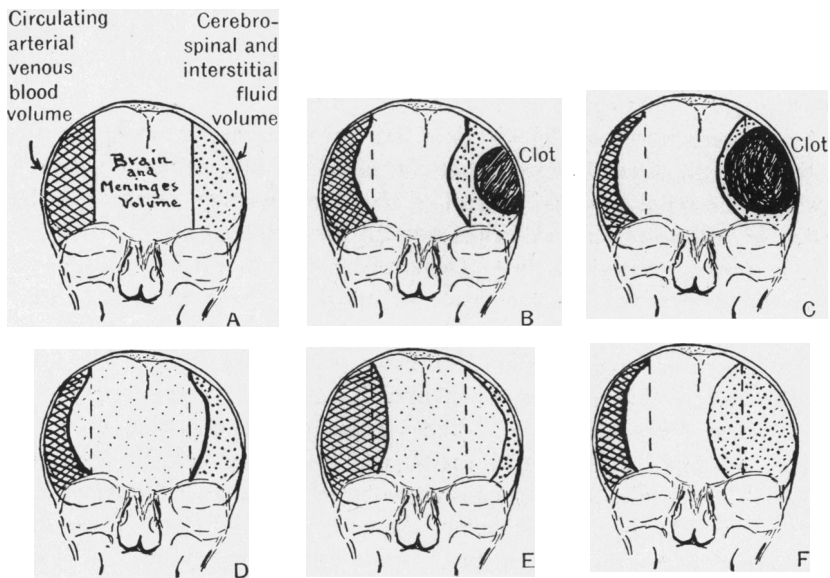


FIG. 4.—Diagrammatic representation of volume relationships within the skull with varying displacements of component volumes in certain types of cerebral trauma. The clinical symptoms are based upon compensation or decompensation between the component volume relationships. The importance of reducing the cerebrospinal fluid component is evident, in order to preserve the vascular and brain volumes, which determine function and the survival of the brain tissue.

A, normal; B, compensated volume displacement; C, decompensation with compression anæmia; D, cerebral œdema with compression anæmia; E, cerebral hyperæmia with œdema, increased intracranial pressure; F, cerebral atrophy, chronic increased intracranial pressure. (Courtesy S. G. O., vol. 54, p. 362, 1932.)

becomes affected by severe local changes in pressure, as pressure is transmitted equally to all parts by the spinal fluid cast.

We are now faced with a simple problem of physics and hydraulics. It is agreed that the cellular mass (b) should be maintained if possible. It is also obvious that the blood volume (a) must remain at its normal level, or may even require additional space for the hyperæmic phase of repair. Volume (c) is the only relatively unimportant and unnecessary component. Spinal drainage or direct ventricular tap forms a means of removing the free fluid portion of (c) volume (limit approximately 120 cubic centimetres or four ounces). Intravenous glucose or hypertonic solutions favor the depletion of the interstitial fluid component of volume (c). *The combina-*

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tion of these measures along with purgation, and the limitation of liquid intake to prevent replacement of the fluid subtracted, constitute the basis of "dehydration" as it applies to various conditions which affect the volume relationships of the craniovertebral cavity.

The control of spinal fluid volume becomes the key to the situation. Subtraction of spinal fluid volume permits needed blood to enter for purposes of repair and nutrition. Readjustments to cerebral volume increases are possible without loss of necessary blood supply within the limits of volume (c). Beyond this point the cranial cavity may be enlarged (decompression) but enlargement of the cavity is of little assistance if the extra space obtained is allowed to be immediately filled by a return of spinal fluid (unrestricted liquid intake, venoclysis, hypodermoclysis, enema, *etc.*). The curtailment of the production of spinal fluid so as to maintain the space secured by drainage or dehydrating measures is of prime importance. Here the experience of the past ten years has shown that adult patients placed upon a total daily intake of twenty ounces (600 cubic centimetres) of liquid and a dry diet (containing not more than twenty ounces of water weight) will not produce sufficient spinal fluid to increase intracranial pressure in the acute traumatic, after the second day of spinal drainage. Only a few cubic centimetres of fluid can be obtained in subsequent spinal taps and frequently a "dry" tap is encountered.

If, however, the patient is placed on thirty-two ounces (1,000 cubic centimetres) of liquid, increased pressure phases will occur and spinal puncture yields as much as thirty to sixty cubic centimetres of fluid with each drainage. Certain variations occur depending on the age of the patient, whether of the fat (hydrated) or thin (dehydrated) type, but in the large majority of cases the principle holds and has been verified in a series of traumatics, as well as chronic idiopathic epileptics.¹⁵

The clinician must recognize that he is dealing primarily with *volume* relationships and not alone with *pressure*. *Pressure is only an index of the force of apposition existing between the component volumes and is made possible because of the limits of the container.*

Confusion at once arises when pressure alone is used as an index, as is evidenced when one is confronted with a patient showing a high spinal pressure without the slightest symptoms of cerebral dysfunction, while on the other hand, a patient with low spinal pressure may be stuporous and show abundant neurological signs with only a few of the remaining brain centres intact. In the former instance (a) and (b) volumes have successfully been maintained in spite of the resistance offered by (c). In the latter, (a) volume has yielded its place to the swelling in (b) or the gradual increase in (c).

In the first instance the treatment concerns the question: What has occurred to prevent or alter the proper escape or adjustment in (c) volume? (Tumor, obstruction, chronic adhesions, meningitis, faulty filtration and drainage.) Assistance in the elimination and control of (c) volume must be

undertaken until the fundamental cause of obstruction is corrected, if pressure is to be returned to normal. Production of spinal fluid must be curtailed if possible.

In the case of low pressure and profound cerebral dysfunction, the problem concerns the vascular system perhaps locally or even from the cardiac and general blood-pressure standpoint. Certain it is in the vast majority of cases of this type, that there is a marked œdema of the brain, the cortex is gray-white, the tissues soggy (syncope, eclampsia, uræmia, terminal toxic states, traumatic and alcoholic wet brain, *etc.* Direct inflammation an exception).

The question that presents itself under these conditions is: How can an improved cerebral circulation (speed of blood-flow important) and increase in circulating blood volume be obtained? It is obvious that an increase in blood volume (a) cannot be obtained until space for this is provided by subtraction from (b) or (c) volumes or enlargement of the cranial cavity. It is likewise obvious that there must exist sufficient blood in the general circulation and that this blood must be pumped into the cranial cavity under sufficient pressure to force its way back into the obliterated cortical capillary network. Furthermore, the appropriate supply of oxygen must be carried by the red cells, and proper oxygen tension maintained to permit delivery to the failing nervous centres.

This problem requires not only the judicious use of all measures at our disposal to directly alter (b) and (c) cranial volumes, but an attack on the etiological factor producing the disturbance of general body water metabolism. Proper elimination of fluids through the skin, kidneys and bowels, regulation of the peripheral capillary bed tone, and stabilization of blood-volume, pressure, and red cells, are considerations which will determine the success or failure in the management of such a case. Under these conditions, "dehydration" plays only an important contributory part in the treatment employed.

The fact that blood cannot enter the cranial cavity unless space is allowed for it, is a difficult idea to convey, because the incident is taken for granted and the adjustment is usually so automatic and perfect.

This space normally is yielded within compatible limits depending upon requirements without a change in pressure, by the fluctuating volume of venous blood in the paravertebral spaces and large dural sinuses.

Spinal fluid may be expelled from the cranial cavity so as to make possible changes in volume at many points in the system. Further space may be obtained when "brain mass" increases, by gradual displacement of spinal fluid volume, obliteration of the subarachnoid spaces and distortion of the ventricles. So long as this adjustment can occur, symptoms of "pressure" do not arise. This mechanism is frequently seen in gradual subdural hæmorrhages (with actual displacement of the pineal gland) or the slow growth of a brain tumor with narrowing of a ventricle. If, however, the point is reached where the volume of spinal fluid can no longer yield, or its path-

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ways of elimination are blocked, pressure symptoms promptly ensue, with the clash that arises between (b) and (c) volumes for space within the cranial cavity. As cerebral circulatory volume, either local or general, must now yield its space instead of spinal fluid, local or generalized symptoms of cerebral pressure and anæmia arise.

One may usually trace the steps of such a process through the history of headache, vomiting, dullness (weakness, paralysis, focal dysfunction), irritability (delirium and convulsions), stupor, and finally coma.

HEADACHE.—There is a corollary to this mechanism of adjustment for increased volumes which is encountered in cases of cerebrospinal fluid leak, and often in post-lumbar puncture headache. When spinal-fluid volume is constantly lost beyond its regulatory level, (a) and (b) volumes must replace the space. This usually favors a marked hyperæmia and congestion in (a) volume above its necessary levels. As the pain fibres responsible for

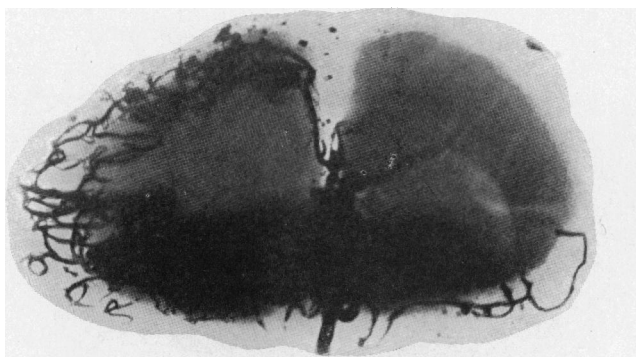


FIG. 5.—Mercury injection of the middle and anterior cerebral artery. The basket-like cortical network has collapsed. When the ventricles are distended this network of vessels is tightly stretched. (Courtesy J. A. M. A.)

this sensation are located upon the arterial branches over the surfaces of the brain, as well as a few on the large venous sinuses, *the symptom of headache arises from vascular congestion and traction on the pain network of these over-filled structures*. In order to *reduce* blood volume (a), volume (b) or (c) must increase. Here, forcing of fluid, pituitrin to shut down renal elimination (Fremont-Smith) and raise spinal pressure through body-tissue retention of fluid (force an increase of (c) volume against (a) volume), vasoconstrictors to cut down the volume of (a), ice to the head, and heat to the extremities favor a better blood distribution to the cranial cavity, bringing about the relief of headache as a symptom, by better readjustment of (a) volume. Spinal pressure in these cases is low and the objective must be therefore to introduce sufficient volume in (b) or (c) to offer a higher pressure resistance to (a). As pressure rises and more spinal fluid is produced than lost (a) volume recedes, the symptoms abate, and a more normal relationship occurs. (“Hydration” instead of “dehydration.”)

Headache in the great majority of cases, however, is due to increased intracranial pressure, because of an increase in (c) volume or its failure to yield

space required by a growth in (b) or hypertension in (a). Here, although (a) volume may be actually diminished, the subarachnoid spaces are over-filled, the arterial vascular tree is stretched by the distended arachnoidal trabeculae and the ventricular tension of fluid expands the convex surface of the brain. The entire cortical vascular network becomes taut and pulsating. This pulsative elasticity produces traction on the vascular tree and the pain fibres register the same warning symptoms of headache as they did when the vessels themselves became overdilated with blood from within (Figs. 5 and 6).

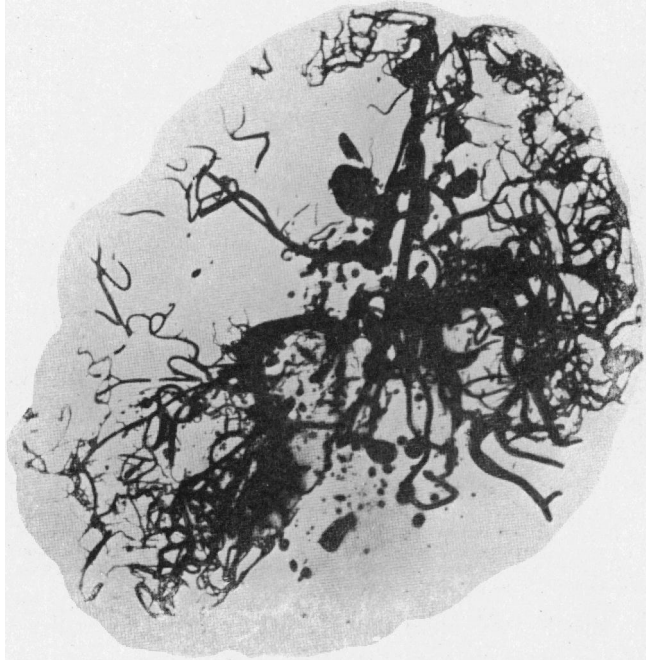


FIG. 6.—Superior view of the brain showing partial distension of the vascular network (X-ray photographs). (Courtesy J. A. M. A., vol. 84, 1927; June 6, 1925.)

This type of pressure headache is immediately relieved by ventricular tap (release of structural tension) because the distended vascular network and cortical brain mass are permitted to relax. Spinal drainage depletes the subarachnoid and ventricular fluid spaces so that the vascular tree is released from its stretched condition. Clinical measures of “dehydration” accomplish the same result in a more gradual manner. Each method has its individual or combined indication as the case may be, but all are directed at the reduction of spinal-fluid volume to relieve the mechanical traction placed on the arterial network from the circle of Willis throughout its branches.

If the objective is to control the symptoms of headache alone, the clinician must not overlook the fact that withdrawal of an appropriate amount of

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spinal fluid will adjust the distention factor and bring about the desired relief. However, if spinal-fluid volume is reduced below its ordinary limits, an increase in vascular volume will be encouraged and with hyperæmia there follows headache of the low pressure type to replace the former, and as the patient is scarcely able to detect the difference between these two extremes, the clinician is frequently puzzled because expected relief has not been apparently obtained. "Dehydrating" measures if added under these overwithdrawal conditions only increase the headache. The operator is convinced that as he found evidences of increased pressure at his initial examination, a similar condition has returned. A recheck of spinal pressure should be done in order to reveal which of these headache states prevail, and treatment may then be instituted accordingly. Fortunately, most pressure-headache mechanisms are of the type caused by some obstruction to the proper circulation of spinal fluid, and the reappearance of headache is in reality due to the prompt return of fluid to its excessive state. "Dehydrating" methods are therefore usually most beneficial, and only when excessive require a reversal of application.

Since the limitation of liquid intake has come into clinical use to control the rate of production of spinal fluid, the factors noted above must be more carefully considered. The general rule of "pressure headache" that formerly prevailed now no longer holds. Following lumbar drainage the patient used to be allowed to indulge freely in liquids, again permitting rapid replacement of the fluid withdrawn and a return of pressure. Consequently, the results of treatment were more variable and the measures utilized more heroic.

The symptom of headache, in order to be properly interpreted and treated, must be considered from two standpoints: (1) Stretch on the pain end-organs of the vascular tree, due to overdistention of the vessels (hyperæmia, increase in volume (a)), or (2) stretch on the pain end-organs, due to overdistention of the subarachnoid spaces and the ventricles ("pressure" headache, increase in volume (c)).

Headache of the first type (hyperæmic) may arise from extracranial conditions such as cellulitis of the neck or scalp; paranasal sinus infection, involvement of the tonsils, pharynx and mastoids. Here the cerebral circulation is the unwilling recipient of an increased blood volume designed primarily for the branches of the external carotid artery, but no mechanism of regulation exists at the bifurcation of the common carotid artery to direct the increased amount of blood sent by the heart and required by the infected areas, so that it is alone confined to the external carotid distribution. The internal carotid shares in this increase of blood volume to some degree, and the consequent rise in (a) volume intracranially is frequently associated with headache.

The author has pointed out¹⁶ the existence of these vascular pain fibres on the internal and external carotid arteries and its branches. The symptoms of deep, dull, throbbing, aching pain in the head, as well as the similar

type of pain encountered deep in the face and known as "atypical facial neuralgia" ("lower half headache," Sluder's syndrome) are, in the author's opinion, due to hyperæmia and irritability of the vascular pain fibres. Frequent observations on patients under local anæsthesia indicate that the brain itself is sensitive to a deep, dull, aching pain *only* when the arterial branches are stimulated by faradic current, or the vessels themselves or their attachments are subjected to irritation and traction.

The patient under local anæsthesia is not conscious of such stimulation when applied to the cortex or meninges. The dura itself appears to be insensitive, excepting at the points where traversed by the branches of the meningeal artery. The two lateral sinuses, the superior and inferior sinuses and portions of the tentorium adjacent to the large veins respond to pain stimuli. The patient usually refers the pain deep in the head, behind the eyes; occasionally to the general area of the vascular distribution on the side stimulated.

These vascular pain fibres follow the vessels out of the skull, to their source along the carotids and vertebrales. Some may be found on the jugular vein and in the carotid sheath. They continue down into the upper thorax and find their way into the spinal cord along the sixth, seventh and eighth cervicals, and the first, second, and third thoracic roots (Fig. 7).

The existence of myelinated and unmyelinated nerve fibres on the cerebral arteries has been demonstrated by Hassin.¹⁷ The course and distribution of these vascular pain fibres have been substantiated by Foerster,¹⁸ and more recently by Cobb.¹⁹

Headache as a symptom, therefore, may be considered as a direct involvement of the vascular pain network by distention and traction, as the most common mechanical cause, although trauma, infection and intrinsic inflammatory disease may also involve them directly. Appropriate measures may be instituted to relieve the condition when the mechanism responsible is definitely established.

VOMITING.—Vomiting may be considered a reaction of defense designed chiefly to protect the individual against: (1) Retention of irritating or poisonous foods; (2) consumption of food or fluid when the gastro-intestinal tract is unable to function or is pathologically obstructed; (3) hydration states usually associated with increased intracranial pressure.

The first two properties of this defense reaction do not concern the subject of intracranial pathology and the specific reactions to toxic drugs; vestibular, ocular responses, psychosensory or emotional manifestations have their own sphere of analysis.

The third consideration of the vomiting reflex has its important part to play in cerebral trauma, as well as other intracranial pathology. Not only is vomiting an indication of the patient's rejection of further food or fluid at a time of cerebral distress, but the mechanism serves the purpose of throwing out from the body a portion of the excess of fluid which threatens the survival of the important nervous centres, thus preparing for the reduction

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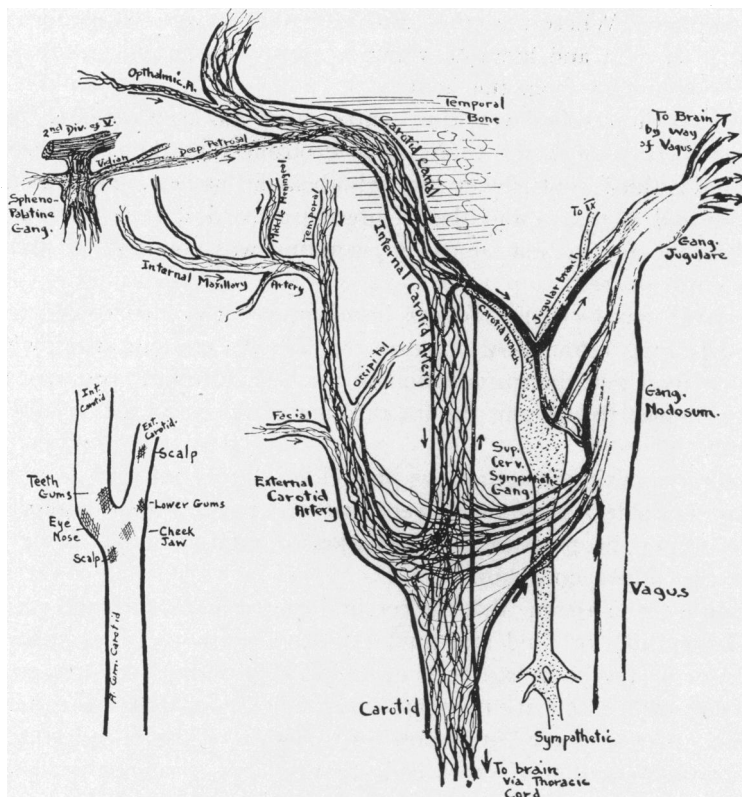


FIG. 7.—Pain network on the vascular tree. These fibres are gathered from the various branches and descend along the carotid arteries in the neck. Some fibres turn back into the vagus while others continue along the jugular and carotid sheath into the thorax entering the lower cervical and upper segments of the thoracic cord (C₆ to T₃). (Courtesy *Annals of Otol., Rhinol. and Laryngol.*, vol. 41, Dec. 4, 1932.)

| Arterial Branches Stimulated | Pain Reference |
|------------------------------|--|
| 1. Meningea Media | Deep in the eye Temporal region; deep |
| 2. Cerebri Media | Deep in the eye Headache |
| 3. Temporalis Superficialis | Ear Scalp temporal region |
| 4. Maxillaris Interna | Eye Malar region |
| 5. Occipitalis | Scalp occipital region Back of neck |
| 6. Maxillaris Externa | Inner canthus of eye Cheek |
| 7. Lingualis | Tongue Tonsillar region |
| 8. Carotis Externa | Scalp, malar region Gums, teeth, jaws |
| 9. Carotis Interna | Deep in ear Deep in eye Headache |
| 10. Bifurcation | Scalp, ear, nose, teeth Gums, cheek, jaws, tongue |

of (c) volume. Where the other important portals of elimination are deficient, such as skin and kidneys, vomiting may arise as an accessory means of fluid elimination from the body.

Although the cerebral vomiting centres have not been definitely localized to a particular area of the brain, it is probable that they lie close to the walls of the third ventricle in the hypothalamic nuclei. The recent work of Fulton and Watts²⁰ established a precentral cortical zone concerned with peristalsis but no reaction similar to vomiting was obtained by stimulation of this cortical area.

Vomiting may be easily induced in the procedure of encephalography by rapidly changing intracranial pressure through the mechanism of withdrawing spinal fluid and the introduction of air. If, however, pressure is carefully maintained throughout the interchange of fluid and air, vomiting does not occur.

Cerebral vomiting is not always projectile in type, but may be responsible for a considerable loss of fluid over what had recently been ingested. The stomach serves the purpose of elimination of fluid from the blood volume as does the kidney and skin.

If one were to assume that the cerebral centres had developed the mechanism of vomiting, or food and fluid rejection, when the total needs of the storehouses of the body had been exceeded, then many symptom-complexes would be brought into a far more understandable light. For instance, a paradox seems to exist where "hydration" of the brain occurs in conjunction with an obvious dehydration of other body tissues (*viz.*, terminal hydrocephalus, meningitis, paresis). The "hydration" here is in reality an overaccumulation of cerebrospinal fluid and not cerebral oedema. As long as the body hydration protective centres are located within the cranial cavity, it would be logical for them to react to pressure states of this type, as though the entire body were affected by a state of overhydration similar to that existing in the brain.

It is here that many differential diagnostic features arise. If the gastrointestinal tract as the offender has been carefully ruled out and the signs of hydration are not in evidence elsewhere, the clinician is driven to suspect the brain and look for increased intracranial pressure. Simple general hydration states of the body are frequently overlooked, and excessive ingestion of liquid not checked. Vomiting under this circumstance (plump infants, alcoholics, eclamptics, *etc.*) may be due entirely to water "intoxication" and the cerebral and gastro-intestinal systems be erroneously suspected of pathological involvement.

Fat, hydrated infants and children are more apt to vomit following head trauma than the adolescent or adult. The symptoms of vomiting may be influenced by the general state of water metabolism throughout the body. Its significance must therefore be judged by the age of the patient and the state of the individual's storage reservoirs. It may be a symptom therefore of only slight importance in some (hydrated types) or of profound significance in others (thin, dehydrated types).

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Prompt and active purgation with hypertonic salines, along with strict limitation of fluid intake, will usually control the general hydrated type. Initial spinal drainage and appropriate fluid regulation will assist the dehydrated group.

It has been a striking fact that post-traumatic and increased intracranial-pressure cases will tolerate small portions of solid food as soon as consciousness has been reestablished, whereas liquids are promptly vomited and subsequently all foods are rejected.

STUPOR AND APHASIA.—A careful neurological examination should be made, with particular reference to the size of the pupils, their reaction to light and the presence of hemianopsia, if this can be demonstrated. Facial weakness should be carefully sought even in semi-stuporous patients. This can be demonstrated usually by pressure upon the supra-orbital nerves.

Aphasia, in contradistinction to stupor, is one of the most important signs to be considered. A patient may give the impression of stupor when he can neither speak nor understand. This may be an important focal sign which is often mistaken for stupor, and in reality the location of the lesion might be determined by this finding alone. If the patient be right-handed the lesion is probably in the left Sylvian area. There may be an associated weakness or paralysis of the right arm and if so aphasia must be carefully considered. The best means of determining the difference between aphasia and stupor is to produce an extremely painful stimulus on the patient and note his reaction. If partly stuporous or wholly unconscious he may rouse sufficiently to make *purposeless* efforts to react in a general way to the pain (groaning, squirming, restlessness). On the other hand, if aphasic, the patient will attempt to remove the painful stimulus in the ordinary way. Thus if severe pressure be made upon the supra-orbital nerve the face may draw up on the side of the pressure (test for facial paralysis), but the patient may also make a definite *purposeful* effort with the unparalyzed hand to remove the painful pressure exerted by the examiner, indicating knowledge of the painful stimulus, its location, and how to prevent it.

When stupor or aphasia is profound the most responsive area to pain will be found in the upper and inner aspect of the thigh, and here, if the folds of the skin are grasped firmly and pinched with force, a marked reaction on the part of the patient is usually forthcoming. If no response can be obtained from this area, the patient may be considered profoundly stuporous. Rectal dilatation at times may be a means of arousing such a case. The test determines the degree of physiological continuity of the cortex, which still remains, and where all efforts fail to rouse the patient, the cerebral injury or pressure may be considered as being extremely widespread.

RESTLESSNESS.—The most frequent finding is that the patient shows extreme restlessness and violently resists restraint, so that if cerebral bleeding is present this may be greatly increased and the condition of the patient becomes worse for the need of quiet and relaxation. Here it is important to give sodium luminal, grains 1 to 3 hypodermatically, and large doses of

chloral hydrate and bromide by rectum. An adult dose may be fifteen grains of chloral and thirty grains of bromide by mouth, or if impossible to administer the sedative in this manner, double the dose by rectum. The same dose may be repeated in four to six hours if necessary. Morphine or its derivative should *never* be given unless other sedatives fail, for there usually ensues respiratory difficulty due to intracranial pressure and oedema with additional depressor effect of the narcotic.

INITIAL OBSERVATIONS.—The neurological examination should also include testing of the reflexes, especially the Hoffmann reflex (snapping the middle fingernail with the hand in mid-pronation. If accompanied by a response of approximation of the index finger and thumb, the sign indicates involvement of the upper motor neuron for the hand centre). The Babinski reflex is extremely important and must be carefully taken, using a variety of degrees of plantar irritation on the outer aspect of the sole. The Oppenheim sign may often be obtained when other signs are absent, and suggests the laterality of the lesion. The tendon reflexes may give a clue if unequal and unilaterally exaggerated, but frequently their entire absence makes these signs unreliable. Clonus is rarely present during the early stages following a head injury. A most important observation when profound stupor is present may be the determination of muscle tone by grasping the patient's wrist and lifting the arms directly upward to their full length, permitting them to fall of their own weight against the sides. On the side of a focal paralysis the extremity will frequently demonstrate a "flail-like" reaction, falling heavily and limply to the bed. The uninvolved extremity will also fall promptly but one may observe the more gradual return of the part to its former position.

The temperature, pulse and respirations should be determined every fifteen minutes, the blood-pressure every half hour and the treatment for shock instituted immediately upon admission by the giving of 50 per cent. glucose solution intravenously. The period of shock should have been passed within the first one or two hours following admission if the patient has been properly treated. The necessary care of the wound, the above neurological observations, the spinal puncture with fluid pressure, and the determination of the character of the fluid now present sufficient evidence to determine the subsequent course of action. This may be considered under three large groupings with occasional overlapping, and consequent modifications of the treatment. Representative cases from each group will be considered in detail, the exceptions and the combinations of treatment depending upon the best judgment of the physician in charge.

It is therefore necessary to outline certain fundamental considerations upon which the basis for the treatment will depend.

THE PULSE RATE.—From the standpoint of an acute cerebral injury we may assume that the patient probably had no organic vascular disease prior to the injury, although frequently exceptions will occur. The pulse rate, therefore, must be viewed in the light of its determining factor, as changes

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in the pulse rate become important considerations in the progress of the patient.

It must be borne in mind that the vagus control of the pulse rate produces normally approximately eighty beats per minute. If irritation of the vagus centre is present or higher centres influence the vagus, the rate may fall as low as forty and I have observed in one case a rate of thirty-eight, present for a period of twenty-six hours (vago-tonic states should be relieved by atropine). It is unusual to find a rate which falls below fifty in the ordinary individual unless heart block has been induced by digitalis, or some disturbance of the auriculoventricular bundle is present within the heart itself. In the post-traumatic, however, when the pulse rate falls to seventy this should be cause for close observation and if it reaches sixty-eight or below the attending physician should search for the cause of this vagus irritation. Bloody spinal fluid, increased intracranial pressure, œdema of the cardiac centre, or subdural hæmorrhage must be considered. Consequently the record of the pulse at fifteen-minute intervals throughout the first forty-eight hours is of extreme value in determining the appearance of complicating factors which require early attention (Fig. 8).

On the other hand, should the vagus centre be paralyzed or temporarily lose control of its regulatory action on the heart, a rate of 120 or over will probably ensue. No reliable information, therefore, can be expected of the pulse above 120 in regard to intracranial pressure. If vagus tone has thus been removed one must not overlook the effect of atropine if given during the period of shock as this drug tends to release vagus influences.

A pulse rate over 120 may be considered as the demand of the heart for more fluid volume, and is frequently found in shock. The rate must be considered in the light of sympathetic irritation, restlessness, or loss of circulatory fluid volume and treated accordingly. In the absence of a demonstrable sympathetic irritant (hyperthyroidism, emotion, restlessness, *etc.*), the pulse rate of 130 or above indicates the need for more fluid to replenish blood volume and this may be met by repeating the injection of 50 per cent. glucose solution intravenously with an appropriate small amount of normal saline solution if necessary.

The clinician must have clearly in mind the fact that fluid, though given intravenously, may rapidly leave the circulating blood-stream (Fig. 9) to be deposited in interstitial spaces or given off by perspiration from the skin surfaces. This loss of fluid volume may be directly from the vascular compartment and the circulation is temporarily unable to reclaim this fluid, so that a rising pulse rate indicates the need for further administration of

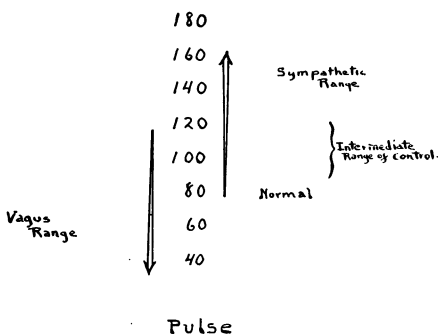


FIG. 8.

appropriate solutions to maintain blood volume. As pointed out above, great care should be taken not to introduce fluid that may be rapidly dissipated into the tissues, for fear of precipitating cerebral oedema; hence hypertonic glucose solution should be resorted to in order to assist in reclaiming vascular volume during this period of temporary disturbance.

Much can be determined by the character of the pulse in regard to the peripheral resistance which blood volume is encountering (vasomotor tone,

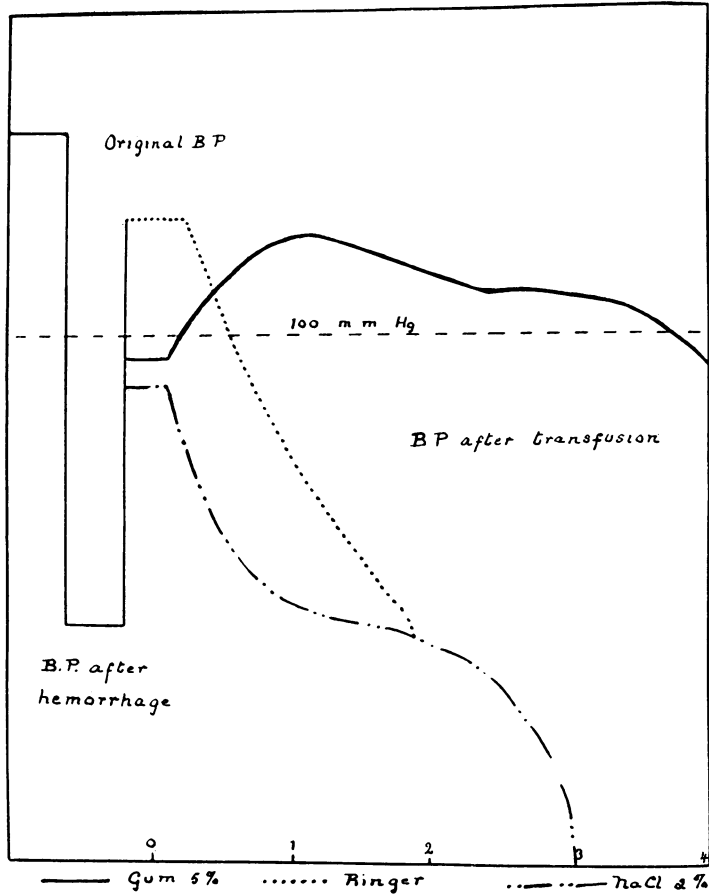


FIG. 9.—Composite curves to show effects on blood-pressure of hæmorrhage, and transfusion with various solutions (N. M. Keith, from Bayliss). The average pressure in the various experiments before and after hæmorrhage is given on the left in a continuous line. The behavior of the pressure after transfusion varied according to the solution used. (Courtesy Macleod, *Phys. and Biochem.*, 6th edit., p. 358. C. V. Mosby & Co.)

capillary bed) in that a rapid, running, thready pulse, easily compressible, signifies either the container of blood volume (vessels) is too large (vasodilatation) or that even with a normal or vasoconstrictor state of the vessels, blood volume is too small to fill the structures. In the former instance vasoconstrictors (pituitrin, ephedrin, ergot) are indicated and not cardiac stimulants. In the latter an increase in blood volume is required (trans-

fusion, glucose, saline). Diastolic-pressure readings help to establish the state of peripheral resistance. In order to relieve the rapid pounding of the heart as well as to raise blood-pressure, maintenance of capillary-bed constriction is important.

The analogy may be drawn to a fire hose without a nozzle. A stream of fluid passing through this hose finds practically no resistance and hence there is no "back pressure." This is similar to the dilated state of the capillaries in the periphery during shock and vasodilatation, the blood running around the circuit without resistance or delay. Returning to the fire hose, if the nozzle be placed upon the end and the outlet thus reduced, "back pressure" becomes sufficient to require a firm fixation of the nozzle by strong hands. The pulsating tortuosity of the hose gives evidence of the fullness of volume working against resistance. This is true of the peripheral capillary bed, which, if properly contracted, produces a rise in diastolic pressure and affords a full resistance for the contracting ventricles of the heart to work against.

The important phase of this physiological consideration is two-fold. In the first place, when diastolic pressure in the peripheral capillary bed has fallen so that it is less than forty millimetres of mercury, oxygen leaves the red blood-cells according to the laws of oxygen dissociation, long before it reaches its intended destination. The outlying cells are thus denied the proper amount of oxygen and cyanosis is usually apparent. With the anoxæmia, increased permeability occurs (Landis²²) and œdema as well as the rapid loss of function of the tissue cells themselves. One must realize that the periphery is not to be considered only the hands and feet, but is as truly represented in a similar degree by organs close to the heart and in the heart itself. Thus the liver, kidneys, brain and other important structures suffer greatly during periods of vasomotor relaxation and anoxæmia. The object should be to maintain oxygen in not only the vital centres but to the entire organism as death is not alone the failure of a single centre but frequently the overwhelming accumulation of gradual functional loss throughout the structures of the entire body.

The character of the pulse, therefore, becomes important in determining the probable vascular volume and the resistance which the cardiac pump meets in the peripheral capillary bed.

We have come to consider the heart rate in intracranial trauma as being significant of vagus irritation if below 70 and as the "cry of the circulation for more fluid" if the rate be above 120, with allowances for age and evident cardiovascular disease, which must be taken into consideration.

RESPIRATIONS.—The respiratory rate is of extreme importance as it frequently gives warning in advance of an impending catastrophe. When the rate is above twenty-six, cerebral irritative factors should be suspected and the lumbar puncture usually reveals bloody spinal fluid. Respiratory rates which reach forty indicate a profound cerebral disturbance and are in themselves not so serious as the fact that such rapid breathing produces hyper-

ventilation and the excessive loss of carbon dioxide, which in turn favors alkalosis and œdema. As alkalosis is undesirable because it favors tissue œdema; it has been our custom in profound cases to permit the rebreathing of carbon dioxide by placing a wet towel fashioned in the shape of a cone over the patient's nose and mouth. A stream of oxygen may be added through a small tube into the cone if rebreathing be confined to a closed system. This method not only stimulates deeper respiration but frequently diminishes the rate and may in border-line cases be extremely beneficial.

The most important significance lies in respiratory rates below eighteen. As the respiratory rate diminishes, there is usually associated increased intracranial pressure, which may be relieved by spinal puncture and drainage. œdema of the respiratory centre may also produce a fall in respiratory rate but this is usually associated with shallow respirations of the Cheyne-Stokes type. Hæmorrhages into the floor of the fourth ventricle frequently produce this type of breathing, as well as hernia of the brain into the foramen magnum and the less recognized condition of hernia of the temporal lobes into the incisura of the tentorium when generalized marked œdema and swelling of the brain are present. If spinal drainage be delayed beyond the forty-eighth hour this type of brain œdema frequently occurs and spinal drainage instituted at this time may be extremely dangerous in that it favors progressive herniation and little fluid can be obtained. Shallow respiratory rates indicate a failing centre.

Respiratory stimulants, such as atropine, caffeine, and carbon dioxide, are indicated when respirations fall below sixteen. Fifty per cent. glucose by vein, or magnesium sulphate by mouth may assist in reducing medullary œdema and thus relieve a respiratory rate which is impaired by pressure or actual œdema. The patient should be turned onto the face and the foot of the bed elevated 45°. Pillows placed under the chest make it possible for satisfactory breathing to be maintained. This measure alone frequently relieves Cheyne-Stokes respirations.

Emergency measures should be introduced when respirations reach ten or twelve per minute as a complete respiratory failure may suddenly occur at this point. One should always be prepared to introduce an intratracheal catheter (woven) so that oxygen may be given directly into the lungs and thus avoid the traumatizing effect of prolonged manual artificial respiration.

In several cases this has been the means of saving the patient's life when sufficient time has not elapsed to deal with the pressure mechanism until after respiratory failure has developed. Sufficient exchange of oxygen and carbon dioxide occurs in the lung when a catheter has been properly placed into the trachea and attached to an oxygen tank to obviate the need of inspiration and expiration. Respirations may spontaneously recur even after a period of an hour or more (seventy-eight hours in one instance) following complete cessation, providing sufficient oxygen has been supplied to the lung bed and circulation maintained. Morphine or its derivatives should never be given where respiratory embarrassment has been indicated by a rate below

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eighteen as prompt respiratory failure has been observed under such conditions (even after the use of one-fourth grain of codeine).

A falling respiratory rate in the presence of clear spinal fluid should give the physician anxiety over the possibility of a progressive subdural hæmorrhage. In uncomplicated cases where œdema is the cause of the fall in respiratory rate, magnesium sulphate crystals, one and one-half ounces in water by mouth, or three ounces by rectum will be found efficacious in relieving the respiratory depression.

A fifteen-minute respiratory record should be maintained during the period of serious manifestations as this curve will serve to indicate the load placed on the respiratory centres and frequently warns the clinician of progressive increased intracranial pressure, when the rate is below eighteen, shallow or irregular. Rates above thirty should be considered in terms of hyperventilation or alkalosis. Results of prolonged hyperventilation are to be avoided, as they favor tetany, decerebrate rigidity and general tissue œdema, augmenting the usual pathological signs.

TEMPERATURE.—Temperature is for the most part influenced by the regulation of evaporation of moisture from the skin surfaces. The subnormal temperature noted in shock is probably due to the rapid loss of fluid through the skin onto the body surfaces. It becomes important, therefore, to maintain a dry skin surface to check this source of heat loss and the application of external heat to assist in the body's reëstablishment of this important factor. *The duration of the subnormal temperature*, therefore, may be considered as *an indication of the period of shock* and the time required for readjustment of vasomotor relaxation associated with a leaky skin. In hyperthermia the skin is usually dry and hot with little available moisture for evaporation and hence insufficient radiation. Temperatures rapidly rising as high as 107° and 108° indicate hæmorrhage or œdema *within* the brain substance, usually between the striate region and the medulla. Transitory disturbances of the vasomotor or heat-regulating centres need offer no concern provided temperature is not permitted to go above 103° . Sponging should be introduced when the temperature reaches 103° , and maintained at half-hour intervals, if there is a tendency toward a further rise. This is best accomplished by applying a wet bath towel to the entire exposed body surfaces, using tepid water and leaving the skin wet and exposed, permitting rapid evaporation, meanwhile protecting the patient from any direct draught. It is useless to expect results if the skin is dried after the sponging; it must be kept wet constantly and exposed without coverings. Too frequently the patient is heavily covered with blankets which do not permit proper radiation.

A fall of 1.5° of temperature within ten minutes following such methods of sponging has been noted. Occasional continuous cold colonic irrigations, composed of hypertonic magnesium sulphate solution, may be resorted to when the danger point is exceeded. Rapidly rising temperatures above 105° usually indicate a terminal condition and strongly suggest an intramedullary

hæmorrhage within the brain substance. The temperatures above 107° rapidly destroy the function of the brain cells, if acquired in a short space of time, and do not yield to methods of body-heat reduction. It is therefore important that rapid rises of temperature be checked in their early stages of progress, and imperative that sponging be resorted to when 103° are registered by the patient. The author has used phenacetin in small doses (grains 3) to assist in the production of a moist skin and thus favor a reduction in temperature.

The temperature, therefore, when taken at fifteen-minute intervals may indicate the duration of the period of shock as well as the presence of a deep-seated intramedullary hæmorrhage. Few cases are on record where a patient with an intramedullary hæmorrhage larger than a walnut has survived,

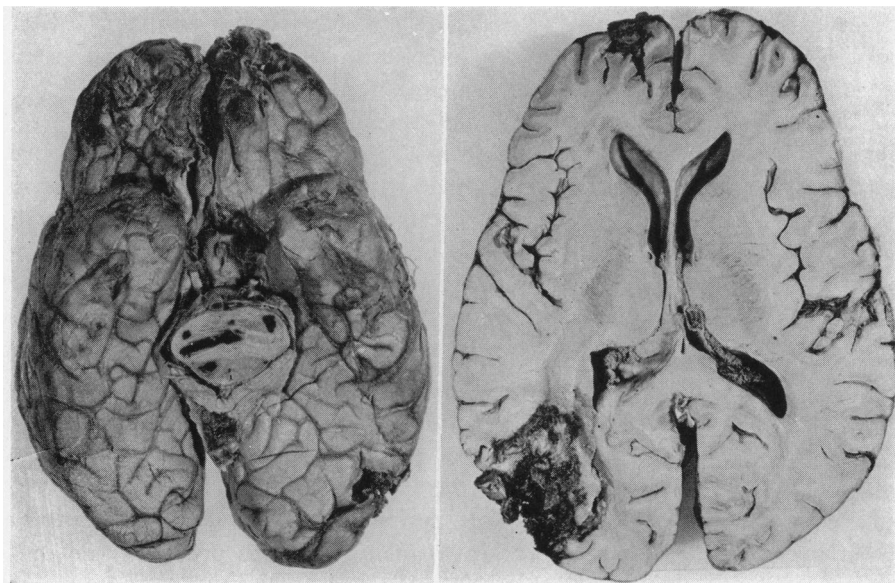


FIG. 10.

FIG. 11.

FIGS. 10 and 11.—The effects of contrecoup injury with compound fracture of the left occipital pole with transmission of the force contrecoup, producing extensive laceration of the under-surface of the right frontal lobe as well as the mesial tip of the left frontal lobe. Note the hæmorrhage in the pons with destruction of the vital centres lying in the diagonal of the transmitted force. (Preparation by Dr. N. W. Winkelman.)

whether spontaneous or secondary to trauma. The profound physiological and neuropathological disturbances created by such a lesion are almost invariably fatal (Figs. 10 and 11).

BLOOD-PRESSURE.—Blood-pressure readings in cerebral trauma become the guide and greatest source of reliance to the clinician in the treatment and management of a case. If frequent readings are taken and charted, a curve is obtained which foretells the approaching dangers and indicates the appropriate time for preventive measures. In the author's opinion this record is of more value than any other. It is important that a blood-pressure reading be made upon the patient at the earliest possible moment after admission

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and where the patient's condition is serious, due to shock or stupor, these records should be taken at fifteen- or thirty-minute intervals.

The *diastolic pressure* and the *pulse pressure* are the outstanding considerations from the standpoint of survival of the patient as well as proper treatment.

Systolic pressures are of little or no concern as long as they are maintained above 75 or below 200 millimetres mercury. The systolic reading represents the driving force of the heart against the peripheral vascular resistance. The cardiac mechanism in cases of cerebral injury is usually organically uninvolved and as long as there is sufficient pressure to drive the blood around the vascular circuit, there is no need for direct cardiac stimulants (caffeine sodium-benzoate, adrenalin, digitalis), as these only overexert the cardiac muscle without correcting the fundamental disturbance in the peripheral capillary bed.

Diastolic pressure, however, represents the peripheral vasomotor bed and its tone. In reality, diastolic pressure is life itself, although too frequently the physician disregards this important figure. It represents the constant low ebb of vascular circulation in terms of the diameter of the peripheral capillaries, thus as already noted above under the pulse, when the capillaries are widely dilated the blood passes through the larger arterioles without entering the smallest capillaries. As the vast majority of functioning tissue cells are supplied from the capillary meshwork, if life is to be maintained, circulation must reach these tissue cells not only with nutrition values but with oxygen, which must be maintained at an oxygen tension of approximately twenty millimetres mercury. Thus at sixty millimetres mercury (Macleod²¹), blood may be nearly saturated with oxygen, whereas at pressures below fifty millimetres it readily loses oxygen, so at ten millimetres there is complete reduction. When diastolic pressure reaches forty the oxygen dissociation curve has reached a point where oxygen on the red cell is no longer available when it reaches the capillaries, and thus "anoxæmia" may be present even though the red cell passes close to the tissue requiring its normal supply of oxygen. As a shift of the pH to the acid side gives a better utilization of the combined oxygen on the red cell, it will be seen that hyperventilation and alkalosis enhance the degree of anoxæmia by increasing the tendency for hæmoglobin to retain its oxygen even under somewhat favorable pressures. Landis²² has pointed out that anoxæmia increases permeability and permits transudation of fluid into the capillary interspaces at *four times* the normal rate. Thus œdema will add to the circulatory deficiency, increasing in a vicious cycle the limitation of oxygen to the tissues, without which they cannot carry on their normal physiological function.

In diastolic pressure, therefore, we are dealing with the crux of the situation and this must be maintained above forty millimetres mercury by every effort within our means, as the function of the cells themselves which determines the patient's existence will depend upon this factor alone. The physician's attention should therefore be directed toward contraction of the

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peripheral capillary bed in shock and at times of vasomotor relaxation. Increase in diastolic pressure favors more uniform oxygen dissociation, better utilization, and thus maintains the functions of the tissue cells. Due regard must be given to conditions that tend to promote alkalosis and oedema, and corrective measures instituted early.

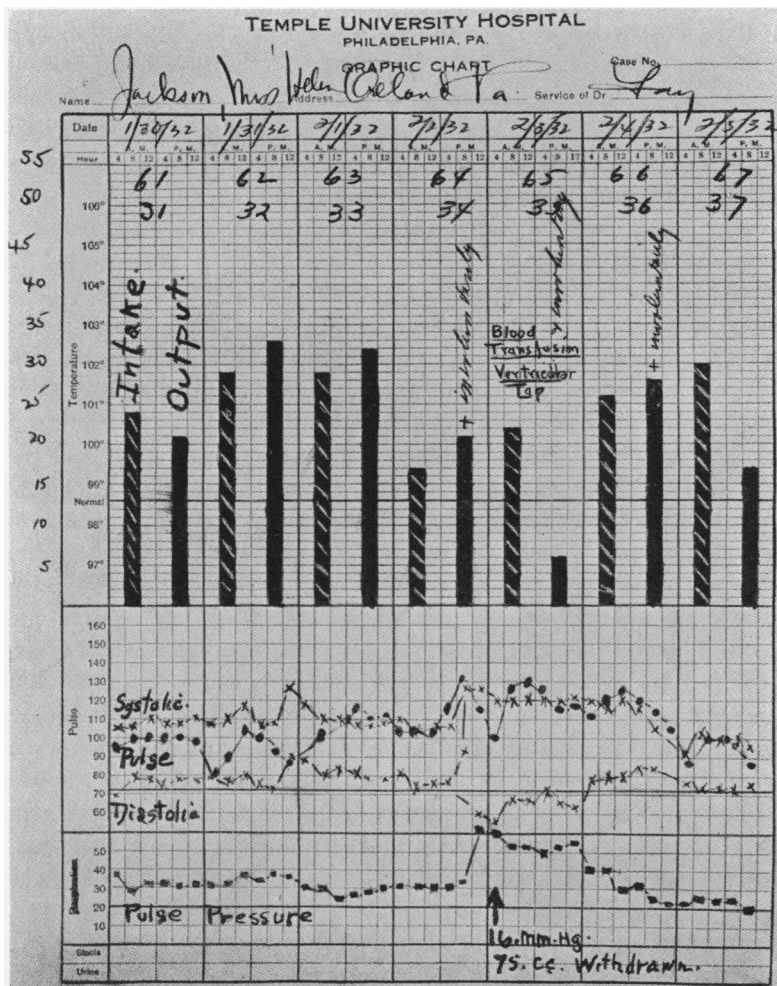


FIG. 12.—(Case I. H. J.).—Hospital record showing acute phase of intracranial pressure with abrupt rise in pulse pressure and fall in diastolic pressure. Note fall in pulse just prior to ventricular tap. Incontinence during the three days of mental obtundity. Improvement after withdrawal of 2½ ounces (75 cc.) under 16 mm. Hg. pressure. Note fluid limitation to 30 ounces (900 cc.) with intake and output record, systolic, diastolic and pulse pressure readings. (Courtesy A. J. Psych., vol. 12, No. 5.)

The cold, cyanotic, clammy extremities clearly tell their story of insufficient oxygen, oedema, capillary permeability and failure of the cells to continue their physiological function. The physician should bear in mind that the periphery is not only the hands and feet, which can easily be observed, but a similar circulatory “periphery” exists in the organs, such as the brain,

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liver, kidneys, and heart. It may be expected, therefore, that if diastolic pressure is not maintained, the functions of these organs will soon be diminished and inevitable accumulative tissue-cell death occur. The summation of each added failing integrant brings the final dissolution of the patient.

For this purpose systolic and diastolic pressure readings are carefully maintained throughout the danger period of the patient. These we consider among the most important observations in the entire management of the cerebral trauma cases. By subtracting the diastolic from the systolic pressure, one obtains *pulse pressure*, and by charting the pulse pressure it is evident "which way the wind blows." A rising pulse pressure usually is obtained when diastolic pressure begins to fall and systolic pressure fails to compensate for the deficiency. Occasionally a rapid fall of both diastolic and systolic pressure may give little change in pulse pressure, but this is usually detectable and when diastolic pressure reaches sixty or below, active means for its correction should be employed (Fig. 12). Stimulation of the peripheral vasomotor bed with pituitrin, strychnine, ergot, and ephedrine may be resorted to. Fifty per cent. glucose solution increases blood volume and viscosity and this in turn assists in raising both diastolic and systolic pressure.

The old adage, "If pulse pressure crosses the 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 approaches the pulse rate, that is the time to dehydrate.*"

By dehydration is meant methods directed toward the relief of intracranial pressure and disturbed tissue fluids, such as ventricular puncture, cisternal or lumbar drainage, hypertonic solutions by mouth, vein or rectum, thus attempting to readjust the proper ratios of the vascular (a) and interstitial (c) compartments. Cerebral oedema, middle-meningeal hæmorrhage and rapidly oncoming intracranial pressure are usually responsible for vasomotor failure. Lesions in the subthalamie area frequently produce incurable vasomotor paralysis. These observations surrounding diastolic pressure and the peripheral vasomotor bed apply chiefly to the period following shock, for during the period of shock itself the vasodilatation and low diastolic pressure noted resemble more closely the toxic peripheral responses similar to those seen in histamine reactions (Dale and Richards²³) than the central types of vasomotor failure due to local brain anoxæmia.

After the period of shock has disappeared, the diastolic pressure and the pulse pressure become the determining factors in the treatment of the case.

LUMBAR PUNCTURE.—This should be performed as soon as possible after the period of shock is over. A twenty-gauge "round-point" Green type of Babcock needle is best. Intracranial pressure should be determined by the use of a mercury manometer (water manometers are frequently inaccurate due to air bubbles, clumsy and breakable).

Bloody Spinal Fluid.—If the spinal fluid is found to be bloody, complete

drainage of all fluid obtainable in the horizontal position is necessary (thirty to seventy cubic centimetres, if possible). Weed^{12, 24} and Bagley^{25, 26} have clearly shown that the red blood-cells produce intense reaction in the subarachnoid spaces and they tend to stop up the normal arachnoid pathways and filters for elimination of cerebrospinal fluid. Thus they temporarily produce 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, the cerebrospinal fluid elaborated will not find proper means of escape and intracranial pressure will probably ensue if fluid intake is not carefully controlled. Every effort should therefore be made to remove the red blood-cells by drainage. Sufficient fluid (thirty ounces, adult) should be given by mouth in the twenty-four-hour

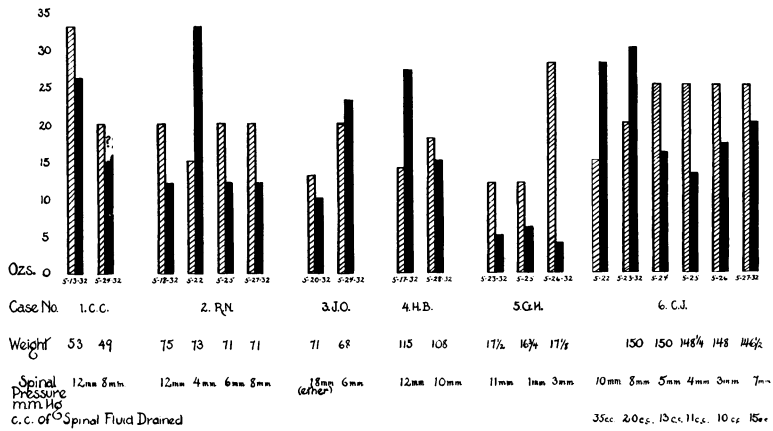


FIG. 13.—Controlled and effective dehydration (patients on service of Dr. Temple Fay). Intake and output record of cases placed on strict dehydration indicating definite loss of body weight as well as gradual decrease in spinal pressure. Note that in Case VI readjustment of spinal pressure occurs at low levels of body weight after intense period of dehydration has been adjusted.

period to permit re-accumulation of cerebrospinal fluid for subsequent drainages, but no more, unless other conditions make it imperative (Fig. 13).

Lumbar drainage must be resorted to as often as a rise in pulse pressure and respiratory changes may indicate (fourth or sixth hour if necessary).

The condition may be considered similar to a ten-day loss of bladder function, during which catheterization is required to prevent overdilatation of the bladder and disturbance in function of the kidneys. Reestablishment of the cerebrospinal fluid circulating function is usually possible by the eighth to tenth day following trauma and the need for lumbar punctures and further drainage becomes unnecessary (Fig. 14).

When daily spinal drainage is discontinued, fluids must be further curtailed for a few days. Sedimentation of ten cubic centimetres of the specimen of fluid taken at each lumbar drainage will give a daily record of the rapidity of hemolysis. Daily red cell count of the spinal fluid may also be resorted to.

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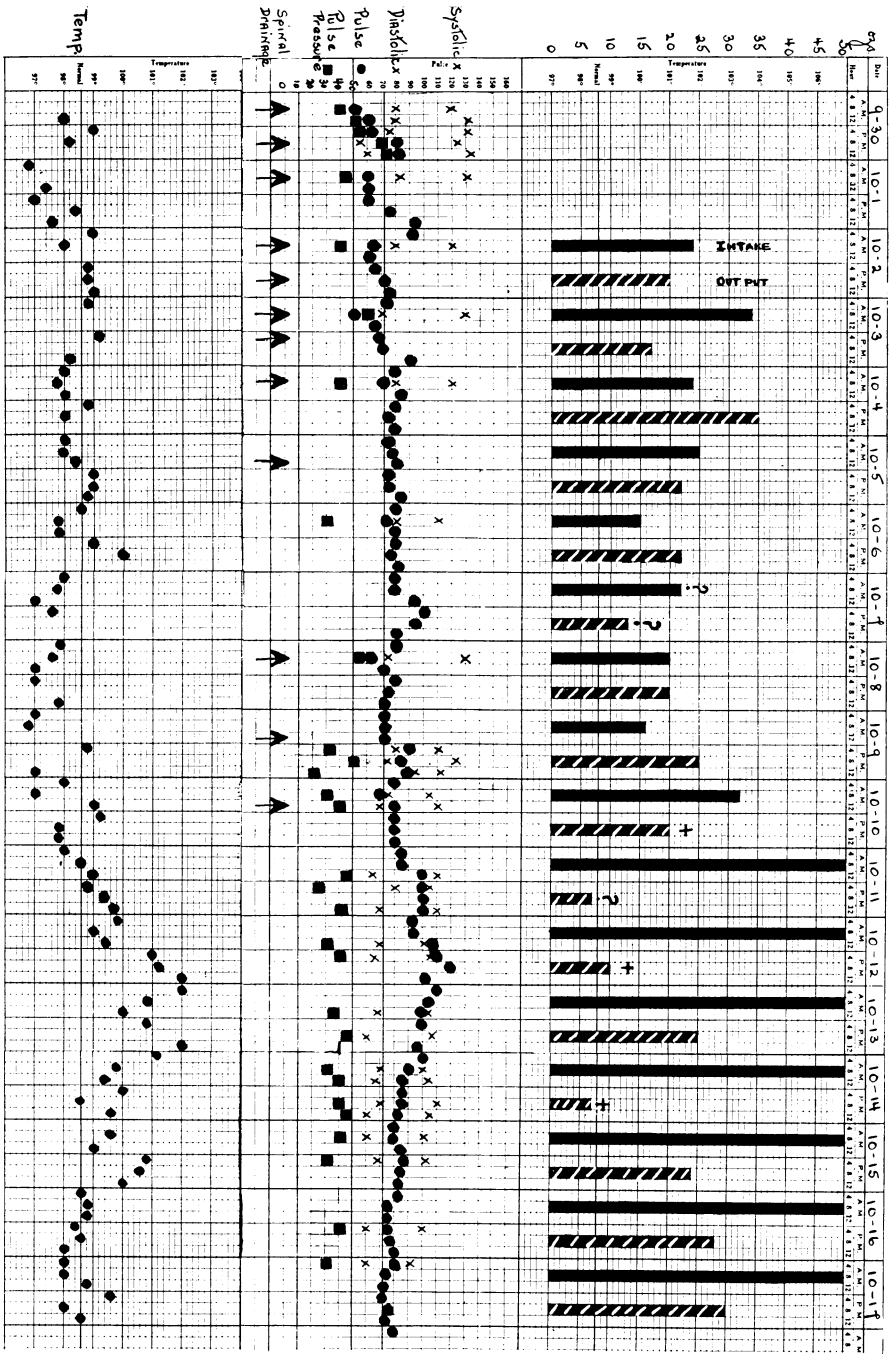


FIG. 14.—Clinical chart indicating the important considerations during the first two weeks following a severe cerebral trauma. Note that no fluids were permitted the patient during the first two days. Black columns represent total intake of fluid. Gray columns total output. Black squares, pulse pressure; gray dots, pulse rate; arrows indicate spinal drainage. On the fourth day, 34 ounces of fluid intake produced crossing of pulse rate and fall in pulse rate, with stupor. Spinal drainage re-introduced. After the tenth day water surreptitiously obtained was followed promptly by rise in pulse pressure and fall in pulse rate, with stupor. Note water storage during this period, indicated by low output.

Extensive fracture of the occipital region, respiratory "failure," and intensely bloody spinal fluid (1,300,000 red cells). Note rising pulse pressure and pulse almost crossing for several hours. (hose and lumbar drainage restored proper relations between pulse pressure and pulse rate. Active dehydration and drainage restored proper relations and patient out of danger. On seventh day patient obtained large quantity of water surreptitiously and stupor returned in four hours, condition became serious, pulse pressure and pulse again crossed, heroic measures, drainage and dehydration again relieved the crisis and patient recovered. Note after the tenth day fluids may be given up to 50 ounces without effect whereas during the first seven days fluids above 20 ounces were not safely tolerated. (Cortney S., G. & O., vol. 54, p. 362, 1932.)

Clear Spinal Fluid.—If clear fluid is obtained at the first 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 should be resorted to if necessary. Control of fluid intake with judicious use of glucose and magnesium sulphate have been found entirely adequate in such cases. 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 Frazier and many others and in the few instances where surgery is necessary, “exploration” (for clot or foreign body) has taken their place.

As the volume of cerebrospinal fluid is largely dependent upon the amount of fluid dialyzed from the blood-stream, which in turn is dependent upon stored fluid in other areas, or quantities of fluid given the patient by mouth, this latter source of fluid must be strictly limited so as to prevent overproduction. Repeated spinal punctures may be resorted to if fluids have been given in excess. It is useless to remove spinal fluid for the purpose of reduction of intracranial pressure only to immediately refill the cerebrospinal fluid spaces by permitting free intake of fluid on the part of the patient. A prompt return of stupor and serious symptoms even on the seventh day has been noted (Fig. 14) following the free unrestricted ingestion of liquids.

The author has established the fact that if liquids are restricted to the minimum (sixteen to twenty ounces per day, adult) little or no excess of spinal fluid occurs (Fig. 13). Ingested liquids above this level yield varying daily amounts of spinal fluid.

Where patients showing clear spinal fluid have been placed upon this allotment there has been no need of further lumbar puncture and symptoms of stupor, headache, vomiting and pressure are absent as long as the proper balance of liquid intake is maintained.

The prompt recovery of the patient from dullness and stupor with the maintenance of an asymptomatic state by proper intracranial pressure and volume regulation is in the author's opinion responsible for the remarkable absence of post-traumatic symptoms in this “dehydration” series and the early economic readjustment of the patient to his former mental levels and physical activity.

On the eighth to tenth day following the injury these patients are gradually allowed more fluid until an intake of thirty-two ounces is reached. They are discharged from the hospital when they are able to measure and regulate their fluids and are taught the basis of the “dry” diet. They are required to follow this fluid and dietary restriction for three months, after which either habits of moderation are formed or they avoid voluntarily former excesses because of the post-traumatic symptoms of dullness, headache, loss of attention and memory, ambition and concentration which arise from hydration states. Once the patients have experienced relief of these symptoms following cerebral

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trauma they will not tolerate their return. In cases where strict limitation has not been practiced their discharge from the hospital before complete recovery occurs only dooms them to a chronic state of disability, not sick enough to be hospitalized and not well enough to work. The lack of instruction and post-traumatic supervision permits them to carry on indiscriminate consumption of food and fluid at home without the possibility of experiencing prolonged freedom from their symptoms so that these indiscretions would be voluntarily avoided.

The need for early and if necessary repeated lumbar puncture becomes of as much importance for the correction of immediate cerebral symptoms as it does the control of the chronic post-traumatic phase. The necessity for this procedure may depend entirely on the physician's management of the fluid intake or the patient's continued lack of coöperation.

Since careful regulation of liquid intake has been found to be of such importance, those cases presenting bloody spinal fluid and consequently requiring thirty ounces of intake to insure daily spinal drainage must be shifted from this level of fluid on the sixth to tenth day when xanthochromia indicates lumbar puncture is no longer necessary. At the time of the last drainage liquid intake is cut to twenty ounces for at least two days and then gradually raised to thirty-two ounces, after which the patient may be discharged with the same restrictions and even more careful supervision than the former clear spinal fluid group.

Where subarachnoid bleeding persists and spinal fluid shows no signs of decrease in the red cell count or sedimentation tests after the third day, two to forty cubic centimetres of air may be introduced into the spinal canal in small quantities (following the removal of at least the same quantity or a greater quantity of spinal fluid—procedure controlled by intermittent spinal pressure readings by manometer as in encephalography).

This manoeuvre usually promptly controls the free subarachnoid bleeding and in the author's experience has proved highly beneficial. In one remarkable instance (Medico-Chi Hospital, 1924) the red cell count in the spinal fluid on the fifth day equalled the blood count of the patient (1,500,000), so persistent was the bleeding. Prompt recovery followed injection of air and the patient seen nine years later was normally engaged in her former duties as charwoman in a rooming house.

The arguments against lumbar puncture and spinal drainage (see Mock's⁷ Survey) in the cerebral traumatic are primarily based on the idea that an increase in the cerebral hæmorrhage may be promoted. All the evidence is to the contrary with the exception of one group. The neurosurgeon has long recognized that when bleeding occurs in the subarachnoid spaces (ventricles, cisternæ, spinal canal) during operative intervention, he must remove the free fluid from about the bleeding point in order to promote clotting. A muscle graft or dry piece of cotton serves to hasten coagulation. As spinal fluid continually dilutes the blood, either the zone about the bleeder must be filled with blood sufficient to permit clotting, or spinal fluid withdrawn from

the area so that tissue surfaces may approximate one to the other or whole blood accumulate without interference.

Rupture of a large vessel (aneurism) either in the subarachnoid space or in the substance of the brain is almost invariably fatal. The spinal fluid, where bleeding of this type reaches the subarachnoid space, resembles almost pure blood and it cannot be denied that spinal drainage under these conditions may hasten the patient's demise. As these patients invariably die with or without lumbar puncture (unless a surgeon is fortunate enough to reach and ligate the vessel in time), those critics of lumbar puncture would use this argument (which applies to a relatively few cases) to deny all others the benefits of reduced spinal pressure, prompt clotting of the usual subarachnoid bleeders, return of necessary blood circulation to uninvolved areas of the brain and the preservation of the vital and gnostic centres required for adequate recovery. The final mortality figures of 13.8 per cent. in this series where lumbar puncture has been practiced and spinal drainage maintained as a routine in such cases, clearly refutes the arguments against its use. Until the "do nothings" of the profession are able to demonstrate results that even approximate these figures the burden of proof rests upon them to justify their neglect of so important a procedure for the good of their patients. Irrespective of the mortality rate the results noted in the post-traumatic group alone in this series with the astonishing preservation of intellectual faculties, clearly establishes the principles of the procedure as not only justifiable but absolutely necessary.

There are those that point out the danger of "foramenal hernia" with sudden respiratory failure, and ascribe this to the effects of lumbar puncture with drainage. In a certain sense this argument is justifiable, but entirely dependent upon the mechanics of the cerebral structures. If the ventricles are not freely communicating with the basal cistern (tumor, occlusion, mass brain oedema), then withdrawal of fluid from about the base favors movement of the brain structures down into the incisura of the tentorium or the foramen magnum. As occlusion by clot in the third or fourth ventricle or aqueduct of Sylvius is *extremely* rare, such a mechanism does not present itself immediately in cerebral trauma.

If, however, the clinician has pursued the course of "do nothing" or adopted purely palliative means during the first twenty-four to forty-eight hours following the trauma allowing a progressive brain oedema to occur with swelling of the cerebral mass and obliteration of the fluid spaces, then foramenal hernia may indeed be a possible sequel to lumbar puncture and spinal drainage. The results should not be blamed upon the procedure, but upon the critical judgment of the physician in charge.

If early spinal drainage and a return of cerebral circulation have been instituted from the first, with appropriate care as to blood-pressure, blood volume and fluid balance of the body, such a cerebral state rarely occurs. If after all measures have failed, the lumbar puncture alone cannot be held entirely responsible.

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To undertake spinal drainage late and only when forced to do so is to court disaster. If the principles underlying the procedure are admitted, then there should be no compromise in their proper application. The frequency with which such œdema complications arise in the untreated cases must be admitted, and for those who insist upon pursuing the "do-nothing" policy, the "in God we Trust" and the "it's all in the deck" attitude, it would be well indeed to avoid lumbar drainages as their mortality is already too high, and the injudicious use of inadequate spinal puncture can only further add to results which Mock, Morrow and Shannon⁷ have shown to exist in the series where the principles of dehydration are not followed out.

In stuporous patients, delayed use of spinal puncture or drainage is always dangerous and other measures (ventricular tap, wide decompression, intensive "dehydration") may have to be resorted to, with the greatest amount of caution and the highest degree of risk.

BLOOD VOLUME.—Blood volume actually holds the key to the entire problem of treatment and survival in cerebral trauma cases, as it does in most others.

As spinal fluid volume arises primarily from blood volume, these two important components of the cranial cavity are complementary and changes in either affect the other reciprocally. Thus, if we wish to reduce cerebrospinal fluid and pressure we may employ the method of Weed by introducing hydragogues into the intestinal tract that draw fluid rapidly away from the circulating blood volume. Such a rapid loss of fluid into the intestinal tract and the vigorous diarrhœa which follows requires prompt replacement of fluid from some source back into blood volume, or serious circulatory deficiency may result. Subtraction of free fluid from the cerebrospinal fluid reservoir as well as from other available tissue reservoirs promptly occurs in order to stabilize blood volume, and the desired effect on intracranial pressure may thus be slowly obtained.

Bleeding, purgation, and sweating have, in common, the property of diminishing blood volume, which in turn may be beneficial to the cerebral centres, in that free fluid volume (c) is drawn upon to replenish the loss from the vascular volume. As spinal fluid volume falls (low tide of fluid), there is space permitted for an increase in the blood volume to the brain and with this possibility (high tide of blood) better oxygenation and nutrition to the cerebral cells with consequent improvement in function.

Probably the accepted beneficial results noted from these age-old procedures have been due primarily to the better adjustment of this cerebral mechanism rather than the elimination of a mythical "toxine," unless the condition of "water intoxication" (Rowntree) be recognized as the real "toxic" agent.

Blood volume enters into the problem from many angles. The period of shock ("surgical shock") which follows severe head trauma gives rise to a series of symptoms which are all primarily due to sudden alteration of blood volume.

In "shock," fluid rapidly leaves the vessels to pass out into the tissue spaces (subcutaneous tissues, cranial and visceral cavities); much fluid is lost onto the skin surfaces (clammy extremities and surfaces, cold because of rapid evaporation and consequently subnormal temperature due to loss of body heat). The pulse becomes rapid as the heart must increase its rate (if its available supply of blood becomes less) so as to fill the arterial tree and maintain adequate blood-pressure. As blood volume becomes insufficient, and the capillary bed dilates (anoxæmia, local vasodilator responses due to histamine-like substances, central vasomotor failure), the pulse becomes weaker and necessarily more rapid; diastolic pressure eventually can no longer be maintained, falls, and pulse pressure rises in consequence.

The symptoms of so-called "surgical shock" (not to be confused with emotional and other types) (Macleod²¹) are those of: (1) Cold, clammy extremities. (2) Subnormal temperature. (3) Rapid, thready pulse. (4) Fall in blood-pressure (especially diastolic below sixty millimetres mercury). (5) Rising respiratory rate (secondary changes in gas dissociation, anoxæmia).

The successful treatment of this condition concerns blood volume alone.

Attempts should be made to: (a) Prevent further loss of fluids from the skin (atropine, warm, dry clothing, heat to restore body loss and counteract effects of evaporation). (b) Restore lost blood volume (saline infusion, transfusion of blood, 50 per cent. glucose or a hypertonic solution by vein to reclaim available fluid from tissues and from body reservoirs). (c) Shrink the size of the vascular bed to fit the blood volume that remains. (Vasoconstrictor drugs.) This allows a rise in blood-pressure, slows the heart rate by providing more blood return to the venæ cavæ and produces a resistance to work against.

The appropriate yielding of these symptoms to treatment depends upon many factors.

The clinician is frequently misled into a false sense of security when a uniform program of treatment is adopted. It is true that the routine treatment may fit the majority of cases, but a patient may be lost who might otherwise be saved, or another receive too rigorous treatment, if each case is not weighed on its own merits. The success of treatment depends upon how much fluid was lost; whether the storage reservoirs are full (hydrated fat types) or scarcely available (thin, emaciated types, old people, *etc.*); how long the state of shock has existed, and how much additional influence the various organs of the body have to play because of complicating pathological states due to chronic disease.

The saline solution introduced into the vein, to refill blood volume, may not remain in the vessels more than thirty minutes to two hours. The original state of shock and depletion may return so promptly that no relief is clinically apparent (Fig. 9).

Much depends upon whether the portals of loss have been closed, or the fluid introduced is made more retainable to the vessels by the addition of gum acacia or 50 per cent. glucose solution.

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From the standpoint of head trauma, it is obvious that to treat the period of shock by introducing large quantities of physiological saline solution alone, is but to favor subsequently a rapid cerebral oedema. This additional fluid will serve to increase the cerebral anæmia by overfilling the cerebrospinal fluid reservoir. In the presence of a low blood-pressure (consequent anoxæmia), an excessive amount of fluid finds its way without opposition into the cerebral fluid spaces, besides that which locally arises from the cerebral injury *per se*. When general circulation and blood-pressure have been reestablished under the method of large intravenous injections of saline, the patient usually fails to regain consciousness after the period of shock, because the available space necessary for the return of blood to the cerebral structures has been temporarily occupied by this free fluid transudate. The resultant struggle for this important space is a clash between (a) and (c) volumes at the expense of (b). Increased intracranial pressure ensues and if the oedema is not rectified, the patient continues unconscious over a long period of time, or the "vital" centres fail, and death supervenes. Too frequently in the past, the patient has been saved from "shock" by large infusions of saline solution only to succumb later to intracranial pressure or oedema induced by this method.

For this reason, cerebral trauma cases require special adaptation of the treatment for shock. If large quantities of saline solution (500–1,500 cubic centimetres) are *required* to combat the phase of shock, then adequate spinal drainage (twenty to sixty cubic centimetres), must follow promptly with the return of blood-pressure to satisfactory levels. Such drainage must be repeated on the second or fourth hour, when pulse pressure rises to fifty or above, unless symptomatic improvement from the cerebral centres has been obtained by the initial measures.

In the majority of cases a small amount of saline solution (100–300 cubic centimetres) *plus* fifty cubic centimetres of fifty per cent. glucose solution (adult dose) serves to restore blood volume temporarily and this dose may be repeated promptly in part or in whole later on, if found to be insufficient. "Better safe than sorry" applies particularly to this period of treatment.

As the objective in the treatment of shock is to restore blood volume to its adequate levels (improve blood-pressure and general circulation) the glucose tends to make the vascular contents hypertonic and draws fluid from the tissue reservoirs and spaces back into blood volume. This not only assists in the desired clinical direction of improvement, but it *prevents temporarily further fluid loss into the tissues and reservoirs* and thus checks cerebral oedema. Of most importance, the cerebrospinal fluid reservoir is prevented from overfilling and frequently may actually be depleted to some degree. As blood-pressure rises by this reversal in the flow of fluid back into the vessels again, the blood circulation is permitted to return to the cerebral structures again because (c) volume is diminishing as (a) volume rises.

The proper manipulation therefore of blood volume during the period of shock in the cerebral trauma group must take into consideration the subsequent problems that will arise concerning intracranial pressure and oedema.

As a general rule, where patients are thin, old, or emaciated, larger quantities of saline must be given, as it is evident that glucose will find little free tissue fluid to draw upon. On the other hand, the fat, hydrated, plump, or alcoholic types may be given glucose alone, and this procedure repeated to become effectual, requiring the patient to utilize his own tissue fluids to replenish blood volume.

Spinal drainage in this hydrated group is almost always necessary in that the movement of fluid from one tissue reservoir to another will tend to cancel the desired result in the cerebrospinal fluid reservoir. After the initial period of shock is over (return of temperature to normal, restoration of blood-pressure) in this hydrated group, it is often necessary to deliberately deplete blood volume again (physiological shock) by giving hypertonic saline (magnesium sulphate) by mouth, (one and one-half ounces of crystals in six ounces of water) or double this dose by rectum. This draws fluid rapidly away from blood volume again and consequently precipitates a mild state of "shock" (rise in pulse rate). If now 50 per cent. glucose is introduced into a vein, blood volume is required to withdraw further fluid from the body reservoirs, and a control of the hydrated type can thus be obtained.

It is of course obvious in this hydrated group that no additional liquids should be permitted the patient by mouth, vein or rectum, if this method is resorted to. A judicious use of the measures at the disposal of the clinician for regulating blood volume, and secondarily controlling cerebrospinal fluid volume, will determine the results which may be expected. To give magnesium sulphate during the period of shock would be to court disaster, as a further loss of blood volume at a critical time would ensue. To give 50 per cent. glucose solution, intravenously, at a time when blood volume had returned to its full state, is only to precipitate an increase of intracranial pressure, because fluid may be forced back into the tissues.

As there exists at the present time a widespread tendency to use hypertonic glucose solution indiscriminately, with the belief that it contains some magic property in these cases, a clear understanding of its true value should be obtained.

We must return to blood volume and its properties for this consideration. It is accepted that blood volume is one of the most "fixed" relationships of the body. Welcher found it to be 7.2 per cent. to 7.7 per cent. of body weight. Keith, Rowntree and Geraghty place blood volume as representing from 5 per cent. to 8.8 per cent of body weight. The accepted constancy of the red cell count around five millions, as normal for all ages, indicates the uniformity of dilution in the blood mass and consequently a suggestive index of its volume. Macleod states, "The newer methods have shown that the volume of the circulating fluid (blood) is maintained *fairly constant in spite of influences tending to alter it*. The body accomplishes this by drawing upon the reserve fluid in the tissues and by varying the rate of water excretion."

There are five general conditions under which blood volume may be pathologically altered: (1) Alteration of cellular content (polycythæmia,

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chlorosis, and the anæmias). (2) Emaciating and “dehydrating” states (asiatic cholera, *etc.*)* (3) Direct loss (severe hæmorrhage).* (4) Pregnancy (increased) obesity (decreased). (5) Shock (Dale-Richards type, “Histamine Shock,” “Surgical Shock” with rapid loss of fluid into tissues).*

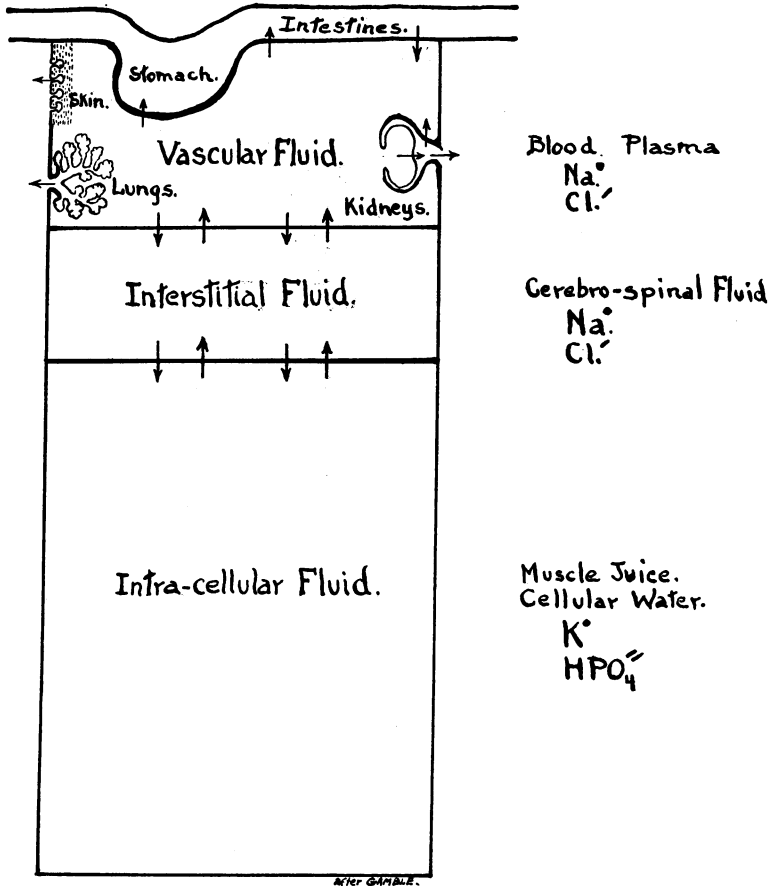


FIG. 15.—Diagrammatic representation of the body fluid compartments (modified after Gamble) showing the directional movement of fluid after entering the gastro-intestinal tract. As blood volume (vascular fluid compartment) tends to remain fixed by means of excretion through its four portals of elimination, or by storage in the interstitial compartment, the proper clinical manipulation of these variables forms the basis of successful treatment. Cerebrospinal fluid represents the largest single interstitial reservoir. Fluid interchange between the vascular and interstitial compartments is facilitated by the fact that both are dependent upon the fixed base Na. for retention, whereas intracellular fluid is maintained by the fixed base K. and exchanges are not reciprocal. Hydration states concern the interstitial fluid compartment and yield to measures of dehydration.

Intracellular œdema on the other hand represents fluid that has become fixed by altered cell metabolism and cannot be reclaimed by practical clinical measures now at our disposal.

Restriction of fluid intake and forced elimination demands that blood volume draw upon the interstitial reservoirs in order to maintain its integrity. (Courtesy J. of Nerv. & Ment. Dis., vol. 71, No. 5, May, 1930.)

In view of the fact that (excepting for the above-noted conditions) blood volume tends to remain unaltered under almost all other circumstances, it seems obvious, therefore, that if blood volume is “normal” the addition of 50

* (2), (3) and (5) intimately concern the problem of trauma.

per cent. glucose solution, in order to draw tissue fluid into blood volume, cannot accomplish this purpose until an equal quantity of fluid has been simultaneously excreted from the blood volume. This must be accomplished through the four normal portals: kidney, skin, bowels or breath. Without medical assistance this process is slow. Glucose in the presence of full blood volume passes out of the blood-stream into the tissues where it is stored and burned. The kidneys rapidly eliminate the remainder. The fluid now tends to pass back into the tissues as the concentration of glucose in the tissues may eventually be higher there than in the blood (due to renal depletion). If the patient is permitted fluids by mouth, skin, or rectum, then repeated doses of glucose in the presence of "Normal" blood volume will promote cellular and tissue oedema and augment an intracranial pressure state that was intended to be diminished. Gamble, Ross and Tisdall,^{27, 28} have pointed out that cellular water storage and increase in water retention of the tissues may be due to carbohydrate and glucose metabolism (Fig. 15).

If hypertonic glucose is to be used as a cerebral "dehydrating" agent, space must be provided in blood volume for the tissue fluid that is to be withdrawn. Purgation, sweating, bleeding, shock, increased kidney output, all offer means of depleting blood volume in order that glucose may be truly effectual. Each of these measures has its special indication and contra-indication. Purgation and increased renal elimination are more easily controllable, and their use more common.

In view of the tendency for blood volume to rapidly return to a normal level if possible, 50 per cent. glucose, if given in the period of shock, is ideal, as blood volume at that time is depleted and needs tissue fluid. After the period of shock has passed, hypertonic glucose solution should not be given *unless preceded by some measure to actively reduce blood volume.*

The first dose of glucose given as soon as the patient reaches the accident ward has been found highly beneficial. The second dose should be given only if indicated, to supplement the first. The third dose requires proper preparation of blood volume for its effectiveness and has been found to be rarely required in the author's experience.

Glucose is one of the most important "dehydrating" adjuncts if properly used where its osmotic pressure effects can be most beneficial. It may become a boomerang if used with careless disregard by those who adopt the slogan, "when in doubt, shoot a little glucose."

SUMMARY OF TREATMENT AND ROUTINE

I. UPON ADMISSION OF PATIENT TO ACCIDENT WARD: (a) Immediate temperature, pulse, respiration and blood-pressure determinations. (b) Temporary dressings to wound—control of hæmorrhage. (*No detailed wound repair.*) (c) Intravenous fifty cubic centimetres (adult dose) of 50 per cent. glucose solution. (If *necessary* give in addition 100–300 cubic centimetres saline solution to combat severe blood-volume loss of fluid.) (d) Shock cabinet, warm, dry blankets, heat to body surfaces, elevate foot of bed 45°. (e)

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Appropriate medication—atropine, pituitrin, ephedrin, ergot, strychnine. (f) Sedatives. Chloral hydrate (grs. xv.), sodium bromide (grs. xxx) by mouth. Double dose if given by rectum. Sodium luminal, grs. i to iii by hypo. Repeat above as indicated. q. 4th hour. (*Avoid morphine and its derivatives, if possible.*) (g) Physical and neurological examination as brief as possible.

II. PERIOD OF SHOCK.—The signs of *subnormal temperature*, cold clammy extremities, low diastolic pressure (usually below sixty millimetres mercury) rapid, thready pulse (usually above 120) increased respiratory rate and abnormal pulse pressure (40 normal) indicate the presence of shock.

The treatment of shock takes precedent over every other consideration (few exceptions) and must be brought promptly under control.

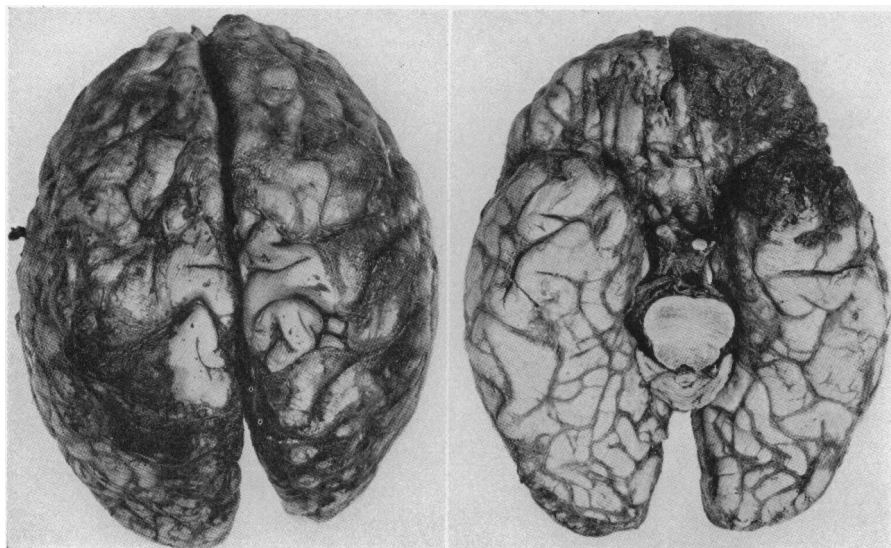


FIG. 16.

FIG. 17.

FIGS. 16 AND 17.—Unilateral contrecoup hæmorrhage from trauma in the parieto-occipital area. Note involvement of the under surface of the left frontal lobe and tip of the temporal lobe with bilateral diffuse subarachnoid hæmorrhage; swollen convolutions and generalized cerebral œdema on the left. (Preparation by Dr. N. W. Winkelman.)

Suture of the wounds, spinal puncture, detailed neurological examination, X-ray examination, or unnecessary movement of the patient should *never* be attempted until adequate recovery from shock has been established. Occasionally, the patient's distress will require immediate lumbar puncture. This should follow the glucose administration and other medication to fortify the additional loss of fluid from lumbar drainage.

The period of shock may be considered over when the *temperature* of the patient returns to normal or above. Temperature, pulse, respiratory and blood-pressure recorded every fifteen minutes. Sufficient sedative to control restlessness and prepare for subsequent suturing, spinal puncture or examinations. No magnesium sulphate should be given at this time.

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III. PERIOD FOLLOWING SHOCK.—Temperature, pulse, respiratory and blood-pressure records continued; readings every half-hour.

Lumbar puncture with pressure reading, and drainage if fluid is bloody or pressure above ten millimetres mercury.

Suture of wound with débridement and necessary reconstruction in a properly equipped operating room, if possible.

X-ray of head and other parts only if absolutely necessary to locate foreign bodies, bone fragments or a marked depression. X-ray evidence of a

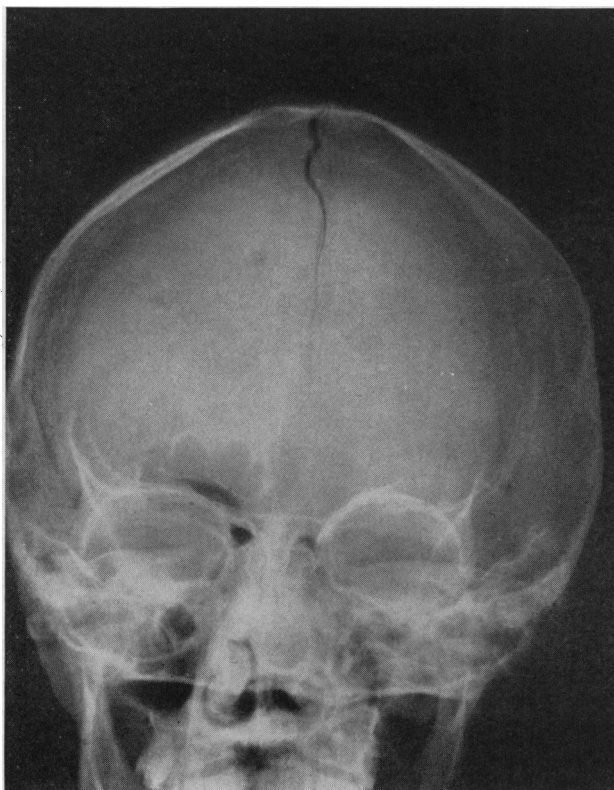


FIG. 18.—Linear fracture of the vault. Compression or thrombosis of the superior sinus may produce venous stasis and profound cerebral œdema in spite of all measures of dehydration and spinal drainage employed. Two verified cases in the author's series were characterized by prolonged and continuous stupor (ten and eighteen days, respectively) up until the time of death. The patients suggested a state of profound narcosis, requiring tube feeding until the end. Slow and delayed ingravescence venous hæmorrhage into the subdural space may occur with fractures which cross the large dural venous sinuses.

skull fracture will offer no aid to treatment unless compounded or greatly depressed. Satisfactory films are not obtained until the patient is coöperative. X-rays taken on the second, third or fifth day after the injury serve to confirm the physician's diagnosis; are excellent medicolegal records and spare the patient from additional shock, as well as the roentgenologist from a difficult duty usually requiring repetition. Patients do not die from "fracture of the

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skull." They do, however, die from shock, œdema and intracranial hæmorrhage. The point and extent of fracture (unless compounded or depressed),

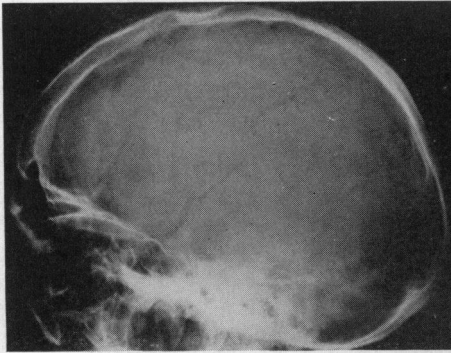


FIG. 19.—Fracture in the temporal region crossing the middle meningeal artery. Subdural hæmorrhage frequently a complication of this area. Stereoscopic X-ray films are required in order to be sure that normal vessel markings in the skull are not mistaken for lines of fracture.



FIG. 20.—Extensive linear fracture of the skull without complications.

do not usually indicate the site of greatest brain damage. Too frequently laceration and hæmorrhage are extensive and contrecoup (Figs. 16 and 17). Frequent neurological examination and careful observation of progressive signs will determine the location and the necessity for surgical intervention rather than the X-ray evidence obtained.

Good X-ray films may be of extreme importance especially if a shift of the pineal gland is shown. The patient must be quiet and coöperative to obtain proper films. Too frequently much valuable time is lost by sending the patient directly to the X-ray laboratory when delay in necessary treatment may mean a life (Figs. 18, 19, 20, 21, 22, and 23).

Detailed neurological examination with especial reference to the pupils (dilated pupil suggests the side of the brain with greatest involvement). *Magnesium sulphate* by mouth or bowel, depending upon general hydration state of the patient and need for dehydration. *Repetition of glucose* intravenously, depending upon requirements of blood volume, pulse and blood-pressure. *Sufficient sedatives* to insure quiet and rest. *Ice-bag to the*

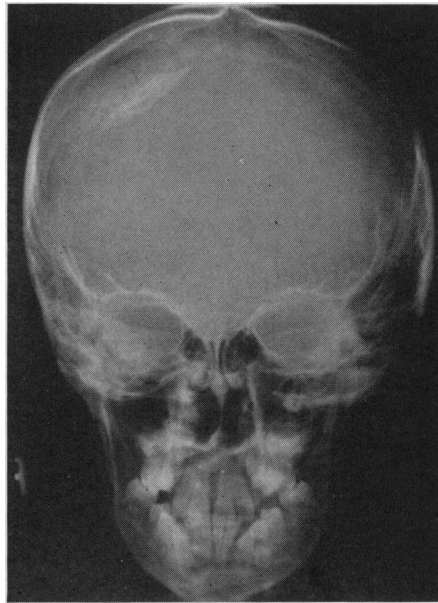
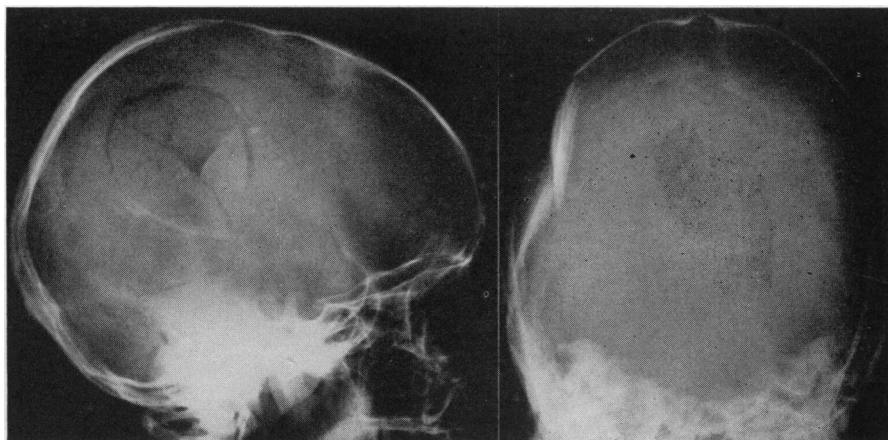


FIG. 21.—Focal depressed fracture of the skull, with satisfactory response to measures of dehydration. Elevation of the fragments on the tenth day following injury, after signs of cerebral œdema and pressure had subsided.

head. *Hexylresorcinol*, grs. x. q. 4th hour, if cerebrospinal leak occurs from nose or ear. Dichloramin T. dressing to ear (t.i.d.) if draining cerebrospinal fluid. Argyrol, 10 per cent. in each eye (t.i.d.) if spinal fluid is draining from the nose. Sponge q. half-hour for temperatures over 103°. Patient placed on twenty ounces of liquid and "dry" or solid diet if spinal fluid clear; thirty ounces of liquid are allowed if spinal fluid is bloody, and during period of daily spinal drainage.

PERIOD OF RECOVERY.—Temperature, pulse, respiratory and blood-pressure readings may be recorded fourth hour after the critical stage is passed. Strict control of liquid intake should be maintained until the eighth to the tenth day (longer if patient is not progressing satisfactorily). Cerebrospinal fluid leak, over-dehydration or emaciation may require forcing of fluid. If given by vein, skin, or mouth, the amounts should be known and excess avoided.



FIGS. 22 and 23.—Anterior and lateral views of skull showing extensive depressed fracture of the skull in the right parietotemporal region. Profound stupor, subarachnoid hæmorrhage and high spinal pressure. No focal neurological signs. Patient's condition successfully managed with repeated spinal drainage and dehydration by Dr. H. M. Jenkins. Conscious on the second day; symptom-free at the third week. Referred to the author for elevation of bone fragments (cosmetic reason only), at the fourth week following the injury.

Enemas or colonic irrigations permit water absorption from the bowel. An ounce of magnesium sulphate to a pint of water will prevent absorption and not produce dehydration, when enema alone is desired. One ounce of glycerine added to the mixture assists in obtaining a prompt return. Daily spinal drainage to be continued until fluid is xanthochromic. The patient may be permitted out of bed from the fifth to ninth day, depending upon the severity of the symptoms. Discharge from the hospital usually occurs on the tenth to twelfth day in patients treated along appropriate lines of dehydration.

It is important that the patient learn to measure his liquid intake and keep a daily record of this as well as his output. Daily weight is a guide to water-storage of the body (one pint weighs approximately one pound). With this

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knowledge the patient may prevent rapid storage of fluid and the subsequent symptoms of headache, dullness, loss of attention and memory defects.

A cheap measuring glass is furnished the patient; a diet regulation (Table V) determined upon, and blank forms for the continuation of his records at home. These must be maintained and submitted for check-up on his return to the follow-up clinic, one and three weeks after discharge.

TABLE V

Diet Form and Instructions Used in Maintaining Dehydration

Total allotment of fluid per twenty-four hours (tea, coffee, milk, water, soup, fruit juices, etc.).....

Total allotment of sugar per twenty-four hours.....

Diet should consist of red and white meats, eggs, fresh water fish, dry cereals and the vegetables listed below in any variety, as well as butter, cheese, nuts, etc.

Portions of food should be moderate.

Meals of equal importance.

Avoid overeating at any time.

Avoid eating between meals.

One uncooked vegetable (carrot, lettuce, celery) *and* one fresh fruit (apple, orange, 1/2 grapefruit, pear, few grapes, etc.) allowed daily. Where juicy fruits or vegetables high in water content are desired, exchange for appropriate amount of fluid can be made by *cutting* total liquid intake allotted for the necessary amount substituted by the fruit or vegetable.

| VEGETABLES RECOMMENDED | VEGETABLES TO BE AVOIDED OR EXCHANGED FOR LIQUID | |
|---|--|----------------|
| Potato (baked, fried, French) | Asparagus | Cauliflower |
| Peas | Tomatoes | Onions |
| Beans, navy and lima | String beans | Spinach |
| Carrots } Drained of juice and served dry | Squash | All greens |
| Beets } as possible. | Turnips | Sweet potatoes |
| Parsnips } | Cabbage | |
| Rice (dry steamed) | | |
| Corn | | |

FOR THIRST

Warm listerine gargle. For water may be substituted orange juice, grapefruit juice, White Rock, buttermilk, milk, tea, coffee, clear unseasoned soup.

AVOID SOGGY DISHES

Stews, puddings, macaroni, gravy, apple sauce.
No creamed preparations.

ALLOTMENT OF LIQUIDS

Breakfast..... ozs.
Breakfast to lunch..... ozs.
Lunch..... ozs.
Between lunch and dinner..... ozs.
Dinner..... ozs.
After dinner..... ozs.

AVOID FRUITS SUCH AS

| | | |
|--------------|---|--|
| Melons | } | These may be exchanged for a proportionate amount of allotted liquids. |
| Peaches | | |
| Plums | | |
| Strawberries | | |
| Cherries | | |

TEMPLE FAY

TABLE V (Continued)

| SUGGESTED DIET PROPORTIONS | RESTRICTIONS |
|---|---|
| Breakfast: dry cereal (shredded wheat, corn flakes, puffed rice or wheat, <i>etc.</i>) with <i>measured</i> amount of milk or cream. Buttered toast, egg, measured amount of coffee. | <i>No Salt Added to Food</i> No salty foods such as fish, chipped beef, saltines, pretzels, olives, salted nuts, <i>etc.</i> |
| Lunch: 1 potato, 1 other vegetable such as listed above. Red meat, fowl, cheese or fish (unsalted). Bread and butter, custard or junket. | <i>No Sweets</i> No ice cream, syrup, honey, jelly, cake, candy, canned fruits with syrup sauce. No sweet puddings, desserts or pie. |
| Dinner: 2 vegetables from above, meat or eggs. Measured allotment of tea or coffee, milk or unseasoned soup. Fresh apple, salad or celery. No desserts. Fluids as above. | DESSERTS (CHOICE OF 1) Unfrosted cake, moderate portion 1 cup custard 1 junket Cookies (without icing or sugar) Jello No sweet puddings, icings, pie, <i>etc.</i> |
| Enemas only as prescribed. | WEIGHT DAILY "A pint is a pound." Gain or loss in fluid can be checked by this means. |

The diet and fluid restrictions are required for three to six months following severe head trauma.

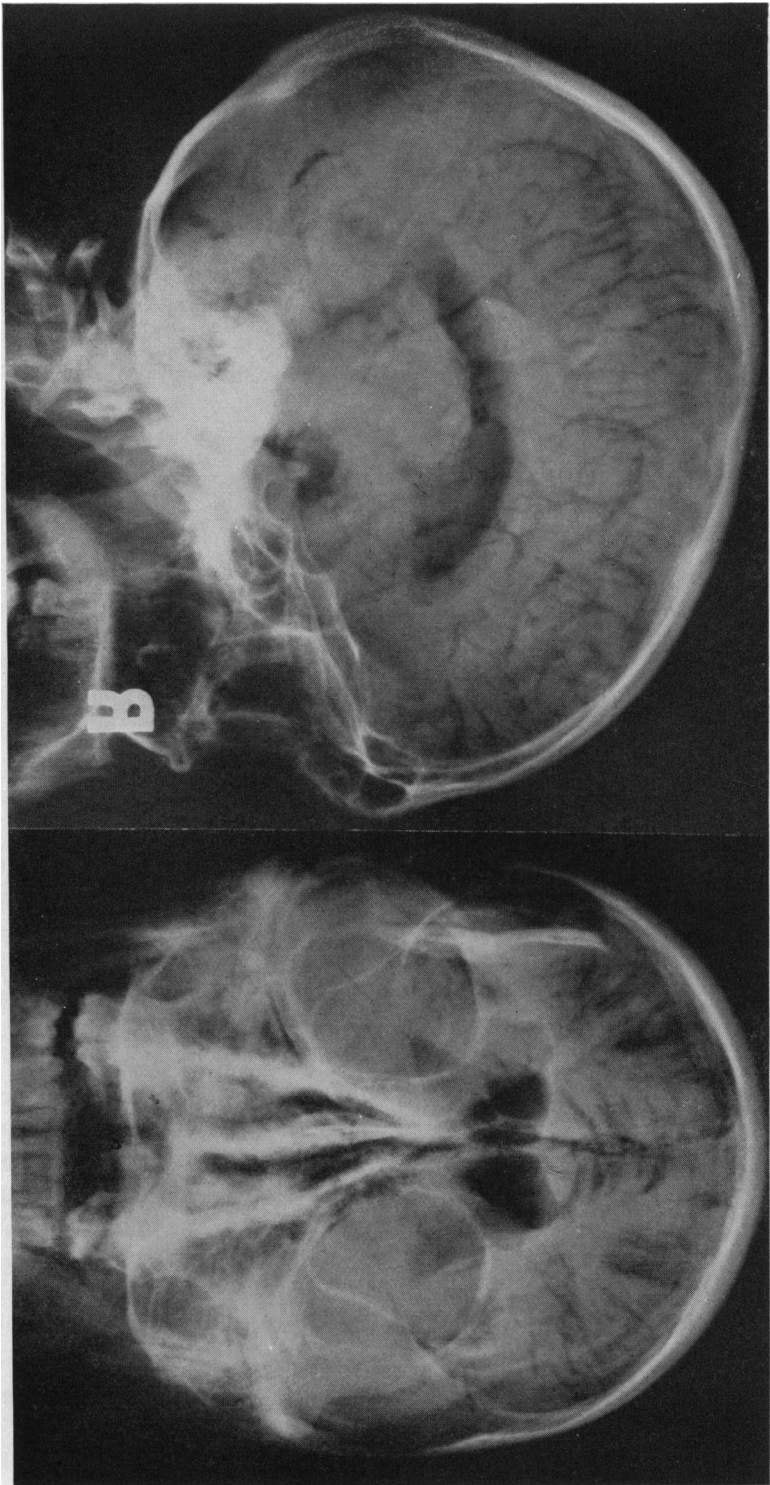
RESULTS OF TREATMENT

It has been found in our series that patients maintained on a solid, dry diet and twenty total ounces of fluid per day, promptly regain consciousness, are free from headache, and little or no spinal fluid can be obtained by lumbar puncture after the second day. This places the cerebral mechanism in a physiological state of rest, free from hydraulic compression, and permits the optimal cerebral circulation during the period of recovery; it shortens the interval of unconsciousness, and preserves the higher centres of intelligence.

After discharge from the hospital (patients with severe cerebral trauma, with or without bloody spinal fluid), the patient is placed upon a total of thirty-two ounces of liquid per day for the ensuing three months. The diet should contain solid foods, and strict avoidance of vegetables, high in water content, or the consuming of excessive volumes of food, should be emphasized.

In this series of observations, the patients have shown rapid improvement following cerebral injury, and have been free from headache, loss of initiative, memory disturbance, and mental fatigue, so common in the former group. They have returned to full activity in many cases within three months of the injury, where formerly a nine months' period of disability was to be expected. The patients have maintained a low fluid level of their own accord. "When they took more liquids they did not feel as well," they had headache, dullness, and voluntarily returned to their former restrictions of fluid and diet.

Encephalography (Fig. 24, A and B) has clearly shown that widespread cortical atrophy of the brain occurs within three weeks following cerebral trauma. Many post-traumatic cases have shown not only focal scars over the cortex of the brain, but an atrophy out of proportion to the site of injury or its extent, and this atrophy of the cerebrum and cerebellum is bilateral and general-



Figs. 24A and B.—Encephalogram of patient at the fifth week after injury. Patient absolutely symptom-free at this time. Note generalized brain atrophy (vertex, midline, subarachnoid spaces) at a distance from the fracture, secondary to subarachnoid bleeding and pressure from spinal-fluid accumulations. Spinal pressure on admission 30 mm. Hg. Fluid very bloody, full drainage. Puncture four hours later, pressure 32 mm. Hg. Full drainage; puncture eight hours subsequently, pressure 18, full drainage. Pressure after second day 10 mm. Hg. on dehydration.

Bone fragments elevated at sixth week after injury, dura found lacerated, small area of local brain destruction (fifty-cent piece). Recovery with return to work in three months. Symptom-free now, one and a half years. Continuing work as gas-station attendant.

The case illustrates depressed fractures are not immediate surgical problems if general pressure problems are capable of conservative treatment. The cerebral atrophy shown occurred within five weeks and is typical of the changes seen by encephalography in the post-traumatic, with or without depressed fractures. Intense pressure during the first few hours following injury is probably entirely responsible for these changes as local damage was comparatively slight. The end-result indicates the importance of control of spinal-fluid pressure and volume from the beginning of treatment. The brain damage is reduced to a minimum; the patient's life assured, and his economic readjustment made possible.

The author considers this case highly important, not only from the standpoint of survival and full recovery in a patient whose condition seemed hopeless from the start, but this is one of the earliest encephalograms recorded, made on a demonstrably focal injury, where all clinical and neurological findings were under constant observation prior to the encephalogram, and for the ensuing eighteen months. The mechanism of post-traumatic cerebral atrophy has become more firmly established by a group of such observations.

ized. Pathologically such brains reveal anæmic changes and degeneration similar to that described by Hassin, and termed *pressure atrophy*. An atrophy of the soft, delicate gray matter of the brain is not surprising in view of familiar observations regarding "pressure atrophy" elsewhere in the body. When a ring is worn upon the finger, when glasses rest upon the nose, or exert pressure upon the cartilage of the ears, when casts or constricting bandages are applied to the surface of the body, characteristic pressure atrophy occurs. The results of pressure from a cast applied to the extremity and the rapid consequent atrophy are readily recognized and accepted by the profession. The fact that the brain is acted upon by an "hydraulic cast" encased by an unyielding skull seems to have escaped the recognition which it should have had from the profession.

The obstetrician depends upon hydraulic pressure for dilatation of the cervix in normal labor.

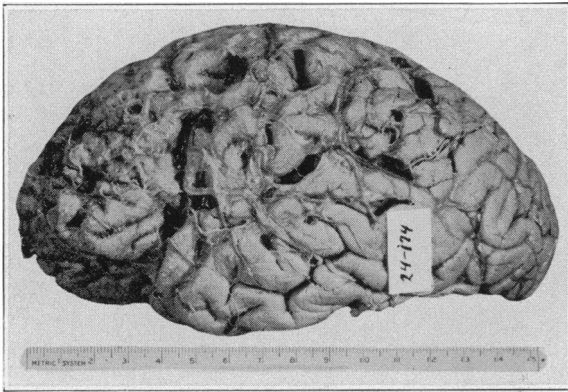


FIG. 25.—Brain of a chronic epileptic in which the arachnoid has been carefully opened to show the dilated fluid pathways and shrunken convolutions. Note how the under surface of the frontal lobe, the occipital lobe and the temporal lobe has escaped this process. The atrophy is confined to the cerebrospinal fluid circulating field and to the pathways themselves. We believe this to be due to the hydraulic "cast" of pressure exerted in this area, producing gradual pressure atrophy of the adjacent cortex. (N. W. Winkelman.) (Courtesy A. M. A.)

The hydraulic atrophy produced in distention of the bladder and renal pelvis is well recognized. The control, therefore, of a similar cerebral hydraulic mechanism is necessary to prevent widespread atrophy of the cortical surfaces surrounded by fluid (Fig. 25).

It is evident that the members of our profession interested in industrial medicine and the economic readjustment of patients suffering from head injuries, must secure the early protection and subsequent preservation of the cortical areas so necessary for intellectual activity. The cerebral hydraulic pressure mechanism should be treated in the same manner in which one would deal with an obstruction to the neck of the bladder or renal pelvis, that is, continued and prolonged fluid drainage must be the first consideration, and the accurate control of fluid production should be maintained during the period of inadequate elimination. Thus, in attacking the problem from both angles, we shall not continue the paradoxical treatment so long in vogue of draining the spinal canal or decompressing the brain, only to follow this beneficial procedure by immediately introducing large quantities of fluid into the individual by mouth, bowel, or vein, destroying the advantages gained through cerebral decompression.

To eliminate surgical decompressions in the treatment of head trauma is to step further forward toward assisting in the industrial and economic readjust-

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ment of the patient, because brain destruction and atrophy invariably occur at the site of surgical decompression, whether inflicted at the time of surgical intervention, or subsequent to the pressure exerted at this point of opening by the resultant cerebral hernia. This leaves the patient with an organic loss of brain in the region of the decompression, superadded to the loss directly due to the injury itself. Not only is this a most important medicolegal "compensation" factor, but a definite inferiority complex develops, characterized by fear and anxiety because of the opening in the skull and the danger of direct injury, in time producing a typical post-traumatic psychosis. Finally, such disfiguring decompressive openings limit the patient in his possibilities of securing work or engaging in activities that offer the slightest danger of trauma to the site of the cranial defect.

In summarizing the important industrial and economic aspects of head injuries, I wish to emphasize that early and continued dehydration as a form of decompression should be immediately instituted and continued to the point of recovery of conscious function. The hydraulic compression from chronic, increased intracranial pressure should be controlled for a period of months following cerebral injury, to prevent the increase of brain atrophy so characteristic in this group. The symptoms of post-traumatic headache, vertigo, mental torpor, mental deficiency, loss of initiative and concentration may frequently be prevented if careful supervision and control of fluid intake have been maintained following severe cerebral injury.

ECONOMIC READJUSTMENT FOLLOWING HEAD INJURIES

In dealing with the economic and industrial aspects of head injuries, it is necessary to dispel at once the conception of certain mysterious and ill-defined consequences resulting from injuries to the brain during the acute period of clinical study, and to recognize the occurrence of certain definite common sequelæ which may be attributed to organic changes not detectable by ordinary neurological examinations.

During the past few years, it has become clear that the early administration of prompt and efficient treatment in cases of severe injury to the brain not only assures the patient of a better chance of survival, but determines to a large degree the resultant mental disability. Too frequently the clinician's attention is directed only toward the preservation of life by palliative means, with the result that the patient is passed along to the neurologist or neurological surgeon as a chronic case of mental deterioration and an almost certain total economic loss.

The close correlation between the acute stage of the problem and the subsequent post-traumatic mental sequelæ has been a matter of careful investigation during the past nine years. We have recognized that not only must the patient be protected from his immediate danger of death, due to involvement of the "vital" centres by pressure, but also that the entire function of the brain, dealing with intelligence, memory, and judgment, requires active consideration during the early hours following a severe cerebral injury.

For our purposes, the brain may be divided into two areas dealing pri-

marily with separate functions: first, those nuclei concerned with maintaining life, such as the vasomotor, cardiac, and respiratory centres which we term "vital" centres; and, second, those portions of gray matter which have to do with intelligence, appreciation, and expression of the individual in relationship to his environment, situated in the cortex of the cerebrum and cerebellum, which we may term the "gnostic" regions. From the immediate clinical standpoint, the *vital* centres are most important, but from the economic and industrial standpoint the *gnostic* areas of the brain are necessary to permit the re-establishment of the individual to his social state.

The vital centres are situated at the base of the brain (Fig. 26) in the region of the third ventricle, the pons and medulla, and definitely indicate the zone of danger toward which the clinician should direct every measure of protection. The cerebral hemispheres and cerebellum are masses of functioning and reacting tissues, which are unnecessary for the actual continuation of life, but are of the highest importance in the intelligent reactions and conscious expression of the individual. Injury or destruction of the former (vital areas) is

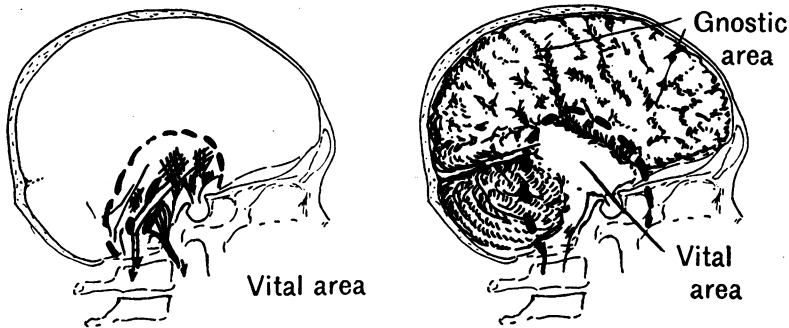


FIG. 26.—Division of the brain into (a) vital and (b) gnostic areas. The vital areas are concerned during the immediate post-traumatic phase. The gnostic areas are not essential to life, but of great importance to economic readjustment. Injuries to the former are fatal; to the latter, they leave evidence of mental and physical disability. (Courtesy S. G. & O., vol. 54, p. 362, 1932.)

followed by death within three to six hours, and all of the present clinical methods at our disposal fail to help the patient. Destruction or injury to the latter (gnostic areas), however, is characterized by a loss of mental function and transient or continued paralysis. The clinician must keep clearly in mind that the neurological examination reveals the signs of loss of function of the higher centres, whether it be inhibitory in nature, controlling reflex, or psychic manifestations, or an apparent paralysis or anæsthesia. The loss of consciousness becomes as important as an actual paralysis in the early phases of the problem. Furthermore, there is a great danger that the factors leading to local cortical compression, which are responsible for the loss of the conscious elements, may spread in the form of œdema to the vital basal centres, and the patient perish from this complication.

Unlike any other organ, the brain is contained within a space of fixed volume (the skull, after closure of the sutures), and the function of this organ must be maintained within these volume relationships. In the presence of adequate oxygen and circulation, brain tissue will survive and tend to function,

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but with the advent of œdema or gross hæmatomata, the blood supply becomes insufficient to supply adequate oxygen, and in this circumstance the "gnostic" areas suffer primarily and most severely, not only because they are at the moment least important, but because they receive five times the blood supply of the basal cells and are exposed to surface compression, and are consequently more sensitive to proportionate diminution in circulating blood volume (oxygen).

As the skull determines the limitation of intracranial volume, and as there can be no increase in volume by any of the components contained therein without an equal withdrawal of one of the factors, the problem of treatment of intracranial injuries resolves itself into attempts to maintain optimal circulation by subtracting from the cranial contents a certain volume of the least important components.

The vital centres at the base of the brain are well protected by solid masses of white matter and receive their blood supply directly from the basilar artery and the circle of Willis, and their capillary network is not exposed to the surface compression which occurs over the cortex during periods of generalized pressure. However, as the process of œdema begins in the cortical layers of the brain, a swelling in the brain volume occurs, with rapid continuation of the œdema into the basilar areas. Thus, the terminal picture is usually one of vasomotor failure associated with respiratory irregularity, and as oxygen becomes less available to the brain, due to a failing circulatory pressure and paralysis of the vasomotor mechanism, the respiratory centre fails, and leaves the intrinsic mechanism of the heart to beat on for a few moments after all other neurological activity has ceased.

If we are to accomplish not only a protection of the vital centres but an early reestablishment of cortical circulation to protect the gnostic areas, the treatment must be vigorously directed toward complete control of the cerebral volume relationships, and the patient must not be permitted to remain in an unconscious state if it is possible to prevent it. This means that the clinician must continue the method of treatment, not only to the satisfaction of the vital centres, but must aim toward sufficient return of circulation to permit cortical function as soon as possible following the injury.

The follow-up results on the post-traumatic group maintained on fluid balance after discharge from the hospital has shown 92 per cent. free from the post-traumatic symptom-complexes usually designated as "neuroses," "compensationitis," "mentally retarded" or "deteriorated" types. The "dehydration" case histories indicate the astonishing readjustments that have been possible in the severely injured group of "brain workers" as well as the laboring class.

The improvement noted in the chronic types of post-traumatic neurosis and deteriorated groups that have been afforded the application of the principle of dehydration even after years of neglect, clearly indicates this group may be improved approximately 30 per cent. (See Whitney and Hunsicker²⁹ and Fay.^{15, 30})

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SUMMARY

The principles of dehydration have been presented as they apply to patients suffering from acute cerebral trauma. It seems justifiable to attribute the definite reduction in mortality (11.4 per cent. in this group) to measures which establish and maintain a better blood supply to the brain organ as a whole, when hæmorrhage, œdema and pressure threaten its survival.

The gratifying results obtained in the post-traumatic group indicate that continuation of the principle itself offers the opportunity of protection to the brain against chronic increased intracranial pressure, with the correction of the so-called post-traumatic syndromes and "neuroses."

The ability of these patients to again engage in their former activities in from three to six months following severe head trauma presents a solution for a staggering economic problem.

In the last analysis, the clinician strives by every means at his disposal to reestablish the necessary blood supply (oxygen and nutrition) required to promote healing of injured tissue and permit retention of all function in the organ affected. As the unyielding craniovertebral boundaries introduce additional problems of physics and hydraulics into the limited environment of the cerebral organ, these obstacles must be removed by clinical and mechanical measures.

The term, "dehydration," has come to be applied to the methods directed toward a reestablishment of *normal functional relationship* between the volume components of the craniovertebral cavity, and in this respect the "dehydration treatment" is obviously indicated only where states of abnormal *hydration* exist. The wise regulation and readjustment of fluids when pathological states exist must be determined for each individual case as one would the dose of a drug, and it has often been said that "the dose of a drug is *just enough*."

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