EXTIRPATION OF THE CHOROID PLEXUS OF THE LATERAL VENTRICLES IN COMMUNICATING HYDROCEPHALUS

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No form of treatment, either medical or surgical, has yet a valid claim to the cure of a single case of hydrocephalus, except in those cases caused by tumor and relieved by tumor extirpation. But hydrocephalus is a curable disease. This is demonstrated by the not infrequent cases which have been cured spontaneously, though usually at a time when cerebral destruction has left the patient a hopeless imbecile. The reason for nature's successes is that the cause has been either circumvented or overcome. The reason for medical or surgical failures is that the cause has not been recognized. All forms of therapy have been entirely empirical. They have been directed toward the effect rather than the cause. They have lacked not only the etiology and pathology of the disease but even a knowledge of the circulation of cerebrospinal fluid before pathological changes have occurred.

Hydrocephalus should no longer be classified as an idiopathic disease. Its pathology and, in large part, its etiology are definitely established.1 With the cause recognized, a rational form of therapy is indicated. I make this statement principally upon the results of our own investigations, which have been conducted in the past five years. These studies include the paths for the circulation of cerebrospinal fluid, the place and manner of formation and absorption of cerebrospinal fluid, the experimental production of hydrocephalus, the pathogenesis of many cases of hydrocephalus studied clinically by the phenolsulphonephthalein test, and the pathology of the various so-called types of hydrocephalus by post-mortem examination. The present communication is directed to the treatment based upon these observations.

A knowledge of the type of hydrocephalus is an absolute prerequisite to its treatment. The timeworn symptomatic classifications are incoördinate and confusing. The classification on the following page is presented, based upon the etiology and pathology of the disease:

The vast majority of cases of hydrocephalus are included in one of the two groups: (1) Communicating hydrocephalus; (2) obstructive hydrocephalus. The other types are rare. It will be noted that no separate subdivision has been made into internal and external hydrocephalus. It is a question whether external hydrocephalus ever really exists as a primary condition. It seems to be a secondary transformation of a primary internal hydrocephalus. This metamorphosis is probably due to a local or general atrophy of the cerebrum permitting escape to the exterior by these artificial channels. Because of the rarity of this condition, however, our facts are not sufficient to make a positive stand in this statement. It is conceivable, though not proven, that it may also result from a transfer of cerebrospinal fluid from the subarachnoid space to the subdural space in communicating hydrocephalus. The exact status of so-called external hydrocephalus is still in doubt. With this rare exception, nearly all hydrocephalus is internal; that is, the accumulation of cerebrospinal fluid is in the ventricles. It is internal for the very good reason that cerebrospinal fluid forms in the ventricles and cannot reach the exterior (obstructive hydrocephalus); or at most it reaches only a small fraction of the external absorbing surface—the posterior cranial fossa and spinal canal (communicating hydrocephalus). In either case the fluid dams back at its source. If cerebrospinal fluid could reach the subarachnoid space over the entire exterior of the brain, it would be absorbed and hydrocephalus could not exist.

Another rare type of hydrocephalus is that due to thrombosis of the vena magna Galeni. Owing to insufficient collateral venous circulation, hydrocephalus then results from an increased production of cerebrospinal fluid by venous stasis, just as ascites often follows stenosis of the inferior vena cava. I have produced this type experimentally, but have seen no instance clinically. Only a few cases have been reported in the literature.

In acute meningitis of inflammatory origin there is undoubtedly an increased production of fluid from the products of the inflammation. It is questionable whether an increase of fluid by exudation should be classified
with true hydrocephalus. It is probably external as much if not more than internal, because the infection has a general but principally external distribution. It subsides with the decline of the infection and is not of practical import in considering the treatment of hydrocephalus. An increase of fluid also follows trauma to the brain in fractures of the skull. It is probably of vascular origin, and usually subsides rapidly.

Fundamentally the two main types of hydrocephalus—obstructive and communicating—are similar. Both are due to an obstruction in the cerebrospinal fluid circulatory system. In our series of cases in children the relative frequency is nearly the same. In the former the obstruction is in the ventricles and in the latter in the subarachnoid space. The only reason for subdividing hydrocephalus into groups is that the anatomical differences in the two types necessitate an entirely different operative procedure for the treatment of each.

In this paper only the treatment of the communicating type of hydrocephalus will be considered. A form of treatment for hydrocephalus with obstruction will be presented in a later communication. For a proper understanding of the basis for the operation herein proposed, a brief explanation of the underlying etiology and pathology is necessary.

The Etiology and Pathology of Hydrocephalus with Communication.—It is the communicating type of hydrocephalus which has caused all hydrocephalus to be considered idiopathic. Though numerous explanations have been proposed, no pathologic findings have been presented until recently. All the ventricles communicate with each other and with the subarachnoid space, and post-mortem examinations of the brain have heretofore revealed nothing to the naked eye. The reason for the negative findings has been an inadequate knowledge (1) of the post-mortem appearance of the normal subarachnoid space and the pia arachnoid membranes; (2) of the alterations produced by pathological changes; (3) of the relation of the subarachnoid space to the absorption of cerebrospinal fluid; and (4) the anatomy and physiology of the cerebrospinal fluid circulatory system.

The only satisfactory time to observe changes in the meninges is when the brain is being removed. Adhesions of an extensive nature will then be seen and divided, but later will show very little in the preserved specimen. In cases of communicating hydrocephalus the meninges will be opaque and thickened, the normal filmy pia arachnoid will be replaced by a firm, fibrous, adherent membrane. This will be especially noted in the cisternæ at the base of the brain. The adhesions are often so dense as to tear the brain during their liberation. It is the distribution or location of these adhesions, not their extent, which determines the production of hydrocephalus (Fig. 5). Adhesions encircling the midbrain where it passes through the incisura tentorii, will destroy all communication between the posterior and middle cranial fossæ and thereby eliminate the entire subarachnoid space over both cerebral hemispheres from participation in the absorption of cerebrospinal fluid. Hydrocephalus will invariably result from such a process. Adhesions which
close the cisternæ at the base of the brain will also produce hydrocephalus just as effectively.

The extraventricular cerebrospinal circulatory system may be compared to the trunk and branches of a tree—the cisternæ representing the trunk, the subarachnoid spaces over the cerebral hemispheres the branches. Obliteration of the cisternæ by adhesions is equivalent to transection of the trunk of a tree. All distal communication will be destroyed and hydrocephalus will follow. Large areas of adhesions may be present over the cerebral hemispheres with only local effects and with no effect upon the general cerebrospinal fluid circulation. This is analogous to the absence of a relationship between the destruction of branches and the life of a tree. The absorbing function of these local areas is easily compensated by the subarachnoid space over the remainder of the brain.

In each of our cases these adhesions have also obliterated either one or two of the three foramina (Luschka and Magendie) by which the ventricles and the subarachnoid space normally communicate (Fig. 5). One foramen remaining patent will maintain an adequate transfer of cerebrospinal fluid. Should the adhesions close all three foramina at the base instead of only one or two, an obstructive hydrocephalus would result. In short, communicating hydrocephalus is due not to a reduction in the avenues of communication between the ventricles and the subarachnoid space but to a blocking of the primary trunks of the subarachnoid space, thus preventing cerebrospinal fluid from passing to the branches. The distribution of cerebrospinal fluid is thereby limited to that small part of the subarachnoid space in the posterior cranial fossa and the spinal canal. It does not include any of the subarachnoid space over either cerebral hemisphere, which is by far the most important area for absorption.

Cerebrospinal fluid is absorbed from the entire subarachnoid space. Adhesions obstructing the main stem of the subarachnoid space therefore limit the absorbing area to a fraction of the normal, and the diminished absorption results in accumulation of cerebrospinal fluid—hydrocephalus.

We have spoken of adhesions almost exclusively as the cause of communicating hydrocephalus. It is conceivable that certain tumors filling or compressing the cisternæ may produce similar results. We have as yet no proof of this possibility. All of the cases observed by Dr. Blackfan and myself have had a meningitis, which has been both prenatal and postnatal. There is frequently a definite history of meningitis preceding the development of hydrocephalus, but this cannot always be obtained.

Studies with the phenolsulphonephthalein test enabled us to predict ante mortem the pathologic findings. By the dye it was possible to measure the amount of absorption from any region by the quantitative excretion in the urine. It was found that normally practically no absorption occurs from the entire ventricular system (less than 1 per cent. in two hours). All the cerebrospinal fluid is absorbed from the subarachnoid space; 40 to 60 per cent. being excreted in a two-hour interval, which has been adopted as an
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arbitrary standard of time. In a large series of cases of communicating hydrocephalus the absorption from the subarachnoid space was found almost invariably to be only 8 to 10 per cent., or about one-fifth of the normal.

In view of the fact that experiments on animals have demonstrated that absorption of cerebrospinal fluid is from the entire subarachnoid space, the lowered absorption in communicating hydrocephalus must mean a reduction in the extent of the absorbing area and at a fairly constant location. The localization of the adhesions blocking the cisternæ or surrounding the mesencephalon harmonized the physiological and clinical tests and the pathological findings. The frequent history of an antecedent meningitis is additional confirmatory evidence, should such be necessary. The experimental production of this type of hydrocephalus by duplicating nature's pathologic processes, leaves no doubt of the etiological and pathological basis of communicating hydrocephalus. This experimental proof will shortly appear, together with that of the other types of hydrocephalus.

In operations upon the brain of cases of communicating hydrocephalus, one is impressed by the absence of fluid in the sulci. The sulci are practically obliterated, and only vascular lines separate the convolutions. If hydrocephalus were due to occlusion of such fanciful structures as stomata into the venous sinuses or the Pacchionian bodies, as has been proposed, one should expect distended subarachnoid spaces up to the points of obstruction. The absence of extracerebral cerebrospinal fluid proves the non-existence of any such mechanism, and indicates the lack of communication of the part of the subarachnoid space with the fluid-containing spaces; in other words, an obstruction must exist at some place nearer the origin of the cerebrospinal fluid.

The Diagnosis of Hydrocephalus with Communication.—Clinically, the two types of internal hydrocephalus are identical. Neither by the history nor the physical examination can they be differentiated. Frequently, the communicating type can be diagnosed by the large quantity of cerebrospinal fluid which can be obtained by lumbar puncture, but there is such great variability in the amounts of fluid obtained in both types of hydrocephalus that the results are frequently of little value. The differentiation between the two types of hydrocephalus can be simply, harmlessly and absolutely made by the phenolsulphonephthalein test.

One cubic centimetre of neutral phenolsulphonephthalein* is introduced into either lateral ventricle; a lumbar puncture is done one-half hour later. If communication exists, the dye will by that time have appeared in the spinal fluid. If an obstruction exists in the ventricular system, the spinal fluid will remain colorless. This test is the only way in which guess-work can be eliminated.

Previous Treatment of Communicating Hydrocephalus.—Because of the

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*This solution has been specially prepared by Mr. H. A. B. Dunning, of Hynson, Westcott & Dunning. A serious reaction will follow the use of the ordinary phenolsulphonephthalein solution used for kidney studies.
great number of operations suggested, tried, and found failures, their consideration individually would require too much space and yield little of practical value. With rare exceptions, no attempt has been made to differentiate the two types of hydrocephalus. The usual treatments have been directed toward a disposition of the fluid, (1) either by transferring the fluid to the exterior of the brain, to the scalp, the retroperitoneal space, the peritoneal cavity, or some other space; or (2) by making a communication with some part of the venous system, either directly or by a vascular transplant; (3) in addition to periodically repeated lumbar and ventricular puncture, attempts have been made to establish a more or less continuous drainage to the exterior, always, of course, with death from secondary infection. Other attempts have been made to reduce the formation of cerebrospinal fluid by ligating one or both carotid arteries, by injection of irritants into the ventricles, compression of the head, etc.

All attempts to drain into body spaces are futile because the tissues wall off the fluid and soon cease to absorb it. Moreover, the openings into the spaces remain patent only temporarily. Puncture of the corpus callosum might at first sight appear to be an ideal procedure, but it is practically identical with punctures elsewhere. Fluid side-tracked by an opening of the corpus callosum or of other parts of the brain does not pass into the subarachnoid space, but into the avascular subdural space, where absorption is little if any greater than in the ventricles or in the scalp. Moreover, the opening in the corpus callosum or elsewhere closes in the course of a few weeks. Fluid can reach the subarachnoid spaces over the brain only through the normally designated distributing channels—the cisternæ. Permanent communication between the ventricles and the cisternæ can be maintained only through the fourth ventricle and by means of the foramina of Luschka and Magendie, or, in their absence, by openings artificially produced in this region.

The fate of vascular communications is similar. The opening into the veins, sinus or transplant functions but briefly. Such openings can hardly be expected to function properly, because they should handle in a few minutes all the fluid which is produced in several hours, and would be functionless the remainder of the time. Normal absorption of cerebrospinal fluid takes place slowly, by a process of osmosis through membranes and not into prepared openings or stomata.

**Scientific Basis for Operative Treatment.**—The logical treatment of any disease is the removal of the cause. In communicating hydrocephalus the treatment of the cause, that is, the removal or liberation of adhesions, is precluded by present surgical limitations. The area of adhesions is too extensive, too diffuse, and in a location inaccessible for operative interference. The ideal treatment would be restoration of the cisternæ through these adhesions, and even if reconstruction were possible reformation of the adhesions must always remain a possibility.

In view of these deterring factors, treatment of the cause must for the
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present at least be deferred. From the results of experiments on animals a new and scientific form of treatment is suggested, by which it is hoped to circumvent the cause. This treatment aims to restore the balance between fluid production and fluid absorption by reducing the production of cerebrospinal fluid to a level where it can be absorbed. By experiments on animals, the results of which will appear shortly, the following facts have been established:

(1) If the foramen of Monro is occluded, a unilateral hydrocephalus results;

(2) But if the entire choroid plexus of this ventricle is removed at the time the foramen of Monro is occluded, this ventricle will be obliterated;

(3) Therefore, cerebrospinal fluid forms from the choroid plexus, and not from the ependyma.

(4) Following total occlusion of the aqueduct of Sylvius the development of hydrocephalus is greatly retarded by the extirpation of the choroid plexus of both lateral ventricles. In this experiment the cerebrospinal fluid, which is produced proximal to the obstruction, can be derived only from the choroid plexus of the third ventricle.

The conclusion from these experiments is if all the choroid plexuses of the four ventricles could be removed, the formation of cerebrospinal fluid would cease and hydrocephalus could not result or if present its development would cease. To cure obstructive hydrocephalus by removal of the choroid plexus, it would be necessary to remove all of the choroid plexuses because there is no absorption in the ventricles; but in communicating hydrocephalus, there is about one-fifth of the normal absorption. It would not therefore be necessary to remove the entire amount of choroid plexus but to reduce its volume until the amount remaining would not produce cerebrospinal fluid faster than it could be absorbed. In other words, it would be necessary to remove roughly four-fifths of the total amount of choroid plexus to reduce the fluid formation to the 8 to 10 per cent. absorption (one-fifth of the normal) which occurs in communicating hydrocephalus.

It is not feasible to remove the choroid plexus from the third ventricle and very difficult to extirpate flocculi in the fourth ventricle. Roughly the choroid plexuses of the combined third and fourth ventricles is about one-fifth of the total amount in all the ventricles. The plexus is in the two lateral ventricles therefore comprise about four-fifths of the total. It is relatively easy to remove the choroid plexus from both lateral ventricles. A bilateral extirpation from the lateral ventricles should, therefore, according to our present conceptions, reduce the formation of cerebrospinal fluid to a point where it can be absorbed by the restricted patent area of the subarachnoid space.

The problem, however, is not purely one of mathematics. Casually, one would expect that the removal of 75 per cent. of the productive structure would reduce the formation to meet absorption which is 20 to 25 per cent.
of the normal, but it should do even more, for in hydrocephalus fluid is formed at a greatly reduced rate because of the changed intracranial pressure from fluid accumulation. It has been demonstrated that the formation of cerebrospinal fluid is to a large extent at least mechanical. This can easily be demonstrated by inducing venous congestion by compression of the veins of the neck. We know that fluid forms at a greatly lessened rate in hydrocephalus due to the increased intracranial pressure, because if it formed at the normal rate (which we know) the head would grow at a tremendous speed. Although nature can modify the rate of fluid production she is unable to reduce its formation to the level at which it can be absorbed. The removal of the choroid plexus from both lateral ventricles should be more than is necessary, but it seems preferable to remove too much with no consequent danger, than to run the risk of an insufficient removal, in which event a progressive destruction of the brain will inevitably result. Hydrocephalus even when developing at the slowest rate causes a rapid atrophy of the brain. It is of course obvious that if more than the necessary amount of choroid plexus is removed, extra- and intravascular pressure differences will produce sufficient fluid to maintain the necessary amount of fluid to fill the ventricles.

The Operation.—The steps in the removal of the choroid plexus are clearly shown in the accompanying drawings by Miss Norris. A small circular bone flap is made over the parietal eminence (Fig. 6). The wound is made well posterior to the Rolandic area and in a salient part of the occipital lobe. After ligating numerous vessels on the cortex by circumvection, the cortex is bloodlessly incised and this incision carried into the ventricle. From the exposure which is over the junction of the body and descending horn of the lateral ventricle, the entire extent of the ventricle can be brought into view (Fig. 7). The opening in the brain is maintained by an open nasal dilator (Fig. 8), or when the ventricle is very large the brain wall must be elevated by a spatula which is inserted into the ventricle. It is necessary to remove all the cerebrospinal fluid in the ventricle to get a view of the choroid plexus; the brownish-red flocculent choroid plexus can then be easily followed from the foramen of Monro to the tip of the descending horn.

The choroid plexus is picked up in forceps at the foramen of Monro (Fig. 8), and the vessels ligated by a silver clip. A pledget of moist cotton is inserted into the foramen of Monro to prevent blood gravitating into the third ventricle. The plexus is then transected and gently stripped backward from its narrow attachment to the floor of the body of the ventricle. When the glomus is reached the stripping from the body of the ventricle is stopped and the choroid plexus picked up at the tip of the descending horn (Fig. 9). This part of the choroid plexus is also stripped backward to the glomus; the remaining attachment of the glomus is then liberated and the entire choroid

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*I am greatly indebted to Professor Halsted for many suggestions in the development of this operative procedure, as well as in the experimental work upon which the operation is founded.
Fig. 1.—Views of case of early communicating hydrocephalus with lumbar meningocele. The meningocele was removed and a bilateral extirpation of the choroid plexus of the lateral ventricles was performed in the first three weeks of life. The head is not enlarged. A most unusual diagnosis of hydrocephalus was made at birth by Dr. J. Whitridge Williams, because of the wide fontanelles and the meningocele.

Fig. 2.—A ventriculogram of the case of communicating hydrocephalus shown in Fig. 1. A well-advanced hydrocephalus can exist even when the size of the head is normal.
FIG. 3.—Same child as Fig. 1, eight months after the bilateral operation. The scar can be seen in the back of the head. The head is still of normal size. The size of the fontanelles is much reduced. The child is apparently perfectly well.

FIG. 4.—Case of advanced communicating hydrocephalus. Beyond this stage the patient seldom survives any operative procedure. This patient died three weeks following the removal of the choroid plexus of one lateral ventricle.
FIG. 5.—Diagram to show the distribution of adhesions over the base of the brain in a case of communicating hydrocephalus. All the cisternæ are obliterated. Communication between the ventricles and subarachnoid space is restricted to one foramen of Luschka (L). The other foramen of Luschka and the foramen of Magendie are sealed by the adhesions. Ablation of the cisternæ prevents cerebrospinal fluid reaching the cerebral subarachnoid space.
Fig. 6.—Shows location, shape and size of incision in scalp and bone for bilateral extirpation of choroid plexus of lateral ventricles. The solid line marks the skin incision, the dotted line the bone flap which is broken and reflected at the base.

Fig. 7.—Skin, bone and dura are reflected. The cerebral vessels are doubly ligated with fine silk by circumvection. Between these two rows of sutures the cortex is incised with the scalpel and this incision carried through to the ventricle.
FIG. 8.—The opening in the cortex is maintained by a nasal dilator. Through this opening the choroid plexus is removed in the manner shown in Fig. 9. The fluid has been entirely removed from the ventricle to permit exposure of the choroid plexus.

FIG. 9.—Coronal section through hydrocephalic brain, showing method of stripping choroid plexus from its attachment to the floor of the ventricle. The right plexus has been stripped from the foramen of Monro to the end of the glomus and at the tip of the descending horn is shown grasped by the forceps in the process of being stripped to the glomus. The entire plexus is then lifted from its bed.
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plexus removed in toto. Bleeding from the denuded area of velum interpositum is slight and easily controlled by moist saline cotton pledgets. Special care must be taken to leave no bleeding points.

The collapse of the brain following evacuation of the ventricular fluid causes a remarkable infolding of the cerebral walls, the extent depending of course upon the size of the ventricle and thickness of the cortex. In advanced cases a tremendous cavity results, which is filled before closure with salt solution to restore the collapsed cerebral wall as nearly as possible to its natural convexity.

A remarkable exposure is obtained during the operation in the ventricle. One can see the third and opposite lateral ventricle and the septum lucidum which is frequently perforated in many places owing to pressure atrophy.

The opening in the cortex is closed with a series of interrupted fine silk sutures which are held by the delicate pia arachnoid membrane. The dura and scalp are carefully closed also with silk, special care being taken to prevent any subsequent leak of cerebrospinal fluid.

Result.—I have extirpated the choroid plexus in four cases of hydrocephalus from Professor Halsted’s Clinic. All of these have survived the operation, although three died two to four weeks after the operation. One patient has survived a bilateral choroid plexectomy ten months, and shows no evidence that the disease is advancing. During and following the operation the reaction seems to vary directly with the grade of the disease. If the ventricle is small the operation will be well tolerated even by a very young baby. When the ventricles are large and the cortex is greatly thinned and marked enlargement of the head has resulted, a very severe reaction occurs during the operation and the convalescence is very slow. In the highest grades of hydrocephalus death will follow almost immediately upon release of the fluid, or a very severe reaction will result at once and death will quickly follow. In the advanced cases we can hold out very little encouragement from operative procedures of any kind which will necessitate release of fluid and consequent collapse of the brain. There is, however, little object in attempting a cure of the disease in this advanced stage because the child would be left a hopeless imbecile. In the three cases of this series which subsequently died the hydrocephalus was of the extreme grade. In each of these there was an immediate operative collapse, beginning with pallor of the face and body, rapid feeble pulse which quickly becomes imperceptible, cold clammy perspiration, rapid, shallow and irregular respirations—in other words, typical shock. These changes invariably begin with evacuation of ventricular fluid, and are undoubtedly due to differences of pressure which affect all the blood-vessels and directly or indirectly the centres in the medulla. Following escape of the fluid the thin brain walls collapse like a wet cloth. In addition to the differences in intra- and extravascular pressures, the mechanical kinking of the large vascular trunks by angulation of the infolding brain must have a pronounced effect upon the circulation.

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In each instance there was a gradual recovery, and death came from two to four weeks later. In two cases death was no doubt due to gradually progressive acute intracranial pressure which we now would be able to recognize and probably alleviate. In a third case the cause of death is still uncertain, though undoubtedly a result of the operation. The temperature rose to 108° four hours after operation; remained around 102° to 106° for two weeks. Though conscious, the ability to swallow was lost and not regained. At autopsy no cause for death could be observed.

In the fourth case, which is still living and well and with no evidence of progress of the disease, no post-operative effects were observed, despite the fact that within three weeks of birth three operations were performed, one for the removal of a large myelomeningocele and two for the bilateral extirpation of the choroid plexus. Feeding was uninterrupted; the temperature at no time rose over 100°, and the rising curve of body weight was not even temporarily affected.

This case was kindly referred to me by Dr. J. Whitridge Williams, who made a most unusual diagnosis of hydrocephalus immediately following the child’s delivery in his clinic. The head was not enlarged but the fontanelles were wider and a trifle fuller than normal (Fig. 1). The myelomeningocele also suggested the possibility of hydrocephalus. A ventriculogram showed a well developed hydrocephalus with complete obliteration of the posterior horn of the lateral ventricle (Fig. 2). In no other way could this very early tentative diagnosis have been substantiated.

Unfortunately in this case our diagnosis of communicating hydrocephalus has been to a large extent conjectural. The large myelomeningocele filling the lumbar region precluded successful lumbar puncture, so that we were unable to determine by the phenolsulphonephthalein test whether communication was present, or indirectly by quantitative absorption whether the hydrocephalus was of the communicating type.

The absorption of phenolsulphonephthalein following injection into the ventricle was 2 per cent., which is a little higher than in obstructive hydrocephalus and about what obtains in communicating hydrocephalus. This is not considered conclusive by any means, as the difference between the ventricular absorption in the two types of hydrocephalus is not great enough to be a decisive test. The meningocele is by no means evidence in favor of a communicating and against an obstructive hydrocephalus.

Wherever possible both choroid plexuses should certainly be removed. This requires two operations, the length of time intervening depending upon the reaction following the first operation. Only one of our cases has had a bilateral extirpation. It is doubtful whether the removal of the choroid plexus of one ventricle would produce more than a retardation of the disease, which, of course, would be of no ultimate benefit.

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Even though one case is apparently well ten months after a bilateral extirpation (Fig. 3), no conclusions are justifiable on the basis of a single case or in such a short period of time. The operation is presented without claims of cure, but because of its apparently sound scientific foundation it is hoped and expected that cures will result.

CONCLUSIONS

1. Any treatment of hydrocephalus must be based upon the etiology and pathology of this disease.
2. Communicating hydrocephalus is caused by an obstruction in the subarachnoid space causing diminished absorption of cerebrospinal fluid.
3. This obstruction is probably nearly always due to adhesions following meningitis.
4. These adhesions close the cisternae through which all cerebrospinal fluid is distributed to the subarachnoid space over the cerebral hemispheres.
5. Absorption is reduced to one-fourth or one-fifth of the normal, roughly corresponding to the volume of subarachnoid space which contains cerebrospinal fluid.
6. It is at present impossible to reestablish the cisternae by surgical means.
7. By experiments it has been demonstrated that cerebrospinal fluid forms from the choroid plexus.
8. An operation is presented for the cure of this type of hydrocephalus by removal of the choroid plexus of both lateral ventricles. This removes, roughly, four-fifths of the total amount of fluid-forming structures. It is hoped the cerebrospinal fluid which forms from the choroid plexus can be absorbed in the small amount of subarachnoid space which remains in the third and fourth ventricles.
9. Choroid plexectomy is of value only in communicating hydrocephalus. Any treatment therefore presupposes an accurate diagnosis of the type of hydrocephalus. This is best made by the phenolsulphonephthalein test.
10. The operation has been performed on four cases. One case is alive and apparently well ten months after the operation.
11. Sufficient time has not elapsed to speculate on the practical results of the operation.
12. The operation can be safely performed in moderately advanced cases of hydrocephalus.