The treatment of nonobstructive (communicating) hydrocephalus by endoscopic cauterization of the choroid plexuses

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The author reviews his personal experience with 39 cases of nonobstructive (communicating) hydrocephalus treated by endoscopic cauterization of the choroid plexuses during the period 1942–1965. The paper includes a brief history of the development of this operative method, and a description of the author’s ventriculoscope. The operative technique has been described in detail. The operative mortality was 10% overall, 5% in the last 20 cases. Hydrocephalus was initially arrested in 26 cases and has remained so in all cases, with a follow-up period of more than 10 years in 23 patients. Only one case developed a late complication requiring reoperation.

This paper is a comprehensive report of my personal experiences in treating nonobstructive (communicating) hydrocephalus by endoscopic cauterization of the choroid plexuses, with detailed descriptions of the ventriculoscope, operating techniques, and results. The merit of this method of treatment lies not so much in its low operative mortality or high percentage of initial successes in arresting hydrocephalus but in the low incidence of late serious complications and high incidence of long-term survivals.

Historical Background

In 1918 Dandy¹ first proposed treating nonobstructive hydrocephalus by destroying the choroid plexuses in the lateral cerebral ventricles, thereby reducing the cerebrospinal fluid formed by the plexuses to an amount which the malformed or damaged subarachnoid system could absorb and return to the general blood stream. His rationale was sound, but the operation which he devised to achieve this end was technically a poor one (Fig. 1 upper left), and resulted in the immediate death of three of the four children upon whom he attempted it. The basic defect in Dandy’s operation was that it involved emptying all of the cerebrospinal fluid from both ventricles before the choroid plexuses could be destroyed; this allowed the thin walls of the ventricles to collapse, which in turn resulted in severe shock.

In 1932 Dandy² described a method of removing the choroid plexuses of each lateral ventricle through a Kelly cystoscope (Fig. 1 upper right) which he referred to as a “ventriculoscope.” The ventriculoscope was further modified³ in 1938 (Fig. 1 lower). Through it, after the cerebrospinal fluid had been completely emptied from both lat-
Fig. 1. Dandy’s operative techniques. Upper Left: Original technique (1918). With thin-bladed nasal speculum and illumination by light reflected from a head mirror, the plexuses were mechanically avulsed. Upper Right: Later technique (1932). A Kelly cystoscope, which Dandy called a “ventriculoscope,” was introduced into the ventricles, and the plexuses were avulsed. Lower: Final technique (1938). All three techniques required complete evacuation of CSF from the two lateral ventricles before destruction of the plexuses. This resulted in collapse of the thin cortex, and shock.

eral ventricles, the plexuses were either avulsed or cauterized, with results comparable to those in 1918.

The high operative mortality and low incidence of success attending “open, surgical” methods for destroying the choroid plexuses, involving the nearly total evacuation of the cerebrospinal fluid from the ventricles, was subsequently confirmed by the definitive reports of Sachs15 and Davidoff7 (Table 1).

Endoscopic cauterization of the choroid plexuses was first described in 1934 by Putnam.11 This marked a great advance in the treatment of nonobstructive hydrocephalus,
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for it enabled the surgeon to destroy the choroid plexuses in the lateral ventricles with minimal loss of ventricular fluid, thereby eliminating the massive collapse of the thin ventriculoscope was of original and unique surgical shock. Putnam reported the use of this ventriculoscope to cauterize the plexuses in seven hydrocephalic infants, with two operative deaths, two children “unimproved,” and three children “improved.” Putnam’s ventriculoscope was of original and unique design, consisting of a solid rod of optical glass 0.7 cm in diameter and 18.0 cm long, to the sides of which were fastened “a light-carrier with a tiny bronchoscopic light,” and a pair of bipolar cauterizing electrodes, which were bent toward each other over the distal end of the glass rod (Fig. 2).

In 1936, I described a new ventriculoscope of original design (Fig. 3)* that resembled in general appearance and construction a modern operating cystoscope, but with certain features specifically adapting it to use within the ventricles of the brain. Incorporated in it were: 1) a “fore-oblique” lens-system, providing not only “forward” vision but a wide angle (70°) of lateral (oblique) vision; 2) a maneuverable unipolar cauterizing electrode; 3) a lighting system; and 4) an irrigating system which could not only clear away blood from the immediate operative field but also continuously replace any fluid escaping from the ventricles along the shaft of the ventriculoscope during cauterization of the plexuses, thereby assur-

* Developed with the generous technical and financial assistance of the late Mr. Frederick Wappler, then President of American Cystoscope Makers, Inc., of New York.

TABLE 1

<table>
<thead>
<tr>
<th>Data</th>
<th>Sachs (1942)</th>
<th>Davidoff (1948)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of cases</td>
<td>90</td>
<td>32</td>
</tr>
<tr>
<td>Operative deaths</td>
<td>46%</td>
<td>43%</td>
</tr>
<tr>
<td>Arrested hydrocephalus</td>
<td>54%</td>
<td>50%</td>
</tr>
<tr>
<td>Follow-up periods</td>
<td>1 to 7 yrs</td>
<td>1 to 4 yrs</td>
</tr>
<tr>
<td>Average survival period</td>
<td>1 yr</td>
<td>2½ yrs</td>
</tr>
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</table>

ing a constant, normal intraventricular pressure throughout the procedure. At the same time, I reported endoscopic cauterization of the choroid plexuses in five infants suffering from verified, nonobstructive hydrocephalus, with one operative death, one failure to arrest the hydrocephalus, and three cases in which the hydrocephalus had apparently been arrested; one of this group survived for 5 years, the longest survival period following endoscopic cauterization to have been reported at that time.

In 1956, Feld† described a ventriculoscope (Fig. 4) comparable to mine but with a simpler “forward-viewing” lens system, employing a tiny mirror fixed obliquely just in front of the fore end of the lens system, to provide lateral (oblique) vision. Feld reported endoscopic cauterization of the choroid plexuses in 14 hydrocephalic children, with no operative deaths, five failures, and successful arrest of hydrocephalus in nine cases.

Clinical results of endoscopic cauterization of the choroid plexuses in the treatment of hydrocephalus have been reported by Put-
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Fig. 3. Scarff's ventriculoscope (1935). Resembling a modern operating cystoscope, the instrument incorporated a "fore-oblique" lens system, a maneuverable unipolar electrode, an illuminating system, and an irrigating system.

nam,11-14 Scarff,16-23 and Feld,8,9 These 95 cases, comprising all of the cases which have thus far been reported, are summarized in Table 2.

The intellectual development of children after arrest of hydrocephalus by cauterization of the choroid plexuses has been less completely reported. Putnam, for example, followed only three of his 42 surgical cases for periods longer than 3 years after operation, two for 6 years, and one for 7 years. In his last report about them, published in 1943, Putnam14 stated that one of these three children was severely retarded but that the other two were functioning satisfactorily in the first grade of school. Feld’s untimely death, just 3 years after his first successful cauterization, ended any significant follow-up studies of his cases.

I have been able to follow the intellectual development of all of my cases fairly closely, from 1934 through 1965. The five follow-up reports18-20,27,28 are summarized in the section of this report entitled "Results."

The Ventriculoscope

The present (1963) model of my ventriculoscope (Fig. 5), here described in medical literature for the first time, is identical to the original (1935) model except that the lighting system used in the original ventriculoscope has been replaced in the later model.

Fig. 4. Feld's ventriculoscope (1956). Comparable to Scarff's ventriculoscope, this instrument had a slightly modified lens system.
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TABLE 2
Results of endoscopic cauterization of choroid plexuses

<table>
<thead>
<tr>
<th>Author</th>
<th>No. of Cases</th>
<th>Operative Deaths (no., %)</th>
<th>Initial Successes (no., %)</th>
<th>Postop Survival of Initially Successful Cases</th>
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</thead>
<tbody>
<tr>
<td>Putnam</td>
<td>42</td>
<td>10 (25%)</td>
<td>17 (40%)</td>
<td>Single Cases (yrs) 1, 1, 1 2, 2, 2, 2 3, 3, 3, 3, 3 6, 6, 7 3 (7%) 3 7</td>
</tr>
<tr>
<td>Scarff</td>
<td>20</td>
<td>3 (15%)</td>
<td>10 (50%)</td>
<td>5, 9, 10, 14, 15 25, 25, 26, 26, 27 10 (50%) 18 27</td>
</tr>
<tr>
<td>Scarff</td>
<td>19</td>
<td>1 (5%)</td>
<td>16 (80%)</td>
<td>8, 10 10, 10, 11, 11, 11, 11 16, 17, 17 17, 17, 18, 19, 19 16 (80%) 13 19</td>
</tr>
<tr>
<td>Feld</td>
<td>14</td>
<td>0 (0%)</td>
<td>9 (65%)</td>
<td>2-27 months, 9 cases living &amp; well at time of Feld's death — — —</td>
</tr>
<tr>
<td>Summary</td>
<td>95</td>
<td>14 (15%)</td>
<td>52 (60%)</td>
<td>— 29 (30%) 8 27</td>
</tr>
</tbody>
</table>

* Not followed.

by the vastly superior “fiber optic lighting system.”*

The ventriculoscope consists basically of two parts, 1) a light-weight tubular metal sheath with obturator, and 2) an “operating element” that incorporates lens, cauterizing, irrigating, and lighting systems.

**Lens System**

The “fore-oblique” lens system used in the ventriculoscope possesses two valuable optical properties, namely, universal focus from zero to infinity (0–∞), which obviates the need for any mechanical focusing adjustment, and an extremely wide angle (70°) of lateral (“oblique”) vision, in addition to forward vision (Fig. 6). This lens system has one minor disadvantage, namely, that its optical field of vision is limited to 180°, or only one-half of the normal physiological field of vision of 360°, a limitation which is easily overcome by simply rotating the ventriculoscope as a whole about its longitudinal axis.

* The new model ventriculoscope is manufactured by American Cytoscope Makers, Inc., Pelham Manor, New York.

**Cauterizing System**

The unipolar, cauterizing electrode can be advanced or withdrawn by simply turning a thumb screw conveniently located on the proximal end of the “operating element.” This electrode is so constructed that it does not advance in a straight line from the tip of the ventriculoscope; instead it advances at an angle of 45° to the longitudinal axis of the ventriculoscope, so that its tip is always in the center of the optical field of vision of the “fore-oblique” lens system (Fig. 6). This arrangement enables the surgeon not only to see but to reach and destroy portions of the choroid plexuses near the foramina of Monro and in the temporal horns; this would otherwise be impossible.

**Irrigating System**

This consists of two small-caliber metal tubes running the full length of the “operating element” of the ventriculoscope on either side of the lens system (Fig. 5). Distally, these tubes open just lateral to the lens objective; proximally, they end in stopcocks firmly mounted on the operating element which accept the male adapters on the ends

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Fig. 5. Scarff's "fiber optic" ventriculoscope (1963). The following are basic parts: a sheath (1), an obturator (2), and an "operating element" (3), in which are incorporated lens, cauterizing, irrigating, and lighting systems. The lens system consists of a multi-element lens enclosed in a narrow metal tube (4), with an eyepiece (5) at the proximal (external) end, and an objective lens (6) at the operating tip of the ventriculoscope. The cauterizing system includes an electric cable (not shown) that leads from the power source to a connection (7) on the external end of a unipolar cauterizing electrode (8), whose cauterizing tip (9) can be advanced or retracted by turning a ratchet wheel (10) located on the proximal end of the ventriculoscope. Shown are connections on the ventriculoscope to the irrigating system for the irrigating tubes (11), stopcocks (12) for controlling the volume and force of the irrigating fluid, and "delivery outlets" for the irrigating fluid (13) on each side of the lens objective. The lighting system consists of a self-contained "fiber optic lighting unit," a flexible glass "fiber optic" light cord (14) for conducting the light from the light unit to a connecting post (15) on the ventriculoscope, and a sheath of fiber optic glass incorporated within the shaft of the ventriculoscope, which conducts the light from the fiber optic electric cord down the shaft of the scope to a "light portal" (16) at the operating tip.

of light rubber tubes coming from a reservoir of irrigating fluid (Ringer's solution) that is suspended 10 to 12 inches above the patient's head. The pressure of the irrigating fluid is regulated by opening or closing the stopcock.

The irrigating system serves two important functions: 1) with the stopcocks widely open, it provides brisk effective irrigation of blood out of the immediate operative field; 2) with the stopcocks only partially opened, it provides continuous replacement of the small amount of ventricular fluid which invariably escapes along the outside of the ventriculoscope during the cauterization of the choroid plexuses (Fig. 7).

Lighting System

The ventriculoscope is equipped with a "fiber optic" lighting system. The source of the light for illuminating the ventricles is a pair of interchangeable 150 W light bulbs with integrated reflectors, housed in a separate light supply unit placed a short distance from the operating table. From this source, a powerful beam of light is conducted to the ventriculoscope by a light-weight, flexible, sterilizable cable composed of optical glass fibers. The light is conducted thence to the operating tip of the ventriculoscope by a second "sheath" of optical glass fibers surrounding the lens system. The light which this system is capable of delivering into the ventricles is 10 times greater than that which could be delivered by the lighting system in my original (1935) ventriculoscope; it is a "cold" light when it reaches the ventricle; and the possibility of its failure during cauterization of the choroid plexuses is almost nil.
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**Operative Technique**

**Essential Preoperative Data**

Before endoscopic cauterization of the choroid plexuses is undertaken, specific diagnosis of nonobstructive (communicating) hydrocephalus must be established. The surgeon must also know the size and shape of the ventricles and the thickness of the cortical mantle. Finally, the presence or absence of a chronic subdural hematoma, hygroma, or congenital neoplasm or cyst as either primary or contributing factors to the elevated intracranial pressure should be determined before operation is undertaken. The simple ventriculogram and dye test, properly performed, will supply the surgeon with all of this essential preoperative information quickly, reliably, and with a minimum of disturbance to the young patient. It has long been my practice to carry out these tests promptly on all infants and young children.
with clinical evidence suggesting hydrocephalus.

Anesthesia

The choice of specific agents and techniques should be left to the experienced anesthesiologist. He should be informed by the surgeon of these facts: 1) the patient will experience no pain during the operation, once the scalp has been infiltrated with 1% procaine; 2) the infant must lie quietly, without movement, for about 3 hours, for a bilateral cauterization, but deep muscular relaxation is not necessary; 3) the loss of blood during the operation should not exceed 10 cc; 4) surgical shock, either of vascular or neural origin, does not result from either the manipulation of the endoscope within the ventricular cavities, or from the actual cauterization of the choroid plexuses. In brief, no special problems of anesthesia should be encountered.

Position

The patient is operated on prone with the head and neck hyperextended, to have the parietal burr holes as high as possible in their relationship to the ventricular cavities, thus minimizing the escape of cerebrospinal fluid from the ventricles along the outside of the shaft of the ventriculoscope (Fig. 8).

Bilateral Trephinations

These are made through the two parietal bossae, that is, more rostral and lateral than the trephinations commonly used for performing ventriculography. If the trephine openings are located too far posteriorly (too low), the raised, central portion of the curved floor of the lateral ventricle will make it difficult for the surgeon to bring the operating tip of the ventriculoscope down and into contact with the choroid plexus in the region of the foramen of Monro (Fig. 8); if the trephine openings are too close to the midline, the medial wall of the ventricle will interfere with the shaft of the ventriculoscope when the surgeon attempts to follow and destroy the choroid plexus as it extends posterolaterally along the thalamus and into the temporal horn of the ventricle.

Incisions

Scalp incisions about 3 cm long, parallel to the midsagittal line, are carried directly down through galea and pericranium to the bone (Fig. 9 A). Maximum care is exercised in identifying, mobilizing, and retracting the galea so as not to tear or fray its edges, or to "button-hole" it. A strong and waterproof closure of the galea, after the cauterization of the plexuses, is essential if postoperative cerebrospinal fluid fistulas are to be avoided. The galeal sutures to be used in closure are "preset" at this time (Fig. 9 B). This is done to minimize the loss of CSF from the ventricles which would occur if the galeal sutures were not placed until the wound was being closed. Interrupted sutures of fine black silk, each about 25 cm (10 in.) long, are placed with small, curved, non-cutting needles. Substantial "bites" of the galea are taken so that the closure will be strong, and the sutures are placed at 2 mm intervals so that the closure will be watertight. Special care is taken to place sutures deeply in the upper and lower angles of the galeal incision, for these are the common sites for leakage of CSF (Fig. 9 A, B, C).

After all of the galeal sutures have been
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Fig. 8. Positioning of the patient's head and neck for operation. *Left:* Incorrect position. The head and neck are flexed, thereby bringing the parietal burr hole below the level of the cerebrospinal fluid in the occipital and posteroparietal portions of the ventricles, and resulting in an increased loss of fluid from those parts of the ventricles. *Right:* Correct position. The head and neck are hyperextended. This brings the parietal burr holes above the top level of the ventricular fluid at all points, and reduces its loss to a minimum. The burr holes properly placed through the parietal bossae enable the cauterizing electrode to reach and cauterize the choroid plexuses both at the foramen of Monro and within the temporal horn of the ventricle.

If a leakage of CSF develops within the first 24 to 48 hours after operation, it is invariably traceable to a poor closure of the galeal incision and usually to the fact that the galeal sutures have not been placed sufficiently close. If such a leak develops, the patient must be returned promptly to the operating room. All of the superficial skin sutures must be removed in order to expose all the galeal sutures. The precise site of the leak between two galeal stitches must be determined, and supplementary galeal sutures added to seal the leaking point.

**Removal of Bone**

The pericranium is stripped off the bone with a sharp periosteal retractor (Fig. 9 F), until a clean bone surface, roughly 2 to 5 cm in diameter, is exposed. No effort is made to preserve the pericranium intact, for no effort to suture the pericranium during closure will be made. Bone is removed to provide an opening approximately 2.5 cm in diameter, which is large enough to accommodate the shaft of the ventriculoscope and allow it free manipulation inside the ventricle.
Fig. 9. Technique for opening the galea: "presetting" the galeal sutures in preparation for final closure. 
A. The scalp incision is carried directly down to bone. The galea is carefully identified, isolated, and 
mobilized for some distance laterally. Great care is taken not to fray the edges of the galea or to "button- 
hole" it. B. Individual sutures of fine black silk, 10 to 12 inches long, threaded on small curved non- 
cutting needles, are placed in the galea, 2 to 3 mm apart. C. The "preset" sutures are then collected into 
two bundles, lifted up and off the pericranium (D); retracted laterally and "anchored" out of the way 
(E); and covered, temporarily, with sterile drapes until time for closure (F).
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Preparation of the Ventriculoscope for Cauterization of the Choroid Plexuses

This is carried out at this time, before the dura is opened on either side. Necessary connections must be made between the operating element of the ventriculoscope and the light source, the electrosurgical unit, and the reservoir of irrigating fluid (Ringers solution) as well as appropriate “settings” for each of these systems.

The most important setting is that for the cauterizing current. Complete reliance for this setting should not be placed on a dial reading on the electrosurgical unit. The best way is to actually test the strength of the cauterizing current directly on some bit of unimportant tissue exposed in the scalp incision, by making a series of gentle “stroking” movements of the tip of the cauterizing electrode across the tissue, at the same time having the strength of the current gradually increased. When the slightest contact of the moving cautery tip causes instant blanching of the tissue, the strength of the current is correct for beginning the cauterization of the plexuses, although it may be found, on actual trial, that slightly more current is needed when the cautery tip is totally immersed in ventricular fluid. A current that is too powerful will char or blacken the tissue, and cause the tip of the electrode to adhere to it; this can set off a train of unpleasant complications.

Opening of the Dura

The dura is now opened on one side of the head only. It is not opened on the second side until after satisfactory cauterization of the choroid plexus has been achieved in the first ventricle, and the galea and skin on that side have been permanently closed. The reason is that in possibly 50% of all cases it may not be feasible for various reasons to proceed to the cauterization of the plexus in the second ventricle at the initial operation session; in such cases cauterization of the plexus in the second ventricle can be carried out far more easily and quickly at a later operation if the dura on that side has not been opened previously.

The dura should be opened widely to provide maximum exposure of the surface of the brain. Any type of dural incision may be used. In closing, no effort will be made to suture the dura, since a watertight closure of any short incision of the dura is practically impossible. Hemostasis should be as complete as possible, for any free blood in the field tends to follow the shaft of the ventriculoscope down into the ventricle; and even very small amounts of blood in the ventricular fluid may at times seriously impair visibility within the ventricle.

Introduction of the Ventriculoscope into the Ventricle

An incision about 0.5 cm long is made just though the pia. A brain cannula is introduced through the midpoint of this incision into the ventricle; the thickness of the cortex, measured directly on the cannula, is noted for future use and the cannula is withdrawn. A small straight hemostat (Mosquito) is then introduced along the tract of the cannula until its tip lies just inside the ventricle; the tips of the hemostat are then opened until they are about 0.5 cm apart, and, with the hemostat maintained in this opened state, it is slowly withdrawn from the brain. The diameter of the transcortical tract thus prepared for the ventriculoscope should not be too great. The sides of the pathway should slightly grip the shaft of the ventriculoscope as it is forced along the tract into the ventricle; this insures minimal leakage of CSF from the ventricle during the cauterization of the plexuses.

It is essential after the ventriculoscope has been passed through the cortex that it be further advanced until its tip lies about 2.5 cm inside the ventricular cavity; this is because the objective element of the lens system is located approximately 2.0 cm proximal to the tip of the ventriculoscope (Figs. 5 and 6). Since introduction of the instrument into the ventricle must be done blindly, it now becomes evident why preknowledge by the surgeon of the thickness of the cortex as measured on the brain cannula, and the size and shape of the ventricles as shown by the ventriculogram, are so important.

After the sheath and obdurator have been introduced for the proper distance into the ventricle, the obdurator is replaced by the “operating element” and the lighting, irrigating, and cauterizing systems activated.
Cauterization

The actual cauteryization of the choroid plexuses is carried out by a series of light “stroking” movements of the cautery tip across or along the plexuses, the movements being made in such a direction that the tip of the cautery is always being pulled away from the plexuses (Fig. 10 upper right), never pushed into them (Fig. 10 upper left). This avoids hooking the plexuses and tearing them, which would start bleeding difficult to control.

The strength of the cauteryizing current is very important. If the current is of proper strength, the choroid plexus should turn a chalky white (blanch) the instant that it is touched by the moving electrode (Fig. 10 lower right); the cautery tip will not adhere to the plexus. If, on the other hand, the current is too strong, the choroid plexus will turn black (Fig. 10 lower left); the cautery tip will then adhere to the plexus, and attempts to free it can result in tearing the blood vessels deeply placed in the plexus, and thus cause troublesome bleeding.

In brief, not only must the current be right, but the cautery tip must always be moving and in the right direction during the

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**Fig. 10. Precise technique for cauterizing the choroid plexuses.** *Upper: Mechanical factors. The cautery tip should always be moving while in contact with the choroid plexus, in repetitive “stroking” movements so directed that the cautery tip is “pulled” away from the plexus (upper right), and not “pushed” into it (upper left), to avoid plexus tearing and bleeding. Only the most superficial portion of the plexus should be touched by the cautery. Lower: Thermal factors. The strength of the cauterizing current must be correct. If too strong, it will blacken the plexus and the cautery tip will adhere to it (lower left); if of proper strength, it will whiten the plexus the instant it touches it, and the cautery tip will not adhere to it (lower right). Only the most superficial portions of the plexuses need be cauterized.*
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time that it is in contact with the plexus in order for cauterization to proceed satisfactorily.

Amount of Choroid Cauterized

As much of the choroid plexuses as possible should be destroyed in both ventricles, preferably at a single initial operative session. Experience has firmly established the fact that it is impossible, from a practical standpoint, to destroy too much plexus; the real mistake is to destroy too little. If the surgeon is prevented, for any reason, from achieving the maximum possible destruction of the choroid plexuses in the two lateral ventricles at the initial operative session, then he should complete the destruction of the plexuses in one or more secondary operations staged about a week apart, until maximum possible destruction of the plexuses has been accomplished.

Only the most superficial portions of the choroid plexuses need be cauterized; it is entirely unnecessary to cauterize the plexuses in depth. Experience has taught that if the superficial portions of even the larger more foliate parts of the plexuses are cauterized, the deeper portions of the plexuses will completely degenerate and never regenerate. By limiting cauterization to the superficial portions of the plexuses, not only are the chances of bleeding greatly reduced but the risk of inflicting thermal damage to the underlying brain (thalamus) is eliminated.

After undergoing a very radical bilateral cauterization of the plexuses in a single operative session, a patient may develop a temporary but acute postoperative intraventricular hypotension, manifested clinically by a retracted anterior fontanel, severe restlessness, and rising pulse and respiratory rates. This condition should be promptly treated by starting a percutaneous intraventricular infusion of Ringer's solution, from a reservoir maintained about 10 cm above the top of the patient's head. The infusion can usually be entirely discontinued after about 12 hours, by which time the intraventricular pressures will have become naturally stabilized at essentially normal pressures.

Intraventricular bleeding during cauterization of the plexuses is never a serious problem but may be an annoying one. The chances of it occurring are small if the precise technique of cauterization just described is followed rigidly. If bleeding from a small vessel in the choroid plexus does occur, a brief brisk irrigation of the immediate operative field will as a rule quickly expose the bleeding vessel and allow its prompt coagulation. If the bleeding vessel cannot be located and controlled, however, there should be no cause for alarm, for bleeding from vessels of the choroid plexuses will always stop spontaneously and the amount of blood lost from such a vessel is never enough to jeopardize the patient's life nor, indeed, the ultimate successful outcome of the surgical treatment.

Even a small amount of blood diffused throughout the ventricular fluid, however, may seriously impair visibility within the ventricle. When this occurs, it is best to terminate the operation at once, lest the cautery tip tear the choroid plexus and set up other bleeding or even damage the walls of the ventricle and important structures of the brain. Cauterization of the plexuses can be completed at a secondary operation a week later, by which time the blood will have disappeared from the ventricular fluid.

It is futile to attempt cauterization of the plexus in the second ventricle at this operative session if blood has already impaired visibility in the first ventricle, because of the rapidity with which blood passes from the first ventricle into the second. It is for this reason that the dura is not initially opened on the second side of the head but only after successful cauterization of the plexus in the first ventricle has been completed and the intraventricular fluid left free of blood.

Closure of Galea and Skin

The scalp wound on the first side must be closed immediately after cauterization of the choroid plexus on that side has been completed, and before cauterization of the plexus in the second ventricle is undertaken, to minimize loss of cerebrospinal fluid. While this closure is being carried out the ventriculoscope is kept in readiness to proceed to the second side, with of course proper attention being paid to the fact that the eye piece of the lens system has probably been in contact with the surgeon's eyebrow or lashes and hence is contaminated.

The special techniques used in closing the
galea and the superficial layer of the skin to minimize loss of ventricular fluid during closure and development of CSF fistulas after operation are described in the following paragraphs.

After cauterization of the plexus has been completed, the operating element with its connections to the light source, electrosurgical unit, and reservoir of irrigating fluid is removed from the sheath of the ventriculoscope and replaced by the obdurator. Then the long free ends of the individual galeal sutures previously set are collected into two essentially equal bundles and pulled upward, away from the wound, until all the slack in them has been removed (Fig. 11 A). As the tip of the ventriculoscope is removed from the brain, a small catheter connected to the reservoir of irrigating fluid is quickly introduced into the ventricle (Fig. 11 A). As this is done, the long free ends of the galeal sutures are drawn briskly and firmly upward away from the wound; this brings the two edges of the galea into snug approximation to each other and produces a temporary “watertight” closure of the galea (Fig. 11 B). The individual sutures are then tied, one at a time, without need for haste. Just before the last galeal suture is tied, the irrigating catheter is withdrawn from the ventricle (Fig. 11 C). By this technique, the galea can be closed without significant loss of intraventricular fluid or fall in intraventricular pressure.

The superficial skin sutures must be so placed that none impales or even makes direct contact with the more deeply placed galeal sutures. The very great importance of this minor technical detail arises from the fact that every skin suture inevitably becomes infected by the microorganisms constantly present in the skin. But, if the skin suture does not make contact with a more deeply placed (galeal) suture (Fig. 12 left), the skin suture is quickly encapsulated successively by fibrin, granulation tissue, and scar tissue, which prevents the spread of infection from the skin suture into adjacent tissues and provides natural drainage paths for infected exudates to the surface of the skin. When the skin suture is finally removed, the infected tract of the suture quickly closes and heals spontaneously.

If, however, a skin suture impales a galeal suture (Fig. 12 right), the infection involving the skin suture is promptly transmitted to the galeal suture. Here, real trouble is in the making, for the deepest portion of the galeal suture is almost invariably bathed by cerebrospinal fluid coming from the ventricle to the galea through the tract left by the ventriculoscope (Fig. 12 right); the infection from the galeal suture is transmitted directly to the cerebrospinal fluid within the ventricle and results in a massive and potentially fatal infection of the ventricular-subarachnoid system. This grave situation is usually compounded by the development on the 4th or 5th postoperative day of an infected CSF fistula, resulting in a rapid loss of cerebrospinal fluid and collapse of the walls of the ventricle (cortex). Attempts to close the fistula by suture are complicated by infection, and the outcome in these cases is usually fatal.

If by rare good fortune a natural tissue barrier has been preserved between the galeal suture and the ventricular fluid, or if such a barrier has developed before the infection from the skin suture reaches the galeal suture, and if this tissue barrier proves to be strong enough, then the infected exudates from the galeal suture will be prevented from reaching the ventricular fluid. Instead, exudates will be discharged through the sinus tract of the original skin suture to the surface of the skin, thus setting up a chronically discharging suppurating “sinus,” which will persist until the infected galeal suture is spontaneously discharged through the sinus to the surface of the skin. No effort should be made to locate and avulse the infected galeal suture with a small hemostat, for any such effort will almost certainly break open the tenuous tissue barrier which has thus far separated the infected suture from the cerebrospinal fluid of the ventricle.

The extra time spent in the meticulous placement of each individual skin suture is therefore extremely worthwhile; in fact, this may determine the success or failure of the entire operation.

Postoperative Care

The postoperative surgical head dressing should be just big enough to protect the two short scalp incisions, but should leave the anterior fontanel of the patient fully exposed for inspection and palpation by the surgeon,
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Fig. 11. Technique for closing the galea without loss of ventricular fluid. A. The "present" galeal sutures are uncovered and lifted gently upward. The ventriculoscope with obdurator is slowly removed from the ventricle. A small rubber catheter, conducting irrigating fluid (Ringer's solution) from the reservoir, is quickly inserted into the ventricle along the path of the ventriculoscope. B. The preset galeal sutures are pulled briskly upward, bringing the two edges of the galea snugly together to form a temporary watertight closure of the galea while the galeal sutures are being tied. C. The galeal sutures are tied, one at a time, without need for haste. Just before the final suture is tied, the irrigating catheter is withdrawn.
thus enabling him to be kept easily apprised of the exact state of the patient's intracranial (intraventricular) pressure during the immediate postoperative period. A narrow strip of surgical gauze, laid over each of the two scalp incisions and held in place by a single layer of surgical gauze "glued" to the immediately adjacent scalp by collodion, forms an excellent dressing.

In general, it is best to keep the patient flat in bed, since this allows the truest evaluation of the intracranial (intraventricular) pressure. However, if the operation had to be terminated before cauterization of the choroid plexuses was completed, so that intraventricular hypertension remains, then it is well to elevate the head end of the bed to slightly reduce the intraventricular pressure and ease tension on the suture lines. If a very radical bilateral cauterization of the choroid plexuses has been achieved at a single operative session, temporary intraventricular hypotension may become evident a short time after the child has been returned to bed; in such cases it is well to lower the head of the bed 6 to 8 in. Many times, this simple maneuver will correct a mild temporary hypotension.

**Results of Treatment**

My personal experience in the treatment of nonobstructive (communicating) hydrocephalus by endoscopic cauterization of the choroid plexuses is comprised of two series of children.

The first series consisted of the first 20 consecutive, unselected children who came to me with active, verified, nonobstructive hydrocephalus, and were operated upon before World War II, during the years 1934 to 1942. They were reported initially in 1942, with subsequent progress reports in 1952, 1959, 1963, 1965, and 1966. The second series of 19 children were operated on after World War II, between 1946 and 1952. They were reported initially in 1952 with subsequent follow-up reports in 1959, 1963, 1965, and 1966. The results in these two series of cases are summarized in Table 2.

**First Series**

In the first series of 20 children, there were three operative deaths, seven failures to arrest the hydrocephalus, and 10 cases in which the hydrocephalus appeared to have been successfully arrested at the time the ini-
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tial report was made in 1942. Follow-up since 1942 shows that five of the 10 patients originally reported as successful cases have died (at 5, 9, 10, 14, and 15 years after operation) of causes not directly related to their hydrocephalus. Of the five remaining patients living and well when last examined by me in 1965 (at 25, 25, 26, 26, and 27 years after operation), all had completed grammar school (8th grade), one had graduated from public high school (12th grade), and two had successfully completed 1 year of college. In one of these children, this achievement had involved unusual strength of mind and character in view of the fact that this youngster had had a complete paralysis of both legs and bladder since birth as the result of a lumbar myelomeningocele, which had been removed before he was referred to me for the treatment of hydrocephalus.23

Second Series

In the second series of 19 children, there was one hospital death, two failures to arrest the hydrocephalus, and 16 cases classified in my original report18 as “successful” operations. Of these 16 patients, two have died, one 8 years after operation of a traumatic rupture of the spleen, the other 10 years after operation due to overwhelming septicemia of unknown origin; neither child had shown evidence of active hydrocephalus between the time of operation and death. Six of the 16 children were living, with hydrocephalus completely arrested, when I last examined them in 1959 (at 10, 10, 11, 11, 11, and 12 years after operation), but they were severely retarded. Three of the 16 children were active, pleasant, socially well-adjusted youngsters, with hydrocephalus completely arrested, when I last examined them in 1965 (at 16, 17, and 17 years after operation), but they were moderately retarded and unable to advance beyond the third grade.

Five of the 16 children were living and well, with hydrocephalus completely arrested, when I last examined them in 1965 (at 17, 17, 18, 19, and 19 years after operation), and they were of normal intelligence. All five had graduated from high school (12th grade); one had gone on to complete 1 year of college, and another, who had maintained a “B” scholastic standing throughout high school and been a regular member of the school football and baseball teams, was serving his second “tour” of active sea duty in the U.S. Coast Guard after having been promoted.23

Analysis of Both Series

The reduction in the operative mortality rate, from 15% in the first series to 5% in the second series, reflects an increased familiarity with the ventriculoscope, improved operating techniques, and a more critical selection of cases for operation. The neurosurgeon should expect to keep his operative mortality at about 5%.

The increased number of patients with a 5-year survival period (from 50% to 80%) reflects the same factors. The neurosurgeon should expect to achieve approximately 80% permanent “arrests.”

The outstanding feature of this experience has been the strikingly low incidence of late, major complications requiring reoperations or “revisions.” Only one patient (3%) in the two series had to have a late reoperation and this not because of a late complication but merely to complete cauterization of the choroid plexus in the second ventricle. There are two reasons for this low incidence of late complications. First, with this technique there are no tubes or valves to become obstructed, no necessity to substitute longer tubes as the child grows. Second, a choroid plexus once destroyed will never regenerate, so the hydrocephalus will remain permanently arrested.

The high incidence of long-term uncomplicated survivals in both series is undoubtedly the result of the low incidence of late serious complications. In the first series, 50% of all children operated on survived 5 years or longer for an average survival (in 1965) of 18 years; five were alive and well 25 to 27 years after operation. In the second series, 80% of all children operated on survived 8 years or longer, for an average survival (in 1965) of 13 years; five were alive and well 16 to 19 years after operation. Most important is the fact that all of these long-term survivals were achieved without late supplementary operations (“revisions”) once satisfactory bilateral cauterizations of the choroid plexuses had been accomplished.

The intellectual development of the chil-

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children who survived a long time has varied widely. Of the 19 long-term survivors of the two series, three completed 1 year of college, four graduated from high school, and three finished eighth grade, making a total of 10 children (50%) with good or at least satisfactory intellectual development. On the other hand, three children, while attractive and socially well-adjusted youngsters, were moderately retarded and never able to get beyond the third grade in school; and six were severely retarded.

A direct relationship between preoperative cortical thickness and postoperative intellectual attainment could not be established. This, of course, suggests that congenital developmental defects in the microscopic structure of the cortex may play an important, or even principal role in determining the ultimate intellectual attainment of the hydrocephalic child.

References

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