THE SURGICAL TREATMENT OF SPONTANEOUS CEREBROSPINAL RHINORRHEA*

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CEREBROSPINAL RHINORRHEA is the drainage of cerebrospinal fluid through the nose. It is a well-known clinical entity, usually resulting from trauma but which may result from various other causes or even without known cause. The term "spontaneous cerebrospinal rhinorrhea" is very loosely used in the medical literature. The present report is intended to set-up as a distinct group those cases that are truly spontaneous and to report the surgical cure of three such cases.

DEFINITION

Cases of cerebrospinal rhinorrhea have been variously classified by numerous authors, but perhaps the most complete classification is that of Cairns.[1] This author divides the cases into four groups: (1) those resulting from acute head injuries; (2) those occurring as a delayed complication of head injuries; (3) those resulting from nasal operations; and (4) spontaneous cases. He states that most of the cases in his fourth group are due to intracranial tumors but some may be due to congenital anomalies. It is with a subdivision of Cairns' fourth group that this report is concerned. Truly spontaneous or primary cerebrospinal rhinorrhea is the discharge of cerebrospinal fluid through the nose that occurs: (1) in the absence of trauma (acute head injuries, delayed complications of head injuries such as fistulous tracts, operative trauma); (2) in the absence of infection of the bones of the paranasal sinuses (ethmoid caries, sphenoid necrosis, *etc.*); (3) in the absence of tumors eroding the base of the cranium (osteoma, pituitary tumors, meningiomas, *etc.*); (4) in the absence of prolonged increased intracranial pressure (cerebral tumors, congenital or acquired hydrocephalus); and (5) in the absence of demonstrable congenital anomalies (nasal cephalocele, *etc.*). In short, primary cerebrospinal rhinorrhea is the drainage of cerebrospinal fluid through the nose without definite demonstrable cause.

ETIOLOGY

The definition of primary cerebrospinal rhinorrhea given in the preceding paragraph naturally precludes the establishment of a definite etiology for this condition. However, several possible pathways for egress of the fluid from the cranium into the nose have been suggested. Loftus,[2] and Johnston,[3] have suggested that the fluid escapes through the craniopharyngeal canal as rem-

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nants of this structure can be demonstrated in humans at term. Britt,[4] and Locke,[5] have postulated that the olfactory bulb might maintain its embryonic ventricular lumen and a fistula form from this lumen into the nose, probably along the olfactory nerves. Britt[4] also suggests an opening from the basal subarachnoid cistern through the cribriform plate but does not mention the possible cause of such an opening. Nothnagel,[6] and Cairns,[1] suggest that fluid may escape along the sheaths of the olfactory nerves. Johnston[3] mentions the possibility of holes in the cribriform plate that are deprived of nerve fibers. Adson[7] and Aubin[8] suggest congenital defects of the cribriform plate that permit extension of arachnoid along nerve fibers through the cribriform plate.

The most probable pathway for the escape of fluid in the cases of primary cerebrospinal rhinorrhea is along the olfactory nerves. Each olfactory nerve consists of approximately 20 separate filaments which are axons of olfactory cells in the mucous membrane of the nose. These filaments pass through the cribriform plate of the ethmoid *en route* to the olfactory bulb and are held together in bundles by extensions of the three layers of the meninges. The dura mater joins the periosteum of the nose while the pia mater fuses into the neurolemma of the nerves. The arachnoid usually does not extend through the cribriform plate and when it does the extension is very short.

Locke and Naffziger[9] injected celloidin masses under pressure into the subarachnoid spaces of dogs and noted that there was frequent leakage into the nose. Although this finding could not be duplicated in the human cadaver, it seems probable that in the cases reported here there is an extension of the subarachnoid space along the olfactory nerves into the nose.

It is possible that there is some defective development of the cribriform plate in the cases of primary cerebrospinal rhinorrhea. In the embryo the ethmoidal cartilage consists of a mesial mass which extends from the sphenoid to the tip of the nasal process and of paired masses lateral to the olfactory sacs. The fibers of the olfactory nerve pass between the unjoined mesial and lateral masses. Cartilaginous trabeculae later surround the bundles of nerve fibers and interconnect the three masses. The perforated parts of the completed ethmoid ossify and are then known as the cribriform plates.

The causative agent of the rupture of the arachnoid with consequent escape of fluid, however, is not known. Jauregg[10] has suggested that sneezing might suddenly force open such a pathway. Every theory in the literature for spontaneous rhinorrhea postulates increased intracranial pressure. In the cases reported here there was no evidence of increase in intracranial pressure. The patients did not have respiratory infections or hay fever so that increases in pressure from coughing or sneezing could not play a part. Therefore, although the pathway for the escape of cerebrospinal fluid into the nose is thought to be along the olfactory nerves, the reason for the sudden onset of the rhinorrhea is still unknown.

Although reports of cases of cerebrospinal rhinorrhea abound in the literature, very few of the cases can be properly included in the group here reported. In the reports of Cairns,[1] German,[11] Eagleton,[12] and many

others,[32, 33] there is a definite history of trauma as the etiologic agent of the rhinorrhea. Ten of the eleven cases of Dandy[13] had histories of trauma or occurred postoperatively. Campbell,[14] Learmonth[15] and Donnelly[16] have reported cases following nasal surgery. Som and Kramer,[17] Adson,[7] and others, have reported tumors eroding the floor of the skull with subsequent rhinorrhea. Locke,[5] Meyer,[18] Cushing,[19] and others, have reported rhinorrhea in cases of intracranial neoplasms causing increased intracranial pressure. Aubin[8] reports minor congenital defects at the cribriform plate. None of these cases fall into the category of truly spontaneous or primary cerebrospinal rhinorrhea set-up in the present report. The great majority of cases reported either have a known etiology or the reports are too meager for proper classification. Of the cases reported by St. Clair Thomson[20] only his own case is definitely one of primary rhinorrhea. The remaining cases reviewed had either a definite etiology or insufficient evidence for classification. Johnston[3] reported a case and reviewed the literature on rhinorrhea through 1920. Although some of these cases are almost surely primary cerebrospinal rhinorrhea, the case reports, for the most part, are too inadequate for definite classification. The cases of primary cerebrospinal rhinorrhea found in the literature since 1920 are found in Table I. The propriety of including the case of Adson[7] might well be questioned, as a definite dural defect was found. However, the defect actually was a minor enlargement of the normal opening around an olfactory nerve and as the lesion in the cases here reported is thought to be around the olfactory nerves, it is thought that this case should be included. Two of the cases reviewed showed hyperostosis frontalis interna by roentgenologic examination, but this may have been a coincidental finding. At any rate, the cases are included with reservation.

DIAGNOSIS

Diagnosis of cerebrospinal rhinorrhea is not difficult. Clear fluid drips from the nose almost constantly and if the diagnosis is in doubt, the fluid can be examined by chemical means and shown to be cerebrospinal fluid. Dye can be injected into the lumbar subarachnoid sac and recovered from the nose, as was done by Fox,[21] and in one of the cases in the present report. This test is also of value in determining the side of the leak, a matter of great importance. After it is established that the fluid is cerebrospinal fluid, it is perhaps best to follow the dictum of Locke[2] and consider each case as a "brain tumor suspect." Careful history and examination, including roentgenologic studies, will decide whether or not tumor or any increased intracranial pressure plays a part in the etiology. Trauma or operative procedures can be evaluated by the history. Infections of the sinuses and osteomyelitis of the skull can be diagnosed by examination and roentgen studies. Congenital anomalies, likewise, will usually be discovered by adequate study. If all of the above conditions have been discarded by clinical studies, the case must be considered as one of primary cerebrospinal rhinorrhea.

TREATMENT

There is no unanimity of opinion as to the proper treatment of cerebrospinal rhinorrhea. The early writers, such as Loftus[2] in 1923, felt that any treatment was definitely contraindicated. Feinblatt and Damrau[22] in 1934,

TABLE I

Author	Age and Sex	Duration of Rhinorrhea	Side Affected	Mode of Onset	Treatment	Remarks	Results
Adson[7] 1941	31 Female	9.5 mos.	Not stated		Craniotomy. Opening 2 mm. in diameter around olfactory nerve. All studies negative.	None	Cure immediately
Friedberg & Galloway[24] 1938	38 Female	4 mos.	Right	Followed severe respiratory infection	Intranasal 20% silver nitrate	All studies negative	Flow ceased in 12 days
Plum[27] 1931	45 Female	18 yrs.	Not stated		Three nasal operations	Had severe headaches. Encephalogram revealed cortical atrophy	Flow has diminished but continues
Fox[21] 1933	33 Female	4 mos.	Left	Followed severe respiratory infection	Intranasal 20% silver nitrate	All studies negative	Flow ceased in 4 mos.
Wessels[26] 1939	49 Female	1 yr.	Left	No definite precipitating factor	Craniotomy. Depression in anterior fossa	Evidence of old papilledema; encephalogram was negative	Cure immediately.
Ballon & Ballon[28] 1937	53 Male	1 yr.	Right	Followed severe respiratory infection	None	Positive WaR. All other studies negative	Flow ceased in 5 mos.
Titche[29] 1941	50 Female	Several mos.	Left	No definite precipitating factor	Told to use silver protein in nose	X-rays revealed hyperostosis frontalis interna	Flow continues
Jobson[23] 1941	50 Female	2 mos.	Right	Followed sneezing	Intranasal 10% silver nitrate	All studies negative	Flow ceased in 1 day. Recurred 1 year later. Stopped after similar treatment
Dandy[13] 1944	39 Male	4 mos.	Not noted	No definite precipitating factors	Craniotomy. Both sides explored	All studies negative	Flow continues
Wurster[30] 1937	29 Female	4 yrs.	Right	Followed severe respiratory infection	Argyrol tampons, mild silver protein solution	All studies negative	Flow ceased in 3 years
Feinblatt & Damrau[22] 1934	59 Female	6 wks.	Not stated	No definite precipitating factor	Rest in bed	All studies negative	Flow ceased in 3 weeks
Ameriso[31] 1942	44 Female	15 days	Right	No definite precipitating factor	None	X-rays revealed hyperostosis frontalis interna	Flow continues

felt that any nasal or surgical treatment was contraindicated. However, the extreme danger to life of cerebrospinal rhinorrhea is generally recognized. Certainly, most of the patients with such a leak will eventually develop meningitis, even in spite of modern drug therapy. Rhinologists have tended to treat

cerebrospinal rhinorrhea by intranasal medication and Fox,[21] Jobson,[23] and Friedberg and Galloway[24] have reported cures following the use of silver nitrate, which is painted around the middle turbinate. This method of therapy causes an intense reaction and is open to the objection that intranasal manipulation might well precipitate a meningitis. In addition, intranasal therapy does not attack the rhinorrhea at its most likely source—the cribriform plate. Intracranial operative intervention is the method of choice in the treatment of this condition as modern neurosurgical methods and chemotherapy have largely dispensed with the old objections of danger of meningitis and operative mortality. The exact operative procedure to be followed is still a matter of some debate. Almost all of the surgical methods reported have been directed towards the closing of traumatic fistulae. Cairns[1] employs direct suture of a dural defect or the use of fascia, which has also been advocated by Dandy,[13] Peet[25] and Learmonth[15] have placed iodoform gauze beneath dural defects, while German[11] has turned down small dural flaps from the covering of the crista galli. Graham[34] plugs the opening in the bone with wax. Adson[7] uses a bilateral bone flap and sacrifices both olfactory nerves in order to overlap dura around the fistula and interposes muscle in the suture line. All of the above methods, particularly those using an extradural approach, are open to the objection that the dura over the cribriform plate is very thin and adherent.

The method of surgical treatment advocated here postulates that the leakage of fluid is along the olfactory nerves. In none of the three cases was a dural or bony defect seen but the method of treatment outlined as follows produced immediate cures in all cases with no recurrences. A small frontal osteoplastic flap is reflected on the side of the leak, the skin incision being entirely behind the hair line. The dura is opened around the edges of the bony opening and the frontal lobe retracted (Fig. 1). In no case was there any evidence of pressure and it was not necessary to tap the ventricles, ample space for exploration being secured by emptying the basal cisternae. Careful exploration for tumor or congenital defect is then carried out, such exploration being negative in the cases here reported. The filaments of the olfactory nerve are then pulled out of the cribriform plate and a piece of muscle inserted into the openings in the cribriform plate (Fig. 2). The frontal lobe is then allowed to fall back over this area and the dura is tightly closed with interrupted silk sutures. The bone flap is replaced and the skin closed in usual fashion. In all of the three cases here reported the cessation of leakage of cerebrospinal fluid has been immediate and there have been no recurrences. An essentially similar method of attack was used successfully by Sachs in a case reported by Wessels,[26] and also by Klein[8] in two cases with congenital defects at the cribriform plate. The surgical attack and repair should be entirely intradural.

CASE REPORTS

Case 1.—A female, age 40, white, was admitted to Medical College of Virginia Hospital, March 29, 1943, with complaint of fluid running from her nose. Family and past histories noncontributory. Present illness began two months before admission, when

clear fluid began to drip from the left nostril at the rate of five to six drops every half hour. No history of trauma or upper respiratory infection. No headaches, convulsions or evidence of any cerebral involvement. Physical examination on admission was completely negative except for clear fluid dripping from the left nostril. Blood pressure 124/90. Neurologic examination was completely negative. Roentgenologic studies revealed the sella turcica to be normal in size, with the posterior clinoids somewhat thin. There was clouding of the left antrum and right frontal sinus. No roentgenographic evidence of increased intracranial pressure was present. Lumbar puncture revealed an initial pressure of 110 mm. water, with clear fluid. No cells found. Protein normal. Wassermann reaction negative. Blood and urinalysis entirely normal. Sulfadiazine therapy was

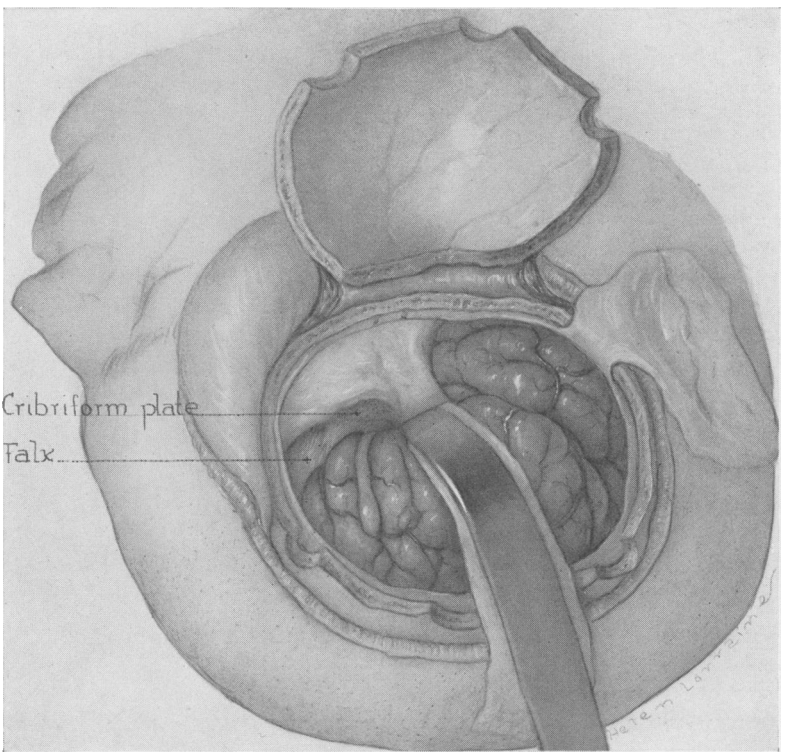

FIG. 1.—Operative exposure of cribriform plate.

instituted. Following lumbar puncture the cerebrospinal fluid ceased to flow. Patient discharged on April 5, 1943. Readmitted April 19, 1943. Eleven days after discharge from the hospital, April 16, fluid again began to drip from left nostril. Patient also complained of severe headache. General and neurologic examinations, again, completely negative. Visual fields full and normal. On April 27 lumbar puncture was done and 1 cc. indigo carmine injected. Fluid from nose was colored blue within 15 minutes. Dry cotton tampons were placed in lateral and superior nasal crevices. Repeated tests showed only the cotton in region of cribriform plate to be discolored. Left frontal craniotomy on April 29, 1943. Technic is outlined in text. No pathology found around sella turcica. The depression of the olfactory bulb seemed a little deep. Cribriform plate sealed-over with

muscle. No drainage of fluid following operation. Patient has continued well in every way (3.5 years postoperative).*

Case 2.—A female, age 50, white, was admitted to Medical College of Virginia Hospital, April 30, 1944, with complaint of watery discharge from left nostril. Family and past histories noncontributory. Present illness began four weeks before admission, with onset of drainage of clear fluid from left nostril. No headache or visual disturbance.

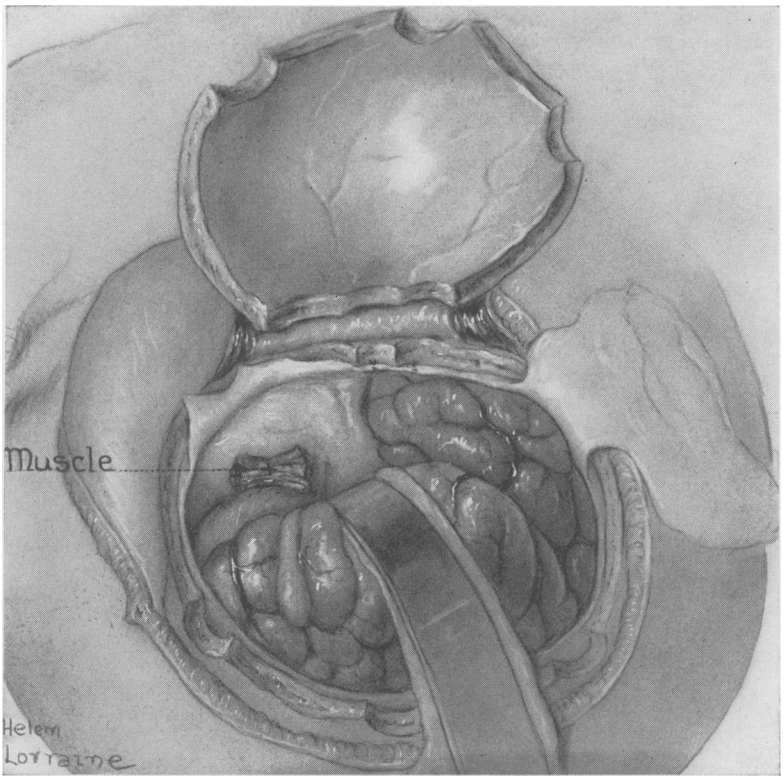

FIG. 2.—Operative exposure illustrating muscle inserted into openings in cribriform plate.

No upper respiratory infection and no history of trauma. Drainage of fluid was constant. General physical examination revealed only drainage of clear fluid from left nostril. Blood pressure 110/80. Neurologic examination was completely negative. Routine blood and urine studies entirely negative. Roentgenologic examination of the skull was entirely negative, the sella turcica being normal in size and not eroded. Ethmoid cells were clear. Rhinologic examination revealed only deflected septum on the left, with deep spur in posterior aspect of middle mentus. Dry cotton localization revealed that fluid was coming from superior nasal space between the middle turbinate and the septum on the left. No anosmia. Operation, May 3, 1944, by technic detailed in text. Operative findings completely negative. Region of left cribriform plate sealed-over with muscle. No further

* See footnote on opposite page.

cerebrospinal rhinorrhea. Discharged May 25, 1944. Patient seen August 22, 1946. Perfectly well (2.25 years postoperative).*

Case 3.—A female, age 50, white, was admitted to Medical College of Virginia Hospital, October 24, 1944, with complaint of drainage of clear fluid from right nostril. Family and past histories were completely negative. Present illness began about two weeks before admission when clear fluid was noted dripping from the right nostril. No upper respiratory infection and no history of trauma. No sneezing. Eleven days after onset of rhinorrhea patient had sudden onset of severe headache and felt that drainage of fluid became more profuse. General physical examination completely negative except for drainage of clear fluid from right nostril. Neurologic examination completely negative. Blood and urine entirely normal by all laboratory studies. Roentgenograms of skull entirely normal. Right craniotomy, October 24, 1944, according to technic detailed in text. No pathology found. Region of cribriform plate sealed-off with muscle. No leakage of fluid following operation. Patient discharged November 10, 1944. October 24, 1946, patient stated that she is perfectly well in every way. (Two years postoperative).

DISCUSSION.—The present report is an attempt to establish as a distinct clinical group those cases of cerebrospinal rhinorrhea that occur without definite demonstrable cause. Although there are innumerable reports of cases of rhinorrhea in the literature, only a few of the cases fall into this category. Undoubtedly, the condition occurs much more frequently than the number of reports indicates, but it seems probable that it is relatively rare. It is desirable to define and segregate these cases as a definite clinical group. The diagnosis, at least for the present, is one of exclusion, as the cases fall into this group only when all demonstrable etiologic agents have been excluded.

A review of the literature reveals 12 cases that fall into the group identified in this report. The great majority of cases have been excluded because of a history of trauma, nasal operations or definite evidence of intracranial tumor. In other instances the cases are too inadequately reported to be sure that they are truly cases of cerebrospinal rhinorrhea or that an intracranial tumor has been eliminated. Five of the cases previously reported had upper respiratory infections either at or immediately before the onset of the rhinorrhea. In none of the cases in this report was there any evidence of such infection. The presence of respiratory infections might well be of some importance because of the suggestion of Werner Jauregg that sneezing might precipitate cerebrospinal rhinorrhea. That such incidents are not the sole precipitating agents is shown, however, by the three cases reported here, in none of which was sneezing a factor.

The etiology of this condition remains in doubt, but it seems most probable that the cerebrospinal fluid escapes along the olfactory nerves. Such a conclusion is given added weight by the cure of the three present cases by intradural occlusion of the points of exit of these nerves with muscle. Very minor congenital anomalies in the region of the cribriform plate can produce openings around the olfactory filaments which allow the arachnoid to extend along the nerves into the nasal cavity. The cause of the rupture of the arachnoid is not

* Cases 1 and 2 were referred by Dr. Roderick MacDonald, of Rock Hill, South Carolina, who reported them in a thesis for the American Laryngological, Rhinological and Otological Society.

as yet determined, however. A marked increase in intracranial pressure could cause the rupture, but in none of the cases in this report was there any evidence of even temporary increased pressure.

Diagnosis of cerebrospinal rhinorrhea is usually not difficult. As mentioned before, it is important to eliminate all possible etiologic agents before the case is considered to be one of primary cerebrospinal rhinorrhea. It is then important to determine the side of the leak, and this can usually be done by inspection of the nasal cavity. Increased certainty is added by the injection of dye into the lumbar canal and viewing its exit into the nose.

Primary cerebrospinal rhinorrhea is an entity that requires prompt treatment. Although some cases have been reported of long duration, they are certainly exceptions to the usual course. The logical point of attack is at the cribriform plate, as this is the almost certain seat of the underlying pathology. The various methods of surgical approach have been reviewed in the text and the authors' method detailed. By this method the presumed seat of pathology is directly attacked and only one olfactory nerve is sacrificed. The efficacy of this method of attack is attested by the results obtained in three cases.

SUMMARY

1. A group of cases exhibiting cerebrospinal rhinorrhea without demonstrable cause is defined as primary cerebrospinal rhinorrhea and set-up as a clinical entity.

2. The literature is reviewed and 12 cases tabulated as probably belonging to this group.

3. Possible sites for escape of cerebrospinal fluid into the nose are discussed and the probable site suggested.

4. A surgical treatment for this condition is detailed, and three cured cases reported.

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