THE DIAGNOSIS AND TREATMENT OF HYDROCEPHALUS DUE TO OCCLUSIONS OF THE FORAMINA OF MAGENDIE AND LUSCHKA

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THE DIAGNOSIS AND TREATMENT OF HYDROCEPHALUS DUE TO OCCLUSIONS OF THE FORAMINA OF MAGENDE AND LUSCHKA

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EVERY case of hydrocephalus has a specific cause which can and should be located by clinical tests during life. In a great many instances this cause is easy of correction by operation with a resultant cure of the disease. I realize that this is a very sweeping statement concerning a disease which has been considered idiopathic and for which no treatment has been successful. The purpose of this paper is to describe a group of cases of hydrocephalus caused by closure of the foramina of Luschka and Magendie, to show the pathology by postmortem specimens, to describe the means by which it can be diagnosed clinically, and particularly to describe an operative procedure which will produce its cure. In previous papers,1 I have described the pathology, methods of diagnosis, and operative procedures for the treatment of other types of hydrocephalus. In this type of hydrocephalus, the treatment is ideal in that the cause can be attacked directly and with every prospect of permanency. It is one of the most common of the types of congenital hydrocephalus, being surpassed in frequency probably only by the group of cases with an obstruction at the aqueduct of Sylvius. To be effective, the treatment must be applied in the early stages of the disease.

A clear conception of the anatomy of the cerebrospinal spaces, within and without the brain, is an absolute prerequisite to the introduction of a successful method of treatment. It must be appreciated that the normal equilibrium of cerebrospinal fluid is absolutely dependent upon communication between the ventricles of the brain and the subarachnoid spaces. In the ventricles, cerebrofluid is produced; in the subarachnoid space, it is absorbed. The balance between the formation and the absorption of cerebrospinal fluid is maintained solely by three openings which connect the fourth ventricle with the cisterna magna. These openings are the paired foramina of Luschka and the median foramen of Magendie. These openings are neither myths nor artefacts. Although they are now incorporated in the modern text books of anatomy, there is a very uncertain attitude toward their acceptance by many authorities, both anatomical and surgical, and their demonstration is usually even more indefinite. By many, they are looked upon as "functional" rather than anatomical openings. There can be no reason for this hesitancy in their acceptance and no basis for a controversy as to their exact nature. They are just as definite and as precisely outlined as the foramen of Monro or the aqueduct of Sylvius. Nor is this an academic discussion. It is one of the greatest practical import, for surely no neurological surgeon can be competent to make a differential diagnosis of lesions of the cerebellar region, and far less to perform cerebellar operations, who has not a perfect understanding of the importance of the foramen of Magendie and who does not know the foramen when he sees it and who does not expose it at every cerebellar operation in which the exploration has not disclosed the lesion. To those who do not question the existence of the foramen of Magendie, these statements, doubtless, seem superfluous, but constantly the question is asked by men of authority: Is the foramen of Magendie a real opening, or is it not an artefact produced by dissection of the delicate membranes? The answers to this query are many and admit of no equivocation.

An anatomical dissection after death shows the three foramina, through each of which a probe can easily be passed without injury to any membranes. The foramen of Magendie is exposed at most cerebellar operations and

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through it the floor of the fourth ventricle is always evident. The three openings can easily be demonstrated at a postmortem examination (without any dissection) by cutting the mid-brain transversely and injecting (without pressure) into the aqueduct of Sylvius a colored solution; the color will pass from the fourth ventricle to the exterior through the foramina of Luschka and Magendie, but through no other openings.

ARE THE FORAMINA OF LUSCHKA AND MAGEN-DIE NECESSARY?

The whole problem of hydrocephalus hinges on this question. As early as 1790, the renowned anatomist, Monro, the discoverer of the foramen of Monro, denied any communication between the ventricles and the subarachnoid space. A curious defect in his argument was that, in a case of hydrocephalus, there were no openings and the ventricular system was intact. So it was in his case, for he missed the cause of the hydrocephalus—the closure of the foramina at the base. Magendie described the foramen which bears his name, in 1825. It seems incredible that, at this late date, a large part of his epoch-making contribution should have been devoted to proving that the cerebrospinal system contained fluid and not air or vapor. His foramen of Magendie was received with skepticism and open hostility. Renauld, 1829, found the foramen of Magendie absent in the horse and dog, and his observations were even confirmed by Magendie, though the latter still thought his foramen important in man. Magendie's explanation of the function of the foramen was not impressive and was incorrect in the light of our present knowledge. He assumed the cerebrospinal fluid to form in the pia and thought it passed upward through his new foramen into the ventricles of the brain. And when he found a case of hydrocephalus (1842) in which the foramen of Magendie was closed, a satisfactory explanation was not forthcoming, though he still thought in some way the closure of the foramen to be the cause of the disease, as it undoubtedly was. Monro had the cause of hydrocephalus before him, but missed it because of his eagerness to prove a theory. Magendie maintained confidence in his anatomical findings, in spite of the conflict with his theories, and should receive credit for the first case of hydrocephalus in which the described pathological findings were the cause of the disease.

Krause (1843) and Todd (1847) described the foramen of Magendie as an artefact produced either by pressure or tearing of the tissues. Virchow (1854) very strongly disclaimed the existence of a foramen of Magendie or any other open communication between the ventricles and the subarachnoid space. With Virchow's strong opposition it is but natural that Magendie's foramen should by that time have been thoroughly discredited. But another discovery of the greatest importance appeared in the same year as Virchow's publication, and gradually the prevailing but erroneous views have been transformed. Luschka discovered a foramen on each side of the fourth ventricle at the point of emergence of the flocculus from the fourth ventricle. He also confirmed Magendie's discovery of a mesial foramen, thus making three foramina which established communication between the ventricles and the subarachnoid space (the cisterna magna). The foramina of Luschka were found to be constantly present in man and all mammals. The absence of the foramen of Magendie in the horse and dog is immaterial because the two foramina of Luschka are ample to assume its function. Nature has made in man the foramen of Magendie as an additional safeguard against possible closure of the fourth ventricle; this is in marked contrast to the absence of such safeguards in case of closure of the aqueduct of Sylvius. Key and Retzius (1875) and Retzius (1896) confirmed the presence of the foramina of Luschka in examinations of many brains. They found the foramen of Magendie absent in two of one hundred cases examined and the foramina of Luschka closed in three instances. No mention is made whether the three foramina were absent in the same brains, nor of the incidence of hydrocephalus in the brains examined. Cannieu (1898), however, made similar examinations and looked upon all the openings as artefacts and considered the ventricles a closed system and everywhere lined by
epithelium. Testut and Schmorl, also, shared this view.

In recent communications I have shown the absolute necessity of communication between the ventricles and the subarachnoid space. I have shown by experiments on animals that cerebrospinal fluid is formed from the choroid plexus which are located in each of the four ventricles, and that in no part of the entire ventricular system is there any appreciable absorption. It is for these reasons that the foramina of Luschka and Magendie are necessary. They permit the fluid to pass to the absorbing area of the brain—the subarachnoid space. The normal ventricular pathway for cerebrospinal fluid is from each lateral ventricle through its foramen of Monro into the third ventricle, thence through the aqueduct of Sylvius into the fourth ventricle and from there into the cisterna magna through the foramina of Magendie and Luschka. There are no other openings which allow communication between the ventricles and the subarachnoid space; all other openings which have been described are artefacts; nor has it been possible to establish artificial openings in other parts of the brain to replace these openings when they become closed, except by the operation which will be proposed; here alone, the anatomical conditions are such as to favor the restoration of these openings.

From another viewpoint, the foramina of Luschka and Magendie can be shown to be indispensable. Closure of these three foramina in the same brain will, without exception, result in hydrocephalus. This we have shown experimentally by producing hydrocephalus in dogs; and clinically it has been observed in a number of patients, in many of whom postmortem examinations have been made.

It is entirely possible, in fact it frequently happens, that one or even two of the three foramina at the base may be occluded and the remaining opening is adequate to allow the ventricular fluid to pass from the ventricles into the subarachnoid space. On the other hand, it does not mean that if one, or even all, of these foramina are open, hydrocephalus may not result. In the communicating type of hydrocephalus precisely this condition is present, but the hydrocephalus is due to an entirely different cause—the obliteration of the cisternae or their branches. The inflammatory process which seals the cisternae or their branches usually obliterates one or two of the three foramina, though not infrequently all three openings are closed, in which case the hydrocephalus becomes obstructive.

THE PATHOLOGY OF OCCLUSIONS OF THE FORAMINA OF MAGENDIE AND LUSCHKA

There are, apparently, two types of occlusions of the foramina of Luschka and Magendie: One which occurs congenitally and is well advanced at birth, the second which occurs at all ages. The latter always follows some form of meningitis; the former may or may not be the result of an intra-uterine inflammation.

In the cases which are apparently of congenital origin, the characteristic large head of hydrocephalus is usually noticed about the second or third month, though, of course, the brain is largely destroyed by this time, showing that the disease has existed for a long time,—undoubtedly far back into intra-uterine life. I have seen four of these cases at postmortem examination; in all, the time of recognition of the hydrocephalus was approximately the same and, therefore, doubtless the disease arose at about the same period of fœtal life. In each of these cases, the aqueduct of Sylvius was treble its normal size, the fourth ventricle greatly enlarged, though there was great difference in the actual size of the fourth ventricle and the shape and size of the cerebellum. In two cases, the vermis was absent (Figs. 2 and 3), the two lateral lobes of the cerebellum were small nubbins, laterally placed in the angles of the posterior cranial fossæ and connected by a broad expanse of a rather thin, transparent membrane which, in the absence of the vermis, was now the roof of the fourth ventricle. This membrane was fastened tightly to the lateral margins of the medulla and pons and

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everywhere the fourth ventricle was hermetically sealed, giving it the appearance of a huge cyst. This cyst was not adherent to the dura and there were no signs of any pre-existing inflammatory process in the posterior cranial fossa. The third and fourth cases differed from this description only in degree; in each the vermis was present, the fourth ventricle was a large cyst everywhere covered by cerebellum and hermetically sealed. One lateral pouch (lateral recess) was very transparent and extremely thin, but everywhere intact; this pouch was really a secondary bulging cyst which had protruded through a ring of denser tissue which corresponded with foramen of Luschka. In these cases no traces of a pre-existing meningitis were seen.

Recently, in all postmortem examinations of cases of hydrocephalus, we have injected a colored solution into the spinal canal to observe its distribution in the subarachnoid space and in the ventricles. In two of these cases (with occlusion of the basal foramina) the solution surrounded the entire brain but did not enter the large dilated fourth ventricle, proving conclusively that the obstruction was complete and the fourth ventricle, therefore, devoid of all communication with the subarachnoid space. One can pass a probe into the regions in which the foramina are normally present and one meets a bulging but closed pouch instead of the usual normal opening. Again, colored solutions injected into the fourth ventricle fail to reach the exterior as in the normal brain.

If one holds the specimen before a light and looks through the enlarged aqueduct of Sylvius into the fourth ventricle, the transparent membrane can be beautifully seen surrounding the margins of the medulla and pons and stretching upward across a space about 1 centimeter wide before attaching itself to the inferior margin of the cerebellum. If the brain is carefully removed, the arachnoid membrane, which forms the outer layer of the cisterna magna, can be dissected from the base of the brain, exposing the cystic membrane which forms the sides of the fourth ventricle and is entirely distinct from the pia-arachnoid. The presence of this delicate arachnoid tissue, not adherent or only slightly adherent to the contiguous brain tissue, may be looked upon as evidence against an old inflammatory process, at least of a severe grade.

We know that the foramina of Magendie and Luschka are secondary openings in a primary, closed, ventricular system. Blake¹ and Heuser² have shown these foramina to

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¹ Blake, J. A. The roof and lateral recesses of the fourth ventricle. J. Comp. Neurol., 1909, x.
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Fig. 2. Advanced hydrocephalus resulting from complete closure of the foramina of Magendi and Luschka. All the ventricles are dilated up to the point of obstruction. Owing to the absence of adhesions, to the absence of the vermis of the cerebellum, together with the presence of hydrocephalus at birth, we have looked upon the occlusion of these foramina to have been due to their failure to develop rather than to a secondary closure. ! Where foramen of Luschka should be; it is closed. m, Where foramen of Magendie should be; it is closed.

Develop in the pig embryo by a gradual thinning of the wall of the ventricle. Owing to the early period of onset of the hydrocephalus in utero, and to the absence of adhesions, I am led to believe that this type of hydrocephalus results from the failure of these foramina to develop, rather than from a secondary closure after their development. On the other hand, the same picture may result from a very mild inflammatory process which has sealed these foramina and has left few, if any, additional traces such as adhesions in its wake. It is not impossible that some very mild type of inflammation may seal these openings, though such an explanation certainly seems less probable.

There is a second group of these occlusions of the basal foramina, which occurs in infants, in which an old inflammatory process is everywhere manifest. We have seen three cases of this kind at necropsy and one at operation. The base of the brain is then sealed to the dura, over areas of considerable extent; the meninges are thickened and tough, and the contiguous brain itself is usually thickened and rigid. The cisterna magna and the other cisternae are usually obliterated by adhesions. The foramina of Luschka and Magendie are occluded either completely or almost completely. The floor of the fourth ventricle also shows marked evidences of the inflammation, ruge of scar tissue are present, and the size of the medulla and pons is increased by the inflammatory tissue. Two of these cases have followed a definite meningitis. In the third case, hydrocephalus was undoubtedly present at birth; the delivery was difficult and the head was unusually large; alburn myelomeningocele was also present. In this latter case, doubtless, an inflammation had been present early in intra-uterine life. This is evident by the presence of the meningocele and by the high grade of hydrocephalus which was manifest soon after birth. In a fourth case, a dorsal meningocele was present at birth and hydrocephalus was observed 2 months after birth. A dense scar covered the foramen of Magendie and the contiguous region. The cerebellum was firmly bound to the medulla and pons, there being no bulging fourth ventricle between. A large cerebellar hernia projected into the spinal canal. The pia-arachnoid was adherent to the dura over most of the area exposed at operation. Undoubtedly, an intra-uterine inflammatory process had produced both the hydrocephalus and the meningocele. In all of the cases with marked evidences of an old inflammatory process, there is a striking difference from those of the non-inflammatory type. Following the inflammatory process, the cerebellum is usually, but not always, firmly bound to the border of the medulla and pons, thus effectually preventing the fourth ventricle from bulging as a cyst between these structures, as occurs in the noninflammatory type. The difference between a total and a partial occlusion of the foramina of Luschka and Magendie is one which is readily made at necropsy and just as easily by a clinical test, and is all-important in the matter of treatment of the individual case.
Fig. 3. Sagittal view of same brain. The entire ventricular system is dilated, the fourth ventricle particularly so. Had the obstruction been at the aqueduct of Sylvius, the fourth ventricle would not be enlarged.

A group of two adult cases completes the pathological picture of occlusions at the basal foramina. The pathology of these cases was seen at operation and not at necropsy. At operation, the foramina of Luschka can be seen directly only with difficulty, but the examination of the foramen of Magendie gives all the necessary information. If the foramen of Magendie is patent, hydrocephalus will not result even should both foramina of Luschka be closed. On the other hand, if the foramen of Magendie is closed, hydrocephalus will result only if both foramina of Luschka are also occluded. But, given a dilated fourth ventricle and a closed foramen of Magendie, the closure of this foramen is always the cause of the hydrocephalus. On the other hand, given a normal fourth ventricle and a closed foramen of Magendie, the closure would not be significant. The latter stipulation, however, is only of theoretical value for there would be no symptom requiring operation.

In one of the two adult cases, a dense scar at the foramen of Magendie (Fig. 7) was the only evidence of a pre-existing inflammation, but this evidence was indisputable. In this young man, who was 19 years old, the pathological features differed from those of the congenital type of occlusion of these foramina only in the presence of this scar; the cystic fourth ventricle bulged on each side between the cerebellum and medulla which were separated by the pressure of the accumulated fluid. The scarred foramen of Magendie was sufficiently extensive and rigid to retain the cerebellum in the posterior cranial chamber and prevent the protrusion of the lobes through the foramen magnum into the spinal canal (cerebellar hernia). In the second adult case, the fourth ventricle was of tremendous size and both lobes of the cerebellum had
photograph of man who has had two useless cranial operations performed. His symptoms resembled closely those of a cerebellopontine tumor. A postinflammatory occlusion of the foramina of Luschka and Magendie was found at operation and relieved by construction of a new foramen of Magendie.

herniated far into the spinal canal. The foramen of Magendie was tightly sealed by adhesions which, however, were not present over the lobes of the cerebellum. The cerebellum and medulla were also tightly adherent, thus effectually preventing the fourth ventricle from bulging between these structures. The operative findings of these two cases were almost exactly like that of the infant in whom an intra-uterine inflammation had closed the foramina of Luschka and Magendie.

To sum up the pathology of these nine cases, the fundamental features were always similar. The foramina of Luschka and Magendie were closed. The absence or presence of the vermis, the position of the cerebellar lobes, the presence or absence of the bulging fourth ventricle between the inferior surface of the cerebellum and the medulla, the presence or absence of a cerebellar hernia into the spinal canal were all dependent upon local differences in type or extent of the causative lesion and, doubtless, also upon the time at which the lesion developed. In 4 cases, the cause of hydrocephalus is presumed to be a failure of the foramina of Magendie and Luschka to develop, though a mild intra-uterine inflammation can not be excluded. In 2 cases, a definite inflammatory process in infancy caused the condition, and in a third case, a definite intra-uterine inflammation had been the cause. Two adult cases were caused by a mild meningitis which, clinically, was not recognized as such but the pathological findings needed no clinical confirmation.

THE DIAGNOSIS OF OCCLUSIONS OF THE FORAMINA OF LUSCHKA AND MAGENDIE

Since occlusions of the foramina of Luschka and Magendie occur at varying periods of life, I shall divide the consideration of the clinical manifestations and the differential diagnosis into the period of infancy and of later life. Seven of our cases occurred in infancy and two in adult life. In infancy, the clinical picture is that of hydrocephalus; in adult life, the clinical picture is hard to differentiate from that of a cerebellar tumor. Infants, of course, give none of the cerebellar signs such as ataxia, adiadochokinesia, nystagmus, Romberg, and disturbances of gait; the signs are purely those of increased pressure of the intraventricular fluid and this is shown solely by enlargement of the head and separation of the sutures. At this early age, an enlargement of the head is almost pathognomic of hydrocephalus. But, as hydrocephalus is a symptom and not a cause, there is nothing about the enlargement of the head which denotes the location of the cause, i.e., the obstruction.

There is no clinical picture which is distinctive of any particular type of hydrocephalus, at least none is recognized as yet. It is only by the tests which have been recently introduced that we are able to determine the location of the obstruction which causes the hydrocephalus. In a recent publication, we have shown how lesions of the aqueduct of Sylvius may be localized. The localization of occlusions of the foramina of Magendie and Luschka is made by the same methods. It is first necessary to determine that hy-

1 Surg., Gynec. and Obst., 1920, xxxi, 340.
drocephalus is not of the communicating type but is due to an obstruction in the ventricular system. This is done by the indigo-carmine test; in obstructive hydrocephalus, the color does not appear in the spinal canal after its injection into a lateral ventricle. There are two locations at which the obstruction can exist: (1) the aqueduct of Sylvius or (2) the foramina of Luschka and Magendie (an obstruction at the foramen of Monro must also be thought of, but its incidence is rare). One can, at times, get a fair indication of the position of the obstruction by the amount of cerebrospinal fluid which can be obtained by lumbar puncture but, at best, such deductions are most capricious. In occlusions of the aqueduct of Sylvius, several cubic centimeters of fluid may be obtained by a simple tapping and by aspiration, whereas in occlusions of the foramina of Magendie and Luschka, usually only a few drops will be obtained and little, if any, more can be aspirated. The reason for this difference is that, in lesion of the basal foramina, the foramen magnum is well plugged by a cerebellar hernia or by the dilated ventricle without a hernia. But this differentiation is not always so simple, for at times only a small amount of fluid is obtained when the obstruction is at the aqueduct of Sylvius, owing to the foraminal herniation of the cerebellum. An absolute determination of the precise location of the obstruction can be made only by the use of ventriculography. Following a complete removal of the ventricular fluid and the substitution of air, the latter will reach the point of the obstruction but can not pass beyond. If the aqueduct of Sylvius is obstructed, the third ventricle will be clearly shown but no air will reach the fourth ventricle. If, in hydrocephalus, the fourth ventricle and aqueduct of Sylvius are filled with air, both will be enlarged and the boundaries of each will be sharply defined. Such findings will eliminate an obstruction at the aqueduct of Sylvius and place the obstruction at the foramina of Luschka and Magendie, provided, of course, an obstructive hydrocephalus has been demonstrated by the indigocarmine or phenolsulphophthalein tests. In obstructions at the foramina of Luschka and Magendie, the air will not fill the cisterna magna, whereas in communicating hydrocephalus, the cisterna magna will not only contain air but will usually be considerably enlarged.

The clinical diagnosis of a postinflammatory occlusion of the foramina of Luschka and Magendie in adult life is more difficult. This is true mainly because the diagnosis of hydro-
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Fig. 7. Operative findings of case shown in Figures 5 and 6. Note the dense scar at a; the medulla d; the vermis e; with widening and flattening of the convolutions, owing to the distention of the fourth ventricle. The fourth ventricle bulges at b between the medulla and cerebellum because these structures are not bound by adhesions. In the insert the new opening is shown at c; this opening represents a newly constructed foramen of Magendie.

cerebral is more difficult and when the diagnosis of hydrocephalus is finally made, the chances are that the obstruction is due to a tumor, particularly when there has been no antecedent history of meningitis. In infants, intracranial tumors need scarcely be considered, so that the diagnosis of hydrocephalus, which is made with relative facility, presupposes an inflammatory condition or one of the congenital defects, both of which lesions are so frequent at that age.

I had never heard of an inflammatory occlusion of the foramina of Luschka and Magendie in adult life, so that the first case came as a surprise, the preoperative diagnosis of a cerebellar tumor having been made. The second case was, likewise, diagnosed a cerebellar tumor because of unilateral localizing signs and symptoms. In neither case, therefore, can we claim credit for making the clinical diagnosis. I have since seen a case which, on examination, gave almost the identical findings which were present in the second case; the diagnosis of an inflammatory occlusion of the foramina of Luschka and Magendie was considered but a cerebellopontine tumor was found. I know of no way that an absolute diagnosis of this type of lesion can be made yet; but it is always possible to diagnose the hydrocephalus, after which the operator must be prepared to find the lesion which is causing the obstruction and thereby producing the hydrocephalus.

The first patient with this inflammation was 18 years old (Fig. 5). He complained of general headache and loss of vision, and, at times, a staggering gait. Eight months before admission to the Johns Hopkins Hospital, the patient was seized with sudden severe headaches in the forehead and suboccipital region. These persisted, though with varying intensity. Seven months ago and one month after the onset of his illness, a sudden severe headache was quickly followed by twitching of his limbs and his right leg became paralyzed and without feeling. This paralysis lasted only a few hours and never recurred, though a similar attack a month later was accompanied by the loss of power in the right arm; this was also transient. A staggering gait was present at times, principally during exacerbations of headaches. Soon an impairment of vision was noticed in the left eye and shortly afterward in the right eye. There had been a rapid loss of sight in both eyes until there was little vision remaining in either
eye, the left being definitely worse than the right. There is complete loss of color vision and great reduction in the fields of vision; (he could not read the large headlines of a newspaper). Diplopia had been present at times. Nausea and vomiting were present only on one occasion. There was a bilateral choked disc which measured six diopters in each eye. The neurological examination was strikingly negative except for a slight, even questionable, Romberg. There was no ataxia, no nystagmus, no adiadokokinesia, no extraocular palsy and the gait was entirely normal during several examinations. There was no difference in the reflexes on the two sides. Aside from the uncertain Romberg, there was only one localizing sign which could be made out objectively and that was a convolutional atrophy of the skull (Fig. 6). This roentgenographic finding is almost pathognomonic of hydrocephalus and, therefore, referred the lesion to the posterior cranial fossa. At operation, the hydrocephalus was found to be due to a dense scar at the foramen of Magendie (Fig. 7).

After operation we inquired into the past history very closely for an illness which could have been responsible for the production of this scar. There was never an illness which at all resembled meningitis, but the mother regarded his present illness as a sequel to an attack of "measles" which antedated his present cranial symptoms by four months. She had even volunteered this when the history was taken before the operation but his recovery from the attack seemed complete and there was a four months' interval of practically normal health. This illness was probably measles; there was an epidemic; a rash, photophobia, and fever were present; he was confined to bed 5 or 6 days. There had been no headaches, cervical rigidity or pains in the legs or back during the illness.

The second case was a man of 36 (Fig. 4) who had wandered about from physician to physician looking for relief from terrific headache and dizziness. Two, not only futile but injurious operations, had been performed by well meaning but thoroughly incompetent surgeons. His illness dated back 4 years when, without warning, he suddenly fell unconscious. Previous to this time he had had only occasional headaches but since then the pain never remitted. Nearly every morning and frequently during the day, vomiting accompanied the headaches and the patient was soon too weak to continue his work. Although he never fell, there was always a feeling as though he would "topple forward on his head." After these symptoms had persisted for a year there was a sudden and unexplained cessation of all symptoms for nearly a year. Then the headaches, dizziness, and vomiting again returned as before but with even greater violence. Vertigo was increased by any change of position or by lying on the back. The headache was then always present and was both frontal and suboccipital.

Bilateral decompressions, previously performed, were bulging knots. The following positive neuro-
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Antecedent meningitis, there could be little certainty in making a diagnosis of occlusion of the foramina of Luschka and Magendie before operation. Of probably the greatest significance is the absence or the relatively slight intensity of the usual signs which are present in cerebellar lesions, such as ataxia, nystagmus, staggering gait and Romberg. In the second case, the complete cessation of all symptoms for a year is suggestive of such a lesion rather than a neoplasm.

The diagnosis of hydrocephalus can frequently be made in the later stages from the roentgenogram but in the early or moderately advanced stages of the disease the diagnosis can only be made by ventriculography. The methods of arriving at the diagnosis are essentially the same in infants and in adults. The diagnosis of hydrocephalus is all-important. The precise localization of the cause of the hydrocephalus before operation is not indispensable, for the same operative approach is necessary for obstructions at the foramina of Luschka and Magendie or the aqueduct of Sylvius. But if a hydrocephalus is proved, it is absolutely required of the surgeon that this obstruction be found at operation and of the findings there can be no equivocation.

Several years ago, I described with Dr. Blackfan occlusion of the foramina of Luschka and Magendie in an infant with hydrocephalus, but, so far as I am aware, this case and those which I have described here are the only instances of this lesion in the literature. I am confident none has been found at operation.

THE TREATMENT OF OCCLUSIONS OF THE FORAMINA OF LUSCHKA AND MAGENDIE

It is obvious that the only satisfactory treatment of any form of hydrocephalus is the treatment of the cause. In occlusions of the foramina of Luschka and Magendie, the entire ventricular system is devoid of any communication with the subarachnoid space and to cure the hydrocephalus it is necessary to make one opening between the fourth ventricle and the cisterna magna to assume the function of the three which are blocked. But before operating it is necessary to know whether the ultimate radicles of the subarachnoid space are open and whether there is a normal absorption of fluid from the subarachnoid space. Following meningitis, particularly in children, the cisternae are blocked as well as the foramina of Luschka and Magendie, so that the reconstruction of a new foramen of Magendie would lead to no beneficial results because the ventricular fluid would have access to only the restricted area of the subarachnoid space posterior to tentorium cerebelli. The subarachnoid space should be tested in one of two ways, first by the intraspinous phenolsulphonephthalein test and second by an intraspinous injection of air. If over 30 per cent of the phenolsulphonephthalein is absorbed in 2 hours, a cure can be expected if the cause is corrected. Intraspinous injections of air will give the same information. The air will reach all the parts of the subarachnoid space which are open. If, therefore, the cerebral sulci show in the roentgenogram, there is a graphic demonstration that the absorbing spaces are open to the reception of fluid if its escape from the ventricles is made possible. The absence of air in the cerebral sulci denotes a closure of the cisternae and precludes an operative cure of the hydrocephalus. Such a finding would be accompanied by a low phenolsulphonephthalein output, usually less than 10 per cent in 2 hours.

PRODUCTION OF A NEW FORAMEN OF MAGENDIE AT OPERATION

The usual bilateral exposure of the cerebellum is made exactly as is done for the extirpation of cerebellar tumors. The operator will quickly learn to recognize the normal foramen of Magendie at a glance and to know whether it is open or closed. I rarely expose the cerebellum without seeing the foramen of Magendie, either because it is necessary to look for it or because after the removal of a tumor from this region, it is usually brought directly into view. It will probably occur to the reader to ask why it is not necessary to expose and determine the patency of the foramina of Luschka. The exposure of these openings is possible but much more difficult and not important. It is necessary to have but one of the three
foramina patent and, if the foramen of Magendie is open, it is not necessary to search further. If the foramen of Magendie is occluded the size of the fourth ventricle will indicate the condition of the foramina of Luschka.

I have operated upon three cases in which a total occlusion of the foramen of Magendie has been found. The findings in each were quite different. In an infant of 3 months, the vermis was entirely absent and the lateral lobes of the cerebellum pushed laterally into the outer angles of the posterior cranial fossa; the remainder of this fossa was filled with the tremendously dilated fourth ventricle covered only by a membrane. The hydrocephalus was extreme, so that puncture of the ventricle quickly resulted in death. The lesion disclosed at operation in the boy of 19 was sharply localized to the foramen of Magendie and the immediately contiguous region to either side. A very dense scar (Fig. 7) bound the cerebellum and medulla and, because of its rigidity, precluded a herniation of the lateral lobes into the spinal canal. To either side of the scar, the tissues were normal. A thin, bluish wall bulged between the cerebellum and medulla and, because of its thinness, offered an ideal situation for the construction of a new foramen (Fig. 7). The absence of any signs of an old inflammatory process in this region seemed to preclude a closure of the newly made foramen, especially in view of the steady flow of cerebrospinal fluid through it into the cisterna magna. In the second case, the procedure was different, the medulla was closely applied to the cerebellum preventing the fourth ventricle bulging between these structures as in the preceding case. It was, therefore, impossible to reconstruct a new foramen of Magendie in the space between the medulla and cerebellum, as in the other case, with any prospect of maintaining a permanent opening. The scar at the foramen of Magendie was much more localized and was confined almost to the normal limits of the foramen of Magendie. A large bilateral cerebellar hernia into the spinal canal made the exposure of the scarred foramen of Magendie difficult. To construct a new foramen, it was necessary to excise the entire scar at the foramen of Magendie. The recurrence of the scar in this case remains a possibility; in the preceding case, recurrence seems impossible.

POSTOPERATIVE COURSE AFTER CONSTRUCTION OF A NEW FORAMEN OF MAGENDIE

Each of the adult cases left the hospital a month after operation, apparently perfectly well. The boy of 19 has since resumed his studies at college. Every symptom has been completely relieved. His vision has greatly improved, both in range and acuity. Color vision, which before operation was entirely gone, has returned. Unfortunately, the older case has been lost track of, so that his ultimate condition can not be reported.

In one of these cases, we have demonstrated by ventriculography not only that the new foramen of Magendie is patent and functioning but that the hydrocephalus has been cured (Fig. 8). Six weeks after leaving the hospital, the patient returned for observation and air was injected into a lateral ventricle in order to test the new foramen. The roentgenogram (Fig. 8) shows the tremendous enlargement of the lateral ventricles; the third ventricle, the aqueduct of Sylvius and the fourth ventricle are sharply outlined. The air has passed through the new foramen of Magendie—which is marked by the silver clips which were placed over a divided blood vessel at operation—into the cisterna magna. The air is even seen to fill the cerebral sulci, showing that the most remote branches of the subarachnoid space receive fluid from the ventricles and that, therefore, the hydrocephalus has been cured.

CONCLUSIONS

1. Blocking of the foramina of Luschka and Magendie invariably produces hydrocephalus. Patency of one of the three foramina prevents the development of hydrocephalus, provided the subarachnoid space is normal.

2. A group of cases is presented with occlusion of these foramina and with a hydrocephalus resulting therefrom.

3. Undoubtedly, the disease, occurring in adult life, follows an inflammatory process
which may or may not have been clinically evident. Probably, failure of the foramina to develop accounts for the hydrocephalus in many cases which are recognized soon after birth; in other infants an intra-uterine or postnatal inflammation is the cause.

4. Although the general results of the occlusion are the same in every case, there are marked local anatomical differences due to the extent of the inflammatory process and the time of its development.

5. There are no clinical features which permit one to make an absolute diagnosis of occlusion of the foramina of Magendie and Luschka. In every instance, both in adults and infants, the diagnosis of hydrocephalus is possible; the exact site and the character of the obstruction causing the hydrocephalus can be determined by ventriculography and by the phenolsulphonephthalein test.

6. The lesion can always be found at operation.

7. An operative treatment is presented. Two adult cases have apparently been cured by this procedure, which attacks the cause directly.