The pathogenesis of post meningitic hydrocephalus

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THE PATHOGENESIS OF POST-MENINGITIC HYDROCEPHALUS

BY

BRUCE R AUSTIN

SENIOR THESIS
1932
INTRODUCTION

It is commonly taught and stated in text-books that post-meningitic hydrocephalus is due to a blockage in the aqueduct of Sylvius or foramina of Magendi and Luschka and that usually spinal fluid is not obtained by lumbar tap. While this may occur, it has been shown that another type of blockage, probably more frequent than the above, may occur. It is, therefore, the object of this paper to present evidence in favor of such a blockage and to confirm the work of earlier investigators apparently unrecognized.

This paper is based upon the work and findings of Dr. Bennett and Dr. Hunt of this institution while I have but attempted to review the literature concerning the subject and to place it in one paper.

I wish to take this opportunity to acknowledge my indebtedness and to express my feeling of deep appreciation to Dr. Bennett and Dr. Hunt for all that they have done.
THE PATHOGENESIS OF POST-MENINGETIC HYDROCEPHALUS

HISTORY

The practitioners of medicine in ancient times had various ideas concerning the cerebrospinal fluid. Galen believed that the fluid contained the animal spirits. Vesalius thought it was a lubricant. Varolius credited the choroid plexus with the property of pumping water into the ventricles. Willis regarded the fluid as a product of distillation of the penial gland and choroid plexuses. Eventoday the exact origin of this fluid is debated.

1774 Cerebrospinal fluid described by Donenico Catugno.
1806 Vieusseux of Geneva gave the first description of meningitis.
1825 Magendie was probably the first to appreciate the physiological importance of the fluid.
1865 Ziemssen and Hess as early as this had observed and verified by necropsy obstructive hydrocephalus.
1876 Keys and Retzius published their famous work in which a comprehensive description of cerebral membranes was given for the first time.
1884 Meningococcus discovered by Marchiofava and Celli.
1887 Anton Weichselbaum identified the organism as the cause of meningitis and named it the diplo-coccus-intracellularis meningitidis.
1891 Henrick Quicken of Keil showed that it was possible to puncture the dura mater and withdraw cerebrospinal fluid.

1906 Antimeningococcic serum began to be used intraspinally with a notable drop in the mortality of the disease.

1908 Cushing and Sladen reported a case of epidemic meningitis with the intraventricular injection of serum.

1918 Herrick described the intravenous serum treatment.

1918-1920 Ayer, Wegeforth and Essick worked out the technique of cisternal puncture and recommended the injection of serum by this route.
ANATOMY AND PHYSIOLOGY OF THE CEREBROSPINAL FLUID
AND THE SUBARACHNOID SPACE

To think of cerebrospinal fluid is to think of Dr. L. H. Weed and the tremendous amount of work that he has done. Concerning the composition of the cerebrospinal fluid, Weed (16), points out that it is a clear limpid liquid of low specific gravity (1.004-1.006), colorless and of a slight but definite viscosity. It contains but few cells—less than 10 normally—but this number is increased greatly in pathological conditions. It is estimated that 100-150 gram are in the cerebral ventricles and about the nervous system. It was termed by Halliburton as "an ideal physiological saline solution".

According to Weed (16), Dandy (5), and many others the cerebrospinal fluid is produced; 1) chiefly by the choroid plexuses of the ventricles, 2) partly from the ependymal cells lining the ventricles and 3) some from the perivascular spaces. However, Hassin (9) & (10) believes it is derived from the tissue fields of the brain and that it enters the ventricles of the brain loaded with waste products which are removed by the choroid plexuses—thus purifying it. His assumptions are based on several cases of hydrocephalus whose choroid plexuses at necropsy were practically absent and seemed too diseased to have caused the disorder. Dandy on the other hand has experimentally
produced hydrocephalus by blocking the foramen of Munro and has also failed to get hydrocephalus in these cases when the choroid plexus was removed before the block was made.

At the present time the circulation of the cerebrospinal fluid is well agreed upon by Weed (16) & (17), Dandy (5) & Globus (8). (Constant reference to diagrams 1 & 11 will help in the visualisation of the subarachnoid spaces and the rout of flow of the cerebrospinal fluid.) The fluid begins its circulation in the lateral ventricles which, as well as the remaining ones, are lined by ependymal cells of ectodermal origin. It passes through the foramina of Monro into the third ventricle and thence by way of the aqueduct of Sylvius into the fourth ventricle. From here the fluid is believed to pass by way of the foramina of Lushka and Magendie into the mesodermal subarachnoid spaces, which at this location are called the cisterna magna and cisterna pontis. From the cisterna magna the fluid slowly seeps efficiently downward in the spinal subarachnoid space. From the cisterna pontis the fluid passes more rapidly forward and upward through the cisterna interpeduncularis to the cisterna chiasmatica from which it passed more slowly up over the hemispheres of the brain. It definitely surrounds the whole central nervous system.

According to Weed (16) the subarachnoid space is that space which lies between the arachnoid membrane and the pia
Plate 1

Plate 11
Schematic representation of the central nervous system with reference to the circulation of the cerebrospinal Fluid. Du., dura; A.V., arachnoid villus; Sd., sub dural space; Ar., arachnoid; A.T., Arachnoid Trabecula; F.M., Foramen of Magendie; Ven., ventrical; Gan., ganglion; Cap., Capillary; G.P., choroid plexus; A.G., arachnoid granulations; Sar., subarachnoid space; C.C. cuboidal cells; Pi., pia; Pv., perivasacular space; Cor., cortex cerebri; Ng., neuroglia.

Taken from--
mater. The former adheres above quite closely to the dura mater and is attached below to the pia mater only by small trabeculae. The arachnoid crosses all sulci of the brain while the pia is closely adherent to the brain thus dipping down into all the brain's sulci. The subarachnoid space is real and according to Weed (16) is lined entirely by a flat, specialized cell fluid retaining in character.

The trabeculae are covered by this type cell and form tortuous meshes in the subarachnoid space. These meshes are smaller over the cerebral hemispheres but get larger in the cerebral sulci and about the spinal cord and reach their maximum in the cisternal dilatation about the cerebro-bulbo angle. The finer the meshed the slower fluid flows and at its slowest movement it comes into closest relationship to the great venous sinuses of the dura matter.

As the origin of the cerebrospinal fluid is disputed so is its point of absorption and the supporters of each theory have done a definite amount of work. Weed (15) & (16) says, "it seems fair to assume that the absorption is two fold process; 1) being chiefly by a rapid drainage into the great dural sinuses, probably by way of the arachnoid villi and 2) in a small part a slow indirect escape into the true lymphatic vessels". Dandy (4) suggests that the absorption of fluid must be by way of "the capillaries which abound in all the radicles of the subarachnoid space". Hassin (10) believes the fluid to escape by way of perineural
spaces into epineural spaces whose rich blood supply receives the fluid.

The pressure of normal cerebrospinal fluid is about 8 mm of mercury and according to Weed (16) is dependent on the blood content, the cerebrospinal fluid and the brain tissue; they all being enclosed in a bony case, the skull. The cerebrospinal fluid pressure is between that of the arteries and the venous sinuses. Normally there is a balance between these but because of the bony case if one is varied the other two must compensate in volume and pressure to keep the pressure constant.
PATHOLOGICAL FINDINGS AT NECROPSY

Regardless of all the experimental work that is done, it is usually the findings at necropsy, associated with signs and symptoms of the disease that has marked the real progress in medicine.

The following will point out where the obstruction of cerebrospinal fluid occurred during the course of meningococci meningitis: Cushing (2), in 1908 reported a necropsy on a child, age 6 months, where he found a thick membrane surrounding the outlets of the ventricular fluid in the neighborhood of the 4th ventricle. The lateral ventricles were distended and contained a purulent fluid, while the ependyma showed a granular inflammation. Blackfan & Dandy (6), in 1917 in four cases brought to necropsy said, "each of the four cases presented exactly the same pathological condition - a barrier of very dense adhesions at the base of the brain. In each case the foramen of Magendie and one foramen of Lushka were sealed by adhesions and the other foramen of Lushka was patent to a certain degree, and through this single channel the fluid escaped from the 4th ventricle and therefore from the ventricular system. The adhesions completely incircled the brain anterior to the patent foramen of Lushka, and the basal cisternae - cisterna magna, cisterna pontomedularis, and cisterna interpeduncularis.
were completely obliterated by these adhesions. The adhesions in each case were more dense in the region of the medulla and the cerebellum and became less pronounced in the chiasmal region. Minor adhesions were scattered over the surface of the brain. In one case the adhesions were present over the base, with additional dense adhesions complete binding the tentorium to the posterior surface of each occipital lobe, and complete encircling the midbrain as it passed through the opening in the tentorium cerebelli. In each instance an apparently complete encircling mass of adhesions sealed off the base of the brain so that the cerebrospinal fluid could not pass forward to the cerebral subarachnoid space but only downward into the spinal canal. The obliteration of the various basilar cisternae, which are the centers from which cerebrospinal is distributed over the subarachnoid space, may in itself be sufficient to eliminate the cerebral subarachnoid space from absorption even without the encircling adhesions."

Refer to plate 111

Globus (8) following up Dandy's work reported a case of hydrocephalus due to similar adhesions at the base of the brain. Hassin (10), also working on hydrocephalus believes that the escape of fluid maybe prevented by occlusions of the cisterna and the perineural spaces. Worster-Drought (20), presents the following autopsy, "the dura mater was found, and on incising it a large quantity of slightly turbid fluid escaped. Over the convexity there was practically no
This shows in diagram form, roughly, the distribution of the adhesions over the base of the brain responsible for the communicating type of internal hydrocephalus.

\( X, X \) represent areas on the cerebellar hemispheres which are relatively free from adhesions.

\( L \) represents the foramen of Luschka, which is patent only on this side. The opposite side is completely obliterated by adhesions. The adhesions extend along the tentorium on both the superior and inferior surfaces.

meninges. At the base, however, tough fibropurulent exudate, was found over the pons, medulla and cerebrum, extending as far forward as the optic chiasma. The exudate also extended downward to the cervical region of the spinal canal. The spinal canal at the foramen magnum being almost completely occluded. The lateral ventricles were only moderately distended with slightly turbid fluid.

From the foregoing it would seem fair to conclude that in meningococcal meningitis an exudate is formed in the large cisternae which later goes on to a fibrous formation which in turn blocks the flow of cerebrospinal fluid upward over the cerebral hemispheres. There following a dilatation of ventricles and cisternae and a thinning of the brain cortex.

EXPERIMENTAL EVIDENCE IN THE LITERATURE

Granting that the above blockage is formed it remains to establish a similar block in some animal and determine the results. It would also be advantageous to determine such a blockage in the human prior to death and autopsy. Dandy (5), in a rather classical experiment produced in dogs a barrier of adhesions in the mesencephalic cisternae which in time resulted in signs of increased cranial pressure. Before necropsy India ink was substituted by cisternal puncture for cerebrospinal fluid and the animal then killed in two hours. At necropsy the ink did not extend beyond the band of adhesions, it being found only in the cisterna and ventricles. Normally the ink would
have been carried to all parts of the subarachnoid space. This in itself shows conclusively what a block in the disterna will do. Weed (17) using kittens and injecting lampblack into the cisterna magna found that they soon developed signs of hydrocephalus. At necropsy dilated ventricles and a thinned brain cortex were found; the lampblack having obstructed the absorption of cerebrospinal fluid.

Dandy (5), following the death of a meningitic patient suspected an obstruction in the flow of cerebrospinal fluid so he carefully substituted by the cisternal route India ink for that fluid. At autopsy the ink was found in the ventricles and extended out into the cisterna to the obstruction but it did not appear beyond the point of adhesions into the subarachnoid space. Globus (8), repeated this method in 5 meningitic cases brought to necropsy and each cause paralleled the work of Dandy.

Dandy (5), going a step farther wished to find evidence of obstruction in the cisternae prior to death. He was able to do this by the use of the x-ray. He substituted air for the cerebrospinal fluid and found in cases of obstruction in the cisternae that the air appeared in the dilated ventricles and cisternae but none showed up in the sulci of the brain. Normally the sulci in a roentgenogram appear as a net work over the cortex. This meant that hydrocephalus must exist, the cerebrospinal fluid evidently was not reaching the absorbing areas of the sub-
arachnoid space due, probably, to a fibrinous blockage in
the cisternae. This method has become quite popular and
is used now in determining the absence or presence of
obstruction in the ventricular or cisternal regions.
whether it be due to tumor, tuberculosis or, as was found
here, to adhesions following a meningococcic meningitis.

CLASSIFICATION OF HYDROCEPHALUS

From the foregoing, hydrocephalus could be said to be
that condition resulting from a marked decrease in the
absorption of cerebrospinal fluid causing an increase in
fluid pressure and this in turn resulting in dilated ven-
tricles, thinned brain cortex and possibly enlargement of
the bony skull. Hydrocephalus can be either acute or chronic
and, it can further be subdivided, as by Dandy, Weed & Globus,
into obstructive or communicating. In the obstructive form
the obstruction to the out flow of cerebrospinal fluid is
located in one or more foramina of the ventricles, either
at the foramen of Munro, Lushka or Magendia or at the
aqueduct of Sylvius; this might occur with a brain tumor,
tuberculosis meningitis or even with meningococcic men-
ingitis. Naturally in this case a roentgenogram would
show no air in the ventricles but at autopsy the ventricles
would be found dilated. In the Communicating type the
obstruction is outside the ventricular system, probably in
the cisternae especially toward the anterior part. In this
A roentgenogram would show air in the ventricles and cisternae but not in the sulci over the brain, providing of course the blockage was complete. If the blockage were incomplete it would be possible to visualize air in the sulci over a part of that hemisphere of the brain. In this type the ventricles and cisternae all communicate. It is assumed that one recognizes the fact that if an obstruction exists the cerebrospinal fluid can not get up to the smaller sub-arachnoid spaces where it can be absorbed.

Dandy (5), says, "obviously an obstruction can exist in any part of the subarachnoid tree and the results in terms of hydrocephalus will depend directly upon the degree and location of the obstruction". He also says, "that the most frequent location for an obstruction in the communicating hydrocephalus is in the cisternae".

THE RELATIONSHIP OF MENINGITIS TO HYDROCEPHALUS

Any disease such as meningococcus meningitis which tends to produce an obstruction to the flow of cerebrospinal fluid is closely related to hydrocephalus whether the obstruction occur during the course of the disease due to pyocephalia or as sequelae due to adhesions. Dandy & Blackfan (6) & Dandy (5), say, "that certainly the majority of all cases of communicating hydrocephalus follow meningitis and, being a post meningitic process, the obstruction of cisternae is in keeping with the basilar involvement of most forms of meningitis." They agree that most of these
cases occur in infants and young children and that at times the meningitic process may be of prenatal origin. They likewise believe that in more than half of their cases of communicating hydrocephalus the disease arises after birth following a disease wrongly diagnosed but latter proven by careful history to be meningitis. Their finding of adhesions at necropsy an operation prove this.

They state that in 7 out of 10 patients with communicating hydrocephalus studied by pneumograph, the obstruction was shown to exist in the pontine or mesencephalic cisternae, the column of air ending at that point and none reaching up into the cerebral sulci. Dandy(5), even goes so far as to say," it is really difficult to understand how communicating hydrocephalus can be caused by any other process than a meningitis.

As to the complications following sporadic meningococceus meningitis treated by serum McLean and Caffey (12), were able to follow 44 cases for 10 years. They found that 69% showed no abnormalities, 22% showed serious sequelae and 11% died. Of the 20% or 9 cases showing sequelae 10% or 4 became deaf mutes, 5% or 2 hydrocephalics, 5% or 2 had impaired vision and 1 was mentally deficient. This is not a large number of cases but it must be remembered that such cases are often difficult to follow.

That hydrocéphalus will develop in the course of the
disease receiving serum treatment is shown by Stetten and Roberts (13), who say, "we find that a case of epidemic meningitis which has been running a relatively normal course rather suddenly develops signs and symptoms pointing to an acute obstructive hydrocephalus." Worster-Drought (20), agrees in the following quotation, "The hydrocephalus occurring in early stages of meningitis, which is responsible for such symptoms as headache, vomiting, stupor, and in children, bludging of anterior fontanelle, is relieved by the lumbar puncture preliminary to the first intrathecal injection of serum".

As regards treatment of epidemic meningitis in the hope of averting a hydrocephalus all, (Bengamin(1), Flexner(7), Kutscher(11), & Stookey (14), along with others), agree that serum given early, more frequently by cisternal route and associated with good drainage of the cerebrospinal fluid is of great value. Then on the other side it is certain that there are others who wonder if too long and too active serum treatment is not conducive to the development of a hydrocephalus.
PRESENTATION OF CASES

1. To confirm Dandy's and Weeds findings three cases of sporadic epidemic meningitis all of which rather suddenly developed signs of increased cranial pressure and whose roentgenogram taken at this time showed a hydrocephalus are presented.

1. George J., 29 months old male was carried to the University Hospital on 3-23-31. He was crying, seemed very irritable and showed a definite dorsal neck retraction.

Onset & Development

3-20-31 patient developed a slight head cold, late the next day he became very irritable, began to vomit and complain of his head hurting. 3-22-31 patients neck became fixed, started to become dorsally extended and he complained of pain in that region.

HISTORY: negative except for having an occasional head cold and pneumonia when 9 mo. old

PHYSICAL EXAMINATION: A boy of stated age lying restlessly in bed on his left side with thighs flexed on body and head noticably dorsally retracted. Eyes showed horizontal nystagmus while the nose and pharynx showed a discharge. His neck was rigid, dorsally extended and its movement elicited much pain. There were course rales in both lungs
while his respirations and pulse were both rapid but regular. Reflexes exacerbated with Kernig and Brudzinski positive.

**PROGRESS & TREATMENT:** Spinal puncture on 3-23-31 showed a cloudy spinal fluid under 9 mm hg. and a cell count of 5,320 with 89% polys. Gram negative intracellular diplococcus seen. The cell count gradually decreased till on 4-5-31 the day after last serum given, 194 polys were counted. He received between 25-30 cc of serum daily in two doses, one half by lumbar puncture and the remaining by cisternal puncture from 3-23-31 to 4-5-31 when serum treatment stopped. On 4-7-31 patients breathing and heart action became irregular and it was necessary to administer hypertonic glucose and stimulants. The temperature ranging from 104 to 101.5 came down to normal when serum treatment was stopped and remained there till patient dismissed. Lumbar punctures were began on 4-8-31 and continued daily till 4-18-31 with 30 to 50 cc of spinal fluid removed each time. On 4-24-31 an encephalogram was done with 60 cc of spinal fluid withdrawn. With the beginning of these lumbar punctures patient began to improve and eventually recovered.

The following is a report on the roentgenogram by Dr. Hunt roentgenologist for the University:

"Encephalographic study demonstrates slight although definite widening of the coronal and sagittal sutures. Both lateral ventricles are well visualized and enormously dilated. The
anterior horns appear forshortened on each side while the posterior horns are unusually long. This maybe the result of a congenital variation. The 3d & 4th ventricules are not well outlined. A small amount of air is visualized in the subarachnoid spaces about the base in the midbrain region. There is no air visualized in the subarachnoid spaces over the cortex. There is no gross lateral displacement or asymmetry of the Ventricular system."

Dr. Hunt
Janith S. four year old girl was carried to the University Hospital receiving room on 3-4-31 complaining of neck rigidity, headache, pain in neck, vomiting and general malaise.

ONSET & DEVELOPMENT: On 2-26-31 patient developed a head cold. On 3-1-31 became restless and her mother thought child had a temperature. On the afternoon of 3-2-31 patient began to vomit, this continuing intermittently till 3-3-31 when she became more restless and that evening mother noticed stiffness in her neck and a slight posterior bowing. Local doctor was called and injected 30cc of antimeningococccic serum intraspinally.

HISTORY: Negative except for measles, mumps, scarlet fever and a head injury, possibly a fractured skull, with which she vomited a great deal but she recovered and no sequelae developed.

PHYSICAL EXAMINATION: A well nourished girl of about stated age lying quietly in bed on left side. Her face markedly flushed, thighs flexed on abdomen and head dorsally extended. Her nose and pharynx showed signs of upper respiratory infection. The neck besides being extended was rigid and cervical lymph nodes were palpable. Respiration and heart beats rapid and regular. Legs flexed. Reflexes exaggerated and Babinski, Kernig and Brudzinski positive. Diagnosis of epidemic meningitis was made.
TREATMENT & PROGRESS: 3-4-31 spinal puncture done and a cloudy fluid obtained with a cell count of 25,600 most of them polys. Gram negative intracellular diplococcus seen and later cultured. From 3-4-31 to 3-12-31 15cc of serum were given in A. M. and 10 cc in P. M. usually by lumbar puncture but occasionally by cisternal route. From 20-40 cc of spinal fluid were withdrawn each time. At the end of this time child was showing improvement and the fluid cell count was down to 2,100 with polys about 94% most of the time.

From 3-12-31 to 3-18-31 15cc of serum given daily and 15-30cc of fluid withdrawn. By this time spinal fluid pressure was down and cell count count running about 128 per c. m. From 3-18-31 to 4-7-31, when encephalogram taken, no lumbar punctures down but from 4-7-31 to 4-29-31 a lumbar puncture done daily with removal of enough fluid to keep the pressure down. A dry diet of less than 500 cc of fluids a day was given patient. On 3-30-31 it was noticed patient was completely deaf. Improvement was slow and child was dismissed on 5-8-31 in fair condition.

The temperature was high and irregular for the first 2 weeks ranging from 104.5 to 100. It gradually approached normal and remained so for next 3 weeks when it went up to around 101. Stayed so for about 1 week, dropped back to normal and remained there till patient dismissed. The blood and urine findings were of no significants.
Roentgenogram of Janith S.

"A study of skull and cerebral fluid spaces was made. There was slight widening of the coronal sutures but no increase of the convolutional impression. There was no apparent change in pituitary fossa. The lateral ventricles were markedly dilated to about 10 times their usual size. No air visualized within the third ventricle, aqueduct or in fourth ventricle. The left lateral ventricle maybe slightly more dilated than the right. There was no significant lateral displacement of the ventricular cisternae. A small amount of air visualized within the chiasmatic cistern. No air is visualized within the subarachnoid spaces distal to the basilar cisternae.

Summary: Marked dilatation of the lateral ventricle, obliteration of subarachnoid spaces."

Dr. Hunt.
Janie G. 12 year old negro girl was brought to the University Hospital receiving room 3-9-31 complaining of: headache, stiffneck, pain in legs and stomach and fever.

DEVELOPMENT & ONSET: 3-6-31 patient had an acute coryza with a sore throat. The following day she complained of a headache in left frontal region and toward evening her legs and neck began to hurt. She had pain in her stomach and became very restless. On 3-8-31 her neck pained her and her temperature rose above normal.

HISTORY: Negative except for measles.

PHYSICAL EXAMINATION: A well developed negro girl about stated age lying restlessly in bed with thighs flexed on abdomen, neck dorsally extended and moaning because of sever frontal headache. Throat injected and showed post nasal discharge. Neck dorsally extended. Heart beats rapid regular and sounds somewhat accentuated. Legs flexed on abdomen and painful to movement. Kernig and Brudzinsky markedly positive. Other reflexes not greatly affected.

TREATMENT & PROGRESS: 3-9-31 spinal puncture showed a cloudy fluid under 22mm hg. and with a cell count of 6,700 in which gram negative intracellular diplococcus were seen. From 3-9-31 to 3-16-31 a cisternal puncture was done in A. M. with about 20cc spinal fluid removed and 15cc serum injected. This was repeated in afternoon only by lumbar puncture.
Spinal fluid gradually became more clear and cell count decreased. On 2-12-31 left ear began to drain. 3-16-31 to 3-23-31 15cc serum given a day by lumbar route. 3-23-31 to 3-30-31 serum given but once but either a lumbar or a costernal puncture was done once a day. On 4-12-31 patient began to complain of headache, neck retraction became more noticeable, and pain in neck became worse. In next 3 days she began to vomit and her condition became worse. A dehydration diet of less than 500 cc daily was ordered. Lumbar puncture showed spinal fluid under 20 mm pressure. On 4-26-31 encephalogram done with 80 cc spinal fluid removed. Patient began to improve slowly and was dismissed 5-16-31. Patient's temperature was high till fourth week when it began to drop and by end of 5th week was down to normal remaining so till dismissal. Her pulse, respiration and laboratory findings showed nothing of significance. The roentgenogram report follows.
"An encéphalographic study demonstrated an extreme dilation of both lateral ventricles to about 10 times their usual volume and a moderate dilatation of 3d ventricle. There is no evident lateral displacement of the ventricular system and no evident filling defects or forshortening. The 4th ventricular and aqueduct of Sylvius are not visualized. The right lateral ventricle appears somewhat less well visualized than the left. There is no apparent air in basalar cisternae or over the cortex in subarachnoid spaces. Summary: Moderate internal hydrocephalus with obliteration of subarachnoid spaces."

Dr. Hunt.
CONCLUSION

In the foregoing cases hydrocephalus appeared clinically about the third week. The roentgenogram confirmed this. That the obstruction causing it was not in any of the foramen or aqueduct of Sylvius is shown by the fact that air injected into the spinal canal appeared in the dilated ventricles but not in the sulci of the brain. Hence, the obstruction must have been in the cisternae, probably interpeduncularis or chiasmatica. This interpretation undoubtedly confirms the work of Dandy, Blackfan and Globus who believed that in the majority of cases of hydrocephalus following meningococcic meningitis the obstruction was found in the cisternae probably the cisterna interpeduncularis or cisterna chiasmatica.
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