THROMBOSIS OF THE DURAL VENOUS SINUSES AS A CAUSE OF "PSEUDOTUMOR CEREBRI"*

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FOR MANY YEARS there have been sporadic reports of a syndrome characterized by increased intracranial pressure and clear spinal fluid without an intracranial neoplasm. Quincke[1] (1893) was perhaps the first to describe cases with this syndrome, and he applied the name "serous meningitis" which is still often used. Nonne[2] (1904) in a similar collection of cases characterized the condition as possessing a more or less chronic development of the picture of brain tumor, with or without localizing signs and without demonstrable pathology. The condition was refered to as "die Nonnesche Krankheit" by other authors[3] (1910) and later by Nonne[4] (1914) as "pseudotumor cerebri," a name also still in use. Other names in common use that have been given the syndrome are "arachnoiditis" (Cushing), "otitic hydrocephalus" (Symonds,[5] 1931)) and "toxic hydrocephalus" (McAlpine,[6] 1937). Dandy[7] (1937) referred to the syndrome, in reporting on 22 accumulated cases, merely as "intracranial pressure without brain tumor."

Clinically, most of the reported cases have, in common, headache, papilledema, increased cerebrospinal fluid pressure and occasionally transient palsies and convulsions. The onset is usually insidious and the patient has the appearance of general good health. Blurred vision and measurable impairment of visual acuity occur if the papilledema persists. Also, diplopia due to abducens nerve palsy secondary to the increased intracranial pressure is common. The spinal fluid, except for the increased pressure, is usually normal and when there are increases in the cell count or protein content these changes are relatively slight. The electro-encephalogram may show abnormal activity of a nonspecific type throughout both hemispheres. Pneumoventriculograms show normal or small ventricles without displacement or distortion and they remain so no matter how long the disease persists.

Some of the cases are associated with infections of the middle ear or mastoid, infections elsewhere in the body, or with recent head injuries or the post partum state. In such cases intracranial venous thrombosis has sometimes been suspected and occasionally demonstrated. But possibly in half the cases there are none of these or other associated conditions. In spite of the many studies of the syndrome, the exact etiology has remained obscure, most observers believing that the condition is caused by some defect in production or absorption of the cerebrospinal fluid. Bailey[8] in the Year Book of Neurology, Psychiatry & Neurosurgery (1949) summarized a report on Pseudotumor Cerebri by Kehrer, F. A. (1949) in which 22 diseases were listed as a basis for such a syndrome, and the list does not include thrombosis of the intracranial venous system.

The majority of the patients with the syndrome recover spontaneously and com-

* Read before the American Surgical Association, Washington, D. C., April 11, 1951.

pletely within a period of eight to 12 weeks. Occasionally the symptoms and signs last for longer periods or may have exacerbations over a period of many months or years. In these cases it may be necessary to perform unilateral subtemporal decompressions in an attempt to allay headache and preserve vision. Some[9] have prefered the use of repeated lumbar punctures with or without surgical decompression in an attempt to maintain a lower intracranial pressure. No other useful therapy seems to have been developed heretofore.

Recently we have studied by sagittal sinus venography four patients presenting this peculiar syndrome. By this method we have been able to demonstrate thrombosis in the posterior half of the superior sagittal sinus or in a dominant transverse sinus with resultant obstruction to the venous outflow of the brain as a cause of the syndrome. On the basis of such established diagnosis certain therapeutic aids appear to be useful.

METHOD

The method of dural sinus venography has been previously published (Ray, Dunbar and Dotter,[10] 1951). Briefly, the method consists of the injection of 35 per cent Diodrast into the anterior portion of the superior sagittal sinus via a catheter introduced into the sinus through a small midline burr hole, with appropriate roentgenograms taken. In the normal, the contrast medium passes backward through the superior sagittal sinus to the torcular herophili and thence through both transverse sinuses into the internal jugular veins. The cerebral veins and other dural sinuses do not fill unless there is some obstruction in the superior sagittal or transverse sinuses. The venous pressure in the sinus is measured at rest and after jugular compression. Normal venous pressure in the sinus is 100-150 mm. of saline.

CASE REPORTS

Case 1.—(I. C., N. Y. H. No. 541269). A 48-year-old woman was admitted to the hospital in May, 1949, complaining of generalized headache, double vision and impaired visual acuity of 6 months' duration. Except for bilateral papilledema, the physical examination was unremarkable. The cerebrospinal fluid pressure was 480 mm.; the fluid was clear, acellular, sterile and of normal protein content. A pneumoventriculogram showed a small ventricular system of normal configuration. Bilateral subtemporal decompressions were performed, but there was no significant alteration of her condition in the following 8 months. The decompressions remained full, though somewhat variable in tension.

She was re-admitted to the hospital for additional study. In the interim she had had several relatively minor convulsive seizures without focal qualities. On one occasion she had for several days seemed confused and given to emotional upsets of mild panic which was out of keeping with her usual placid nature. The corrected visual acuity was 20/70 in one eye and 20/50 in the other eye. There was bilateral papilledema and the decompressions were full. The rest of the neurologic examination was normal. The spinal fluid pressure was 480 mm.

A bilateral carotid arteriogram was performed which showed good filling on the left with some of the contrast medium passing to the right cerebrum. On the right side there was an obstruction in the internal carotid in the neck, presumably from a thrombosis of that artery. The contrast medium injected on this right side passed down the common carotid artery and back up the right vertebral artery, outlining the right posterior cerebral artery.

A sagittal sinus venogram (Fig. 1) showed obstruction at the junction of the posterior and middle one-third of the sinus. There was filling of the superior cerebral veins anterior to the point of obstruction. The inferior sagittal sinus, straight sinus and some diploic and scalp veins also filled well with the dye. Venous pressure in the sagittal sinus measured 480 mm. of water.

A retrograde jugular venogram on the right side showed filling of the internal jugular vein only. None of the contrast medium passed into the intracranial veins or dural sinuses. The pressure in the right internal jugular vein at the superior bulb was 32 mm. of water.

A retrograde jugular venogram on the left side showed good filling of the basilar sinuses, including the superior and inferior petrosal and cavernous sinuses, as well as filling of the pterygoid plexus, vertebral veins and deep facial veins. Because of technical difficulties, venous pressure was not measured on this side.

Several weeks later (February, 1950), the posterior one-third of the superior sagittal sinus was

exposed through a craniotomy. A thrombus which completely occluded the sinus was removed with forceps and suction until a brisk flow of venous blood was obtained from both directions. The opening in the sinus was closed with silk sutures and sealed with Gelfoam. Culture of the thrombus was sterile and microsiopic examination showed it to be aseptic. The patient was given anticoagulant therapy with heparin and Dicumarol for 3 weeks postoperatively and she steadily improved. At the end of 3 weeks the spinal fluid pressure had fallen to 200 mm., the decompsessions were soft, the papilledema subsided, diplopia disappeared and headaches were less.

Six weeks after removal of the clot the sagittal sinus venogram was repeated. The venous pressure in the sinus had fallen to less than 300 mm. A block still existed in the same part of the sinus as before, but there was a significant increase in the filling of the anastomotic veins and sinuses. Three months after the clot was removed the spinal fluid pressure was normal and the decompressions soft. A year later, from reports submitted by the patient, there appeared not to have been any return of increased intracranial pressure or development of thrombophlebitis elsewhere in the body.

Comment. This case has been referred to, but less completely described, in previous publications.[10, 11] It was the first case studied and possesses a number of interesting and unusual features, though none out of keeping with the clinical syndrome under discussion. There is no question about the diagnosis of a sterile thrombus in the posterior third of the superior sagittal sinus, and there may also have been thrombosis of one of the lateral sinuses. Since there was no recognizable associated infection in the body, it must be assumed that the thrombotic processes in the right carotid artery and in the intracranial venous system were spontaneous and in the nature of aseptic thrombosis. There is no information about when the carotid thrombosis occurred with relation to the venous thrombosis or, for that matter, how the two conditions are related. It might be reasoned that in the presence of a pre-existing arterial occlusion ve-

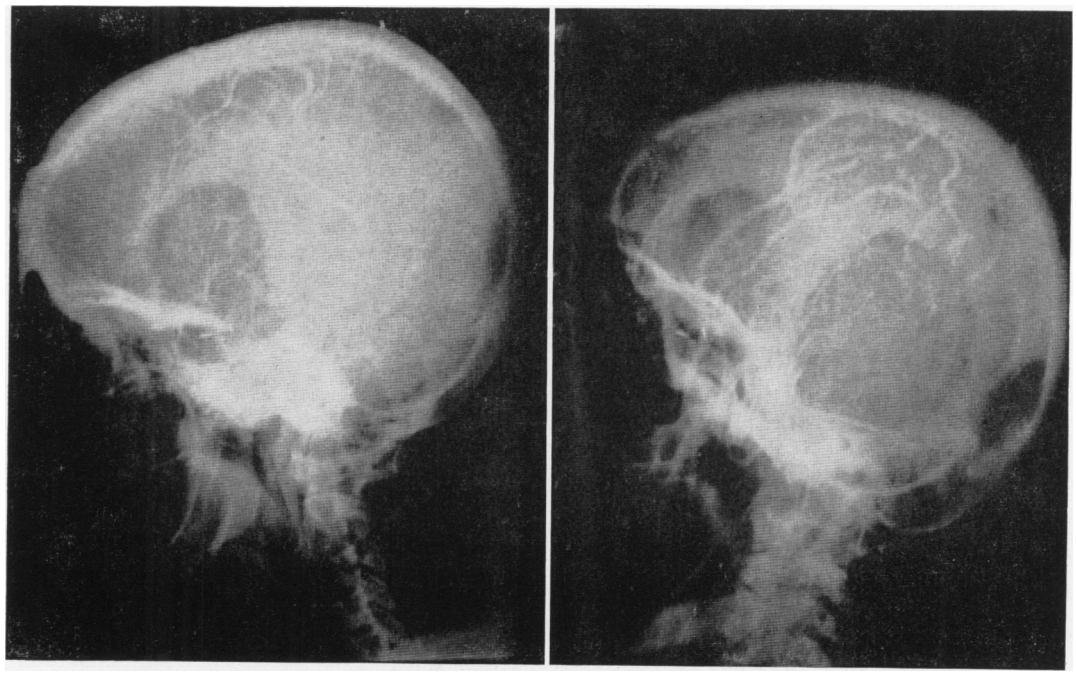

FIG. 1.—Case 1. (A) Lateral venogram before and (B) after removal of thrombus posterior half of the superior sagittal sinus. Thrombus has reformed but filling of collateral channels has greatly increased.

nous thrombosis might be more prone to occur, but it is notable that the carotid obstruction did not embarrass the cerebral circulation to the extent of causing any focal neurologic changes.

Even bilateral subtemporal decompression did not benefit the intracranial pressure to an impressive degree since she continued to have headache, papilledema and steady visual loss 14 months after the onset of her disease and eight months after the decompressions. The opening of the sinus and removal of the thrombus seemed the only procedure that held any hope of improving her condition. While there is ample precedent for such a procedure in operations performed elsewhere in the body, the procedure seems not to have been employed before in the head region except in the evacuation of thrombi from the lateral sinus in conjunction with mastoid operations.

The striking improvement which followed the evacuation of the clot and the administration of anticoagulant therapy at first suggested that the lumen in the sagittal sinus had remained patent. The subsequent venogram showed that this was not the case but the filling of many more venous channels, together with the subsidence of intracranial pressure and of venous pressure in the sinus, indicated that the combined treatment had accomplished some good.

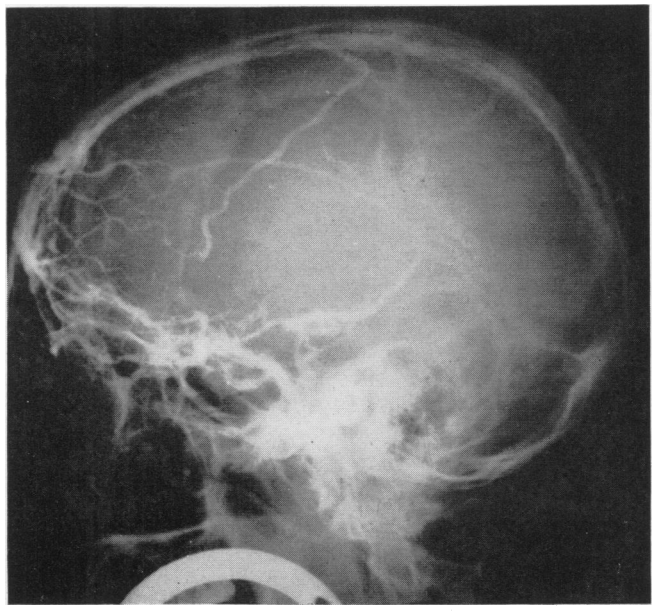

FIG. 2.—Case 2. Lateral venogram showing obstruction (by thrombosis) in the posterior one-third of the superior sagittal sinus.

Case 2.—(J. V., N. Y. H. No. 516917). A 28-year-old man was first admitted to the hospital in August, 1948, with complaints of headache and blurred vision of 3 weeks' duration. The headaches were generalized and constant and, for the first 3 days of his illness, accompanied by slight stiffness of the neck and vomiting. There was no history of infection in the ears or elsewhere in the body and no history of thrombophlebitis.

The examination revealed papilledema of 3 diopters and hemorrhages in both occular fundi. The visual fields were constricted and the blind spots enlarged. Visual acuity was 10/15 bilaterally. The remainder of the physical examination was normal.

The spinal fluid was under 300 mm. of pressure, but was clear and acellular. Repeated lumbar punctures during a three weeks' period of observation revealed similar findings. The protein content of the fluid varied from 26 to 50 mg. per 100 cc. Roentgenograms of the skull and chest were normal. Pneumoventriculograms showed small, but normal, ventricles without displacement or distortion.

A right subtemporal decompression was followed within one month by cessation of headaches and improvement in vision, but by the end of the month the papilledema had not completely subsided and the decompression was full though not tense.

Seven months later the patient returned with symptoms and signs of deep phlebitis in one leg. At this time he was free of his former complaints, the decompression was nearly flat and the spinal fluid pressure was 200 mm. He recovered from the phlebitis of the leg, but in the subsequent 18 months he returned to the hospital 3 times for a perforated peptic ulcer, a schistosomiasis infestation and a recurrence of the thrombophlebitis of the leg that resulted in pulmonary infarction, from which he recovered. Through all these latter illnesses there were no complaints referrable to the central nervous system or any evidence of return of increased intracranial pressure.

In June, 1950, nearly 2 years after his original admission to the hospital, a sagittal sinus venogram (Fig. 2) was performed. It showed a complete block at the juncture of the posterior and middle third of the sinus. The contrast medium filled numerous scalp veins and superior cerebral veins anterior to the block, and also the inferior sagittal sinus. The venous pressure in the sinus was 200 mm. No complications followed the sinus venography.

Comment. Following the experience with Case 1, an attempt was made to persuade patients to return for sinus venography who had had unexplained increased intracranial pressure in the past, and Case 2 represents the only one willing to do so. His initial illness, nearly two years previously, had been typical of the syndrome and he had made a complete recovery aided or not by the decompression operation. The later development of phlebitis in the leg led particularly to the suspicion that he might have had an intracranial thrombosis also.

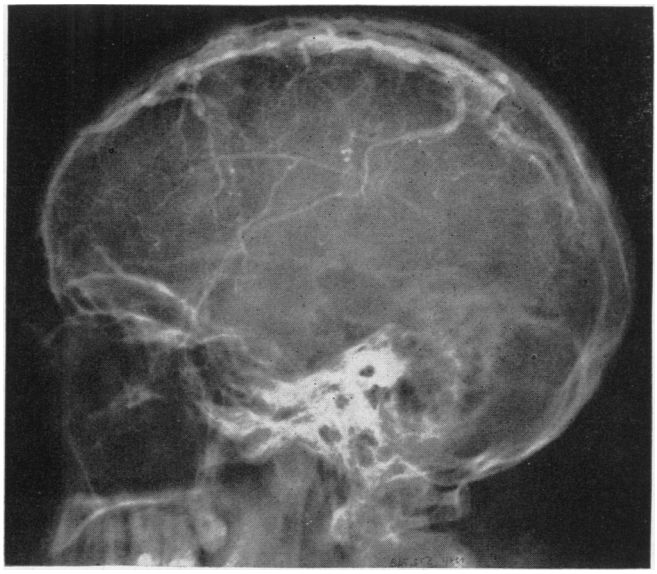

FIG. 3. — Case 3. Thrombosis in the posterior one-half of the superior sagittal sinus. Collateral channels including the inferior sagittal and straight sinuses, cerebral and scalp veins are well filled.

The sinus venogram, two years later, clearly demonstrates the persistence of a block in the posterior third of the superior sagittal sinus so that recanalization of the sinus had not occurred to aid in the recovery. But the clinical recovery and the subsidence of the intracranial and intrasinus pressures had come about through the development of ample collateral and compensatory pathways for venous drainage from the brain.

Case 3.—(W. L., N. Y. H. No. 572748). A 29-year-old man was admitted to the hospital in June, 1950, complaining of several generalized convulsive seizures, beginning in the left arm and leg, in the previous 6 hours. He appeared acutely ill and had a temperature of 39°C. His white blood count was 16,000. The examination showed a left hemi-

paresis and normal ocular fundi. A lumbar puncture showed 180 mm. of spinal fluid pressure; the fluid was clear, colorless, acellular and normal in its protein content.

The convulsions were controlled with barbiturates and standard doses of penicillin were administered. Within 24 hours the temperature had returned to normal, the left hemiparesis had improved and the patient appeared to be recovering rapidly. Further questioning now disclosed that he had had 2 episodes of thrombophlebitis of the lower extremities, one and 5 years previously. He did not show evidence of phlebitis in the extremities at this time.

A presumptive diagnosis of cerebral thrombophlebitis was made, and treatment with Dicumarol instituted. In the following 5 weeks a low grade papilledema appeared and the spinal fluid pressure gradually rose to 300 mm.; but the patient felt well, his hemiparesis had subsided and he left the hospital against advice.

Ten days later he returned to the hospital because of generalized headache and several more convulsive seizures. The spinal fluid pressure was now over 400 mm. but still normal in other respects.

A pneumoventriculogram showed small ventricles in normal position and with normal configuration. A sagittal sinus venogram (Fig. 3) revealed a complete block in the posterior third of the sinus and filling of numerous scalp veins, superior cerebral veins and other collateral venous channels. The venous pressure in the sinus was 500 mm.

Anticoagulant therapy was reinstituted and continued as an ambulatory treatment for 3 months. Within a month after discharge from the hospital he was asymptomatic, had normal ocular fundi and returned to work. Ten months after his original admission to the hospital he was free of any symptoms or signs suggestive of intracranial disease, but had recently had a transient recurrence of phlebitis in one leg.

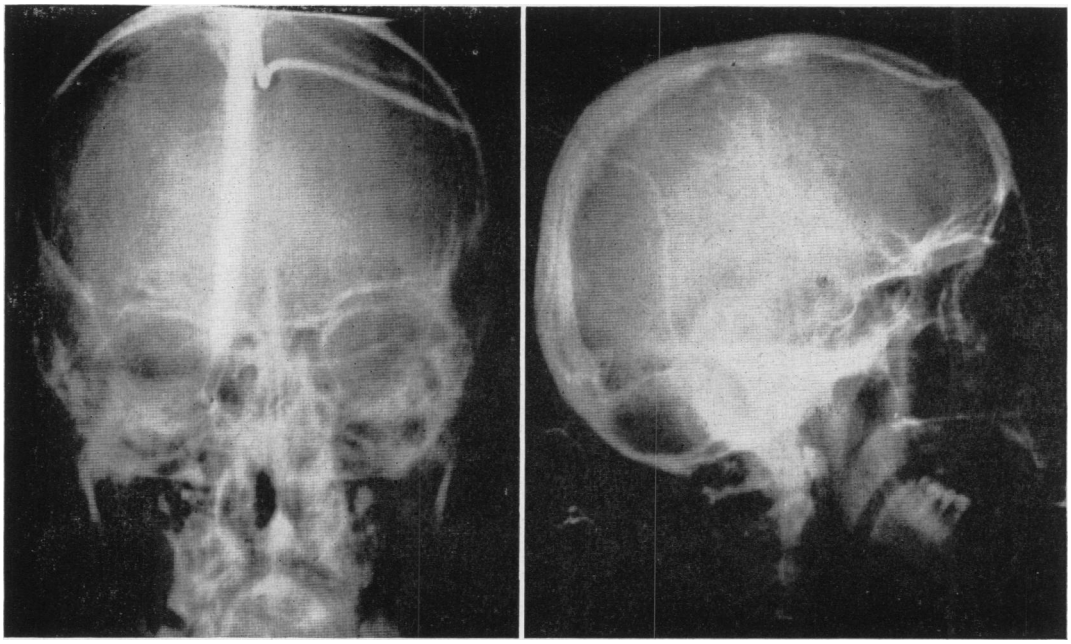

FIG. 4.—Case 4. Lateral and AP venograms showing obstruction by thrombosis in the dominant right transverse sinus and development of collateral circulation. The left transverse sinus is extremely small.

Comment. The history of thrombophlebitis in the legs prior to the onset of acute symptoms of cerebral disease simplified the initial diagnosis of intracranial thrombosis. The point should be made in this case, however, that the focal convulsions and hemiparesis, without increased intracranial pressure at the onset, suggest that the thrombosis began in cerebral veins or possibly in the wall of the superior sagittal sinus at the openings of cerebral veins. If the latter obtained, it must be assumed that in the be-

ginning the lumen of the sinus was not blocked since there was no increase in intracranial pressure until some days later.

Dicumarol, given for its anticoagulant effect, and penicillin for its antibacterial effect, were seemingly well suited in the treatment of this case, since it may be assumed because of the acute febrile onset that he had a thrombophlebitis. Cases of this kind should be easier to recognize than the more benign and insidious phlebothrombosis, yet the similarity in the effect on the dynamics of the intracranial venous system and secondarily on the intracranial pressure cannot be denied.

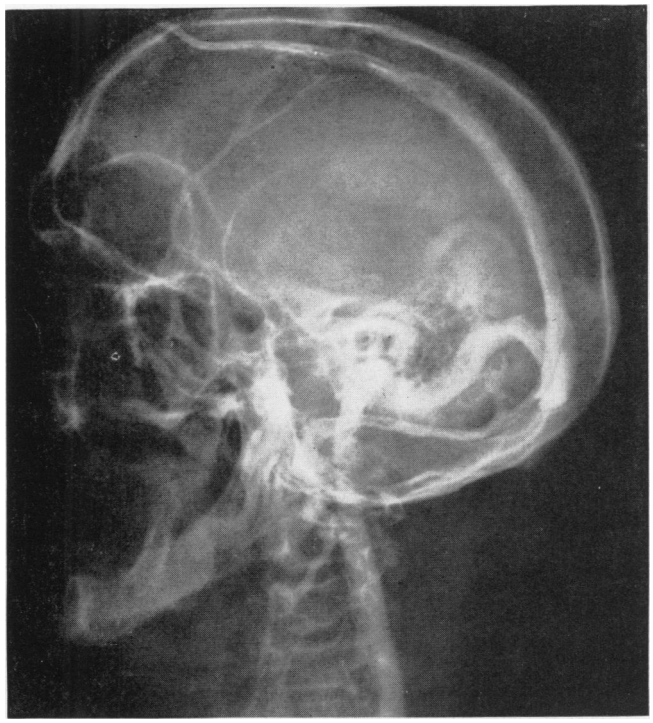

FIG. 5.—Lateral venogram showing thrombosis in the right transverse sinus (sigmoid portion). The left transverse sinus is huge and collateral circulation has not developed.

Case 4.—(P. Z., N. Y. H. No. 581433). A 33-year-old man was admitted to the hospital in November, 1950, with a history of earache and purulent discharge from the ear canal on the right side for 2 weeks. On the day of admission he had developed a high fever (40°C) and a blood culture taken on that day was positive for *Proteus vulgaris*. Roentgenograms showed evidence of acute right mastoiditis.

Antibiotic treatment was instituted and a right mastoidectomy performed, at which time the transverse venous sinus was found to contain an infected thrombus which was not removed. There was rapid improvement in the patient's condition, but within a few days he began to experience blurred vision and generalized mild headache. A funduscopic examination disclosed the presence of about 2 diopters of papilledema and a few small retinal hemorrhages. The neurologic examination was otherwise normal. The spinal fluid pressure was 400 mm. and the fluid was clear, colorless and acellular.

A sagittal sinus venogram (Fig. 4) showed complete obstruction of the right transverse sinus just lateral to the torcular herophili and a small left transverse sinus. The contrast medium entered some of the scalp veins and superior cerebral veins. The venous pressure in the sagittal sinus was 320 mm.

The patient improved gradually. The headache, blurred vision and papilledema subsided steadily and had disappeared after two months. There were no recurrent symptoms in the subsequent six months.

Comment. This case corresponds to that variation of the syndrome more commonly referred to as "otitic hydrocephalus," since there is an associated ear infection. Symonds,[5] who proposed the name, concluded

that obstruction of the transverse dural sinus is not a factor. He tended to explain the "hydrocephalus" on the basis of extension of mural thrombi from the transverse sinus to the superior sagittal sinus, with resultant interference with cerebrospinal fluid absorption through the arachnoid villi.

In this case, it is shown that the obstruction is limited to one transverse sinus (in this case the right) while the superior sagittal and other transverse sinus are both patent. But the increased venous pressure in the sagittal sinus, the increased intracranial pressure and the development of collateral circulation through the cerebral veins all point to the inadequacy of the remaining transverse sinus to compensate for the obstruction of the other transverse sinus. This reasoning is in keeping with evidence that occasionally the ligation of one internal jugular vein, as in a radical neck dissection, may lead to increased intracranial pressure if the transverse sinus on that side is the dominant one and the transverse sinus on the opposite side happens to be unduly small or absent.

On the other hand, if the obstruction or thrombosis occurs in a small transverse sinus, the opposite relatively large transverse sinus provides an adequate channel for drainage of the venous blood from the superior sagittal sinus and increased intracranial pressure does not develop. Figure 5 is a venogram in such a case. This patient's spinal fluid and sagittal sinus venous pressures remained normal and there was no papilledema.

There undoubtedly are some cases that fall in the group of otitic hydrocephalus in which the thrombosis involves the superior sagittal sinus alone or in conjunction with a transverse sinus, and possibly other cases in which both transverse sinuses are involved, but the sagittal sinus spared. But the important point is that the increase in intracranial pressure is purely mechanical as a result of obstruction of the intracranial venous system and has little or nothing to do with spinal fluid over-production or reduced absorption.

SUMMARY AND CONCLUSIONS

The study of these four cases brings to attention several variations in the clinical pattern of thrombosis of the dural venous sinuses which results in increased intracranial pressure. Case 1 exemplifies the protracted and remitting aspects sometimes seen in the disease. Case 2 is more nearly like the majority of reported cases of increased intracranial pressure which develop insidiously, cause little or nothing in symptoms or signs except the secondary effects of the increased pressure, and subside within two or three months. Case 3 typifies the acute onset sometimes seen with cases in which there are fever and convulsions followed some days later by gradually increasing intracranial pressure as the acute stage subsides. Case 4 is representative of "otitic hydrocephalus" in that the syndrome of increased intracranial pressure is associated with or preceded by an ear infection.

There must be other variations in the syndrome, depending principally on whether the thrombus is aseptic (phlebothrombosis)or infected (thrombophlebitis) and on how rapidly and extensively the occlusion of the venous system occurs. It is believed that the occurrence of convulsions or palsies implies the thrombosis of cerebral veins initially or by extension from the primarily thrombosed sagittal sinus. If the lumen of the posterior portion of the superior sagittal sinus is quickly occluded by the thrombus the lag in development of collateral venous channels accounts for the more rapidly developing and severe aspects of increased intracranial pressure. Surgeons are aware of the danger to life that accompanies division or resection of the patent posterior half of the superior sagittal sinus and the relative safety of such a procedure if the sinus has been gradually occluded by

tumor and collateral circulation has had an opportunity to develop. An extensive or extending thrombus, by diminishing the possibilities for development of collateral circulation, would result in more protracted increased intracranial pressure. We have seen one patient with the syndrome, but with the diagnosis unproved, who still had papilledema and elevated spinal fluid pressure four years after the onset of his illness. Dandy[7] reported a similar case in which the patient's vision had become greatly impaired and whose symptoms had been present for five years when first seen by him.

More emphasis has been made in the past on the dire effects that may be expected from occlusion of the posterior portion of the superior sagittal sinus than to the possible similar effects that may occasionally attend the occlusion of one of the transverse sinuses. Attention has been called (Woodhall)[12] to the variation in size and importance of the two transverse sinuses and to the occasional absence of one transverse sinus. Numerous discourses indicate that the sinus on the right side is the dominant one for drainage of the superior sagittal sinus and its tributary veins, though studies thus far with sinus venography do not entirely support this assumed disproportion. But the fact remains that thrombosis in one transverse sinus will lead to increased venous pressure and secondarily to increased intracranial pressure if the other transverse sinus is absent or inadequate to serve as a channel for drainage of the superior sagittal sinus.

Additional experience is needed to throw light on the significance of the Tobey-Ayer[13] test in the presence of thrombosis of the supeior sagittal sinus. Dandy,[7] in the discussion of his reported cases with the syndrome, concluded that thrombosis causing occlusion of the large venous sinuses could be ruled out by this test. It is to be expected that thrombosis of a transverse sinus, particularly if the thrombus extends to the jugular bulb, may abolish or diminish the response of a rise in intracranial venous pressure when the internal jugular vein is compressed on the same side. But the circumstances are different in the case of thrombosis of the sagittal sinus, since the collateral circulation which is set up is dependent on the patency of the jugular veins and is further facilitated if the transverse sinuses are also patent. Thus the Tobey-Ayer[13] test is of limited value in the diagnosis of this syndrome.

Recovery from thrombosis of the posterior portion of the superior sagittal sinus or of a dominant transverse sinus appears to be largely dependent on the development of collateral channels for venous drainage of the brain. Possibly recanalization of the thrombosed sinus may sometimes play a part, but in two of the cases studied (Cases 1 and 2) the late venograms do not indicate that recanalization of the sinus occurred to contribute to the recovery.

The anastomotic venous channels available for the development of collateral circulation are voluminous and in our opinion are the most important factors in the recovery. These channels include the following: (1) diploic and scalp veins draining into the external jugular system; (2) anastomosing superior cerebral veins which short circuit about the obstruction in the sinus; (3) veins in the falx cerebri which connect the superior and inferior sagittal sinuses; (4) veins extending forward from the superior sagittal sinus into the orbital and facial veins; (5) anastomotic veins of Labbé and other inferior cerebral veins connecting with the transverse, cavernous and petrosal sinuses; and (6) more remotely, the straight sinus, occipital sinus, vertebral veins and pterygoid veins. Individual variations in the size and presence of these collateral pathways must exist just as in the transverse sinuses, where variations are known to be common.

While spontaneous recovery may be expected of most patients with thrombosis of the dural sinus, subtemporal decompression has undoubtedly been helpful at times in minimizing headache and the threat to visual loss that exists in the cases with prolonged increased intracranial pressure. In suspected cases, sinus venography should be employed early without fear of its causing complications, and if the diagnosis is established, anticoagulant therapy should be instituted. Thus far, heparin and Dicumarol have been employed for this purpose. Although a larger experience is desirable before attempting to evaluate this treatment, it appeared to have been useful in Cases 1 and 3. The treatment is directed at prevention of extension of the thrombus and thus should have greater advantage the earlier it is instituted. The length of time the therapy should be continued is questionable, but under proper control it might reasonably be extended to a period safely beyond the time when collateral circulation becomes established and intracranial pressure begins to fall.

If, in the occasional case, there should be progression of the disease in spite of anticoagulant therapy or inadequate improvement with its use, there may be an indication for surgical removal of the thrombus, as was employed in Case 1. While the lumen of the sinus apparently again became occluded by reformation of the thrombus, the extent of the secondary thrombus appears not to have been as great.

When the thrombus is infected (thrombophlebitis) the onset of symptoms should be characterized by acute illness with chills, fever and leukocytosis in the blood. At other times, when infection exists simultaneously or previously in other parts of the body, it should be suspected that the intracranial thrombosis is infected or may become so. In these cases anti-bacterial therapy should be instituted but, if possible, not until a blood culture has been made in an effort to establish the identity of the infecting organism.

It is only fair to say that although it may reasonably be expected that the majority of cases heretofore classified as increased intracranial pressure of unknown etiology will, with sinus venography, prove to be thrombosis of the superior sagittal or transverse venous sinuses, much more experience is necessary. In the investigation of suspected cases, the venogram should be reserved for the last step in the examination. It is best first to establish by pneumoventriculography and other tests that the clinical picture is not due to infection of the brain or meninges or to a space-occupying lesion before looking for evidence of sinus thrombosis by sinus venography. The ventricles are normal, or possibly small, but of normal configuration, and enlargement of the ventricular system should lead to a suspicion of some diagnosis other than thrombosis of a venous sinus. The term "hydrocephalus" applied to the condition under discussion is a misnomer.

The increased intracranial fluid pressure accompanying thrombosis of the superior sagittal sinus or a dominant transverse sinus is largely, if not wholly, the result of increased intracranial venous pressure. Recovery is dependent on the development of adequate collateral channels for venous drainage of the brain. To this end, anticoagulant therapy may be helpful, particularly if employed early, and in infected thrombi anti-bacterial therapy should be used as well. Subtemporal decompression may still be of advantage in the occasional case and there may also prove to be an advantage to surgical removal of the thrombus in the protracted case.

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