The details of pre-operative treatment, operation, and post-operative care are described, and subsequent injection treatment is outlined.

The whole varicose circulation can be completely obliterated in from six to eight weeks from the time of operation, and the patient rendered fit even for heavy work.

Patients with the severe type of varicosity should be reviewed at intervals of a year or so whenever possible.

The factors governing prognosis are outlined. Of the controllable factors the most important is the completeness of the obliteration of the varicose circulation.

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THROMBOSIS IN THE SUPERIOR LONGITUDINAL SINUS FOLLOWING CHILDBIRTH

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Thrombosis in the superior longitudinal (sagittal) sinus has long been known to pathologists as one of the conditions that may give rise to fatal cerebral symptoms in the puerperium or after abortion. With the exception of a case reported by Symonds in 1937 little has been written in English on the subject, for a good many years past, but five cases were published in the *Zeitschrift für Gynäkologie* between 1925 and 1928, and these and seven others were collected by Mondré (1928). Other verified instances are to be found among the numerous cases of paralysis associated with pregnancy collected by von Hösslin (1904, 1905). In none of these instances was the diagnosis made during life. Headache and gradually increasing coma were the outstanding symptoms; paralysis was not always observed; convulsions occurred in some cases; and amaurosis is mentioned by Zangemeister (1925) in his description.

Within the last few years cases in which the superior longitudinal sinus is obstructed have become subject to clinical diagnosis. This has resulted partly from observation of these puerperal cases, but chiefly it has been a consequence of Symonds's studies of "otitic hydrocephalus"—another condition in which this sinus is thrombosed. Now that they can be recognized clinically it appears that puerperal cases of sagittal sinus thrombosis end more often in recovery than in death, but so far only one non-fatal case has been published (Symonds, 1940; Martin and Sheehan, 1941). Clinical accounts are given here of three cases which are believed to be of this nature: death ensued in one of them, but it was not due to the thrombosis in the sagittal sinus. In addition to these three I have seen during the last five years two cases in which similar symptoms were associated with stillbirth, a case following abortion in which the right cavernous sinus was evidently thrombosed, and a case of long-standing residual disabilities from a puerperal illness which must have involved very extensive thrombosis in the superior longitudinal sinus.

The moment is appropriate for consideration of these cases because Batson's work, recently published, has provided the probable explanation of the means by which thrombosis following childbirth or abortion is determined to this seemingly extraordinary site.

Applied Anatomy

The diagnosis of thrombosis in the superior longitudinal sinus depends on the signs of raised intracranial pressure and the signs of obstruction of superior cerebral veins.

Signs of Raised Intracranial Pressure.—It will be recalled that the sinus is triangular in cross-section (see diagram).

Diagram showing the superior longitudinal sinus in cross-section.

In many subjects numerous trabeculae cross it, and in some instances a continuous membrane divides it horizontally for a portion of its length (O'Connell, 1934). At its upper angles irregular lateral diverticulae—the lacunae laterales—contain within their walls numerous Pacchionian bodies through which the cerebrospinal fluid is absorbed into the blood stream. If the circulation in the sinus is obstructed, or if the upper story of the sinus becomes filled with clot, absorption of the cerebrospinal fluid is prevented and the intracranial pressure consequently rises, with the production of headaches, papilloedema, and perhaps vomiting. The significance of this hydrocephalus as an indication of sinus thrombosis was recognized by Symonds (1937).

Signs of Obstruction of Superior Cerebral Veins.—The sinus receives the superior cerebral veins, and these enter its walls near the inferior angle. If a thrombus occludes the whole cross-section of the sinus opposite the entrance of a vein, or if it is situated in the trough of the sinus or extends down into the trough, it may obstruct one (or more) of these veins. Thrombosis of the affected vein is then likely to occur. Many thrombi, however, are not so situated as to obstruct veins. Moreover, it seems that an obstructed vein does not invariably become thrombosed, and this is doubtless because of the easy collateral circulation. When thrombosis of an obstructed vein occurs it gives rise to paralysis corresponding to the area of cerebral cortex which the vein drains, and this paralysis may or may not be ushered in by a series of convulsions: Convulsions seem to be less usual when venous thrombosis is secondary to obstruction in the sinus than they are when a vein is thrombosed primarily. By no means all cases of thrombosis of the superior longitudinal sinus are recognizable in the present state of our knowledge, and it is certain that a large thrombus may exist in the sinus without giving rise to any clinical signs.

Case I

This is an instance of the most straightforward type of case.

Fits seventeen days after delivery; right hemiparesis; papilloedema; complete recovery.

An 8-para aged 40 was confined on June 25, 1936, labour being normal and the puerperium uneventful. During the night of July 12-13 she had four fits, and next day was admitted to hospital. She was then conscious, but in a stuporous state, was very drowsy, and barely understood what was said to her. She had paresis of the right arm and slight drooping of the right side of the face; there was also slight weakness of the right leg, and the right plantar reflex was extensor. The temperature on admission was 100° F. and the pulse rate 110. There was no neck rigidity. Examination of the heart disclosed nothing abnormal. The blood pressure and urine were normal, as were

the lungs and abdomen. Lochia had ceased. The C.S.F. was normal except that it contained a considerable number of red blood cells; but it was thought to be under increased pressure, and examination of the ocular fundi a few days later showed slight swelling (1 D) of both optic disks and a few small haemorrhages over the left one.

On the second morning after admission the patient had several more fits, with twitching beginning in and being chiefly confined to the right arm. The next day she was conscious enough to obey simple commands; her right arm was then found to be almost completely paralysed and the weakness of her face and leg had increased. Her pupils were unequal, the right being the greater. Her temperature was normal. A week later she was very much better. She was quite alert and clear mentally, her right arm had regained a great deal of its power, and her right leg was normal. Her papilloedema, however, remained, and fresh haemorrhages occurred, this time at the side of the right disk. A fortnight after the onset her right arm could be considered normal. As soon as she was well enough she complained of pain in the right leg, but nothing abnormal was seen on examination of it. Later, when after nearly eight weeks she began to get up, this leg swelled, and it is possible that there had been some thrombosis in the deep veins.

She was discharged from hospital, feeling well, after three months, but it was another six weeks before her ocular fundi were passed by the ophthalmologist as clear.

Comment.—This case showed symptoms of the two groups mentioned. The sudden onset of convulsions in the right side, followed by right hemiplegia and accompanied by the presence of a small quantity of blood in the C.S.F., can be attributed to thrombosis of a vein on the surface of the left cerebral hemisphere. The symptoms and circumstances are identical with those in certain puerperal cases described by Martin and Sheehan (1941), in which such thrombosis of veins was verified post mortem. The co-existing papilloedema showed that the intracranial pressure was considerably raised, and this would not be caused by thrombosis of veins alone. It is, however, accounted for by prevention of the absorption of cerebrospinal fluid by clot in the superior longitudinal sinus. The differentiation from cerebral tumour could not be made with certainty in the early stage, but the blood cells in the C.S.F. and the association with childbirth were points greatly in favour of thrombosis, while the normal protein content of the C.S.F. was a point against tumour. With cerebral arterial embolism, thrombosis, or haemorrhage there would have been no papilloedema.

Case II

Femoral thrombosis seven days after delivery. Two weeks later, abrupt onset of visual disturbance and headache, followed by left hemiplegia; papilloedema; considerable recovery, but death in consequence of a spinal lesion.

The patient, aged 43, was delivered of her seventh child on June 12, 1938. On June 19 she had thrombosis in her left thigh; this was treated with hot fomentations, and she got up on June 25. On July 3 she went to church, and on returning home "felt a blackness behind the eyes" and had a headache. She went to bed and slept. The next morning on waking she could not see, and could not move her left arm and leg satisfactorily.

The patient was admitted to hospital the same day. She was then unable to see light and her pupils were dilated and did not react to light. She had weakness of the left arm and slight weakness of the left side of the face. The tendon reflexes were exaggerated on the left side; the left plantar reflex was extensor and the right one was weakly extensor also. Nothing abnormal was found in the heart, lungs, abdomen, or urine. The lochia had ceased. On July 8 she developed signs of a spinal lesion at the sixth dorsal segment, with complete paralysis of both legs, loss of sensation up to the lower ribs, and incontinence of urine and faeces. Meanwhile some vision had returned, but her optic disks were swollen and there were haemorrhages on the right

one. Paralysis of the left arm had become almost complete. Mentally she was rational and fairly alert. A week later her sight was much better and the visual fields seemed full, but the papilloedema remained. During the next few weeks the condition of the patient's left arm improved greatly, but as a result of the spinal paralysis her general condition gradually deteriorated, and she died on September 28, 1940. No post-mortem examination was made.

Comment.—Signs of intracranial pressure (papilloedema and headache) and hemiplegia indicated thrombosis of the superior longitudinal sinus. The sequence of events suggests that a clot present in the sinus had suddenly occluded it and that extension of the clot had afterwards gradually obstructed the mouth of a vein which drained the motor area of the right cerebral hemisphere.

The severe disturbance of vision at the onset of the illness calls for some comment. A somewhat similar disturbance will be described in the next case also, and, as has already been mentioned, amaurosis occurred in at least one of the German cases in which massive thrombosis of the superior longitudinal sinus was found post mortem. It seems most likely that this amaurosis is due to acute venous congestion of the optic disks consequent upon the abrupt rise of intracranial pressure which results from occlusion of the longitudinal sinus. There is no reason to suppose that the ophthalmic veins are affected more directly by thrombosis in this sinus: the disturbance cannot be satisfactorily explained by postulating venous lesions or congestion in the occipital lobes, nor would such a supposition account for the loss of the pupillary reaction to light which was observed in this case. Here and in Case III the sight was largely restored within a week or two while papilloedema was still present. I have encountered similar, though less severe, amaurosis in another case of sinus thrombosis in which the intracranial pressure was 200 mm., and in that instance also the vision quickly improved, although the intracranial pressure remained high and papilloedema persisted. We must suppose that after the sudden onset circulatory conditions within the nerve head adapt themselves in spite of the continuance of a high intracranial pressure.

In nearly all the cases of primary venous thrombosis described by Martin and Sheehan convulsions marked the onset of the illness and ushered in the paralysis, but in this case the hemiplegia developed without convulsions. There is some reason to believe that convulsions are less common if the sinus is occluded before the vein. An explanation of this may possibly be found in the fact that obstruction in the sinus leads to opening up of the collateral venous circulation, so that when the mouth of a vein becomes blocked at the sinus the vein itself may not become thrombosed and venous congestion in its drainage area may be much less intense. The absence of convulsions may, however, simply be due to the gradualness of the onset of the paralysis and of the obstruction causing it.

The diagnosis in Case II admits few alternative suggestions. The suddenness of the onset strongly indicates a vascular lesion, and there are not many that would cause papilloedema. In particular, arterial embolism would not account for it.

The spinal lesion is an unusual complication on which I do not wish to comment here, beyond stating that it, too, was probably due to venous thrombosis, and its occurrence does not modify the diagnosis of the cerebral condition.

Case III

In this case the signs of cerebral venous thrombosis were not at first evident. Some clinically similar cases that were fatal are recorded in the literature.

Sudden onset of stupor. Amaurosis, headache, vomiting, papilloedema. Word-blindness. Recovery with residual symptoms.

This patient was confined on August 28, 1938, and ten days later one leg (? which) became painful and remained so until her admission to hospital on January 1, 1939. Both legs were then greatly swollen, and there were tender veins in the right thigh and in the left calf.

On January 12 she was found in a stuporous condition. When roused she carried out simple commands but "rambled." No paresis of her limbs was found. The next day she was still more drowsy and could not be made to understand what was said to her. She had no paralysis. The drowsy and stuporous state lasted for more than a week. Occasionally she mumbled incoherently. Eight days after the onset she was more sensible and said she had a headache. She complained that her sight was "faint" and that she could not recognize the ward sister. She vomited twice on that day (? before), and the margins of her optic disks were blurred. The drowsiness gradually passed off in the course of another week; but headache continued, and she still complained that her sight was misty, though it had improved. Her general condition and her sight gradually became better, and by the end of another month she seemed very well, but was unable to read; she could write, but could not read what she had written (word-blindness). Her acuity of vision was about normal and she could recognize small objects at a distance; her visual fields were full. When she left hospital after a stay of twelve weeks her papilloedema had entirely subsided.

Eighteen months later she was readmitted, having had two fits and become weak in her legs. Her word-blindness was still present. Her legs were slightly weak and spastic, with typical reflex signs, and her abdominal reflexes were absent. She was discharged much improved after a fortnight.

Comment.—In this case the connexion with childbirth is less immediate, but the series of events is continuous. During the first admission her superior longitudinal sinus was probably obstructed by clot, and hydrocephalus was thus caused; to this obstruction her prolonged drowsiness or stupor must be attributed. Her earliest visual disturbance was probably due, as has been said, to venous congestion of the optic nerve heads. It would seem that about the same time at least one superior cerebral vein was blocked, thereby giving rise to her word-blindness. In this case also the absence of convulsions is to be noted. Eighteen months later further thrombosis evidently occurred, causing some venous obstruction more anteriorly, with consequent temporary weakness of the legs. Two features of interest are the presence of a permanent residual disability and the occurrence, after a prolonged interval, of signs suggestive of further thrombosis. I have seen two other probable cases with severe residual disabilities, and in both of them the history indicated that there had been more than one acute attack.

Pathology

The form of the clot in the longitudinal sinus which may give rise to such symptoms is subject to great variation. In a number of the fatal cases a massive clot has been found filling the whole sagittal sinus and even the lateral sinuses as well (Mondré, 1928), but we can exclude such conditions in considering these less severe cases. There may, however, be a massive clot filling a portion of the sinus. Alternatively there may be multiple mural thrombi, some becoming large enough to obliterate the lumen, as in Symonds's (1937) case and in several of the older instances (Collier, 1891). Martin and Sheehan described a long "rat-tail" thrombus swimming free in the blood stream, except for an attachment to the wall of the sinus at one point, and such a clot might enlarge until it occluded the sinus. Other conditions which may occur have been described in non-puerperal cases—e.g., a continuous clot along the roof of the sinus, leaving the trough unobstructed (Bailey and Hass, 1937), and a layer of

clot on the walls, with a channel for blood persisting in the centre (Gowers, 1892-3).

The primary thrombus is usually in the longitudinal sinus, but it may be in one of the lateral sinuses, or clot may extend into the longitudinal sinus from a vein (Virchow).

Diagnosis and Clinical Variations

The principles for the diagnosis of clot on the superior longitudinal sinus have already been given, and if signs of raised intracranial pressure and signs of cerebral venous thrombosis coexist its presence may be assumed. Granted that there is no severe heart disease to provide a source of emboli and that the blood pressure is within normal limits, venous thrombosis is the most probable cause of hemiplegia occurring during the puerperium (von Hösslin). If this be remembered, and if papilloedema be looked for, the diagnosis will seldom be missed. Cerebral tumour is the most likely condition to cause confusion, and it may not always be possible to exclude it from the diagnosis at the beginning. A little blood in the cerebrospinal fluid on microscopical examination is strongly in favour of thrombosis, while with tumour an increase of the protein content is usual. Meningitis may have to be considered, and this can be excluded by examination of the cerebrospinal fluid.

It is not necessary for the diagnosis that both the principal groups of signs should be present. As with otitic hydrocephalus, there must be cases in which no thrombosis of veins occurs. The sudden onset in the puerperium of signs and symptoms of increased intracranial pressure should therefore arouse a strong suspicion of thrombosis in the longitudinal sinus, especially if they are accompanied by a drowsy or stuporous state. In a severe case of this kind the symptoms would be the same as in Case III without the residual word-blindness—namely, drowsiness or stupor, headache, amaurosis, and papilloedema. On the other hand, there are cases in which the intracranial pressure is not raised and the symptoms are exclusively those of venous thrombosis. If the symptoms merely indicate thrombosis of a single vein it is impossible to carry the diagnosis further, although clot may be present in the sinus; but if there is evidence of two or more superior cerebral veins becoming thrombosed, and especially if they are on opposite sides of the brain, there is a strong presumption that the veins are being obstructed by clot in the sinus. The validity of this deduction is illustrated by the fatal case described by Symonds (1937), in which there was paralysis first of the left arm, then of the right side of the face, and later complete left hemiplegia; there was no papilloedema; the cerebrospinal fluid was slightly bloodstained but its pressure was normal; post mortem there were mural thrombi of different ages in the longitudinal and lateral sinuses.

In cases with less extensive clot there may be neither any severe rise of intracranial pressure nor venous obstruction, and in the present state of our knowledge such cases are not recognizable. Some impairment of C.S.F. absorption may cause headache and nothing else, but there must be a number of cases in which relatively small clots on the walls or roof of the sinus give rise to no symptoms at all.

Treatment

When symptoms have appeared the treatment that is indicated is (1) to allay convulsions if they are present, (2) to reduce intracranial pressure, and (3) to prevent further thrombosis.

To allay convulsions a dose of three grains of luminal should be given, by mouth or by injection according to whether the patient is conscious or not. It may be repeated

as required at intervals of six hours. For the reduction of intracranial pressure the same palliative measures may be used as in cases of cerebral tumour—namely, the intravenous injection of glucose solution (50 to 100 c.cm. 50%) or an enema of concentrated magnesium sulphate solution (3ij-3iv). Symonds (1937) used frequent lumbar punctures in cases of sagittal sinus thrombosis resulting from ear disease; and in the first few days, especially if amaurosis is present, this is perhaps the most advisable procedure, because the pressure of the cerebrospinal fluid can be measured at the time of the puncture and can definitely be reduced temporarily to a normal level; it should be brought down to 100 mm. To prevent the thrombosis spreading the only means at our disposal is the administration of heparin, and of this I have no personal experience. In recent articles Dockeray and Kawerau (1940) and others have described the giving of this drug by the method of continuous intravenous drip, but if this method is not available repeated injections may be used.

Aetiology

In the active period of life cases such as these are extremely rare apart from child-bearing, and the association with child-bearing is so constant that it cannot be mere coincidence. Difficult labour, manual removal of the placenta, post-partum haemorrhage, uterine sepsis, and other complications of delivery can be exonerated as causes except in so far as they may give rise to pelvic or femoral thrombosis. In nearly all the obstetrical cases of longitudinal sinus thrombosis that I have seen there has been evidence of venous thrombosis elsewhere in the body, and the same has applied in many, if not all, of the fatal cases that have been described. Can it be that the clot in the longitudinal sinus is in some way "metastatic"—that some fragment transmitted from elsewhere forms the nucleus of a new clot? The superior longitudinal sinus would be a likely place for any element carried in the blood stream to settle, because the flow of the blood there is so slow that it would probably be deposited, and because the walls of the sinus are rough and irregular, with many small depressions in which it might lodge.

I had written this paper and stopped baffled at this question when I became acquainted through an annotation in the *British Medical Journal* with certain findings of Batson (1940). This worker, interested in the distribution of metastases from prostatic carcinoma, investigated the connexions of the pelvic veins by injecting radio-opaque material into them. He found that in addition to their known connexions with the caval system, these veins had free anastomosis with the veins in the vertebral canal and around the vertebral column, and that when material was injected into the veins in the pelvis of the cadaver this vertebral plexus of veins provided a free pathway for it right up to the dural sinuses. "We . . . expect to find fairly well filled such veins and sinuses of the cranial cavity as the superior longitudinal sinus, the cavernous sinus, Trolard's anastomotic vein, and others." Carrying this investigation further, Batson injected radio-opaque material into the dorsal veins of the penis of a monkey and showed that it followed the same paths as in the cadaver. If the flow to and in the inferior vena cava was obstructed by compression of the abdomen by means of a towel tied round the animal, some of the thorium medium passed into the vertebral system of veins, and could be followed in the x-ray films upwards past the zone of compression. Batson deduced from his experiments that during coughing and straining blood was actually squeezed out of the abdominal (and pelvic—J. P. M.) veins into the vertebral vein system. In the parturient woman the conditions for the upward passage of any blood-borne fragment are still more favourable than in Batson's monkeys. In

fact, when she strains on the bed-pan they seem to be simply ideal. Because of the movements of the bowel and bladder and her active straining, that is the time when a fragment of clot is most likely to be dislodged; because of her straining, to which is added compression of her abdomen by a firm binder, such a fragment would be directed into the vertebral vein system and propelled upward by the movements of blood induced by her repeated straining. As if those factors were not enough, her posture on the bed-pan calls in gravity to aid the process. It is well known that fragments of clot are liberated from thrombosed pelvic or femoral veins and give rise to pulmonary embolism, and it would be remarkable, under the conditions prevailing, if they did not sometimes pass into the vertebral venous system, and occasionally reach the cranial cavity.

In a cerebral vein a fragment of clot might cause actual embolism (plugging), but in the longitudinal sinus it obviously cannot do so, since all the means of entrance into the sinus are narrower than the channel itself. It is therefore necessary to suppose that a deposited fragment would serve as the nucleus of a new clot. Batson's work, however, obviously provides an explanation also of those puerperal cases in which thrombosis is limited to cerebral veins and those in which sinuses other than the superior longitudinal are involved.

The occurrence of a spinal lesion in my second case receives its explanation, too, from the hypothesis that fragments are transmitted by way of the vertebral veins if we suppose that thrombosis first took place in the extrathecal veins at the affected level and consequently spread into the spinal veins.

The prophylactic measures which these considerations suggest are obvious. When a patient is suspected of having pelvic thrombosis every precaution should be taken to avoid straining, the binder should not be tight, and the patient should be kept well propped up in bed.

Summary

Thrombosis in the superior longitudinal sinus occurs in the puerperium and after abortion.

The clinical diagnosis depends on signs of raised intracranial pressure and signs of obstruction of superior cerebral veins. Under certain conditions the diagnosis may be made in the presence of only one of these groups of signs.

Clinical accounts are given of three cases which are believed to be of this nature.

It is suggested that a fragment of clot carried up to the superior longitudinal sinus from the pelvic veins by way of the vertebral venous system acts as the nucleus for the clot which forms in the sinus.

All the cases described in this paper were seen at Whipps Cross Hospital, and I am indebted to the medical superintendent, Mr. O. R. M. Kelly, for permission to describe them.

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J. H. Levy (*Tri-State med. J.*, 1941, **13**, 2642) states that diabetic neuritis, the incidence of which according to different observers ranges from 0.6 to 57.3% of the total number of diabetics, is probably not dependent upon vitamin B₁ lack. Supplementary or properly organized diet with the vitamin in pure chemical form is therefore not indicated.