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PRIMARY THROMBOSIS OF CEREBRAL VEINS (FOLLOWING CHILDBIRTH)

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Using Gowers's (1892-3) classification, thrombosis of a cerebral vein is "primary" if it is due to "the state of the blood and circulation generally" and is not consequent upon disease adjacent to the brain or venous sinuses—e.g., in the ears. Evidence is gradually accumulating that primary thrombosis of a vein (or veins) on the surface of the brain is not a very uncommon condition. Few uncomplicated cases have, however, been described in which its occurrence has been established by post-mortem examination or by exploratory operation, and its clinical and pathological features are not at all widely known.

We are able to present three cases which came to necropsy and one in which the thrombosed vein was seen at operation, and to refer to other cases in which the clinical features were so similar to those of the verified cases that there can be little doubt that they had the same pathology. Since we are concerned only with the effects of thrombosis of the veins we shall exclude from consideration cases in which, according to our judgment, the venous obstruction was due to occlusion of a cranial sinus, even if the thrombosis of the sinus was "primary."

Primary thrombosis of cerebral veins happens sometimes in childhood and less often in old age; during the active period of life its most common association seems to be with childbirth, but it occurs also with general and local infective conditions, and sometimes without any such recognizable causal factor. At a recent discussion the two of us referred, independently of each other, to cases arising during the puerperium (Martin, 1939; Sheehan, 1939), and this paper will be made more compact if we limit ourselves here, so far as is possible, to cases arising under such circumstances. The puerperal cases will serve to illustrate the symptoms and pathology of primary cerebral venous thrombosis in general; at the same time they have an importance of their own in that they demonstrate the nature of certain alarming cases usually regarded as due to "late eclampsia" or to cerebral embolism.

Anatomy

According to O'Connell (1934) eight veins, on the average, pass up from the superficial middle cerebral vein (which lies in the Sylvian fissure), and make anastomoses over the frontal and parietal lobes with the superior longitudinal sinus. Veins arising on the surface of the brain join on to them. The positions of four of these veins are relatively constant, one lying in each of the precentral, central (Rolandic), and post-central sulci, and one in the parieto-occipital sulcus. The more posterior

members of the group bend forward as they approach the upper border of the hemisphere and enter the sinus obliquely, in a direction which is thus partly contrary to that of the blood flow in it. Each of the larger superior cortical veins as it passes from the lateral (superior) surface of the brain receives a large tributary from the medial surface of the hemisphere.

Case I

Convulsions ten days after delivery; death one and a half days later. Necropsy: thrombosed veins over right prefrontal area.

A 5-para aged 41 admitted to hospital December 9, 1935, had a normal pregnancy and delivery. The puerperium was uneventful for the first nine days with normal lochia and no pyrexia; nine days after delivery she complained of slight headache. The next day she suddenly became unconscious and had a fit followed by a series of fourteen other fits in the course of twenty-four hours. The temperature rose rapidly to 107° F. The cerebrospinal fluid was clear and under normal pressure; the urine contained albumin and blood; the blood pressure was 145/90 mm. Hg. She remained comatose but without any evidence of hemiplegia, and died thirty hours after the onset of the fits.

Necropsy revealed early bronchopneumonia, healed tuberculosis of the mesenteric glands, some old scars of healed pyelonephritis in both kidneys, and old pleural adhesions on the left side. The uterus was healthy, with normal involution. The other organs, apart from the brain, were also healthy.

The brain showed an area of early cortical softening on the outer aspect of the right prefrontal region about 5 cm. in diameter. In the pia arachnoid over this softening there were two or three thrombosed veins and some small areas of haemorrhage. The superior longitudinal sinus was not examined.

In this case the vessels thrombosed were some of the variable veins in the right prefrontal region, and the onset of convulsions probably coincided with the time of their obstruction. Although a softening was produced, no paralysis was observed, and this was evidently because the lesion was well in advance of the motor area of the cerebral cortex.

Case II

Fits from fourth to eighth day after delivery. Right hemiplegia. Death fifteen days after first fits. Necropsy: thrombosed veins over precentral gyrus; "rat-tail" thrombus in superior longitudinal sinus.

A 10-para aged 39 was admitted on September 11, 1937. She had a normal pregnancy and labour. For the first three days of the puerperium she was apparently well, with normal

temperature and pulse and normal lochia. On the fourth day after delivery she had a fit and developed a right-sided hemiplegia. During this day she had several further fits, during which the right arm was rotated outwards, but the right leg remained still; there was no loss of consciousness. On the seventh day after delivery four similar fits occurred, and on the eighth day two fits.

The blood pressure was 155/85 to 165/95 mm. Hg on the fourth day post partum, but thence varied between 135/80 and 120/75. The temperature rose on the seventh and eighth days to 100° F., and afterwards there were occasional temperatures of 99° F. to 99.5° F. The pulse rate was 70 to 80 per minute for the first week of the puerperium, and afterwards ran at 100 to 110. The urine contained a slight amount of albumin and lactose; the blood urea was 32 mg. per 100 c.cm. seven days after delivery. The lochia were always normal.

The hemiplegia remained unaltered, but the patient seemed to be improving until nineteen days post partum, when she developed cyanosis and a sense of oppression in the chest. This was partly relieved by oxygen; but after about twelve hours she became suddenly more distressed, and died rapidly. At the necropsy the immediate cause of death was found to be a true thrombosis of both pulmonary arteries. The thrombus was firm and grey and occupied most of the branches of these arteries; it appeared to be a few days old. There was no evidence of pulmonary embolism or of lung infarcts; the lungs were dry and rather tough. The right ureter showed a pregnancy dilatation of considerable degree but was not inflamed. The right ovarian veins in the pelvis were thrombosed. The uterus was healthy, with normal involution. The other organs, apart from the brain, were normal.

The brain showed an area of reddish softening of the cortex of the upper part of the left hemisphere, centred near the upper end of the precentral gyrus. This was about 5 cm. in diameter at the surface and about 2 cm. deep; it was surrounded by a zone of yellow staining and softening about 1 cm. in thickness. In the pia arachnoid over the softening there were three large veins distended with red thrombus. The superior longitudinal sinus contained a pale thrombus along its whole length back to the torcular Herophili; this was thicker and whiter, and was adherent near the junction of the thrombosed meningeal veins, but did not occlude the lumen of the sinus.

Microscopically the cerebral softening was typical of advanced necrosis, and the surrounding yellow zone was packed with compound granular corpuscles full of lipid.

In this instance the onset of fits and of right hemiplegia is presumably to be attributed to thrombosis in the veins over the left cerebral hemisphere and the resulting softening. It is not possible to say when the clot in the superior longitudinal sinus developed, and as it did not obstruct the lumen it is doubtful whether it gave rise to any symptoms.

Case III

Weakness of left arm fourteen days after delivery, followed by repeated convulsions. At a later stage, high intracranial pressure. Thrombosed Rolandic vein seen at operation. Eventual excellent recovery.

This woman, aged 32, was admitted to hospital on March 18, 1934, under the care of one of us. Her first baby was born on March 3, and the puerperium was normal except for a rise of temperature to 99° F. on the third day. On March 17 she complained of a slight headache and had some temporary weakness of the left arm. Next morning on waking she had a headache, her left hand was paralysed, and her tongue felt too big for her mouth. (She may have bitten it during a fit in her sleep.) Soon after waking she had a fit, which began with jerking of the left arm and left side of the face. Four similar fits occurred during the morning, and she was then admitted to hospital, and during the next twenty-four hours had seventeen fits. She had complete paralysis of the left hand and gross loss of sense of position in it. The left side of the face was paralysed; there was only slight weakness of the left leg, but the plantar reflex was extensor.

The central nervous system was otherwise normal, and the other systems showed no abnormalities. The cerebrospinal fluid pressure was normal; the fluid contained 1,700 blood cells per c.mm. but was not otherwise abnormal. The fits practically ceased on the second day and the patient made good progress until the sixteenth day. She then had a fit, and another on each of the two succeeding days; these were followed by a rise of intracranial pressure to 240 mm. of fluid, with morning headaches and papilloedema, signs which eventually led to an exploratory operation. At the operation, performed by Mr. Geoffrey Jefferson on the sixty-ninth day of the patient's illness, the dura was found slightly adherent over the fissure of Rolando, and instead of the normal appearance of the vein in the fissure there was a white opaque streak. The motor cortex appeared softened, but stimulation of it produced responses in the contralateral limbs. The patient made further improvement after the operation, and left hospital a month later without disability.

This case shows two phases. There can be little doubt that the alarming symptoms which occurred at the beginning of the illness were due to thrombosis of the right Rolandic vein, which ten weeks afterwards was seen as an opaque streak in the arachnoid. The cerebrospinal fluid contained numerous blood cells, but the pressure of the fluid was normal. The second phase began on the sixteenth day, when the patient had a recurrence of fits, and thereafter signs of raised intracranial pressure appeared. This rise of pressure may be taken as an indication that the absorption of cerebrospinal fluid by way of the Pacchionian bodies and the superior longitudinal sinus had been obstructed, and thus as evidence of occlusion of the sinus, or at any rate of its "upper story," by clot. Hydrocephalus in connexion with thrombosis of the superior longitudinal sinus has been described in cases of various kinds by Bailey and Hass (1937), Frenckner (1934), Symonds (1937), Ellis (1937), and others.

Case IV

Symptoms of thrombosis of cerebral veins seven days and forty-two days after delivery. Death three years later. Necropsy.

About a week after her sixth confinement, when she was 27 years old, this patient, a 7-para aged 30, had a single convulsion. There are, unfortunately, no further details available about her condition at that time and during the next few days. Three weeks later she had a slight thrombosis of the superficial veins of the right leg; this cleared up satisfactorily. Two weeks afterwards (i.e., six weeks post partum) she suddenly developed paresis of the right arm and leg. The right plantar reflex was extensor; the other reflexes and the eyes were normal. The blood pressure was 110/70. Blood examination showed a marked anaemia: R.B.C., 2,540,000; haemoglobin, 20%; W.B.C., 5,000; urea, 49 mg. per 100 c.cm. Wassermann negative. No abnormality was found in any other system. The hemiplegia gradually disappeared during the next month, though subsequently she had occasional attacks of fainting with unconsciousness, but without any convulsions.

This patient died three years later (April 26, 1940) as a result of ante-partum haemorrhage and uterine sepsis in her seventh pregnancy, and a post-mortem examination was made. The brain showed three depressed scars in the cortex, one over the upper part of the left parietal region and one on the upper aspect of each frontal lobe. These scars were all about 2 to 3 cm. in diameter and 1 cm. deep. The overlying pia arachnoid was brown, thick, and moderately adherent. No remaining thrombosis of cerebral veins or sinuses could be distinguished. On section the depressed areas of the brain surface showed only a thin layer of cortex, which was dark in colour; beneath this the white matter was soft and shrunken. Microscopically these portions showed, almost exclusively, loosely formed glia with few remaining nervous elements.

The history and the pathological findings in this case indicate that three separate cortical veins had thrombosed after the sixth delivery. The convulsion seven days after

delivery was presumably related to one of the frontal lesions; the hemiplegia which occurred later was due to thrombosis of a vein over the left parietal cortex. It is of interest that the latter thrombosis was not accompanied by convulsions, and this brings out the important point that convulsions are not an essential element of the symptomatology. In our experience they are usual, but have not been a prominent feature of all the verified cases of primary cerebral venous thrombosis hitherto published. They occurred in Davis's (1933) case and in Money's (1890), but Dowman's (1926) patient merely had slight attacks of a twitching sensation in the right upper eyelid: fits are not mentioned by Irish (1938) in regard to two cases of primary thrombosis in his series (Nos. 1 to 4), but one of the patients had cramp. A presumptive case without fits has recently been described by Symonds (1940).

Another point of interest in this case is the virtual disappearance of the thrombosed veins three years after the event. In the preceding case the affected vein was seen evidently in process of being absorbed; while Bailey and Hass (1937) have described, in the case of an infant, both the formation of new vessels and canalized thrombi in the superior cerebral veins two and a half months after the presumed time of thrombosis.

Previously Reported Cases.—The puerperal cases reported up to 1904 were included in v. Hösslin's collection (1904, 1905). Of other, more recent, cases Dowman's (1926) was confirmed at operation, and ended in complete recovery. Davis's (1933) case was confirmed, both at operation and post mortem. Waggoner's (1928) was complicated by the presence of an angioma. Two uncomplicated cases of primary venous thrombosis occurred among a group of pathological cases described by Irish (1938). None of these cases was puerperal. Cases in children are in a somewhat different category, and we have not taken account of them all. Money's (1890) case was referred to by Gowers (1892-3), and in the more recent literature there are two post-mortem cases in children with heart disease described by Lhermitte and his co-workers (1936).

For the purpose of establishing the pathological nature of these cases we have necessarily made use of those in which the diagnosis was verified. But we do not wish to give the impression that the condition is always a fatal one. On the contrary, the majority of patients make good recovery. We have seen three other puerperal cases, and a number not associated with childbirth, which in their initial clinical features were closely similar to those already described, but ended happily. The following account, though it describes the most severe of the three puerperal cases, may be taken as typical of all of them.

Case V

Cramp and fits ten days after delivery: incomplete left hemiplegia. Good recovery in three weeks

A primipara aged 25 was confined on June 30, 1938. Labour was normal and the puerperium was uneventful until July 10, when she complained of "cramp" in her left hand and a feeling of faintness. During that evening she had three epileptiform fits, with the twitching more pronounced on the left side, and next morning she found that her left arm was weak. On admission to hospital at 12.50 p.m. that day the patient was able to speak but was stuporous: she had obvious weakness of the left side of her face, and did not move her left arm. There was no stiffness of the neck, and nothing abnormal was found on examination of the heart, lungs, and abdomen, or in the ears; her urine, taken by catheter, had a high specific gravity (1036) but was otherwise normal. Her temperature on admission was 100° F. and her pulse rate 108. The temperature was normal by the following morning and remained so until the next day, when three more brief fits

occurred involving loss of consciousness; her temperature rose to 100.2° F., but had returned to normal by 10 o'clock next morning. Her blood pressure was 120/80 mm. Hg; the blood Wassermann was negative.

When examined on the sixth day of her illness she was listless and moderately drowsy. She had incomplete left hemiplegia, the face and hand being most affected and the leg much less, but the left plantar reflex was extensor. There was a loss of sense of position in the fingers of the left hand. The cerebrospinal fluid pressure was not measured, but there was no suggestion, either then or later, that it was increased. The patient's condition steadily improved. In a few days she was quite alert mentally, and within a fortnight of her admission her left hand had recovered a great deal of its power and postural sensibility. She was discharged from the hospital on August 9 after a stay of four weeks and a day, and was then using her left hand normally. There was still very slight weakness in the left side of her face, and the tendon jerks on the left side of her body were distinctly greater than those on the right; the left plantar reflex was indefinite.

When seen again three months later she declared herself quite well except for slight attacks of numbness in the left hand.

There can be little doubt that in this case, as in Case III, the right Rolandic vein was involved. It was probably not thrombosed up to its entry into the superior longitudinal sinus, because the absence of paralysis in the left leg indicates that the tributary from the medial surface of the hemisphere was not obstructed: this tributary enters the main vein as it leaves the surface of the brain to cross over in the arachnoid to the sinus. There were no complications, and recovery was made much quicker than in Case III.

When veins over the more posterior parts of the cerebral hemisphere become thrombosed visual disturbances are likely to ensue. In the following instance, which was not associated with childbirth, it is likely that part of the vein which runs up to the parieto-occipital fissure or one of its tributaries was occluded. Word blindness, attributable to disturbance of function in the adjacent angular gyrus, was among the symptoms.

Case VI

Typhoid fever. Epileptiform attacks followed by word blindness and impairment of postural sense in right hand. Good recovery.

A girl aged 18, recovering from typhoid fever, had seven epileptiform attacks one morning and was afterwards stuporous. When seen four days later she showed no loss of power, but recognized objects in the right hand with less confidence than in the left. She was quite unable to read, although her acuity of vision and visual fields seemed normal. Her tendon jerks were brisk and her plantar reflexes flexor. Her condition gradually improved, and in about six weeks she seemed to have completely recovered.

Comments

These cases show the clinical picture of primary cerebral venous thrombosis and its variation according to the position of the vessels involved. Regarding the subject in its larger aspect the main divisions that call for discussion are: (1) the thrombosis; (2) its effect on the brain; and (3) the clinical features to which these jointly give rise.

The Thrombosis

Aetiology.—The special anatomical factors which favour thrombosis in the veins on the surface of the brain were stressed by Gowers (1892-3). We are inclined to attach less importance than he did to their vertical direction, but the slowness of the blood-flow in them and the fact that the blood leaving them enters the longitudinal sinus in a direction contrary to the current are

probably significant. The more general factors which favour the thrombosis are not well known. In an important group of cases primary cerebral venous thrombosis occurs in the puerperium, which is a time at which thrombosis is particularly apt to arise elsewhere in the body. Nearly all the other cases are associated with conditions in which diarrhoea and vomiting is an added element—typhoid fever, ulcerative colitis, gastro-enteritis—but also (as we have seen in another instance) such a mild infection as “gastric influenza” in a vigorous young subject. In the puerperal cases there is certainly no relation to sepsis which can be recognized clinically or at necropsy. Nevertheless, even though the puerperium has apparently been normal up till the onset of a convulsion, the possibility of a symptomless infection cannot be excluded: positive blood cultures are found in a high percentage of normal women for an hour or two after ordinary delivery. Cerebral venous thrombosis is certainly not a result of difficulty at the delivery: in nearly all the cases this has been quite normal.

Formation of the Thrombus

The exact mechanism by which this thrombosis occurs in cerebral veins has not been fully worked out. An analogy may, however, be drawn with the thrombosis sometimes found in femoral or ovarian veins after delivery, in which the probable sequence is as follows: (a) A small pale thrombus forms at the junction of a tributary vein with a large vessel. It is attached along a very narrow longitudinal line at the cardiac side of the junction. (b) It enlarges until it rather suddenly obstructs the mouth of the tributary vein, which immediately becomes distended by a firm red thrombus made up mainly of packed red corpuscles. (c) The primary thrombus also enlarges into the lumen of the main vein, where it forms a grey “slug” on the wall; later this may gradually extend in either direction along the main vein as a long grey thrombus, sometimes with branches down other tributary veins. This secondary thrombus is formed in blood which is still flowing (though probably somewhat sluggishly), and it usually lies free in the lumen.

The conditions which have been found in the cerebral veins correspond with this interpretation. The distension by red thrombus of one or two of the veins in the arachnoid over the hemisphere is the most striking appearance on opening the skull in these cases. The manner of its development may be considered according to the three stages mentioned.

(a) In Case II the site of the primary mural thrombus was identified by the grey slug at the junction of the thrombosed arachnoid vein with the superior longitudinal sinus. But it is not clear whether this is the usual site; it seems probable that in other cases the primary thrombosis occurs at a junction of two arachnoid veins some distance from the sinus. (Since our necropsy technique was modified to study this point we have not had a case of this kind.) The sparing of the leg in Cases III and V must be taken as evidence that the tributary vein from the medial surface of the hemisphere was not obstructed, which suggests that the Rolandic vein was not thrombosed up to its point of entry into the sinus. In some cases in the literature (especially that of Davis, 1933) it is expressly stated that the thrombosis of the arachnoid vein did not extend up to the junction with the superior longitudinal sinus, and in these it may be accepted that the primary thrombosis was at the junction of one arachnoid vein with another. On the other hand, it seems that the point of entry of a vein into the sinus is a likely site for the initial thrombosis owing to the partly opposed directions of flow of the joining blood streams, with the consequent formation of an eddy. In a fatal puerperal case described by Symonds (1937) multiple mural thrombi were seen post mortem on the side of the sinus, and Gowers (1892–3) states that a layer of clot is sometimes found on the wall of the sinus.

(b) The red thrombus in the arachnoid vein presumably forms suddenly when the vein is blocked. After a few weeks it becomes decolorized and shrinks as in Case III; after a few years it is no longer recognizable, as in Case IV.

(c) When the primary thrombus is on the wall of the superior longitudinal sinus, or if it eventually reaches up to it, the secondary grey thrombus may extend, as in Case II, in the blood flowing along the sinus. It is easy to believe that in some cases this may become so large as to produce mechanical obstruction of the sinus, or that it may become adherent in places to the wall of the sinus. Such occurrences may produce further effects—interference with the absorption of cerebrospinal fluid into the superior longitudinal sinus (as indicated by the clinical signs in Case III) or the obstruction of the mouth of other meningeal veins and consequent red thrombosis of these veins. The latter process may have been responsible in our Case IV for the late thrombosis which caused necrosis of the parietal cortex. A possibility which must not be neglected is that certain of the results apparently due to the secondary thrombus may actually be caused by more than one primary thrombus. This is suggested by the multiple mural thrombi in the sinus recorded by Symonds (1937). On the other hand, in our Cases III and IV the time of development of the later symptoms is in accordance with the explanation that these were secondary.

The Cerebral Lesion

Thrombosis of a vein in the retina provides a dramatic illustration of the effects of venous occlusion in a delicate tissue: numerous small haemorrhages are seen where venules draining into the obstructed vein have ruptured. Something similar evidently occurs in the meninges and in the brain. Frequently one of the first effects is the development of small subarachnoid haemorrhages about the size of a postage stamp or smaller (Case I). This has also been described by Davis (1933), Lhermitte, Lereboullet, and Kaplan (1936), and Irish (1938). The amount of blood may be sufficient, as in Case III, to cause easily recognizable blood in the cerebrospinal fluid obtained at lumbar puncture. Haemorrhages of various sizes may also occur within the brain substance as in Symonds's (1937) case, in two cases in children described by Lhermitte and his co-workers (Armand-Delille and others, 1936; Lhermitte and others, 1936), and in Davis's case.

Necrosis of cerebral tissue without haemorrhage seems, however, to be a more common effect. Our fatal cases show that there is a rapid softening of the area of the hemisphere drained by the affected vein. It is an ordinary reddish necrosis affecting cortex and underlying white matter, and in extent varies from 2 to 5 cm. in diameter. In the fatal cases there is no doubt about the complete nature of the necrosis, either to the naked eye or microscopically, but in non-fatal cases it may be less intense, and in Case III the altered cortex was still capable of response to electrical stimulation. After some days it is surrounded by a broad yellow zone of compound granular corpuscles loaded with lipids. In the healed stage it becomes a shrunken depressed area 1 to 2 cm. in diameter, with the pia arachnoid somewhat thickened and the arachnoid lightly adherent over it; the general build of the atrophied convolutions can be made out, but the microscopical structure consists of little else but glial fibres.

In the operated cases reported in the literature (Downman, 1926; Davis, 1933) the condition found was non-haemorrhagic, as in Case III.

Clinical Features

In puerperal cases the onset of the acute illness is most commonly between four and twenty days post partum; usually the delivery has been normal and the puerperium quite uneventful up to that time. The first symptom in most cases is the occurrence of one or more fits. If,

however, a detailed history can be obtained it is often found that the first fit was preceded by some symptoms such as headache or intermittent cramp or weakness of a limb. The fits probably coincide with the sudden obstruction of the meningeal vein and the formation of red thrombus in it. They are to be ascribed to cellular disturbances in the area of cortex in which the venous drainage has been abruptly arrested. The exact site of the cortical lesion is not of great moment: fits seem to occur almost as frequently when non-motor cortex is involved as when motor cortex is concerned. In many cases the fits continue over several hours or days; they are most evident in, and sometimes limited to, the part or side first affected. Recurrence of fits after an interval may indicate that the thrombosis is spreading. The incidence of coma in relation to the fits is very variable. Some patients appear to be conscious immediately after the fit. Prolonged coma is of bad prognostic significance.

After the fits the condition depends on the site of the thrombosis. If the lesion is in the frontal or occipital area no paralysis may be observed. But if the motor cortex is involved, as is commonly the case, there is usually complete paralysis of one limb and weakness of the rest of that side of the body. In our experience the arm is generally the part most severely affected.

The cerebrospinal fluid is as a rule normal unless the subarachnoid haemorrhages have been extensive, when a little blood may be present. There may be slight pyrexia, some albuminuria, and mild hypertension, but these conditions usually clear up in the course of a few days. A transient rise of blood uric acid due to the convulsions is also to be expected (Crawford).

When the convulsive stage is over the patient is in no direct danger from the cerebral lesion, though she is still exposed to the risks of spreading thrombosis in the veins or in the superior longitudinal sinus, or of thrombosis of vessels in other parts of the body. In many cases the paralysis has almost completely gone in the course of a few weeks. Evidently, with the re-establishment of venous drainage the parts of the cortex forming the margin of the lesion are capable of recovery, and the residual lesion, although anatomically considerable, as is shown by our Case IV, is not sufficient to cause paralysis. Two factors possibly help to explain why recovery is much better in these cases than in arterial thrombosis or haemorrhage. The first is that the lesion is on the surface of the brain, where a larger lesion is required to produce the same degree of paralysis than if the lesion is, say, in the internal capsule. Secondly, in cases in which haemorrhage occurs the blood is effused under a lower pressure than in cases of arterial bleeding and so causes less disruption of fibres, and greater recovery is consequently possible when the blood is absorbed.

Recovery is not so complete in all instances, and in more than one presumptive case we have seen residual symptoms. There can be little doubt that in all cases gross anatomical lesions of the cortex are left, and the degree of clinical recovery that is usual provides an illustration that a considerable cortical defect can exist with few or no symptoms.

Diagnosis

The signs and symptoms of cerebral venous thrombosis are not pathognomonic, but the syndrome, taken in conjunction with the conditions in which it arises, is fairly clear. The differential diagnosis of the cases which occur in the puerperium ranges over all the conditions which may cause fits or paralysis at that time, whether peculiar to obstetric cases or not, and it is manifestly impossible to discuss it fully here. Many of these cases have in the past been regarded as "late eclampsia"; the existence

of this disease is doubtful, and most of the signs and symptoms on which it is diagnosed are seen in cerebral venous thrombosis, as noted above. Other cases have been ascribed to cerebral embolism from thrombi in the pelvic or femoral veins; this presupposes a patent foramen ovale—an assumption that is rarely justified by the anatomical findings. A further cerebral condition involving hemiplegia associated with blindness of one eye which may occur several days after delivery and thus give rise to difficulties of diagnosis has been fully discussed by Wright Thomson (1940).

Treatment

Much of the treatment is on obvious lines. Luminal, 2 or 3 grains by mouth or intravenously, will usually control the convulsions, and afterwards smaller doses should be administered for a week or two, and massage or passive movements should be given to paralysed limbs. Unless other complications supervene the patient will in most cases have made a good recovery in a few weeks. Since there is often evidence of extension of the thrombosis after the first symptoms the administration of heparin should be considered as soon as the condition is recognized. We have, however, had no opportunity of trying it. In the puerperal cases the danger of uterine haemorrhage would seem to be the principal risk entailed in its use, and the possible danger, in all cases, of the liberation of emboli will have to be taken into account.

Summary

The clinical features and the pathology of primary cerebral venous thrombosis are illustrated by a group of six cases.

In two of these cases the diagnosis was established post mortem and in one case at an exploratory operation. A fourth case came to necropsy three years after the onset, and in the other two cases the diagnosis is presumed because of similarity of symptoms and circumstances with those in the verified cases.

In five of these cases the symptoms occurred after childbirth. Such cases have usually been regarded as instances of "late eclampsia" or cerebral embolism.

Primary cerebral venous thrombosis occurs in the puerperium and in conditions of general and local infection—especially in those associated with vomiting or diarrhoea.

As a result of the venous blockage, softening, with in some cases haemorrhage, occurs in the brain and a permanent residual scar is left on the cerebral cortex.

The outstanding clinical features are fits and paralysis. The fits may be local or general, and in occasional cases may be absent. Premonitory symptoms occur in many cases. A small amount of blood may be found in the cerebrospinal fluid. The relation of these symptoms to the pathological features is briefly discussed.

It is suggested that when the condition is recognized heparin might be tried with a view to preventing spread of the thrombosis.

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