Cerebral Venous and Sinus Thrombosis*

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The general pathology and clinical picture of cerebral venous thrombosis have been well recognized entities since the 19th century. But from our own experience of 92 cases, and from the study of cases reported in the literature, it is obvious that the diagnosis of cerebral venous thrombosis could be made with reasonable certainty only in a very few cases at the onset of the illness. In the great majority of the cases the correct diagnosis was revealed by the subsequent course of events. The cases associated with childbirth are usually best known and the easiest to diagnose. The fact, described by *Symonds*⁽²⁰⁾, that increased intracranial pressure associated with ear disease is due to venous thrombosis is recognized in some cases as "otitic hydrocephalus". But in the great majority of cases the diagnosis remains difficult when the symptoms first begin to manifest themselves. The study of our case material has been undertaken with a view to showing whether cerebral angiography has a special diagnostic value at the onset of the disease. Since the introduction of modern chemotherapy and treatment with anticoagulant drugs, an accurate diagnosis is extremely necessary from the onset of symptoms. Furthermore, the role of surgical intervention must be carefully evaluated. Only by early introduction of these therapeutic means will the situation improve to the point that the prospects of more or less complete recovery are fairly good. Experiences detailed herein are described from a purely clinical point of view.

Pathogenesis and Pathology

In 92 patients the diagnosis of cerebral venous and sinus thrombosis has been made clinically by cerebral angiography, by operation, and at necropsy. In Fig. I.1 the causative factors are enumerated. As for sex and age distribution, 46 cases were men and 46 cases women, and thrombosis may develop at any age, ranging in our series between 2 and 71 years. It is shown that thrombosis generally follows another pathological condition or a change in the integrity of the body (infection, pregnancy, puerperium). The important infectious group comprises 37 patients

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Fig. I.1. Etiology and pathogenesis of cerebral venous and sinus thrombosis.

with venous thrombosis which was associated with local septic conditions or generalized infections. Of these, 13 cases were infectious thromboses resulting from bacterial infection of the nasal sinuses (six cases), the middle ear (three cases), or of the upper eyelid (four cases). All but four of them have shown the clinical syndrome of cavernous sinus thrombosis and eight patients died from bacterial meningitis. In the remaining 24 cases, venous thrombosis developed in the course of infections elsewhere in the body.

The bulk of our material is presented by 55 cases of non-infectious thrombosis. This developed in 17 women during pregnancy, after abortion, or (5 and 8) in the puerperal state. The neurological syndrome occurred also in four patients receiving synthetic steroids as oral contraceptives¹⁹⁾. There seems to be a close correlation between the alterations of the clotting factors seen in pregnancy and those produced by the progestational steroids. Pregnant women show an increase in prothrombin, Factor VII and Factor X. Fibrinogen is elevated and fibrinolytic activity is decreased, which *Phillips* and his associates¹⁸⁾ found to be an effect of estrogen preparations on the fibrinolytic enzyme system. *Jürgens* and *Stein*¹⁰⁾ suggested that puerperal venous thrombosis might be caused by a sudden activation of intravascular coagulation through the inflow of a thromboplastin-like substance from the placenta in the blood stream.

Infections in the neighborhood of the skull, especially sinuses, ear, and neck or elsewhere in the body (23 cases), injuries (three cases), and cardiac diseases (four cases including tetralogy of Fallot and endocarditis) may precede the intracranial venous thrombosis. Venous and arterial thromboses in other regions of the body may precede or occur beside the cerebral venous thrombosis. In two cases a venous thrombosis was found in the lower extremities. In one of these, the cerebral complication immediately followed the interruption of an anticoagulant treatment of a venous thrombosis in a leg. The extracerebral thromboses have been verified at necropsy in the lower extremities, in the jugular vein, in the internal carotid artery, in the posterior tibial artery, and in the aorta. On two occasions thrombosis was associated with hemolytic anemia and ulcerous colitis

respectively. Quite often, however, no causative factor can be ascertained. That is the case with the important group showing thrombosis of a "primary nature" (22 cases, i.e. 23.9 per cent of this series).

The venous thrombosis affects the cerebral vascular circulation. The resistance in the cerebral circulation is increased and functional impairment of the cerebral cortex results. Thrombosis in the superior longitudinal sinus is followed by signs of increased intracranial pressure, and obstruction of superior cerebral veins give rise to paralysis corresponding to the area of cerebral cortex which the vein drains. This paralysis may or may not be associated with convulsions. From a section showing the superior longitudinal sinus (Fig. I.2), it is evident that obstruction in the upper half of the sinus prevents absorption of the cerebrospinal fluid through the Pacchionian bodies, raising the intracranial pressure, that mural thrombosis below may obstruct one or more entering veins, and that thrombosis of the affected vein is then likely to occur. But a vein may also be thrombosed primarily without thrombosis in the sinus. The degree of functional impairment depends upon the localization and extension of the venous thrombosis and upon the velocity of its development. An extensive, increased resistance in the venous part of the blood circulation leads, furthermore, to an increase of the blood pressure in the small arteries and capillaries with extravasation of blood and plasma. Brain edema

Fig. I.2. Section through the superior longitudinal sinus. The lacunae laterales contain within their walls numerous Pacchionian granulations through which the cerebrospinal fluid is absorbed into the blood stream. However, there is also a direct venous drainage from the subarachnoid space through the endothelial cells of capillaries and venules in the cranial and spinal subarachnoid space (5). According to O'Connell (17), in many subjects numerous trabeculae cross the sinus, and in some instances a continuous membrane divides it horizontally for a portion of its length. At its upper angles are the lacunae laterales and the superior cerebral veins enter its walls near the inferior angle (12). (From A. Hafferl: *Lehrbuch der topographischen Anatomie*, Springer-Verlag, Berlin, 1953, as used by J.W. Millen and D.H.M. Woollam: *The Anatomy of the Cerebrospinal Fluid*, Oxford University Press, London, 1962.)

Fig. I.3. Hemorrhagic infarct, purpura and edema of the left frontal lobe with displacement of ventricular system in the case of a 29-year-old woman with puerperal thrombosis. Occlusion of the superior longitudinal, transverse, superior and inferior petrosal sinuses of the left jugular vein, and parasagittalerterring veins demonstrated at necropsy.

and intracerebral hemorrhage (Fig. I.3) with the signs of an intracranial space-occupying lesion are the consequences⁸⁾. In the infectious cases the thrombi are often infected with germs and a purulent meningitis may result.

Symptomatology

From the study of these anatomopathological changes it seems clear that the occurrence of headaches, mostly unilateral, convulsions, and varying degrees of paresis is likely to be the result. Headaches generally are the first sign (Fig. I.4). The convulsive phenomena are often of dramatic severity and fulminating in their onset. Generalized seizures are slightly more frequent than Jacksonian attacks. In 42 cases the electroencephalogram has been analyzed and the electrical alterations were studied in the acute stage of the illness. According to the study made by my associates *Burkhardt* and *Regli*²⁾, there is a severe general disturbance of the electrical activity, which is irregular over the hemispheres as a rule and more marked on one side than on the other. Delta foci, often combined with depression of the basic rhythm, rhythmic, symmetrical frontal 3 per sec. delta waves, or focal epileptogenic potentials are found (Fig. I.5). It is most likely that the depression of the basic activity is a consequence of the blood stasis in the region of the thrombosed veins and, in the case of a definite delta focus, the development of a sub-

Fig. I.4. Symptomatology of cerebral venous and sinus thrombosis.

Fig. I.5. The electroencephalogram (EEG) demonstrates a severe generalized slowing, depressed activity in left anterior temporal region (Channel 6), and a sharp wave focus in right frontal region. This is evidence of an extensive pathological condition having first severely damaged the left hemisphere and subsequently encroaching on the right side (case of Fig. I.6.).

cortical hemorrhage must be kept in mind. In our experience the convulsions also occur when the lesions are posterior to the cerebral gyri. The hemiparesis may be partial or complete and its greater incidence is, in our cases, on the arm and not on the leg. In all cases in which the symptoms rapidly became generalized in the convulsive stage or in which the convulsions were generalized from the very beginning, the immediate prognosis was alarming, and the mortality was high. In these cases the symptoms involve both hemispheres. The five patients with bilateral flaccid hemiplegia died. The 14 cases with oculomotor palsies 13 belong to the group of cavernous sinus thrombosis and are frequently associated with a unilateral exophthalmos. The body temperature may be normal or slightly or markedly increased. As a rule, the blood sedimentation rate is moderately or greatly increased. This is of some interest, also *Gilligan* and *Ernstein*⁶⁾ have observed that the increase of the plasma fibrinogen is proportional to the sedimentation rate of the red blood cells. The leukocyte count is often pathological. The findings vary between a leukocyte count cu.mm. of 10,000 and more than 15,000. There is no difference, in these findings, between the noninfectious and infectious group. The lumbar puncture demonstrates the cerebrospinal fluid usually under increased pressure; the cerebrospinal fluid may be xanthochromic or bloody and the total protein is, in about 18 per cent of the cases, increased, the same being true for the increase in cells. Papilledema in most of the cases was absent in spite of the evidence of raised intracranial tension demonstrated by lumbar puncture or at operation. Its absence is obviously explained by the rapid and fulminating course of the disease.

Cerebral Angiography

The presumable clinical diagnosis of a cerebral venous and sinus thrombosis has to be verified by angiography¹³⁾. This should be carried out as early as possible in the course of the disease. Direct puncture of the longitudinal sinus with injection of a contrast medium may spread thrombosis of the sinus directly to the cerebral cortex by way of the ascending veins. Therefore, sinography seems to us a dangerous diagnostic procedure in cases in which the thrombotic process is still in evolution.

The review of our cases, on the other hand, leaves no doubt that we can expect detailed data concerning the delayed cerebral circulation from speedy cerebral angiography. For correct interpretation of the angiograms, it is important that the angiographic technique be correct. In every case of suspected venous thrombosis the contrast is injected twice for stereoscopic view. Six pictures are taken in lateral projection and at 2-sec. intervals over a period of 12 sec., and three pictures in the anteroposterior projection at an interval of 3 to 4 sec.

The significant angiographic indication suggesting venous involvement is slowing of the circulation with stagnation of contrast medium in the small vessels and capillaries. The impairment of cerebral circulation gives, in the capillary and

Fig. I.6. Right carotid angiogram of a 16-year-old youth with pansinusitis, left-sided proptosis, and motor hemiplegia. It demonstrates nonfilling of the right anterior cerebral artery (A), and a corkscrew-like appearance of the capillaries in the frontal region in the capillary phase (B). The capillaries do not extend to the parasagittal region; the frontal ascending veins and the anterior third of the longitudinal sinus are not visualized in the late venous phase (C). The internal cerebral vein and basal veins including the vein of Labbé are demonstrated. Recovery after evacuation of pus from the frontal sinus and antibiotic treatment with residual motor hemiparesis.

Fig. I.7. Absence of the anterior third of the superior longitudinal sinus in a child. Normal variation. The frontal ascending veins are well visualized.

venous phase of the angiogram, the corkscrew or nail pressure-like appearance to the capillaries and venous vessels, which do not reach the surface of the hemisphere. The venous circulation is delayed also in circumscribed thrombosis, and the ascending veins of a certain brain area are not filled with contrast.

In Figure I.6 such a lesion in partial and limited thrombosis consists of the frontal ascending veins and the anterior third of the longitudinal sinus with no venous opacification of the frontal lobe. This 16-year-old patient recovered completely, his only defect being a left motor hemiparesis. However, it must be emphasized that absence of the anterior third of the superior longitudinal sinus is not, in itself, pathological. Hypoplasia or asplasia in this part is well known, especially in children (Fig. I.7)¹⁴⁾. The same isolated thrombosis may also occur in the parieto-occipital region. In Figure I.8 the ascending parieto-occipital veins are missing on the left side. This 13-year-old boy suffered from cerebral venous thrombosis in the course of an angina with high temperature, presenting right-sided Jacksonian fits with motor and partial sensory loss on the right side, blindness, and increase of blood sedimentation rate up to 41/46 mm. With antibiotic and anticoagulant treatment, the illness cleared up completely after 4 weeks, without any sequelae. Figure I.9 demonstrates a case of bilateral Jacksonian epilepsy with thrombosis of these veins on both sides and of the posterior third of the longitudinal sinus with drainage of the cortical veins through multiple parasagittal emissary veins of the rate mirabile bilaterally into the veins of the scalp and through the

Fig. I.8. Circumscribed cerebral thrombosis of the left ascending parieto-occipital veins in a 13-year-old boy during the course of an angina, presenting right-sided Jacksonian seizures, motor and partial sensory loss, and blindness. Complete recovery with antibiotic and anticoagulant therapy.

Fig. I.9. Bilateral thrombosis of the ascending parieto-occipital veins and of the posterior third of the longitudinal sinus, drainage through the rete mirabile into the veins of scalp (arrow), and through the deep and basal cerebral veins. This 36-year-old patient was suffering from bilateral Jacksonian epilepsy, rightsided motor hemiparesis, and papilledema. Recovery with bilateral subtemporal decompression and antibiotic treatment.

system of the deep and basal cerebral veins into the tentorial venous plexuses. The thrombosis may also be visualized in a larger vein such as the vein of Labbé (Fig. I.10). The temporal veins are not filled and at operation the vein of Labbé was thrombosed and there was a subcortical temporal hematoma which is shown by the upward displacement of the Sylvian vessels in the phlebogram.

Nonfilling of the transverse sinus is not invariably pathological, as its absence may represent an anatomical variation. However, absence of the sigmoid sinus, in the presence of a transverse sinus and venous engorgement of the cortical and deep veins of the same side and visualization of the transverse and sigmoid sinuses of the opposite side as shown in Figure I.11, is a definite pathological condition known as otitic hydrocephalus. This 5-year-old boy had a normal pneumo-encephalogram and a positive Queckenstedt sign on the affected side. But there is no doubt that this syndrome develops in only a very small fraction of cases in which sinus thrombosis has been proved. Therefore, other factors such as extension of the thrombosis into the superior longitudinal sinus and other sinuses or variations in the size of the sinuses at the level of the torcular (Fig. I.12) may be causal. We know that one sinus, usually the left lateral, may be hypoplastic or absent and that the sagittal sinus drains into the larger or single lateral channel,

Fig. I. 10. Thrombosis of the left vein of Labbé with subcortical hemorrhagic infarction in the temporal lobe, verified at operation. Upward displacement of the left Sylvian group, nonfilling of the vein of Labbé in the late venous phase. This 46-year-old patient recovered from his aphasia after removal of the intracerebral blood clot and with antibiotic treatment.

e., the right. Its occlusion, therefore, might well cause enough venous engorgement and consequent cerebral edema.

In the more serious cases of thrombosis in the longitudinal sinus, the ascending veins of one or both hemispheres and the sinus are missing in the second phase phlebogram even after 10 sec. In Figure I.13 the angiogram of a 39-year-old patient with no filling of the cortical veins and of the sinus is shown. But after 3 days of antibiotic and anticoagulant treatment the venous system is of a normal appearance in the phlebogram and the clinical signs (papilledema and left-sided hemiparesis) gradually cleared up completely. As a rule, the drainage is effected predominantly in the direction of the basal and internal cerebral veins. This is shown in Figure I.14 of a 46-year-old man with very scarce cortical veins but an enormous anastomosis between the Sylvian vein and the vein of Labbé. At operation the typical black appearance of the hard veins with subcortical cerebral softening was well demonstrated. As a consequence of the drainage in the direction of the basal veins and basal sinuses is the visualization of the superior ophthalmic veins. *Blauenstein* and *Lévy*¹⁾ have first demonstrated this venous drainage through the superior ophthalmic veins from the engorged cavernous sinus into the facial

Fig. I. 11. Angiogram of a case of otitic hydrocephalus. Venous engorgement of the cortical and deep veins, nonfilling of the sigmoid sinus on one side, and visualization of both transverse sinuses and of the sigmoid sinus of the opposite side (arrow) in a 5-year-old boy. Normal pneumoencephalogram. Complete recovery from bilateral papilledema and Jacksonian epilepsy after anticoagulant and antibiotic treatment.

Fig. I. 12. Schematic drawing of the anatomical variations in the development of the torcular Herophili; 23 cases (From K.-A. Hossmann: Zur Pathogenese des "otitischen Hydrozephalus". Fortschr. Neurol. Psychiat., 34: 236-246, 1966.)

Fig. I.13. Right-sided cerebral venous and sinus thrombosis in a 39-year-old patient. Nonfilling of cortical veins and longitudinal sinus in the late phlebogram (A). After 6 days of antibiotic and anticoagulant treatment, normal phlebogram (B), and recovery from motor hemiparesis and papilledema.

Fig. I.14. Angiogram showing venous drainage through the basal cortical veins in a case of a 46-year-old patient with right frontal venous thrombosis verified at operation (Fig. I.22). Complete recovery.

vein and pterygoid plexus. In normal cerebral angiography these veins are missing. Figure I.15 shows both superior ophthalmic veins in the case of a 31-year-old patient receiving over a period of many years oral contraceptives. In the lateral projection the engorgement of the deep cerebral veins and of the transverse and sigmoid sinus is well demonstrated.

In most cases of cerebral venous thrombosis the disease is confined to one cerebral hemisphere. Therefore, bilateral angiography demonstrates the absence of the ascending veins and of the longitudinal sinus on one side, whereas these venous structures are shown clearly on the opposite side (Fig. I.16). Isolated thrombosis of the deep cerebral veins is very rare⁴⁾. Figure I.17 represents such

Fig. I.15. Right carotid angiogram of a 31-year-old female patient receiving oral contraceptives and presenting severe bilateral papilledema and bilateral sixth nerve palsy. It demonstrates non-filling of the whole longitudinal sinus and only partial filling of the ascending veins, but venous engorgement of both superior ophthalmic veins (arrows) (A), of the deep veins and of the transverse and sigmoid sinus (B). Recovery from bilateral papilledema and sixth nerve palsy with antibiotic and anticoagulant therapy and repeated lumbar punctures. (C,) retinal hemorrhages and papilledema on the right side. (D,) retinal hemorrhages and papilledema on the left side.

In a case, a 23-year-old pregnant patient in whom all the signs—bilateral papilledema, 5th nerve palsy, and left-sided sensory hemiparesis—cleared up with anticoagulant treatment and abortion. A similar situation is shown in the phlebogram (Fig. I.18) of a 32-year-old patient receiving, over a 3-year period, oral contraceptives. The capillaries are hardly visible and the main venous drainage is effected in the direction of the basal lateral veins and through the veins of the scalp, but the internal cerebral vein is not visualized. With antibiotic and anticoagulant treatment, the

Fig. I.16. Cerebral venous thrombosis over the left hemisphere in a 40-year-old patient with right-side hemiparesis and aphasia. A, phlebogram of the left-side with no filling of ascending veins and sagittal sinus; B, normal phlebogram on the right side.

patient was discharged from the hospital without any abnormal neurological signs.

The tortuosity and enlargement of the cortical veins may be considerable, as in the case of a traumatic thrombosis of the superior longitudinal sinus. This is shown in Figure I.19 with engorgement of all ascending veins and venous drainage into the basal and internal cerebral veins. Control angiography 1½ years later demonstrates normal venous circulation in the 30-year-old patient who completely recovered from a traumatic state of unconsciousness and a flaccid tetraplegia over a 3-month period.

Therapeutic Measures and Prognosis

With regard to treatment, this consisted of either anticoagulation therapy (Marcoumar) reducing the prothrombin time to between 15 and 25 per cent or of antibiotic therapy with penicillin at a daily dose varying generally between 1 and 10 million units, partially combined with streptomycin (1 to 2 gm. daily), or chloramphenicol (1 to 3 gm. daily); when epileptic seizures occurred, an antiepileptic treatment was started. This medical treatment was applied to the infectious and

the noninfectious group²¹⁾. In addition in many cases repeated lumbar punctures were performed. This treatment is in agreement with the conclusion of *Kalbag* and *Woolf*¹¹⁾ on page 248 of their monograph "the management of dural venous sinus thrombosis is essentially medical, relying on anticoagulants, anticonvulsants and antibiotics".

There are differences in the mortality rate in the different treatment groups (Fig. I.20). On the whole, we get the impression that the combined antibiotic and anticoagulant treatment gives the best results. We have no proof that cerebral hemorrhages occur more often and are more severe in anticoagulated cases. The group without any treatment has the highest mortality. But included in this group are cases in very serious condition already, mostly comatose on admission to the hospital. Death usually occurred some hours or a few days after admission.

As neurosurgeons we have now to discuss the question: What is the neuro-surgical contribution to a curative treatment? The answer may be furnished by our own experiences. In twelve patients with noninfectious intracranial venous thrombosis and in one case with multiple subcortical abscesses, surgical intervention was undertaken. The 53-year-old patient with the abscess formation in the right insular region died from brain edema and remaining abscesses (Figs. I.21 and 22). In the noninfectious group the removal of a cerebral hemorrhage rapidly improved

Fig. I.17. Thrombosis of deep cerebral veins in a 23-year-old pregnant patient with bilateral papilledema, 6th nerve palsy, and left-side sensory hemiparesis. Complete recovery after abortion and anticoagulant therapy.

the condition of four patients and there was no mortality. In the remaining eight cases a decompressive craniotomy was performed for the relief of generalized or local cerebral edema. Four of them showed the typical appearance of a more or less marked swelling of the cerebral convolutions, patches of cerebral purpura, and bluish dark, thickened veins without circulation of blood. Sometimes there is a concomittant, yellowish infectious or noninfectious leptomeningitis (Fig. I.23). However, surgical intervention is not unaccompanied by risk. In one case, our earliest one, we incorrectly interpreted the findings as a space-occupying lesion. Coagulation of the thrombosed veins was followed by multiple extensive hemorrhages, and the patient died finally from extensive cerebral edema. We believe, therefore, that one must avoid resection of thrombosed cortical veins. From these experiences we conclude that surgery has its definite place for removal of large intracerebral hemorrhages and abscess formation and for relief of localized or generalized increased intracranial pressure.

The mortality rate with the infectious and noninfectious cerebral venous and sinus thrombosis is high. In our series of 92 cases it is 38 per cent and the prognosis is bad when the patient becomes comatose. Therefore, very early diagnosis is imperative when the symptoms first begin to manifest themselves.

Fig. I.18. Cerebral venous and sinus thrombosis in a 32-year-old patient receiving oral contraceptives. Venous drainage through the rete mirabile into the veins of the scalp (arrows) and through the basal lateral cortical veins.

Fig. I.19. Traumatic thrombosis of the longitudinal sinus with engorgement of the ascending cortical veins. A, post-traumatic angiogram; B, normal control angiogram 1 1/2 years later.

Fig. I.20. Mortality rate in the different treatment groups.

However, if the patient survives, the end results are encouraging. Of our 48 survivors, only three were more or less disabled by a motor hemiparesis and some amnesic aphasia, and a mild residual epilepsy was observed in three patients.

Conclusions

A distinction is to be made between noninfectious and infectious cerebral venous and sinus thrombosis. The diagnosis is very likely in the presence of signs

Fig. I.21. Right frontal venous and sinus thrombosis with subcortical abscess formation in a 53-year-old patient. Displacement of insular arteries, no phlebogram obtained.

Fig. I.22. A and B. At operation thrombosed veins and subcortical abscess in the right insular region. Death from cerebral edema and multiple, small subcortical abscesses.

Fig. I.23. Typical operative findings in cerebral venous thrombosis: swelling of the cerebral convolutions, patches of cerebral purpura, and dark, thickened veins without circulation of blood (case of Fig. I.14).

of obstruction of superior cerebral veins and of increased intracranial pressure which occur in the puerperium and after abortion or in the course of local or remote infective conditions. But in the great majority of cases the diagnosis remains difficult. The thrombosis may be limited to a few veins or a part of a sinus, or it may be more generalized.

The cerebral circulation is damaged by the venous thrombotic process, with consecutive brain edema, cerebral infarction with a more or less extensive subcortical hemorrhage, and, occasionally, intracerebral abscess formation.

The diagnosis of cerebral venous thrombosis can be made with reasonable certainty by means of serial cerebral angiography at the onset of the disease. The study of the capillary and venous phase of the angiogram makes it possible to diagnose the changed conditions of cerebral venous circulation. This radiological method, and electroencephalographic examination as a further auxiliary method, provides an accurate diagnosis and makes possible an early introduction of antibiotic and anticoagulant treatment.

A series of 92 patients with infectious and noninfectious cerebral venous thrombosis is reported, the clinical course of the illness is described, and the symptoms and signs are evaluated. The typical angiographic appearance of the disease is given. The results of treatment, partially with antibiotics and with anticoagulants, partially with antibiotics alone and combined with surgery, are evaluated. The mortality rate of the 71 treated cases was 20 per cent. Surgical intervention was confined to decompressive procedures and to removal of intracerebral hemorrhages in twelve cases and of multiple intracerebral abscesses in one case. As sequelae of the disease, slight hemiparesis, amnesic aphasia, and epilepsy were noted. An anticonvulsive treatment was applied in three cases.

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