SPECIAL ARTICLE

CONSIDERATIONS ON A SERIES OF MENINGEAL SUBARACHNOIDAL HEMORRHAGES IN CHILDREN ABOVE THE NEONATAL PERIOD*

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Our understanding of meningeal subarachnoidal hemorrhage has undergone considerable change in the last twenty years. Formerly, the diagnosis of meningeal hemorrhage was considered an end in itself, on a par with cerebral softening or hemorrhage. Aside from cases of arterial hypertension of the newborn and certain well known blood diseases, its etiology remained uncertain and more than half of the meningeal hemorrhages of adolescence or young adulthood were classified as «idiopathic».

Now, however, thanks to modern techniques of investigation, such as electroencephalography and angiography, the majority of the so-called idiopathic hemorrhages have been assigned a cause: vascular malformations, arterial aneurysms or arteriovenous angiomas, sufficient to account for the bleeding. Among adults, all authors agree, such malformations account for 50 per cent of meningeal hemorrhages.

However, few recent works have been devoted to the meningeal hemorrhages of children, apart from those of the early neonatal period. It is therefore to this phase of nervous system pathology of childhood that we have dedicated this work.

We have collected fifteen cases of children who presented a clinical picture of meningeal hemorrhage and in whom lumbar puncture revealed bloody cerebrospinal fluid. We included all the children whose major original symptoms were meningeal and in whom bloody cerebrospinal fluid was found but we excluded patients who, at the onset, presented a clinical picture of cerebral hemorrhage even though the hemorrhage later became cerebromeningeal.

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Incidence

The incidence of meningeal hemorrhage in children, after the neonatal period, is low. All authors make a distinction between the hemorrhages of the neonatal period, the incidence of which is well known and which present a special therapeutic problem not discussed here, and the meningeal hemorrhages of adults. These, too, are well known, and numerous works have been devoted to them. However, little has been done, to date, on the problem of meningeal hemorrhages in childhood. Meningeal hemorrhage is very frequent at birth but quite rare in early childhood. Subsequently, the frequency of meningeal hemorrhage increases with age; in fact, much of the literature concerns the prepubertal period. The frequency then continues to increase after puberty and reaches a maximum level towards the forties. Our series is no exception to this observation: two thirds of our patients were between ten and fifteen years old.

In 1945, in a general review of world medical literature, Brandt (5) assembled 58 cases of subarachnoidal hemorrhage in childhood which could not be attributed to vascular malformation. The etiologies suggested by the various authors concerned were: vitamin K deficiency, general infection, convulsions and encephalitis of undetermined origin. In actual fact the majority of these suggestions were not convincing.

Our personal material comprises fifteen cases of meningeal hemorrhage in children collected during the last ten years.

It is obviously difficult to compare statistically the cases of meningeal hemorrhage arising in childhood with adult cases owing to differences in the origin of the clinical material. For example, ten of our fifteen cases came from a neurosurgical clinic where, during the same period, some 200 cases of meningeal hemorrhage were observed in adults. If we were to base a discussion of the incidence of this condition on these figures we should arrive at the conclusion that the incidence among adults is greater by a ratio of twenty to one, which would be in accord with a good deal of the statistical material published in the literature. It is our feeling, however, that this presents a misleading picture and that although rare in childhood, meningeal hemorrhage is by no means without exception and that its study is worthwhile, particularly in view of the therapeutic possibilities.

Clinical Study

Due to the rupture of a vessel in the pia mater, arachnoid, or even in the cortex and to the diffusion of blood into the arachnoidal spaces,

meningeal hemorrhage produces a meningeal syndrome which has been frequently discussed. However, damage to the adjacent parts of the brain can often disturb the clinical picture so that it is not at all typical. We shall therefore examine, successively, the onset of the hemorrhage, the period of its development, and its clinical evolution, and shall attempt to present the typical picture in each phase together with variations from it.

1. The Onset

Classically the onset is sudden. The patient is struck as though by a thunderbolt. Two major signs generally accompany the onset: cephalalgia and vomiting. Cephalalgia is sudden and usually extremely violent, giving the impression of extreme tension. It sometimes appears following physiologic cerebral congestion during exertion, but more often than not is totally unexpected and sometimes attacks during sleep. The episode is often described as feeling «as though something had burst», and the precise localization of this initial sensation should always be looked for.

These very specific symptoms which are valid among adults may not be so typical in the child due to the lack of ability to make precise descriptions. But even here cephalalgia remains the chief symptom, and we have, in fact, found it in most all of our cases.

From the point of view of frequency, vomiting is the second most common sign of the onset of a meningeal hemorrhage. It usually commences suddenly and is projectile in type. This sign never fails to appear in children, and although appearing more frequently during the initial stage of the disease it sometimes occurs at a later time.

An insidious beginning with cephalalgia and a clouded sensorium, which becomes progressively worse in a few days, is also a possibility. Our case number nine was an example of this type of onset. The patient had his first attack at the age of eight in 1948. This was followed by coma which persisted for 48 hours with a residual right hemiplegia and aphasia. An exploratory burr hole at the left temple, which was performed at this time apparently did not reveal meningeal hemorrhage. The patient recovered gradually and was able to finish school. On December 1, 1956, when the patient was fourteen years old, cephalalgia which became increasingly worse appeared accompanied by vomiting. This state was followed in a few days by one of stupor. A physical examination on December 3,

revealed a meningeal syndrome with stiffness of the neck and a positive Kernig's sign. Lumbar puncture revealed meningeal hemorrhage. Due to an increasing state of stupor, the child was admitted to the neurosurgical clinic of the hospital. At this time there was a discrete right hemiplegia. An electroencephalogram (E.E.G.) showed slow left temporofrontal waves and left carotid angiography revealed prefrontal angioma. On December 10, 1956, surgical intervention was performed with ablation of a left frontal angioma and an adjacent hematoma. The patient then followed a normal postoperative course. In 1958 the boy was well and an E.E.G. showed only a slow focus in the left frontotemporal region.

A third sign which may appear at the onset is a generalized convulsion. Although this sign is rare at the onset, it is said to be more frequent in children than in adults. Our case number five, a long - time epileptic, was an example of this presenting symptom. This child was hospitalized on account of a meningeal hemorrhage which was observed following a convulsive crisis.

2. The Period of Development

This period is comprised of many series of symptoms which we have, perhaps artificially, grouped in the following fashion, and which we will discuss separately : a. disturbances of consciousness, b. classical signs of meningeal hemorrhage, c. vegetative signs and d. neurological localization signs.

Disturbances of consciousness are classically very frequent occurrences progressing rapidly from a simple clouded sensorium to coma. The most frequent type of disturbance is simple stupor and marked asthenia. Even in children, the frequency of cases where the consciousness is only slightly disturbed, is quite large. Our case number fifteen is a typical example of this type of disturbance. Sometimes disturbances of consciousness may be absent at the beginning and only manifest themselves later on. Case number eight is an illustration of this: M.C., an eleven year old boy, developed violent and brutal right temporal cephalalgia accompanied by vomiting, in December 1957. Six days later he showed progressive stupor and definite meningeal signs. Lumbar puncture revealed xanthochromic fluid. Ankle areflexia and bilateral mydriasis were present. An E.E.G. demonstrated theta activity in the middle and posterior temporal regions on the right. Right carotid angiography produced an image

of a right temporal hematoma with a small angiomatous knot. Surgical intervention was performed on December 13, with ablation of the temporal hematoma and angioma which occupied the tip of the lobe. In 1960, the child had returned to normal school life and the clinical examination was negative.

Classical signs of meningeal irritation include cephalalgia and vomiting. These signs, which appear at the onset of an episode of meningeal hemorrhage, may also persist in the development period. In general this is a negative sign and means that the bleeding is persisting. This is especially true if the vomiting continues at this time. The physical examination may also reveal contraction of the spinal muscles, i.e., stiffness of the neck, Kernig's sign, Brudzinski's sign and limitation of Lasègue's maneuvre. None of these signs seems to be more significant than any of the others, but it is always possible that any of these symptoms may appear 24 to 48 hours later, and this well known fact should always be taken into consideration before eliminating the possibility of meningeal hemorrhage in a diagnosis. Our case number six is an example of the classical meningeal signs: A.S., an eight year old girl with no previous symptoms, developed frontal cephalalgia with a brutal beginning accompanied by vomiting, in December 1959. At this time the child also collapsed without losing consciousness.

Physical examination revealed a discrete right-sided hemiparesis, and 48 hours later severe meningeal signs appeared which made hospitalization necessary. Examination of the cerebrospinal fluid revealed that it was bloody and at this point the child was transferred to the neurosurgical service. Fifteen days later the meningeal signs had almost totally regressed but neurological examination showed the persistence of the right hemiparesis. A left carotid angiogram performed at this time revealed nothing abnormal. Ventriculography revealed displacement of the ventricles to the right with deformation of the roof of the lateral left ventricle. Surgical intervention made possible the evacuation of a hematoma situated in the superior section of the left parietal region. In the internal face of the cavity there was an angioma which was ablated simultaneously with the hematoma. Recovery was complete with the disappearance of the right hemiparesis.

The search for these various classical signs does not present a major problem in children, but their verification is easier in older children than in babies.

The severity of the vegatative signs is important from the point of view of prognosis. Besides the disturbances of consciousness, these signs are characterized by injury to the great vital functions: respiration, deglutition, temperature, and pulse. These should always be very carefully studied.

Irregularity of the temperature with an arrest at 38 C. or 39 C. is quite a frequent occurrence. This is not in itself a sign of the gravity of the condition, but this cannot be said of a rapid or progressive elevation in temperature over 40 C., which is indeed a grave sign.

A slight slowing down of the pulse rate is quite frequent, and is a significant diagnostic sign. A severe slowing down is usually correlated with the severity of the hemorrhage. On the other hand, an excessive acceleration usually means a ventricular inundation.

Respiration is generally not greatly affected, at the most a slight tachypnea is heard. When the tachypnea is highly accentuated or there are disturbances in rhythm with respiratory pauses, it means that there is a serious irritation of the diencephalic structures.

A close study of deglutition makes it possible to estimate the depth of the coma, and the gravity of the vegetative attack. It may also prevent fatal errors at the time of attempted feeding by mouth.

The study of all of these signs should be very detailed and frequently repeated. In this way estimation of the evolution of the disease and its prognosis should be possible. These very important signs may be closely related to the amount of bleeding, but they more often mean a local attack caused either by overflow which erupts into the ventricular cavities or by dilaceration of the cerebral parenchyma in the deep structures of a hemisphere.

The detailed study of the signs of neurological localization will sometimes make possible the localization of the lesion. Many clinical forms may be differentiated according to the results of this study. It has been found helpful to group these signs into four general categories.

In the first category the neurological examination is entirely negative, or shows nothing but signs which indicate pyramidal irritation. In this instance a bilateral Babinsky and an exaggeration of the tendon reflexes in the lower extremities can be observed. Also, the abolition of the Achilles tendon reflex or even of the patellar reflex may be noted. This is due, according to the majority of the auth-

ors, to the accumulation of erythrocytes around the spinal nerve roots. In this case a pure meningeal hemorrhage may be the cause.

In the second category the neurological examination shows minor but positive neurologic signs which imply the existence of a small cortical lesion. A decrease in unilateral motor ability, an isolated Babinsky sign and a lateral homonomous hemianopsia can be noted. An isolated attack on certain cranial nerves is also possible.

In the third category major neurological signs, generally hemiplegia with or without aphasia, dominate the clinical picture. We are then faced with a cerebromeningeal hemorrhage. Normally this picture is not the same as that of subarachnoidal hemorrhage but is more like cerebral hemorrhage. Indeed, most often this group of signs indicates an intracerebral hematoma which has erupted into the subarachnoidal spaces.

In the fourth category the examination reveals signs of medullary localization. This clinical picture, which is associated with a stiffness at the level of the dorsal or lumbar spine and with an insignificant headache, indicates the presence of a spinal hemorrhage. We have had two such cases.

3. Clinical Evolution of the Hemorrhage

The progression of symptoms in cases of meningeal hemorrhage varies considerably according to the patient, but is always serious from the point of view of survival. Mortality varies according to the statistics reviewed and the type of patients recruited. The statistics of the classical authors indicated a 50 per cent mortality rate. At the present time, it averages about 30 per cent. Relapses are frequent, and are said to be always more serious than the initial onset. Recovery without sequelae is certainly possible, but it is less frequent than recovery with sequelae. It is difficult to compare the various statistics published on this subject. The gravity of the hemorrhage varies according to age but also according to the cause. Arterial aneurysms are certainly the most serious of the etiologies. However, the foregoing statements apply only to adults. The absence of statistics concerning children makes difficult all kinds of prognostic estimation. The majority of the published cases remain isolated, but seem to have a favorable prognosis. Hess reported two deaths among six cases published in 1942, one case with multiple relapses and three with isolated hemorrhages. Hollenhorst (11), in ten cases, reported two deaths. The eventual outcome of this condition is very poor

according to this author and he feels that the frequency of sequelae (hemiplegia, epilepsy and blindness) is particularly high. In our experience, we had only one death and four children who suffered from sequelae, which were not very distressing from a social point of view. We believe that the prognosis and the ultimate outcome depend especially on the causal lesion and its localization.

Etiology

A general observation about the etiology of meningeal hemorrhages concerns the antecedent episodes in the patient's prior medical history. When a meningeal hemorrhage is suspected, one must always look for former analogous episodes. The proportion of patients who have had former episodes is always small, and the episodes are usually minor although their significance is interpreted in various ways by various authors. Meningeal hemorrhages which occur during thrombopenic purpura, for instance, are very well known. It is also necessary to make a thorough physical examination in order to eliminate a general disease as the cause of a meningeal hemorrhage. In our experience, we encountered one case where the patient had had two previous hemorrhages, confirmed by lumbar puncture, before the one that led to the diagnosis.

Case number three, A.A., a girl, was born in 1939. In 1952 she had two meningeal hemorrhages confirmed by lumbar puncture from which she recovered fully without sequelae. In March, 1959, sudden coma with vomiting and meningeal stiffness occurred. A lumbar puncture revealed the existence of a third meningeal hemorrhage. The coma returned fifteen days after recovery from this episode and the physical examination revealed a slight right hemiparesis and a hemianopsia in the right superior field. Left carotid angiography revealed an angioma of the «pli courbe», between the wall and the midline.

The search for epileptic antecedents is always practical, because the appearance of seizures during the years before the hemorrhagic accident favors a vascular malformation. This fact has been noted by several authors.

Our case number five is an example of epileptic antecedent to meningeal hemorrhage : This girl, M. A. P., had had increasingly frequent generalized epileptic seizures. In 1957, at the age of ten, she was hospitalized at a neurosurgical clinic for a prolonged

post-ictal coma. Physical examination revealed a temperature of 39 C., stiff neck, bilateral Babinsky and tendinous areflexia in the arms and legs. Lumbar puncture 48 hours later revealed hemorrhagic fluid and the physical examination at this time showed the presence of right hemiplegia. An E.E.G. showed asymmetric traces with alpha rhythm on the left and theta on the right, mixed with generalized slow waves in the entire right hemisphere. Bilateral carotid angiography indicated a small angioma of the left anterior cerebral artery with an adjacent hematoma on the right and a voluminous arteriovenous angioma at the posterior part of the Sylvian artery without an apparent hematoma. Surgical intervention through a left frontal craniotomy on July 12, 1957, made possible evacuation of an intracerebral hematoma in the internal capsule whence the angioma originated and its exeresis was accomplished. The postoperative period was not marked by any definite amelioration. Slowly, severe cachexia developed and finally death followed.

We have purposely limited the subject of meningeal hemorrhages to those of spontaneous appearance. For several years our knowledge of this subject was rather limited. Hemorrhages of the aged or arteriosclerotic persons did not present any problem in etiology but the hemorrhages occurring in young adults without hypertension and even more, those occurring in children, were considered idiopathic. This term hid our ignorance. Various etiologies were considered by different authors: syphilis (Audry), tuberculosis (Lafforgue), epidemic encephalitis (Cordier, Achard), and neurotropic viruses (Sovel). These hypotheses were all abandoned because of lack of evidence. In 1924, C. P. Symonds suggested the idea, supported by his necropsy observations, that the etiology could be related to the frequency of aneurysms of the circle of Willis. Real enlightenment occurred in 1927 when Ega Moniz discovered the technique of cerebral angiography. This technique, permitting investigation on living persons, made possible the study of the frequency of arterial aneurysms and of angiomas, whose occurrence had been known since Virchow. Cl. Vincent, in 1938, was one of the first to realize the importance of these vascular malformations which were later studied by Bailey, Dandy, Olivecrona, Richardson, Jefferson and others. After that time, the problem became one of determining the statistical incidence of vascular malformations in meningeal hemorrhages. Originally estimated at 80 per cent, the present statistics are in general accordance in making them responsible for 50 per cent of the cases of meningeal hemorrhage.

The causes of these hemorrhages are the same for adults as for children in most studies, except for the relatively unimportant role of vascular disease at this early age, although atheroma can exist in adolescence and even in childhood.

Frank Ford, in the latest edition of his book on pediatric neurology, cites the following etiologies : 1. vascular anomalies : arterial aneurysms, and especially arteriovenous angiomas which he considers the most important cause, and of which he presents several personal observations; 2. thrombophlebitis of the sinuses chiefly responsible for subdural hematomas; 3. certain blood diseases which can cause subarachnoidal hemorrhages; 4. periarteritis nodosa and infantile arteriosclerosis which are not without exception at that age and which may cause meningeal hemorrhages. These hemorrhages are not easy to detect in the midst of the extremely complicated clinical picture of the accompanying diseases.

Moreover, ideas concerning the etiology of meningeal hemorrhages in children have undergone the same modifications as those concerning the adult. Thus Brandt (5), notes in 58 personal observations which did not correspond to malformations, that in some of them a malformation could well have existed but that no examinations were made to prove it. However, the cases published recently all show vascular malformations. Our own statistical survey also reveals the frequency of this etiology. In fact, in fifteen meningeal hemorrhages we found nine arteriovenous encephalic angiomas, one intracerebral hematoma which had diffused into the subarachnoidal spaces for an unexplained reason, three pure meningeal hemorrhages whose etiology was not discovered and two spinal hemorrhages due to medullary tumors. This last group, to which we shall refer again, is in general neglected by the majority of other authors. The literature shows, however, that it is not entirely uncommon. We shall discuss separately each of these etiologies.

Vascular encephalic malformations comprise arterial aneurysms, angiomas, of which the most frequent is the arteriovenous angioma, and the various systemic vascular malformations of the nervous system (Sturge - Weber disease, or Lindau - Von Hippel's disease). In the adult, arterial aneurysm is the malformation which most frequently causes meningeal hemorrhages. In children this does not seem to be the case.

An arterial aneurysm can be defined as a blind pocket formed from the altered interior wall of an artery, filled with coagulated or

non - coagulated blood, and communicating with the lumen of the vessel. We will not discuss fully the problem posed by these aneurysms, but will cite Pluvinage (21) and the recent report of Allegre and Vigouroux (1) as references concerning this lesion.

At present this anomaly is considered to be congenital and in the great majority of cases represents one of the essential causes of meningeal hemorrhage in young adults. Even though it manifests itself very rarely at this age, arterial aneurysm can cause meningeal hemorrhage even in children. This etiological hypothesis should never be overlooked because of its gravity and the therapeutic intervention which it necessitates. In order to make this diagnosis, complementary investigations are necessary and the treatment is surgical more often than not. These problems will be discussed later in this paper.

The term cerebral angioma encompasses vascular malformations consisting mainly of voluminous vascular knots formed by arteries, veins and capillaries with the special characteristic of more or less fluent arteriovenous anastomosis. Classically cerebral angiomas appear less frequently than arterial aneurysms, but this opinion seems to have less support at present, due to more frequent diagnosis of arteriovenous angiomas by angiography in neurosurgical centers. Their congenital origin seems to be demonstrated according to Bailey, Bergstran, Olivecrona, Tonnis and Van Bogaert. The classification of cerebral angiomas has been much discussed and therefore we shall not go into the details of these classifications but refer to the thesis of Winninger (24). The most frequent of all these malformations is one of the arteriovenous type with an arteriovenous shunt, which is the most important characteristic of the condition, and which is responsible for the frequently severe anoxic disturbances. These lesions have a variable location and, though they often have a conical shape with a parietal base and a central summit, all forms are seen. They may remain latent for a long time and they are likely to produce three types of clinical signs : 1. epileptic accidents; the percentage of epilepsy due to angioma is certainly low but the existence of an angioma should not be neglected when there is a case of focal epilepsy. Angioma can cause epilepsy of a central encephalic type such as we saw in our case number five, previously discussed in this paper. 2. Signs of permanent neurological deficiencies; hemiplegia, hemianesthesia and hemianopsia, are of considerable value in localizing the lesion. 3. Hemorrhagic accidents which could lead to or follow an epileptic history. These are most often unexpected (we have observed nine such cases), and they may be of various types: pure meningeal

hemorrhage; meningocerebral hemorrhage; and cerebromeningeal hemorrhages.

Pure meningeal hemorrhage is very rare and is only seen in cases of very superficial malformations and the hemorrhage is usually of small volume. It is most often seen in the adult and Laine feels that these cortical malformations remain latent more often than the more severe malformations. He reports, however, two infantile cases and we have also found two such cases.

In the first case, the child twice suffered from a pure meningeal hemorrhage after which he recovered very quickly. It was only at the third episode that a more severe clinical picture brought the child to the neurosurgical department where the diagnosis was made.

In the second case, J.L.A., case number two, an eight year old boy, developed a violent cephalalgia with vomiting, in June 1954. The next morning a meningeal stiffness with a temperature of 38.5 C. was present. Lumbar puncture, performed twice, revealed hemorrhagic fluid. On June 30th, there was persistence of stiffness with a hyperextended neck. A neurological examination was normal and skull radiography showed a thickened vault of normal texture. An E.E.G. revealed slow rhythms with peaks in all leads at 6c/S and even slower waves in the interior part of the left hemisphere. Left carotid angiography demonstrated a large arteriovenous angioma occupying the frontal pole and nourished by a voluminous branch originating from the left anterior cerebral artery; right carotid angiography also showed the angioma. In an operation on July 2, 1954, the prefrontal cortex was discovered to be completely covered by a network of large arterialized veins. The nourishing pedicle was approached internally and ligated one centimeter from the cerebral artery. Complete ablation of the angioma, which almost meant a prefrontal lobectomy. was performed. There were no postoperative complications and in January 1961, no neuropsychic signs were present and an E.E.G. was normal.

Meningocerebral hemorrhage is more frequent in the presence of an arteriovenous angioma than in the presence of an arterial aneurysm, and this can be explained in the following manner. An arterial aneurysm is most often situated in the circle of Willis or at the point of issue of the arterial trunks, a region where the vessels are not in very intimate contact with the cerebral parenchyma. Therefore, the occurrence of hemorrhages here would cause few parenchymatous lesions. The same cannot be said about an arteriovenous

angioma which almost always concerns a portion of the cerebral parenchyma. Its rupture will always damage the parenchyma causing destruction or anoxia of varying degrees. We have observed two cases which fall into this pattern. In the first case, (A.A., case number three, cited previously), the third hemorrhagic accident which led to the diagnosis was of the meningocerebral type. The same can be said of the second case: H.F., a nine and a half year old boy, (case number one) developed sudden cephalalgia which was extremely violent and accompanied by vomiting, in December 1955. Some hours after the first symptoms appeared the child was in a state of deep coma. Physical examination revealed a definite meningeal stiffness and the cerebrospinal fluid was hemorrhagic. Fifteen days after admission to the hospital, the child came out of the coma. Physical examination revealed a bilateral mydriasis and a homonomous right lateral hemianopsia. An E.E.G. showed generalized dysrhythmia which was stronger and increased more in the left temporal region. Left carotid angiography demonstrated a very small saccular aneurysm and an angiomatous pellet hanging from the posterior temporal artery. An operation was performed on December 17, 1955, with evacuation of a superficial and deep occipital hematoma, and complete ablation of the aneurysm and of the sclerotic tissue surrounding them. This was equivalent to an external occipital lobectomy sparing the calcarine region. A smooth postoperative period followed with no sequelae except for a right hemianopsia. In this less typical case, there was a hematoma going into the depth of the hemisphere but the superficial seat of the aneurysm caused us to think that the rupture had occurred at the same time both in the subarachnoidal spaces and in the cerebral parenchyma.

From this observation it can be seen how difficult it is to distinguish between cerebral and meningeal hemorrhages and the mixed forms. All of our observations are based on direct anatomical verification of the lesion. Before this kind of study, diagnoses of pure meningeal hemorrhages were based on simple clinical examinations. Now with angiographic or further paraclinical examinations and anatomical verification, it is possible to differentiate more accurately the various types of lesions. For the general practitioner faced with a patient in coma with bloody cerebrospinal fluid, if there is not significant hemiplegia, the case is one of meningeal hemorrhage. A paraclinical examination and surgical intervention would probably show that the cause was a cerebral hemorrhage which had reached the ventricle or the subarachnoidal spaces but the significant factor would have been the hemorrhagic cerebrospinal fluid. This type of

accident is the one most frequently encountered in the study of arteriovenous angiomas in children. This was Laine's opinion and we have also seen it in six out of nine cases of angioma (cases five, six, eight and nine, cited previously, and cases four and seven, to follow).

Case number four, P.C., an eight year old girl, on September 4, 1957, developed a sudden onset of meningeal signs with stiffness of the neck, cephalalgia and vomiting. The E.E.G. on September 12, showed generalized delta anomalies, more severe in the right posterior temporal region. Ventricular puncture revealed hemorrhagic fluid from both sides. Bilateral carotid angiography demonstrated a left temporal hematoma. Surgical intervention was performed through a posterior temporal burr hole. After the evacuation of the hematoma, it was seen that the infero-internal part of its sac was formed by a cluster of angiomatous arterioles which occupied the infero-external cortex and the hippocampus. Resection after cleavage of this cluster was performed. A smooth postoperative period was followed by recovery, without sequelae, which has persisted one year following surgery.

Case number seven, M.L., a nine year old girl, suddenly developed signs of meningeal hemorrhage with immediate coma. Lumbar puncture confirmed the presence of meningeal hemorrhage. When the patient came out of the coma, right hemiplegia was apparent. Faced with the persistence of the meningeal hemorrhage, left internal carotid ligature was performed. Symptomatology regressed one month after this prccedure. Two years later Bravais - Jacksonian epilepsy occurred on the right side with secondary generalization. Six years later, after increasing severity of the seizures, left carotid angiography was performed. This revealed a left arteriovenous tempororollandic aneurysm which was responsible for the meningeal hemorrhage in the patient's childhood and the succeeding epileptic attacks.

In all these cases the clinical examination showed definite meningeal signs. Neurologic signs (hemiplegia) dominated the clinical picture in only two cases. In all the cases, however, surgical intervention made possible the evacuation of a hematoma due to the rupture of an aneurysm. It is the formation of this hematoma which marked the beginning of the clinical history. Its rupture into the subarachnoidal spaces resulted in the clinical picture of meningeal irritation. These cerebromeningeal hemorrhages are certainly the most serious ones among hemorrhagic accidents due to arteriove-

nous angiomas. In fact, in our six cases, we have noted one death, two recoveries with secondary epilepsy despite medical treatment and three recoveries without notable sequelae. However, these children are receiving barbiturate treatment.

Arteriovenous angiomas are therefore an important cause of meningeal hemorrhage in children. Their presence must be suspected especially if focal neurological signs or a past history of epilepsy are associated with a meningeal hemorrhage. Arteriovenous angiomas have a severe prognosis if left untreated. The patient is always subject to the possibility of a sudden rupture which could cause a severe cerebromeningeal hemorrhage. In this case the prognosis from the vital point of view is dubious. The length of the latent phase between the first hemorrhagic accident and the following ones varies in the published cases, from a few months to about twenty years. The average seems to be between three and five years. Whatever the length of time between them, recurrences are frequent and always more severe than the first hemorrhage. Chronic evolutionary changes, on the other hand, may await the patient: epilepsy caused by the formation of scar tissue in the involved area; other disturbances caused by anoxia and gliosis surrounding the malformation, whose evolutionary character is certain even though slow. It is important, therefore, to be able to make a diagnosis from the first hemorrhagic incident in order to give the patient necessary treatment. From this point of view, it can be said that arteriography represents one of the most important diagnostic tools, to which we shall return subsequently.

Systemic vascular malformations of the nervous system are the last of the groups which make up vascular encephalic malformations, and they have been gathered together here because of their particular clinical aspect. Vascular malformations in close proximity to the brain, involving the eye and the integuments of the face, can exist together with a similar malformation of the brain. Three syndromes have been described : 1. Lindau - Von Hippel's disease with cystic hemangioma of the posterior cerebellar fossa and cystic hemangiomas of the retina. Meningeal hemorrhage occurs rarely with this syndrome. 2. Sturge - Weber's syndrome with a cutaneous angioma in the region of the trigeminal nerve; ocular anomalies, mainly glaucoma and choroid angioma; vascular cerebral malformations of cortical origin and particular structure, more like telangiectasis than arteriovenous angiomas. Becoming progressively calcified, they are not very likely to cause hemorrhages. Meningeal hemorrhage in children has been seen with this syndrome according to Laine, but

in actuality it is quite rare. We have personally observed no such case among the children with Sturge - Weber disease which we have studied. 3. Bonnet Dechaume Blanc's syndrome, or encephalo - oculo - trigeminal angiomatosis, is comprised of a cutaneous trigeminal angioma, a cirsoid aneurysm of the retina, often causing blindness, and a cerebral arteriovenous angioma which is usually found on the dorsal side of the superior cerebral trunk.

We do know that the classical authors considered the search for a cutaneous malformation as most important when an intracranial angioma was suspected. Such a discovery can, of course, be useful, but its absence is of no particular value.

Spinal meningeal hemorrhages must be considered in the etiology of subarachnoid meningeal hemorrhages. Even though these hemorrhages can have various causes of their own, this group deserves individual attention because of the particular clinical signs, and the specific examinations necessary to reach a correct diagnosis and therapeutic confirmation. These hemorrhages are not rare. Gros and Girard have reported such cases among adults and we have, ourselves, observed two such cases in children.

Case number ten, R.D., an eight year old boy, developed meningeal signs with a severe onset, accompanied by lumbar pain, spinal stiffness, obstipation and febrile spikes at 38 C. He made a rapid recovery from these symptoms in two weeks. In October 1947, he again developed lumbar pain, associated with difficulty in running, and enuresis. In February 1948, he had difficulty in walking, stiffness of the trunk with lumbar hyperlordosis, diffuse pains on pressure of the lumbar spinous processes without any localization. Deep tendon reflexes were present in the legs and there was no sensory disturbance. Spinal radiography was normal, but the cerebrospinal fluid was xanthochromic, and contained 2.4 grams albumin per 100 ml. and five cellular elements per c. mm. Lipiodol given suboccipitally indicated a massive blockage at the level of the lumbosacral joint. Surgical intervention on March 10, 1948, made possible the ablation of an intradural tumor penetrating the intervertebral foramina. Histologic examination of the tumor revealed malignant meningoblastoma. There was a recurrence of the tumor in 1949 at the level of the occipital foramen.

Case number eleven, a fourteen year old girl, suddenly developed meningeal signs with lumbar pain and a temperature of 39 C. but without vomiting. Physical examination revealed intense spinal con-

traction and Lasègue's sign at 30 degrees. Lumbar puncture confirmed the presence of a meningeal hemorrhage. The child recovered from these symptoms in six weeks. Three months later she had a recurrence of the lumbar pain from which she recovered, by resting, after a few weeks. A year after the first hemorrhage, another painful episode, in the S1 dermatome, recurred, with hypesthesia on the external border of both feet. Spinal contraction was also present and myelography with lipiodol demonstrated an abrupt blockage at the level of L4. Surgical intervention made possible the ablation of a giant tumor of the cauda equina, which was identified as an ependymoglioma. Postoperative radiotherapy was used and to date there has been an apparent recovery.

Clinical Characteristics of Spinal Meningeal Hemorrhage.

Two special features appear to dominate the clinical picture : 1. at the time of the occurrence of the hemorrhage, cephalic signs (vomiting, cephalalgia, stiff neck, somnolence) seem to be less apparent, whereas spinal signs are more obvious (spinal pain, constipation, spinal stiffness and a diminished Lasègue's maneuvre). 2. The clinical examination or the antecedent symptoms usually point towards spinal cord involvement: pain, «like a belt» around the trunk, paraparesis and sciatic pain. However, the symptoms do not always appear in this manner as can be seen from case number ten, cited above.

Etiology of Spinal Meningeal Hemorrhage

Only the causes which require surgery are known and will be discussed here. Almost always an intraspinal or an intradural tumor is the cause and this tumor is most frequently an arteriovenous angioma. Girard and Garde estimate its frequency at three to four per cent of the medullary tumors. These angiomas cause repeated hemorrages with medullary or radicular compression. The combination of meningeal hemorrhage with signs of medullary compression should lead to this initial diagnosis. Lipiodol confirms medullary compression, but only surgical intervention makes it possible to specify the nature of the compression. The prognosis of these disturbances must be somewhat cautious as it is not possible to promise complete recovery or survival. Surgery is, in fact, often very difficult or even impossible in view of the frequent extension of the malformation. Radiotherapy does not prevent the danger of thrombosis, and spontaneous evolution of the disturbance can

lead to a clinical picture of subacute necrotic myelitis of the adjacent segment. We have observed no cases of this kind among children.

All medullary tumors can give rise to meningeal hemorrhages in the same way as cerebral tumors. However, this rarely occurs and medullary tumors have not been widely studied. We have seen them twice in our experience, in connection with highly embryonic tumors.

Medical and Cryptogenetic Hemorrhages

We have grouped these hemorrhages together, and include here also those hemorrhages whose origins have not been proven. These constitute four of our cases in this paper.

Case numer twelve, a fourteen year old boy, in 1959, developed, without notable previous symptoms, a sudden meningeal syndrome (cephalalgia, vomiting and a stiff neck). This was followed a few hours later by a right hemiplegia. Lumbar puncture made a few hours after the onset revealed bloody cerebrospinal fluid. The child was hospitalized the next day, and physical examination on admission confirmed the meningeal syndrome and the right hemiplegia. Lumbar puncture once more confirmed the meningeal hemorrhage and an E.E.G. revealed a certain alteration of the structures of the base and the left hemisphere. Arteriography demonstrated the presence of a left intracerebral hematoma situated in the Sylvian region. Surgical intervention was performed on February 15, 1959, and made possible the evacuation of the intracerebral hematoma. No vascular malformation was noted either at operation or on the angiograms. Following the operation there was almost complete regression of the hemiplegia.

Case number thirteen, a fourteen year old girl, in November 1958, developed violent cephalalgia accompanied by vomiting and fever. There were no known prior symptoms and the clinical examination at this time was negative. Forty-eight hours later, the family doctor suspected a subarachnoid hemorrhage which was the motive for hospitalization. Lumbar puncture verified the diagnosis of meningeal hemorrhage but the neurological examination did not show any signs of localization. An E.E.G. showed discrete alteration of the structures of the base. Recovery occurred in eight days. Further investigations were refused by the family, and two years later there had been no further recurrence.

Case number fourteen, a girl, developed sudden temporal cephalalgia accompanied by projectile vomiting. Half an hour later the child went into coma. A few hours later, at the time of hospitaliza-

tion, the clinical examination was negative apart from a sub-comatose state, and at this time there were no meningeal signs. Lumbar puncture and analysis of the cerebrospinal fluid revealed uniformly bloody fluid. Meningeal signs appeared the next day, at the same time as the abolition of the deep tendon reflexes of the lower limbs. Eight days later, the clinical examination was entirely negative and bilateral carotid angiography did not reveal any malformation. We have not had any news of the child since that time.

Case number fifteen, a girl, experienced a transient febrile episode which only lasted one day, in August 1956. A month later, she collapsed suddenly and was hospitalized immediately. Examination on admission showed total areflexia of the lower limbs, a temperature of 38 C., and clear meningeal signs were present (stiff neck and Kernig's sign). Lumbar puncture was performed at once and revealed grossly hemorrhagic fluid. Forty-eight hours later the child was hospitalized at the neurosurgical clinic for further investigations. Meningeal signs disappeared after ten days, and the reflexes reappeared after the fifth day. The E.E.G. was practically normal and bilateral carotid angiography did not reveal any malformation. The child recovered without sequelae and there was no relapse after four years.

We know which etiologies should be investigated before arriving at the diagnosis of cryptogenetic or idiopathic hemorrhage. Arterial hypertension is a frequent cause in the adult, but it was not the cause in any of our four cases. In adults, arterial atheroma is frequently associated with hypertension. Although atheroma is not usually a disease of children, according to Ford, it can exist. We might have taken this cause into consideration in the diagnosis of our case number twelve, but we had nothing to prove it. We must, all the same, note that some authors have proved histologically that arterial atheromas can cause cerebral hemorrhages in children. We have observed this condition in an adolescent of seventeen years but in spite of this, arterial hypertension and atheroma are exceptional in children.

The other causes which were mentioned at the beginning of this section on etiology were not found among our cases. In fact, they seem quite rare.

There remains nothing now but to call this type of hemorrhage «idiopathic». Actually, what does this term mean? Formerly this category used to include 50 to 70 per cent of the adult hemorrhages, and

at present it still encompasses twenty per cent of the cases. Which etiologies were used to explain these hemorrhages? They were the same for both adults and children. Bailey and Woodard (2), in studies of necroscopic material suggest the possibility of malformations which are too small to be visible with arteriography but which can be seen in anatomical sections. Ingraham and Matson also share this point of view. Brandt (5), in a review of published cases, considers the possibility of encephalitic crises of viral origin, insufficiency of vitamin K, vague infections which might cause a tendency toward blood disorders and the possibility of hemorrhagic accidents in the course of convulsive attacks. But these explanations do not, in fact, satisfy the authors and they wonder whether the cause might not be small microscopic malformations which have passed entirely unnoticed. Hollenhorst (11) and Hess, each of whom reported a few cases without vascular malformations, find no satisfactory cause. Evidently an infection can nearly always be found previous to the incident and all means (cerebrospinal fluid examination, inoculation, culture and general examination) should be undertaken in order to detect it.

In this question of etiology, two hypotheses have been suggested: 1. the presence of small vascular malformations, too small to be diagnosed by present methods of exploration and which repair themselves after the first incident, and 2. transitory disturbances of the vascular permeability, of unknown origin. In fact, no valid proof can be found to verify these hypotheses and we refrain here from drawing conclusions in any way. We would like to note, however, that two out of the three children in whom no cause could be discovered, have had no recurrence during the two years following the episode. This period is obviously too short to reject the possibility of an angioma. Our own observations and the data from the literature suggest the conclusion that in children, as in adults, the most important cause of meningeal hemorrhage is vascular cerebral malformation. However, in children, unlike adults, the arteriovenous angioma is practically the only causal malformation. It is difficult to define the cause of the hemorrhage if this malformation is absent.

Treatment of Meningeal Hemorrhages

We have grouped all treatment under a single heading even though it might have been more clear if treatment had been discussed along with etiology. However, by grouping the treatment thus, we want to try to indicate the procedures to be followed in all cases regardless of the etiology of the hemorrhage. The following steps should

be taken successively: 1. symptomatic treatment of the hemorrhage itself, and 2. etiologic treatment which should be started as soon as possible.

Symptomatic Treatment of the Hemorrhagic Accident

In a sudden accident which suggests the diagnosis of an intracranial hemorrhage, several problems arise. First, should lumbar puncture be performed immediately? Although study of the cerebrospinal fluid clearly reveals the hemorrhage and confirms the diagnosis, this procedure is not free from danger and could cause recurrence of the hemorrhage if it is done too early. Most of the children we observed had a spinal puncture in the initial stages of their illness and we did not believe that it aggravated the clinical picture. We feel that this procedure can be successful, if carefully done. The child should not be seated or handled roughly and only a few drops of the fluid should be taken out.

Second, should coagulant therapy be systematically prescribed? It is advised by many authors; but, in the case of vascular malformations it could encourage a thrombosis near the malformation and thereby enlarge the area of the softening. We did not prescribe this treatment for our own patients routinely, and only four of them received minor coagulant therapy. These patients recovered quickly, but three of them had presented no detectable lesion.

It is obvious that in a case of severe hemorrhagic shock a transfusion of fresh blood will always be helpful. Could the lowering of the blood pressure also prove useful? This problem arises especially in the adult with hypertension. It is true that high blood pressure can cause recurrence of the hemorrhage but a too sudden lowering could also cause softening of the brain tissue through thrombosis. The removal of blood would be definitely inadvisable in this case.

As to other therapeutic agents currently being used, their effect is not very well known from the aspect of cerebral circulation, and we have not personally used them on our patients. We would only recommend them in cases of arterial hypertension.

What treatment should then be undertaken? We feel that the approach should be cautious and one which does not impose any further risks on the patient. The child should be put on strict bed rest in the prone position. A few analgesic agents of the salycylate series

should be prescribed in order to relieve the cephalalgia. If vomiting hinders their administration, they should be given by rectum. Barbiturates should be prescribed automatically in order to prevent convulsive attacks which risk aggravation of the clinical picture. Only in the case of severe neurovegetative disturbances, should suppression of the neurovegetative reflexes be performed, and this should be done with great caution. We personally prefer the mixture Hydergine - diparcol to the «classical cocktail», Largatyl - Phenergan - Dolosal, whose effect on the respiratory centers can be disturbing. The dosages vary according to the condition of the patient.

Etiological Investigation

In this section etiological investigation and treatment will be discussed. This type of investigation usually consists of verbal interrogations and a complete clinical examination of the patient with special attention to the nervous system. We have mentioned previously the particular points which should be developed during a study of the past history, but a number of paraclinical examinations should be given to each patient. Especially important are: 1. ophthalmological examination, 2. cranial radiography, 3. electroencephalogram, 4. cerebral angiography, and 5. ventriculography.

The ophthalmologic examination should include examination of the eye grounds, which could reveal signs of meningeal hemorrhage and papilledema. The possible presence of a retinal aneurysm should not be overlooked in any case. When the patient is conscious this examination should include study of his visual fields. In two of our patients, cases one and three, we were able to discover, in this manner, homonomous lateral hemianopsia, a valuable clue in locating the lesion.

The use of simple cranial radiography should be automatic, even if it is only a matter of control to eliminate the possibility of an undiscovered fracture, and it should be performed before angiography. It may show dilatation of some of the foramina of the base of the skull in the case of a large arteriovenous angioma. It did not reveal anything significant in any of our patients.

An electroencephalogram, a harmless procedure, should be done as soon as possible. The less the clinical examination gives signs of localization, the more interesting the E.E.G. is apt to be. It can demonstrate two series of signs: 1. signs of diffuse damage due to the involvement of all the formations of the base and sometimes cerebral

circulatory deficiency (leakages through an arteriovenous fistula); 2. signs of more or less localized hemispheric abnormality indicating local cerebral attrition which is most often related to a vascular malformation. Signs of damage to the structures of the base are of various intensities, but in general are in proportion to the severity of the hemorrhage and to the degree of involvement of the consciousness.

They can be minimal, as is the case with hemorrhages which are of little importance. Localization of the hemorrhage by E.E.G. is sometimes difficult in cases of severely disturbed tracings. However, the predominance of slow waves, with more definite slowing on one side than the other, unilateral flattening of the tracing and a focus of slow peaks are extremely useful signs in localization. Almost all of our patients have had at least one E.E.G. examination. Sometimes the E.E.G. was only slightly disturbed, or as in case number fifteen almost normal, but these cases were ones of insignificant hemorrhage whose etiology was not proven. In all cases of arteriovenous angioma the E.E.G. showed a focus of localized cerebral damage and usually signs of quite severe damage to the structures of the base. This enabled us to direct radiologic investigation when the clinical examination did not show signs of localization. In one of these cases, for instance, the E.E.G. showed two contralateral foci, and bilateral angiography revealed an angioma on both sides. The E.E.G. is then a very useful procedure making possible a precise topographic diagnosis, as well as indicating the direction of further investigations.

Cerebral angiography has modified our knowledge of meningeal hemorrhages and has considerably increased therapeutic success, as we have already indicated. Its technique is well established for the adult patient except for vertebral angiography, but in children it is necessary to modify the procedure. A general anesthetic is almost always required, and we have used it consistently except when the patient was in a deep coma. Injection of the carotids is generally easy after three years of age, but this is not so with injections of the vertebral system. Accidents are extremely rare, and insignificant in expert hands. Our experience in a neurosurgical department where cerebral angiography has been practiced almost daily for the last three years, has shown us that accidents have not altered the expected results and that no deaths have occurred in this period. Twelve of the children had one or several angiograms with no accidents; the youngest was five years old. Cerebral angiography usually makes possible the detection of a malformation if one exists. An arterial aneurysm, for

instance, shows as an opaque spot hanging from an artery which remains visible during the time when the venous system is injected and visible. It is often necessary to perform the procedure several times in order to visualize its size and shape, to differentiate the aneurysm from an abnormal vascular ring, and especially to determine how it is inserted, and the size and morphology of its pedicle. An arteriovenous angioma appears as a more or less diffuse, spread - out opaque spot with dilated vasculature and prematurely filled drainage veins due to a short-circuiting of the blood through an arteriovenous shunt. Angiography demonstrates the shunt, and also shows the topography and the nourishing pedicles of the malformation. Finally, angiography might show no pathological image or only a vascular gap with displacement of the vessels which is an indication of an intracerebral hematoma. Some authors advise the regular practice of bilateral angiography because of the possibility of multiple malformations. This is not always advisable but should be performed if the first angiogram does not elucidate the diagnosis. From our twelve cases we have obtained the following results: in two, a normal bilateral angiography; two cases of intracerebral hematoma, (one of these patients was suffering from an angioma); seven typical pictures of arteriovenous angioma with or without associated hematoma; and one case which showed no pathological picture in spite of the presence of a hematoma and an angioma. Although this examination has its limits, it is still of considerable value.

Ventriculography is, at present, of secondary importance since angiography tends to be more revealing. It is not essential in every case since it only reveals displacement or deformity of the ventricules. It should be performed only in cases of suspected intracerebral hematoma, such as we saw in our case number six, where ventriculography made possible the diagnosis.

Myelography with lipiodol, performed most often through the suboccipital route, makes possible the determination of the level of the compression by indicating the point at which the opaque material is blocked.

By etiological treatment we mean all kinds of surgical intervention, but we shall not undertake here a full study of the indicated and proposed techniques, (see Allegre and Vigouroux (1) on arterial aneurysm and Winninger (24) on arteriovenous angiomas).

Among the proposed methods, ligation of the carotid is only indicated in cases where direct extirpation of the lesion is contra-indicated. We feel that in children we must be even more cautious than in the

adult. Early correction of a malformation which may continue to evolve and lead to some serious developments is desirable. The ligation of the carotid does not prevent arterial aneurysms from rupturing. Therefore this procedure is only recommended in aneurysms which have developed at the root of the carotid, where the cavernous sinus begins. This is a rare but sure indication for «trapping» (Dandy). In children then, ligation of the carotid is a more risky procedure than in the adult. It was performed in only one case of our series, case number seven, at a time when our knowledge of this subject was not what it is today.

Direct approach to the lesion, that is surgical intervention, remains, therefore, the treatment of choice. There is no question of any other form of treatment in the case of a hematoma, but at the very least the hematoma should be evacuated. In our opinion surgical intervention is necessary even in the absence of a hematoma. However, serious consideration must be given to the usefulness of an operation if the lesion is so situated that any intervention would jeopardize vital brain functions. If a rollandic arteriovenous angioma is the cause of initial hemorrhage in childhood it will sooner or later almost certainly cause hemiplegia. It is better to risk creating hemiplegia ourselves at an age when motor re-education is less difficult, than to risk a later recurrence which might be fatal. In arterial aneurysms radical cure includes the dissection of the sac, ligation of the pedicle and ablation of the aneurysm. Such a thorough operation is not always possible. It is sometimes necessary to make do with a reduction or another intermediary technique. In arteriovenous angiomas intervention should include, besides evacuation of the hematoma, the complete ablation of the malformation after ligation of the pedicles. The advantages of angiography are obvious here. The operation should almost always be completed with the removal of the cerebral tissue which has softened after the onset of the symptoms. The operation will sometimes include partial lobectomy depending on the location of the malformation. An almost ideal treatment, it cures the lesion and prevents epileptic sequelae. Sometimes only the pedicles need be ligated. This technique is rarely used because the ligation must be total in order to be effective, and is as perilous as the direct approach. Moreover, it is quite useless when incomplete.

Among our nine cases of angioma, one was not operated on, one had ligation of the ipsilateral carotid, and seven were approached directly by surgical intervention. Only this procedure made possible the ablation

of the angioma in all cases and of the hematoma when one existed. Six of these patients recovered completely; the seventh, with a double malformation, died of cachexia. These results lead us to the conclusion that surgical intervention can and should be attempted. Ingraham and Matson are also of this opinion.

Conclusions

Meningeal hemorrhage in children, however rare it may be, is not without exception. We have observed personally fifteen such cases, of which two were related to a spinal cord tumor (spinal hemorrhage). We have not studied these cases at length but mention them because the symptoms are sometimes similar to meningeal hemorrhage. Among the thirteen remaining cases, nine were arteriovenous angiomas, three could not be explained, and one was related to a cerebromeningeal hemorrhage of uncertain origin. Unfortunately, one death occurred due to bilateral angiomas. Seven patients had neurosurgical treatment in which the lesion was directly approached and ablated. This method gave complete satisfaction in six cases. We did not encounter any arterial aneurysms in this series, but their existence in children has been noted by others. Cerebral vascular malformations are, therefore, the essential cause of meningeal hemorrhage in children. In contrast to the situation with adults, an arteriovenous angioma is the most common lesion among children. It explains the high frequency of meningocerebral hemorrhages.

All meningeal hemorrhages in children should be explored by angiography because of the frequency and the prognostic gravity of vascular malformations, especially when the E.E.G. reveals focal anomalies. When a malformation is present, surgical intervention is advisable in almost every case. In the absence of such a malformation, the prognosis seems to be good without surgical intervention.

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