CONTENTS

TREATMENT OF ANTERIOR COMMUNICATING ANEURYSMS

Professor J. Lawrence Pool, M.D.*

First of all I wish to extend the warmest thanks to President Ameli and the Middle East Neurosurgical Society for the honor of being invited to participate in such a splendid meeting. It has been highly rewarding from the scientific point of view and also because of the opportunity of meeting so many distinguished colleagues from Iran and other countries.

Because we have been discussing intracranial hemorrhage of various kinds it may be noted that intracranial aneurysms, our present topic, generally give rise to subarachnoid hemorrhage or rupture. Rupture may also lead to an intracerebral or an intraventricular hematoma and occasionally to a subdural hematoma. The most common cause of subarachnoid hemorrhage, according to a recent survey by Zimmerman of New York, is vascular hypertension, while rupture of an intracranial aneurysm is the next most common cause, with an incidence of approximately 33%. Cerebral angiomas (arteriovenous malformations) account for another 9%. From a diagnostic point of view, it is worth noting that about 25% of all patients with a ruptured aneurysm have vascular hypertension. Less frequent causes of subarachnoid hemorrhage are blood dyscrasias such as leukemia, certain metastatic or glial brain tumors, occasionally a pituitary adenoma, spinal varices and ependymomas of the spinal cord. In some cases the cause of hemorrhage is never known.

Subarachnoid hemorrhage from a ruptured aneurysm has a more serious prognosis than that following most other causes because an aneurysm is so likely to bleed again, usually with a fatal outcome. Rebleeding is especially frequent 7 to 14 days after the presenting hemorrhage.

Most intracrainal aneurysms are of the saccular berry type. They are commonly believed to be congenital in origin. Some are undoubtedly subject to enlargement and rupture because of arteriolsclerotic changes.

* Professor of Neurological Surgery, College of Physicians and Surgeons, Columbia University; Director, Service of Neurological Surgery, The Neurological Institute, The Presbyterian Hospital, New York, New York.

Approximately 85% arise from the circle of Willis or its major branches and the remainder from arteries of the vertebral-basilar system. Multiple aneurysms have an incidence of about 11%.

Aneurysms of the anterior communicating artery comprise nearly one-third of all intracranial aneurysms. Because of their frequency, dangerous location at the base of the brain, and the fact that they seldom give rise to localizing signs, they are especially difficult problems.

Their rupture leads to the classical syndrome of subarachnoid hemorrhage: sudden excruciating headache, often followed by lapse of consciousness and perhaps convulsive seizures. Stiffness of the neck and legs, and photophobia are then noted. If the hemorrhage is not too severe, recovery of consciousness is rapid and headache soon subsides. Some patients complain of flashing lights or other scotomata in each tempral field of vision at the time of rupture. Such symptoms (rare), or demonstrable field cuts (5%) of unilateral or bitemporal character are due to involvement of the optic chaiasm or optic nerves which lie immediately under these aneurysms. A, more common sign of the location of these lesions after rupture is weakness of one leg, which occurs, it is thought, because of the vasospasm that so commonly affects an anterior cerebral artery. Subjective as well as objective sensory disturbances may also be noted in one or both legs. Unlike carotid aneurysms that usually give rise to pain about the eye and 3rd nerve paresis, or middle cerebral aneurysms that lead to contralateral motor and sensory signs, the ruptured anterior communicating aneurysm characteristically gives rise to no symptoms or signs other than those of subarachnoid hemorrhage.

Diagnosis:

Any patient who has suffered signs of meningeal irritation that could be due to subarachnoid hemorrhage, especially if there are no focal neurological signs, should be suspected as possibly having an aneurysm of the anterior communicating artery. Such patients should not be regarded as suffering from meningities or encephalitis unless lumbar puncture confirms the diagnosis. Lumbar puncture is often overlooked in children, for example, who may have had only a minor bleeding episode that did not clinically suggest hemorrhage, because it is not widely recognized that aneurysms occur and may bleed even in the very young, ranging from ages of a few hours old up to 12 years and more. Adults, known to have vascular hyper-

tension should also be considered as possibly having a ruptured aneurysm unless there is clear evidence that hemorrhage is due to a vascular accident, based on high blood pressure. Finally, pregnant women with intracranial bleeding, papilledema and convulsions should not necessarily be considered as having toxemia of pregnancy unless the possibility of a ruptured aneurysm has been ruled out.

Arteriography should be done in all cases suspected of having a ruptured aneurysm, regardless of age, pregnancy, or blood pressure, provided their condition permits the test. Arteriography should be done without delay, I feel, and such patients should be transferred by plane or ambulance promptly to the nearest center equipped to do the test. Delays of even a day may cost the life of a patient who could be saved by prompt operation before their condition deteriorates. Delays of over a week are certainly not wise, I feel, because aneurysms so frequently bleed again, often fatally, 8 to 14 days ofter their first hemorrhage. Carotid arteriography should be bilateral even though an aneurysm is found on the first side tested and should preferably include vertebral studies also. This is because multiple aneurysms may be present. If more than one is discovered, the presence of vasospasm or a clot next to one will generally indicate which one has just bled. Complete angiography is also important in order to know whether or not the circle of Willis is normal or abnormal, as certain anomalies may alter the therapeutic approach.

Cerebral vasospasm deserves a brief comment since it is so often recognized close to an aneurysm that has recently bled. It lasts about 2 weeks, and may increase during the first week so that the condition of the patient is made worse as the cerebral circulation becomes impaired and results in cerebral edema which may prove fatal.

The cause of cerbral vasospasm is unknown. One must suspect several factors. Extravasation of blood into or along the walls of parent arteries may be one cause. An irritating effect of extravasated blood may be another cause, perhaps due to breakdown products of blood, such as hematin, or serotonin released from platelets. (We have found that old but not fresh blood and dilute serotonin applied to the cerebral arteries of animals may result in local vasospasm.) The cerebral vasospastic effects of serotonin have recently been confirmed by Kirgis et al of California.

Mechanical stresses secondary to rupture may serve as mechanical stimuli to parent vessels and thus initiate spasm. We, and others, have shown that simple mechanical stimuli applied to the cerebral arteries of animals and man cause vasospasm. It is known that aneurysms prior to,

and after rupture, as proved by arteriographic study, usually alter their contour. I have actually seen this happen at operation. It seems possible therefore that a sudden alteration in the shape of the sac may impose a sudden strain upon the parent artery that sets off spasm due to this type of mechanical stimulus. Finally, the fact that spasm may extend widely from the site of rupture, often without the spread of blood to affected remote arteries, suggests a neurogenic spread via the nerve network of the cerebral arteries. Therefore, it seems to me, we may consider three factors: chemical (from blood); mechanical (from new stresses); and neural (from intrinsic vasomotor reflex effects) as possible causes of cerebral vasospasm.

Treatment

There are many ways of treating any ruptured aneurysm, and this applies to those of the anterior communicating artery. Bed rest with or without hypothermia and induced hypotension sooner or later invites rebleeding and, therefore, should not be considered except for patients who are so old or in such poor condition that surgery would be foolish. Carotid ligation in the neck has proved effective for some anterior communicating aneurysms but others have been known to bleed fatally in spite of this treatment. The same applies to wrapping the sac with muscle or to intracranial ligation of the anterior cerebral artery that is the main source of blood to the aneurysm, in site of considerable success with these or similar methods in skilled hands such as those of Dott, Krayenbuhl, Poppen, Falconer and Gillingham. Pilojection (the injection of hairs by a special air gun) as advocated by Gallagher, has not yet proved very satisfactory. Coating the aneurysm with fine gauze or plastics is usually satisfactory, but if this can be done, it seems to me simpler and better to clip it, being careful to spare the main arterial trunks.

In general the treatment of intracranial aneurysms depends on the location of the aneurysm, its size, the condition of the patient, the experience of the surgeon and his preference for a given therapeutic approach. These principles apply to the treatment of aneurysms of the anterior communicating artery, for which intracranial surgery is preferable to carotid ligation in the neck. There are several accepted ways of treating these aneurysms intracranially: 1) the anterior cerebral artery that is the main supply of the aneurysm may be occluded proximal to the aneurysm with a clip or ligature in the hope that reduction of its blood supply will prevent the aneurysm from enlarging and rupturing again. This has been done by Dott, then Logue, Poppen and others. 2) The aneurysm may be permanently excluded from the circulation by clipping or ligating its neck. 3) The

aneurysm may be invested with muscle, gauze or a plastic material, Dott having been the pioneer in this type of procedure as in the others just mentioned.

The surgical approach differs widely also. Some neurosurgeons use a unilateral craniotomy flap, others a bifrontal flap. Some remove a wedge from one frontal lobe, others do not, and still others separate the two frontal lobes in approaching the lesion.

The prevention of premature rupture of the aneurysm at operation is also managed in various ways. Some surgeons deliberately lower the blood pressure for a brief period with hypotensive drugs, others use temporary slings or clips to shut off the circulation to the aneurysm in case of need, and still others, like the speaker, do this as a routine measure.

The approach I prefer is simply one way of operating on these difficult aneurysms, and is done almost always at the Neurological Institute of New York because of our feeling a) that it is better to occlude the aneurysm by clipping it rather than run the known risk of rebleeding that sometimes occurs after a proximal arterial occlusion or an investing procedure; and b) because of our occasional sad experiences in the past when an aneurysm ruptured at operation before one could expose it properly, because both anterior cerebral arteries were not temporarily shut off.
For these reasons the following technique has been evolved and used with considerable success.

Technique of clipping by bifrontal flap and use of temporary clips. The main points for success are the following:

1. Adequate exposure of the aneurysm
2. Prevention of rupture at operation
3. Slow, gentle retraction of the frontal lobes
4. Preservation of the circulation through both anterior cerebral arteries after clipping the aneurysm

Adequate exposure means a view of both sides of the aneurysm so that one can delineate without any doubt the arterial supply from each side, and control it before the sac itself is even seen. This requires a bilateral exposure in my opinion, best obtained by making a small (4-hole) bifrontal flap as anteriorly placed as possible. (Figure 1) A transverse scalp incision permits burr holes (3) along the supraorbital ridge, and one in the midline about 4 cm. posteriorly. Saw cuts pass directly through the frontal sinuses, which are later sealed over in water-tight fashion with a periosteal flap. No infections have occurred in over 200 cases for whom this flap was used for tumors as well as aneurysms. The dura is opened only transversely along the anterior extent of the flap.
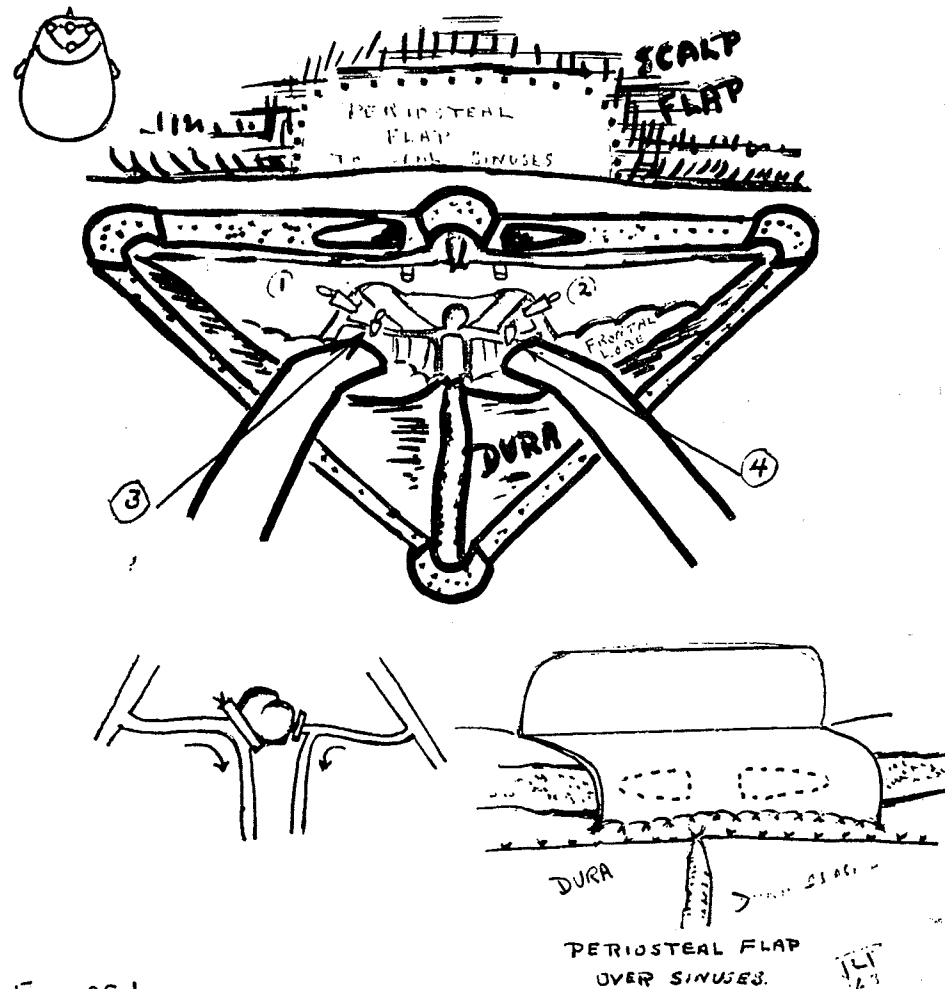
FIGURE 1

The superior longitudinal sinus is doubly lighted and divided. The dura is then covered with cottonoid to prevent it from crumpling and thus possibly causing thrombosis of the venous sinus. First one and then the other frontal pole is gently retracted until each olfactory tract is seen and divided. (Loss of the sense of smell does not bother these patients.)

The patient's head, moderately elevated up to this point, is not tilted down so that the frontal lobes fall away from the floor of the anterior fossa. By this time hypothermia of 28°C. (which is always used) and intravenous urea or spinal drainage, facilitate the exposure. The arachnoid around one and then the other carotid artery is opened to allow CSF to escape and improve the exposure, until no more CSF flows out. A temporary Mayfield-Kees clip is now gently placed across one, and then the other carotid artery. A small temporary clip is then placed across one, and then the other anterior cerebral artery, whereupon the carotid clips are removed (usually after only 3 to 4 minutes). Not until now are attempts made to expose the aneurysm. Its exposure is carried out as swiftly and boldly as possible, using a small suction tip to clear away clot and adherent brain from the sac and at the same time to expose each anterior cerebral artery fully, so that one can be absolutely certain that it is well seen and, there, will not be included in the clipping of the aneurysm. Perforating vessels should not be touched, if possible. The neck of the aneurysm is now clipped, or isolated by clipping the anterior communicating artery. If the aneurysm has no discrete neck its sac is crimped down by clips, so that its lumen is excluded from the circulation, but in such a manner as to leave an ample channel for each anterior cerebral artery. A small bit of muscle is then wrapped around the whole site for reenforcement. The two temporary clips are then removed and the wound is carefully closed. Watertight closure of the dura is followed by sealing (with periosteum) the frontal sinuses. The head is of course re-elevated during this stage. The dura is then tented up to the bone flap, and drains are placed. Obviously there must be no bleeding or oozing whatsoever from brain or dura on closure.

With this technique there is no need to worry if the aneurysm ruptures after its major circulation has been shut off for the necessary 10 to 20 minutes. Indeed, it is often helpful to rupture it at this stage deliberately (to facilitate placement of the permanent clips) and when this is done there is usually a retrograde flow of blood from the sac that already indicates some degree of collateral flow, presumably supplied by the middle cerebral artery. It is best to clip a second aneurysm, if one is present near the site, at the same time. If a second sac is not easily reached, it should be clipped by an appropriate craniotomy 3 to 6 weeks

leater, since a second aneurysm sooner or later is so apt to rupture fatally.

Timing of surgery: Surgery may usually be safely done of the patient is in good or reasonably good condition, as soon after hemorrhage as possible. We prefer to operate on the day of rupture if hemorrhage has not been severe, and the patient is alert and in good condition and not over 50 years old. This is because the condition often fails if surgery is delayed, presumably because of progressive cerebral vasospasm that leads to cerebral edema.

If, however, the condition is deteriorating or is poor, or the patient is over 50 years old, we prefer to wait until the condition improves or reaches a plateau or stabilizes. In some cases this may mean a delay of 1 to 3 weeks. As a rule we do not like to delay surgery over 7 days because 7 to 14 days after rupture there is apt to be rebleeding that is often fatal. For this reason most of our patients are operated upon within the first week after hemorrhage.

Patients in coma or stupor are not operated upon unless their condition becomes stabilized, or a clot is suspected. Operation on such poor risk cases is called salvage surgery and seldom leads to good results.

Results: For 25 **favorable risk** patients that I have personally operated upon (for the most part within 8 days after rupture) the operative mortality has been 5%. Ages have ranged from 12 to 61 years. The morbidity has been close to 20% but there have been no late deaths from recurrent hemorrhage. Thus 75% of patients with a ruptured anterior communicating aneurysm who have been in at least reasonably good condition for surgery, have survived early surgery (mostly 1 to 8 days after rupture) in good mental and physical condition, and are now active one to seven years after surgery, as biochemist, physician, banker, librarian, linguist, salesman, shop foreman, student, etc.

Summary

One method of treating ruptured anterior communicating aneurysms is presented. This permits early clipping through a bifrontal flap. Key branches of the circle of Willis are temporarily occluded by special removable clips before exposing and permanently clipping the aneurysm. This requires the use of hypothermia at 28°C. and other technical aids described. The risk of a fatal recurrent hemorrhage appears to be removed by this method with a reasonable operative mortality (5%) and morbidity (20%) that compare favorably with the mortality rate for un-operated patients of 34% or higher.

Résumé

Une nouvelle methode de traitement de l'anevrysme de l'artère communicate antérieure romppe est presentés. Ceci permet une clippage rapid de l'anevrysme à travers d'un valet bifrontal. Les branches principales de 1 Hexagone de Willis sont fermées par des clips temporaires avant d'exposer et clipper definitivement l'anevrysme.

Ceci nécessite une hypothermie à 28°C. et d'autre techniques decrites. Les risques de recidive d'hemoragie sont évités de cette maniére. avec une mortalitè (5%) et morbidité (20%) raisonnable comparé avec la mortalité des sujets non operés de 34% ou plus.