Anesthesia for the Surgery of Intracranial Aneurysms: Part I

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Abstract

INTRODUCTION

The anesthetic and surgical handling of cerebral aneurysms has changed significantly during the last few years. The use of a surgical microscope, the advances in neuroradiology, the temporary clipping of aneurysms, the aggressive treatment of vasospasm, and cerebral protection with hypothermia have permitted a change towards clipping of aneurysms during the first three days after the subarachnoid hemorrhage. Subsequently, rebleeding could be reduced, allowing treatment of vasospasm. All these changes resulted in an improvement of recovery.

ANATOMIC REMEMBRANCE

In the one month old human embryo, the encephalic messenchyme is perfused by 4 arteries: 2 dorsal (vertebrals a.) and 2 ventrals (carotids a.). These two arterial systems are continuous and anastomozed.

Later on, the vertebral arteries merge to originate the basilar artery. This union of the vertebral arteries is sometimes not uniform, but eventually, it is completed. The posterior communicating artery remains as a sign of the union of both systems.

As the development progresses, an anastomosis is formed between the two anterior cerebral arteries creating a communicating anterior artery, thus completing the cerebral arterial or circle of Willis (Fig 1).

Figure 1

Figure 1

- A.C.-Anterior Communicating Artery.
- A.C.A.-Anterior Cerebral Artery.
- A.I.C.-Anterior Inferior Cerebellar Artery.

- B.- Basilar Artery.
- C.A.-Choroidal Artery.
- I.A.A.-Internal Auditory Artery.
- I.C.A.-Internal Carotid Artery.
- M.C.A.-Middle Cerebral Artery.
- P.C.-Posterior Communicating Artery.
- P.C.A.-Posterior Cerebral Artery.
- P.I.C.-Posterior Inferior Cerebellar artery.
- S.C.A.-Superior Cerebellar Artery.
- V.A.-Vertebral Artery.

The anterior cerebral artery and the middle cerebral artery receive blood mainly from the carotid artery, while the posterior cerebral artery receives it from the vertebral system.

All the encephalon except the occipital lobe and a portion of the temporal lobe are areas perfused by the carotid artery. The diencephalon is perfused by both arterial systems. The rombencephalon with the cerebellum is in the territory of the vertebral system.([1])

ANEURYSMS

An aneurysm is a disease of the vessel caused by abnormal dilatation and located at the site of weakness in the elastic layer of the artery. Cerebral aneurysms represent saccular dilatations that appear more frequently in the bifurcations of intracranial cerebral vessels. Although the etiology is originally congenital they can develop subsequently due to degenerating changes in the walls of the vessels associated with hypertension.([2])

Some hereditary conditions and vascular injuries are associated with intracranial aneurysms, such as in the of Ehlers-Danlos syndrome, coarctation of aorta, polycystic kidney disease, arteriovenous malformations, fibromuscular dysplasia, and sickle cell disease.

Aneurysms can also be caused although rare, by trauma, infection, arteriosclerosis, and damage to the arterial wall.([3,4])

Predisposing factors for the rupture of an aneurysms are: use of tobacco, abuse of alcohol, pregnancy, strenuous activity andhypertension([5,6]).

Classification of aneurysms

Cerebral aneurysms can by classified according to size in:

- Small. If less than 12 mm in diameter (78 %)
- Large. From 12 to 24 mm in diameter (20 %)
- Giant. If more than 24 mm in diameter (2 %)

90 % of then belong to the anterior circulation. Of these:

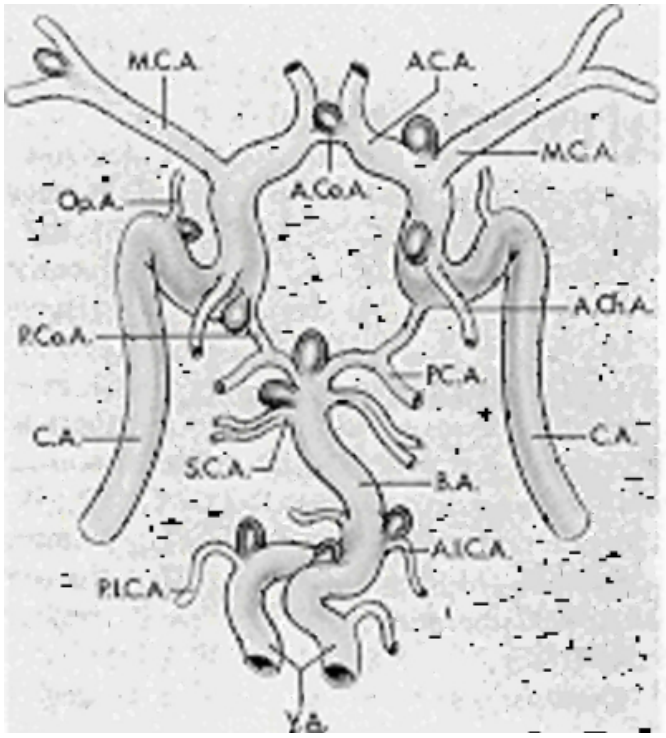
- 39 % are at the junction of the anterior communicating artery and the anterior cerebral artery.
- 30 % along the carotid artery.
- 22 % along the middle cerebral artery.
- 8 % in the posterior circulation.

Aneurysms can also be classified in:

1. Mycotic: They are caused by a septic degeneration of the elastic and muscular layer of the cerebral arteries. These aneurysms are not frequent.
2. Arteriosclerotic fusiformed: They are produced by serious arteriosclerosis and arterial hypertension. The vessels of the circle of Willis are mainly affected. They are more frequent in the territory of the basilar and middle cerebral artery.
3. Congenital saccular: They are arterial dilatations of less than 2.5 mm that appear in the bifurcation of the arteries of the circle of Willis.
4. Giants: There have a multifactorial origin and a diameter in excess of 2.5 mm and are more frequent in the vertebral-basilar system.

LOCALIZATION ANEURYSMS

Figure 2



The majority of the aneurysms are close to the circle of Willis. Approximately 90 % are localized in the following places:

1. Internal carotid artery (C.A.) at the level of the posterior communicating artery (P.Co.A.).
2. Junction of the arterial cerebral artery (A.C.A.) and anterior communicating artery (A.Co.A.).
3. Near the bifurcation of the middle cerebral artery (M.C.A.).
4. Junction of the posterior cerebral artery (P.C.A.) and the basilar artery (B.A.).
5. The bifurcation of the carotid artery into anterior cerebral artery and middle cerebral artery.

Other places for aneurysms along the carotid artery are at the origin of the ophthalmic artery (Op.A.) and the anterior choroidal artery (A.Ch.A.). Other locations along the vertebral artery (V.A.) and basilar artery include the origin of the postinferior cerebellar artery (P.I.C.A.), postanterior cerebellar artery (A.I.C.A.), superior cerebellar artery (S.C.A.), and junction of the basilar artery with vertebral arteries.($_{7}$)

FORMATION MECHANISM OF ANEURYSMS

Formation mechanisms of saccular aneurysms are subject of debate. Investigation of origin of saccular aneurysms is difficult because their size is less than one mm. in diameter . Experimental models of gigantic aneurysms have been developed and conclusions have been made based on studies by Ferguson, Coll and Wright that could be valid and applicable to saccular aneurysms as well. Important for the formation of aneurysms is the influence of the change from laminar to turbulent flow in the bifurcation of arteries. Other contributing factors are the speed of the flow along the wall of the artery, the irregular distribution of flow inside the aneurysm and the distribution of flow in the curves of the arteries.($_{8}$)

SUBARACHNOIDAL HEMORRHAGE

Subarachnoidal hemorrhage is defined as presence of blood in the subarachnoid space. The rupture of an aneurysm is the most common cause of subarachnoidal hemorrhage (SAH). The rupture of the aneurysm occurs normally in the fundus of the aneurysm. Other causes of SAH are:

- Trauma.
- Dissection of vertebral and carotid arteries.
- Arteriovenous dura mater and spinal malformations.
- Rupture of a mycotic aneurysm.
- Sickle cell disease.
- Coagulopathy.
- Cocaine abuse.
- Pituitary apoplexia.

EPIDEMIOLOGY

It is estimated that about 5 million people in the U.S. population suffer an intracranial aneurysm (ICA).($_{9}$)

The rupture of an aneurysm is the most common cause of SAH (80 - 90 %), with an annual frequency of 10 to 11 cases per 100.000 people and year (9,$_{10}$). In the U.S., 25.000 cases take place each year and in 10 % of the cases astroke occurs (9). Arteriovenous malformations appear in 4 to 5 % of thecases ($_{11}$).

Of the 28.000 annual ruptured cases in the U.S. : 12.000

survive without sequels, 8.000 die or become invalid due to the initial hemorrhage. Out of 20.000 patients that could be treated, 40 % remained handicapped, mainly due to rebleeding or arterialvasospasm([12])

Arterial hypertension is among the potential factors and risks for the rupture of an ICA. In addition, it is one of the causes for formation of aneurysm.

PATHOPHYSIOLOGY OF THE RUPTURE OF ANEURYSM

CEREBRAL HEMODYNAMICS

The rupture of intracranial aneurysm produces an increase of the intracranial volume due to the accumulation of blood in the subarachnoidal space. Consequently, intracranial pressure (ICP) increases. The increase of ICP produces a secondary reduction of cerebral perfusionpressure (CPP) ([13]). The increase of ICP is the factor that reduces or stops bleeding within the intracranial space. The clinical consequence is a reduction in consciousness due to global cerebral ischemia ([14]).

Cerebral hemodynamics can be altered in two different ways ([15]):

1. The ICP rises toward the diastolic arterial pressure causing a reduction of the CBF. This is followed by a reduction of the ICP, resulting in an increase of the CBF with subsequent hyperemic reaction which improves the cerebral function.

2. Persistent increase of ICP with lack of recovery of CBF and subsequent loss of consciousness may be due to alterations in the dynamics of cerebrospinal fluid (CSF) caused by thrombi in the basal cisterns. The lack of recovery of CBF has been associated with cerebral swelling and vasospasm.

The reduction of CPP can produce ischemia in the insufficiently perfused zones, alteration of autoregulation and increase in ICP due to rupture of the blood-brain barrier (BHE)([16]).

ELECTROCARDIOGRAPHIC ALTERATIONS

Electrocardiographical alterations appear in 50 to 58 % of the patients who suffer an SAH([17]).

The electrocardiographic changes can affect the P wave, the U wave, Q-T interval and demonstrate dysrythmias such as ventricular tachycardia and fibrillation which can be life-threatening during the first 48 hours of the SAH.

The most frequent changes that appear affect the the S-T segment and the T wave, simulating electrocardiographic ischemic changes. The dysrythmia can appear as premature ventricular complexes in 80 % of the patients with an SAH.

The etiology is controversial and is related with the hyperactivity state of the autonomous nervous system that is present in an SAH. The electrocardiographic changes appear more frequently in the first 48 hours of the SAH.

The differentiation of electrocardiographic alterations caused by SAH or produced by a coronary disease can by difficult. It is necessary to obtain several electrocardiograms, determine cardiac enzymes and judge echocardiographic images in order to differentiate the etiology of the electrocardiographic alterations.

The question can be raised if the intervention should be postponed in patients with SAH and concomitant coronary disease. This problem might be solved with the cooperation of the neurosurgeon considering the neurosurgical situation and the prognosis , the characteristics of the operation, the risk of rebleeding and vasospasm, the possible intraoperative changes of hemodynamics, and the evaluation of the possibility of cardiac ischemia with subsequent arrhythmia's and congestive cardiac failure.

Surgery can be performed quickly if necessary in a patient with no history of coronary disease presenting with a neurologic grade of 2 or less and showings an abnormal electrocardiogram, a normal CPK-MB and a negative echocardiogram. The increase in CPK and specifically in CPK-MB appears in 50 % of the patients with SAH ([18]). The total CPK and the index of CPK-MB is rarely constant in myocardial transmural infarct. It appears in up to 27-33 % of the patients with SAH ([19], [20]).

If patients with a history of coronary disease are admitted with a low neurological degree and an increase of CPK-MB it is preferable to postpone surgery and to perform further cardiac studies. If urgent surgery is necessary, invasive hemodynamic monitoring (Swan-Ganz catheter) and anesthetic techniques similar to those used in cardiovascular anesthesia are required.

HYDROCEPHALIA

Hydrocephalia appears in 10 to 20 % of the patients who survive the initial hemorrhage. It is caused by an obstruction of the circulation of cerebrospinal fluid due to blood clots in

the cisterns of the base and the subarachnoidal space as well as the presence of blood in places of absorption of CSF (arachnoidal villi). It normally presents with a reduction of level of consciousness and is diagnosed with computer tomography (TC).

If there is no spontaneous improvement, a ventricular drainage is inserted. Some patients require a permanent ventriculo-peritoneal drainage . Ventricular drainage should not be excessive in order to avoid extreme reductions in ICP with consequent rebleeding due to rupture of theaneurysm ([21]). The swelling process of the arachnoid villi and fibrosis of the leptomeninges may necessitate a derivation of the CSF ([22,23]).

SEIZURES

Seizures appear in over 13 % of the patients after an SAH and in 40 % of the patients with neurological deficit ([24]). Seizures are very likely caused by rebleeding. Vasospasms rarely produce seizures ([25]). Seizures increase the ICP, the blood pressure, theO2 consumption and the production of lactate, therefore increasing the risk of rupture of the aneurysm.

Treatment of the acute phase include benzodiazepines or barbiturates and later phenytoine. It is recommended to use prophylactic seizure treatment with anticonvulsives during the posthemorrhagic period.

ARTERIAL HYPERTENSION

The hypertension which often appears in SAH can be due to hyperactivity of the vegetative nervous system caused by cerebral ischemia or by direct injury of the autonomic control system. The transmural pressure of the aneurysm is in relation to the difference between the mean arterial pressure (MAP) and the ICP. An increase in MAP raises transmural pressure and can result in rupture of the aneurysm. The decline in MAP provokes a reduction in transmural pressure and thus reduces the risk of rupture. It also reduces the CPP in patients with vasospasms, leading to ischemic zones in badly perfused areas with alteration of autoregulation and an increase of the ICP due to disruption of the blood-brain barrier. Some surgeons prefer to maintain pressures between 120 and 150 mm Hg. Both, the sudden increase of MAP and the reduction of ICP can rupture the aneurysm and produce rebleeding.

HYPONATRIEMIA

Hyponatriemia frequently appears in patients who suffer an SAH. It is seen in 10 to 34 % of the patients usually in the days following the hemorrhage and coinciding with the vasospasm. It usually takes place between the 3rd to the 15th day of the SAH ([26]). Hyponatriemia has been attributed to an inadequate secretion of antidiuretic hormone. It can be due to “salt wasting” syndrome in which a high concentration of natriuretic factor is produced. Large amounts of sodium are lost in theurine (>40 mmol/L)([27,28]), provoking hypovolemia and hyponatremia. Hyponatriemia lowers of the level of consciousness, leads to muscular weakness, seizures and coma. Dehydration along with hypotension increases the risk of vasospasm ([29,30]).

Treatment of hyponatremia using serum with isotonic sodium chloride in order to achieve an adequate intravascular volume should be performed cautiously.

OTHER COMPLICATION

- Neurogenic Pulmonary Edema.

It appears in 1 to 2 % of the patients with SAH. It can be caused by an alteration in the pulmonary capillary due to the presence of blood in the subarachnoid space resulting in hyperactivity of the sympathetic nervous system.

The following complications usually appear in patients hospitalized in the Intensive Care Unit.

- Pneumonia.

It appears in 7 to 12 % of the patients with SAH.

- Gastrointestinal Hemorrhage.

It appears in 2 to 4 % the patients perioperatively. The incidence is reduced by prophylactic treatment with blockers H2.

- Deep Venous Thrombosis.

It appears in 1 to 5 % and pulmonar embolism in 0.8 - 2.2 % ([31]).

An intermittent pneumatic calf compression should be used as prophylaxis, reducing the probability of thromboembolic disease.

Low doses of subcutaneous heparin for prophylaxis are rarely used before or immediately after surgery. They can be used in patients who have to remain in bed during extensive

periods of time after surgery.

- Hypermetabolism.

Patients have an exacerbated metabolism after an SAH([32,33]). The nutritional requirements specific for these patients after an SAH have not been established.

In patients having a reduction of the level of consciousness it is preferable to use enteral alimentation for nutritional support. The risk of aspiration of gastric contents is decreased with continuous bolus of feedings, by raising the head, by paying attention to gastric residuals, and by keeping the pH high.

References

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