Anesthesia For The Surgery Of Intracranial Aneurysms: Part II

J Crespo

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Abstract

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PREOPERATIVE MANAGEMENT

Patients who suffer subarachnoidal hemorrhage (SAH) have been classically classified in two groups: Classification by Botterell et al. (1956)(table 1) ([34]) and the modification made be Hunt and Hess (table 2) ([35]). The Hunt and Hess scale was also modified including the 0 grade, which is the one related to patients with aneurysm and no SAH. Recently, the World Federation of Neurological Surgeons has introduced a scale based on the Glasgow Coma Scale (table 3) ([36]).

Figure 1

Table 1. Clinical Grades in the Botterell et al. Scale.

Grade	Criteria
I	Consciousness with or without meningeal signs
II	Drowsiness without significant neurologic deficit
III	Drowsiness with neurologic deficit and probable cerebral clot
IV	Major neurologic deficits presents
V	Moribund with failing vital centers and extensor rigidity

Figure 2

Table 2. Clinical Grades according to the Hunt and Hess Scale

Grade	Criteria	Index of Perioperative Mortality (%)
0	Aneurysm is not ruptured	0-5
I	Asymptomatic or with minimal headache and slight nuchal rigidity	0-5
II	Moderate to severe headache, nuchal rigidity, but no neurologic deficit other than cranial nerve palsy	2-10
III	Somnolence, confusion, medium focal deficits	10-15
IV	Stupor, hemiparesis medium or severe, possible early decerebrate rigidity, vegetative disturbances	60-70
V	Deep coma, decerebrate rigidity, moribund appearance	70-100

Figure 3

Table 3. Grades of the WFNS Scale

WFNS Grades	CGS Score	Motor Deficit
I	15	Absent
II	14-13	Absent
III	14-13	Present
IV	12-7	Present or absent
V	6-3	Present or absent

CLINICAL CHARACTERISTICS

NEUROLOGIC

Blood in the subarachnoid space produces a swelling in the meninges which produces a clinical effect similar to meningitis. These symptoms appear frequently within 4 hours of the SAH:

- Headaches. Appear in 85 - 95 % of the patients (38).
- Level of consciousness. They can suffer brief losses of consciousness alternating with periods of reduction of the consciousness.

- Convulsions. Approximately 20 % of the patients suffer convulsions.
- Nausea and vomiting.
- Altered mentality.
- Photophobia.
- Motor deficit.
- Sensitive deficit.
- Visual field deficit.
- Loss of reflexes.

Grades I and II are characterized by an increase of headache; grades III and IV, by deterioration of consciousness and focal deficit; grade V by a deep coma. Patients with the worst prognosis are those who arrive at the hospital with poor grades.

Paralysis of the ocular nerve can appear in aneurysms of the posterior communication artery. Paralysis of the external motor ocular nerve (abducens) can be seen in cases with increase of intracranial pressure ICP resulting in traction of the nerve due to cerebral herniation. Compression of the trigeminal nerve produces irritation with pain in its distribution area. It appears more frequently in giant aneurysms of the cavernous sinus.

CARDIOVASCULAR

The electrographic alterations that can appear in SAH have been previously described. (Part I)

An injury of the posterior hypothalamus during SAH can result in release of norepinephrine from the medulla and the sympathetic cord (39). Subendocardial ischemia can result from the increase of myocardial afterload.

OTHER SYSTEMATIC MANIFESTATIONS

* Reduction of total blood volume and blood cells can occur (40,41). This reduction can help to prevent the occurance of vasospasms. Some surgeons maintain an adequate blood volume in order to maintain cerebral perfusion in patients who suffer vasospasm. Patients with a high grade in the Hunt-Hess classification with vasospasm and a great quantity of blood in the subarachnoid space need to have their central venous pressure CVP or pulmonary artery pressure controlled in order to monitorize intravascular volume.

- Pulmonary neurogenic edema.
- Electrolytic alterations.
- Gastric erosion.
- Pulmonary aspiration.
- Deep vein thrombosis.

DIAGNOSTIC STUDIES

AXIAL COMPUTERIZED TOMOGRAPHY (ACT) AND MAGNETIC RESONANCE IMAGING (MRI)

Cerebral ACT without contrast is the diagnostic procedure of choice in diagnosing SAH. ACT can detect the presence of blood in 95 % of the patients within 24 hours of the a SAH. Positive testing is reduced as time goes by: within 48 hours it can be detected in 90 % within 5 days in 80 % and within a week in 50 % (42).

The ACT can give important information such as the localization of the SAH, its magnitude, clues as to the possible localization of the aneurysm, and evaluation of the size of ventricles.

- In the cases of rupture of aneurysms of the anterior communicant artery, blood is distributed inside the ventricles.
- Rupture of aneurysms of the anterior cerebral artery in its distal portion and the middle cerebral artery usually result in intracerebral bleeding.
- There is suspicion of rupture of aneurysm when blood appears in the fourth ventricle.

Sections of 1 to 5 mm can be explored with the high resolution ACT. Contrast injection may facilitate the location of the aneurysm. Patients with blood in the base cisterns have a probability of developing vasospasm .

MRI is not used frequently in the acute phase of the SAH. MRI can detect the presence of blood under the pia mater near the place of rupture of the aneurysm. MRI combined with angiography shows the aneurysm if it is big enough (43).

LUMBAR PUNCTURE

Lumbar punction can confirm the diagnosis of the SAH when the ACT has been negative. Xanthochromic cerebrospinal fluid CSF appears within 4 hours of the SAH

and becomes negative after 3 weeks (44). Lumbar punction has the risk of producing cerebral herniation and rebleeding of the aneurysm due to reduction of ICP (45).

ANGIOGRAPHY

Angiography of the 4 cerebral vessels identifies the source of the bleeding and can detect the presence of other cerebral aneurysms. Multiple cerebral aneurysms have an incidence of 5 to 33.5 % (46). Angiography can be repeated in patients with xanthochromic CSF and with ACT plus negative front angiography. The angiography image of vasospasm appears as a narrowing of the cerebral vessels in the area of the rupture of an aneurysm, in remote zones of the brain or appear diffusely. A diffuse pattern is correlated with the worst prognosis (47).

TRANSCRANIAL DOPPLER (TCD)

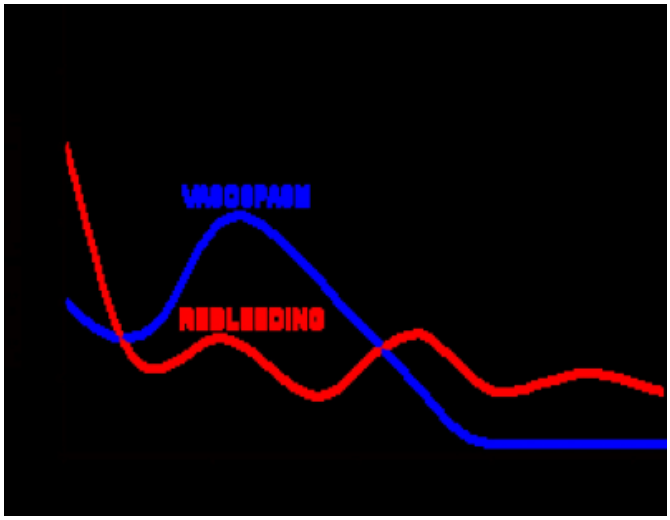
TCD is a non invasive method used with variable success to identify and quantify vasospasms. The profiles of speed detected by Doppler sonography increases with the reduction in diameter of the affected vessels. Great speeds of 200 cm/s have been associated with great risk of stroke after SAH and patients having speeds of lees than 100 cm/s have little probability of having clinic vasospasms ([48]).

REBLEEDING

Rebleeding is a serious and devastating complication after rupture of an aneurysm. Following vasospasms, it is the second worst complication regarding morbidity. Mortality is even worse than the one caused by vasospasms. The risk of rebleeding varies with the time elapsed after an SAH (Fig.1) It has been found that the peak of the risk of rebleeding appears at the end of the first week ([49]). The International Cooperative Aneurysm Study found a peak of risk of rebleeding of 4.1 % within the first day of the SAH and of 1.5 % in the following days, the accumulative risk being 19 % in the first 14 days ([50]) and 50 % in the 6 months following the SAH ([51,52]). After 6 months rebleeding decreases at 3 % per year (52).

Figure 4

Figure 1. The risk of rebleeding and vasospasm varies with the time elapsed after an SAH



The high incidence of rebleeding after conservative handling of SAH leads to a trend towards surgical treatment. Only clipping the aneurysm offers a successful treatment to prevent rebleeding. Medical treatment to prevent rebleeding in patients awaiting clipping of the aneurysm has been discussed in several publications ([53,54]). In order to avoid rebleeding it is necessary to try to reduce the transmural pressure of the aneurysm. Hypotension, dehydration and sedation of patients increases the risk of vasospasm. The present tendency is to maintain arterial pressure and volume in a normal range. Hypertension should be treated with antihypertensive drugs of short action such as Esmolol or Nitroprusside.

Antifibrinolytic agents, tranexamic acid and aminocaproic acid significantly decrease the incidence of rebleeding compared to placebo, but they don't reduce mortality ([55]). The incidence of rebleeding can be reduced by using antifibrinolytic therapy. However, this at the expense of increased ischemic neurological deficit and a proportional increase of mortality ([56]). Therapy with antifibrinolytics is associated with an increase in incidence of hydrocephalia. Risk for pulmonary embolization and deep venous thrombosis shows no differences compared to placebo (55,56).

VASOSPASM

Vasospasm is the most important cause of mortality and morbidity after SAH. Vasospasm can be seen angiographically in 60 % of the patients, but only half of these suffer a clinic syndrome. Approximately 35 % of the

patients with SAH have a secondary neurological deterioration due to vasospasms.

The prevention and treatment of vasospasms is one of the most important endpoints in treating patients with SAH. Neurological deficit appearing in SAH is not always associated with the evidence of vasospasms in arteriography. The relationship between the clinical syndrome and angiographic vasospasm is disputed. The neurologic deficit is due to ischemic cerebral stroke. The pathophysiology of vasospasm is not totally understood. The period of maximum spasm occurs between the 4th and the 11th day after the SAH and reversible symptoms do not persist for more than 2 weeks.

The clinical syndrome occurs in a matter of hours, with an inclination to sleep, confusion and stupor. Normally the symptoms appear between the 5th to the 7th day of an SAH and rarely occur after two weeks (22,[57]). The risk of a vasospasm is correlated with the location and amount of blood in the subarachnoidal space shown by the ACT ([58], [59]).

PATHOPHYSIOLOGY

The cause of vasospasm is unknown and has probably multiple factors. In the development of vasospasm, the relationship of hemoglobin and products of its degradation are important. Other substances such as histamine, serotonin,catecholamines, prostaglandins, angiotensin, and free radicals have also been considered to take part in the development of vasospasms. There is also evidence of changes in the tissues of the vessels walls ([60]).

Fall of CBF below the critical point produces ischemia with neurological deficit in the territories which are perfused by vessels affected by the vasospasm ([61]) . Autoregulation is affected by vasospasms. There is also a reduction of reactivity to CO2 in patients with SAH. The magnitude of the reduction is related to ischemic deficits ([62]).

Blood vessels affected by vasospasms have structural alterations. The following can be found in the vessel walls: red cells, leukocytes, macrophages, mediators such as interleucin-1, eicosanoids and immuno complexes, which show the presence of a swelling process ([63]).

The quantity and location of the blood in the subarachnoidal space as seen by the ACT can predict the incidence and severity of the vasospasms. The aggressive elimination of blood from the subarachnoidal space reduces the incidence and severity of the vasospasms ([64],[65]). Treatment with antifibrinolytics may prevent lysis of the clot and rebleeding. However, it can increase the risk of vasospasm ([66],[67]).

DIAGNOSIS

Vasospasm may be followed by a reduction of the level of conscience, disorientation or transitory neurologic focal deficit ([68]). It depends on the location of the vasospasm and the collateral circulation. Vasospasms of the anterior cerebral artery can produce akinesia, mutism and dysplegia. If the middle cerebral artery of the dominating hemisphere is affected it can produce hemiparesis and aphasia. If the structures of the posterior fossa are affected, hemodynamic and respiratory alterations can result ([69]).

The clinic picture of vasospasm is completed with an increase of headache, meningism, fever and tachycardia.

It is not common for vasospasms to appear in the first three days following the SAH. Most often they appear between the 7th and 10th day of the SAH and resolve within 10 to 14 days. They rarely occur after the 12th day.

Neurologic deterioration after rupture of an intracranial aneurysm can be caused by rebleeding, edema, hydrocephalia, convulsions, hyponatremia, drug intoxication and other medical complications added to vasospasm. Rupture of an intracranial aneurysm can be diagnosed clinically, with ACT, electroencephalographic studies, and Transcranial Doppler (TCD). These studies are useful to evaluate the severity of vasospasms. Angiography is used to confirm the diagnosis of vasospasms but its popularity is being reduced by using non invasive methods such as TCD. TCD can identify and quantify vasospasms. The capability of TCD to predict vasospasm is questioned. It measures the speed of blood flow in the cerebral vessels (more commonly in the middle cerebral artery). During vasospasms an increase in the speed of the blood flow of more than 120 cm·sec-1 can be measured. The presence of high speed is suggestive of vasospasm but finding normal speeds dues not exclude the diagnosis of vasospasm.

The use of angiography increases due to its utilization in angioplasty. The image of vasospasm can be located in a specific area of the brain or appear as a diffuse image. The diffuse form is associated with the worst prognosis.

TREATMENT

The treatment of vasospasm has three endpoints:

1. Prevent or revert the reduction of caliber of the vessels.

2. Prevent or revert ischemia.
3. Protect the brain from the effects of ischemia (infarction).

In order to increase the CBF in cerebral areas affected by vasospasm and thus avoid the damage caused by ischemia, a combination of hypervolemia and hypertension is used.

HYPERVOLEMIA/HYPERTENSION/HEMODILUTION

The idea is to increase the cardiac output and arterial pressure by increasing intravascular volume. The increase in intravascular volume produces hemodilution, with an Hkt between 30 and 50%. If the increase of intravascular volume does not increase the cardiac output , it can be necessary to add vasoactive drugs. Control of volume can be obtained by measuring CVP, but it is more accurate by measuring pulmonary artery pressures. It is necessary to maintain pulmonary artery pressures of 15 - 16 mm of Hg and central venous pressures of 8 to 9 mm of Hg.

Increased mean arterial pressures will increase the cerebral blood flow CBF in the vessels affected by vasospasm and in the ischemic cerebral areas. The ischemic zones of the brain have an CBF which is pressure-depending after loss of autoregulation. The application of the 3 H's (hemodilution, hypervolemia and hypertension) is showing promising results. According to some studies, the combination of hypertension and hemodilution has been effective in 60 to 70 % of the patients in reversing neurological deficits of vasospasms before the vasospasm can produce cerebral infarction (60,[70],[71],[72]). The success of this therapy is more effective if combined with surgery. (70,60).

Mannitol is used for hemodilution and expansion of plasma , thus improving the rheologic conditions of the blood.

Different levels of arterial pressure have been recommended to treat vasospasm: Levels of 10 to 20 mmHg above normal systolic pressure, systolic pressures above 240 mm Hg and average pressures of 150 mmHg , systolic pressures of 160-200 mmHg for clipped aneurysm and 120- 150 mmHg for unclipped ones, systolic pressures of 150-175 mmHg for clipped aneurysms and 130-150 for unclipped ones (71,[73],[74],[75]). In general, arterial pressure should be limited to less than 160 mmHg in patients whose aneurysms have not been clipped. The level of cardiac index should be maintained at 3 to 3.5 l/min/m2.

The principal goal of this therapy is to reverse neurologic deficits. Once this purpose has been achieved it is not clear if further increase of therapy can be beneficial ([76]). Prophylactic use of hypervolemia /hypertension to prevent the development of symptomatic vasospasm is questionable (73,[77]).

This treatment is dangerous in elderly patients or in those with preexisting cardiac disease. It bears also the risk of cerebral edema and rebleeding.

The most common complication of treatment with hypertension/hypervolemia is pulmonary edema which appears in 26 % of the patients (77). Patients with coronary disease can develop myocardial ischemia due to the increase of preload and afterload with subsequent increase in myocardial O2 demand (76).

Other complications are: dilutional hyponatremia, myocardial infarct and rebleeding in patients whose aneurysms have not been clipped.

Further complications that can appear are those related to invasive hemodynamic monitoring or preoperative/postoperative rebleeding of a second unclipped aneurysm.

A variety of drugs have been used without verified good results in trying to revert vasospasms after SAH: adrenergic drugs (sympathetic and parasympathetic), phosphodiesterase-inhibitors, serotonin-antagonists, nitrates, prostaglandin's and inhibitors, adenosine, oxygen free radical scavengers and local anesthetics ([78]). The prophylactic administration of the calcium channel blocker dihidroperidine demonstrated promosing results in SAH I(the method of action does not appear to be by cerebral vasodilatation) (76).

Nimodipine is a powerful cerebral vasodilator and has proved in several studies to produce a significant improvement three months after SAH ([79],[80]). Nimodipine decreases neurological deficits and mortality, and reduces the index of cerebral infarction by vasospasm after SAH in patients with rupture of an aneurysm (79,80,[81]). The beneficial effects of nimodipine can be compared with the neuroprotective effects of calcium channel blockers([82]). Nicardipine reduces the clinical and angiographical incidence of vasospasm ([84]). There is interaction between calcium antagonists (nicardipine and nimodipine) and anesthetic drugs. High intravenous doses of nimodipine have a depressing effect on arterial pressure when volatile

anesthetics are present([83]).

Hypotension produced by calcium channel blockers may be counterproductive if the patient is treated with the hypertension/hypervolemia regimen.

Early surgical cleaning of the blood accumulated in the cerebral cisterns can prevent vasospasm ([85],[86]).

Anti-inflammatory agents may help to prevent the development of vasospasm. High doses of glucocorticoids have been used, in some uncontrolled studies resulting in improved outcome in high risk patients ([87]). 21-aminoesteroid U-7400 6F is a new experimental drug with great expectations in reversing vasospasms in models of SAH ([88],[89]).

Vasospasm not responsive to medical treatment can be treated with transluminal angioplasty in order to expand the spastic vessels (121,122).

TIMING OF SURGERY

The timing of intervention in patients with ruptured aneurysms has been subject of debate for years.

The advantages of processing with the intervention early include:

1. Prevention of rebleeding.
2. Evacuation of blood from the subarachnoid space thus reducing the risk of vasospasm.
3. Subsequent use of aggressive treatment with hypertension and hypervolemia to fight the formation of a vasospasm.

The advantages of postponing the intervention include:

1. Reduction or disappearance of the cerebral edema, increased cerebral retraction and facilitation of surgery due to better surgical exposure.
2. Stabilization of the clot at the aneurysm, thus reduced risk of intraoperative rupture and facilitation of surgical dissection.

Results of the International Cooperative Study on the Timing of Aneurysm Surgery (46) show that postoperative results in patients in which the intervention was postponed (from 11 to 14 days after SAH) are superior. Favorable results are obtained in patients with a good grade if surgery takes place within three days after SAH or after the 11th day; however, mortality in this group reduces only if surgery is delayed. The International Cooperative Study on the Timing of Aneurysm Surgery could not detect any difference of mortality between patients who had experienced an early intervention (from 0 to 3 days) and those who had it later (from 11 to 14 days). The results were worse if surgery took place between day 7 and 10 after SAH, which is the period of highest risk for vasospasm. The reduction of mortality caused by rebleeding obtained trough early surgery, is counteracted by the mortality associated with ischemia by vasospasms. In contrast with the results of the Parent International Study, a separate analysis of a subset of patients treated in North American centers reveal that results are better if surgery takes place between the 0 and the 3rd day after SAH ([90]).

Patients whithou interventions demonstrated the same mortality (20 %) and good results (60 %) both with early planned surgery (from day 0 to the 3rd) and delayed surgery (from the 11th to the 14th). A greater mortality and morbidity was found in patients who were surgically treated between the 7th and the 10th day after SAH (Table 4. Modified by Kassell NF, Torner JC, Jane JA, et al. The international cooperative study on the timing of aneurysm surgery, II-surgical results. J Neurosurg 1990;73:37,28,[39,40,41,42,43,44,45,46,47]).

Figure 5

Table 4. Results 6 months after SAH (46).

	Surgical treatment (%)		Non-surgical treatment (%)	
Days after SAH	Good recovery	Mortality	Good Recovery	Mortality
0 a 3	66	17	63	20
4 a 6	66	19	60	24
7 a 10	64	18	56	28
11 a 14	77	7	62	21
+ de 15	72	8	63	20

An early operation does not appear to reduce the risk of vasospasm and cerebral infarct (46,[91]).

Early interventions revealed a higher incidense of technical difficulties for the neurosurgeon. The occurancel of intraoperative ruptures of aneurysms is not related to the timing of surgery (46).

An early intervention after SAH should only be performed in patients with a good grade.

The presence of ischemia is the most frequent cause for

mortality and morbidity in these patients, requiring a therapeutic plan designed to prevent or revert vasospasm and thus protect the brain from ischemia.

Interventional neuroradiology is an alternative way of handling aneurysms. Endovascular treatment of the aneurysm sack is realized by occlusion with a balloon or by insertion of platinum rolls. This technique is normally used if surgical clipping is not possible because their aneurysm is located in an inaccessible location. This technique, now increasingly used, widens the field of action of the neuroanesthesiologist (Anesthesia for Interventionist Neuroradiology). At the same time, this technique offers a valuable alternative for patients with serious associated pathology such as unstable myocardial ischemia.

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Author Information

Jose Luis Martinez-Chacón Crespo, M.D.
M□co Adjunto, Servicio de Anestesiología y Reanimación, Hospital Universitario de la Princesa