

# **Endovascular Management of Subarachnoid Hemorrhage Induced Vasospasm**

*Essay*

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Master degree of science in Radiodiagnosis

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## Introduction

*A*neurysmal subarachnoid hemorrhage (aSAH) is a devastating disease and leads to the development of poor outcome and high mortality. The worldwide incidence is about 10.5 cases per 100,000 person-years. The incidence increases with age, with a mean age at presentation of 55 years. The risk for women is 1.6 times that of men, and the risk for blacks is 2.1 times that of whites (*Suarez et al., 2006*).

Neurologic complications are common and include hydrocephalus (20 percent), rebleeding (7 percent) and symptomatic vasospasm (46 percent of patients) (*Solenski et al., 1995*).

Cerebral vasospasm is a major cause of morbidity and mortality in patients who survive aneurysmal subarachnoid hemorrhage (SAH), (*Jabbour et al., 2009*). Angiographic vasospasm occurs in 30–70% of patients following aneurysm rupture, most commonly between days 3 and 12, and results in permanent morbidity or mortality in up to 20% of these patients (*Komotar et al., 2008*).

It can present with clinical manifestations of cerebral ischemia which include: decreased consciousness or focal neurological deficit (new symptoms or worsening of previously detected manifestations), Disorders of the anterior cerebral artery (ACA) (apathy, akinetic mutism and

occasional paraparesis) and internal carotid artery (ICA) or middle cerebral artery (MCA) (hemiparesis or hemiplegia, which may be associated with sensory deficit, hemianopsia and aphasia) (*Bederson et al., 2009*).

Today, patients are treated with oral nimodipine and a combination of induced hypertension, hemodilution, and volume expansion (Triple-H therapy) to minimize the effects of cerebral vasospasm. Nimodipine, a dihydropyridine calcium channel blocker, which blocks L-type, slow conducting, voltage dependent calcium channels, has been shown to reduce cerebral infarction, when compared to untreated patients, while triple-H therapy focuses on maintaining high cerebral perfusion pressures to increase cerebral blood flow during vasospasm (*Appelboom et al., 2012*).

Symptomatic vasospasm refractory to hemodynamic augmentation is an indication for endovascular treatment. Endovascular solutions including intra-arterial administration of vasodilators such as nimodipine and transluminal balloon angioplasty (TBA), have proved to be effective as a more aggressive approach for such patients, these endovascular techniques have their own associated risks and benefits, and controversy exists over the best method. At what point to intervene with endovascular treatment has also been controversial (*Brisman et al., 2008*).

Percutaneous transluminal angioplasty and intra-arterial verapamil are generally safe, with a low complication rate but further studies are required to determine appropriate patient selection and treatment efficacy (*Jun et al., 2010*).

Intra-arterial nimodipine (IAN) in patients with severe vasospasm has been reported by several authors. Data from a larger series by Biondi et al., suggested that IAN is effective and safe for treatment of vasospasm after subarachnoid hemorrhage. However no information regarding the influence of IAN on cerebral perfusion is available. Moreover there is lack of information on about the duration of effect (*Hanggi et al., 2008*).

We still have an incomplete understanding of the pathophysiology of vasospasm, and it remains challenging in its treatment and is responsible for significant morbidity and mortality in patients with ruptured cerebral aneurysms.

## **Aim of the Work**

**T**o assess the safety and efficacy of endovascular treatment of cerebral vasospasm with different modalities in preventing delayed neurologic deficits in patients with symptomatic vasospasm.

## Chapter (1): Anatomy of Cerebral Arteries

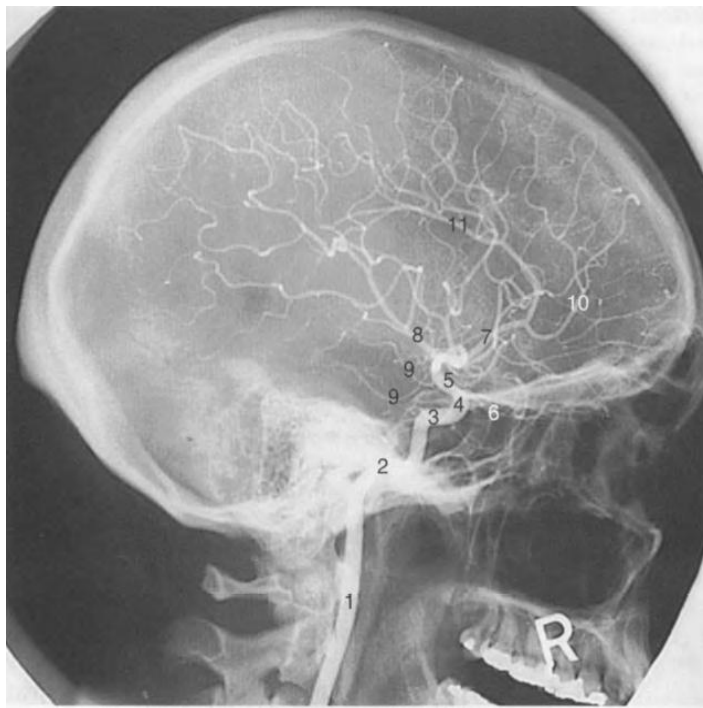
The arterial supply of the CNS is derived from the internal carotid and vertebral arteries. Anastomoses exist between the internal and external carotid arteries but little or no supply to the normal brain is derived from the latter. All arteries entering the surface of the brain are end arteries, that is, they have no precapillary anastomoses with other arteries and obstruction of them causes infarct of the supplied territory (*Rayn et al., 2004*).

### I. The internal Carotid Artery and Branches:

Nomenclature varies, but the four-part division of the internal carotid, designated as **C1–C4** and described in the radiology and surgical literature, is useful (*Johnson et al., 2008*).

The cervical segment (**C1**) begins proximally at the origin of the ICA with the common carotid artery (CCA) and extends cephalic to the external orifice of the carotid canal. The petrous segment (**C2**) traverses the carotid canal and enters the cavernous sinus (Dura), where the cavernous segment (**C3**) begins. The cavernous segment ends where the ICA pierces the dural roof of the cavernous sinus. The supraclinoid segment (**C4**) begins where the ICA exits the dural ring and enters the subarachnoid space, and it ends at the internal carotid bifurcation into anterior and middle cerebral artery branches. Together, the **C3** and **C4** segments form the characteristic “S” shape seen on lateral and oblique angiographic views of the skull base (**fig. 1**) (*Johnson et al., 2008*).

**C1** does not normally provide any branches. **C2** gives rise to three potential branches: the caroticotympanic branch supplying the middle and inner ear; the vidian artery, or the artery of the pterygoid canal, which goes through the foramen lacerum; and the artery of the foramen rotundum (*Johnson et al., 2008*).



**Figure (1):** Internal carotid angiogram, lateral view:

1. Internal carotid artery in the neck
2. Internal carotid artery in the petrous bone
3. Internal carotid artery in the cavernous sinus
4. Carotid siphon
5. Intracranial part of the internal carotid artery
6. Ophthalmic artery
7. Anterior cerebral artery
8. Middle cerebral artery
9. Anterior choroidal branches
10. Callosomarginal artery
11. Pericallosal artery.

*(Quoted from Ryan et al., 2004).*

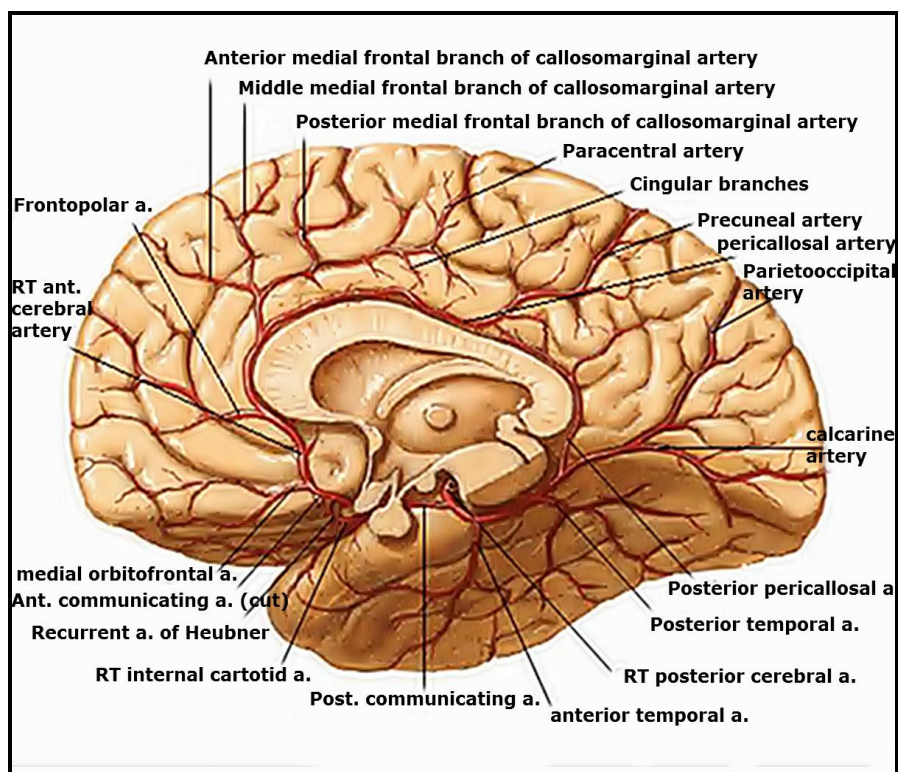
The principal branches of the **cavernous segment** are: The posterior trunk (meningohypophyseal artery) arises posteriorly from the superior aspect of the first bend of the cavernous segment and gives off branches to the pituitary gland and tentorium cerebelli (marginal tentorial artery). The inferolateral trunk arises more anteriorly and laterally from the horizontal portion of the cavernous segment. It supplies the third, fourth and fifth cranial nerves and has important anastomoses with the external carotid system: with the maxillary artery through the foramen rotundum and ovale and with the middle meningeal artery through the foramen spinosum (*Saunders et al., 2008*).

The principal branches of the **supraclinoid segment** are: The ophthalmic artery is usually given off just after the carotid artery leaves the cavernous sinus, but its origin is variable and it can sometimes arise from the middle meningeal artery (*Saunders et al., 2008*).

The posterior communicating artery passes on each side from the internal carotid to the posterior cerebral arteries. The anterior choroidal artery arises from the internal carotid artery just above the posterior communicating artery. The circle of Willis is situated in the suprasellar cistern and links the internal carotid arteries with each other and with the vertebrobasilar system. By facilitating “cross-flow”, it affords some protection in the event of major arteries occlusion (*Butler et al., 2007*).



**The anterior cerebral artery (ACA)** passes above the optic nerve. In the longitudinal fissure, it is connected by a short trunk (the anterior communicating artery) to the opposite one. This landmark subdivides the artery into segments A1 (or horizontal or precommunicating segment), and A2 (or vertical or postcommunicating segment). Distal and cortical branches are also termed A3. The artery runs in the longitudinal fissure around the corpus callosum until its posterior part, where they end by anastomosing with the posterior cerebral arteries (*Fig 2*) (*Gallucci et al., 2007*).



**Figure (2):** Medial view of the brain showing Right ACA & PCA and their branches (*Quoted from Netter et al., 2002*).

## Branches of anterior cerebral artery

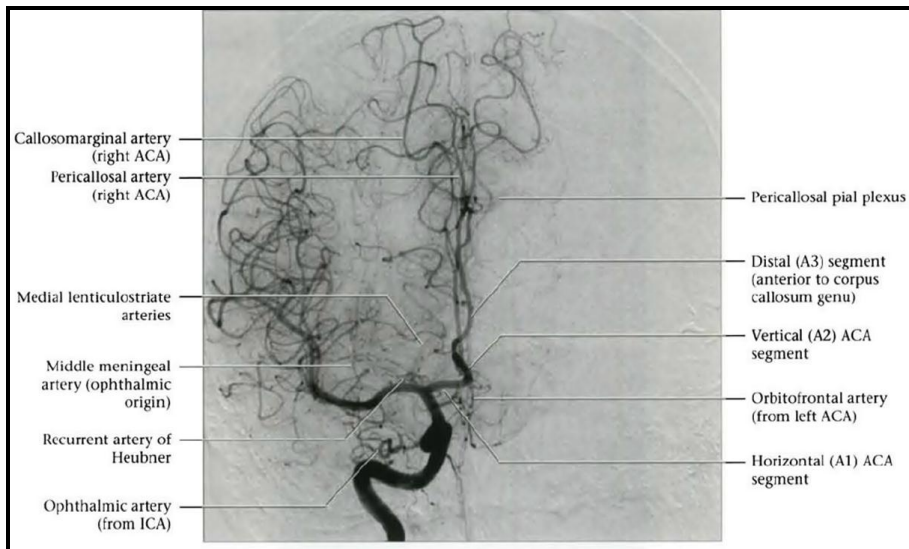
### ➤ Cortical branches

- 1- Orbitofrontal artery, Arises from proximal A2 and Ramifies over inferior surface of frontal lobe.
- 2- Frontopolar artery: Arises from mid-A2 and Extends anteriorly to frontal pole.
- 3- Pericallosal artery: Arises from A2 near corpus callosum genu, it is larger of two major distal ACA branches and Courses posterosuperiorly above corpus callosum, below cingulate gyrus.
- 4- Callosomarginal artery: Smaller of two distal ACA branches that Courses posterosuperiorly in cingulate sulcus, above cingulate gyrus.

### ➤ Perforating branches

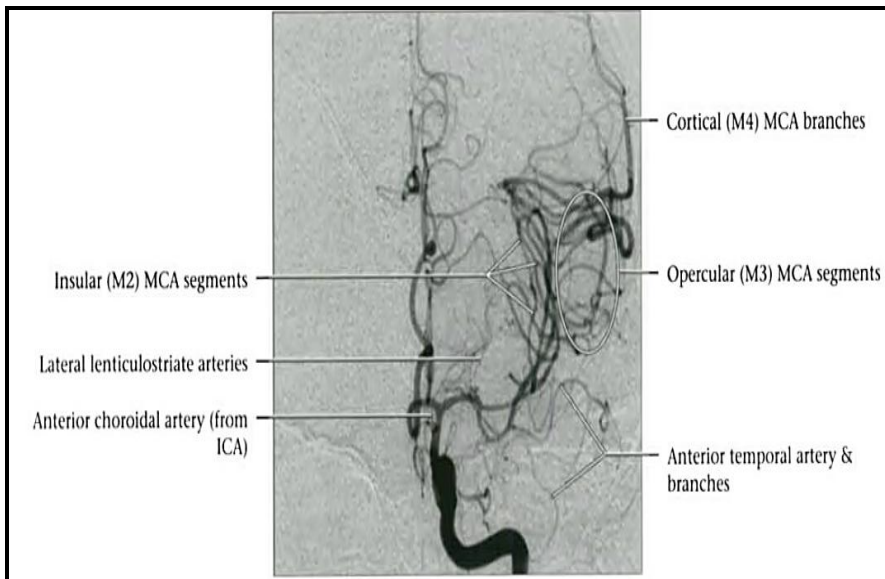
- 1- Medial lenticulostriate arteries: course superiorly through anterior perforated substance.
- 2- Recurrent artery of Heubner: Arises from distal AI or proximal A2 and Curves back laterally above AI to enter anterior perforated substance (**Fig. 3**).

*(Harnsberger et al., 2006).*



**Figure (3):** AP view of right internal carotid DS showing anterior cerebral artery and its branches (*Quoted from Harnsberger et al., 2006*).

**The middle cerebral artery (MCA)** is the largest and most direct of the branches of the internal carotid artery and is therefore the most prone to embolism. It passes laterally and ends as branches on the insula, the overlying opercula and most of the lateral surface of the cerebral hemisphere (**Fig. 4**) (*Rayn et al., 2004*).



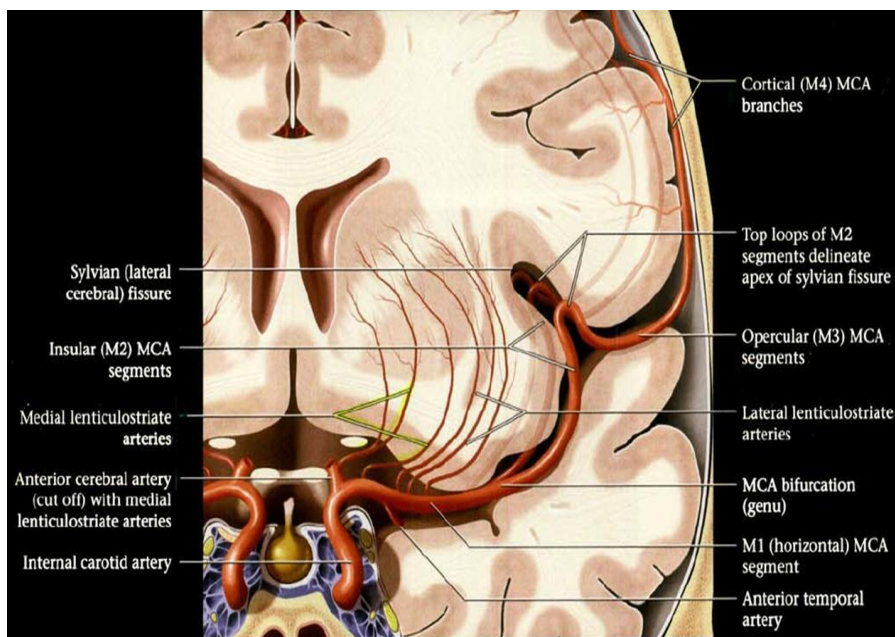
**Figure (4):** AP view of left internal carotid DSA showing middle cerebral artery (*Quoted from Harnsberger et al., 2006*).

The middle cerebral artery can be divided into four segments: M1 or horizontal segment, which runs horizontally below the anterior perforated substance, bifurcates or trifurcates in its lateral third and ends at the limen insulae, by turning postero-superiorly; M2 or insular segments are 6 to 8 in number, lie on the cortical surface of the insula and terminate at the top of the circular gyrus; M3 or opercular segments follow and terminate at the surface of the lateral cerebral fissure; M4 or cortical segments are the distal branches extended over the hemispheric surface (*Gallucci et al., 2007*).

The branches of the middle cerebral artery (**Fig 5**) are: **Medial and lateral striate (or lenticulostriate) arteries** - these arise at the anterior perforated substance and are seen

on AP angiograms arising from the upper surface of the middle cerebral artery trunk. They have a tortuous course superiorly, running at first medially for a short distance, then laterally and finally medially again. On lateral angiograms they are usually hidden by overlying branches of the middle cerebral vessels (*Rayn et al., 2004*).

One of these branches tends to be larger than the others and is called the artery of cerebral haemorrhage, as it is the artery of the brain most frequently ruptured. The striate arteries supply the basal ganglia and the anterior part of the internal capsule (*Rayn et al., 2004*).



**Figure (5):** Medial view of the brain showing Right ACA & PCA and their branches (*Quoted from Harnsberger et al., 2006*).

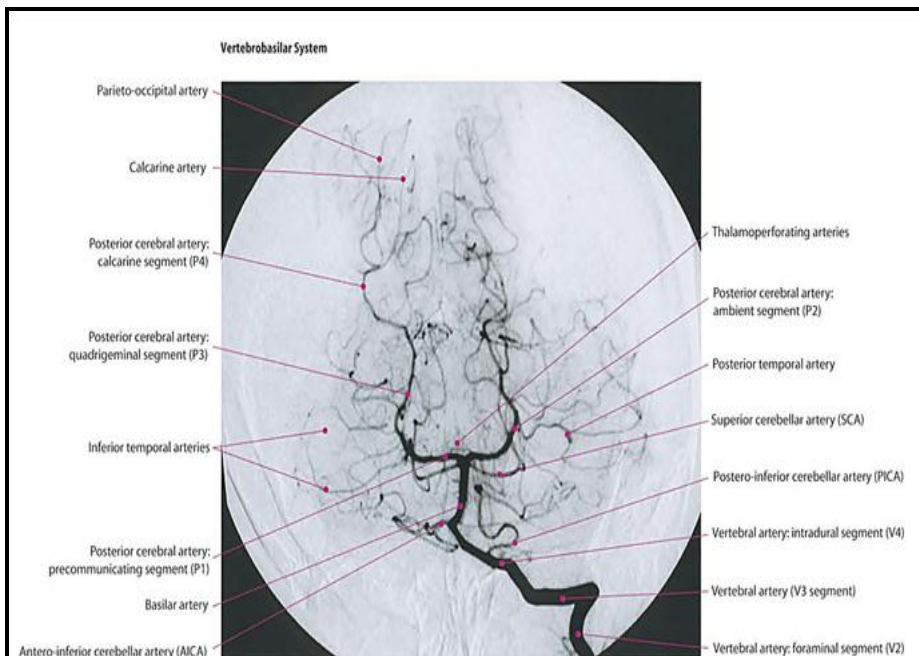
**Cortical branches** - these arise in the insula and pass superiorly and inferiorly to supply its surface before turning at a sharp angle to supply the inner surface of the overlying opercula and then exiting from the lateral fissure. They then turn again to emerge as branches that supply most of the lateral surface of the hemisphere as follows: Frontal branches, Parietal branches, Angular branches; and Superior temporal branches (*Rayn et al., 2004*).

## **II. Vertebral Artery and Branches:**

The vertebral artery can be divided into four segments: VI or extraosseus (from the subclavian artery to C6), V2 or foraminal (from C6 to axis), V3 or extraspinal (from C1 to foramen magnum), V4 or intradural (from foramen magnum to basilar junction). The artery gives rise to cervical branches destined to spine and muscles; meningeal branches (anterior and posteriormeningeal branches of the V.A.), and intracranial arteries: anterior and posterior spinal arteries; postero-inferior cerebellar artery (**Fig. 6**) (*Gallucci et al., 2007*).

The anterior spinal artery arises from both distal vertebral arteries from two branches that fuse on the midline. It supplies the anterior surface of the medulla. The posterior spinal artery originates from the distal VA or from the PICA (*Gallucci et al., 2007*).

The PICA has four segments and originates from the distal part of the VA. The first or anterior segment goes lateralward toward the cerebellar olive; the second or lateral continues posteriorly in the cerebello-medullary cistern, forming a caudal loop; the third or posterior medullary segment turns upward behind the posterior medullary velum. The fourth or supratonsillar segment gives the second, cranial loop and courses above the cerebellar tonsil. By the fusion on the midline of both vertebral arteries, the basilar artery is formed (*Gallucci et al., 2007*).



**Figure (6):** Vertebrobasilar system angiogram: AP view (*Quoted from Gallucci et al., 2007*).