

Anesthetic Management Of Intracranial Aneurysm surgery

An Essay

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Anesthesia

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List of Abbreviations

CO ₂	Carbon dioxide
COX	Cyclooxygenase
CPP	Cerebral perfusion pressure
CSF	Cerebrospinal fluid
CT	Computed tomography
ECG	Electrocardiographic
EDHF	Endothelium-derived hyperpolarizing
ICP	Intracranial pressure
MAP	Mean arterial blood pressure
NO	Nitric oxide
NOS	NO synthase
PCO ₂	Partial pressure of carbon dioxide
PGE ₂	Prostaglandin E ₂
PGD ₂	Prostaglandin D ₂
PGF ₂ α	Prostaglandin f ₂ α
PGI ₂	Prostaglandin I ₂
PKC	Protein kinase C
SAH	Subarachnoid hemorrhage
SSS	Superior sagittal sinus
TCD	Transcranial Doppler ultrasonography
TMP	Transmural pressure
Triple-H	Hypervolemic, Hypertensive, and Hemodilution
TXA ₂	Thromboxane A ₂

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Introduction

The prevalence of cerebral aneurysms is estimated at 1% to 5%. Ruptured aneurysms occur in middle age, between the ages of 40 and 60 years with a slight female predominance. The overall mortality rate is 25%, and more than 50% of survivors suffer significant neurologic morbidity (*Kim, 2011*).

The etiology of cerebral aneurysms is structural abnormality caused by hemodynamic factors and genetic predisposition. Cerebral aneurysms are divided into three groups: saccular, fusiform, and dissecting. Saccular aneurysms are the most common type. Fusiform aneurysms are often associated with atherosclerosis. A dissecting aneurysm develops at a tear between the endothelial and medial layers (*Kim, 2011*).

The leading causes of death and disability are, in descending order, vasospasm, the direct effects of the initial bleed, permanent ischemic effects of increased intracranial pressure, rebleeding, and surgical complications (*Kassell et al., 1999*).

Because of the associated systemic effects and the surgical requirements, patients with cerebral aneurysms

Introduction

present a unique challenge to the anesthesiologist. Successful anesthetic management of patients with cerebral aneurysms requires a thorough understanding of the natural history, pathophysiology, and surgical requirements of the procedures (*Molyneux et al., 2005*).

Two modalities are now used for treatment of intracranial aneurysm: endovascular coiling or surgical Clipping. Multiple factors must be evaluated, including aneurysm factors such as location and anatomy and patient factors such as age, comorbidities, and patient wishes (*Campiet al., 2010*).

There is a significant overlap in anesthetic management between clipping and coiling. The depth of anesthesia required for endovascular procedures is much less than that for open surgery (*Kim, 2011*).

Aim of Essay

This review is to formulate anesthetic plan for management of intracranial aneurysm surgery through comprehensive understanding of intracranial aneurysm pathophysiology, one's knowledge of anesthetic drugs and clinical experience.

Anatomy of Cerebral Circulation

The brain is one of the most highly perfused organs in the body. It is therefore not surprising that the arterial blood supply to the human brain consists of two pairs of large arteries (Figure 1), the internal carotid and the vertebral arteries (*Barrett et al., 2009*).



FIGURE 1

The internal carotid and vertebral arteries:
right side.

The internal carotid arteries principally supply the cerebrum, whereas the two vertebral arteries join distally to form the basilar artery. Branches of the vertebral and basilar arteries supply blood for the cerebellum and brain stem. Proximally, the basilar artery joins the two internal carotid arteries and other communicating arteries to form a

complete anastomotic ring at the base of the brain known as the circle of Willis. The circle of Willis (Figure 2) gives rise to three pairs of main arteries, the anterior, middle, and posterior cerebral arteries, which divide into progressively smaller arteries and arterioles that run along the surface until they penetrate the brain tissue to supply blood to the corresponding regions of the cerebral cortex (*Colman et al., 2006*).

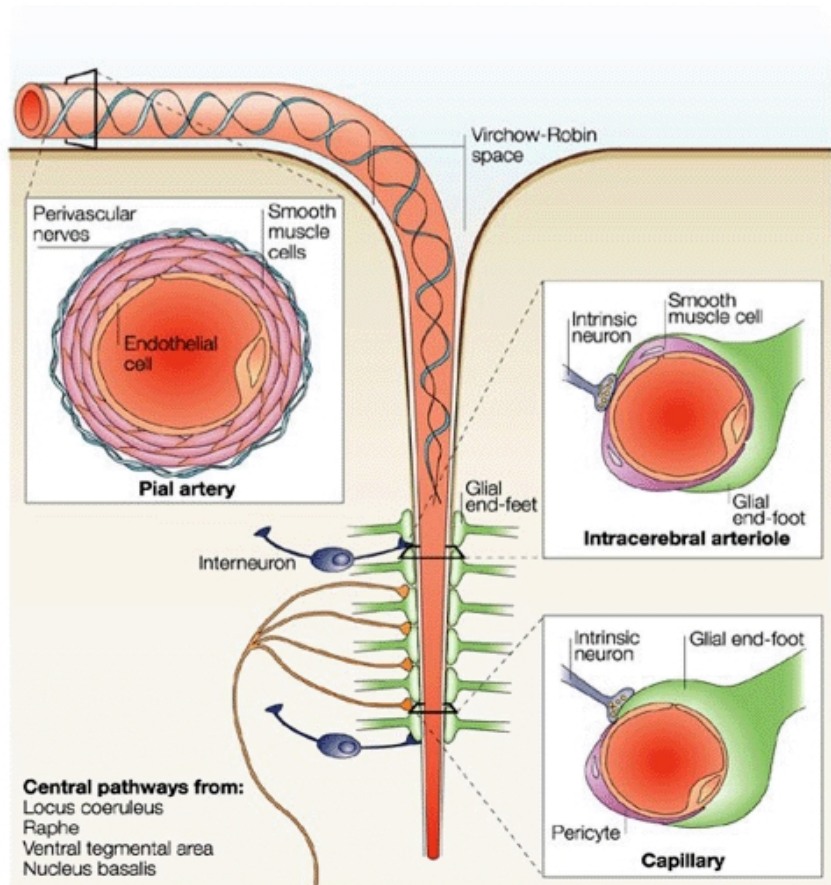


FIGURE 2

The arteries of the base of the brain. The temporal pole of the cerebrum and a portion of the cerebellar hemisphere have been removed on the right side.

The pial vessels are intracranial vessels on the surface of the brain within the pia–arachnoid. Pial vessels are surrounded by cerebrospinal fluid (CSF) and give rise to smaller arteries that eventually penetrate into the brain tissue (Figure 3). Penetrating arterioles lie within the

Virchow–Robin space and are structurally between pial and parenchymal arterioles. The Virchow–Robin space is a continuation of the subarachnoid space (*Jones, 1970*).



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FIGURE 3

Pial arteries on the brain surface have perivascular nerves that give rise to penetrating arteries within the Virchow–Robin space. As penetrating arterioles become parenchymal arterioles within the brain neuropil, they become associated with neurons and astrocytes. Parenchymal arterioles supply the cerebral microcirculation, known as the neurovascular unit.

There are several differences between pial arteries and smaller parenchymal arterioles. First, pial arteries receive innervation from the peripheral nervous system (extrinsic innervation), whereas parenchymal arterioles are innervated from within the brain neuropil (intrinsic innervation). Lastly, pial vessel architecture forms collateral network such that occlusion of one vessel does not appreciably decrease cerebral blood flow. However, parenchymal arterioles are long and largely unbranched such that occlusion of an individual arteriole results in significant reductions in flow and damage (infarction) to the surrounding local tissue (*Nishimura et al., 2007*).

The cerebral venous system is a freely communicating system comprised of dural sinuses and cerebral veins. Two groups of valveless veins drain cerebral hemispheres: the superficial cortical veins and the central veins (Figure 4). The superficial cortical veins (located in the pia matter) drain the cerebral cortex and subcortical white matter. The central veins consist of subependymal veins, internal cerebral veins, basal vein, and the great vein of Galen (Figure 5) (*Kiliç et al., 2008*).

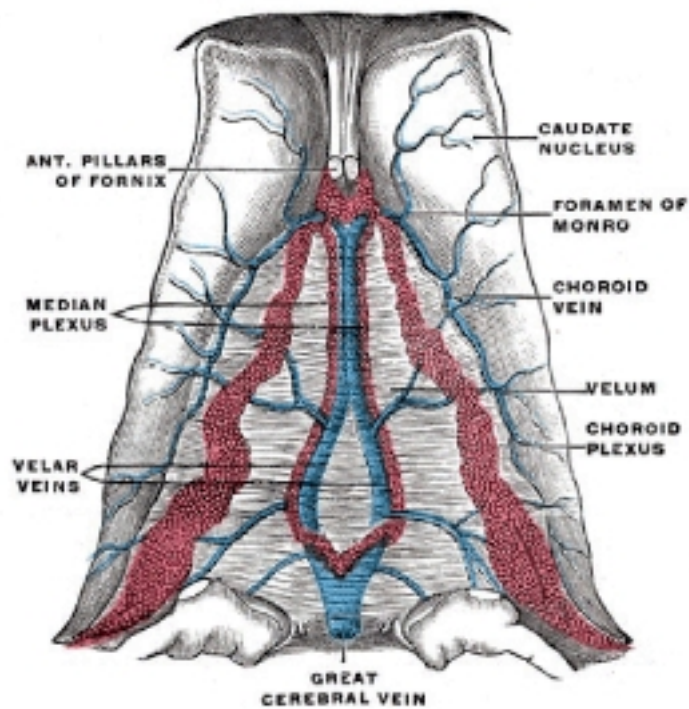


FIGURE 4

Superficial cortical veins and dural sinuses.

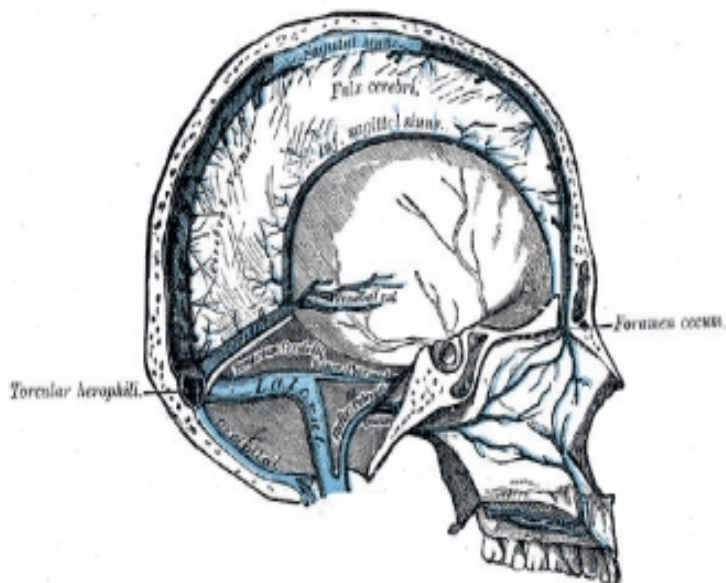


FIGURE 5

Central veins.

The central veins drain the brain's interior and anastomose with the cortical veins, emptying into the superior sagittal sinus (SSS). The SSS and deep veins is drained by the sigmoid sinuses and jugular veins. The cerebellum is drained primarily by two sets of veins, the inferior cerebellar veins and the occipital sinuses. The brain stem is drained by the veins terminating in the inferior and transverse petrosal sinuses (*Kiliç et al., 2008*).

The collateral circulation in the brain consists of vascular networks that allow for maintenance of cerebral blood flow when principal inflow conduits fail due to occlusion or constriction. The circle of Willis allows for redistribution of blood flow when extracranial or large intracranial vessels are occluded (Figure 6). This provides low-resistance connections that allow reversal of blood flow to provide primary collateral support to the anterior and posterior circulations (*Liebeskind, 2003*).