

# Anesthesia and intracranial arteriovenous malformation

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Anesthetic management of intracranial arteriovenous malformation (AVM) poses multiple challenges to the anesthesiologist in view of its complex and poorly understood pathophysiology and multiple modalities for its treatment involving different sub-specialties. The diagnosis of AVM is based on clinical presentation as well as radiological investigation. Pregnant patients with intracranial AVM and neonates with vein of Galen malformation may also pose a special challenge and require close attention. Despite technological advancement, reported morbidity or mortality after AVM treatment remains high and largely depends on age of the patient, recruitment of perforating vessels, its size, location in the brain, history of previous bleed and post-treatment hyperemic complication. Anesthetic management includes a thorough preoperative visit with meticulous planning based on different modalities of treatment including anesthesia for radiological investigation. Proper attention should be directed while transporting the patient for the procedure. Protection of the airway, adequate monitoring, and maintaining neurological and cardiovascular stability, and the patient's immobility during the radiological procedures, appreciation and management of various complications that can occur during and after the procedure and meticulous ICU management is essential.

**Key Words:** Intracranial arteriovenous malformation, anesthesia, intraoperative monitoring

## Introduction

Arteriovenous malformation (AVM), a congenital vascular malformation, deprives the surrounding tissues of blood supply and nutrients; produces venous hypertension and localized edema; and by overloading the heart can cause congestive cardiac failure (CCF).<sup>1</sup> Anesthetic management of intracranial AVM poses a great challenge in view of its diverse nature involving different modalities of treatment. Recently, stereotactic radiosurgery (SR) has been introduced as a sole

or adjunctive treatment modality for AVM, however, there are very few reports describing the anesthetic management of the patients.

## Surgical consideration

AVMs are errors in the development of the vasculature that, together with the effects of blood flow, may lead to a focal arteriovenous shunt.<sup>2</sup> It consists of abnormal direct communications between small arteries and veins without intervening capillaries producing low resistance and high-flow shunt.

AVMs most commonly present between 20-45 yrs of age with a peak in the 4<sup>th</sup> decade.<sup>2</sup> In spite of their congenital origin only 18-20% of cerebral AVMs are diagnosed during infancy and childhood.<sup>3</sup> The presenting signs and symptoms of cerebral AVMs are intracranial bleed (30-86% in adults,<sup>4</sup> and 75-80% in pediatrics<sup>3</sup>), seizure (8-46%),<sup>4</sup> focal neurological deficits (4-23%),<sup>4</sup> hydrocephalus or rarely CCF.<sup>4</sup> Cerebral damage may result from the presence of an AVM due to (i) "Steal" phenomenon, (ii) Ischemia from failure of perfusion from CCF, (iii) Hemorrhagic infarction from thrombosis of the aneurysm of the great vein of Galen, (iv) Cerebral atrophy and (v) Alterations of flow caused by surgery.<sup>5</sup> Intracranial AVMs are located in supratentorial areas (70-97%), infratentorial (3-30%) or in deeper brain structures (5-18%).<sup>4</sup>

There are prognostic features of AVMs, which are related to higher intra- and postoperative complications.<sup>6</sup> These include (i) Volume of AVM (>20 cm),<sup>7</sup> (ii) Presence of deep feeding vessels and deep draining system, (iii) Shunt flow >120 cm.sec<sup>-1</sup> and pulsatility index <0.5 as estimated by Transcranial Doppler, (iv) Eloquent area of brain and (v) History of previous bleed. Langer et al suggested that smaller the AVMs greater the risk for hemorrhage because of high perfusion pressure inside the smaller AVMs.<sup>8</sup> The diagnosis of AVM is by the angiographic demonstration of arteriovenous shunting and is sine qua non of an AVM. However, MRI or MR angiography can also confirm it. The AVM has been graded by Spetzler et al and the gradation is based on

its size, pattern of venous drainage and neurological eloquence of the adjacent brain.<sup>9</sup>

### **Treatment modalities**

The decision to treat AVMs is based on the patient's age, neurological condition, and the characteristics of AVMs. The different treatment modalities are (a) Conservative management, (b) Microsurgical removal with or without endoscopic assistance, (c) Endovascular embolisation and (d) Stereotactic radiosurgery. Management strategies differ according to local preferences.<sup>2</sup> Despite this, many controversies still exist regarding the different modalities of treatment, however, the multimodal approach has been shown to improve clinical outcome.<sup>10-11</sup> If an angiogram after the treatment of AVM shows that the AVM has disappeared, then the patient is essentially cured and recurrence is extremely rare except in Spetzler's Grade IV and V AVMs.<sup>12</sup>

(a) Conservative management: It is primarily considered when the risk of other management options is thought to be excessive, based either on the nature of the AVM or the patient's physical condition. With the introduction and development of intravascular obliteration technique, neuroanesthesia, and radiosurgery, true indication for conservative management has decreased.

(b) Surgical resection: It is indicated for surgically accessible lesions in non-eloquent areas, where the risk of surgery is less than the risk of the natural history. Emergency surgery is indicated to remove increasing intracerebral hematoma that may pose a significant risk of brain herniation or to place a ventricular drain to treat acute hydrocephalus. Morbidity and mortality after AVM resection are based on the size, deep venous drainage, and the Spetzler-Martin grade.<sup>13</sup> In a recently published series, the reported rates of morbidity and mortality after AVM resection are 1% and 0.5% for patients with Grade I & II AVMs, 18.9% & 2.7% respectively for Grade III AVMs, and 25.6% & 7.7% respectively for patients with Grade IV & V AVMs.<sup>14</sup> Sometimes, a saccular aneurysm may coexist with AVM, the reported incidence ranges from 3.7-8.7%.<sup>15</sup> The aneurysm may rupture before, during and immediately after the treatment of an AVM.<sup>16</sup> Therefore, the presence of aneurysm should be ruled out before commencing the treatment of AVMs.

(c) Endovascular embolisation: The goal may be total obliteration of the lesion via embolisation alone, or to decrease the size and blood flow through the AVMs to facilitate surgical resection or radiosurgery later. Usually, the arterial endovascular approach is preferred for the endovascular embolisation of true AVMs.<sup>17</sup> The transfemoral approach is used to deliver embolic agents. A super selective angiography should be performed to identify all feeding arteries. After embolisation with N- butyl-cyanoacrylate (NBCA), the AVM becomes a solid, brittle mass and hence can be resected surgically if required. NBCA is most commonly used because it

appears to be most resistant to recanalization.

The overall goal of this technique is to reduce the shunt, prepare the patient for surgery, obliterate the lesion completely, and to embolise the deeper feeder vessels without causing damage to the eloquent areas of the brain, and to reduce the hyperemic complications. However, endovascular embolisation is not without risk. Frizzel RT and associates documented embolisation in 1246 patients with brain AVMs, showed cure in 5% patients and was associated with an 8% permanent morbidity and 1% mortality.<sup>18</sup> They further stressed that the morbidity after embolisation depends on the embolisation technique. Actually, the newer technologies have decreased the complications of the embolisation by one-third.<sup>19</sup> The complications that may result following embolisation are transient ischemic attack (2-30%), stroke (2-20%), hemorrhage (4-90% with 30% mortality), gluing of the catheter to the brain with liquid adhesive agents, recanalization and pulmonary embolisation.<sup>4</sup> Despite the possibilities of these complications, various studies suggest that endovascular embolisation is safe and cost-effective and shows better results if combined with surgery than surgery alone.<sup>20</sup>

(d) *Stereotactic radiosurgery (SR)*: SR is clearly an important adjunct in the multimodality treatment approach for large AVMs,<sup>21</sup> however, its role in the treatment of AVMs remains somewhat controversial, partially because of the relative lack of long-term, prospective studies comparing radiosurgery with surgical and/or endovascular treatments.<sup>22</sup> It has been advocated in the treatment of AVMs not amenable to surgical resection and as an adjunct in treating unusually large and complex AVMs to reduce their size before endovascular or surgical therapy. The risk of complications appears to be related to total radiation dose, volume of tissue irradiated, clinical history, the AVM's location and history of prior irradiation.<sup>22</sup>

The typical radiosurgery sequence involves: (i) the application of the stereotactic frame after induction of anesthesia in the CT scan room, (ii) definition of the lesion by CT scan with or without cerebral angiography, (iii) a waiting period for the computation of the dose and lesion surface contours, spent either in the ICU, anesthetic induction room in the operation theatre or in the general ward, (iv) accurate performance of radiosurgery and (v) removal of head frame and recovery from prolonged general anesthesia or sedation.

### **Anesthetic consideration**

Patients with AVM may need anesthesia for preoperative investigation (CT or angiography), for endovascular embolisation, for SR or for surgical resection of the lesion.

#### ***(A) Anesthetic drugs and their effects on the cerebral physiology and brain protection:***

Most of the intravenous anesthetics reduce the cerebral metabolic rate (CMR). This depression is accompanied by an in-

crease in vascular resistance and a decrease in cerebral blood flow (CBF). In contrast, most inhalation anesthetics except  $N_2O$  cause an increase in CBF (vasodilator effect) and a decrease in CMR. This effect does not necessarily mean an uncoupling of flow-metabolism.<sup>23</sup> Of all the available inhalational agents, isoflurane remains the primary agent for use in neurosurgical patients because of its limited potential to increase CBF and ICP and also because it is supposed to have the best cerebral protective effects due to its potent cerebral metabolic depressive effects.<sup>23</sup> On the other hand, intravenous anesthetics cause a dose-dependent decrease in CBF and cerebral metabolic rate of oxygen ( $CMRO_2$ ), except ketamine. As a result of its membrane stabilizing property and inhibition of free fatty acid liberation, etomidate may provide protection against ischemia.<sup>24-26</sup> Barbiturates, etomidate and propofol are found to provide brain protection during AVM embolisation and/or surgical resection, when the brain is at risk of focal ischemia.<sup>27-28</sup>

Although not confirmed for every anesthetic, those that dilate vessels may produce the steal phenomenon and the anesthetics that constrict the vessels have the opposite effect, resulting in inverse steal, and may protect the brain or ameliorate the damage,<sup>23</sup> apart from their effects on CBF. Effects of opioids on CBF,  $CMRO_2$  and ICP are variable, however, the combination of fentanyl and droperidol (neuroleptanaesthesia) has no significant effect on CBF and  $CMRO_2$  and can be safely used during embolisation.<sup>23</sup> However, whenever these opioids are used, slow administration and care to maintain mean blood pressure are recommended.<sup>29</sup>

With the exception of succinylcholine, the muscle relaxants cause a minimal change in CBF and ICP if respiration is well controlled and an increase in  $PaCO_2$  is avoided, whereas succinylcholine causes an increase in ICP because of fasciculation.<sup>23</sup>

### **(B) Anesthesia for investigational procedure**

For diagnostic CT, appropriate sedation and monitoring is all that is needed. This technique is also useful for cooperative children, however, some of them may need general anesthesia. If there are no signs of raised ICP, IV ketamine with midazolam may suffice with prior premedication with glycopyrrolate. In the presence of increased ICP, a mixture of pethidine and phenergan may be safely used intravenously.<sup>30</sup> If the procedure is prolonged, sedation can be supplemented with IV midazolam.

For angiography, the patient may come as an emergency or on an elective basis and may require either conscious sedation or general anesthesia. During preoperative assessment, attention should be paid especially to evaluate the neurological status (viz. ICP), airway, fasting status, cardiovascular stability and any therapeutic measures already taken.

Anesthetic induction techniques are dictated by the urgency and the clinical presentation. In most of the situations where there is no urgency, intravenous induction with thiopentone

or propofol with endotracheal intubation may be all that is needed. In an emergency, a rapid-sequence induction and intubation can be used. Intravenous atropine is advised before the use of succinylcholine to prevent bradycardia.<sup>31</sup>

### **(C) Anesthesia for endovascular embolisation**

We administer general anesthesia with tracheal intubation at our institute, however, in many western centers, the basic anesthetic approach in otherwise cooperative adult patients is conscious sedation under monitored anesthesia care (MAC).<sup>4,32-33</sup> General anesthesia is required for those patients who are uncooperative, need controlled ventilation (for good image quality as a slight movement may cause image distortion or if there is a sign of increase in ICP), have a pre-existing medical condition which precludes lying supine for long hours (arthritis, chronic cough, asthma), those suffering from claustrophobia or for those having an unstable neurological or cardiovascular status.

**(I) Preoperative evaluation and Premedication:** Apart from the usual pre-anesthetic evaluation, particular attention should be paid to evaluate neurological status, physical condition of the patient (CCF), drug history, protamine allergy (including insulin use, fish allergy),<sup>33</sup> history of prior anticoagulation or coagulation disorders, recent steroid use and contrast reaction. A history of transient ischemic attacks or cerebrovascular occlusion would militate against vigorous hypotension during embolisation.<sup>32</sup> If a procedure is contemplated under MAC, the patient's tolerance to lying supine for several hours should also be enquired. In female patients, the possibility of pregnancy should also be noted. We routinely administer oral diazepam or trichlorophos along with intramuscular or oral glycopyrrolate in appropriate doses and continue antiepileptic drugs (AEDs) and other medications necessary for the stability of the patient.

**(II) Room preparation:** Ideally, an interventional radiology suite should be equipped for anesthetic care exactly as standard operating rooms. All intravenous lines including the central venous catheter should have sufficient slack by attaching long extension tubing to their ports to have free movement of the C-arm of fluoroscopy, and tubing should be pre-filled with the desired drugs or saline. Continuous arterial line monitoring should be started before the induction of anesthesia. The femoral artery catheter which is placed by the radiologist should be flushed continuously with an intraflow device with heparinised saline. Finally, through this co-axial catheter, the pressure at the tip of the super selective catheter can be monitored during AVM embolisation. The patient should be wrapped in a blanket or placed in a warming suite and care should be taken to administer only warm fluid to avoid excessive cooling. The bladder should also be catheterized to monitor urine output that helps in fluid management because the contrast medium produces an osmotic load and often leads to vigorous diuresis.<sup>34</sup>

**(III) Anesthesia technique:** The choice of agents for the induction and maintenance of anesthesia should reflect the need for hemodynamic stability, rapid emergence at the end of the procedure, and the medical and neurological requirements of the patients. Factors favoring the choice of general anesthesia are age (infants or young children), uncooperative patients, potential airway problems (chronic cough, smoking, snoring), back pain, anticipated long procedure where complete immobility is needed and suspected increase in ICP where sedation is contraindicated.<sup>32</sup>

MAC is induced in the form of neuroleptanesthesia with combinations of fentanyl, droperidol, promethazine and midazolam, until the patient is sedated and drowsy but arousable. We generally employ general anesthesia using either thiopental or propofol for induction and tracheal intubation with any muscle relaxant except succinylcholine. Ventilation is controlled to achieve moderate hypocapnia, as spontaneous ventilation with the possibility of hypercarbia may be detrimental. A baseline activated clotting time (ACT) is also obtained<sup>32</sup> and the patient is then anticoagulated with heparin 1 mg/kg IV to keep the ACT 1½ times normal. Heparin should be continued for a further 24 hours.<sup>33</sup> The rationale for post-procedure anticoagulation is to protect the patient against both the thrombogenic effects of endothelial trauma and the inherently thrombogenic materials instilled which can cause retrograde thrombosis in the embolised vessels.<sup>33</sup> A period of 24 hours is believed to be sufficient for a “pseudo-epithelial” layer to form and to prevent either retrograde or antegrade thrombus formation that may propagate along the arterial tree and venous system in AVMs with a potentially disastrous result.<sup>33</sup>

**(IV) Deliberate hypotension:** The blood pressure during embolisation should be tightly controlled; however, deliberate hypotension is required at the time of glue injection. The goal is to slow the flow through AVM feeding artery and to prevent systemic embolisation of glue. To create hypotension, sodium nitroprusside (SNP) can be infused keeping in mind that SNP is also a cerebral vasodilator and has theoretic potential for creating cerebral steal. However, of the drugs currently used for hypotension, SNP probably maintains cerebral perfusion the best.<sup>35</sup> Cardiac pause has also been used by infusing adenosine to decrease the flow through AVM during glue placement.<sup>36</sup>

**(V) Deliberate hypertension:** Not infrequently, a situation arises in which cerebral ischemia develops; during this period the brain can be protected by increasing the flow through the collateral pathways by raising the blood pressure by infusing phenylephrine with or without dopamine.

**(VI) Post-procedure management:** After the procedure, in an uncomplicated case, the trachea is extubated and patient is transported to ICU on O<sub>2</sub> via mask. Any episode of nausea and vomiting as well as headache during this period may warrant immediate neurological consultation. Deliberate hypo-

tension or hypertension is employed as per the merit of the case in the post-embolisation period.

#### **(D) Anesthesia for SR:**

Providing safe anesthesia for SR poses a unique challenge for the anesthesiologist, especially in infants and young children because of the following reasons:

- (i) It requires anesthetic management in four locations, as well as during the transportation of patients to and from these sites.
- (ii) Infant and young children must be “calm” and “cooperative” for a long period.
- (iii) The patient must be readily and safely transported to multiple sites within the hospital or sometimes between the two hospitals.
- (iv) Underlying pathophysiology and the risk of neurological deterioration during anesthesia.
- (v) Problems related to radiation exposure and the potential for intravenous contrast complication.
- (vi) Procedure-related problems.

(a) Application of stereotactic frame: The procedure is usually performed with the infiltration of the scalp with local anesthetics with or without brief intravenous sedation. However, general anesthesia with endotracheal intubation is required for children because of the following reasons.<sup>37-39</sup> First, the nature of the disease may warrant modification of intracranial hemodynamics; second, in the presence of the stereotactic device, to ensure a patent airway may be difficult and lastly, once the stereotactic device is placed, which overlies the nose and mouth, intubating the trachea is possible but may be challenging. It is important that the head frame wrench should always accompany the patient allowing its rapid removal from the patient’s head in case it becomes urgently necessary to secure emergency access to the patient’s airway.

(b) Other concerns: A closed-circuit television camera is helpful in allowing the anesthesiologist to view the patient’s head and chest and to monitor the monitors on a screen from outside the room during the delivery of the radiation while the patient is inside the SR suite.<sup>38</sup>

Routine anti-emetics should be used in view of the potential problem arising out of SR with head frame in-situ. Further, the anesthesiologist needs to be familiar with the potential problems of prolonged general anesthesia. These include development of hypoglycemia, hypothermia, nerve compression and fluid and electrolyte abnormality. There is a need for an indwelling urinary catheter as well as proper positioning of the patient in view of the long duration of the procedure.

(c) Anesthetic options: Inhalational anesthesia may be used during all the components of radiosurgery especially during induction and fixation of the head frame, however, the requirement of the longer duration of the procedure (8-12 hours) as well as the unavailability of vaporisers in different

areas and during transportation makes it unsuitable for SR. Intravenous anesthesia with propofol may be an ideal agent especially during transfer or in difficult remote locations viz. ICU or radiosurgery treatment room, where there may be no vaporisers and/or an anesthetic machine available. A total intravenous anesthetic technique is also described which offers cardiovascular stability and adequate depth of anesthesia for the various stages required in stereotaxis.<sup>40</sup>

(d) Extubation and recovery: Once the procedure is completed, the patient should be assessed for fitness for extubation on the basis of cerebral and cardiovascular hemodynamics and skin temperature. It is better to ventilate the patient electively for a few hours if the patient is not adequately warmed, especially infants and smaller children.

### **(E) Anesthesia for surgical resection**

Surgical resection, either alone or in combination with preoperative or intraoperative embolisation, remains the treatment of choice for AVM. Intraoperative ultrasonographic examination during the surgical treatment of AVM is gaining popularity because of its ability to detect the components and the accompanying part of the lesion.<sup>41</sup> However, the gold standard for the documentation of surgical cure of an AVM is a postoperative angiogram. Intraoperative angiography is used in many western centers for assessing the surgical obliteration of the AVM, however, it does not replace postoperative angiography to confirm AVM removal because of false negative findings.<sup>42</sup> The use of microsurgical technique, combined with embolisation, with improvement in neuroanesthesia techniques, has enabled the total resection of lesions previously considered inoperable.

(i) Preoperative evaluation and premedication: Preoperative evaluation and premedication are similar to the patients scheduled for embolisation (as discussed above). However, the development of any new neurological insult during embolisation, condition of hydration status as well as kidney status keeping in view that a large amount of dye might have been injected during angiography and/or embolisation should especially be evaluated and stabilized. Although preoperative endovascular embolisation has been shown to reduce intraoperative blood loss during surgical resection,<sup>20</sup> when it occurs, it can be rapid, massive and difficult to control. Hence, appropriate measures should be taken beforehand, including the availability of blood and blood product.

(ii) Technique of Anesthesia: Principle goals are smooth induction and intubation with tight blood pressure control. The major differences with embolisation are the presence of intense surgical stimulation and the possibility of sudden and profuse blood loss during surgical resection of AVMs. Consequently, the technique of anesthesia should be directed to suppress the noxious stimuli and to evaluate the volume status. Thiopental, etomidate and propofol would all be appropriate except in small children where we generally use sevoflurane

for induction. The hemodynamic response to laryngoscopy, intubation, and the placement of pins for head fixation and skin incision must be anticipated and the appropriate use of drugs (viz. lidocaine,  $\beta$ -blocker, nitroglycerine, SNP) at an appropriate time may be beneficial. If the patient is planned for operation in a sitting position, proper measures should be undertaken to avoid, diagnose and treat venous air embolism quickly should this occur.

Monitoring devices are applied before the induction of anesthesia. After induction but before tracheal intubation, especially in children, an indwelling arterial catheter should be placed for direct measurement of blood pressure. A central venous pressure catheter, pulmonary artery catheter or trans-esophageal echocardiography may be helpful in monitoring fluid replacement therapy, particularly in patients with underlying cardiovascular disease. In some cases neurological monitoring, especially somatosensory-evoked potential may be required.

(iii) Brain protection: Apart from AEDs, hyperventilation, furosemide, mannitol, and corticosteroids are administered during surgery to reduce the ICP. High-dose barbiturate can be used on a case-to-case basis; however, slow emergence is always a concern. The use of mild hypothermia has become popular in recent times. Meyer FB et al have used deep hypothermic circulatory arrest during the resection of large AVMs successfully.<sup>43</sup>

(iv) Emergence: Early emergence after tracheal extubation is desirable to have a quick and reliable assessment of the neurological function of the patient. Tight blood pressure control is needed to avoid bleeding from AVM bed. According to one report, the incidence of delayed hemorrhage leading to mortality and morbidity can be significantly reduced to 1% from 4.4% following resection for AVMs Grade > II and sizes >3.5 cm in diameter, if postoperative blood pressure is aggressively controlled.<sup>14</sup>

## **Special consideration**

### **(i) AVMs and pregnancy:**

The presentation of AVM during pregnancy is usually a result of hemorrhage following rupture.<sup>44</sup> Whether pregnancy is a risk factor for hemorrhage from AVMs is controversial.<sup>44</sup> An earlier study showed it carries 87% risk of hemorrhage, with poor outcome of baby in subsequent pregnancy if the AVM is untreated.<sup>45</sup> However, a more recent study found that the risk of first hemorrhage for pregnant women with an unruptured AVM was only 3.5%, similar to the known annual bleeding rate in the non-gravid population with an unruptured AVM.<sup>46</sup> Apart from AVM morphology, other factors that increase bleed from AVMs during pregnancy are younger age (20-25 years) and primigravida.<sup>47</sup> Once hemorrhage occurs, it accounts for 5-12% of all the maternal deaths and remains the third most common non-obstetric cause of maternal mor-

bidity.<sup>46</sup> AVMs during pregnancy may present with severe headache, meningism and photophobia and can be confused with eclampsia.<sup>48</sup> The confirmation of the diagnosis is made by computed tomography (CT) or lumbar puncture and cerebral angiography.<sup>48</sup>

Once the diagnosis of AVM in pregnancy is made, further management should be based on a team approach involving an obstetrician, neurosurgeon and anesthesiologist. The goals to maintain optimal fetal and maternal oxygenation on the one hand and to ensure a sufficient depth of anesthesia to prevent intracranial hypertension and re-bleeding on the other hand are problematic and challenging. The obstetric problems are those of raised intragastric pressure, aortocaval compression, fetal placental transfer of drugs, maternal oxygenation and maternal hypotension. In most neurosurgical practices, patients with unruptured AVMs or stable post-hemorrhage are allowed to reach term with an elective postpartum excision of AVMs. The best mode of delivery (vaginal or caesarean) in the patients with untreated AVMs still remain controversial, however, it appears to have little influence on either the fetal or maternal outcome.<sup>46</sup> The indications for surgical intervention are the same as for the non-gravid patient and are based on neurosurgical rather than obstetrical considerations.<sup>48-49</sup> In those patients with a high operative risk or inoperable lesions, a conservative management course should be adopted during the pregnancy allowing stereotactic radiosurgery or embolisation options to be pursued after delivery.<sup>44</sup>

The choice of the anesthetic technique for caesarean section is influenced by the need to maintain a stable cardiovascular system and is decided on a case-to-case basis. A regional anesthetic technique may be preferred if delivery is considered before neurosurgical intervention for AVM. After delivery, oxytocin in small doses or by infusion rather than ergometrine can be administered. In this fashion, oxytocin is unlikely to have an adverse effect on CBF or ICP.<sup>47</sup> If the resection of the AVM and the delivery of the fetus are considered simultaneously, general anesthesia is essential. In general anesthesia, normocarbia should be maintained. Excessive hyperventilation causes uteroplacental vasoconstriction, however, the decrease in uterine blood flow may also be due to the effect of intermittent positive pressure ventilation (IPPV) on maternal cardiac output.<sup>50</sup> The sequence of operation (delivery followed by surgical resection or vice-versa) in the same sitting depends on the condition of and the risk involved for the mother and the fetus, urgency of the particular intervention and the position of the patient required for neurosurgical intervention. If a sitting position is necessitated, the caesarean section should precede the neurosurgery. During general anesthesia one should intermittently look for vaginal bleeding, however, inhalational agents should be cautiously used to avoid uterine atonia.

(ii) AVMs of the great vein of Galen:

Cerebral AVMs involving the great vein of Galen are rare,

but nevertheless represent a significant proportion of AVMs presenting in infancy. Clinical presentation varies with the age of patients. In one series of 128 patients, 96% of the neonates presented with CCF and 92% had hydrocephalus whereas older children and adults presented with hydrocephalus (30%), hemorrhage (38%), neurological deterioration (15%), intracranial hypertension or CCF (2%).<sup>4</sup> The AVMs formed by the great vein of Galen have the largest shunt, have the poorest prognosis, and are nearly always fatal without treatment. Surgical ligation of the fistula has been tried with an extremely high mortality rate. However, with the rise in skills of the interventional radiologists, improved embolisation technologies and tools, and the non-invasive nature of the endovascular procedures, the role of surgery has since been diminished. Nevertheless, if interventional techniques cannot reach or obliterate significant feeding vessels, operative surgery may be indicated. However, its management is best achieved by a multidisciplinary team approach involving interventional radiologists, neurosurgeons, anesthesiologists, and neonatologists.<sup>51</sup> Mortality and morbidity are high even with these modalities of treatment and range from 23-75% and 21-88% respectively.<sup>52</sup> The cornerstone of approaching the vein of Galen malformation for its embolisation is the transvenous route through the femoral vein. The end point of the treatment is not the complete occlusion of the fistula but is related to improvement in cardiac function.<sup>17</sup> Often, more than one stage is required to reach the goal.<sup>17</sup>

CCF in these patients is secondary to both pressure and volumes overload and hence presents with a picture similar to persistent fetal circulation. The signs and symptoms of these malformations may be local, either from compression of brain tissue or from obstruction of CSF flow, or systemic as a result of cardiovascular decompensation. Patients present with signs of left-and/or right-sided heart failure. Laboratory tests will reveal electrolyte and pH imbalance.<sup>53</sup> These children may be on digoxin, furosemide, and inotropic drugs and often on mechanical ventilation. Drugs for the treatment of CCF and to control the ICP and seizure should be continued during the procedure; however, premedication in the form of sedatives should be best avoided.

Inhalation or intravenous induction may be performed in the child without evidence of increased ICP, however, neonates with CCF should have intravenous access before induction. Despite the potential adverse effects of ketamine on intracranial compliance, because of its potential beneficial effects on myocardium in CCF, its use as an induction agent in neonates with CCF is recommended.<sup>5</sup> Sometimes, hypotensive technique is required at the time of AVM ligation; however, one should be very careful in instituting the technique because neonates having CCF could not tolerate it, rather they needed vasoactive drugs to control their pressure, as the systemic vascular resistance in these patients is low. Fluid management in these patients is challenging. On the one hand, neonates may not

tolerate fluid load at all because of the presence of CCF, while on the other hand, children with a contracted intravascular compartment from attempts at brain dehydration may experience rapid circulatory collapse following brisk intraoperative bleeding. Hence, a triple lumen CVP catheter is inserted. Neonates with CCF may especially benefit from monitoring the cardiac filling pressure with the help of TEE, available beforehand along with the arrangement of adequate blood and blood products. These patients should be ventilated postoperatively until they demonstrate hemodynamic and neurological stability.

## Complications and their management

(I) Management of neurological catastrophes during embolisation:

The catastrophes during embolisation of AVMs can be rapid and dramatic and that needs urgent and planned attention. The primary responsibility of the anesthesia team is to secure the airway and preserve gas exchange, especially if conscious sedation is used and to communicate effectively with the radiologist in determining the extent and nature of the problem. In case of an occlusive problem, blood pressure should be augmented with or without direct thrombolysis to increase distal perfusion. If the problem is hemorrhagic, immediately, the blood pressure should be reduced by starting SNP infusion and heparin is reversed with protamine without undue regard for the systemic cardiovascular effect of protamine. The rapidity with which heparin is reversed is directly proportional to the good outcome from the bleed.<sup>33</sup> The patient's trachea should be intubated with the help of a muscle relaxant to secure the airway and to control the ventilation. This will also prevent a seizure. Further management is based on the patient's clinical and radiological findings.

The morbidity and mortality from these complications vary between 5-27% and 0-4% respectively in one series,<sup>19</sup> whereas between 8% and 1% respectively, in another.<sup>18</sup> The contrast medium can cause a variety of adverse reactions. It may include allergic reaction, or osmotic load that further aggravates CCF, especially in neonates, hypovolemia, electrolyte imbalance (osmotic load) and renal impairment, especially in patients with renal compromise.

(II) Complications after surgical resection:

Hyperemic complication:

Cerebral edema or hemorrhage may occur either during endovascular embolisation or surgical resection, and constitute hyperemic complication. Following hyperemic complication, 46% patients have good outcome as compared to 92% without it.<sup>54</sup> Two theories have been proposed to explain its pathogenesis, one is "normal perfusion pressure breakthrough" (NPPB) and other is "occlusive hyperemia" as proposed by Spetzler et al<sup>55</sup> and Al Rodhan et al<sup>56</sup> respectively. Among the two, the NPPB, is the most popular theory to explain its pres-

ence. With the abrupt removal of the shunt (i.e. AVM) from the circulation either by embolisation or by surgery, the increase in CBF into previously hypoperfused areas can lead to cerebral edema and hemorrhage at normal perfusion pressure leading to all the signs and symptoms related to it. Several mechanisms have been proposed to explain the NPPB syndrome; however none is conclusive.<sup>57</sup> The reported overall incidence of NPPB leading to postoperative morbidity may vary widely and ranges from 1.4-18%.<sup>54</sup>

In the last one decade, the overall rate of hyperemic complication has decreased, which may be attributed to the increased use of endovascular technique in combination with surgical resection to permit the staged removal of AVMs. Various authors have used SPECT imaging preoperatively to identify the patients who are at risk of the development of NPPB<sup>58-59</sup> or NIRS (near-infrared spectroscopy), to detect the hyperemic status of the adjacent brain after the resection of AVMs by measuring cortical oxygen saturation.<sup>60</sup>

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