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# Periprocedural management of patients presenting for neurointerventional procedures using flow diverters for complex intracranial aneurysms: An anesthetist's perspective - A narrative review

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**Abstract:**

Complex intracranial aneurysms pose significant challenges in the realm of neurointervention, necessitating meticulous planning and execution. This article highlights the crucial roles played by anesthetists in these procedures, including patient assessment, anesthesia planning, and continuous monitoring and maintaining hemodynamic stability, which are pivotal in optimizing patient safety. Understanding these complex procedures and their complications will aid the anesthetist in delivering optimal care and in foreseeing and managing the potential associated complications. The anesthetist's responsibility extends beyond the procedure itself to postprocedure care, ensuring a smooth transition to the recovery phase. Successful periprocedural anesthetic management in flow diverter interventions for complex intracranial aneurysms hinges on carefully orchestrating these elements. Moreover, effective communication and collaboration with the interventional neuroradiologist and the procedural team are emphasized, as they contribute significantly to procedural success. This article underscores the essential requirement for a multidisciplinary team approach when managing patients undergoing neurointerventions. In this collaborative framework, the expertise of the anesthetist harmoniously complements the skills and knowledge of other team members, contributing to the overall success and safety of these procedures. By providing a high level of care throughout the periprocedural period, anesthetists play a pivotal role in enhancing patient outcomes and minimizing the risks associated with these intricate procedures. In conclusion, the periprocedural anesthetic management of neurointervention using flow diverters for complex intracranial aneurysms is a multifaceted process that requires expertise, communication, and collaboration.

**Keywords:**

Anesthesia, complex intracranial aneurysm, flow diverter, neurointervention

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## Introduction

Flow diversion is an endovascular procedure aimed at treating conditions mainly cerebral aneurysms. It involves the deployment of a soft, braided, self expanding, and flexible mesh-like stent known as a flow diverter (FD) inside the parent artery with an aneurysm. This stent effectively redirects blood flow away from the aneurysm sac, reducing the risk of rupture. These procedures are vital for patients with cerebral aneurysms and entail the placement of specialized devices within the brain's blood vessels to redirect blood flow, thereby preventing the potential for rupture or bleeding. In this



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context, the role of an anesthetist is pivotal in managing complex neurointerventional procedures involving FD placement. This article offers an overview of the crucial responsibilities of an anesthetist in the management of neurointerventions, specifically those involving the placement of flow diverters.

## Evolution of Intracranial Flow Diversion

The endovascular management of intracranial aneurysms has emerged as the preferred treatment approach for patients with these conditions. Despite significant advancements in endovascular techniques, such as stent or balloon-assisted coiling, and the introduction of new-generation coils, this method still has limitations, primarily its inability to achieve complete and permanent occlusion of aneurysms.[1] This limitation becomes particularly relevant in the case of complex, large, and giant aneurysms, which carry a higher risk of recurrence. The concept underpinning the development of "endovascular flow diversion" techniques are grounded in the idea that modified stents can be employed to redirect blood flow "away" from the aneurysmal sac and "back" into the parent vessel, offering a promising alternative to address these challenges.

## How Do FD Devices Work?

FD reduces the parent artery–aneurysmal sac interface by altering inflow and outflow jets to induce sac thrombosis. This results in occlusion of the aneurysmal sac in 70%–80% aneurysms at 6 months.[2] The mechanism of action of FD relies on a complex interplay of FD properties, the parent vessel anatomy, the size of the aneurysm, presence of side branches and perforators. Notably, early research, both *in vivo* and *in vitro*, has highlighted the paramount importance of two key FD properties: porosity and pore density. While pore density quantifies the number of pores within a unit surface area, porosity is the ratio of the metal-free surface area to the total surface area of the device.[3] The mechanism underlying FD's effectiveness can be delineated into three distinct stages: the hemodynamic stage, thrombus formation, and endothelization, as elucidated in Table 1.[4] The subsequent neointimal overgrowth covers the stent effectively, thereby reconstructing the parent artery and eliminating the aneurysm/parent vessel interface, as depicted in Figure 1. This process is typically designed to spare the origins of perforators. Moreover, in the setting of fusiform aneurysms, these processes facilitate the creation of a seamless endothelial-covered channel that seamlessly continues with the parent artery, as depicted in Figure 2. These distinctive features are believed to significantly reduce rupture rates in clinical practice.[5]

**Table 1: Three stages by which the aneurysmal sac occlusion occurs in the flow diverter management of complex intracranial aneurysms**

| Stages | Mechanism of aneurysmal sac occlusion |
|---|---|
| Hemodynamic stage | Immediately after FD placement during which there is a disruption of blood flow into and out of the aneurysm from parent artery to the resistance created by mesh |
| Stage of thrombus formation | Phase of immediate platelet activation and progressive formation of stable thrombus over days to week |
| | Thrombus formation depends on multiple factors - aneurysm neck size, FD properties, patient's blood rheology and platelet response to antiplatelet medication |
| | During this phase there may be local inflammation or mass effect which can cause headache if previously present |
| Endothelization stage | Conversion of thrombus to fine collagen and endothelization of FD augmented by CD34+ progenitor cell which takes months to years |

FD: Flow diverter

## Indications of Flow Diversion Procedures

1. Giant or large intracranial aneurysms[6]
2. Wide-neck intracranial aneurysms[6]
3. Aneurysms arising from the internal carotid artery (ICA) (petrous to the superior hypophyseal segments).[6]

## Extended indications include

1. Posterior circulation, blister, fusiform, dissecting, bifurcation, small, distal aneurysms[7]
2. Intra- and extracranial vessel reconstruction (e.g., dissections, pseudoaneurysms, and cavernous carotid fistulas)
3. Bifurcation aneurysms[7]
4. Recurrent aneurysms postclipping or coiling.

Further studies are required to assess whether the benefits outweigh the potential risks when employing FD for treating blister-like aneurysms, bifurcation aneurysms, small aneurysms, and aneurysmal dysplastic arterial segments with multiple small aneurysms.

## Variants of FD are Currently in Use

1. Pipeline embolization device (ev3/Covidien, Irvine, California)[8]
2. Silk flow diverter (SILK; Balt Extrusion, Montmorency, France)
3. Surpass flow diverter (SURPASS; Stryker Neurovascular, Fremont, CA)[8]
4. Other devices in use include: derivo mini embolization device (Acandis, Germany), Fred – microvention (California, USA), P64 MW/P48MW flow modulation device (Phenox – Germany), Tubridge FD (MicroPort Medical Company, Shanghai, China).

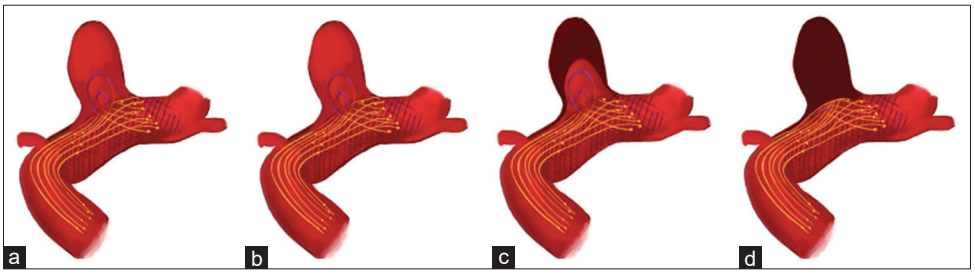


**Figure 1:** The stages involved in the obliteration of the intracranial aneurysm using the flow diverter (a) aneurysmal sac (b) flow diverter deployment in the parent vessel (c) parent vessel remodeling as the new arterial endothelial lining covers the device (d) aneurysmal sac thrombosis and obliteration

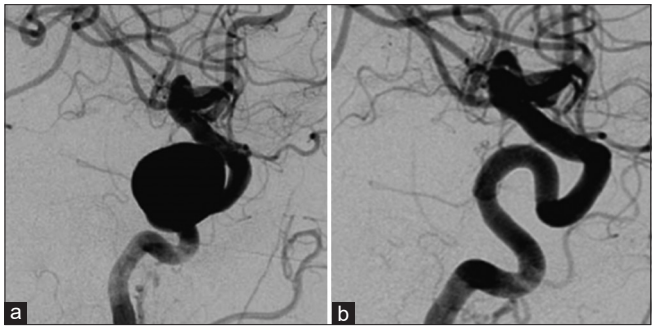


**Figure 2:** Digital subtraction angiography showing the aneurysmal sac in the internal carotid artery (a) and the obliteration of the sac postflow diverter deployment (b)

## Anesthetic Management of Patients Presenting for Flow Diversion Procedure

### The anesthesia goals for placement are

1. Maintaining stable hemodynamics, cerebral perfusion pressure (CPP), and transmural pressure gradient (TMP)
2. Platelet function assessment and management of periprocedural antiplatelet and anticoagulation therapy
3. Anticipation and management of intraprocedural complications
4. Facilitate early and prompt recovery to assess the neurological status
5. Facilitate safe transport of patients from radiology suite to intensive care units
6. Following radiation safety rules.

### Preoperative evaluation and workup

Flow Diversion is mainly indicated in the occlusion of unruptured aneurysms and in selected ruptured aneurysms. Before the neurointervention, it is essential to take a detailed preanesthetic evaluation, which includes a detailed history of current disease and comorbidities, along with a clinical examination of focal neurological deficits, Glasgow Coma Score (GCS), World Federation of Neurosurgeons (WFNSs) grading and Fisher grading of subarachnoid hemorrhage (SAH) in a case of the ruptured aneurysm.[9] A detailed drug history is essential, which includes screening for drugs affecting coagulation and screening for drug allergy. Patients with an allergy to iodine are at a risk for contrast-induced allergy.[10] Systemic heparinization is done intraoperatively, which is usually reversed by protamine. However, systemic heparinization is cautiously done in the setting of acute SAH. History regarding prior protamine allergy, prior anticoagulation, and coagulation disorder should be noted. Patients with a known history of hypersensitivity reactions to fish or prior exposure to protamine or protamine-containing insulin therapy may experience hypersensitivity reactions to protamine sulfate.[11] History of contrast allergy should also be noted, and these patients can be treated with antihistamines and steroids before the procedure.[12] Factors that increase the risk of contrast-induced nephropathy include preexisting renal impairment, uncontrolled hypertension, congestive heart failure, hypersensitivity conditions such as bronchial asthma, age over 70 years, diabetes mellitus (particularly when on metformin), dehydration, and the concurrent use of nephrotoxic medications such as aminoglycosides and nonsteroidal anti-inflammatory drugs.[13] Up to 300 ml of contrast agents might be used during the intervention, which poses a significant risk of contrast-induced nephropathy. Currently, there is insufficient evidence to recommend the prophylactic use of either N-acetyl cysteine or intravenous (IV) sodium bicarbonate. Notably, newer contrast agents like iodixanol (Visipaque), characterized by its iso-osmolarity to plasma, are known to be less nephrotoxic.[14]

A complete hemogram, baseline renal function test and serum electrolytes, coagulation profile, and comorbid-specific investigations must be done. In addition, patients undergoing FD will be on preprocedure antiplatelet therapy. The use of antiplatelet agents has become increasingly common in the placement of flow diverters (FDs) to mitigate the risk of thromboembolic complications. These agents, which include aspirin, glycoprotein IIb/IIIa receptor antagonists like abciximab, and thienopyridine derivatives such as clopidogrel, prasugrel, and ticagrelor, play a crucial role in preventing platelet aggregation and ensuring the success of the procedure.[15] Abciximab, eptifibatide, and tirofiban are

among the glycoprotein IIb/IIIa receptor antagonists, with abciximab exhibiting a potent but prolonged effect, which can lead to an increased risk of periprocedural bleeding. In contrast, eptifibatide and tirofiban are smaller-molecule agents with shorter half-lives, making them competitive blockers.[15] Thienopyridine derivatives, on the other hand, bind irreversibly to the platelet's adenosine diphosphate receptor, altering it permanently and ensuring an extended duration of action, typically lasting the life span of the platelet.

Dual antiplatelet therapy, often comprising aspirin in combination with clopidogrel, prasugrel, or ticagrelor, is a standard approach post-FD deployment to minimize thromboembolic risks.[15] Clopidogrel is frequently incorporated due to its established efficacy.[16] Although Prasugrel, a newer-generation thienopyridine, has a swifter onset of action and increased efficacy compared to clopidogrel, it has a higher risk of periprocedural bleeding. Replacing clopidogrel with prasugrel in antiplatelet therapy during endovascular treatment for nonruptured cerebral aneurysms has the potential to reduce the clinical impact of intraoperative and postoperative thromboembolic complications without a corresponding increase in hemorrhagic events.[17] The prasugrel group demonstrated superior and dependable platelet inhibition. It may be unnecessary to conduct the antiplatelet response assay when administering low-dose prasugrel before endovascular treatment for patients with unruptured aneurysms.[18] However, Prasugrel demands prudence in patients with a history of stroke or transient ischemic attacks due to the higher incidence of periprocedural complications. Ticagrelor, akin to its thienopyridine counterparts, delivers effective inhibition of P2Y12 with a moderate half-life of approximately 8 h, enhancing the array of antiplatelet options for FD placement procedures. Furthermore, pregnancy screening should be done in females of reproductive age group considering the radiation risk and the last menstrual period should be documented in female patients undergoing the procedure. It is advisable to defer the FD procedure during the active bleeding phase of the menstrual cycle, given the increased risk of menorrhagia due to concomitant antiplatelet and anticoagulant therapy.

## Preoperative platelet aggregometry test

Thromboembolic complications pose a significant risk of postprocedure morbidity following flow diversion procedures. In certain patients, the effectiveness of clopidogrel in inhibiting platelet aggregation may be compromised due to genetic variations in CYP2C19, the enzyme responsible for converting the drug into its active form.[19] Consequently, many medical institutions routinely conduct platelet function tests before proceeding with FD procedures. Optical aggregometry is routinely employed to evaluate clopidogrel's antiplatelet effects, whereby it inhibits ADP-induced aggregation in platelet-rich plasma (PRP).[20] PRP exhibits high optical density and poor light transmission due to the presence of platelets, which hinder the passage of light. Platelet-poor plasma, on the other hand, allows for better light transmission, resulting in low optical density. When a substance promoting platelet aggregation is introduced to PRP, normal platelets become activated and aggregate.[20] A platelet aggregometer tracks changes in optical density or light transmittance at 37°C over time. As platelets aggregate in PRP, optical density decreases (light transmittance increases) as the aggregated platelets move out of the path of light.[20] Consequently, patients nonresponsive to clopidogrel exhibit reduced platelet aggregation when they do not respond to clopidogrel.[20] In clinical practice, for individuals undergoing intracranial flow diversion, prasugrel is often favored, especially in cases where the patient does not respond adequately to clopidogrel.[19] Ticagrelor serves as a safe and effective alternative to clopidogrel but is less appealing due to its higher cost and requirement for twice-daily dosing.[15]

## Anesthetic management of flow diversion procedure

Routine preinduction monitoring comprises electrocardiography, noninvasive blood pressure measurement, and pulse oximetry. As flow diversion procedures have a potential for catastrophic periprocedural bleeding, it is advisable to secure an IV access with a wide-bore cannula. The induction goal is to preserve the TMP (mean arterial pressure-intracranial pressure [ICP]).[21] Therefore, any hypertensive episodes should be avoided to prevent any increase in TMP, which can cause aneurysm rebleed. IV induction is preferred in adults and inhalational in the pediatric age group. Intubation should be performed in a deeper plane of anesthesia to maintain the TMP. Intubation responses can be obtunded using lignocaine, esmolol, or fentanyl. Care should be taken during endotracheal intubation as any potential injury to the lips, teeth, or oropharyngeal cavity can be a source of significant bleeding due to the underlying antiplatelet and anticoagulant therapy. For the same reasons, an invasive arterial line for arterial pressure monitoring should be attempted in arteries that can be easily compressible and preferably under ultrasound (USG) guidance. In difficult IV access scenarios, USG-guided central venous access is preferred over landmark techniques to avoid accidental arterial injuries. Bladder catheterization is mandatory owing to the diuretic effect of contrast and to measure the hourly urine output.

## Anesthetic maintenance

Cerebral protection strategies include maintenance of CPP and TMP. Periprocedural hypertension can result

in rebleeding, and hypotension could lead to cerebral infarction. Therefore, the 6 N's-normoxia, normocarbia, normotension, normothermia, normonatremia, and normoglycemia should be maintained. Hypotensive episodes are encountered while the radiologist administers nimodipine boluses during various intervention stages. Vasopressors such as noradrenaline or phenylephrine can be used to maintain blood pressure.

Anesthesia is routinely with total IV anesthesia (TIVA) or inhalation anesthesia as per institutional practice. Few studies have explored the comparative impact of these techniques on patient outcomes in cases of SAH.[21,22] Bhagat *et al*. conducted a study comparing TIVA to inhalational anesthesia in patients undergoing aneurysm repair following SAH.[21] Their findings revealed no discernible difference between the two groups concerning long-term neurological outcomes and biomarkers of neuronal injury. Another study by Umesh *et al*.[22] retrospectively analyzed the effects of inhalational anesthesia versus TIVA in the context of SAH-induced delayed cerebral ischemia (DCI).[22] They observed that patients who received inhalational anesthesia experienced significant protection against SAH-induced angiographic vasospasm and DCI.[22] In addition, it is recommended to avoid nitrous oxide, as it can lead to the expansion of air bubbles inadvertently introduced into cerebral arteries through catheters.

## Analgesia

Interventional radiology procedures are usually less painful and can be managed using intermittent boluses or infusion of short-acting opioids, for example, fentanyl/remifentanil in addition to paracetamol (15–20 mg/kg) and local site infiltration of local anesthetics.

## Anticoagulation and reversal

At the outset of the procedure, a baseline activated clotting time (ACT) is measured, with the normal value falling within the range of 90–130 s. Subsequently, unfractionated heparin is administered at a dose of 70–100 U/kg, with the aim of achieving an ACT that is 2–3 times the baseline value. This approach is adopted to prevent the occlusion of vessels during the procedure. Arterial dissection leading to intracranial hemorrhage is a rare but dreaded complication that may occur during FD procedures. Similarly, silent hemorrhage into the retroperitoneal compartment or the soft-tissue compartment of the thigh is also known to occur. This should be ruled out if there is an intraprocedural refractory hypotension with signs of hypovolemia. In addition, hematuria and airway bleed can occur. In such scenarios, heparin reversal is done with IV protamine sulfate after removing the interventional catheter [Table 2]. Patients undergoing FD are at a higher risk as they will be on antiplatelets.[23]

**Table 2: Dosage of protamine for reversal of heparin**

| Protamine dose for reversal | |
|---|---|
| **Time since last dose (min)** | **Dose of protamine per units of heparin** |
| <30 | 10 mg/1000 U heparin |
| 30–60 | 0.5–0.75 mg/100 U heparin |
| 60–120 | 0.375–0.5 mg/100 U heparin |
| 120 | 0.25–0.375 mg/100 U heparin |
| Maximum dose | 50 mg |
| Infusion rate | 10 mg/ml, solution should not exceed 5 mg/min |

## Extubation and Recovery

Patients with good grade SAH (WFNS Grades I or II) may be extubated in the radiology suite. Smooth emergence is of utmost importance to avoid a rise in ICP and the risk of rebleeding of the recently secured aneurysm.[24] To minimize coughing during extubation, a patient with normal airway anatomy can be extubated in a deeper plane of anesthesia, or IV lidocaine (1.5 mg/kg) can be administered. Labetalol and esmolol are both effective in controlling emergency hypertension. Gentle suctioning is to be done due to the risk of mucosal bleeding. In patients with WFNS Grade II, the decision to extubate depends on the preoperative GCS and level of preoperative ventilatory support received. Patients with preoperative WFNS Grades IV or V usually require preoperative ventilatory support, which can be continued in neurointensive care unit (NICU).[25] These patients often require intrahospital transfer from the radiology suite to the NICU for close hemodynamic and neurological monitoring and ventilatory support.

## Periprocedural and delayed complication

1. Side branch occlusion:
   a. During ICA deployment, occlusion of the ophthalmic artery, posterior communicating artery, and anterior choroidal artery can occur
   b. During basilar artery deployment, occlusion of the anterior inferior cerebellar artery and posterior cerebral artery can occur.
2. Perforator occlusion: Higher incidence with basilar artery aneurysms (3% symptomatic occlusion)[25]
3. Intraprocedural vessel rupture: To prevent this complication, it is imperative to continuously monitor the distal delivery wire position, exercise careful manipulation, and perform angioplasty to confirm the appropriate stent-to-vessel wall distance
4. Perianeurysmal edema: Common in giant aneurysms and areas of the brain without intervening cerebrospinal fluid space due to inflammatory processes that occur along with aneurysmal thrombosis. It usually lasts 48–72 h postprocedure and requires antiedema measures

5. Distal infarction: Due to excessive manipulation during stent deployment
6. Delayed hemorrhage: Intraparenchymal and SAH[25]
7. Flow divertor thrombosis: Adequate antiplatelet therapy for at least 7 months is mandatory to prevent FD thrombosis.[26] In addition, measures to reduce antiplatelet failure should be considered
8. Contrast-induced encephalopathy: Contrast-induced encephalopathy (CIE) is an uncommon complication resulting from the use of contrast agents. It manifests with symptoms such as headaches, altered consciousness, seizures, cortical blindness, and temporary focal neurological deficits. The primary risk factors associated with CIE include the use of high-dose contrast agents and the direct injection into the posterior circulation. Managing CIE typically involves symptomatic treatment and ensuring proper hydration, which are the primary approaches for addressing this condition.[27]

## Conclusion

Flow diverters have expanded the therapeutic options for the treatment of giant and large cerebral aneurysms and aneurysm's with wide neck. Anesthetists are essential members of the neurointerventional team and play a critical role in optimizing patient safety and comfort during complex procedures like flow diverters. Their expertise in anesthesia management and understanding of the procedures involved in FD is vital for successful outcomes and minimizing complications.

### Author contributions

Dr. Sree: Concepts, design, definition of intellectual content, literature search, clinical studies, experimental studies, data acquisition, data analysis, manuscript preparation, manuscript editing and manuscript review; Dr. Hrishi: Concepts, design, definition of intellectual content, literature search, clinical studies, data acquisition, data analysis, manuscript preparation, manuscript editing, manuscript review and guarantor; Dr. Praveen: Concepts, design, definition of intellectual content, literature search, data analysis, manuscript preparation, manuscript editing and manuscript review; Dr. Sethuraman: Concepts, definition of intellectual content, clinical studies, data analysis, manuscript preparation, manuscript editing and manuscript review.

### Ethics committee approval and declaration of Helsinki

Not applicable.

### Patient consent

Not applicable.

### Data availability statement

Data sharing not applicable to this article as no datasets were generated and/or analyzed during the current study.

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### Conflicts of interest

There are no conflicts of interest.

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