

## Anesthetic Management for Wingspan Stent

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### ABSTRACT

**Background:** The bare metal self-expanding Wingspan stent (Boston Scientific, Natick, MA) was approved by the Food and Drug Administration under the Humanitarian Device Exemption in August 2005 for patients with intracranial atherosclerotic disease (ICAD) who are refractory to medical therapy. Relatively low rates of periprocedural morbidity and mortality have been reported.

**Methods:** After receiving institutional review board approval, we conducted a retrospective chart review to examine the anesthetic management and perioperative mortality and morbidity for all Wingspan stent insertions performed at our institution from 2005 to 2007.

**Results:** A total of 72 patients with a history of intracranial stenosis had angioplasty and Wingspan stent insertion: 34 male and 38 female, with an average age of  $64 \pm 11.6$  years. Preoperative systolic blood pressure was  $200 \pm 45$  mmHg, and diastolic blood pressure was  $100 \pm 23$  mmHg. All patients received general anesthesia for stent insertion. Five patients died (6.9%), 4 had perioperative stroke (5.5%), and 9 had recurrent stenosis (12.5%).

**Conclusions:** Anesthetic management for Wingspan stent insertion for ICAD is challenging. Maintenance of hemodynamic stability with optimum brain perfusion during the stent

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**Keywords:** Anesthetic management, bare metal, intracranial atherosclerosis, self-expanding, stent, Wingspan stent

*The authors have no financial or proprietary interest in the subject matter of this article.*

deployment is crucial to patient safety. A prospective study is warranted to assess the optimal anesthetic choice during Wingspan stent insertion.

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### INTRODUCTION

Atherosclerotic disease of the major intracranial arteries is a major cause of stroke. The Warfarin-Aspirin Symptomatic Intracranial Disease (WASID) trial<sup>1</sup> revealed that aspirin is as effective as and safer than warfarin for preventing stroke or vascular death in patients with atherosclerotic disease and that patients with  $\geq 70\%$  intracranial stenosis are at particularly high risk of stroke despite antithrombotic therapy.<sup>2</sup> Therefore, the use of cerebral stents seems to be an effective therapeutic tool for managing high-grade cerebral stenosis.

The bare metal self-expanding Wingspan stent (Boston Scientific, Natick, MA) was approved by the Food and Drug Administration (FDA) under a Humanitarian Device Exemption in August 2005 to be used in patients with intracranial stenosis ( $>50\%$ ) who are refractory to medical therapy. The preliminary results from Europe and Asia for using the Wingspan stent showed very low rates of periprocedural morbidity and mortality (4.4%) in a series of 45 patients undergoing treatment.<sup>3</sup>

More recently, a multicenter registry in the United States led by Cleveland Clinic reported relatively low rates of periprocedural complications (6.1%) and a high rate of successful deployment ( $>98\%$ ).<sup>4</sup> We describe for the first time our experience with anesthetic management of patients with intracranial atherosclerotic disease (ICAD) who underwent Wingspan stent insertion.

### METHODS

After receiving institutional review board approval, we conducted a retrospective chart review to assess the anesthetic management of all Wingspan stent insertions performed at our institution over a 2-year

**Table 1. Patients' Demographic Characteristics**

<b>Age</b>
64 ± 11.6 years (range: 37-85)
<b>Sex</b>
Male (n = 34, 47.2%)
Female (n = 38, 52.8%)

period, as well as these patients' perioperative mortality and morbidity.

## RESULTS

A total of 72 patients with a history of intracranial stenosis underwent angioplasty and Wingspan stent insertion. Preoperative systolic blood pressure (SBP) was 200 ± 45 mmHg, and preoperative diastolic blood pressure was 100 ± 23 mmHg.

The patients' demographics and comorbidities are shown in Tables 1 and 2. Pathological diagnoses and vessels involved are shown in Tables 3 and 4.

All patients received general anesthesia during stent insertion. Anesthetic drugs and medications used during the perioperative period are shown in Table 5. Nitrous oxide was not administered to avoid enlarging minute air emboli that commonly occur during angiography and irrigation. Inhalation anesthetics were used in most patients to ensure the patient's immobility during the procedure.<sup>5</sup> Inhalation anesthetics have neuroprotective effects via preconditioning and post-conditioning that might be helpful during the ischemic events of the procedure.<sup>6-9</sup> All patients had an arterial line inserted prior to the induction of anesthesia to ensure continuous blood pressure (BP) monitoring and to facilitate tight control. Arterial blood gas analysis was obtained at regular intervals to ensure proper levels of arterial oxygenation.

During the procedure, the patients were kept normocapnic or slightly hypercapnic to ensure proper

**Table 3. Patients' Pathological Diagnoses**

<b>Diagnosis</b>	<b>N =</b>	<b>%</b>
Basilar artery occlusion/stenosis	6	8.3
Carotid dissection/occlusion/stenosis	6	8.3
Vertebral artery stenosis	2	2.8
Left internal carotid artery occlusion/stenosis	2	2.8
Left middle cerebral artery stenosis	3	4.2
Medically refractory mid-basilar stenosis	1	1.4
Right internal carotid artery occlusion/stenosis/stroke	2	2.8
Right middle cerebral artery stenosis/stroke	3	4.2
Symptomatic intracranial atherosclerotic disease refractory to medical treatment	47	65.3

cerebral perfusion both before angioplasty and during temporary cerebral blood flow reduction at angioplasty and stent insertion. More than 50% of our patients required vasopressor support. This is understandable because 65.3% of our patients had a history of systemic hypertension that predisposes to relative hypotension after the induction of anesthesia. Phenylephrine was used in 40 patients (55.6%) to keep the SBP above 160 mmHg and the mean arterial pressure above 95 mmHg to promote collateral circulation before and during stent deployment. If the patient had impaired left ventricular function and low ejection fraction, norepinephrine was used instead because of its inotropic and vasopressor properties.

Postoperative complications occurred in 9 patients and included gastrointestinal bleeding, stroke, transient ischemic attacks, infarction, and hemorrhage (Table 6). Recurrent stenosis occurred in 9 patients (12.5%), and postoperative death occurred in 5 patients (6.9%).

## DISCUSSION

The Wingspan stent gained FDA approval in 2005 for the treatment of symptomatic intracranial stenoses

**Table 2. Patients' Preoperative Comorbidities**

<b>Comorbidity</b>	<b>N =</b>	<b>%</b>
Hypertension	47	65.3
Hyperlipidemia	39	54.2
Diabetes mellitus	24	33.3
Coronary artery disease	17	23.6
Congestive heart failure	4	5.6
Atrial fibrillation	3	4.2
Carotid occlusion	18	25.0
Peripheral vascular disease	7	9.7
Cerebral aneurysm	2	2.8
Cerebrovascular accident	29	40.3

**Table 4. Patients' Vessels Involved**

<b>Vessel</b>	<b>N =</b>	<b>%</b>
Basilar artery	19	26.8
Intracranial stent redo	1	1.4
Left internal carotid artery	11	15.5
Left middle cerebral artery	7	9.9
Left vertebral artery	4	5.6
Proximal M2 middle cerebral artery stenosis	1	1.4
Right internal carotid artery	10	14.1
Right middle cerebral artery	12	16.9
Right vertebral artery	6	8.5

Data were not available for one patient.

**Table 5. Anesthetic Management and Medications Used for Blood Pressure Control**

Medications	N =	%
<b>Induction drugs</b>		
Etomidate	6	8.3
Sodium thiopental	4	5.6
Propofol	40	55.6
Others	22	30.5
<b>Maintenance drugs</b>		
Desflurane	12	16.7
Dexmedetomidine	4	5.6
Isoflurane	21	29.2
Remifentanil	3	4.2
Sevoflurane	10	13.9
Others	24	33.3
<b>Intraoperative agents</b>		
Phenylephrine	40	55.6
<b>Antihypertensive agents used in the postanesthesia care unit</b>		
Hydralazine	20	27.8
Labetalol	21	29.2
Nicardipine	16	22.2

(>50%) refractory to medical therapy. The substantial reduction in periprocedural complications with Wingspan stents (6.1%) in comparison to symptomatic vertebrobasilar ICAD undergoing treatment with traditional stent (23.1%) reported in the Fiorella et al study is because of the recommended treatment strategy and the device design.<sup>4</sup> Before deployment of the Wingspan stent, angioplasty is performed with the Gateway balloon (Boston Scientific). The balloon is only inflated to 80% of the normal parent vessel diameter, reducing the risk of target vessel perforation and downstream embolization of atheromatous debris caused by plaque disruption. In terms of design, the Wingspan microstent is composed of nitinol and is housed in a low-profile, hydrophilic microcatheter delivery system. These properties make the stent delivery system considerably easier to navigate to and across an intracranial target lesion. The enhanced navigational ability of the Wingspan stent is thought to result in decreased perioperative complications.<sup>2,4,10</sup>

The anesthetic management for Wingspan stent insertion in patients suffering from ICAD requires an understanding of the technical aspects of the stent insertion, the ability to anticipate periprocedural complications, and the recognition of cerebral hemodynamic principles.

In our retrospective study, most of the patients undergoing Wingspan stent insertion were elderly and had multiple comorbidities related to atherosclerosis

**Table 6. Incidence of Neurologic and Other Complications**

Complication	N =	%
Acute infarct in the left dorsolateral midbrain	1	1.4
Cerebellar stroke	1	1.4
Ischemic stroke embolic shower	1	1.4
Postprocedure stroke in middle cerebral artery territory	1	1.4
Gastrointestinal bleeding	2	2.8
Right groin hemorrhage	1	1.4
Transient ischemic attacks	2	2.8

4 cases of stroke = 5.5%

and age, potentially complicating anesthetic management. Although all patients received general anesthesia, different agents were used for anesthetic induction, and maintenance of anesthesia was accomplished primarily with inhalation anesthetics and muscle relaxants. The anesthetic technique used most frequently consisted of propofol for induction and isoflurane for maintenance. Total intravenous anesthesia, monitored anesthesia care, and sedation were not used.

General anesthesia may be preferable for this procedure. Immobility is critical during mapping, while crossing the atherosclerotic lesion with the microwire, and during stent insertion. Moreover, the upper airway can be secured effectively with general anesthesia, reducing the risk of hypoxic episodes that might exacerbate brain ischemia during angioplasty and stent insertion.

We are aware of only one report of Wingspan stent insertion without the use of general anesthesia.<sup>11</sup> Sedation may result in unpredictable effects on patients' ability to lie still for the procedure. Sedation may also compromise upper airway patency and result in startle reactions that jeopardize safe stent deployment. Some patients may also experience paradoxical effects with benzodiazepine sedation.

After successful stent insertion, SBP should be reduced to  $\leq 140$  mmHg to avoid reperfusion syndrome or hemorrhage. In our institution, we prefer using nicardipine infusion to maintain the BP within the required target range because of its primary physiologic effect as opposed to vasodilation that has limited effects on chronotropy, dromotropy, and inotropy.<sup>12</sup>

The ischemic brain causes an increase in plasma norepinephrine and epinephrine to maintain its perfusion.<sup>13</sup> Increased serum catecholamine levels may have adverse consequences such as elevating the risk of myocardial ischemia in susceptible patients. During the procedure, anesthetic management aims to maintain proper perfusion to the brain and other vital organs while at the same time counteracting the surge of catecholamines.

Dexmedetomidine infusion was used in 4 patients in this study as an adjunct to inhalation anesthesia. The experience from this very limited number of patients was encouraging. Dexmedetomidine induced smooth reduction of BP in patients with severely elevated BP. Also, we noticed that patients on dexmedetomidine during the immediate postoperative period needed less medication to keep SBP  $\leq$ 140 mmHg. Another potentially beneficial effect of dexmedetomidine may be a neuroprotective effect mediated through sympatholysis. The mechanisms of such a neuroprotective effect may involve improvements in the balance between cerebral oxygen demand and oxygen supply; dexmedetomidine may also lessen direct toxic catecholamine effects and improve perfusion in the ischemic penumbra.<sup>13</sup>

However, the use of dexmedetomidine as anesthetic agent for patients with ICAD is still controversial. Dexmedetomidine has been reported to decrease cerebral blood flow (CBF) in humans.<sup>14</sup> This observation, taken with the fact that cerebral metabolic rate (CMR) was reported unchanged in an experimental study,<sup>15</sup> gave rise to the concern that dexmedetomidine might result in an unfavorable cerebral oxygen balance. However, in a study of human volunteers, Drummond et al<sup>16</sup> showed, contrary to previously reported animal studies, that human CMR/CBF coupling is preserved during dexmedetomidine administration. Also, the incidence of intracarotid shunting in patients undergoing awake carotid endarterectomy who received dexmedetomidine during the procedure did not increase.<sup>17</sup> Further studies regarding the safety of dexmedetomidine during stenting procedures for ICAD are necessary.

During the stent insertion procedure, patients are usually given heparin to maintain activating clotting time between 250 seconds and 300 seconds, and dual antiplatelet therapy is initiated or maintained with aspirin and clopidogrel. Anticoagulation and antiplatelet therapy presents a challenge when vessel rupture occurs during the procedure. Rapid reversal of anticoagulation by the administration of protamine and platelet transfusion is a standard practice.

## CONCLUSIONS

Periprocedural neurological complications in our report were 5.5%, comparable to the incidence of 6.1% previously reported in a US multicenter trial of 70 patients.<sup>4</sup> Awareness of periprocedural complications is critical for anesthesiologists managing patients undergoing interventional neuroradiologic procedures for ICAD. The rupture of intracerebral vessels is the most severe complication, resulting in intracerebral hemorrhage. Close communication with the

neurointerventional team is essential. Decisions regarding the reversal of anticoagulation, the control of BP, and management of temporary cerebral ischemia may become necessary and should be made jointly. Vessel rupture can often be treated endovascularly but might also require urgent craniotomy. The availability of blood products, adequate intravenous access, and management of expanding intracerebral hematoma with attention to intracranial volume control and cerebral perfusion pressure maintenance are of utmost importance.

The use of the Wingspan stent will likely expand over the coming years. Anesthetic management may well contribute to enhanced periprocedural patient safety and outcome. Our experience highlights the importance of conducting further prospective studies to evaluate the best anesthetic technique to be used during cerebral stenting procedures.

## REFERENCES

- Chimowitz MI, Lynn MJ, Howlett-Smith H, et al. Comparison of warfarin and aspirin for symptomatic intracranial arterial stenosis. *N Engl J Med.* 2005 Mar 31;352(13):1305-1316.
- Derdeyn CP, Chimowitz MI. Angioplasty and stenting for atherosclerotic intracranial stenosis: rationale for a randomized clinical trial. *Neuroimaging Clin N Am.* 2007 Aug;17(3):355-363, viii-ix.
- Henkes H, Miloslavski E, Lowens S, Reinartz J, Liebig T, Kühne D. Treatment of intracranial atherosclerotic stenoses with balloon dilatation and self-expanding stent deployment (WingSpan). *Neuroradiology.* 2005 Mar;47(3):222-228. Epub 2005 Mar 15.
- Fiorella D, Levy EI, Turk AS, et al. US multicenter experience with the wingspan stent system for the treatment of intracranial atheromatous disease: periprocedural results. *Stroke.* 2007 Mar;38(3):881-887. Epub 2007 Feb 8.
- Yang J, Chai YF, Gong CY, et al. Further proof that the spinal cord, and not the brain, mediates the immobility produced by inhaled anesthetics. *Anesthesiology.* 2009 Mar;110(3):591-595.
- Kapinya KJ, Löwl D, Fütterer C, et al. Tolerance against ischemic neuronal injury can be induced by volatile anesthetics and is inducible NO synthase dependent. *Stroke.* 2002 Jul;33(7):1889-1898.
- Tanaka K, Ludwig LM, Krolikowski JG, et al. Isoflurane produces delayed preconditioning against myocardial ischemia and reperfusion injury: role of cyclooxygenase-2. *Anesthesiology.* 2004 Mar;100(3):525-531.
- Preckel B, Schlack W, Comfère T, Obal D, Barthel H, Thämer V. Effects of enflurane, isoflurane, sevoflurane and desflurane on reperfusion injury after regional myocardial ischaemia in the rabbit heart in vivo. *Br J Anaesth.* 1998 Dec;81(6):905-912.
- Lange M, Redel A, Lotz C, et al. Desflurane-induced postconditioning is mediated by beta-adrenergic signaling: role of beta 1- and beta 2-adrenergic receptors, protein kinase A, and calcium/calmodulin-dependent protein kinase II. *Anesthesiology.* 2009 Mar;110(3):516-528.
- Fiorella D, Woo HH. Emerging endovascular therapies for symptomatic intracranial atherosclerotic disease. *Stroke.* 2007 Aug;38(8):2391-2396. Epub 2007 Jun 21.

11. Kelly ME, Turner RD, Moskowitz SI, et al. Revascularization of symptomatic subacute cerebrovascular occlusions with a self-expanding intracranial stent system. *Neurosurgery*. 2009 Jan;64(1):72-78; discussion 78.
12. Tobias JD. Nicardipine: applications in anesthesia practice. *J Clin Anesth*. 1995 Sep;7(6):525-533.
13. Engelhard K, Werner C, Kaspar S, et al. Effect of the alpha<sub>2</sub>-agonist dexmedetomidine on cerebral neurotransmitter concentrations during cerebral ischemia in rats. *Anesthesiology*. 2002 Feb;96(2):450-457.
14. Zornow MH, Maze M, Dyck JB, Shafer SL. Dexmedetomidine decreases cerebral blood flow velocity in humans. *J Cereb Blood Flow Metab*. 1993 Mar;13(2):350-353.
15. Karlsson BR, Forsman M, Roald OK, Heier MS, Steen PA. Effect of dexmedetomidine, a selective and potent alpha 2-agonist, on cerebral blood flow and oxygen consumption during halothane anesthesia in dogs. *Anesth Analg*. 1990 Aug;71(2):125-129.
16. Drummond JC, Dao AV, Roth DM, et al. Effect of dexmedetomidine on cerebral blood flow velocity, cerebral metabolic rate, and carbon dioxide response in normal humans. *Anesthesiology*. 2008 Feb;108(2):225-232.
17. Bekker A, Gold M, Ahmed R, et al. Dexmedetomidine does not increase the incidence of intracarotid shunting in patients undergoing awake carotid endarterectomy. *Anesth Analg*. 2006 Oct;103(4):955-958.

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