REVIEW ARTICLE

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Perioperative Hemodynamic Management of Carotid Artery Surgery

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STENOSIS of the extracranial carotid artery is known to be responsible for 15% to 20% of ischemic strokes.[1,2] Carotid artery surgery has been proven to be the best secondary prevention for reducing the risk of recurrent stroke.[3] Therefore, surgical treatment has become the standard practice to prevent stroke and its devastating clinical features.

Hemodynamic instability is a common perioperative finding caused by both an intrinsic dysfunction and an iatrogenic manipulation of the baroreceptors.[4] In addition, hemodynamic instability depends on the timing and type of surgery and is influenced by the anesthetic management.[4–13]

The available literature providing practical recommendations for perioperative management of hemodynamic instability is fragmented. This review aims to provide an update of the pathophysiology of perioperative hemodynamic instability during carotid endarterectomy (CEA) and carotid artery stenting (CAS), together with a practical perioperative hemodynamic management strategy. In addition, although the incidence of cerebral hyperperfusion syndrome (CHS) in this clinical setting is rather small, it is one of the most feared complications of carotid artery surgery.[14,15] Therefore, specific attention has been paid to its diagnosis, prevention, and therapy. The aim of this review is to provide more robust anchors and recommendations that may help improve the perioperative management of patients undergoing carotid artery surgery.

PERIOPERATIVE HEMODYNAMIC INSTABILITY

To decrease the risk for the development of a new persistent neurologic deficit, a tight timeframe must be kept between the appearance of neurologic symptoms and surgery. The European Carotid Surgery Trial and the North American Symptomatic Carotid Endarterectomy Trial demonstrated that a maximal stroke prevention, and therefore a better neurologic outcome, are obtained if surgery is performed within 2 weeks after the onset of neurologic deficits.[13,16–18] As a consequence, sparse time remains for an optimal preoperative evaluation and optimization of the patient's clinical condition. Furthermore, surgery in symptomatic patients is associated with increased intraoperative and postoperative hemodynamic instability, related to dysfunction of the baroreceptors during a 7- to 10-day period after a transient ischemic attack (TIA) or stroke.[5] As such, perioperative hemodynamic management may be intensive, and obtaining stable hemodynamics often may be difficult to achieve.

Pathophysiology of Perioperative Hemodynamic Instability in Carotid Artery Surgery

The arterial baroreflex contributes to the short-term regulation of blood pressure and may be a cause of cardiovascular variability. It modulates blood pressure and heart rate by alteration of the parasympathetic and sympathetic activity in response to acute changes of arterial blood pressure. This baroreflex is altered in patients with chronic hypertension, coronary artery disease, carotid artery disease, diabetes mellitus, and advanced age.[19,20]

The presence of a carotid atheroma reduces blood flow through the affected carotid artery, thereby decreasing cerebral perfusion. It also reduces baroreceptor sensitivity and affects cerebrovascular autoregulation, even in asymptomatic patients.[21] If only 1 site is affected, the overall functioning still can be considered as normal.[19] If both carotid arteries are involved, a bilateral baroreceptor dysfunction exists, making the patient more prone to intraoperative and postoperative hemodynamic instability.[22]

When a standard CEA is performed, a longitudinal arteriotomy is done to remove the calcified plaque, usually followed by patch angioplasty.[23] CEA is associated with a surgical stripping of sensory nerve endings from the arterial wall, resulting in an impairment of baroreceptor sensitivity. This causes hemodynamic instability during and after the surgical procedure in 12% to 54% of patients.[4,6] The instability may last for several hours to days and is associated with an increased risk for the development of cardiac and neurologic complications.[4,24] Afterwards, the baroreceptor function normalizes spontaneously, resulting in a much better hemodynamic control during the late postoperative period.[7,25]

CAS is an alternative procedure to CEA. It is performed in patients considered to be at high surgical risk and in patients with difficult surgical access to the internal carotid artery due to neck irradiation or previous neck surgery. CAS may be associated with hypotension and bradycardia, occurring in 29% to 51% of patients. These symptoms may last for 12 to 24 hours.[7] It has been suggested that hypotension and bradycardia are caused by predominantly parasympathetic

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activity during placement of the stent.[26,27] However, when comparing patients undergoing CAS with a control group of risk patients without stenting, similar decreased baroreceptor sensitivity was found in both groups.[28] This finding suggested that the alteration of baroreceptor function was related more to the underlying disease than to the stenting procedure.[29]

Perioperative Hypertension

Perioperative hypertension occurs in 11% to 56% of patients undergoing CEA. It is observed more frequently after eversion CEA when compared with conventional CEA.[6,30–34] An eversion CEA involves an oblique circumferential transection of the internal carotid artery at the level of the carotid bulb. The longitudinal transection of nerve fibers finally results in decreased baroreceptor sensitivity.[34] Perioperative blood pressure management after carotid artery surgery often represents a delicate balance among hypertension, clinically acceptable blood pressure, and hypotension.

Intraoperative Management of Blood Pressure

During carotid artery cross-clamping, an adequate perfusion pressure must be maintained across the circle of Willis to reduce the risk of the development of ischemic neurologic deficits.[35,36]

The systolic blood pressure (SBP) should be kept between its normal preoperative value and 20% above that value.[37] However, it must be taken into account that intraoperative blood pressure elevation is associated with an increased risk of myocardial ischemia and intracranial hemorrhage and that these factors might render the insertion of a shunt technically difficult.[19,38,39] As will be discussed in the next section, the use of cerebral monitoring techniques might help in obtaining the right balance. In the presence of adequate cerebral perfusion, a more liberal approach (eg, relative hypotension) may be tolerated.[19]

Postoperative Management of Blood Pressure

Postoperative severe hypertension, which is defined as an SBP of at least 180 mmHg, is observed in 37% to 66% of patients undergoing CEA.[40–43] The most important risk factor for postoperative hypertension is a preoperative SBP greater than 160 mmHg.[4] Other risk factors include peripheral vascular disease, intraoperative shunting, intracranial carotid artery stenosis, renal insufficiency, neurologic instability, and cardiac arrhythmia.[44] Hypertension after carotid artery surgery typically peaks in the first hours after surgery.[19] Tight monitoring is mandatory because hypertension can lead to potentially life-threatening complications, such as postoperative bleeding, CHS, and myocardial infarction.[19,45–47] Generally, invasive blood pressure monitoring is strongly advised. Keeping SBP less than 160 mmHg or within 20% of the preoperative value is recommended, although other strategies exist.[19] To reduce the risk for the development of CHS or wound hematoma, lower blood pressures have been targeted.[47,48]

When hypertension develops in the postoperative period, pain or urinary retention first should be excluded as a cause. It is mandatory to treat perioperative hypertension in a controlled and titrated manner using short-acting antihypertensive drugs (Table 1). Data from literature comparing the efficacy among antihypertensive agents after carotid artery surgery are scarce. In addition, the high interindividual variability in responses makes it difficult to predict the most efficient drug.

Direct-acting vasodilators, such as sodium nitroprusside, nitrates, or nicardipine, are used widely and have been proven to be very efficient in treating hypertension. However, these drugs cause cerebral vasodilation, which might result in an increased cerebral blood flow (CBF) in patients with an altered baroreceptor function, eventually leading to intracranial hypertension.[49–52] Compared with nitroprusside, nicardipine has been shown to provide a more stable hemodynamic profile with less need for additional antihypertensive treatment.[53]

It has been suggested that central-acting α_2-receptor agonists might be superior in the treatment of postoperative hypertension after CEA.[54] Immediately after carotid cross-clamp release, elevated cranial norepinephrine levels were observed,[54] suggesting a central sympathomimetic mechanism causing postoperative hypertension. No elevation of intracranial and peripheral norepinephrine levels was observed in patients who did not suffer from postoperative hypertension.

The efficacy of α-and β-blocking agents, such as labetalol and esmolol, have been shown to be suitable for the treatment of perioperative hypertension.[55,56] These agents have no cerebral vasodilating effects and do not influence intracranial pressure.[57] According to Greene et al,[58] β-lytics most commonly are used to treat hypertension after carotid artery surgery.

Sublingual nifedipine is contraindicated in this setting because it may induce sudden, severe, and uncontrolled hypotension.[59,60]

Table 1. Overview of Commonly Used Antihypertensive Agents in Carotid Artery Surgery

Drug	Mechanism of Action	Recommended Intravenous Dose
Esmolol	Selective β_1-antagonist	Loading dose: 0.5-1 mg/kg Continuous infusion: 25-300 μg/kg/min
Labetolol	Selective α_1-antagonist, nonselective β-antagonist	Loading dose: 20 mg Continuous infusion: 0.5-2 mg/min
Nicardipine	Calcium channel blocker	2-4 mg/min until desired effect, Maintenance: 3-30 mg/h
Clonidine	α_2-agonist	Loading dose: 2-4 μg/kg Continuous infusion: 1-4 μg/kg/h
Sodium nitroprusside	NO release => vasodilation A>V	Continuous infusion: 0.25-2 μg/kg/min
Nitroglycerine	NO release => vasodilation V>A	Continuous infusion: 0.25-2 μ/kg/min

Abbreviations: A, arterial; NO, nitric oxide; V, venous.