Hemodynamic Changes during CAS Cause Half of the Complications: How Can They Be Prevented?

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Hemodynamic instability is well recognized in patients after carotid endarterectomy (CEA). With CEA, postoperative hypertension has been associated with stroke or death. Studies of hemodynamic alterations associated with CEA suggest that bradycardia is a frequent but benign finding, and it has been associated with hypotension.Carotid angioplasty and stenting (CAS) is currently being investigated as a promising alternative to percutaneous therapy, particularly suitable for patients at high risk for CEA. Despite favorable results from initial series, hemodynamic instability may complicate the carotid stent procedure. Hemodynamic instability is reported in the literature to occur in up to 30%.
Hemodynamic changes during CAS can be caused by two -mechanisms:

1. Hemodynamic instability owing to triggering of the baroreceptors of the carotid sinus
2. Release of catecholamines

Different techniques have been used to prevent hemodynamic instability. Prophylactic use of atropine (a muscarinic antagonist that abolishes the effect of acetylcholine) causes an effect on the sympathic system resulting in a mild tachycardia. Unfortunately, atropine does not prevent hypotension.

In a retrospective study, the effect of the prophylactic use of another vasoactive drug during CAS is investigated. Isoprenaline is a b-adrenergic-agonist. It stimulates both the heart rate and contractility by stimulating b1-receptors in the heart. Stimulation of b1-receptors causes blood vessel dilatation and relaxation of smooth muscle in organs. In this study, we compared retrospectively a group of patients who underwent CAS with atropine on indication, with another group of patients who underwent CAS under prophylactic isoprenaline with regard to the systolic blood pressure and heart rate. The use of isoprenaline resulted in a relevant decrease in the occurrence of bradycardia, asystole and hypotension.

In another study, we evaluated the patterns of catecholamine release in CAS compared with CEA. Patterns of adrenaline and noradrenaline release were significantly different in patients undergoing CAS and CEA with much higher and more variable surges of adrenaline and noradrenaline occurring in CEA patients. Adrenaline and noradrenaline levels (expressed as ratio from baseline) increased significantly following carotid artery clamping in patients undergoing CEA (noradrenaline: pre-clamp 1.54 ± 0.36; 24 hours post-unclamp 8.38 ± 5.5. $p < .001$; adrenaline: pre-clamp 1.12 ± 0.14; 60 minutes post-unclamp 17.59 ± 5.3, $p < .001$). Conversely, in patients undergoing CAS, catecholamine levels remained unchanged (noradrenaline: pre-clamp 0.96 + 0.06; 24 hours post-unclamp 0.92 + 0.12, p = NS; adrenaline: pre-clamp 0.83 + 0.09; 60 minutes post-unclamp 0.56 + 0.12, p =NS).

Clinical results of our institutional series were assessed. In a series of 121 patients two major strokes and five minor strokes occurred. The majority of complications were related to hemodynamic instability during the procedure.

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