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Post-carotid Endarterectomy Hypertension. Part 2: Association with Peri-operative Clinical, Anaesthetic, and Transcranial Doppler Derived Parameters

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WHAT THIS PAPER ADDS

This paper examines the influence of intra-operative patient, transcranial Doppler ultrasound and anaesthetic variables on the incidence of post-carotid endarterectomy (CEA) hypertension. The data show that patients requiring treatment for post-CEA hypertension had significantly higher pre-induction systolic blood pressure, the greatest decrease in blood pressure on induction of anaesthesia, a lower need for ephedrine to maintain blood pressure after induction of anaesthesia, and higher post-operative pain scores. Logistic regression analysis of all of the pre- and intra-operative data identified impaired baroreceptor sensitivity and pre-induction systolic blood pressure to be independent predictors of post-endarterectomy hypertension. Other patient, transcranial Doppler ultrasound, and anaesthetic variables were not associated with post-CEA hypertension.

Objective/Background: The first paper in this series observed that pre-operative baroreceptor dysfunction and poorly controlled hypertension were independently predictive for identifying patients who went on to require treatment for post-endarterectomy hypertension (PEH). The second paper examines the influence of intra-operative patient, transcranial Doppler (TCD) ultrasound, and anaesthetic variables on the incidence of PEH.
Methods: In total, 106 patients underwent carotid endarterectomy (CEA) under general anaesthesia. Systolic blood pressure (SBP) changes, anaesthetic and vasoactive agents, analgesia, and post-operative pain scores, as well as TCD derived changes in middle cerebral artery (MCA) velocity during surgery were recorded. Patients who met pre-existing unit criteria for treating PEH after CEA (SBP $>$ 170 mmHg without symptoms or SBP $>$ 160 mmHg with headache/seizure/neurological deficit) were treated according to an established and validated protocol.
Results: In total, 40/106 patients (38%) required treatment for PEH following CEA (26 in theatre recovery [25%], 27 back on the vascular surgery ward [25%]), whereas seven (7%) had SBP surges $>$ 200 mmHg on the ward. Patients requiring treatment for PEH had significantly higher pre-induction SBP (174 $\pm$ 21 mmHg vs. 153 $\pm$ 21 mmHg; $p < .001$), the greatest decreases in SBP after induction of anaesthesia (median decrease 100 $\pm$ 32 mmHg vs. 83 $\pm$ 24 mmHg; $p = .01$) and were significantly more likely to experience moderate/severe pain scores post-operatively ($p = .003$). Logistic regression analysis of the pre- and intra-operative data revealed that higher pre-induction mean SBP and lower pre-operative (impaired) BRS were the only independent predictors of PEH.
Conclusion: This analysis of intra-operative variables has demonstrated that patients with poorly controlled and/or labile hypertension at induction of general anaesthesia were those at greatest risk of requiring treatment for PEH in the post-operative period after CEA. No other variables, including use of vasopressors, treatment of hypotension, anaesthetic agents, or changes in MCA velocity after clamp release and restoration of flow were

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able to predict who might go on to require treatment for PEH. Identification of at-risk individuals and aggressive blood pressure control in the post-operative period remains the mainstay of treatment.

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INTRODUCTION

Carotid endarterectomy (CEA) is beneficial in selected patients with symptomatic and asymptomatic carotid stenoses, with maximum benefit being observed in those with recent onset symptoms.[1–4] However, the paradox remains that CEA is associated with a small but significant risk of procedural stroke. Previous work from this unit has shown that a targeted strategy of intra-operative monitoring, quality control assessment, and dual antiplatelet therapy was associated with a significant and sustained reduction in both intra-operative stroke and stroke due to post-operative carotid thrombosis.[5] However, there remained a subgroup of patients who continued to suffer stroke due to hyperperfusion syndrome (HS) or intracranial haemorrhage (ICH), while also being at risk of fatal cardiac events. One factor common to all three of these important complications is post-CEA hypertension (PEH).[6,7]

A project was undertaken to evaluate a wide range of pre-operative and/or intra-operative clinical, physiological, and anaesthetic parameters that might be associated with the development of PEH. The first paper evaluated pre-operative imaging and clinical parameters and observed that baroreceptor dysfunction and pre-operative hypertension (especially poorly controlled hypertension)[8] were significantly predictive of patients who were at highest risk of going on to require treatment of PEH in the post-operative period. This second paper examines the influence of intra-operative patient, transcranial Doppler (TCD) monitoring, and anaesthetic variables on the incidence of PEH requiring treatment in the post-operative period.

METHODS

The overall design and methodology of this project has been detailed in a previous paper.[8] The Leicestershire Research and Ethics Committee approved this study and 108 patients gave informed consent between October 2010 and January 2012. The current paper specifically deals with intra-operative variables and their ability to predict which patients required treatment for PEH in the post-operative period. In summary, all patients underwent CEA under general anaesthesia (GA), using routine thin walled polyester patching, routine shunting (Pruitt-Inahara), systemic heparinisation (5000 U unfractionated heparin) without protamine reversal, routine proximal and distal intimal tacking sutures, and subcutaneous infiltration of 20 mL 1% lidocaine with adrenaline immediately before the skin incision was made. Intra-operative TCD monitoring was undertaken in all patients with an accessible temporal window and all patients underwent completion angioscopy before restoration of flow.[5] Additional cervical plexus blockade prior to commencing surgery was at the discretion of the anaesthetist but was not routine practice in all patients. The use of vasoactive medications intra-operatively was also at the discretion of the anaesthetist, but the agreed aim was to maintain intra-operative blood pressure (BP) within 20% of the pre-operative BP,[7] to always treat systolic BP (SBP) $<$ 100 mmHg and/or to maintain mean middle cerebral artery velocity (MCAV) on TCD $>$ 15 cm/s.[9] Ephedrine was usually the first line vasoactive agent (3–6 mg boluses), especially if the heart rate was low, and was preferentially used after induction and intubation to maintain BP before the start of surgery. Phenylephrine (50–100 μg boluses or continuous infusion) was mainly used to maintain intra-operative BP. Angiotensin converting enzyme inhibitors were not routinely withheld prior to surgery.

Non-invasive BP was measured after entry into the anaesthetic room at 5 min intervals until a radial arterial catheter was sited under local anaesthesia. Invasive arterial BP was then recorded throughout the procedure. During induction of anaesthesia the maximum decrease in BP was recorded and the intra-operative BP was then documented at 10 min intervals, until closure of the neck wound. All patients were extubated in the operating theatre before transfer to the post-anaesthesia care unit (PACU), once the patient was awake and initial BP stabilised.

The type and dose of anaesthetic agents used, the use of supplementary superficial cervical plexus blockade by the anaesthetist, intra-operative analgesics, vasoactive medications (doses and volumes), carotid clamp times, shunt time, overall duration of surgery, and post-operative pain score in the PACU—as recorded by the recovery nurse—(mild/moderate/severe) were documented. In addition, continuous TCD monitoring of middle cerebral artery velocities (MCAV) was undertaken in patients with an accessible cranial window (see below).

TCD measurements of mean and peak MCAV and the pulsatility index (PI) were recorded at regular time points throughout the procedure. PI reflects the difference between the peak systolic and minimum diastolic velocities divided by the mean velocity during the cardiac cycle. The time points used for specific TCD analyses were similar to those used in previous studies (Table 1),[10–12] and were: (i) pre-clamp (V1), immediately before shunt removal, and re-clamping of the ICA; (ii) 1 minute post-clamp (V2), 1 minute after restoration of flow (ROF) after completion of the endarterectomy; and (iii) 10 minute post-clamp (V3), 10 minutes post-ROF. Using these measurements, the percentage change in mean MCAV and peak MCAV and PI were calculated for 1 and 10 minutes post ROF. A $>$ 100% increase in mean MCAV, peak MCAV, or PI after de-clamping

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