Prevention of Postoperative Wound Haematomas and Hyperperfusion Following Carotid Endarterectomy


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Objective: to study the incidence of wound haematoma and hyperperfusion following carotid endarterectomy and the effect of changes in perioperative management.

Methods: we undertook a prospective audit of the postoperative outcome of 300 consecutive carotid endarterectomies performed for a symptomatic stenosis of the internal carotid artery, under the care of a single consultant.

Results: audit of the first 100 operations between 1990–93 resulted in 4 changes to clinical practice. These included the use of Dacron instead of vein because of 3 vein patch blowouts, invasive postoperative monitoring of blood pressure, and the use of intravenous beta-blockers to control hypertension, because of 4 hyperperfusion injuries. The use of 10F suction drains was discontinued, because they did not prevent 8 wound haematomas. The results of the second 100 cases between 1994–97 and the third 100 cases between 1998–2000 confirmed no further hyperperfusion injuries or patch blowouts (p = 0.01 and 0.04 respectively). Larger 14F suction drains were reintroduced for the third series because of thirteen haematomas in the second series (p = 0.09). Only 4 haematomas occurred in the third series (p = 0.05). The need for beta-blockers fell in the third series due to the introduction of local anaesthesia (p = 0.0001).

Conclusion: the use of Dacron patches and postoperative control of hypertension has reduced the incidence of haemorrhage and hyperperfusion after carotid endarterectomy. Larger suction drains may also help.

Key Words: Carotid endarterectomy; Haemorrhage; Hyperperfusion; Beta-blockers; Patches; Drains.

Introduction

The European Carotid Surgery Trial (ECST) and the North American Symptomatic Carotid Endarterectomy Trial (NASCET) have both shown that carotid endarterectomy (CEA) confers a six to ten-fold reduction in the long-term risk of stroke, compared to best medical therapy alone, for symptomatic patients with a severe (70–99%) stenosis of the internal carotid artery.1,2 However, the advantage of CEA over medical treatment is dependent upon a perioperative disabling stroke and death rate of <6%. A recent national audit in the U.K., by the Vascular Surgical Society,3 reported an encouraging perioperative stroke rate of 3%. Although stroke and death seem important outcome measures, other complications including haemorrhage contribute to the morbidity of the procedure.

Methods

This study was undertaken to establish the incidence of postoperative complications following carotid endarterectomy and the effect of changes in management. Since 1990, we have maintained a prospective audit of all patients undergoing CEA, under the care of a single consultant surgeon (JDB). The information recorded included demographic details, referral source, presenting symptoms, associated medical conditions, preoperative medication, clinical examination findings, preoperative investigations, operative details, postoperative complications and outcome at the time of discharge. A neurologist reviewed all patients preoperatively and postoperatively. The indication for surgery was a symptomatic internal carotid artery stenosis >70%. Nine patients with stenoses between 50 and 70% were included in the first series before the NASCET and ECST trials were reported. Three audit cycles were undertaken on each consecutive series of 100 patients.

The demographic details and risk factors for each series are summarised in Table 1. Comparison of the demographic details of the three groups showed few
Table 1. Demography and risk factors.

<table>
<thead>
<tr>
<th></th>
<th>1st 100</th>
<th>2nd 100</th>
<th>3rd 100</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>73</td>
<td>64</td>
<td>59</td>
<td>196</td>
</tr>
<tr>
<td>Median age (range)</td>
<td>64 (45–78)</td>
<td>68 (34–82)</td>
<td>69 (48–93)</td>
<td>67 (34–93)</td>
</tr>
<tr>
<td>Median stenosis (range)</td>
<td>79% (50–99)</td>
<td>82% (79–99)</td>
<td>85% (70–99)</td>
<td>82% (50–99)</td>
</tr>
<tr>
<td>Contralateral occlusion</td>
<td>7</td>
<td>5</td>
<td>8</td>
<td>20 (7%)</td>
</tr>
<tr>
<td>Previous stroke</td>
<td>28</td>
<td>34</td>
<td>37</td>
<td>99 (33%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>53</td>
<td>61</td>
<td>55</td>
<td>169 (56%)</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>41</td>
<td>40</td>
<td>34</td>
<td>115 (38%)</td>
</tr>
</tbody>
</table>

Table 2. Complications.

<table>
<thead>
<tr>
<th></th>
<th>1st 100</th>
<th>2nd 100</th>
<th>3rd 100</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patch rupture*</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>3 (1%)</td>
</tr>
<tr>
<td>Haematoma (explored)†</td>
<td>8 (2)</td>
<td>13 (4)</td>
<td>4 (2)</td>
<td>25 (8.3%)</td>
</tr>
<tr>
<td>Hyperperfusion injury‡</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>4 (1.3%)</td>
</tr>
<tr>
<td>Disabling stroke§</td>
<td>6</td>
<td>3</td>
<td>1</td>
<td>10 (3.3%)</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>5 (1.6%)</td>
</tr>
<tr>
<td>Death</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>7 (2.3%)</td>
</tr>
<tr>
<td>Disabling stroke and death</td>
<td>7</td>
<td>4</td>
<td>2</td>
<td>13 (4.3%)</td>
</tr>
</tbody>
</table>

* 1st 100 to 2nd and 3rd 100 \( p = 0.04 \).
† 1st 100 to 2nd 100 \( p = 0.09 \), 2nd 100 to 3rd 100 \( p = 0.05 \).
‡ 1st 100 to 2nd and 3rd 100 \( p = 0.01 \).
§ 1st 100 to 2nd 100 \( p = 0.16 \), 2nd 100 to 3rd 100 \( p = 0.25 \).

differences apart from an increasing number of women, and an increasing number of strokes as the indication for surgery.

We used the Fisher Exact Test rather than Chi-Square because of the small numbers (i.e. less than 5) in some of the cells.

Results

The first 100 procedures between 1990–93 were all performed under general anaesthetic by the consultant (JDB) or a supervised registrar, using transcranial Doppler monitoring, shunts and vein patches for all cases. Pruitt Inahara or Javid shunts were used as part of a randomised study of shunt blood flow. All patients received 5000 units heparin IV during the operation and a 10F suction drain was inserted at the end of the procedure. Three patients awoke with a disabling ischaemic stroke (1 new, 2 extensions) and two with a minor stroke that had resolved by the time of discharge. In the postoperative period, hyperperfusion syndrome occurred in three patients (two of whom developed disabling haemorrhagic strokes), and cerebral oedema (headache, vomiting and photophobia) in the other. CT scanning confirmed the presence of cerebral oedema in all four cases.

Three vein patch ruptures occurred between three and five days after surgery. The origin of the patches that ruptured were arm vein (\( n = 1 \)), great saphenous vein from the ankle (\( n = 1 \)) and great saphenous vein from the thigh (\( n = 1 \)). All patients required emergency endotracheal intubation, surgical exploration and replacement of the vein with a synthetic patch. Two of the three patients recovered without further incident, but one suffered a disabling ischaemic stroke. There were eight wound haematomas severe enough to cause a delay in discharge, despite the presence of the suction drain. Two of these required surgical evacuation. Three deaths occurred, two following strokes and one following a myocardial infarction.

Four changes were instigated after this first audit. Thin-walled, collagen impregnated, Dacron patches replaced the vein patches. All patients were transferred directly from theatre to a High Dependency Unit, and remained there for invasive blood pressure monitoring for at least 24 h. Hypertension (systolic pressure >10% above preoperative level) was treated with an intra-
venous infusion of the short-acting beta-blocker “Esmolol”, as this had been reported to reduce the risk of hyperperfusion injury. Patients with persistent postoperative hypertension were treated with oral beta-blockers. The use of 10F suction drains was discontinued, as a randomised trial of vacuum drainage of groin wounds after vascular surgery had shown no benefit.

The next 100 procedures were undertaken between 1994–97 (Table 2). Fifty-three patients required IV beta-blockers for postoperative hypertension. There were no patch ruptures or hyperperfusion injuries in the second or third series (p = 0.04 and 0.01 respectively), and the number of disabling strokes fell to three (p = 0.16). Two of these developed intraoperatively (1 new ischaemic stroke and 1 extension of an existing deficit), and one following a postoperative wound haematoma. One patient developed a minor stroke intraoperatively, with full recovery. The number of wound haematomas rose to thirteen (p = 0.08), four of which required surgical evacuation and one patient developed a disabling ischaemic stroke due to hypoxia caused by difficulty in re-intubation. Two deaths occurred, one following a stroke and one from a myocardial infarction, resulting in a death and disabling stroke rate of 4%.

The last 100 operations were undertaken between 1998–2000 (Table 2). Forty-six procedures were performed under local anaesthesia, with awake monitoring, resulting in less use of transcranial Doppler monitoring and fewer shunts (42% and 54% respectively). The indication for a shunt, when employing local anaesthesia, was the onset of a neurological deficit or severe hypertension (systolic >200 mmHg) due to reduced cerebral perfusion. Larger, 14F suction drains were used to counter the continuing problem of wound haematomas. Only twenty-seven patients required IV beta-blockers for postoperative hypertension (p = 0.0001). There was one disabling intraoperative ischaemic stroke (p = 0.24), two minor strokes with full recovery and two deaths, one from the stroke and one from a myocardial infarction, resulting in a death and disabling stroke rate of 2%. Four haematomas occurred, two requiring drainage without any untoward effects (p = 0.05).

**Discussion**

We use patch closure for all patients undergoing CEA. The theoretical advantages of patch closure include a lower incidence of perioperative stroke due to carotid artery thrombosis and a lower incidence of significant restenosis. A meta-analysis of randomised trials suggests that patching reduces the risk of postoperative occlusion and restenosis. Carotid vein patch rupture seems an infrequent but widely reported phenomenon with a reported incidence of 0.5% to 4%. This may be a reflection of its use in a high-pressure, large-calibre artery. A prospective study by Archie et al., suggests that rupture can be reduced by rejecting small calibre veins <3.5 mm diameter and ensuring that the reconstructed carotid artery does not exceed 13 mm in diameter. Rupture causes catastrophic neck swelling, resulting in compromise of the patient’s airway due to direct pressure and laryngeal oedema and necessitating immediate return to theatre. We found no relationship with the site of vein harvest as one rupture each occurred with the use of arm, ankle and thigh veins. In the light of our experience with vein patch ruptures in the first 100 patients, we switched to synthetic Dacron patches which avoided the problem.

Hyperperfusion syndrome is characterised by poorly controlled postoperative hypertension, cerebral oedema, convulsions and cerebral haemorrhage, caused by increased perfusion of the brain after carotid endarterectomy. It is more likely to occur in patients with high grade carotid stenoses and contralateral occlusions, due to defective cerebral autoregulation. Careful postoperative blood pressure monitoring and control of hypertension with intravenous beta-blockers has been reported to reduce the risk. We encountered no further episodes of hyperperfusion after adopting this strategy. The use of local anaesthesia for almost half of the last 100 patients appears beneficial in this respect as local anaesthesia may improve cerebral autoregulation and postoperative hypertension. This may explain the significant reduction in the need for IV beta-blockers in this group.

We routinely used small caliber 10F suction drains in the first series of patients but they did not appear to prevent wound haematomas. Surgery in a vascular region such as the neck, together with the use of heparin, seems likely to risk bleeding with potentially disastrous consequences. Non-reversal of heparin has been reported to increase the risk of haematoma formation. However, a randomised trial of 64 patients by Fearn et al. showed that although protamine reversal after carotid endarterectomy reduced wound drainage volumes, it significantly increased the risk of carotid thrombosis. Another retrospective study agreed with these findings. Small calibre suction drains were discontinued in the second series of patients as a
randomised trial of such drains had shown lack of benefit for groin wounds following vascular surgery. However, wound haematomas increased in the second series, resulting in one ischaemic stroke due to hypoxia, and therefore larger 14F suction drains were re-introduced for the last 100 patients. This seemed to reduce, but not abolish the incidence of wound haematoma. Fibrin sealant has been shown to reduce suture line bleeding during carotid endarterectomy, when PTFE patches are used. However, in our experience, postoperative bleeding, requiring re-exploration, usually arises from the wound edges rather than the Dacron patch.

In conclusion, a prospective audit has enabled us to identify the incidence of haemorrhage and hyperperfusion following carotid endarterectomy. Repeated audit suggests that changes to clinical practice, i.e. the use of Dacron patches, and postoperative control of hypertension (using beta-blockers and/or local anaesthesia), have significantly reduced the incidence of such complications. Larger suction drains may also help.

References

1 European Carotid Surgery Trialists Collaborative Group MRC. European Carotid Surgery Trial: Interim results for symptomatic patients with severe (70–99%) or mild (0–29%) carotid stenosis. Lancet 1991; 337: 1235–1243.

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