Brachial plexus injury is an unusual and under-recognised complication of coronary artery bypass grafting especially when internal mammary artery harvesting takes place. It is believed to be due to sternal retraction resulting in compression of the brachial plexus. Although the majority of cases are transient, there are cases where the injury is permanent and may have severe implications as illustrated in the accompanying case history.

Neurological complications after coronary artery bypass grafting (CABG) are common (see the illustrative case history in box 1). In a study of 312 patients by Shaw et al., 61% of patients showed evidence of neurological complications with detailed investigation, although serious morbidity was rare with “severe disability” (defined as incapable of leading an independent existence) only occurring in four patients (1.3%). The commonest neurological complications were primitive reflexes—that is, development of palomental reflex, pout reflex or grasp reflex (39%), ophthalmological abnormalities (25%), and disorders of the peripheral nervous system (12%). Seven percent (21 patients) of the total number of patients had brachial plexopathy. Death as a result of hypoxic brain injury accounted for 0.3%, whereas definite stroke accounted for 5% of neurological complications. Mild cognitive impairment and memory loss are also frequent complications of cardiac surgery but these tend to be subtle. There is an extensive literature on central nervous complications which is beyond the scope of this article.

Only brachial plexus injury will be discussed in detail in this article. Other nerves that are not infrequently injured during CABG include the phrenic, sympathetic, ulnar, and radial nerves. More rarely, injury to the recurrent laryngeal nerve may also occur. While phrenic nerve palsy is relatively harmless if unilateral, bilateral phrenic nerve palsy has significant impact on the patient’s recovery and also morbidity status postoperatively. This is particularly so if the patient also suffers from respiratory disorders whereby tidal volumes are already reduced such as chronic obstructive airways disease. Phrenic nerve injury is thought to be due to hypothermia induced by ice slush during cardioïglia and hence may be preventable by phrenic nerve insulation intraoperatively. Injury can also be avoided by warm cardioplegia. Injury to the sympathetic nerve resulting in Horner’s syndrome is a further recognised complication of CABG. Moreover, radial nerve injury may also be caused by sternal retraction during internal mammary artery harvesting.

INCIDENCE OF BRACHIAL PLEXOPATHY

Brachial plexus injury after CABG is not an infrequent complication but it is under-diagnosed and under-reported as it is often transient. The risk of brachial plexus injury in this context has been reported to be between 5.5% and 10% (table 1). It is thought that the injury is a consequence of the sternal split and retraction transmitting compressive pressure to the nerve roots. Brachial plexus injury is also more common if internal mammary artery preparation is required. In a prospective study of 1000 consecutive patients, Vahl et al. reported that CABG was associated with a 1% risk of brachial plexus injury if no internal mammary artery preparation was involved, with the risk rising to over 10% otherwise. This prevalence occurs despite preventive measures used by the investigators to minimise this particular complication such as using a lower position and least possible opening of the sternal retractor. Bearing in mind that the use of internal mammary arteries as conduits in CABG is increasing, one can only surmise that this complication will become more common and patients will need to be made aware at the time consent is obtained, even more so if their livelihood depends on it. More importantly, steps need to be taken to minimise this complication.

MECHANISMS OF BRACHIAL PLEXOPATHY

Several mechanisms have been proposed as the cause of brachial plexus injury during CABG. These include hyperabduction of the arms, direct trauma during needle catheterisation of the internal jugular vein, and traction and compression during sternal retraction. During harvesting of the internal mammary artery, asymmetrical traction of the sternal halves appears to be associated with a higher risk of brachial plexopathy. Other factors associated with a higher risk of brachial plexopathy are diabetes mellitus and hypothermia during surgery, with diabetes also being associated with a longer duration of disability. The predisposition of the lower roots (C7–T1) to be injured in comparison with the upper roots lends support to the theory
that sternal retraction is the cause, as the lower roots would be compressed most in the retroclavicular space.

The utilisation of somatosensory evoked potential (SEP) monitoring of bilateral ulnar and median nerves intraoperatively may be used to predict peripheral nerve injury during cardiac surgery. In a study involving 30 patients undergoing cardiac surgery, Hickey et al reported a 13% incidence of SEP changes during internal jugular venous cannulation. However, the changes were intermittent and transient, and none of the patients eventually suffered any neurological deficit. Significant SEP changes were observed in 70% of patients with the use of the Canadian or Favoloro retractors but the majority reverted to normal intraoperatively. In five patients, however, the changes persisted to the end of the operation and these five patients subsequently demonstrated neurological deficits. Another study by Jellish et al showed large decreases in SEP on insertion of Rultract and Pittman retractors in 85% and 68.75% of patients respectively. Although this improved after removal, the amplitudes did not return to baseline. Only mild decreases were noted in their control group where Cooley retractors were used in patients not undergoing internal mammary artery harvesting. They concluded that SEP was a sensitive marker to detect nerve injury.

In a further study of 36 patients by Baisden et al, the removal of the uppermost blades of the conventional Ankeney retractor was associated with a reduced incidence of occult rib fractures (evident on bone scans but not chest radiographs) overall. In particular, occult posterior first rib fractures were found to be associated with brachial plexus injury. These findings would be consistent with the notion that increased traction and compression by sternal retractors have a role in brachial plexus injury. However, the number of patients in the published studies were relatively small. With the advent of thoracoscopic harvesting of the internal mammary artery and minimally invasive CABG (that is, minithoracotomy), sternal retraction may be minimised and perhaps brachial plexus injury reduced. Totally endoscopic CABG would be something to look forward to in the future.

Finally, arm positioning during internal mammary artery harvest may also influence brachial plexus injury. Indeed, some suggest that the “hands-up” position may actually reduce the occurrence compared with the usual “arms-at-the-side” position. It is hypothesised that the “hands-up” position may in fact reduce brachial plexus stress during asymmetric sternal retraction. However, in a study of 80 patients, Jellish et al showed that the “hands-up” position offered little protection against brachial plexus stress based on SEP. An interesting finding in that study was that although patients in both groups suffered with brachial plexus injury, those who also show ulnar nerve problems were in the “arms-at-the-side” group only. This suggests that the “arms-at-the-side” position may also predispose to ulnar nerve compression but not the “hands-up” position.

**BRACHIAL PLEXOPATHY OR ULNAR NEUROPATHY?**

It is important to distinguish brachial plexopathy from ulnar neuropathy, the main differential diagnosis, as the underlying mechanisms appear to be different as well as the prognosis. In the study by Vahl et al, eight of 27 patients who developed brachial plexus injury had persistent symptoms three months after referral to the cardiothoracic surgeon for CABG to his right coronary artery, which filled retrogradely from the left coronary artery. The left ventricular function was good. An elective mammary artery harvesting.

### Table 1: Studies of brachial plexus injury and CABG

<table>
<thead>
<tr>
<th>Investigators</th>
<th>Study</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vahl et al23</td>
<td>n=1000; prospective study of 1000 consecutive patients</td>
<td>Brachial plexus injury occurred in &lt;1% of patients not requiring internal mammary artery preparation with the risk rising to 10.6% if internal mammary artery preparation was required</td>
</tr>
<tr>
<td>Shaw et al8</td>
<td>n=312; prospective study</td>
<td>Brachial plexus damage occurred in 6.7% of patients</td>
</tr>
<tr>
<td>Lederman et al23</td>
<td>n=421; Cleveland clinic study</td>
<td>Brachial plexus injury occurred in 5.5% of patients undergoing CABG</td>
</tr>
<tr>
<td>Baisden et al23</td>
<td>n=36 (small patient numbers)</td>
<td>Removal of the uppermost blades of the Ankeney retractor reduced risk of posterior 1st rib fractures and brachial plexus injury</td>
</tr>
</tbody>
</table>
16 patients developed brachial plexus injury with only one possible method to reduce brachial plexus injury postoperatively. The cause of postoperative ulnar nerve palsy is not one that is readily identifiable but is thought to be a result of local nerve compression/trauma in the posterior con- dylar groove. This could be due to malposition of the arm, either intraoperatively or postoperatively when the patient is immobile. It has also been suggested that it may be due to prolonged ischaemia of the upper limb during CABG. Its prognosis tends to be less favourable than brachial plexus injury, although these data come from non-cardiac surgery. In one prospective study of 335 patients undergoing median sternotomy for cardiac surgery by Tomlinson et al, 16 patients developed brachial plexus injury with only one patient still symptomatic at the time of discharge. However, they placed all their patients in the “hands-up” position and were careful with sternal retraction. This may have prevented more significant damage, thereby producing a more favourable outcome. Utilisation of nerve conduction studies allows differentiation between ulnar neuropathy and brachial plexus injury quite readily. Accurate diagnosis allows steps to be taken to reduce these complications which may not be consequential as illustrated in this case. Further investigation is required on the effects of reduction of sternal retraction force on SEP and if this will actually prevent brachial plexopathy (table 2).

CONCLUSION
It is important to inform patients of the potential neurological complications that may occur after CABG. Care should be taken to minimise the risk of these complications during surgery and, to that end, the mechanism by which they occur requires further investigation. This is particularly appropriate with the increasing use of internal mammary artery conduits that is associated with a higher incidence of brachial plexus injury. More recently, however, the introduction of thoracoscopic harvesting of the internal mammary artery and minimally invasive CABG obviates the need for excessive sternal retraction and hence, may prevent brachial plexus injury.  

Authors’ affiliations
A Y Chong, G Y H Lip, University Department of Medicine, City Hospital, Birmingham  
C E Clarke, University Department of Neurology, City Hospital, Birmingham  
W R Dimitri, Department of Cardiothoracic Surgery, Walsgrave Hospital, Coventry

REFERENCES

Table 2
Mechanism and prevention of nerve injury

<table>
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<tr>
<th>Nerve injury</th>
<th>Proposed mechanism</th>
<th>Prevention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brachial plexopathy</td>
<td>Traction and compression of nerves during sternal retraction (internal jugular venous cannulation)</td>
<td>Reduction of sternal retraction and at a lower position; may be reduced by thoracoscopic harvesting of the internal mammary artery</td>
</tr>
<tr>
<td>Ulnar neuropathy</td>
<td>Arm position resulting in compression of the ulnar nerve</td>
<td>Avoidance of local compression either by arm position or other means of protection of the humeral groove</td>
</tr>
<tr>
<td>Phrenic nerve palsy</td>
<td>Hypothenar from ice slush for cardioplegia (perhaps also stretching)</td>
<td>Phrenic nerve insulation intraoperatively and warm cardioplegia</td>
</tr>
</tbody>
</table>

31 Miller RG, Camp PE. Postoperative ulnar neuropathy. JAMA 1979;242:1636–9  
34 Miller RG, Camp PE. Postoperative ulnar neuropathy. JAMA 1979;242:1636–9  
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