REVIEW

Brachial plexus injury as an unusual complication of coronary artery bypass graft surgery

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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.

drome is a further recognised complication of CABG.^{13 14} Moreover, radial nerve injury may also be caused by sternal retraction during internal mammary artery harvesting.^{15 16}

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.^{13 16–19} The risk of brachial plexus injury in this context has been reported to be between 5.5% and 10% (table 1).^{1 13 20} 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 with the sternal retractor.¹³ 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.^{19 20} During harvesting of the internal mammary artery, asymmetrical traction of the sternal halves appears to be associated with a higher risk of brachial plexopathy.¹⁸ 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.^{17 20} The predisposition of the lower roots (C7–T1) to be injured in comparison with the upper roots lends support to the theory

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 palmomental 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.¹ 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 complication 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,² sympathetic, ulnar, and radial nerves.^{3–7} More rarely, injury to the recurrent laryngeal nerve may also occur.⁸ 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.⁹ This is particularly so if the patient also suffers from respiratory disorders whereby tidal volumes are already reduced such as chronic obstructive airways disease.¹⁰ Phrenic nerve injury is thought to be due to hypothermia induced by ice slush during cardioplegia and hence may be preventable by phrenic nerve insulation intraoperatively^{6 7}; injury can also be avoided by warm cardioplegia.^{11 12} Injury to the sympathetic nerve resulting in Horner's syn-

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Box 1: Illustrative case history

A 61 year old man first presented with an inferior myocardial infarction in 1998, which was treated with streptokinase. He had a total cholesterol level of 5.2 mmol/l on admission. There was no history of hypertension, diabetes, or prior coronary artery disease. He had a peak creatine kinase rise of 3259 IU/l and was discharged from hospital a week later without complication. An exercise tolerance test performed a month later was clinically positive with less than 1 mm ST depression in the inferolateral leads. Coronary angiography showed a virtually normal left coronary system but an occluded proximal right coronary artery, which filled retrogradely from the left coronary artery. The left ventricular function was good. An elective percutaneous transluminal coronary angioplasty of the right coronary artery was unsuccessful and he was therefore referred to the cardiothoracic surgeon for CABG to his distal right coronary artery.

Nine months after his myocardial infarction, he underwent a right internal mammary artery bypass graft to his distal right coronary artery, off pump. His postoperative course was complicated by a chest infection on the third day which responded to oral antibiotics. He complained of left arm weakness in his early postoperative course. On review two months later, he was doing very well from a cardiovascular aspect; but his left arm weakness persisted and marked muscle wasting was noted in the small muscles of his left hand and medial aspect of his left forearm. The distribution of muscle weakness was clinically consistent with either ulnar neuropathy or brachial plexopathy. There was no sensory loss detected clinically. He was no longer able to work as a joiner.

Nerve conduction studies were consistent with a proximal high brachial plexus lesion located at the root level of C7, C8, and T1 on the left. Profuse denervation was noted in the left first dorsal interosseous, abductor pollicis brevis, and patchy denervation in the long extensors of the forearm and triceps muscles. Although there was no sensory loss detected clinically, neurophysiology revealed at least a 50% reduction in the size of the sensory nerve action potential in the corresponding dermatomes. Subsequent follow up at eight and 14 months after surgery showed no improvement in his clinical condition.

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.²¹ 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.²² 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.²³ 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 minimised²⁴⁻²⁸ 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.²⁹

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

Table 1 Studies of brachial plexus injury and CABG		
Investigators	Study	Comment
Vahl <i>et al</i> ¹³	n=1000; prospective study of 1000 consecutive patients	Brachial plexus injury occurred in <1% of patients not requiring internal mammary artery preparation with the risk rising to 10.6% if internal mammary artery preparation was required
Shaw <i>et al</i> ¹	n=312; prospective study	Brachial plexus damage occurred in 6.7% of patients
Lederman <i>et al</i> ²⁰	n=421; Cleveland clinic study	Brachial plexus injury occurred in 5.5% of patients undergoing CABG
Baisden <i>et al</i> ²³	n=36 (small patient numbers)	Removal of the uppermost blades of the Ankeney retractor reduced risk of posterior 1st rib fractures and brachial plexus injury

Table 2 Mechanism and prevention of nerve injury

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

postoperatively.¹³ 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 condylar 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.³⁰ Its prognosis tends to be less favourable than brachial plexus injury,³¹ although these data come from non-cardiac surgery.^{32–34} 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.³⁵ 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 inconsequential 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 which 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^{24–28} and hence, may prevent brachial plexus injury.

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