Peripheral Nerve Injuries During Cardiac Surgery: Risk Factors, Diagnosis, Prognosis, and Prevention

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Neuropathies involving the brachial plexus, phrenic, recurrent laryngeal, and saphenous nerves, as well as the sympathetic chain are complications of cardiac surgery. The reported frequency of nerve injuries varies from 1.5% to 24% for the brachial plexus and 10% to 60% for the phrenic nerve (1-5). Neuropathies of the recurrent laryngeal, lower limb (saphenous) nerves, and the sympathetic chain are less frequent.

This review will focus on the risk factors, diagnostic modalities, prognosis, and preventive measures that can be used to reduce the severity and frequency of neuropathies after cardiac surgery. Particular attention will be directed to discussion of brachial plexus and phrenic nerve injury.

Brachial Plexus Anatomy

The brachial plexus is formed by the anterior rami of the fifth to eighth cervical and the first thoracic spinal nerves (6). After exiting from the intervertebral foramina, the roots of the brachial plexus pass inferiorly between the middle and anterior scalene muscles combining to form the superior, middle, and inferior trunks. In this position, the brachial plexus is invested in a fascial covering that represents an extension of the prevertebral fascia. As the three trunks pass over the first rib and under the clavicle, they split into divisions, recombining to form the cords that pass down through the axilla.

There are three principal anatomical features that make the brachial plexus susceptible to neuropathy. First, its superficial location makes it susceptible to direct damage. Second, the nerve roots of the brachial plexus are fixed both proximally at their site of origin (the intervertebral foramina) and distally where they are tethered to the investing fascia, muscles, and other tissues. As a result, force applied between these points increases the likelihood of producing a stretch neuropathy (7). Third, the space between the first rib and the clavicle is limited. Thus, fracture and/or displacement of the first rib can directly damage the brachial plexus (7).

Cell bodies in the dorsal root ganglion provide a continuous supply of nutrients distally to the peripheral axons. Any injury that interferes with this supply can damage the nerve. In 1973, Upton and McComas (8) described the “double crush” hypothesis to explain nerve entrapment syndromes. This hypothesis states that two relatively minor nerve lesions, which by themselves are incapable of producing significant nerve damage, can combine to produce a significant lesion. For example, clinically silent ulnar nerve entrapment in the cubital tunnel (present preoperatively) may be converted to an overt ulnar neuropathy by a minor stretch neuropathy to the lower trunk of the brachial plexus during sternal retraction. This hypothesis may hold true for many brachial plexus injuries after cardiac surgery. However, despite the widespread acceptance of this double crush hypothesis by the medical community, serious questions have been raised about this concept because no experimental studies have shown conclusively that double lesions across a nerve cause magnified damage (9-12).

Risk Factors for Brachial Plexus Neuropathy

Sternal Retraction and Internal Mammary Artery Dissection.

Sternal retraction is one of the key factors responsible for brachial plexus neuropathy. As discussed previously, the anatomical features of the brachial plexus render it susceptible to stretch injury. In 1971, Kirsh et al. (7) performed sternal retraction in human cadavers and demonstrated that retraction produced superior rotation of the first rib and pushed the clavicles into the retroclavicular space, thereby stretching the brachial plexus (Figure 1). This stretching may be made worse by subsequent turning of the head to the contralateral side (13).

Figure 1. A, Normal course of the brachial plexus as it passes over the first rib. B, Opening the sternum widely causes superior rotation of the first rib that pushes the clavicles into the retroclavicular space leading to stretching of the brachial plexus. Reprinted with permission (7).
Direct trauma of the brachial plexus can occur by first rib fracture fragments or the associated fracture hematoma directly compressing the nerves (Figure 2) (14,15). Posterior fractures of the first rib can easily remain undiagnosed by routine radiography. The incidence of upper rib fractures after median sternotomy is uncertain. The radiographically reported frequency of first and second rib fractures varies from 4% to 16% (16-18). Greenwald et al. (16) prospectively studied 24 patients undergoing cardiac surgery with pre- and postoperative chest radiography and postoperative bone scans. Bone scans revealed 44 rib fractures, 30 on the left and 14 on the right. In contrast, chest radiography revealed only one rib fracture. On retrospective review of chest radiography, only three new fractures could be identified. Posterior fractures of the first rib were the most common site of fractures. In addition to their potential adverse effects on the brachial plexus, the authors suggested that rib fractures should be included in the differential diagnoses of unexplained postoperative nonincisional chest pain and that bone scans were more sensitive than chest radiography in diagnosing rib fractures.

There are several commonly used sternal retractors. Most of the studies have been conducted with the Cooley and the Ankeney retractor. Several studies have shown that the more caudal placement of the retractors within the sternotomy is associated with a reduced incidence of first rib fractures (1,2,19). The optimal placement of the retractor is controversial.

Vander Salm et al. (14) studied the placement of sternal retractors and first rib fractures in 20 noncardiac patients who had autopsies performed through a median sternotomy. They found that the frequency of first rib fractures was completely eliminated by placing the cephalad blade of the sternal retractor in the fourth intercostal space. In contrast, 11 patients had first rib fractures demonstrated by chest radiography when the cephalad blade was placed in the second intercostal space. These authors suggested that, by placing the retractor in the cephalad position, the upper portion of the sternum is spread less and may result in less pressure transmitted to the first rib with a resultant decreased frequency of first rib fractures.

Tomlinson et al. (19) prospectively studied 335 cardiac surgery patients undergoing median sternotomy both preoperatively and five to seven days postoperatively for evidence of brachial plexus injury. Patients with abnormal neurological findings were subsequently examined at two-day intervals until discharge. Diagnosis was made after detailed sensory and motor evaluation of muscle groups innervated by the brachial plexus. They attributed the decreased frequency of brachial plexus neuropathy (4.8%) on the relative caudal placement of the Cooley-type sternal retractor. Baisden et al. (1) prospectively studied 36 consecutive cardiac surgery patients undergoing median sternotomy. All patients had preoperative and postoperative chest roentgenograms as well as radionuclide bone scans 7 to 14 days after surgery. By removing the uppermost pair of blades from the Ankeney retractor, they were able to reduce the frequency of first rib fractures from 50% to 16%.

Some of the commonly used internal mammary artery (IMA) dissection retractors are the Pittman™ (Minnesota Scientific Instruments Inc., Minneapolis, MN) (Figure 3A), Rultract™ (Rultract Inc., Cleveland, OH) (Figure 3B), and Delacoix-Chevaller™ (Delacoix-Chevaller, Paris, France) (Figure 3C). Jellish et al. (20) prospectively studied the effect of these three asymmetrical sternal retractors on brachial plexus dysfunction using intraoperative somatosensory–evoked potentials (SSEP) in 60 patients undergoing Coronary Artery Bypass Graft (CABG) surgery with IMA harvest. Preoperatively, an investigator blinded to the retractor type performed detailed motor and sensory examination of the brachial plexus. Intraoperatively, SSEPs were also monitored by an investigator blinded to the type of retractor used. In contrast to the patients in the Pittman™ and Rultract™ groups, they demonstrated that fewer patients in the Delacoix-Chevaller™ group had decreases in SSEP amplitudes after retractor.
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Placement suggesting that not all IMA dissection retractors produce similar effects on the brachial plexus. This may be related to the fact that the Pittman™ and the Rultract™ retractors pull the sternal halves laterally while the Delacroix-Chevalier™ pushes the operative sternal half up and the opposite sternal half down, with little lateral traction on the sternum and brachial plexus. Despite decreases in the degree of SSEP amplitude reduction associated with the use of the Delacroix-Chevalier™ retractor, there was no difference in frequency of postoperative brachial plexus neuropathy among the three groups.

In a prospective, randomized study of 44 patients, the same group of investigators (21) further evaluated the effect of the Rultract™ and Pittman™ IMA retractors on brachial plexus SSEPs. They demonstrated that, although both these retractors were associated with large decreases (>50%) in SSEP amplitudes (P < 0.05) as compared with controls, such changes were more severe and more common in the Rultract™ group than in the Pittman™ group (85% vs 69%). Again, despite these findings, no clinical differences in the frequency of brachial plexus neuropathy could be ascertained between the two groups.

The preparation of IMA conduits has been linked to brachial plexus neuropathies. Vahl et al. (22) studied 1000 cardiac surgery patients prospectively and demonstrated that the frequency of neuropathies in patients without IMA grafts was 1% as compared with 10.6% in those receiving IMA grafts. They concluded that IMA dissection requires a wider, more asymmetric sternal opening for adequate visualization thus predisposing to an increase in brachial plexus traction and subsequent neuropathy. The authors suggested that asymmetric retractors should be used with caution. Other investigators have questioned the degree of association between brachial plexus neuropathy and IMA dissection (19,23).

Positioning.

Studies attempting to correlate arm positioning with brachial plexus neuropathy have also produced inconsistent results. Early studies by Jackson and Keats (24) showed that hands-up positioning (defined as abduction of the arm to no more than 90°, anterior flexion of the elbows, and elevation of the elbows 6 inches above the table) was associated with less tension and compression of the brachial plexus. The authors evaluated the stress produced by various positions on the brachial plexus in 15 cadavers. Tension was estimated by observation, and palpation and was graded arbitrarily from 0 to 4. In their prospective study of 335 patients, Tomlinson et al. (19) attributed the low frequency (4.8%) and benign course of brachial plexus neuropathy to this hands-up position. There was no control group in this study. Vander Salm et al. (14) prospectively studied 180 patients with respect to arm positioning during cardiac surgery and brachial plexus neuropathy. The frequency of neurological deficits was greater with the arms by the side (23.5%) than with arms abducted to 90° (14.5%), although this was not statistically significant (P = 0.10).

In a study regarding this issue, Jellish et al. (25) prospectively studied the effect of either hands-up (defined, as arms abducted to no more than 90° and elevation of elbows above the horizontal plane [Figure 4]) or arms by the side positioning on brachial plexus SSEP during asymmetric sternal retraction for IMA harvest in 80 patients. Although their results showed that hands-up positioning was associated with a lower frequency of SSEP amplitude decreases, this did not reflect in a lower frequency of clinical postoperative brachial plexus neuropathies. This study, however, was likely underpowered to definitely resolve this issue.

Central Venous Catheter Placement.

The internal jugular vein is closely related to the brachial plexus. While cannulation of the internal jugular vein may lead to direct needle injury to the brachial plexus, cannulation of the internal jugular vein has not consistently been shown to be associated with an increased frequency of brachial plexus neuropathies. Hanson et al. (26), in a prospective study of cardiac surgery patients ($n = 531$) found that, in 73% of the patients, the side of the brachial plexus injury corresponded with the side of internal jugular vein cannulation. This study was criticized, however, because brachial plexopathies after CABG surgery occur predominantly on the left side while most often the right internal jugular vein is cannulated for central venous access. In contrast to this study by Hanson et al. (26) in which internal jugular vein cannulation was performed after the patients were anesthetized, many central venous catheters are inserted with the patient awake, and thus severe pain would be expected if the needle entered the brachial plexus alerting the physician of possible brachial plexus injury. Additionally, most patients who undergo internal jugular vein cannulation are not having cardiac surgery. The frequency of brachial plexus neuropathies in this noncardiac surgery population is very small. Other investigators have not found the same correlation between internal jugular vein cannulation and brachial plexus neuropathy (2,27,28).

**Patient and Operation Characteristics.**

Advanced age has been linked to brachial plexus neuropathy (14,15), but diabetes mellitus, sex, height, weight, history of smoking, and presence of carotid bruit do not appear to be significant risk factors (26,30). The duration of cardiopulmonary bypass (CPB), aortic cross-clamp times, total anesthesia times, hematocrit during CPB, or type of oxygenator used have also not been associated with increased frequency of brachial plexus neuropathies after CABG surgery (2,26,28,29).

**Diagnosis of Brachial Plexus Neuropathy**

The majority of symptomatic brachial plexus neuropathies after cardiac surgery involve stretching of the lower roots (C8- T1) related to use of sternal retractors and present postoperatively as an ulnar neuropathy (21). Other possible sites where the ulnar nerve can be damaged include the edge of the supinator muscle below the elbow, ulnar groove at the elbow, cubital tunnel, and the medial base of the palm.

Examination of the brachial plexus includes a detailed history of upper extremity pain, paraesthesia, assessment of sensation to pin prick, and examination of motor function of muscle groups innervated by the brachial plexus. Motor evaluation includes elevation of the scapula (C5), adduction (C5-7), abduction (C5-8), lateral and medial (C5-8) rotation of the arm, flexion and supination of the forearm (C5-6), ulnar hand flexion (C7-8 and T1), thumb flexion and opposition (C7-8 and T1), extension of the forearm and phalanges (C6-8), and radial extension of the hand and extension of the thumb (C7-8). Sensory and motor symptoms associated with ulnar neuropathy can differ remarkably depending on the site of nerve damage (31) (Table 1).

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<th>Clinical Signs Associated with Ulnar Neuropathy</th>
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**Warner et al. (32)** extensively studied ulnar neuropathy in 1,129,692 patients retrospectively. The frequency of persistent ulnar neuropathy in noncardiac patients (defined as sensory or motor deficits greater than 3 months duration) was identified in 414 patients; a rate of 1 per 2729 patients (0.04%). In contrast, the frequency of ulnar neuropathy (the most common form of brachial plexus neuropathy) in cardiac surgery patients is much higher (1.9% to 18.3%) (31,32). Ben-David and Stahl (33) retrospectively reviewed patients who presented to a hand clinic with brachial plexopathy over 6 years. They identified 22 cases of documented brachial plexus neuropathy during this time and concluded that a) although controversial, cardiac surgery is associated more with brachial plexus neuropathy to the lower roots as compared with upper and middle root association with noncardiac surgery, b) brachial plexus neuropathy after cardiac surgery results primarily in sensory deficits as compared with relatively painless motor deficits associated with noncardiac surgery, c) brachial plexus neuropathy after cardiac surgery is generally associated with a more rapid recovery as compared with noncardiac surgery. Warner et al. (32) identified male sex, low (<24) and high (>36) body mass index, and longer duration of hospital stays as independent predictors for the development of persistent postoperative ulnar neuropathy in noncardiac patients. Such an association has not been identified with cardiac surgery patients.

Electrophysiologic studies can detect changes in nerve function during the perioperative period, but these changes are very sensitive and often do not reliably predict postoperative neuropathic symptoms. Large, prospective trials demonstrating the importance of electrophysiologic studies in the early diagnosis and prevention of brachial plexus neuropathy are lacking. Measurement of motor and sensory conduction velocities, SSEP's, and electromyography are some of the common modalities used.

Maximum motor and sensory conduction velocities (MMCV/MSCV) can be measured across the arm, elbow, and forearm. Major neuropathies are associated with slowing of nerve conduction. Watson et al. (34) prospectively studied bilateral ulnar nerve MCCV in 20 patients preoperatively, immediately after cardiac surgery, and 4 to 6 weeks postoperatively. They considered slowing of MCCV more than 20% in the elbow relative to the forearm significant for ulnar neuropathy. Preoperatively, 7 patients (35%) had ulnar nerve slowing of more than 20% (as compared with healthy volunteers) and more than 30% in 2 of the 7. Early postoperative examination showed MCCV slowing of more than 20% in 3 of 33 normal preoperative ulnar nerves. However, there were no significant changes in the 7 ulnar nerves in which preoperative slowing was more than 20%. Only one of three cases with significant MCCV slowing showed clinical evidence of ulnar neuropathy. This small study demonstrated that 25% of CABG patients had preoperative MCCV slowing across the ulnar nerve. However, ulnar neuropathy after surgery was uninfluenced by the preoperative status of the ulnar nerve.

In contrast, Hickey et al. (35) prospectively studied intraoperative bilateral SSEP's from median and ulnar nerves in 30 patients undergoing cardiac surgery. They considered a persistent three SD increase in latency and accompanying decrease in amplitude indicative of postoperative brachial plexus neuropathy. In their study, an individual blinded to the results of intraoperative SSEP monitoring evaluated brachial plexus neuropathy in...
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the postoperative period. They demonstrated that central venous cannulation was associated with brief SSEP changes in 13% (4 of 30) of patients. Use of sternal retractors was associated with significant SSEP changes in 70% (21 of 30) patients. In 16 of 21 patients, SSEP changes reverted to normal intraoperatively, and none demonstrated postoperative brachial plexus neuropathies. The remaining 5 of the 21 patients demonstrated postoperative neurological deficits. In all of these five patients, SSEP changes associated with sternal retraction persisted until the end of surgery. The authors suggested that upper extremity SSEP monitoring might be used to predict postoperative peripheral nerve injury. However, Seal et al. (36) found no relationship between transient compared with persistent SSEP changes and symptomatic postoperative brachial plexus neuropathy. Initial results of intraoperative SSEP monitoring are promising, but further larger prospective studies are needed before recommending routine intraoperative SSEP monitoring in an attempt to reduce the frequency of postoperative brachial plexus neuropathies.

Unfortunately, the true value of electrophysiologic monitoring is not fully understood, as the majority of studies have generally had insufficient patient enrollment to obtain adequate statistical power.

**Prognosis of Brachial Plexus Neuropathy**

The overall prognosis of brachial plexus neuropathies after cardiac surgery is generally good. However, prolonged recovery (up to one year), at times with residual symptoms, has been reported (33). In a prospective study of 335 patients, Tomlirson et al. (19) reported severe persistent brachial plexus neuropathy in only 1 of 16 (0.3%) patients considered to have postoperative brachial plexus neuropathy. The remaining 15 patients (4.8%) were symptom--free at the time of discharge. Hanson et al. (26) studied 531 patients prospectively and the clinical diagnosis of brachial plexus neuropathy was made in 5.0% (26 of 531) patients. Similarly only 1.0% (6 of 531) of patients had persistent symptoms for more than four months. Vahl et al. (22), in a prospective study of 1000 patients, showed that 0.8% (8 of 1000) patients had symptoms persisting for more than three months.

**Prevention of Brachial Plexus Neuropathy**

It appears that, despite optimal surgical and anesthetic techniques, brachial plexus neuropathies can still occur. Factors that may reduce the frequency of brachial plexus neuropathies include a) precise midline sternotomy to avoid subsequent asymmetrical sternal traction, b) more caudal placement of the sternal retractor, c) cautious use of asymmetrical sternal retractors, and d) maintenance of neutral head position. None of these steps, however, have consistently reduced the frequency or severity of brachial plexus neuropathies.

**Phrenic Neuropathy During Cardiac Surgery**

**Phrenic Nerve Anatomy**

The phrenic nerve arises from the fourth cervical nerve with additional contributions from the third and fifth cervical nerves. The left phrenic nerve passes cephalad through the thoracic cavity, lying between the lung and the mediastinal aspect of the pleura, in close association with the pericardium. In contrast, the right phrenic nerve is situated more deeply, lying lateral to the right innominate vein and superior vena cava (6).

**Risk Factors for Phrenic Neuropathy Topical Hypothermia.**

In 1957, Cross et al. (37) used topical hypothermia for myocardial protection. Topical hypothermia, alone or in combination with cardioplegia, is a mainstay in cardiac surgery. Phrenic neuropathy, attributable to hypothermia, was described by Scanell et al. (38) who referred to cold--induced phrenic neuropathy as "frost-bitten phrenic." Hypothermia--induced neuropathy is well recognized with temperatures below 17°C. Hypothermia is associated with a steady decline in nerve conduction velocities with complete blockade occurring at 5°C (39,40). On histopathologic examination, cold--injured nerves display a wide spectrum of pathologies ranging from minimal localized demyelination to extensive axonal degeneration (39-41).

Several studies have demonstrated topical hypothermia as the principal cause of postoperative phrenic neuropathy (42-44). Benjamin et al. (44), in both a prospective and retrospective analysis of chest radiographs from cardiac surgery patients, examined the frequency of new left lower lobe infiltrates. They attributed left lower lobe infiltrates to left hemidiaphragm dysfunction caused by left phrenic nerve paresis or paralysis. They demonstrated a 30% (12 of 40) frequency of left lobe atelectatic changes when topical myocardial cooling was not used. In contrast, in cases in which topical cooling with ice was used, the frequency of left lower lobe infiltrates was 43.4% (53 of 122) in the retrospective group and 65% (26 of 40) in the prospective group \( P < 0.001 \). In the prospective group, 69.2% (18 of 26) had left diaphragm paresis or paralysis as demonstrated by diminished motion of the left hemidiaphragm on radiography. In a separate study, Witte et al. (45) demonstrated diminished motion of the left hemidiaphragm with fluoroscopy in nearly 70% of patients after topical cardiac cooling. In the majority of these studies, the left phrenic nerve was involved. The likely explanation for this observation was the close proximity of the left phrenic nerve with the ice-slush filled pericardium. Although topical myocardial cooling can be achieved by the use of either ice slush or cold saline solution, it appears that ice slush is more injurious than cold saline solution (42,43,46-48).

**Internal Mammary Artery Dissection.**

IMA dissection during CABC has been implicated in postoperative phrenic neuropathy. Abd et al. (48) performed phrenic nerve conduction studies (NCS) in CABC surgery patients that were difficult to wean from mechanical ventilation postoperatively. In 90% of the patients, there was concordance between the side of diaphragmatic dysfunction and IMA dissection. The explanation for the association of IMA dissection with phrenic neuropathy could be either direct surgical damage during dissection or phrenic nerve ischemia after ligation of IMA branches supplying the phrenic nerve. However, the association between IMA dissection and phrenic neuropathy is controversial and confounded by the fact that the left IMA is the most frequently used IMA, and the left phrenic nerve is more susceptible to cold injury, as discussed previously. Dimpoulou et al. (42) prospectively investigated 63 patients undergoing CABC surgery and found no association between left IMA dissection and phrenic neuropathy.
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In an adult swine model, O'Brien et al. (49), studied the effects of left IMA dissection on phrenic nerve perfusion. In the first group, the left IMA was dissected completely with ligation of all its branches. This resulted in a 70% reduction in left phrenic nerve perfusion with minimal decrease on the right side. In the second group, left IMA dissection was achieved without sacrificing the pericardiophrenic artery, a small proximal branch that supplies the phrenic nerve. In this group, left phrenic nerve perfusion was decreased by only 20%. The authors concluded that complete left IMA dissection with ligation of the pericardiophrenic artery leads to disruption of the blood supply to the phrenic nerve, which may exaggerate the effect of hypothermic phrenic neuropathy. The origin of the pericardiophrenic artery in humans is quite variable and includes multiple small branches originating from the upper 2 to 3 cm of the IMA.

Other.

Age, diabetes mellitus, left ventricular ejection fraction, CPB time, and the number of coronary grafts appear to have no relationship to postoperative phrenic neuropathy (42,47,50).

Diagnosis of Phrenic Neuropathy

Some of the commonly used modalities to diagnose phrenic neuropathy include chest radiography, fluoroscopy, spirometry, ultrasonography, and transcutaneous phrenic nerve stimulation.

The criteria used to diagnose unilateral phrenic neuropathy include a consistent postoperative elevation of the affected hemidiaphragm in relation to the opposite hemidiaphragm by one intercostal space greater than that seen on preoperative chest radiographs (45,46,48,51). This however is not necessarily synonymous with phrenic neuropathy because pleural disease, atelectasis, and postoperative ileus can also elevate the diaphragm. Thus, the specificity of chest radiograph findings for phrenic nerve injury is not clear (50). In fact, an elevated diaphragm is observed in 30% to 70% of patients after cardiac surgery. However, chest radiograph findings of phrenic neuropathy can be improved when taken at end inspiration in the spontaneously ventilating patient in the recumbent position. This is because, in the upright position, active contraction of the abdominal muscles can passively elevate the diaphragm during exhalation. Similarly, during inspiration in the upright position, abdominal muscle relaxation can stimulate downward movement of the diaphragm despite paralysis. Therefore, upright chest radiographs have a higher false negative rate. Elevation of the paralyzed diaphragm can be missed with deep exhalation and positive-pressure ventilation because deep exhalation elevates the unaffected hemidiaphragm without affecting the paralyzed one, and positive-pressure ventilation pushes the paralyzed diaphragm downward toward the normal position.

Fluoroscopy during spontaneous ventilation in patients with phrenic neuropathy can demonstrate either immobility or a paradoxical movement of the diaphragm (Kienboeck's sign). However, fluoroscopic assessment can be made difficult by the presence of pleural fluid, pulmonary consolidation, atelectasis, and pleural fibrosis (50). In contrast to fluoroscopy (that demarcates the interface between two densities), ultrasonography directly visualizes the diaphragm. In patients with phrenic neuropathy, ultrasonography can demonstrate a hypokinetic, immobile, or paradoxically moving diaphragm (50). Spirometry can also aid in the diagnosis of phrenic neuropathy. Wilcox et al. (52) demonstrated inspiratory muscle weakness with a restrictive pattern on spirometry in patients with phrenic nerve injury.

NCS in the setting of suspected neuropathies demonstrate an increased latency period (>9.0 to 10.0 ms; normal, 6 to 8 ms) and decreased amplitude of the compound diaphragmatic action potential (CDAP) (50). Unfortunately, CDAP amplitude has a 15% intrasubject variation, thus decreasing its utility (50). A more invasive technique of using needle diaphragmatic electromyography can specifically evaluate axonal loss. Electromyography variables evaluated in phrenic neuropathies include motor unit recruitment, spontaneous activity, motor unit potential amplitude, duration, and morphology (50). The presence of fibrillation potentials is indicative of axonal damage.

In a prospective study, DeVita et al. (50) examined the incidence of phrenic neuropathy in 92 CABG patients, noting that 85% of the patients had abnormal postoperative chest radiographs. Atelectasis was the most common chest radiograph finding, followed by an elevated diaphragm. Of these, 54% (42 of 78) had abnormal diaphragm motion by ultrasonography. Fifty-seven percent (24 of 42) of the patients with abnormal diaphragm motion also had abnormal nerve conduction studies. Thus, of the 92 patients, 26% had findings on chest radiograph, ultrasonography, and NCS together consistent with phrenic neuropathy, 75% of these involved the left phrenic nerve. In addition, patients in this study with normal diaphragm motion and nerve conduction improved faster than those with abnormal diaphragm motion and nerve conduction.

Bilateral diaphragmatic paralysis in cardiac surgical patients is very rare and requires a high degree of suspicion to diagnose (53). Similar to unilateral phrenic neuropathy, bilateral diaphragmatic paralysis after CABG surgery is thought to be caused by hypothermic injury to the phrenic nerves. The first indication of bilateral phrenic neuropathy may be difficulty in weaning patients from mechanical ventilation (53). Because of the need for positive pressure ventilation, chest radiography and fluoroscopy is usually nondiagnostic in bilateral diaphragmatic dysfunction (54). In such situations, measurement of the transdiaphragmatic pressure by using gastric and esophageal catheters may be the only diagnostic tool available for definitive diagnosis (54,55). Esophageal pressure closely reflects intrapleural pressure, and gastric pressure reflects intraabdominal pressure (53). Kohorst et al. (53) found in patients with bilateral diaphragmatic paralysis that inspiration lead to a decrease in both esophageal and gastric pressures, and that during inspiration, transdiaphragmatic pressure failed to increase above baseline (normal > 20 cm H2O). With a normally functioning diaphragm, inspiration leads to diaphragmatic contraction, compression of intraabdominal contents, and an increase in intraabdominal pressure.

Phrenic nerve function can be monitored intraoperatively by direct percutaneous electrical stimulation of the nerve at the cervical level and recording CDAP at the seventh intercostal space. Mazzoni et al. (56) prospectively monitored CDAP in patients (n = 12) undergoing CABG surgery. Two of the three patients with abolished CDAP developed overt phrenic neuropathy. In this small study, the authors suggested that postoperative diaphragmatic dysfunction may be predicted in patients when intraoperative CDAP was abolished and did not recover during rewarming from CPB. Further larger prospective studies are needed to demonstrate if changes in surgical technique based on intraoperative phrenic nerve monitoring can reduce postoperative phrenic neuropathy.

In summary, short of postmortem studies, there is no "gold standard" for the diagnosis of phrenic neuropathy (50). Chest radiography has a high false positive rate. NCS have the least false positive and false negative rate (50). Prolonged latency is very specific of neuropathy. An absent response may be indicative of axonal degeneration or technical failure (50). Mild phrenic neuropathies can be undetected by NCS but may be identified by using ultrasonography and fluoroscopy.

**Prognosis of Phrenic Nerve Neuropathy**

In the otherwise healthy adult, recovery and long-term outcome from unilateral phrenic neuropathy is generally good. Unilateral diaphragmatic involvement is usually associated with minimal to no symptoms because of recruitment of accessory, abdominal, and intercostal muscles to assist with ventilation. The most common complaints of patients diagnosed with unilateral phrenic neuropathy are nocturnal orthopnea as well as dyspnea with moderate physical activity (42). Dimopoulos et al. (42) found that there was no difference in the duration of mechanical ventilation, intensive care unit stay, and postoperative complications at hospital discharge in patients with normal phrenic nerve function compared with those with phrenic neuropathy.

In some CABC patients, recruitment of accessory respiratory muscles to assist with ventilation may be hampered as a result of pain from multiple chest tubes and a sternotomy incision. This may eventually lead to diminished cough, resulting in atelectasis and increased chest infections (48). It is most likely that unilateral diaphragmatic dysfunction caused by phrenic nerve involvement is underreported because of minimal patient symptoms. There are no large studies that have addressed the rate of recovery of phrenic neuropathy. The time required for phrenic nerve healing depends on the type of injury and the distance over which regeneration is required (52). Cases of mild phrenic nerve demyelination recover rapidly. In cases with severe demyelination, healing occurs with the synthesis of myelin from schwann cells, and recovery may take 12 weeks or longer. Recovery after complete axonal degeneration is complicated, involves several steps, takes several months, and may be incomplete. The majority of phrenic neuropathies after CABC surgery recover by 3 to 6 months (50). There are no studies that have investigated the time course of phrenic neuropathies in relation to the suspected etiology (i.e., hypothermia versus IMA harvest).

In the patient with chronic obstructive airway disease (COPD), unilateral phrenic neuropathy can have greater sequelae than in the absence of lung disease. Patients with COPD have a limited ventilatory reserve, and the added insult of phrenic nerve injury can lead to further deterioration in lung function. Cohen et al. (57) in a retrospective, case–matched study of 1303 CABC patients with severe COPD, demonstrated that patients with COPD who had phrenic neuropathy during CABC surgery had longer hospital and intensive care unit stays, more frequent reintubations, and hospital readmission as well as decreased overall survival.

Bilateral diaphragmatic involvement is a serious medical problem; death caused by respiratory failure can occur. Depending on the strength and recruitment of accessory respiratory muscles, patients with bilateral diaphragmatic involvement may compensate during waking hours but have serious respiratory embarrassment during sleep. Efthimiou et al. (47), reported 2 cases of bilateral diaphragmatic paralysis among 360 elective CABC patients. Both these patients had severe orthopnea, paradoxical abdominal movement, and a 50% decrease in vital capacity in the supine position. Bilateral phrenic neuropathy has a much longer time course of recovery with significant associated mortality. Of the four patients studied by Efthimiou et al. (47) and Kohorst et al. (53), one patient died of cardiopulmonary arrest after six months and in the other three, phrenic nerve regained function by the tenth month. Two of these patients received intermittent nasal positive pressure ventilation at night to assist with ventilation.

The prognosis of phrenic neuropathy in the pediatric population differs from adults. Mickell et al. (51) found the frequency of unilateral phrenic nerve paralysis in pediatric cardiac surgery patients (n = 1891; age 1 day–14 year) to be 1.7%. In younger children, unilateral phrenic nerve paralysis was a major factor resulting in ventilatory failure and prolonged mechanical ventilation. The authors enumerated several reasons why infants do poorly after phrenic nerve paralysis. First, the intercostal muscles of the infant are weaker, and their contraction does not necessarily result in an increase in intrathoracic cavity size. Therefore, the infant relies on proper diaphragmatic function for effective ventilation. Second, as a result of narrower airways in the infant, atelectasis is more common in the event of disturbed ventilatory mechanics. Third, the mediastinum of the infant is extremely mobile, and elevation of the effected diaphragm can push the mediastinum to the opposite side, further limiting pulmonary ventilation. Lastly, compared with adults, the supine position in infants causes a much greater reduction in vital capacity.

**Prevention of Phrenic Neuropathy**

Nerves can be protected from hypothermia–induced damage by the presence of an insulating layer of fat or close proximity of a major blood vessel. This may be the reason why hypothermia–induced neuropathy includes such a wide spectrum of pathologic features and does not occur in all patients. To minimize hypothermic phrenic neuropathy, Wheeler et al. (46) used a cardiac insulation pad (CIP). They prospectively studied 120 patients undergoing CABC surgery, randomizing patients to two groups: Group I consisted of 60 consecutive patients undergoing CABC surgery without the use of CIP, and Group II consisted of the next 60 patients, in whom the CIP was used. Phrenic nerve injury was diagnosed on the basis of elevation of the hemidiaphragm in the postoperative chest radiograph after comparison with the preoperative chest radiograph. In their study, 60% of the patients who received topical ice slush for myocardial protection (without CIP) demonstrated left hemidiaphragm paresis and left lower lobe atelectasis. In contrast, only 8% of the patients demonstrated evidence of diaphragmatic elevation after the use of CIP (P < 0.0001) (37). The authors proposed that the application of the CIP before the application of ice slush decreased the risk of the phrenic nerve to hypothermic neuropathy.

Several other steps can be taken that may reduce postoperative phrenic neuropathy. Careful dissection of the IMA with particular attention to avoiding ligation of the pericardiophrenic artery may be helpful. Avoiding ice slush collection in the pericardial well for long periods of time also seems prudent. Avoiding entry into the pleural cavity during IMA dissection to prevent collection of ice slush in the pleural cavity, may also be useful. In high risk COPD patients, consideration may be given to the use of warm cardioplegia or performing coronary grafts on a beating heart. Larger prospective, randomized studies are needed to more thoroughly address this issue.
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Recurrent Laryngeal Neuropathy During Cardiac Surgery

Compared with brachial plexus and phrenic neuropathies, vocal cord dysfunction caused by damage of the recurrent laryngeal nerve after cardiac surgery is less common. The frequency of vocal cord dysfunction in adult CABG patients postoperatively varies from 1.9% to 7.8% (58,59).

The left recurrent laryngeal nerve lies very close to the parietal pleura in the upper and posterior part of the thoracic cavity as it circles around the arch of the aorta. If the pleura is opened during IMA dissection and large quantities of ice slush are introduced in the pleural cavity, the left recurrent laryngeal nerve can suffer hypothermic neuropathy (58,60).

Recurrent laryngeal nerve can also be damaged during endotracheal intubation (61), central venous catheter placement (62), and surgical dissection (63). A malpositioned or over-inflated endotracheal tube balloon may come to lie against the vocal cords. Both conditions may cause damage to the anterior branch of the recurrent laryngeal nerve that is closely related to the vocal cords (61). The use of transesophageal echocardiography (TEE) has also been examined as a possible etiologic factor. Kawahito et al. (59) reported that 5 of 64 patients (7.8%) in whom a TEE probe was placed and 3 of 52 (5.8%) in whom TEE was not used were diagnosed with recurrent laryngeal neuropathy postoperatively. This difference was not statistically different, and the authors concluded that the intraoperative use of TEE in cardiac surgery patients was not associated with an increased frequency of recurrent laryngeal neuropathy. The authors noted, however, that the duration of surgery, CPB, and anesthesia administration were significantly longer in patients with recurrent laryngeal neuropathy. This condition should be suspected in patients who develop respiratory insufficiency, ineffective cough, and hoarseness after extubation (58). Vocal cord dysfunction is often associated with dysphagia, which in turn may be associated with aspiration pneumonia.

The diagnosis of recurrent laryngeal neuropathy can be confirmed by using laryngoscopy. Unilateral vocal cord neuropathy usually takes 8 to 12 months to recover (58). Most patients can be treated conservatively, whereas some severe cases may require reintubation and tracheostomy. Long-term management includes potential fat or teflon injection into the vocal cords to increase tension and possibly arytenoidectomy to increase the laryngeal opening (58).

Saphenous Neuropathy After Cardiac Surgery

Despite increasing use of the IMA as a graft conduit, the long saphenous vein continues to be widely used for CABG surgery. The saphenous nerve, a sensory branch of the femoral nerve (supplied by the nerve roots L2,3,4) descends below the inguinal ligament in the subsartorial canal. It continues becoming superficial in the lower leg, descending with the long saphenous vein toward the medial malleolus (64-66). It supplies the anteromedial aspect of the leg through its two major divisions: sartorial and the infrapatellar branches (64-66). Multiple branches of the saphenous nerve cross the long saphenous vein in the distal two thirds of the leg. The saphenous nerve is in close approximation with the adventitial sheath of the vein at the ankle level. Avulsion injury to the saphenous nerve, particularly the infrapatellar branch, is a possibility during saphenous vein harvest (67).

Injury to the saphenous nerve commonly presents as saphenous neuralgia that is characterized with the triad of anesthesia, hyperesthesia, and pain along the medial side of the calf and foot to the level of the great toe (64,65). Nair et al. (68) reported that the frequency of saphenous neuralgia varies from 90% immediately after surgery to 10% at 14-18 months postoperatively. Mountney and Wilkinson (69) demonstrated that nearly 90% of the lower limbs examined after long saphenous vein harvest showed some degree of anesthesia at 3 days with 72% remaining symptomatic after 20 months.

Nair et al. (68) found that the incidence of postoperative neuralgia after saphenous vein harvest was increased in patients whose leg incision was closed in two layers (i.e., subcutaneous and subcuticular) as compared with one layer (i.e., subcuticular closure only). They attributed this finding to a neuropaxia from the pressure exerted by the tightened sutures across the wound. They consequently recommended single-layer subcuticular closure of the leg wound after harvest of the long saphenous vein.

The direction of long saphenous vein dissection may also influence the frequency of saphenous neuralgia. Ramasastry et al. (67) demonstrated that patients, in whom the long saphenous vein was dissected in the upward direction (i.e., dissection starting from the medial malleolus and continuing upwards) as compared with the downward direction (i.e., dissection starting from the saphenofemoral junction and continuing downward), had significant sensory deficits in the saphenous nerve distribution at 12 weeks postoperatively ($P < 0.0001$). At six months the results were similar. After cadaveric studies, the authors concluded that long saphenous vein dissection upward leads to the avulsion of the pre Tibial and/or the infrapatellar branch of the saphenous nerve more frequently than in dissection performed in the downward direction.

Recently in many cardiac centers, endoscopic saphenous vein harvest is being increasingly used. The endoscopic technique permits harvesting of the long saphenous vein with minimal handling and, thus, may result in less local inflammation and hematoma formation. Folliguet et al. (70) prospectively randomized 60 CABG patients to either open or endoscopic long saphenous vein harvest. Between the seventh and the tenth postoperative days, pain in the distribution of the saphenous nerve was assessed on a numerical scale from 0 to 10 (10 being described as excruciating pain, and 0 as no pain at all). Their results demonstrate that local pain was reduced in the endoscopic vein harvest group, with a mean score of 1.8 in the open and 0.6 in the endoscopic groups. The authors did not comment whether this difference in pain scores was statistically significant. Larger, randomized studies are needed to address whether endoscopic harvesting techniques result in less saphenous nerve injury and resulting saphenous neuralgia.

Sympathetic Chain Neuropathy (Horner’s Syndrome) After Cardiac Surgery

The cervical sympathetic chain lies medial to the inferior trunk of the brachial plexus as it crosses over the first rib, making it vulnerable to injury much the same way as the brachial plexus. Vander Salm et al. (14) demonstrated that excessive sternal retraction could lead to posterior first rib

fractures, which could directly damage the cervical sympathetic chain. Sympathetic chain neuropathy produces Horner's syndrome characterized by miosis, ptosis, and anhidrosis. Shaw et al. (71) identified 4 of 312 CABG patients with postoperative Horner's syndrome. In each case, it was unilateral and associated with neuropathy to the inferior trunk of the ipsilateral brachial plexus. Horner's syndrome persisted for more than six months in two of these patients while the other two recovered within six months (71). Although not studied specifically, preventive strategies similar to those for brachial plexus neuropathies may also decrease the frequency of sympathetic chain neuropathies after cardiac surgery.

In conclusion, nerve injuries after anesthesia are well recognized complications. They are also a significant source of morbidity for patients and potential liability for anesthesiologists. Cheney et al. (72) showed in the Closed Claims Project database that besides death, neuropathies after surgery were the second most common claim. CABG surgery is frequently associated with neuropathies involving the brachial plexus, phrenic, and saphenous nerve. Fortunately, the majority of patients recover without further treatment. In a minority of patients, these neuropathies can be associated with increased postoperative morbidity. Focused preventive strategies may reduce both the frequency and severity of these lesions.

Footnotes

Accepted August 7, 2000.

References

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**Articles citing this article**

- Ventricular Fibrillation With Diminished Internal Mammary Artery Graft Flow During Sternal Retraction
  - [Abstract] [Full Text] [Full Text (PDF)]

- Postoperative Care of Cardiac Surgery Patients
  - [Full Text]

- Recognizing Chronic Postsurgical Pain Syndromes at the End of Life
  - *AM J HOSP PALLIAT CARE* September 1, 2007 24:319-324
  - [Full Text (PDF)]

- Prevalence, characteristics, and predictors of chronic nonanginal postoperative pain after a cardiac operation: A cross-sectional study
  - [Abstract] [Full Text] [Full Text (PDF)]

- Chronic postoperative pain: the case of inguinal herniorrhaphy
  - *Br J Anaesth* July 1, 2005 95:69-76
  - [Full Text] [Full Text (PDF)]