Paraplegia After Coronary Artery Bypass Graft Surgery
Case Report of a Rare Event

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The authors discuss paraplegia after CABG surgery, including spinal cord anatomy and physiology, pathophysiology, clinical findings, and complications. In addition, a case study as well as nursing implications and interventions are presented.

Paraplegia is an unpredictable complication that can occur after repair of coarctation of the aorta, thoracotomy (e.g., lobectomy, pneumonectomy), repair of aortic dissection, valvular surgery, heart transplantation, and coronary artery bypass graft (CABG) surgery.1–12 Paraplegia after surgeries of the thoracoabdominal or abdominal aorta has a reported incidence of 4% to 80%, depending on the complexity of the surgical procedure required and alterations in spinal cord perfusion.3,14 However, paraplegia after CABG surgery is rare. An extensive literature search of the CINAHL and MEDLINE databases reported incidence of 4% to 80%, depending on the complexity of the surgical procedure required and alterations in spinal cord perfusion.3,14 However, paraplegia after CABG surgery is rare.

In this article, we discuss paraplegia after CABG surgery, including spinal cord anatomy and physiology, pathophysiology, clinical findings, and complications. We use a case study approach to provide an understanding of this type of paraplegia and present nursing implications and interventions.

Spinal Cord Anatomy and Physiology

The spinal cord acts as a relay center between the central and peripheral nervous systems for sensory and motor receptors. The spinal cord is supported and protected by various structures. Encased in the vertebral column, the cord originates at the medulla and ceases at the second lumbar vertebra. Within the vertebral column, the meninges and cerebrospinal fluid (CSF) surround and protect the cord.15

The spinal cord consists of 31 pairs of nerves: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal.16,17 (Figure 1). Each pair of nerves consists of a motor (anterior) root and a sensory and touch (posterior) root.3,15 Upon exiting the spinal cord, the pairs of nerves are structured into a plexus. The plexus then forms the various peripheral nerves. The cervical plexus, composed of the cervical nerves, innervates the shoulder, diaphragm, bowel, and bladder. The brachial plexus, composed of the cervical and thoracic nerves, innervates the upper extremities. The lumbosacral plexus, originating from the lumbar and sacral nerves, innervates the lower extremities.15

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Figure 1
Spinal nerves.
The arterial blood supply to the spinal cord varies depending on the anatomical location. The arterial blood supply consists of a rich capillary network and longitudinal, medullary feeder, segmental, and radicular arteries (Figure 2). The anterior spinal artery and 2 small posterior arteries make up the 3 longitudinal vessels, which are derived from the vertebral artery. The anterior spinal artery is large and supplies the frontal two thirds of the spinal cord; the posterior arteries perfuse the remainder of the cord.

The medullary feeder arteries, which arise from the segmental arteries, reinforce blood supply to the 3 longitudinal vessels. The Adamkiewicz artery (radicularis magna) is the largest medullary feeder artery and is a key contributor to the anterior spinal artery and the lumbar region of the spinal cord. The Adamkiewicz artery enters the spinal cord at an acute angle. In 85% of the population, the Adamkiewicz artery is a single vessel on the left side of the spinal cord supplying the T9 to L2 distribution.

The segmental arteries are located as pairs, one pair at each intervertebral level. The anatomical location within the spinal cord determines the source of the segmental arteries. Within the cervical vicinity, the arteries arise from the subclavian and vertebral arteries. The thoracoabdominal segmental arteries are derived from the intercostal and lumbar arteries; the caudal aspects of the spinal cord arteries arise from the lumbar, iliolumbar, and lateral sacral arteries of the hypogastric circulation. The radicular arteries provide blood supply to the spinal nerves at every segmental level.

In addition to the circulatory pattern described, 4 collateral pathways are possible, although they are not well defined. One pathway is located between the intercostal and lumbar arteries; another is positioned between the internal iliac or middle sacral arteries and the lower aspect of the spinal cord. The internal mammary artery, via the inferior epigastric artery, may provide collateral circulation to the lower aspect of the spinal cord. Finally, the anterior spinal artery may increase in size and length (ie, between the lower cervical spinal artery and the upper thoracic spinal artery).

Etiology of Paraplegia After Cardiac Surgery

Paraplegia after cardiac surgery in adults is rare. Only 7 cases were reported between 1978 and 2002. Other neurological complications, such as cerebral stroke or transient decreased level of consciousness, are more common. Unlike the more prevalent paraplegia caused by trauma of the spinal cord (fracture or penetration by a foreign body such as a bullet), paraplegia after cardiac surgery is related to hypoperfusion of the spinal cord arteries. Paraplegia after cardiac surgery may be related to formation of a hematoma, hypotension (intraoperative blood loss, use of anaesthetic or epidural agents, and aortic dissection), use of an intra-aortic balloon catheter, thrombosis or embolization, use of aortocoronary grafts, electrolyte imbalances, and the toxic effects of drugs (Table 1).

Table 1

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<th>Causes of paraplegia after cardiac surgery</th>
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<td>The first reported case of paraplegia after CABG surgery was attributed to the formation of a subadvential hematoma, found at autopsy, just below the left subclavian artery and extending to the celiac artery. Sub-advential and epidural hematomas have also been reported after mitral valve replacement. Paraplegia after CABG surgery has also been reported in patients with a history of peripheral vascular disease who experience severe hypertension (mean arterial blood pressure &gt;100 mm Hg) during surgery and in the postoperative period. In patients with severe peripheral vascular disease who do not have aortic dissection, hypertension is thought to lead to plaque rupture and embolization to the spinal cord arteries. In addition, hypotension during the postoperative period in patients with a history of peripheral vascular disease and hypertension may lead to hypoperfusion and ischemia of the spinal cord arteries. Reversible paraplegia due to causes other than trauma has been reported. One case was attributed to hypokalemia (serum potassium level 1.0 mmol/L) after burn injury caused by a sustained release of catecholamine with increased intracellular movement of potassium ions.</td>
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in the shock state. A similar event may occur in patients who have cardiothoracic surgery. Hypokalemia is common in the early postoperative period because of fluid and electrolyte shifts and diuresis. Fluid shifts are due to the destruction of platelets by the cardiopulmonary bypass machine; the subsequent decrease in oncotic pressure leads to movement of fluid into the extravascular space. Mannitol may be used during cardiac surgery to help protect the renal tubules from occlusion by cellular debris and ultimately prevent renal dysfunction, third spacing of fluids, and electrolyte imbalances.

Pathophysiology of and Clinical Findings in Paraplegia After Cardiac Surgery

Paraplegia occurs at or below the T4 spinal level. Full upper body strength and use of the upper extremities may be preserved; only the lower extremities are affected. Evidence of functional limitations depends on the level of injury of the spinal cord (Table 2). Injury of the spinal cord because of ischemia or infarction typically occurs at the T4 to T9 spinal level; this area is most susceptible to the causes given in Table 1. This region (T4 to T9) is the narrowest part of the spinal cord and has the poorest collateral blood supply. The typical cause of paraplegia associated with damage in this area is ischemia and infarction of the anterior spinal artery. Hypoperfusion or compression of the arteries at the T4 to T9 spinal level during surgery and in the postoperative period could lead to a spinal cord infarct with concomitant paraplegia. As well, the Adamkiewicz artery, which supplies the T9 to L2 spinal levels, enters the spinal cord at an acute angle and is susceptible to compression. Decreased perfusion to or occlusion of the Adamkiewicz artery can cause paraplegia below the T9 spinal level.

Methods to decrease oxygen demands and limit hypoperfusion during surgery can diminish the risk of paraplegia. Using hypothermia, maintaining an adequate blood pressure distal to the aortic clamp, keeping the duration of aortic cross-clamping as short as possible, and limiting the duration of anesthesia can diminish the risk. An appropriate balance between hypertension and hypotension during surgery is required. However, the recommended levels for blood pressure control have not been well defined and are determined in the clinical setting on the basis of the unique needs of each patient.

Diagnosis of Paraplegia

Before sudden paralysis is ascribed to a surgically induced mechanism, other causes such as trauma, cerebral stroke, or an arteriovenous malformation must be ruled out. Laboratory investigations are required to determine if the patient has an underlying disease process such as a neoplasm or blood dyscrasia. The signs and symptoms of both conditions can mimic those of spinal cord infarction, although neoplasms and blood dyscrasias have a more insidious onset. A neoplasm can lead to compression of the spinal cord with resultant paraplegia.

Paraplegia after cardiac surgery is typically not diagnosed soon after onset because patients have postoperative pain and are given analgesics and sedatives in the immediate postoperative period. Possible diagnostic studies include myelograms, electromyographic studies, computed tomography, and magnetic resonance imaging.

A myelogram may provide the location of spinal cord compression. An electromyographic study may help in assessing denervation. Computed tomography can be used to locate an intimal flap or hematoma, differentiate flow rates between arterial true and false lumens, determine an increase in the size of the aorta, detect displacement of intimal calcification, and, possibly, locate a spinal cord infarct. Magnetic resonance imaging may be the best diagnostic tool for detecting enlargement or abnormalities of the spinal cord or locating a spinal cord infarct; however, use of magnetic resonance imaging after cardiac surgery is limited because the patients often have epidural pacemaker wires, permanent pacemakers, and implantable pacemaker cardioverter defibrillators.

Case Report

L.W., a 74-year-old man, was referred to the cardiovascular surgical team for CABG surgery after he experienced an acute coronary syndrome, for which he had been admitted to the coronary care intensive care unit (ICU). He had a history of cigarette smoking with emphysema, benign prostatic enlargement, hypertension with renal impairment, and severe peripheral vascular disease. He had previously had bilateral carotid endarterectomies and aortobifemoral bypass grafting. Echocardiograms and stress testing with technetium Tc 99m 2-methoxy-isobutyl-isonitrile indicated that he had
L.W. was treated for acute anterior myocardial infarction and congestive heart failure and underwent coronary angiography, which showed severe triple-vessel coronary disease. He was given inotropic agents and diuretics to optimize cardiac and renal function in preparation for surgery. Sixteen days after his admission, L.W. had 4-vessel CABG surgery, with grafts from the left internal mammary artery to the left anterior descending artery and saphenous vein grafts to the obtuse marginal, ramus, and right coronary artery. Cardiopulmonary bypass time was 175 minutes, with an aortic cross-clamp time of 121 minutes. Difficulty was encountered in terminating the bypass, and L.W. was transferred to the cardiovascular surgical ICU with his sternum open; treatment with high-dose vaspressors, antiarrhythmics, diuretics, and sedatives continued. Treatment with intra-aortic balloon counterpulsation was not used because of his history of peripheral vascular disease and aortofemoral bypass surgery and the severe aortic calcification discovered during the CABG procedure.

L.W.’s postoperative course was extremely complex, with recurrent life-threatening complications. A transesophageal echocardiogram done at the time of admission to the cardiovascular surgical ICU indicated 2+ mitral regurgitation, severe aortic atherosclerosis, and an ejection fraction of 0.15 to 0.17; only the lateral wall of the left ventricle was contracting. L.W. was sedated with propofol; his scores on the Glasgow Coma Scale and the Ramsey Sedation Scale were 2/10 and 5 to 6, respectively. His sternum remained open, so minimal movement was desired. Analgesia was provided by a fentanyl infusion. His hemodynamic status remained unstable, and he was acidic and oliguric. Continuous renal replacement therapy was started 2 days later when the serum creatinine level reached 189 μmol/L (2.1 mg/dL).

Sternal closure was achieved on postoperative day 6. At that time, blood pressure was 100/60 mm Hg, pulmonary artery pressure was 65/25 mm Hg with inhaled nitric oxide at 60 ppm, and the score on the Glasgow Coma Scale was 3/10. During the next few days, scores on the Glasgow Coma Scale were 6/10 to 8/10, and L.W.’s movements were negligible. Because he was being treated with fentanyl for pain control, his central nervous system depression was attributed to an accumulation of fentanyl and propofol in a patient with renal and hepatic dysfunction. His neurological status was difficult to assess because he was extremely agitated at night, a situation attributed to a long history of alcohol abuse and subsequent withdrawal, and he required benzodiazepines in addition to his other central nervous system depressants.

On postoperative day 9, when L.W. was fully awake, flaccid paralysis of both lower extremities and severe weakness of the left upper extremity were apparent. Because he had been treated with steroids for adrenal insufficiency after surgery, the profound muscle weakness was initially attributed to steroid use. The neurology service was consulted, and paraplegia at the T9 spinal level with sensation to the T7 to T8 levels was diagnosed. The cause was thought to be related to either multiple emboli or hypoperfusion leading to spinal cord ischemia. Computed tomography of the brain showed multiple areas of infarction in the right middle cerebral artery territory, which accounted for the severe weakness in the left upper extremity. Anticoagulation therapy was started.

Continuous renal replacement therapy was discontinued on postoperative day 14, and nonoliguric renal failure occurred. L.W. was still receiving mechanical ventilation; a tracheostomy was done on postoperative day 16. Moreover, L.W. continued to require a dobutamine infusion for decreased cardiac output and poor left ventricular function. A conference with L.W. and his family was held on postoperative day 18, and a do-not-resuscitate order was established because of L.W.’s neurological disability, poor respiratory and cardiac function, ongoing renal dysfunction, and overall debilitated state.

An echocardiogram 26 days after surgery showed no recovery of cardiac function with an ejection fraction of 0.10 to 0.15 once the dobutamine infusion was discontinued and maximal medical therapy for congestive heart failure was started: an angiotensin-converting enzyme inhibitor, a diuretic, a β-blocker, and spironolactone. Trials of weaning from mechanical ventilation were done as L.W. could tolerate them, and mobilization to a cardiac chair began. Approximately 1 month after the CABG surgery, L.W. was transferred to the general systems ICU for further rehabilitation and convalescence. Despite the best efforts of the multidisciplinary healthcare team, further complications developed, and ultimately when it was evident that L.W. would not recover, comfort care was chosen and he died soon thereafter.

**Discussion**

Patients with paraplegia induced by either spinal cord infarction or spinal cord trauma have similar pathophysiological changes that affect multiple systems. Autonomic dysfunction, heterotopic ossification, muscle spasticity, osteoporosis, sensory impairment,
and voluntary muscle paralysis with associated complications are evident below the level of the spinal cord injury (Table 3). However, spinal cord infarction may not involve full transection. Because the anterior spinal artery is affected, the patients have loss of motor movement and loss of pain and temperature sensations to the areas below the level of the spinal cord injury (Table 2). However, often the posterior spinal cord root functions of vibration and proprioception (position) remain intact.

Postoperative considerations for paraplegia and implications for nursing care are listed in Tables 3 and 4. L.W. had several risk factors for postoperative spinal cord ischemia and paraplegia. Severe peripheral vascular disease and hypertension made him especially sensitive to alterations in blood flow and reduction in blood pressure during surgery. In addition, the prolonged duration of cardiopulmonary bypass, use of an internal artery mammary graft, the presence of severe aortic calcification and the requirement for high-dose vasopressors to maintain a borderline blood pressure contributed to spinal cord ischemia and resultant paraplegia. Although the cause of the paraplegia could never be radiographically confirmed, because L.W.’s condition was not stable enough for him to undergo magnetic resonance imaging, most likely the cause was a combination of low perfusion and embolic plaque rupture.

L.W. required sedation and analgesia for several days in the immediate postoperative period because of his compromised hemodynamic status and his open sternum. Minimal movement and a decreased level of consciousness were desired because of his critical condition and the use of multiple invasive catheters. Consequently, the paraplegia went undiagnosed until several days after its occurrence. As nurses are aware, providing “sedation holidays” for even the most critically ill patients is imperative, to permit assessment of neurological status and detection of any deviations from the norm. Once recognized, a plan of care can be rapidly implemented to prevent further complications, even if the paraplegia is irreversible.

Cross-clamping of the aorta decreases perfusion of the spinal cord by simultaneously increasing CSF pressure and decreasing distal aortic perfusion pressure. These changes may result in intrathecal compartment syndrome. Therefore, using drainage to decrease CSF pressure theoretically should increase spinal cord perfusion.

At University of Alberta Hospital in Edmonton, we are in the initial stages of planning for CSF pressure monitoring and drainage in the cardiovascular surgical ICU. The effect of CSF drainage is based on the hypothesis that lowering CSF pressure increases spinal cord pressure and perfusion. When excess fluid is drained, spinal cord compression is released and perfusion is enhanced. The drainage can be accomplished via a monitored lumbar drain, maintaining a normal CSF pressure of 10 to 15 mm Hg.

Deep vein thrombosis may also be a problem for patients with paraplegia. The risk of prophylaxis with subcutaneous heparin must be considered in postoperative cardiac surgical patients who have bleeding and disseminated intravascular coagulopathy. In addition, nurses must be cognizant of the risk for heparin-induced thrombocytopenia, which may be more common in patients such as L.W. who have previously been treated with heparin than in patients never treated with the agent.

As patients with paraplegia start to participate in rehabilitation, it is important to acknowledge that paraplegia can have a profound effect on increasing cardiac workload. When patients move to a chair or to a wheelchair, energy expenditure and cardiac output increase, and upper body strength must be maximized, the stress of which may affect the sternotomy incision and wound healing. In patients such as L.W. who have depressed cardiac function, the expectation for them to be able to mobilize independently may be unrealistic and may influence decision making about quality-of-life issues.

In addition to recognizing the physiological consequences of paraplegia, nurses must also be aware of the psychological devastation of this rare complication and must be prepared to help patients and patients’ families in grieving and acceptance. Denial, anger, depression, loss of self-esteem, and ineffective coping often occur. A multidisciplinary team approach for care of these patients is vital. This approach may require additional team members from social services, psychiatry, physiotherapy, occupational therapy,
nutrition, and pastoral care. To be successful, the multidisciplinary team must include patients and the patients’ family members in all phases of rehabilitation. This inclusion will give patients and their families a feeling of power and a sense of control in a situation that otherwise might be overwhelming.

As patients’ advocates, nurses can participate in the process of informed consent by affirming that patients are aware of all potential complications after CABG surgery, even complications as unlikely as spinal cord infarct resulting in paraplegia. By doing so, nurses may have an important effect in improving the level of knowledge of patients and patients’ families, ensuring that expectations are realistic as far as risks of surgery and long-term outcomes and facilitating acceptance of postoperative paraplegia and its sequelae.

Conclusion

All critical care nurses should have a basic understanding of spinal cord anatomy and physiology. Paraplegia after CABG surgery is a rare, yet devastating complication. Aortic cross-clamping, use of an internal mammary artery conduit, embolism from aortic calcification, and hypotension are the most common risk factors for spinal cord ischemia in these patients, but aortic dissection, an intimal tear at the cannulation or cross-clamp site, or disruption of the graft suture line are also possible causes of such ischemia. Decreased perfusion or occlusion of a segmental artery could have dire consequences, because the medullary feeder and longitudinal arteries would be affected. If the internal mammary artery is used as a conduit for aortocoronary surgery, paralysis of the lower extremities is possible.

Elderly patients with severe atherosclerotic disease are at highest risk for spinal cord ischemia if even a slight decrease in perfusion to the spinal cord occurs. However, prolonged hypotension must be avoided in all cardiac surgical patients, because spinal cord infarction can occur in young, healthy individuals, especially in the T4 to T9 spinal levels. Early recognition and prevention of paraplegia-related complications via meticulous nursing care will help patients with paraplegia after cardiac surgery in the long process of recovery.

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Footnotes

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9. Archer AG, Choyke PL, Zeman RK, Green CE, Zuckerman M. Aortic dissection
Paraplegia is an exceedingly rare neurologic complication after off-pump coronary artery bypass graft (OPCAB) surgery commonly caused by spinal cord ischemia (SCI). SCI has also been reported as a rare complication among the other neurologic sequelae of cardiac surgery. We report a case of paraplegia in a patient with metabolic syndrome and multiple risk factors after an OPCAB. This patient may be considered as an interesting case as no any ischemic event was found in the imaging modalities and culprit lesion may be attributed to left internal thoracic artery use as an important blood perfusion to anterior spinal artery vasculature and 2-Disc herniation at intervertebral space of T as a culprit lesion 3-transient intraoperative hypotension.