Case Report and Review

Unique Considerations for the Spinal Cord Injured Patient Undergoing Cardiac Surgery Utilizing Cardiopulmonary Bypass

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Abstract: A 37-year-old male with mitral valve regurgitation presented for mitral valve replacement. He has been a C5 quadriplegic for 13 years. The patient had been discharged 2 months before to this admission after a complicated hospital course for Staphylococcus aureus infection of the left hip. His course was complicated by adult respiratory distress syndrome (ARDS) requiring prolonged intubation, acute renal failure (ARF) requiring dialysis, 10-day coma, and bacterial endocarditis now requiring mitral valve replacement. After initial stabilization with antibiotics and gradual improvement of the multiorgan system failure, the patient presented for valve replacement and worsening congestive heart failure (CHF). Para- and quadriplegic patients rarely undergo cardiac surgery requiring cardiopulmonary bypass (CPB). The explanation for this low incidence of heart surgery in this patient population ranges from physiologic changes from the spinal cord injury to their relatively short life span. Therefore, there is no vast knowledge of how these patients with spinal cord injury will physiologically respond to CPB. Chronic paraplegia presents unique anesthetic and perfusion challenges. General anesthesia for a patient with prolonged spinal cord damage can be difficult because of dysreflexia, muscle wasting, and potassium changes with depolarizing muscle relaxants. For the perfusionist, chronic paraplegia also accentuates hemodynamic responses to nonpulsatile flow with low peripheral vascular resistance common and difficult to treat. Dramatic increases in circulating catecholamine levels are a secondary result of the initiation of CPB that can cause a hypo- and hypertensive state. Depending on the level of spinal cord injury, one might expect acute hypo- or hypertension with the various phases of open-heart surgery and CPB. A viscous circle may occur because the hypertensive state is exaggerated because of inhibitory signals not passed below the spinal cord lesion and, therefore, the vasoconstrictive reflex continues unabated. The attack usually occurs abruptly and can lead to cerebrovascular hemorrhage and death if not controlled. Fortunately, we found this patient did not develop mass autonomic dysreflexia and was not difficult to wean from CPB. The problems associated with spinal cord injury present potential complications to this patient population. Numerous triggering mechanisms may lead to a variety of clinical complications. Consideration of a response/treatment management plan for potential problems must be exercised by the surgical team. Keywords: cardiopulmonary bypass, spinal cord injury, cardiac surgery, autonomic dysreflexia.

The problem of spinal cord trauma with resultant spinal cord shock and para/quadriplegia represent a major additional threat to the patient should he or she require cardiac surgery. The cord-injured patient presents with a spectrum of pathological and physiological changes, depending on the level of injury and the time since injury. There are several reports and reviews of anesthesia literature on patients with a spinal cord trauma and para/quadriplegia (1). Based on these reports, it is reasonable to subdivide the paraplegia syndrome into phases: acute (under than 3 weeks); intermediate (3 days to 3 months); and chronic (more than 3 months) (1). This division is useful because it defines a group of patients in the intermediate phase that may have elevated serum potassium and an abnormal response to depolarizing blocking agents (note the overlap of intermediate and acute phases). The spinal cord injury (SCI) patient also is divided into low injury (below T7 motor level) and high (above T7 motor level). Those patients with transections above the level of T7, demonstrate significant vascular irritability with both hypotensive and hypertensive responses reflexly initiated by changes in vasculature below the lesion (1).
With each passing year, there is an increased likelihood of renal complications, and 60% of SCI patients have impaired renal function after 10 years (1,2). Amyloidosis is seen frequently with protein loss and reduction in total body sodium and potassium.

Respiratory compromise in SCI patients with high spinal lesions accounts for a large portion of the mortality and morbidity associated with anesthetizing these patients. This decreases vital capacity and expiratory reserve volume, predisposing the patient to postoperative complications (1). Preoperatively, these patients have varying degrees of hypoxia and hypercarbia (2–5).

Temperature regulation is dependent to a large extent on control of circulation to the skin, and this regulatory mechanism is impaired in SCI patients. The quadriplegic patient is poikilothermic below the level of the spinal cord lesion from the loss of autonomic control of sweating, shivering, and cutaneous vasoactivity (1).

With impaired cutaneous circulation, decubitus ulcers may develop after 2 hours of continuous pressure of a skin area and after only 20 minutes if the patient has a fever. The incidence of this condition is higher in anemic and quadriplegic patients (2).

Patients in the intermediate stage often have elevated serum potassium and respond to depolarizing muscle relaxants with further increases in potassium. This high level of potassium has been reported to lead to ventricular fibrillation (1,6,7). Electrolyte and metabolic disturbances may occur as a result of frequent use of enemas preoperatively.

Autonomic dysreflexia is an acute syndrome characterized by widespread reflex sympathetic discharge in patients with chronic spinal cord lesions. Classical clinical manifestations are sudden paroxysmal hypertension and bradycardia (2). If occurring during surgery, the hypertensive changes of autonomic dysreflexia may lead to increased blood loss. The patient can lose as much as 10–15% of his or her plasma volume during an attack of autonomic dysreflexia (6). If unabated, the severe hypertension may lead to unconsciousness, seizures, hypertensive encephalopathy, retinal cerebral and subarachnoid hemorrhage, cerebrovascular accident (CVA), and death. Beside bradycardia, cardiac changes may include left ventricular failure resulting in pulmonary edema, cardiomegaly, and myocardial ischemia (1,2,5,6,8–10).

Characterization of the pharmacokinetic behavior of many drugs used in the treatment of SCI patients has never been studied in an SCI patient population. There is some evidence to suggest that the volume of distribution and possible the clearance of many of these drugs may be altered in SCI patients (11).

Because of its effect on multiple organ systems, SCI has the potential to affect the pharmacodynamics of a number of drugs. The effect of SCI on drug pharmacodynamics and receptor sensitivity can depend on the length of time since the injury (11).

The patient with a high spinal cord lesion presents many challenges, and the surgical team must work together to be cautious in providing the adequate level of anesthesia and the least amount of disruption of a delicate physiological equilibrium.

**CASE REPORT**

The patient is a 37-year-old male who sustained a gunshot wound to the neck 12 years ago and is a C5 quadriplegic. He has developed problems with ulcer pressure sores on his sacrum and trochanteric areas and, as a result, he developed endocarditis from presumed hematogenous spread of the organism, with a 2–3-week history of increased shortness of breath. As the patient became more septic, he developed renal failure and slipped into a coma for a period of 10 days. The pressure sores required repair. However, because of his unstable cardiac status, this could not be completed before his mitral valve replacement. Upon admission, he was found to have severe mitral regurgitation with increased atrial pressure and size, mild to moderate tricuspid regurgitation, and normal aortic and pulmonic valvular function. This combination of physiological insults required inotropic support and afterload reduction, followed by mitral valve repair/replacement.

Preoperatively, routine arterial blood gases revealed the following: pH 7.32, Pco2 74, Po2 68, saturation 90%, hematocrit 34%, K+ 4.7, and base excess +10. Anesthetic induction was carried out using fentanyl, versed, and pancuronium. Isoflurane in 100% O2 was used as a maintenance inhalation agent at 0.7% initially and increased to 1.2% for a period before cardiopulmonary bypass (CPB). Sodium nitroprusside was started shortly after induction and titrated to maintain a mean arterial pressure of 70–80 mm Hg throughout the initial stages of induction to the initiation of CPB. Pulmonary artery pressure was steady at 125/75 mm Hg before commencement of CPB.

Before heparinization, the activated clotting time (ACT) of 149 seconds was measured (Hemotec, Medtronic Cardiopulmonary, Anaheim, CA). The patient was heparinized with 300 IU of porcine mucosal heparin and the repeat ACT was 793 seconds. A pump flow of 2.4 L/m² was constant throughout the case, and normothermia was maintained. Mean arterial pressure was low, in the range of 35–38 mm Hg, for approximately the first 30 minutes of CPB. The arterial blood pressure was treated with phenylephrine and maintained for the duration of CPB. Arterial and venous blood gas measurements...
were done at 30-min intervals, and attempts were made to maintain his physiologic acid-base balance (see Table 1). For the remaining 45 minutes of bypass, the mean arterial pressure was maintained in the range of 50–55 mm Hg. Initial hematocrit after the start of CPB was 18%. A hemococoncentrator (COBE Cardiovascular, Inc., Arvada, CO) was used to remove 1100 mL of volume during CPB. This volume removal, in addition to the administration of one unit of packed red blood cells (250 milliliters), increased the hematocrit to 24% before the termination of CPB. No other volume was added during CPB, and urine output was 40 mL. Hypothermic (10°C) blood cardioplegia was used for myocardial protection with the patient’s final systemic potassium level of 5.5 mEq/L. Total bypass time was 80 minutes with an ischemic time of 55 minutes, and the patient successfully weaned from CPB.

PATHOPHYSIOLOGY OF SPINAL CORD INJURY AND CARDIOVASCULAR COMPLICATIONS

As a result of a spinal cord injury, the demand from blood flow by various organs and tissues will change. This necessitates a control system that can modify blood supply to meet local demands in varied situations. Control of the circulation occurs both at a local and regional level through autoregulation and metabolic effects of individual tissues and systematically through neural and humoral mechanisms. The later consists of several components including peripheral receptors monitor demand, integrating centers in the brain stem and spinal cord, and peripheral effects to alter supply to meet demands (2).

An altered and, sometimes, defective control of the cardiovascular system is a common complication of SCI because there may be a destruction of spinal nerves, resulting in an interruption of communication between certain receptors and effectors and controlling and modifying centers in the brain stem (9,12).

With SCI, the disruption of the autonomic nervous system (ANS) accounts for specific defects that may occur with a given level of spinal cord transaction. The major cranial parasympathetic supply is via the vagus nerve, which exits the central nervous system (CNS) through the brain stem. Thus, vagal supply is spared in spinal cord injuries; whereas, sacral parasympathetic influences are frequently disturbed. In contrast, all sympathetic outflow occurs below cervical segments. Injury occurring above T1 results in loss of excitatory and inhibitory input to all pre-ganglionic sympathetic neurons. Cervical spinal cord transaction usually results in significant alteration of cardiovascular system control; whereas, thoracic lesions produce fewer alterations. The heart and blood vessels above the diaphragm are largely innervated through thoracic cord segments T1–T4. With the loss of communication between higher centers in the brain stem and the CNS, important cardiovascular regulatory mechanism via reflexes is lost. Spinal cord neural activity becomes unrestrained and dysfunctional. Primitive reflexes that occur at the spinal cord level and are limited functional significance when the spinal cord is intact become more important when the spinal cord is disrupted. The most relevant cardiovascular problem resulting from SCI is autonomic dysreflexia or autonomic hyperreflexia.

ANESTHETIC MANAGEMENT AND AUTONOMIC DYSREFLEXIA

The important objective of anesthesia in these cases is the prevention of autonomic dysreflexia. An understanding of the pathophysiology of this syndrome is crucial in its successful management. Autonomic dysreflexia poses the only medical emergency related to spinal cord injury (13).

The syndrome of autonomic dysreflexia is encountered often in patients with cervical or high thoracic spinal cord injury. High spinal cord lesions disconnect the central (medullary) autoregulatory mechanisms from the great bulk of capacitance and resistance vessels that ordinarily regulate blood pressure and flow in the body (Figure 1).

Clinical signs of autonomic dysreflexia include hypertension, tachycardia followed by bradycardia, hyperhidrosis above the lesion, and various visual signs. Symptoms include local flushing of the face and neck, piloerection, nasal congestion, and paresthesias. If hypertension is severe, headaches may occur along with transient or chronic neurologic deficiency, and rarely, acute cerebral hemorrhage.

The mechanisms underlying the autonomic dysreflexia syndrome are only partially understood. Cutaneous stimulation below the lesion or distention of hollow viscera may

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result in increased activity of sensory endings in these organs, which, in turn, results in sympathetic activation caused by stimulation of spinal reflexes resulting in blood pressure elevation and other symptoms (14).

Dysreflexia has reportedly been triggered by spontaneous or induced muscle spasm, range of motion exercises, and electrical stimulation during collection of semen for artificial insemination (15), spontaneous labor (16), pulmonary embolism (17), and during surgery (18). The syndrome seems to be influenced by increased activity in certain organs, innervated by visceral afferents, activation of which results in increased adrenergic activity. Regional vasoconstriction and venoconstriction result from these increased in adrenergic activity (2). Increases in peripheral vascular resistance combined with increased cardiac output lead to a sudden marked rapid increase in arterial blood pressure. Plasma norepinephrine levels rise immediately and are linearly related to the magnitude of blood pressure rise. It is known that patients with spinal cord transaction have an enhanced pressure response to norepinephrine. With the elevation in blood pressure, arterial baroreflexes are activated resulting in a reduction in heart rate, mediated through an increase in vagal tone. In contrast, sympathetic activity is not buffered or modulated because of the absence of descending influences to the interrupted spinal cord. Vasoconstriction, hypertensive
crisis, and other manifestations of sympathetic overactivity seen in autonomic dysreflexia are medicated via postganglionic release of norepinephrine with an alpha-adrenergic activation of vascular smooth muscle. Patients with spinal cord lesions below T6 do not manifest autonomic dysreflexia with cutaneous or visceral stimulation because descending tracts to the preganglionic sympathetic neurons that innervate a large part of the cardiovascular system remains intact.

Severe and prolonged episodes of autonomic dysreflexia may result in considerable morbidity and may occasionally lead to death. Appropriate management requires understanding of the complex reflex mechanisms that mediate the cardiovascular and noncardiovascular responses. The manifestations of the syndrome indicate excessive activation of the sensory endings in certain organs and may represent a warning sign of an underlying process (16,17).

The induction process should proceed slowly; the blood pressure should be manipulated by direct vasoactive inotropic and chronotropic drugs and infusion of intravenous fluids. Indirect acting sympathetic agonists and antagonist should be avoided (2,14).

The use of succinylcholine in SCI patients is well documented (2,7,6,8). After the first days of injury and up to and even after 18 months, the entire membrane of the muscle cell acts as a motor endplate. Succinylcholine depolarizes the entire membrane, and a large amount of potassium is released into the circulation, potentially causing ventricular fibrillation.

The initial approach to management of autonomic dysreflexia is the recognition of symptoms of the syndrome followed by identification and removal of the precipitating stimuli. This will often result in the termination of many of the symptoms.

The SCI patient can lose as much as 10 to 15% of his plasma volume during an attack of autonomic dysreflexia because of increased capillary filtration, so acute volume loading may be necessary. This will lead to an increase in total extracellular fluid that may require diuresis.

Autonomic dysreflexia is a serious clinical syndrome that is common in SCI patients. A stimulus or a combination of multiple stimuli often precipitates the clinical manifestations of the syndrome. Prompt recognition of the causative stimulus is critical to proper management of these patients. Acute hypertension associated with autonomic dysreflexia should be treated aggressively, while keeping in mind that it is difficult to limit increases and decreases in arterial pressure. Such drugs as alpha and beta-blockers will dilute the severity of symptoms by blocking massive sympathetic affects.

PULMONARY COMPLICATIONS IN SPINAL CORD INJURY

The most serious problem facing the SCI patients is respiratory failure in the acute phases and pneumonia and atelectasis in both the acute and chronic periods. Respiratory failure is defined as a condition requiring intubation and mechanical ventilation. Another common complication is pulmonary embolism in the presence of complete extremity paralysis (4,8). Factors other than muscle movement may be required to dislodge a deep vein thrombosis. Shifts in intrathoracic and intra-abdominal pressures create secondary effects in the venous circulation of the lower extremities.

The degree of respiratory impairment in SCI depends on the level of the normally functioning neurologic segment. Cervical lesions are associated with the greatest dysfunction. Low cervical lesions (C6–C8) effect abdominal muscles, intercostal and some accessory muscles (scalene), while leaving intact the diaphragm, trapezi, and sternocleidomastoids. Lumbar injuries cause little if any compromise. Lesions affecting the thoracic cage will cause paralysis of intercostal and abdominal muscles.

Studies vary as to the degree of hypoxemia that is presenting quadriplegia; these differences represent the time past injury. Sinha and associates studied C5–C8 quadriplegic patients and found 52% had $\text{PaO}_2 > 80 \text{mmHg}$, 38% 60–80 mmHg, 8% 40–60 mmHg, and 2% <40 mmHg (3).

Possible etiologies of hypoxemia include ventilation/perfusion mismatch, shunting secondary to atelectasis, cardiogenic or noncardiogenic pulmonary edema, or, as in cases of acutely injured patients, there can be other associated injury, such as lung contusion or hemothorax (3,4).

ENDOCRINE—METABOLIC COMPLICATIONS IN SPINAL CORD INJURY

Spinal cord injury affects the endocrine system in several ways. Sympathetic denervation may directly modify the function of the adrenal medulla, the pancreas, and the renin–aldosterone complex. The physical inactivity found in quadriplegia results in muscle atrophy and fat deposition that impair the action of insulin. As a chronic disease process with many associated medical conditions such as renal failure and malnutrition, spinal cord injury may produce nonspecific effects on endocrine function.

The higher the lesions, the more profound the endocrine abnormality will tend to be. A complete high cervical cord lesion produces, in addition to paralysis of most of the voluntary musculature, sympathetic denervation of the pancreas, the adrenals, and the juxtaglomerular apparatus of the kidney, allowing unopposed parasympathetic activity. In contrast, a thoracic lesion preserves the motor activity and the sympathetic innervation of the pancreas, adrenal, and kidney remains relatively intact.

Endocrine secretion does not rely on an intact spinal cord, resulting in essentially normal basal and stimulated levels. Subtle abnormalities in the hypothalamic pituitary–adrenal complex such as abnormal adrenocorticotropic
hormone (ACTH) diurnal variations have been found in SCI. Hypercalcemia and hypercalcinuria may occur early in SCI as a result of immobilizations, not attributable to altered parathyroid hormone or vitamin D status (20).

**INFECTIOUS COMPLICATIONS IN SPINAL CORD INJURY**

Infection, such as pneumonia, is the major cause of morbidity and mortality in SCI patients (21). Sugarman and colleagues found that fever, infection, or both occurred at some time during hospitalization in 67% of the patient groups followed (21). Because of advances in antibiotic treatment of infection, mortality from infection has decreased; however, infection remains the major cause of hospitalization of SCI patients.

Spinal cord injury does not directly affect the immune system (20). During the acute phase of SCI nutritional demands are increased and malnutrition may result in impaired cellular and antibody responses to infection. In addition, renal failure may result in a decreased immunological response to infection.

In persons with cervical or high thoracic cord lesions, there is a marked reduction in the ability to cough and a reduction in chest expansion during breathing. Decreased capacity to clear secretions predisposes them to pneumonia. Inability to void results in urinary stasis that promotes bacterial growth.

**PHARMACOKINETIC AND PHARMACODYNAMIC ALTERATIONS CAUSED BY SPINAL CORD INJURY**

Pharmacokinetics is the mathematical relationship that exists between the dose of the drug and the concentration of the drug in a readily accessible site in the body, usually blood or plasma. Clinical pharmacokinetics involves the application of pharmacokinetic principles and data to the individual and optimization of drug dosing requires. Pharmacological therapy is directed toward reducing the afferent nerve activity that triggers the spinal reflexes, blocking, and ganglionic activity, and blocking effector mechanisms. For example, calcium channel blockers, which cause vasodilatation, may be used. Directly acting vasodilators that influence blood pressure control are useful in the treatment of severe crisis condition (2,9,10,19).

Many drugs used in the treatment of SCI patients and their subsequent pharmacokinetic behavior have never been studies in a SCI population. Currently, SCI patients are having their drug dosages adjusted using homograms and recommendations developed in non-SCI patients (11). It is important to be aware of these changes, particularly when using potentially toxic drugs with narrow therapeutic indices.

Changes in pharmacokinetics may or may not lead to changes in the response to the drug. The known and potential effects of SCI on drug pharmacodynamics may ultimately be found to have more clinical significance that changes in pharmacokinetics. There is potential for clinically significant changes in drug concentrations and/or drug response in SCI.

**DISCUSSION**

Chronic spinal cord injured patients have disorders of many organ systems, and the alteration to normal physiology caused by SCI is widely recognized. These disorders combined with the pathophysiology of cardiopulmonary bypass present a unique challenge to the perfusionist.

Sympathetic denervation in the SCI patient may directly modify the function of the adrenal medulla, the pancreas, and the renin–aldosterone complex. The higher the lesions, the more profound the endocrine abnormality will tend to be. A compete high cervical cord lesion produces, in addition to paralysis of most of the voluntary musculature, sympathetic denervation of the pancreas and the adrenals and the juxtaglomerular apparatus of the kidney, allowing unopposed parasympathetic activity. Plasma renin activity and angiotensin II are elevated in SCI patients as a result of removal of the inhibitory effects of the sympathetic nervous system. These patients may have a tendency toward retention of sodium and wasting of potassium (20). Cardiopulmonary bypass results in an increase in serum catecholamine levels resulting from the stimulation of both the carotid sinus and aortic baroreceptors. Hypothermia also seems to be a major stimulus for catecholamine release and can adversely affect postoperative hemodynamics and increase oxygen consumption during rewarming (22). Vasopressin levels increase considerably during CPB because of the decreased stimulus of baroreceptors. Renin secretion from the kidney is also increased in response to a decreased mean arterial pressure and left atrial pressure. This renin secretion in turn leads to angiotensin activation and subsequent aldosterone secretion. The increase of catecholamine vasopressin and angiotensin II combine to increase blood pressure.

Hypotension is common after the initiation of bypass and has been attributed to several factors (23); 1) flowrate is usually established at 2.4 L/m², which is less than normal perfusion; 2) elevated histamine levels have been shown when CPB is initiated and hypotension attributable to vasodilation is a known response to histamine; 3) activation of the alternative complement pathways; and 4) a sudden decrease in systemic vascular resistance caused by abrupt hemodilution, which affects blood viscosity. Hypotension was experienced in this patient, with the mean arterial pressure being elevated over a period of time with pharmacological intervention. This prolonged low arterial
blood pressure was thought to be the result of extensive loss of muscle tone, initiation of CPB, and the lack of spinal cord reflexes. Activated neutrophils may injure endothelial cells and subsequently, lead to an increase in microvascular permeability during routine CPB (22). Venous return is impaired if blood volume is not adequately replaced, leading to decreased arterial flow. Tissue perfusion is not optimal if this occurs and can lead to metabolic acidosis. Decreased water content of plasma has been observed during CPB.

Fluid management in a SCI patient during CPB is difficult. This patient was anemic before CPB, and a critical hematocrit level was observed at the onset of CPB. The addition of one unit of packed red blood cells and hemoconcentration throughout the bypass procedure enabled the hematocrit to return to an acceptable level. If an autonomic dysreflexic episode had occurred during CPB, his plasma volume could drop rapidly and require acute volume replacement. The volume replacement must be done remembering that not to increase the already hemodiluted state, but also to maintain proper hemodynamics.

High cord transection, above T1, prevents the action of the effector mechanisms of temperature control in two ways. By damaging the connections between the temperature-regulating centers of the hypothalamus and the sympathetic outflow from the cord, vasoconstriction and sweating are prevented and vasodilatation occurs (2,8,10). In addition, damage to the motor tracts prevents shivering except in nonparalyzed muscles. In a cold environment, such as an operating room, the SCI patient may not be able to conserve heat and become hypothermic. In a warm environment, the SCI patient may not be able to dissipate body heat loss by increased vasodilatation and, therefore, becomes hyperthermic. Care must be taken to provide a stable environment for the SCI patients. The operating room was warmed when the patient entered and again before discontinuing CPB. Normothermic bypass was carried out with the patient core temperature maintained at 37–37.5°C. SCI patients with high cord lesions, such as this presented case, experience body temperatures greatly influenced by the environment.

In the presence of end-stage renal disease, SCI patients can be hemodialyzed using essentially the same protocol as for ambulatory patients on CPB requiring hemodialysis. The SCI patient presented in this case suffered from renal insufficiency and was not a hemodialysis patient except for the episode of acute renal failure he suffered during his hospital admission 2 months previous to this valve surgery. It was felt that his renal failure had been resolved and hemoconcentration during CPB was performed successfully.

Cerebral blood flow metabolism during many pathological conditions and during CPB can become altered from that in the normal person. Hypoxemia and more importantly hypercarbia increase cerebral blood flow (24). Carbon dioxide reactivity is mediated by cerebral spinal fluid (CSF) pH variations; whereas, cerebral lactic acidosis leads to abolishment of cerebral blood flow (24). Although autoregulation and changes in arterial PCO2 occur rapidly, regulation of the SCF pH can take up to 24 hours. Every attempt was made to maintain the preoperative PCO2 level in this patient for the reasons just described. This was difficult to maintain because of the high efficiency of the oxygenator. No supplemental carbon dioxide was bled in to the gas flow, and the increased PCO2 was a direct result of decreased sweep gas flow rate.

CONCLUSION

Many articles and textbooks have been written on the subject of spinal cord injury and its resulting effects on the human body; however, cardiac surgery utilizing cardiopulmonary bypass in a patient suffering from SCI is currently undocumented in the literature. Autonomic dysreflexia is a potentially life-threatening complication that may develop during surgery in this patient population. Chronic spinal cord injured patients have disorders of multiple organ systems and present a unique challenge to the cardiothoracic surgical team. Extensive discussion preoperatively should occur between team members and include a thorough historical review of patient injury. Increased awareness of the complications that can occur with the SCI patient can greatly decrease negative outcomes.

REFERENCES