# Management of High Spinal Injury: An Anaesthetic Riddle

*Sukanya Mitra, MD**
*Vinod Kumar Grover, MD, MNAMS***

**Cord injured patients pose unique difficulties, autonomic dysreflexia merits special concern. A sixty six year female patient with a history of compressive myelopathy at the level of $T_1$-$T_2$ was placed for anterolateral decompression under general anaesthesia. During intraoperative period the patient developed several episodes of bradycardia and hypertension. Towards the end of surgery she developed hypothermia and hypotension needing the support of vasopressors. On the 3rd postoperative day she developed left sided collapse of the lung, which responded to chest physiotherapy and mucolytics. This case report demonstrates several issues confronting the anaesthetist while dealing with patients of high spinal cord lesion. An adequate knowledge and quick anticipation of complications can however avert a major disaster.**

***Bahrain Med Bull 1999:21(1): 33-34***

Surgery on the spine and spinal cord makes up approximately 60%of the neurosursical procedures[1]. Injury to spine and spinal cord often lead to pathophysiological changes not only involving the neuromuscular system but also cardiovascular, respiratory, thermoregulatory and genitourinary systems[1,2]. Thus anaesthesia in the cord injured patients poses unique difficulties, autonomic dysreflexia merits special concern, as it brings forth a dilemma in the anaesthetic management of these patients[3]. We present such a case report.

## THE CASE

A 66 years female, weighing 66 kg belonging to ASA physical status II presented with a history of compressive myelopathy at $T_1$-$T_2$ level and was posted for elective anterolateral decompression. Her preoperative history was unremarkable except for the history of paraparesis of both lower limbs for last 20 days. Her preoperative investigations were within normal limits except that her pulmonary functions revealed mild restrictive defect.

The patient was premedicated with tablet diazepam 5mg a night before and 5 mg on the morning of surgery. She also received morphine 7.5mg, glycopyrrolate 0.2mg and promethazine 25mg IM one hour before surgery. In the operating room the patient was connected to noninvasive blood pressure monitor (NIBP), ECG, pulse oximeter and Capnogram and her baseline parameters were recorded. Anaesthesia was induced using sleep dose of thiopentone and morphine 6mg. Trachea was intubated 4 minutes following pancuronium 8mg with a 7.5 mm cuffed endotracheal tube. Lungs were ventilated using Bain's anaesthetic system and anaesthesia was maintained with 0.4-0.6% isoflurane and supplemental doses of pancuronium.

During the intraoperative period patient had several episodes of hypertension and bradycardia. Her blood pressure increased up to 220/120 mmHg and her heart rate dropped to 48 beats/min. During these episodes of hypertension and bradycardia surgical manipulation was stopped and blood pressure was controlled using isofluorane 1.5-2%. Intravenous glyceryl trinitrate was started at the rate of 2 µg/kg/min and the drip rate was titrated according to the pressor response. Surgical procedure lasted four hours and during this period patient received 2 units of blood (700 ml) and 3 litres of balanced salt solution against an estimated blood loss of 1000 ml. At the end of surgery patientís temperature dropped to 35°C and pulse oximeter showed signs of poor peripheral perfusion (reduced plethysmographic wave form) and her $EtCO_2$ also decreased. Ambient temperature of the operating room was immediately raised and the patient was infused warm IV fluids. After 2 hours the patient's body temperature increased to 37°C, but a fall in blood pressure to 80/60 mmHg was observed. At this time patient had CVP of 12 cm H20 and an adequate urine output. Another unit of blood (350 ml) was transfused and a litre of normal saline was quickly infused. Dopamine infusion was started at rate of 5µg/kg/min which was subsequently increased to 8 µg/kg/min.

Patient was then shifted to Intensive Care Unit and was connected to a Evita-2 (Drager) ventilator using $FiO_2$ of 0.4, tidal volume 700 ml, RR 12/min. ABG after 30 minutes of ventilation showed base deficit - 8 which was corrected using 80 ml of sodabicarb. Patient was weaned off ventilator using SIMV mode and was extubated 24 hours postoperatively. Once the blood pressure stabilised dopamine was reduced to 4 µg/kg/min and finally weaned off in next 48 hours.

* Senior lecturer
Department of anaesthesia
Government Medical College
** Additional Professor
Department of anaesthesia
Postgraduate Institute of Medical Education and Research
Chandigarh, India

On the 3rd post operative day patient had dyspnoea with difficulty in coughing out sputum. Her X-ray chest revealed collapse of left lung which was treated with physiotherapy, antibiotics, mucolytics and bronchodilators. Subsequent course of her recovery was uneventful and she was discharged from ICU on 5th PO day.

## DISCUSSION

The pathophysiologic sequelae of spinal cord injury involves not only the neuromuscular system, but also cardiovascular, respiratory, thermoregulatory and genitourinary systems[1]. Autonomic dysreflexia is a pathological sequelae in spinal cord lesions above $T_7$[1,2]. Our patient presented with compressive myelopathy at the level of $T_1$-$T_2$. She had no history of headache, flushing or fluctuations in blood pressure suggestive of autonomic dysfunction[2]. During the intraoperative period she developed several episodes of hypertension and bradycardia, requiring the support of vasopressors. Thus, autonomic dysreflexia can manifest intraoperatively without any previous history suggestive of the same.

Treatment of such an episode should always begin with the removal of the precipitating stimulus and by pharmacological intervention[3,4]. In our patient the surgical stimulus was temporarily stopped and hypertension was managed by increasing the concentration of isoflurane and glyceryl trinitrate infusion.

Induction of general anaesthesia in these patients is fraught with pit falls; too little anaesthesia and, hypertensive crisis may be initiated, too much anaesthesia and hypotension may occur[3]. Basal low sympathetic outflow resulting in instability of vascular tone, increased venous capacitance, decreased venous return to heart[6] may trigger a precipitous drop in blood pressure on induction. The hazardous use of succinyl choline in these patients is well documented[7,8]. Thus, in our patient anaesthesia was induced with sleep dose of thiopentone and morphine, while muscle relaxation for endotracheal intubation was facilitated with IVpancuronium.

Disruption of the sympathetic pathways carrying temperature sensation and loss of vasoconstriction below the level of injury cause spinal cord injured patients to be poikilothermic. Temperature is difficult to control in poikilothermic[9] patients. Heat conservation can be achieved using warming blankets, heated humidification of gases, by increasing ambient temperature and infusing warm intravenous solutions[3]. However the temperature dropped to 35°C in our patient inspite of all precautions.

These patients often have a reduced blood volume, some times as low as 60 ml $kg^{-1}$. The changes in their autonomic function often lead to an abnormal response to valsalva test[10]. There is often a tendency in overshooting of blood pressure when such pressure is released. Towards the end of anaesthesia, our patient also developed hypotension and needed vasopressors for maintenance of blood pressure. These could have been possibly triggered by abnormal valsalva response, during 'bucking' on the endotracheal tube. Other contributory factors might have been the reactive vasodilatation following correction of hypothermia and inadequate release of norepinephrine[3].

Patients with lesions between $C_5$ and $T_7$ will suffer significant alterations in respiratory function owing to the loss of abdominal and intercostal support. The indrawing of flaccid thoracic muscles during inspiratory phase produces paradoxical respiration and vital capacity is reduced by 60 percent[1]. Inability to cough and effectively clear secretions result in atelectasis and infection. Thus, atelectasis, bronchial occlusion, and pneumonia may occur postoperatively[1,11]. Treatment consists of chest physiotherapy, postural drainage, mucolytic agents, and humidified oxygen, in case of active problems in respiratory system[3,11].

## CONCLUSION

**In conclusion, this case report demonstrates several issues confronting the anaesthetist while dealing with a patient with high spinal cord lesion for surgery. An adequate knowledge and quick anticipation of impending complications or problems can thus go a long way in averting a major disaster.**

## REFERENCES


1. Horlocker TT, Cucchiara RF, Ebersold MJ. Vertebral column and spinal cord surgery. In: Cucchiara RF, Michenfelder JD, eds. Clinical Neuroanaesthesia. Churchill, Livingstone: New York, 1990:325-51.
2. Hambly PR, Martin B. Anaesthesia for chronic spinal cord lesions. Anaesthesia 1998;53: 273-89.
3. Amzallag M. Autonomic hyperreflexia. Int Anaesth Clin1993;31:87-102.
4. Colachis SC. Autonomic hyperreflexia with spinal cord injury. J Am Paraplegia Soc 1992;15:171-86.
5. Schonwald G, Fish KJ, Perkash I. Cardiovascular complications during anaesthesia in chronic spinal cord injured patients. Anesthesiology 1981;55:550-8.
6. Mathias CJ, Frankel HI. Cardiovascular control in spinal man. Annu Rev Physiol 1988;50:577-92.
7. Gronert GA, Theye RA. Pathophysiology of hyperkalemia induced by succinylcholine. Anesthesiology 1975; 43:89-99.
8. Tobey RE. Paraplegia, succinylcholine and cardiac arrest. Anesthesiology 1970;32:359-64.
9. Desmond J. Paraplegia: problems confronting the anaesthesiologists. Can Anaesth Soc J 1970;17: 435-57.
10. Dismond JW, Laws AK. Blood volume and capacitance vessel compliance in the quadriplegic patient. Can Anaesth Soc J 1974;21: 421-6.
11. Giffen JP. Anaesthesia for patients with spinal cord injury : acute and chronic.42nd Annual American Society of Anaesthesiologists Refresher Course Lectures 1991;152:1-7.