

**Q15 Outline the respiratory and cardiovascular consequences of an acute complete spinal cord transection at C6 (Sept 2012)**

Complete spinal cord transection at C6 → complete interruption of ascending and descending pathways at that level.

**CARDIOVASCULAR CONSEQUENCES:**

- Immediately post injury → early massive sympathetic stimulation resulting from catecholamine release from the adrenal medulla causing hypertension.
- Minutes later → spinal shock resulting from loss of sympathetic activity below the lesion. The result is physiologically similar to spinal anaesthesia and leads to
  - Vasodilatation and fall in TPR
  - Venodilatation, pooling and reduction in venous return
  - Fall in cardiac output and BP
  - Bradycardia due to unopposed parasympathetic activity, bradyarrhythmias, AV nodal block
- Weeks to months →
  - Reflex activity may return below the level of the lesion over a period of days to weeks
  - Patient is now at risk of autonomic dysreflexia → somatic or visceral stimulation below the level of the lesion (eg, a full bladder, constipation) results in massive sympathetic stimulation below the level of the lesion. Intense vasoconstriction causes a rise in SVR and BP. Interrupted descending pathways are unable to inhibit the sympathetic stimulation so parasympathetic compensation occurs with bradycardia. Compensatory vasodilatation above the level of the lesion causes flushing, headache and diaphoresis.
  - Decreased muscle pump activity and venodilatation increases the risk of VTE.

**RESPIRATORY CONSEQUENCES:**

- Phrenic nerves arise from the C3,4,5 nerve roots, hence a lesion at C6 will preserve phrenic nerve function.
- Lesions below C6 cause variable weakness of the internal and external intercostal and abdominal wall muscles due to interruption of spinal nerves below that level, resulting in paradoxical respiration (indrawing of flaccid intercostal muscles during inspiration). Consequently:
  - Forced expiratory lung volumes such as FRC and FVC fall
  - The reduction in FRC impairs gas exchange and causes V/Q mismatch
  - Maximum insp/exp pressures fall
  - Vital capacity is reduced to ~1500ml
  - Tidal volume is reduced up to 60%
- The overall effect is severe hypoventilation producing type 2 respiratory failure with severe hypoxaemia and hypercapnoea
- The inability to cough and clear secretions leads to atelectasis and pneumonia
- The absence of SNS activity can cause bronchoconstriction