Neurological complications following regional anaesthesia in obstetrics
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Neurological complications may complicate regional anaesthesia. The incidence has been reported as 0–36 per 10 000 blocks after epidural anaesthesia and 35 per 10 000 blocks after spinal anaesthesia. The most common complication is a reversible neuropathy due to direct needle/catheter trauma or intraneural injection of local anaesthetic presenting as a radiculopathy involving a single spinal nerve root. In a prospective study of over 10 000 obstetric epidural anaesthetics, there were problems with insertion in 13% of cases. The only neurological sequelae reported was one case of neuropathy. A retrospective postal survey in the UK reported a total of 56 cases of prolonged neurological sequelae following spinal or combined spinal-epidural anaesthetics, of which 18 could be attributed to the regional procedure, 29 were of uncertain origin and 9 could be attributed to other factors. The incidence of neurological sequelae in this study was estimated as being around 1 in 1000.

It is recognised that under-reporting may occur and the situation is complicated in that neurological dysfunction may also occur co-incidentally or in association with the process of childbirth. Indeed, transient neurological dysfunction in the postpartum period occurs in up to 20% of women, although this is only clinically important in 1 in 500. Any neurological complication in the postpartum period (Table 1) requires careful assessment and, in the majority of cases, reassurance and explanation is all that will be needed. Occasionally, urgent investigation and treatment is required in consultation with a neurologist. Post-dural puncture headache and its differential diagnosis has been discussed recently in this journal (see key references) and will not be considered further.

Key points
Neurological problems in the postpartum period are most commonly due to obstetric causes.
Nerve root damage can be caused by direct trauma from a needle or catheter.
The conus medullaris terminates at a variable level and Tuffier’s line is not reliable.
Serious neurological sequelae are rare but need urgent assessment.
An epidural haematoma or abscess requires early appropriate treatment to prevent permanent neurological sequelae.

Fig. 1 Nerves and nerve trunks which may be involved in compression injuries. [Reproduced with the kind permission of Blackwell Science from Holdcroft A, Thomas TA. In: Principles and Practice of Obstetric Anaesthesia. 2000, Oxford: Blackwell Science.]
Peripheral nerve lesions

The process of childbirth may lead to a maternal nerve palsy, usually associated with prolonged labour and the use of forceps. The anatomy of the nerves arising in the pelvis is shown in Figure 1. The majority of maternal nerve palsies are due to neuropraxia and recovery can be expected within 3 months. However, if there has been disruption of the nerve (axonotmesis), full recovery of long nerves may take 3 years.

Lateral cutaneous nerve of thigh

The lateral cutaneous nerve of thigh can be injured at several points along its course but most often as it passes under the inguinal ligament. During the second stage of labour, there is an increase in intra-abdominal pressure and abdominal wall tension leading to compression of the nerve. The lateral cutaneous nerve of the thigh is a purely sensory nerve (L2 and L3) and damage is characterised by numbness on the anterolateral aspect of the thigh. Symptoms may also occur in pregnancy as the exaggerated lumbar lordosis stretches the nerve making it more susceptible to trauma. Symptoms usually resolve within 3 months of delivery.

Postpartum foot drop

Postpartum foot drop is caused by damage to the lumbosacral nerve trunk or, less frequently, the common peroneal nerve. The lumbosacral trunk (L4 and L5) is compressed between the ala of the sacrum and the descending fetal head. It may also occur during a forceps delivery. Typically, it occurs in a mother of short stature with a large baby. The result is a unilateral foot drop with loss of sensation and/or paraesthesia along the lateral calf and foot. Common peroneal nerve damage may occur due to improper or prolonged positioning during lithotomy and sensory deficit may be limited to the dorsum of the foot. Nerve conduction studies are required to identify the site of neural damage with any certainty.

Femoral neuropathy

Pressure from the fetal head in the pelvis may damage the femoral nerve (L2–L4) and this neuropathy also occurs more often after forceps delivery. It may also occur after Caesarean delivery where the use of retractors may compress the nerve. The woman will have problems climbing stairs due to loss of hip flexion and knee extension. Sensory loss will be over the anterior aspect of the thigh and the anterior and medial side of the leg. An absent or reduced knee reflex is the most reliable sign of femoral neuropathy.

Obturator nerve palsy

The obturator nerve (L2–L4) may also be damaged during forceps delivery. In this case, there will also be sensory loss over the medial side of the thigh and loss of adduction of the hip joint. The femoral nerve may also be damaged at the same time.

Nerve root damage

This is caused by direct needle/catheter trauma or by intraneural injection of local anaesthetic. Painful paraesthesia is often (but not always) elicited during the epidural or spinal procedure. It usually presents with skin hypoaesthesia in the dermatome supplied by the nerve root and occasional muscle weakness (Table 2).

The area of sensory deficit differs depending on whether there is damage to a nerve root or to a peripheral nerve. An epidural or spinal needle may traumatise a nerve root but peripheral nerve damage is almost certainly due to obstetric causes. If symptoms are caused by trauma to a nerve root, the woman may be re-assured that major symptoms usually resolve in weeks, although complete recovery may take months or longer. Occasionally, a chronic pain syndrome can result and follow-up after discharge from hospital is essential.

Mild paraesthesia often occurs during the insertion of an epidural/spinal needle or catheter but is not usually followed by

| Table 1 Neurological complications following regional anaesthesia in obstetrics |
|-------------------------|------------------|
| Neurology               | Causes           |
| Peripheral nerve injury | Obstetric palsies|
| Nerve root/cord damage  | Needle or catheter trauma |
| Cranial nerve palsy     | CSF leakage      |
| Spinal cord compression | Epidural haematoma|
| Meningitis              | Infective        |
| Cord ischaemia          | Anterior spinal artery syndrome |
| Cauda equina syndrome   | Compression      |
| Arachnoiditis           | Mainly chemical, now rare |

| Table 2 Neurological deficit following nerve root trauma |
|-------------|------------------|
| Root        | Sensory loss     | Motor weakness  |
| L2          | Upper anterior thigh | Hip flexion   |
| L3          | Lower anterior thigh, medial thigh | Thigh adduction |
| L4          | Lateral thigh, knee, medial leg | Leg extension |
| L5          | Lateral leg, dorsum of foot | Ankle dorsi-flexion |
| S1          | Lateral foot     | Ankle plantar flexion |
neurological sequelae. However, an epidural catheter should be removed if paraesthesia persists. Injection of local anaesthetic should never be performed in the presence of pain or paraesthesia.

Lumbar disc prolapse may occur in the peripartum period, usually at L4/L5 or L5/S1. As well as the signs of nerve root irritation, this classically presents with backache and pain down the lateral aspect of the thigh.

**Spinal cord damage**

When performing a spinal anaesthetic, the injection should occur below the level of the conus medullaris. Cases of conus damage following spinal anaesthesia have been reported, as the conus medullaris does not always terminate at the lower border of L1. In 2–20% of individuals it ends at the lower border of L2, more commonly in women. Even senior anaesthetists may underestimate the interspace where injection occurs. Tuffier’s line may cross the midline at L3/L4 not L4/L5 and is not a reliable indicator. Therefore, before attempting spinal anaesthesia, it is prudent to choose the lowest possible interspace and avoid using the L2/L3 interspace (or higher).

**Cranial nerve palsies**

The aetiology of cranial nerve palsy is usually CSF leakage following dural puncture. Thus it is more likely to occur after dural puncture with a Tuohy needle. Any cranial nerve may be affected but sixth nerve palsy is the most common, presenting with diplopia. Resolution is usually spontaneous (within weeks) but patching the affected eye in the interim may be required. Early blood patching for post-dural puncture headache may help prevent cranial nerve palsies but, once established, the response to patching is poor.

Facial nerve palsy also occurs during pregnancy because of oedema in the facial canal. The incidence is greatest in the third trimester, though some cases have been reported in the first 48 h after delivery. Complete recovery usually occurs within 6 weeks. Epidural blood patching may occasionally precipitate a facial nerve palsy. The suggested mechanism is that the increase in CSF pressure compromises an already oedematous nerve.

**Epidural haematoma**

This is a rare but potentially devastating complication. Epidural haematomas can occur spontaneously in pregnancy with no apparent predisposing factors. However, there is an increased risk in patients with a bleeding tendency. Therefore, before a regional block is sited, it is important to assess the potential risks and benefits. Regional blockade should never be performed in the presence of full anticoagulation or within 10 h of an injection of a low molecular weight heparin.

Similarly, following a regional procedure or removal of an epidural catheter, there must be a delay before heparin is given. Each unit should have clear, written guidelines regarding the timing of anticoagulation and thromboprophylaxis in relation to the timing of regional anaesthesia and epidural catheter removal.

Thrombocytopenia occurs in pregnancy for several reasons. In general, a woman with a platelet count > 80 × 10^9 litre⁻¹ in the absence of pre-eclampsia is unlikely to have abnormal platelet function. Even in pre-eclampsia, a platelet count > 80 × 10^9 litre⁻¹ is not a contra-indication as the benefits of regional analgesia usually outweigh the risks. Unfortunately, the platelet count itself is a poor predictor of bleeding tendency. Bleeding time is operator dependent and thrombo-elastography is not yet in routine clinical practice.

An epidural haematoma should be suspected if the effects of epidural anaesthesia persist for > 8 h after the last dose of local anaesthetic and if there is backache and local tenderness. Although epidural or spinal anaesthesia may occasionally be prolonged, delayed recovery should always be followed up. The presence of a bilateral distribution of motor and sensory abnormalities and/or disturbance of bowel or bladder function should arouse suspicion of a catastrophic lesion. Urgent imaging is required and, if a haematoma is present, emergency decompression is indicated as soon as possible. Any delay increases the likelihood of permanent neurological sequelae.

**Epidural abscess**

Epidural abscesses are rare (< 4 per 100 000) but do occur after epidural anaesthesia and can have serious consequences. However, they may also occur spontaneously in the absence of regional anaesthesia. Epidural abscesses may arise from endogenous bacteria circulating at the time of childbirth but infection may also be introduced from an external source. *Staphylococcus aureus* is the most common infecting organism in both spontaneous and epidural induced abscesses. The risk increases with the duration of epidural catheterisation.

Presentation is similar to that of epidural haematoma; the most consistent symptom is backache with localised tenderness. Pyrexia and systemic symptoms of sepsis may also be present. Typically, after 3 days of back pain, the patient will develop nerve root pain followed rapidly by lower limb weakness and
then paraplegia. An epidural abscess needs urgent treatment with intravenous antibiotics and aggressive surgical management. Neurological deterioration prior to surgery carries a poor progress despite the use of intravenous antibiotics.

**Meningitis**

This may occur after spinal anaesthesia with an incidence of up to 1.5 in 10 000 blocks. The infecting organism in iatrogenic meningitis is different from those found in meningitis in pregnancy in the absence of regional anaesthesia. In the latter case, the infecting organism is usually *Streptococcus pneumoniae* or *Neisseria meningitidis* whereas in iatrogenic meningitis *Streptococcus viridans* is more likely. In one non-obstetric case, the infecting organism was identical to that isolated from the throat swab of the doctor who had performed the lumbar puncture. Thus, it is important to observe strict asepsis, including the wearing of a face mask whenever performing spinal anaesthesia. The prognosis of meningitis is better than that of epidural abscess.

Aseptic (chemical) meningitis may also occur but is rare nowadays. It is caused by the injection of contaminated local anaesthetic. It produces a syndrome of fever, headache, neck stiffness and photophobia that resolves spontaneously. The contaminants in past reports were residual traces of detergent on needles and syringes but, more recently, inadvertent contamination of epidural equipment with chlorhexidine spirit has been implicated. Differentiation from bacterial meningitis is difficult and it is prudent to prescribe antibiotics empirically in all cases.

**Anterior spinal artery syndrome**

The anterior spinal artery supplies approximately two-thirds of the spinal cord at all levels supplemented with radicular arteries arising from the aorta. However, there are ‘watershed’ areas of poor supply. This syndrome is usually associated with severe hypotension, arteriosclerosis and disturbance of aortic blood flow. It is extremely rare in obstetric practice but the combination of severe hypotension and local anaesthetic solutions containing epinephrine have been implicated.

**Cauda equina syndrome**

Smaller nerve fibres are the first to be affected by pressure, ischaemia or contact with toxic chemicals. Thus, the autonomic nerve fibres of the cauda equina are often the first to be involved. Damage to the S2–S4 sacral nerve roots leads to bladder atony and loss of voluntary control of micturition. This lower motor neurone lesion may progress upwards leading eventually to paraplegia. The sensory loss classically occurs in the ‘saddle’ distribution and low back pain may also occur. Onset of this syndrome immediately after spinal or epidural anaesthesia is usually due to compression and may result when inappropriately large volumes are injected into the epidural space. However, it may also be caused by the injection of neurotoxic chemicals and the use of spinal microcatheters with 5% lidocaine has been implicated.

**Arachnoiditis**

Arachnoiditis presents days, weeks or even months after regional anaesthesia as a gradually progressive weakness and sensory loss in the lower extremities. It is an inflammatory process and carries a poor prognosis despite surgery. It can progress to complete paraplegia. Causes of arachnoiditis include meningitis, spinal trauma and injection of neurotoxic chemicals. Many are idiopathic. Drugs containing preservative must never be used for neuroaxial anaesthesia and epidural catheter lines must be clearly labelled to prevent confusion with intravenous lines.

**Key references**


See multiple choice questions 80–84.