MANAGEMENT OF NEUROLOGICAL DEFICIT IN POSTPARTUM PATIENTS

Dr R.Bird, Dr S.K.Backe

The document deals with neurological deficits occurring below umbilicus. PostDural Puncture Headache is discussed elsewhere in the handbook.

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- Background
- Non-Anaesthetic causes
- Anaesthetic causes
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- Management of Neurological complications
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Background

The majority of neurological complications are due to compressive neuropathy as a result of prolonged labour or poor patient positioning or mode of delivery.

Incidence of neurological problems due to maternal and fetal variables are estimated to be 0.92%(3).

Incidence of permanent harm following Regional block is estimated to be 1 in 80000 to 1 in 320425(4).

The temporal association between anaesthetic intervention with neuraxial block and onset of neurological symptoms often means that anaesthetists are consulted early in the presentation of a neurological complication following childbirth.

When neurological complications do occur, prompt recognition and management can reduce the risk of permanent neurological deficit.
Therefore, despite the majority of nerve injury being related to factors other than regional anaesthesia/analgesia it is important to have knowledge of diagnosis, investigation and management of neurological injury.

### Non-anaesthetic causes

<table>
<thead>
<tr>
<th>Type of neuropathy</th>
<th>Incidence</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compressive Neuropathy</td>
<td>(1 in 100)</td>
<td>▪ Fetal head compressing lumbosacral trunk</td>
</tr>
<tr>
<td></td>
<td></td>
<td>▪ Positioning</td>
</tr>
<tr>
<td></td>
<td></td>
<td>▪ Instrumental delivery</td>
</tr>
<tr>
<td>Ischaemic neuropathy</td>
<td>1 in 500,000</td>
<td>▪ Prolonged hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>▪ Obstruction of Internal Iliac arteries by fetal head in prolonged labour</td>
</tr>
</tbody>
</table>
**Compressive neuropathy**

This is the most common postpartum neurological complication. It results often due to compression of nerves secondary to poor patient positioning, foetal head or instrumental delivery. There is **usually a unilateral sensory and motor deficit** which is related to the damaged nerve, although the **deficit can be rarely bilateral**, examples in table 1.

<table>
<thead>
<tr>
<th>Injury</th>
<th>Nerves affected</th>
<th>Common Causes</th>
<th>Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lateral cutaneous nerve of the thigh</td>
<td>L2-3</td>
<td>Compression of the nerve as it passes under the inguinal ligament</td>
<td>Sensory loss over the anterolateral aspect of the thigh.</td>
</tr>
<tr>
<td>Lumbosacral plexus</td>
<td>L4,5 S1-5</td>
<td>Compression of lumbosacral plexus against sacral ala. Usually from foetal head in second stage. It arises on the opposite side to the fetal occiput.</td>
<td>Numbness over lateral aspect of thigh, lower leg and dorsum of foot. Results in foot drop. (Weak ankle dorsiflexion and plantarflexion). The foot drop is almost always unilateral and on the opposite side to the fetal occiput resulting in weak dorsiflexion and</td>
</tr>
<tr>
<td>Common peroneal nerve due to peripheral nerve compression</td>
<td>L4-5, S1-2</td>
<td>Prolonged lithotomy position. The nerve is compressed as it passes over the head of the fibula (inappropriate positioning of the patient in stirrups).</td>
<td>Numbness over lateral aspect of lower leg and dorsum of foot, foot drop. Ankle reflex intact.</td>
</tr>
<tr>
<td>---</td>
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<td>---</td>
</tr>
<tr>
<td>Femoral nerve</td>
<td>L2-4</td>
<td>Compression of nerve against inguinal canal during forceps delivery or LSCS. Femoral neuropathy can occur bilaterally 25% of the time, is often mistaken for an intraspinal lesion.</td>
<td>Sensory loss over anterior thigh and inner aspect of lower leg. Weak knee extension. Often presents with difficulty climbing stairs. Loss of knee jerk. The reduced or absent patellar reflex is the most reliable objective sign in femoral neuropathy</td>
</tr>
<tr>
<td>Obturator nerve</td>
<td>L2-4</td>
<td>Compression of nerve by fetal head or forceps. Obturator neuropathy, which occurs bilaterally 25% of the time, is often mistaken for an intraspinal lesion.</td>
<td>Usually unilateral sensory loss over inner thigh and weak hip adduction and rotation.</td>
</tr>
</tbody>
</table>

**Differential Diagnosis of Foot Drop**

<table>
<thead>
<tr>
<th></th>
<th>L5 nerve root</th>
<th>Lumbar plexus</th>
<th>Sciatic involvement</th>
<th>Peroneal nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weakness of paraspinous muscles</td>
<td>Weakness of gluteal muscles and sphincter</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ankle inversion weak</td>
<td></td>
<td>Normal or weak</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>normal</td>
<td>normal</td>
<td>Normal or weak</td>
<td>Normal</td>
</tr>
<tr>
<td>--------------------------</td>
<td>----------------</td>
<td>-----------</td>
<td>----------------</td>
<td>--------</td>
</tr>
<tr>
<td><strong>Plantar flexion</strong></td>
<td>weak</td>
<td>weak</td>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Toe flexion</strong></td>
<td></td>
<td></td>
<td>Normal or weak</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Sensory loss</strong></td>
<td>Poorly demarcated predominantly big toe</td>
<td>Well demarcated to L5 dermatome</td>
<td>Lower 2/3 of lateral leg and dorsum of foot.</td>
<td>Lower 2/3 of lateral leg and dorsum of foot</td>
</tr>
<tr>
<td><strong>Ankle jerk</strong></td>
<td>normal*</td>
<td>normal*</td>
<td>Normal or weak</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td>Common, radicular</td>
<td>Common, may be radicular</td>
<td>Can be severe</td>
<td>Rare</td>
</tr>
</tbody>
</table>

¶ Attempt inversion with the foot dorsiflexed passively to 90°
* Can be weak with S1 involvement

The duration of symptoms for compressive neuropathies is 6 weeks to 2 months with symptoms in almost all patients resolving in this time.

Despite the myriad mechanical options for nerve injuries, good advice would be to change lower extremity position frequently during a prolonged second stage of labour, avoid prolonged thigh flexion, avoid extreme thigh abduction and external rotation, and minimize motor and inappropriately dense sensory block by using lower concentrations of local anaesthetic during regional anaesthesia in labour.
### Table 2. Types of nerve damage and recovery

<table>
<thead>
<tr>
<th>Types of Nerve Damage</th>
<th>Definition</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurpraxia</td>
<td>superficial type of nerve injury</td>
<td>Spontaneous recovery within weeks to a few months is the rule.</td>
</tr>
<tr>
<td>Axonotmesis</td>
<td>the axon is disrupted</td>
<td>Recovery (through axon regeneration) is generally achieved over several months or sometimes even 3 years (Howells 2013)</td>
</tr>
<tr>
<td>Neurotmesis</td>
<td>complete nerve transection</td>
<td>Spontaneous recovery is unlikely without surgical repair</td>
</tr>
</tbody>
</table>

**Anaesthetic Causes**

Neurological complications secondary to central neuraxial block (CNB) are as a result of damage to the spinal cord or nerve roots. This may be due to trauma, neurotoxicity, infection, ischaemia or haematoma. Important complications to be aware of and act upon are in table 3.

Central lesions, usually associated with anaesthetic interventions, are more commonly accompanied by back pain (supplied by posterior rami) as the anterior and posterior rami of the nerve root are affected.
## Anaesthetic Causes

### Table 3. Potential complications of central neuraxial block.

<table>
<thead>
<tr>
<th>Causes</th>
<th>Symptoms &amp; Diagnosis</th>
<th>Prevention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nerve damage</td>
<td>This causes paraesthesia, loss of sensation, and muscular weakness in the distribution of the nerve</td>
<td>Clearly avoiding contact with nerves</td>
</tr>
<tr>
<td>Secondary to direct needle or injection trauma disrupting the fibres of a single nerve.</td>
<td>Pain on needle insertion Prolonged motor and sensory weakness at and below the level of injury. Can be unilateral or bilateral. May have urinary symptoms.</td>
<td>If pain on needle insertion or injection of LA withdraw needle. Scanning of the back can identify the correct interspace</td>
</tr>
<tr>
<td>Spinal cord trauma</td>
<td></td>
<td>The conus medullaris usually ends at L1 but may extend to L2,3 in 10% of patients. L3,4 should be highest landmark.</td>
</tr>
<tr>
<td>Direct trauma when placing spinal, epidural or CSE.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| **Cauda Equina Syndrome** | Damage to cauda equina nerve fibres due to compression or trauma. | Backache  
Nerve root pain  
Saddle anaesthesia  
Paraplegia  
Sphincter dysfunction | There have been links made with use of hyperbaric lignocaine - avoid its use.  
Strict asepsis on epidural insertion.  
Observe cautions in patients with coagulopathies, bleeding disorders, and who are on anticoagulants. |
|--------------------------|-------------------------------------------------|----------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| **Arachnoiditis**        | Inflammation of the arachnoid meningeal layer and subarachnoid space. There have been associations made with this and needle contamination with chlorhexidine. | Variable presentation.  
Progressive symptoms of paraesthesia, numbness or leg weakness.  
Most common symptom is pain. | Use preservative free drugs  
Use low concentration (0.5%) chlorhexidine and ensure it is kept on a separate surface away from epidural or spinal needle and allow to dry on skin before needle insertion.  
Aagbi guideline reference-https://www.aagbi.org/sites/default/files/skin%20antisepsis%20for%20central%20neuraxial%20blockade_0.pdf |
| **Epidural Abscess**     | On insertion of epidural:  
• Prolonged catheter insertion  
• Presence of sepsis  
• Inadequate aseptic technique | Backache  
Nerve root pain  
Weakness  
Paralysis  
Fever  
Raised inflammatory markers | Caution in placement of epidural in infection, especially if pyrexial (WCC may be raised secondary to labour)  
Strict asepsis on epidural insertion |
| **Meningitis** | Complication of dural puncture  
Usually seen following spinal or CSE  
(incidence 1:10,000)  
Causative organism often streptococcus viridans | Headache  
Fever  
Backache  
Nausea  
Can be confused with PDPH | Asepsis on placement of central neuraxial block including use of facemask. |
| **Haematoma** | Mainly occurs in epidural space because of prominent venous plexus.  
Haematoma causes neural ischaemia due to | Back pain  
Nerve root pain  
Weakness  
Paralysis (late feature) | Risk factors:  
• Coagulopathy  
• Difficult CNB insertion  
• Anticoagulants  
Consider timing of regional block if on anticoagulants and |
<p>| compression |  | whether coagulopathy excludes patient from CNB |</p>
<table>
<thead>
<tr>
<th>History &amp; Documentation</th>
<th>Technique</th>
<th>Periprocedure care</th>
</tr>
</thead>
</table>
| Careful assessment before performing a regional block is essential. | Technique  
• Aseptic technique - handwash, hat, mask, gloves, gown  
• 0.5% chlorhexidine should be prepared away from epidural and spinal needles and be allowed to dry once applied to the skin.  
• A bacterial filter for drawing up and administering drugs for CNB  
• Avoid touching equipment that enters patient  
• Avoid prolonged epidural catheterisation  
Location of CNB - lowest palpable space. Above L3/4 should be avoided - it is well known that anaesthetists are inaccurate in identifying level, and are often one space higher than they think. | Prevent prolonged periods of hypotensive episodes to maintain spinal cord perfusion.  
Take care in patient positioning to avoid compressive neuropathy.  
Consent - Inform patient of risks of procedure and document risks discussed.  
All patients undergoing CNB should be reviewed following the procedure. Any neurological deficit should be promptly assessed by a senior anaesthetist. |
| Enquire about medical history  
• Document existing neurological deficit after careful examination  
• Anticoagulants / coagulopathy / thrombocytopenia - (see annexe2)  
• Infection/sepsis - (see annexe 1) - avoid neuraxial procedures in the septic patient or in the presence of local infection. Discuss with consultant on call where unsure.  
Prognosis depends on the degree of the initial neurological deficit and evidence of improvement within the first 24 h. | If persistent pain on insertion of needle, placement of catheter or injection of drugs - remove needle or discontinue drug administration. |
6. Management

REFER TO FLOWCHART BELOW

1. History

   i. Neurological - including conditions predisposing to neuropathy e.g. backache, obesity, disc
disease, diabetes, malignancy, coagulopathy, infection, previous trauma)

   ii. Labour/Mode of delivery - Instrumental delivery(type), posture during labour, use of
retractors or diathermy, period of full dilatation, injections given by obstetrician, and
hypotension.

   iii. Drugs - particularly anticoagulants, steroids, hypoglycaemics

   iv. Anaesthetic - type of block, degree of technical difficulty, possibility of inadvertent dural
puncture, bloody tap, spinal catheters, type/baricity/concentration of anaesthetic, additives,
details of aseptic technique, site of injection, pain/paraesthesia during procedure

2. Examination

   i. Physical examination including full neurological examination and examination of back.

To establish an accurate diagnosis and guide to treatment a detailed neurological examination
should take place followed by appropriate investigations where necessary.(Contact the medical
team if you don’t feel able to do a neurological assessment).

Dysfunction of lower extremity should be evaluated rapidly and limb or life-threatening aetiologies
excluded.

Deteriorating symptoms or onset after a symptom-free interval should be treated seriously. This
implies changing pathology such as increasing compression from an enlarging mass (i.e haematoma/
abscess).
3. Investigation

In event of sinister symptoms including acute onset back pain and radicular leg pain, urinary and anal dysfunction, and lower leg numbness and weakness it is essential to perform immediate MRI to exclude a central lesion. Rapid referral to Neurosurgery at the RVI may be necessary for decompression - permanent injury occurs between 6 and 12 hours from the onset of symptoms. Bear in mind that our spine surgeons may be more immediately available and able to perform the decompression sooner.

ROLE OF EMG(nerve conduction studies)- Arrange after discussion with on call Neurology SpR at the RVI, Newcastle
Electrophysiological investigations can differentiate between central and peripheral nerve injury, identify the muscles affected and give a likely prognosis of neural recovery. It may be able to identify the precise lesion site. They can also produce a temporal estimate as to the timing of the injury, which is of particular note with regards to litigation. However EMG only measures large nerve fiber changes and may take as long as 3 weeks after injury to show changes. An obstetric palsy will have EMG changes occurring distally to any point of anaesthetic intervention. Therefore, assessment within 72 h of neurological deficit is important to demonstrate preexisting pathologies as new electrophysiological changes take the time to evolve.

(Faecal incontinence: Faecal Incontinence can occur after Vaginal delivery even in those patients without perineal tears (2-5%) and is very rarely due to Epidural or Spinal. The common causes are sphincter damage or damage to the innervation of the sphincter during difficult vaginal delivery. In lower Lumbosacral plexopathy incontinence can even accompany perianal sensory disturbance. In any case MRI has to be done on all such patients to rule out compression by a space occupying lesion). Misoprostol is a prokinetic agent which can cause diarrhoea. It can therefore exacerbate faecal incontinence.
Potential neurological deficit identified to anaesthetist by patient / midwife / obstetrician

**History** - attention to neurology, drugs, labour and procedures in labour delivery

**Examination** - Physical examination and full neurologic examination including examination of back

**Anaesthetic** - review of technique - refer to dermatome map where necessary

**In hours** - Discuss with Obstetric Anaesthetic consultant

**Out of hours** - discuss with on call Anaesthetic consultant urgently if concerned about central pathology. Discuss in the morning if not urgent

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**Clinical Diagnosis suggestive of?**

**Central pathology:**
- Spinal Epidural Haematoma (SEH)
- Spinal Epidural Abscess (SEA)

1. Urgent consultant review
2. Refer for Urgent MRI

NSECH if 8am - Midnight

RVI Newcastle if Midnight - 8am

3. Inform Obstetric Consultant
4. Early liaison with Neurosurgical SpR, RVI Newcastle

If results suggest above pathology discuss with Neurosurgical SpR in Newcastle and anticipate early transfer

Document all findings and ensure referrals and transfers timely

Complete critical incident report

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**Suspected peripheral nerve damage**

1. Discuss findings with patient
2. Inform Obs/Anaes consultant
3. Discuss with on call Neurology SpR in Newcastle

If motor deficit or urgent may need MRI +/- nerve conduction studies (NCS)

If sensory deficit and non-progressive may need nerve conduction studies delayed by at least 3 weeks - (refer to Northumbria NCS service)

Follow up as deemed necessary

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**No evidence of neurological deficit**

Reassure patient of clinical findings

Arrange further senior anaesthetic review at 24 hours

Document all findings

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**Clinical Signs of Central Pathology**

SEH: Acute onset back and radicular leg pain, lower extremity weakness and numbness, urinary and anal dysfunction

SEA: Backache, localised tenderness, fever, raised WCC & CRP, headache, neck stiffness, late signs of loss of lower limb and sacral sensation, decreased reflexes, bladder dysfunction

Newcastle Hospitals switchboard: 0191 233 6161
Figure 1. Dermatomes of the lower limb and peripheral nerve supply distribution.
7. References


5. Howells, AC. Neurological complications in obstetric regional anaesthesia. Anaesthesia and intensive care medicine 2013;14(8):331-332