NEUROLOGICAL DEFICIT POSTPARTUM – DIAGNOSIS AND MANAGEMENT

This LOP is developed to guide clinical practice at the Royal Hospital for Women. Individual patient circumstances may mean that practice diverges from this LOP.

1. AIM

- Diagnosis and management of woman with a potential postpartum neurological deficit

2. PATIENT

- A woman who may or may not have had a neuraxial block for analgesia or anaesthesia in the peripartum period who:
  - describes postpartum numbness, weakness or significant back pain that was not present prior to delivery
  - has a pre-existing neurological injury which appears to have worsened

3. STAFF

- Medical, nursing and midwifery staff

4. EQUIPMENT

- Nil

5. CLINICAL PRACTICE

Refer to neurological deficit management flowchart (see Appendix 1)

Initial Assessment and Management

- Take a comprehensive history of the symptoms being experienced. This initial assessment may be done by midwifery/nursing staff, obstetric/anaesthetic senior or junior medical staff and should include:
  - antenatal neurology
  - onset of symptoms
  - medications
  - type and length of labour and
  - mode of delivery
- Notify treating obstetric team or on-call obstetric team (out of hours) of woman with symptoms
- Notify anaesthetic registrar (outside team) if woman has had a neuraxial block
- Check anaesthetic technique (if applicable) from obstetric anaesthetic intervention form, obstetric epidural chart, or anaesthetic chart, including complications on insertion
- Perform a full neurological examination including an examination of the back
- Consider any evidence of central pathology which include:
  - acute onset back pain particularly rapidly worsening (see Appendix 2)
  - localised back tenderness
  - radicular leg pain
  - lower leg numbness and weakness
  - decreased reflexes
  - loss of sacral sensation
  - headache
  - neck stiffness
  - urinary and/or anal sphincter dysfunction

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Referral

- Discuss woman with obstetric consultant
- Discuss woman with anaesthetic consultant if has had neuraxial block
- Refer for urgent magnetic resonance imaging (MRI) at Prince of Wales Hospital (POWH) if deemed necessary
- Liaise with neurosurgical registrar at POWH if necessary
- Discuss woman with obstetric medical registrar in hours if suspected peripheral nerve injury (see Appendices 2 and 3). The obstetric medical registrar can further discuss the woman with the neurology registrar at POWH if required. The neurology registrar to advise if woman needs consultation as an inpatient or outpatient and whether should be referred for nerve conduction studies

Ongoing assessment

- Review woman again the following day, reassessing whether symptoms have improved or not
- Reassure woman if clinical findings suggest non-specific neurological injury these are expected to improve
- Ensure ongoing (post discharge) management by obstetric medical and/or neurology teams if suspected peripheral nerve injury

6. DOCUMENTATION

- Medical record

7. EDUCATIONAL NOTES

Background

- Most neurological complications are due to compressive neuropathy because of prolonged labour, poor patient positioning or mode of delivery. The incidence of neurological problems due to maternal and fetal variables are estimated to be 0.92%[1] and the incidence of permanent harm following neuraxial anaesthesia is estimated to be 1:80000 to 1:320425[2,3]. However, despite most nerve injuries being related to factors other than neuraxial analgesia, when serious neurological complications do occur, prompt recognition and management can reduce the risk of permanent neurological deficit
- Be aware that urinary and faecal incontinence can commonly occur after vaginal delivery even in those patients without perineal tears (2-5%) and are very rarely due to a complication of an epidural or spinal. The common causes are ongoing action of neuraxial anaesthesia, ongoing hormonal effects, any birth canal trauma including direct anal sphincter damage or damage to the innervation of the anal sphincter during a difficult vaginal delivery

Causes of postpartum neurological deficit

- Maternal and fetal variables (see Appendix 2 and 4)
    - Compressive neuropathy: this is the most common postpartum neurological deficit due to poor maternal positioning, fetal head, or instrumental delivery. Commonly a unilateral sensory and motor deficit is noted, although rarely it can be bilateral.
    - Ischaemic neuropathy

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- Anaesthetic variables (refer to Appendix 5)
  - Direct trauma to nerve, spinal cord, or cauda equina
  - Arachnoiditis
  - Epidural abscess
  - Meningitis
  - Epidural haematoma

Investigation

- A full blood count (FBC) and C-reactive protein (CRP) may help determine where there is an infective cause.
- In event of sinister signs and symptoms together suspicious of central pathology, it is essential to perform immediate MRI to exclude a central lesion. Rapid referral to neurosurgery may be necessary for decompression as permanent injury occurs between 6-12 hours from the onset of signs and symptoms
- Role of nerve conduction studies: Electrophysiological investigations (electromyography - EMG) can differentiate between central and peripheral nerve injury, identify the muscles affected and give a likely prognosis of neural recovery. They may be able to identify the precise lesion site, and produce an estimate as to the timing of the injury. However, EMG only measures large nerve fibre changes and it may take as long as three weeks after an injury to show changes. An obstetric palsy will have EMG changes occurring distally to any point of anaesthetic intervention

Prognosis

- Neuropraxia – superficial type of nerve injury. Usually there is spontaneous recovery in weeks to a few months.
- Axonotmesis – axon disruption. Recovery through axon regeneration is usually achieved over several months but sometimes can take up to 3 years
- Neurotmesis – complete nerve transection. Spontaneous recovery is unlikely without surgical repair if possible

Prevention

- Compressive neuropathies:
  - change lower extremity position frequently during the second stage of labour
  - avoid prolonged thigh flexion, extreme thigh abduction or external rotation
  - avoid motor block during epidurals by using lower concentrations of local anaesthetic where possible (0.1% Ropivacaine).
- Ischaemic neuropathies - avoid hypotension.
- For prevention of anaesthetic causes see Appendix 4

8. RELATED POLICIES / PROCEDURES / CLINICAL PRACTICE LOP

- Epidural analgesia guideline
- Epidural Analgesia Programmed Intermittent Epidural Bolus (PIEB) and Patient Controlled Epidural Analgesia (PCEA)- Delivery Suite
- Correct identification of medication and solutions for regional anaesthetic procedures. SESLHDPR/242 https://www.seslhd.health.nsw.gov.au/sites/default/files/documents/SESLHDPR242.pdf

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9. RISK RATING

- High

10. NATIONAL STANDARD

- Standard 5 – Comprehensive Care

11. REFERENCES

1. Wong CA. Neurological deficits and labour analgesia. Regional Anaesthesia and Pain Medicine 2004; 29:341-352.
2. The third national audit project of the Royal College of Anaesthetists. Available from: www.rcoa.ac.uk; January 2009.
3. Richards A. Immediate postpartum neurological deficits in the lower extremity: a prospective observational study. Int J Obstet Anesth. 2017;31:5-12.
4. Howells, AC. Neurological complications in obstetric regional anaesthesia. Anaesthesia and intensive care medicine. 2013;14: 331-332

REVISION & APPROVAL HISTORY
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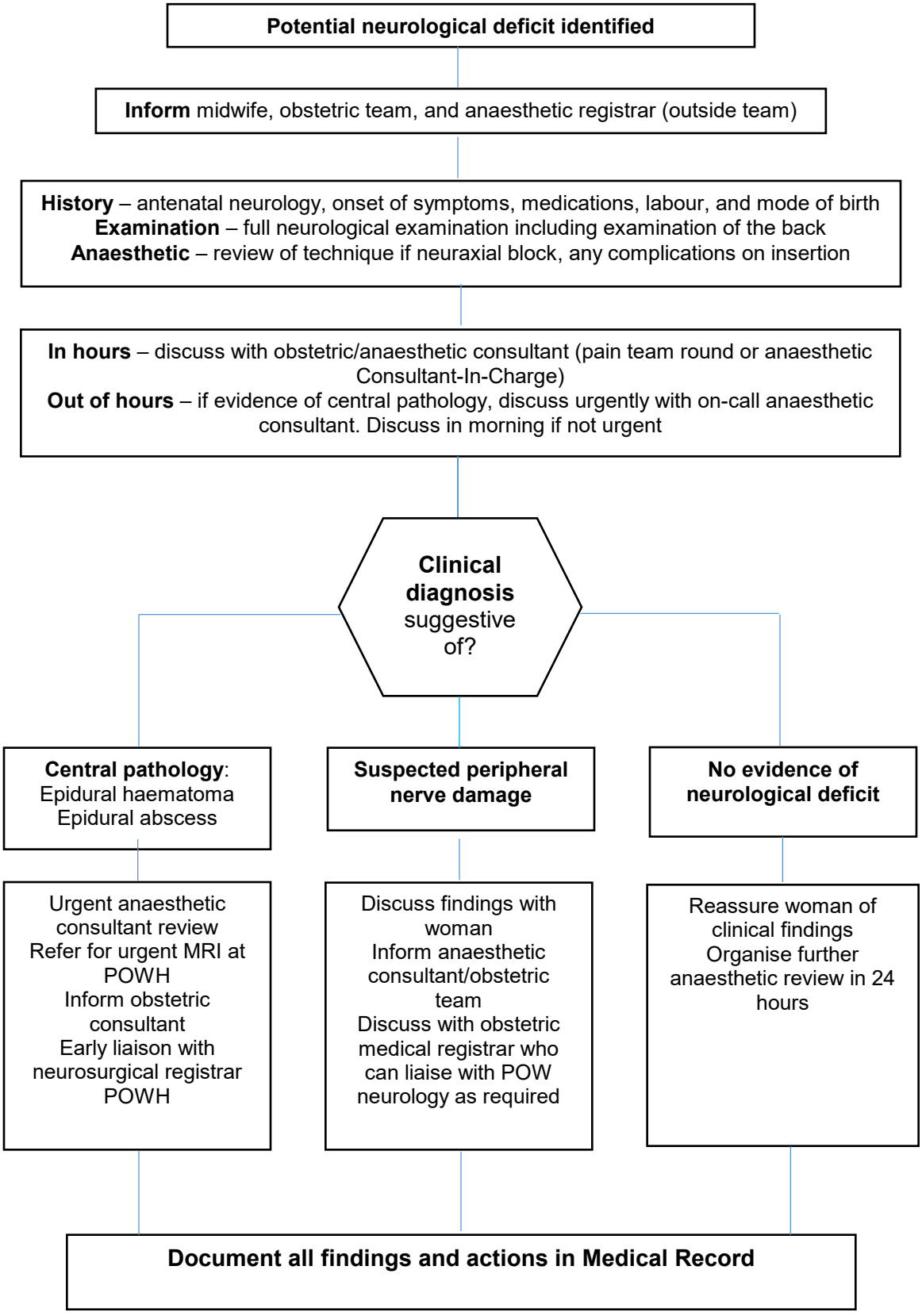
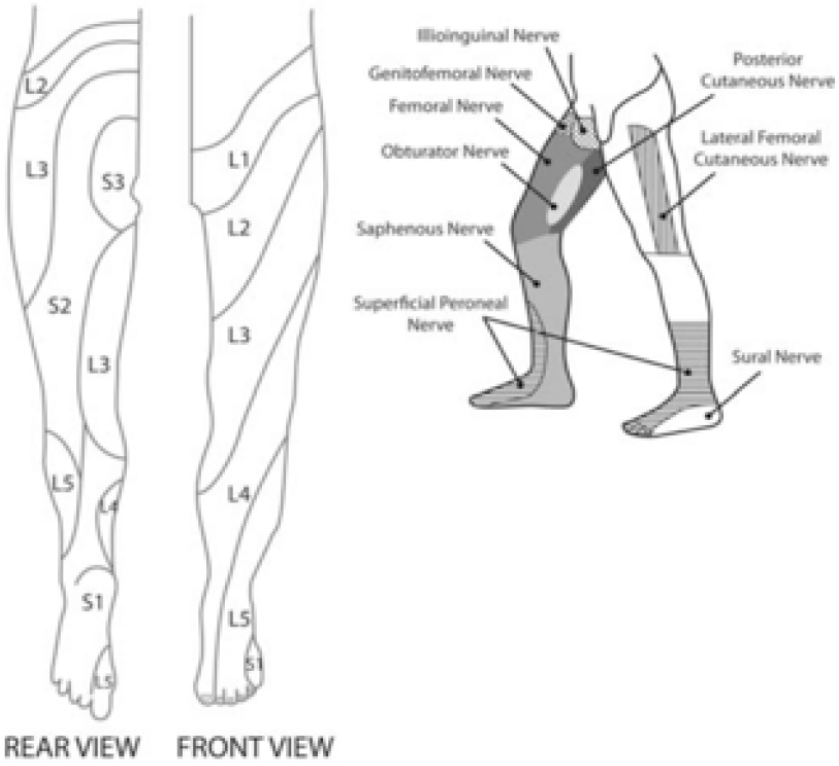
APPENDIX 1 – Neurological deficit management



APPENDIX 2 – Dermatomes of lower limb and peripheral nerve distribution



APPENDIX 3 - Compressive nerve injuries[4]

Injury	Nerves affected	Common Causes	Presentation
Lateral cutaneous nerve of the thigh	L2-3	Compression of the nerve as it passes under the inguinal ligament	Sensory loss over the anterolateral aspect of the thigh.
Lumbosacral plexus	L4,5 S1-5	Compression of lumbosacral plexus against sacral ala. Usually from foetal head in second stage. It arises on the opposite side to the fetal occiput.	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 eversion with decreased sensation on the lateral lower leg and dorsal foot
Common peroneal nerve due to peripheral nerve compression	L4-5,S1-2	Prolonged lithotomy position. The nerve is compressed as it passes over the head of the fibula (inappropriate positioning of the patient in stirrups).	Numbness over lateral aspect of lower leg and dorsum of foot, foot drop. Ankle reflex intact.
Femoral nerve	L2-4	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.	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
Obturator nerve	L2-4	Compression of nerve by fetal head or forceps. Obturator neuropathy, which occurs bilaterally 25% of the time, is often mistaken for an intraspinal lesion.	Usually unilateral sensory loss over inner thigh and weak hip adduction and rotation.

APPENDIX 4 - Maternal and fetal causes[4]

Type of neuropathy	Incidence	Cause
Compressive Neuropathy	(1 in 100)	■ Fetal head compressing lumbosacral trunk ■ Positioning ■ Instrumental delivery
Ischaemic neuropathy	1 in 500,000	■ Prolonged hypotension ■ Obstruction of Internal Iliac arteries by fetal head in prolonged labour

APPENDIX 5 - Anaesthetic causes of neurological deficit[4]

	Causes	Symptoms & Diagnosis	Prevention
Nerve damage	Secondary to direct needle or injection trauma disrupting the fibres of a single nerve .	This causes paraesthesia, loss of sensation, and muscular weakness in the distribution of the nerve	Clearly avoiding contact with nerves
Spinal cord trauma	Direct trauma when placing spinal, epidural or CSE.	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.	If pain on needle insertion or injection of LA withdraw needle. The conusmedullaris usually ends at L1 but may extend to L2,3 in 10% of patients. L3,4 should be highest landmark. Scanning of the back can identify the correct interspace
CaudaEquina 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 asespsis on epidural linsertion. 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.
Epidural Abscess	On insertion of epidural: · Prolonged catheter insertion · Presence of sepsis · Inadequateaseptic 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

	Can also occur spontaneously Causative organism is commonly Staph. aureus	Needs urgent MRI	
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 compression.	Back pain Nerve root pain. Weakness Paralysis (late feature)	Risk factors: • Coagulopathy • Difficult CNB insertion • Anticoagulants Consider timing of regional block if on anti-coagulants and whether coagulopathy excludes patient from CNB