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OBSTETRIC ANESTHESIA (LR LEFFERT, SECTION EDITOR)

Postpartum Neuropathies

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Abstract

Purpose of Review This review discusses neuropathies intrinsic to the process of labour itself, and those related to obstetric central neuraxial block. The focus is on lower limb pathology. A general approach to the evaluation of patients is provided, emphasising the importance of identifying time-critical conditions.

Recent Findings Recent work has focussed on the incidence of postpartum neuropathy and confirms how rarely anaesthesia is a causative factor. International guidelines have been developed to minimise the risk of neuraxial-related injury, and to optimise both monitoring of patients with neuraxial block and investigation of abnormal findings.

Summary Obstetric anaesthetists are frequently asked to see patients with abnormal neurology in the postpartum period. A clear understanding of common pathology and the core tenets of evaluation and management are crucial for safe patient care. While rare, complications related to neuraxial anaesthesia can be devastating, and appropriate preventative measures should be employed to minimise risk.

Keywords Postpartum neuropathy · Intrinsic obstetric neuropathy · Central neuraxial block (CNB) · Vertebral canal haematoma · Epidural abscess · Chronic adhesive arachnoiditis

Introduction

Neurologic symptoms affecting the lower limbs are commonly encountered by an obstetric anaesthetist in the postpartum period. Neuraxial techniques are often assumed to be the cause, despite their rarity compared to injuries that are intrinsic to the process of childbirth itself [1, 2]. They are a common cause of litigation [3]. Intrinsic obstetric injury is usually related to the stretch or compression of nerves at ligaments and bony prominences [4–6], and generally carries a good prognosis [7, 8]. An understanding of common injury patterns will help aid diagnosis and allow early treatment implementation.

Anaesthetic-related complications, although rare, may be catastrophic. Nerve injury may be due to direct needle trauma, compression by a space-occupying haematoma or abscess, infection, or chemical irritation [4, 6, 9]. The unique physiology of the parturient, commonly encountered co-morbidities, and pressured environment of the delivery suite provide a unique challenge for the obstetric anaesthetist. Meticulous attention to preventative measures, monitoring, and timely investigation of abnormalities help to ensure risk related to neuraxial block remains low.

Intrinsic Obstetric Injury

Intrinsic obstetric nerve palsies represent the majority of postpartum neurological injuries [6, 10, 11], and are directly caused by the process of childbirth itself, whether or not an anaesthetic intervention has taken place [2]. The incidence of intrinsic obstetric injury has been estimated at 0.3–2%, depending on the nature of the study (prospective vs retrospective) and the method of screening (active or relying on patient self-reporting) [7, 12–14]. Most injuries are sensory in nature, with a motor deficit being much less common [13, 14].

General Mechanism of Injury

Intrinsic obstetric neuropathies usually result from nerve compression by the fetal head or forceps or stretching of

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nerves from extremes of positioning maintained for prolonged periods [1, 2, 4, 9–11, 15]. Rarely, hypotension may compromise the blood supply to nerves [15]. Most commonly, a demyelinating injury results, which generally recovers provided the axon remains intact [2, 9, 13].

General Risk Factors

Factors which increase the risk of compression by the fetus itself include macrosomia (large baby), reduced maternal height, cephalopelvic disproportion, and non-vertex presentations such as breech [1, 8]. High or mid-cavity forceps delivery (e.g. using Kielland's forceps) posed the greatest risk of forceps-induced injury, but this practice is becoming less common in favour of cesarean delivery (CD) [11].

A prolonged labour second stage is also associated with an increased risk [1, 4, 7, 8, 14–16] which may be because extremes of lower limb positioning (e.g. hip flexion, abduction, and external rotation) are maintained for longer periods, or the fetal head may be in contact with nerves for longer. Intrinsic obstetric neuropathies are more common amongst primiparous women [1, 4, 7, 8, 13, 14, 16].

While not a direct cause of injury, decreased sensation resulting from central neuraxial block (CNB) can reduce awareness of the symptoms of developing nerve injury, while motor block may mean parturients are less able to move position to relieve pressure on nerves [4, 13, 16]. Higher concentrations of local anaesthetic (LA) can increase second-stage labour duration [9, 15]. Stretch injury risk may be increased with the thighs hyper-flexed on the abdomen—the position often adopted for the active second stage ('pushing') when CNB is present [1, 2, 4, 6].

Many of the physiological changes of pregnancy also increase the risk of nerve injury, including weight gain and tissue oedema, as the chance of nerve compression at bony and ligamentous sites is increased [7].

Treatment and Prevention

Treatment is largely supportive. Anti-inflammatories, anti-neuropathic medications, and nerve blocks may be employed for pain (although consider seeking obstetric advice if breastfeeding due to the risk of drug transfer) [8, 9, 17]. Physiotherapy may strengthen affected muscles and allow compensation for weakness, and occupational therapy (including ankle and knee braces) aims to prevent secondary injury [8, 9, 18]. Psychotherapy may be considered if there is significant emotional distress [18]. Managing subsequent pregnancies is challenging if severe neuropathies persist. The rarity of intrinsic obstetric neuropathies means estimating the risk of recurrence is impossible, and CD may be reasonable for subsequent deliveries [19].

Prevention generally centres on frequent repositioning during labour, particularly during the second stage, and avoiding extremes of hip flexion, abduction, and external rotation [1, 4, 14, 18]. The use of low-concentration LA for epidural analgesia may minimise motor block, and avoid prolongation of the second stage [1, 20].

Prognosis

Several studies have shown very good recovery of intrinsic obstetric injuries, which generally occur within weeks but may rarely take up to 3.5 years [4, 7, 8, 10, 11, 13–15, 18, 21–23]. The duration of symptoms is usually dictated by the severity of the initial lesion [24]. While this should generally reassure physicians and patients alike, the presence of axonal injury may worsen the prognosis [8], and the development of chronic regional pain syndrome has been reported [25].

Specific Neuropathies

The lumbosacral plexus (Fig. 1) gives rise to the nerves of the lower limb and may be injured directly by compression from the fetal head at the posterior pelvic brim or sacral ala [4, 9–11, 15, 27]. Signs and symptoms depend on the specific nerve roots involved and may be difficult to differentiate from more distal individual nerve lesions, or more central polyradiculopathies [8, 9]. The characteristic features of specific lower extremity nerve injuries are described in Table 1.

Injury Secondary to Central Neuraxial Block

Nerve injury secondary to CNB may be due to direct needle trauma, space-occupying lesions including vertebral canal haematoma (VCH) and epidural abscess, infection, and chemical injury. This is particularly relevant for patients with a pre-existing neuropathy as the 'double crush phenomenon' states that they are particularly vulnerable to a secondary insult [7, 9, 29].

Incidence

Nerve injury related to CNB is extremely rare. In 2014, the Serious Complications Repository Project of the Society for Obstetric Anesthesia and Perinatology (SOAP) [30] reported an incidence of 'serious neurologic injury' (defined as any central or peripheral nervous system injury requiring neuroimaging or consultation) as 1 in 35,923 (which equates to 2.8 per 100,000). Prior to this, the 3rd National Audit Project of the Royal College of Anaesthetists in the UK found an incidence of 'permanent harm' of between 0.3 and 1.2 per 100,000 cases following CNB in obstetric

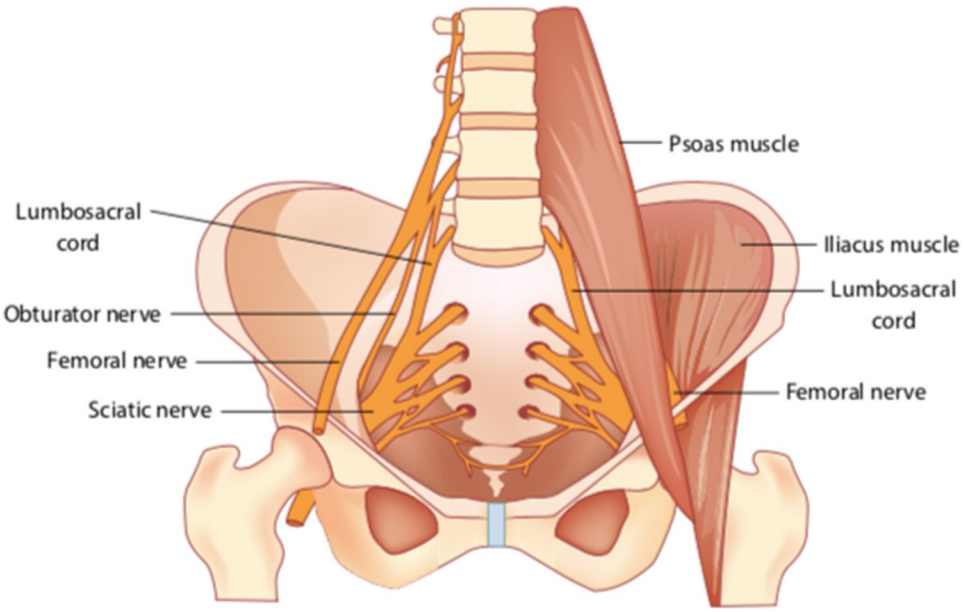

Fig. 1 Branches of the lumbosacral trunk within the pelvis. Reprinted with permission from Springer Nature: Springer. Quick Hits in Obstetric Anesthesia by Fernando R, Sultan P, Phillips S (Eds) [26]

patients [21, 31]. This was notably lower than in the general peri-operative population, a finding which has been attributed to the relative health of parturients and short epidural catheter insertion duration [21]. An earlier meta-analysis found the incidence of persistent neurologic injury in obstetric patients receiving epidural procedures of 1:237,000 to 1:256,979 (0.39–0.42 per 100,000) [32], while a more recent population-based descriptive study using the UK Obstetric Surveillance System found the incidence of vertebral canal haematoma and epidural abscess—two of the most serious complications of CNB—to be as low as 6.7 per 1,000,000 cases (0.67 per 100,000) [33].

Patterns of Injury

Nerve Root Injury

Radiculopathies (injury to the spinal nerve root) are most commonly seen at L4, L5, and S1. They are associated with back pain, radicular weakness, dermatomal sensory loss, and diminished deep tendon reflexes [7, 9]. Lasègue's sign (eliciting pain with straight leg raise to ≤ 45°) will be positive [7]. In the presence of multiple nerve root injuries, a polyradiculopathy will present with more extensive weakness and sensory disturbance, depending on the nerve roots affected [9], while in extreme circumstances cauda equina syndrome presents with bilateral limb involvement, urethral and anal sphincter disturbance and back pain [9].

Conus Medullaris Syndrome

Damage to the conus, affecting both somatic and parasympathetic nerves, presents with perineal anaesthesia, and sphincter dysfunction [9]. In contrast to a cauda equina syndrome, back pain is usually absent, sphincter dysfunction occurs early, and there is minimal involvement of the lower limbs [9].

Spinal Cord Injury

Spinal cord injury usually presents with paraparesis (lower extremity weakness) which progresses to paraplegia (lower extremity paralysis) [34••]. It is often painless, unless there is meningeal irritation or blood vessel displacement [9]. Further symptoms depend on the underlying pathology and are discussed in more detail below. Of note, blood in the subarachnoid space may cause irritation of the posterior columns of the spinal cord, and Lhermitte's phenomenon of shooting electric shock-like pains involving the occiput, thoracic region, and peripheral extremities may be associated with neck flexion [9].

Prognosis

Prognosis often depends on the severity of the initial presentation, with significant weakness and bladder and bowel involvement a poor prognostic sign [9]. In the presence of a space-occupying lesion—e.g. an abscess or haematoma—urgent surgery is needed, as delays worsen the prognosis [9].

Trauma

Trauma to the spinal cord, conus, or nerve root can be caused directly by an epidural or spinal needle, an epidural catheter, or the intraneural injection of drugs [4, 6, 15, 16].

Table 1 Key features of individual intrinsic obstetric neuropathies of the lower limb (*ASIS* anterior superior iliac spine, *CD* cesarean delivery). *Supportive measures include analgesics, physio- and occupational therapy, and psychological support. Adapted from [1, 2, 4, 6–11, 13, 15, 17, 23, 27, 28]

Nerve (roots)	Mechanism of injury	Signs and symptoms	Risk factors	Specific treatment measures	Other
Lateral femoral cutaneous nerve (L2–L3)	Compression/stretch at inguinal ligament or ASIS; iatrogenic (surgical retractors during CD)	Sensory loss over anterolateral thigh; no motor deficit; neuropathic pain ('meralgia paraesthetica')	Increased intraabdominal pressure from pregnancy, fetal macrosomia, or excess weight gain; increased lumbar lordosis; prolonged hip flexion	Supportive measures*	Rarely injured directly by spinal/epidural needle insertion as nerve origin lateral to transverse processes
Femoral nerve (L2–L4)	Compression/stretch at inguinal ligament or ASIS; iatrogenic (Pfannenstiel incision or retractors during CD); compression by retroperitoneal pathology including haemorrhage	Sensory loss +/− neuropathic pain in anterior thigh, medial leg, and foot; quadriceps and psoas weakness—difficulty climbing stairs or standing from sitting; patellar reflex loss	Increased intraabdominal pressure; prolonged hip flexion, abduction, and external rotation	Knee braces for stabilisation	Most common lower limb neurologic deficit; bilateral in up to 25% of cases
Obturator nerve (L2–L4)	Compression at lateral wall of pelvis by fetal head or forceps	Sensory loss to upper medial thigh; weakness of hip adductors; difficult ambulation with wide-based gait	Cephalopelvic disproportion; forceps delivery	Supportive measures*	May be associated with femoral neuropathy
Sciatic nerve (L4–S4)	Exact mechanism unknown, possibly impingement between ischial tuberosity and lesser trochanter	Sensory loss to posterior thigh, lateral calf, and foot; weakness of knee flexors, ankle invertors, evertors, plantar- and dorsiflexors	Prolonged lithotomy	Supportive measures*	Rare as intrinsic obstetric palsy; often mistaken for more proximal lumbosacral trunk injury
Common peroneal nerve (L4–S2)	Compression at fibular head	Sensory loss to anterolateral leg and dorsum of foot; weakness of ankle dorsiflexors and evertors; foot drop and high-stepping gait	Prolonged positioning in lithotomy stirrups	Ankle brace to reduce falls risk	
Pudendal nerve (S2–S4)	Compression by fetal head or forceps	Sensory loss to perineum; urethral and anal sphincter dysfunction	Forceps delivery	Pelvic floor muscle physiotherapy, laxatives, anticholinergics for urinary urgency, neuromodulation with sacral or peripheral nerve stimulators for refractory symptoms	Difficult to distinguish from normal symptoms in immediate postpartum period, diagnosis may require urodynamic testing and anorectal manometry

It is usually associated with pain or paraesthesia at the time of injection [4]. Presentation of resulting neuropathies will depend on the site of injury (see above) but is most commonly a radiculopathy involving a single nerve root [35, 36]. Magnetic resonance imaging (MRI) may show signs of spinal cord trauma including oedema, haemorrhage, or a needle track in the affected area [36].

Prevention

Traditional landmark palpation of the intervertebral level is often inaccurate [4, 37]. Tuffier's line is unreliable in pregnancy due to weight gain, lumbar lordosis, and pelvic rotation [38]. The conus lies lower than L1/2 in a significant minority of patients [3, 4, 39]. Therefore, clinicians should use the lowest possible interspace to minimise the risk of spinal cord contact [6, 10, 11], and the L2/3 interspace should not be used for spinal anaesthesia [39, 40].

The use of pre-procedural ultrasound, although not yet a standard of care, can help to accurately determine the intervertebral level, and is particularly useful in challenging patients—e.g. with obesity or kyphoscoliosis [36, 41, 42].

Even with an appropriate level of insertion, however, lateral needle deviation may still injure nerve roots [41]. It is therefore crucial to withdraw, redirect, or change the level of insertion if any pain or paraesthesia is reported on needling or injection, particularly if distant to the site of insertion, or if cerebrospinal fluid (CSF) is not free flowing [3, 6, 27].

Vertebral Canal Haematoma

The incidence of VCH is estimated to be between around 1:150,000 and 1:250,000 [30, 32, 43, 44]. While it can occur spontaneously [4], it is typically due to damage to the epidural venous plexus during CNB, particularly epidural catheter placement or removal [10, 11, 15, 41, 45]. The resulting haematoma can cause both direct trauma to the spinal cord and ischaemia [15]. Classically, it causes back pain with sensory and motor deficit outlasting the expected duration of the block [10, 11, 15, 36]. There may be bladder and bowel dysfunction [41]. However, symptoms are variable [45]. It is rarer amongst obstetric patients than the general surgical population [43, 46, 47], despite the presence of larger, more dilated blood vessels [1]. This is thought to be due to the procoagulant effect of pregnancy [1, 46]. Younger patients also have larger intervertebral foramina, facilitating the exit of accumulating blood [47], and more compliant spinal cords, with increased tolerance to volume expansion [46]. As such, although traumatic insertion is often cited as a risk factor for development, most VCH have been reported in patients with a pre-existing coagulopathy (e.g. secondary to pre-eclampsia), or those taking anti-coagulant medications [4, 6, 16, 27, 34••, 46]. The risk is higher in those with pre-existing spinal abnormalities, e.g. stenosis [34••, 36]. MRI is the investigation of choice [3, 34••, 36, 48]. Rapid surgical decompression is key to management [48], with permanent neurological injury occurring as rapidly as 8–12 h after symptom onset [34••, 48]. Prognosis is linked to the severity of symptoms at presentation and is worse with increasing delays to surgery [45, 48].

Prevention

Assessment of Coagulopathy

There are of a number of conditions commonly encountered in obstetric patients which may cause a low platelet count. These include gestational thrombocytopenia, hypertensive disorders of pregnancy, and (more rarely) immune thrombocytopenia [49••]. A precise safe limit for CNB is unknown; however, recent consensus guidelines suggest that with these etiologies, in the absence of signs or symptoms of coagulopathy, a platelet count $> 70 \times 10^9$/L is likely to be safe [49••]. The evidence for a concurrent coagulation screen when thrombocytopenia is present is limited [49••]. The optimal timing of testing is unknown, but in severe diseases, it should be as close to the planned procedure as possible [49••, 50]. There is insufficient evidence to recommend the routine use of viscoelastic haemostatic assays for predicting the risk of VCH development [49••]. Risk should be seen as a spectrum, and the risk of not siting a neuraxial block must also be considered [49••, 50].

Management of Anti-coagulants

The prevalence of venous thromboembolism as a leading cause of maternal death [51] is reflected in the increasing numbers of women prescribed anti-coagulant medications in pregnancy [52••]. Recent guidelines from SOAP provide recommendations on the timing of neuraxial block around commonly used anti-coagulant medications in both elective and emergency settings to minimise the risk of VCH [52••]. A full discussion of the guidelines is outside the scope of the current article, but they are unique from previous guidance in that they take account of the pharmacokinetic effects of the physiological changes of pregnancy to give advice specific to the parturient. They also provide decision-making aids to guide physicians in emergency situations when the risks of not providing CNB must also be considered [52••].

Monitoring

Early detection is crucial to successful outcomes when managing VCH [34••, 48]. Early symptoms may mimic

a normal neuraxial block, so a high index of clinical suspicion is required [34••, 41, 53]. Findings which should prompt early anaesthetic assessment include an unexpectedly dense motor block when using low dose epidural mixture in labour, a block that is slow to regress (particularly after 4 h has elapsed), the return of a previously resolved block, and an unexpected block distribution [3, 34••, 41]. Leg strength may be used as a marker of spinal cord health. Therefore, the straight leg raise has been suggested as a pragmatic tool for monitoring adequate motor function. It should be checked hourly in labour alongside other routine midwifery observations, and 4 h after the last dose [34••]. Clinicians should be aware that the distractions of a busy delivery suite must not detract from the rapid assessment of symptomatic patients [34••, 53]. There should be clear pathways in place for urgent imaging, including when out of hours or requiring inter-hospital transfer, and referral to surgical teams [3, 34••, 41], accepting that the majority of scans will be negative [34••]. Epidural abscesses (considered below) should be detected in the same way, but are most likely to present insidiously, often after the patient has been discharged [34••].

Infection

The incidence of infective complications—namely, epidural abscesses and meningitis, is rare. It has been estimated at 1:50,000 to 1:150,000 [30, 32]. In a recent analysis of over 2 million obstetric epidurals, no epidural abscesses occurred, emphasising the rarity of this complication [44]. The two infective processes have different aetiologies and typical causative organisms. Meningitis tends to follow spinal anaesthesia as there is a dural breach [47] and is usually caused by *Streptococcus viridians* [47]. Epidural abscesses may occur spontaneously [10, 11, 35], or via haematogenous spread from other infected sites [41, 54]. When associated with CNB, they are generally the result of epidural catheterisation [16, 47], and usually caused by staphylococci [10, 11, 15, 35, 47].

Epidural Abscess

Risk factors for epidural abscess development include immunosuppression, pre-existing spinal pathology, sepsis, prolonged catheterisation, and poor aseptic technique resulting in contaminated drugs or equipment [1, 10, 11, 15, 34••, 41, 54]. Infection overlying the insertion site is an absolute contra-indication [41].

Symptoms are often out of proportion to the degree of spinal cord compression, which is thought to be due to associated vessel thrombosis and compression [54].

A classical triad of back pain, fever, and neurologic deficit [6, 41] usually presents hours to days after epidural catheterisation [15]. Typically, there is bladder and bowel dysfunction, motor deficit, and variable sensory involvement [6, 10, 11, 15]. However, presentation is variable, and reliance on these classic symptoms may result in delayed diagnosis [54].

An MRI will show the abscess and associated cord compression, and inflammatory markers will be raised [36]. Treatment consists of broad-spectrum antibiotics which are continued for several weeks, and early surgical decompression [6, 10, 11, 41, 54]. Lumbar puncture should be avoided in the diagnostic stage due to the risk of coning and introducing infection to the subarachnoid space [47, 54].

Meningitis

Meningitis presents with neck stiffness, photophobia, headache, back pain, fever, nausea, and lethargy [10, 11, 15, 47]. Symptoms may be confused with Post Dural Puncture Headache (PDPH) [10, 11, 41], except there is no postural element to headache [41].

The common causative organisms of *Streptococcus viridians* colonises the female genital tract and gastrointestinal and upper respiratory tracts [2]. Its presence in the upper respiratory tract highlights the importance of wearing a facemask when performing CNB [16, 41]. CSF provides the ideal culture medium [2, 35, 47], but it requires a dural puncture to breach the blood–brain barrier [2, 41]. It appears to occur more commonly in women who have laboured rather than after elective CD, potentially due to vaginal trauma and bacteraemia, or the less sterile conditions of a labour room [47]. Other risk factors include maternal sepsis, and poor asepsis resulting in contaminated drugs or equipment [15]. Neurological consultation is warranted, since early presentation may be similar to epidural abscess, and consideration should be given to performing an MRI before lumbar puncture [47].

Prevention

Due to the rarity of infective complications, definitive evidence from randomised controlled trials is not feasible [47]. Expert consensus suggests that the risk of infective complications can be minimised by [47, 55]:

- Pre-assessment of patients to identify those at high risk of infective complications and pre-procedure antibiotic administration in the bacteraemic patient.
- Full aseptic technique (hand wash, face mask, sterile gown, gloves, and drape, skin preparation with chlorhexidine and alcohol).
- Use of a bacterial filter for epidural catheters.

- Minimising epidural catheter insertion times and frequency of disconnections, and removing catheters which have been accidentally disconnected.
- Careful daily evaluation of the epidural catheter insertion site with awareness that infective complications may be increased if there are co-existing risk factors.

Chemical Injury

Adhesive Arachnoiditis

This is a non-specific inflammation of the meninges, resulting in collagen deposition with interrupted blood and CSF flow [4, 10, 11, 15, 16, 36, 41, 56]. It is associated with back and leg pain and lower limb neurologic impairment which may be severe and permanent [4, 15]. In extremis, it causes complete CSF flow obstruction with associated raised intracranial pressure and hydrocephalus [40]. The prognosis is poor, as there are few treatment options [41]—focus must be on prevention. It is thought to be due to an idiosyncratic reaction to injected irritants [41]. Chlorhexidine (discussed further in 'prevention', below) is an important cause [3, 40, 41, 57], and blood, contrast media, epidural steroids, vasoconstrictors, and preservatives have also been implicated [4, 15, 41, 56]. Low-concentration, preservative-free bupivacaine and opioids injected using disposable equipment in a standard way have not been linked to the condition [56].

Prevention

Prevention focusses on measures to ensure culprit drugs are not injected during CNB. The primary concern is chlorhexidine as this is used for skin preparation immediately prior to CNB placement. Measures to reduce the risk include using 0.5% rather than 2% solutions, as there is no evidence for reduced infection risk, but greater neurotoxicity with higher concentrations [40, 57]. Care should be taken to avoid contamination of gloves and equipment, and to prevent accidental injection of chlorhexidine epidurally—which may be aided by avoiding open containers [3, 36, 40, 41, 57]. The solution should be allowed to dry completely prior to performing the block [36, 40, 57].

Other Chemical Injury

Intrathecal hyperbaric 5% lidocaine has been associated with a syndrome of transient buttock and lower limb pain. It is unclear whether there is true neurologic injury, and the syndrome usually self-resolves within a few days [1, 2, 6, 15, 27]. Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) provide effective pain relief, and early mobilisation may help symptom resolution [1, 27].

Supra-normal doses of maldistributed LA may cause a cauda equina syndrome, and excessive sacral block must be excluded prior to repeating single-shot spinal injections [36].

Finally, tranexamic acid, increasingly used for postpartum haemorrhage management, has a very high mortality if accidentally injected intrathecally, with permanent neurologic damage in survivors [58, 59].

Miscellaneous Neurologic Injury

Spinal Cord Ischaemia

Anterior spinal artery syndrome usually occurs during aortic surgery but may rarely be seen in the parturient if there is prolonged hypotension alongside local vasoconstriction caused by LA solutions containing adrenaline. Pre-existing pathology affecting epidural compliance may contribute as epidural and CSF pressures become abnormally raised following otherwise normal epidural LA volume administration. The usual presentation is bilateral lower limb weakness and loss of temperature and pain sensation, but preserved vibration, light touch, and proprioception [6, 8, 16, 35].

Prolapsed Intervertebral Disc

This is a rare cause of neurologic injury in an obstetric patient, with an incidence of approximately 1:10,000 [11]. Radicular symptoms caused by disc impingement on local nerve roots may be mistakenly attributed to neuraxial block. Cauda equina syndrome is a neurosurgical emergency which must be managed without delay to avoid permanent neurological injury [60].

Psychogenic Paresis

A diagnosis of exclusion, this rare conversion disorder has been described in the postpartum patient [61]. There is no underlying organic cause of the neurological symptoms, although they are not intentionally produced. Early diagnosis with psycho- and physiotherapy is key to management [61].

Approach to Evaluation and Management

Any patient reporting neurological symptoms in the postpartum period requires prompt review [2]. It is crucial to identify those time-critical conditions which carry the highest

Table 2 Core investigations for patients with postpartum neurological symptoms (*CT* computed tomography, *MRI* magnetic resonance imaging). *Wallerian degeneration is the active degeneration which develops from the distal end of an axon after nerve injury. Adapted from: [2–5, 15, 33, 34••, 36, 48, 49••, 50]

Investigation	Role
Blood tests—full blood count, coagulation screen, inflammatory markers	• Severe thrombocytopenia and coagulopathy predispose to vertebral canal haematoma • Inflammatory markers will be raised in infective pathology
CT spine	• May identify those space-occupying lesions most amenable to surgical management if MRI is not available
MRI spine	• Gold standard for diagnosis, all departments should have policies in place to allow timely access to MRI imaging • May show evidence of direct trauma to spinal cord, presence of haematoma or abscess, and degree of cord compression
Electrophysiologic studies	• Electromyography determines muscle units affected • Nerve conduction studies localise site of injury-related conduction block • Together can identify underlying pathology and likely prognosis • Assist with timing of injury (Wallerian degeneration* can take up to 3 weeks, so positive results within 72 h–1 week imply pre-existing injury)

morbidity and which are amenable to early management, in particular space-occupying lesions and meningitis [6, 15]. Early liaison with a neurologist is key to guide diagnosis, treatment, and follow-up plans.

Patient evaluation is complicated by the fact that many of the symptoms are the same as normal issues in the early postpartum period, including back pain, fever, and bladder and bowel dysfunction [2, 6, 9].

A thorough history of intrapartum events is required, considering patient positioning both during labour and delivery, duration of the second stage, mode of delivery, and the details of any anaesthetic interventions [2, 9, 15]. The presenting symptoms should be explored, especially their timing of onset, duration, and progression [36].

A full neurological and musculoskeletal examination is required, signs of sepsis must be sought, and the back should be examined carefully for point tenderness and skin changes over the insertion site [9, 15]. Severe pain on palpation of the back implies a central cause as the posterior primary rami of the spinal nerves are affected, while the absence of pain or dysfunction of the paraspinal musculature points to a more distal injury [2, 4, 15].

While history and examination alone are often insufficient to pinpoint the diagnosis [2], they allow the identification of red flags which require urgent imaging of the spine and discussion with a neurologist. These include [4, 9, 36]:

- Pre-existing risk factors such as a coagulopathy or immunosuppression
- Severe back pain
- Unexplained fever
- Bladder or bowel involvement (often a late sign)
- Motor or sensory deficits which:
  - Follow a central or radicular pattern
  - Are bilateral (although peripheral nerve injuries may also present bilaterally)
  - Are progressive
  - Have returned after initial resolution
- Lhermitte's sign which signals potential pathology affecting the posterior columns of the spinal cord.

Following the exclusion of immediate, life- or limb-threatening pathology by the absence of red flags, or negative imaging, further investigation is guided by consultation with neurologists and often includes electrophysiologic studies [2, 4, 5, 15, 36]. The role of different investigations in the workup of a patient with postpartum neurologic disturbance is detailed in Table 2.

Management of individual injuries has already been discussed but since most are self-limiting it usually involves patient reassurance and measures to prevent secondary injuries [2, 6, 15].

Conclusion

It is often not possible to provide a precise diagnosis when assessing patients with abnormal neurology postpartum. Anaesthetists must be aware of red flags which require urgent investigation, but it is reassuring that most conditions are self-limiting and unrelated to anaesthesia. Although rare,

complications from CNB are serious, and prevention must be the aim of all obstetric anaesthetists.

Declarations

Conflict of Interest Matthew Sinnott and Roshan Fernando declare they have no conflicts of interest.

Human and Animal Right and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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•• Of major importance

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