Painless childbirth? Epidural and spinal techniques in obstetric anesthesia

Schyns-van den Berg, A.M.J.V.

Chapter 10

Postdural puncture headache: Beyond the evidence

A.M.J.V. Schyns-van den Berg,
D.N. Lucas,
L.R. Leffert

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ABSTRACT

Despite advances in procedural techniques and equipment, postdural puncture headache (PDPH) remains a serious complication of labour epidural analgesia after accidental dural puncture (ADP). Often considered a temporary inconvenience, PDPH can be debilitating in the short term. It can also be associated with chronic manifestations and serious complications.

The precise underlying mechanisms of PDPH are still incompletely understood, and long-standing beliefs of dysregulation of cerebrospinal fluid (CSF) homeostasis due to CSF fluid loss are currently being challenged. The existence of orthostatic headaches unrelated to CSF loss demands consideration of other mechanisms involved, for instance, related to the autonomic nervous system or the release of calcitonin gene-related peptide (CGRP) associated with activation of the meningeal and cerebral arteries.

A multi-society international working group recently provided evidence-based recommendations on the prevention, diagnosis, and management of PDPH resulting from neuraxial procedures. According to the recommendations, there was insufficient evidence to support the routine use of intrathecal catheters or sphenopalatine ganglion blocks to prevent or treat PDPH. Both evidence-based and experimental strategies include stabilizing CSF dynamics through preventing CSF loss, supplementing CSF, or increasing production, and reducing cerebral vasodilation.

Future research in PDPH preventive and therapeutic strategies can be facilitated with standardised definitions, interventions, and outcome measures. Analyses should consider various confounding factors and recognise the complex multifactorial nature of pain experience across diverse populations. Clinical care and research on PDPH will demand a multidisciplinary collaborative effort to elucidate the complexities of its pathophysiology and further improve patient outcome and quality of care.

INTRODUCTION

Postdural puncture headache (PDPH), occurring after intentional or accidental dural puncture (ADP), remains a significant challenge in obstetric anaesthesia due to the high proportion of neuraxial techniques, and prevalence of risk factors such as younger age and female sex. While PDPH has traditionally been considered a relatively benign, self-limited condition, cohort and case-control studies suggest an association between PDPH and long-term morbidities, such as chronic headache, backache, neck pain and depression [1]. Additionally, studies demonstrate an association between PDPH and rare but serious complications such as intracranial subdural hematoma and cerebral venous thrombosis [2,3].

Unfortunately, our understanding of PDPH is incomplete. There is a paucity of high-quality evidence to support many existing management strategies [4]. It is not feasible to conduct sufficiently powered prospective studies for which large numbers of patients would be needed, and ethical considerations prevent randomisation [5]. Here we review the current understanding of PDPH including strategies for prevention, pathophysiology, treatment, and long-term concerns with inclusion of lesser-known strategies for which further research is warranted.

PDPH definition, epidemiology, and diagnosis

a. Definition

The International Classification of Headache Disorders, ICHD-3, classifies PDPH (7.2.1) as a headache developing within 5 days of lumbar puncture, caused by cerebrospinal fluid (CSF) leakage through the dural puncture, which remits spontaneously within 2 weeks, or after sealing of the leak with autologous epidural lumbar patch [6]. As with other headaches caused by low CSF pressure, headache symptoms may be more severe in the upright position (orthostatic or postural), but this feature is not required for a PDPH diagnosis. A PDPH is frequently accompanied by neck stiffness, tinnitus, photophobia and/or nausea. In anaesthesia practice or after diagnostic lumbar puncture, the diagnosis of PDPH is based on clinical symptoms, without the need to confirm reduced CSF pressure or CSF leakage through imaging.

b. Epidemiology

The incidence of PDPH varies based on procedure, needle type, and patient population [7]. Patient-related risk factors for PDPH include female gender, younger age, low body mass index, chronic headache, pregnancy, vaginal delivery, and history of PDPH [4,8]. A PDPH occurs after 50-60% of spinal techniques when large (<24G) cutting spinal needles are used, but this risk falls dramatically to 0.16-2% with the use of pencil-point needles or smaller (>24G) cutting needles [9]. Accidental dural puncture with a large (16-18G) epidural needle results in PDPH in 50-80% of cases [10,11]. With increasing utilization of labour epidural analgesia,

it has been estimated that up to 15,000 women in Europe and 50,000 in the United States develop PDPH after childbirth every year [12,13].

c. Clinical presentation and diagnosis

The presentation of a PDPH is typically dull, throbbing, and bilateral, often with worsening in the upright position. However, up to 5% of PDPH may present without orthostatic changes [14]. Common symptoms include neck stiffness, nausea, vomiting, tinnitus, hearing changes, photophobia, visual disturbances, and vertigo [12]. These symptoms are thought to be the result of descent of brain tissue, engorgement or activation of meningeal or cerebral vessels, meningeal inflammation and temporary dysfunction of the first three cervical and cranial nerves [4,15]. Auditory symptoms, reported in 20% of cases, result from either involvement of the vestibulocochlear nerve or cochlear dysfunction [16].

Table 1. Differential diagnosis of headaches in the postpartum period.

Condition	Common Presentation
Tension headache	Bilateral, tightening headache
Migraine headache	Unilateral, pulsating headache
Preeclampsia with severe features/eclampsia	Headache associated with hypertension or seizure attributed to preeclampsia
Posterior Reversible Encephalopathy Syndrome (PRES)	Headache, visual disturbances, altered mentation, hypertension, ± seizure
Cerebral venous thrombosis (CVT)	Severe headache of gradual onset, typically non-positional
Meningitis/sepsis	Severe headache, fever, nuchal rigidity, vomiting, + Kernig or Brudzinski's sign
Subdural hematoma (SDH)	Progressively worsening headache, with possible decreased consciousness
Postdural puncture headache (PDPH)	Fronto-occipital headache, often worse when upright
Cerebral venous infarction +/-	Headache, focal neurologic deficits
Intracerebral or subarachnoid haemorrhage (SAH)	Headache with focal neurologic deficits, "worst headache ever" (SAH)
Idiopathic intracranial hypertension	Headache, visual symptoms, ± nausea
Reversible cerebral vasoconstriction syndrome	Thunderclap headache

Postpartum headaches are common, affecting approximately 40% of women, with the majority being classified as tension headaches or migraines (Table 1) [17]. It is crucial to consider other potential causes when assessing postpartum headaches. In cases where hypertension is present, new-onset or worsening preeclampsia should be evaluated as a possible diagnosis. Such headaches may involve posterior reversible encephalopathy

syndrome (PRES), though this is rare. Severe and rapidly worsening headache symptoms, especially when accompanied by focal neurological signs or altered consciousness, require immediate neurological assessment and neuroimaging to rule out rare but potentially life-threatening conditions, such as subarachnoid haemorrhage, subdural hematoma, cerebral venous thrombosis, ischemic stroke, or an undiagnosed intracranial tumour [17-20]. To aid in the accurate diagnosis of postpartum headaches, Lim et al. recommend using the PARTUM mnemonic (Table 2) [19].

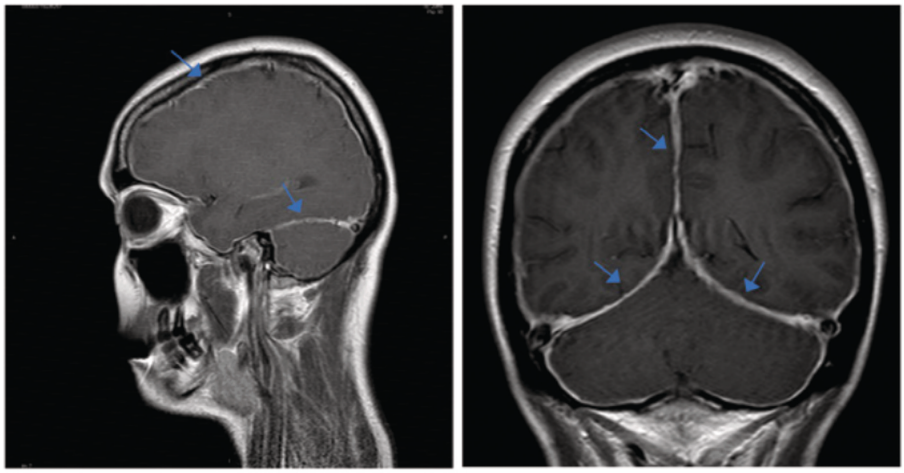
Table 2. PARTUM mnemonic for diagnosis of postpartum headaches. Adapted from Lim et al [19]

P	Pressure	Blood pressure for pre-eclampsia/eclampsia
A	Anaesthetic	Postdural puncture headache (PDPH)
R	Reversible	Reversible vasoconstriction syndrome or posterior reversible encephalopathy syndrome (PRES)
T	Thrombosis	Cerebral venous sinus thrombosis (CVT), ischemic stroke
U	Use your brain	There are so many other causes of headache: musculoskeletal, tension-type, meningitis, caffeine withdrawal, etc.
M	Migraine	if it improved during pregnancy, likely to recur in the first week postpartum

Although PDPH has often been described as self-limiting, early studies reported chronic headache after spinal anaesthesia and recent cohort and case-control studies in obstetrics describe an increased risk of chronic headache, backache, postpartum depression and post-traumatic stress disorder (PTSD) [21-23]. A recent anonymous online survey of individuals on a PDPH-patient advocacy website reported severe long-term consequences such as persistent headache and disability in patients previously diagnosed and treated for PDPH after lumbar puncture, epidural anaesthesia, or spinal surgery [24]. Given the retrospective nature of data on chronic headache after PDPH, the possibility of bias is significant [23]. Nonetheless, mounting evidence for an association of PDPH with chronic headache and other sequelae warrant further attention and consideration for longer-term follow-up.

In obstetric PDPH, additional diagnostic tools [e.g., radiologic evidence of CSF leak or lumbar puncture demonstrating CSF pressure <60 mm H2O)] are not typically used, and PDPH remains a clinical diagnosis [4]. If the headache presentation is atypical, changes over time, or symptoms appear which cannot be explained by CSF loss or raise suspicion of serious complications, then imaging is warranted [4]. Neuroimaging can rule out other pathology and either directly or indirectly demonstrate signs of CSF leakage. Spinal MRI, CT, or digital subtraction myelography can display the amount and site of CSF leakage. However, this finding can be nonspecific, often unaccompanied by clinical PDPH symptoms, or clinical symptoms can be present without signs of spinal CSF leakage [25,26]. Cranial MRI using a gadolinium-based contrast agent may show pachymeningeal (dural) enhancement, venous engorgement, brain sagging, or pituitary enlargement. In one study, 100% of patients undergoing MRI within 3 days of PDPH onset showed dural enhancement (Fig. 1) [27].

Fig. 1. Brain MRI: Pachymeningeal enhancement (arrows)



A B

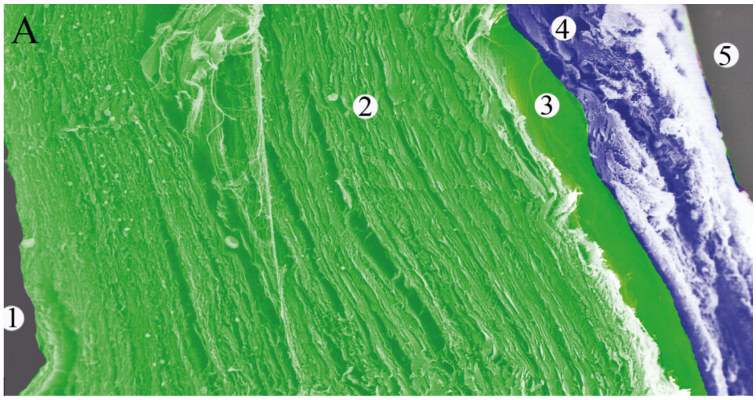
(A) Sagittal gadolinium-enhanced TSE, T1-weighted brain MRI.
(B) Coronal gadolinium-enhanced TSE, T1-weighted brain MRI.
From: Brain MRI features of postdural puncture headache, Sanchez Garcia et al., Reg Anesth Pain Med 2024 [27].

Transorbital ultrasound for optic nerve sheath diameter (ONSD) measurement could possibly aid in the diagnosis and monitoring of PDPH, as changes in CSF pressure are transmitted along the optic nerve sheath [28]. Increased ONSD has been demonstrated after successful epidural blood patch (EBP) or closure of CSF leaks and is associated with a decrease in intracranial venous volume and blood flow [28,29].

The pathophysiology of PDPH

Postdural puncture headache (PDPH) was first accurately described by Dr. August Bier in 1899, who linked the condition to CSF loss and emphasized minimizing CSF loss during procedures. A similar orthostatic headache can occur with spontaneous intracranial hypotension (SIH), even without prior trauma or medical intervention [30]. The CSF leak is often perceived to result from a dural tear; however, the primary barrier preventing CSF leakage is not the dura mater, with its 70-80 permeable layers of randomly arranged collagen and fibrinogen fibres, but rather the impermeable arachnoid mater (Fig. 2) [31].

Fig. 2. Human dura and arachnoid mater thickness.



Dura mater in green and arachnoid mater in blue
Magnification A: x300
(1) Epidural space; (2) Dura mater; (3) Acquired subdural space; (4) Arachnoid mater; (5) Subarachnoid space.
With permission from Prof. Miguel A. Reina M.D., Ph.D., private archive.

When spinal CSF loss exceeds the body's replacement rate (estimated to be about 300-1000 mL/day), the reduction in intracranial CSF volume—and eventually pressure, once the compensatory cerebral vasodilation capacity is surpassed—can cause a downward shift of brain structures, known as brain sagging [32,33]. This phenomenon can be accentuated in the upright position. The headache symptoms are thought to relate in part to traction on pain-sensitive structures such as meninges, arteries near the base of the brain, and anchoring veins draining into the sagittal, transverse, and other venous sinuses [34-36].

Another feature of PDPH appears to involve cerebral vasodilation, as a *reflex response* to traction on intracranial pain-sensitive vessels and/or a *compensatory mechanism* caused by a reduction in CSF volume. This compensatory mechanism of haemostatic intracranial volume regulation (Monro-Kellie doctrine) is displayed when a loss in intracranial CSF volume is accompanied by an increase in cerebral blood volume (via vasodilation) [33,37].

Dilation of the meningeal arteries and activation of the trigeminovascular system (TVS) involving release of calcitonin gene-related peptide (CGRP) may also contribute to headache symptoms like those of migraine headaches [33,38]. Another hypothesis suggests that lumbar CSF loss increases spinal compliance caudally, leading to low intracranial CSF pressure in the upright position, and subsequently to intracranial venous dilation [39-41]. Lastly, neurotransmitters and modulators involved in pain perception, such as substance P, may play a role. Reduced CSF levels of substance P have been associated with an increased incidence of PDPH following lumbar puncture [42].

It is notable that orthostatic headaches can occur without CSF disturbances in autonomic nervous system (ANS) dysfunction syndromes like orthostatic hypotension or postural orthostatic tachycardia syndrome (POTS), when compensatory baroreflex mechanisms fail to adapt to postural changes [43,44]. In over 40% of cases, POTS coexists with other headache disorders, such as migraine or SIH, or develops after successful SIH treatment, which suggests a contribution of autonomic dysfunction [43-45]. Further research is needed to elucidate the ANS mechanisms involved in the evolution of PDPH, and to identify potential therapeutic targets that modulate this activity.

Current evidence-based preventive and therapeutic management strategies for PDPH

a. Summary

The rare occurrence of PDPH and variability in clinical presentation make gathering of high-quality evidence difficult. Recently, a multidisciplinary group published expert- and evidence-based recommendations using the US Preventative Services Task Force grading guidelines [46]. Recommendations assigned levels of certainty give clinicians a framework for the prevention, identification and management of PDPH (Table 3) [4,46]. The main procedural recommendation for spinal anaesthesia/analgesia or lumbar puncture to minimize the risk of PDPH is to use a high gauge (small calibre), pencil point needle whenever feasible [4,47]. There is currently insufficient evidence to support other suggested preventative strategies, such as particular patient or needle positioning, postprocedural bed rest, hydration, or medications [4].

Intrathecal insertion of the epidural catheter (intrathecal catheter, ITC), once an accidental dural puncture (ADP) occurs, may provide a functional mode of labour analgesia or caesarean delivery anaesthesia, but does not decrease the risk of PDPH and/or the need for an EBP [4]. Likewise, the neuraxial injection of other substances (e.g., preservative free morphine, saline) into the epidural space or the use of prophylactic EBPs have not consistently shown benefit [4,48].

Once PDPH develops, recommended conservative management includes maintenance of adequate hydration and the provision of a multimodal analgesic regimen including paracetamol (acetaminophen), nonsteroidal anti-inflammatory drugs (NSAIDs), and limited use of opioids [4]. Oral caffeine may temporarily reduce symptom severity, but the ingested amount should be limited in obstetric patients and breastfeeding women [4]. While maintaining the supine position reduces the severity of symptoms and provides temporary relief, prolonged bedrest is undesirable due the increased risk of thrombosis associated with immobility [4]. Of the more invasive procedures for PDPH treatment, limited evidence suggests that a greater occipital nerve block (GONB) might be considered after spinal anaesthesia with small gauge spinal needles [4,49]. The EBP is the most effective invasive procedure for providing temporary or permanent relief from PDPH resulting from various dural

puncture interventions. It should be considered in patients with PDPH that is impairing the activities of daily life and that does not respond to conservative therapies [4].

Table 3. Consensus practice guidelines: preventive and therapeutic measures with level A-C grading recommendations.

	Recommendation Level	
Procedural factors		
Non-cutting spinal needles	A	
Narrower gauge cutting spinal needles	A	
Conservative treatments		
Paracetamol (acetaminophen) and NSAIDs offered to all patients with PDPH	B	
Short-term opioid use if regular analgesics ineffective	C	
Caffeine offered in the first 24 h of symptoms	B	Maximum dose of 900 mg/day, 200-300 mg if breastfeeding
Adequate hydration maintained with oral fluids	C	
Bedrest to lower severity of symptoms	C	Beware of increased risk of thromboembolism
Invasive treatments		
Greater occipital nerve block (after spinal anaesthesia with 22-G or smaller spinal needle)	C	Headache recurrence risk
Epidural blood patch	B	Offered if PDPH does not respond to conservative treatment and impairs daily activities

Adapted from: Uppal et al. [4]
Abbreviations: NSAIDs, non-steroidal anti-inflammatory drugs; PDPH, postdural puncture headache; mg, milligrams; G, gauge

b. The epidural blood patch (EBP)

The EBP has been the cornerstone of PDPH treatment, since Gormley observed that bloody taps during spinal anaesthesia were associated with a lower PDPH incidence. An EBP involves the injection of autologous blood into the epidural space, with a hypothesized two-fold effect: increasing epidural and lumbar CSF pressure and stimulating fibroblastic repair in proximity to the dural defect. The immediate but transient increase in epidural volume increases the spinal and intracranial CSF pressure. Notably, there is no demonstrated relationship between the final epidural pressure generated during the procedure and its success [50,51]. An epidural

hematoma is known to be a potent cerebral vasoconstrictor which may contribute to the positive outcome [38,51]. MRI studies have shown that the mass effect of injected blood lasts only a few hours, and clot forms within 7 h, which stimulates fibroblastic and collagen repair of the dural defect, preventing further CSF leakage [52-54]. Absolute contraindications for an EBP are patient refusal, coagulopathy (thrombocytopathy, active anticoagulation), sepsis, systemic infection without CNS involvement, local infection at the site of injection, and increased intracranial pressure [4,55,56].

Efficacy of an EBP procedure varies significantly, likely reflecting procedural and patient-related factors and the extent of dural damage. For PDPH after spinal anaesthesia, EBP success rates of 75-96% have been reported. After ADP with large gauge Tuohy needles in obstetrics, complete and permanent resolution of symptoms after one EBP occurs in less than 50% of patients [4,56,57]. A large prospective international cohort study of 647 obstetric patients (EPiMAP) investigating risk factors for EBP failure after ADP found that complete success (defined as an NRS score of 0 in upright position at 4, 24 and/or 48 h) was achieved in only 33.0% of cases [58]. Complete failure (NRS scores ≥7 at one of these time points or the need for a second EBP) occurred in 28.7% of patients, with 19.8% ultimately requiring a second EBP [58].

The optimal volume and timing of an EBP is unknown. The EPiMAP study could not provide a definitive answer as most patients received approximately 20 mL of blood, according to common practice [58,59]. The study found no association between EBP failure and EBP lumbar level, position during EBP, or operator experience. The only associations with failure of EBP were timing of the EBP, a history of migraine which was specifically associated with an increased provision of a second EBP, and (unexpectedly) a higher lumbar level of the original ADP.

As reported, the EPiMAP study confirmed that patients receiving an early EBP, within 48 h after ADP, were more likely to require a repeat procedure: 37.8% of women who received an EBP within 48 h of the ADP needed a second EBP, compared to 23.4% who received their first EBP between 48 and 72 h, and 7.1% after 72 h. Importantly, it is not known whether receipt of an earlier EBP was associated with more severe presenting PDPH symptoms [4,56,58]. These findings suggest that if an early EBP is recommended to patients, they should be informed of the possibility of a repeat EBP to achieve complete resolution of symptoms [4]. However, delaying an EBP for patients with severe symptoms is not recommended, as it prolongs patient suffering and may increase the risk of rare severe complications such as subdural hematoma [3].

The most common reported complication of EBP is back pain, with an estimated incidence of 80%. Chronic headache, present a year after ADP in 28% of patients, is not significantly reduced in patients who were treated with an EBP [57,60]. Arachnoiditis, radiculitis, meningitis and spinal subdural hematoma, all serious neurologic conditions, have been described both after ADP alone, and after EBP [61-63].

Alternative current and future management strategies

Over the years clinicians have explored numerous additional interventions to prevent and treat PDPH based on the presumed pathophysiological mechanisms described above. Sufficient evidence is lacking to promote their recommendation for routine use.

a. Reducing cerebrospinal fluid leakage with an ITC

There appears to be no consistent association between the volume of CSF loss and the degree of PDPH symptoms, and interindividual variability is significant. Some patients develop severe symptoms after an uneventful procedure with a 27 G pencil-point needle, while others remain asymptomatic even after ADP with a large bore epidural needle. Transient headache symptoms do often occur during drainage of CSF in lumbar punctures and are associated with the volume collected. However, headache immediately after lumbar puncture procedures is unrelated to persistent PDPH at 24 h or the need for an EBP [36,64].

One intervention to reduce CSF loss following ADP during epidural labour analgesia procedure is the immediate insertion of an intrathecal catheter (ITC). The ITC can be used to provide rapid labour analgesia without the need for another procedure and is thought to potentially reduce CSF leakage by partially occluding the dural hole. An ITC should only be considered in the presence of an institutional protocol which includes strict aseptic techniques, meticulous monitoring, labelling and communication to prevent drug errors, and recommendations of drugs and dosages to be used intrathecally under the various obstetric scenarios [65,66]. Although several studies and meta-analyses have suggested that ITCs reduce PDPH severity and EBP use, Heesen et al.'s trial sequential analysis determined evidence to be insufficient to exclude a type 1 error (false positive, rejecting the null hypothesis when it is actually true), despite finding a reduced PDPH incidence (RR 0.82, 95% CI 0.71-0.95) and need for EBP (RR 0.62, 95% CI 0.49-0.79) [5]. Two subsequent retrospective studies have suggested benefits of ITC, with or without intrathecal saline administration [67,68]. One showed no additional benefit in leaving the ITC in place postpartum [68]. Notably, the best practices outlined in the 2024 Guidelines on ITC placement after ADP from the Obstetric Anaesthetists' Association conclude that there is no evidence-based indication for leaving the ITC in situ after delivery is complete [66].

b. Increasing cerebrospinal fluid volume

Various strategies aim to increase the production of CSF. Increased fluid intake, administration of caffeine or other methylxanthines, hydrocortisone and ACTH-analogues, have all been considered but have not proven to contribute to increased CSF production [4]. There is currently renewed interest in the ACTH-analogue cosyntropin, which is hypothesized to contribute to analgesia and increased CSF production through stimulation of glucocorticoid and mineralocorticoid synthesis [69]. However, the results of studies demonstrating cosyntropin's effectiveness in preventing PDPH or reducing its severity are inconsistent. A randomized controlled trial in obstetric patients after ADP did find that cosyntropin reduced

the incidence of PDPH and the need for an EBP compared to placebo [70]. In a retrospective study of 578 women with ADP, the combination of cosyntropin, an ITC, and prophylactic administration of 10 ml intrathecal (IT) saline did not reduce the incidence of PDPH, but did decrease the need for an EBP [68,71]. Other recent retrospective analyses failed to find any benefits from the use of cosyntropin in preventing or reducing severity of PDPH [69,72].

Epidural single shot saline boluses and continuous epidural or caudal saline infusions have been used as an option for the treatment of PDPH. Sterile saline, injected epidurally, produces a short-lived mass effect which is thought to temporarily reduce CSF flow through dural hole, thus facilitating repair [73]. One study demonstrated that epidural saline injections resulted in either resolution or temporary relief of PDPH after spinal anaesthesia, even though there was only a 3-to-10-min increase in epidural and subarachnoid pressure, depending on volume, injection rate and site of injection [50]. Various regimens in different patient populations have been studied, and while severity of symptoms reduces, the effect is transient, compared to EBPs [4,73].

IT saline injection may relieve stretching of pain-sensitive structures and secondary vasodilation. Intrathecal injection of saline, either during a spinal procedure, during ADP, or through an ITC before removal, appears to contribute to reduced incidence and severity of PDPH, though current evidence is not sufficient to recommend this as standard practice [4,67,68].

c. Reduction of cerebral vasodilation

Various treatments for migraine and cluster headache, such as triptans, gabapentinoids, sphenopalatine ganglion block (SPGB), and greater occipital nerve block (GONB), have shown some effectiveness in relieving the symptoms of PDPH, suggesting potential similarities in underlying pathophysiologic mechanisms. Interruption of central sensitization, reduction of inflammation, and modulation of pain pathways may contribute to relief of symptoms [74]. Likely, cerebral vasoconstriction is an important mechanism of action. For example, sumatriptan is thought to inhibit presynaptic serotonin release which reduces cerebral vasodilation and interferes with vasoactive peptides release and trigeminal pain [75-77]. There is insufficient evidence to recommend caffeine and other xanthine derivates such as aminophylline or theophylline, although a recent meta-analysis found the last two medications associated with lower pain scores [4,78]. A prospective randomized controlled study showed that neostigmine in combination with atropine reduced the severity of PDPH and accelerated recovery, while nebulized dexmedetomidine significantly reduced PDPH severity and reduced mean flow velocity in the middle cerebral artery of postpartum patients [79,80]. Further exploration of these therapies for PDPH are warranted.

d. Peripheral nerve blocks to modulate the pain pathway

The SPGB, typically used in patients with cluster headache and trigeminal neuralgia, is considered to be a minimally invasive alternative treatment option for PDPH [38,81]. The

sphenopalatine ganglion (SPG) is the main extracerebral parasympathetic ganglion, located bilaterally in the pterygopalatine fossa, containing sensory, sympathetic, and parasympathetic fibres that innervate the lacrimal glands, nasal glands, and cerebral blood vessels [82]. The SPG is connected to the trigeminovascular system, which is implicated in various headache disorders. While its mechanism of action is not fully elucidated, the SPGB appears to temporarily reduce or reverse cerebral reflex vasodilation due to low CSF volume and potentially interrupt trigeminal activation, or modulate inflammatory neurotransmitters involved in pain signalling [38,82]. The precise mechanism is not completely understood, as an RCT has demonstrated that a SPGB with saline is as effective as LA in reducing headache symptoms and the need for an EBP in patients with PDPH after intended or ADP [83]. An effective SPGB typically provides only temporary symptomatic relief, and repeated treatments may be necessary before PDPH symptoms resolve [84]. Techniques include percutaneous and trans-nasal approaches, the latter being the less invasive, but likely less effective as well [38,85]. Although there is insufficient high-quality evidence to support the routine use of SPGB for PDPH, it may be an alternative strategy when EBP is contraindicated or refused. Further refinement and standardization of this less invasive technique may increase its utility [85].

The GONB has been shown to effectively reduce PDPH severity in patients with PDPH after spinal anaesthesia with a traumatic needle and might be effective after ADP [4,49]. A typical GONB includes bilateral injection of local anaesthetic and corticosteroid near the greater occipital nerves and may modulate trigeminocervical signalling to effect central pain processing pathways. The anti-inflammatory effect of the corticosteroid dose may augment the efficacy of the GONB. Other medications with similar actions are gabapentin which might reduce headache severity through modulating pain pathways, and hydrocortisone with its anti-inflammatory properties [86].

e. Oxygen therapy

Oxygen therapy can be effective in treatment of cluster headache where it may have similar actions as in high altitude headache: inhibiting trigeminal-vascular and autonomic pathway innervation by acting specifically on the PS/facial nerve projections to the cranial vasculature [87]. One small retrospective study described the use of high flow oxygen in conjunction with metoclopramide as a pro-serotonin agent to treat PDPH resulting from various diagnostic and therapeutic lumbar punctures [88]. In 10 out of 12 patients, PDPH symptoms resolved rapidly, without additional metoclopramide administration [88].

Overall, while cerebral vasoconstriction is considered to be the primary mechanism of action of these therapies, central sensitization interruption, anti-inflammatory effects, and pain pathway modulation may have additional important effects.

f. Promotion of dural healing

In treatment resistant PDPH, other means of repairing the meningeal breach have historically been considered, such as the use of fibrin glue. While the limited evidence present does not support routine use, and complications include anaphylaxis and aseptic meningitis, a 2024 prospective, randomized non-blinded study in 70 obstetric patients with refractory PDPH after ADP compared EBP with epidural fibrin glue injection (6 mL fibrin sealant (Tissucol Duo®, Baxter S.L. Valencia, Spain) [4,89]. Epidural fibrin glue injection resulted in 100% complete relief at 12 h and at 30 days, compared to an EBP (15 mL blood) which only provided complete relief in 65.7% of patients at 12 h and in 22.9% at 30 days [89]. The most common complication, lumbar radiculopathy, occurred 6 times more frequently in the EBP group which also had a significantly longer hospital stay.

CONCLUSION

The unpredictable occurrence and the variety in signs, symptoms, and severity of PDPH suggest additional patient risk and mechanistic factors, which require further investigation. To date, our understanding of PDPH has relied on retrospective or small cohort studies hindered by variable definitions, heterogeneous strategies, and inconsistent outcomes. Pain outcomes may be impacted by individual, societal, social, and culturally determined factors. Variable timing of interventions, placebo effects, and the spontaneous resolution of symptoms can introduce confounding. Lastly, there are ethical issues preventing optimal study design, such as randomisation to include using larger or cutting needles for dural puncture, or withholding treatment with an EBP, given the lack of equipoise in these strategies as they relate to PDPH risk and treatment.

The path forward requires a multidisciplinary, international effort by experts from anaesthesiology, neurology, neuroradiology, neurosurgery, pain medicine and basic science. Use of a Delphi method could achieve consensus on a global, standardized definition of PDPH, diagnostic criteria and identification of outcome measures. Collaboration on studies should address both fundamental mechanisms and clinical challenges. A multi-centre PDPH registry could provide a method to collect comprehensive data on PDPH cases, and integrate patient reported outcomes and experiences to capture the full impact of PDPH. In this manner, we can meaningfully work to improve the quality of our care related to ADP, an important complication of neuraxial procedures.

Research agenda

Investigate potential strategies to reduce ADP during epidural procedures.

Develop standardized definitions and diagnostic criteria for PDPH and therapeutic outcome measures.

Develop a multi-center PDPH registry to collect prospectively relevant data.

Evaluate the role of new diagnostic tools such as optic ultrasound in PDPH diagnosis and treatment.

Design a multi-center RCT comparing treatment strategies for PDPH after ADP.

Practice points

Pencil-point spinal needles should be used for neuraxial procedures worldwide, as they significantly reduce PDPH incidence

Postpartum headaches should be systematically evaluated to differentiate PDPH from both more benign and more serious causes

If a PDPH does not respond to conservative measures, an EBP should be recommended

An EBP is the most effective treatment for PDPH, but a repeat EBP might be necessary to achieve complete resolution of symptoms

An ITC should only be considered in the presence of strict institutional protocols to ensure safe use

When an EBP is contraindicated of refused, alternative strategies like a SPGB or GONB can be considered

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