Mini topic review: Postdural puncture headache

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Introduction

Every year, about 707,000 central neuraxial blocks are performed in the UK. Post-dural puncture headache (PDPH) is an iatrogenic complication of puncture of the dura mater. This could be secondary to spinal anaesthesia, epidural analgesia, combined spinal-epidural anaesthesia, diagnostic lumbar puncture, myelography or intrathecal chemotherapy. PDPH can be severe, incapacitating and potentially last for days to weeks or even months. It can cause a significant increase in anaesthetic workload and result in prolonged hospitalisation. A recent systematic review conservatively estimated the incidence of severe PDPH in the obstetric population alone as about 800 cases per year in the UK. Hence the financial implications for this condition could be enormous.

Clinical Presentation

The International Headache Society has defined PDPH as a headache that develops within five days of dural puncture. The headache improves within 15 minutes of assuming the supine posture and worsens within 15 minutes of assuming a sitting or erect posture. It may be associated with nausea, vomiting, dizziness, tinnitus, hyperacusis, neck stiffness or photophobia. Occasionally, visual and auditory disturbances have also been reported. In rare cases cranial nerve palsies can occur due to the stretching of vulnerable nerves secondary to cerebrospinal fluid (CSF) redistribution. The most common visual disturbance is diplopia due to stretching of the VI cranial nerve over the petrous part of the temporal bone. Transient palsies of the III, IV, VII and VIII cranial nerves have also been reported. Auditory disturbances include transient hearing loss and tinnitus because of the direct connection between CSF and perilymph across the cochlear aqueduct.

The headache can be dull or throbbing, starting in the occipital or frontal region and later become generalised. It may radiate to the neck or shoulders and be exacerbated by coughing, straining or any manoeuvre that increase intracranial pressure.

Incidence

After diagnostic lumbar puncture with 22Gauge spinal needle, the incidence of PDPH with a standard needle and atraumatic needle was quoted as 36% and 3% respectively. In a meta-analysis of PDPH in parturients, a group considered to be at the highest risk, the incidence of PDPH from small atraumatic spinal needles (Whitacre 27G) was calculated to be 1.7%. The accepted rate of accidental dural puncture (ADP) associated with best practice for epidural analgesia during labour is < 1%; 50-80% (in some studies unto 100%) of these patients subsequently develop PDPH.

Aetiology and pathogenesis

CSF is secreted by the choroid plexus and the total CSF volume in the central nervous system (CNS) is approximately 150 ml. Following dural puncture (intentional or unintentional), there is a possibility of leakage of CSF from the puncture site. When this leakage is greater than the CSF production rate (0.35 ml/min) intracranial hypotension occurs. Although the exact mechanism for PDPH is not clear, two hypotheses are well known:

- Loss of CSF in the spinal space depletes the intracranial CSF cushion of the brain. Traction is exerted on the pain sensitive intracranial structures during upright posture. Pain arising on or above the tentorium cerebelli is transmitted via the V cranial nerve to the frontal region; pain from below the tentorium is transmitted via the IX & X cranial nerves and upper three cervical nerve fibres to the occipital region and neck.
- Changes in cerebrovascular mechanisms (mediated via adenosine receptors), include compensatory cerebral venous dilatation secondary to loss of CSF (as explained by Monro-Kellie doctrine). This vasodilatation is considered to be responsible for PDPH.

Risk factors for PDPH

Patient characteristics

Incidence of PDPH is inversely related to age. Highest incidence is between 20–30 years of age and the incidence starts to decrease after the age of 40. It is rare after 60 yrs. Previous history of PDPH is associated with higher risk and so is...
the female sex. Lower BMI has been shown to be associated with higher risk of PDPH. Ironically, PDPH incidence is noted to be low in morbid obesity. This may be because of the large abdominal panniculus acting like an abdominal binder and raising the intra-abdominal pressure, thus reducing the rate of leak of CSF through the dural defect.

### Needle characteristics (size and design)

Incidence is directly related to the needle size. The incidence is 30% -36% with a 22G Quincke needle. The incidence of PDPH and epidural blood patch (EBP) rates for different types of needles are given in Table 1. Due to technical difficulties, needles of size 29G or smaller are not commonly used for spinal anaesthesia.

<table>
<thead>
<tr>
<th>Needle size &amp; Type</th>
<th>Bevel</th>
<th>Incidence of PDPH%</th>
<th>% of PDPH cases needing EBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>25G Quincke</td>
<td>cutting</td>
<td>8.7</td>
<td>66</td>
</tr>
<tr>
<td>26G Atraucan</td>
<td>cutting</td>
<td>5</td>
<td>55</td>
</tr>
<tr>
<td>24G Gertie Marx</td>
<td>atraumatic</td>
<td>4</td>
<td>12.5</td>
</tr>
<tr>
<td>24G Sprotte</td>
<td>atraumatic</td>
<td>2.8</td>
<td>0</td>
</tr>
<tr>
<td>25G Whitacre</td>
<td>atraumatic</td>
<td>3.1</td>
<td>0</td>
</tr>
</tbody>
</table>

Atraumatic needles with pencil-point tip separate the dural fibres rather than cut them, reducing the incidence of PDPH compared to needles with a cutting bevel. Lower incidence of PDPH with atraumatic needles has been reiterated in a recent study in paediatric population. In addition, the need for epidural blood patch has been shown to be lower for the atraumatic needles when compared to the cutting bevel needles; this is most probably related to lower leak rates with atraumatic needles which lead to milder degrees of PDPH.

In-vitro studies have confirmed that both fluid loss and leakage rates after dural puncture were high with 22G compared to 26G spinal needles, whilst there is no or minimal leak with 29G needle. Also there was less fluid loss observed with atraumatic needle (Whitacre) compared to cutting needle (Quincke) of same size.

A prospective randomized trial reported that after ADP with a 17G epidural (Tuohy) needle, 100% of patients developed PDPH whereas only 55.5% developed PDPH with 18G epidural (Special sprotteTM) needle.

### Needle orientation

Arrangements of dural fibres are thought to be longitudinal, but electron microscopy views revealed that dural collagen fibres are arranged in different directions. Clinical studies favour use of parallel orientation of needles, both spinal and epidural. With the use of pencil-point needles for spinal anaesthesia, needle orientation may not be important. However, the pathophysiology of PDPH is more related to the size and shape of the hole made in the arachnoid than in the dura mater due to the fact that arachnoid is the actual fibrous sac containing the CSF. It has been shown that the pencil point needle makes an irregular hole than that caused by cutting needle; in addition, they tend to cause more local oedema or tissue reaction than the cutting needle. This inflammatory reaction caused by the tearing of the collagen fibres after meningeal penetration may result in significant edema which might act as a plug, thus lowering the CSF leak rate through the defect.

It has been shown that angle of puncture of the dura mater was not a significant factor in the development of PDPH.

### Diagnostic LP and stylet replacement

After diagnostic lumbar puncture with 21G needle replacement of the stylet has been shown to reduce the incidence of PDPH from 16 to 5%. The subarachnoid granulation is thought to enter the hole in the dura, by keeping it open there is a comparatively larger volume of CSF leak. If the stylet is replaced, the subarachnoid granulation is pushed out of the hole and CSF loss through the hole in the dura is minimised. Though 25G needles have been tried with negative pressure for CSF collection, the American Academy of Neurologists has emphasised that diagnostic lumbar punctures should be done with at least 22G atraumatic spinal needle.
Parturients

The obstetric population is at increased risk due to young age and widespread application of neuraxial techniques. Labouring women are particularly at high risk of developing PDPH after dural puncture due to the following factors: (i) peripartum dehydration and postpartum diuresis reducing the levels of CSF production, (ii) bearing down during second stage of labour increases CSF leak, (iii) abrupt release of intra-abdominal pressure and venacaval compression at delivery reducing epidural venous pressure, (iv) early ambulation and (v) anxiety. Interestingly, it has been shown that good quality labour analgesia is feasible with a 19G Tuohy needle and 23G epidural catheter. For obvious reasons, the PDPH rates would be lesser with smaller gauge epidural needle compared to 16G needle.

Intrathecal catheter

Following inadvertent dural puncture with an epidural needle, insertion of an epidural catheter into the subarachnoid space would prevent further leak and provoke an inflammatory reaction around the puncture site. However, evidence to support this is not clear. One study has shown reduced incidence of PDPH in obstetric patients when intrathecal catheter was left in situ for 24 hours. Advantages of leaving an intrathecal catheter after ADP in a labouring parturient are threefold: (i) rapid establishment of analgesia, (ii) avoiding the risk of another dural puncture, (iii) reduction in the development of PDPH after ADP. However, this practice is associated with the risk of infection and accidental injections into the subarachnoid space.

Factors related to regional technique

Using loss of resistance to air (as opposed to saline) to identify the epidural space has been shown to increase the risk of PDPH. A recent meta-analysis noted decreased incidence of PDPH in chronic pain setting when saline was used but there was no difference in obstetric population. A Cochrane review reports no significant difference in the incidence of PDPH between patients who received epidural and those who received combined spinal-epidural.

Operator factors

Operator inexperience, fatigue, sleep deprivation and multiple attempts might contribute to a higher incidence of accidental dural puncture and PDPH.

Natural course of PDPH after dural puncture

Usually, symptoms develop within 48 hours and may be delayed upto five days. But, presentations as late as 12 days after uneventful labour analgesia has been reported. Generally, there is gradual improvement in the symptoms with time. 70% of them tend to resolve within one week, 95% within 6 weeks and 96% within six months. With early hospital discharges becoming increasingly common, these patients often present to non-anaesthetists in the community.

Management

PDPH is usually a self-limiting process. Most treatments are aimed at reducing the symptoms until the hole in the dura can heal. The treatment options are discussed as conservative and invasive.

Conservative

The fact that several treatment options are available implies that there is no consensus in the management of PDPH.

Bed rest

Remaining supine after dural puncture was once thought to prevent the occurrence of PDPH. Although bed rest does not affect the incidence of PDPH, when it occurs bed rest reduces the severity of symptoms.

Hydration

Dehydration can result in decreased CSF production. Maintaining hydration (oral or intravenous) improves the ratio of CSF production to CSF leak and may improve the clinical picture. However, if the patient is appropriately hydrated CSF production would be normal and overhydration is not beneficial.

Analgesics

Oral NSAIDs and opioids such as codeine and tramadol are often prescribed for symptomatic relief.
Caffeine

Caffeine sodium benzoate can reduce the headache by causing cerebral vasoconstriction or by limiting cerebral vasodilatation. It is available in oral or intravenous formulation and the dose is 300-500 mg once or twice daily. If unavailable, advising the patient to drink caffeine containing beverages may be useful but caffeine levels vary between 66 and 142 mg in 150 ml of coffee. Potential side effects include central nervous system toxicity and atrial fibrillation. There is a reported case of posterior reversible encephalopathy syndrome (PRES) triggered by intravenous caffeine in a patient with multiple sclerosis. However, the effects of caffeine are only temporary.

Theophylline

Single dose oral theophylline is found to be effective in treatment of PDPH, presumably due to its lasting action and potent cerebral vasoconstriction. However, its narrow therapeutic range and side effects preclude its routine use.

5-HT1D receptor agonists

Sumatriptan (subcutaneous) and the newer, Frovatriptan (oral) could theoretically improve PDPH by promoting cerebral vasoconstriction, at present there is no strong evidence to support the use of these drugs.

Methergine

Oral methergine, 0.25 mg, eight hourly for 48 hours was found to be effective for PDPH following spinal anaesthesia in obstetric patients. No studies on its use for PDPH following epidural have been published to date.

ACTH and corticosteroids

ACTH is thought to increase CSF production through active sodium transport mechanism and also raise the pain threshold through an increase in beta-endorphin levels. Multiple doses of adrenocorticotropic hormone have proven effect whilst single depot injection was not effective. A double blind controlled study found that Hydrocortisone 100 mg eight hourly for 48 hours was effective.

Gabapentin & Pregabalin

Gabapentin 400 mg eight hourly and Pregabalin 50 mg eight hourly have been shown to be useful within 24 hours in 2 different case reports. But there are no randomized trials to prove their usefulness.

Invasive

Epidural blood patch (EBP)

EBP is the definitive treatment for PDPH and is dealt with in detail later.

Epidural saline or colloids

Epidural saline or Hartmann's solution or dextran-40 (bolus or infusion) increases the pressure in the epidural space thereby reducing the CSF leak. 100% success rate has been reported in a series of 56 patients who received 20-30 ml of epidural dextran-40 over two minutes without any reported complications. This impressive result has not been reproduced elsewhere, though a case report describes successful treatment of intracranial hypotension with epidural dextran-40 and paramethasone followed by oral steroids.

Epidural or intrathecal opioids

There are case reports that claim epidural morphine as beneficial for treatment of PDPH. But there is no strong evidence to confirm this.

Fibrin glue

Although fibrin glue injection is found to be an alternative to EBP, it is associated with aseptic meningitis and poor success rate (65%) despite being performed under CT guidance.
Occipital Nerve block (ONB)

Nerve stimulator guided blockade of greater and lesser occipital nerves with a mixture of local anaesthetics, fentanyl and clonidine has been shown to be superior in relieving symptoms compared to conventional analgesics in a randomised controlled trial. Greater occipital nerve was blocked for PDPH in the parietal and occipital areas whereas lesser occipital nerve was chosen for PDPH in the frontal and temporal distribution. ONB was tried on the physiological basis that the clinical features of PDPH are very similar to those of cervicogenic headache that responded well to this nerve blockade. ONB is thought to act by interrupting the pain transmission via the greater and lesser occipital nerves or their nerve roots or ganglia. ONB relieved the PDPH and accompanying symptoms in two-thirds of patients after 1-2 injections with the rest requiring up to 4 injections. ONB has also resulted in earlier hospital discharge and shorter sick leave duration.

Surgical repair

For PDPH remaining unresolved after the above treatments, surgical repair of the dura may be necessary.

Table 2: Summary of treatment options for PDPH

<table>
<thead>
<tr>
<th>Conservative Measures</th>
<th>Invasive Measures</th>
<th>Prophylactic Measures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bed rest &amp; Hydration</td>
<td>Epidural blood patch</td>
<td>Epidural morphine</td>
</tr>
<tr>
<td>Analgesics: NSAIDS, Opioids</td>
<td>Epidural saline / colloid</td>
<td>ACTH Analogue</td>
</tr>
<tr>
<td>Caffeine</td>
<td>Epidural opioids</td>
<td>Epidural blood patch</td>
</tr>
<tr>
<td>Theophylline</td>
<td>Epidural fibrin glue</td>
<td></td>
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<tr>
<td>5-HT1D receptor agonist (Sumatriptan / Flavitriptan)</td>
<td>Occipital nerve block</td>
<td></td>
</tr>
<tr>
<td>Methergine</td>
<td>Surgical repair of the dura</td>
<td></td>
</tr>
<tr>
<td>ACTH &amp; Corticosteroids</td>
<td></td>
<td></td>
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<tr>
<td>Gabapentine &amp; Pregabatine</td>
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Epidural blood patch (EBP)

The first EBP was reported by Gormley in 1960. EBP is the gold standard treatment for PDPH against which other treatment options are compared. The first EBP was reported by Gormley in 1960.

Indications

EBP is indicated for moderate to severe PDPH when conservative treatment is unsatisfactory, durocutaneous fistulae and spontaneous low CSF pressure headache (Schaltenbrand’s syndrome).

Contraindications

Contraindications include patient refusal, coagulopathy, systemic sepsis, fever, localised infection in the back and anatomical abnormalities that could lead to higher risk of dural puncture. There is a theoretical risk of seeding the neuraxis with neoplastic cells and hence risks and benefits should be analysed in oncology patients. However, human immunodeficiency virus (HIV) infection is not a contraindication.

Practical conduct of the procedure

Ideally, EBP should be performed in a sterile environment with two operators, one aspirating blood from a sterile site and the other injecting 15 to 20 ml of blood using Tuohy needle into the epidural space, at the level of previous puncture or one level below it. The exact volume to be injected is not conclusive, but the injection should be stopped if the patient complains of any pain or discomfort in the back. Interestingly, one study has shown that low volume (7.5ml) EBP is as effective as high volume (15 ml) and is associated with less nerve root irritation pain in the low volume group. A recent randomised controlled trial reiterated that EBP is an effective treatment of PDPH. Sending blood cultures at the time of EBP is controversial.
Remaining in decubitus position for two hours after the EBP improves the success rate compared to bed rest for 30 minutes. This period of decubitus gives sufficient time for the organisation of the clot and the regeneration of CSF to mitigate the effects of intracranial hypotension.

**Timing of the EBP**

The timing of the EBP in relation to the dural puncture is important. The general consensus is that EBP is less effective if performed within 24 hours after dural puncture. This window of 24 hours is useful because it allows time for (i) confirmation of diagnosis, (ii) trial of conservative management, (iii) complete regression of the neuraxial block, if present and (iv) disappearance of local anaesthetic from epidural space. Epidural injection of blood while residual neuraxial block is still present might result in an extensive neuraxial block and the local anaesthetic might inhibit blood coagulation. It might also confound the clinical picture if the patient develops any neurological deficit. However, if the symptoms of PDPH are incapacitating, there is no strong evidence to delay EBP, especially if it occurs after ADP with wider gauge epidural needles. There are reported cases of PDPH that have been treated successfully with EBP even months after the dural puncture.

**Success rate**

The success rate following the first EBP is 61-75%, even though more than 90% of patients had initial resolution of symptoms. If unsuccessful, repeating the EBP has been shown to increase the success rate up to 97%.

**Complications**

Complications of EBP include back pain (25%), paresthesia, radiculitis, temporary cranial nerve palsies, permanent paralysis, cauda equina syndrome, pneumocephalus, epidural abscess and possible late arachnoiditis. There is no evidence to show that remote EBP could affect the subsequent conduct of epidural analgesia.

**Jehovah’s witness**

EBP for Jehovah’s Witness may require a continuous circuit established from antecubital vein to the epidural. All the options must be discussed with each individual patient and informed consent obtained.

**Mechanism of action of epidural blood patch**

It has been shown that blood clotting was accelerated (four times faster than activated clotting time) by the presence of CSF, but even instantaneous clotting of epidurally injected blood cannot explain the rapid relief EBP provides. EBP has two distinctive effects: (i) The immediate effect due to volume replacement by compression of dura that will restore CSF pressure and relieve headache. This effect lasts up to seven hours. (ii) The sustained effect (after seven hours and up to 18 hours or possibly longer) is due to the sealing of the dural defect by the blood clot. An EBP also augments the fibroblast proliferation which is a normal response to a dural tear and forms a network for collagen deposition. While the organised clot is occluding the hole in the dura, the ‘repair work’ continues to seal the dural tear. When the clot starts to resolve, the dural defect has been completely repaired or the defect is so small that the symptoms of PDPH are less intense. Unfortunately, if the blood clot gets dislodged for any reason before the repair of dural tear could be completed, failure of EBP occurs. The extent of spread of the injected blood has been shown to positively correlate with the total volume of the blood injected, an average of one spinal segment for every 1.6 ml of blood. The clot resulting from EBP spreads significantly over three to five segments around injection site and it spreads principally upwards from the site of injection. Hence, EBP should be performed below the level of dural puncture or at least at the level; for the same reasons, performance of EBP by injecting blood through an epidural catheter in situ is less likely to be successful.

**Repeat EBP:**

If EBP did not relieve the symptom, then the original diagnosis of PDPH should be questioned, especially after two consecutive failed EBPs. Detailed neurological assessment and investigation should be considered to rule out other sinister causes of headache. The clinician should always look for any change in the clinical features (e.g., change from postural headache to constant headache), which might give a clue to the development of complications of intracranial hypotension. Development of cranial subdural haematoma following untreated PDPH has been reported. CT guided EBP directed to the exact site of CSF leak might be a safe option in selected patients when repeated EBPs failed to resolve PDPH.
Causes of epidural blood patch failure:

- Injection at inappropriate level or injection through an epidural catheter
- Inadequate blood volume
- Presence of local anaesthetics or steroids in epidural space
- Incorrect timing of EBP (too early or too late)
- Headache due to other cause(s) (Table 3)

Use of substances other than blood for epidural patching:

Different substances including dextran, hetastarch, gelatine, fibrin glue and cryoprecipitate are administered instead of autologous blood for epidural patching due to various reasons including religious beliefs, concerns of infectious complications related to blood, arachnoiditis, cancer and thrombocytopenia.

Table 3: Differential diagnosis of headache after dural puncture (intentional or unintentional).

<table>
<thead>
<tr>
<th>Primary</th>
<th>Secondary</th>
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<tbody>
<tr>
<td>Migraine</td>
<td>PDPH</td>
</tr>
<tr>
<td>Tension / non-specific headache</td>
<td>Pre-eclampsia or eclampsia</td>
</tr>
<tr>
<td>Sinus headache</td>
<td>Cerebral arterial or venous sinus thrombosis</td>
</tr>
<tr>
<td>Orgasmic headache</td>
<td>Ischaemic or haemorrhagic stroke</td>
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<tr>
<td></td>
<td>Subarachnoid or intracranial haemorrhage</td>
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<tr>
<td></td>
<td>Hypertensive encaphalopathy or bleeding</td>
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<tr>
<td></td>
<td>Posterior Reversible Encephalopathy Syndrome (PRES)</td>
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<tr>
<td></td>
<td>Postpartum cerebral angiopathy</td>
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<tr>
<td></td>
<td>Pituitary apoplexy</td>
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<tr>
<td></td>
<td>Intracranial tumour or pseudo-tumour</td>
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<tr>
<td></td>
<td>Uncal herniation</td>
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<tr>
<td></td>
<td>Meningitis (viral, bacterial, chemical)</td>
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<tr>
<td></td>
<td>Caffeine withdrawal headache</td>
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</tbody>
</table>

Strategies to prevent PDPH following accidental dural puncture

Various strategies including prophylactic epidural injection of saline, dextran or autologous blood, intrathecal placement of epidural catheter, avoidance of bearing down in the second stage of labour, subarachnoid opioid injection, cerebral vasoconstrictors and abdominal binders have been suggested as prophylaxis to reduce the incidence of PDPH after unintentional dural puncture, but there is no strong evidence favouring these interventions. A recent Cochrane review concluded that the benefit of prophylactic EBP to prevent PDPH remains unclear.

A recent systematic review and other studies have shown that prophylactic epidural morphine decreases the incidence of PDPH after ADP.

A recent randomised controlled trial has claimed that a single prophylactic intravenous administration of cosyntrophin (ACTH analogue) after ADP has halved the incidence of PDPH.
Conclusion

PDPH could potentially increase the workload of anaesthetists and physicians. In addition, the resultant increase in hospital stay, investigations and treatment required could have significant financial repercussions. Also the physicians and general practitioners should be aware of late presentations in the community.

EBP is the gold standard treatment for PDPH. But conservative management should be considered for the first 24 hours, while the diagnosis is being confirmed. EBP performed within the first 24 hours may not be very effective. But certainly, EBP should not be delayed more than 24 hours in a patient with incapacitating symptoms. Other pathologies that may coexist in these patients should be excluded and hence a structured multidisciplinary approach is advisable, especially if EBP fails twice.

For diagnostic lumbar puncture, larger needles (20G) are still widely used outside theatres. This practice needs to be addressed and the physicians should be encouraged to use atraumatic needles with at least 22G. Smaller gauge spinal and epidural needles would not only reduce the incidence of PDPH, but also the rate of EBP required. While smaller gauge Tuohy needles are available and could be used to provide good quality labour analgesia, 16G needles are still widely being used. Routine use of smaller gauge epidural needles should be considered especially, strongly considered for labour analgesia. It remains to be seen if further explorations on ACTH analogues and lesser invasive procedures, such as occipital nerve blocks, would alter our current management of PDPH.

References


32. Arendt K, Demaerschalk BM, Wingercuk DM, Camann W. Atraumatic lumbar puncture needles: after all these years, are we still missing the point?. Neurologist 2009; 15(1):17-20.[Link to abstract]


38. Simmons SW, Cyna AM, Dennis, AT, Hughes D. Combined spinal-epidural versus epidural analgesia in labour. Cochrane Database of Systematic Reviews 2007; 18(3): CD003401.[Link to full text]


40. Vandam ID, Dripps RD. Long term follow up of patients who received 10,098 spinal anaesthetics. Anesthesia 1974; 44(5):415-8.[Link to full text]

41. Reynolds F. Dural puncture and headache. British medical journal 1993; 306:874-76.[Link to full text]


44. Camann WR, Murray RS, Mushlin PS, Lambert DH. Effects of oral caffeine on postdural puncture headache. A double-blind, placebo-controlled trial. Anesthesia and analgesia 1990; 70 (2):181-4.[Link to abstract]


76. Diaz JH. Permanent paraparesis and cauda equine syndrome after epidural blood patch for postdural puncture headache. Anesthesiology 1991; 75: A1082. [Link to abstract]


55. Foster P. ACTH treatment for post-lumbar puncture headache. British journal of anaesthesia 1994; 73:42. [No abstract available]


52. Foster P. ACTH treatment for post-lumbar puncture headache. British journal of anaesthesia 1994; 73:42. [No abstract available]


89. Hakim SM. Cosyntropin for prophylaxis against postdural puncture headache after accidental dural puncture. *Anaesthesia* 2010; 113(2):413-20. [Link to abstract]

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