A rational approach to the cause, prevention and treatment of postdural puncture headache

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Objective: To review the current research and formulate a rational approach to the cause, prevention and treatment of postdural puncture headache (PDPH).
Data sources: Articles published from January 1980 to April 1992 were obtained through a search of MEDLINE and *Index Medicus*. Key reference articles published before 1980 were also reviewed.
Study selection: All pertinent studies were included and critically analysed.
Data synthesis: PDPH occurs when a slow leak of cerebrospinal fluid leads to contraction of the subarachnoid space and compensatory expansion of the pain-sensitive intracerebral veins. Female sex and an age between 20 and 40 years have been shown to be independent risk factors for PDPH, but pregnancy has not. The rate of PDPH is directly proportional to the diameter of the needle used and also depends on the design of the needle tip. Prophylactic epidural blood patching or saline infusion after dural puncture can decrease the incidence of PDPH, but both are invasive procedures. Intravenous caffeine sodium benzoate therapy effectively relieves PDPH, but the headache may recur. An epidural blood patch is an invasive but effective, permanent treatment for PDPH in most cases; resistant cases may respond to epidural saline infusion.
Conclusion: The rate of PDPH after lumbar puncture can be minimized through strict attention to technique and the employment of a 25-gauge needle with the bevel parallel to the dural fibres. A reliable diagnosis and stepwise approach to treatment will minimize complications.

Objectif : Passer en revue les études actuelles et établir une démarche rationnelle en matière de cause, de prévention et de traitement de la céphalée de ponction durale (CPD).
Sources des données : On a obtenu des articles publiés de janvier 1980 et avril 1992 en consultant MEDLINE et *Index Medicus*. On a aussi examiné des articles de référence clés antérieurs à 1980.
Sélection d'études : On a tenu compte de tous les articles utiles et on les a soumis à une analyse critique.
Synthèse des données : Il y a CPD quand un écoulement lent de liquide céphalo-rachidien fait se contracter l'espace sous-arachnoïdien et cause ainsi une dilatation compensatrice des veines intracérébrales sensibles à la douleur. Le sexe féminin et un âge de 20 à 40 ans sont des facteurs de risque indépendants pour la CPD, contrairement à la grossesse. La fréquence du mal est directement proportionnelle au diamètre de l'aiguille utilisée et dépend en outre de la forme de la pointe de cette aiguille. L'hémo-obturation ou l'infusion saline épidurale à titre prophylactique après une ponction durale peuvent diminuer l'incidence, mais l'une et l'autre sont effractives. Une thérapie par intraveineuse à la caféine benzoate sodique a pour effet de soulager cette céphalée, qui peut cependant récidiver. L'hémo-obturation épidurale est un

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traitement effractif, mais efficace et durable dans la plupart des cas. Les sujets résistants pourront réagir à une infusion saline épidurale.

Conclusion : On peut réduire au minimum la fréquence de la CPD en se conformant strictement à la technique et en employant une aiguille de diamètre 25 dont le biseau est parallèle aux fibres durales. Les complications seront faibles grâce à un diagnostic sûr et à un traitement progressif.

The syndrome of postdural puncture headache (PDPH) was first described by Dr. August Bier, in 1898.[1] At that time he was conducting his initial experiments on the subarachnoid injection of cocaine. After undergoing the procedure himself he suffered a violent headache and provided the first written account of this, the most common complication of dural puncture. The classic "spinal headache" is described as a severe, dull, nonthrobbing pain usually fronto-occipital in location that is aggravated in the standing position and diminishes in the supine position. It is now recognized that the patient may also suffer from associated nausea, vomiting, visual disturbances, tinnitus or deafness.[2,3]

Modern anesthetic techniques have reduced the incidence of PDPH considerably. However, because of the current popularity of spinal and epidural anesthesia in fields such as obstetrics and orthopedics and the widespread use of dural puncture in radiologic and diagnostic procedures, PDPH continues to be a major problem for inpatients and outpatients. Furthermore, despite nearly 100 years of research there is still considerable controversy about the cause and treatment of PDPH. Much of the research has been complicated by several inherent problems: for example, there is no suitable animal model, and the range of possible human experimentation is necessarily limited; as well, it can be difficult to reproducibly quantify pain in a way that can be usefully compared between individuals. With these limitations in mind, I review the current research findings and attempt to synthesize a rational approach to the cause, prevention and treatment of PDPH.

Method

Articles published from January 1980 to April 1992 were obtained through a search of MEDLINE and *Index Medicus*. Key reference articles published before 1980 were also retrieved. All studies pertinent to the topic were included and critically analysed.

Results

Cause

Some reasonable hypotheses as to the cause of PDPH can be formulated from several observations made during the last century.

Age: The incidence of spinal headache is inversely proportional to the age of the patient after the age of 20 years.[4-7] The risk for a 25-year-old patient is roughly three to four times that for a 65-year-old one.[4-6] It is interesting that patients less than 20 years of age have been found to have a lower relative risk of PDPH than those aged 20 to 40, a reversal of the trend.[4,5] The physiologic reason for this pattern has not been determined.

Sex: A retrospective study in 1956 by Vandam and Dripps[4] involving 9277 patients given a spinal anesthetic demonstrated that women suffered PDPH twice as often as men. When Lybecker and associates[5] failed to reproduce this statistical difference among 1021 such patients they suggested that the previous results had been confounded by age because of the large number of young obstetric patients in the larger study. However, at least two recent well-constructed studies have shown a higher incidence of PDPH among women than among men regardless of age.[6,8] In one, there was a threefold increase in the risk of PDPH among women in their 30s relative to men in the same age group.[6] This difference disappeared almost completely by the fifth decade. Thus, it is reasonable to conclude that in young women there is a disproportionately high risk of PDPH, which decreases gradually until menopause, when men and women are equally susceptible.

Pregnancy: Although pregnancy is widely quoted as an independent risk factor, an extensive search of the literature provided no scientific evidence that pregnancy alone increases the relative risk of PDPH. The high incidence found in studies of obstetric samples may be a result of age and sex. Interstudy comparisons are invalid because of the wide variation of PDPH rates between centres. A single-centre cohort study involving obstetric patients and a group matched for age and sex is required to identify the risk attributable to pregnancy.

Antiseptics: Gurmarnik[9] reported an increased incidence of PDPH among patients who did not have povidone-iodine removed from the skin before dural puncture. This finding undoubtedly does not explain the cause of PDPH in most cases, but skin cleansers are plausible aggravating agents. An effort should be made to avoid introducing any preparatory agents into the subdural space by allowing them to dry before procedures involving intentional or potential dural puncture.

Anesthetic agents: In a randomized, double-blind study involving 2511 patients Naulty and collaborators[10] found a statistically significant difference in the incidence of PDPH depending on the local anesthetic solution used: lidocaine–glucose (9.54%), bupivacaine–glucose (7.64%) and tetracaine–procaine (5.85%). They hypothesized that the difference could be due to either

the different chemical structure (amides being more irritating than esters) or the glucose content (which would cause an instantaneous hyperglycemic peak in the cerebrospinal fluid [CSF]). Quaynor, Corbey and Berg[11] failed to show a difference in PDPH incidence between lidocaine and bupivacaine in a nonblind study involving 106 patients. However, their study may not have been sensitive enough to detect the small difference noted by Naulty and collaborators. Similarly, Vandam and Dripps[4] found no difference between tetracaine, procaine and dibucaine in their retrospective study. Like skin cleansers, anesthetic agents are probably not the sole cause of spinal headache but, rather, an aggravating factor. Even if a difference between anesthetics exists it is likely small and is outweighed by other considerations in the selection of the anesthetic.

CSF volume: It has been shown that a depletion of CSF volume is directly related to the development of headaches. In an elegant series of experiments in 1943 Kunkle, Ray and Wolff[12] demonstrated that removal of 15 to 20 mL of CSF by lumbar puncture rapidly and uniformly produced a headache, the characteristics of which were identical to those now described in PDPH. It was also demonstrated that replacement of the CSF volume with sterile crystalloid relieved the headache rapidly (in less than 4 minutes) and completely. Finally, a young woman suffering from a headache after a diagnostic lumbar puncture was found to have a low CSF pressure at repeat puncture. She reported resolution of her headache less than 2 minutes after intrathecal injection of saline; the withdrawal of CSF then caused an exact reproduction of the headache.[12] The design of the experiments was such that the patients were unaware of each manipulation as it was performed. On the basis of these results and findings published by Ray and Wolff,[13] two mechanisms to explain "drainage headache" were proposed. First, drainage of CSF results in the intracranial veins dilating to maintain a constant intracranial volume. These veins are pain sensitive, and dilation may directly result in discomfort. Second, extensive venodilation may result in increased brain volume to the extent that direct pressure is exerted on the pain-sensitive meninges.

The hypothesis of Kunkle and colleagues[12] remains the most plausible biologic explanation for PDPH. If a slow leak of CSF occurs through the dural puncture site, it is conceivable that sufficient volume could be lost to produce a headache. Furthermore, all the modern methods of prophylaxis against and treatment of PDPH can be explained with the use of this model.

Prevention

The prevention of PDPH usually revolves around minimizing the postpuncture leakage of CSF. Traditional methods, such as restricting the patient to a supine position for 24 hours and applying tight abdominal binders, have been shown to be marginally effective at best and have now largely been discarded because of patient discomfort.[2,3] Current efforts are centred on defining the optimum needle size, needle tip and technique of insertion. Most recently, debate has intensified about the use of an epidural blood patch or saline infusion for prophylaxis.

Posture: The length of postoperative recumbency has not been shown to be related to the incidence of PDPH.[5,14–16]

Needle design: There are three basic needle points available commercially: Quincke, Whitacre and Sprotte. The Quinke point is a simple cutting needle similar in design to the common venipuncture needle. The theoretic advantage of the Whitacre is its tapered "pencil-point" tip with lateral displacement of the distal orifice; this is intended to bring about an atraumatic splitting of the dural fibres rather than the cutting action of the Quincke. The Sprotte attempts to improve on this principle with a blunted ogival tip. In-vitro experiments have shown that a 22-gauge Whitacre produces less leakage than a 22-gauge Quinke after dural puncture.[17,18] However, the same studies demonstrated that a 22-gauge Whitacre produces markedly more leakage than a 25-gauge Quincke. Thus, in the laboratory the needle design has been found to be significant but of secondary importance to the needle calibre. Lynch and coworkers[8] performed a clinical trial using 22-gauge and 25-gauge Whitacre needles; both were associated with a lower incidence rate of PDPH (4% and 2% respectively) than is generally found with Quincke needles of comparable size (see "Needle diameter"), but neither offered a clear advantage over 26-gauge Quincke needles.[6,7,9–11,20–27] In a comparison of the 25-gauge Whitacre needle with the 24-gauge Sprotte needle for use in patients undergoing cesarean section the Sprotte design was found to produce PDPH in only 1 (0.4%) of 216 patients.[19] The conclusion from these trials is that needle size is of primary importance in preventing PDPH, but given two needles of identical size a Whitacre point will produce fewer cases of PDPH than a Quincke needle. A Sprotte needle may result in a significantly lower rate of PDPH than a Whitacre or Quincke needle, but more clinical trials are needed.

Needle diameter: The relation between needle size and PDPH incidence is difficult to quantify. Factors such as age, sex and operator play such an important role that widely varying rates are reported for each needle gauge. In general, however, the relative risk of PDPH decreases with each successive reduction in needle diameter. In the articles reviewed, a Quincke needle point caused PDPH with the following frequencies: 20 gauge 11% to 28%, 25 gauge 3% to 25%, 26 gauge 3% to 8% and 29 gauge 0% to 2%.[6,7,9–11,20–27] PDPH has been reported to occur in as many as 75% of patients suffering inadvertent dural puncture with a 16-gauge or 18-gauge Tuohy needle.[28,29] These clinical findings are supported by in-vitro studies of lumbar dura that have demonstrated a direct relation between needle diameter, residual dural hole diameter and CSF flow.[17,18,30]

The disadvantage of a smaller needle is the increased technical difficulty of dural puncture. Smaller needles tend to bend during insertion, do not give the characteristic "pop" upon puncture of the dura and require a longer period for the confirmatory CSF to appear at the hub after successful placement. The failure rate of attempted spinal anesthesia is significantly higher when a 29-gauge Quincke rather than a lower gauge needle is used and becomes prohibitive with a 30-gauge needle.[22,24-27]

Therefore, the correct size of needle for any dural puncture is the smallest gauge that allows the operator to perform the intended procedure with a reasonable success rate. Given its widespread availability and low cost the best needle overall for routine spinal anesthesia is probably a 25-gauge needle of Whitacre design.

Bevel direction: The elastic fibres in human dura run predominantly in a longitudinal direction. This has little consequence when symmetrical needle points such as the Whitacre or Sprotte are used but becomes important in the case of the Quincke point. In-vitro studies revealed that the shape but not the size of the dural hole varied according to the Quincke bevel direction in needles ranging in gauge from 20 to 29.[30] Two further studies demonstrated a higher rate of flow through isolated human dura when the needle bevel was perpendicular rather than parallel to the dural fibres.[17,18] These findings correlate with those from clinical trials that showed a significant increase in PDPH incidence when the needle bevel was perpendicular to the dural fibres.[5,31] Thus, care should be taken to ensure that Quincke point and epidural needles are passed with the bevel parallel to the longitudinal fibres of the dural membrane during intentional or potential dural puncture.

Angle of insertion: It has been suggested that an acute angle of approach to the dural membrane during puncture could create a flap valve and thus result in a lower rate of PDPH.[18] Although an in-vitro study demonstrated support for this theory[18] a limited in-vivo trial in published 1990 failed to show the clinical efficacy of this technique:[32] when 81 patients were randomly assigned to undergo puncture with a 22-gauge Quincke needle at either a midline (80° to 90°) or a laminar (20° to 55°) angle of approach, a similar rate of PDPH was found in both groups (2.8% and 6.7% respectively). Thus, further supporting evidence is needed before this method can be recommended.

Epidural blood patch: The introduction of 5 to 20 mL of homologous blood into the epidural space has been used to treat PDPH in clinical practice for at least 30 years.[33] More recently, this technique has undergone evaluation as prophylaxis against PDPH, especially when puncture results from large-bore (gauge 16 to 18) needles. Theoretically, a prophylactic blood patch would coagulate in the epidural space, sealing the dural rift before enough CSF could escape to produce a headache. In two studies[34,35] a total of 13 patients were treated with a prophylactic blood patch of 15 to 20 mL after accidental dural puncture during epidural anesthesia. No PDPH was reported in either series. A prospective randomized study published in 1989 involving 39 patients demonstrated a PDPH rate of 80% among patients suffering inadvertent dural puncture during epidural anesthesia.[36] The rate was only 21% when a prophylactic epidural blood patch was applied.

Reports of major acute complications after an epidural blood patch are rare and have never demonstrated a causal relation.[37,38] A concern is the effect of such a patch on future epidural anesthesia. A retrospective analysis by Ong and associates[39] showed that a block of satisfactory distribution was achieved in only two thirds of patients undergoing epidural anesthesia after a remote accidental dural puncture. However, this was true for both conservatively treated patients and those who had received an epidural blood patch. Thus, the authors postulated that the lower success rate was the result of the accidental dural puncture rather than the blood patch.

Other complications are rare. Two cases of facial nerve (cranial nerve VII) paralysis after a blood patch were described, both of which resolved spontaneously.[40,41] One patient was reported to have suffered protracted dizziness, tinnitus, vertigo and ataxia.[40] The immediate side effects on application of a blood patch include discomfort or pain in the back, buttocks or legs, presumably secondary to nerve-root compression. This can be minimized by the use of slow injection and the patient-limited technique, first advocated by Crawford:[42] a volume of up to 20 mL is used, and application is stopped as soon as radicular symptoms are encountered. A significant proportion (up to 19%) of patients experience mild lower back discomfort for several days after the patch.[40,42]

Although this procedure appears effective, objections to its use have arisen on the grounds that PDPH is a self-limited condition and does not occur in all patients.[43] Given the swift and effective treatment available with caffeine, prophylactic blood patches are probably not justified even in groups at high risk for PDPH.

Epidural placement of saline: The epidural placement of saline as a constant infusion or as repeated boluses has been advocated as an alternative to an epidural blood patch. The following studies refer to prophylaxis in patients suffering accidental dural puncture during epidural anesthesia. In 1972 Crawford[44] reported the infusion of 1.0 to 1.5 L of Hartmann's solution over 24 hours after anesthesia. PDPH developed in 31% patients (5 out of 16), as compared with 77.5% in an identical untreated population reported previously by Crawford.[28] In 1973 Craft, Epstein and Coakley[29] used two 60-mL boluses of normal saline within 24 hours to treat 17 patients and followed 16 others as controls. The incidence of PDPH was found to be 12.5% in the treatment group and 76.5% in the control group; however, the trial was neither randomized nor blinded. Smith[45] repeated the experiment in 1979, modifying it to include 30 to 60 mL of

normal saline (limited by patient discomfort) every 6 hours for a total of 24 hours after puncture. One (8%) of 13 patients had PDPH, as compared with an overall rate of 64% before prophylactic measures were instituted. Most recently, Okell and Sprigge[46] repeated Crawford's original experiment using 1 L of Hartmann's solution over 24 hours. Eleven patients were treated, and 10 patients were selected as controls. It was concluded that epidural infusion did not significantly reduce the incidence of PDPH in the treatment group but did delay the onset of headache by 24 to 48 hours.

From these studies the effect of prophylactic saline on the relative risk of PDPH cannot easily be quantified. It seems that some reduction in incidence does occur; however, it is difficult to recommend such an invasive procedure, since a large number of healthy patients will receive unnecessary treatment.

Treatment

Therapy for PDPH begins with proper diagnosis. Only headaches that are substantially affected by posture, as originally described by Bier,[1] should be considered PDPH. Other headaches are likely not the result of the pathophysiologic mechanisms proposed by Kunkle and associates[12] and therefore will not respond to the specific therapies for PDPH. Misdiagnosis is a strong possibility, because after spinal anesthesia the rate of headaches that are not PDPH varies between 5% and 16%[5,6,10,24] (Table 1). In particular, the physician should take care to rule out treatable catastrophic medical problems such as meningitis and subarachnoid bleeding.

Once the diagnosis is made, most authors recommend 24 hours of conservative therapy, since the natural history is one of spontaneous resolution.[2,3,47] If the headache persists and is disabling to the patient, or if nausea, vomiting, visual disturbance or tinnitus occurs the diagnosis should be reconsidered. Once other important causes of severe headache are again ruled out, the therapeutic options include caffeine given intravenously, an epidural blood patch and epidural administration of saline.

Conservative measures: Bed rest is effective in that it avoids the upright position, which aggravates PDPH. Analgesics given orally, including mild opiates, also routinely bring some relief during the observational period. Aggressive hydration and tight abdominal binders are no longer recommended.

Caffeine: According to the mechanism proposed by Kunkle and associates[12] PDPH results in part from dilation of the intracranial veins. Caffeine is a methylxanthine known to produce vasoconstriction specific to the cerebral vasculature.[48] These facts, coupled with empirical evidence of efficacy, prompted Sechzer and Abel[49] to carry out a randomized double-blind study of intravenous treatment with caffeine sodium benzoate. Of 104 patients suffering from PDPH 63 responded to conservative therapy. The remaining 41 patients were randomly assigned to receive either 0.5 g of caffeine sodium benzoate or 2 mL of normal saline, each delivered as a slow intravenous bolus. Two hours after the first bolus all patients with persistent headache received the caffeine. Three (15%) in the control group reported relief of their headache after administration of the saline, as compared with 15 (75%) in the group initially given caffeine. Of the 38 subjects eventually receiving caffeine 27 (71%) responded, but 8 (21%) later suffered a recurrence of their headache. Jarvis, Greenawalt and Fagraeus[50] produced a modified protocol for intravenous therapy with caffeine in 1986. Their technique called for 0.5 g of caffeine sodium benzoate in 1 L of isotonic fluid over the first hour, to be repeated if the headache is not relieved after 4 hours. Relief was reported in 14 (78%) of 18 patients, but no mention was made of PDPH recurrence.

Table 1: Differential diagnosis of headache after dural puncture

Postdural puncture headache
Tension headache
Migraine headache
Subarachnoid hemorrhage
Meningitis

Intravenous caffeine sodium benzoate therapy is less expensive and more easily administered than an epidural blood patch. Furthermore, there is no evidence that this dosage of caffeine will produce adverse side effects.[48,51,52] Thus, the rates of response (roughly 75%) and of permanent relief (50%) probably justify the employment of this technique when conservative management fails and before more invasive methods are used. Another future alternative is the oral administration of anhydrous caffeine, which should produce a serum level profile similar to that after a 1-hour infusion of caffeine sodium benzoate. A well-designed clinical study is needed to demonstrate the efficacy of the oral route.

Epidural blood patch: When a patch is used in patients suffering from moderate to severe or prolonged PDPH a success rate of 96% to 98% can be expected.[40,42,53] Two mechanisms likely bring about this effect: formation of a plug in the dural defect, which stops the loss of CSF, and simultaneous reduction in the volume of the subarachnoid space through expansion of the epidural space, which eliminates the relative CSF deficiency. Although it is invasive, the immediate adverse effects are mild, and long-term complications are rare. This therapy is justified for patients with moderate to severe PDPH that has not responded quickly to conservative measures and caffeine.

Epidural placement of saline: The epidural administration of saline to relieve PDPH predates epidural blood patching by at least a decade. Rice and Dabbs[54] pioneered a bolus technique, as described in 1950, and found that 100% of their patients suffering from PDPH obtained relief. However, roughly 50% were later found to have a recurrence of their symptoms. In 1973 Craft,

Epstein and Coakley[29] reported a similar experience using a bolus technique. As with epidural blood patching, the initial saline bolus into the epidural space would result in compression of the subarachnoid space and thus improve the relative CSF deficiency. However, saline would be more prone to dissipation through tissue plains and to rapid reabsorption than would whole blood. Therefore, saline would be expected to disperse quickly from the epidural space, allowing re-expansion of the subarachnoid space and return of the headache.

Epidural saline infusion would theoretically avoid this problem. Two patients in whom patching had failed to relieve PDPH received epidural infusions of normal saline, and success was reported after about 24 hours.[55] In 1988 Stevens and Jorgensen[56] reported the successful treatment of a particularly stubborn case of PDPH by means of a 24-hour infusion followed by an epidural blood patch. Although the technique of epidural saline infusion has met with initial success, few data are yet available regarding efficacy. Similarly, there is little to suggest that fewer short-term or long-term complications result from this method than from blood patching. Moreover, the patient suffers at the very least from the inconvenience of undergoing epidural saline infusion for 24 hours. Therefore, until further clinical trials are performed, an epidural blood patch is preferred when invasive therapy is required. However, epidural saline infusion should be considered when PDPH is resistant to more proven forms of therapy.

Recommendations

In light of the reviewed data a number of recommendations can be made about the prevention and treatment of PDPH.

Prevention

1. Pay particular attention to technique in patients between the ages of 20 and 40 years. Women in this age group are highly prone to PDPH.
2. Ensure that antiseptics are allowed to dry as completely as possible before dural puncture.
3. Use a 25-gauge Whitacre needle point for dural puncture unless specific factors dictate the use of a larger instrument.
4. Orient the needle bevel parallel to the longitudinal dural fibres during the needle insertion.
5. Avoid invasive prophylactic measures such as epidural blood patching and epidural saline infusion.

Treatment

1. Ensure that the diagnosis of PDPH is correct.
2. Initiate conservative therapy with bed rest and analgesics administered orally.
3. If the headache persists beyond the time of planned discharge, if it becomes moderate to severe in intensity and restricts the patient's activities or if nausea, vomiting, visual disturbance or tinnitus occurs, reconsider the diagnosis before proceeding with therapy.
4. If conservative measures fail, attempt control with an intravenous bolus of 500 mg of caffeine sodium benzoate.
5. The intravenous caffeine sodium benzoate therapy may be repeated if the headache is not relieved within 2 to 4 hours.
6. An epidural blood patch should be used for headaches resistant to intravenous caffeine sodium benzoate therapy.
7. Failing all else, epidural saline infusion can be instituted.

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References

1. Calverley RK: Anesthesia as a specialty: past, present, and future. In Barash PG, Cullen BF, Stoelting RK (eds): *Clinical Anesthesia*, Lippincott, Philadelphia, 1989: 12.12
2. Reid JA, Thorburn J: Headache after spinal anaesthesia [E]. *Br J Anaesth* 1991; 67: 674–677
3. Weeks SK: Spinal headache — prevention and treatment. *Can J Anaesth* 1990; 37 (4 pt 2): Sliii–Slviii
4. Vandam LD, Dripps RD: Long-term follow-up of patients who received 10,098 spinal anesthetics. Syndrome of decreased intracranial pressure (headache and ocular and auditory difficulties). *JAMA* 1956; 161: 586–591
5. Lybecker H, Moller JT, May O et al: Incidence and prediction of postdural puncture headache. A prospective study of 1021 spinal anesthesias. *Anesth Analg* 1990; 70: 389–394
6. Flaatten H, Rodt S, Rosland J et al: Postoperative headache in young patients after spinal anaesthesia. *Anaesthesia* 1987; 42: 202–205
7. Rasmussen BS, Blom L, Hansen P et al: Postspinal headache in young and elderly patients. Two randomized, double-blind studies that compare 20- and 25-gauge needles. *Anaesthesia* 1989; 44: 571–573
8. Lynch J, Krings-Ernest I, Strick K et al: Use of a 25-gauge Whitacre needle to reduce the incidence of postdural puncture headache. *Br J Anaesth* 1991; 67: 690–693
9. Gurmarnik S: Skin preparation and spinal headache [C]. *Anaesthesia* 1988; 43: 1057–1058
10. Naulty JS, Hertwig L, Hunt CO et al: Influence of local anesthetic solution on postdural puncture headache. *Anesthesiology* 1990; 72: 450–454
11. Quaynor H, Corbey M, Berg P: Spinal anaesthesia in day-care surgery with a 26-gauge needle. *Br J Anaesth* 1990; 65: 766–769
12. Kunkle EC, Ray BS, Wolff HG: Experimental studies on headache. Analysis of the headache associated with changes in intracranial pressure. *Arch Neurol* 1943; 49: 323–358
13. Ray BS, Wolff HG: Experimental studies on headache: pain-sensitive structures of the head and their significance in headache. *Arch Surg* 1940; 41: 813–856

14. Thornberry EA, Thomas TA: Posture and post-spinal headache. A controlled trial in 80 obstetric patients. *Br J Anaesth* 1988; 60: 195–197

15. Cook PT, Davies MJ, Beavis RE: Bed rest and post lumbar puncture headache. The effectiveness of 24 hours' recumbency in reducing the incidence of post lumbar puncture headache. *Anaesthesia* 1989; 44: 389–391

16. Carbaat PAT, VanCrevel H: Lumbar puncture headache: controlled study on the preventive effect of 24 hours' bed rest. *Lancet* 1981; 2: 1133–1135

17. Cruickshank RH, Hopkinson JM: Fluid flow through dural puncture sites. An in vitro comparison of needle point types. *Anaesthesia* 1989; 44: 415–418

18. Ready LB, Cuplin S, Haschke RH et al: Spinal needle determinants of rate of transdural fluid leak. *Anesth Analg* 1989; 69: 457–460

19. Cesarini M, Torrielli R, Lahaye F et al: Sprotte needle for intrathecal anaesthesia for caesarean section: incidence of postdural puncture headache. *Anaesthesia* 1990; 45: 656–658

20. Thomas DV: Spinal anaesthesia in obstetrics: headache and special needles [C]. Ibid: 1100–1101

21. Barker P: Are obstetric spinal headaches avoidable? *Anaesth Intensive Care* 1990; 18: 553–554

22. Geurts JW, Haanschoten MC, Van Wijk RM et al: Post-dural puncture headache in young patients. A comparative study between the use of 25-gauge and 29-gauge spinal needles. *Acta Anaesthesiol Scand* 1990; 34: 350–353

23. Sarma VJ, Bostrom U: Intrathecal anaesthesia for day-care surgery. A retrospective study of 160 cases using 25- and 26-gauge spinal needles. *Anaesthesia* 1990; 45: 769–771

24. Flaatten H, Rodt SA, Vamnes J et al: Postdural puncture headaches. A comparison between 26- and 29-gauge needles in young patients. *Anaesthesia* 1989; 44: 147–149

25. Dahl JB, Schultz P, Anker-Moller E et al: Spinal anaesthesia in young patients using a 29-gauge needle: technical considerations and evaluation of postoperative complaints compared with general anaesthesia. *Br J Anaesth* 1990; 64: 178–182

26. Collins PD: Obstetric spinal headache [C]. *Anaesth Intensive Care* 1991; 19: 477–478

27. Lesser P, Bembridge M, Lyons G et al: An evaluation of a 30-gauge needle for spinal anaesthesia for caesarean section. *Anaesthesia* 1990; 45: 767–468

28. Crawford JS: Lumbar epidural block in labour: a clinical analysis. *Br J Anaesth* 1972; 44: 66–74

29. Craft JB, Epstein BS, Coakley CS: Prophylaxis of dural-puncture headache with epidural saline. *Anesth Analg* 1973; 52: 228–231

30. Dittmann M, Schafer HG, Ulrich J et al: Anatomical re-evaluation of lumbar dura mater with regard to postspinal headache. Effect of dural puncture. *Anaesthesia* 1988; 43: 635–637

31. Norris MC, Leighton BL, DeSimone CA: Needle bevel direction and headache after inadvertent dural puncture. *Anesthesiology* 1989; 70: 729–731

32. Stasiuk RBP, Jenkins LC: Post spinal headache: a comparison of midline and laminar approaches. *Can J Anaesth* 1990; 37 (4 pt 2): S58

33. Gormley JB: Treatment of postspinal headache. *Anesthesiology* 1960; 21: 565–566

34. Quaynor H, Corbey M: Extradural blood patch — Why delay? *Br J Anaesth* 1985; 57: 538–540

35. Ackerman WE, Colclough GW: Prophylactic epidural blood patch: the controversy continues [C]. *Anesth Analg* 1987; 66: 913

36. Colonna-Romano P, Shapiro BE: Unintentional dural puncture and prophylactic epidural blood patch in obstetrics. *Anesth Analg* 1989; 69: 522–523

37. Leivers D: Total spinal anesthesia following early prophylactic epidural blood patch. *Anesthesiology* 1990; 73: 1287–1289

38. Leighton BL: A complication following prophylactic blood patch: Spinal or subdural anesthesia [C]? *Anesthesiology* 1991; 74: 1166–1167

39. Ong BY, Graham CR, Ringaert KRA et al: Impaired epidural analgesia after dural puncture with and without subsequent blood patch. *Anesth Analg* 1990; 70: 76–79

40. Abouleish E, de la Vega S, Blendinger I et al: Long-term follow-up of epidural blood patch. *Anesth Analg* 1975; 54: 459–463

41. Lowe DM, McCullough AM: 7th nerve palsy after extradural blood patch. *Br J Anaesth* 1990; 65: 721–722

42. Crawford JS: Experiences with epidural blood patch. *Anaesthesia* 1980; 35: 513–515

43. Woodworth GE: Prophylactic epidural blood patch in obstetrics [C]. *Anesth Analg* 1990; 70: 568–569

44. Crawford JS: The prevention of headache consequent upon dural puncture. *Br J Anaesth* 1972; 44: 598–599

45. Smith BE: Prophylaxis of epidural "wet tap" headache [abstr]. *Anesthesiology* 1979; 51: S304

46. Okell RW, Sprigge JS: Unintentional dural puncture. A survey of recognition and management. *Anaesthesia* 1987; 42: 1110–1113

47. Parnass SM, Schmidt KJ: Adverse effects of spinal and epidural anaesthesia. *Drug Saf* 1990; 5: 179–194

48. Rall TW: Drugs used in the treatment of asthma. In Gilman AG, Rall TW, Nies AS et al (eds): *Goodman and Gilman's The Pharmacological Basis of Therapeutics*, 8th ed, Pergamon Toronto, 1990: 619–630

49. Sechzer PH, Abel L: Post-spinal anesthesia headache treated with caffeine. Evaluation with demand method. Part 1. *Curr Ther Res* 1978; 24: 307–312

50. Jarvis AP, Greenawalt JW, Fagraeus L: Intravenous caffeine for postdural puncture headache [C]. *Anesth Analg* 1986; 65: 316–317

51. McEvoy GK (ed): Respiratory and cerebral stimulants. In *American Hospital Formulary Service Drug Information 91*, Am Soc Hospital Pharmacists, Bethesda, Md, 1991: 1317–1319

52. Ellenhorn MJ, Barceloux DG: Over-the-counter products. In *Medical Toxicology, Diagnosis and Treatment of Human Poisoning*, Elsevier, New York, 1988: 508–514

53. Shah JL, Veness AM: Epidural blood patch using a catheter. Diagnosis of an unrecognized dural tap. *Anaesthesia* 1985; 40: 1120–1123

54. Rice GG, Dabbs CH: The use of peridural and subarachnoid injections of saline solution in the treatment of severe postspinal headache. *Anesthesiology* 1950; 11: 17–23

55. Baysinger CL, Menk EJ, Harte E et al: The successful treatment of dural puncture headache after failed epidural blood patch. *Anesth Analg* 1986; 65: 1242–1244

56. Stevens RA, Jorgensen N: Successful treatment of dural puncture headache with epidural saline infusion after failure of epidural blood patch. *Acta Anaesthesiol Scand* 1988; 32: 429–431