Complications associated with regional anaesthesia for Caesarean section

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Introduction

Spinal anaesthesia (SA) has been used for obstetric analgesia since the early 1900's. Accordingly, it has been recognized for over a century that this technique is not without significant side effects. Postdural puncture headache due to the use of large bore spinal needles was common, but more serious was the high mortality rate associated with SA. This was largely due to the misconception that untrained personnel could administer SA, and that monitoring was not required. By the 1930's the mortality rate for Caesarean section (CS) under SA was reported as 1 in 139, and by the 1950's SA was cited as the most dangerous form of obstetric anaesthesia (OA).[1]

Since the 1960's there have been major scientific developments that have improved the safety of both SA and epidural anaesthesia in parturients. Regional anaesthesia (RA) now has a mortality rate seventeen times less than GA for CS, and is internationally advocated for this use.[2]

Despite this known relative safety of SA for CS, there is now evidence that in South Africa, the number of maternal deaths secondary to SA is increasing.[3] This may be because inexperienced and unsupervised junior doctors are being sent to peripheral hospitals where they are required to perform OA. As they are better trained in the administration of SA and feel safer with this technique than GA, these junior doctors predominantly use SA. Due to the practitioner's inexperience, complications, when they arise, are difficult to overcome. This is in contrast with more experienced general practitioners, who are more likely to use GA, since they were trained to use this technique, and better equipped to deal with complications.

It is probable that although there is a significant morbidity and mortality associated with SA, the complication rate would be higher if inexperienced doctors were to only use GA for CS.

The aim of this paper is to promote awareness of the complications of RA for CS so they may be recognized early and appropriately managed, thereby reducing morbidity and mortality in South Africa.

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Complications of Regional Anaesthesia for CS

The three categories of RA currently employed for CS are:
1. Spinal (subarachnoid),
2. Epidural, and
3. Combined Spinal-Epidural (CSE) anaesthesia.

In the public sector most Caesarian Sections are performed using SA. In this setting, epidural anaesthesia for labour is infrequently used, so relatively few epidurals are extended for CS.

Both maternal and fetal complications may occur as a consequence of the use of RA. These may develop during administration of the RA, intraoperatively or in the postpartum period.

Complications arising during administration of RA:

1. Anxiety related

The preparation required for RA is stressful for the patient, and can result in anxiety-related physiological changes. Vasovagal episodes are not uncommon and are more severe when the patient is in the sitting than the lateral position. Severe episodes can result in maternal asystole[4] and fetal compromise.[5] Anxiolytic premedication is usually omitted lest it causes neonatal depression, but when the mother is very anxious, 1mg of midazolam IV may help prevent vasovagal episodes.[6]

2. Equipment related

Technical problems related to the spinal needle are more frequent when the CSE needle through needle technique is used. If the epidural space is located with a curved Huber tip epidural needle and a fine gauge spinal needle inserted through this, the curve may bend the spinal needle tip resulting in the inability to withdraw the stylet[7], or causing no cerebrospinal fluid (CSF) to flow, due to blockage by the kinked tip.[8]

Blocked epidural catheters secondary to a defective component have resulted in technical failure. The obstruction is often located in the catheter connector and remedied simply by changing the connector.[9,10]

3. Nerve damage

Pain or paraesthesia during needle insertion or anaesthetic injection is worrying as it may be a sign of iatrogenic nerve damage.[11] A previously unrecognised spinal malformation may be unmasked[12], or the anaesthetist can easily misidentify the lumbar interspace level and insert the needle too high and into the spinal cord. Wherever possible, the spinal needle should be inserted below the spinous process of L3.[13] Care must also be taken when subcutaneous skin infiltration of local anaesthetic is given prior to the spinal needle insertion, as spinal cord injury has resulted from the patient inadvertently rolling onto the infiltration needle.[14]

If an epidural catheter is being sited, loss of resistance to saline is preferable to air to identify the epidural space, as the use of air has been associated with a higher incidence of dural puncture and pneumoencephalus.[15,16]

Complications arising soon after administration of RA or during CS

1. Failure of technique

Reasons for having to convert to GA preoperatively after SA has been attempted, include inadequate block height, inability to locate a space, paraesthesia on injection and communication problems. Panic attacks due to claustrophobia may also require conversion to GA. Intraoperatively, pain is the commonest reason for technique failure. Figures quoted for the incidence of failure of SA range from 1.7[17] to 2.9%.[18]

The conversion rate for epidural anaesthesia to GA varies considerably between centers, with a mean of 6% from a UK national survey.[17] The commonest reason for conversion when an epidural catheter is in situ, is lack of time due to a non-reassuring fetal heart trace, rather than inadequate epidural anaesthesia. Exteriorisation of the uterus is best avoided, since the associated pain may also necessitate conversion to GA.[18]

CSE offers greater flexibility than SA alone, as the SA gives a denser block , which can be extended using the epidural catheter. Failure rates with either component of the CSE depend on the type of CSE and equipment used.[19]

2. Hypotension

All methods of RA can cause hypotension, but it is more troublesome after the rapid onset of sympatholysis following SA. Depending on the dose used, it can occur in over 80% of parturients having SA or CSE for CS, and may be detrimental for both fetus and mother.[20] The uterine blood supply is not subject to autoregulation, hence a maternal systolic pressure of less than 100mmHg can cause "pathologic" fetal bradycardia.[21]

Methods commonly used to prevent or treat hypotension, include fluid preloading, the use of hyperbaric bupivacaine, positioning to relieve aorto-caval compression and vasopressor therapy.[22]

All of these methods have their advocates and critics.[23,24,25] Even the recommended 15° left table tilt position is associated with aorto-caval compression and may be inadequate if hypotension occurs.[26] Large volumes of crystalloid preload (>1000ml) usually do not eliminate hypotension as the fluid is rapidly redistributed, and should be avoided in the pre-eclamptic parturient as pulmonary oedema may ensue. Colloids are more expensive, and a major anaphylactoid reaction due to hydroxyethylstarch has occurred when given as the preload

to a mother with HELLP syndrome.[27] In the healthy parturient 20ml/kg of a crystalloid (an isotonic balanced salt solution) administered at the time of induction of SA may be associated with a lower requirement for vasopressor than a conventional preload.[28] A maximum of 10ml/kg is advisable in the severe pre-eclamptic parturient.

Ephedrine, the standard first-line vasopressor, should be given early in response to hypotension, to keep maternal blood pressure close to baseline values.[29] Ephedrine use prior to delivery has been related to fetal acidosis (due to a β-adrenergic fetal metabolic effect), but neonatal outcomes appear unaffected. There is a suggestion that in the compromised, already acidotic foetus, this ephedrine related acidosis could become clinically relevant, and some authors now recommend phenylephrine as the first line vasopressor.[30]

Phenylephrine has traditionally been avoided in pregnancy after studies in pregnant ewes showed that it caused impaired uteroplacental blood flow. Subsequent studies of phenylephrine in human pregnancy, support its value as a vasopressor during SA for CS.[31]

Ephedrine is in practice a safer drug to administer as small inaccuracies in dose administration are less likely to cause severe hypertension than phenylephrine. Phenylephrine must be diluted to 50µg/ml and only small boluses (25-50µg) given. A baroreceptor mediated bradycardia can result from a raised afterload and hypertension if larger doses are given; atropine or glycopyrrolate should be avoided when the blood pressure is normal or raised, since severe tachycardia and hypertension may result.[32]

Rarely, severe hypotension with bradycardia can occur after SA, secondary to activation of the Bezold-Jarisch reflex (acute underfilling of left ventricle leading to activation of vagal afferents).[33] Prompt treatment with adequate doses of vasopressors is required, as cardiac arrest may follow. If cardiac arrest does occur, the fetus should be rapidly delivered in order to improve both maternal and fetal survival prospects.[34] Delivery benefits the mother by alleviating aortocaval compression, which makes cardiopulmonary resuscitation more effective, and also reduces maternal oxygen requirements and carbon dioxide production.

At delivery oxytocin is given to contract the uterus. This drug has other significant pharmacological actions: it causes systemic vasodilatation (and coronary vasoconstriction) and must be cautiously administered, if at all, if the patient has become hypotensive from SA or haemorrhage, as the combination of vasodilatation and circulatory insufficiency can prove fatal.[35]

3. High Motor Block

The classic symptoms of a total spinal block are generally well known to anaesthetists and include a rapid cephalad progression of sensory block, paralysis, apnoea and hypotension. The standard principles of resuscitation (airway, breathing, circulation) are required. Airway protection to avoid aspiration and circulatory support with relief of aortocaval compression are additional resuscitative requirements in the parturient.

High motor blocks of a lesser degree may also result in difficulties in talking, swallowing and breathing, according to the level of the block. Intubation may be required to prevent aspiration and adequately oxygenate the patient. This complication is often related to high doses of intrathecal local anaes-

thetic, but may occur when standard doses are used. Although a high motor block usually occurs soon after the induction of SA, it may be delayed, and result from a change in the position of the patient. Hence the patient must at no times be left unmonitored for at least 90 minutes after SA.

High motor block can also occur after accidental SA injection via a previously functional epidural catheter, and after a SA is given when an epidural block fails. The latter situation often arises when an urgent CS is required in a patient whose epidural block for labour analgesia has failed, or was associated with an accidental dural puncture. The use of spinal anaesthesia in this situation is controversial.[36] Epidural injections of fluid (saline or local anaesthetic) can significantly compress the lumbar subarachnoid space and cause significant rises in level of block of SA.[37] If time permits, the residual epidural block can be extended with a second, low thoracic epidural catheter or a CSE with a reduced dose of SA bupivacaine. If a single-shot SA is used, careful patient positioning restricting the spread of hyperbaric bupivacaine should be employed.[38]

4. Loss of Consciousness
This is usually due to severe hypotension from a high spinal block. On occasion a patient can lose consciousness while haemodynamically stable, and this has been attributed to subdural spread of anaesthesia. Here the signs of cephalad extension of the block are usually slower than with an immediate total spinal block, and patients may complain of dyspnoea, weakness of the arms or dysarthria. There may be no warning before the loss of consciousness (LOC) suddenly occurs some time after the block has been inserted.[39]

Other causes of LOC during CS include air[40] or amniotic fluid embolism, pulmonary embolism[41], inadvertent sedative drug administration or hysteria. These require systematic exclusion.

5. Intravenous injection of local anaesthetic
With the advent of multi-orifice epidural catheters, the widespread use of aspiration to test for intravascular placement, and the fractionation of epidural doses, this complication of EA for CS is now infrequent. Test-doses with adrenaline may give confusing results and should be avoided in pre-eclamptics as a hypertensive crisis can be precipitated or uterine blood flow reduced.

6. Shivering
This can be a troublesome side effect of RA for CS for both the mother and the anaesthetist. It interferes both with maternal comfort and monitoring. Moreover, shivering increases maternal oxygen consumption and particular efforts should be taken to avoid shivering when other pathology resulting in maternal or fetal hypoxaemia is present. The aetiology of shivering associated with SA is complex and poorly understood. It occurs in women who have both low core temperatures (thermoregulatory shivering) and core temperatures>38.0˚C (nonthermoregulatory shivering).

    (a) Thermoregulatory shivering: RA causes loss of sympathetically mediated peripheral vasoconstriction, with redistribution of heat from the central core to the periphery. This can result in a rapid fall in core temperature and thermoregulatory shivering. Skin warming with a forced-air warmer can prevent this by increasing skin temperature and thus reducing heat redistribution.[42]

    Other standard measures such as maintaining an adequate environmental temperature and avoidance of prolonged skin exposure and cold intravenous fluids, are also of benefit. EA causes more shivering during CS than SA, as the latter is associated with a more rapid increase in skin temperature from vasodilatation, and thus blunting of the hypothermic response.[43]

    (b) Non-thermoregulatory shivering:
    Epidural anaesthesia for labour is known to cause a rise in maternal temperature.[44] In 5% of mothers the temperature may exceed 38˚C, and whilst generally not of significance, this has been associated with fetal tachycardia.
    Shivering can occur with high core and high skin temperatures, which suggests "passive" hyperthermia, as opposed to shivering with a high core but low skin temperature, which indicates "febrile" hyperthermia, being actively maintained by the patient's thermoregulatory system. Predictive factors and mechanisms for hyperthermia with shivering during epidural anaeshesia are unknown.[45]

In clinical practice one must be aware that shivering can be due to both low and high maternal core temperatures. Therefore, core temperature should be measured before active warming.

Pharmacological treatment of shivering includes epidural fentanyl(25µg)[46] and intrathecal pethidine.[47] Intravenous pethidine and clonidine (30µg)[48] are effective, but may be associated with fetal and maternal sedation..

7. Adverse effects related to intrathecal opioids
Fentanyl and/or morphine are frequently administered in combination with intrathecal bupivacaine, to enhance and prolong intra- and postoperative analgesia.

Intrathecal (IT) morphine is clinically useful for postoperative analgesia, but is associated with significant side effects. Even the currently recommended dose of 0.1mg, which provides good analgesia for up to 11 hours postoperatively, will result in an estimated incidence of 43% for pruritus, 10% for nausea and 12% for vomiting.[49] Using this low dose, delayed respiratory depression is very uncommon in the obstetric population, although its exact incidence is unknown. In one review it was observed in only one patient (out of 485 patients) and the number needed to harm (NNH) for respiratory depression calculated as 476 (95% confidence interval 164 - ∞).[50] Higher doses may be associated with a higher incidence of clinically significant respiratory depression after CS.[51] Rarely, IT morphine for CS may cause postoperative shivering, hypothermia and excessive sweating. The mechanism probably involves a disturbance of hypothalamic thermoregulatory mechanisms after cephalad spread of morphine, since naloxone antagonised these effects.[52]

Intrathecal fentanyl (12.5µg) for CS provides better intraoperative analgesia than IV fentanyl, with fewer side-effects such as nausea, vomiting and hypotension. However fentanyl provides little benefit in terms of postoperative analgesia.[49] Pruritus is dose-dependent, and the incidence is higher when given IT (26%) than IV (8%).[53]

8. Myocardial ischaemia/infarction

Electrocardiographic (ECG) changes suggestive of myocardial ischaemia are common during CS.[54] Although these changes occasionally may be associated with symptoms of chest pain and dyspnoea, measurement of cardiac enzymes and echocardiographic analysis suggest that these ST changes are not associated with myocardial injury, but related to tachycardia induced ECG changes.[55,56]

Excessive doses of vasopressors during SA for CS have led to myocardial infarction, secondary to coronary artery plaque rupture, in a patient with no previous history of coronary artery disease. Whilst this is a rare event, it is prudent to maintain perioperative haemodynamic stability.[57]

Postoperative complications

1. Postdural puncture headache (PDPH)

Dural puncture can lead to CSF loss with resultant intracranial hypotension, compensatory venodilatation and headache.[58] The larger the size of the needle puncturing the dura, the greater the CSF loss, and incidence of headache. The incidence of PDPH can be as high as 70% if a Tuohy needle has been used.[59] Pencil-point needles separate rather than cut dural fibres and produce less PDPH than cutting needles, but may be associated with a higher incidence of paraesthesia and potential nerve damage, since the orifice is proximal to the needle tip. The Atraucan® needle with an atraumatic and narrow cutting bevel has recently been marketed as a compromise. It has a lower incidence of paraesthesia than pencil-point needles and a relatively low incidence of PDPH for a beveled needle.[60]

PDPH usually starts within three days of dural puncture. It is characteristically postural, exacerbated by standing. In a few cases, more severe neurological complications have developed, including intracranial haematomas due to rupture of thin-walled dural blood vessels following reduction of CSF pressure. The diagnosis may be difficult, since a chronic subdural haematoma may be mistaken for psychiatric disease[61], and seizures may be diagnosed as eclamptic rather than secondary to raised intracranial pressure associated with haematoma.[62] If the diagnosis is in doubt, a computerized tomographic (CT) brain scan is of value.

Untreated, a PDPH usually resolves after a week, but can persist indefinitely in a minority of cases. Treatment is with an epidural blood patch. An early report suggesting that a better success rate is achieved by delaying the blood patch for 24 hours[63] has been criticized. It was an uncontrolled, retrospective study, in which the patients who had dural punctures with smaller needles were only in the late treatment group. Unless PDPH symptoms are severe, it is recommended to wait 24 hours before performing a blood patch, in order to confirm the diagnosis and ensure the spinal block has completely regressed.[64] The success rate for the blood patch is then estimated at 70-80%[65], and this is also the success rate for repeating the patch.

Intrathecal saline (10ml) injected immediately after an accidental dural tap (or through an intrathecal catheter inserted after the inadvertent dural tap) has also been shown to reduce the incidence of PDPH and the need for blood patch.[66] Most other treatments are ineffective.

2. Intraspinal haematoma

Only 2 cases in over 900,000 spinal blocks in two combined series have been reported, in part due to the avoidance of RA in the presence of coagulopathy or thrombocytopaenia. Most units permit RA where the platelet count is >75 000 x109/dl providing no other coagulopathy or clinical bleeding exists.[38]

Complications may arise when epidural catheters have been inserted and the platelet count subsequently decreases, for example in severe pre-eclampsia. The timing of catheter removal is then important, since the trauma of removal may precipitate a spinal haematoma.[68]

3. Infective complications

Meningitis and epidural abscesses occur infrequently. Strict aseptic techniques should always be used when administering RA, including wearing face masks, as Streptococcus viridans from the oropharynx of anaesthetists has been associated with meningitis in the parturient.[69]

There is often concern about introducing spinal infection when a patient has another infection such as chorioamnionitis. If the patient is haemodynamically stable, the fever low-grade and antibiotics have been administered, then SA is considered safe.[70]

Only one case of discitis has been reported after SA for CS, and one after epidural anaesthesia for labour. Both parturients were otherwise healthy. CT guided fine-needle biopsy was used to aspirate and isolate Streptococcus bovis of uncertain source from the affected disc of the first patient. Propionibacterium acnes was isolated in the second case, with a probable skin source related to multiple attempts at epidural insertion. Both patients recovered well after six week courses of antibiotics.[71,72]

4. Chronic adhesive arachnoiditis (CAA)

Obstetric epidural anaesthesia may be implicated in this condition. The diagnostic criteria include back pain aggravated by exertion, with or without leg pain, neurological abnormalities, and characteristic MRI findings.

Additives to epidural solutions are a possible cause of CAA, hence only preservative-free solutions may be used. In addition, epidural adrenaline has associated with CAA, but the evidence for cause and effect is not clear. There is no evidence that the preservative-free, low concentration bupivacaine and opioid combinations in current use, lead to this condition.[73]

5. Hearing Loss

Low-frequency hearing loss has been reported after SA. Usually this is minor and transient, but it can be permanent and disabling, particularly when associated with vertigo and tinnitus.[74] This problem has been attributed to CSF leakage, causing a reduction in perilymph pressure in the cochlea.[75] Cutting type needles of larger gauges (22G)are associated with a higher incidence of this complication than when finer gauge Quincke needles (25G) or pencil point needles are used.[76] As in the case of PDPH, an epidural blood patch may be used successfully to treat this complication.

6. Reactivation of herpes simplex virus (HSV)

Patients with previous HSV given epidural morphine for CS, have a 9% incidence of reactivation of this virus, typically 2-5 days postoperatively.[78] This complication also occurs after IT morphine. The infection reappears in the same distribution as the primary infection, typically on the mouth as herpes labialis as it is often the trigeminal region that is originally

infected. In addition to the unpleasant sensation for the mother, there may be serious ocular involvement, or neonatal infection. Neuraxial morphine should thus be avoided in patients with a history of HSV infection.

Conclusion

RA is safe for CS, provided that the anaesthetist is aware of the complications associated with the various techniques, takes precautions to prevent complications where possible, carefully monitors the patient, and treats complications timeously and appropriately.

References

1. Gogarten W, Van Atken H. A Century of Regional Analgesia in Obstetrics. Anesth Analg 2000; 91: 773-5.

2. Hawkins JL, Koonin LM, Palmer SK, Gibbs CP. Anesthesia-related deaths during obstetric delivery in the United Sates, 1979-1990. Anesthesiology 1997; 277-84.

3. Saving Mothers 1999 – 2001. Report of the Committee for the Confidential Enquiry into Maternal Deaths in South Africa. Department of Health, Pretoria. 2002.

4. McConachie I. Vasovagal asystole during spinal anaesthesia. Anaesthesia 1991; 46: 281-2.

5. Watkins EJ, Dresner M, Calow CE. Severe vasovagal attack during regional anaesthesia for Caesarean Section. Br J Anaesth 2000; 84; 118-20.

6. Burke D, Wildsmith JAW. Severe vasovagal attack during regional anaesthesia for caesarean section (letter) Br J Anaesth 2000; 84: 824-5.

7. Sturgess JE, Browne D. Complication of the combined spinal epidural technique 1. Anaesthesia 2003; 58: 486.

8. Lok C, Kirk P. Complication of the combined spinal epidural technique 2. Anaesthesia 2003; 58: 486-7.

9. Gupta S, Singh B, Kachru N. "Blocked" epidural catheter: another cause. Anaesth Analg 2001; 92: 1617-18.

10. Singh B, Solanki SS. Blocked epidural catheter: time to look beyond the catheter. Anaesthesia 2003; 58: 400-1

11. Horlocker TT, McGregor DG, Matsushige DK et al. A retrospective review of 4767 consecutive spinal anaesthetics: central nervous system complications. Perioperative Outcomes Group. Anesth Analg 1997; 84: 578-84.

12. Wenger M, Hauswirth CB, Brodhage RP. Undiagnosed adult diastematomyelia associated with neurological symptoms following spinal anaesthesia. Anaesthesia 2001; 56; 764-7.

13. Damage to the conus medullaris following spinal anaesthesia. Reynolds F. Anaesthesia 2001; 56: 238-47.

14. Absalom AR, Martinelli G, Scott NB. Spinal cord injury caused by direct damage by local anaesthetic infiltration needle. Br J Anaesth 2001; 87: 512-5.

15. Yentis SM. Time to abandon loss of resistance to air (letter). Anaesthesia 1997; 52: 184.

16. Lucas DN, Kennedy A, Dob D. Dural puncture and iatrogenoc pneumocephalus with subsequent transverse myelitis in a parturient. Can J Anaesth 2000; 47: 1103-6.

17. Shibli KU, Russell IF. A survey of anaesthetic techniques used for caesarian section in the UK. IJOA 2000; 9: 160-7

18. Garry M, Davies S. Failure of regional blockade for caesarian section. IJOA 2002; 11: 9-12.

19. Cook TM. Combined spinal-epidural techniques. Anaesthesia 2000; 55: 42-64.

20. Rout CC, Rocke DA. Prevention of hypotension following spinal anesthesia for caesarian section. International Anesthesiology Clinics 1994; 32: 117-35.

21. Hon EH, Reid BL, Hehre FW. The electronic evaluation of fetal heart rate II. Changes with maternal hypotension. American Journal of Obstetrics and Gynaecology 1960; 79: 209-15

22. Burns SM, Cowan CM, Wilkes RG. Prevention and management of hypotension during spinal anaesthesia for elective Caesarian section: a survey of practice. Anaesthesia 2001; 56: 777-798.

23. Kinsella SM, Whitwam JG, Spencer JAD. Reducing aortocaval compression: how much tilt is enough? BMJ 1992; 305: 539-40.

24. Vallejo MC, Ramanathan S. Should a-agonists be used as first line management of spinal hypotension? International Journal of Obstetric Anesthesia 2003; 12(4): 243-245.

25. Aveling W (Proposer). P. Howell (Opposer). Controversies. Heavy bupivacaine has no advantage over plain bupivacaine in spinal anaesthesia for caesarian section. International journal of Obstetric Anaesthesia 1999; 8: 260-5

26. Rees SGO, Thurlow JA, Gardner IC, Scrutton MJL, Kinsella SM. Maternal cardiovascular consequences of positioning after spinal anaesthesia for Caesarian section: left 15∞ table tilt vs. left lateral. Anaesthesia 2002; 57: 15-20.

27. Vercauteren MP, Coppejans HC, Sermeus L. Anaphylactoid reaction to hydroxyethylstarch during Cesarean delivery in a patient with HELLP syndrome. Anesth Analg 2003; 96: 859-61.

28. Dyer RA, Farina Z, Joubert IA, du Toit P, Meyer M, Torr G, Wells K, James MF. Crystalloid preload versus rapid crystalloid administration after induction of spinal anaesthesia (colloid) for elective Caesarean section. Anaes Int Care 2004; 32(3) (in press).

29. Datta S, Alper MH, Ostheimer GW, Weiss JB. Method of ephedrine administration and nausea and hypotension during spinal anaeshesia for Caesarian section. Anesthesiology 1982; 56: 68-70.

30. Cooper DW, Carpenter M, et al. Fetal and maternal effects of phenylephrine and ephedrine during spinal anaesthesia for Caesarian delivery. Anesthesiology 2002; 97: 1582-90.

31. Ngan Kee WD, Lee A. Multivariate analysis of factors associated with umbilical arterial pH and standard base excess after Caesarian section under spinal anaesthesia. Anaesthesia 2003; 58: 125-30.

32. Bythell VE, Mowbray P, Cooper DW. Phenylephrine in obstetric regional anaesthesia. Anaesthesia 2003; 58: 288-9.

33. Kinsella SM, Tuckey JP. Perioperative bradycardia and asystole; relationship to vasovagal syncope and the Bezold-Jarisch reflex. Br J Anaesth 2001; 86(6): 859-68

34. Dildy GA, Clarke SL. Cardiac arrest during pregnancy, Obstet Gynecol Clin North Am 1995; 22: 303-14.

35. Thomas TA, GM Cooper, on behalf of the Editorial Board of the Confidential Enquiries into Maternal Deaths in the United Kingdom. Maternal deaths from anaesthesia. An extract from Why Mothers Die 1997-1999, the Confidential Enquiries into Maternal Deaths in the United Kingdom. Br J Anaesth 2002; 89: 499-508.

36. Adams TJ, Peter EA, Douglas MJ. Is spinal anaesthesia contraindicated after failed epidural anesthesia? Anesth Analg 1995; 81: 659.

37. Takiguchi T, Okano T, Egawa H, Okubo Y, Saito K, Kitajima T. The effect of epidural saline injection on analgesic level during combined spinal and epidural anesthesia assessed clinically and myelographically. Anesth Analg 1997; 85: 1097-100.

38. Paech M. Regional Analgesia and Anaesthesia. In: Anaesthesia for Obstetrics and Gynaecology. Ed. R. Russell. BMJ Books 2000:

109-177.

39. Chan YK, Gopinathan R, Rajendram R. Loss of consciousness following spinal anaesthesia for Caesarean section. Br J Anaesth 2000; 85: 474-6.

40. Davis FM, Glover PW, Maycock E. Hyperbaric oxygen for cerebral arterial air embolism occurring during Caesarean section. Anaesth Intens Care 1990; 18: 403-5.

41. Vadhera RB, Hankins GDV. Embolism during pregnancy: thrombus, air, and amniotic fluid. Anesthesiology Clin N Am 2003; 21: 165-182.

42. Glosten B,, Hynson J, Sessler DI, McGuire J. Pre-anesthetic skin-surface warming reduces redistribution hypothermia caused by epidural block. Anesth Analg 1993; 77: 488-93.

43. Davies SJ, Paech MJ, Welch H, Evans SF, Pavy TJG. Maternal experience during epidural or combined spinal-epidural anaesthesia for caesarean section: a prospective, randomized trial. Anesth Analg 1997; 85: 607-13.

44. Philip J, Alexander JM, Sharma SK, Leveno KJ, McIntire D, Wiley J. Epidural analgesia during labor and maternal fever. Anesthesiology 1999; 90: 1271-1275.

45. Shivering and shivering-like tremor during labor with and without epidural analgesia. Panzer O, Ghazanfari N et al. Anesthesiology 1999; 90: 1609-16.

46. Shehabi Y, Gatt S, Buckma T, Isert P. Effect of adrenaline, fentanyl and warming of injectate on shivering following extradural analgesia in labour. Anaesth Int Care 1990; 18: 31-7.

47. Roy J-D, Girard M, Drolet P. Intrathecal meperidine decreases shivering during Cesarean delivery under spinal anesthesia. Anesth Analg 2004; 98: 230-4.

48. Capogna G, Celleno D. IV clonidine for post-extradural shivering in parturients: a preliminary study. Br J Anaesth 1993; 71: 294-5.

49. Dahl JB, Jeppesen IS, Jorgensen H, Wetterslev J, Moiniche S. Intraoperative and postoperative analgesic efficacy and adverse effects of intrathecal opioids in patients undergoing cesarean section with spinal anaesthesia. Anesthesiology 1999;91: 1919-27.

50. Swart M, Sewell J, Thomas D. Intrathecal morphine for caesarean section: an assessment of pain relief, satisfaction and side-effects. Anaesthesia 1997; 52: 373-7.

51. Abouleish E, Rawal N, Rashad MN. The addition of 0.2mg subarachnoid morphine to hyperbaric bupivacaine for cesarean delivery: a prospective study of 856 cases. Reg Anesth 1991; 16: 137-40.

52. Sayyid SS, Jabour DG, Baraka AS. Hypothermia and excessive sweating following intrathecal morphine in a parturient undergoing cesarean delivery. Reg Anesth Pain Med 2003; 28: 140-3.

53. Siddik-Sayyid SM, Aouad MT, Jalbout MI, Zalaket MI, Berzina CE, Baraka AS. Intrathecal versus intravenous fentanyl for supplementation of subarachnoid block during cesarean delivery. Anesth Analg 2002; 95: 209-13.

54. Palmer CM, Norris MC, Giudici MC, Leighton BL, DeSimone CA. Incidence of electrocardiographic changes during cesarean delivery under regional anaesthesia. Anesth Analg 1990; 70: 36-43.

55. McLintic AJ, Pringle SD, Lilley S, Houston AB, Thorburn J. Electrocardiographic changes during cesarean section under regional anesthesia. Anesth Analg 1992; 74: 51-6.

56. Ross RM, Baker T. Cardiac enzymes in patients undergoing Caesarean section. Can J Anaesth 1995; 42: 46-50.

57. Kulka PJ, Scheu C, Tryba M, Oberheiden R, Zenz M. Coronary artery plaque disruption as cause of acute myocardial infarction during cesarean section with spinal anesthesia. J Clin Anesth 2000; 12: 335-8.

58. Grant R, Condon B, Hart I, Teasdale GM. Changes in intracranial CSF volume after lumbar puncture and their relationship to post-LP headache. J Neurol Neurosurg Psychiatry 1991; 54: 440-2.

59. Costigan SN, Sprigge JS. Dural puncture: the patients' perspective. A patient survey of cases at a DGH maternity unit 1983- 1993. Acta Anaesthesiol Scan 1996; 40: 710-14.

60. Sharma SK, Gambling DR, Joshi GP, Sidawi JE, Herrera ER. Comparison of 26-gauge Atraucan and 25-gauge Whitacre needles: insertion characteristics and complications. Can J Anaesth 1995; 42: 706-10.

61. Campbell DA, Varma TRK. Chronic subdural haematoma after lumbar subarachnoid analgesia for caesarean section, presenting as puerperal psychosis. Br J Obstet Gynaecol 1993; 100: 782-4.

62. Vaughan DJA, Stirrup, Robinson PN. Cranial subdural haematoma associated with dural puncture in labour. Br J Anaesth 2000; 84: 518-20.

63. Loeser EA. Hill GE, Bennett GM, Sederberg JH. Time vs. success rate for epidural blood patch. Anesthesiology 1978; 49: 147-8.

64. Duffy PJ, Crosby ET. The epidural blood patch. Resolving the controversies. Can J Anesth 1999; 46(9) : 878-886.

65. Abouleish E, Vega S, Blendinger I, Tio TO. Long-term follow-up of epidural blood patch. Anesth Analg 1975; 54: 459-63.

66. Charsley MM, Abram SE. The injection of intrathecal normal saline reduces the severity of postdural puncture headache. Reg Anesth Pain Med 2001; 26(4): 301-5.

67. Turnbull DK, Shepherd DB. Post-dural puncture headache: pathogenesis, prevention and treatment. Br J Anaesth 2003; 91: 718-29.

68. Yuen TST, Kua JSW, Tan IKS. Spinal haematoma following epidural anaesthesia in a patient with eclampsia. Anaesthesia 1999; 54: 350-371.

69. Pindr AJ, Dresner M. Meningococcal meningitis after combined spinal-epidural analgesia. International Journal of Obstetric Anesthesia 2003; 12: 183-7.

70. Goodman EJ, deHorta E, Tagiuam JM. Safety of spinal and epidural anesthesia in parturients with chorioamnionitis. Reg Anesth 1996; 21: 436-41.

71. Bajwa ZH, Grush HC, Kleefield J, Warfield CA. Discitis associated with pregnancy and spinal anesthesia. Anesth Analg 2002; 94: 415-6.

72. Hernandez-Palazon J, Puertas-Garcia JP, Martinez-Lage J, Tortosa JA. Lumbar spondylodiscitis caused by Propionibacterium acnes after epidural obstetric analgesia. Anesth Analg 2003; 96: 1486-8.

73. Rice I, Wee MYK, Thomson K. Obstetric epidurals and chronic adhesive arachnoiditis. Br J Anaesth 2004; 92(1): 109-20.

74. Kilickan L, Gurkan Y, Ozkarakas H. Permanent sensorineural hearing loss following spinal anaesthesia. Acta Anaesthesiol Scand 2002; 46: 1155-7.

75. Kilickan L, Gurkan Y, Aydin O, Etiler N. The effect of combined spinal-epidural (CSE) anaesthesia and size of spinal needle on postoperative hearing loss after elective caesarean section. Clin Otolaryngol 2003; 28: 267-72.

76. Finegold H, Mandell G, Vallejo M, Ramanathan S. Does spinal anesthesia cause hearing loss in the obstetric population? Anesth Analg 2002; 95: 198-203.

78. Lee CM, Peachman FA. Unilateral hearing loss after spinal anesthesia treated with epidural blood patch. Anesth Analg 1986; 65: 312.

79. Boyle RK. Herpes simplex labialis after epidural or parenteral morphine: a randomized prospective trial in an Australian obstetric population. Anesth Int Care 1995; 23: 433-7.