Backache, headache, and neurologic deficit after regional anesthesia

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Back pain

Low back pain is the most common musculoskeletal complaint during pregnancy and the puerperium. Epidural analgesia is gaining popularity in providing effective pain relief for women during labor and delivery all over the world. The existence of a causal relationship between epidural anesthesia and backache has been suggested by a few studies [1,2] and contradicted by others [3–7].

Etiology

Etiology of backache is unknown, but drugs, labor, muscle relaxation, and abnormal postures all have been implicated. Gestation places a considerable strain on the spine and pelvic joints and shifts the center of gravity of the body. Relaxin, a hormone released during pregnancy, causes the ligaments to stretch. The exaggerated lumbar lordosis of pregnancy and other factors may predispose a woman to backache after delivery. Labor and prolonged expulsive efforts also may cause or exacerbate the back pain.

Incidence

The prevalence of back pain during pregnancy was studied from the twelfth week of pregnancy until childbirth in Sweden. Forty-nine percent of women complained of back pain during their pregnancy. Back problems before pregnancy increased the risk of back pain, as did young age, multiparity, and several physical and psychological work factors [8].

The debate continues as to whether there is a causal or a casual relationship between back pain and labor epidural analgesia. Initial retrospective studies from the United Kingdom suggested an association between epidural analgesia in labor and the development of new-onset long-term backache after delivery [1,2]. The first large retrospective study included 11,701 patients. Nineteen percent of women who had labor epidural analgesia complained of backache, compared to 10.5% who did not. The authors found a statistically significant association between backache and labor epidural analgesia and concluded that the relation was

probably causal and resulted from a combination of effective analgesia and stressed posture during labor. This retrospective study suffered from patient recall and selection bias in the epidural and nonepidural groups. Patients who selected epidural analgesia also may have had obstetric, orthopedic, social, or other unidentified factors that predisposed them to postpartum back pain [1].

Another retrospective study reported an incidence of new onset backache of 18% after epidural analgesia, compared to 11.7% without epidural analgesia [2]. The nature of the backache was no different in patients who did or did not receive epidural analgesia during labor and was postural and mild in nature. Both studies were criticized for a low patient response rate and for being based on recall of events several years postpartum.

A prospective study determined the incidence of postpartum back pain and its relationship to epidural analgesia [3]. The incidence of back pain 1 to 2 months postpartum was 44% in the epidural group and 45% in the nonepidural group. The incidence of back pain in women who delivered vaginally was 45% and in cesarean section deliveries was 42%.

The principal findings of the study were as follows: (1) Postpartum back pain occurred with equal frequency in women who did or did not receive epidural analgesia. (2) Postpartum back pain was equally distributed between vaginal delivery and cesarean section patients. (3) The most significant predictor of postpartum back pain 1 to 2 months after delivery was back pain during the pregnancy. (4) The development of new-onset postpartum back pain was not associated with epidural analgesia for labor or delivery. (5) Postpartum and new-onset postpartum back pain was associated with greater body weight [3].

Recently, more prospective trials have demonstrated that new-onset postpartum back pain is not associated with regional anesthesia. Among women who received either 0.125% bupivacaine or 0.0625% bupivacaine, when compared to controls, the incidence of new long-term back pain was 7.6% with no difference between the groups [7].

Women with a previous history of back pain have an increased risk of subsequent prolonged back pain. Back pain after epidural analgesia is most frequently seen on the first postpartum day and may be caused by tissue trauma during needle placement [6].

The evidence from these prospective clinical studies indicates that ongoing postpartum back pain is not caused by epidural analgesia. Obstetric anesthesiologists, obstetricians, and midwives can reassure women confidently

that any increased risk of back pain after epidural analgesia for labor and delivery is minimal and limited to only the first postpartum day.

Chemical backache

Since 1980, reports have described neurologic deficits after presumed intrathecal injection of 2-chloroprocaine. The 2-chloroprocaine used in these reports contained sodium bisulfite as an antioxidant, which also lowered the pH. Covino was the first to suggest that a low pH and the presence of sodium bisulfite was probably responsible for the reported neurologic deficits [9].

In 1987, Astra Corporation (Westborough, MA) marketed a new formulation nesacaine-MPF (methyl paraben-free 2-chloroprocaine) that contained disodium ethylenediaminetetraacetic acid (EDTA) for epidural and caudal use. Subsequently, sporadic reports of severe back pain were reported in patients who received large volumes of epidural nesacaine-MPF [10,11]. The proposed mechanism of this back pain was attributed to disodium EDTA, which binds to tissue calcium and results in hypocalcemic tetany of paraspinous muscles [10]. The currently used nesacaine-MPF contains no preservatives and is stored in brown bottles for protection from light.

Postdural puncture headache

August Bier was the first to describe the signs and symptoms of postdural puncture headache (PDPH) [12]. The closed claim studies show that PDPH is the third most common cause for litigation in obstetric anesthesia [13]. PDPH is always bilateral and occurs in frontal and occipital regions [14]. The headache is usually postural in nature. Although the severity of headache varies from mild to severe, it is often incapacitating. Pain is exaggerated in the upright position and relieved in the supine position and can radiate to the neck and cause neck stiffness. The absence of a postural component makes the diagnosis of PDPH questionable.

The headache also can be associated with nausea (60%), vomiting (24%), ocular symptoms (13%) (photophobia, diplopia, difficulty in accommodation), and auditory symptoms (12%) (hearing loss, hyperacusis, tinnitus) [14]. Cranial nerve palsy is also present occasionally. The sixth cranial nerve is most susceptible because of its long intracranial course.

Onset and duration

Although PDPH may occur immediately after puncture of the dura mater, it usually starts 24 to 48 hours after the dural puncture. The rapidity of onset of symptoms seems to be related to the amount of cerebrospinal fluid (CSF) that is lost during and after the puncture [15]. The headache can resolve spontaneously, with approximately 50% resolving in 5 days, and more than 90% resolve in 10 days [16]. PDPH generally lasts less than a week, although there have been rare reports of headache that lasted for months or even years [17].

Pathophysiology

The cause of PDPH is not entirely certain. The best explanation is that low CSF pressure results from CSF leakage through a dural and arachnoid tear, a leakage that exceeds the rate of CSF production [18]. CSF volume is estimated to be 150 mL, and the production rate is approximately 0.35 mL/min. When the rate of CSF leakage exceeds production, there is a loss of a cushion effect by CSF of the brain. CSF hypotension can produce headache and cranial nerve symptoms through downward descent of the brain and stretching of pain sensitive structures, including dura, nerves, and bridging veins. Pain is referred from the pain-sensitive structures via the trigeminal nerve to the frontal region and the glossopharyngeal, vagus, and cervical nerves to the occiput, neck, and shoulders [16].

The pain of PDPH may be caused in part by cerebrovasodilation as a consequence of low CSF pressure [19]. The beneficial effect of cerebral vasoconstrictor drugs, such as caffeine, theophylline and sumatriptan, supports a vascular cause for PDPH [20].

Risk factors

Women, particularly during pregnancy, are considered at increased risk for PDPH. The incidence is highest between 18 and 30 years and declines in children younger than 13 years and adults older than 60 years. The incidence is greater in patients with lower body mass index (weight/height2). Younger women may be at a greater risk because of increased dural fiber elasticity that maintains a patent dural defect compared to a less elastic dura in older patients [18]. Patients with a headache before lumbar puncture and a prior history of PDPH are also at increased risk. There is no known relationship between the diagnosis of migraine headaches and increased incidence of PDPH after regional anesthesia [21].

Needle size

The incidence of PDPH is directly related to the needle diameter that punctures the dura mater [22]. Although smaller diameter needles (29–32 gauge) decrease the risk of PDPH, they are technically difficult to use and associated with a lower success rate of dural puncture [16]. The incidence of PDPH with a 26-gauge Quincke needle is 5.2%, 2.7% with a 27-gauge Quincke needle, and 1.2% with a 25-gauge Whitacre needle. The incidence of PDPH with the 25-gauge Whitacre is less than with the 27-gauge Quincke needle [22]. Morbidity associated with lumbar puncture can be decreased by the proper selection of appropriate needle gauge and tip configuration. Use of the smallest gauge needle with a noncutting tip produces the lowest incidence of PDPH in parturients [16]. The risk of PDPH recently was evaluated by neurologists using 22-gauge Quincke (cut bevel tip), 22-gauge Whitacre, and 22-gauge Sprotte (pencil point tip). The incidence of PDPH was 24.4% for the Quincke needle versus 12.2% for the pencil point needles [23]. Norris et al, in a prospective study of 2183 laboring women, did not find an increase in risk of PDPH after using the combined spinal-epidural technique with a 27-gauge Whitacre needle compared to epidural analgesia (without dural tear) [24].

Direction of bevel

Spinal needles are designated as cut bevel, as in the Quincke type, or pencil point, as in the Whitacre type spinal needle. Dural fibers were once believed to run longitudinally [25]; however, microscopic dissection of the dura mater from fresh cadavers and the use of scanning electron microscopy revealed that the dural fibers do not run longitudinally or in parallel fashion. The dura is a laminated structure built up from well-defined layers oriented concentrically around the medulla spinalis [26]. Orienting the bevel of a cutting needle probably needs further consideration.

Electron microscopy has shown that pencil point needles are more traumatic to the dura than the cut bevel needles. It is postulated that a pencil point needle produces an irregular tear in the dura, and the subsequent inflammatory reaction reduces CSF leakage more effectively than the clean U-shaped puncture seen with cut bevel needle, which decreases the risk of PDPH [27].

Conservative treatment

Bed rest and hydration

Patients with PDPH tend to rest in bed to avoid worsening of headache in the sitting or standing position. Bed rest may postpone the occurrence of the headache but does not prevent it. Patients need encouragement to ambulate early after delivery. The headache appears during the hospital stay and treatment can be started early.

Oral hydration remains a popular therapy for PDPH, but there is no evidence that vigorous hydration has any therapeutic benefit. No patient with PDPH should be allowed to become dehydrated, however.

Pharmacotherapy

Oral and intravenous medications

If the headache is mild, intravenous fluids, caffeine, and theophylline can be attempted as a first-line treatment. Methylxanthines may block cerebral adenosine receptors that lead to cerebral vasoconstriction. The efficacy of oral caffeine for the treatment of PDPH was evaluated in 40 postpartum patients [28]. A single oral dose of 300 mg was demonstrated to be safe and effective and should be considered in the early treatment of mild PDPH. It is convenient, less expensive than intravenous caffeine, and provides temporary relief from PDPH. Caffeine sodium benzoate, 500 mg, administered as an intravenous bolus or as an infusion of caffeine sodium benzoate 500 mg in 1 L of balanced salt solution over 1 hour also can be used to treat PDPH. Caffeine is a potent central nervous system stimulant. The use of caffeine must be avoided in patients with diagnosis of pregnancy-induced hypertension or seizure disorder. Bolton et al described a seizure shortly after the administration of caffeine therapy and epidural blood patch (EBP) in a patient diagnosed with mild pregnancy-induced hypertension [29].

Dodd et al evaluated cerebral blood flow changes using ^{133}Xe washout technique and intravenous caffeine sodium benzoate [30]. Caffeine was 75% to 80% effective in the initial treatment of PDPH; however, follow-up 48 hours later revealed that all patients had a return of their headache [30]. Caffeine relieves PDPH by vasoconstriction of dilated cerebral blood vessels, but the effect is transient. The CSF pressure still remains low because of the continued leakage of CSF, and the headache returns once the effect of caffeine wears off.

Theophylline

Theophylline is also a cerebral vasoconstrictor and is available in oral and parenteral forms. Theophylline is more effective than placebo for treatment of PDPH but has not become popular [31].

Sumatriptan

Sumatriptan is a serotonin type 1-d receptor agonist and is useful in the effective treatment of migraine and cluster headaches. Sumatriptan administered subcutaneously is effective in the treatment of PDPH, with complete resolution of symptoms [20]. The dose is 6 mg subcutaneously and is available in oral form. The drug is expensive, however. Side effects include pain at the site of injection and chest tightness. Caution must be used in patients with ischemic heart disease [20]. Controlled trials are needed to evaluate further the use of sumatriptan for PDPH.

Adrenocorticotropic hormone

Cosyntropin, a synthetic form of adrenocorticotropic hormone, is less antigenic than native adrenocorticotropic hormone and is the preferred agent for clinical use [32]. There have been reports of success in treating refractory PDPH with adrenocorticotropic hormone. The dose, 0.25 to 0.75 mg, is administered as an intravenous infusion over a 4- to 8-hour period. The intramuscular route also has been used. Adrenocorticotropic hormone is believed to work by stimulating the adrenal gland to increase CSF production and betaendorphin output [33].Caution should be used in patients with diabetes [34].

Definitive treatment

Epidural saline infusion

Epidural saline is a less effective therapy for PDPH than EBP, and the benefit is usually transient. Successful use of prolonged (24-hour) epidural saline infusion has been reported for the treatment of PDPH in patients with failed EBP [35,36].

Epidural fibrin glue injection

Fibrin glue is a preparation of pooled human plasma obtained from plasmapheresis. The fibrin clot forms a temporary biologic seal of the dura until healing occurs. The clot does not retract because of the lack of corpuscular blood

components. Fibrin glue has been used successfully in the treatment of persistent CSF leak in three preterminal cancer patients [37]. It also has been used in the treatment of persistent PDPH after two failed EBPs in a woman who had received spinal anesthesia [38].

Epidural dextran patch

Epidural administration of 20 to 30 mL of dextran-40 is as effective and safe as EBP for the relief of PDPH. It relieves the PDPH by means of mechanical tamponade at the dural puncture site. The viscosity and high molecular weight of dextran-40 delays its absorption from the epidural space and leads to long-lasting local compression [39]. It has been used successfully in the treatment of PDPH in a Jehovah's Witness (unreported case) at the authors' institution. Additional information is needed before this technique can be adopted widely. Fear of anaphylaxis might limit its use.

Prophylactic epidural blood patch

Performing a prophylactic EBP after a dural tap has been controversial. It has been supported by some [40] and questioned by others [41]. Its effectiveness depends on the proximity of the catheter tip to the dural rent and on the certainty that the catheter is sited within the epidural space. The risk of infection could increase because blood is injected through a nonsterile epidural catheter compared to an epidural needle placed de novo. Proponents of prophylactic blood patch recommend it to avoid subdural hematoma [42,43], cranial nerve palsies [44,45], and malpractice law suits [13].

Epidural blood patch

An EBP is the most effective treatment for PDPH and is considered the ”gold standard” treatment. It is indicated when conservative measures have failed, the headache is severe, or when it affects the length of stay in the hospital. The success rate after a first EBP is 85%; this rate reaches 98% after the second EBP.

Gormley introduced autologous blood patch into clinical practice in 1960. He reported relief of headache after administration of 2 to 3 mL of blood in the epidural space [46]. Subsequently, Giovanni and Dunbar reported an immediate cure of

headache in 41 of 45 patients who received 10 mL blood in the epidural space [47]. Their success later led to the widespread use of this technique to relieve PDPH.

The optimal volume of injected blood remains controversial. Two to 3 mL of blood injected into the epidural space was reported to relieve PDPH in 100% of patients [46]. Crawford used 20 mL of blood for EBP and reported a 96% success rate [48]. Although there is no general consensus about the optimal blood volume, the common practice is to inject 20 mL blood or stop when the patient complains about discomfort or pain in the back, legs, or buttocks.

Based on an MRI study, EBP injection produced a focal hematoma mass around the injection site. The mass initially compressed the thecal sac and nerve roots. The main bulk of the epidural blood clot extended only three to five spinal segments from the injection site. Spread from the injection site was principally cephalad, although a small amount of blood spread distally [49,50]. Mass effect was present at 30 minutes and 3 hours, but clot resolution occurred by 7 hours [49].

Technique of epidural blood patch

The anesthesiologist must provide a thorough explanation of the risks and benefits of a blood patch. The patient should give written informed consent. The lateral position is more comfortable for the patient with a severe headache, but if there is difficulty in identifying the space, the sitting position can be used. The EBP should be performed at the level caudad to the level of dural puncture because blood tends to spread in the cephalad direction in the epidural space [49,50]. The epidural space is identified in the usual manner. Using a sterile technique, an assistant withdraws 15 to 20 mL of autologous blood from an antecubital vein, which is then injected slowly through the epidural needle. The injection is terminated once the patient complains of back, neck, or buttock pain. Usually, the headache is relieved immediately after the blood patch. Current belief is that the injected blood raises pressure in the epidural and subarachnoid spaces, forces CSF back inside the cranium, and restores the cushioning effect of CSF on the brain, which provides rapid relief of headache [51].

After the procedure, the patient assumes the supine position. The legs are elevated by placing pillows or blankets underneath the knees. This position eliminates the lumbar lordosis and helps in even spreading of the blood. The patient should rest quietly in this position for 1 to 2 hours. Later, the patient can resume ambulation but should avoid vigorous physical activity for several days.

Severe complications from EBP are rare but sometimes can be alarming. Some of the reported complications include transient back pain at the site of the blood patch, radicular pain, transient bradycardia [52], lumbovertebral syndrome [53,54], and facial palsy [55]. There has been a case report of exacerbation of PDPH after blood patch that then responded to 100 mg per rectal diclofenac [56]. Radicular pain and backache can be explained by the mass effect of injected blood on the nerve roots.

Blood patch in HIV-positive patients

Fear of infectious complications has led to debate regarding the safety of EBP on HIV seropositive patients. Studies suggest that the HIV crosses the blood-brain barrier and infects the central nervous system early in the clinical course, often before any symptoms appear. EBP is unlikely to introduce HIV into the central nervous system. The likelihood of central nervous system inoculation by a small amount of viremic blood, via EBP, remains uncertain [57].

Neurologic complications

No anesthetic procedure is entirely without risk of death or injury, and so the choice of general or regional anesthesia must be judged on the inherent risk-benefit ratio in each individual patient [58]. The incidence of catastrophic neurologic injury in the United States after regional anesthesia is low and believed to be approximately 1 in 10,000. The incidences of neurologic deficits from various studies are included in Table 1.

Table 1. Incidence of neurologic deficits

Author	No. of anesthetics	Incidence
Vandam [77]	10,098 spinals	19 neuropathies
Scott [78]	505,000 epidurals	38 single nerve neuropathy
		1 paraplegia
Scott [79]	108,133 epidurals	46 neuropathies
	14,856 spinals	
Dahlgren and Tornebrandt [80]	8501 spinals	7 neurologic deficits
	9232 epidurals	13 neurologic deficits
Horlocker et al [61]	4767 spinals	6 persistent paresthesias
Auroy et al [59]	30,413 epidurals	6 (5 radiculopathy, 1 paraplegia)
	40,640 spinals	24 (19 radiculopathy, 5 CES)
Aromaa et al [81]	170,000 epidurals	7 (1 paraparesis, 1 CES, 5 nerve deficits)
	550,000 spinals	31 (5 paraparesis, 1 CES, 25 nerve deficits)
Paech [82]	10,995 epidurals	1 traumatic mononeuropathy L3 sensory loss

Abbreviation: CES, cauda equina syndrome.

Etiology of neurologic complications

Needle trauma and local anesthetic neurotoxicity are the most common causes of neurologic complications related to neuraxial anesthesia. A prospective survey in France recently evaluated the incidence and characteristics of serious complications related to regional anesthesia. All patients with a neurologic deficit that lasted more than 2 days were examined by a neurologist. Patients with cauda equina syndrome were further evaluated with a CT scan to rule out any compressive etiology. A total of 71,053 regional anesthetic agents, including 40,640 spinal and 30,413 epidural anesthetic agents, were performed over a 5-month period. Neurologic complications occurred in 34 patients; recovery was complete within 3 months in 19 of 34 patients. Serious complications occurred even in the presence of experienced anesthesiologists. Continued vigilance in patients who undergo neuraxial anesthesia is warranted at all times [59].

Cheney et al examined the American Society of Anesthesiologists Closed Claims database to determine the role of nerve damage in malpractice claims filed against anesthesia care providers. A total of 4183 claims were reviewed, out of which 670 (16%) claims involved anesthesia-related nerve injuries. Lumbosacral nerve root injuries accounted for 105 claims and spinal cord injuries accounted for 84 claims. Paresthesias during needle insertion or injection of drug and multiple attempts to perform a block were the major factors associated with lumbosacral nerve root injuries [60].

Nerve injury from needle

Direct needle-induced trauma and intraneuronal injection of local anesthesia are avoidable causes of nerve injury, but fortunately severe or disabling neurologic complications are rare. A recent retrospective study of 4767 spinal anesthetic agents noted the presence of a paresthesia during needle placement in 298 (6.3%) patients [61]. Four of the six patients with a persistent paresthesia postoperatively complained of a paresthesia during needle placement. Elicitation of a paresthesia is considered to be a risk factor for a neurologic deficit. In a prospective study, two thirds of patients with neurologic complications experienced pain during needle placement or injection of local anesthesia [59]. In all cases, the neurologic deficit had the same distribution as the elicited paresthesia. The needle should be replaced in the event a paresthesia is elicited to avoid the risk of nerve injury.

Although the cord commonly ends opposite the lower border of L1 or L1-2 interspace, it may extend as low as L3. It is safer to avoid upper lumbar interspaces at all times. Reynolds et al recently described seven cases in which neurologic damage followed spinal or combined spinal-epidural technique using an atraumatic spinal needle. All patients experienced pain during needle insertion. The sensory and motor deficit of lower motor neuron distribution relating to one leg was followed by pain on spinal needle insertion on the same side. MRI showed a spinal cord of normal length in all of the cases. In no case did the spinal cord appear to be unduly long [62].

Recently, a case report of spinal cord injury caused by a 4-cm long 21-gauge needle used for local anesthetic infiltration was published [63]. During subcutaneous local anesthetic skin infiltration, the patient rolled toward the anesthesiologist onto her back, which caused the needle to be advanced to its hub. The patient felt a shock-like sensation that ran from the site of insertion of the needle down both her legs but was worse on the left. After the needle was withdrawn, she had transient sharp pain in the lower half of her body and numbness in her left leg down to the foot. The MRI showed an area of spinal cord edema at the level of penetration by the infiltrating needle, consistent with a needle-stick injury [63]. In most patients, the distance from the skin to the epidural space is between 4 and 6 cm. In thin patients, however, it can be less than 3 cm. This case suggests that when a thin patient is encountered, caution must be exercised even with the infiltrating needle.

Transient neurologic syndrome

Transient neurologic syndrome (TNS) was first described in 1993 after single injection of intrathecal hyperbaric 5% lidocaine [64]. This phenomenon is associated with pain or sensory abnormalities in the lower back, buttock or lower extremities. The symptoms of burning pain and dysesthesia in L5 and S1 dermatomes usually start within 1 day of surgery and often resolve spontaneously within 1 month. Schneider reported four cases of severe radicular back pain that occurred after resolution of hyperbaric lidocaine spinal anesthesia [64]. All four patients had undergone surgery in the lithotomy position. No sensory or motor deficits were detected on examination, and the symptoms resolved spontaneously within several days.

The incidence of TNS ranges from 0% to 37% [65] and depends on local anesthetic used, surgical procedure, and patient factors. A prospective, randomized, double-blinded study reported a 16% incidence of TNS in patients who received either hyperbaric 5% lidocaine with epinephrine or 2% isobaric

lidocaine without epinephrine [66]. None of the patients who received 0.75% hyperbaric bupivacaine developed TNS. The incidence was higher in patients with knees or hips flexed (arthroscopy) during their procedure, when compared to patients positioned supine (herniorraphy). Flexion of hips is believed to cause additional stretch of the nerve roots, which produces TNS. A subsequent study was conducted to compare the incidence of TNS in knee arthroscopy patients with hyperbaric spinal lidocaine using different concentrations. The incidence of TNS did not differ among the groups with dilution of lidocaine [67].

A large multicenter epidemiologic study that involved 1863 patients was conducted to identify potential risk factors for TNS [68]. The incidence of TNS with lidocaine (11.9%) was significantly higher than that with tetracaine (1.6%) or bupivacaine (1.3%). The pain associated with TNS is often severe and more than 90% of cases resolve completely within 1 week. The factors associated with increased risk of TNS with lidocaine include lithotomy position, outpatient status, and obesity. The following factors do not increase the risk of TNS: gender, age, history of back pain or neurologic disorder, lidocaine dose or concentration, spinal needle size, aperture, direction, or addition of epinephrine.

A recent study was conducted in pregnant women comparing hyperbaric lidocaine to hyperbaric bupivacaine for cesarean section under spinal anesthesia [69]. The incidence of TNS was 0 in both the groups. During pregnancy, the engorgement of epidural veins and exaggerated lumbar lordosis favor a more cephalad spread of the local anesthesia. This is probably the reason for less pooling of local anesthesia in the L5-S1 roots involved in the pain distribution of TNS.

Transient neurologic symptoms are most often associated with spinal anesthesia, but some recently reported cases of TNS include symptoms associated with bupivacaine use in epidural analgesia [70].

Neurophysiologic evaluation in volunteers during TNS did not reveal any abnormalities in somatosensory evoked potential, electromyography, or nerve conduction studies [67]. No treatment is required if the pain is mild. If the pain is severe, the recommended therapy for TNS is nonsteroidal antiinflammatory drugs or oral opioid analgesic agents.

Cauda equina syndrome

The potential causes for cauda equina syndrome include direct or indirect trauma, ischemia, infection, and neurotoxic reaction of locally injected drugs.

Repeated injections of local anesthetic via an indwelling intrathecal catheter or by a repeat spinal injection to improve a patchy or failed block have been associated with this syndrome. A patient with this syndrome presents with varying degrees of bowel and bladder dysfunction, perineal sensory loss, and lower extremity motor weakness. The signs and symptoms are localized to the areas innervated by the lumbar and sacral nerves.

Maldistribution and sacral pooling, with exposure of the vulnerable cauda equina nerve roots to toxic concentrations of hyperbaric local anesthetic, are hypothesized to cause cauda equina syndrome [71]. Using a spinal model, it has been demonstrated that the repeated injection of hyperbaric lidocaine intrathecally through a spinal catheter resulted in accumulation of the anesthesia near the cauda equina [72,73]. Local anesthetic agents implicated in its cause are lidocaine, 2-chloroprocaine, procaine, bupivacaine, and tetracaine [74,75]. Reports of cauda equina syndrome led the Food and Drug Administration to recall spinal microcatheters smaller than 24 gauge from the US market in 1992 [76].

Summary

Back pain, chemical backache, PDPH, and neurologic deficit all may be reported after regional anesthesia for childbirth. Back pain is common during pregnancy, but epidural analgesia during labor does not increase the incidence of long-term back pain. Chemical backache caused by 2-chloroprocaine is probably a result of hypocalcemic tetany of paraspinous muscles. The mechanism is presumed to be chelation of calcium by sodium bisulfite, an antioxidant present in nesacaine-MPF. PDPH after dural puncture is caused by leakage of CSF, which causes cerebral hypotension. Cerebral hypotension leads to traction on pain-sensitive intracranial structures and cerebral vasodilation. Initial therapy includes hydration, caffeine, and sumatriptan. EBP is the most effective treatment in severe PDPH. If the first EBP fails, a second blood patch can be performed. Neurologic deficits after regional anesthesia are rare. Meticulous technique and vigilance are the keystones in avoiding major neurologic complications of regional anesthesia. Rapid diagnosis and appropriate treatment are essential to optimize a successful outcome if complications do develop.

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