

Case Report

Severe adhesive arachnoiditis resulting in progressive paraplegia following obstetric spinal anaesthesia: a case report and review

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Summary

A 27-year-old woman developed severe adhesive arachnoiditis after an obstetric spinal anaesthetic with bupivacaine and fentanyl, complicated by back pain and headache. No other precipitating cause could be identified. She presented one week postpartum with communicating hydrocephalus and syringomyelia and underwent ventriculoperitoneal shunting and foramen magnum decompression. Two months later, she developed rapid, progressive paraplegia and sphincter dysfunction. Attempted treatments included exploratory laminectomy, external drainage of the syrinx and intravenous steroids, but these were unsuccessful and the patient remains significantly disabled 21 months later. We discuss the pathophysiology of adhesive arachnoiditis following central neuraxial anaesthesia and possible causative factors, including contamination of the injectate, intrathecal blood and local anaesthetic neurotoxicity, with reference to other published cases. In the absence of more conclusive data, practitioners of central neuraxial anaesthesia can only continue to ensure meticulous, aseptic, atraumatic technique and avoid all potential sources of contamination. It seems appropriate to discuss with patients the possibility of delayed, permanent neurological deficit while taking informed consent.

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Case report

A 27-year-old woman with no significant medical history and in her first pregnancy underwent spinal anaesthesia for caesarean section for fetal compromise at another hospital at 42 weeks' gestation, after going into spontaneous labour. With the patient sitting, the skin over the L4-5 interspace was cleaned once by a consultant anaesthetist using a SOLU-I.V.[®] MAXI swabstick (Solumed, Laval, Canada) impregnated with 2% chlorhexidine gluconate and 70% isopropyl alcohol. There was no delay between opening the swabstick packaging and its use on the patient's skin. It is not clear whether the swabstick and its packaging were disposed of immedi-

ately. The prepared area was allowed to air-dry for 3 min before a 24-G needle (Becton, Dickinson & Company, Franklin Lakes, NJ, USA) was placed at L4-5 on the first attempt by a consultant anaesthetist wearing a sterile surgical gown and gloves, mask and hat, using an aseptic technique. After aspiration of free-flowing, clear cerebrospinal fluid (CSF), 2.5 ml bupivacaine 0.5% with 12.5 µg fentanyl was administered over approximately 15 s. A few seconds after the end of the injection, the patient complained of severe, burning pain in the lower back radiating into the legs bilaterally, worse on the right than the left. The pain began to recede as the block took effect and this was formally assessed 10 min after the

spinal injection, when it was recorded as 'perfect to umbilicus' bilaterally (to touch and cold). The degree of motor block was not recorded on the anaesthetic chart. Surgery was uncomplicated and no intra-operative analgesics or other drugs were required. Intra-operative blood pressure was constant at 91/51–106/62 mmHg. Thirty minutes into surgery, after delivery of a healthy boy, but before the end of surgery, the patient developed a sudden-onset, constant, occipital headache with bilateral parietal radiation. This persisted after transfer to recovery, where the patient was serially assessed and found to be haemodynamically stable (blood pressure 118/70–127/77 mmHg) and afebrile, with neurological findings consistent with a normally resolving block. Her Glasgow Coma Scale was 15 and her pupillary responses were normal. The block resolved over 2 h and the back pain returned; this and the ongoing headache were initially refractory to two intravenous boluses of fentanyl 50 µg 15 min apart, followed by intravenous paracetamol 1 g 20 min later, but receded gradually and completely over the ensuing 4 h.

The rest of the mother's postoperative course was unremarkable apart from a single episode of asymptomatic hypotension (90/62 mmHg) on the third day, which responded to a single 250-ml colloid bolus. She and her baby were discharged on the fifth postoperative day, but the patient returned later that day with sudden-onset of vomiting and intermittent, severe headache. She was assessed in the emergency department and found to have a blood pressure of 145/95 mmHg and pulse rate of 80 beats.min⁻¹. There was no proteinuria, oedema or abnormal blood results, but given her blood pressure reading and headache, she was treated for pre-eclampsia with cilazipril and labetalol (details unavailable). Neurological examination at this stage remained normal. She was discharged the following day on labetalol with regular obstetric and general practitioner follow-up.

She returned 11 days postpartum with constant, generalised, intermittently severe and occasionally pulsatile headache and low back pain, but no objective neurological signs. A computed tomography scan showed marked communicating hydrocephalus. She was transferred to our centre where a thorough neurological examination demonstrated no abnormality and she underwent ventriculoperitoneal shunting with a Strata adjustable valve, resulting in immediate symptomatic

relief on emerging from general anaesthesia. Analysis of CSF showed a mild lymphocytosis with scanty fibrous debris, but viral serology was unremarkable and no tumour cells were seen. Magnetic resonance imaging (MRI) of the head and spine showed an abnormal cervicothoracic cord contour and what was felt to be probable clumping of the cauda equina anteriorly, although underlying spina bifida occulta could not be excluded. No disc or vertebral pathology was noted and the canal was capacious. There was no evidence of multiple sclerosis.

Initially, given the patient's lack of symptoms, an expectant approach was adopted. Headaches, nausea and nystagmus, gradually worsening over the ensuing month and refractory to adjustment of the shunt valve, mandated repeat MRI (Fig. 1). This showed recurrent hydrocephalus, 6.3 mm of cerebellar tonsillar descent and extensive cervicothoracic syringomyelia. An urgent foramen magnum decompression and C1 laminectomy with duraplasty were performed; once again, good resolution of symptoms was achieved and the patient was discharged 6 days later with a plan for serial MRI



Figure 1 Sagittal T2 magnetic resonance images of (a) cervicothoracic and (b) thoracolumbar spine 6 weeks after spinal anaesthesia, showing clumped nerve roots and disrupted cord contour with anterior tethering at multiple levels.

and neurosurgical outpatient review at 6 weeks as well as follow-up by her general practitioner and local neurology service.

The patient returned 2 weeks later via her local emergency department, now 3 months postpartum, with dramatic, progressive loss of bowel and bladder function and leg weakness. She was unable to walk and required urinary catheterisation. Serial clinical examination over 24 h revealed a rapidly ascending sensory level that stabilised at the T10 dermatome, 3/5 power, hypotonia and hyporeflexia in the lower limbs and reduced anal tone. An MRI showed persistent cervicothoracic syringomyelia, clumped lumbar nerve roots and fixation of the cord anteriorly at multiple levels. She underwent an urgent exploratory L5-S1 laminectomy with a view to treating any congenital tethering of the spinal cord, as dysraphism had not yet been fully excluded as a contributory factor. On opening of the non-pulsatile theca, grossly abnormal, matted clumps of thickened nerve roots, tightly adherent to the dura and one another, were encountered. No CSF was present at this level and there was no evidence of spina bifida. The dura was closed and a course of high-dose dexamethasone (4 mg intravenously, 6 hourly) was commenced. This was discontinued after 5 days of no clinical benefit and the development of a florid thoracic shingles rash, treated with acyclovir. In the face of ongoing clinical deterioration, a second laminectomy, at T9, was performed 48 h after the first, to permit the placement of an external drain in the syrinx, with the rationale that draining this cavity may effect neurological improvement. Once again, at operation, an overwhelming, adhesive inflammatory process involving the meninges, nerve roots and spinal cord was observed, with no CSF flow. The catheter was passed into the grossly oedematous cord, with no true syrinx cavity encountered, although this did yield a small amount of CSF for analysis. Biopsies of the cord and arachnoid were also taken. The CSF was clear and colourless, and contained $2 \times 10^6 \cdot \text{l}^{-1}$ white cells and a mildly elevated protein level of $0.67 \text{ g} \cdot \text{dl}^{-1}$. Cord biopsy showed degenerate white matter with demyelination around blood vessels associated with perivascular accumulations of CD3+ lymphocytes and CD68+ monocyte/macrophages. Over the next 4 weeks, the patient deteriorated further, losing all power in the lower limbs and developing 4/5 weakness

in the C8-T1 myotomes of the left hand. She was discharged to a spinal rehabilitation unit where she received a course of methylprednisone, with minimal clinical improvement. At 21 months' follow-up, she has no power in the lower limbs and reduced sensation up to T6 on the left and T10 on the right. The left hand remains weak. She uses a wheelchair to mobilise and has a suprapubic catheter. She is unable to work and dependent on others for most daily activities.

Discussion

Chronic adhesive arachnoiditis

Adhesive arachnoiditis is a debilitating disorder in which the pia-arachnoid undergoes an inflammatory reaction to an injurious stimulus, ultimately resulting in intrathecal scarring that can impede subarachnoid CSF pathways, disrupt blood supply and tether neural elements leading to atrophy. Authors agree that it is a rare entity, although its precise incidence remains uncertain [1–3]. Clinically, it has been defined as back pain that increases with activity in the context of uni- or bilateral leg pain, an abnormal neurological examination and MRI changes consistent with the disease [1]. The MRI findings (see Table 1) are highly characteristic and demonstrate good sensitivity (92%) and excellent specificity (100%) [4]. Presentations are highly variable in terms of severity and timing – there is a delay between the putative initial insult and the development of neurological signs and symptoms ranging from a week to more than a decade [5–8].

Pathophysiology

Pathologically, it has been proposed that an initial, noxious insult to the pia-arachnoid precipitates a response to injury with adhesion formation similar to that seen in other serous membranes [7]. Initially, this takes the form of pia-arachnoid inflammation and nerve

Table 1 Magnetic resonance imaging criteria for diagnosis of adhesive arachnoiditis [4].

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|----|----------------------------------------------------------------------------------|
| 1. | Conglomerations of adherent nerve roots residing centrally within the thecal sac |
| 2. | Nerve roots adherent peripherally giving the impression of an 'empty sac' |
| 3. | Soft tissue mass replacing the subarachnoid space |

root swelling with hyperaemia, causing radicular symptoms [9]. This is followed by arachnoiditis, characterised by the development of a relatively oligocellular, fibrinous exudate. The lack of arachnoid vasculature results in few enzyme-bearing leucocytes while the presence of circulating CSF serves to dilute and carry off phagocytes and fibrinolytic enzymes – a situation that permits the unrestricted build-up of fibrinous bands upon which collagen is deposited. This process leads to the adhesive phase: adhesions between the contents of the theca, reduced blood supply and encapsulation and tethering of the nerve roots and cord, ultimately resulting in recurrent micro-trauma and atrophy. Disruption of CSF flow resulting from arachnoiditis significantly reduces nutrient provision to the neural elements [10]. Whether genetic tendencies to keloid scarring or aberrant fibrinolytic pathways can predispose individuals to the development of adhesive arachnoiditis is uncertain, nor is much known about the role of the immune system in sustaining the reaction.

When adhesive arachnoiditis occurs adjacent to the spinal cord, syringomyelia may result [11, 12]. In rabbit and rat models, chemically induced spinal arachnoiditis was observed to result in syrinx formation in 37.5% of patients [13]. In these animals, ischaemia, cavitation and demyelination were observed in spinal cord adjacent to areas of arachnoiditis, correlating with the clinical observation that syrinx formation starts at the level of CSF flow blockade [14]. Theoretical models have proposed that disrupted CSF flow at the level of the adhesive arachnoiditis produces a pressure gradient between the spinal subarachnoid space and the CSF in the intramedullary canal, causing interstitial seepage of CSF into the cord with every cardiac cycle, generating cord oedema and cavitation [11, 14].

Precipitating agents

In the modern era, adhesive arachnoiditis is most commonly encountered as a complication of multiple revisions of lumbar spine surgery or disc herniation [15, 16], or in patients who underwent contrast myelography in the decades when this was prevalent [1, 8, 17]. Historically, central nervous system infections were the most significant cause and remain so in some parts of the developing world, particularly as a complication of tuberculosis [8]. The evidence for these factors in

precipitating arachnoiditis is strong and generally accepted.

Central neuraxial anaesthesia and adhesive arachnoiditis
Spinal and epidural anaesthesia are safe, well established, widely utilised anaesthetic techniques that facilitate many hundreds of thousands of obstetric, orthopaedic, urological, general and other surgeries per year [18–20]. Neurological complications are uncommon and almost invariably temporary [17, 18, 21]. These can result from direct trauma to neural elements or, more rarely, through introduction of infection, hypoxic cord injury, expanding epidural haematomas or injection of anaesthetic into or adjacent to a canal already critically stenosed by osteophytes, disc protusion or less common abnormalities, such as arteriovenous fistulae [22]. Adhesive arachnoiditis is a rare, but recognised cause of neurological deficit following central neuraxial anaesthesia [5, 12, 17, 22–29].

Case series describing surgically proven adhesive arachnoiditis following spinal or epidural blocks first emerged in the 1950s. Kane summarises a syndrome of ‘gradual progressive weakness and sensory loss ... beginning several weeks to several months after spinal anaesthesia [that] may lead to complete paraplegia’ and details the characteristic findings at laminectomy [5].

More recently, published reviews of arachnoiditis and epidural and spinal anaesthesia have concluded that the available evidence proposing central neuraxial anaesthesia as a cause of adhesive arachnoiditis is weak [1, 5, 30], although the authors of one admit that to exclude a link statistically would require an impractically large prospective survey [1]. Two of the larger population studies to date also argue against an association [20, 28]. These countrywide, 5-year retrospective population surveys of obstetric anaesthesia performed in Finland [20] and the UK [28] reported no definite cases from a total of almost 750 000 central neuraxial anaesthetics, of which a majority were epidurals. In 2009, a UK-wide audit of over 700 000 central neuraxial anaesthetics did identify a single case of severe adhesive arachnoiditis resulting in paraplegia following a spinal anaesthetic, but could not attribute causality [21]. However, as our case demonstrates, a significant interval of weeks to months can elapse between the putative insult and development of symptomatic arachnoiditis. Anaesthetists are not

expected to follow-up apparently uncomplicated cases and so rely on primary or secondary care providers to report back delayed neurological deficits. A prospective multidisciplinary attempt to assess the rate of neurological deficit following central neuraxial anaesthesia in obstetrics was not designed to detect cases of adhesive arachnoiditis [19].

Cases of delayed neurological deficit resulting from adhesive arachnoiditis following apparently routine neuraxial block continue to be sporadically reported in the literature. Those published since 1990 are listed in Table 2. Obstetric procedures predominate and development of symptoms severe enough to warrant neurosurgical referral is usually in the order of days to weeks following the anaesthetic. The temporal relationship between the neuraxial anaesthetic and the development of adhesive arachnoiditis is compelling in all cases, but the proposed mechanisms of initial injury remain speculative and controversial [1, 5, 17]. They include an inflammatory response to blood in the subarachnoid space, the action of the local anaesthetics themselves and accidental contamination of the injectate.

Blood. Rarely, adhesive arachnoiditis occurs in the context of subarachnoid haemorrhage [31]. It has been theorised that oxidative blood breakdown products can cause damage to neural elements and precipitate adhesive arachnoiditis [32]. Conclusive laboratory evidence for an inflammatory response to haemoglobin products is lacking and the use of a pencil-point needle and aspiration of free-flowing, clear CSF before administration of the local anaesthetic in our case argues against haemorrhage being a factor, although significant occult bleeding cannot be excluded.

Local anaesthetics. Local anaesthetics themselves have been proposed as a cause of adhesive arachnoiditis [1, 17], although this suggestion is controversial. Laboratory studies have demonstrated dose-dependent damage to neural elements exposed to supra-clinical concentrations of local anaesthetic [33]. There appears to be no good evidence, however, that such insults can precipitate arachnoiditis, with animal studies using injections of various local anaesthetic agents showing no sign of a meningeal reaction [34, 35].

Chemical contamination. Contamination of intrathecal or epidural local anaesthetic solution with noxious chemicals has been proposed as an aetiological factor in many cases of adhesive arachnoiditis, mostly dating from before the era of disposable equipment [5]. Many reported cases developed adhesive arachnoiditis over days to weeks and some died of hydrocephalus secondary to CSF block [36]. Phenolic, acidic and detergent contaminants have all been implicated [1, 5, 36], with the best known cases from that era resulting in the 1953 Wooley and Roe trial in the UK [37], the finding of which – that phenol seeped through microscopic cracks in local anaesthetic ampoules – has been roundly criticised as implausible, it being more likely that acidic descaler was present in the syringes and other equipment due to an oversight [38].

In 2008, alcoholic chlorhexidine, a ubiquitous and effective bactericidal cleansing agent, was ruled the cause of a severe case of adhesive arachnoiditis in *Sutcliffe vs Aintree Hospitals* – a trial taken to the UK Court of Appeal [39, 40]. The detailed judgement contains a clinical account of the case and it is remarkably similar to that reported herein. The patient underwent spinal anaesthesia with bupivacaine for elective caesarean section. She experienced peri-operative back pain and headache, and went on to develop catastrophic adhesive arachnoiditis with hydrocephalus, cord cavitation and permanent paraplegia. Chlorhexidine was poured into a receptacle on the sterile field and the verdict hinged on conjecture that at least 0.1 ml splashed from the tray and came into contact with the bupivacaine, that was then injected. Controversially, the hospital was ruled negligent and compensation awarded. Few clinical details are available regarding a further case in 2011 in Australia, in which 8 ml clear (i.e. untinted) chlorhexidine was accidentally substituted for local anaesthetic and injected epidurally, again resulting in paralysis and neurosurgical intervention [41].

Experimentally, various cleansing agents have been shown to cause arachnoiditis in dogs and monkeys [42, 43]. Adhesive arachnoiditis developed in all five monkeys given a spinal injection of tetracaine deliberately contaminated with unspecified detergents, one of which became paraplegic [42]. Such studies are naturally precluded in humans and the specific effect

Table 2 Cases of adhesive arachnoiditis causing delayed neurological deficit following central neuraxial anaesthesia, with no declared previous spinal surgery or myelography, reported since 1990.

Year* and reference	Patient's age and sex	Anaesthetic agent(s)	Type of block	Obstetric?	Skin preparation	Time to neurosurgical referral	Treatment	Outcome
1992 [25]	30 F	Unknown	Epidural	Y	Unknown	7 months	Adhesiolysis, steroids	Sphincter disturbance, leg numbness
1993 [22]	38 M	15 ml bupivacaine 0.5%	Epidural	N	Unknown	n/a	Unknown	Lower limb symptoms
1996 [30]	36 F	15 ml lidocaine 2% + adrenaline 1:200 000	Epidural	Y	Unknown	12 h	Adhesiolysis, cyst fenestration	Sphincter disturbance, cyst formation
2007 [24]	30 F	20 ml lidocaine 2% + 5 ml bupivacaine 0.5%	Epidural	Y	Unknown	6 days	Steroids	Sphincter disturbance, lower limb symptoms
2008 [39]†	40 F	2.8 ml bupivacaine 0.5%	Spinal	Y	0.5% chlorhexidine in 70% alcohol	41 days	Ventriculoperitoneal shunt	Permanent paraplegia
2009 [26]	29 F	Ropivacaine 0.2% + morphine (epidural; doses not specified), 2.4 ml bupivacaine 0.5% (spinal)	Combined epidural-spinal	Y	Unknown	5 months	Repeated adhesiolysis, cyst fenestration, cystoperitoneal shunt insertion	Sphincter disturbance, leg weakness, cyst formation
2011 (present case)	27 F	2.5 ml bupivacaine 0.5% + 12.5 µg fentanyl	Spinal	Y	2% chlorhexidine in 70% isopropyl alcohol (swabstick)	11 days	Ventriculoperitoneal shunt, foramen magnum decompression, steroids, trial of external syring drain	Permanent paraplegia

n/a, not applicable.

*Estimated date of case determined from case report.

†Case information taken from the High Court judgement. The speculative cause was chlorhexidine contamination.

of alcoholic chlorhexidine on the meninges and neural elements does not appear to have been modelled in animals.

Accordingly, we considered chlorhexidine contamination as a possibility in the present case. Acute back pain and subacute occipital headache following injection is consistent with the presence of a noxious agent circulating in the CSF and is strikingly similar to the peri-operative headache described in Sutcliffe vs Aintree Hospitals [39] and one of the patients in the Wooley and Roe case [37]. A pre-packaged chlorhexidine-impregnated swabstick 'lollipop' [44] was used for disinfecting the skin, which was left to dry over 3 min as advised by the manufacturers. There was no bowl of liquid chlorhexidine solution nearby to permit significant contamination of syringe or needle (both disposable and removed from pre-sterilised packaging immediately before use) and the procedure was performed on a slim patient in a seated position, precluding significant pooling of chlorhexidine on the skin surface to be accidentally aspirated and injected. This said, in our experience, such chlorhexidine swabsticks are prone to dripping and a volume of alcoholic chlorhexidine pools inside the packaging. Either could conceivably represent a source of contamination, especially if the packaging remains near the sterile field.

In our case, 2% chlorhexidine in 70% alcohol was used on the patient's skin. The manufacturer specifically advises against use of its chlorhexidine products in 'lumbar puncture or in contact with meninges' [44], although the evidence for chlorhexidine's antiseptic superiority over the alternatives, principally povidone-iodine, is clear, and usage for central neuraxial block is supported by anaesthetic Societies in several countries [40, 45, 46]. Many practitioners would use 0.5% preparations [47], although use of 2% solution finds advocates in the literature [48]. Of course, a higher concentration of chlorhexidine means a smaller volume of contaminant may be required to precipitate a meningeal reaction, and using this preparation may increase the risk of adhesive arachnoiditis. This small, theoretical risk must be set against that of introducing infection when considering, which concentration to use [47].

We have no evidence that contamination with alcoholic chlorhexidine occurred in this case. Given the

severity of the potential outcome and the relative ease with which contamination can be avoided, however, several preventative steps are advisable. It is already standard practice to avoid the pooling of chlorhexidine on the skin and to allow it to dry completely before proceeding. Chlorhexidine swabsticks, packaging or receptacles should be placed as far as is practical from needles and syringes, opened or unopened, ideally on a separate tray. Opened impregnated swabsticks or vessels containing detergents should not be passed over the sterile tray to prevent dripping.

Treatment

Few treatment options are available for the management of adhesive arachnoiditis and the most severe cases are likely to develop lasting disability despite aggressive intervention [8]. The associated psychosocial burden is large and depression and suicide are common [6, 8]. Some authors advocate decompressive laminectomy and careful microsurgical lysis of the nerve roots but outcomes remain generally poor [6, 25, 26]. Cases, such as the one reported herein, with longitudinally extensive arachnoiditis, are not amenable to this treatment. In occasional cases complicated by space-occupying cystic inclusions, surgical management of the cysts has a recognised role [6, 26, 29]. For associated syringomyelia, various shunting procedures are possible, although case series have shown benefit rates of only 31% [14], and complications [23] and long-term patency problems are well recognised. In light of this, we chose a less invasive trial of an external drain, which was unfortunately not helpful.

As other authors have done [24, 25], we unsuccessfully trialled a course of high-dose dexamethasone in an attempt to arrest our patient's rapid neurological decline, although, 2 months into the disease process, adhesions had already formed and irreversible neural ischaemia and micro-trauma, rather than inflammation, were likely to be driving the bulk of the deterioration. Earlier steroid therapy may well yield better results, but is dependent on practitioners' recognising adhesive arachnoiditis in its often ambiguous early stage and distinguishing it confidently from more common, particularly infective, complications of spinal anaesthesia.

Conclusion

This case and other reports of delayed neurological deficit secondary to adhesive arachnoiditis following apparently routine central neuraxial anaesthesia share significant similarities [22, 24–26, 29]. Within the broad spectrum of adhesive arachnoiditis, a subgroup of patients may be emerging who are prone to develop severe neurological symptoms relatively rapidly – within weeks or months of central neuraxial anaesthesia – in response to blood, perhaps local anaesthetics or traces of contaminants. They may be distinct from those patients with adhesive arachnoiditis whose symptoms, of which pain is often primary, are more nebulous and may take years to manifest. It is possible that this former group of patients share a common predisposition to adhesive arachnoiditis in the form of abnormal fibrolytic pathways, allergy and other immune disorders or genetic factors.

A second possibility is that gross contamination with disinfectants, such as chlorhexidine, is responsible. Given the number of procedures performed worldwide and the ubiquity of chlorhexidine, it is unlikely that trace amounts of chlorhexidine are sufficient to cause adhesive arachnoiditis and instead measurable volumes are probably required. While this would normally be noticed by anaesthetists or those assisting them, significant accidental mixing or administration, while highly improbable, can never be impossible. Such lapses may have occurred in some of the unexplained cases listed in Table 2.

It is sensible to sound a note of caution – this disturbing case and the handful of others listed here do not establish a causative link between central neuraxial anaesthesia and adhesive arachnoiditis, and the suggested aetiologies remain unproven. That these unexplained yet devastating outcomes in healthy patients – usually young mothers – may be avoidable, however, mandates a call for further research and reporting. In particular, animal experiments to define better the volume and concentration of commonly used skin preparations required to cause a meningeal reaction and/or adhesive arachnoiditis would greatly inform the debate as to the currently (largely speculative) role of contamination. In the meantime, practitioners of spinal or epidural anaesthesia are strongly encouraged to discuss this hugely disabling, but, fortunately, exceed-

ingly rare complication as part of obtaining informed consent. Every conceivable effort must be taken to avoid contamination of the injectate, including removing any sources of chlorhexidine and avoiding dripping or splashing, or passing impregnated swabs or disinfectant containers across the sterile field. Ensuring meticulous technique and taking reports of peri-procedural paraesthesia and pain seriously are also essential.

Competing interests

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