Obstetric epidurals and chronic adhesive arachnoiditis

Editor—We welcome Rice and colleagues’ review of obstetric epidurals and chronic adhesive arachnoiditis (CAA), but are disappointed with the confusing and imprecise presentation of data and concepts, some of which have now been superseded. There is indeed no definite evidence for a link between CAA and epidural analgesia as currently practised in the UK with low-concentration bupivacaine. However, insufficiency of evidence does not mean that obstetric epidural analgesia does not pose a significant risk for the development of CAA. Nor, given modern systems for data collection and analysis, would it require an ‘enormous prospective study...over many years’ to obtain such evidence. In fact, Holdcroft and colleagues’ 1995 study over one year confirmed seven cases of persistent neurological deficit after 48,006 deliveries, of which 13,633 were associated with epidural or spinal analgesia. These were unfortunately not followed up with clinical interview or MRI, but the time scale and numbers involved are certainly manageable.

Although many physicians may be unclear about the precise pathology and clinical picture of CAA, the disease itself is clearly defined and has a range of neurological symptoms that are similar to, but less diverse than, for example, those of multiple sclerosis. It is no more ‘nebulous’ than relating different radicular symptoms to the site of radicular involvement in the pathological process. In clinical practice the more useful neurological abnormality in CAA is a sensory one, determined by finding neuropathic pain, usually associated with allodynia and hypoesthesia, within approximate dermatomal territories. Together with the presence of a spinal procedure or trauma in the past medical history, this would already give conspicuous pointers towards the diagnosis of CAA and a short list of differentials.

For radiological corroboration of the clinical diagnosis, a sensitivity of 92% with a specificity of 100% from MRI (though taken from a paper published in 1987) is superior to the figures quoted for disc prolapse and for recognition of early multiple sclerosis. Although rare, with an estimated prevalence of 1 per 3000 population, CAA is not ‘extremely rare’. This estimate derives from 25,000 spinal surgeries per year, and does not include other causes of arachnoiditis such as intraspinal injections and spinal trauma. CAA is underdiagnosed by an unaware clinical community, not by difficulties arising in making the diagnosis.

Turning to available estimates of the risk of neurological injury after epidural and spinal anaesthesia or analgesia, the figures from the study by Dahlgren and Tornebrandt have not been cited. They found an incidence of 1 in 1300 for persistent neurological symptoms after epidural anaesthesia. Palot and colleagues’ review of 288,351 epidurals given for labour over a 5 yr period produced an incidence of 1 in 4005 for serious complications, though they note the difficulty of identifying isolated radiculopathies. It would be reasonable to take a figure of 1 in 2000 as a working estimate for the potential risk of neurological complications from obstetric analgesia. There is no confirmation of CAA as a pathological cause for these cases, but then, it was not looked for.

If this is the sort of figure that arises for neurological injury from obstetric epidurals, should the young mother be informed about such a risk? With ~200,000 spinal and epidural analgesic procedures each year in the UK, there is the potential for 100 young mothers to be chronically disabled. One factor we miss in Rice and colleagues’ paper is discussion of the implications of a ~2% incidence of dural puncture during epidural catheterization. Consequences remain speculative, but the potential risk is introduced.

In conclusion, CAA is not a nebulous disease entity, nor is the diagnosis difficult, providing the clinician is aware of the disease. Sadly, persistent neurological deficit does occur after epidural and spinal analgesia, although the pathological mechanism remains speculative in the absence of clinical and radiological examination.

Underdiagnosis of CAA after obstetric analgesia and epidural and spinal anaesthesia is a certainty. Neurological complications are often not followed up as a method of self-audit with clinical examination and MRI. Furthermore, arachnoiditis may present months to years after the causative event. As Holdcroft and colleagues pointed out, ‘significant morbidity is not being recognized in hospitals where women are being delivered and it is within the community that these disorders are recognized. This has implications for training, audit and risk assessment.’

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Editor—In the very useful review of CAA, the statement by Rice and colleagues that ‘the Woolley and Roe case of 1954 famously advanced contamination with detergents as a cause of neurological abnormalities after spinal anaesthesia’ is incorrect.

In 1947, at the hospital treating Woolley and Roe, chemicals and detergents were not used to clean spinal needles, and in Cope’s account of the trial, the possibility of detergent contamination is not even mentioned. What, in fact, was famously advanced in Cope’s text as the cause of the post-spinal paralyses (and accepted by the Court) was Professor Macintosh’s theory of phenol seepage through invisible cracks in glass ampoules of nupercaine; an opinion which was rejected in a strongly worded editorial in this journal.

At Cecil Roe’s post mortem, the pathologist found classical signs of CAA, and the most likely and now generally accepted explanation is acid contamination of the syringes and needles. Failure by Rice and colleagues to identify this has led to their omission of acidity as another cause of CAA.

This cannot be dismissed as a historical nicety. Gissen and colleagues have demonstrated the dangers to neurological function from a very low pH in association with modern local anaesthetic agents. Although anaesthetists are aware of the risks associated with neurotoxic preservatives when giving drugs intrathecally or epidurally, the dangers associated with extremes of pH tend to be forgotten. Yet again, we must be reminded that the lessons of history are forgotten at our peril.

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Editor—Thank you for the opportunity to reply to the letter of Talbot and Lewis, and that of Hutter. The syndrome of CAA is neither clear-cut, nor well defined. There is no consistent clinical pattern, and the relationship between pathological findings and symptomatology has not been determined. One of our aims in writing the review article was to highlight the clinical features and diagnostic criteria of CAA to those who perform obstetric epidurals. Without this information, CAA will continue to be a
nebulous disease entity and one that is difficult to diagnose. We agree that MRI is a sensitive and specific tool in the diagnosis of CAA, which is why we also quoted the work of Ross and colleagues in our review,1 and recommended that MRI should be used to diagnose CAA.

We do not agree with Talbot and Lewis' interpretation of the studies of neurological deficit following epidural analgesia. Palot's review of nearly 300 000 labour epidurals over a 5yr period found an incidence of 1:4005 serious complications after epidural analgesia.2 Dural puncture (1:156), massive subarachnoid injections (1:8010), and convulsions (1:9011) are quoted as the most frequent of these. Palot quotes an incidence of 1:3277 for transient neurological disturbance, but does not report any permanent neurological deficit or CAA.

Dahlgren and Tornebrandt's retrospective study4 of 17 733 central neuraxial blocks looked at 8501 spinals and 9232 epidurals, none of which was obstetric. They found three cases of permanent neurological damage that were disputably attributable to epidural anaesthesia; an incidence of 1:3077.

In Holdcroft's retrospective study5 of 48006 deliveries in London, 13 007 patients had epidural analgesia. Thirteen patients were found to have neurological problems after delivery, seven lasting for >1yr. However, after extensive investigation, only one was attributable to epidural analgesia; an incidence of 1:13 007 permanent neurological sequelae after an obstetric epidural, but none of these cases were linked to CAA. Holdcroft herself points out that this incidence is comparable with that found by Usubiaga (1:11 000).6 It would also correlate with the incidences reported by Scott and Hibbard (0.1:10 000),7 Aromaa (0.47: 10 000),8 and Kane (0.6: 10 000).9 Talbot and Lewis are therefore wide of the mark quoting a 'working estimate' of 1:2000. As we highlight in our article, it is not possible to use the evidence from any of these studies to comment on a link between CAA and obstetric epidurals. A study to determine this would need to be prospective, and more extensive both in depth and duration than those already undertaken.

We are interested that Talbot and Lewis quote a figure of 1 in 3000 for the incidence of CAA, and that this figure appears to have been taken from a population who have undergone spinal surgery. Previous spinal surgery is a known risk factor for CAA, and so it is not surprising that such a population would be predisposed towards the development of CAA.

We also dispute the 2% incidence of dural puncture ‘during epidural catheterization’ quoted by Talbot and Lewis. In the UK, a recent study quoted the risk of inadvertent dural puncture during epidural insertion for labour analgesia of 0.1–0.5%.10 Talbot and Lewis feel that the consequences of dural puncture remain ‘speculative’. However, they have been extensively investigated. Speculation and inaccurate reporting of the facts is not appropriate when considering the risks of epidural analgesia. We still have not found any evidence that obstetric epidurals as currently used in the UK predispose parturients to CAA.

Albert Woolley and Cecil Roe underwent spinal anaesthesia performed to routine practice at the time. Their paralysis has been attributed to contamination by chemicals used in the descaling of spinal needles and syringes.9 We realize that, by their chemical structure, these are acids, not detergents. We referred to them as detergents, as they were used in the cleaning process. We apologize to Dr Hutter for the inaccuracy and thank him for the correction.

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