Interlaminar approach for epiduroscopy in patients with failed back surgery syndrome

Editor—We read with interest the article by Avellanal and Diaz-Reganon.1 We acknowledge that they have demonstrated that the interlaminar approach for epiduroscopy is possible. However, we would argue with their conclusions that its diagnostic efficacy was clear, due to flaws in the design of the study and their high rate of complications.

There were small numbers involved in this study, a short duration of follow-up for a chronic condition, wide variations in pre-procedure management, no blinding, and no attempt to provide control subjects. There appears to be no attempt made to adhere to the core outcome measures as detailed in the IMMPACT (Initiative on Methods, Measurement and Pain Assessment in Clinical Trials) recommendations2 making acceptance of the findings more difficult. We have reservations over a technique which offers a 31.6% chance of no improvement and a 10% chance of making symptoms worse. The group who showed improvement (7/13) only had a one to two point improvement in their VAS (visual analogue scale). Is such a small decrease in VAS clinically significant? Possibly if the authors could have demonstrated a reduction in other core outcome measures such as physical and emotional functioning or drug use reduction. Unfortunately, they did not. In addition, they describe what we feel is an unacceptably high rate of complications. The authors describe 4/19 (21%) patients suffered dural puncture. This does not compare favourably in comparison with Igarashi and colleagues3 who had 1/58 (1.7%) patients suffer a dural puncture. A further 4/19 patients suffered transient neurological symptoms of headache or hypoacusia meaning that 8/19 (42%) patients had suffered a serious complication from the procedure. At no point in their study they mention other previously described complications which were felt to not be uncommon. This includes intravascular injection4 and visual disturbance.5

We have other reservations after reading the description of the technique. Using a solid 14 G Tuohy needle to introduce the epiduroscope raises two problems. First, how were they able to safely secure its position within the epidural space? Secondly, did they experience problems with shearing of the epiduroscope when they advanced and withdrew the catheter through the needle or when they rotated the Tuohy needle itself to help steer the epiduroscope? These are problems which one would not anticipate using the previously described route via a soft-tipped introducer placed through the sacral hiatus over a guidewire.

In conclusion, we felt that this approach for epiduroscopy, although physically possible, was not clinically appropriate on the grounds of weak evidence which shows low success rates and high complication rates. This paper is not going to alter the NICE guidelines6 which states that there is inadequate safety and efficacy evidence to support epiduroscopy other than for research and audit. This stance is further supported by the findings that targeted placement of epidural steroid is no better than an untargeted caudal epidural injection which is associated with a lower risk of complication.7

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Editor—We are grateful to Dr Fai and colleagues for their comments. The design of the study could possibly have been better set out and included more measures, but we used a system as in previous studies.7 8 In our study, 31.6% of the patients showed significant improvement which was maintained in all cases at 6 months, and we can now confirm that the improvement was maintained 1 yr later. These patients had previously had all the treatments available, including epidurolysis, without benefit. If we had not performed epiduroscopy, these patients (1/3) would have been required spinal cord stimulation. This means that one-third of our patients had relief from a serious pain problem and returned to their work and normal life without implantation of spinal cord stimulation systems. Moreover, in no case did the procedure make the patient worse. Therefore, we believe it to be clinically significant.

In relation to the rate of complications, we had 21% incidence of dural puncture. This is high in comparison with other studies.3 These are our initial results. We think that our patients cannot be compared with other series due to their high number of operations at lumbar level. On the other hand, only one patient suffered post-dural puncture headache. If you want to break adhesions down and reach the nerve roots in patients with strong adhesions, dural puncture during catheter advance must be considered as a side-effect. It can probably be diminished with practice. In fact, we have reduced the incidence of dural puncture to 10% in the last 20 procedures performed; in these cases, no patient suffered headache.
Fai and colleagues consider transient neurological symptoms such as headache or hypoacusia related to injection of saline boluses to be ‘serious complications from the procedure’. The mean duration of such ‘serious complications’ was 10 s, and in no case, these lasted for more than 30 s. These are well-known transient symptoms in patients during epiduroscopy or epiduralysis, especially in patients who are not under sedation, and are directly related to the pressure of injection. We did not mention cases of visual disturbance or intravascular injection because we did not have these complications. However, we included in the discussion a paragraph related to these transient symptoms and the importance of a more accurate control of the epidural pressure in order to avoid them. As for the possible risk of shearing the epiduroscope, it was not a problem in any case. We used an epidural needle (14 G RX COUDÉ) with special tip design that makes it possible to advance and withdraw the catheter without shearing it.

We think epiduroscopy will find a place among the diagnostic and therapeutic tools in patients with chronic low back pain with or without radiculopathy, and especially in patients with failed back surgery syndrome. We have described a new method for performing epiduroscopy, which allows an interlaminar approach. It requires training and practice. In fact, we have improved on our personal results, now reaching 40% of very significant improvement in the last series with a very low incidence of side-effects.

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Pulmonary oedema after high infusion rate of sulprostone

Editor—The use of sulprostone [prostaglandin (PG) E2] for atonic uterine haemorrhage is increasing. Despite the potential of the drug to cause pulmonary oedema and coronary artery spasm, few clinical reports of side-effects have been published. We present a case of a 35-yr-old healthy Jehovah’s Witness requiring emergency surgery for a postpartum haemorrhage of at least 1 litre of blood. Rapid sequence induction and intubation were uneventful. Bleeding was controlled by manual compression and insertion of a uterine balloon device. Sulprostone was infused at a rate varying from 2.1 to 8.2 μg min⁻¹. During the operation, which lasted ~2 h, estimated total blood loss reached 3 litre, and 1 litre of lactated Ringer’s solution and 3 litre of 6% hydroxyethylstarch (HES) (Voluven®) were administered. Blood was withheld in accordance with the patient’s expressed wish. Adequate haemostasis was achieved, facilitated by the use of tranexamic acid 2 g, calcium 2.2 mmol, and desmopressine 24 μg. The patient remained haemodynamically stable throughout the procedure with a final core temperature of 37.2°C. We therefore decided to extubate her trachea in the operating theatre. At this stage, ~600 μg of sulprostone had been infused. Just before extubation, the patient showed signs of agitation, such that pulse oximetry readings became unreliable. After extubation, the patient remained restless and did not respond to verbal commands. Owing to persistent cyanosis, despite oxygen 100% by face mask, we decided to re-intubate her trachea. This proved very difficult due to flooding of the laryngeal inlet with colourless fluid. Saturation was restored to 100% with 20 cm H₂O positive end-expiratory pressure (PEEP) and an FiO₂ of 0.6. Furosemide 60 mg was given. In intensive care, she was successfully weaned from the ventilator the next day and subsequently discharged to the ward. No blood or blood products were administered and the lowest haemoglobin concentration measured was 3.8 mmol litre⁻¹.

We have learned three lessons from this experience. First, we suspect that the rate of infusion of sulprostone and not only the total dose might be a determinant of the development for pulmonary oedema. Secondly, restlessness at emergence after administration of sulprostone should alert the anaesthetist to the possibility of pulmonary oedema, even if no fluid is visible in the tube as was the case with our patient, and that re-intubation might be difficult. Thirdly, since the mechanism of pulmonary oedema after sulprostone infusion is not completely understood, mainstay of therapy remains supportive including intubation and ventilation with PEEP.

PGE₂ in animal models can increase the hydrostatic pressure and vascular permeability of the pulmonary vascular bed, which can lead to pulmonary oedema.