FIGURE Pain relief in labour after initial dose of epidural anaesthesia.

TABLE Patient's characteristics

Age (yr)	29.4 ± 0.8
Weight (kg)	77.9 ± 2.5
Height (cm)	165.4 ± 1.2
Parity	
– Primiparous	39 (60%)
– Multiparous	26 (40%)
Infant weight (g)	3551.8 ± 82.5
Before epidural :	
– Cervical dilatation (cm)	3.8 ± 0.3
– Range of dilatation (cm)	1–8
– Visual analogue scores (0–10)	7.6 ± 0.3

Values are mean ± SEM.

obtaining direct evaluation of the drugs and techniques that we use daily.

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Spinal anaesthesia and gravity

To the Editor:

Lui *et al.* emphasise the importance of gravity on the distribution of isobaric and hyperbaric solutions in a "sitting" model of the spinal canal.¹ *In vivo*, the situation is more complex with gravity playing a limited role, and then only with "heavy" solutions.^{2,3} The posturally induced spread of previously stable analgesic levels noted with plain bupivacaine^{4–6} cannot be explained by gravitational theories nor by the behaviour of solutions in spinal canal models.

There is considerable evidence that marked venous volume changes occur within the lower spinal canal (without any change in CSF pressure),⁷ and these, by displacing CSF cranially have a major effect on the spread of intrathecal drugs.^{8–10}

In his original article on the glass spine, Barker¹¹ implied that, in the absence of gravitational effects, the only way solutions injected at the second lumbar interspace could affect the "mid dorsal region or even higher" was "by the shifting of the whole column of cerebrospinal fluid in which it is suspended upwards." This prediction seems to have been largely ignored.

Clinically, such movements of CSF will modify significantly the effects of gravity on spinal drugs. Unless models of the spinal canal are multi-compartmental and allow for differential volume changes and fluid movement within the compartments then *in vitro* research into the mechanisms underlying the spread of intrathecal drugs will be limited and misleading.

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REPLY

There is no question that an in vitro study is a simplification of the in vivo situation. Just as each individual plant species in a forest may be examined individually or collectively, we chose to study only the single factor, baricity, and its effect on the distribution of local anaesthetic solution in spinal anaesthesia. The study was not designed to incorporate the effects of cerebral spinal fluid volume on the spread of lidocaine, but only to determine whether the molecules of local anaesthetic actually migrate with the molecules of dextrose when the two are injected into the cerebrospinal fluid together, as is so commonly done.

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Trigemino-cardiac reflexes

To the Editor:

The recent clinical report by Lang *et al.*¹ and the accompanying editorial by Blanc² are timely discussions of a problem faced jointly by anaesthetists, maxillofacial, oral, plastic and neurological surgeons during procedures which manipulate the trigeminal system. The nature of these autonomic changes is best understood if they are categorized simply as pressor or depressor responses. Kumada *et al.* studied the response of the trigeminal brainstem complex and nerve to low frequency electrical stimulation and coined the term "trigeminal depressor response" in rabbits to describe the significant bradycardia, hypotension, respiratory depression and gastric hypomotility which they observed. Higher frequency stimulation causes a pressor response.³ We have also observed a significant depressor response upon percutaneous compression of the trigeminal ganglion in rabbits.⁴ These findings are not specific to the trigeminal nerve and have been observed in the sciatic nerve of cats.

In a study of 21 patients who underwent percutaneous compression of the trigeminal nerve and ganglion for the treatment of trigeminal neuralgia significant drops in heart rate and blood pressure were observed in 70% of procedures. We recommended the use of a non-invasive temporary external pacemaker fitted preoperatively in these procedures.⁵ The depressor response is probably from A-delta fibre excitation at energy levels lower than that needed to excite C-fibres. C-fibre injury will cause a pressor response. The depressor response is from stretching or compression of the trigeminal nerve during maxillofacial surgery and also is a warning to the surgeon that the nerve is being injured. The advantage of using the temporary non-invasive pacemaker is that unnecessary injury to the trigeminal system can be detected and avoided without the need for the use of atropine, which will then mask the effects of further manipulation. Atropine should, however, always be available, as should sodium nitroprusside, for treatment of the pressor response. We also recommend use of the term "trigeminal depressor response" rather than "trigemino-cardiac" or "trigeminorespiratory reflex" since the autonomic response to the trigeminal system injury is a generalized one.

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REPLY (1)

I wish to thank Dr. Brown et al. for their thoughtful letter. Their observations and review of the literature support the existence of