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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

and clinical significance of trigemino-cardiac reflexes. We have not found it necessary to use external pacemakers fitted pre-operatively for maxillo-facial procedures. Episodes of bradycardia and/or asystole respond to cessation of the surgical stimulus and intravenous atropine. However, in some instances, it may well be prudent to place an external cardiac pacing unit before surgery. A suggestion that the trigeminocardiac reflex can be used as an index of potential injury to the trigeminal nerve is interesting. Firm recommendations cannot be made, however, as there is no conclusive experimental evidence to support Dr. Brown's contention that injury to the trigeminal nerve can be avoided in this fashion.

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REPLY (2)

Drs. Brown, Preul and Nimr raise two interesting and uncommon issues: the semantics of the terms "trigeminal depressor response," "trigeminocardiac" and "trigeminorespiratory reflex," and the advantage of using a non-invasive temporary external pacemaker, instead of intravenous atropine, during percutaneous trigeminal ganglion compression for unilateral trigeminal neuralgia or tic douloureux.

First, I found no reason to choose the term "trigeminal depressor response" rather than "trigeminocardiac" or "trigeminorespiratory reflex" because the so-called "trigeminal system" is not a single physiological unit. Equal electrical stimuli do not elicit equal responses when applied to the spinal trigeminal tract and its nucleus or to each of the trigeminal nerves or branches.¹⁻² Secondly, the term "trigeminal depressor response" (TDR) was coined by Kumada et al.¹ to describe a unique tetrad of symptoms (arterial hypotension, bradycardia, apnoea and gastric hypermotility – not hypomotility) following electrical stimulation of discrete sites of the "spinal trigeminal complex" in anaesthetized or decerebrated rabbits. Most important, arterial hypotension is related to reduction of peripheral resistance and not to decreased cardiac output. Brown, Preul and Nimr restrict the term TDR to the cardiovascular responses. Their studies in humans show that when a needle was guided to the foramen ovale and a Fogarty embolectomy catheter was advanced, through the needle, among the fibres of the mandibular nerve and of the Gasserian ganglion, and/or when the balloon of this catheter was inflated, bradycardia was elicited in 70% of procedures and arterial hypotension occurred in 55% of procedures.³⁻⁴ Brown and Preul observed episodes of sudden bradycardia usually but not always accompanied by episodes of arterial hypotension but they did not prove that arterial hypotension was due to a reduction of peripheral resistance and not to sudden and/or profound bradycardia. If arterial hypotension was not secondary to bradycardia it should neither be prevented with intravenous atropine nor easily corrected with an external pacemaker. Moreover, Kumada et al.¹ emphasize that the TDR appears to be an unique response differing from all other autonomic reflexes which are well established as being mediated through the trigeminal nerves, including the diving reflex, the nasopharyngeal reflex and the oculocardiac reflex (OCR). Thirdly, the occurrence of "cardiovascular pressor responses" (arterial hypertension, tachycardia, and/or tachyarrhythmias)

seems to depend mainly upon the stimulus strength, the stimulus frequency, the anatomical structure being stimulated, and the anaesthetic chosen. High intensity and high frequency electrical stimuli tend to activate nociceptive pathways and to elicit a sympathetic response.^{1,3,5,6} Trigeminal stimulation is more often associated with bradycardia.¹⁻³ In the series of patients reported by Brown and Preul,³⁻⁴ stimulation of the mandibular nerve and/or of the Gasserian ganglion was frequently followed by bradycardia with or without arterial hypotension. Only two episodes of transitory arterial hypertension (with moderate bradycardia) were observed.³ On the contrary, in the series reported by Kehler et al.,⁷ and by Sweet et al.,⁸ trigeminal rhizotomy and radiofrequency heating of trigeminal rootlets, were complicated with marked increases in systolic blood pressure when the surgeon stimulated the trigeminal ganglion. Finally, it is important to recognise the differences between an oculocardiac reflex (OCR) and an oculo-respiratory reflex (ORR) because: (1) their efferent pathways and effector organs are different; (2) it is easier to elicit an ORR than to elicit an OCR;⁹ (3) hypercapnia augments the incidence of the OCR in spontaneously breathing infants and children¹⁰ and hypoxaemia must be prevented when facing the possibility of cardiac arrhythmias; (4) intravenous atropine abolishes the OCR while it does not change the incidence of the ORR in humans,^{10,11} and seems to enhance the ORR in dogs.^{12,13}

I have no experience with the use of an external pacemaker. If there is a contra-indication to atropine, an external pacemaker programmed to respond when the heart rate decreases to below 50 beats · min⁻¹ seems to be an elegant alternative to prevent excessive bradycardia, arterial hypotension and cerebral hypoperfusion, if a trigeminocardiac reflex – rather than a TDR – is elicited.

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