and clinical significance of trigemino-cardiac reflexes. We have not found it necessary to use external pacemakers fitted preoperatively 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 pheripheral 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 oculorespiratory 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 children[10] 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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