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Facial Pain Induced by Music

To the Editor:—The term "musicogenic epilepsy" first was used by Critchley[1] to describe seizures that were induced by certain types of music. Accordingly, my case might be called "musicogenic facial pain." A 31-year-old man complained of a four-month history of right-sided facial pain combined with bilateral involuntary eye blinking induced by listening to rock music on a tape recorder. The pain was characterized as an electrical shock radiating to the right side in the distribution of the second branch of the trigeminal nerve and lasted 2–5 s. The facial pain with eye blinking occurred frequently at several second intervals during the music. Immediately upon cessation of the music, the facial pain with eye blinking disappeared completely. This type of pain with eye blinking was completely reproduced by rock music but could not be evoked by tactile or other sensory stimulation involving the face and mouth nor by other types of music. Following the administration of 200 mg carbamazepine a day, po, the facial pain with involuntary eye blinking during rock music disappeared promptly. This patient may have experienced this facial pain as a part of an episode of musicogenic epilepsy. There is no doubt that musicogenic epilepsy is a rare form of epilepsy. Furthermore, partial sensory seizures induced by music are very rare. Among several theories concerned with the pathogenesis of trigeminal neuralgia, an epileptiform type of pain attack is worth considering. The character of the pain (sudden onset, short duration, trigger mechanisms) and the therapeutic effect of antiepileptic drugs appear to favor this hypothesis. This case report strongly suggests that the pathogenesis of atypical facial pain as well as true trigeminal neuralgia may be related to some type of epilepsy.

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Some Corrections Concerning the Precipitation of Local Anesthetic Drugs in Cerebrospinal Fluid

To the Editor:—In a recent article, Moore[1] summarized our study[2] which appeared in *Der Anaesthesist* as follows:

> Bupivacaine, etidocaine, mepivacaine, and tetracaine solutions have been stated to precipitate in CSF (cerebrospinal fluid). This conclusion was based on an *in vitro* aerobic study, which human CSF was frozen, then reconstituted at a later date, mixed with solutions of the local anesthetic drugs, titrated to the *p*H of CSF. The authors cautioned that injection of these drugs into the subarachnoid space might cause spinal cord damage.

I regret that none of these three statements represents an accurate interpretation of content, methods, or conclusions in our paper.

First, we have never concluded that "Bupivacaine, etidocaine, mepivacaine, and tetracaine solutions . . . precipitate in CSF." It is true that we have investigated the solubility of local anesthetics in CSF *in vitro* and its dependence on the hydrogen ion concentration. We have shown that under control conditions of *p*H 7.371, P_{CO_2} 40 mmHg, and temperature 37° C, the solubilities of the local anesthetics in CSF are as follows: bupivacaine HCl: 0.83 ± 0.10 mg/ml CSF; carticaine HCl: 27.00 ± 2.80 mg/ml CSF; lidocaine HCl: 24.00 ± 1.30 mg/ml CSF; mepivacaine HCl: 14.80 ± 0.20 mg/ml CSF; and tetracaine HCl: 1.40 ± 0.12 mg/ml CSF.

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Second, our method did not involve freezing CSF, its later reconstitution, or mixing it with solutions of local anesthetics. The quantitative analysis of solubility involved the use of buffer solutions by Britton Robinson, *p*H 6.05–9.9 and a 25% ammonia solution. The measurements of *p*H were made with an Orion® model 701 *p*H meter using a microelectrode type HA 405 MS-NS and a spectrophotometer. The method is described fully in the paper.

Third, we have never "cautioned that the injection of these drugs into the subarachnoid space might cause spinal cord damage." This warning was contained in a letter from the Astra Corporation, Germany, in 1976, as follows: "Late lesions of the spinal cord after injection of the subarachnoid anesthesia with any local anesthetic, including bupivacaine, cannot be excluded" (author's translation). This comment naturally aroused our interest and concern and gave rise to the study we conducted; it was the impetus for our study, not the conclusion of it.

In closing, we propose the following problem: Under control conditions the maximal solubility of bupivacaine is 0.83 ± 10 mg/ml CSF; it is possible that 15 mg of bupivacaine injected in the subarachnoid space would precipitate, since the subarachnoid space volume from T5 to S2 is reported to contain approximately 15 ml of CSF,[3] thus producing a bupivacaine concentration of 0.833 mg/ml. The volume of CSF is a critical question; we accepted an estimate of 15 ml, but is that accurate? We would, therefore, very much like to know of other investigations regarding the volume of the subarachnoid space which either substantiate or shed new light on the previous study.

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The Optimal Test Dose for Epidural Anesthesia

To the Editor:—We read with interest the recent clinical report by Moore and Batra[1] and the subsequent correspondence from Morrison.[2]

We agree entirely with the addition of 1:200,000 epinephrine to the epidural test dose. Since this became our routine in December 1981, we have observed significant tachycardia in four patients undergoing epidural analgesia for cesarian section. We had no hesitation in resiting the epidural catheters in these patients. While its use in the hypertensive parturient perhaps is unwise, the addition of epinephrine to the epidural test dose in the normotensive pregnant woman should substantially reduce the number of inadvertent intravascular injections of local anesthetic. We would, however, dispute the use of isobaric bupivacaine as the local anesthetic test solution. Clearly, the rationale for using a local anesthetic is to test for an inadvertent subarachnoid injection. While we have no experience of injecting 0.75% solution[3] in the pregnant patient, we have studied 25 patients in whom cesarian section was conducted under subarachnoid block using 2–3 ml of 0.5% bupivacaine (specific gravity 1:007) injected at the L3–4 interspace without barbotage. We found this to be unsatisfactory for two reasons: 1) the variability in the level of block achieved; and 2) the long length of time required for the block to become fully established. One patient developed a total spinal after 3 ml of 0.5% bupivacaine: this occurred within two minutes of injection. Two other patients developed a T1 level; one patient received 3 ml while the other received 2 ml of bupivacaine; the block for the latter patient took thirty minutes to reach its maximum level.

Although 4 ml of 0.5% bupivacaine have been used successfully for subarachnoid blocks in nonpregnant patients,[4,5] we feel that this is a dangerous volume to use in the parturient, and that even 2 ml of bupivacaine injected into the subarachnoid space may produce a dangerously high block which will not always be apparent in the five minutes usually allowed to elapse before injecting the main epidural dose. Our experience suggests that there is probably little place for the use of subarachnoid isobaric bupivacaine for cesarian section and that isobaric 0.5% bupivacaine should not be used as an epidural test dose in the pregnant patient.

We agree with Moore[6] that a single-dose vial containing 2–3 ml of a rapidly acting hyperbaric local an-

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