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## Facial Pain Induced by Music

*To the Editor:*—The term "musicogenic epilepsy" first was used by Critchley<sup>1</sup> 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

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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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1. Critchley M: Musicogenic epilepsy. *Brain* 60:13-27, 1937

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

*To the Editor:*—In a recent article, Moore<sup>1</sup> summarized our study<sup>2</sup> which appeared in *Der Anaesthetist* 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 pH 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 pH 7.371, P<sub>CO<sub>2</sub></sub> 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.