disengagement of all domes for the P23Db transducer is somewhat less than two turns. Sisco et al. reported that the dome was discovered to be one turn loose. This means a separation of the diaphragms of 1/16 inch, and that the dome was actually half off the transducer.

A locking mechanism for the dome should be unnecessary. When properly tightened, the dome will not easily work loose, and a latching device could complicate more than benefit the operation. Confirming that the dome is properly tightened is just as easy as checking any of the other connections in the line. It is to be hoped that Sisco et al. and our communication will alert others to the importance of this maneuver.

Methodology for Studying Cerebral Evoked Potentials Challenged

To the Editor—Chapman and Benedetti1 are very likely correct in their prediction that measurements of evoked central nervous system activity will be useful in studying pain. Unfortunately, several problems in study design leave their specific conclusions open to question.

First, both subjects and observers knew when nitrous oxide was being administered and seemingly expected analgesia and changes in cerebral evoked potentials (CEP). They also knew when naloxone was given and that it was expected to change CEP; at least the observers (and very possibly the subjects) anticipated reversal of analgesia after injection. Evoked potentials in the latency range considered (100–500 msec) could surely have been affected by the subjects’ expectations. Attention level, vigilance, and expectancy are known to alter long-latency, sensory-evoked waveforms.2 Double-blind administration of nitrous oxide and naloxone would have lent considerable credibility to the authors’ conclusions.

Second, administration of nitrous oxide by nasal mask with the mouth open leaves considerable doubt as to actual inspired concentrations, and duration of inhalation prior to testing is not stated.

Third, constancy of stimulus application is not at all assured with the method employed. Not only was the stimulating probe hand-held; it was held by the subject! Monitoring the stimulating waveform on an oscilloscope does not ensure uniform delivery of a constant stimulus in this setting. Nitrous oxide, 8–12 per cent, has been shown to impair psychomotor performance.3 What part might the subject’s impaired probe-holding performance have played in changes in CEP and pain reporting found with nitrous oxide?

Finally, room air is compared with nitrous oxide in oxygen, 67 per cent. Room air itself may have some anesthetic effect,4 and we do not know whether increased inspired concentrations of oxygen alter long-latency CEP.

Evoked potential measurement may well prove to be a valuable tool in pain research. The work reported by Chapman and Benedetti invites replication under a more stringent experimental protocol.

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