**Spreading depression**

In a well-designed study, Piper et al. demonstrate that the induction of a cortical spreading depression (SD) by a stab wound to the cortex of the cat is dependent on the anesthetic employed. Isoflurane and halothane, commonly used volatile anesthetics in humans, inhibit SD. In contrast, α-chloralose, an injectable anesthetic, permits the induction of SD. The authors suggest that failure to observe SD in patients during neurosurgical procedures may be attributed to the use of volatile anesthetics.

SD has been hypothesized as playing an important role in cerebrovascular pathophysiology. It is present in rat brain after focal cerebral ischemia, and the numbers of spreading depressions correlate with ischemic cell damage. SD has also been hypothesized as contributing to the pathogenesis of migraine. Although the literature contains provocative studies on the role of SD in cerebral pathophysiology, the lack of evidence supporting the presence of SD in the human dampens enthusiasm for these laboratory studies. The present work provides a possible explanation for the inability to document SD in neurosurgical patients by suggesting that anesthetic agents block SD, and thus keep alive the clinical relevance of SD as an important pathophysiological event.

The results of the present study are also important for investigations of experimental cerebrovascular pathophysiology such as stroke and head trauma. If SD contributes to the cell damage in stroke and head trauma, the effect of anesthetic agents on SD in experimental models must be determined. A secondary benefit of the present study is that it provides insight into the mechanisms that facilitate the induction of SD. The authors discuss the effects of halothane and isoflurane on K⁺ conductance and concentration, and the role of glial cells contributing to the induction of SD.

In summary, this is an important study because it encourages further investigations into the role of SD in migraine and cerebrovascular injury, and provides opportunities to modify SD by mechanisms analogous to those provided by anesthetic agents.

-Michael Chopp

**Idiopathic stabbing headache**

Transient stabbing pains not uncommonly occur spontaneously in the head in the absence of organic disease of underlying structures or of the cranial nerves. Pains are mainly felt in the distribution of the first division of the trigeminal nerve, occurring as single stabs or series of stabs, lasting for less than a second. In this issue, Pareja et al. in great detail describe their clinical features from a series of 38 consecutive patients, collected from two hospitals during a one-year period, and report an annual incidence of 33 per 100,000. However, the authors believe that the real incidence is still higher because of the selection of patients. This is supported by studies of Birthe Krogh Rasmussen and Jes Olesen (1), who found a lifetime prevalence of idiopathic stabbing headache (ISH) of 2%, and by Raskin and Schwartz (2), who reported three ISH sufferers out of 100 adult control subjects, and as many as 42 ISH sufferers out of 100 migraine patients. Drummond and Lance (3) obtained a history of icepick pains in 200 out of 530 patients with recurrent headache (migraine and tension-type headaches). Thus, ISH is a rather common symptom when specifically asked for. The results of the present study also show that coexistence of jabbing pains and other primary headaches is characteristic. Pareja et al. did not report any case of cluster headache suffering from ISH, but it has been noted that such paroxysms of stabbing or pricking sensations may occur during attacks in one-third of cluster headache patients, usually indicating that the cluster attack is coming to an end (4).

Pareja et al. point out that ISH is a primary benign headache and that it has a definite clinical relevance in the differential diagnosis of cranial neuralgias. In two-thirds of ISH patients indomethacin may be useful. The mechanism of the pain remains to be clarified, but a vascular basis has been hypothesized (3). It appears that the primary pathogenetic mechanisms may be localized centrally. Transient increase of cranial sensory input via the trigeminal nerve might facilitate nervous reflex arches in critical centres of the brain stem. Thus, ISH represents both clinical and scientific interest as a marker of vascular headaches.

**REFERENCES**


**Tension headache**

The IHS criteria for tension headache allow for a subclass of patients to be identified who not only have the symptoms of tension headache but also have signs of a pericranial muscle disorder. These criteria accept either tenderness or increased EMG levels as the signs of muscle disorder. The study of Jensen and Rasmussen provides data that demand a revision of these criteria. The authors have dissected the relationship between tenderness, pain threshold, and EMG, and their prevalence in patients with tension headache.

From their results, it is quite clear that pressure-pain threshold and EMG offer no discriminating value in tension headache; abnormalities of any degree are just as common in patients with tension headache as in subjects without headache. The only discriminating feature that emerged from this study is tenderness upon manual palpation. Marked tenderness occurred in 37% of patients with episodic tension headache. If there is to be a subclass of patients with muscular disorders it is these patients.