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 suffers 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 prickling 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


KARL EKBRON

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.
The implication of the Jensen and Rasmussen study is that EMG should not be used as a criterion for subdividing patients; it simply does not discriminate patients. Those who wish to persevere with this criterion should note that their faith in this feature ignores the false-positive rate demonstrated by Jensen and Rasmussen.

Having clarified the position clinically, what the study now calls for is a concerted investigation into the basis of this tenderness. It may indeed be a feature of muscle disorder, as believed by many, but Jensen and Rasmussen have also shown that there is no regular relationship between tenderness, pain and EMG that would be consistent with a primary disorder of muscle. What haunts this field is the prospect that tension headache may be a central disorder in which tenderness is but a feature of dysnociception, and in which EMG changes are irregular epiphenomena.

**Nikolai Bogduk**

**Pattern-reversal VEP**

Rossi et al. have studied 71 pediatric patients who suffered from migraine (with and without aura) or tension-type headache. Data obtained from this sample were compared with data obtained from 19 controls without headache. The authors recorded pattern-reversal VEPs, but failed to observe differences in P100 latencies between groups. This paper is interesting particularly because of the conflicting reports in the existing scientific literature. Specifically, it has been reported that adult migraineurs may or may not demonstrate VEP abnormalities intra- and interictally. The present investigators suggest that their sample of children may not have had headache long enough for the previously reported VEP effects to be observed. However, it is possible that the pattern-reversal VEP is insensitive to the effects of headache on the function of the occipital cortex. This paper adds to the existing body of knowledge chiefly because the subjects were children and because they had a number of different types of headache.

**Gary Jacobson**

**Migraine and tension-type headache in children and adolescents**

In the last few years a team from the Department of Neuropsychiatry of Childhood and Adolescence in Vienna has reported several interesting studies of migraine and tension-type headache in children and adolescents, some of them recently published in the proceedings of the 7th International Headache Congress in Toronto in September 1995.

One of these studies (by C. Wöber-Bingöl et al.) is published in this issue, where it is investigated whether IHS criteria for migraine and tension-type headache depend on gender.

The authors state that girls more often have migraine with aura than boys, who more often have vomiting and phonophobia than girls.

In tension-type headache, females more often reported mild intensity of headache. All other criteria were similar in both sexes. Their study also suggests that tension-type headache may occur more often in girls, and that gender has some influence on the IHS criteria for migraine, but almost no influence on those of tension-type headache.

As there are very few clinical studies in the past dealing with tension-type headache in children and adolescents, this and other studies from this ambitious Vienna team are very welcome.

**Bo Bille**