Neurology 2001;56:428–429

Time for more attention to migrainous vertigo?

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Patients with migraine frequently have vestibular complaints, ranging from frank vertigo to less specific symptoms of dysequilibrium and head motion intolerance. Numerous studies have documented the association of migraine and vestibular symptoms.[1-7] In this issue of *Neurology*, Neuhauser et al. rigorously reassess the association in three groups—200 patients with migraine presenting to a headache clinic, 200 dizzy patients presenting to a neurotology clinic, and a control population of 200 orthopedic inpatients hospitalized for problems unrelated to headache or dizziness.[8] Their study is unique by virtue of a relatively large study population, use of International Headache Society (IHS) criteria to define migraine, a prospective design, and proposed new criteria to define "probable" and "definite" migrainous vertigo. The authors' major findings include the following: 1) migraine was more common in dizziness clinic patients than in orthopedic controls, which supports a connection between migraine and vestibular dysfunction; 2) although many patients complained of episodic vertigo and headaches, only a small portion met criteria for definite or probable migrainous vertigo. In fact, in 75% of dizziness clinic patients with migraine, investigation revealed other causes for the complaints of dizziness, which demonstrates that vertigo can only be attributed to migraine after other causes have been excluded; and 3) the majority of patients with definite migrainous vertigo did not fulfill IHS criteria for migraine with aura or basilar migraine, often because the timing and duration of vestibular symptoms did not meet the IHS criteria for aura.

Do migraine-associated vestibular symptoms warrant special recognition and study, or is it sufficient to regard them as another type of aura (albeit with temporal characteristics that differ somewhat from aura as defined by the IHS)? Neuhauser et al. argue for special attention, because migraine is an underrecognized cause of vertigo. They attribute this lack of recognition to a number of factors, including the frequent temporal dissociation between headache and vestibular symptoms, the sometimes subtle nature of the migrainous symptoms, and the generally poor fit between the characteristics of migrainous vertigo and any existing IHS headache categories. There are obvious clinical benefits to heightening awareness of migrainous vertigo. Clinicians would be more likely to query dizzy patients about migraine symptoms, which patients may not volunteer spontaneously if the symptoms are subtle or temporally dissociated from the vestibular complaints. Physicians might also consider prescribing antimigraine treatments, which are often useful in treating the vestibular complaints.[1-3]

Further development of rigorous criteria for migrainous vertigo will also facilitate the study of the pathophysiology of this disorder, which may in turn elucidate the mechanisms of migraine in general. In terms of both anatomy and physiology, the vestibular system is one of the best understood neural systems, with sophisticated techniques available to quantify its function. Thus, of all forms of migraine-associated neural manifestations, vestibular dysfunction is particularly amenable to study, and it may share a common pathophysiology with other, less experimentally approachable, types of aura.

The study of migrainous vertigo may also afford insight to the linkage between genes and migraine. In 1996, the Dutch Migraine Genetics Research Group[9] demonstrated that familial hemiplegic migraine can result from mutations in the gene for the $\alpha 1$ subunit component of a voltage-gated calcium channel. They predicted that calcium channelopathies may be the basis of more prevalent types of migraine. To date, the pursuit of this prediction has yielded mixed results,[10] doubtless because migraine is pathophysiologically and genetically heterogeneous. A study population drawn from migrainous patients with vestibular complaints would have a better chance of capturing patients with a calcium channelopathy: neuronal calcium channelopathies are often associated with cerebellar dysfunction, and, due to the interconnections of the cerebellum and

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vestibular system, these patients often have vestibular symptoms. However, even a population fulfilling the criteria for definite migrainous vertigo will probably be heterogeneous. In addition to patients whose vestibular symptoms and headache spring from a shared cause such as a channelopathy, there will be others in whom vertigo and migraine are only related because the discomfort of vertigo triggers migraines in a nonspecific fashion. There may also be migraine patients who have vestibular symptoms because the migraine has caused peripheral vestibular damage.[1]

What criteria should be chosen to define migrainous vertigo? With respect to this question, the goals of research and patient care conflict. Research demands stringent criteria to ensure identification of patient groups that represent, to the extent possible, a "pure culture." Patient care requires less rigid definitions that maximize the ability to identify dizzy patients who would benefit from migraine treatments. By their proposal of two separate categories—probable and definite migrainous vertigo—Neuhauser et al. provide a compromise between the requirements of research and patient care. Further studies may argue for modification of their diagnostic criteria, but the probable/definite distinction seems both essential and helpful at this time.

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Editorial

Neurology 2001;56:429–430

CME

Improving brain function with transcranial magnetic stimulation?

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Stimulation of the cerebral cortex has played an integral role in the development of clinical neuroscience, from Penfield's delineation of the motor homunculus, to the therapeutic techniques of electroconvulsive therapy, to the current use of stimulation to determine the function of epileptic cortex or to identify the language-dominant hemisphere. In 1985, Barker et al.[1] introduced transcranial magnetic stimulation (TMS), a technique for electrical stimulation of the cerebral cortex by inducing a strong, rapidly changing magnetic field adjacent to the scalp. Because the calvarium has no impedance to a magnetic field, TMS induces intracortical electrical currents with little or no discomfort. TMS can theoretically be applied to any superficial cortical area. Because the effects of motor cortex stimulation are readily quantified with electromyography (EMG), TMS has been used predominantly in the study of the motor system. For example, Liepert et al.[2] used TMS to map the motor cortex to show that exercise of the paretic arm after a stroke, along with restraint of the nonparalyzed arm, increased the size of the motor area for the affected hand.

TMS can be used not only to map motor cortex, but also to study the function of cortical areas. TMS causes synchronous excitation of neurons followed by a period of inhibition during which ongoing cortical activity is disrupted. With stimulation of the motor

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