MUMS the word. Migraine with unilateral motor symptoms: what can you say?

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Is migraine with unilateral motor symptoms a motor complication of disordered sensory attentional mechanisms?

Migraine is a complex disorder of the nervous system with some well described clinical features of the attacks, such as unilateral throbbing pain that is worsened by physical activity and associated with sensory sensitivity, typically photophobia and phonophobia.¹ Willis thought it was a disorder of the cranial blood vessels but the current view is that migraine is a disorder of the brain.² Migraine has many secrets to uncover, and in the paper by Young and colleagues,³ in this issue, unilateral motor symptoms that do not easily fit classical descriptions for hemiparetic weakness are described (*see p 600*). Are the patients just crazy, perhaps even malingering, or are there features clinical neurology has yet to understand about migraine?

Young and colleagues³ identified 24 subjects from their tertiary referral service and compared them with 48 controls. Nine subjects had episodic migraine and 15 had chronic migraine. Each index case had a complaint of weakness in the limbs, two-thirds of which were homolateral to unilateral headache, and in each there was the phenomenon of give-away weakness. One patient with familial hemiplegic migraine was excluded. More than half had weakness between attacks, and nearly all had concomitant sensory symptoms. There was frequently a rostro-caudal

march of symptoms. Depression, as measured by the Beck inventory, was no more common in the affected patients. The authors controlled for resource use and referral base such that the comparison with the control group becomes a challenge to what we might think about these symptoms.

It is easy to dismiss these patients as being either benignly or malignantly crazy, elaborating symptoms for the purpose of attention to a problem that is not medically serious. However, they seem to have no excess of psychopathology, and frankly their problem (migraine) is serious and it is high time it is considered as such. Young and colleagues³ offer several possibilities, including that this weakness is a form of delayed or prolonged aura, although these symptoms are unlike classical sporadic hemiplegic migraine.⁴ They suggest it is like "clasp-knife" weakness, as in classical spasticity, but my experience is that it is not velocity dependent⁵ but rather almost attention dependent. These patients very often perform normal tasks, such as walking, better than one might predict, unless one predicted that the more they are asked to attend the worse the weakness gets, which is what happens. If this correlation is accepted, is the problem a motor complication of

disordered sensory attentional mechanisms that seem a fundamental feature of migraine?⁶ It is my clinical experience that these symptoms dissipate with good control of migraine with appropriate preventives.

I have had the privilege of working with some of the greatest clinical neuroscientists of the 20th century; neurologists bridging the bench-to bedside valley that separates our clinical observations from scientific explanation. The enduring lesson of that studentship is that sometimes it is better to err on the generous side when judging the worthiness of symptoms, so-to-speak, rather than to jump to the judgment that symptoms not easily explained are just modified madness. A fair wind of interest and a modicum of curiosity may be well rewarded in understanding perhaps one of the many secrets of neurology's most interesting opportunity and perhaps greatest current challenge—migraine.

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