30 seconds. On several occasions it was impossible to precipitate the involuntary movements by persistent thumb to finger movements on the right hand; however, on relaxing the right limb and taking up a similar manoeuvre with the left, the right would immediately develop the involuntary movements as described. Passive movement did not precipitate an attack, nor did voluntary movement of either leg. This woman demonstrated unilateral paroxysmal kinesigenic dystonic movements only. This dyskinesia was most commonly precipitated by voluntary movement of the affected limb; however, unlike those cases reported by Plant, it was also possible to evoke similar attacks by voluntary movement of the contralateral hand. This suggests that whatever the specific precipitant of the dyskinesia, whether it be the movement itself or the anticipation of movement it does not have to be limited to the limb affected by the choreoathetosis.

ANTHONY E LANG
Division of Neurology,
Toronto Western Hospital,
Toronto, Canada M5T 2R2

References


Plant replies:
Dr Lang’s interesting comments raise two important issues. The first concerns the nosology of the paroxysmal dystonias. The case described demonstrates features indicating that this may not be the same disorder as that first clearly distinguished from the other paroxysmal dystonias by Kertesz.1 In particular most reported cases are normal on examination between attacks and also the frequent occurrence of attacks when not moving—particularly when lying in bed—would be most unusual. I would also suggest that certain features reported in most cases of paroxysmal kinesigenic choreoathetosis are likely to prove distinctive of a specific disorder. These are the importance of the abruptness of movements which precipitate attacks, the importance of prior inactivity and the occurrence of a refractory period following attacks. It is not clear from Dr Lang’s account whether these characteristic attributes were present.

The second issue concerns the importance of detailed analysis in cases of dystonia in which involuntary movements are induced by various stimuli. Such disorders are of potential importance in understanding the pathophysiology of dystonia and if others follow Dr Lang’s example of careful observation a clearer picture may emerge from what is currently something of a can of worms.

As for Dr Lang’s specific point I agree that the less rigidly linked focal activity and focal attacks are, then the more likely it is that a mechanism related to the initiation of movement is the primary precipitant rather than the motor activity per se or feedback from the limbs. I have observed a large number of attacks in cases 1 and 2 described in my article and although I have never seen an attack in the contralateral limbs induced by hopping I have witnessed, in case 2, attacks provoked apparently by the anticipation of movement where no movement took place other than the attack itself. In the act of hopping all four limbs are moved: it was my intention to point out the importance of either the initial component of a complex movement or, conceivably, the side of the body to which attention is directed in determining the laterality of the attacks in my cases.

References


Indomethacin-responsive episodic cluster headache

Sir: Since more attention has been paid to cluster headache various other forms of this disease have been distinguished besides its classic or episodic form.2,3 The localisation of the pain is the basis of the definition of this disease while the frequency and character of attacks take various forms.3 Apart from the classical types, we can also distinguish so-called transitional forms.4,5 The interesting description of the case presented by Geaney* can not, I think, be classified as the episodic form, but is the transitional form between the episodic and the CPH forms. The effectiveness of indomethacin in cases of CPH is regarded as one of the criteria for diagnosis. However, I would like to point out that indomethacin is also effective in transitional forms of cluster headache. In 1976 I described a case which was transitional between the chronic and the CPH forms.7 She was a 50-year-old woman who suffered from classic migraine in her childhood. When she was 45 yr she started to suffer from the episodic form of cluster headache, which then turned into the secondarily chronic one. After 2 years, during which the patient reported 1–3 attacks per day, exacerbation of the disease occurred when the number of attacks per day increased to 7 with simultaneous shortening of duration. When she was treated with indomethacin the attacks ceased completely after a few days. My case and the one presented by Geaney are not the only atypical forms of this disease, but it should be noted that indomethacin often proves to be effective when attacks are frequent.

A KLIMEK
Department of Neurology,
School of Medicine,
Köpïckieskiego 22,
90–153 Łódź, Poland

References

5 Bogucki A, Niewodnicz A. Chronic cluster headache with unusual high frequency of attacks. Differential diagnosis with CHP. Headache (in press).

Geaney replies:
Chronic paroxysmal hemicrania (CPh) was reviewed in 1980 and it was apparent that prior to the development of the chronic stage, in which multiple attacks occur every day, there is frequently a preliminary stage during which the headaches may occur in episodic or cyclical form (pre-CPh stage). This episodic stage has lasted for up to 19 years before evolving into the chronic stage.
Indomethacin-responsive episodic cluster headache.

A Klimek

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