Hand-held myometry

In their two recent articles, Vander Ploeg *et al* provided information that should prove useful to clinicians who use hand held dynamometers. The reference values presented by the authors provide a much needed basis for establishing the normality of a patient's strength.¹ The ratios between the forces measured during make tests and break tests may, as the authors suggest, be diagnostically discriminating.² The purpose of this letter is to provide some additional information relevant to reference values and make test and break test forces.

Reference values, based on hand held dynamometer measurements, have been published before. The values (which were for make tests rather than break tests) were limited, however, to 10 upper extremity muscle groups of healthy young women.³ Comparisons of the ratio of break test forces to make test forces have also been published previously. The ratios, however, are higher than those reported by Vander Ploeg *et al*. Specifically, Bohannon reported that the force measured at the elbow during break tests was a mean 1.3 times as great as that measured during make tests. The ratio was demonstrated in both healthy subjects⁴ and on the nonparetic side of patients with stroke.⁵ On the paretic side of the stroke patients, the break test to make test force ratio was a mean 1.7 to 1.0. Why the ratios reported by Bohannon are so different from those of Vander Ploeg *et al* is uncertain. What I believe is certain is that hand held dynamometry is a much underused clinical measurement procedure and that further research needs to be conducted on the procedure.

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- 1 Vander Ploeg RJO, Fidler V, Oosterhuis HJGH. Hand-held myometry: reference values. *J Neurol Neurosurg Psychiatry* 1991;54:244-7.
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Paroxysmal kinesigenic choreoathetosis

A recent letter¹ discussed paroxysmal kinesigenic choreoathetosis (PKC) as a presenting of symptom multiple sclerosis. Exertion, emotion (stress or hyperventilation) and sudden movements have been reported to provoke such attacks in multiple sclerosis. There is a lack of consensus on their nomenclature: terms include tonic spasms, tonic seizures, paroxysmal dystonia, tetanoid attacks and sensorimotor seizures. For simplicity in the case of multiple sclerosis, one might suggest the label tonic spasms, reserving the term PKC for idiopathic attacks, often familial, and with onset in youth. Semantic distinctions aside, the stereotyped attacks described

are likely a result of "white matter (axonal) irritability", rather than grey matter (neuronal) dysfunction. By contrast, the latter pathology appears responsible for many of the less paroxysmal, and the hereditary dystonias and choreas.

As recognised, the MRI of the patient shown had too many lesions to allow unambiguous assignment of the right-sided spasms to a specific anatomic area. However, due to recent reports on MRI localisation of the anatomical basis of tonic spasms,²⁻⁴ it is likely that the causative lesion is that seen in the left (contralateral) posterior limb of the internal capsule, rather than the grey matter lesions.

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- 1 Roos RAC, Wintzen AR, Vielvoye G, Polder TW. Paroxysmal kinesigenic choreoathetosis as presenting symptom of multiple sclerosis. *J Neurol Neurosurg Psychiatry* 1991;54:657-8.
- 2 Honig LS, Wasserstein PH, Adornato BT. The anatomic basis of tonic spasms in multiple sclerosis (MS). *Neurology* 1988;38:236.
- 3 Honig LS, Wasserstein PH, Adornato BT. Tonic spasms in multiple sclerosis: anatomic basis and treatment. *West J Med* 1991;154:723-6.
- 4 Maimone D, Reder AT, Finocchiaro F, Recupero E. Internal capsule plaque and tonic spasms in multiple sclerosis. *Arch Neurol* 1991;48:427-9.

Roos replies:

In his letter Dr Honig mentioned the well known problem of descriptions of movements. Video monitoring these motor problems would evade most semantic problems. Tonic spasms are different from choreoathetotic movements induced by motion or other stimuli. Also in the article by Maimone *et al* tonic spasms are said to be known as tonic seizures or paroxysmal dystonia! His article is illustrated with an MRI showing a very similar white matter abnormality as in our patient. A causal relationship therefore between the central white matter lesion and tonic spasms is suggested. The interesting point made by Dr Honig cannot be solved because we saw a patient with paroxysmal kinesigenic choreoathetosis.

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Angiostrongylus cantonensis abscess in the brain; what do we learn?

It was interesting to read, in the article by Purohit *et al*,¹ that *Angiostrongylus cantonensis* abscess was mistakenly treated initially as tuberculoma with antituberculous drugs, because of the CT picture of enhancing disc lesion. The lesion had apparently not increased in size even after two months, although the authors do not mention about the repeat CT findings. It would be altogether an interesting issue to know the natural history of such lesions.

Unfortunately, in India an enhancing single small ring or disc lesion on CT of the brain is presumed to be tuberculoma and

antituberculous therapy is indiscriminately instituted without histological verification. The fact that such lesions "disappear" after a few months of antituberculous therapy falsely reinforces the physicians' faith in continuing such medication. It has been found, however, that such lesions are cysticercosis or parasitic granulomas and not tuberculomas.^{2,3}

Is there a role of empirical antituberculous therapy while treating such lesions? The answer to that crucial question is definitely no. It is hoped that one considers the parasitic diseases affecting the brain as the first possibility in diagnosing single, small, enhancing ring or disc lesions especially in countries like India where hygienic conditions are extremely poor.

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- 1 Purohit AK, Dinakar I, Sundaram C, Ratnakar KS. Angiostrongylus cantonensis abscess in the brain. *J Neurol Neurosurg Psychiatry* 1991;54:1015-6.
- 2 Chandry MJ, Rajshekhar V, Ghosh S, Prakash S, Joseph T, Abraham J, Chandi SM. Single small enhancing CT lesions in Indian patients with epilepsy: clinical, radiological and pathological considerations. *J Neurol Neurosurg Psychiatry* 1991;54:702-5.
- 3 Prakash S, Abraham J, Chandry MJ, Rajshekhar V, Ghosh S, Joseph T. Intracranial tuberculomas (Letter). *Paediatric Neuroscience* (In Press).

Purohit *et al* reply:

Dr Prakash has correctly pointed out that antituberculous treatment is indiscriminately instituted in India without histological confirmation. In the present case, however, the antituberculous treatment was started because of the following genuine reasons.

- 1 The enhancing disc shaped solid morphology on CT scan is a common feature of tuberculoma aetiology.¹
- 2 The patient belongs to that part of India where cysticercosis is not at all a common disorder but tuberculosis is surely a more common disease.

Lastly, we would like to draw the attention to the inference which has been quoted from the study of other cases² and cannot be applied to the present case because the cases they have studied had CT lesion of less than 10 mm in size whereas with our case the diameter was 20 mm.

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- 1 Umesh SV, *et al*. Intracranial tuberculoma and the CT scan. *J Neurosurg* 1986;64:568-74.
- 2 Chandry MJ, *et al*. Single small enhancing CT lesions in Indian patients with epilepsy: clinical, radiological and pathological considerations. *J Neurol Neurosurg Psychiatry* 1991;54:702-5.