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. The relations between the forces measured during make tests and break tests may, as the authors suggest, be diagnostically discriminating. 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. 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 of 1.3 compared to 1.6 as that measured during make tests. The ratio was demonstrated in both healthy subjects and on the nonparetic side of patients with stroke. 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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Paroxysmal kinesigenic choreoathetosis

A recent letter1 discussed paroxysmal kinesigenic choreoathetosis (PKC) as a presenting symptom of 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 clamp, paroxysmal dystonia, tetonoid attacks and sensorimotor seizures. For simplicity in the case of multiple sclerosis, one might suggest 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 assignation of the right-sided spasms to a specific anatomical 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 seen that in the left (contralateral) posterior limb of the internal capsule, rather than the choreoathetotic lesions.

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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 of Maimone et al tonic spasms are said to be known as tonic seizures or paroxysmal dystonias! 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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Matters arising

Angiostrongylus cantonensis abscesses 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 tuberculosis 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 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 tuberculosis and antituberculous therapy is indiscriminately instituted without histological confirmation. The fact that such lesions "disappear" after a few months of antituberculous therapy falsely reinforces the physicians' faith in continuing such treatment. It has been found, however, that such lesions are cystercerosis or parasitic granulomas and not tuberculosis.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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Purohit et al reply:

Dr Purohit 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 tuberculous aetiology.

2) The patient belongs to that part of India where cystercerosis 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 cases 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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