MATTERS ARISING

Cerebral localisations in articulatory dyspraxias

Clarke et al reported a case of pure articulatory dyspraxia (pure anarthria, PA) without orofacial apraxia (OFA), in a patient with a predominantly cortical haemorrhagic contu- sion involving a part of the precentral gyrus. The patient had developed a syndrome that they have termed 'pure' PA (as opposed to apraxia, PA). They suggested that the components of PA may be related to specific localisations. Carden et al reported an anatomoclinical correlation in cases of partial PA. We also recently described a case of pure-dysprastic PA without OFA and, like Clarke et al, located the lesion in the inferior part of the dominant precentral gyrus. Such cases of partial PA and related cases reported in the literature suggest precise anatomoclinical correlations: a) A predominantly cortical lesion of the inferior part of the dominant precentral gyrus responsible for pure PA or motor apraxia PA; b) A cortical or a motor but not ideomotor OFA may be seen; b) A predominantly subcortical lesion of the dominant opercular responsible for dyspraxic PA without OFA or palsy; c) A lesion of the lower part of the middle frontal gyrus responsible for dyspraxic PA or ideomotor apraxia PA; d) A lesion of the lower part of the dominant inferior precentral frontal gyrus responsible for dyspraxic PA or ideomotor apraxia PA; e) A lesion of the lower part of the dominant inferior frontal gyrus associated with OFA. The case of Clarke et al appears to agree with such a hypothesis.

Neurocysticercotic versus idiopathic epilepsy

I read with interest the letter by Arruda. The conclusion drawn by the author that "Any patient with late onset epilepsy with normal neurological examination and living in an endemic area of taeniasis/cysticercosis should be considered as possibly having neurocysticercosis regardless of seizure type and EEG findings" is erroneous. The data presented by the author and literature on the subject do not support such contention. Epilepsy has varied causes in any age group. Ahuja et al in a prospective study of late onset epilepsy from India (an endemic area) found cysticercosis as a cause in only 5-1% cases, while no cause could be detected to 75-1% patients. To say that all these patients had neurocysticercosis because they had normal neurological examination and living in an endemic area would be stretching the imagination too far. It is interesting that none of the patients in the latter group on follow up which extended to 18 months showed any evidence of neurocysticercosis. To label all patients of late onset epilepsy with normal examination as neuro-cysticercosis just because the authors did not find any difference in certain parameters studied, is logically incorrect. Since the prognosis and length of treatment required in neurocysticercosis and idiopathic epilepsy may be different, the practical implications of the statement are obvious. In my opinion, the conclusion drawn by the author is not supported by facts, is unscientific and dangerous.


Arruda replies: I appreciated the comments by Dr Ahuja and I quite agree that the conclusion as presented is misleading. In fact, a single but essential word is missing in the final sentence: possibly! It should read: "Any patient with late onset epilepsy with normal neurological examination and living in an endemic area of taeniasis/cysticercosis should be considered as possibly having neurocysticercosis regardless of seizure type and EEG findings." This conclusion is supported by previous studies showing neurocysticercosis (NC) in 25-5% to 50% of patients with late onset epilepsy and living in endemic areas of these diseases in Brazil and Mexico. Ahuja and Mohanta report a much lower prevalence of NC in Northern India (5-1%). Nevertheless, NC should always be considered in the differential diagnosis of patients with late onset epilepsy even in these populations due to its possible therapeutic and prognostic implications.


Isolated muscle hypertrophy as a sign of radial or peripheral nerve injury

The article by Martle et al showed that hypertrophy of a single muscle can be a sign of partial nerve or ventral root lesion, and this diagnosis is confirmed by the presence of "profound spontaneous activity in the hypertrophied muscle". However, I am concerned about the proposed diagnostic value of this somewhat arbitrary EMG sign and the authors' suggestion that knowledge of this clinical sign might become necessary, costly, and sometimes invasive investigations", in search for, for example, "focal myositis or muscle neoplasms". We have seen such lipomatous tumours of the calf with profound spontaneous activity in the "hypertrophic" muscle. Case 1

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P Humphrey and M D Shaw

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