MATTERS ARISING

Cerebral localisations in articulatory dyspraxias
Clarke et al reported a case of pure articulatory dyspraxia (pure anarchia, PA) without orofacial apraxia (OFA), in a patient with a predominantly cortical haemorrhagic contu- sion. 1 Likewise, PA is considered a very special form of OFA. Finally, OFA is interpreted as an ideomotor or a motor (melokinetic) apraxia, the latter being a motor dysfunction intermediate between "pure" palsy and apraxia. 2 Lecours et al suggested that the components of PA may be related to specific localisations. 2 Cardebat et al reported such anatomoclinical correlations in cases of partial PA. We also recently described a case of pure-dystonic PA without OFA 3 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 4 suggest precise anatomoclinical correlations: a) A predominantly cortical lesion of the inferior part of the dominant precentral gyrus responsible for parietal PA or motor apraxia PA; b) an orofacial palsy or a motor but not ideomotor OFA may be seen; b) A predominantly subcortical lesion of the dominant opercular responsible for dystonic PA without OFA or palsy; c) A lesion of the lower part of the ventral frontal gyrus F2 responsible for dyspraxic PA or ideomotor apraxic PA, with ideomotor OFA; d) A lesion of the lower part of the dominant inferior frontal gyrus F3 responsible for PA without OFA or palsy. 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. 1 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 regarding type and EEG findings" is supported by previous studies 2-4 showing neurocysticercosis (NC) in 25-50% of patients with late onset epilepsy and living in endemic areas of these diseases in Brazil and Mexico. Ahuja and Mohanta 5 report a much lower prevalence of NC in Northern India (5%). Nevertheless, NC should always be considered in patients with late onset epilepsy even in these populations due to its possible therapeutic and prognostic implications.

Isolated muscle hypertrophy as a sign of radialar or peripheral nerve injury
The article by Martle et al 1 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 "profuse spontaneous activity" in the hypertrophied muscle. However, a study about the proposed diagnostic value of this somewhat arbitrary EMG sign and the authors' suggestion that knowledge of this clinical sign might be of great necessity, costly, and sometimes invasive investigations, in search of for example, "focal myositis or muscle neoplasms" has been seen with both lippatous tumours of the calf with profuse spontaneous activity in the "hypertrophic" muscle. Case 1


Arruda replies: I appreciated the comments by Dr Ahuja and I quite agree that the conclusion as presented is misleading. 1 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 regarding type and EEG findings." This conclusion is supported by previous studies 2-4 showing neurocysticercosis (NC) in 25-50% of patients with late onset epilepsy and living in endemic areas of these diseases in Brazil and Mexico. Ahuja and Mohanta 5 report a much lower prevalence of NC in Northern India (5%). Nevertheless, NC should always be considered in patients with late onset epilepsy even in these populations due to its possible therapeutic and prognostic implications.


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Neurocysticercotic versus idiopathic epilepsy
I read with interest the letter by Arruda. 1 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 2 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 lived 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 implementation of the mentioned statement are obvious. In my opinion, the conclusion drawn by the author is not supported by facts, is unscientific and dangerous.

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Non-invasive diagnosis of internal carotid artery dissections.

P Humphrey and M D Shaw

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