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 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." is not accurate.

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Arruda replies: I appreciated the comments by Dr Ahuja and I quite agree that the conclusion as presented is misleading, especially since a single but essential word is missing in the final sentence: possibly! It should read: "Any patient with late onset epilepsy with normal examination as neurocysticercosis is probably more likely to have neck trauma, Horner's syndrome. All patients with late onset epilepsy 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%). 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.

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Isolated muscle hypertrophy as a sign of radicular 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 hypertrophic muscle. However, awareness about the proposed diagnostic value of this somewhat arbitrary EMG sign and the authors' suggestion that knowledge of this clinical sign might be useful for timely treatment of such a muscle commentator about muscle hypertrophy interest has been raised in such lipomatous tumours of the calf with profuse spontaneous activity in the "hypertrophic" muscle. Case 1

Non-invasive diagnosis of internal carotid artery dissections

We read the article by Mullges et al with interest.2 Carotid artery dissection is probably one of the commonest causes of young stroke.3 Many centres operate a selective policy for angiography in young stroke. The presence of neck pain, Horner's syndrome or history of trauma are widely thought to be useful pointers to the diagnosis of carotid dissection. We would agree with Mullges et al.1 that these signs are only present in a small number of cases, if an alternative screening test is used to detect carotid dissection. We have used a Computed-Doppler to screen all possible young strokes (age range 14-55 years).4 All dissections had either a considerably reduced internal carotid artery flow or a too and fro signal. During the period of 1986-91, only one dissection had been seen at this unit. A history of trauma, neck pain or Horner's syndrome was only present in five cases. All cases were confirmed by angiography. The small number of cases with these clinical parameters may have been accentuated by our policy of not performing angiograms on patients with a severe deficit who have little to lose from a further stroke. Patients with a severe completed stroke are probably more likely to have neck pain and a Horner's syndrome.

We would endorse the view that clinical parameters are a poor guide to the presence of carotid dissection and that carotid dissec-
tion should be considered the most likely diagnosis in all young strokes if no cardinal source of emboli is found. The ultra sound findings act as a useful screening test and when combined with MRI scanning would seem to obviate the need for conventional angiography.

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Cerebral localisations in articulatory dyspraxias

Clarke et al.1 reported a case of pure articulatory dyspraxia (pure anarthria, PA) without orofacial apraxia (OFA), in a patient with a predominantly cortical haemorrhagic contusion.3 Likewise, PA is considered a very special form of OFA. Finally, OFA is interpreted as an ideomotor or a motor (melokinetik) apraxia, the latter being a motor dysfunction intermediate between "pure" palsy and apraxia.2,4 Alajouanine et al.5 suggested that the components of PA may be related to specific localisations. Cardebat et al.6 reported such anatomoclinical correlations in cases of partial PA. We also recently described a case of petro-dystonic PA without OFA.7 and, like Clarke et al.,8 located the lesion in the inferior part of the dominant precentral gyrus. Such cases of PA and related cases reported in the literature9 suggest precise anatomoclinical correlations: a) A predominantly cortical lesion of the inferior part of the dominant precentral gyrus responsible for petrotic PA or motor apraxic PA; b) An orofacial palsy or a motor but not ideomotor OFA may be seen; b) A predominantly subcortical lesion of the dominant operculum responsible for dystonic PA without OFA or palsy; c) A lesion of the lower part of the frontal lobe is responsible for middle 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 of Broca's aphasia, without OFA. The case of Clarke et al.10 appears to agree with such a hypothesis.


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