MATTERS ARISING

Cerebral localisations in articulatory dyspraxias

Clarke et al1 reported a case of pure articulatory dyspraxia (pure anarthria, PA) without orofacial apraxia (OFA), in a patient with a predominantly cortical haemorrhagic contusion, and one of the earliest reports of the phonetic, ideomotor and ideokinetic (PA) apraxia. 

It is noted that the components of PA may be related to specific localisations.2 Cardebat et al2 reported such anatomoclinical correlations in cases of partial PA. We also recently described a case of paretic-dystonic PA without OFA and, like Clarke et al,1 located the lesion in the inferior part of the dominant precentral gyrus.

Such cases1 of partial PA and related cases reported in the literature1 5 suggest specific anatomoclinical correlations: a) A predominantly cortical lesion of the inferior part of the dominant precentral gyrus responsible for paretic PA or motor apraxia PA, and 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 third frontal gyrus, middle frontal gyrus F2 responsible for dyspraxic PA or ideomotor apraxia PA, with ideomotor OFA; d) A lesion of the lower part of the dominant inferior frontal gyrus responsible for PA or ideomotor apraxia PA, with ideomotor OFA; e) A lesion of the part of the dominant inferior frontal gyrus F3 responsible for dyspraxic PA or ideomotor apraxia PA, with ideomotor OFA; f) A lesion of the lower part of the dominant precentral gyrus responsible for PA or ideomotor apraxia PA, with ideomotor OFA; and g) A lesion of the lower part of the dominant inferior frontal gyrus responsible for PA or ideomotor apraxia PA, with ideomotor OFA and/or Broca’s aphasia, without OFA. The case of Clarke et al1 appears to agree with such a hypothesis.

YD DE SMET
Clinique Saint-Louis,
Hôpital Régional du Nord,
BP 103 1-9020 Etsbünderg,
Luxembourg

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 taeiniasis/cystercerosis should be considered as 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 al2 in a prospective study of late onset epilepsy from India (an endemic area) found cystercerosis as a cause in only 5.1% cases, while no case 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 implications of the statement are obvious. In my opinion, the conclusion drawn by the author is not supported by facts, is unscientific and dangerous.

G K AHUJA
All India Institute of Medical Sciences,
New Delhi-110029, INDIA

Arruda reply: 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 taeiniasis/cystercerosis should be considered as possibly having neurocysticercosis regardless of seizure type and EEG findings.” This conclusion is supported by previous studies2-4 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 Mohanta6 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.

WO ARRUDA
Rua Gonçalves Dias 713, 80-240 Curitiba PR, BRAZIL


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

The article by Martle et al1 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, an article appeared about the proposed diagnostic value of this somewhat arbitrary EMG sign and the authors' suggestion that knowledge of this clinical sign might be seen as unnecessary, costly, and sometimes invasive investigations," in search of for example, "focal myositis or muscle neoplasms". We have seen two such lipomatous tumours of the calf with profuse spontaneous activity in the "hypertrophic" muscle. Case 1...
Neurocysticercotic versus idiopathic epilepsy.

G K Ahuja

*J Neurol Neurosurg Psychiatry* 1992 55: 1220
doi: 10.1136/jnnp.55.12.1220-b

Updated information and services can be found at:
http://jnnp.bmj.com/content/55/12/1220.3.citation

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/