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 contusion of the precentral, part of the phonetic integration syndrome (PA) of Alajouanine, Ombredane and Durand proceeds from a triple dysfunction: motor, dystonic and dyspraxic. Likewise, PA is considered a special form of PA. OFA can be interpreted as an ideomotor or a motor (melokinetin) apraxia, the latter being a motor dysfunction intermediate between "pure" palsy and apraxia. Cardebat et al. suggested that the components of PA may be related to specific localisations. Cardebat et al. reported such anatomoclinical correlations in cases of partial PA. We also recently described a case of pure PA. The left part of the frontal lobe was normal. The middle frontal gyrus F2 is responsible for dyspraxic PA or ideomotor apraxia. Clarke et al. agreed with such a hypothesis.

Neurocysticercotic versus idiopathic epilepsy

We 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 type and EEG findings" is erroneous. Data presented by the author and literature on the subject do not support such contention. Epilepsy has varied causes in any age group. Arruda et al. in a prospective study of late onset epilepsy from India (an endemic area) found cysticercosis at a cause in only 51% cases, while no cause could be detected in 75% 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 latter group 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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Arruda repuie: 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 paragraph: 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 type and EEG findings". This conclusion is supported by previous studies 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 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. showed that hypertrophy of a single muscle can be a sign of partial nerve or root lesion, and this diagnosis is confirmed by the presence of "profound spontaneous activity in the hypertrophied muscle. However, it is important to consider the proposed diagnostic value of this somewhat arbitrary EMG sign and the authors' suggestion that knowledge of this clinical sign might be unnecessary, costly, and sometimes invasive investigations", in search for example, "focal myositis or muscle neoplasms". We have seen two such lipomatous tumours of the calf with profound spontaneous activity in the "hypertrophic" muscle. Case 1
Iron and Akathisia

The report by O'Loughlin et al. on the serum iron status of patients with acute neuroleptic-induced akathisia is of interest to us as our own similar investigation revealed no association between the two. Since the publication of our findings, we have repeated the study on a further 50 patients recently started on neuroleptic medication, with further negative results (unpublished). Studies of serum iron status in "chronic akathisia" have reported similarly conflicting findings. It would therefore be worthwhile at this stage to re-examine the rationale and the methodology of the studies. We commend Dr O'Loughlin and colleagues for their attention to detail, except for the following points: 1) The definition of akathisia used in their study was based on scale scores. Not only have the psychometric properties of this scale not been published, it was also designed to rate and not "diagnose" akathisia. As we point out elsewhere, the issue of appropriately defining akathisia is of paramount importance. 2) The small number of patients with akathisia involved; 3) The failure of the authors to report the differential neuroleptic doses of the two groups. The dose can be an overriding factor that can skew the contribution of any other factors difficult to determine; 4) The lack of any correlation between Simpson and Angus scores and akathisia scores is surprising, suggesting that their patients may not have typical acute akathisia.

There are some important theoretical considerations. What constitutes iron deficiency, and whether serum biochemical measures accurately reflect the state of brain iron, is debatable. While transferrin is important for the exchange of iron between tissues, its saturation in the serum fluctuates between 20–50% in normal subjects (due to mononuclear cells), and small variations in saturation as seen in the above study are unlikely to affect iron delivery. The most important determinant of tissue iron delivery is the total number of transferrin receptors, and an efficient homeostatic mechanism exists to maintain the rate of iron transported to the tissues. The turnover of iron in the brain is low, and it has a tendency to resist iron depletion, raising further doubts about the pathological significance of the reported findings. It is also possible that neuroleptic drugs either mobilise iron from peripheral tissues into the brain, or decrease its turnover in the brain, making peripheral measures of iron status even more suspect in revealing the iron status in the brain. MRI may be one method of overcoming this problem, but only good animal models are likely to provide the final answer.

Despite these objections, we feel that there are compelling reasons to study the interaction of iron and neuroleptic drugs. The fall in serum iron and transferrin in the patients with akathisia reported by O'Loughlin et al. is intriguing, even though its significance for the development of akathisia is questionable. Rosebush and Mazurek reported somewhat similar findings for patients who developed neuroleptic malignant syndrome. Little is known about the effect of neuroleptics on ferrokinetics. Clearly this area needs further informed investigation.  