Cerebral localisations in articulatory dyspraxias

Clarke et al 1 reported a case of pure articulatory dyspraxia (pure anaesthesia, PA) without orofacial apraxia (OFA), in a patient with a predominantly cortical haemorrhagic contusion in the region of the precentral-inferior parietal integration syndrome (PAI) of Alajouanine, Ombredane and Durand proceeds from a triple dysfunction: paretic, dystonic and dyspraxic. 2 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 (stereotypic apraxia). Clarke et al 1 suggested that the components of PA may be related to specific localisations. 2 Cardebat et al 3 reported such anatomoclinical correlations in cases of partial PA. We also recently described a case of paretic-dystonic PA without OFA 4 and, like Clarke et al 1, located the lesion in the inferior part of the dominant precentral gyrus.

Such cases 2 of partial PA and related cases reported in the literature 5 suggest precise anatomoclinical correlations: a) A predominantly cortical lesion of the inferior part of the dominant precentral gyrus responsible for paretic 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 F2 in cases of non-paretic apraxia, of carotid dissection, was only reported in 1985. 6 7 We would endorse the view that clinical and pathological parameters are likely to be assessed in all young stroke patients with normal examination as neuro-cytotrophic necrosis 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.

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/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 al 2 in a prospective study of late onset epilepsy from India (an endemic area) found cystercerosis as a cause in only 51% cases, while no cause could be detected to 751% 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-cytotrophic necrosis 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 neurocysticerciosis 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.

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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/cystercrosis should be considered as possibly having neurocysticercosis regardless of seizure 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 disorders in Mexico and Brazil. Ahuja and Mohanta 4 report a much lower prevalence of NC in Northern India (5%). Nevertheless, NC should always be considered in these patients, even in those populations due to its possible therapeutic and prognostic implications.

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Isolated muscle hypertrophy as a sign of radialic 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, it is not clear whether the proposed diagnostic value of this somewhat arbitrary EMG sign and the authors' suggestion that knowledge of this clinical sign might be of a necessary, costly, and sometimes invasive investigations", in search for for example, "focal myositis or muscle neoplasms". We have seen both lipomatous tumours of the calf with profuse spontaneous activity in the "hypertrophic" muscle. Case 1

MATTERS ARISING

Non-invasive diagnosis of internal carotid artery dissections

We read the article by Mullges et al with interest. 1 Carotid artery dissection is probably one of the most common causes of young stroke. 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 like to congratulate 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 Doppler ultrasound to screen all possible young strokes (age range 14-55 years). 2 3 All dissections had either a considerably reduced internal carotid artery flow or a to and fro signal. During the period 1986-95, only one dissection has 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 and pathological parameters are a poor guide to the presence of carotid dissection and that carotid dissection should be considered the most likely diagnosis in all young strokes if no cardiac source of emboli is found.

The ultra sound findings act as a useful screening test. A dissection can be confirmed with MRA scanning would obviate the need for conventional angiography.

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was a 48 year old man who presented with the history and clinical signs of L5 and S1 radiculopathy including fibrillations, positive sharp waves, and bizarre low-frequency discharges in all peroneal, and some in the gluteal muscles, associated with unilateral calf enlargement. Distal motor latencies to the peroneal muscles were mildly prolonged. CT scans showed a 9 × 3 × 3 cm lipoma, which was excised, (fig A) compressing both the deep and superficial peroneal nerves, and a homolateral S1 root compression by a slipped disc. Case 2, a 33 year old housewife, noted progressive painless calf enlargement over several years. Eventually, the circumference of the right calf exceeded that of the left one by 9 cm (fig B). Her neurological status was normal. Electromyography revealed dense fasciculations, bizarre low-frequency discharges, and chronic neurogenic alteration of motor unit potentials exclusively in the right triceps surae muscle. On surgery, diffuse lipomatosis was found and confirmed by microscopical examination. In both cases intramuscular areas with "silent" EMG—close to areas with profuse spontaneous activity—were indicative of a non-myogenic nature of the calf enlargements.

Large series suggest that intramuscular tumours are far more common than denervation hyper trophy.1 2 Focal myositis or muscle neoplasms, if invading the terminal nerve branches, might induce "profuse spontaneous activity".

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Mattle et al reply:

We thank Dr Meinck for his comments and the presentation of two additional cases of isolated muscle hypertrophy. However, comparing his and our cases, the EMG results were substantially different. As Dr Meinck states, both his patients showed intramuscular areas which were silent on EMG close to areas with profuse spontaneous activity. Silent EMG areas were not present in our patients. We intended our cases to make a contribution to the understanding of the pathophysiology of isolated muscle hypertrophy after peripheral nerve or root lesions. We showed that this phenomenon is not restricted to the calves and may affect other muscles as well, where the stretch hypothesis for generation of muscle hypertrophy might not be true and profuse spontaneous activity of the muscle fibres might have caused hypertrophy. We agree that tumours are much more common causes of muscle masses and might cause spontaneous activity on EMG when invading terminal nerve branches. However, as stated, there are silent EMG areas within tumours. In these and doubtful cases we also perform MRI.

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Iron and Akathisia

The report by O'Loughlin et al1 on the serum iron status of patients with acute neuroleptic-induced akathisia is of interest to us as our own similar investigation revealed no associated between the two.2 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.1 3 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.3 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,4 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 affect 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,5 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.6 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 mono- and dimer forms), 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,7 raising further doubts about the biophysical significance of the reported findings. It is also possible that neuroleptic drugs either mobilise iron from peripheral tissues into the brain, or decrease iron turnover in the brain,8 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,9 but only good animal models10 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 al1 is intriguing, even though its significance for the development of akathisia is questionable. Rosebush and Mazurek12 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.

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

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