MATTERS ARISING

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. The pure form of the phonetic disintegration syndrome (PA) of Alajouanine, Ombredane and Durand proceeds from a triple dysfunction : paretic, dystonic and dyspraxic.[2 3] 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 "pure" (ideomotor) apraxia. Alajouanine *et al* suggested that the components of PA may be related to specific localisations.[2] Cardebat *et al*[2] reported such anatomoclinical correlations in cases of partial PA. We also recently described a case of pareto-dystonic PA without OFA [3] and, like Clarke *et al*, [1] located the lesion in the inferior part of the dominant precentral gyrus.

Such cases [2 4] of partial PA and related cases reported in the literature [3 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 apraxic PA; and orofacial palsy or a motor but not ideomotor OFA may be seen; b) A predominantly subcortical lesion of the dominant opercule responsible for dystonic PA without OFA or palsy; c) A lesion of the lower part of the dominant 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 Broca's aphasia, without OFA. The case of Clarke *et al*[1] appears to agree with such a hypothesis.

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1 Clarke CE, Holder DS, Currie S, Stone J. Cerebral localisation in articulatory dyspraxia. *J Neurol Neurosurg Psychiatry* 1992; 55:168–9.
2 Cardebat D, Puel M, Nespoulous JL, Rascol A. Trois observations de désintégration phonétique :étude neurolinguistique. In: Messerli P, Lavorel P, Nespoulous JL, eds. *Neuropsychologie de l'expression orale*. Paris: CNRS, 1983:37–51.
3 Lecours Ar, Lhermitte F. The "pure form" of the phonetic disintegration syndrome (pure anarthria): anatomo- clinical report of a historical case. *Brain and Language* 1976;3:88–113.
4 De Smet YD. Pure anarthria without simultaneous buccofacial apraxia in a Luxemburgish patient. In: Poeck K, Hacke W, Schneider R, eds. *Verhandlungen der Deutschen Gesellschaft für Neurologie 4*. Berlin: Springer-Verlag, 1987:217–8.
5 Tonkonogy J, Goodglass H. Language function, foot of the third frontal gyrus, and Rolandic operculum. *Arch Neurol* 1981;38:486–90.

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 commonest causes of young stroke.[2] 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* 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 continuous wave Doppler to screen all possible young strokes (age range 14–55 years).[3] All dissections had either a considerably reduced internal carotid artery flow or a to and fro signal. During the period 1986–91, 22 cases of carotid dissection have 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 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 and when combined with MRI scanning would seem to obviate the need for conventional angiography.

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1 Mullges W, Ringelstein EB. Leibold M. Non-invasive diagnosis of internal carotid artery dissections. *J Neurol, Neurosurg Psychiatry* 1992;55:98–104.
2 Bogousslavsky J. Ischaemic Strokes in adults younger than 30 years of age. *Arch Neurol* 1987;44:479–82.
3 Eljamel MSM, Humphrey PRD, Shaw MDM. Dissection of the cervical internal carotid artery: the role of doppler/duplex studies and conservative management. *J Neurol, Neurosurg Psychiatry* 1990;53:379–83.

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 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 implications of such a 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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1 Arruda WO. Neurosysticercosis versus idiopathic epilepsy: a comparative study of 175 patients. *J Neurol Neurosurg Psychiatry* 1991;54:1015.
2 Ahuja GK, Mohanta A. Late onset epilepsy. A prospective study. *Acta Neurol Scandinav* 1982;66:216–26.

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 regardless of seizure type and EEG findings". This conclusion is supported by previous studies [2–4] 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 Mohanta [5] report a much lower prevalence of NC in Northern India (5·1%). 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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1 Arruda WO. Neurocysticercotic versus idiopathic epilepsy: a comparative study of 175 patients. J Neurol Neurosurg Psychiatry 1991;54:1015.
2 Arruda WO. Etiology of epilepsy - a prospective study of 210 cases. *Arq Neuro-psiquiatria* 991;49:251–54.
3 Vasconcelos D, Lombardo L. Crisis epilepticas del adulto: estudio prospectivo de 65 casos. *Gac Med Mex* 1983; 119:23.
4 Medina MT, Rosas E, Rubio-Donnadieu F, Sotelo J. Neurocysticercosis as the main cause of late epilepsy in Mexico. *Arch Intern Med* 1990;150:325.
5 Ahuja GK, Mohanta A. Epilepsy of late onset—a prospective study. *Acta Neurol Scand* 1982;66:216–26.

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

The article by Mattle *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, I am concerned about the proposed diagnostic value of this somewhat arbitrary EMG sign and the authors' suggestion that knowledge of this clinical sign might help to avoid "unnecessary, costly, and sometimes invasive investigations", in search of for example, "focal myositis or muscle neoplasms".

I have seen two cases with lipmatous tumours of the calf with profuse spontaneous activity in the "hypertrophic" muscle. Case 1