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 left anteroposterior integration syndrome (PA) of Alajouanine, Ombredane and Durand proceeds from a triple dysfunction: paretic, dystonic and dyspraxic. 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 (idalop). Apraxia. The authors, Clarke et al suggested that the components of PA may be related to specific localisations. Cardetab et al 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, located the lesion in the inferior part of the dominant precentral gyrus.

Such cases of partial PA and related cases reported in the literature 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) 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 anterior 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 F3 responsible for dyspraxic PA or ideomotor apraxia PA, with ideomotor OFA; E) an area of the main sensory area of the cervical region of the sensory cortex responsible for aphasia. The case of Clarke et al appears to agree with such a hypothesis.

Neurocysticercotic versus idiopathic epilepsy

I 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 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 in a prospective study of late onset epilepsy from India (an endemic area) found cysticercosis as a cause in only 5% cases, while no case could be detected to 75% patients. To say that all these patients had neurocysticercosis because they had normal neurological examination and living 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 above statement are obvious. In my opinion, the conclusion drawn by the author is not supported by facts, is unscientistic and dangerous.

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