ANALYSIS OF LESION LOCALIZATION AND SIZE

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ABSTRACT. Methodological issues on the study of the brain / behaviour relationships are reviewed. Present studies intended to evaluate the structural correlates of childhood aphasia should describe the selection of the study sample, aetiology, time interval from onset to neuropsychological examination and imaging studies and the presence of previous and concomitant disorders. CT and MRI make possible a detailed anatomical analysis of lesion site and size. The extent of damage to each relevant anatomical structure should be quantitated.

Correlation of neuropsychological and imaging data can be made through visual methods - overlapping individual lesions - or through chi square, t tests and multiple regression analysis. Whenever available, functional studies, such as SPECT or PET should also be performed, specially in subcortical lesions.

The study of the structural correlates of aphasia has been, since Broca (1861), one of the major aims of those who study brain / behaviour relationships. In the following pages we will address some of the methodological problems of this research. Appropriate selection of subjects, careful analysis of lesion and adequate statistical methods are necessary to relate language disturbance to damage to localized brain sites.

Aetiology, time from onset and referral bias should be considered when describing the study population. Bias should be avoided when selecting comparable controls. For the study of childhood aphasia cases
and controls should be matched not only for sex and age, but also for school grade, social class and, if possible, IQ, (or performance IQ).

Aetiology interacts with lesion location. This may be specially relevant if a center is working with a particular selected population. Non penetrating head trauma tends to affect mainly the basofrontal and the temporal lobes. Herpes encephalitis causes damage to both temporal lobes. With ischemic stroke, which is the best aetiology to study acquired aphasia, the chances of getting lesions on different parts of the brain depends mainly on the physiopathology of stroke. Embolism produces cortical or large subcortical infarcts, while those having an hemodynamic pathogenesis are localized in terminal or border zones (Zeumer and Ringlestein, 1987). In children, the majority of strokes are secondary to intracranial occlusive disease are localized in the basal ganglia (Zimmerman et al, 1987), while those related to congenital heart disease or cardiac surgery are either cortical or of the hemodynamic type (Furlan and Jones, 1987).

What methods can we use to detect and localize brain lesions? They can be divided in morphological and functional. Morphological methods include CT scan and MRI. MRI does not expose subjects to radiation and it is clearly superior to CT scan, specially for posterior fossa imaging, grey-white matter differentiation, visualization of gyri details, detection of small deep lesion. MRI provides not only conventional axial slices, but also coronal and sagittal cuts. Functional methods include electrophysiological procedures, such as EEG and evoked potentials, and perfusion / metabolic studies such as measurements of regional cerebral blood flow that can be imaged in axial tomographic slices, SPECT and PET. The main problem with electrophysiological studies is that they cannot differentiate between the activities of cortical and subcortical structures, while this is possible using with metabolic / perfusion methods. PET is much more expensive and less available than SPECT. With those functional methods patients can be studied at rest and while performing some simple tasks (Rousseau et al, 1989). These activation procedures may be of great interest to study the mechanisms of recovery.

Several parameters should be considered and described when reporting the results of localization studies namely: time from onset, lesion location and size, distant effects of localized lesions and concomitant disorders.

Despite data from several adult series (cf: Kertesz, Harlock and Coates, 1979; Knopman et al, 1983; Ferro and Crespo, 1988) has clearly demonstrated that neuropsychological defects improve over time and, consequently, that the structural correlates of these disturbances are