Disorders vs. neuropsychological developmental difficulties. Relevance of the evolutive relationships in the differential diagnostic

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Keywords: Developmental disorders; Developmental difficulties; Differential diagnosis

Developmental neuropsychological disorders – DNPD – are alterations that affect to psychological processes and skills – attention, memory, language... – by a dysfunctional Nervous System. Mental retardation, learning disorders, communicative disorders, autism, or attention deficit disorder with hyperactivity are considered DNPD, among others (Weiss and Landrigan, 2000). It is obvious that all of them constitute very different kind of disorders in aetiology, characteristics, course and epidemiology. Currently, the available data on the topic are confusing because of: (1) disputes in the delimitation of neurological damage – anatomical, neurochemical, functional... –, (2) multicausal aetiology – genetic factors, voluntary and involuntary environmental... –, (3) lack of knowledge on the interactions between maturation of the nervous system and environmental factors and, consequently, (4) difficulties in the process of detection and differential diagnosis. For this reason, questions as if, in fact, there is an increase of DNPD or if the above-mentioned rise is just explained on the basis of the adoption of a wider diagnostic criterion, still remain without an answer. Only in those cases where congenital alterations can be traced, some certainties can be established. However, there are many other disorders without a clear connection to genetic alterations, about which little can be said in a definite way.

Thus, from the knowledge provided by developmental psychology is possible to consider a proposal that clarifies some gaps with the help of a multidisciplinary approach. The main areas from which the distinction between developmental disorders and neuropsychological difficulties can be established are: the number of fields of development that are involved, the type of clinical or evolutive manifestations, and the progress or course.

For the study of the differentiation disorder vs. difficulty, retrospective and prospective studies are proposed (by perinatal risk analysis (López Gómez et al., 2008a, López Gómez et al., 2008b) and by analyzing the tendency to disorder/difficulty of the neuropsychological development respectively). This work, besides its implications for establishing the differential diagnosis –disorder vs. difficulty–, also presents relevant conclusions for the intervention -therapeutic vs. optimizing –.

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doi:10.1016/j.ijdevneu.2010.07.044

Dendritic bundles, minicolumns, columns, and cortical output units

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Keywords: Pyramidal neuron; Corticocortical neuron; Corticocollicular neuron; Corpus callosum

The search for the fundamental building block of the cerebral cortex has highlighted three structures, perpendicular to the cortical surface: (i) columns of neurons with radially invariant response properties, e.g., receptive field position, sensory modality, stimulus orientation or direction, frequency tuning etc. (ii) minicolumns of radially aligned cell bodies and (iii) bundles, constituted by the apical dendrites of pyramidal neurons with cell bodies in different layers. The latter were described in detail, and sometimes quantitatively, in several species and areas. By retrograde labeling analysis and using MAP-2 immunostaining and Voronoi tessellation in the rat visual cortex, we recently suggested that the dendritic bundles consist of apical dendrites belonging to neurons projecting their axons to specific targets. We would suggest that another structural and computational unit of cerebral cortex is the cortical output unit (COU), i.e., an assembly of bundles of apical dendrites and their parent cell bodies including each of the outputs to distant cortical or subcortical structures, of a given cortical locus (area or part of an area). This somato-dendritic assembly receives inputs some of which are common to the whole assembly and determine its radially invariant response properties, others are specific to one or more dendritic bundles, and determine the specific response signature of neurons in the different cortical layers and projecting to different targets.

doi: 10.1016/j.ijdevneu.2010.07.045

Abnormal cortical development and cell division defects in magoo mutant mice

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Keywords: Cortex; Progenitor; Microcephaly

The formation of the cerebral cortex is a complex process requiring a number of coordinated steps including appropriate temporal and spatial control of progenitor divisions. The size and shape of the cortex are largely determined through a program of tightly controlled neural-progenitor cell divisions. Using forward genetics, we have isolated a mouse mutant called magoo that dies at birth and is microcephalic, with the forebrain showing severely defective brain morphology. The magoo mutation was mapped to a microtubule motor of the kinesin family which has been implicated to function during cell division. The point mutation results in a splicing defect which causes the apparent loss of protein as seen in both immunoblot and immunostaining. magoo mutant mice have thinner cortices across all embryonic ages and immunohistochemistry reveals a significant increase in apoptosis occurs in mutant brains. Cultures of magoo mutant fibroblasts showed an increase in binucleate cells, multipolar mitotic spindles, and chromatin lag. Taken together, these results suggest that cell division defects and loss