To the Editor.

Goldberg (1985) has argued that disruption of the mesolimbic/mesocortical dopamine system by long-term neuroleptic treatment may be responsible for producing the tardive dysmentia syndrome (Wilson et al. 1983), which can be seen as similar to the pseudopsychopathic presentation sometimes observed following damage to orbitomedial frontal cortex (Blumer and Benson 1975; Damasio 1979). Similarly, the akinesia syndrome of Rifkin, Quitkin, and Klein (1975) is viewed as analogous to the pseudodepressed variety of frontal syndrome that follows damage to the dorsolateral frontal convexity. These can be considered cognitive or “psychic” analogues to the more purely motoric tardive dyskinesia syndromes that result from disorders of the nigrostriatal dopamine system.

I would extend these observations to include the possibility that dysfunction in the nigrostriatal system, as well as in the mesolimbic/mesocortical system, may also play a role in the cognitive, affective, and behavioral manifestations of schizophrenia and allied disorders, and that the basal ganglia and associated structures may play important roles in broader aspects of human behavior.

The basal ganglia are frequently implicated in neurologic disease states that produce delusional and psychotic symptomatology (Bowman and Lewis 1980; Cummings 1985), and MacLean (1985) has suggested that the neostriatal portion of the basal ganglia may play a role in the coordination of behavioral subroutines that enable the organism to negotiate the demands of the external environment vis-a-vis its own needs and desires—a capacity that has been associated with frontal lobe functioning (Nauta 1971; Luria 1980) and with the psychotic disorders that may attend its dysfunction (Muller 1985).

In man, although the basal ganglia have traditionally been considered a component of the motor system, clinical evidence exists to implicate these structures in the modulation of recursive goal-directed behavior and thought, i.e., in the processes of “motivation” in a broad sense (Caine et al. 1978; Eccles 1982). Indeed, the akinesias ascribed by Goldberg (1985) to dysfunction of the mesolimbic/mesocortical-prefrontal system bear a striking resemblance to akinetic syndromes, psychic as well as motoric, that occur with neostriatal disease or damage (Caine et al. 1978; Laplane et al. 1984).

Thus, the nigrostriatal system may possess points of functional as well as anatomic interface with the limbic-cortical systems regulating complex behavior.

Goldberg (1985) points out that the frontal lobes, more than any other system, are likely to be the point of summation of dysfunctions generated by many morphologically or chemically diverse systems due to a multifocal or diffuse CNS disease, or to reflect anatomically specific but remote CNS events or disturbances (p. 259).

As neuropsychologists working in the field of psychiatry, we must resist the temptation to infer structure from function in a literal, correlational manner, based on reference to the brain-behavior rubrics traditionalized over time with neurological populations. Luria (1973) warned against this “narrow localizationism” with respect to brain-injured subjects; how much more cautious must we be when dealing with psychiatric groups where structure-function relationships are only beginning to be understood (Miller, in press).
Goldberg (1985) has made a valuable contribution to this effort by emphasizing the multidimensional nature of brain-behavior interactions with respect to “frontal lobe” deficits in schizophrenia. A similar point has been made with reference to the hemisphericity issue in schizophrenia (Miller 1984). It is to be hoped that this approach will diffuse through research and theory in psychiatric neuropsychology as a whole.

References


Miller, L. “Narrow localizationism” in psychiatric neuropsychology. Psychological Medicine, in press.


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