Childhood Autism from the Point of View of Behavioral Neurology

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Analysis of the abnormalities present in autistic patients, using standard methods of clinical inference, leads to the conclusion that the manifestations of childhood autism correlate best with the manifestations of dysfunction in the frontal tier of the ring of phylogenetically older cortex located on the mesial surface of the frontal and temporal lobes ("mesocortex" or "mesolimbic cortex") and with the manifestations of dysfunction in the striatum. These structures are distinctive in ways that suggest possible etiologies. The observations and inferences that lead to these conclusions are outlined and are supplemented by data from recent studies by the authors. Emphasis is placed on disorders of motility, communication, and attention and perception.

Nowadays most investigators accept that childhood autism is an organic disorder of the central nervous system. The morbid anatomy, pathophysiology, and etiology are as yet unknown, but it is possible to make plausible hypotheses about them by applying standard methods of clinical inference. These hypotheses, in turn, provide a conceptual framework for understanding the disorder and for guiding further research. This communication is a description of the path we have followed in undertaking to make such hypotheses and apply them, and of what we have found along the way. It parallels the arguments of our original communication (Damasio & Maurer, 1978) and is an outline of our observations and in-

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ferences, supplemented by data from two studies that grew out of that work (Damasio, Maurer, Damasio, & Chiu, 1980; Vilensky, Damasio, & Maurer, 1981) and by some additional inferences and background material. For the details of our reasoning, for data from the studies, and for citations, the original articles should be consulted. Statements are referenced below, with a few exceptions, only if they are newly made here or if they draw upon previously uncited sources.

The task we initially set for ourselves was to determine the anatomical and etiological inferences that could be made from the abnormalities that we observed in our autistic patients or that were described in the literature on autism. Our method of reasoning was the classical clinical method (Feinstein, 1967) used in the everyday practice of neurology (Adams & Victor, 1977). We collected and categorized clinical data, and we designated these data by the names or descriptions of signs and symptoms in order to correlate them with similar signs and symptoms in better understood neurological disorders. By making analogies to disorders in which the underlying anatomy or physiology is known, and by reasoning from knowledge about the structure, function, and connections of various brain regions, we interpreted the signs and symptoms in terms of disturbed anatomy and physiology, and estimated the localization that could account for the data most parsimoniously. Finally, taking into consideration the hypothesized anatomic localization, the natural history of the disorder, and accessory data such as association with other medical conditions, we deduced possible causes for the syndrome.

The criteria we used for autism were those of Rutter (1975) and included (a) the characteristic triad (as redefined by Wing & Gould, 1979) of impairment in two-way social interaction, impairment in the comprehension and use of language, both verbal and nonverbal, and impairment of true, flexible, imaginative activities, with the substitution of a narrow range of repetitive, stereotyped pursuits; (b) onset before the age of 30 months; and (c) a chronic course. It became apparent, however, that autism the syndrome or symptom complex (i.e., the triad) needed to be distinguished from autism the disorder or disease entity (i.e., the triad occurring in the context of a specific onset and course), a point recently reinforced by case reports of typical autistic syndromes with atypical onset and course (DeLong, Bean, & Brown, 1981; Weir & Salisbury, 1980). The distinction between “syndrome,” which refers to a set of concurrent symptoms, and “disease,” which as a clinical concept refers to symptoms evolving in a correlated pattern over time, is an old and important one. This distinction has become blurred in psychiatry, where “syndrome” is used in both senses, but is still important in neurology, where the concurrence of symptoms (the immediate manifestations of the disorder) is often the key to anatomic localization. Symptoms may correlate because they share the same functional