

Autism and the Cerebellum:

A Neurophysiological Basis for Intervention

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During the last decade imaging and autopsy studies of people with autism have found abnormalities in the cerebellum and in other brain regions that are components of the cerebellar system^{1,2,3,4}. Particularly striking are findings that lobules VI and VII of the vermis, the central "trunk" of the cerebellum, are abnormal⁵. Whether these are real findings or are methodological artifacts still is in dispute^{6,7}, but even the possibility that this part of the brain is dysfunctional in autism leads down a tantalizing and useful trail. Vermal lobules VI and VII are the phylogenetically oldest part of the mammalian cerebellum⁸, and would be involved in the primitive behaviors that differentiate mammals from reptiles, from whom mammals evolved. These behaviors are nursing in conjunction with maternal care, audiovocal communication for maintaining maternal-offspring contact, and play^{9,10}. Disruption of these behaviors, from the child's side of the interactions, is the autistic triad.

The findings of abnormalities in the cerebellar system were surprising: no one expected autism to involve that part of the brain, which traditionally has been thought to be concerned only with the smooth and effective control of movement. Even studies of movement disorders in autism, such as those Antonio Damasio and I did^{11,12,13}, pointed toward involvement of a different part of the "movement" brain, the basal ganglia. Recently, however, a colleague of mine, Philip Teitelbaum, a physiological psychologist who for years used the Eshkol-Wachmann system of movement notation¹⁴ to study disintegration and reintegration of movement after brain damage in animals, began to use this system to reanalyse films from those old studies, and to analyse videotapes of children learning to walk who later turned out to be autistic. He finds that the coordination of movement across joints and the integration of composite movements, both functions of the cerebellum, are abnormal, and are abnormal at an early age. He also finds that movements of different parts of the body come from different developmental stages, as if development itself is uncoordinated, and that the odd movements and postures of children with autism often are appropriate to earlier stages of development, but are inappropriately preserved. For example, the limp, dystonic posture of one hand, that children with autism often have when they walk, is similar to the normal posture of the hands of an infant just learning to walk.

A characteristic feature of the cerebellum is the homogeneity of its neural architecture. Other, very different parts of the nervous system connect in complex ways to the different parts of it, but these parts all have the same regular, repeating microarchitecture, so that of all the structures in the brain its architecture most closely resembles that of a computer. This suggests that the role it plays is the same for each functional system of which it is a part, however different the functions of those systems are^{15,16}. But how could disruption of processes involved in the coordination and integration of movement fundamentally disrupt mammalian social behavior?

Some research done in the 1960s and 1970s is relevant to this question. Adam Kendon¹⁷, Daniel Stern¹⁸, William Condon^{19,20}, and others^{21,22} wanted to understand what happens in interactions, and did frame-by-frame microanalyses of filmed interactions. They found remarkable synchronies and symmetries among body movements and speech sounds, and found many of these present already in the interaction between mother and infant, which Stern likened to a waltz. He and others proposed that in autism this dance was disrupted, and Condon had film to show that it was. In addition, the Israeli ethologist Ilan Golani used the Eshkol-Wachmann notation to study the courting behavior of both a primitive species and an advanced species of mammal, found that courting couples of both species moved together, even when not in contact, as if they were connected by a joint, and concluded that interactional "joints" were a fundamental feature of mammalian interaction²³.

This research fell out of fashion, and the trails have not been followed further, but the phenomena are easy to see or hear when one watches or listens to people interacting, and looks or listens for the elements of a "dance" or, in the case of autism, a disrupted "dance". The fundamental role this "dance" plays in relationship and the extremely corrosive effects of its disruption also are easy to grasp, when one considers how uncomfortable one feels when one's partner in an interaction does not "dance", and how one automatically either pushes one's own rhythm, or falls into the rhythm of one's partner, if one wants to maintain the interaction. The cerebellum would have to be involved in coordinating this dance, and its role would be a natural extension of its role in coor-

dination of movement across joints and in integration of composite movements, by adding information about movements of the other to the information it has about movements of the self. Significantly, vermal lobules VI and VII and related paravermal regions are the parts of the cerebellum to which the auditory and visual senses connect²⁴, the senses the cerebellum would have to use to play this additional role.

Autism is more than just the autistic triad. As Temple Grandin²⁵ and other people with autism eloquently point out, the disorder includes significant sensory abnormalities as well. These used to be a focus of autism research, but this research also fell out of fashion. Any theory of autism has to explain them, however. Could disruption of cerebellar processes like those involved in the coordination and integration of movement also disrupt perceptual processing?

Interestingly, the cerebellum, long considered a motor structure because of its apparent function, is phylogenetically and embryologically a sensory structure. In all vertebrates it is more closely associated with sensory systems used for tracking movements of targets in the environment than it is with motor systems. It seems to be a tracking system that predicts sensory consequences of all types, and feeds the predictions to motor and other output systems. Dysfunction would lead to a specific type of deficit, the inability to correctly estimate the "trajectory" of a stimulus²⁶. When fed to the systems that modulate motor function these incorrect estimates would produce the characteristic undershoot and overshoot of cerebellar motor abnormalities. When fed to the systems that modulate other functions, such as sensation, they would produce an analogous "undershoot" and "overshoot" in the regulation of these systems, the principal feature of sensory abnormalities in autism. Again, significantly, vermal lobules VI and VII and related paravermal regions are the parts of the cerebellum to which the auditory and visual senses connect. They also are parts to which the other senses connect, and thus would be the parts involved in the modulation of one sense by one another, a type of regulation also abnormal in autism.

The neurodevelopmental approach to intervention in cerebral palsy is grounded in a comprehensive neurophysiological model of the dysfunction involved²⁷. Does the model outlined above similarly ground a neurodevelopmental approach to autism? There are indications that it does. A serious problem in the autism field, particularly evident to parents, has been the balkanization of interventions. A common theme in many useful interventions, across many different disciplines, is the use of environmental accommodations that provide a consistent pattern of sensory, motor, attentional, emotional, or some other type of modulation. This is true of sensory integration, the "squeeze machine", auditory training, the "Lovaas method", facilitated communication, and the Erlen lenses, to name a few of the more controversial interventions. Sometimes the use of such accommodations is an incidental aspect of the intervention, yet may be the actual active ingredient. All of these interventions compensate for one actual or potential type of cerebellar disability or another, and thus the theory of cerebellar dysfunction in autism may provide the comprehensive model we need to coordinate and integrate the interventions that different groups of us in the field have found to be useful.

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