

Language after Hemispherectomy

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We studied the spoken language of 49 children who had undergone hemispherectomy as part of the UCLA Pediatric Epilepsy Surgery Research Program and analyzed, among a number of clinical factors, the relation between acquired vs developmental pathology and spoken language outcomes. In this paper we will briefly review the results of our study and attempt to explain (1) why “the early” is not always better, (2) why so many *right* hemispherectomies fail to develop language, and (3) why some *left* hemispherectomized children develop remarkably good language despite removal of the “language” hemisphere. This account will rest on the proposed model of brain maturation and progressive lateralization.

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As a part of the UCLA Pediatric Epilepsy Surgery Research Program over 50 children have undergone hemispherectomy for catastrophic childhood epilepsy in the past few years (Peacock, Wehby-Grant et al. 1996). Several studies have shown that intractable seizures may be eliminated or reduced in children following hemispherectomy (removal of central cortical regions and disconnection of the frontal and occipital lobes). The primary goal of this drastic procedure is to save a child’s life and/or improve her quality of life (Shields, Duchowny et al. 1993; Wyllie, Comair et al. 1996). No firm criteria exist to predict which patients will benefit from the surgery and even less so in predicting the possibility of normal cognitive/linguistic development. However, regarding the latter, it has been conventionally assumed that (1) the earlier the surgical intervention the better due to neural plasticity and (2) the left hemisphere is dominant for language, therefore, a right hemispherectomy will be less damaging as far as language is concerned. Based on these assumptions we predicted that those children who underwent hemispherectomy very early in life would show better neuropsychological outcome due to functional reorganization, and that the right hemispherectomized children would have better chances of developing normal language. Both predictions were not fully confirmed, however, in our follow-up study of the linguistic status of 20 right and 30 left hemispherectomies.

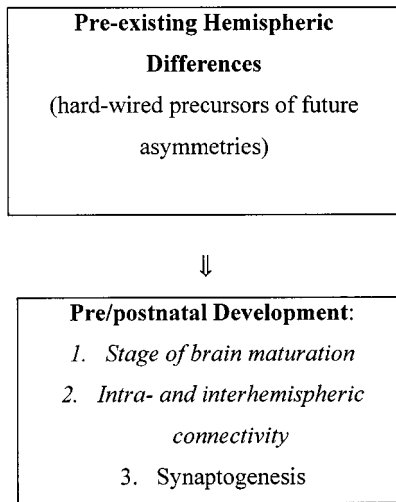


FIGURE 1

In this paper we will briefly review the results of our study and attempt to explain why (1) “the early” is not always better, (2) why so many *right* hemispherectomies fail to develop language, and (3) why some *left* hemispherectomized children develop remarkably good language despite removal of the “language” hemisphere. We propose an account of the neurobiology underlying language acquisition in which interhemispheric inhibition and axonal loss contribute critically to the process of language acquisition and lateralization in both clinical and normal populations. Our hypothesis rests on the following assumptions: first, each hemisphere has its own hardwired, predetermined program for functional specialization. Second, to instantiate this predetermined program as functional asymmetries, normal prenatal and postnatal neurobiological development is crucial (we listed just a few potentially relevant processes). We hypothesize that part of this normal development is establishing normal interhemispheric connectivity. Our account regarding the hemispherectomy findings, and more generally, regarding the neurobiology of language acquisition thus encompasses the assumptions presented in Fig. 1.

We will attempt to show how factors like etiology, time of the insult, and presence of epilepsy, can be factored into the proposed model and what seem like surprising findings in the literature discussing linguistic outcomes after hemispherectomy of either side become predicted, neurolinguistic findings.

For example, we will argue that “the early” is not always better because the functional consequences of cortical injury vary with the *developmental stage* at the time of injury (Kolb, 1990; Kolb and Wishaw, 1998). For example, we would expect a worse outcome as a consequence of an injury that

TABLE 1
Neurobiological Mechanism Leading to the Unique Cognitive Outcome in SWS

Neuropathology of hemi megalencephaly	Normal brain development	Neuropathology of Sturge Weber
Abnormal (much thicker cortex, neurons in the white matter result in the increased number of synapses); (O'Kusky, Akers et al., 1996; Robain & Gelot 1996)	<i>I. Intrahemispheric cortical organization</i>	Normal (despite vascular tumor formation of the two protective brain tissue layers)
Abnormal (due to a significant increase in the total number of synapses)	<i>II. Interhemispheric connectivity</i>	Normal
Does not occur	1. Establishing connections between homotopic areas of the two hemispheres (pre- and postnatally)	
	2. Postnatal axonal loss (fixes predetermined functional asymmetries in place), (Trevarthen 1990; Witelson & Nowakowsky 1991)	Does not occur due to the known effect of seizures on corpus callosum fibers (Grigonis & Murthy 1994)
	↓	
Aberrant connectivity prevents a healthy hemisphere from normal functioning even after resection	<i>Functional Asymmetries</i>	↓
		Functional asymmetries fail to get established but both hemispheres stay "on line" due to extensive and normal connectivity. Surgical resection releases inhibition and allows a remaining hemisphere to develop normally.

occurred during the end of the mitotic phase and neuronal migration (the third trimester of gestation and the part of the first year in humans). On the contrary, the most favorable outcome can be expected if cortical injury occurs during the period of maximal dendritic differentiation and synapse overproduction. We will show that although the factor of the brain developmental stage can be critical for acquired damage (stroke, trauma), it cannot be automatically applied to developmental disorders. For the latter, it is the phenom-

enon of *intra- and interhemispheric connectivity* that will play the most important role in the brain maturation and language development. Finally, all the factors will be analyzed within a bigger timeframe of developing functional asymmetries and progressive lateralization.

To illustrate our model we will use a variety of etiologies, for example, we will compare two different developmental diseases, megalencephaly and Sturge Weber and explain why the Sturge Weber syndrome allows for greater linguistic development than megalencephaly (Table 1).

In a similar vein we will attempt to predict linguistic outcomes after hemispherectomy following a number of different developmental and acquired insults.

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Priming Arithmetic Reasoning in an Amnesic Patient

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The present study elucidates the role of implicit memory in problem solving by evaluating priming effects in a severely amnesic patient. An arithmetic series com-