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Brain imaging studies of developmental stuttering☆

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Abstract

This paper reviews recent brain imaging research on stuttering against a background of studies that the writer and colleagues have been conducting at the University of Texas Health Science Center in San Antonio. The paper begins by reviewing some pertinent background to recent neuroimaging investigations of developmental stuttering. It then outlines the findings from four brain imaging studies that the San Antonio group has conducted using $H_2{}^{15}O$ positron emission tomography (PET). Finally, some of the principal findings that are emerging across brain imaging studies of stuttering are reviewed, while also highlighting — and attempting to resolve — some apparent across-study inconsistencies among the findings. Research on stuttering using magneto-encephalogaphy (MEG) and transcranial magnetic stimulation (TMS) is also considered. The findings increasingly point to a failure of normal temporal lobe activation during speech that may either contribute to (or is the result of) a breakdown in the sequencing of processing among premotor regions implicated in phonologic planning.

Learning outcomes: As a result of this activity, the participant will become familiar with some recent neurophysiological correlates of stuttering and what they suggest about the nature of this disorder.

Keywords: Stuttering; PET imaging; Neurological correlates

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1. Introduction

A brief comment on the origins of much of the San Antonio group's neuroimaging research on stuttering may help to understand the direction of the research program that colleagues and I have been following. This program actually emerged from some studies on the "chorus reading effect" that colleagues and I were conducting during the 1970s (Ingham & Carroll, 1977; Ingham & Packman, 1979). The chorus reading effect is the well-documented dramatic reduction in stuttering that occurs when an accompanist and a stuttering speaker read aloud the same material at the same time (Barber, 1939; Johnson & Rosen, 1937). While conducting these studies, it became apparent that the chorus reading effect had to rely on much more than just an induced speech-pattern change. During the 1970s — and even in recent years — this was the generally accepted explanation for the chorus reading effect (Wingate, 1969). Yet, every attempt to confirm that the effect is due to an induced speech pattern has essentially failed (Adams & Ramig, 1980; Ingham & Packman, 1979; Stager, Denman, & Ludlow, 1997; Stager & Ludlow, 1998). There is simply no compelling evidence that the effect depends on the speaker using an unusual speech pattern. It seemed to be the case then — and it still does — that whenever chorus reading is introduced and withdrawn, its remarkable effectiveness must be due to "something" that is literally switched "on" and "off" within the brain. However, it was not until the late 1980s that the then-evolving neuroimaging technologies made it possible to investigate the neurologic processing of that effect in stuttering speakers. Colleagues and I also speculated that imaging this effect might make it possible to identify concomitantly changing neural regions that might have functional control over stuttering.

2. Some relevant history

2.1. Cerebral Dominance Theory

The notion that chronic stuttering is due to an abnormal neurologic system is certainly not new; it has had a long and checkered history. Beginning with observations by Sam Orton and Lee Travis during the 1920s (Orton, 1927; Orton & Travis, 1929), this notion was then fostered by their well known Cerebral Dominance Theory (Travis, 1931, 1978). Actually, it was a theory of *failed* cerebral dominance, or nondominance, because it argued that stuttering was the direct consequence of a developmental failure to achieve lateral dominance of the speech centers. This theory waxed and waned in popularity, but somehow it survived because its central proposition — that stuttering is functionally related to failed lateralization — was never thoroughly repudiated.