Stuttering: dysfunction in a complex and dynamic system

Stuttering is a developmental speech disorder that usually appears between 3 and 8 years of age and often remits before puberty. When it persists past the close of the period of developmental plasticity, around puberty, it becomes a chronic adult speech disorder throughout the life span (Andrews et al., 1983). The emergence of brain imaging has increased interest in exploring the long-held concept that persons who stutter have greater right hemisphere involvement in speech production than fluent speakers (Travis, 1978). By contrasting fluency-enhancing speaking conditions with those exacerbating stuttering, brain imaging studies have found increased activation in premotor regions bilaterally and greater right hemisphere involvement during stuttering. Comparisons between fluency and stuttering conditions are not clear contrasts, however, because fluent speech occurs in both conditions and stuttering only occurs intermittently during the dysfluent condition. Therefore, Fox and colleagues (Fox et al., 2000) in this issue and Braun and colleagues previously (Braun et al., 1997) conducted correlation analyses between stuttering frequency and activation in different brain regions. Braun et al. measured stuttering during a sentence construction task, while Fox et al. used stuttering measures from rest, fluent and dysfluent speech tasks, and therefore had increased statistical power.

Both authors attempted to separate brain-performance relationships within the adult speech motor control system as either being associated with either stuttering or attempts to avoid or compensate for stuttering. Positive relationships with increased stuttering were used to identify particular regions of the speech system as associated with the state of stuttering. Braun et al. found significant blood flow increases in the putamen, ventral thalamus and inferior anterior cingulate, all on the left, with increased stuttering. Fox et al. found increased stuttering related with increased activation in the right premotor regions and the non-dominant left cerebellum. These differences cannot be accounted for by the differences in statistical power between the two studies.

Both authors found an inverse relationship with increased stuttering related to decreased activation in the primary auditory and association areas in the right hemisphere. The decreased activity in the auditory area during increased stuttering suggests that feedback of one’s own speech is suppressed with stuttering.

Fox et al. also examined brain activation relationships with increased syllable production in the stuttering group to determine which brain mechanisms might be compensatory by aiding subjects to avoid stuttering. They identified increases in the cerebellum and inhibition in the superior temporal area. Braun et al. found activation in some speech motor regions in the right hemisphere were inversely related to stuttering, and interpreted these as compensatory. Some of these same regions, however, also had significant and similar relationships with the frequency of stuttering in either of the two studies. Thus, activity in some regions was related to both stuttering and fluent speech in stuttering subjects.

Speech production involves a highly distributed system which includes the left insula for speech articulatory planning (Dronkers, 1996) and the bilateral motor control of vocal tract musculature in the two hemispheres. These must interrelate with other motor control systems including the supplementary motor area, the basal ganglia, thalamus (McClean et al., 1990) and cerebellum. This entire system is capable of precise timing of articulator movement to control air flow changes for rapid acoustic transients of a few milliseconds (Borden and Harris, 1984). Adults have attempted to compensate for stuttering disruptions in this system during fluent speech since childhood. Persons who stutter report that they continuously monitor their speech to prevent dysfluency. Stuttering and fluent speech probably employ the same functionally disturbed system within an individual. The greater cerebellar involvement and stronger relationships between frontal pre-central regions and syllable rate in the stuttering group may manifest this struggle as Fox et al. suggest. Brain activation patterns observed in stuttering adults are perhaps the result of individually adapted systems that evolved during childhood and early adolescence in an effort to produce fluent speech. Individually adapted systems, when combined for correlation analyses, may then lead to varying results across studies using different tasks and measures.

The limited temporal resolution of PET technology used in the Braun and Fox studies may have obscured dynamic interrelationships within and between brain regions important for the production of fluent speech. Magnetoencephalography (MEG) has been used to study fluent speech in adults who stutter and controls while reading single words (Salmelin...
et al., 2000). When a 400-ms time period between word presentation and a delay in motor execution was examined, predominant processing occurred in the left hemisphere in both stutterers and controls, but the pattern and timing of activation differed between groups. The sequence of left inferior region processing prior to left central processing found in the controls was reversed in the stuttering subjects before fluent production. The stuttering subjects activated the left central MI region for motor execution before the left inferior region for articulation programming. These subjects’ abnormalities within the left hemisphere indicate that the dynamic relationships may be functionally different in adults who stutter even during fluent speech.

Several authors have found a reduction in auditory region activation in the stuttering group. Fox et al. have interpreted this as being an abnormal inhibition of auditory feedback. However, during oral reading, Numminen and colleagues found MEG responses to auditory stimuli are normally suppressed in control subjects by 44–71% in comparison with responses during silent reading (Numminen et al., 1999). In a similar study of subjects who stuttered, responses to auditory stimuli during silent reading were less suppressed on the left and more suppressed on the right than those of the controls (Salmelin et al., 1998). Surprisingly, the greater auditory suppression on the right and reduced suppression on the left were normalized when the subjects were stuttering. Dynamic abnormalities are present, then, in both the left speech motor areas and the right auditory areas even when subjects who stutter are fluent or not speaking.

In conclusion, the findings of Fox and colleagues (Fox et al., 2000) and others studying adults who stutter provide intriguing new information about the dynamic interplay among complex cortical and sub-cortical systems involved in the planning, production and monitoring of speech expression. The results indicate a variety of complex dysfunctional systems. In the adult system it may be difficult to distinguish between mechanisms responsible for stuttering and those developed to compensate. It is likely that the dysfunction is not one of a simple laterality difference as was suggested many years ago (Travis, 1978). As new technologies emerge which are non-invasive and have improved temporal resolution, studies in children who stutter during the critical period for speech development may provide understanding of how this dysfunctional system emerges.

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References