Matters arising

The response of the apparent receptive speech disorder of Parkinsonism to speech therapy

Sir: In two studies,^{1,2} Scott and Caird argue for a sensory speech disorder involving prosody in apparent idiopathic Parkinson's Disease. Although sensory findings are not uncommon in either extrapyramidal or pyramidal illness, it is misleading to isolate prosody and suggest that "abnormality of prosody dominates the disorder of speech and often gives a false impression of dysarthria" (p 840).²

A widely accepted definition of dysarthria has been the one set forth by Darley, Aronson, and Brown based in part on their classic research.³⁻⁵ Specifically, dysarthria is defined as a group of speech disorders resulting from disturbances in muscular control due to damage to the central or peripheral nervous system and characterised by weakness, slowness, incoordination or altered muscle tone involving the speech mechanism. The term implies and their research suggests that one or more of the substrates of motor speech including respiration, phonation, articulation, resonance, and prosody may be involved.

In general, it is agreed upon that prosody is manifest as patterns of acoustic intensity, vocal fundamental frequency, duration and timing of vowels and consonants. In this vein, Darley, *et al*'s original work^{4,5} as well as the work of others⁶ indicates that prosodic aberrations, particularly reduced stress, inappropriate silences, short rushes of speech, and variable to increased rate are *integral components* of Parkinsonian dysarthria and are considered representative of altered motor control of the speech mechanism. The finding that Scott *et al*'s² patients could not verbally demonstrate prosodic changes representing anger, questions, or statements would appear likewise to be consistent with a deficit in motor control rather than a sensory abnormality. Further, it is difficult to conceive of a motor disorder in articulation (dysarthria in the historical sense) that would not influence prosody as well. For example, the acoustic intensity variations underlying prosody are implemented both by the respiratory system via manipulation of subglottal air pressure⁷ and tongue-jaw movements^{8,9} (that is, degree of oral opening). Similarly, intonation (vocal fundamental frequency) changes are accomplished by extrinsic and intrinsic laryngeal muscle actions¹⁰ but also

are influenced by tongue movements (for example high tongue vowels have a greater vocal pitch than low tongue vowels).¹¹ Given these considerations, it is difficult to argue that a motor disorder even with involvement confined to the jaw and tongue would not manifest disturbances to prosody as well as in articulation.

Patients with Parkinson's disease may indeed have prosodic problems independent of a co-occurring dysarthria. Whether these deficits can be ascribed to focal involvement of the neuraxis, for example the right hemisphere as suggested in other research¹² or, diffuse/multifocal involvement as frequently occurs in Parkinson's disease, requires further investigation.

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Scott *et al* reply

As we clearly stated,¹ we have used the widely accepted definition of prosody by Monrad Krohn² and Crystal.³ The disagreement with Hartman and Abbs is that largely trans-Atlantic and semantic. One reason for emphasising the importance of prosody in Parkinson's disease was to focus attention on a treatment method, which Hartman and Abbs do not deny is effective.

We agree that tests of production angry and questioning tones are not evidence of a possible sensory abnormality but suggest that the failure to match facial expression and prosody is. Our paper deliberately entitled "Evidence for an apparent sensory speech disorder in Parkinson's disease" because that is all that has been shown. Further work is obviously necessary to confirm or deny the reality and significance of our findings.

References

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Lithium-induced improvement myotonia: relevance of prostaglandin blockade by lithium

Sir: Gerst *et al*¹ recently reported lithium-induced improvement of myotonia. Although they proffered no concrete explanation for this phenomenon, they suggested that lithium ions may affect the kinetics of the sodium channel in skeletal muscle. I would like to suggest that Prostaglandin E1 blockade by lithium might be involved.