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 thus 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 of 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 was 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.

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Lithium-induced improvement myotonia: relevance of prostaglandin E1 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.

Mitochondrial calcium overload has been suggested as the general mechanism for cell necrosis in muscle disease.² There is excess calcium entry into the cell as well as a failure of calcium removal. Prostaglandin E1 is well known for its bell-shaped dose response curve with very low and very high levels having the same effect.³ Very high levels of Prostaglandin E1 can inhibit Thromboxane A2 (TXA2) activity which is involved in both calcium release and calcium removal. A substantial overproduction of Prostaglandin E1 has been proposed to exist in myotonic dystrophy as it explains the presence in myotonia of weakness, reproductive abnormalities, reduced glucose tolerance, increased metabolic effect of growth hormone, and anatomical motor end plate abnormalities.⁴ Finally, lithium inhibits the production of Prostaglandin E1 by an opiate-like effect and has, in fact, been suggested by Horrobin⁴ to improve the clinical state in myotonic dystrophy.

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Brumback and Gerst reply

We appreciate the comments by Dr Backon. Although it is attractive to hypothesise that lithium-mediated changes in prostaglandins modified the myotonia, we suggest that a lithium effect on other "second messengers" such as adenine and guanine nucleotides^{1,2} might also alter myotonic phenomena. Prostaglandins apparently do have a role in calcium regulation, and calcium overload has been proposed as a mechanism of muscle fiber destruction in certain neuromuscular diseases;³ however, necrosis is not a feature of myotonia congenita (the disease in our

reported case), and atrophy and myofibrillar network disturbances are the major histologic changes in myotonic dystrophy.^{4,5} Also, in myotonia congenita the disturbance is confined to muscle, while myotonic dystrophy is a multisystem disorder. The 20,25-diazacholesterol model of myotonia appears to be more analogous to myotonia congenita than to myotonic dystrophy.⁶

We have previously postulated that the disturbance underlying myotonic dystrophy is abnormal function of cellular aminergic and peptidergic receptors.⁷ We originally proposed an intrinsic defect in the receptor mechanism itself, but it is equally possible that abnormality in a second messenger system could explain the symptomatology of myotonic dystrophy. Additionally, we have found that patients with myotonic dystrophy manifest significant neuropsychiatric impairment due to chronic depression (affective disorder) that is responsive to tricyclic antidepressant pharmacotherapy.⁸⁻¹⁰ Lithium pharmacotherapy has been shown to be beneficial in many patients with primary unipolar depressive illness,¹¹ and it is possible that the depression associated with myotonic dystrophy might also respond to lithium administration.

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