Matters arising

The “pacing board” in selected speech disorders of Parkinson’s disease

Sir: Critchley1 and Downie et al2 have recently discussed the severe communication disturbances present in some patients with Parkinson’s disease. Because these difficulties are particularly resistant to traditional speech therapy, the latter authors suggested the use of a delayed auditory feedback apparatus in the management of selected cases. We would like to draw to the attention of physicians caring for patients with Parkinsonism a simple and inexpensive alternative form of therapy which can result in striking improvements. The “pacing board”3 is an easily portable wooden or plastic board segmented into eight sections by raised dividers. The patient moves a finger along the board from one segment to another pronouncing one syllable per segment. The usefulness of this device has been previously reported only in palilalia.4 We would suggest that patients with additional severe communication disorders also may benefit as exemplified by the following case.

This 53-year-old man had suffered from Parkinson’s disease since the age of 37. He did not have a history of encephalitis and had never experienced oculogyric crises. He had undergone a right thalamotomy in 1965 with some improvement and had been treated with levodopa preparations since 1969 and had also undergone unsuccessful trials of bromocriptine. Over several years he had experienced frequent fluctuations in his clinical state throughout the day. In the few months prior to admission, he estimated that he was normal 10% of the day, “on” with dyskinesias 40% of the time and akinetic 50% of the time when he was usually chair or bed bound. During both phases of “on” and “off,” he experienced severe communication difficulties. While “on” he had very rapid, festinating speech and palilalia. Communication was most markedly impaired by frequent hesitations lasting an average of 6 seconds with sound and word repetitions causing a reduction in his rate of speech to 30% of normal values.5 When “off” voice amplitude was reduced and he was often unable to do more than repeat a single syllable over and over again. Much of his speech was unintelligible independent of which clinical phase he was in. The addition of pergolide mesylate6 2-2 mg per day in divided doses resulted in a marked reduction in his akinetic periods. However, speech continued to be a major problem. Using the “pacing board” to enforce syllabic speech, during the “on” phases rate of coherent speech increased by 63% and all dysfluencies were virtually eliminated. However, during the “off” phases the patient was often too akinetic to use the board effectively.

The similarities between disordered speech and gait in Parkinson’s disease have been recognised for many years. Critchley6 discussed the similarity in response of locomotion and speech disturbances to external stimuli. It was because of these similarities and the response of walking difficulties to visual cues such as stairs, or lines painted across the floor that Helm3 designed the “pacing board”. She reported its use in a single patient with palilalia. Our experience suggests that other Parkinsonian speech disorders also may benefit from the technique; however, it is likely that only a select group of patients will respond and we are in the process of defining the limits of its usefulness. Because of the simple and inexpensive nature of this device, we would recommend a trial of the “pacing board” before turning to more complicated and costly delayed auditory feedback speech aid used by Downie et al.2

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2 Downie AW, Low JN, Lindsay DD. Speech disorder in Parkinsonism; Use of delayed auditory feedback in selected cases. J Neurol Neurosurg Psychiatry 1981;44:852.
agreement among well qualified neurologists about the presence or absence of a certain sign in a particular patient, and some of the "focal signs" referred to in these patients could have been seen differently by another colleague. I would like to bring a study reported in this journal to Dr Weisberg's attention about the unreliability of our interpretation of the most precious of the neurological signs, that of Babinski. Radiologists interpreting CT scans are not infallible, nor is the technique itself. It certainly is an objective test, and leaves much less room for arguments among interpreters than the great variety of signs encountered during neurological examination among clinicians.

His second conclusion is even more controversial. He quotes two reports from pathology journals about the high occurrence of subclinical pituitary adenomas and incidental meningiomas. CT scan would probably not detect—at least not at this stage—the microscopic pituitary adenomas, and with the newer neurosurgical techniques even those are treated. As for meningiomas I believe all "incidental" cases in accessible location should be removed. A recent study presented at the 1982 meeting of the Society of British Neurological Surgeons shows that a high percentage of meningiomas become symptomatic if left untreated, and mortality from surgery increases with age. It would be difficult not to conclude that surgery at an early stage is highly recommended. It is probably true that early detection of malignant tumours does not influence the eventual outcome, but is there anything that does? At the time of ordering a scan we are more interested in finding lesions that are curable and really need early detection, than finding an incurable malignant tumour. The same applies to cerebrovascular disease; the clinical picture might be quite typical for an ischaemic process, and haemorrhage or other non-vascular abnormality is found on the CT scan. Three of the patients in his study had chronic subdural haematoma. The report he quotes about non-surgical treatment of chronic subdural haematomas was not well received by neurosurgeons. I wonder if Dr Weisburg would ever think about not referring his patients with a chronic subdural haematoma—especially the more recent cases—to a neurosurgeon for evacuation of the clot. If they are to be treated, they are to be found. They can be clinically very deceiving, and CT scan has been a great help to me in finding the clinically borderline or unsuspected cases.

It was around the time Dr Weisberg's publication was reported that I received the report on my third patient with colloid cyst of the third ventricle in 18 months and my second patient with a meningioma in six months—all without or with questionable clinical manifestations. I had a lot more reports on "incidental" abnormalities, but these five cases made me wonder how many people in my community are walking around with similar treatable and—in the case of colloid cysts—potentially lethal lesions. I am also deeply disturbed by my patients with subarachnoid haemorrhage from ruptured aneurysms with its devastating personal and social effects on people in their prime years. The risks and costs of angiography are too high to detect them before bleeding occurs, and I am very hopeful that the day is not far when the sensitivity of CT scan will reach the point that even small aneurysms will be detected with no risk to the patients, and enable us to operate on them before they rupture.

Dr Weisberg's study is based on retrospective analysis of patients referred for CT scan, and this is a step in the right direction, even if our opinions about the conclusions are so different. If we are to resolve the question of who should be referred for CT scan and with what indications, a minimum number of studies is needed: (1) to determine the frequency of incidental CT abnormalities in a well defined neurological patient population and (2) to do the same in a sufficiently high number of healthy people. My anticipation is that unexpected correctible CT abnormalities are frequent enough to warrant liberal use of this test in our patients, and I am hopeful that in the reasonably near future it will be used as a highly reliable screening procedure without specific indications for the general population as well. For the time being I do not think we should put restrictions on who is and who is not allowed to ask for a CT scan. During my years as a consultant neurologist it has not been my impression that non-neurologists would have had a great deal of difficulty recognising basic neurological syndromes and initiating the appropriate investigation.

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References

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Weisberg replies:

Sir: Dr Suranyi raises many complex and provocative issues. The difference in our opinions is due to a deep philosophical schism. I welcome the opportunity to respond to the general philosophical differences; however, a point for discussion would require more space and would be more than I an allowed in this letter. Our major point of difference is that I do not believe that CT should be used as a screening procedure. I believe that clinical neurological practice should remain an art and it should be the responsibility of neurologists to teach our colleagues, for example internists, general practitioners, psychiatrists, family practitioners, paediatricians, how they may be well versed in the art of neurological history taking and examination. To become a "Picasso" in this art, physicians without formal training in neurodiagnostic skills need to interact with their colleagues who do have training in neurodiagnosis so as to learn how well they can approach most patients with neurological disease. If nonneurologically trained physicians cannot approach common neurological conditions with confidence, neurologists have failed in a primary function, that is, education. However, there are multiple textbooks which are well written and concise whose purpose is education of the non-neurologist in the approach to the patient with neurological disease. Two books are mentioned because I use them, however there are at least 10 other good books.

The importance of the clinical neurological examination has been stressed by Dr Oldendorf. The clinical assessment of a patient by a neurologist requires 30 to 60 minutes and the physician is mobile, that is,