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Short report

Neurological stuttering—a clinical entity?

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SUMMARY Stuttering associated with neurological pathology in normal adult speakers is uncommon, has no consistent clinicopathological picture, and its significance is too easily dismissed. A case is reported showing that stuttering may be a presenting symptom of progressive neurological disease, and another case demonstrates that a speech disorder which is indistinguishable from common stuttering may follow cerebral injury in adulthood.

Recent evidence that genetic factors are important in the aetiology of typical stuttering (Andrews and Harris, 1964; Howie, 1975) suggests that a neurological abnormality may underlie this common speech disorder, although the detailed aetiology is still unknown. The rarer syndrome of stuttering after neurological pathology in previously normal speaking adults is not universally accepted as a clinical entity. Perhaps this is because relevant case reports are frequently anecdotal in nature, do not present a consistent clinicopathological picture, and are so rare that they may represent merely a predictable background rate of eccentric case reporting.

We know of six cases in which apparently permanent stuttering was either a presenting symptom or a complication of neurological pathology in adults. In two cases the stuttering preceded the eventual diagnosis of presenile dementia, whereas it followed the onset of neurological lesions in two cases of stroke, one of thalamotomy, and one of head injury. Two of these cases are described in some detail.

Case 1

STUTTERING AFTER HEAD INJURY

This 30 year old man was intensively investigated in an attempt to clarify the nature of his speech disorder and the associated neurological deficit. There was little doubt that the subject's speech had once been stutter-free since, quite fortuitously, he was looked after by one of the present authors (GA) both before and after the onset of stuttering. There was a clear history of stutter-free speech in childhood, and there was no family history of stuttering.

After a high-speed motorcycle accident in which he was thrown headfirst into a speedway retaining wall, he was admitted to hospital comatose with multiple fractures of the upper limbs. Burrholes revealed considerable extradural clot extending over the left frontal lobe. During his slow rehabilitation it was noted in his medical records that he was disorientated in time and space and had post-traumatic amnesia for more than two months, as well as expressive dysphasia and a tendency to perseverate in speech. He had left amblyopia, left optic atrophy, right temporal hemianopia, and nystagmus with a fast component to the right. His pupils responded directly and consensually to light directed to the right eye but not to the left. He showed behavioural problems characterised by occasional aggression, and was easily distracted. At six months his speech was nearly normal although he tended to perseverate when tired.

Six years after the accident the patient presented for treatment of stuttering, which he had first noticed four years earlier. It had become progressively worse. Speech pathologists diagnosed his speech disorder as stuttering, characterised more by repetitions similar to those of childhood stutterers than by the hesitations and mannerisms of adult stutterers.

On examination he was stuttering on 20% of his syllables and his speech rate was reduced to 108 syllables per minute. His stuttering showed the adaptation effect (Index=68) and consistency effect (scores=3.06, 3.88, 3.19) described by Johnson *et al.* (1963) as characteristic of typical stuttering.

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His intellectual and language function had, in six years since the accident, returned to virtually normal levels. His WAIS intelligence score had improved by 24 points to 110 (Verbal 103, Performance 118), and aphasia was now only evident in weakness and concrete responses in the verbal expression subtest of the Illinois Test of Psycholinguistic Abilities.

This subject was included in an investigation of cerebral dominance in stutterers using the Wada intracarotid sodium amylobarbitone technique (Andrews *et al.*, 1972). This technique usually produces marked dysphasia after dominant hemisphere injection, although the minority of left handers who showed bilateral speech representation also demonstrate unusually mild dysphasia (Milner *et al.*, 1966). The present subject not only was unable to sustain speech after the injection of either hemisphere, but also experienced severe dysphasia. These findings suggested a post-traumatic inter-hemispheric redistribution of language function which may represent a process of division rather than of reduplication.

Dichotic listening tests are widely employed in the investigation of cerebral dominance. There is some evidence that those subjects with left hemispheric lesions who show unusually powerful 'paradoxical' right ear superiority on dichotic testing may have lesions of a trans-callosal pathway which, under dichotic conditions, transfers auditory information from the right hemisphere to the left (Sparks *et al.*, 1970). The scores of the present subject on a dichotic test used in this laboratory (Quinn, 1972) were comparable with those of the above individuals. His scores were: left ear 2; right ear 30. Six months later, his scores were: left ear 4; right ear 27.

The evidence for bilateral representation of language in the present subject, however, confounds the model of Sparks *et al.*, which presupposes unilateral left hemispheric language output. While this subject's left hemisphere may be more receptive of dichotically dispersed auditory information than the right, alternative explanations for the poor report of left ear words, in a subject with proven left hemispheric injury, would include the possibility of additional neurological lesions.

Case 2

PRESENILE DEMENTIA PRESENTING WITH STUTTERING

A 62 year old successful businessman presented because of the recurrence of a stutter that had afflicted him transiently during his childhood. No neurological or psychiatric abnormalities were noted at this time, although in retrospect it is evident that his business acumen was failing and uncharacteristic errors of judgement were being made.

Seven months later his stutter had become so severe that he was again referred for treatment. When assessed he was stuttering on 48% of his syllables, and his speech rate was reduced to 31 syllables per minute. The stutter was unusual in that there were multiple simple repetitions on each affected syllable as is common in childhood stuttering and similar to that of the first case. In contrast with the latter, the stutter was not improved when the subject attempted to speak under a masking tone, with delayed auditory feedback or with prolonged speech. By this time there was evidence of an early dementing process, with mild expressive aphasia, poor concentration, and impaired judgement. Six months later the stutter was still present but was overshadowed by the impaired comprehension and grossly impaired intellectual performance. Evidence that his father and sister had died from a similar rapidly progressive presenile dementia was also obtained.

The most probable diagnosis for this man and his relatives appears to be Alzheimer's disease. Stuttering, similar in form to his earlier childhood stutter, was presumably the reiterative utterance of parts of words which is so characteristic of Alzheimer's disease. The condition continued to be progressive and this man died 18 months after the appearance of the first symptom. Necropsy was not performed.

Discussion

Lately there have been attempts (Canter, 1971) to establish a clinical syndrome of stuttering associated with neurological insult in previously normal speakers. The present report provides clinical and investigatory evidence to support such a concept.

We have seen a steady trickle of adults who would fit this syndrome. Our experience is consistent with impressions derived from the literature that neurological stuttering is uncommon, has no consistent clinicopathological picture, and is too easily dismissed when it does occur. Since the first two characteristics do not render a clinical entity invalid, and in view of evidence concerning speech details in our first case, we would suggest that a speech disorder which is indistinguishable from common stuttering may be evidence of specific neurological disorder.

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