SPASMODIC DYSPHONIA: EVIDENCE CONCERNING A NEUROLOGICAL CAUSE

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The term spasmodic dysphonia is a recent one, suggested by Aronson et al. (1968b), as a replacement for the term spastic dysphonia. The new term was advocated because "the term **spastic** is commonly used to imply disease of . . . pyramidal pathways", while the neurological evidence in the Aronson research concerned the extrapyramidal system. Further, Aronson et al. observed that "the strained voice quality in this disorder waxes and wanes from moment to moment in a spasmodic fashion". Spasmodic dysphonia, then, seems an appropriate descriptive term. Since pyramidal involvement appears to be minimal, and since even extrapyramidal involvement in the disorder is not widely acknowledged, the new term for the disorder may well enjoy widespread use in future.

Critchley (1939) described the voice of spasmodic dysphonia as "harsh and constrained, the pitch at times unduly low and at other times too high" (Aronson et al, 1964). Further characteristics of this disorder, sometimes called laryngeal stuttering, were seen as an interruption of the word sequence, uncontrolled loudness, tremor, monotone, excessive movements of the facial expression muscles, tic-like contractions, and a forced character in the speech.

Three subtypes of spasmodic dysphonia have been isolated by Kiml (1963). His classification is based on whether the abnormal laryngeal movements concern the intrinsic muscles of the larynx, or both the intrinsic and extrinsix muscles. This classification, however, has not been useful in establishing the cause of the disorder; nor has it yet been of any use in determining appropriate treatment for each of the subgroups, since no effective treatment has been found for spasmodic dysphonia in general (Boone, 1971; Aronson et al., 1968b).

There is considerable doubt regarding the origin of the disorder, with some authorities viewing the problem as psychological, and others suspecting a central nervous system cause. Boone (1971) reported that "most voice clinicians appears to view spastic dysphonia as a symptom of personality maladjustment and not as a disorder originating in the central nervous system." However, Boone specified neither the population of voice clinicians whose reports contributed to this statistic, nor the basis for their opinions, which might have been informal clinical observations rather than systematic research. Aronson et al. (1968a) reported that a review of the literature showed the consensus that spasmodic dysphonia "is a vocal expression of psychoneurosis", and that it is usually classified as a conversion reaction.

Evidence for a psychological cause appears to be based largely on observation, rather than research. Boone (1971) cited no studies in support of his statement that "the onset of spastic dysphonia is relatively abrupt and frequently follows an unpleasant, traumatic event", nor for his statement that the spastic dysphonic patient "usually can isolate some situations in which he enjoys a normal voice". Both of these statements can be questioned on the basis of the study involving 34 subjects reported by Aronson et al. (1968a, 1968b). Boone further observed that such patients "give their vocal and personal histories with a rather fixed smile"; this statement appears to be offered in support of a psychological basis for the disorder. While it is recognized that clear psychological or psychiatric characteristics may be difficult to pinpoint, it is also clear that more than casual, undocumented statements are needed to provide evidence for a psychogenic basis of the disorder.

Aronson et al. (1968a, 1968b) have provided the most comprehensive research to date concerning spasmodic dysphonia. They attempted to describe the quality variations and severity range, and to study both the neurologic and psychiatric characteristics of the disorder. Their subjects were 34 patients who had been diagnosed as having spastic dysphonia on the basis of "the presence of intermittent . . . strained hoarseness or complete momentary vocal arrest" (1968a). Patients known to have related neurologic or general medical diseases were excluded from the study.

Aronson et al. (1968a) first isolated ten commonly occurring voval factors observed among their subjects. These included the intermittent voice stoppages, both irregular and regular, by which the subjects had originally been diagnosed. Other voice factors which occurred in more than 70% of their subjects were excessively low pitch, monopitch, harshness, intermittent strain-strangle dysphonia, and voice tremor. Several listeners rated the severity of each subject's voice along each of the ten voice dimensions.

The second aspect of the Aronson et al. (1968a, 1968b) research, the investigation of neurological characteristics via direct neurological examination of the subjects, suffered somewhat in that there was no control group. The result,

that 74% of the 27 subjects examined showed neurological abnormalities, is potentially significant. The surprisingly high incidence of voice tremor in their subjects led the researchers to compare spasmodic dysphonia with the essential tremor syndrome, which is characterized by voice tremor, and with other neurologic syndromes, as discussed below. Two other neurological measures, EEG readings and a review of the subjects' general medical symptoms, revealed no findings that "appeared to have any logical organic bearing on the spastic dysphonia" (1968a).

The severity and incidence ratings for the ten voice factors provided a means of comparing spasmodic dysphonia with essential voice tremor, five other neurologic disorders, and with the psychogenic disorder of functional aphonia. The comparison made by Aronson et al. (1968b) revealed that spasmodic dysphonia is more similar to essential tremor than to the other five neurologic diseases investigated. Differences between spasmodic dysphonia and essential tremor were significant, however, on six of the ten voice factors. It was further pointed out those voice and articulation factors that can differentiate spasmodic dysphonia from each of the six recognized neurological diseases. These research results can be extremely valuable in diagnosis, since occasionally the only sign of early essential tremor, pseudobulbar palsy, or amyotrophic lateral sclerosis is a voice disorder. The wide range of valuable results reported in this study is in clear contrast to the undocumented reports of characteristics such as Boone's "fixed smile" observation, which masquerade as evidence for a psychological etiology of the disorder.

The third aim of the Aronson (1968a) study was to investigate the psychiatric characteristics of the spastic dysphonia subjects. This aspect of the research on spasmodic dysphonia is of particular interest, since a similar psychiatric evaluation had been carried out by the same researchers for a group of subjects with functional aphonia (Aronson et al., 1964). The previous study had consistently revealed a distinct precipitating emotional conflict or trauma among the functionally aphonic patients. In contrast, the less sharply defined emotional conflicts of the spastic dysphonic group "had no particular temporal relationship to the onset of the spastic dysphonia" (1968a). Further, many of the 34 spastic dysphonia subjects were stable and well-adjusted, in contrast to the aphonic subjects, all of whom showed instability and "the unconscious utilization of their voice symptom to attempt to deal with an unsatisfactory life situation" (1968a), as determined by the psychiatric interview. In summary, then, there was no psychiatric evidence relating directly to spasmodic dysphonia.

Aronson et al. (1968b) were not satisfied with the results of their psychiatric investigation of spasmodic dysphonia. They recommended further psychiatric and psychological studies, particularly long-term ones.

Aronson et al. concluded tentatively that "the syndrome of spastic dysphonia is predominantly a result of central nervous system dysfunction, and

that psychiatric factors may be viewed as complicating rather than primary" (1968a). They subsequently qualified even this tentative statement by proposing that those subjects who show both neurological and psychiatric signs may have an unstable motor system predisposing them to spasmodic dysphonia, which is manifested when life events precipitate the symptoms. Thus, a single cause for spasmodic dysphonia was not demonstrated, although there was evidence of probable neurologic cause in half the subjects, and of probable psychiatric cause in one-fifth.

Another interesting study which adds support to the theory of neurogenic dysfunction in spasmodic dysphonia was reported by McCall (1973). He measured the acoustic impedance in 14 patients with a diagnosis of spasmodic dysphonia. He found evidence of middle ear dysfunction in all 14 subjects, with three patterns of abnormal response to the acoustic reflex noted. In one group (2 Ss), the contraction of the middle ear muscles was not sustained throughout the 30-second sound stimulation. The second group (7 Ss) showed a prolonged time in the relaxation of the muscles after the stimulation had ceased. The third group (5 Ss) all exhibited tremor in the bulbar innervated muscles. Their impedance measurements showed corresponding shifts in middle ear impedance under baseline conditions (no sound stimulation).

In devising the study, McCall (1973) made use of the known connection between middle ear and laryngeal muscle function during vocalization. Consequently, he was able to introduce a new concept into the small body of research studies involving spasmodic dysphonia. In an earlier study by Robe et al. (1960), audiometric testing of the subjects had been used, but this consisted only of pure tone testing and, for some of the subjects, tests for central dysfunction. The acoustic reflex had not been investigated.

McCall reported abnormalities of middle ear function in all 14 of his subjects. He did not specify how these subjects were chosen for the study, except to say that no subject had a hearing complaint. An additional statement, however, that the subjects were chosen randomly, or at least independently of their acoustic reflex responses, would have been reassuring. Such a statement is particularly desirable considering that abnormal results were obtained for all of the subjects. Secondly, McCall did not use the same instrumentation for testing all 14 subjects; nor did he explain why this was the case. A third problem with the study was the fact that there was no control group of subjects, despite McCall's own statement that "adequate normative data concerned with the on-time, steady rate, and off-time characteristics of the acoustic reflex response have not yet been reported in the literature". The range of normal off-time characteristics is particularly important, since half of McCall's subjects showed a normal response except for a "prolonged" return to baseline, which might prove not to be prolonged when adequate normative data are considered.

McCall (1973) concluded that acoustic impedance measurements should be used in the evaluation of spasmodic dysphonic patients. This might in fact be a reasonable step, once a large body of normative data has been established. In order that the procedure might have any use in differential diagnosis, corresponding acoustic impedance measurements must also be made for groups of subjects with various psychogenic and neurogenic disorders. Further, the study as it stands is not useful in determining the treatment required for the patient. Its biggest contribution is in taking one more step toward solving the riddle of the etiology of spasmodic dysphonia. If the study can be replicated, and if normative data do not destroy the results, then a new neurological sign, middle ear dysfunction, can be added to the common neurological signs of spasmodic dysphonia, such as voice tremor, which Aronson has discussed.

Successful therapy techniques can often be developed for disorders of speech and language, even if the disorder itself is not well understood. Unfortunately, this has not been the case with spasmodic dysphonia. Boone (1971) reported that spasmodic dysphonia is highly resistant to both psychotherapy and voice therapy techniques. He stated that behavioral modification techniques, of incrementally extending good phonation such as a laugh, have been unsuccessful, as has the hierarchical analysis technique. Virtually **no** patient has experienced a permanent restoration of normal voice, according to Boone. Aronson et al. (1968b) expressed a similar view concerning the general failure of voice therapy in treating spasmodic dysphonia, but stated that "we have no data on the effects of psychotherapy." They advocated further research in both areas of therapy.

Thus, much research remains to be done to investigate spasmodic dysphonia. Additional neurologic and psychiatric measures are needed, and must be applied to large groups of subjects. Long term studies may prove useful. It seems that such research is necessary, not just in order to establish the etiology of the disorder, but also in order that the possibility of developing effective therapy techniques might be established.

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