Spasmodic Dysphonia: Case Studies of Three Patients

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Introduction

Aronson(1) reported in 1973 that there are two types of spasmodic dysphonia: the adductor type and abductor type. From a symptomatic viewpoint, the physiology of the vocal cords in case of abductor spasmodic dysphonia (hereafter referred to as 'abductor S.D.') is completely opposite to that in case of adductor spasmodic dysphonia ('adductor S.D.). Aronson defined the abductor S.D. as the 'abrupt breathy release of air owing to a sudden hyper abduction of the true vocal folds. Although the abductor type arises far less frequently than the adductor type(2). We have observed three cases of what seemed to be Aronson's abductor S.D. This is a report on their clinical characteristics, the results of voice functional tests and the findings of an acoustic analysis.

Test Method

At first, each patient's history was taken and then minute observations were made with a laryngofiberscope and a storoboscope. Maximum phonation time (MPT), vocal range and speaking fundamental frequency were investigated next. A phonolaryngograph (SH-01 Rion) was used to examine the flow rate, intensity and pitch at comfortable phonation. Recording was made using a microphone (SONY Electret condenser microphone IMP 600 ohm) and a digital audio tape deck (SONY DTC-100ES). At the same time an EGG was checked with a laryngograph (KAY Corp.) and recorded on a digital audio tape (DAT). The tape used was DT 60R (SONY). The voices recorded on DAT were analyzed with an analysis program developed at the Research Institute of Logopedics and Phoniatrics, School of Medicine, Tokyo University, in order to produce a narrow band sound spectrograph. The recorded voices were also used to analyze the pitch and intensity of the running speech with Visi Pitch 6095/6097 (KAY Corp).

Case 1

This patient was a 23-year-old female. She had a high pitched voice and sang normally in a chorus group when she was in elementary school. She started to experience momentary breathy voice suddenly at the age of 14 when she was in the eighth grade. Gradually, the occurrence of this intermittent breathy voice increased in frequency. The

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patient consulted a local medical doctor, who was not able to find any cause. She rarely had the voice disorder in talking with her family members or her friends, and the symptom did not bother her until she graduated from a vocational school which she attended after completing her senior high school education. After graduation, she started workng for a business firm, and she began to feel higher tension in dealing with her boss and other people at her work place. Her symptoms became profuse under such circumstances, and she could not speak loudly. The patient visited our hosptal in September 1988.

Her previous history included asthma in childhood and whiplash due to a traffic accident. The latter led to torticollis (wryneck) in the course of treatment but healed spontaneously about a year later. There was nothing particular about her family history. Except for her voice problems, she showed no systemic neurological problems as those concerning the cerebellum or involuntary motion. Typically, no morphologically or functionally abnormal findings were observed for the larynx either with or without a stroboscope when she produced a sustained vowel /e/ or /i/. When she read aloud a few sentences, she tended to become breathy suddenly, involuntarily and momentarily in pronouncing words which started with A, NA, NI, NU, NE, NO, HA, HI, HU, HE, HO, MA, MI, MU, ME, or MO. Laryngo-stroboscopic observations of the vocal cords during concversation showed sudden abductions of the vocal cords and a wider glottal chink. A laughing voice did not cause breathy phonation. She didn't necessarily have her voice disorder always at the same place in reading aloud the sentences. When she got used to our hospital two months into her periodic visits, she did not show breathy voice at a low pitch very often in her conversation with us.

Figure 1 is a sound spectrograph of the sentense ARUHI KITAKAZE TO TAIYO. F0 and the harmonics were lost at A in ARUHI, and at HI KITAKA. The same findings were obseerved at KA in KAZE, and at IYO in TAIYO. It was observed that the interval between TO and TA in KITAKAZE TO TAIYO was extended, during which time the patient inspired and delayed her voice onset. This longer time for voice onset made the time required for the utterance of a given sentence longer compared to that of a healthy person.

Figure 2 is an EGG of a sustained phonation of /e/. The waves were undisrupted, uniform and rhythmical. It can be seen that the contact area of the vocal cords is larger. No abnormal phonation was observed in sustained voice as shown here.

Figure 3 is an EGG of the word MAZU in the sentence YOSHI HITOMEKURINI SHITEYARO TO MAZU. The EGG wave patterns disappeared between MA and ZU. There was a portion where both edges of the vocal cord did not contact with each other when MA and ZU were produced.

Case 2

This patient was a 24-year-old male. Since January 1984, when he was 21 years old, after severe coughing, he noticed that his voice intermittently and momentarily became breathy. This tendency gradually got worse. One year later, his voice became almost aphonic, he was admitted to several hospitals; but he wasn't diagnosed correctly. When he was 22 years old, he was employed at a pharmacy. When he felt fatigue or stress while speaking to his customers, the voice disorder arose more frequently. But in relaxed enviroments, his voice changed little. In September 1987 he visited our hospital and was diagnosed as having abductor spasmodic dysphonia. There was nothing particular in his past or family history. He had no neurological or psychological signs. When he produced a sustained vowel /e/ or /i/, his vocal cords intermittently abducted abruptly and momentarily. His voice had a tendency to more momentary breathiness when he produced words which started with HA, HI, HU, HE, HO, MA, MI, MU, ME or MO. As a result he had atended to avoid words beginning with these syllables.

The results of the EGG and the soundspectrography were to be abnormal, as in case 1. In the EMG, his PCA's wave exhibited abnormal spikes, while he was speaking (Fig.4). In general, the PCA's action potential was restricted while voiced syllables were spoken.

He was given voice therapy, but he had no control over his voice.

Case 3

This patient was a 25-year-old female. About four years ago she realized a change in her voice. At first, she was not able to say SAN and HACHI, which mean three and eight in Japanese. After she graduated from university, she worked as a gymnastics teacher at a high school. She always had difficulty saying SAN and HACHI when she spoke to her students. She had many opportunities to speak with her boss and older colleagues in stressful environments, and under such circumstances her voice became intermittently breathy. When she relaxed, she was able to speak with less difficulty.

In March 1989 she visited our hospital. There was no particular past history or family history. There were no abnormalities found in her vocal cords under fiberscopic and stroboscopic examinations while she produced a sustained vowel /e/ or /i/. However, while she was speaking, intermittent abrupt breathiness occurred. Her peculiar breathy voice arose at the initial part of words which began with SA, SHI, SU, SE, SO, TA, CHI, TSU, TE, TO, HA, HI, HU, HE, and HO while she was giving lectures to her students. We prescribed a minor tranqilizer, but no effect was seen.

Discussion

All three cases discussed above satisfied the definition of abductor S.D.(1): 'an abrupt breathy release of the true vocal folds.' Seventeen cases have been examined and reported by Hartman et al.(3) in terms of clinical characteristics of Abductor S.D.

Hartman et al.(3) call the disorder 'intermittent breathy dysphonia' (hereafter reffered to as IBD). Their patients ranged from 16 to 56 years of age with an average of 37-38 years, compared to our relatively young patients who are all in their twenties. The male/female ratio was 8 vs 9 in Hartman et al.'s study. An average of five to eight years passed for Hartman's patients from the onset to their first medical examination, compared to a relatively shorter delay for our three cases. Why do such patients wait so long before going to see a doctor compared to other diseases? Our conjecture based on from our three cases is that the frequency of sudden breathy phonation is low in the early stages, and every day activities are not disturbed also. the disorder is easily mistaken for a temporary one caused by the upper respiratory infection.

Our three patients saw the onset of their problems in junior high school and during university. They conversed with family members and close friends most of the time. Breathy phonation did not arise so often in a relaxing atmosphere. Even when it did arise, they re-uttered those words that were disrupted by breathy phonation. They did not feel it was troublesome in their every day life. This could be another reason why they did not visit the hospital for a longer time. Other reports(4) suggest that voice disorders worsen gradually although they may be mild in their early period. When a person graduates from a university and starts working, he or she has more occasions to talk under tension, speaking to bosses and customers, or giving lectures to students. This pushes up the frequency of breathy phonation. A patient finally comes to see a doctor as the disorder interferes with his or her work. We believe that mental and/or phychological factors are somewhat involved in this process.

The vocal abnormality was in no way beneficial to our patients. Rather, they were upset with the voice diorder happening all of a sudden. They were afraid of the vocal change. Thus, they tended to keep quiet. The disorder seems to be different from hysteria in this regard. A percentage of IBD patients are reported to have complained of some sort of neurologic signs, including tremors or tics on the head or the hands. Our three cases did not show any neurologic or psychiatric signs. CT scan and MRI of the brain, therefore, were not conducted.

Diagnosis of Abdoctor S.D.

The diagnosis of abductor S.D. starts with psychoacoustic analysis. The way breathy phonation is generated involuntarily, irregularly and suddenly is similar to how a

radio is suddenly switched off and again turned on. The voice after a breathy voice is characteristically strained, and its voice onset delayed. This is probably because of the patients' efforts to regain normal voice when they get auditory feedback after a sudden breathy phonation. The sudden vocal abnormality tends to be triggered by the aforementioned mental stress, physical fatigue(3, 4), soft voice, loud voice (Case 2 and 3) and words which start with a voiceless consonant.

On the other hand, mental relaxation(3), laughter, loud voice and phonation of voiced consonants work as factors which diminish the vocal disorder. The fact that mental and environmental factors come into play indicate that the abnormality could be psychogenetic, but the causes do not seem to lie in the brain stem nor the cerebellum. This is not a neurologically explained disturbance.

Findings on the larynx

It has been reported that the vocal cords showe a bowing on them. No bowing or sulcus vocalis was observed in our cases. The movement of the vocal cords was good. Sudden breathy phonation did not take place during sustained phonation of /e/. Furthermore, stroboscopic examinnations revealed that the closure of the vocal folds was good. The wave movement of the mucous membrane was symmetrical and showed regular movements. No abnormality was found. When a flexible fiberscope was inserted from the nasal foramen and a few sentences were read aloud, both edges of the vocal cords showed considerable abduction suddenly and intermittently, making the voice breathy. The movement of the vocal cords was not the continuous, periodic changes of the essential tremor and pharyngolaryngeal myoclonus(5). As Hartman et al.(3) state, the "steady state of the prolonged vowel prevented sudden abduction of the vocal folds." Breathy voice does not arise in a sustained phonation but it appears suddenly in running speech. This is an important point in diagnosing this disorder.

Evaluation of Phonatory Function Test

The EGG results showed that EGG wave patterns were completely normal and that the glottis closed when a sustained phonation /e/ was made (Fig.2). In case of reading aloud a few sentences, however, EGG waves suddenly disappeared as shown in Fig.3 and reappeared later. The interval between the disappearance and the re-appearance was approximately 20-50 msec. The EGG waves did not become smaller in the disappearance, nor did smller waves re-appear and become larger. The waves vanished more or less suddenly before re-manifesting themselves at around the same, original size. Thus we realized that the pitch didn't change. This is considered to be a result of the involuntary abduction of the vocal cords, and was another distinctive feature of the abnormality of our patients.

A sound spectrograph showed, as has already been reported by Zwitman(6), that there are two types: one type is that findings look like a picture in a zigsaw puzzle with a few missing pieces and another type is that exhausted exhalation due to abnormal phonation induces inhalation at unusual points which leads to a delayed start of utterance of words(6). Some researchers reported(4,7) that the time delay owing to inspiration and the sudden breathy voice make patients require a longer time to utter a given sentence than the healthy subjects. Others have reported(4) that the time required was the same for both patients and healthy subjects. Our findings showed a slight delay. This may be partly because the patients were nervous about their breathy voice when they got feedback from their ears. Another cause could be that the patients tried to tighten the larynx when they felt they were not making voice in producing unnatural inspiration and pronouncing voiceless consonants. The delay was about 1000 msec(8). Muscles in the facial and cervical regions do not produce spasms. The possibility of the disorder being Meige syndrome(7) was clearly ruled out.

In the EMG, the PCA was a dilator muscle for the vocal cord. In general, action potential of the PCA is restrained in pronouncing voiced syllables, but in this case, it was not (Fig.4). Here, the PCA could have been in a state of abnormal myotonia. We could not so conclude positively regarding this point. Our findings may offer us a clue to the native of abductor S.D. We intend to continue to look into this with more cases.

Treatment

It's relatively popular to treat adductor S.D. by surgery but for abductor S.D. only a pessimistic view is given in most reports. In our approach, we told our patients about the above mentioned possible inducers and reducers and encourage them to avoid words that may lead to abnormal phonation, and to talk in a low or loud voice. This guidance has been found to be the most effective so far. As far as voice training in our clinic is concerned, we give patients training for relaxed phonation and laughter centering on the accent method.

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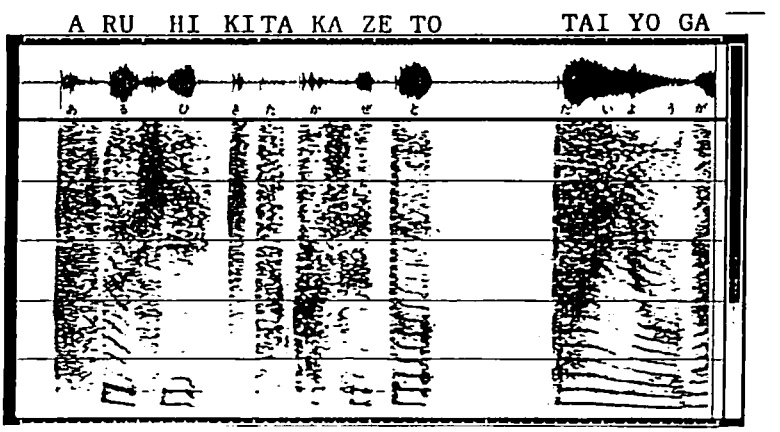

Fig. 1 A sound spectrograph of the sentence uttered by Case 1.

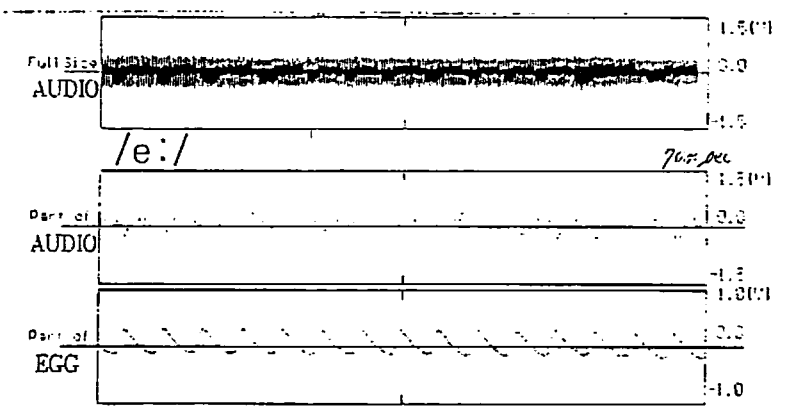

Fig. 2 An EGG of a sustained phonation of /e/ uttered by Case 1.

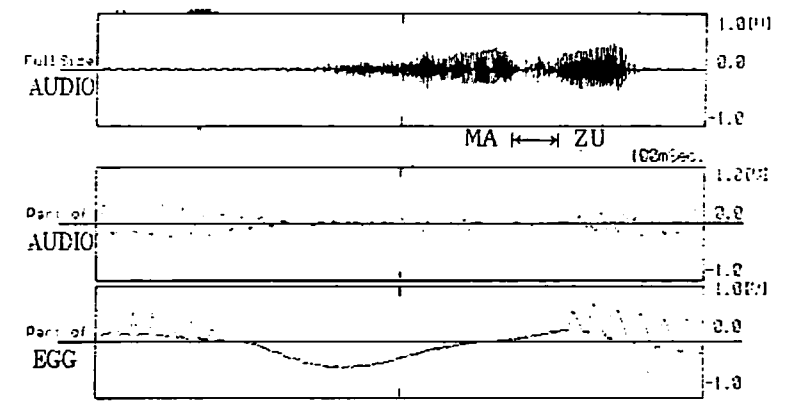

Fig. 3 An EGG of the word MAZU uttered by Case 1.

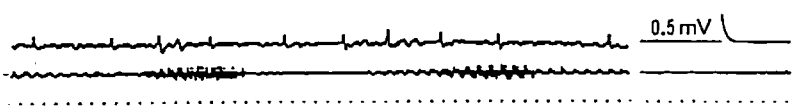

Fig. 4 The PCA in the EMG of Case 2.