Laryngeal Function in Postpolio Patients

Brian P. Driscoll, MD; Carol Gracco, PhD; Carl Coelho, PhD; Jonathan Goldstein, MD; Kiyoshi Oshima, MD; Elizabeth Tierney; Clarence T. Sasaki, MD

Of the 250,000 survivors of the polio epidemics, approximately 25% experience progressive muscle weakness known as postpolio syndrome (PPS). Laryngeal function in postpolio patients previously has not been studied. This paper presents data detailing laryngeal function in a group of postpolio patients who had been evaluated for swallowing complaints. Nine patients underwent comprehensive history and physical exam, acoustical voice analysis, and laryngeal videostroboscopic endoscopy. Three patients underwent laryngeal electromyography (EMG) evaluation. Results indicated some degree of phonatory or laryngeal deficit in all subjects. Subjects with dysphagia also demonstrated vocal fold paralysis. EMG revealed decreased recruitment and increased amplitude, findings consistent with EMG studies in skeletal muscle in postpolio patients. Results suggest that postpolio patients who complain of swallowing difficulties are at risk for laryngeal pathology.

INTRODUCTION

The last polio epidemic in New England occurred in 1955. Since that time, the disease has been virtually eliminated due to successful vaccination programs.[1] The virus, however, continues to plague its original victims of 30 to 40 years ago. It is estimated that there are at least 250,000 postpolio survivors. These patients have gained recent publicity with the recognition of the postpolio syndrome (PPS). This syndrome is characterized by the new onset of progressive muscle weakness, fatigue, and pain. It occurs in patients 30 to 40 years after their original infection with polio. The prevalence of the syndrome is estimated at about 25%.[2] The predisposing factors are being female and having a residual deficit, with a severe residual deficit greatly increasing the risk for developing the syndrome.[2] Most of the data on the postpolio syndrome come from examination of extremity muscles.

Once the virus infects a neuron, that neuron is either damaged or destroyed. The virus has a particular affinity for the anterior horn cell motor neuron.[1] Destruction of a motor neuron leads to orphaning of the muscle fibers. These orphaned muscle fibers can be detected about 2 weeks after injury by the electromyographic (EMG) finding of fibrillation potentials. These low-voltage potentials are detected when the muscle is at rest. They arise from the spontaneous discharge of single denervated muscle fibers. Normally these muscle fibers are then reinnervated by the terminal axonal sprouting of surviving motor neurons. If all denervated fibers are reinnervated, the fibrillation potentials disappear and the motor unit stabilizes. The reorganization of the motor unit by the disease leads to several EMG findings which are characteristic of previous denervation. The motor units are larger (more fibers per neuron). This leads to increased amplitude and duration of the motor unit action potential. An increase in the percentage of the long duration polyphasic motor units can also be seen. Another finding characteristic of denervation and seen in the postpolio motor unit is a decreased recruitment pattern. Normally, as the functional load on a muscle increases, more motor units are recruited to increase the power output of the muscle. Patients with previous denervation have a decreased number of motor units and thus a decreased recruitment pattern is seen.[3]

In postpolio patients the aforementioned findings are seen in muscles previously weakened by the disease and in muscles clinically uninvolved with the disease.[4–8] EMG is unable to distinguish between those patients who complain of new-onset weakness (postpolio syndrome) and those patients who are clinically stable.[7,8] Interestingly, EMG studies also reveal fibrillation potentials and positive waves in most postpolio patients, indicating ongoing denervation in the postpolio motor unit. Similar indictions of instability in the postpolio motor unit have been detected by use of single-fiber electromyography showing increased jitter and blocking. These findings are similar in weakened and stable muscles.[4–8] This instability of the motor unit years after the original insult led Wiechers to speculate that, in the postpolio patient, there are increased metabolic demands placed on the enlarged

Presented at the Meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Inc., Ottawa, Ontario, Canada, January 29, 1994.

From the Section of Otolaryngology (B.P.D., C.G., E.T., C.T.S.) and the Department of Neurology (J.G.), Yale School of Medicine, New Haven, Conn.; the Department of Communication Disorders (C.C.), Gaylord Hospital, Wallingford, Conn.; and Haskins Laboratories (C.G., K.O.), New Haven, Conn. Dr. Driscoll is now with the Department of Otolaryngology, University of Texas Medical Branch at Galveston.

Supported by grants from Haskins Laboratories and Gaylord Hospital, and the McFadden, Harmon, and Mirikitani endowments.

Send Reprint Requests to Brian P. Driscoll, MD, Department of Otolaryngology, University of Texas Medical Branch at Galveston, Room 7.104, John Sealy Annex, 301 University Blvd., Galveston, TX 77555-0521.

motor units. This could lead to an inability of the axon to meet the metabolic demands of all its muscle fibers, causing them to be continuously denervated and reinnervated.[6,9] At some point there may be a net denervation and eventually new onset of muscle weakness. This concept of late denervation is supported by muscle biopsy reports.[7,10]

There have been three reports documenting swallowing difficulties in postpolio patients.[11–13] Sonies and Dalakas[11] found that 75% of the patients who complained of swallowing difficulties had evidence of bulbar polio at original infection. However, on detailed study of 32 randomly selected patients with PPS, 31 showed some swallowing abnormality. Only 2 patients showed any evidence of aspiration. In the study by Coelho and Ferranti,[12] 27% of postpolio patients polled complained of swallowing difficulties. Of the 21 patients tested, 20 had documented abnormalities on videofluoroscopic examination. Swallowing, airway maintenance, and phonation share common anatomy and innervation. A pathologic process affecting one function will likely affect the other functions. The purpose of this study was to document the types of abnormalities seen in laryngeal function in a group of postpolio patients who had been evaluated for dysphagia.

MATERIALS AND METHODS

Patient Selection

The patients were selected from a group of 21 postpolio patients who had been evaluated for swallowing complaints by Coelho and Feranti.[12] These patients are now enrolled in a 2-year follow-up study by the same authors. As part of this study, the patients were offered the opportunity for laryngeal evaluation. Nine of the 21 patients agreed to all or part of the laryngeal evaluation, which consisted of a history and physical examination, comprehensive voice evaluation, and laryngeal EMG. The group comprised 3 men and 6 women ranging in age from 47 to 66 years. Eight of the nine patients had a residual deficit from their initial illness with polio. Five of the 9 patients had bulbar polio as their initial symptomatogy. Six of the 9 carried the diagnosis of postpolio syndrome. Seven of the 9 had complaints specific to voicing or the larynx. Four of the nine had swallowing deficits.

Laryngeal Evaluation

A general medical and surgical history was obtained. The details of the history of polio and any evidence of the postpolio syndrome were recorded. The patients were carefully questioned about possible vocal disturbances. They also underwent a full head and neck examination along with a neurological exam which concentrated on the function of the cranial nerves.

Videostroboscopic Evaluation

Videostroboscopy was performed using a 70-degree Machida (LY-C30) telescope or flexible fiberoptic scope. These instruments were attached to a CCD camera (Computar WA 0422) and the image was displayed on a Sony color video monitor (PVM 1343MD) and recorded on a Mitsubichi super VHS videotape recorder. A Bruel and Kjaer 4914 rhino-larynx stroboscope was used to evaluate vibratory characteristics of the vocal folds during phonation. The stroboscopic data were then evaluated using an adapted version of the University of Wisconsin videostroboscopic rating system.[17]

Acoustical Analysis

Acoustic assessment and perceptual assessment were accomplished in a quiet setting using a (Telex PU-91) head-mounted microphone and a Panasonic professional DAT SV-3700 digital tape recorder for voice input. Mouth-to-microphone distance was constant at 5 cm. Extended speech samples, sustained phonation, and a phonetically balanced speech sample were recorded. Speech samples were randomized for perceptual rating. Three trained judges rated the overall quality of voicing on a five-point equal-interval scale.

For computerized acoustic analysis of sustained phonation, the voice signal was filtered at 1 kHz and transferred via an RC Electronics analog-to-digital board coupled with an IBM 386 computer. The Computer Assisted Speech Evaluation and Rehabilitation (CASPER) software was used to analyze specific measures of variability in the voice signal.[19] These included jitter percent (perturbation in frequency of vibration), shimmer percent (perturbation in amplitude of the signal), and harmonics-to-noise ratio (the ratio of noise components in the signal compared to the noise). Specific formulas for calculating these values based after Koike are as follows[18]:

Jitter Percent-Frequency Perturbation:

$$\frac{100}{n-2}\sum_{1-2}^{n-1}\left|\frac{\frac{(P_{i-1}+P_i+P_{i-1})}{3}-P_i}{\overline{P}}\right| \qquad \overline{P}=\left(\frac{1}{n}\right)\sum_{i=1}^{n}P_i$$

Shimmer Percent-Amplitude Perturbation:

$$\frac{100}{n-10}\sum_{1=6}^{n-5}\left|\frac{\frac{(A_{i-5}+\ldots A_i+\ldots A_{i+5})}{11}-A_i}{\overline{A}}\right| \qquad \overline{A}=\left(\frac{1}{n}\right)\sum_{i=1}^{n}A_i$$

Ai=Peak-Peak amplitude in the cycle

Harmonics-To-Noise Ratio:

$$\frac{n\int_0^T[f_A(t)]^2dt}{\sum_{i=1}^{n}\int_0^{Ti}[f_i(t)-f_A(t)]^2dt}$$

These calculations were made on samples of the midportion of sustained vowel production for the vowel */a/* produced at instructed normal pitch and loudness levels. Four samples from four vowels were analyzed to ensure consistency of these values.

Laryngeal EMG

Laryngeal EMG was performed using a 26-gauge bipolar concentric needle electrode. The cricothyroid, vocalis, and lateral cricoarytenoid muscles were sampled. Thus, data from the superior laryngeal and recurrent laryngeal

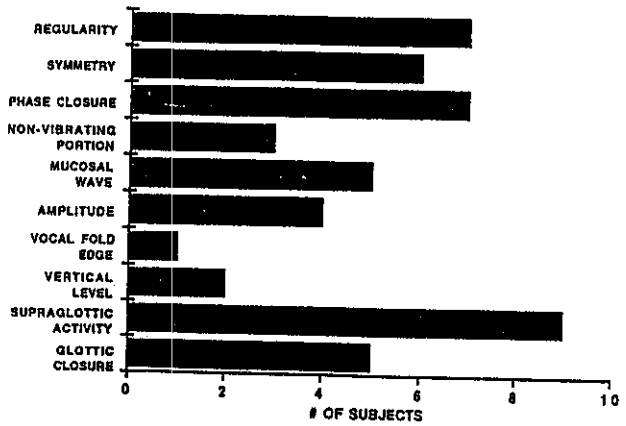

Fig. 1. The distribution of vibratory deficits observed on videostroboscopic exam for the nine patients studied. The y-axis lists the parameters evaluated. The x-axis shows the number of subjects who displayed that abnormality.

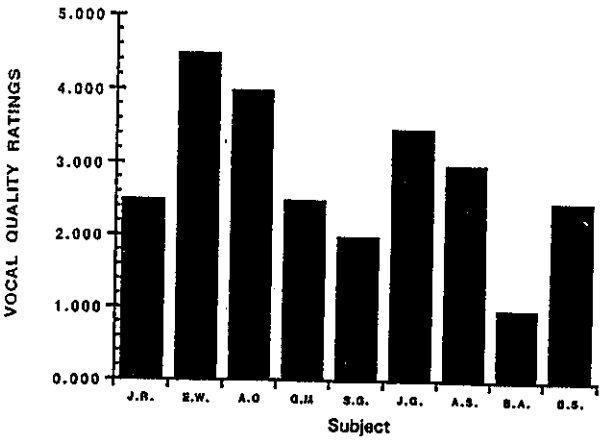

Fig. 2. Perceptual ratings indicating judgments of overall vocal quality. Patients E.W. and A.O. were the only subjects who had no vocal complaint.

nerves were obtained. The technique used for electrode insertion was described by Hirano.[14] Verification gestures following electrode insertion were performed to assure proper placement based on muscle function. Once placement was confirmed, a recording with the muscle at rest was obtained to look for abnormal spontaneous activity. Additional conditions for recording included vocalization of /i/ in falsetto to elicit cricothyroid activity and modal pitch phonation to elicit vocalis muscle activity. Data were sampled and recorded using a Dantec Counterpoint electromyography unit.

RESULTS

Laryngeal Videostroboscopic Assessment

All nine patients had some degree of abnormality on videostroboscopic evaluation. The most striking finding was that four of the nine patients presented with evidence of unilateral vocal fold paralysis based on paucity of movement of the arytenoid cartilage, fixed presentation of a vocal fold during phonation, and atrophic appearance of the vocal fold. All four of these patients had initial bulbar polio and some residual deficits in the trunk or limbs. Figure 1 shows the pooled results from the videostroboscopic data. All nine of the patients were observed to have some level of supraglottic activity. Visual inspection of the records from patients with no evidence of paralysis showed changes in symmetry and regularity of vibration which were attributed to slight to moderate bowing of the vocal folds bilaterally. Supraglottic compensation often at the level of the false vocal fold was also observed in patients with this type of reduction in glottic closure.

Acoustical Analysis

The results of the perceptual data are shown in Figure 2. There were only two patients who scored 4.0 or better. Interestingly, these were the only two patients who had no vocal complaints. As expected, the patients with vocal fold paralysis faired poorly, all having scores of 2.5 or less.

The results of the acoustical analyses are shown in Figures 3 through 5. The harmonics-to-noise ratio (Fig. 3) indicated four patients with values greater than one standard deviation from the mean. A review of the stroboscopic data indicates that three of these four patients demonstrated vocal fold paralysis, while the other patient had a predominately open phase closure which may add noise to the voice. The data for shimmer are shown in Figure 4. Five of the nine patients had values greater than one standard deviation from the mean. Videostroboscopic data show that three of the five patients had vocal fold paralysis, while the other two had a predominately open phase closure with evidence of phase asymmetry. Data reflecting percent jitter or frequency perturbation are illustrated in Figure 5. Two of the nine subjects had jitter scores greater than one standard deviation from the mean. Both of these patients had a vocal fold paralysis.

Laryngeal EMG

Three of the nine patients underwent laryngeal EMG evaluation. The patient with initial history of spinal polio showed no evidence of laryngeal neurologic abnormality on videostroboscopic examination or on electrodiagnostic assessment. Two patients with history of bulbar polio were observed to have evidence of a vocal fold paralysis based on absence of arytenoid activity on videostroboscopic examination. These two patients also demonstrated neurogenic changes evidenced by neuronal dropout in both the "normal" and paralyzed vocal folds (Figs. 6 through 8).

DISCUSSION

The interaction between phonatory, respiratory, and swallowing processes often prompts cross-disciplinary assessment of these functions. In the case of postpolio patients, the initial confirmation of swallowing deficit and anecdotal complaints of deviant vocal quality suggested the need for thorough otolaryngological and voice evaluation. Prior to this study, there

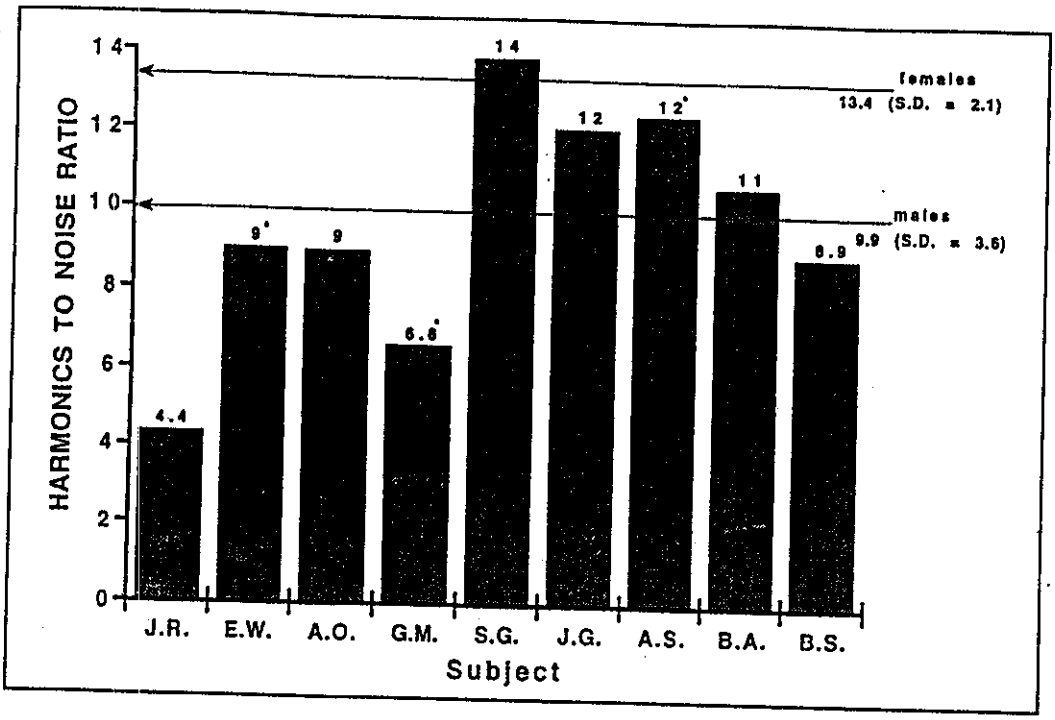

Fig. 3. Harmonics-to-noise ratio is displayed with normative data based on Computer Assisted Speech Evaluation and Rehabilitation (CASPER) analysis for each subject. The asterisks mark the male patients.

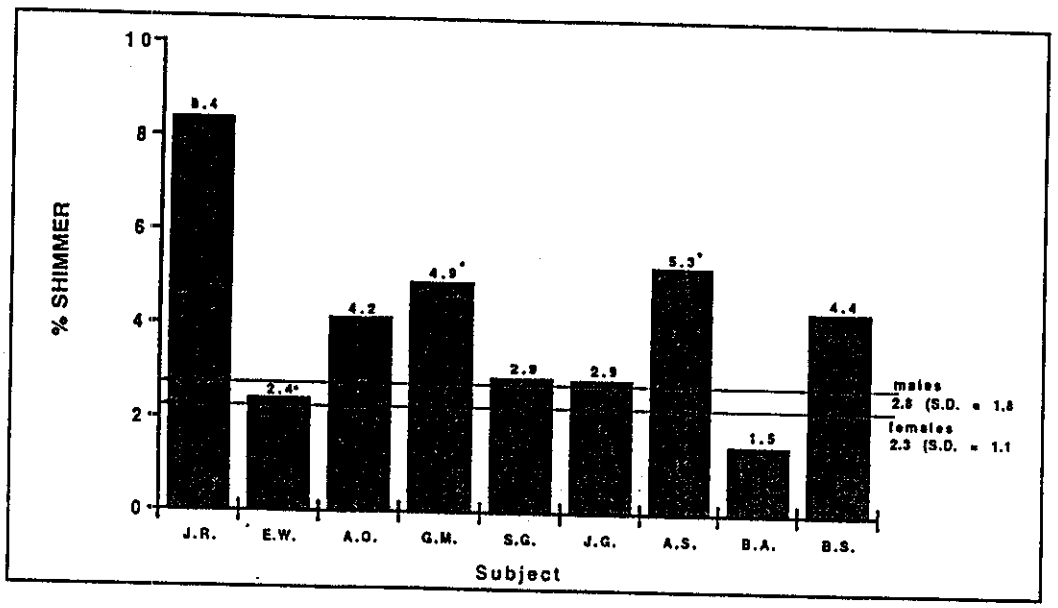

Fig. 4. Percent shimmer, or perturbation in amplitude, for each subject is illustrated with normative data based on CASPER analysis. The asterisks mark the male patients.

were no reports characterizing laryngeal function in postpolio patients. The only data implicating laryngeal abnormalities in this population were two reports of bilateral vocal fold paralysis.[15,16]

Although this report is a limited study, the patients who reported severe swallowing deficits were those who also presented with the most severe voice and laryngeal deficits. All four patients with persistent and frequent swallowing deficits presented medical histories with bulbar polio as initial symptomatology. It was consistently reported in the record that these patients had early swallowing deficits and often reported laryngeal paralysis as well. Although voicing had improved over the years, these patients were the four who had confirmed unilateral vocal fold paralysis and often abnormal electrodiagnostic exam for both vocal folds. In the latter two cases, the EMG exam was simply less abnormal for one of the vocal folds. This likely represents severe involvement of the bulbar neurons at original infection with a resultant residual deficit.

The reasons for increased severity of symptoms 20 years postonset is suggested to be related to the original weakened muscle tissue and normal factors of aging in the vocal folds. The literature reports loss of collagen and elastic tissue and changes in muscle

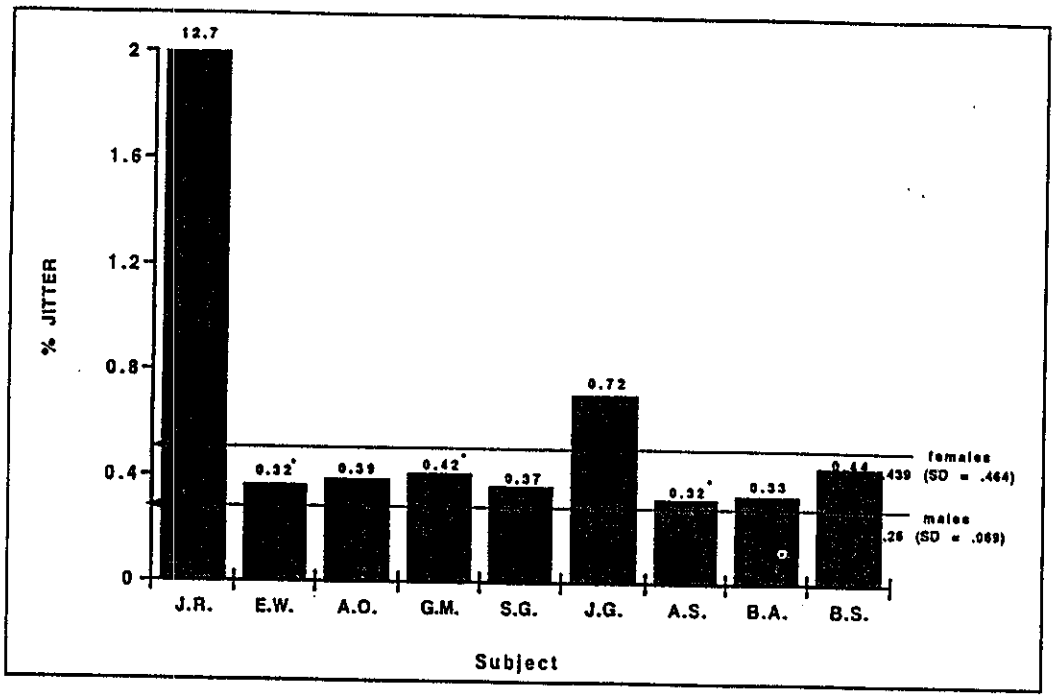

Fig. 5. Jitter, or perturbation in frequency, is shown for each subject. Normative data are based on the CASPER analysis system. The asterisks mark the male patients.

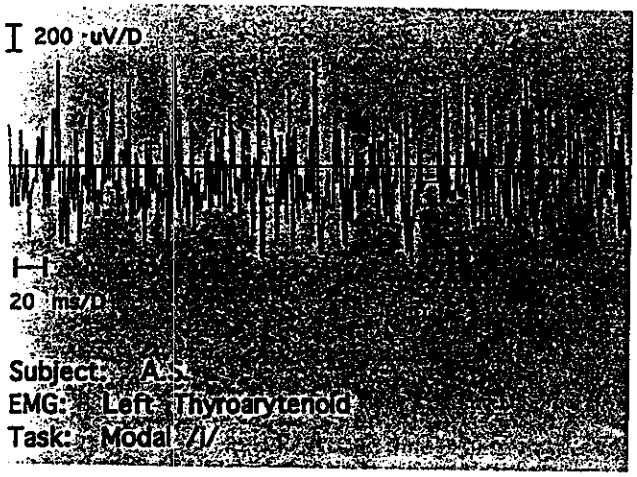

Fig. 6. The electromyography (EMG) tracing of the left thyroarytenoid muscle for patient A.S. during vocalization of modal /i/. Note the full phase interference pattern of this normal EMG. This patient had no movement abnormality as observed on videostroboscopic examination.

fibers which render glottic closure less dynamic and vibration less stable.[20]

These findings also suggest that postpolio patients who have active swallowing difficulties are currently not well served in terms of referral and rehabilitation. Although only one of the four patients gave a history consistent with aspiration, all nine subjects demonstrated some form of untreated vocal pathology.

The laryngeal function in our patients was most influenced by the finding of a vocal fold paralysis. It is interesting, however, that some patients with vocal fold paralysis were found to have acoustic measures of voicing which approached normal values. These patients were those who demonstrated compensatory strategy by the opposing vocal fold and/or supraglottic structures during closure.

The varied nature of electrodiagnostic exams for these patients deserves comment. The one patient (A.S.) with a normal videostrobe exam also had a normal laryngeal EMG. He exhibited normal-amplitude EMG signals and had a full field interference pattern with vocalization (Fig. 6). This is not surprising since this patient suffered only spinal polio based on record of medical history. The two patients with evidence of vocal fold paralysis and history of swallowing and voice deficit each demonstrated abnormal EMG. However, the EMG was abnormal for both the mobile and nonmobile vocal folds. The findings of decreased recruitment in both the involved and the uninvolved muscle are consistent with the EMG data on extremity muscles.[4–8] Such was the case with subject J.R., who demonstrated a right vocal fold paralysis. EMG revealed decreased recruitment with very-high-amplitude motor units on the involved, immobile vocal fold. This may indicate restructuring of the motor unit and replacement by a decreased number of very large motor units. On the functional side (left), the EMG showed normal-sized motor units with evidence of decreased recruitment of a less severe nature than on the contralateral side (Fig. 7). The observed movement of the left vocal fold may simply represent a lesser degree of neuronal damage. However, other possibilities include laryngeal synkinesis (perhaps the posterior cricoarytenoid is also firing, giving no net movement) or arytenoid fixation.

Subject B.A. was a complex patient who had a his-

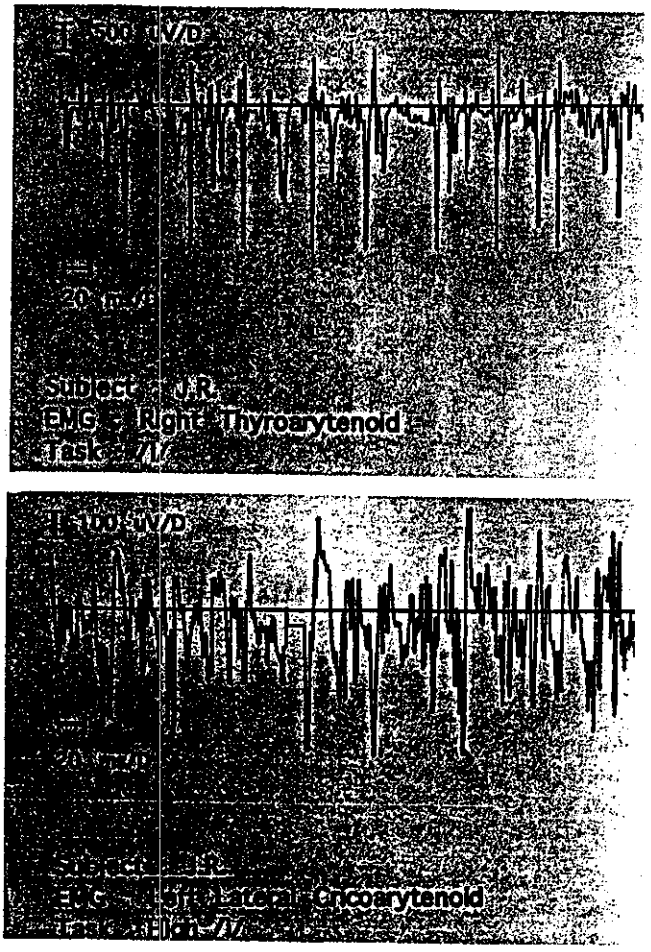

Fig. 7. Patient J.R. has a right vocal fold paralysis. **Top.** The EMG signal from the right thyroarytenoid muscle characterizing a decreased interference pattern along with high voltage motor units. **Bottom.** The EMG signal from the left lateral cricoarytenoid displaying normal voltage with a decreased interference pattern.

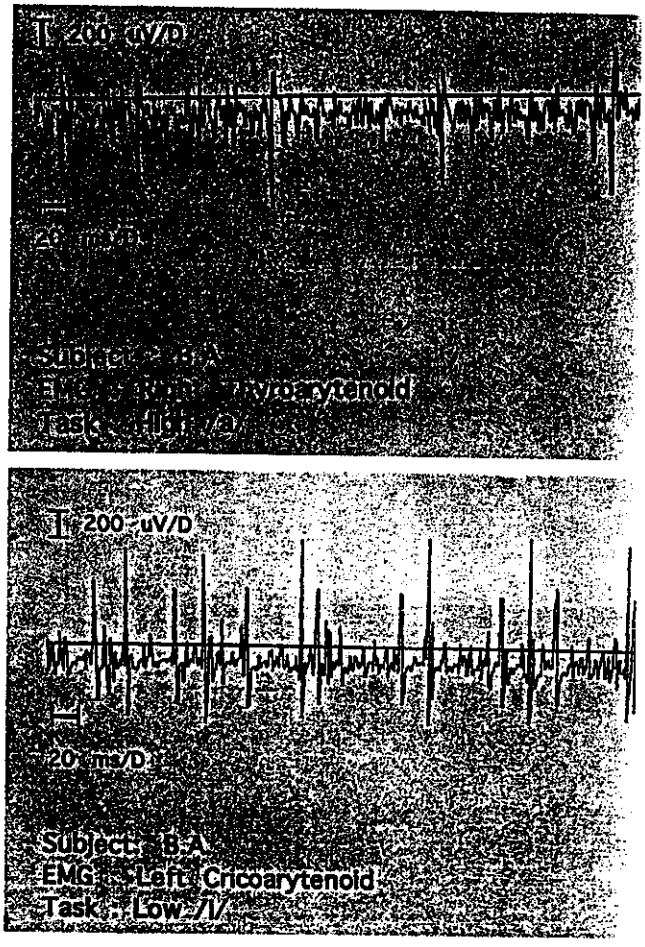

Fig. 8. Patient B.A. has a left vocal fold paralysis and a history of bilateral vocal fold paralysis. EMG signals from the right thyroarytenoid **(top)** and the left lateral cricoarytenoid **(bottom)** both show severely decreased interference patterns.

tory of bilateral vocal fold paralysis which required tracheotomy. On videostroboscopy there was evidence of a left vocal fold paralysis, with clearly demonstrated movement of the right vocal fold. Her laryngeal EMG indicated a bilateral neuropathic pattern with severe neuronal dropout in both vocal folds (Fig. 8). Again the movement of the lesser-damaged vocal fold may be related to aforementioned factors. Of these electrodiagnostic exams, there was no evidence of fibrillation potentials or positive waveforms. Although many postpolio patients have this finding, it is not as universal as a decreased recruitment pattern.[6]

CONCLUSIONS

In conclusion, several points can be made from this study's data. Electrodiagnosis of neuronal dropout or axonal loss in these patients is consistent with neurogenic change. Patients with previous bulbar symptoms showed evidence of neurogenic change.

The progressive nature of this disease is variable. However, if the disease leads to a progressive weakening as suggested in the reports by Dalakis, *et al.*,[10,14] the patients with vocal fold paralysis and EMG evidence of contralateral cord involvement may be at high risk for bilateral vocal fold paralysis and subsequent acute respiratory distress.

Further, each of the nine subjects in this group demonstrated some form of voice deficit. In the case of reduced or weakened glottic closure, voice treatment alone or in combination with surgical intervention such as thyroplasty to improve closure seems highly appropriate. In many cases, neither diagnostic examination nor rehabilitation had been suggested during the 20-year postpolio period, even in the face of overt symptomatology. Clearly those patients who have active swallowing complaints require thorough laryngeal and voice assessment to evaluate the occurrence and extent of coexistent laryngeal pathology, in addition to appropriate therapy.

ACKNOWLEDGMENTS

This paper is dedicated to the memory of Bernice Alexander. The authors gratefully acknowledge the participation of subjects for this study.

BIBLIOGRAPHY

1. Raymond, D.A. and Victor, M.: *Principles of Neurology* (4th ed.). McGraw-Hill, New York, pp. 591–697, 1989.
2. Ramlow, J., Alexander, M., LaPorte, R., *et al.*: Epidemiology of the Post-Polio Syndrome. *Am J Epidemiol*, 136:769–785, 1992.
3. Raymond, D.A. and Victor, M.: *Principles of Neurology* (4th ed.). McGraw-Hill, New York, pp. 1017–1024, 1989.
4. Trojan, D.A., Gendron D. and Cashman, N.R.: Electrophysiology and Electrodiagnosis of the Post-Polio Motor Unit. *Orthopedics*, 14:1353–1361, 1991.
5. Daube, J.R.: Electrophysiologic Studies in the Diagnosis and Prognosis of Motor Neuron Diseases. *Neurol Clin*, 3:473–491, 1985.
6. Weichers, D.O. and Hubbell, S.L.: Late Changes in the Motor Unit After Acute Poliomyelitis. *Muscle Nerve*, 4:524–528, 1981.
7. Cashman, N.R., Maselli, R., Wollmann, R.L., *et al.*: Late Denervation in Patients With Antecedent Paralytic Poliomyelitis. *N Engl J Med*, 317:7–12, 1987.
8. Agre, J.C., Rodriguez, A.A. and Tafel, J.A.: Late Effects of Polio: Critical Review of the Literature on Neuromuscular Function. *Arch Phys Med Rehabil*, 72:923–931, 1991.
9. Weichers, D.O.: New Concepts of the Reinnervated Motor Unit Revealed by Vaccine-Associated Poliomyelitis. *Muscle Nerve*, 11:356–364, 1988.
10. Dalakas, M.C., Elder, G., Hallett, M., *et al.*: A Long-Term Follow-up Study of Patients With Post-Poliomyelitis Neuromuscular Symptoms. *N Engl J Med*, 314:959–963, 1986.
11. Sonies, B.C. and Dalakas, M.: Dysphagia in Patients With the Post-Polio Syndrome. *N Engl J Med*, 324:1162–1167, 1991.
12. Coelho, C. and Ferranti, R.: Incidence and Nature of Dysphagia in Polio Survivors. *Arch Phys Med Rehabil*, 72:1071–1075, 1991.
13. Jones, B., Buchholz, D.W., Ravich, W.J., *et al.*: Swallowing Dysfunction in the Postpolio Syndrome: A Cinefluorographic Study. *AJR Am J Roentgenol*, 158:283–286, 1992.
14. Hirano, M.: Clinical Examination of the Voice. In: *Disorders of Human Communication*. F. Winckel and B.D. Wyke (Eds.). Springer-Verlag, New York, p. 122, 1981.
15. Cannon, S. and Ritter, F.N.: Vocal Cord Paralysis in Postpoliomyelitis Syndrome. LARYNGOSCOPE, 97:981–983, 1987.
16. Nugent, K.M.: Vocal Cord Paresis and Glottic Stenosis: A Late Complication of Poliomyelitis. *South Med J*, 80:1594–1595, 1987.
17. Bless, D.M., Hirano, M. and Feder, R.J., Videostroboscopic Evaluation of the Larynx. *Ear Nose Throat*, 66:289–296, 1987.
18. Koike, Y., Takahashi, H. and Calcaterra, T.C.: Acoustic Measures for Detecting Laryngeal Pathology. *Acta Otolaryngol (Stockh)*, 84:105–117, 1977.
19. Till, J.: Computer-Assisted Evaluation of Speech Disorders: Rationale and Directions for Future Development. *Journal of Computer Users in Speech and Hearing*, 2:134–148, 1990.
20. Hirano, M., Kurita, S. and Nakashima, T.: Growth, Development and Aging of Human Vocal Folds. In: *Vocal Fold Physiology*. D.M. Bless and J.H. Abbs (Eds.). College Hill Press, San Diego, pp. 22–24, 1983.