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Guest Editorial

POST-POLIO DYSPHAGIA: ALARM OR CAUTION?

David W. Buchholz, MD
Bronwyn Jones, FRACP, FRCR

Dysphagia is among the residual and potentially progressive problems encountered by polio survivors.[1-4] Post-polio dysphagia is due primarily to pharyngeal and laryngeal muscle weakness as a consequence of bulbar (brain stem) motor neuron destruction at the time of acute polio. It has been estimated that 10% to 15% of patients with acute polio develop dysphagia during the acute attack.[5]

The prevalence of residual dysphagia among polio survivors may be in the range of 10% to 20% according to questionnaire studies.[6,7] Less is known about the extent of functional disability and the risk of complications, such as aspiration pneumonia, related to dysphagia among polio survivors. Similarly, although it has been suggested that pharyngeal dysphagia may be among the progressive problems facing polio survivors, there have been no convincing data.[3,4]

Over the last few years we have evaluated 28 patients at the Johns Hopkins Swallowing Center with a remote history of polio and recent or increasing dysphagia. The Johns Hopkins Swallowing Center is a multidisciplinary group of physicians and allied health professionals who have a special interest in patients with the symptom of dysphagia (difficulty in swallowing, sensation of food sticking); clinicians include gastroenterologists, neurologists, radiologists, otolaryngologists, head and neck surgeons, rehabilitation medicine specialists, and speech/language pathologists. The Center evaluates over 100 patients with dysphagia per year and actively consults on many more than that number. Patients are referred from local, national, and international sources.

We recently analyzed the cinefluorographic data of 25 post-polio patients with dysphagia (Table). Two had minimal findings, including one who flexed the head to facilitate swallowing and another who had minimal laryngeal penetration; the others had abnormalities varying from mild pharyngeal weakness with minimal retention in valleculae or pyriform sinuses following swallowing to complete loss of pharyngeal muscle contraction and aspiration (sometimes without a protective cough reflex). Some of the abnormalities could be ascribed to pharyngeal weakness.

Despite the finding of poor or absent pharyngeal contraction, in most cases the swallow was "safe" or "compensated."[8] Aspiration was found in five patients and moderate laryngeal penetration without aspiration in two others; minimal or occasional laryngeal penetration without aspiration was present in the remaining abnormal studies.

Importantly, other abnormalities contributing to symptoms such as a cervical esophageal stricture, Zenker's diverticulum, a lateral diverticulum, or pharyngeal pouches were present in one third of patients and esophageal abnormalities were found in almost half (44%) (Table). In addition, several patients had other neuromuscular processes that could have been causing or contributing to the patients' symptoms, including Parkinson's disease, cerebrovascular accident, and cerebellar atrophy. This study did not address progression of symptoms but warned against ascribing dysphagic symptoms to polio without excluding other potentially treatable structural lesions or other neuromuscular disorders.

Because of our experience with this group of polio survivors and many other patients with dysphagia, we felt that some comment should be made on a recent publication that deals with progressive symptoms of dysphagia in the post-polio population and raised the question of swallowing "abnormalities" even in the asymptomatic post-polio population.[9] Sonies and Dalakas are to be congratulated for their efforts. Nonetheless, we are concerned that this paper may be misleading and perhaps unnecessarily alarming. The authors studied 32 patients randomly selected from a cohort of 72 polio survivors. As the authors noted, "Of the 32 patients, 14 had symptoms of new swallowing difficulties, and 18 were asympto-

Dr Buchholz is Associate Professor, Department of Neurology, and Neurologist, the Johns Hopkins Swallowing Center, The Johns Hopkins Medical Institutions, Baltimore, Md; Dr Jones is Professor, the Russell H. Morgan Department of Radiology and Radiological Science, and Director, the Johns Hopkins Swallowing Center.

Address reprint requests: David W. Buchholz, MD, the Johns Hopkins Swallowing Center, The Johns Hopkins Medical Institutions, Baltimore, MD 21205.

Table

OTHER ABNORMALITIES IN 25 PATIENTS WITH DYSPHAGIA AND A REMOTE HISTORY OF POLIO

Pharynx or cervical esophagus	
Zenker's diverticulum	1
Lateral diverticulum	2
Bilateral pharyngeal pouches	8
Stricture	1
Esophageal disease	
(hiatal hernia, reflux or spasm)	11
Other diseases	
Neuromuscular disorders (Parkinson's, cerebellar atrophy, multiple cerebrovascular accidents)	3
Supraglottic laryngectomy	1

Numbers not cumulative; several findings may be present in a single patient.

matic in this respect." Remarkably, the combination of detailed oral sensorimotor examination, ultrasonography of swallowing, and videofluoroscopy of swallowing revealed some abnormality in all but one of the 32 patients.

Our concerns relate to three main points. First, these authors do not clearly differentiate between the post-polio syndrome and progressive post-poliomyelitis muscular atrophy (PPMA). Post-polio syndrome is variably defined but is generally regarded as a constellation of symptoms such as fatigue, muscle and/or joint pain, and muscle weakness and/or wasting occurring usually several decades after recovery from acute polio. It has many causes, only one of which is PPMA.[10] The mechanism of PPMA is thought to be gradual exhaustion of the ability of surviving motor neurons to maintain their abnormally large axon trees after several decades of reinnervation following polio.[11,12] The incidence of PPMA is unknown, but its rate of symptomatic progression in affected individuals seems to be very slow, in the range of 1% per year decline in muscle strength.[11]

It is critically important to distinguish between post-polio syndrome and PPMA; PPMA is presently untreatable, whereas many of the other causes of post-polio syndrome can be treated. Additionally, confusion between post-polio syndrome and PPMA has probably led to an overestimation of the extent to which PPMA occurs in polio survivors, and that has led many individuals to become unnecessarily anxious about their prognosis.

Our second concern regarding their paper pertains to its potential to provoke exaggerated impressions of the magnitude of the problem of post-polio dysphagia. Their study did not utilize a control group for videofluoroscopy. Recent evidence suggests that the incidence of unexpected "abnormalities" detected by videofluoroscopy is quite high, especially among the asymptomatic elderly,[13] suggesting that the standards of normality in the young should not be applied in a stringent fashion to the older person. Accordingly, it is unclear to what extent the videofluoroscopic findings among post-polio patients are truly "abnormal." Moreover, the findings were relatively mild in that only one dysphagic and one non-dysphagic post-polio patient demonstrated "trace aspiration after swallowing." In this light, references to "often life-threatening complications of choking and aspiration pneumonia" and "potentially life-threatening aspiration" appear to be inappropriate. The data in their paper do justify caution, but not necessarily alarm, in considering the presence and implications of dysphagia among polio survivors.

The third and final concern pertains to the conclusion, "These abnormalities suggest that in bulbar neurons there is a slowly progressive deterioration similar to that in the muscles of the limbs." This seems to be based on two sources of data. First, as in the 25 patients we have studied, Sonies and Dalakas found that a large percentage of their post-polio patients have, by history, symptoms suggestive of progressive pharyngeal impairment, often beginning several decades after the acute illness. It must be kept in mind that these are subjective data provided by patients who have sought medical attention in the setting of widespread fear about "reactivated" polio, and it is understandable that anxiety may be influencing their perception of symptoms, especially those involving a function as essential as swallowing (Clark M, Witherspoon D. A new scare for polio victims. *Newsweek*, April 23, 1984, p 83).

The other source of information suggesting to Sonies and Dalakas that post-polio dysphagia may be progressive is follow-up information on four of their patients within 1 to 2½ years after the initial evaluation. Careful review of the subjective and objective data indicating progressive dysphagia among these four patients raises doubt in our minds that it can as yet be stated that PPMA causes progressive dysphagia. More extensive, longitudinal, objective studies are needed before that conclusion can be firmly drawn. For now, it is puzzling that a condition such as PPMA, which allegedly causes slowly progressive weakness at the rate of only 1% per year, should result in substantial worsening of pharyngeal muscle function over an interval as short as 1 or 2 years as Sonies and Dalakas seem to suggest. If that observation is valid, the explanation may be that even slight additional weakness can cause symptomatic decompensation of marginally compensated pharyngeal dysphagia.[8]

Sonies and Dalakas imply that post-polio bulbar dysfunction may offer a unique opportunity to study PPMA, because "new bulbar dysfunction can be quantified" and such quantification has been difficult in the limbs. Our experience indicates that quantification of videofluoroscopic findings is difficult, and refinement of this technique is necessary before it will be useful as a reliable quantitative tool. On the other hand, videofluoroscopy can be helpful in evaluating individual patients; the question is, which post-polio patients should have swallowing studies. Should we study all patients with a past history of polio, even if presently asymptomatic, or should we be more selective? Based on our experience with swallowing disorders, our recommendations are that there are several groups who definitely need to be evaluated.

First, the patient with residual dysphagia *since* the acute polio requires a baseline study. Such patients' residual dysphagia may have resulted in either the need to substantially modify diet and/or feeding habits, symptoms of airway penetration (such as choke/cough episodes), or may have caused a complication such as aspiration pneumonia or airway obstruction. This group would include patients who had severe bulbar involvement at the time of the acute attack with quadriparesis, breathing or swallowing problems, and who may have required treatment in an iron lung. It is apparent from the work of Sonies and Dalakas that individuals with prior *bulbar* polio are at higher risk for dysphagia and its complications than are those who experienced only spinal polio.

The second group requiring evaluation includes patients with mild residual deficits in swallowing since the acute polio who are noticing a change or deterioration in the ability to eat or swallow (such as food sticking, coughing, choking, swallowing, and slowing of swallowing or eating ability).

The third group includes those patients who have new difficulty with eating or swallowing.

The purpose of swallowing evaluation of these patients is to diagnose the cause or causes of dysphagia and to provide optimal management of the problem. It should not be assumed that progressive dysphagia in a post-polio patient is due to progressive muscle weakness; survivors are not immune to other, potentially treatable conditions such as a superimposed structural lesion (Table).[14] Even if no abnormality other than pharyngeal weakness is demonstrated, there is evidence that patients can benefit from tailored swallowing therapy that allows the patient to improve swallowing.[15] Therapy includes changing or restricting diet to certain "safe" consistencies (substances that can be safely swallowed without airway entry, such as purees). These substances need to be individually determined by a swallowing therapist aided by videofluoroscopy. Special breathing techniques or head positioning also may be useful.

In summary, much remains to be learned about the prevalence and impact of post-polio dysphagia. There is as yet no good evidence that patients need be alarmed about the prospect of developing disabling or life-threatening dysphagia. On the other hand, it is prudent to formally evaluate polio survivors with dysphagia, using videofluoroscopy, if dysphagia symptoms are serious, progressive, or new. Treatment such as supervised swallowing rehabilitation is available to enhance the ease and safety of feeding.

REFERENCES

1. Bosma JF. Studies of disability of the pharynx resultant from poliomyelitis. *Ann Otol Rhinol Laryngol.* 1953; 64:529-547.

2. Ardran GM, Kemp FH, Wegelius C. Swallowing defects after poliomyelitis. *Br J Radiol.* 1957; 30(353):169-189.

3. Buchholz D. Dysphagia in post-polio patients. In: Halstead LS, Wiechers DO, eds. *Research and Clinical Aspects of the Late Effects of Poliomyelitis.* White Plains, NY: March of Dimes Birth Defects Foundation; 1987:55-62.

4. Buchholz D, Jones B. Dysphagia occurring after polio. *Dysphagia.* 1991; 6:165-169.

5. Baker AB, Matze HA, Brown JR. Poliomyelitis III: a study of medullary function. *Arch Neurol.* 1950; 63:257.

6. Halstead LS, Wiechers DO, Rossi CD. Late effects of poliomyelitis: a national survey. In: Halstead LS, Wiechers DO, eds. *Late Effects of Poliomyelitis.* Miami, Fla: Symposia Foundation; 1985:11-31.

7. Speier JL, Owen RR, Knapp M, Canine JK. Occurrence of post-polio sequelae in an epidemic population. In: Halstead LS, Wiechers DO, eds. *Research and Clinical Aspects of the Late Effects of Poliomyelitis.* White Plains, NY: March of Dimes Birth Defects Foundation; 1987:39-48.

8. Buchholz DW, Bosma JF, Donner MW. Adaptation, compensation, and decompensation of the pharyngeal swallow. *Gastrointestinal Radiology.* 1985; 10:235-239.

9. Sonies BC, Dalakas MC. Dysphagia in patients with the post-polio syndrome. *N Engl J Med.* 1991; 324:1162-1167.

10. Dalakas MC. New neuromuscular symptoms after polio (the post-polio syndrome): clinical studies and pathogenetic mechanisms. In: Halstead LS, Wiechers DO, eds. *Research and Clinical Aspects of the Late Effects of Poliomyelitis.* White Plains, NY: March of Dimes Birth Defects Foundation; 1987:241-265.

11. Dalakas MC, Elder G, Hallett M, et al. A long-term follow-up study of patients with post-poliomyelitis neuromuscular symptoms. *N Engl J Med.* 1986; 314:959-963.

12. Cashman NR, Maselli R, Wollmann RL, et al. Late denervation in patients with antecedent paralytic poliomyelitis. *N Engl J Med.* 1987; 317:7-12.

13. Ekberg O, Feinberg MJ. Altered swallowing function in elderly patients without dysphagia: radiographic findings in 56 patients. *AJR.* 1991; 156:1181-1184.

14. Buchholz DW, Marsh BR. Multifactorial dysphagia: [illegible] *Dysphagia.* 1986; 1:88-90.

15. Silbergleit AK, Waring WP, Sullivan MJ, Maynard FM. Evaluation, treatment, and follow-up results of post polio patients with dysphagia. *Otolaryngol Head Neck Surg.* 1991; 104:333-338.