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DYSPHAGIA IN PATIENTS WITH THE POST-POLIO SYNDROME

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Abstract *Background and Methods.* Dysphagia may develop in some patients many years after an attack of acute paralytic poliomyelitis. To identify clinical or subclinical signs of oropharyngeal dysfunction, we examined 32 patients (mean age, 48.9 years) with the post-polio syndrome (defined by new weakness in the limbs). Of the 32 patients, 14 had symptoms of new swallowing difficulties, and 18 were asymptomatic in this respect; 12 had a history of bulbar involvement during acute poliomyelitis. Swallowing function was assessed objectively by ultrasonography, videofluoroscopy, and an oral motor index score for 10 components of oral function.

Results. All but 1 of the 32 patients, regardless of whether they had new symptoms or previous bulbar involvement, had some abnormality on detailed testing of oropharyngeal function; only 2 patients had any signs of aspiration. The mean oral motor index score (a quantitative measure of oral sensorimotor function) in the patients was higher than that in age-matched normal subjects ($P<0.001$). Videofluoroscopy showed abnormalities of varying severity, including unilateral bolus transport through the pharynx, pooling in the valleculae or pyriform sinuses, delayed pharyngeal constriction, and impaired tongue movements. On ultrasonography, the mean (±SD) duration of wet swallows was significantly longer in the symptomatic patients than in the asymptomatic patients (2.67±0.70 vs. 1.65±0.42 seconds). The four patients who were reexamined two years later had objective signs of worsening oropharyngeal function and corresponding new symptoms.

Conclusions. In patients with the post-polio syndrome, the bulbar muscles often have clinical or subclinical signs of dysfunction. These abnormalities suggest that in bulbar neurons there is a slowly progressive deterioration similar to that in the muscles of the limbs. (N Engl J Med 1991; 324:1162-7.)

THE post-polio syndrome consists of a variety of musculoskeletal symptoms with muscular atrophy that create new difficulties with activities of daily living 25 to 30 years after the original attack of acute paralytic poliomyelitis.[1-11] Post-poliomyelitis muscular atrophy is characterized by slowly progressive weakness in muscles previously affected or, less often, in muscles seemingly spared during the original illness.[1-8]

Although attention has focused primarily on new weakness affecting the limbs, symptoms involving the muscles of the oropharynx (i.e., the bulbar muscles) have been increasingly recognized in isolated case reports.[12] This complication is not unexpected because in acute paralytic poliomyelitis there is widespread infection of the motor neurons not only in the spinal cord but also in the brain-stem nuclei.[13,14] Symptoms due to the involvement of the bulbar neurons have in fact been recorded in at least 15 percent of patients with acute paralytic poliomyelitis.[13,14] When post-poliomyelitis muscular atrophy affects the limbs, it causes difficulties with mobility and functional activities. By contrast, weakness in the bulbar muscles, which activate the oropharynx and larynx, may cause occult and often life-threatening complications of choking and aspiration pneumonia.

In the limb muscles of patients who have had polio, the motor units are in a state of denervation and reinnervation, even in clinically stable muscles.[1-11,15,16] It is believed that clinical signs of new weakness develop when these chronically overstressed motor neurons can no longer support the newly reinnervated muscle fibers.[1-11,15,16] This process is very slow and can be detected by electromyography and muscle biopsy.[1-8] Because the bulbar muscles cannot be evaluated with these techniques, their status remains unknown until the patient suddenly becomes aware of sometimes life-threatening dysphagia or choking.

In this study, we examined the oropharyngeal and esophageal musculature of 32 patients with post-poliomyelitis muscular atrophy. Fourteen of these patients had new symptoms of swallowing difficulties, and the other 18 were asymptomatic. Our goal was to identify neuronal bulbar dysfunction similar to the dysfunction that occurs in the limbs of patients who have had polio.[1-8] Our observations should have practical implications for the prevention of choking or aspiration in patients with post-poliomyelitis muscular atrophy.

METHODS

We studied 32 patients (mean age, 45.7 years) who had a history of paralytic poliomyelitis with the recent development of post-poliomyelitis muscular atrophy, as defined by the clinical criteria of new weakness in previously affected or unaffected limbs.[1-8] These patients were selected at random from a cohort of 72 patients studied consecutively. All 32 patients selected for this study presented clinically with post-poliomyelitis muscular atrophy that affected the skeletal musculature and that had developed a mean of 33 years after the initial attack of polio (range, 12 to 55). Only two patients presented with dysphagia as the predominant symptom. The possible involvement of the bulbar muscles during the original attack of polio was not a consideration in the selection of the patients. Assessment included blood chemistry studies, electromyography, cervical and lumbar x-ray films, and in most of the patients, lumbar puncture and muscle biopsy. Patients with other medical, neurologic, psychiatric, or orthopedic illnesses that could explain the new symptoms were excluded. All the patients who agreed to undergo a swallowing study gave their written informed consent. The protocol was approved by the institute's clinical investigational review committee.

Fourteen of the 32 patients with post-poliomyelitis muscular atrophy (7 men and 7 women, ranging from 27 to 64 years of age; mean, 46.5) had symptoms of oropharyngeal dysfunction, whereas 18 others (11 men and 7 women, ranging from 25 to 71 years of age; mean, 50.7) had no such symptoms. The progression of symptoms

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was assessed by follow-up examinations of four patients after a mean of two years. The findings in the patients who had had polio were compared with those in 12 normal, healthy subjects matched for age (mean age, 53.5 years; range, 37 to 71).

All 72 patients had a neurologic examination and were then interviewed to determine whether they were aware of any changes in speaking, swallowing, or oral sensorimotor ability that were associated with the onset of post-poliomyelitis muscular atrophy. The 32 study patients completed a checklist of potential problems and symptoms associated with dysphagia.[17] The checklist consisted of 18 items designed to identify problems associated with dysphagia that occurred during a typical meal, including difficulty swallowing, the need to avoid certain foods, food catching in the throat, the feeling of having a lump in the throat, and coughing or choking before, during, or after swallowing.

Oral sensorimotor examination of cranial-nerve function was performed as described previously.[18-20] Oral motor function was assessed for 10 measures of oral sensorimotor activity (oral structure; symmetry; volitional and reflexive movement of the tongue, lips, jaw, and palate; strength of the tongue and lips; swallowing ability; voice quality; oral sensation; fluency; speech articulation; and oral diadochokinesia). The oral motor function of each patient was rated on each of the 10 measures as 1 (normal), 2 (mild deficit), 3 (moderate deficit), or 4 (severe deficit), and a mean oral motor index was calculated. This rating system, which is objective and reproducible, is based on earlier studies.[18,19] For example, a mild-to-moderate score of 2.5 for the strength of the tongue and lips suggests that the strength or motion of either the tongue or the lips is impaired sufficiently to affect speech or swallowing, whereas a moderate-to-severe rating of 3.5 suggests that both strength and motion of the tongue and lips are sufficiently diminished to impair speech and swallowing.

Ultrasonography

The real-time ultrasound assessment of swallowing involves submental placement of a 5-mHz ultrasound transducer to visualize the activity of the tongue, the hyoid bone, and a bolus of water.[21,22] The motion of the hyoid bone as it moves superiorly and anteriorly toward the mandible indicates the start of the oropharyngeal phase of swallowing. The subsequent descent of the hyoid bone to its original resting position as the bolus enters the esophagus at the moment of the swallow is an objective index of the end of the oropharyngeal phase of swallowing. The duration of the oropharyngeal phase was therefore determined to be the time from the initial motion of the hyoid to its return to a resting position. Because ultrasonography provides clear images of the soft tissues, the tongue and floor muscles of the mouth can easily be imaged, and any unusual motions of the tongue or compensatory swallowing gestures can be examined.[21]

Ultrasonography was performed in 16 patients from each group (8 symptomatic and 8 asymptomatic). All 16 patients completed a standard swallowing protocol of three consecutive dry swallows followed by three consecutive wet swallows with a 10-ml bolus of water.[21,22]

Videofluoroscopy

The oropharynx was visualized with a modified barium swallow and fluoroscopy. Each study was recorded on videotape, and stop-frame analyses (Sony U-Matic VO-5800) were made to determine the presence and frequency of a set of clinical symptoms of oropharyngeal dysphagia (e.g., pharyngeal pooling, unilateral transport of the bolus of barium, aspiration, and laryngeal penetration of the bolus).[23]

Statistical Analysis

For the swallowing checklist, the chi-square test was used to compare the differences in the percentage of items checked by the symptomatic and the asymptomatic groups. A single-factor analysis of variance was used to compare the differences in the mean oral motor index scores among symptomatic and asymptomatic patients and normal controls. For all pairwise examinations, the t-test was used to compare the differences in the mean durations of the dry and wet swallows between symptomatic and asymptomatic patients and between all patients and normal controls. For all analyses a P value of 0.05 or less was considered to indicate statistical significance.

Results

Frequency of New Dysphagia and Relation to the Original Bulbar Disease

Of the 72 patients evaluated for the post-polio syndrome, 24 (33 percent) had involvement of the bulbar muscles during the original paralytic illness. Twenty-four (33 percent) also reported new swallowing difficulties; 18 of these symptomatic patients (75 percent) had previous involvement of the bulbar musculature, and 6 (25 percent) had nonbulbar (spinal) paralytic disease during the initial illness. The frequency of these symptoms in relation to previous bulbar involvement was similar in the group of 32 patients selected at random for detailed studies from our cohort of 72 patients. New swallowing difficulties were reported by 14 of the 32 patients (44 percent); 12 (38 percent) had involvement of the bulbar muscles during the original disease. New swallowing difficulties were noted by 9 of the 12 patients (75 percent) with previous bulbar involvement and by 5 of the 20 patients (25 percent) with nonbulbar (spinal) paralytic poliomyelitis.

Although some patients in the asymptomatic group were aware of a feeling of discomfort after meals due to a hiatal hernia or peptic ulcer or had ill-fitting dentures and difficulty chewing, none indicated on the checklist any awareness of difficulty with the oral or pharyngeal phase of swallowing. In contrast, all patients in the symptomatic group reported a variety of symptoms of oral and oropharyngeal dysphagia. There was a significant difference ($P<0.001$) between the two groups in the percentage of items checked. The asymptomatic group checked 0 to 19 percent of the 18 items, and the symptomatic group checked 20 to 61 percent of the items.

Oral Motor Examination

Although some patients were symptomatic and some were not, we found signs of mild-to-moderate abnormalities of oral motor function in all but one of them. As compared with normal values, the values were significantly different ($P<0.001$) for 6 of the 10 measures (Table 1). Both groups of patients had mild-to-moderate deficits in tongue, lip, palate, and jaw movement, slowing of oral diadochokinesia, and a mild-to-moderate reduction in the strength of the tongue, lips, and jaw, with changes in the pitch and volume of speech. There was also a significant difference ($P<0.001$) in swallowing ability between the symptomatic patients and the asymptomatic patients, with index scores of 2.32 and 2.31, respectively (Table 1). Among all studied patients, whether symptomatic or asymptomatic, those with bulbar involvement during the original illness had a high mean oral motor index score of 1.95 (range, 1.4 to 2.9), as compared

Table 1. Mean Oral Motor Index Scores in Patients with the Post-Polio Syndrome.*

Oral Motor Function	Oral Motor Index Score		
	Symptomatic Patients (N = 14)	Asymptomatic Patients (N = 18)	Controls (N = 12)
Oral structure	1.36±0.63	1.25±0.59	1
Symmetry	1.80±0.69	1.60±0.77	1†
Volitional and reflexive movement of tongue, lips, jaws, tongue	2.30±0.61	1.78±0.67	1‡
Tongue and lip strength	2.50±0.44	1.67±0.77	1‡
Oral sensation	1.32±0.72	1.08±0.06	1
Swallowing ability	2.82±0.42	2.31±0.54§	1‡
Fluency	1.13±0.02	1.13±0.03	1
Voice quality	2.11±0.74	1.64±0.61	1¶
Speech articulation	1.11±0.32	1.14±0.36	1
Oral diadochokinesia	1.82±0.87	1.25±0.39	1¶

*Plus–minus values are mean ±SD scores for 10 measures of oral sensorimotor activity. A score of 1 indicates normal oral motor function; 2, a mild deficit; 3, a moderate deficit; and 4, a severe deficit.

†P<0.09 for the comparison with values for both symptomatic and asymptomatic patients.

‡P<0.0001 for the comparison with values for both symptomatic and asymptomatic patients.

§P<0.001 for the comparison with values for symptomatic patients.

¶P = 0.001 for the comparison with values for both symptomatic and asymptomatic patients.

with a score of 1.53 (range, 1.0 to 2.4) for the patients with nonbulbar (spinal) paralytic disease.

Ultrasonography

There were significant differences (P<0.05) in the duration of the wet swallow between symptomatic and asymptomatic patients and between symptomatic patients and controls (Table 2). The mean (±SD) time required to swallow three 10-ml boluses of water was 2.67±0.70 seconds for the symptomatic group, 1.65±0.42 seconds for the asymptomatic group, and 1.67±0.88 seconds for the controls (P<0.05). Although the duration of dry swallows was not significantly different in the three groups (Table 2), the patients required more time to complete a dry swallow than the normal subjects.

Videofluorography

Videofluorography revealed a variety of signs of oropharyngeal dysphagia in both groups of patients (Table 3). The most common sign of dysfunction in both groups was impaired tongue activity: the patients required a disproportionate number of tongue-pumping gestures to initiate a swallow. This occurred in 64 percent of the symptomatic patients and 55 percent of the asymptomatic patients. Uncontrolled bolus flow into the pharynx due to impaired tongue movements was noted in 50 percent of the symptomatic and 17 percent of the asymptomatic patients. Unilateral transport of the bolus through the pharynx was the second most common sign in the symptomatic group, occurring in 57 percent of the patients (Fig. 1A); this sign was also seen in 22 percent of the asymptomatic group. Delayed esophageal motility was noted in 57 percent of the symptomatic patients and 39 percent of the asymptomatic patients. Pooling in the valleculae and pyriform sinuses was common in both groups of patients (Fig. 1B) and was observed in 43 percent of the patients in the symptomatic group. In the asymptomatic group, 44 percent had pooling in the pyriform sinus and 39 percent had pooling in the valleculae (Fig. 2). Bilateral pooling in the pyriform sinuses, as well as overflow into the laryngeal vestibule without aspiration, was found only in the symptomatic group. Other common signs in both groups were delayed initiation of the swallowing reflex in 43 per-

Table 2. Duration of Wet and Dry Swallows in Patients with the Post-Polio Syndrome.*

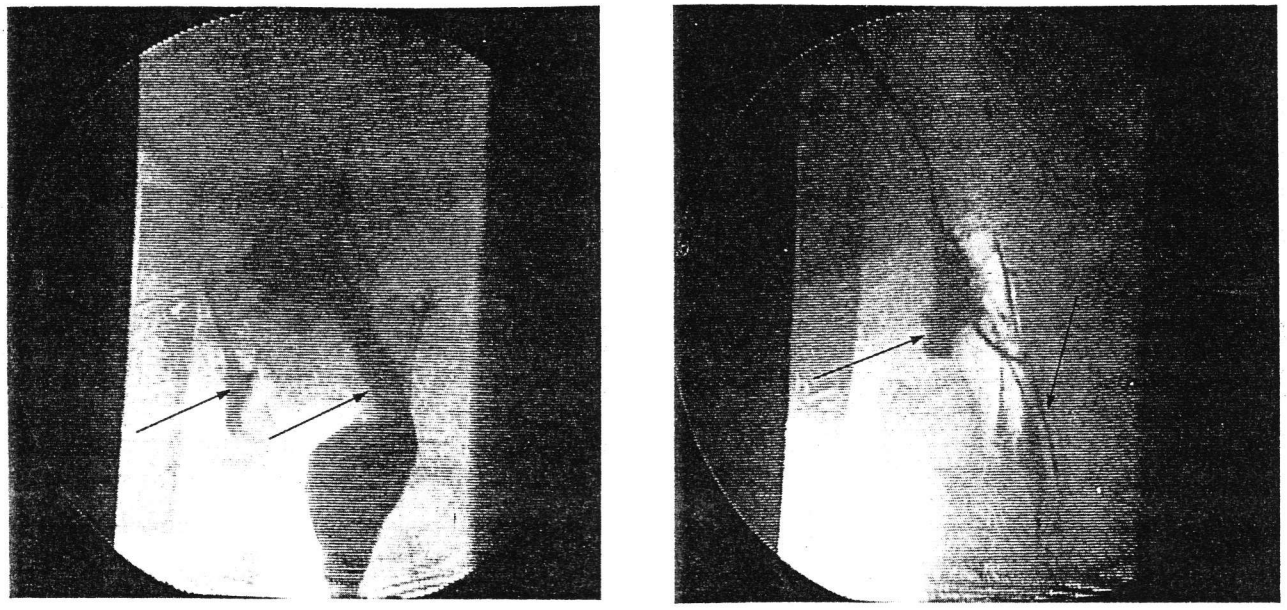
Type of Swallow	Duration of Swallow		
	Symptomatic Patients (N = 9)	Asymptomatic Patients (N = 11)	Controls (N = 12)
	seconds		
Wet	2.67±0.70†	1.65±0.42	1.67±0.88
Dry	2.55±0.84	2.97±1.98	2.13±1.13

*Plus–minus values are means ±SD. Values in each group were calculated from a mean of three swallows in each subject (see text for details).

†P<0.05 for the comparison with values for asymptomatic patients and for controls.

Table 3. Signs of Oropharyngeal Dysphagia in Patients with the Post-Polio Syndrome.

Signs of Dysphagia	Symptomatic Patients (N = 14)	Asymptomatic Patients (N = 18)
	no. (%)	
Tongue pumping and tongue gestures	9 (64)	10 (55)
Unilateral bolus transport through pharynx	8 (57)	4 (22)
Delayed esophageal motility	8 (57)	7 (39)
Uncontrolled bolus flow into pharynx	7 (50)	3 (17)
Pooling in valleculae	6 (43)	7 (39)
Pooling in pyriform sinuses	6 (43)	8 (44)
Delayed initiation of swallowing reflex	6 (43)	4 (22)
Delayed pharyngeal constriction	5 (36)	5 (28)
Impaired tongue activity	4 (28)	1 (5)
Difficulty swallowing liquids	4 (28)	2 (11)
Esophageal reflux	4 (28)	6 (33)
Overflow into laryngeal vestibule without aspiration	2 (14)	0 (0)
Pooling in cricopharyngeal area	2 (14)	2 (11)
Nasal reflux	2 (14)	2 (11)
Trace aspiration after swallowing	1 (7)	1 (5)



A B

Figure 1. Videofluorogram of the Oropharynx of a 37-Year-Old Symptomatic Man.

In Panel A, the anteroposterior view, there is poor bolus control, with pooling in the right pyriform sinus (left arrow) and unilateral transport of the barium bolus through the pharynx (right arrow). In Panel B, the lateral view, there is uncontrolled bolus flow, pooling in the valleculae (left arrow), and coating of the pharyngeal walls after the barium swallow (right arrow). The valleculae are located in the hypopharynx at the base of the tongue above the epiglottis; the pyriform sinuses are located in the laryngopharynx at the level of the false vocal folds. Both are cul-de-sacs that protect the bolus from overflowing into the laryngeal vestibule.

cent of the symptomatic patients and 22 percent of the asymptomatic patients and delayed pharyngeal constriction in 36 percent and 28 percent, respectively. Twenty-eight percent of the symptomatic patients and 11 percent of the asymptomatic patients had difficulty swallowing liquids. Pooling in the cricopharyngeal area was noted in 14 percent of the symptomatic patients and 11 percent of the asymptomatic patients. Nasal reflux was seen in two patients in each group, and trace aspiration after swallowing occurred in one patient in each group. Four of the symptomatic patients (28 percent) and six of the asymptomatic patients (33 percent) had esophageal reflux. The nonspecific finding of heartburn and indigestion was noted in four patients (28 percent) in the symptomatic group and in six patients (33 percent) in the asymptomatic group. One patient from the asymptomatic group had none of the signs of dysphagia on videofluorography.

Follow-up

Follow-up examinations were performed in four patients, each of whom had previous bulbar involvement, after 1 to 2.5 years (mean, 2.0) (Table 4). Patient 1, who was initially asymptomatic, still reported no symptoms two years later in spite of a subtle worsening of the oral motor index score and of the results of swallowing studies. Patient 2, who was initially asymptomatic, became aware one year later of mild difficulties with [illegible]. This was consistent with the finding of new mild-to-moderate weakness of the tongue and jaw and a worsening of the oral motor index score from 1.35 to 2.00. Patient 3, who was also asymptomatic initially, became symptomatic two years later. Her clear perception of new difficulties in oropharyngeal function was consistent with

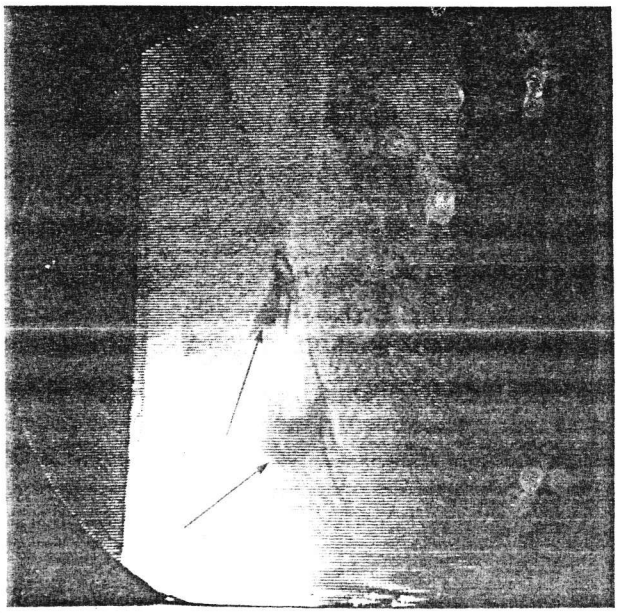


Figure 2. Lateral View of a Videofluorogram of the Oropharynx of a 45-Year-Old Asymptomatic Woman.

Note pooling in both the valleculae (top arrow) and the pyriform sinus (bottom arrow) after a barium swallow.

progression of the objective measures of dysphagia. Patient 4, who was initially symptomatic and had signs of reduced epiglottal descent without aspiration (Fig. 3), reported increased difficulty swallowing, which was confirmed by worsening of the oropharyngeal signs and the development of aspiration two years later.

Discussion

Objective analysis of oropharyngeal signs in patients with the post-polio syndrome, whether symptomatic or asymptomatic, revealed frequent abnormalities of swallowing. Symptomatic and asymptomatic patients had similar findings on the oral motor examination that were unrelated to a history of bulbar involvement during the original illness. The patterns of dysphagia on ultrasonography and videofluoroscopy were similar in the symptomatic and asymptomatic patients, but not surprisingly, the latter were not as severely affected. Follow-up of four patients two years later showed objective signs of worsening that corresponded to new clinical symptoms. These findings suggest that, as in the limb muscles, the bulbar muscles in patients who have had polio show subclinical signs of slowly progressive and quantifiable dysfunction that, after a certain degree of deterioration, become symptomatic and are perceived as difficulty swallowing.

The difference in the duration of wet swallows between the symptomatic and the asymptomatic patients appears to be related to the higher incidence of delayed initiation of the swallowing reflex coupled with excessive pumping of the tongue and lingual movements in the patients with symptoms. The higher incidence of pooling in the valleculae and pyriform sinuses, delayed pharyngeal constriction, and uncontrolled bolus flow into the pharynx is most likely related to the higher degree of paresis of the oropharynx noted in the symptomatic group. This paresis may cause the bolus to move asymmetrically and with reduced force.

Aspiration was rare in all patients, suggesting that the airway is adequately protected and that hyoid and epiglottic activity is largely unaffected, at least in the early years after the onset of post-poliomyelitis muscular atrophy. This may be due to the patients' ability to compensate for the longstanding dysfunction of the bulbar muscles by using other accessory muscles or by a subconscious adjustment of their swallowing posture. It is possible that minor episodes of aspiration pneumonia occurred but were not symptomatic because of the ability of the lungs to clear small amounts of aspirated material.

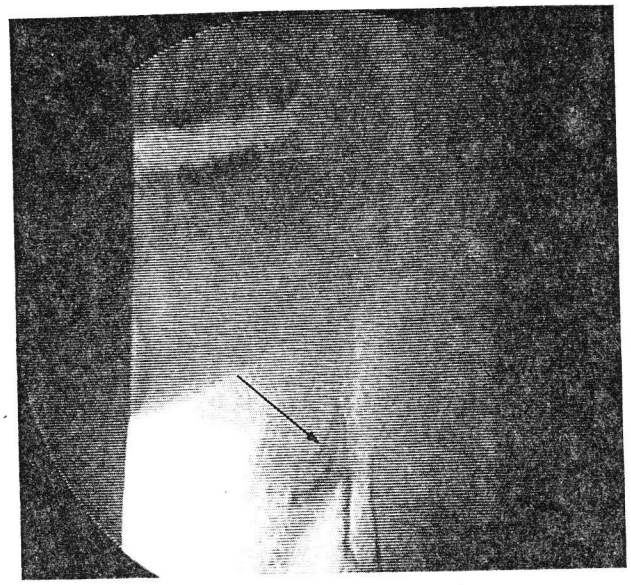


Figure 3. Lateral View of a Videofluorogram of the Oropharynx of a 37-Year-Old Symptomatic Man.

Note the absence of epiglottal descent after a barium swallow that obliterates the vallecular space (arrow). The airway remains protected. This patient had minimal aspiration on follow-up examination two years later (see text for details).

The post-polio syndrome is a clinical diagnosis, and there is concern about the lack of objective evidence of new signs of dysfunction and deterioration.[1-11,15,16] Electrophysiologically, there is instability with unstable neuromuscular synapses even in the muscles of clinically stable patients who have had paralytic polio.[4,7,11,15] Histologically, there are signs of new and old denervation in the muscle-biopsy specimens, as well as signs of inflammation, with neuronal chromatolysis in the spinal cord of all the patients regardless of whether there are new symptoms.[1-8,16,24] These observations have provided a strong argument that post-poliomyelitis muscular atrophy is an appropriate designation for the clinically apparent late stage of an ongoing neuronal reaction that is subclinical for many years until neuronal reserves substantially diminish, new motor-nerve ter-

Table 4. Follow-up of Patients with the Post-Polio Syndrome for Signs of Oropharyngeal Dysphagia.

Patient No.	Age/Sex	Clinical Findings		Oral Motor Index		Signs of Dysphagia
		Initial	Follow-up*	Initial	Follow-up*	
1	37/M	Asymptomatic	Asymptomatic	2.00	2.58	More severe pharyngeal and esophageal signs
2	59/M	Asymptomatic	Symptomatic	1.35	2.00	New signs of mild-to-moderate weakness of tongue and jaw
3	45/F	Asymptomatic	Symptomatic	1.91	2.16	New symptoms: gurgling, gasping, pharyngeal signs
4	37/M	Symptomatic	Symptomatic	1.91	2.25	Worsening of symptoms, increased pooling in pharynx, development of minimal aspiration

*Follow-up ranged from 1 to 2.5 years (mean, 2.0).

minals cannot survive, and further reinnervation cannot be supported.[1-8,16,24] Our findings of subclinical bulbar dysfunction and new dysphagia suggest that the same phenomena that we observed in the limbs are also operative in the bulbar muscles. Bulbar symptoms have been observed in at least 15 percent of patients with acute paralytic polio, but on autopsy, subclinical involvement of the bulbar nuclei is a more common finding.[13,14] This observation may explain the subclinical dysfunction we found even in muscles historically spared during the acute illness. New dysphagia was more severe in patients with previous bulbar involvement, implying an earlier exhaustion of the already overtaxed remaining bulbar motor neurons.

In the limb muscles, the new weakness cannot be measured accurately, and the margin separating the clinically stable state from that associated with clinically overt signs of weakness cannot be defined.[7,8,11,15,16] By contrast, our study shows that new bulbar dysfunction can be quantified and the threshold beyond which weakness ensues can be defined by an objective clinical score. Findings from the symptomatic and asymptomatic groups (Table 1) and the follow-up study (Table 4) indicate that a clinically critical point associated with new symptoms of oropharyngeal dysfunction is reached when the oral motor index score is ≥ 2. The oral motor index score may therefore be helpful in assessing whether the observed abnormalities pose a high risk of symptomatic dysphagia and possible aspiration, requiring therapeutic intervention, or whether they are clinically unimportant. Furthermore, long-term monitoring of bulbar function, with the use of the objective and quantifiable methods described, not only may predict impending dysphagia but also may help to assess the progression of the disease accurately. The rate of progression of post-poliomyelitis muscular atrophy is quite variable. With the rather subjective manual testing of the limb muscles based on the Medical Research Council scale, the annual progression may range from 1[4,5,7] to 7 percent,[25] or according to a quantitative measurement,[26] there may even be no progression.

Symptoms of dysphagia are progressive and appear to be more severe in patients who had bulbar involvement during the original attack of polio. Therefore, periodic dynamic imaging studies, regardless of whether the patients are aware of new symptoms or not, may be warranted to identify progressive dysfunction and to protect patients from unexpected and potentially life-threatening aspiration. For seven of the symptomatic patients that we followed, simple modifications in swallowing position, a change in diet to foods with medium-to-soft consistencies, and the introduction of a variety of compensatory techniques[27-29] effectively improved their ability to swallow safely.

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