

## Outcomes of Swallowing Rehabilitation in Chronic Brainstem Dysphagia: A Retrospective Evaluation

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**Abstract.** This study examines the functional and physiologic outcomes of treatment in a group of 10 patients with chronic dysphagia subsequent to a single brainstem injury. All patients participated in a structured swallowing treatment program at a metropolitan teaching hospital. This program differs from more traditional swallowing treatment by the inclusion of surface electromyography biofeedback as a treatment modality and the completion of 10 hr of direct treatment in the first week of intervention. A retrospective analysis of medical records and patient questionnaires was used to gain information regarding medical history, site of lesion, prior interventions, and patient perception of swallowing recovery. Physiologic change in swallowing treatment, as measured by severity ratings of videofluoroscopic swallowing studies, was demonstrated in nine of 10 patients after 1 week or 10 sessions of treatment. Functional change was measured by diet level tolerance after 1 week of treatment, at 6 months, and again at 1 year posttreatment. Eight of the 10 patients were able to return to full oral intake with termination of gastrostomy tube feedings, whereas two demonstrated no long-term change in functional swallowing. Of the eight who returned to full oral intake, the average duration of tube feedings following treatment until discontinuation was 5.3 months, with a range of 1–12 months. Six patients who returned to oral intake maintained gains in swallowing function, and two patients returned to nonoral nutrition as the result of a new unrelated medical condition.

**Key words:** Dysphagia — Brainstem — Rehabilitation — SEMG biofeedback — Efficacy — Deglutition — Deglutition disorders.

Individuals with brainstem injury are particularly susceptible to oral pharyngeal swallowing deficits. Although dysphagia secondary to cortical stroke frequently improves to functional levels or resolves completely as a function of time and spontaneous recovery, this trend is not as frequently observed in the dysphagia subsequent to brainstem injury [1–3]. The compact clustering of cranial nerve nuclei, nerve tracts, and reticular interneurons within and between brainstem structures that are critical to swallowing [4] has the potential to produce dysphagia that is severe and resistant to spontaneous recovery. Thus, the need for development and substantiation of behavioral and rehabilitative interventions is of particular consequence to this population.

Current state of the art in the management of dysphagia includes a wide variety of multidisciplinary practices. These practices range from simple diet adjustment to more invasive surgical interventions, depending on the nature and severity of the disorder. Because management of dysphagia is a relatively new specialization, the direct therapeutic interventions offered by the speech language pathologist have consisted largely of compensatory mechanisms by which the patient compensates for disordered swallowing with adjustments in diet consistency or behaviorally based maneuvers. These behaviorally based maneuvers include techniques designed to improve airway protection, such as the supraglottic swallow, and maneuvers designed to facilitate transfer of the bolus through the pharyngeal cavity by maximizing gravitational forces and minimizing resistance to the bolus, such as chin-tuck posturing [5]. These interventions, although serving an important role, provide an immediate but only transient approach to the underlying physiologic deficit. They are compensatory in nature. Development of rehabilitative exercises and substantiation of efficacy are needed to address long-term changes in swallowing physiology.

Several techniques have been proposed as com-

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pensatory in intent, but clinical experience suggests that these techniques, when repeated in the context of an exercise regime, may facilitate overall change in swallowing physiology. The effortful, or modified valsalva, swallow consists of a normal swallow in duration and sequence but with greater effort or force during the muscle contraction [6,7]. This technique was initially conceptualized to facilitate bolus clearance from pharyngeal recesses as a result of increased tongue-base approximation to the posterior pharyngeal wall. However, no identified study has documented the long-term effects of the maneuver. The Mendelsohn maneuver [8] was designed to address deficits associated with reduced opening of the upper esophageal sphincter. Execution involves the prolongation of a pharyngeal swallow when the thyroid cartilage is at its point of highest excursion, with the intent of maintaining opening of the upper esophageal sphincter. More recently, a tongue-holding maneuver, or the Masako maneuver, was developed based on the observation that patients with base of tongue resection demonstrate spontaneous increase in posterior pharyngeal wall excursion to compensate for the resected base of the tongue [9]. Further research has indicated that in normal swallows, execution of this maneuver significantly facilitates posterior pharyngeal wall movement and thus may provide a direct rehabilitative exercise to address pharyngeal contraction [10]. The head-lifting exercise is the newest technique in the arsenal of interventions for dysphagia, presented by Shakir et al. [11,12]. This study demonstrated that a very simple technique of isotonic and isometric head-lifting movements from a supine position resulted in facilitation of cricopharyngeal sphincter opening and decreased bolus pressure when compared with a nontherapeutic placebo exercise.

Although the immediate, short-term effects of the Mendelsohn and tongue-holding maneuvers and the long-term effects of the head-lifting maneuver have been documented in isolation, there have been few studies that document the cumulative effects of a rehabilitation program on abnormal swallowing physiology. This is particularly the case in the patient population with chronic brainstem dysphagia given its resistant nature to spontaneous recovery [2].

Logemann and Kahrilas [13] provided a case report of a single 45-year-old patient with severe dysphagia secondary to medullary infarct who presented months postonset at the initiation of intervention. A series of compensatory maneuvers, including head rotation, supraglottic swallow, and the Mendelsohn maneuver, was provided sequentially, beginning at 4 months postonset and continuing over the course of 4 years. The short-term physiologic effects of these techniques were documented by videofluoroscopy. Ultimately the patient was able to

return to a full oral diet with discontinuation of gastrostomy tube feedings at 50 months postonset. Although this study provides valuable information about the effects of isolated compensatory maneuvers on swallowing efficiency in a single patient with chronic dysphagia subsequent to brainstem injury, it does not address cumulative effects from rehabilitative efforts.

Neumann and colleagues published a series of papers reporting on the efficacy of intervention in a large group of patients with dysphagia of neurogenic origin [14–16]. The initial study [14] reported on a group of 66 patients with dysphagia subsequent to a broad range of neurologic etiologies, including brainstem and cortical stroke, tumor, trauma, Parkinson's disease, amyotrophic lateral sclerosis (ALS), and cerebellar ataxia. At the initiation of intervention, 61% were on exclusive tube feedings with no oral intake. Using a combination of rehabilitative and compensatory mechanisms, only 8% remained on full nonoral feedings, with 74% tolerating full oral feedings posttreatment. The median duration of treatment was 17 weeks, with a range of 1–60 weeks. The time postonset at the initiation of treatment ranged from 2 weeks to 32 years, with the median time postonset of 18 weeks. Five of the patients were reported to be between 5 and 32 years postonset. Although it was not specifically reported how this subgroup responded to intervention, chi-square analysis suggested that time postonset did not correlate with outcome of treatment. Although functional outcome in terms of diet adjustment is valuable information, this study did not provide information about physiologic outcomes or severity pre- and posttreatment. No information was provided about long term follow-up.

These findings were supported in an additional study by Neumann et al. [15] in which the outcome of treatment on 58 patients who were on nonoral feedings secondary to neurologic disease was presented. For this study, the median time postonset was 10 weeks, with a range of 3–156 weeks. Provision of treatment was better controlled in this study, with subjects receiving therapy 5 days weekly for 1-hr sessions. Again, by using a combination of rehabilitation and compensation in the treatment plan, 67% of the patients returned to oral feedings after a median of 15 weeks of treatment (range = 2–52 weeks). A subset of 11 patients with time postonset of greater than 2 years had a similar success rate of 64%, again supporting that lengthy time postonset is not contraindicated for swallowing recovery. As with the prior study, no information was provided about long-term follow-up, severity of the disorder, or physiologic effects of intervention based on videofluoroscopy.

A third study by Bartolome and Neumann [16] reported on a subgroup of the neurologically impaired patients in their previous study [14], with specific dys-

function associated with cricopharyngeal sphincter opening. Of this subgroup of 28 patients, treatment was initiated from 5 weeks to 5.3 years postonset, with a median of 17.5 weeks. Nonoral feeding was required in 21 of the patients due to the severity of the disorder. Treatment was provided for a median of 16 weeks (range = 2–52 weeks) and included the Mendelsohn maneuver, supra-glottic swallow, dietary adjustment, and head positioning. Of this group, 65% demonstrated gains as shown by upgrades in diet or route of nutrition, and an additional 25% reported subjective improvement without associated improvement in diet tolerance. Although this study provided useful information, no information was provided about long-term follow-up, severity of the disorder pre- or posttreatment, or physiologic effects of intervention based on videofluoroscopy.

The afore-mentioned studies, particularly those with data from large clinical samples, provide valuable information regarding efficacy of dysphagia management services. In particular, data collected from a relatively large sample of neurologically impaired patients is valuable. However, further questions beg to be addressed directly. Efficacy research that includes mixed diagnoses and a wide variation in time postonset cloud the issue of spontaneous recovery versus the rehabilitative effects. Although several of the prior studies have addressed this statistically, efficacy research limited to patients with “chronic” dysphagia would be of benefit to factor out spontaneous recovery. More specifically, defining the nature and frequency of the treatment provided would also be helpful. There is support that some type of intervention provides positive outcomes, but replication in clinical practice with similar results will be more difficult without greater control and definition. Outcome of treatment as measured by functional tolerance of diet is of benefit in the clinical realm; however, concomitant documentation of physiologic change is needed to specifically identify the neuromuscular effects of the treatment. Finally, documentation of long-term follow-up would be advantageous. Are the benefits derived from therapy stable or, after the return to oral feeding and discontinuation of treatment, is there a regression in swallowing function?

Crary [17] addressed many of these issues in a report on the outcome of a direct intervention program for chronic neurogenic dysphagia secondary to brainstem stroke. Six patients with time postonset ranging from 5 to 54 months were on gastrostomy tube feeding prior to intervention. Specific findings from clinical and videofluoroscopic examinations were reported. All received dysphagia treatment using surface electromyographic (SEMG) biofeedback monitoring of swallowing rehabilitation exercises. Frequency of treatment was initially daily treatment for all but one patient, and duration of

treatment ranged from 3 weeks to 7 months. Of the six patients, all had received some prior swallowing therapy. At the conclusion of treatment, five of the six patients had resumed total oral nutrition. At follow-up, between 18 and 24 months posttreatment, all patients continued with full oral feedings with the gastrostomy tube removed.

An adequate understanding of swallowing recovery as a response to intervention optimally requires four criteria, according to Logemann [18]. These criteria include (a) quantification of the disorder and outcome of treatment, (b) standardization of the testing procedures, (c) careful selection of the patient population to be studied, and (d) definition of the treatment protocol. Crary [17] addressed more of these criteria than did prior studies by documenting pretreatment physiology on videofluoroscopy, addressing a single etiologic population that was past the acute recovery period, standardizing the nature of the treatment, and documenting maintenance of gains through long-term follow-up. However, despite the significant contribution of the Crary study in addressing these criteria, there remain some unanswered questions.

Although Crary documented pretreatment status on videofluoroscopy, posttreatment status was not similarly evaluated; thus, the functional gains that were reported cannot be specifically attributed to a change in underlying physiology and may be explained by compensatory swallowing behaviors. Crary attempted to address this issue by evaluating patterns of SEMG activity during swallowing recovery. However, so little is known about SEMG recordings and their direct correlation with physiologic features that the interpretation of this information is questionable. No report has been provided regarding functional change in terms of pulmonary function. Functional change regarding diet was addressed at the termination of treatment. Although the patients in this study were reported to return to full oral intake and the duration of treatment was reported, the duration of time until return to full oral intake was not clearly defined. Three patients in that study were reported to receive subsequent interventions following the discontinuation of treatment, suggesting an incomplete recovery subsequent to the rehabilitation program. Clarification of extent and duration of recovery would be of interest.

The purpose of the present report, therefore, was to provide a partial replication and extension of the study by Crary [17] to evaluate additional relevant clinical issues. The present retrospective report documents outcomes of swallowing rehabilitation in a group of patients with chronic dysphagia subsequent to brainstem injury. This study provides further information regarding the severity of dysphagic physiology pre- and posttreatment as measured by videofluoroscopy, duration of tube feeding following intervention, documentation of pulmonary status pre- and posttreatment, and controls for the fre-

quency and intensity of treatment provided. Data were collected through a retrospective review of medical records, videofluoroscopic swallowing studies, and a follow-up patient questionnaire.

## Methods

### *Research Participants*

Ten patients with chronic dysphagia subsequent to brainstem injury served as participants in this study. These patients were selected to meet several criteria. To eliminate the contaminating effects of spontaneous recovery on treatment efficacy, all patients selected for inclusion in the study were at least 8 months postonset, with no substantial recovery of swallowing function. No patient had a history of preexisting dysphagia or neurologic disease prior to the onset of the current disorder.

The characteristics and diagnosis of each patient are summarized in Table 1. There were seven men and three women, ranging in age from 42 to 76 years, with a mean age of 62 years. Seven of the 10 patients were diagnosed with dysphagia subsequent to brainstem infarct. Two of those were confirmed by magnetic resonance imaging (MRI) and five were determined by neurodiagnosis of the attending neurologist. Neurodiagnosis by the physician was based on presentation of symptoms in the absence of confirmatory radiographic findings on computed tomography (CT) when MRI was not available. The three remaining patients presented dysphagia subsequent to tumor compression and/or resection. Two were diagnosed and received resection of a foramen magnum meningioma, and the final patient was diagnosed and received resection of a large foramen of Luschka choroid plexus papilloma with involvement of cranial nerves 9–12. Seven of the 10 patients had received prior swallowing therapy services without measurable change in swallowing physiology or functional return to oral intake. The three patients who did not receive prior treatment were those with the shortest time postonset. At the initiation of the Outpatient Accelerated Swallowing Treatment program, the mean time postonset of dysphagia was 26.9 months, with a range of 8–84 months. Eight of 10 patients were greater than 1 year postonset of dysphagic symptoms.

### *Procedures*

Data for this retrospective report were collected through a review of medical records, videofluoroscopic swallowing studies, and a patient completed questionnaire (Appendix 1). All patients participated in a near identical outpatient rehabilitation program at a major university teaching hospital. The Outpatient Accelerated Swallowing Treatment Program was developed to target those patients with chronic dysphagia who had failed prior intervention efforts. The primary differentiating features of this program were the requirement of 10 hr of direct rehabilitation within the first week of treatment, paired with a rigorous home program and the utilization of SEMG biofeedback monitoring as an adjunct to treatment. The treatment program policies and procedures are fully outlined in Appendix 2.

Prior to acceptance in this outpatient program, history information and a video copy of the most recent diagnostic swallowing study were reviewed to determine the appropriateness of treatment and develop the treatment plan. Patients who were accepted into the program received 1 hr of direct therapy each morning and 1 hr of direct therapy each mid-afternoon, with an intervening 3–4 hour rest period, for 5 consecutive days. Thus, this accelerated treatment course consisted of

10 sessions within a 1-week period. A rigorous home program was prescribed which included three additional independent sessions of 15 min duration. Home programming activities during the week of intensive treatment were reviewed daily through discussion with the patient and family and review of computer-stored biofeedback tracings saved after each home session. Although specific treatment plans for the patients in this study were individualized to reflect the needs of each patient, the physiologic deficits were quite similar for all patients; thus, the treatment provided was likewise quite similar. The following physiologic abnormalities were prevalent across patients: weakened and/or inefficient pharyngeal contraction with postswallow diffuse residual, severely impaired passage of the bolus through the upper esophageal sphincter with pronounced postswallow pyriform sinus residual, postswallow aspiration of liquids and occasionally puree, and strong volitional airway protection in the absence of a consistent reflexive cough. As appropriate for each patient's impaired physiology, treatment focused on concentrated repetition of swallowing rehabilitative maneuvers including the effortful, or modified valsalva, swallow [6,7], the Mendelsohn maneuver [8], and for some patients the Masako maneuver [10] and head-lifting maneuver [11,12]. For all patients, SEMG biofeedback was heavily used to facilitate teaching and execution of the therapy tasks. For some patients, auditory feedback of the acoustic signal associated with swallowing was also provided. Because instrumental biofeedback modalities are not typical components of rehabilitation of swallowing, they will be discussed in further detail in the next paragraph. In addition to the execution of rehabilitative exercises, direct oral intake was included as a component of the treatment regime as soon as possible for airway protection. This component of treatment was considered critical for the neurosensory stimulation it provided to the oropharyngeal cavity and the sense of meaningful progress it provided the patient. Attempts at oral intake also provided the benefit of early incorporation of transfer of training to functional tasks. During therapeutic oral feeding, airway-protection maneuvers and compensatory strategies were reinforced to maximize airway protection and bolus transfer. All patients were realistically counseled regarding the risks associated with aspiration pneumonia and were requested to make an independent decision as to whether oral trials would be included as a component of their treatment. All patients expressed a desire to proceed with oral trials with compensatory strategies. After the initial first week of concentrated rehabilitation, a repeat videofluoroscopy was performed to assess changes in swallowing physiology. If measurable change was documented, the patients were recommended for follow-up treatment on a schedule of once or twice weekly. Despite recommendations for follow-up and referrals to clinicians in the geographic region of the patients, only three of the 10 patients continued formal, direct treatment after the initial week. All patients, however, reported that they continued rehabilitative exercises independently through a home program.

To foster rapid mastery of rehabilitative exercises and provide measurable short-term objectives, exteroceptive biofeedback was used extensively. Biofeedback modalities have been evaluated extensively in other realms of physical medicine and rehabilitation, with numerous studies demonstrating clinical efficacy for a variety of neuromuscular disorders. Wolf documented more than 300 clinical studies addressing the efficacy of SEMG biofeedback in physical rehabilitation of neuromuscular disorders, not including associated disorders of pain [19]. Wolf further posited that utilization of SEMG during motor relearning provides an alternative sense of proprioception for the patient with neurological disorders, by allowing the patient greater insight into the sensory recognition of motor patterns [19]. Kasman [20] added that biofeedback serves as "an extension of the patients or clinicians senses." As the patient begins to reestablish sensory mechanisms, the necessity for external feedback diminishes with increased reliance on internal feedback mechanisms. This type of information may be par-

**Table 1.** Summary of research participant characteristics

Subject	Age at start of treatment	Months after onset	Etiology	History of pulmonary illness	Prior therapy
1	68	12	Brainstem CVA <sup>a</sup>	Yes	Yes
2	74	18	Brainstem CVA <sup>a</sup>	Yes	Yes
3	76	13	Brainstem CVA <sup>a</sup>	Yes	Yes
4	61	84	Brainstem CVA <sup>a</sup>	Yes	Yes
5	72	60	Brainstem CVA <sup>a</sup>	No	Yes
6	51	8	Brainstem CVA <sup>a</sup>	No	No
7	74	26	Brainstem CVA <sup>a</sup>	Yes	Yes
8	48	28	Foramen magnum meningioma	Yes	Yes
9	54	12	Foramen of Luschka papilloma	No	No
10	42	8	Foramen magnum meningioma	No	No

<sup>a</sup>CVA = cerebrovascular accident.

ticularly useful in the patient with brainstem injury to facilitate integration of sensory information into the executable motor program for swallowing.

These theories of biofeedback application have been supported by empirical research. Adrian and Bonk [21], in a study of normal subjects, determined that the electrical responses in individual muscles provide an accurate reflection of the actual functional activity of the muscle, i.e., there is a direct linear correlation between the EMG tracing and muscle force. Reliable measurements of functional activity are prerequisite for clinical usefulness. Two other studies have provided early documentation of the subject's ability to consciously control the biofeedback tracing. Lindsley [22] and Smith [23] in a series of similar studies documented that subjects could exert conscious control on even smallest motor unit potential and at rest demonstrated no inherent muscular tension. In addition, they documented that normal subjects could achieve complete relaxation as demonstrated by no measurable motor response with little difficulty.

Evidence of the physiologic correlation of the SEMG signal and swallowing was provided by Sonies et al. [24] at the Fifth Annual Meeting of the Dysphagia Research Society. Using simultaneous ultrasound imaging and submental SEMG measurements, a very high correlation ( $R = 0.99$ ) was noted between peak EMG amplitude and maximal hyoid elevation during all bolus consistencies. This observation suggests that the peak EMG waveform indicates maximum submental muscle contraction and maximal hyolaryngeal elevation.

Two biofeedback modalities were emphasized with the patients described in the present report. SEMG biofeedback as an adjunct to dysphagia rehabilitation used primarily submental surface electrodes to monitor activity of the collective suprahyoid muscle group or those muscles largely responsible for laryngeal excursion during swallowing [25,26]. Using this electrode placement, the relative strength and timing of contraction of this muscle group was visually displayed on a computer screen for observation by the patient and clinician during treatment. The effortful swallow and Masako maneuver were optimally represented by a high peak amplitude of short duration, representing the rapid contraction and relaxation of the suprahyoid muscle group associated with swallowing. In contrast, the Mendelsohn maneuver was optimally represented with a rapid onset rise in amplitude, followed by a sustained high amplitude tracing for several seconds before the abrupt offset of amplitude associated with completion of the maneuver and return to resting posture. Although there are no valid normative values for SEMG signals at this location, for rehabilitative purposes the patient served as his or her own referent. The relative shape of the waveform targeted minimization of extraneous, nonfunctional motor activity, whereas the amplitude of the waveform targeted progressively increased peak thresholds until the dysphagic symptoms were resolved or

the patient reached a clinical plateau in waveform and amplitude. Auditory feedback was paired with the visual tracing of the waveform, with an electronic auditory signal, or "reward," provided when the patient achieved the targeted EMG threshold.

Additional auditory feedback of the acoustic signal associated with swallowing was provided for most patients by using an amplified stethoscope bell as a transducer. An inexpensive amplified speaker served as the output for a lapel microphone that was connected through flexible plastic tubing to the housing of a stethoscope bell. The stethoscope bell was placed over the lateral lamina of the thyroid cartilage to monitor the acoustic signal associated with swallowing. Although "cervical auscultation" has been the focus of significant recent research as a clinical diagnostic tool [27–31], its application as a biofeedback modality is significantly less complex and has not previously been reported in the literature. Execution of a Mendelsohn maneuver requires sustaining laryngeal excursion during the swallow or prolonging the swallow. Acoustic feedback was used to monitor tracheal sounds, in particular evaluating for the absence of air flow, during completion of this maneuver to facilitate correct execution. Acoustic feedback was also used during execution of supraglottic swallow to provide feedback regarding vocal fold adduction. The decision to use acoustic biofeedback was based primarily on the patient's response to the feedback and personal preference. In addition, the device for acoustic feedback was created and included in the therapeutic repertoire after some of the patients had already been seen for treatment, thus limiting its availability for the first patients accepted into the program.

### Outcome–Assessment

Pretreatment status and posttreatment progress were measured by using a combination of radiographic documentation of swallowing physiology and evaluation of functional ability to safely consume oral intake. Videofluoroscopic swallowing studies were obtained on all subjects before treatment and after 1 week of intensive treatment, or 10 therapeutic sessions. Additional videofluoroscopic swallowing studies after the immediate posttreatment were not available because they could not be justified based on the patient's clinical presentation. The admission videofluoroscopic swallowing study was completed within 2 weeks prior to onset of treatment for the two patients less than 1 year postonset, within 1 month for those patients between 12 and 28 months postonset, and within 6 months for the two patients who presented for treatment more than 28 months postonset of dysphagia. The posttreatment videofluoroscopy was completed on the afternoon of the last day of the intensive week of treatment. Videofluoroscopic swallowing stud-

ies were evaluated by using a 9-point equal-appearing interval scale, with a rating of 0 representing no significant dysphagia and a rating of 8 representing profound dysphagia. The overall severity rating was derived by selection of a series of 17 very specific physiologic severity descriptors, associated with impairment in oral, oral pharyngeal transit, pharyngeal, cricoesophageal, and laryngeal parameters. The overall severity rating thus represents an estimated "clinical average" of the physiologic severity ratings [32].

Functional outcome was measured by a rating of method of nutritional intake by using a 5-point scale and notation of respiratory symptomatology in the medical record. Nutritional level ratings were made at the initiation of the intensive treatment program, at the conclusion of 1 week of treatment, at a 6-month follow-up, and at the conclusion of data gathering for this project. The time after treatment of the final rating differed among subjects. All subjects were at least 1 year posttreatment, and several were more than 4 years posttreatment. The rating scale used is presented in Table 2. Indications of respiratory symptomatology related to aspiration were noted as either present or absent based on information gleaned from a review of medical records and patient reports of status posttreatment. In the pretreatment condition, those patients with pneumonia were diagnosed with aspiration pneumonia by their attending physician. In this retrospective study, no controls were provided for how that diagnosis was derived. In the posttreatment data, no incidence of pneumonia was reported, regardless of etiology.

## Results

### *Pretreatment Characteristics*

Table 3 summarizes the pretreatment characteristics of this group of subjects prior to completion of the Outpatient Accelerated Swallowing Treatment program. Videofluoroscopic severity levels ranged from moderate (level 4) to profound (level 8), with a median level of 6.5, or severe impairment. All 10 subjects demonstrated radiographic evidence of aspiration on the pretreatment videofluoroscopic swallowing study. The median diet level was 1, with all of the subjects fed via gastrostomy or jejunostomy. Only one of the 10 was able to tolerate oral intake of any texture, placing that subject at diet level 2 on initiation of treatment. With significant effort, this patient (no. 5) was able to ingest approximately 4 oz. of thin liquid in successive gulps but relied on nonoral feedings as the primary route of nutritional intake. Six of the 10 subjects reported at least one episode of diagnosed pulmonary symptoms related to aspiration which occurred following their discharge from acute rehabilitation, as diagnosed by their attending physician.

### *Posttreatment Characteristics*

Table 4 summarizes the characteristics of the subjects after 1 week, or 10 sessions of direct treatment. Based on the end-of-week videofluoroscopic swallowing study, nine of the 10 subjects demonstrated a measurable change in swallowing physiology. A comparison of pre-

**Table 2.** Scale for functional outcomes related to nutritional route

Level	Description
1	Feeding tube only; no oral intake
2	Feeding tube for primary nutrition; oral intake secondary
3	Oral intake for primary nutrition; feeding tube secondary
4	Oral intake only, feeding tube removed; restricted diet texture
5	Oral intake only, feeding tube removed; minimal texture restriction

and posttreatment swallowing severity based on videofluoroscopic studies is shown in Figure 1. Severity levels ranged from mild (level 2) to severe-profound (level 7), with a median severity level of 5.5, or moderate to severe.

Functional changes in recommended and tolerated diet levels were measured after 10 sessions, again at 6 months and at final outcome. Figure 2 summarizes these findings. At the conclusion of 10 treatment sessions, all but one subject (patient 5) demonstrated a change in diet level. The range of diet level ranged from feeding tube for primary nutrition with secondary oral intake (level 2) to oral intake for primary nutrition with feeding tube for secondary (level 3), with a median diet level of 2.0. Interestingly, the patient who failed to increase diet level after the initial week of treatment was the same patient who tolerated small quantities of oral nutrition at the initiation of treatment. The second posttreatment measurement at 6 months indicated that seven of the 10 patients continued with measurable progress and had their feeding tubes removed, two subjects remained at the same diet level, and one subject discontinued trial feedings and returned to full nonoral status. At 6 months, the range of diet level ranged from feeding tube for primary nutrition with secondary oral intake (level 2) to full oral intake of minimal texture restriction with feeding tube removed (level 5), with a median diet level of 4.0. The final diet level rankings, at long-term follow-up, indicated that six of the 10 subjects continued further progress and achieved full oral nutrition of a minimally restricted diet, two of the subjects who had achieved full oral intake with feeding tube removed required reinitiation of the tube feeding, and two patients demonstrated no long-term change in functional oral intake. Given that some patients continued to progress whereas others declined, the median diet level ranking was essentially unchanged from the prior measurement at 6 months (median level = 5.0).

As a measure of pulmonary tolerance of oral intake, the occurrence of pulmonary symptoms related to aspiration was noted (Table 4). Subsequent to participation in the swallowing treatment program, no patient reported symptoms of pulmonary illness as assessed at the

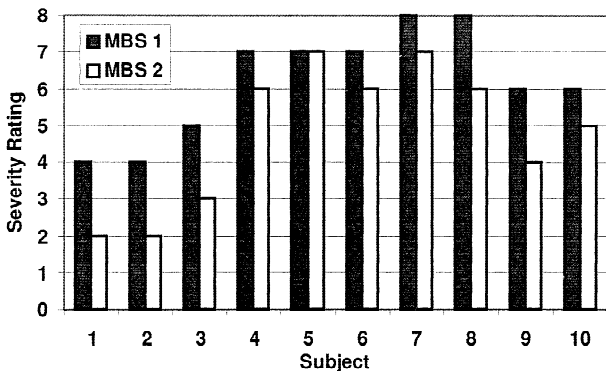
**Table 3.** Summary of subject pretreatment characteristics

Subject	Severity at start of treatment based on videofluoroscopy	History of aspiration-related pulmonary symptoms	Pretreatment functional diet level
1	Moderate level 4	Yes	Level 1, NPO <sup>a</sup>
2	Moderate level 4	Yes	Level 1, NPO <sup>a</sup>
3	Moderate to severe level 5	Yes	Level 1, NPO <sup>a</sup>
4	Severe to profound level 7	Yes	Level 1, NPO <sup>a</sup>
5	Severe to profound level 7	No	Level 2, with a tube, then orally
6	Severe to profound level 7	No	Level 1, NPO <sup>a</sup>
7	Profound level 8	Yes	Level 1, NPO <sup>a</sup>
8	Profound/absent level 8	Yes	Level 1, NPO <sup>a</sup>
9	Severe level 6	No	Level 1, NPO <sup>a</sup>
10	Severe level 6	No	Level 1, NPO <sup>a</sup>

<sup>a</sup>NPO = nothing by mouth.

**Table 4.** Summary of subject posttreatment characteristics

Subject	Severity rating at end of 10 sessions	Pulmonary symptoms by final outcome	Diet at 1 week	Diet at 6 months	Diet final	Feeding tube removed (months)
1	Mild level 2	No	3	4	5	4
2	Mild level 2	No	2	4	1	5
3	Mild to moderate level 3	No	2	5	1	4
4	Severe level 6	No	2	1	1	—
5	Severe to profound level 7	No	2	2	2	—
6	Severe level 6	No	2	2	5	13
7	Severe to profound level 7	No	2	4	5	6
8	Severe level 6	No	3	4	5	6
9	Moderate level 4	No	2	4	5	5
10	Moderate to severe level 5	No	2	4	5	5



**Fig. 1.** Summary of change in swallowing physiology as measured by the severity level rating on videofluoroscopic swallowing studies. MBS 1 = admission videofluoroscopy; MBS 2 = posttreatment videofluoroscopy.

final outcome (1–4 years posttreatment). This result is in contrast to that in six subjects who reported at least one episode of diagnosed pulmonary symptoms related to aspiration since their discharge from acute rehabilitation but prior to the initiation of treatment.

### Statistical Analyses

Due to the small sample size and the nominal and ordinal nature of the data, nonparametric statistical procedures were employed. Statistical analyses of the data included Wilcoxon signed-ranks analysis of changes in pre- and posttreatment videofluoroscopy severity ratings. As diet level changes were measured over several ratings, a nonparametric Friedman test for several related samples was used to evaluate the presence of overall significance in diet level changes. Post hoc Wilcoxon signed-ranks tests were then used to identify sources of differences within the dataset. To evaluate the significance of time postonset and severity, a series of Spearman rho correlation coefficients were computed. A binomial sign test was used to evaluate for the significance of changes in presence or absence of pulmonary symptomatology.

Physiologic changes in swallowing based on a shift in severity level ratings were found to be highly significant. When comparing pretreatment and posttreatment severity scores, Wilcoxon signed-rank test produced a Z score of  $-2.739$ ,  $p = 0.006$  ( $n = 10$ ). Figure

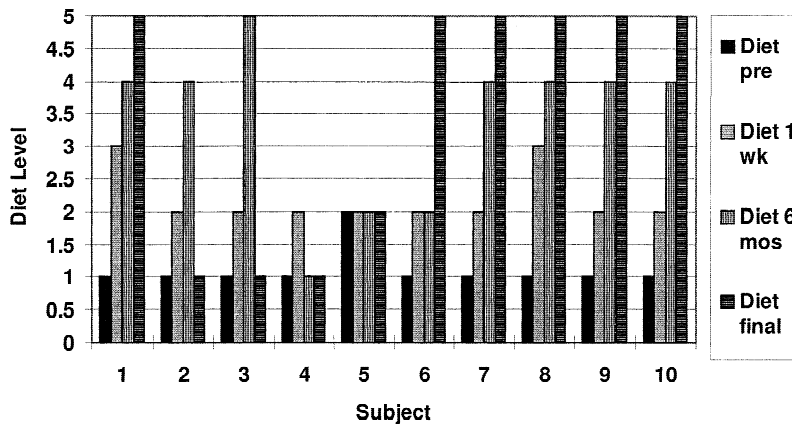


Fig. 2. Summary of change in functional diet level across measurement periods.

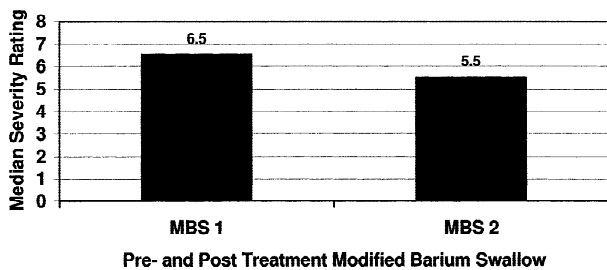


Fig. 3. Summary of median change in swallowing physiology as measured by the severity level rating on videofluoroscopic swallowing studies across subjects. Wilcoxon  $Z = -2.739$ ,  $p = 0.006$ .

3 illustrates changes in severity ratings on consecutive videofluoroscopic swallowing studies.

Functional change in diet level across the four ratings (pretreatment, after 1 week, after 6 months, and final outcome) was also significant, as indicated by a Friedman chi-square result ( $df = 3$ ) of 14.422,  $p = 0.002$ . Post hoc Wilcoxon signed-rank tests were conducted to identify the source of this difference. The change in diet level from the pretreatment rating to the rating at the end of 1 week (10 sessions) was statistically significant ( $Z = -2.810$ ,  $n = 10$ ,  $p = 0.005$ ). The median change in diet level across subjects from the measurements taken after 1 week and at 6 months was also statistically significant ( $Z = -2.280$ ,  $n = 10$ ,  $p = 0.023$ ), suggesting that the research participants continued to improve during this period. The change in diet level across subjects from the pretreatment measurement to the final outcome measurement was also statistically significant ( $Z = -2.449$ ,  $n = 10$ ,  $p = 0.014$ ), despite the loss of oral intake for two subjects associated with new onset diagnoses. These results are depicted in Figure 4.

Functional change in patient status, pre- and post-treatment as measured by the presence or absence of pulmonary symptomatology, was also evaluated by using the sign test. The reduction in pulmonary symptomatol-

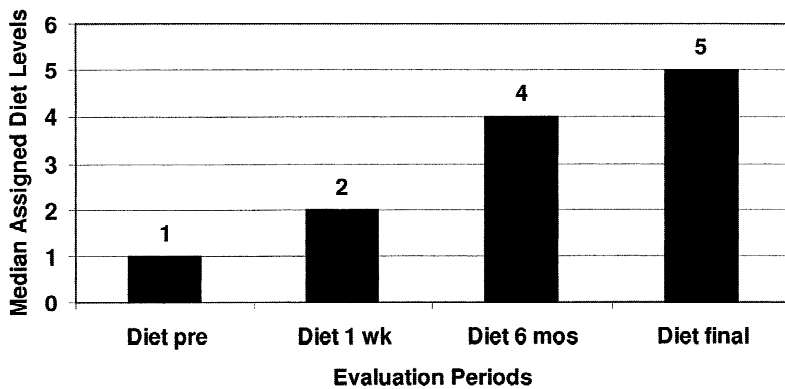
ogy was significant, with a  $p$  value of 0.031, with six patients reporting pulmonary complications prior to onset of treatment and no patients reporting pulmonary complications subsequent to treatment.

Spearman rho correlation coefficients were used to further evaluate the data for trends in the patterns of recovery and to identify features that may influence recovery. Time postonset did not significantly correlate with any measurement of the recovery process. Pretreatment severity level and posttreatment severity level (i.e., videofluoroscopy after 10 sessions) were significantly correlated with one another ( $\rho = 0.918$ ,  $n = 10$ ,  $p < 0.001$ ). These two severity level ratings did not significantly correlate with any measurement of the recovery process, with the exception of the duration of time after treatment until the gastrostomy tube was removed. Pretreatment severity correlated with time of gastrostomy tube removal ( $\rho = 0.811$ ,  $n = 8$ ,  $p = 0.014$ ), as did posttreatment severity ( $\rho = 0.825$ ,  $n = 8$ ,  $p = 0.012$ ). A significant negative correlation was identified between duration until gastrostomy tube removal and diet level at the 6-month evaluation ( $\rho = -0.736$ ,  $n = 8$ ,  $p = 0.037$ ).

## Discussion

Of this group of patients with chronic dysphagia secondary to single brainstem injury, the majority responded well to rehabilitative management. Statistically significant improvements were observed in swallowing physiology, diet level, and pulmonary status. Despite these changes, individuals with more severe initial impairments tended to be relatively more severely impaired immediately after treatment. Further, the more severe the physiological impairment, the longer the time until gastrostomy tube removal. Longer times until gastrostomy removal were, predictably, associated with poorer diet levels at 6 months posttreatment. It was noteworthy,





**Fig. 4.** Summary of median change in functional diet level across subjects. Diet before treatment and after 1 week: Wilcoxon signed rank sum test,  $Z = -2.810$ ,  $p = 0.005$ ; Diet after 1 week and after 6 months: Wilcoxon signed rank sum test,  $Z = -2.280$ ,  $p = 0.023$ ; Diet before treatment and at the final treatment: Wilcoxon signed rank sum test,  $Z = -2.449$ ,  $p = 0.014$ .

however, that diet levels continued to improve beyond the 6-month interval.

Eight of the 10 subjects were able to return to full oral intake with termination of gastrostomy tube feedings, and only two demonstrated negligible changes in swallowing physiology. Of the eight who returned to full oral intake, the average duration of tube feedings following treatment until discontinuation was 5.3 months, with a range of 1–13 months. Interestingly, all but two subjects had gastrostomy tube feedings discontinued 5–6 months posttreatment. The subject who was able to remove the gastrostomy tube after 1 month was subject 1 who was 12 months postonset and demonstrated one of the milder initial disorders. The other outlier in terms of tube feed discontinuation at 13 months was subject 6 who had the shortest time postonset (8 months) and demonstrated severe dysphagia. Six of these eight maintained their gains and continued progress until reaching a near normal diet. At final outcome, the only restrictions reported included dry bread and heavy meats by three subjects and rapid ingestion of thin liquids by three subjects. They all reported weight gain and denied pulmonary symptoms. Two of those who had reached full oral intake subsequently experienced onset of a new etiology and associated dysphagic symptoms. Subject 2 was in an automobile accident and was diagnosed with Parkinson's disease within 1 month of the accident, resulting in a decline in swallowing function and necessitating a return to nonoral feedings. Subject 3 was diagnosed with an atypical variant of Sjögren's disease and likewise returned to full nonoral feedings. Thus, in this group of 10 patients, if substantial progress was demonstrated after 1 week of intensive treatment, gastrostomy tube feedings were ultimately discontinued, and the patients returned to near normal diet and maintained their gains, unless their course was complicated by new onset of medical conditions.

Two of the original 10 subjects did not respond favorably to intervention (subjects 4 and 5). Although time postonset and severity and initiation of treatment

were not found to be significant predictors of outcomes, these two patients were the longest time postonset at the initiation of treatment (84 and 60 months) and demonstrated severe to profound dysphagia at the initiation of treatment. Subject 4 appeared to demonstrate slight improvement both functionally and radiographically after the initial treatment week. However, the gains in this patient were not as pronounced as those in the other subjects who continued to progress. This patient had a history of depression and did not follow up regularly with home programming and per oral trials according to the report of his spouse. In addition, this patient demonstrated an atypical SEMG waveform characterized by an elevated and unstable resting EMG tracing. This apparent inability to relax suprahyoid musculature raises the question of oral-pharyngeal spasticity as a contributor to dysphagia [33]. This EMG pattern was not observed in the other nine subjects. One could argue that the progress observed by subject 4 was not a direct effect of intervention but rather reflected further spontaneous changes that occurred, but were not documented, in the 6-month period after the most recent pretreatment videofluoroscopy. Subject 5 entered treatment with the ability to ingest large quantities of liquid rapidly and continued with that ability throughout the treatment course. No substantial functional or radiographic changes were noted in this patient subsequent to the first week of treatment. He continued on limited oral intake of rapidly ingested liquids.

Despite the severe impairments initially exhibited by subjects 4 and 5, the severity of the disorder does not appear to necessarily predict outcome. Three other subjects (subjects 6–8) who did recover demonstrated dysphagic physiology that was at least as severe as that of the two who did not recover. Time postonset may be an issue in recovery. However, from these data it appears to be a complex issue. Although the two patients who did not respond to treatment were at the longest time postonset (subjects 4 and 5), the subject who took the longest to have the tube feeding discontinued in the recovered

group was among those with the shortest time postonset (subject 6).

What accounts for recovery in this group of chronic patients? At a functional level, the combination of intensive treatment paired with maximal auditory and visual feedback facilitated recovery in eight of the 10 patients in this study. Five of the eight patients had failed prior rehabilitation efforts. Crary [17] used a very similar treatment approach by combining intensive treatment with SEMG biofeedback. When functional outcome data from the present study are combined with those from the study by Crary [17], 13 of 16 patients with chronic dysphagia were able to respond to rehabilitation by using a combination of intensive treatment and exteroceptive biofeedback modalities.

In support of the inclusion of biofeedback modalities in swallowing rehabilitation, the literature provides considerable discussion regarding the role of biofeedback in motor learning. Rubow [34] identified two models of learning that represent conceptually different approaches to the rehabilitative process, the cybernetic model of learning and operant conditioning. He elaborated that a critical issue in differentiating these two models and applying them to treatment is the relative importance assigned to reinforcement and dynamic feedback. The cybernetic model of learning relies in concept on a continuous, closed-loop learning process. This model requires that continuous and immediate reinforcement regarding performance is the key to perceptual-motor learning. The cybernetic model thus reflects the immediacy of instrumental feedback in that the feedback is continuously integrated into the patients ongoing motor control processes. Conversely, the operant conditioning model implies that learning and behavioral adaptation occur as a consequence of reinforcement or punishment that is provided in temporal association with a given task. This model of learning suggests that reinforcement is effective if it is provided within a few seconds of a desired response. Thus, the operant conditioning model of learning would typically represent a clinician-guided treatment wherein the patient's rehabilitative behavior is followed by initially consistent, albeit delayed, feedback provided by the clinician. The consistency of the feedback is progressively withdrawn as the patient assumes greater independence in the learning process. Rubow suggested that feedback in accordance with the cybernetic model is important in the early stages of rehabilitation; however, the role of delayed and even intermittent reinforcement, via operant conditioning, increases in later stages.

Another model of motor learning has been provided by Wolf [19], which further supports the benefits of biofeedback monitoring in rehabilitation processes. He outlined two stages of motor learning, the acquisition

and transfer phases, that correlate well with the cybernetic models and operant conditioning models. In his review of motor learning theory, Wolf acknowledged data that suggest that normal learning and retention are enhanced with periodic rather than with continuous reinforcement. However, he questioned the validity of these data when applied to the initial motor relearning process in patients with neurologically based disorders. He suggested that the feedback signal in the initial phases of treatment may serve as a substitute for a patient's inadequate proprioceptive signals, which in normal settings are instantaneous and consistent, and that these exteroceptive signals ultimately engage the internal sensorimotor networks. Thus, the acquisition phase of relearning a motor skill requires continuous reinforcement, whereas the transfer phase begins with engagement of the internal sensorimotor networks, thus requiring less immediate or continuous external feedback. The success of rehabilitative programs, when following this model, is considered to be secondary to a relearned appreciation of internal cues and to potential "recalibration" of the proprioceptive system [19]. The use of biofeedback modalities is thus considered a temporary adjunct to treatment, with the inherent goal of internalization of the feedback signal and extinction of the need for external feedback. Given the disruption of sensorimotor integration, largely mediated by nucleus solitarius and its interconnections [35], in hindbrain lesions such as these, relearning and recalibration may represent a type of intrasystemic reorganization [36].

Although the effects of audiovisual biofeedback appear to be favorable, the recovery of this group of patients may also reflect the frequency with which treatment was provided. It will require further study to determine whether the additional information provided by biofeedback modalities, the frequency of treatment, or a combination of the two influenced outcome of swallowing treatment. Although in this group of chronic patients, an argument may be made for using each patient as his or her own control, no studies have systematically compared this treatment regime with a more typical therapy approach. A prospective, randomized study of chronic patients which carefully evaluates the variables of intensity of treatment and modalities used would be of benefit to factor out the most efficacious approach. By using this approach, an "optimal treatment approach" in terms of both ultimate outcome and cost effectiveness could be determined.

Although efficacy data are critically needed, there is also a need to explore the neurobiologic mechanisms responsible for recovery in the dysphagic population. In the present sample, the subjects who were able to return to full oral intake demonstrated physiologic change as seen radiographically and reported that com-

pensatory techniques were no longer needed to maintain safety and bolus transfer following tube feeding discontinuation. Can one reasonably say that these patients recovered instead of compensated? If so, what is the neurobiologic foundation for that recovery in these patients, many of whom were past the presumed point of spontaneous recovery or rehabilitation? Subject 8 raises very puzzling questions. At more than 2 years postonset, this patient presented profound dysphagia, with total absence of a synergistic response to the bolus, despite a full year of rehabilitation efforts that included many of the exercises that were used in the accelerated program. However, after 1 week of treatment, she demonstrated well-coordinated, although weakened, swallowing and was able to ingest up to 4 oz. of a puree consistency. She continued to make rapid progress without formal treatment until she returned to a near normal diet with minimal compensatory maneuvers and discontinued tube feedings at 6 months. Because she had failed treatment previously, a question could be raised as to whether there is a "critical period" for neurologic recovery that is perhaps not within the first year of treatment. Moreover, rehabilitation efforts in this case appeared to influence not only the strength but also the synergy of the swallowing response. At this late point in the postoperative course, it would seem reasonable to improve muscular strength, but what accounts for the improved synergy in swallowing?

The neuroscience literature offers some explanations for neural recovery [37,38]. However, little attempt has been made to incorporate this information into an explanation for recovery in patients traditionally seen for speech pathology services. Keefe [39] reviewed much of this information and speculated as to how known neural recovery mechanisms may influence rehabilitation of the aphasic patient. Three categories of neural change are summarized below that may be susceptible to behaviorally based treatments: changes in synaptic function, structural adaptations, and alteration of neural networks. According to Keefe [39], optimizing the temporal relationship between stimulus presentation of impaired and nonimpaired modalities may facilitate the process of long-term potentiation, which may influence alteration in synaptic function. Structural changes of the nervous system, such as collateral sprouting, have been demonstrated to be highly influenced by experience and use and thus may be heavily influenced by the selection and sequencing of treatment modalities. Keefe speculated that reorganization of neural networks, or cortical reorganization, will require intensive task repetition or a directly trained task with rigorous carryover. Although the concepts presented by Keefe have yet to be systematically validated with controlled research, scholarship of this nature is needed to formulate hypotheses and guide fu-

ture research. With respect to swallowing, an introduction to this line of research has been presented by Hamdy et al. [40,41]. An initial study [40] using magnetic cortical stimulation and topographic mapping documented interhemispheric cortical asymmetry associated with swallowing. Furthermore, in comparison with normal swallowers, dysphagia appeared to be associated with smaller cortical pharyngeal representation of the intact hemisphere. A subsequent study by the same group documented, in a group of eight dysphagic patients, a significant trend toward an increase in cortical pharyngeal representation in the unaffected hemisphere as a function of swallowing recovery [41].

The data presented in this retrospective report raise some interesting questions regarding the provision of dysphagia management services, which will require further prospective evaluation. As with management in other areas of speech and language pathology, management of the dysphagic patient has typically been to initiate rehabilitation efforts within the first months after onset of the disorder to maximize spontaneous recovery. These initial rigorous attempts at rehabilitation are then characteristically discontinued if progress is not identified within 6 months after initiation of treatment. This approach is driven by the conventions of clinical practice and third-party reimbursement. Data such as those presented in the study by Crary [17] and in the present report challenge this approach by identifying the potential for recovery of swallowing function in patients who are well past the initial subacute period of their illness and have entered the chronic phase. Thus, it may be prudent to reconsider the timing of intervention, and schedule the dysphagic patient for brief trials of treatment that are discontinued if the patient does not respond and reschedule at a later date to determine if there may be an ultimate response to treatment. In addition, only three patients in this sample continued formal treatment after the initial intensive treatment program. Five of the patients who returned to full oral intake did not receive any formal treatment after the intensive treatment program, relying instead on continued home programming. Thus, the question could be posed as to the necessity of continuing direct treatment in this population. Further research is needed to identify prognostic factors and systematically compare treatment regimens. This type of research will be critical to drive the patterns of tightening reimbursement that we currently practice under. For the patients included in this study, reimbursement was procured for all through a series of lengthy letters of justification and patient advocacy. However, with the ever-shrinking health care dollar, reimbursement may no longer be available for programs such as this unless prospective studies can strongly support their efficacy. It behooves us

to identify rehabilitation programs that provide maximal results in a limited number of treatment sessions.

## Summary

This paper retrospectively reviews the outcomes of a clinical swallowing treatment program for 10 patients with chronic dysphagia secondary to single brainstem injury. Although these data provide support for the implementation of an intensive treatment program with multimodality biofeedback technology, there are many unanswered questions that are not addressed through retrospective reporting methods. These will need to be addressed in future prospective research endeavors. Specifically, the following questions are posed:

1. What accounts for the recovery in this group of patients, the intensity of the treatment regime, or the inclusion of instrumental biofeedback modalities? A prospective comparison of treatments that isolate the intensity of treatment and the inclusion of biofeedback would be very informative.
2. What affect do the variables of home programming and follow-up treatment have on outcomes? Would a program including only the intensive program be as effective and less expensive as an intensive program that encourages follow-up treatment?
3. Do the slight variations in the treatment provided that were dictated by patient physiology influence outcomes? A retrospective study, by nature, reports on the treatment provided to a group of patients based on the clinical needs of those patients. Prospective treatment efficacy studies will require more rigorous controls of the treatment provided and clearer definitions of other potentially influential variables.
4. This paper addresses only patients with brainstem injury. How applicable would a program such as this be for patients demonstrating dysphagia secondary to other etiologies?

Of the group of 10 patients reported on in this paper, six provided comments on their patient questionnaire similar to those of subject 8: "Since I equated eating with living, I thought I was just marking time until I died." The impact of chronic dysphagia on the emotional, physical, and financial well-being of the individual is significant. Thus, further prospective research is critical to justify ongoing clinical practice in this area so that patient needs can be met. In the interest of those patients who have demonstrated little return of swallowing as a function of spontaneous recovery or who have failed initial attempts at rehabilitation, clinical practice

and efficacy research should include study of long-term rehabilitation and recovery patterns.

## Appendix 1: Patient Questionnaire (Reformatted for Purposes of Publication)

Patient ID:

DOB:

Relationship to patient of person completing this questionnaire?

1. When did your swallowing problem begin?
2. What was the diagnosis associated with the start of your swallowing problem (be as specific as possible)?
3. If you had surgery associated with the onset of your swallowing problem, what was the type of surgery and the date of your surgery?
4. How long was the hospitalization associated with your swallowing problem?
5. During this hospitalization, did you suffer from respiratory failure?
6. Did you require intubation (a tube through your mouth and into your airway to help you breathe)?
7. Did you have a tracheostomy (a tube through an incision in your throat)?
8. If so, how long did you have your tracheostomy?
9. Have you had pneumonia since your discharge from the hospital?
10. Did you receive any surgical intervention to treat your swallowing disorder?
11. If so, what surgery did you receive and what was the date of the surgery?
12. Was the surgery helpful?
13. Did you receive therapy for your swallowing disorder prior to your treatment in the Outpatient Accelerated Swallowing Program (OASP)?
14. If so, how long did this therapy last?
15. How often were you seen for therapy?
16. Did you consider this therapy to be helpful?
17. Why did you discontinue this treatment?
18. Were you ever told that your swallowing would come back on its own?
19. Were you ever told that your swallowing would not improve and you would never eat again?
20. Did you consider your week of therapy in the OASP to be helpful?
21. Following your week of therapy in the OASP, did you begin any oral eating?
22. Did you receive any therapy after your treatment in the OASP? If so, for how long and how often?
23. Did you follow the recommendations regarding oral feeding and treatment given to you in the OASP?

24. Did you have any medical complications after your treatment in the OASP (pneumonia due to aspiration, further neurologic injury, etc.)?
25. How do you receive your nutrition now (feeding tube, oral feeding, combination)?
26. Have you had your feeding tube removed? If so, please give the date if was removed.
27. If you are eating by mouth, what kind of diet are you on? Please list specific foods/liquids that you cannot eat.
28. Have you had any significant weight loss or weight gain since you completed your swallowing treatment? If so, approximately how many pounds loss or gain?
29. Are you still going to swallowing therapy? If not, when did you discontinue therapy and why?
30. Have you had any complications since your treatment in the OASP that you feel affected your ability to return to oral intake (i.e., any other medical problems, stressors, etc.)?
31. In your own words, how has your swallowing problem affected your life?
32. Please add any additional information regarding the course of your illness that you think may be helpful.

## Appendix 2: Outpatient Accelerated Swallowing Treatment Program; Clinical Policy and Procedures

### *Focus of the Program*

This program is conceptually intended to provide patients with chronic and often severe pharyngeal phase dysphagia the opportunity to benefit from an intensive course of directed rehabilitation. The combination of highly concentrated rehabilitation and instrumental visual and auditory feedback provides maximal opportunities for swallowing recovery.

### *Treatment Candidacy*

Prior research has suggested that intensive, daily rehabilitation efforts result in significant changes in swallowing physiology and functional diet tolerance in a significant number of patients with chronic dysphagia subsequent to brain stem injury [17,32]. Thus, the targeted patient population for this program includes patients who:

- are more than 9 months postonset
- demonstrate pharyngeal phase dysphagia secondary to a single unilateral neurological event
- exhibit adequate strength and stamina to tolerate the rigors of an outpatient intensive rehabilitation program.

Data are limited regarding prognosis for recovery. Given this limitation, patients with long-standing dysphagia should not be excluded on the basis of time postonset. The data support that patients with even profound deficits may respond to intervention; thus, the severity of the disorder should not necessarily influence admission to the program. Further-intact cognition is not necessarily a prerequisite to benefit from treatment because the additional visual and auditory feedback and the intensity of intervention have the potential to overcome the barriers of poor language and cognition in many patients.

In addition to these criteria, all patients who are considered appropriate candidates for rehabilitation of pharyngeal-phase deficits are considered appropriate for SEMG-facilitated treatment, although the intensity of intervention may be adjusted to reflect the needs of the individual patient. For example, an acute patient may be appropriate for intervention, but the intensity of 2 hr of treatment daily may likely exceed the patient's ability to participate. Patients who are undergoing radiation treatment may benefit from the visual feedback and the structured repetition of swallowing to maintain flexibility, but the intensity of treatment should be significantly reduced with much more relaxed goals.

Exclusionary criteria would include patients who:

- are diagnosed with neurodegenerative disease processes underlying the swallowing disorder (e.g., ALS, multiple sclerosis, myasthenia gravis)
- demonstrate swallowing physiology that is not considered amenable to rehabilitative efforts, in particular the literature does not support any *rehabilitative* maneuver that will significantly alter the timing of onset of swallow; patients with this type of physiologic deficits are appropriate for compensatory rather than rehabilitative management
- do not exhibit adequate strength or stamina to tolerate the full treatment regimen
- are undergoing radiation treatment for oral pharyngeal carcinoma
- have failed a brief trial of intensive intervention secondary to inadequate cognition or attention.

In addition, careful consideration and specific, written physician's orders should be obtained for patients who are:

- currently are exhibiting unstable cardiac or neurologic function
- immediately postoperative oral pharyngeal surgery for resection of carcinoma or repair of traumatic injury.

### *Treatment Regimen*

Prior to acceptance into the program, history information and a video copy of the most recent instrumental swallowing study will be reviewed by the dysphagia team to determine the appropriateness of rehabilitation and develop the treatment plan (refer to previous section on Treatment Candidacy). The admission videofluoroscopic swallowing study should be completed:

1. within 2 weeks prior to onset of treatment for patients less than 1 year postonset
2. within 1 month for patients between 12 and 28 months postonset, or
3. within 6 months for patients who presented for treatment more than 28 months postonset of dysphagia.

The dysphagia team should consist minimally of the patient, family, referring physician, and treating speech pathologist and should include, as appropriate and available, the dysphagia team physician, dietician, and social worker. The treatment plan will be further developed and reviewed at length with the patient and appropriate caregivers with acceptance into the program or during the initial session.

Patients admitted into the program will be scheduled for treatment for 5 consecutive days, two sessions per day, for a total of 10 hr per week of direct therapy. Patients will be scheduled for 1 hr of therapy in the morning and 1 hr in the afternoon, with an intervening 3–4 hr for rest. These hour-long sessions will include a combination of direct rehabilitative training and compensatory management of oral feeding trials. These two components will be approximately evenly divided during each therapy session in 15-min increments. As an example, the beginning of each session will focus on approximately 15 min of rehabilitative training, following by an approximate 15 min of oral trials. This sequence will then be repeated, allowing for rigorous completion of exercises interrupted by frequent repetition of compensatory techniques. In addition, education and review of home programming will be incorporated into the therapeutic hour.

Although specific treatment plans for the patients in the program will be individualized to reflect the needs of each patient, treatment for pharyngeal phase deficits will tend to focus in on the following areas, as appropriate:

- mastery of compensatory mechanisms to improve airway protection and facilitate transfer of the bolus through the pharyngeal cavity by maximizing gravitational forces and minimizing resistance to the bolus
- relaxation of musculature involved in swallowing (inhibition of extraneous movement, spasticity)

- increasing strength and efficiency of the oral pharyngeal swallow
- maximizing sensory input and response to the oral pharyngeal cavity
- initiation of trials of oral intake and a return to oral diet.

SEMG and cervical auscultation biofeedback will be used to facilitate teaching and execution of the therapeutic exercises, including:

1. effortful, or modified valsalva, swallow: used when the patient demonstrates radiographic evidence of weakened or dyscoordinated pharyngeal contraction or reduced laryngeal excursion resulting in postswallow vallecular residual or diffuse pharyngeal residual and inadequate epiglottic deflection for airway protection
2. Mendelsohn maneuver: used when the patient demonstrates radiographic evidence of weakened or dyscoordinated pharyngeal contraction or specific impairment of upper esophageal sphincter opening resulting in postswallow diffuse pharyngeal residual or residual isolated to the pyriform sinuses with impaired bolus transport into the cervical esophagus
3. vocal adduction exercises: used when the patient demonstrates deficits in laryngeal airway protection and/or vocal dysphonia
4. oral motor exercises: used when the patient clinically demonstrates oral motor weakness or dyscoordination with radiographic evidence of poor bolus manipulation, formation, and noncohesive transfer secondary to poor oral control
5. head-lifting maneuver: used when the patient demonstrates radiographic evidence of impairment in upper esophageal sphincter opening resulting in postswallow pyriform sinus residual; this technique is used primarily as a home programming task and does not use instrumental biofeedback modalities.

In addition to the execution of rehabilitative exercises, direct oral intake will be included as a component of the treatment regimen as soon as possible with respect to assurance of airway protection. This component of treatment is considered critical for the neurosensory stimulation it provides to the oropharyngeal cavity and the sense of meaningful progress it provides the patient. During therapeutic oral feeding, airway protection maneuvers and compensatory strategies will be reinforced to maximize airway protection and bolus transfer. All patients will be realistically counseled regarding the risks associated with aspiration and potential subsequent pneumonia and will be encouraged to make an independent decision as to whether oral trials would be

included as a component of their treatment. Compensatory mechanisms that may be used include, but are not limited, to:

1. supraglottic swallow
2. pharyngeal expectoration
3. vocal quality checks
4. head rotation
5. chin-tuck posturing
6. head tilt
7. dry swallow
8. cyclic ingestion
9. liquid wash

If the patient begins oral intake during the treatment week, calorie counts should be taken to monitor intake and adjust tube feedings. Consultation with a dietitian is thus strongly recommended. In addition, respiratory sounds should be carefully monitored by appropriately trained personnel. General precautions should be taken to minimize risk of pulmonary infiltrate. After all oral trials, patients should be encouraged to be upright and ambulatory for at least 30 min and should monitor temperature at the same time daily.

### *Home Programming*

Home programming is considered an integral component of a successful treatment regimen. As the patients learn rehabilitative and compensatory maneuvers during the treatment session, these techniques will be carried out in an independent program. Optimally, patients who are attending treatment twice daily will complete additional three 15-min sessions independently. The patient's family or caregiver is considered to be an integral component of the home program to aid the patient in completing exercises, monitoring progress, and documenting oral intake. Compliance with the home program will be reviewed daily during direct treatment. If the patient is able to safely tolerate oral intake, nutritional intake should be scheduled to follow all home programming sessions. Patients will keep an intake diary including amounts, textures, and relative tolerance of oral intake. A portable biofeedback device will be available to all patients for use during the home program. Education regarding electrode placement and device set-up will occur during direct treatment session prior to independent use. Compliance with completion of exercises will be monitored by reviewing stored SEMG data on the portable biofeedback device.

### *Follow-up*

After the first week of concentrated rehabilitation, a repeat videofluoroscopy will be performed to assess

changes in swallowing physiology. If a measurable change is documented, the patients will be scheduled for follow-up treatment at a rate of once or twice weekly, but will continue rigorous home programming five times daily for 15 min each prior to oral intake, if tolerated. If no measurable change is identified on posttreatment videofluoroscopy and no functional clinical gains are noted, the patients candidacy for continued treatment will be discussed by the patient, family, clinician, and physician.

### *Documentation*

Careful documentation is required to assure reimbursement from third-party payors and to gather departmental program efficacy data. The videofluoroscopic swallowing study that is evaluated for admission into the program should be rated by using a departmental evaluation form or some objective rating instrument, even if a report for the examination is generated from another referring facility. Subsequent studies should use the same format. The initial diagnostic summary and treatment plan should be comprehensive and include history information, prior treatment attempts, diagnostic information, and a detailed treatment plan with long-term and short-term objectives. Daily progress notes should include a statement of the treatment objectives and a subjective measure of progress toward those objectives. It is important that progress notes reflect *clinically measurable* objectives that are supported by objectives measured with SEMG biofeedback.

A final treatment summary and diagnostic report should include a summary of progress toward short-term objectives and long-term goals. Posttreatment videofluoroscopic findings should be detailed, with particular emphasis on changes in physiology. The treatment plan should be revised and updated to reflect changes in swallowing physiology.

Copies of all documentation and any additional referral sources should be forwarded to the patient/caretaker and to the referring physician with the patient's signed consent.

### *Billing*

Patients should be billed for speech pathology services. Using CPT coding for biofeedback treatment may reduce the likelihood of reimbursement. The treatment regimen includes many other facets of treatment, with biofeedback serving only as an adjunct to facilitate the process. It may be beneficial from a marketing perspective to develop a package price for two diagnostic examinations and 10 treatment sessions.

Private insurance companies may require precer-

tification for treatment. Medicare may make the request for chronic patients. Points to highlight:

- prior research that documents improvement in chronic patients
- costs of maintaining patients on tube feeding
- availability of new techniques (head lifting, Masako) and new technologies (SEMG) that may improve the patients chances of recovery.

The patient and family member should advocate for themselves by requesting the treatment.

Portable devices, if elected for use, are generally reimbursable under a “durable medical equipment” clause of the insurance company. Medicare will not reimburse for a portable device. If the patient’s insurance company will not reimburse for a portable device, the patient can rent it directly from the biofeedback distributor. It is unlikely that the patient will require or desire a portable device for more than 1 month.

Biofeedback electrodes can be considered consumable materials and must be purchased by the department on an ongoing basis. Expense for electrodes can be either absorbed by the department or offset by separating standard packs of 100 electrodes into packages of 10 and bill a portion of the total cost to the patient.

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## References

1. Barer DH: The natural history and functional consequences of dysphagia after hemisphere stroke. *J Neurol Neurosurg Psychiatry* 52:236–241, 1989
2. Chapman D: Dysphagia recovery in stroke patients. Presented at the *American Speech Language Hearing Association Annual Convention*, Seattle, WA, 1996
3. Horner J, Massey EW, Riski JE, Lathrop DL, Chase KN: Aspiration following stroke: clinical correlates and outcome. *Neurology* 38:1359–1362, 1988
4. Miller A, Bieger D, Conklin JL: Functional controls of deglutition. In: Perlman A, Schulze-Delrieu C (eds.): *Deglutition and its Disorders: Anatomy, Physiology, Clinical Diagnosis and Management*. San Diego: Singular Publishing Group, 1997, pp 57–63
5. Logemann JL: Management of the patient with disordered oral feeding. In: Logemann JA (ed.): *Evaluation and Treatment of Swallowing Disorders*. Austin: Pro-Ed, 1983, pp 127–159
6. Kahrilas PJ, Lin S, Logemann JA, Ergun GA, Facchini F: Deglutitive tongue action: volume accommodation and bolus propulsion. *Gastroenterology* 104:152–162, 1993
7. Kahrilas, PH, Logemann JA, Lin S, Ergun GA: Pharyngeal clearance during swallow: a combined manometric and video-fluoroscopic study. *Gastroenterology* 103:128–136, 1992
8. Kahrilas PJ, Logemann JA, Krugler C, Flanagan E: Volitional augmentation of upper esophageal sphincter opening during swallowing. *Am J Physiol* 260:G450–G456, 1991
9. Fujii M, Logemann J, Pauloski B: Increased Post-operative posterior pharyngeal wall movement in patients with anterior oral cancer: preliminary findings and possible implications for treatment. *Am J Speech Lang Pathol* 4:24–30, 1995
10. Fujii M, Logemann J: Effect of a tongue-holding maneuver on posterior pharyngeal wall movement during deglutition. *Am J Speech Lang Pathol* 5:23–30, 1996
11. Shaker R, Kern M, Bardan E, Taylor A, Stewart E, Hoffmann R, Arndorfer R, Hofmann C, Bonnevier J: Augmentation of deglutitive upper esophageal sphincter opening in the elderly by exercise. *Am J Physiol* 272:G1518–G1522, 1997
12. Shaker R, Kern M, Bardan E, Arndorfer RC, Hofmann C, East-erling C: Effect of isotonic/isometric head lift exercise on hypopharyngeal intrabolus pressure. Presented at the Fifth Annual Dysphagia Research Society Meeting, 1997 [abstract]. *Dysphagia* 12:107, 1997
13. Logemann JA, Kahrilas PJ: Relearning to swallow after stroke—application of maneuvers and indirect biofeedback: a case study. *Neurology* 40:1136–1138, 1990
14. Neumann S: Swallowing therapy with neurologic patients: results of direct and indirect therapy methods in 66 patients suffering from neurological disorders. *Dysphagia* 8:150–153, 1993
15. Neumann S, Bartolome G, Buchholz D, Prosiegel M: Swallowing therapy of neurologic patients: correlation of outcome with pretreatment variables and therapeutic methods. *Dysphagia* 10:1–5, 1995
16. Bartolome G, Neumann S: Swallowing therapy in patients with neurological disorders causing cricopharyngeal dysfunction. *Dysphagia* 8:146–149, 1993
17. Cray MA: A direct intervention program for chronic neurogenic dysphagia secondary to brainstem stroke. *Dysphagia* 10:6–18, 1995
18. Logemann JA: Criteria for studies of treatment for oral-pharyngeal dysphagia. *Dysphagia* 1:193–199, 1987
19. Wolf SL: Biofeedback. In: Downey JA, Myers SJ, Gonzales EG, Lieberman JS (eds.): *The Physiological Basis of Rehabilitation Medicine*, 2nd ed. Stoneham, MA: Butterworth-Heinemann, 1994, pp 563–572
20. Kasman G: Motor learning with EMG biofeedback: an information processing perspective for rehabilitation. *Biofeedback* 24:4–7, 1996
21. Adrian ED, Bonk DW: The discharge of impulses in motor nerve fibres. Part II. The frequency of discharge in reflex and voluntary contractions. *J Physiol* 67:119–151, 1929
22. Lindsley DB: Electrical activity of human motor units during voluntary contraction. *Am J Physiol* 114:90–99, 1935
23. Smith O: Action potentials from single motor units in voluntary contractions. *Am J Physiol* 108:629–638, 1934
24. Sonies BC, Gottlieb E, Solomon BI, Mathews K, Huckabee ML: Simultaneous ultrasound and EMG study of swallowing. Presented at the Fifth Annual Dysphagia Research Society Meeting, 1997 [abstract]. *Dysphagia* 12:106, 1997
25. Bryant ML: Biofeedback in the treatment of a selected dysphagic patient. *Dysphagia* 6:140–44, 1991
26. Huckabee ML: SEMG biofeedback: an adjunct to swallowing therapy. *Biofeedback* 25:20–29, 1997
27. Stott FD: The laryngeal microphone as an aid to treatment of bulbar poliomyelitis. *Br Med J* 20:1416, 1953
28. Hamlet S, Nelson RJ, Patterson RL: Interpreting the sounds of swallowing: fluid flow through the cricopharyngeus. *Ann Otol Rhinol Laryngol* 99:749–752, 1990



29. Takahashi K, Groher ME, Michi K: Methodology for detecting swallowing sounds. *Dysphagia* 9:54–62, 1994
30. Zenner PM, Losinski DS, Mills RH: Using cervical auscultation in the clinical dysphagia examination in long-term care. *Dysphagia* 10:27–31, 1995
31. Boiron M, Rouleau P, Metman EH: Exploration of pharyngeal swallowing by audiosignal recording. *Dysphagia* 12:86–92, 1997
32. Huckabee ML, Cannito MP: Analysis of swallowing physiology: reliability of interpretation using a structured rating index. Manuscript in preparation, 1998
33. Huckabee ML, Rozehnal S: A case comparison in lateral medullar infarct: does suprahyoid spasticity effect recovery in dysphagia? Presented at the Third Annual Dysphagia Research Society Meeting, 1994 [abstract]. *Dysphagia* 10:145, 1995
34. Rubow R: Role of feedback, reinforcement, and compliance on training and transfer in biofeedback based rehabilitation of motor speech disorders. In: McNeil M, Rosenbek J, Aronson A (eds.): *The Dysarthrias: Physiology, Acoustics, Perception and Management*. San Diego: College-Hill Press, 1984, pp 207–229
35. Jean A: Control of the central swallowing program by inputs from the peripheral receptors. A review. *J Autonom Nerv Syst* 10:225–233, 1984
36. Luria AR: *Traumatic Aphasia: Its Syndromes, Psychology and Treatment*. The Hague: Mouton, 1970
37. Held JM: Recovery after damage. In: Cohen H (ed.): *Neuroscience for Rehabilitation*. Philadelphia: JP Lippincott, 1995, pp 388–406
38. Seltzer ME: Mechanisms of functional recovery in traumatic brain injury. *J Neurol Rehabil* 9:72–83, 1995
39. Keefe KA: Applying basic neuroscience to aphasia therapy: what the animals are telling us. *Am J Speech Lang Pathol* 4:88–93, 1995
40. Hamdy S, Aziz Q, Rothwell JC, Singh KD, Barlow J, Hughes DG, Tallis RC, Thompson DG: The topography of human swallowing musculature in health and disease. *Nat Med* 2:1217–1224, 1996
41. Hamdy S, Aziz Q, Rothwell JC, Crone R, Hughes DG, Tallis RG, Thompson DG: Explaining oropharyngeal dysphagia after unilateral hemispheric stroke. *Lancet* 350:686–692, 1997