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Prepharyngeal Dysphagia in Parkinson's Disease

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Abstract. Dysphagia in patients with Parkinson's disease (PD) is most often attributed to pharyngeoesophageal motor abnormalities. In our study of patients with idiopathic PD, attention was focused on prepharyngeal symptoms and motor functions. Using the Hoehn and Yahr disease severity scale, patients were grouped into those with mild/moderate disease [subgroup I (n = 38)] and those with advanced disease [subgroup II (n = 34)]. Dysphagia symptoms were present in 82% of all patients, but subgroup I patients voiced significantly more complaints. Conversely, many prepharyngeal abnormalities of ingestion, including jaw rigidity, impaired head and neck posture during meals, upper extremity dysmotility, impulsive feeding behavior, impaired amount regulation, and lingual transfer movements were statistically more frequent in subgroup II patients. Impaired mastication and oral preparatory lingual movements were the most common aberrations observed during dynamic videofluoroscopy (48/71), with most patients being concordant for both. The motor disturbances of ingestion reported herein reflect the disintegration of volitional and automatic movements caused by PD-related akinesia, bradykinesia, and rigidity.

Key words: Parkinson's disease — Ingestion — Mastication — Lingual dysmotility — Deglutition — Deglutition disorders.

James Parkinson described dysphagia in his 1817 essay on "The Shaking Palsy." [1] In addition to difficulty swallowing, he observed several prepharyngeal abnormalities of ingestion including difficulty initiating and maintaining self-feeding, impaired oral containment of both saliva and food, and labored lingual movements. Almost 150 years later, Eadie and Tyrer [2] reported the first systematic investigation of dysphagia in Parkinson's disease (PD). While documenting sialorrhea and chewing difficulty, their report, like much of the subsequent related literature in the next decade, focused on pharyngeo-esophageal abnormalities [3–6]. Palmer [5], referring to upper esophageal sphincter dysmotility, editorialized that dysphagia in PD was ". . .almost always hypopharyngeal dysphagia."

In 1967, Donner et al. [7] observed oral and lingual stage abnormalities in patients with PD including hesitant deglutition, impaired bolus formation, inefficient mastication, and glottic tremor. Blonsky et al. [8] recorded the global motor dysfunctions of slow oral transit time and impaired lingual motility in 85 of 100 PD patients. Uncoordinated "posterior tongue" movement was the only descriptor. Calne et al. [9] studied pharyngeal deglutition in patients with PD. Finding no pharyngeal cineradiographic dysfunction, the authors suggested that dysphagic symptoms were related partly to defective tongue movements observed in many of their patients. This supposition was recently supported by Bushmann et al. [10] and Robbins et al. [11] who reported defective lingual motility including lingual "rocking" and "pumping," delayed swallowing reflex, decreased tongue mobility, and piecemeal deglutition.

The recent refocus on prepharyngeal motor dysfunctions in PD patients questions the primacy of pharyngo-esophageal dysphagia in this disease. The current study, part of a comprehensive investigation of dysphagia in basal ganglia diseases, reports our observations of prepharyngeal abnormalities of ingestion [12] in PD.

Subjects and Methods

Between 1975 and 1992, 1,160 patients with Parkinson's disease were evaluated by the Dysphagia Center of Crozer-Chester Medical Center.

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Table 1. Demographic data

	Parkinson's disease (n = 72)	Subgroup I (n = 38)	Subgroup II (n = 34)
Age (± SEM)	73.0 (10.0)	73.8 (9.6)	73.0 (10.4)
Disease duration (± SEM)	8.7 (6.2)	5.3 (4.5)	12.5 (5.6)
Hoehn & Yahr score (SEM)	3.4 (0.8)	2.8 (0.4)	4.2 (0.4)
Sex (M/F)	51/21		

The dysphagia chart and videoradiography results were reviewed in all patients. Patients were excluded from the study for the following reasons: insufficient clinical data, unconfirmed diagnosis, or other central nervous system illnesses that could affect the clinical dysphagia or radiologic evaluations, such as stroke or metabolic encephalopathy. Patients with dementia were not specificly excluded; neuropsychological or other formal tests that quantify dementia were completed inconsistently. The results were tabulated for the remaining patients with idiopathic Parkinson's disease (50 men and 22 women). Ages ranged from 50 to 88 (mean age = 72.1). (Table 1) The Hoehn and Yahr (H&Y) scale [13] was used to stage PD disease severity; it ranges from 0 (no signs of disease) to 5 (wheelchair bound or bedridden). The H&Y group mean disease severity score was 3.4 (range 2–5); the disease duration was 8.7 years (range 2–30). All but 2 patients were receiving a variety of antiparkinsonian medications when first evaluated, the majority (69.4%) on an inpatient basis.

Methods

The following Crozer-Chester Medical Center Dysphagia Center evaluation was completed on all patients: dysphagia history, clinical assessment of oral motor and sensory system functions with and without bolus inclusion, and an activity of daily living (ADL) self-feeding assessment. A dynamic video-fluoroscopic swallowing function study (DVSFS) prior to and coincident with compensatory techniques was performed on all but 1 patient.

Dysphagia History

Information regarding dysphagic symptoms was obtained from interviews with patients and their families or caretakers. Questions concerning the existing dietary level, choking, coughing, or other aberrant behavior during ingestion were addressed.

Clinical Prefeeding Oral Motor and Sensory Examination

This examination assessed the motility, range, strength, and coordination of oral structures. Thermal, gustatory, tactile, and proprioception sensory responses and their effects on the oral preparatory and lingual phases of ingestion were evaluated.

Activities of Daily Living Self-feeding Assessment

Self-feeding assessments were conducted to determine functional feeding status and its relationship to prepharyngeal phases of ingestion, ascertain effects on swallowing sequence positioning and sequence execution requirements, verify supervision/assistance needs, and confirm adaptive equipment necessity.

Clinical Assessment

Patients were examined in a stimulus-controlled environment to reduce ambient distractions. A variety of bolus substances were presented: pureed, mechanical soft, solid foods, and thin liquids. Volume, texture, temperature, taste, and weight properties were controlled and systematically modified, and the responses were analyzed. Existing overt dysphagic symptoms were documented. Unusual feeding patterns were recorded.

Dynamic Videofluoroscopic Swallowing Function Study

A DVSFS was completed on all but 1 patient to confirm the location, severity, type, and cause of overt dysphagic symptoms, and to identify covert abnormalities. Each DVSFS used a video positioning chair (VPC), designed by one of the authors (MCK), specifically to ensure focused erect lateral and anteroposterior observation during all stages of deglutition.

Mastication and transfer functions were assessed with barium-impregnated foods of varying textures ranging from pureed to solids, introduced in teaspoon amounts. Thin liquids followed, from regulated cup and straw sips to continuous drinking of unregulated amounts. Mastication was classified into anterior munching/graded, lateral, or rotary patterns in the lateral or anterior-posterior (A-P) projections, respectively. Bolus acceptance, containment, transfer, and transport functions associated with oral functions were analyzed in the lateral projection.

Statistics

Because our patient samples with H&Y stages II and V patients were small, patients with mild/moderate or more severe PD were grouped together for exploratory statistical purposes: subgroup I—H&Y stages II, III (n = 38); subgroup II—H&Y stages IV, V (n = 34). χ^2 testing was used for testing the hypotheses that differences in categorical variables were related to disease severity. Differences between proportions were examined with the Z test. To study the differences between means, the t test was used.

Results

History

The dysphagia questionnaire uncovered complaints related to all phases of ingestion (Fig. 1). Although prepharyngeal ingestion symptoms were primarily sought, results related to the pharyngeal and esophageal stages of ingestion are included for completeness. There were no

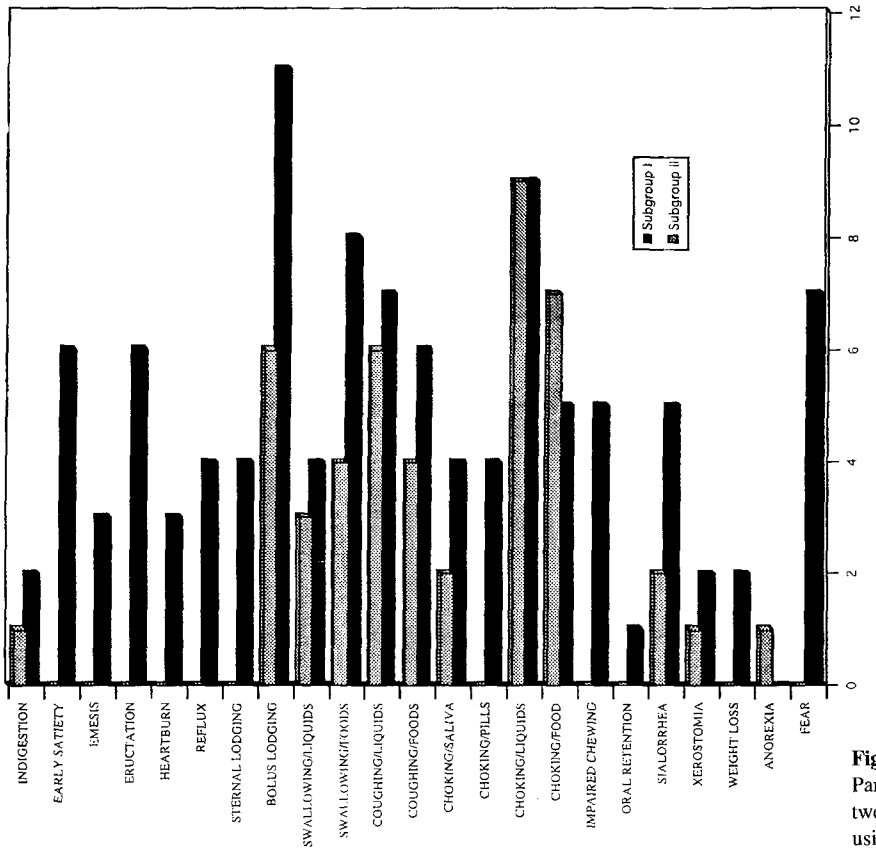

Fig. 1. Occurrence of symptoms in patients with Parkinson's disease (n = 72). Differences between the means of symptoms/patient were tested using the Student's *t*-test ($p < 0.01$).

statistically significant differences in discrete variables between subgroups I and II. Among the 72 subjects, the number of symptoms per patient ranged from 0 to 11 (mean = 2.5). Patients with the least severe PD (H&Y stage II) averaged more complaints (4.0) than stage III (2.8), stage IV (2.4), and stage V (2.0) patients. The mean (± SD) number of symptoms for subgroups I and II, respectively was 3.03(0.39) and 1.85(1.64). Testing the hypothesis that the means were statistically different, the resulting t-ratio was 2.46 ($p < 0.01$). Indeed, 8/12 patients who denied any complaint were stage 4 or 5 disease. The most common symptoms related to the pharyngeal stage of ingestion. Coughing on food was reported most frequently, followed by subjective lodging of food in the oropharynx, coughing on liquids, choking on liquids and food, and difficulty swallowing. Less frequent prepharyngeal phase symptoms included fear of eating, impaired chewing, sialorrhea, xerostomia, and oral retention of food after completing swallowing.

Clinical Prefeeding Oral Motor/Sensory System Examination

Pre-oral functions were observed during the oral motor/sensory examination (Fig. 2). Jaw rigidity, xerostomia, sialorrhea, lip tremor, and jaw tremor were registered most commonly. Only jaw rigidity was significantly more common in patients with advanced disease ($\chi^2 = 4.80$; df = 1, $p < 0.03$).

Self-feeding Examination

Patients were given the opportunity to feed themselves under the observation of the dysphagia specialist (Fig. 3). Though 33/72 patients fed independently, 13/72 patients needed some compensatory cuing, and 9 others were independent feeders after their meals were set up for them or after adaptive equipment was provided; an additional 12 patients were dependent feeders. In testing the hypothesis that feeding independence was directly related to disease severity, subjects were deleted for small cell frequency; the differences between subgroups I and II were significant ($\chi^2 = 12.99$, df = 2, $p < 0.002$). Multiple abnormalities of feeding behavior were observed during this task (Fig. 4). Pre-oral motor dysfunctions that impaired feeding efficiency included impaired regulation of food quantities ($\chi^2 = 9.24$, df = 1, $p < 0.003$), impulsive feeding ($\chi^2 = 8.05$, df = 1, $p < 0.005$), impaired hand to mouth motility excluding tremor ($\chi^2 = 7.64$, df = 1, $p < 0.005$), head/neck malpositioning

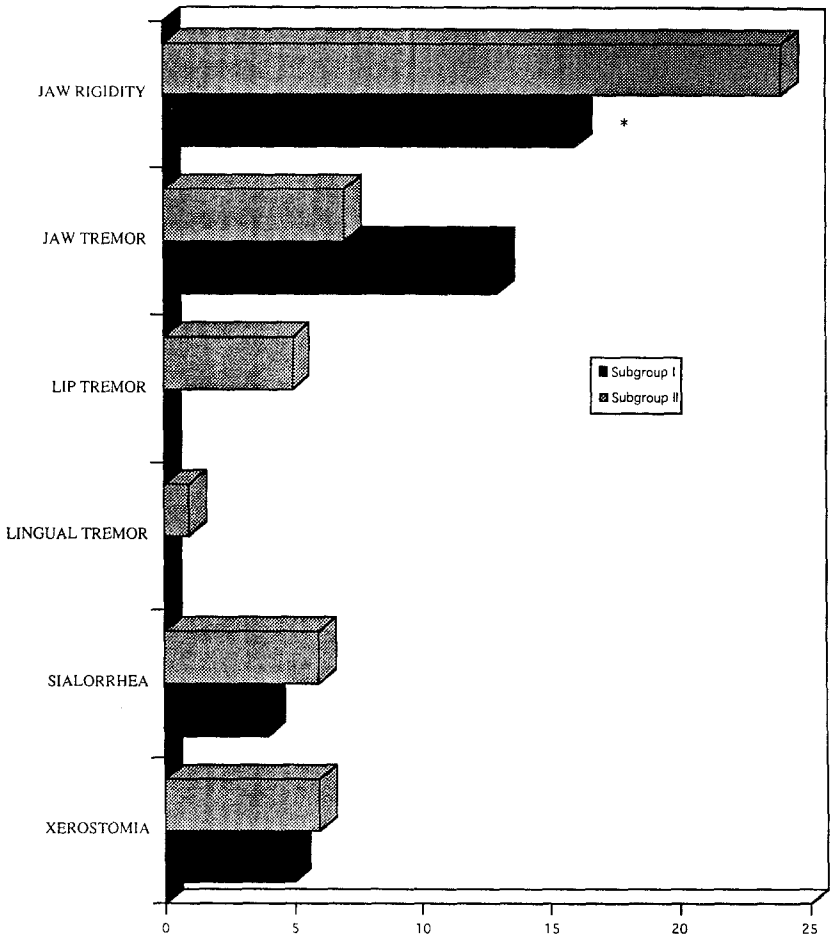

Fig. 2. Occurrence of abnormalities of the prefeeding oral motor/sensory examination in patients with Parkinson's disease. Differences between subgroups I and II were tested using the Chi-square test. (p:* < 0.03).

($\chi^2 = 15.98$, df $= 1$, $p < 0.0001$), impaired utensil use (grasping utensils, cutting food, or securing the bolus), upper extremity tremors, truncal anteroflexion, and tachyphagia.

Adaptive equipment was used by 7 patients and included scoop dishes, straws, and built up, rocker, weighted, or two-handed utensils.

Dynamic Videofluoroscopy Swallowing Function Study

Preparatory and lingual stage motor abnormalities were identified during DVSFS in 71 patients. Impaired mastication and dysfunctional lingual motility prior to lingual transfer were seen most frequently (48/71). Testing the concordance of observed proportions against that expected by change (50%), patients were likely to be concordant for lingual and chewing competency ($Z = 5$; $p < 0.00001$). Mastication was slow, hesitant, and delayed with ineffectual movements. Normal rotary moments were frequently absent and replaced with graded (20/71) or lateral (7/71) movements. Impaired oral containment was recorded in 9/71 patients. Lingual transfer was impaired in most patients, with many patients manifesting multiple abnormalities (Fig. 5). Latent ($\chi^2 = 10.64$, df $= 1$, $p < 0.001$), uncoordinated ($\chi^2 = 3.75$, df $= 1$, $p < 0.05$), premature, and segmented lingual transfers were seen most frequently; posterior bolus leakage prior to active transfer, suck-negative transfer, visceral swallow, and nontransfer were also encountered. After transfer efforts stopped, excessive oral retention of test meals was observed in 15/71 patients. Normal lingual transfer was present in only 12/71 patients, 75% of whom were subgroup 1.

Comment

Early reports of dysphagia in PD patients emphasized radiologic abnormalities but often failed to record patient symptoms. When noted, the number of complaints seriously understated the frequency and severity of radiologically documented swallowing dysfunction [2,11,14]. Logemann et al. [14] reported that 15%–20% of PD patients voiced swallowing complaints, but 95% had cineradiographic disturbances. Bushmann et al. [10] identified 53% of their patients who denied dysphagic symp-

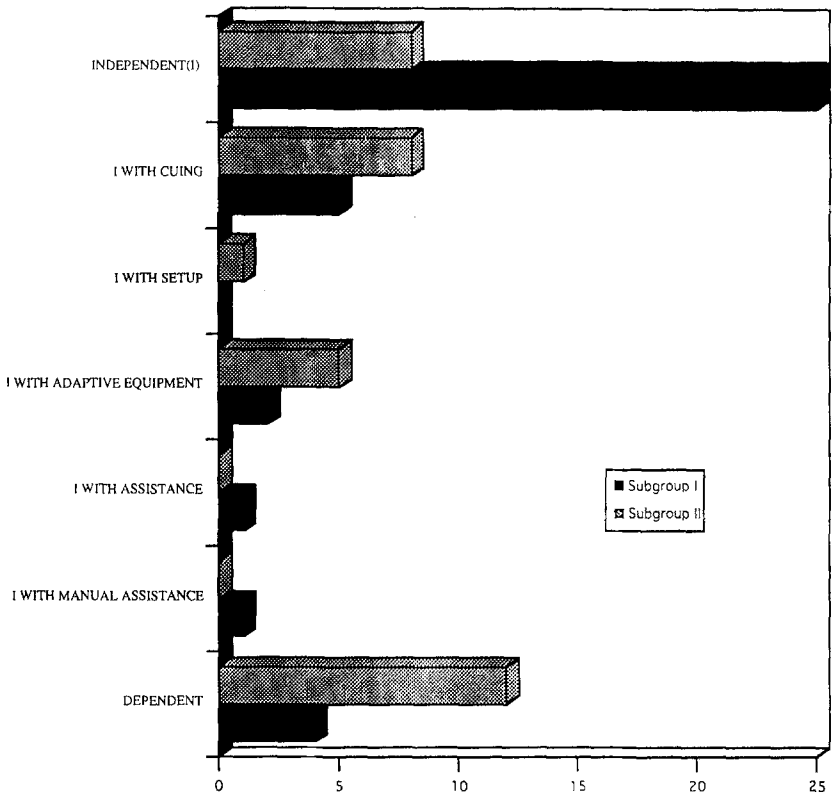

Fig. 3. Self-feeding status in patients with Parkinson's disease (n = 72). Differences between subgroups I and II were analyzed using the Chi-square test after eliminating categories with two or less members.

toms but had an abnormal videofluoroscopic swallowing study; only 1 symptomatic patient in their cohort reported complaints attributable to the prepharyngeal stages of ingestion. Others recount that almost 50% of PD patients voice dysphagic symptoms [2,15].

Using an extensive questionnaire, we obtained an 82% affirmation rate for ingestion-related complaints, most of which reflect pharyngeo-esophageal dysfunction. However, sialorrhea (10%) and impaired mastication (7%) relate specifically to deficits of prepharyngeal phases of ingestion. In 1965, Eadie and Tyrer [2] reported disordered salivation in 78% and chewing difficulty in 12% of their cohort. This prevalence difference may relate to a disparity in disease severity between our two cohorts and to more effective drug therapy available to our patients; the levodopa treatment option for Parkinson's disease began in 1969 [16].

The comparatively small number of dysphagic symptoms relative to abnormalities on barium swallow cineradiography in the PD literature can be explained by several factors. Many dysphagic symptoms are not elicited because limited questionnaires, such as the United Parkinson Disease Rating Scale (UPDRS), are too insensitive [17]. This disability scale questions deglutition function by asking only if patients choke while swallowing. Because dysphagia clinicians interviewed our patients specifically for abnormalities of ingestion, we obtained more frequent reporting and improved detailed description of dysphagic complaints. Dementia also interferes with symptom ascertainment. The dementia prevalence rate increases to almost 32% in patients with more advanced disease [18]. This may explain the large number of our patients with advanced disease who denied any ingestion-related complaint. In some patients, as in normal elderly subjects, aberrant swallows may be too minor to induce symptoms [19]. Other patients may ignore or consider even coughing during meals as "normal" mealtime behavior. Instead, PD patients' spouses or caretakers complain of slowness at meals, thus exemplifying a preoccupation with more obvious, socially disrupting symptoms.

Physicians too may ignore symptomatic dysphagia, particularly when complaints do not include choking, gagging, and impaired swallowing. In several surveys of patients with PD, excess oral saliva or drooling was reported 30%–53% more frequently than difficulty swallowing [2,14,20]. Drooling, though routinely recorded as part of the UPDRS, does not usually evoke physician concern that the patient is dysphagic. Sialorrhea in PD patients is not caused by secretory overproduction [21], but is the likely consequence of reduced swallow initiation and frequency, deficient oral containment, and cervical anteroflexion. Therefore, sialorrhea may be the first complaint of dysfunctional prepharyn-

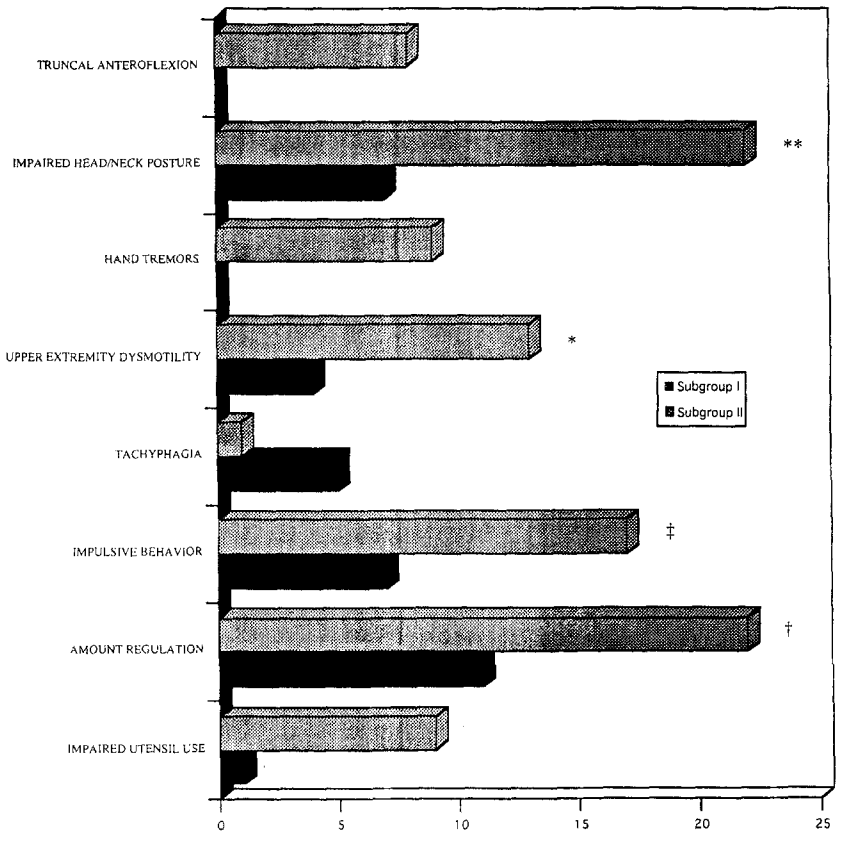

Fig. 4. Occurrence of abnormalities during self-feeding examination in patients with Parkinson's disease (n = 72). Differences between subgroups I and II were analyzed with the Chi-square test. (p:* < 0.006; ** < 0.001; ‡ < 0.005; † < 0.003).

geal ingestion to herald more obvious and critical dysphagia.

"Whilst at meals the fork not being duly directed frequently fails to raise the morsel from the plate: Which, when seized, is with much difficulty conveyed to the mouth." ("an Essay on the Shaking Palsy," James Parkinson, 1817 [1].)

The observational segment of our ingestion evaluation begins by examining its anticipatory phase including those facets of eating that precede the intra-oral placement of a bolus [12]. Half of the studied cohort and all but 2 patients who fed independently, usually those with less advanced disease, manifested no dysfunction during this phase. Patients with severe PD often demonstrated multiple abnormalities. In our study group, aberrations of body positioning, impaired utensil use, and upper extremity dysmotility reflect the influence of PD-related bradykinesia and rigidity on the patterned movements of feeding. However, tachyphagia, common in the hyperkinetic malady Huntington's disease [22], was an unanticipated finding. In some PD patients, this maladaptive motor behavior may represent a benign premorbid feeding behavior that, when combined with pathologic oral motor functions or dementia, increases choking and aspiration risk.

Several of our patients exhibited jaw or lip tremor during meals. One patient had a lingual tremor, a feature reported in 3/6 PD patients studied by Robbins et al. [11] but absent in patients of Bushmann et al. [10]. Our patient's lingual tremor, at times so coarse as to dislodge food from the approaching utensil, stopped or diminished markedly during mastication and transfer initiation. Silbiger et al. [3] also observed suppression of tongue tremor during the volitional phases of swallowing; the tremor returned as the bolus passed into the oropharynx. Volitional effort typically stops or markedly suppresses PD-related limb tremor.

Other anticipatory phase abnormalities in PD patients have been reported by Athlin et al. [23]. Most of their patients, regardless of dementia, displayed difficulty preparing food on the plate, manipulating utensils, placing appropriate quantities of food on utensils, or merely initiating feeding. Half their nondemented patients were apraxic. We observed but did not record some of these features in our study group.

". . .but when the food is conveyed to the mouth, so much are the actions of the muscles of the tongue, pharynx, &c. impeded by impaired action and perpetual agitation, that the food is with difficulty retained in the mouth until masticated; and then as difficultly swallowed." ("An Essay on the Shaking Palsy," James Parkinson, 1817 [1].)

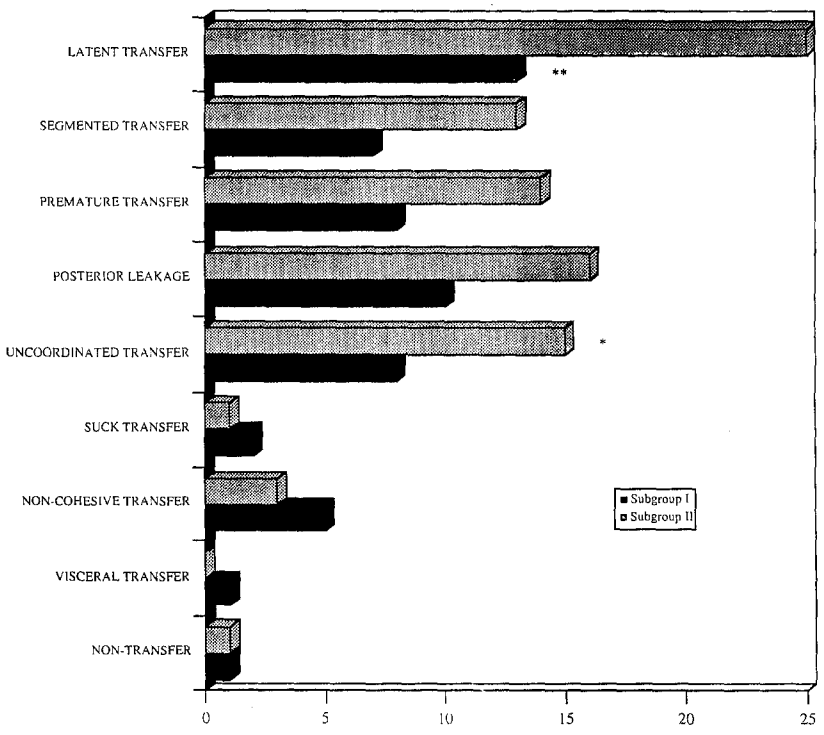

Fig. 5. Occurrence of lingual stage abnormalities in Parkinson's disease (N = 71). Differences between subgroups I and II were analyzed with the chi square test. (p:* < 0.05; ** = 0.001).

The evaluation of oral prepatory and lingual phases of ingestion is both clinical and radiographic. During the oral preparatory phase, buccolingual intentional, automatic, and autonomic motor and sensory functions prepare and position food properly before swallow initiation. The great majority of our PD patients manifested several abnormalities including inefficient oral containment, deficient mastication, and impaired lingual searching and transfer behavior. Other authors may have subsumed the latter two abnormalities in less specific terms including "impaired oral mobility" [10].

In his 1817 monograph, James Parkinson alluded to abnormal mastication [1], the most common motor dysfunction in our study. Eadie and Tyrer [2] noted an increased frequency of impaired mastication coincident with disease progression. Few subsequent reports of dysphagia in PD mention masticatory function; none include radiologic data that characterize anomalous mastication [10,23]. Karlsson et al. [24] measured impaired jaw velocity and displacement in PD patients; both parameters benefitted from levodopa therapy. Further, although not specifically addressed by the authors, their figure exemplifying rhythmic chewing in one of their unmedicated patients also demonstrated fatigue of this repetitive movement, a clinical feature usually elicited in PD patients by repetitive hand or foot tapping.

Impaired mastication is an anticipated motor abnormality in patients with PD, an illness responsible for the gradual loss of associated movements that may leave the primary movement relatively unaltered until later in the disease process. The loss of normal arm swing during ambulation that precedes eventual gait failure exemplifies this concept. Mastication appears to undergo a similar but less consistent and obvious fate. Chewing typically includes both vertical and rotational vectors, the latter being an important but less critical component. When mastication was affected in our subjects, the associated (rotary) mandibular movements were often lost first, usually with no appreciable consequence. However, impaired vertical movements caused ineffectual mastication.

In normal subjects, lingual movement is partly responsible for efficient mastication. During early chewing cycles the tongue guides the bolus between the upper and lower teeth, then moves synchronously with the mandible during later chewing cycles to further help particulate food [25]. Subsequently, synergistic tongue and jaw movements transport food to the tongue before swallow initiation [26]. Impoverished jaw movement during mastication may restrict associated tongue motility during the oral preparatory phase of ingestion. Lingual and masticatory abnormalities during this phase occurred with equal frequency in our cohort, the majority being concordant for both.

The lingual phase of ingestion propels food or liquid into the oropharynx. James Parkinson reported lingual phase dysfunction in his original description of "The Shaking Palsy." Subsequent authors identified delayed swallowing initiation, hesitant deglutition, and impaired lingual peristalsis [10,27,28]. The latter term ap-

pears generic for a variety of disturbed swallow-initiated tongue movements observed in patients with PD such as lingual pumping, prolonged lingual elevation blocking the normal bolus trajectory [8,13], piecemeal deglutition, and tongue thrusting. Although our terminology differs, we also observed many similar anomalies of lingual movement in a majority of our patients. It is unclear if any of these lingual abnormalities is specific for Parkinson's disease or is particularly more injurious to efficient deglutition. Indeed, similar cineradiographic glossal abnormalities have been reported in normal subjects [18,29,30] and in patients with other neurologic diseases such as amyotrophic lateral sclerosis, myasthenia gravis, and Alzheimer's disease [27,31,32].

The pathophysiology of prepharyngeal ingestion dysfunction in PD patients is, like other volitional motor functions, secondary to impaired basal ganglia motor control. Diminished nigrostriatal dopamine activity dominates the neurochemical deficiencies that results in parkinsonian rigidity and akinesia. Muscular rigidity, usually evaluated by passively moving a joint, disrupts volitional movements. We detected temporomandibular joint (TMJ) rigidity, a feature not previously reported in PD patients. However, rigidity assessment of the TMJ is less secure because its relaxation is often difficult to achieve, especially in the more demented, less cooperative patient. Other than minor TMJ movement, there is no joint motion during the lingual phase of ingestion. Although unproven, Denny-Brown [33] and Martin [34] suggested lingual "rigidity" to explain reduced tongue movements seen in patients with PD.

Without invoking rigidity, akinesia and bradykinesia may satisfactorily explain most aspects of prepharyngeal dysmotility during ingestion in PD patients. Although demonstrable during simple movements, akinesia is most clinically relevant during simultaneous and sequential movements [35] such as those of the oral preparatory and lingual phases of ingestion. Marsden [36] predicted increasing inefficiencies of more prolonged sequential motor programs. When compared with the motor sequence played during ingestion of liquids, the motor melody of food ingestion is longer, more complex, and therefore at greater risk to be disrupted. Indeed, Robbins et al. [11] recorded many more cineradiographic abnormalities of lingual motility in their PD patients using semisolids than liquids. Impaired perioral sensorimotor integration, as evidenced by deficient jaw proprioception and lingual tactile localization in patients with PD [37], may enhance akinesia and further compromise prepharyngeal ingestion.

Although we acquired our study data prospectively, our interest in dysphagia as a reportable topic developed *post facto,* thus resulting in several methodological limitations. Our patients' neurologic disability status was determined by the H&Y disability scale, a relatively unrefined scale that is heavily weighted on gait and postural stability [12]. The UPDRS [16] or other detailed functional rating scales would have permitted us to correlate various motor and cognitive deficits with the presence, severity, and characteristics of prepharyngeal dysphagia. However, in agreement with Edwards et al. [15], our H&Y-based data permit us to anticipate an increased prevalence and severity of dysphagia with advancing disease.

Drug therapy modulates volitional movements in patients with PD. Except in the early stages of the disease when patients are "long-responders," levodopa therapy often provokes a variety of motor fluctuations, including a "wearing off effect" (increased bradykinesia) and dopa-induced dyskinesias (hyperkinesias). Except for Bushmann et al. [10], we and other investigators made no effort to consider motor fluctuations by conducting the clinical dysphagia examination or barium cineradiography at a consistent period after levodopa dosing. Motor fluctuations are of particular importance if the beneficial effect of drug treatment on dysphagia is to be assessed. We believe that dysphagia therapy may be best conducted when it coincides with a drug schedule that promotes symptomatic improvement of PD. Likewise, a dysphagia treatment schedule that anticipates peaks of drug-related parkinsonian improvement may also enhance therapy effectiveness.

This report provides a profile of dysphagia characteristics in patients with PD while identifying abnormalities in the early phases of ingestion not previously reported. Using a similar diagnostic format, and hopefully a standardized nomenclature, confirmatory results will enhance our understanding of basal ganglia influences on buccolingual and consequent pharyngeo-esophageal function.

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