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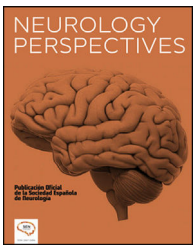

ORIGINAL ARTICLE

Findings in orofacial praxis in clinical swallow examination in patients with neurogenic oropharyngeal dysphagia

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Abstract
Introduction: Praxis facilitate the execution of learned motor acts, including swallowing. Inability to perform praxis movements, or apraxia, may be classified according to different criteria. Buccopharyngeal apraxia includes orofacial apraxia. Clinical swallow evaluation does not typically include assessment of oropharyngeal/orofacial praxes; however, this would be of great semiological value, particularly in patients with neurological and neuromuscular disorders who present with dysphagia. The objective of this study was to explore and compare findings from the assessment of 3 orofacial praxis in healthy individuals without dysphagia and in patients with neurogenic oropharyngeal dysphagia, as part of the clinical swallow examination.
Methods: We designed a case–control study based on a clinical swallow examination that included an assessment of 3 orofacial praxis. Comparisons were made, and odds ratios with 95% confidence intervals were calculated. Non-performance of the 3 orofacial praxis was recorded separately and jointly, adjusting for sex and age in both groups, as well as for other clinical variables of interest in the patient group.
Results: Our study included 86 patients and 80 controls. Oral motor apraxia was associated with oropharyngeal dysphagia; more specifically, the presence of neurogenic oropharyngeal dysphagia was found to be associated with difficulty in moving both lips together to either side, as well as with such other physical examination findings as tongue fasciculations and atrophy, and impaired tongue coordination. No statistically significant association was observed with age or sex in either group.
Conclusion: Assessment of orofacial praxes can help to identify patients with neurogenic oropharyngeal dysphagia.

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PALABRAS CLAVE
Apraxias;
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Hallazgos en praxias orofaciales en la evaluación clínica de la deglución en pacientes con disfagia orofaríngea neurogénica

Resumen
Introducción: Las praxias facilitan la ejecución de actos motores aprendidos, incluyendo el acto de tragar. Su alteración o apraxia se clasifica de varias formas, donde están las apraxias bucofaríngeas que incluye las orofaciales. Habitualmente la evaluación clínica de la deglución no contiene la ejecución de praxias bucofaríngeas/orofaciales, de valour semiológico, potencialmente útil en la valoración de pacientes neurológicos y neuromusculares que cursan con disfagia. Es objetivo explorar y comparar los hallazgos en la evaluación de tres praxias oro faciales entre sanos sin disfagia y pacientes con disfagia orofaríngea neurogénica, como parte de la evaluación clínica de la deglución.
Métodos: Estudio de casos y controles donde se aplicó por igual una evaluación clínica de la deglución que incluyó tres praxias orofaciales. Se realizaron comparaciones, obteniendo odds ratio acompañados de intervalos de confianza del 95%, identificando la no ejecución de forma individual y agrupada de apraxias, ajustando por sexo y edad, más otras variables clínicas de interés en los casos.
Resultados: 86 casos y 80 controles. El conjunto de apraxias oro motoras se relacionó con la disfagia orofaríngea, puntualmente la alteración en la praxia oro facial de llevar labios juntos a los lados se asoció con la presencia de disfagia orofaríngea neurogénica, más otros hallazgos al examen físico como fasciculación, atrofia e incoordinación lingual. En ambos grupos ni la edad y el sexo influyeron en las asociaciones.
Conclusiones: Las praxias oro faciales en la evaluación clínica de la deglución puede ayudar en la identificación de pacientes con disfagia orofaríngea neurogénica.

Introduction

The term praxis refers to neurological processes that facilitate and support the performance of a previously learned motor act or the ability to formulate movements in a non-paretic limb or other part of the body, through the planning of a schema based on complex representations of learned movements.[1]

Apraxias are a series of cognitive disorders that are not attributed to an alteration in motor, sensory, and/or coordination function. In general terms, apraxia is the inability to produce voluntary movements or learned motor acts on command[1] due to lesions that cause a disconnect between an idea (cognition of movement) and its execution (muscular materialisation).[2] Patients with apraxia are unable to perform specific, predefined actions nor learned, purposeful movements, independently of sensory, motor, and/or cognitive deficits impairing recognition of the stimulus, understanding of the task, or performance or the response,[3] with a discrepancy between the planned action and actual performance.

Apraxias are typically classified as ideational, ideomotor, limb-kinetic, dressing, or constructional.[1] However, other types exist, such as buccopharyngeal, speech, gait, oculomotor, eyelid-opening, and arm apraxia.[1] They can also be classified clinically, according to the affected part of the body: limb, trunk, or facial apraxia (the latter includes ocular and oral apraxias).[1]

Buccopharyngeal apraxia, including buccofacial, orofacial, oral, and bucco-linguo-facial apraxias, is defined as the inability to perform voluntary movements of the laryngeal, pharyngeal, lingual, labial, and/or facial muscles, in the absence of paresis or paralysis, and despite preservation of automatic movements of the same muscles.[4] They are assessed by verbally instructing the patient to open their mouth, stick out their tongue, blow, show their teeth, purse/pucker their lips, touch their nose or chin with their tongue, bite their lower lip, move their tongue in and out, click their teeth together, smile, carry the tongue to the roof of the mouth, cough, puff out their cheeks, wiggle their tongue from side to side, or hum.[1]

Dysphagia is the impairment of swallowing, in which patients have difficulty safely moving the food bolus from the oral cavity to the stomach.[5] It may present in isolation or as part of a systemic disease, and most frequently has an underlying neurological cause.[6,7] Dysphagia can be classified clinically as oropharyngeal or oesophageal, and aetiologically as structural, motor, or functional.[8] The latter form is characterised by impairment of swallowing physiology by entities that compromise the neurological control of the oral and pharyngeal phases, modulation of peristalsis, neuromuscular coordination of sphincters, and/or muscle action.[8,9]

Functional dysphagia includes neurogenic oropharyngeal dysphagia (NOD),[8,10] one of the most frequent forms of dysphagia, which can lead to respiratory and nutritional

secondary complications.[9,11,12] Swallowing impairment in neurological diseases can range from mild to severe.[13]

Assessment of praxis is part of neurological examination, the fundamental element of clinical practice that identifies whether a patient has a neurological problem, indicating the level of involvement and the possible aetiology. Detection of apraxia in the neurological examination demonstrates involvement of the cerebral cortex, until evidence to the contrary is identified.[3,4]

Clinical swallow examination (CSE) is the most widely used standard formal, clinical assessment of swallowing disorders, and frequently constitutes the first or even the only assessment used to investigate signs of suspected dysphagia.[14] However, CSE usually does not include assessment of praxis in general, or buccopharyngeal praxis in particular.

This study aims to explore and compare findings from the assessment of 3 orofacial (oral motor) praxis included in the CSE in patients with NOD and in healthy controls without dysphagia.

Methods

We conducted a case–control study based on semiological findings from CSE including orofacial praxis, which was performed in healthy individuals (no dysphagia) and patients with NOD of neurological or neuromuscular aetiology.

Using the Epidat® software, we calculated a minimum sample size of 76 patients with NOD and 76 healthy controls, based on a reported sensitivity of CSE of 80%,[15] with a statistical power of 80% and confidence interval of 95%.

Patients were recruited at 12 private speech therapy and swallowing clinics, 10 healthcare centres with dysphagia services, 4 healthcare centres for elderly people, and 3 patients' associations located in Valle de Aburrá y de San Nicolas in Antioquia, Colombia. Controls were recruited at 2 social and leisure centres for elderly people, 2 universities, and one community action association, located in Valle de Aburrá (Medellín).

Inclusion criteria for the patient group were as follows: age $\geq$ 18 years; either sex; presence of NOD of at least one month's progression; and diagnosis of some neurological or neuromuscular disorder causing oropharyngeal dysphagia. The presence of dysphagia symptoms was defined as a total score $\geq$ 3 points on the Eating Assessment Tool–10 (EAT-10), an instrument validated for use in Colombia.[16,17] We excluded patients with oesophageal, mechanical, propulsion, or iatrogenic dysphagia; history of face and/or neck radiotherapy for cancer; history of surgical procedures involving the skin of the neck in the last 3 months; or advanced dementia hindering comprehension of the commands to chew or swallow. We also excluded patients undergoing active procedures for endodontic treatment; with congenital malformations of the oral cavity, tongue, and/or neck; or history of Sjögren syndrome.

Inclusion criteria for the control group were as follows: age $\geq$ 18 years; either sex; no diagnosis of dysphagia or central/peripheral nervous system or neuromuscular disorders; no history of cancer in the head or neck; no history of surgical procedures in the lower two-thirds of the face or neck; and no history of botulinum toxin treatment. We excluded healthy individuals undergoing active procedures for endodontic treatment; with congenital malformations of the oral cavity, tongue, and/or neck; and with history of Sjögren syndrome and/or cognitive decline.

Selection criteria for the patient group were applied by a neurologist with experience in the management of swallowing and NOD, supported by a speech therapist with training in swallowing/dysphagia. In the control group, criteria were applied by a physician specialised in neurological rehabilitation, with training in swallowing and NOD.

We verified that controls and patients with orofacial apraxia did not present sensory or motor involvement of the trigeminal or of the facial nerve, to ensure that impaired performance of any of the praxes evaluated could not be attributed to impaired orofacial motor and/or sensory function.

In both groups, the same CSE protocol was applied, with a clinical history interview focused on swallowing and symptoms of dysphagia; examination of the anatomy, functioning, sensitivity, and reflexes of the swallowing apparatus (oral and pharyngeal phase of swallowing)[18]; and examination of the oral cavity and the lower cranial nerves plus the trigeminal and facial nerves, which also included the command to perform 3 orofacial (buccopharyngeal) praxis[1]: puckering the lips (pressing them together and pushing them forward), smiling (retracting the lips and showing the teeth), and moving the lips together to either side (left and right). If the subject did not initially perform the action after the verbal command, they were instructed to imitate the action. The CSE protocol was administered by a different professional in each group: by a neurologist in the patient group, and by a physician specialising in neurological rehabilitation in the control group.

We performed a descriptive statistical analysis of the results. Quantitative variables were tested for normal distribution using the Shapiro–Wilk test; we also performed a comparative analysis of the findings from the physical examination to identify variables showing significant differences. Crude odds ratios (OR) were calculated with 95% confidence intervals. Qualitative variables were compared using the chi-square test or the Fisher exact test. Non-performance of the 3 orofacial praxes (apraxia) was recorded separately and jointly, and adjusted for sex and age in both groups. In the patient group, we also compared apraxia adjusted for the other potentially relevant variables, such as progression time of the disease responsible for NOD, progression time of dysphagia, and self-perceived dysphagia symptoms according to EAT-10 score. Statistical analysis was performed using the Jamovi statistics software (version 2.5.5).

Due to statistical considerations, age groups of <60 and ≥ 60 years were established. Similarly, progression time was classified as ≤ 4.5 and >4.5 years for neurological/neuromuscular disease and ≤ 1.3 and >1.3 years for dysphagia; EAT-10 score was classified as ≤ 16 and >16 points. None of these 4 variables were normally distributed; the median was used as the cut-off point for establishing these groups.

Results

From March 2019 to December 2021, we established a total sample of 166 individuals: 86 patients and 80 controls. All participants underwent CSE including facial praxis.

Men accounted for 53% of the total sample (88/106) and 59.3% of the patient group (51/86). The majority of controls were women (43/80; 53.8%). Age was not normally distributed (Shapiro–Wilk test: $P < .001$), with a median of 61 years (Q_1–Q_3: 51–68) in the total sample, 60.5 years in the patient group, and 61.5 years in the control group. At the time of the study, feeding was oral in all controls and practically all patients. Table 1 presents detailed information on the sociodemographic and clinical characteristics of the study population.

In the patient group, NOD involved central nervous system mechanisms in 88.4% of cases (76/86) and neuromuscular mechanisms in 11.6% (10/86).

The subgroup of NOD due to central nervous system involvement included 18 patients with Parkinson's disease (23.7%), 14 with amyotrophic lateral sclerosis (18.4%), 13 with stroke (17.1%), 10 with multiple sclerosis (13.2%), 7 with dementia (9.2%), 5 with traumatic brain injury (6.6%), 4 with cerebral palsy (5.3%), 3 with ataxia (3.9%), and 2 patients (2.6%) with other causes (one with thalamotomy due to abnormal movements and one with hydrocephalus secondary to neurocysticercosis).

Neuromuscular causes of NOD in our sample included muscular dystrophy (5/10; 50%), dermatomyositis (2/10; 20%), neuropathy (2/10; 20%), and myasthenia gravis (1/10; 10%).

In the patient group, we observed a median progression time of 4.5 years for the main diagnosis responsible for oropharyngeal dysphagia (Table 2).

The median progression time of dysphagia was 1.33 years. The most frequent symptoms of dysphagia were the sensation of food sticking in the throat after swallowing, coughing after eating, and the sensation of food sticking in the neck. The median EAT-10 score was 16. The progression times of dysphagia and of the disorder responsible for dysphagia did not follow a normal distribution (Shapiro–Wilk test: $P < .001$ in both cases). Table 2 shows the clinical characteristics of oropharyngeal dysphagia in the patient group.

Several CSE findings were only detected in the patient group, including impaired control of the head, sensory alterations of the mandibular branch of the trigeminal nerve and impaired jaw closing, facial nerve alterations (motor and gustatory fibres), difficulty with tongue protrusion and retrusion, tongue atrophy, deviated uvula, dry mouth, and impairment in protruding the lips and smiling (orofacial apraxia). However, other signs assessed in the physical examination were observed in both groups. Being a healthy control (no NOD) did not rule out the presence of any physical and/or neurological signs in the physical examination, without these necessarily being indicative of swallowing disorders or having pathological significance.

Among the set of signs observed in the physical examination, we found statistically significant differences between groups (with greater frequency among patients with NOD) in the following signs: impaired jaw jerk reflex and sensory impairment in the mandibular region, central facial palsy, alteration of tongue strength and coordination, tongue atrophy and fasciculations, dry mouth, and impaired performance in the oral praxis of moving the lips together to either side. The majority of variables related to the mouth, cooperation, and interaction, control of the head, the motor branch of the trigeminal nerve (jaw movement), and movement of the tongue showed no statistically significant differences between groups. Table 3 presents detailed data on the comparison between groups of the signs assessed in the physical examination.

When alteration in any of the 3 praxis assessed was treated as a single category (oral motor apraxia), the OR for dysphagia was 12.8 (Table 4).

The control group showed no statistically significant differences or associations between oral facial praxis performance (either individual or combined) and such other variables as sex or age.

The patient group showed no statistically significant differences or associations between the performance of any individual oral facial praxis and the variables sex, age, progression time of dysphagia or of the disorder responsible for dysphagia, or EAT-10 score. However, the presence of oral motor apraxia (alteration in any of the 3 movements assessed) was significantly more frequent in men (presence of some oral motor apraxia increased the odds of being a man with NOD). Table 5 presents detailed information on the comparisons performed between apraxias and the variables sex, age, progression time of dysphagia or the disorder responsible for dysphagia, and EAT-10 score in the patient group.

Table 1 Characteristics of the study population.

Variable		Total (n = 166)			P
		Patients (n = 86)	Controls (n = 80)		
Age (years)	Median (Q_1–Q_3)	60.5 (48.3–68)	61.5 (54–66.3)	3361	0.798*
	Range	18–84	22–90		
Sex	Women, n (%)	35 (40.7)	43 (53.8)	2.83**	0.092
	Men, n (%)	51 (59.3)	37 (46.3)		
Feeding	Oral, n (%)	81 (94.2)	80 (100)	4.8**	0.091
	Gastrostomy, n (%)	2 (2.3)	0 (0)		
	Mixed, n (%)	3 (3.5)	0 (0)		

Q_1–Q_3: quartiles 1 and 3.
Source: original data.
* Mann–Whitney U test.
** Chi-square test.

Table 2 Clinical characteristics of oropharyngeal dysphagia in the patient group.

Variable		Patients (n = 86)
Reports problems initiating swallowing, n (%)		28 (32.6)
Sensation of food sticking in the seconds after swallowing, n (%)		77 (89.5)
Location of sensation of stuck food, n (%)	Mouth	14 (18.2)
	Neck	55 (71.4)
	Chest	1 (1.3)
	Not specified	7 (9.1)
Coughing before swallowing, n (%)		15 (17.4)
Coughing after swallowing, n (%)		63 (73.3)
Sensation of breathlessness after swallowing, n (%)		40 (46.5)
Nasal regurgitation of food, n (%)		11 (12.8)
Difficulty chewing, n (%)		35 (40.7)
Type of food with greatest difficulty swallowing, n (%)	Liquid	16 (18.6)
	Solid	52 (60.5)
	Both	18 (20.9)
Total EAT-10 score	Mean (SD)	17.3 (8.57)
	Median (Q_1–Q_3)	16 (10.5–21)
	Range	3–40
Progression time (years) of the disorder responsible for dysphagia	Mean (SD)	9.75 (12.2)
	Median (Q_1–Q_3)	4.5 (1–16.5)
	Range	0.1–65
Progression time (years) of dysphagia	Mean (SD)	3.18 (5.48)
	Median (Q_1–Q_3)	1.33 (0.62–3)
	Range	0.08–31

EAT-10: Eating Assessment Tool–10; Q_1–Q_3: quartiles 1 and 3; SD: standard deviation.

Discussion

NOD, as a subtype of dysphagia, can result from various different conditions affecting swallowing physiology.[9] Neurological aetiologies cause disruption of the neurological mechanisms of swallowing secondary to alterations in the central nervous system, whereas in neuromuscular causes, swallowing impairment is caused by alterations in nerves, the motor endplate, and/or pharyngeal or oesophageal muscles.[19] NOD presents with various sensory and motor signs, which can be detected in CSE.

CSE, or clinical non-instrumental evaluation of dysphagia, aims to establish the presence and severity of dysphagia, identify the causes, assess the need for instrumental examination, propose a rehabilitation plan, and evaluate treatment outcomes.[18]

In our study, findings (signs) from physical examination of the trigeminal, facial, and hypoglossal nerves, the oral cavity, and the tongue showed statistically significant differences between patients and controls, supporting the use of CSE in patients with neurological and neuromuscular diseases. CSE typically involves physical examination of the cranial nerves and oral cavity, but not assessment of orofacial or buccopharyngeal motor praxis.

A novel result was the impaired performance observed in orofacial praxes, which are relatively simple to assess during neurological physical examination. Difficulty in moving the lips together to either side (a form of orofacial apraxia) was significantly associated with NOD. This association persisted when alteration in any of the 3 movements assessed was treated as a single variable (oral motor apraxia). This is a preliminary finding, and further study is needed. However, we may tentatively suggest that it may be useful in differentiating patients with NOD from healthy individuals without dysphagia. Methodologically, we verified that alterations in praxis performance in patients and in controls were not secondary to motor or sensory impairment of the facial muscles or muscles of mastication.

Oral apraxias are a subtype of ideomotor apraxia (impaired performance in the use of tools or gestures) that involves the orofacial muscles,[20] which are involved in swallowing. Performance and clinical assessment of orofacial or buccofacial praxes (through instructing patients to perform or to imitate an action: smiling, puckering/protrusion of the lips, moving the lips together to either side) constitute a neurophysiological assessment of the ability to perform oral and facial sequences, that is, a means of clinical evaluation of the integration of cognitive aspects of cortical control and motor execution in swallowing.

Swallowing is a complex neuromuscular process and event,[21] and requires precise coordination of over 25 pairs of muscles, intact pharyngeal sensation, and central control in the brainstem and cortex,[22,23] as well as intact cognition,

Table 3 Physical examination findings in patients and controls.

Variable		Patients (n = 86) n (%)	Controls (n = 80) n (%)	OR	P	95% CI
Problems with cooperation		1 (1.2)	0 (0)	2.82	1**	0.113–70.3
Problems with interaction		2 (2.3)	0 (0)	4.76	0.498**	0.225–101
Difficulty controlling the head		5 (5.8)	0 (0)	10.9	0.06**	0.591–200
Olfactory nerve alterations		31 (36)	23 (28.7)	1.4	0.316*	0.726–2.69
Trigeminal nerve	V3 sensory impairment	16 (18.6)	0 (0)	37.7	**<0.001***	2.22–640
	Impaired jaw closing	6 (7)	0 (0)	13	0.029**	0.720–235
	Impaired jaw laterotrusion	11 (12.8)	5 (6.3)	2.2	0.154*	0.729–6.64
	Impaired jaw protrusion	14 (16.3)	5 (6.3)	2.92	0.043*	0.999–8.51
	Impaired jaw retrusion	16 (18.6)	16 (20)	0.91	0.82*	0.423–1.98
	Impaired jaw jerk reflex	34 (39.5)	2 (2.5)	25.5	**<0.001***	5.87–111
Facial nerve	Gustatory alteration	8 (9.3)	0 (0)	17.4	0.007**	0.989–307
	Central facial palsy	12 (14)	0 (0)	27.0	**<0.001***	1.57–464
	Peripheral facial palsy	6 (7)	0 (0)	13.0	0.029**	0.72–235
Hypoglossal nerve and tongue	Impaired tongue strength	16 (18.6)	1 (1.3)	18.1	**<0.001***	2.33–140
	Difficulty raising the tongue	10 (11.6)	3 (3.8)	3.38	0.059*	0.984–12.8
	Difficulty in tongue protrusion	8 (9.3)	0 (0)	17.4	0.007**	0.989–307
	Difficulty in tongue retrusion	8 (9.3)	0 (0)	17.4	0.007**	0.989–307
	Impaired tongue motility	7 (8.1)	3 (3.8)	2.27	0.332**	0.567–9.12
	Impaired tongue coordination	11 (12.8)	1 (1.3)	11.6	**0.004***	1.46–91.9
	Tongue fasciculation	24 (27.9)	1 (1.3)	30.6	**<0.001***	4.03–232
	Tongue atrophy	10 (11.6)	0 (0)	22.1	**0.002****	1.27–384
Oral cavity	Partial prosthesis	25 (29.1)	22 (27.5)	1.08	0.823*	0.549–2.13
	Full prosthesis	11 (12.8)	10 (12.5)	1.03	0.955*	0.411–2.57
	Edentulism	10 (11.6)	10 (12.5)	0.92	0.863*	0.362–2.35
	Inflamed gums	7 (8.1)	1 (1.3)	7	0.065**	0.842–58.2
	Dry mouth	19 (22.1)	0 (0)	46.5	**<0.001***	2.76–785
	Deviated uvula	8 (9.3)	0 (0)	17.4	0.007**	0.989–307
Performance of oral motor praxes	Difficulty in lip protrusion	4 (4.7)	0 (0)	8.78	0.121**	0.465–166
	Difficulty smiling	4 (4.7)	0 (0)	8.78	0.121**	0.465–166
	Difficulty in moving both lips together to either side	9 (10.5)	1 (1.3)	9.23	**0.019****	1.14–74.6

CI: confidence interval; OR: odds ratio; V3: third trigeminal branch or mandibular nerve.
* Chi-square test.
** Fisher exact test.

adequate sensory processing, reward and motivation mechanisms, sensorimotor control, protection of the respiratory tract, and intact involuntary function.[13] Unlike other voluntary movements, successful swallowing concludes with a reflex stage,[21] but begins with a conscious stage involving learned, voluntary orofacial movements.

Table 4 Oral motor apraxia in patients and controls.

	Patients (n = 86) n (%)	Controls (n = 80) n (%)	OR	P	95% CI
Oral motor apraxia	12 (14)	1 (1.3)	12.8	0.002*	1.63–101
No oral motor apraxia	74 (86)	79 (98.8)			

CI: confidence interval; OR: odds ratio.
* Chi-square test.

Neurophysiological studies have shifted our conception of swallowing from the idea of a purely reflexive, automatic mechanism towards the idea of a modelled response involving several levels of the nervous system, both cortical and subcortical,[24] with different brain regions performing different functions in anticipation, preparation, and execution.[21]

Apraxias in general constitute dysfunctions of motor acts, unrelated to alterations in the primary motor cortex or pyramidal tracts, with orofacial apraxias representing a loss of voluntary control of muscles of the face, tongue, pharynx, and/or mastication despite spared muscle, reflex, spontaneous, and automatic function.[25]

Few studies have focused on the assessment of apraxia in patients with dysphagia in general and NOD in particular. A study including 60 patients with first ischaemic stroke and signs of dysphagia found buccofacial apraxia to be associated with lesion laterality,[26] suggesting the existence of a dominant brain hemisphere in swallowing: the left hemisphere was more frequently dominant in the early, voluntary stage of swallowing,[27] with the left parietal

Table 5 Comparisons between orofacial apraxias and the variables sex, age, progression time of dysphagia or the disorder responsible for dysphagia, and Eating Assessment Tool−10 score in the patient group.

Sex	Women (n = 35) n (%)	Men (n = 51) n (%)	OR	P	95% CI
Difficulty in lip protrusion	0 (0)	4 (7.8)	0.149	0.142**	0.007−2.85
Difficulty smiling	1 (2.9)	3 (5.9)	0.471	0.643**	0.046−4.72
Difficulty in moving both lips together to either side	1 (2.9)	8 (15.7)	0.158	0.076**	0.018−1.33
Presence of some orofacial motor apraxia	1 (2.9)	11 (21.6)	0.107	**0.023****	0.013−0.871
Age	<60 years (n = 40) n (%)	≥60 years (n = 46) n (%)	OR	P	95% CI
Difficulty in lip protrusion	3 (7.5)	1 (2.2)	3.65	0.334**	0.364−36.6
Difficulty smiling	2 (5)	2 (4.3)	1.16	1**	0.156−8.62
Difficulty in moving both lips together to either side	4 (10)	5 (10.9)	0.911	1**	0.227−3.65
Presence of some orofacial motor apraxia	5 (12.5)	7 (15.2)	0.796	0.717*	0.231−2.74
Progression time of the neurological or neuromuscular disease, n (%)	≤4.5 years (n = 40) n (%)	>4.5 years (n = 40) n (%)	OR	P	95% CI
Difficulty in lip protrusion	2 (5)	2 (5)	1	1**	0.134−7.47
Difficulty smiling	2 (5)	2 (5)	1	1**	0.134−7.47
Difficulty in moving both lips together to either side	3 (7.5)	6 (15)	0.459	0.481**	0.106−1.98
Presence of some orofacial motor apraxia	4 (10)	8 (20)	0.444	0.210	0.122−1.62
Progression time of dysphagia, n (%)	≤1.3 years (n = 42) n (%)	>1.3 years (n = 41) n (%)	OR	P	95% CI
Difficulty in lip protrusion	2 (4.8)	2 (4.9)	0.975	1**	0.131−7.27
Difficulty smiling	3 (7.1)	1 (2.4)	3.08	0.616**	0.307−30.9
Difficulty in moving both lips together to either side	5 (11.9)	3 (7.3)	1.71	0.713**	0.381−7.68
Presence of some orofacial motor apraxia	7 (16.7)	4 (9.8)	1.85	0.353*	0.498−6.87
Self-perception of symptoms (EAT-10 score)	≤16 (n = 46) n (%)	>16 (n = 40) n (%)	OR	P	95% CI
Difficulty in lip protrusion	2 (4.3)	2 (5)	0.864	1**	0.116−6.43
Difficulty smiling	2 (4.3)	2 (5)	0.864	1**	0.116−6.43
Difficulty in moving both lips together to either side	4 (8.7)	5 (12.5)	0.667	0.728**	0.166−2.67
Presence of some orofacial motor apraxia	6 (13)	6 (15)	0.850	0.794*	0.251−2.88

CI: confidence interval; OR: odds ratio. EAT-10: Eating Assessment Tool-10.
* Chi-square test.
** Fisher exact test.

cortex being dominant in the execution of ideomotor praxis.[28]

Several brain regions have been implicated in swallowing processes, such as the inferior primary motor cortex (M1) and primary somatosensory cortex (S1), bilateral insula, cingulate gyrus, and supplementary motor area.[29,30] The latter region appears to be relevant in the pathophysiology of neurogenic dysphagia,[31] given the fact that praxis shows a greater neuroanatomical relationship with association areas. The somatosensory association area, frontal operculum, and superior temporal gyrus play a greater role in preparation and execution of voluntary phases of swallowing,[32] which respond to cognitive or perceptual aspects that are affected in patients with NOD, where impairment of these mechanisms can cause apraxia.

Buccopharyngeal apraxias are disorders of voluntary movement of the tongue, jaw, and lips in tasks other than speech; they differ from apraxia of speech in that the latter involves impairment of the ability to voluntarily perform movements involved in speech articulation.[1]

Oropharyngeal apraxia has been identified as a pathophysiological factor involved in premature bolus spillage,[23,33] one of the 7 neurogenic dysphagia phenotypes typically observed in patients with supratentorial and acute stroke, according to studies using flexible endoscopic evaluation of swallowing.[23]

Different studies have reported buccofacial apraxia secondary to stroke (patients with stroke tend to present apraxia in the upper and lower third of the face, including the lips, cheeks, and tongue),[34] Alzheimer's disease

(severity of dementia is associated with orofacial apraxia and apraxia of speech),[35] corticobasal degeneration,[36] and motor neuron disease.[25]

A retrospective study reporting videofluoroscopy findings from 130 patients with supratentorial stroke observed an association between oral apraxia and dysphagia, particularly in the oral phase with thick liquids and in the pharyngeal phase with thin liquids. The authors concluded that there is a need for formal evaluation of oral apraxia in patients with stroke and dysphagia.[37]

Impaired orofacial motor execution, or orofacial apraxia, appears not to be associated with age, either in patients with NOD or in controls. In the patient group, apraxia was not associated with progression time of dysphagia or of the neurological or neuromuscular cause, or with self-perceived symptoms (EAT-10 score). However, the single variable orofacial apraxia, defined as impairment in any of the movements assessed, did show an association with male sex. A literature search identified no studies reporting sex-related differences in praxis execution or the presence of a given type of apraxia.

The strengths of this study include the participant recruitment and selection process (which achieved groups of similar age and sex, in which physical examination findings were comparable) and the application in both groups of the same assessment protocol, which included oral motor praxis. We controlled for selection and information bias in both groups by checking a range of eligibility criteria and through the use of patient assessments by different professionals (specialists in both cases) with training in swallowing and dysphagia, who administered CSE according to the same protocol. As a limitation of the study, we should mention the small number of patients with NOD caused by neuromuscular disorders; a larger group would have enabled comparison between patients with neurological and neuromuscular aetiology.

Building upon the assessment of oral motor apraxia in patients with NOD and healthy controls, future studies could analyse the degree of apraxia in patients with NOD of different aetiologies (eg, oral motor apraxia in Parkinson's disease vs in amyotrophic lateral sclerosis); compare orofacial apraxia findings in patients with NOD of neurological vs neuromuscular aetiology and with other types of dysphagia (eg, mechanical, propulsion, or other types of functional dysphagia); and evaluate the capacity of apraxia assessment in distinguishing and classifying the cause of dysphagia in general.

Conclusions

Beyond the semiological value of the alterations or signs observed in the physical examination of the trigeminal, facial, and hypoglossal nerves, orofacial apraxias may be helpful in identifying patients with NOD; this examination could be implemented by different healthcare professionals responsible for screening and assessing patients with neurological and neuromuscular disorders that progress with oropharyngeal apraxia.

Clinical assessment of patients with NOD should include cognitive aspects of swallowing, such as buccopharyngeal praxis, as neurological and neuromuscular aetiologies may cause a disconnect between the idea and the muscular execution of swallowing.

Informed consent

All participants from both groups gave written informed consent to participate in the study and for the publication of the results.

Ethical considerations

This study was approved by the research ethics committees of Universidad Pontificia Bolivariana (meeting no. 7, 1 June 2017), Fundación Hospitalaria San Vicente Paúl (meeting no. 35–2018, 21 December 2018), and Clínica Somer (meeting no. 1–2019, 8 February 2019), and endorsed by several institutions, private clinics, and patients' associations. We respected the right to privacy and complied with all ethics regulations.

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Declaration of competing interest

None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.neurop.2025.100191.

References

1. Chandra SR, Issac TG, Abbas MM. Apraxias in neurodegenerative dementias. Indian J Psychol Med. 2015;37(1):42–7. doi:10.4103/0253-7176.150817.
2. Torrentera M, Lazcano G. Capítulo 102: Enfermedad vascular cerebral y sus secuelas.In: García R, Botello G, editors. Práctica de la Geriatría 3e. New York: McGraw-Hill; 2015. p. 1–26.
3. Cubelli R. Definition: Apraxia. Cortex. 2017;93:227. doi:10.1016/j.cortex.2017.03.012.
4. Morihara K, Ota S, Kakinuma K, Kawakami N, Higashiyama Y, Kanno S, et al. Buccofacial apraxia in primary progressive aphasia. Cortex. 2023;158:61–70. doi:10.1016/j.cortex.2022.10.010.
5. López-Liria R, Fernández-Alonso M, Vega-Ramírez FA, Salido-Campos MÁ, Padilla-Góngora D. Treatment and rehabilitation of dysphagia following cerebrovascular disease. Rev Neurol. 2014;58(6):259–67. doi:10.33588/rn.5806.2013335.
6. Altman KW, Richards A, Goldberg L, Frucht S, McCabe DJ. Dysphagia in stroke, neurodegenerative disease, and advanced

dementia. Otolaryngol Clin North Am. 2013;46(6):1137–49. doi:10.1016/j.otc.2013.08.005.
7. Ney DM, Weiss JM, Kind AJH, Robbins J. Senescent swallowing: impact, strategies, and interventions. Nutr Clin Pract. 2009;24(3):395–413. doi:10.1177/0884533609332005.
8. Suárez-Escudero JC, Porto KSL, Patiño DP, Moreno MI, Londoño CLB. Disfagia orofaríngea neurogénica: concepto, fisiopatología, clínica y terapéutica. Arch de Neurocien. 2022;27(4):44–56. doi:10.31157/an.v27i4.347.
9. Rommel N, Hamdy S. Oropharyngeal dysphagia: manifestations and diagnosis. Nat Rev Gastroenterol Hepatol. 2016;13(1):49–59. doi:10.1038/nrgastro.2015.199.
10. Ickenstein GW, Höhlig C, Prosiegel M, Koch H, Dziewas R, Bodechtel U, et al. Prediction of outcome in neurogenic oropharyngeal dysphagia within 72 hours of acute stroke. J Stroke Cerebrovasc Dis. 2012;21(7):569–76. doi:10.1016/j.jstrokecerebrovasdis.2011.01.004.
11. Duncan S, Gaughey JM, Fallis R, McAuley DF, Walshe M, Blackwood B. Interventions for oropharyngeal dysphagia in acute and critical care: a protocol for a systematic review and meta-analysis. Syst Rev. 2019;8(1):283. doi:10.1186/s13643-019-1196-0.
12. Gallegos C, Brito-de la Fuente E, Clavé P, Costa A, Assegehegn G. Nutritional aspects of dysphagia management. Adv Food Nutr Res. 2017;81:271–318. doi:10.1016/bs.afnr.2016.11.008.
13. Ciucci M, Hoffmeister J, Wheeler-Hegland K. Management of dysphagia in acquired and progressive neurologic conditions. Semin Speech Lang. 2019;40(3):203–12. doi:10.1055/s-0039-1688981.
14. Carnaby-Mann G, Lenius K. The bedside examination in dysphagia. Phys Med Rehabil Clin N Am. 2008;19(4):747–68 viii: https://doi.org/10.1016/j.pmr.2008.05.008.
15. Cook IJ. Diagnostic evaluation of dysphagia. Nat Clin Pract Gastroenterol Hepatol. 2008;5(7):393–403. doi:10.1038/ncpgasthep1153.
16. Giraldo-Cadavid LF, Gutiérrez-Achury AM, Ruales-Suárez K, Rengifo-Varona ML, Barros C, Posada A, et al. Validation of the Spanish version of the eating assessment tool-10 (EAT-10spa) in Colombia. A blinded prospective cohort study. Dysphagia. 2016;31(3):398–406. doi:10.1007/s00455-016-9690-1.
17. Belafsky PC, Mouadeb DA, Rees CJ, Pryor JC, Postma GN, Allen J, et al. Validity and reliability of the eating assessment tool (EAT-10). Ann Otol Rhinol Laryngol. 2008;117(12):919–24. doi:10.1177/000348940811701210.
18. Ricci Maccarini A, Filippini A, Padovani D, Limarzi M, Loffredo M, Casolino D. Clinical non-instrumental evaluation of dysphagia. Acta Otorhinolaryngol Ital. 2007;27(6):299–305.
19. Suárez-Escudero JC, Rueda Vallejo ZV, Orozco AF. Disfagia y neurología: ¿una unión indefectible? Acta Neurol Colomb. 2018;34(1):92–100. doi:10.22379/24224022184.
20. Foundas AL, Henchey R, Gilmore RL, Fennell EB, Heilman KM. Apraxia during Wada testing. Neurology. 1995;45(7):1379–83. doi:10.1212/wnl.45.7.1379.
21. Leopold NA, Daniels SK. Supranuclear control of swallowing. Dysphagia. 2010;25(3):250–7. doi:10.1007/s00455-009-9249-5.
22. Miller AJ. The neurobiology of swallowing and dysphagia. Dev Disabil Res Rev. 2008;14(2):77–86. doi:10.1002/ddrr.12.
23. Warnecke T, Labeit B, Schroeder J, Reckels A, Ahring S, Lapa S, et al. Neurogenic dysphagia: systematic review and proposal of a classification system. Neurology. 2021;96(6):e876–89. doi:10.1212/WNL.0000000000011350.
24. Malandraki GA, Johnson S, Robbins J. Functional MRI of swallowing: from neurophysiology to neuroplasticity. Head Neck. 2011;33(Suppl 1(01)):S14–20. doi:10.1002/hed.21903.
25. Lobo PP, Pinto S, Rocha L, Reimão S, de Carvalho M. Orofacial apraxia in motor neuron disease. Case Rep Neurol. 2013;5(1):47–51. doi:10.1159/000349895.
26. Steinhagen V, Grossmann A, Benecke R, Walter U. Swallowing disturbance pattern relates to brain lesion location in acute stroke patients. Stroke. 2009;40(5):1903–6. doi:10.1161/STROKEAHA.108.535468.
27. Teismann IK, Dziewas R, Steinstraeter O, Pantev C. Time-dependent hemispheric shift of the cortical control of volitional swallowing. Hum Brain Mapp. 2009;30(1):92–100. doi:10.1002/hbm.20488.
28. Makuuchi M, Kaminaga T, Sugishita M. Brain activation during ideomotor praxis: imitation and movements executed by verbal command. J Neurol Neurosurg Psychiatry. 2005;76(1):25–33. doi:10.1136/jnnp.2003.029165.
29. Hamdy S, Rothwell JC, Brooks DJ, Bailey D, Aziz Q, Thompson DG. Identification of the cerebral loci processing human swallowing with H2(15)O PET activation. J Neurophysiol. 1999;81(4):1917–26. doi:10.1152/jn.1999.81.4.1917.
30. Kern MK, Jaradeh S, Arndorfer RC, Shaker R. Cerebral cortical representation of reflexive and volitional swallowing in humans. Am J Physiol Gastrointest Liver Physiol. 2001;280(3):G354–60. doi:10.1152/ajpgi.2001.280.3.G354.
31. Li S, Ma Z, Tu S, Zhou M, Chen S, Guo Z, et al. Altered resting-state functional and white matter tract connectivity in stroke patients with dysphagia. Neurorehabil Neural Repair. 2014;28(3):260–72. doi:10.1177/1545968313508227.
32. Dziewas R, Sörös P, Ishii R, Chau W, Henningsen H, Ringelstein EB, et al. Neuroimaging evidence for cortical involvement in the preparation and in the act of swallowing. Neuroimage. 2003;20(1):135–44. doi:10.1016/s1053-8119(03)00285-4.
33. Schimmel M, Ono T, Lam OLT, Müller F. Oro-facial impairment in stroke patients. J Oral Rehabil. 2017;44(4):313–26. doi:10.1111/joor.12486.
34. Bizzozero I, Costato D, Sala SD, Papagno C, Spinnler H, Venneri A. Upper and lower face apraxia: role of the right hemisphere. Brain. 2000;123(Pt 11):2213–30. doi:10.1093/brain/123.11.2213.
35. Cera ML, Ortiz KZ, Bertolucci PHF, Minett TSC. Speech and orofacial apraxias in Alzheimer's disease. Int Psychogeriatr. 2013;25(10):1679–85. doi:10.1017/S1041610213000781.
36. Ozsancak C, Auzou P, Dujardin K, Quinn N, Destée A. Orofacial apraxia in corticobasal degeneration, progressive supranuclear palsy, multiple system atrophy and Parkinson's disease. J Neurol. 2004;251(11):1317–23. doi:10.1007/s00415-004-0530-0.
37. Myung JH, Pyun SB. Effect of oral apraxia on dysphagia in patients with subacute stroke. Dysphagia. 2023;38(1):227–35. doi:10.1007/s00455-022-10458-w.