

DYSPHAGIA AND SIALORRHEA: the relationship to Parkinson's disease

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ABSTRACT - *Context* - Dysphagia and sialorrhea in patients with Parkinson's disease are both automatically accepted as dependent on this neurological disease. *Objective* - The aim were to establish if these two complaints are a consequence or associated manifestations of Parkinson's disease. *Method* - Two Parkinson's diseases groups from the same outpatients' population were studied. Patients in the first group, with dysphagia, were studied by videofluoroscopy. The second, with sialorrhea, were studied by the scintigraphic method, *Results* - Videofluoroscopic examination of the oral, pharyngeal and esophageal phases of swallowing showed that 94% of Parkinson's diseases patients present, structural causes, not related to Parkinson's diseases, able to produce or intensify the observed dysphagia. The scintigraphic examination of Parkinson's diseases patients with sialorrhea showed that there is no increase of serous saliva production. Nevertheless, showed a significantly higher velocity of saliva excretion in the Parkinson's diseases patients. *Conclusions* - Dysphagia can be due to the muscular rigidity often present in the Parkinson's diseases patient, or more usually by non Parkinson's disease associated causes. In Parkinson's diseases patients, sialorrhea is produced by saliva retention. Nevertheless, sialorrhea can produce discomfort in swallowing, although without a formal complaint of dysphagia. In this case, subclinical dysphagia must be considered. Sialorrhea is indicative of dysphagia or at least of subclinical dysphagia. As final conclusion, Parkinson's diseases can be an isolated cause of dysphagia and/or sialorrhea, but frequently, a factor unrelated to Parkinson's diseases is the main cause of or at least aggravates the dysphagia.

HEADINGS - Deglutition disorders. Sialorrhea. Fluoroscopy. Radionuclide imaging. Parkinson disease.

INTRODUCTION

Dysphagia, i.e., difficulty in swallowing, and sialorrhea, understood as an excess flow of saliva into the mouth, with or without escape but in an unpleasant volume, are both dependent on a primary disease affecting the upper digestive tract. Among the primary diseases, neurological diseases are recognized as the most frequent causes of these problems. Considered as common digestive manifestations in Parkinson's disease (PD)^(1, 2, 6, 15, 16, 19, 21, 27), dysphagia and/or sialorrhea are both naturally assumed to be dependent on PD when they are present in association with this neurological disease.

Parkinson's disease, the most frequent cause of parkinsonism, is an idiopathic, asymmetrical and slowly progressing disease, resulting mainly from the progressive loss of neurons in the ventrolateral portion of the pars compacta of the substantia nigra in the midbrain^(1, 2, 6). This neurological disease is characterized by the presence of bradykinesia and at least one other of the three cardinal signs: rigidity, resting tremor, and loss of postural reflexes, usually

occurring in persons 50 years old or older^(8, 9). Recently, the importance of the presence of non-central motor symptoms was recognized, such as those produced by the affected autonomic nervous system^(11, 13) especially in the digestive system where constipation, dysphagia and sialorrhea may be observed^(14, 15, 18).

Pathological difficulty in swallowing, when present in a Parkinson's patient, is usually considered to be caused by the PD, without further investigation^(9, 13, 18). Nevertheless, as mentioned in the literature, several other causes of dysphagia may occur in association with PD⁽²⁷⁾.

In Parkinson's disease, the prevalence of dysphagia is estimated to be from 32% to 70%⁽²⁸⁾. Nevertheless, the risk posed by dysphagia is neglected or underestimated until the patient suffers the first episode of pneumonia^(17, 20), despite the known high risk of aspiration and pneumonia associated with oropharyngeal dysphagia in PD patients^(10, 26).

In association with dysphagia, because of the difficulty of clearing the oral saliva, sialorrhea or at least sialostasis, can be observed. James Parkinson, in his classic monograph "An essay on the shaking palsy",

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referred to excess salivation as an unpleasant occurrence caused by difficulty in appropriate transfer of saliva to the oropharynx, resulting in excessive drooling⁽²³⁾.

Described as an event of unknown pathophysiology⁽²⁵⁾, sialorrhea occurs in from 45% to 80% of PD patients. Its presence is associated with the severity of the disease⁽⁶⁾, although this association has been contested⁽²¹⁾. In theory, sialorrhea can result from excessive production or retention of saliva by dysphagia, or both^(2, 16, 21, 27). Through a comparative scintigraphic study of PD patients with sialorrhea and healthy volunteers, the PD sialorrhea was identified as produced by retention of oral saliva⁽²¹⁾.

Sialorrhea and dysphagia, were symptoms present in two of three patients with moderate PD, and the presence of each symptom was significantly correlated one with the other⁽²⁴⁾. Although sialorrhea and dysphagia has been associated with PD since its original description, advances in understanding of the pathophysiology of this association have been slight⁽²⁴⁾.

Our aims were to correlated the observed results from PD patients with dysphagia as the main complaint studied by videofluoroscopy with the results from PD patients with sialorrhea as the main complain studied by scintigraphic method.

METHODS

This study was conducted in full agreement with the ethical guidelines proposed by the World Medical Association (WMA, Declaration of Helsinki, 1995; amended by the 52nd WMA General Assembly, Edinburgh, UK, October, 2000). Our protocol was approved by the Ethics Committee for Scientific Investigation of the Federal University of Rio de Janeiro (UFRJ), and all volunteers gave their informed consent to participate.

PD patients of both sexes, with a disease evolution of 4-19 years and in the 3rd to 5th stages of the Hoehn & Yahr scale (H&Y)⁽⁸⁾ were analyzed. The patients were selected in distinct time based in its main complaint. PD patients complained of dysphagia was studied by videofluoroscopy, and PD patients complained of sialorrhea was studied by scintigraphy. All the patients were from the same outpatients population.

Videofluoroscopic study

This procedure was accomplished on 17 dysphagic patients, between 34 and 86 years of age, clinically evaluated by the UPDRS. The videofluoroscopic study utilized Siemens equipment with a TV Videomed 2 system, image intensifier RBV 23/13, vidicom tube with 525 lines, frequency 60 Hz, interlace 2:1. The images from the dynamic events of the oral, pharyngeal and esophageal phases during swallowing of contrast media (liquid, solid, and paste) were recorded in VHS medium and analyzed using a video monitor. The liquid medium was a commercial 100% barium solution. The solid medium was moistened bread crumbs mixed with barium powder and divided into balls 0.5 to 1 cm in diameter, to swallow as pills. The paste medium was obtained from a piece of the same solid medium, about 1.5 cm in diameter, to be chewed into a paste for swallowing.

Scintigraphic study

This procedure was accomplished in two subgroups. The first subgroup was formed by 14 PD patients with sialorrhea, between 42 and 76 years of age, and clinically evaluated by UPDRS - part II and by scintigraphy. The second subgroup was composed by 8 non-PD control volunteers between 39 and 77 years of age. The dynamic studies of the salivary glands were accomplished in a Diacam-Siemens wide-field gamma camera. All individuals were placed in the frontal position for acquisition of the dynamic image, which was started simultaneously with a venous injection, in bolus, of 37 MBq of Tc-99m (pertechnetate). The images were recorded in a matrix of 128 x 128 (total of 40) sequential frames. A small amount (0.5 mL) of lemon juice was administered through a probe into the oral cavity, 20 min after the examination started. Graphs of the uptake dynamics and excretion of the parotid glands were generated, reproducing the gland activity over a certain time period. The parotid-gland activity generated graphs with three components: CP1, the first ascending curve, which corresponds to the uptake phase of the gland; EP, the descending curve, which corresponds to the excretion phase; and CP2, the second ascending curve, which corresponds to the uptake activity, immediately after the excretion phase following acid stimulation (lemon juice). After the examination started, there was a 5 min period before the first curve, CP1, began to be generated. CP1 was then recorded for 15 min. Then, at 20 min, EP recording followed, with the administration of the acid stimulus (lemon juice). Finally, after excretion subsided, CP2 was generated until the end of the examination after 40 min. Statistical analysis was done with the nonparametric t test (Mann-Whitney at $P = 0.05$) using GraphPad Prism 4 for Windows (GraphPad Prism version 4.00, GraphPad software, 2003, La Jolla, California, USA).

RESULTS

Videofluoroscopic study – PD patients with dysphagia as the main complaint

The videofluoroscopic evaluation of the swallowing phases of PD patients with dysphagia allowed visualization of structural and functional disarrangements that can produce and/or increase difficulty in swallowing.

In the oral phase, structural disarrangements were identified that interfere with chewing, organization of the bolus (especially fluid) on the tongue, and the intensity of oral ejection over the bolus to be transferred from the oral cavity to the pharynx.

During the examination, five patients showed muscle rigidity, which was considered to produce a negative effect on the chewing process, the bolus organization, and the bolus ejection to the pharynx. Seven patients had great difficulty in swallowing the contrast pills (solid medium) without a sip of water (videofluoroscopic indicative of xerostomy); i.e., they had difficulty in producing sufficient saliva to aid in swallowing. All of them had referred for xerostomy in the previous clinical evaluation. Eight patients had lost most

Table 1. Results from videofluoroscopy evaluation of oral, pharyngeal and esophageal phases in 17 patients with PD and dysphagia.

ORAL PHASE			PHARYNGEAL PHASE	ESOPHAGEAL PHASE
CHEWING	BOLUS ORGANIZATION	BOLUS EJECTION		
most or all teeth missing (8)	moderate deficiency (14)	by gravity action (7)	aspiration (2)	slow transit (4)
xerostomy (7)	Severe deficiency (2)	twice (3)	open deficiency in PET UES (2)	abnormal enlargement of aortic arch (8)
rigidity (5)	normal (1)	slow (4)	PET compression from osteophyte (2)	motor disturbance with nonpropulsive contraction (9)
normal (1)		without synchrony (2)	pharyngeal diverticula (4)	
		dental prosthesis poorly adjusted (1)	functional opening of UES (13)	

UES = upper esophageal sphincter; PET = pharyngoesophageal transition

or all of their teeth, with extensive absorption of mandible and maxillary bone, which impeded chewing, organization, and ejection, and also affected the pharyngeal phase. Four of these eight patients also showed muscle rigidity; one of them had a poorly adjusted upper dental prosthesis.

Of 17 PD patients with dysphagia, only 1 did not show structural compromises that could cause difficulty in chewing and oral bolus organization (94,11%). Nevertheless, in this patient, oral ejection was slow due to muscle rigidity. Fourteen patients showed some difficulty in oral bolus organization, which affected oral ejection. This interference was partially compensated in seven patients by the action of gravity, in three by swallowing twice, and in four others by a weaker ejection that was still able to conduct the bolus through the pharynx without disturbing the airway respiratory function. Thirteen patients (76,5%) had a functional upper esophageal sphincter (UES); they remained open during the passage of the entire fluid bolus. Two patients showed an absence of synchrony between the oral and pharyngeal phases; these two patients also showed apparent deficiency in the UES and aspiration. The patient who had a poorly adjusted dental prosthesis also showed apparent deficiency in the UES, however without airway compromise.

In the pharyngeal-phase analyses, two patients were found with a cervical osteophyte that increased the resistance to pharyngeal flux. In the pharyngeal study we also observed four patients with lateral laryngopharyngeal diverticula, also called a lateral pharyngeal pouch, formed in the less anatomically resistant area where the inner branch of the superior laryngeal nerve passes through the pharyngeal wall. These pouches do not cause difficulty in pharyngeal flux, but are a result of the same high resistance that causes this difficulty.

In regard to the esophagus, nine patients showed motor disturbances that produced diffuse esophageal spasms, varying in intensity; these did not depend on the oral and pharyngeal findings. Four of these patients also showed slow transit. The anatomical relationship between the aortic arch and the esophagus showed a pathological change in eight patients, without significantly affecting flux. Nevertheless, some resistance was certainly imposed on the esophageal flux, resulting in oral and pharyngeal overcharge (Table 1 and Figs. 1 and 2).

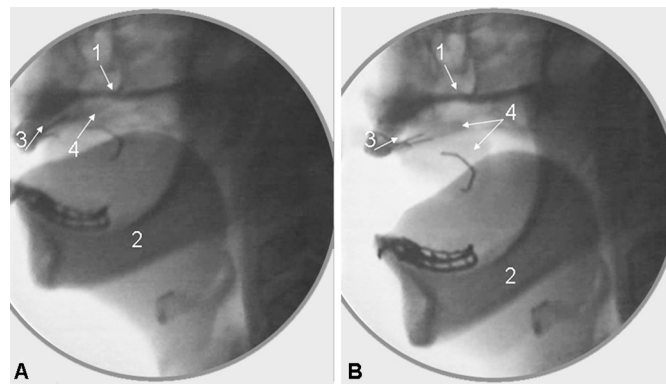


FIGURE 1. Images obtained from dysphagic PD patients with rigidity, associated with bone absorption and lack of teeth in the maxilla (1) and mandible (2). In (A) the prosthesis (3 and 4) is apparently adjusted on the hard palate (1), in B we see a space between the hard palate (1) and the prosthesis (4). The observed rigidity certainly could have caused difficulty in swallowing, but the condition of the mandible and maxilla bone and the poorly adjusted dental prosthesis certainly exacerbated the difficulty in swallowing.

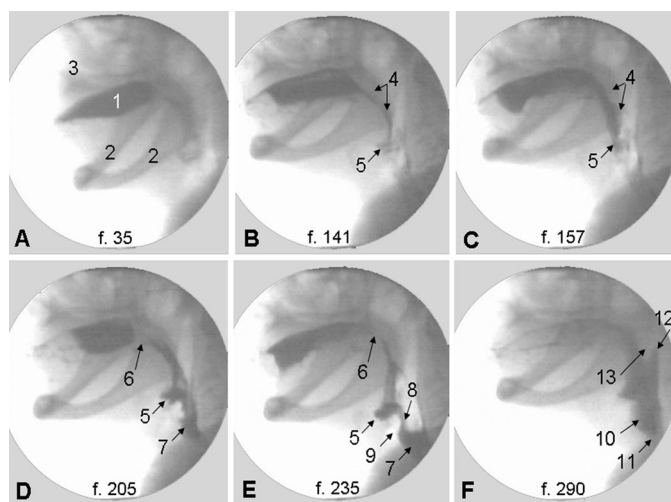


FIGURE 2. Sequence of 8.5 seconds from frames (f.) 35 to 290 obtained from dysphagic PD patient with resting tremor and without rigidity. In (A) 1- the barium solution enters the mouth, 2- mandible with severe bone absorption, also seen in 3- the maxilla. In (B) the contrast medium begins to pass over 4- the tongue from the mouth to the pharynx, until 5- vaeclula. In (C) 4- a larger volume passes to 5- vaeclula. Subsequently, (D) 5-vaeclula and 7- pyriform recess with greater volume of contrast medium and also 6- interruption of the oral escape. In (E) 6- interruption of the oral escape is still present, and we can see 5- vaeclula and 7- pyriform recess collecting the contrast medium, drawing 8- the ariepiglottic fold without 9- vestibule penetration. In (F) All the oral volume was transferred to (10) pharynx which remains distended, starting the opening of the (11) pharyngoesophageal transition (PET). We could also see 12- contrast medium insinuation behind 13- soft palate. From B to F, 5 seconds passed and the pharyngeal phase was not concluded. This oral and pharyngeal dysfunction was dependent on the oral structural disarrangement and not on the PD.

Scintigraphic study – PD patients with sialorrhea as the main complain

Fourteen PD patients presenting sialorrhea, with symptoms of the disease for 1 to 16 years, were selected. This group represented 10.45% of the same population from which dysphagic patients were obtained. Complaints of sialorrhea were mentioned spontaneously by three patients, and obtained by the questioner in 11 others. The severity of Parkinson's disease, based on the H&Y⁽²⁴⁾ modified scale (UPDRS – part V) revealed that two patients were in the H&Y- I stage, six in H&Y- II, and six in H&Y-III, with no correlation with the severity of sialorrhea. According to the UPDRS sialorrhea quantification standard, nine patients were classified as grade 1, four as grade 2, and one as grade 4. Complaints of dysphagia were not mentioned spontaneously by any of the patients.

Comparison between scintigraphic analyses from both subgroups, normal volunteers and PD patients, indicated no differences in the first uptake (CP1) of Tc-99m [pertechnetate] by the parotid gland, nor in the second one (CP2). Nevertheless, the excretion velocity of the parotid gland in 71.4% of the PD patients was more rapid than that observed in normal volunteers.

The nonparametric *t test* (Mann-Whitney at *P* = 0.05) using the software GraphPad Prism 4 for Windows indicated a significantly higher excretion velocity in the PD group than in the control group (*P*<0.05). The first uptake (CP1) and the second uptake (CP2) showed no significant difference (*P*>0.05).

The CP1, CP2 and EP curves for the submandibular gland, similarly to the parotid gland, sometimes allowed adequate observation; nevertheless, its anatomical location usually mixes its radioactive tracer with that present in the oral cavity, limiting scintigraphic analysis of its function. For this reason we did not include its behavior in the results, but we believe that at least its serous component behaved similarly to the parotid glands (Table 2, Figs. 3 to 5).

TABLE 2. Parotid gland pertechnetate first uptake, second uptake and excretion velocity in the control subgroup and in patients with Parkinson's disease.

Control subgroup	CP1%	EP %	CP2%	PD Subgroup	CP1 %	EP %	CP2 %
1	2.70	13.74	2.04	1	3.08	12.45	2.36
2	2.98	14.58	2.00	2	2.10	21.00	1.76
3	2.30	13.16	1.17	3	2.56	10.63	1.14
4	3.48	8.96	1.70	4	3.40	25.70	1.90
5	2.84	12.34	3.22	5	3.32	16.60	1.97
6	2.89	10.58	1.33	6	4.68	12.00	2.68
7	1.92	9.62	2.50	7	2.81	20.60	2.18
8	2.54	9.03	1.22	8	2.00	14.80	1.06
				9	2.25	15.20	1.58
				10	3.22	15.20	0.98
				11	3.16	32.70	2.24
				12	1.88	11.96	1.02
				13	3.12	14.90	2.62
				14	3.86	22.20	2.14
\bar{X}	2.70	11.50	1.9	\bar{X}	2.96	17.57	1.83
± SD	± 0.47	± 2.33	± 0.71	± SD	± 0.77	± 6.21	± 0.59

CP1 = first uptake of the parotid gland; CP2 = second uptake of the parotid gland; EP = excretion phase of the parotid gland; \bar{X} = mean ± SD = Standard deviation; % = "k" values from 0 to 1 expressed as percentages

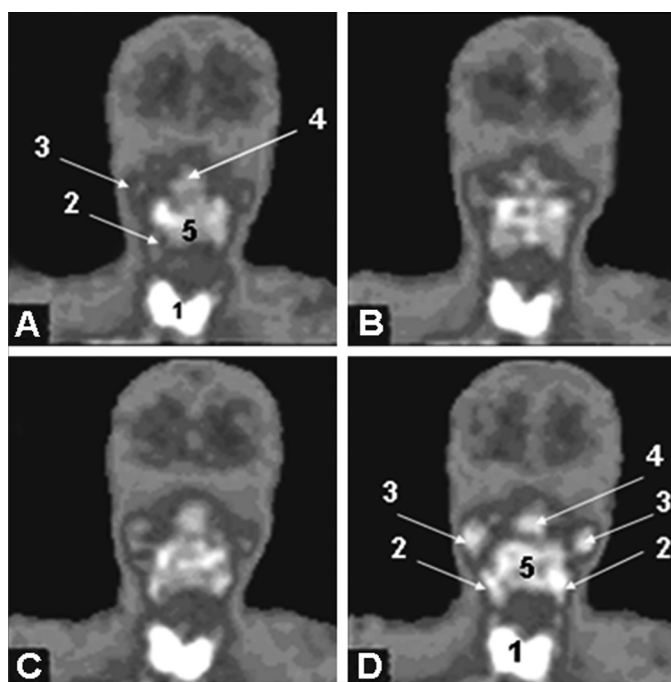


FIGURE 3. Scintigraphic study of the salivary glands in patients with PD. Following venous injection of pertechnetate ($^{99m}\text{TcO}_4$), the images show 1- Uptake of the thyroid, 2- submandibular and 3 - parotid glands. Also shows 4 - nasal cavity and 5 - mouth. The increase of radioactivity uptake, frame by frame, in serous salivary glands is observed from A to D.

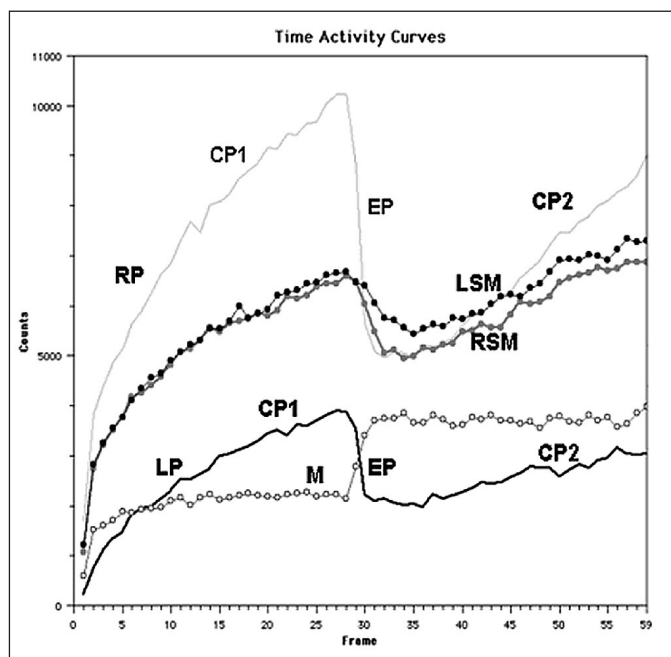


FIGURE 4. Time activity curve of the scintigraphic examination of the salivary glands (parotid and submandibular) showing the first uptake phase (CP1), the excretion phase (EP) and the second uptake phase (CP2), where RP is the right parotid gland, RSM the right submandibular gland, LSM the left submandibular gland, LP the left parotid gland and M the mouth.

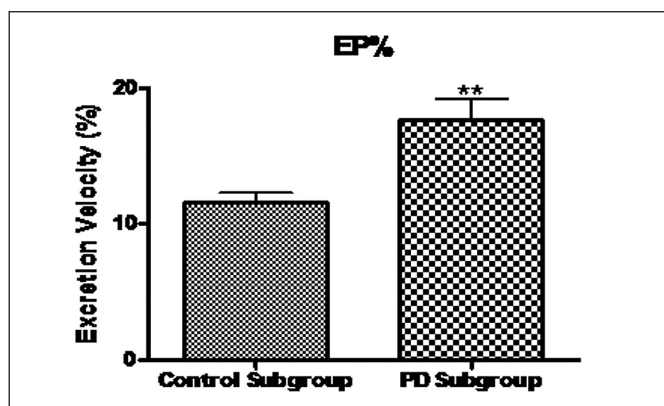


FIGURE 5. Obtained with Mann-Whitney test from Table 2, showing a significantly higher excretion velocity in the PD subgroup than in the control subgroup ($P < 0.05$).

DISCUSSION

Dysphagia and or sialorrhea are both considered common digestive manifestations in PD^(1, 2, 6, 15, 16, 19, 21, 27), but sialorrhea has seldom been related to dysphagia in PD patients^(14, 21). These two digestive manifestations, in addition to causing discomfort and social embarrassment, can cause aspiration and pneumonia. Aspiration related to swallowing is a major cause of morbidity and mortality in PD⁽¹⁵⁾. Therefore, it is important to establish the role of PD in the production of these two common digestive manifestations.

Dysphagia, similarly to PD, is more frequently found in elderly patients. Dysphagia can be caused by structural and/or neurological subjacent diseases; nevertheless, a neurological cause is the most frequent etiology. For a patient with any neurological disease associated with dysphagia, difficulty in swallowing is automatically attributed to the associated neurological disease.

PD is a neurological disease, usually occurring in persons more than 50 years of age, who usually present a combination of sialorrhea and dysphagia. Despite the slight advance in knowledge of their pathophysiology, both these digestive manifestations, when present in PD patients, are routinely categorized as dependent on this neurological entity, usually without further investigation^(8, 23).

The selection of the two PD groups, with dysphagia and with sialorrhea, were also done without further investigation. In the dysphagic group all of them complained and showed a clear swallow difficult. Sialorrhea was not thoroughly investigated. In the same way in the PD patients, where sialorrhea was the objective dysphagia was not thoroughly investigated. In this group, where sialorrhea was the objective, only 3 patients complaints sialorrhea spontaneously, the 11 others patients were obtained by questioner (UPDRS - part II). The relationship of dysphagia with sialorrhea and of these two digestive manifestations with PD and other, non-neurological etiologies was not considered initially.

To videofluoroscopic study, our attention was directed toward dysphagia associated with PD. Videofluoroscopy of

PD patients showed a clear neurological compromise such as rigidity in association with several other disarrangements, also clearly independent from the neurological compromise, and able to reveal or to produce dysphagia without any neurological contribution. Therefore, at least in the PD dysphagic patients, we must consider the possible existence of non-neurological causes in association with the neurological one.

The difficulty in swallowing explains the complaint of sialorrhea. Nevertheless, in our dysphagic group we found seven PD patients who complained of xerostomy, which was confirmed during the videofluoroscopy by the especially marked difficulty in the solid swallowing which is more dependent on saliva support. This xerostomy is probably more dependent on the patients' age than on any other factor. No patients were using medications able to produce xerostomy.

Dysphagia related to missing teeth with extensive mandible and maxillary bone absorption obviously can interfere with chewing, organization, and also oral ejection that may, through inadequate oral transference, hamper the response of the pharyngeal phase. Missing teeth have harmful consequences because of the loss of muscle mass, tonus and force, even with a well-adjusted dental prosthesis. Of course, oral dysfunction is strongly aggravated when the dental prosthesis is poorly adjusted, as we found in one PD patient.

The opening of the pharyngoesophageal transition (PET), also called the upper esophageal sphincter, is an important condition for passage from the pharynx to the esophagus, and is produced primarily by the upward and forward motion of the hyolaryngeal complex by the action of the suprahyoid muscles⁽³⁾. In the great majority of cases of dysphagia associated with neurological disease, we find some apparent limitation of PET, especially in duration, through compromise of the fifth and seventh cranial nerves that are mainly responsible for controlling the suprahyoid muscle. In contrast to the others neurological diseases where PET usually are compromised, 76.5% of the our studied PD patients have a functional PET. We could identify a compromised PET in only four PD patients. Two of them had a loss of synchrony between oral ejection and the PET function, leading us to consider this dysfunction as primarily dependent on oral-ejection deficiency. The other two showed the presence of a cervical osteophyte at the level of the PET, limiting its free function. These two osteophytes alone were probably not able to cause dysphagia, but if they are combined with other deficiencies, this is an additional element to be consider.

The lateral laryngopharyngeal diverticula found here are formed in a less anatomically resistant area in the pharyngeal wall, and they do not cause dysphagia. These diverticula are considered to result from pharyngeal overpressure, which is often produced by dysphagia^(4, 5, 7). Their presence in the PD patients indicates a long-term pharyngeal pressure overcharge. These kind of diverticula usually do not present symptoms^(4, 5, 7).

The videofluoroscopy study used to analyze the esophageal movements revealed nine patients with motor disturbances producing a diffuse esophageal spasm that varied in

intensity; four of these patients also showed a slow transit of the esophagus. This esophagus motor dysfunction is probably caused by a presbyesophagus and is not dependent on PD, despite the acknowledged compromise of the autonomic nervous system in this disease^(11, 13). This compromise, independently of its etiology, can affect the oropharyngeal flux and must be considered in dysphagic patients. In eight patients, the relationship of the aortic arch and esophagus was more intense than normal, but did not materially affect the flux. This aortic distension is probably dependent on arterial pressure, which is usually high in older persons. As a complementary consideration, the aortic-esophageal relationship observed here certainly did not have an important negative effect on the oropharyngeal flux.

To scintigraphic study, our attention was directed toward sialorrhea. By means of scintigraphic analysis, we studied 22 volunteers after a venous injection of a bolus of pertechnetate, comparing 14 PD patients with sialorrhea against 8 healthy control subjects in the same age range. In the frontal images, the acquired radioactive emission showed the salivary serous glands (parotid and submandibular), thyroid gland, nasal cavity (in a rich venous sub-mucosa plexus) and oral cavity. The dynamic study conducted with the use of a Diacam-Siemens wide-field gamma camera was limited to the parotid gland.

The gamma camera program generated the CPI curve as the first curve for parotid-gland activity, which corresponds to the uptake phase of the gland. In the PD patients and in the control group, the CPI curves were similar, indicating that there was no increase of saliva production in the PD patients. Nevertheless, after acid stimulation, a descending curve was produced, corresponding to the excretion phase, and showing in PD patients an increase in the excretion velocity of the parotid glands. A second ascending curve generated immediately after the excretion phase, which corresponds to the uptake activity, behaved similarly to the first curve.

Because saliva production did not increase in PD patients, it is valid, as considered elsewhere⁽²¹⁾, to assume that the saliva accumulation (sialorrhea) in PD results from some degree of compromise of swallowing.

The submandibular activity seemed to be similar to that observed in the parotid glands. Nevertheless, its anatomical location usually mixes the radioactive tracer with that present in the oral cavity, limiting the scintigraphic analysis of its function. Therefore, an increase in the velocity of saliva excretion to the mouth will produce an overcharge in the saliva swallowing efficiency, defining a sialorrhea by retention, which can vary in severity according to the associated oral and pharyngeal compromise.

In agreement with our videofluoroscopic results, were in association with PD found 94,11% of structural causes of dysphagia; there are a study, using the videofluoroscopic method in PD patients with dysphagia and sialorrhea found changes in the oral stage of swallowing in all patients studied; and in the pharyngeal phase in 94% of the patients. The authors concluded that the patients with more severe dysphagia had more copious drooling⁽²²⁾.

As already mentioned, sialorrhea was defined as a usual excess of saliva in the mouth, with or without escape, but in unpleasant volume. Dysphagia was defined as difficulty in swallowing the patient's usual meals, at such a level that this difficulty must be recorded as an additional complaint. Nevertheless a discreet swallow difficult can be incorporated by patients as normal event only observed after hard and necessary interrogatory. It is important to consider this swallow discreet difficult at least as a subclinical dysphagia. On the other hand, there is a functional limit for chewing and organizing the bolus in the mouth without additional difficulty, and this limit is anatomically and physiologically determined. To attempt to chew, organize and swallow a bolus that exceeds this limit will produce some difficulty, which must not be considered as dysphagia.

PD is a pathology of elderly people that can, especially because of muscle rigidity, produce dysphagia and sialorrhea by saliva retention. Because of their age, elderly people may present several associated disturbances able to produce or aggravate difficulty in swallowing. It is possible to find PD patients without dysphagia and/or sialorrhea. It is also possible for PD patients to present dysphagia without sia-

lorrhea. Nevertheless, if a PD patient presents sialorrhea, he or she will also present some level of dysphagia, at least at the subclinical level. A subclinical level of dysphagia is due to a reduction of the functional limit observed in old persons. Young persons have a large functional reserve and are able to exceed this limit without causing a problem. Older persons have their functional limit closely adjusted to their diet, and any overcharge will therefore pose a problem. This consideration might explain the occurrence of complaints of sialorrhea without a formal complaint of dysphagia; the patient believes that his or her usual small or partial difficulty in swallowing is normal for his/her age and condition.

CONCLUSIONS

Parkinson's disease can be an isolated cause of dysphagia and/or sialorrhea. Nevertheless, we found that 94,11% of our dysphagic patients had a non-neurological cause of dysphagia associated with PD.

The cause of sialorrhea in PD patients is oral retention produced by associated dysphagia. Sialorrhea is indicative of dysphagia or at least of subclinical dysphagia.

Nicaretta DH, Rosso AL, Mattos JP, Maliska C, Costa MMB. Disfagia e sialorreia na doença de Parkinson. *Arq Gastroenterol.* 2013;50(1):42-9.

RESUMO - Contexto - Disfagia e sialorreia em pacientes com doença de Parkinson são automaticamente entendidos como decorrentes do comprometimento neurológico produzido pela doença de Parkinson. **Objetivo** - Estabelecer se estas duas queixas são consequências ou manifestações associadas à doença de Parkinson. **Método** - Dois grupos de pacientes com doença de Parkinson provenientes da mesma população ambulatorial foram estudados. O primeiro grupo com queixa de disfagia foi estudado pelo método videofluoroscópico. Um segundo grupo com sialorreia foi estudado pelo método cintigráfico. **Resultados** - O exame videofluoroscópico das fases oral, faríngea e esofágica da deglutição mostrou que 94% das disfagias nos pacientes com doença de Parkinson eram devidas a causas estruturais não relacionadas com a doença de Parkinson e capazes de produzir ou intensificar a disfagia observada. Os exames cintigráficos dos pacientes com doença de Parkinson e sialorreia mostraram que não ocorre aumento da produção de saliva. No entanto mostrou significativo aumento na velocidade de excreção da saliva nesses pacientes. **Conclusões** - A disfagia pode ser devido à rigidez muscular frequentemente presente nos pacientes com doença de Parkinson ou mais frequentemente por causas associadas que independem desta. Nos pacientes com doença de Parkinson a sialorreia é produzida pela retenção oral da saliva. Contudo é possível observar queixa de sialorreia sem formal queixa associada de disfagia. Nesses casos, disfagia sub-clínica deve ser considerada. Sialorreia é um indicativo de disfagia ou pelo menos de disfagia sub-clínica. Como conclusão final, a doença de Parkinson pode ser causa isolada de disfagia e ou sialorreia, mas frequentemente um fator não relacionado com a doença de Parkinson pode cursar como a principal causa ou pelo menos como causa agravante da disfagia.

DESCRIPTORIOS - Transtorno de deglutição. Sialorreia. Fluoroscopia. Cintilografia. Doença de Parkinson.

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