

Changes in esophageal motility and transit after a stroke

Alterações da motilidade e trânsito pelo esôfago no acidente vascular cerebral

Cambios en la motricidad esofágica y el transito después un accidente cerebrovascular

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Resumo

Introdução. O Acidente Vascular Cerebral (AVC) é uma causa frequente de disfagia neurogênica, com mais importante comprometimento funcional e clínico das fases oral e faríngea da deglutição. No entanto, a doença também pode afetar a função esofágica. **Objetivo.** Revisão das alterações esofágicas descritas em pacientes que sofreram acidente vascular cerebral. **Método.** Foram revisados na PubMed, de 1990 a 2022, artigos que avaliaram a motilidade esofágica e o trânsito esofágico em pacientes que sofreram acidente vascular cerebral. **Resultados.** Observou-se no esôfago aumento de contrações não peristálticas, tempo de trânsito esofágico prolongado, diminuição da proporção de peristaltismo em pacientes com disfagia, em comparação com pacientes sem disfagia, aumento do tempo de depuração esofágica, retenção esofágica e fluxo retrógrado. Estas alterações diminuem em frequência com o tempo decorrido após o acidente. **Conclusão.** O acidente vascular cerebral pode afetar o trânsito e a contração esofágica, com maior tempo de passagem do bolo deglutido pelo esôfago e aumento do número de contrações não peristálticas. Estas alterações diminuem de frequência com o decorrer do tempo após o acidente.

Unitermos. Acidente Vascular Cerebral; Esôfago; Doenças do esôfago; Deglutição; Transtornos da deglutição

Abstract

Introduction. Stroke is a frequent cause of neurogenic dysphagia, more importantly causing functional and clinical impairments in the oral and pharyngeal phases of swallowing. However, the disease may affect esophageal function as well. **Objective.** Review of esophageal changes described in patients who had a stroke. **Method.** The study reviewed articles from 1990 to 2022 in PubMed that assessed esophageal motility and esophageal transit in patients who had a stroke. **Results.** The review found increased non-peristaltic esophageal contractions, longer esophageal transit, decreased peristalsis proportion in patients with dysphagia than in those without it, longer esophageal clearance, esophageal retention, and retrograde flow. These changes became less frequent with time after the stroke. **Conclusion.** Stroke may affect esophageal transit and contraction, taking more non-peristaltic contractions and longer for the bolus to pass through the esophagus. These changes became less frequent with time after the stroke.

Keywords. Stroke; Esophagus; Esophageal diseases; Deglutition; Deglutition disorders

Resumen

Introducción. El Accidente Vascular Cerebral (AVC) es una causa frecuente de disfagia neurogénica, con más importante comprometimiento funcional y clínico de las fases oral y faríngea de la deglución. Sin embargo, la enfermedad también puede afectar la función esofágica. **Objetivo.** Revisión de las alteraciones esofágicas descritas en pacientes que sufren accidente vascular cerebral. **Método.** Fueron revisadas en la PubMed, de 1990 a 2022, artículos que avalúan la motilidad esofágica y el transito esofágico en pacientes que sufren

accidente vascular cerebral. **Resultados.** Se observó en el esófago aumento de contracciones no peristálticas, tiempo de tránsito esofágico prolongado, disminución de la proporción de peristaltismo en pacientes con disfagia, en comparación con pacientes sin disfagia, aumento del tiempo de depuración esofágica, retención esofágica y flujo retrogrado. Estas alteraciones disminuyen en frecuencia con el tiempo recorrido después del accidente. **Conclusión.** El accidente vascular cerebral puede afectar el tránsito y la contracción esofágica, con mayor tiempo de pasaje del bolo deglutido por el esófago y aumento del número de contracciones no peristálticas. Estas alteraciones disminuyen con frecuencia con el transcurso del tiempo después del accidente.

Palabras clave. Accidente Cerebrovascular; Esófago; Enfermedades del Esófago; Deglución; Trastornos de Deglución

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INTRODUCTION

Gastrointestinal symptoms in neurological diseases include sialorrhea, dysphagia, gastroparesis, gastrointestinal pseudo-obstruction, constipation, diarrhea, and fecal incontinence, caused by diseases that affect from the cerebral hemispheres to the peripheral autonomic nerves¹.

Stroke is a frequent cause of neurogenic dysphagia, causing important clinical impairments of the oral and pharyngeal phases of swallowing^{2,3}. About 64% to 78% of stroke patients have dysphagia⁴, with frequency in Brazil ranging from 59% to 76%³, depending on the dysphagia assessment method.

The disease may also affect esophageal function, contributing to the occurrence of dysphagia. In humans, esophageal motility in the proximal esophagus is neurologically controlled by the activation of motor units in the swallowing center, which is in direct contact with striated muscle through vagal fibers⁴. In the middle and distal

esophageal smooth muscles, the esophageal peristalsis is controlled by the enteric nervous system⁴.

The initiation of peristalsis in the proximal esophagus has the control of the central nervous system⁵. The oral, pharyngeal, and esophageal phases of swallowing are independent of each other and dependent on sensory feedback⁶. The neurologic lesion caused by ischemic or hemorrhagic cerebrovascular disease may impair the control of all phases of swallowing – more intensely in the oral and pharyngeal phases due to the direct connection of the central control with the mouth and pharynx, although the esophageal phase of swallowing may also be affected.

The objective of the review was to describe the results of changes in esophageal motility and/or transit after a stroke. The hypothesis was that the stroke may also affect the esophageal phase of swallowing, thus contributing to dysphagia.

METHOD

This review included papers that evaluated esophageal motility and/or esophageal transit in patients who had a stroke, cited in PubMed from 1990 to 2022. The search terms were “Esophageal body – Stroke” and “Esophageal body – Cerebrovascular Disease”.

Hence, it included papers with evaluation results of esophageal transit and/or motility in patients with ischemic or hemorrhagic stroke.

RESULTS AND DISCUSSION

There were seven articles⁷⁻¹³ about esophageal function after a stroke, published from 1999 to 2022. Table 1 has a summary of the results.

Table 1. Alterations described in the esophageal motility and/or transit after a stroke.

Authors	Esophageal alterations
Miles <i>et al</i> ⁷	Longer esophageal transit
Silva <i>et al</i> ⁸	Shorter distal esophageal transit in upright position Longer esophageal clearance
Alves <i>et al</i> ⁹	Increase in the frequency of esophageal residues Longer esophageal clearance
Micklefield <i>et al</i> ¹⁰	Decrease in the proportion of peristaltic contraction
Micklefield <i>et al</i> ¹¹	Longer and faster contractions
Aithal <i>et al</i> ¹²	Decrease in the proportion of peristaltic contractions
Ready <i>et al</i> ¹³	Longer esophageal clearance

Post-stroke oral, pharyngeal, and esophageal function were assessed with videofluoroscopy^{7,14}, scintigraphy^{8,9}, and manometry¹⁰⁻¹². The findings report increased esophageal non-peristaltic events¹⁰, prolonged esophageal transit time⁷, decreased peristalsis proportion in patients with dysphagia¹⁰, increased esophageal clearance time^{12,13}, esophageal retention, and retrograde flow¹³.

Esophageal transit duration in patients with stroke was 17 seconds, compared with 11 seconds in controls (p<0.001)⁸, and 58% of patients had longer esophageal clearance, esophageal retention and retrograde flow¹³.

Evaluated in the upright position 10 to 56 days (median: 43 days) after the ictus, stroke patients had shorter distal esophageal transit duration of liquid bolus

($1.74 \pm 0.84s$) compared to controls ($2.68 \pm 1.65s$; $p=0.028$)⁸. The position of evaluation may be the cause of shorter distal transit duration of patients with stroke. In this position, with no effective peristalsis but with relaxation of the lower esophageal sphincter, the distal bolus transit may be faster. In the mid-esophagus, with ischemic lesions in the vertebrobasilar territory, esophageal clearance took longer in stroke patients ($3.80 \pm 1.28s$) than in controls ($2.92 \pm 1.49s$; $p=0.027$)⁸.

Another investigation found, after swallowing evaluation performed one to 84 months (median: 5.5 months) after the ictus, that a sour bolus caused longer clearance duration in post-stroke patients compared with neutral, sweet, and bitter boluses – a similar behavior to that of controls, without differences between patients and controls⁹, a suggestion that the perception of different bolus taste may be present. With neutral bolus, esophageal residues were highly frequent in stroke patients⁹.

Differences in the intensity of changes in stroke patients' esophageal transit, as described in the investigations, may be due to differences in time between the ictus and esophageal motility and transit evaluation and different method of swallowing assessment. The modern methods to evaluate esophageal contractions and transit are the videofluoroscopy, high-resolution manometry and impedanciometry^{15,16}.

Esophageal manometry in patients with stroke performed within two days after hospital admission shows

that the proportion of peristaltic waves in the distal esophagus was $93.5 \pm 1.1\%$ in patients without dysphagia and $53.5 \pm 4.4\%$ in those with dysphagia ($p < 0.001$). In the proximal esophagus, the proportion was $93.2 \pm 3.4\%$ in patients without dysphagia and $62.1 \pm 7.3\%$ ($p < 0.050$) in patients with dysphagia¹⁰.

Hence, it indicates that dysphagia may also result from impaired esophageal contraction peristalsis. Manometric measures taken 2 to 10 days after a stroke found longer contractions (stroke: 4.2 ± 1.0 s; controls: 2.2 ± 0.7 s; $p < 0.001$) and faster contractions through the esophageal body (stroke: 6.3 ± 1.1 cm/s; controls: 3.2 ± 0.8 cm/s, $p < 0.001$)¹¹.

Differences in oral and pharyngeal phases of swallowing between patients with stroke and controls tend to improve or disappear with time after the ictus, which is also expected from changes in esophageal motility. In patients with stroke and no important involvement of the oral and pharyngeal phases of swallowing, the mean percentage of complete esophageal peristaltic events increased from 58.7(5.9)% – assessed three to five days after the ictus – to 77.3(3.9)% – three weeks after the ictus ($p = 0.005$)¹².

Results describing longer esophageal clearance after a stroke are important because it may affect the treatment. Sour liquids (citrus) increase esophageal clearance time in healthy^{9,17} and post-stroke subjects⁹. Thus, sour liquids may not be the best option to increase total fluid intake by

patients with post-stroke dysphagia days after the ictus, because it may affect esophageal transit.

However, in the pharynx, sour and cold liquids, a thermal and chemical modification of pure water^{18,19} may improve the liquid ingestion. This possibility has not been fully clarified yet and requires further investigation.

As previously pointed out¹⁴, all patients with oropharyngeal dysphagia should have their esophageal transit and/or esophageal motility assessed.

CONCLUSION

Stroke may affect the esophageal transit and contraction, with longer clearance time and more non-peristaltic contractions. These changes became less frequent with time after the ictus.

REFERENCES

1. Camilleri M. Gastrointestinal motility disorders in neurologic disease. *J Clin Invest* 2021;131:e143771. <https://doi.org/10.1172/JCI143771>
2. Martino R, Foley NC, Bhogal S, Diamant N, Speechley M, Teassel R. Dysphagia after stroke: incidence, diagnosis and pulmonary complications. *Stroke* 2005;36:2756-63. <https://doi.org/10.1161/01.STR.0000190056.76543eb>
3. Pacheco-Castilho AC, Vanin GM, Dantas RO, Pontes-Neto OM, Martino R. Dysphagia and associated pneumonia in stroke patients from Brazil. *Dysphagia* 2019;34:499-520. <https://doi.org/10.1007/s00455-019-10021-0>
4. Clavé P, Shaker R. Dysphagia: current reality and scope of the problem. *Nat Rev Gastroenterol Hepatol* 2015;12:259-70. <https://doi.org/10.1038/nrgastro.2015.49>
5. Nikaki K, Sawada A, Ustaoglu A, Sifrim D. Neuronal control of esophageal peristalsis and its role in esophageal disease. *Curr Gastroenterol Rep* 2019;21:59. <https://doi.org/10.1007/s11894-019-0728-z>
6. Lang IM. Brain stem control of the phases of swallowing. *Dysphagia* 2009;24:333-48. <https://doi.org/10.1007/s00455-009-9211-6>

7. Reedy EL, Herbert TL, Bonilha HS. Visualizing the esophagus during modified barium swallow studies: a systematic review. *Am J Speech Lang Pathol* 2021;30:761-71. https://doi.org/10.1044/2020_AJSPL-20-00255
8. Miles A, Bennett, K, Allen, J. Esophageal transit times vary with underlying comorbid disease. *Otolaryngol Head Neck Surg* 2019;161:829-34. <https://doi.org/10.1177/0194599819874342>
9. Silva ACV, Fabio SRC, Dantas RO. A scintigraphic study of oral, pharyngeal, and esophageal transit in patients with stroke. *Dysphagia* 2008;23:165-71. <https://doi.org/10007/s00455-007-9117-0>
10. Alves, LMT, Fabio SC, Dantas RO. Effect of bolus taste on the esophageal transit of patients with stroke. *Dis Esophagus* 2013;26:305-10. <https://doi.org/10.1111/j.1442-2050.2012.01366x>
11. Micklefield GH, Jorgensen E, Blaeser I, Jorg J, Kobberling J. Esophageal manometric studies in patients with an apoplectic stroke with/without oropharyngeal dysphagia. *Dtsch Med Wochenschr* 1999;124:239-44. <https://doi.org/10.1055/s-2007-1024278>
12. Micklefield GH, Jorgensen E, Blaeser I, Jorg J, Kobberling J. Motility disorders of the esophagus in patients with apoplectic infarct during the acute illness phase. *Med Klin* 1999;94:245-50. <https://doi.org/10.1007/BF03045048>
13. Aithal GP, Nylander D, Dwarkanath AD, Tanner AR. Subclinical esophageal peristaltic dysfunction during the early phase following a stroke. *Dig Dis Sci* 1999;44:274-8. <https://doi.org/10.1023/A:1026690030900>
14. Reedy EL, Simpson NA, O'Rourke AK, Bonilha HS. Abnormal esophageal clearance identified during modified barium swallow study in an acute poststroke cohort. *Am J Speech Lang Pathol* 2022;31:2643-62. https://doi.org/10.1044/2022_AJSLP-22-00029
15. Gascon L, Bryon PC, Benninger M, Brodsky MB. Assessing dysphagia in the adult. *Otolaryngol Clin N Am* 2024;in press. <https://doi.org/10.1016/j.otc.2024.03.003>
16. Tack J, Pawels A, Roman S, Savarino E, Smout A. European society for Neurogastroenterology and Motility (ESNM) recommendations for the use of high-resolution manometry of esophagus. *Neurogastroenterol Motil* 2021;33:e14043. <https://doi.org/10.1111/nmo.14043>
17. Alves LMT, Secaf M, Dantas RO. Oral, pharyngeal, and esophageal transit of an acidic bolus in healthy subjects. *Esophagus* 2013;10:217-22. <https://doi.org/10.1007/s10388-13-0389-1>
18. Handy S, Jilani S, Price V, Parker C, Hall N, Power M. Modulation of human swallowing behavior by thermal and chemical stimulation in health and after brain injury. *Neurogastroenterol Motil* 2003;55:69-77. <https://doi.org/10.1046/j.1365-2982.2003.0039.x>
19. Cola PC, Gatto AR, Silva RG, Spadotto AA, Schelp AO, Henry MACA. The influence of sour taste and cold temperature in pharyngeal transit duration in patients with stroke. *Arq Gastroenterol* 2010;47:18-20. <https://doi.org/10.1590/S0004-28032010000100004>