

The deglutition and conditions affecting it.

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ABSTRACT: Deglutition is a very complicated neuromuscular function starting from the very first moment the man is born, providing to the organism necessary nutrients for growing and maintaining in life.

It takes place in three stages (Anatomical aspects) commencing with a voluntary one and followed by other two involuntary successive reflexes coordinated from the deglutition centre in the medulla near the vagal nucleus and the respiratory centre. However, this fine function may be affected by various conditions (Clinical aspects) analysed in the text.

Key Words: Neuromuscular function voluntary, Involuntary, Oesophageal stenosis, Vascular ring.

INTRODUCTION

ANATOMICAL ASPECTS

First stage (voluntary)

It starts by introducing food into the oral cavity. The anterior part of the tongue raises **and presses the hard palate**, the soft palate close, the solid food is masticated, mixed with saliva, form the bolus and pushed to the posterior part of the oral cavity by the intrinsic muscles of the tongue (superior and inferior longitudinal, transverse and vertical) mainly by the superior longitudinal, and the transverse. At the same time, by contraction of the geniohyoid, mylohyoid, digastric and stylohyoid, the hyoid bone ascends and becomes fixed. By the action of styloglossi the posterior part of the tongue elevates posterosuperiorly and by the contraction of palatoglossi and approximation of the palatoglossal arches the bolus is squeezed backward into the oral part of the pharynx.

In case of fluids the intrinsic tongue muscles push the fluid back through the mouth into the oral pharynx.

Second stage (involuntary)

It begins as soon as the bolus passes through the oropharyngeal isthmus into the oral pharynx with the el-

evation of soft palate by the elevator muscles - veli palatini - and by the tensor muscles - tensor veli palatini.

Furthermore, by the contraction of the palatopharyngeal sphincter and the upper fibres of the superior constrictor the bolus approximates to the posterior pharyngeal wall and the pharyngeal isthmus closes preventing its upwards passing.

By the action of stylopharyngeus, salpingopharyngeus, thyrohyoid and palatopharyngeus muscles the larynx and pharynx drawn upwards.

At the same time, in order to prevent the entrance of the bolus into the larynx, the aryepiglottic, oblique arytenoids and thyroarytenoid muscles contract, the aryepiglottic folds are approximated and the arytenoids cartilages are drawn upwards and forwards.

The descend of the bolus into the oesophagus is facilitated by the contraction of the palatopharyngeal muscles which convert the posterior pharyngeal wall into an inclined surface directed downward and backward.

Third stage (involuntary)

The inferior constrictor compresses the bolus into the oesophagus. (Gray's 1973).

CLINICAL ASPECTS

The function of deglutition may be altered:

In cases of palatine cleft (Gray's 1973, Snell 1995, Ger et al 1996), the frequency of which, in general, is 1 in 750 births (Sinnatomby 1999). Deglutition malfunction grade depends on the degree of malformation.

In case of oesophageal stenosis due to vascular ring (Godtfredsen et al 1977, Backer et al 1989, Morris et al 2000) or to extrinsic oesophageal compression by regional carcinomas and/or enlarged lymph nodes. There are also cases of intrinsic stenosis due to trauma, infections or radiotherapy (Camidge 2001).

In case of mediastinal vascular anomalies. Normally the right subclavian artery arises from the brachiocephalic trunk and the left one from the aortic arch; however, sometimes the right subclavian artery originates as a last branch from the aortic arch arising directly from it. In its course then to the neck, in order to get in its normal position (Sclavounos 1934-1938, Ellis 1997), it passes behind the trachea and oesophagus and may compress the oesophagus (Kasimos 1976) causing difficulty in swallowing - «dysphagia lusoria» - (Gray's 1973, Ellis 1997, Sinnatomby 1999).

It has also been reported (Gupta and Winslet 2005) that tortuosity of the right common carotid artery; coil of the proximal common carotid artery (Lin et al 2000); aneurysms of the common carotid; aneurysms of the internal carotid artery; aberrant subclavian arteries may also cause extrinsic oesophageal compression (Rowe et al 1994, Nicolosi et al 1995).

In diphtheria soft palate may paralyse, due to the action of the toxin on the nerve cells in the medulla oblongata, with an end result of regurgitation of fluids or food into the nose when swallowing is attempted (Mericas 1973, Gray's 1973, Ellis 1997). These symptoms, usually, last for one or two weeks, rarely more, and finally they disappear (Mericas 1973). Typically patients with different types of oesophageal tumours (Table 1) (McBeth et al 2008), exhibit progressive dysphagia to solids and liquids and about 50% of them experience pain (odynophagia). Other causes of dysphagia may due to acute or late side effects of radiotherapy to the oesophagus. The acute side-effect is mucositis and the late one is benign stricture that is needed endoscopic evaluation, biopsy and dilatation.

In case of hypercalcaemia, which is a common

metabolic complication of malignancy, probably because it reduces the contractility of smooth muscles. If its properly treated and the serum calcium normalizes (2,1-2,65 mmol/l) the symptoms of dysphagia rapidly improve (Grieve and Dixon 1983, Balcombe 1999, Amin 2002).

In case of injuries of the hypoglossal nerve (12th cranial nerve) which cause difficulty in swallowing (Gray's 1973).

In cerebral trauma involving the 9th, 10th and 11th cranial nerves (glossopharyngeal, vagus and accessory) the swallowing reflex is depressed (Gray's 1973, Elli 1997) and **foreign material may be aspirated into the pulmonary trunk**, especially in cases the patient lies on his/her back position (Ellis 1997).

In patients being unconscious or anaesthetized normal swallowing cannot take place, because the first stage of swallowing is a voluntary one (McMinn et al, 1996). In those patients the tongue muscles relax and the tongue may fall backwards and obstruct the pharyngeal part of the airway (Ellis 1997, Sinnatomby 1999). In such cases the tongue must be pulled forwards in order to restore a patent airway (Sinnatomby 1999). The narcotics depress the central nervous component of the swallowing reflex and normal deglutition is «disturbed» having as a result the aspiration of food or liquids administered (Ellis 1997) with serious consequences.

Table 1. Types of oesophageal tumour.

Type	Example
Benign neoplasms	Adenoma Leiomyoma Haemangioma
Primary Malignant Neoplasms	Adenocarcinoma (65%) Squamous carcinoma ($\approx 25\%$) Others ($\approx 5\%$) Small cell carcinoma BCC (Basal Cell Carcinoma) Adenoid cystic carcinoma Mucoepidermoid carcinoma Carcinoid Lymphomas Melanoma Leiomyosarcoma Gastrointestinal stromal tumour (GIST)
Secondary Malignant Neoplasms	Lung carcinoma Breast carcinoma Melanoma

Η κατάποση και οι παράγοντες που την επηρεάζουν.

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ΠΕΡΙΛΗΨΗ: Η κατάποση είναι μια τέλεια και σύνθετη νευρομυϊκή λειτουργία, που αρχίζει από την στιγμή που γεννιέται ο άνθρωπος, παρέχοντας στον οργανισμό τα απαραίτητα συστατικά διατροφής για τη διατήρηση στη ζωή.

Περιλαμβάνει τρεις φάσεις (από πλευράς ανατομικής), η πρώτη είναι εκούσια και οι άλλες δύο ακούσιες και αντανακλαστικές με εντολή του κέντρου κατάποσης στο μυελό κοντά στον πυρήνα του πνευμονογαστρικού και του αναπνευστικού κέντρου. Αυτή η τέλεια λειτουργία μπορεί να προσβάλλεται από διάφορες παθολογικές καταστάσεις. Στο κεφάλαιο που ακολουθεί γίνεται μια προσπάθεια να αναλυθούν οι ανατομικές και οι κλινικές καταστάσεις που επηρεάζουν την κατάποση.

Λέξεις Κλειδιά: Νευρομυϊκή λειτουργία, Εκούσια, Ακούσιες, Οισοφαγική στένωση, Αγγειακός δακτύλιος.

REFERENCES

1. Amin R.: Dysphagia in lung cancer ; 2002. JRSM, 95, (1): 55-56.
2. Backer C L, Hbawi M N, Idriss F S and DeLeon S Y.: Vascular anomalies causing tracheoesophageal compression; 1989. J Thorac Cardiovasc Surg, 97 (5): 725-31.
3. Balcombe N R.: Dysphagia and hypercalcaemia; 1999. Postgrad. Med. J., 75:373-374.
4. Camidge D R.: The causes of dysphagia in carcinoma of the lung; 2001. JRSM, 94, (11): 567-572.
5. Ellis H.: *Clinical Anatomy*; 1997, 9th Edition, Blackwell Science, p. 291,303, 304, 323.
6. Ger R, Abrahams P and Olson T R.: *Essentials of Clinical Anatomy*; 1996. 2nd Edition, The Parthenon Publishing Group, p. 190-191.
7. Godtfredsen J, Wennevold A, Efsen F, Lauridsen P: Natural history of vascular ring with clinical manifestation; 1977. Scand J Thor Cardiovasc Surg 11: 75-77.
8. Grieve R J and Dixon P F.: Dysphagia: a further symptom of hypercalcaemia? 1989. BrMJ, 286, p. 1935-6.
9. Gupta A and Winslet M C.: Tortuous common carotid artery as a cause of dysphagia; 2005. JRSM, 98, (6): 275-276.
10. Kasimos C.: *Paediatrics (in Greek)*, 1976. Thessaloniki, Vol. 2, p. 689.
11. Lin P H, Bush R L, Reddy P and Lumsden A B.: An unusual cause of dysphagia: coil of the proximal common carotid artery; 2000. J Vase Surg, 34 (6): 521-6.
12. McBeth F. et al «*Practical Clinical Oncology*», 2008 ISBN 10:0521618169 Chapter 9.
13. McMinn M H, Gaddum-Rose P, Hutchings R T, Logan B M.; McMinn's *Functional and Clinical Anatomy*; 1996. Mosby, p. 185.
14. Mericas G E.: *Internal Medicine (in Greek)*, 1973. Paschalidis, Vol. 2, p. 324-325.
15. Morris C D, Kanter K R and Miller J I.: Late-onset dysphagia tusoria; 2000. Ann Thorac Surg, 71: 710-2.
16. Nicolosi A C and Cambria R A.: Late development of esophageal compression from a vascular ring; 1995. Arm Thorac Surg, 60: 1413-5.
17. Rowe J G and Hosni A A.: A common carotid artery aneurysm causing severe dysphagia; 1994. J Laryngol Otol, 108: 67-68.
18. Sclavounos G.: *Human Anatomy (in Greek)*, 1934-1938. 3rd Edition, Tarousopoulos, Vol 3, p. 147.
19. Sinnatombay C S.: *Last's Anatomy Regional and Applied*; 1999. 10th Edition, Churchill Livingstone, p. 28, 375.
20. Snell R S.: *Clinical Anatomy for Medical Students*; 1995. 5th Edition. Little Brown and Company, p. 743-744.
21. Warwick R and Williams P.: *Gray's Anatomy*; 1973. 35th Edition, Longman, p. 119,639-641, 1027, 1208-1209, 1246.