OESOPHAGEAL MOTILITY DISORDERS: RAPID FUNCTIONAL DIAGNOSIS USING COMPUTERISED RADIONUCLIDE OESOPHAGEAL TRANSIT STUDY

M Paramsothy, K L Goh, P Kannan

ABSTRACT

Ten patients presenting with central chest pain and/or dysphagia were diagnosed to have oesophageal motility disorders (OMD) with an incoordinate motor function using computerised radionuclide oesophageal transit study (RT). The criteria for diagnosis of OMD with incoordination using RT were: an'incoordinate' or 'to and fro' pattern characterised by multiple peaks of activity, prolonged total transit time or radionuclide bolus through entire length of oesophagus and a significant portion of bolus entering the stomach. These features are characteristic but not pathognomonic of diffuse oesophageal spasm (DES) as they are also seen in non-specific motility disorders (NSMD) and occasionally in other oesophageal motility disorders. Mechanical obstruction in the oesophagus and coronary artery disease were excluded appropriately in these patients. When manometry is not available, RT is a sensitive, safe, simple, rapid and non-invasive alternative modality in confirming certain oesophageal motility disorders.

Keywords: *oesphageal motility disorders, diagnosis, radionuclide oesophageal transit study.*

SINGAPORE MED J 1995; Vol 36: 309-313

INTRODUCTION

Oesophageal motility disorders can be characterised as primary or secondary. Primary dysmotility include diffuse oesophageal spasm (DES), achalasia, non-specific motility disorders (NSMD), hypertensive lower oesophageal sphincter, acid-induced motility disorders, 'Nutcracker' oesophagus and 'vigorous' achalasia. Secondary dysmotility may occur in diabetes, scleroderma, other connective tissue disorders, Chagas disease and from surgery[1].

The diagnostic criteria for oesophageal motor disorders have been defined by oesophageal manometry. For example in achalasia, there is an elevated lower oesophageal sphincter (LES) pressure with incomplete sphincter relaxation and aperistalsis of the entire oesophagus. In diffuse oesophageal spasm, LES function is normal but there is repetitive high amplitude and non-progressive contractions along with some normal peristaltic contractions.

Oesophageal motility disorders, notably diffuse oesophageal spasm, clinically present with central chest pain and/or dysphagia. These retrosternal pains often mimic that of cardiac origin[2]. Many such patients have been misdiagnosed, investigated over and over and mistreated as due to having ischaemic heart disease or functional disorder for example cardiac neurosis. Oesophageal dysmotility causing such symptoms have been uncommonly diagnosed due to lack of clinical suspicion and to unavailability of oesophageal manometry.

Division of Nuclear Medicine
University Hospital
59100 Kuala Lumpur
Malaysia

M Paramsothy, FRCP (Lond)
Professor

Department of Medicine
University Hospital

K L Goh, MRCP (UK)
Associate Professor

P Kannan, FRCP (Lond)
Associate Professor

Correspondence to: Prof M. Paramosthy

Radionuclide oesophageal transit studies (RT) have been used to screen for suspected oesophageal motor disorders and to assess response to treatment[3,4]. Over the past one year we have diagnosed oesophageal motility disorders with incoordinate motor function in ten patients using computerised radionuclide oesophageal transit scintigraphy. We report these ten cases.

MATERIALS AND METHODS

Ten patients with complaints of intermittent central chest pain and/or dysphagia suspected clinically to have oesophageal motility disorders after exclusion of other diseases were studied with RT. Six patients complained of intermittent central chest pain only, two patients had dysphagia only, and two patients complained of both intermittent chest pain and dysphagia (Table I).

RT studies were carried out as described previously[3]. Nitrates and calcium antagonists were discontinued 48 hours before tests were carried out. The patients in fasting state (> 4 hours) were positioned supine (to avoid effect of gravity) beneath a gamma scintillation camera interfaced to a computer. Divergence collimation was used to allow the events in the mouth, the entire oesophagus and the stomach to be recorded. The position of the cricoid cartilage to mark the upper end of the oesophagus was made using a radioactive marker. A 10 ml homogenous liquid bolus of water and 0.25 mCi of Technetium-99m-colloid was introduced into the mouth from a syringe and ingested on demand with a single swallow. The activity in the oesophagus during the swallowing sequences were recorded at the rate of one frame every 0.5 second for 60 seconds. The recorded images were visualised in the form of cine display. Simultaneous analogue 1 sec x 36 sequential images were also obtained on X-ray films. The mouth and pharynx were monitored for residual radioactivity to prevent artifacts resulting from delayed entry of the bolus into the oesophagus as a result of an incomplete initial swallow or hesitant double swallow of the bolus in succession. This was determined by generation of activity-time (A/T) curve of a region of interest (ROI) over the mouth area. A clean single swallow is demonstrated as a sharp fall in radioactivity. In the event of an incomplete swallow or a double swallow, the study was repeated with another identical

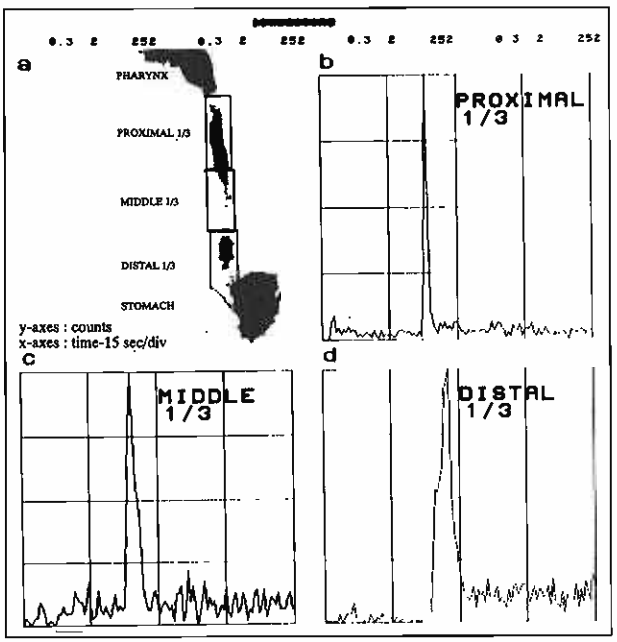
Table I - Clinical summary of ten patients

Patient	Age (yrs)	Sex/ Ethnic	Clinical History	Upper Gastro-Intestinal Ensodcopy	Barium Swallow	Stress ECG	Coronary Angiogram	RT studies
1. NSY	45	F/C	Gripping retrosternal pains, usually at rest, not related to meals and swallowing for 18 months. No dysphagia or heart burns.	Not done	Not done	Normal	Normal	Normal RT pattern in proximal third and inco-ordination in distal two-thirds of oesophagus. TTT = 54 sec
2. VD	56	M/I	Intermittent dysphagia to both solids and liquids for 2 years. No chest pains.	Normal	Normal	Not done	Not done	Inco-ordination in entire length of oesophagus. TTT > 60 sec
3. AR	49	M/M	Intermittent severe, 'crushing' central chest pain, usually at rest. No dysphagia. Exercise 201 Thallium and exercise. RVG studies normal.	Not done	Not done	Normal	Normal	Normal RT in proximal thirds and incoordination in distal two-thirds of oesophagus. TTT > 60 sec
4. ND	57	F/I	Episodic retrosternal 'pressing' pain relieved by GIN for 1st year. No dysphagia.	Normal	Not done	Normal	Normal	Normal RT in upper two-thirds and inco-ordination in distal third of oesophagus. TTT > 60 sec
5. RE	50	F/C	Intermittent dysphagia, worse to solids than liquid. Central chest pains 'pulling' in nature and unrelated to meals for last 6 months. No heart burn or loss of weight.	Normal	Typical cocks-screw oesopha-gus	Normal	Not done	Inco-ordination in entire length of oesophagus TTT > 60 sec
6. WFP	30	F/C	Intermittent central chest pain, 'gripping' or 'pressing' in nature. Not related to exertion for 5 yrs. No dysphagia.	Normal	Typical cock-screw oesopha-gus.	Normal	Not done	Normal in proximal third and inco-ordination in lower two-thirds of oesophagus
7. KA	60	M/M	Intermittent dysphagia associated with episodic severe central chest pain 'pulling' in nature and not related to meals or exertion for 10 years. Had myocardial infarction 9 months ago. Free from angina pectoris since percutaneous transluminal coronary angioplasty. Ex 201 Thallium and Ex RVG. Post PTCA show no ischaemia.	Normal	Normal	Normal post Angio-plasty (PTCA)	Coronary artery disease with PTCA done	Inco-ordination in entire length of oesophagus. TTT > 60 sec
8. KTB	48	F/C	Intermittent dysphagia for 10 years. No chest pain.	Normal	Not done	Not done	Not done	Normal RT in proximal third. Inco-ordination in distal two-thirds of oesophagus. TTT > 60 sec
9. G	38	F/I	Recurrent gripping retrosternal pain radiating to left shoulder for 6 months. No dysphagia. Ex 201 Thallium and Ex RVG studies normal.	Not done	Not done	Normal	Normal	Normal RT in proximal two-thirds. Inco-ordination in distal third of oesophagus. TTT > 60 sec
10. SH	47	F/M	Recurrent crushing retrosternal pain. No dysphagia.	Not done	Not done	Normal	Normal	Inco-ordination in entire length of oesophagus. TTT > 60 sec

radionuclide liquid bolus. When none of the bolus enters the stomach, a second study is performed in the erect position. A/T curves of the transit pattern of bolus radioactivity (representing volume) through the whole length of the oesophagus and through each equal third of the oesophagus and the stomach were generated. The total transit time (TTT) as the interval between first appearance of activity in the oesophagus to its final clearance was calculated. Clearance was considered when the radioactive counts had fallen below 10% of the peak value as a steady low level of activity in each patient after the passage of the bulk of bolus through the oesophagus. The individual TTT in each equal third of the oesophagus (proximal, middle, and distal) were also calculated in a similar manner.

A 'normal' RT pattern (Fig 1) is defined as a smooth coordinated temporal progression of bolus in an aboral direction and characterised by three distinct sequential peaks of activity representing proximal (Fig 1b), middle (Fig 1c) and distal (Fig 1d) oesophageal regions respectively[2]. Reported values for mean TTT for entire oesophagus is 7.7 sec ± 1.7 (SD)[3] and 7.3 sec ± 2.3 (SD) with a range 5 - 15 seconds[5]. Mean transit times for individual thirds are: proximal 2 sec ± 0.8 (SD); middle 4.4 sec ± 1.7 (SD); and distal 7.2 ± 1.7 (SD). There is increasing time with distal progression[3]. RT abnormality is considered severe when TTT is greater than 50 seconds[2]. The A/T curves were examined for abnormal RT patterns. An 'adynamic' or 'static retention' pattern is characterised by complete loss of normal distinct sequential peaks of activity with a prolonged TTT and very little radioactivity reaching the stomach even when repeated in the upright position[2]. An 'incoordinate' or 'to and fro' pattern is characterised by multiple peaks of activity showing the disorganised bolus transit with periods of retrograde movement, and a prolonged TTT and significant portion of bolus entering the stomach in the first 50 seconds even in the supine position[2]. The A/T curves are also examined for combination of both patterns occurring.

Fig 1 – Normal RT study (a) sum of serial frames of passage of radionuclide bolus through the oesophagus after a single swallow showing regions of interest for activity-time (A/T) curve generation. Transit shown through proximal (b), middle (c), and distal (d) thirds of oesophagus as three distinct sequential peaks of activity indicate a smooth passage of bolus in an aboral direction.



Note: Not shown are A/T curves generated over pharynx and stomach to check on quality of bolus ie no double swallow or fragmented bolus and for early and complete entry of bolus into stomach. Vertical and horizontal axes are radioactivity and time in seconds respectively.

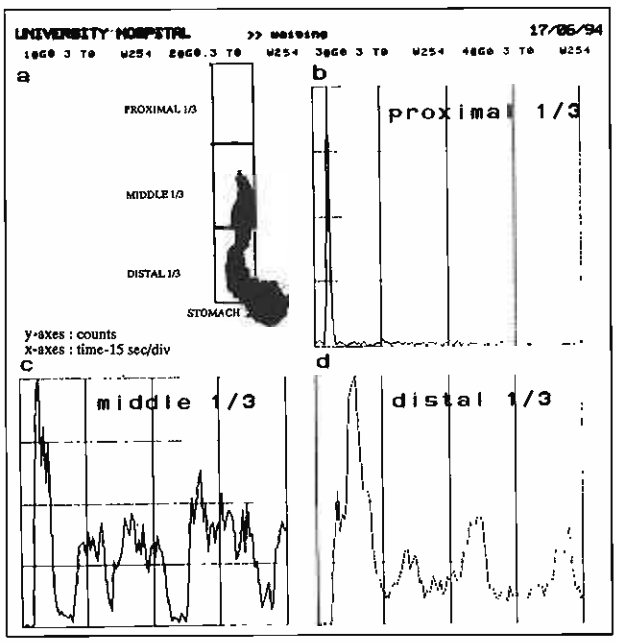
RESULTS

The clinical information and the results of the various investigations performed on the ten patients are summarised in Table I. Six patients only had intermittent retrosternal or central chest pains variously described as 'pulling', 'pressing', 'gripping' or 'crushing' in nature occurring at rest and not related to exertion except in one patient who had 'gripping' central chest pain both at rest and rarely during exertion. Two patients, had both intermittent dysphagia and central chest pains, the former being major symptom. Two patients complained of intermittent dysphagia only and no chest pains.

All ten patients had abnormal RT pattern typical of an 'incoordinate' or 'to and fro' pattern. Concurrent 'adynamic' pattern was not detected in all ten patients. Four patients had 'incoordinate' pattern throughout the entire length of the oesophagus, and with a prolonged total oesophageal transit time of more than 52 seconds.

Significant portion of bolus activity was demonstrated in the stomach within the first 36 seconds even with the patient in the supine position. Four patients demonstrated a normal RT pattern characterised by a distinct peak of activity in the proximal third (Fig 2b) of the oesophagus and an abnormal transit with an 'incoordinate' pattern characterised by multiple peaks of activity in the middle and distal thirds of oesophagus (Fig 2c and d). The TTT was prolonged to more than 52 seconds. The transit time in the proximal third was 2.5

Fig 2 – Abnormal RT study after a single swallow. 'Incoordinate' RT pattern characterised by multiple peaks of activity represent disorganised 'to and fro' intraoesophageal movement of bolus in middle (c) and distal (d) thirds of oesophagus.



Note: Initial normal bolus transit represented by a single distinct peak of activity in proximal third of oesophagus. Axes as in Fig. 1.

seconds and more than 50 seconds both in the middle and distal third of the oesophagus.

Significant bolus activity was seen in the stomach within the first 36 seconds. The analogue images of the bolus transit demonstrated temporary 'hold-up' of disorganised bolus with periods of retrograde bolus activity.

For two patients normal RT pattern demonstrated in the proximal and middle third was replaced by the typical 'incoordinate' pattern in the distal third of the oesophagus. The transit times in both patients were less than 2.5 seconds in the proximal third, less than 5 seconds in the middle third and more than 52 seconds in the distal third of the oesophagus.

DISCUSSION

Oesophageal motility disorders with 'incoordinate' motor function, notably diffuse oesophageal spasm, occurs in all ages and classically presents with chest pain and/or dysphagia occurring intermittently and varying in severity and frequency. The pains may be precipitated by ingestion of food but often occur unrelated to meals and sometimes occurring at night during sleep, mimicking pains due to myocardial ischaemia. Dysphagia is also of variable severity and lacks the persistence seen in achalasia or an organic stricture.

In the typical patient with DES, peristalsis progresses in a normal fashion down to the middle third of the oesophagus where it is replaced by simultaneous non-progressive contractions of the remaining oesophagus. These abnormal contractions may be of high amplitude and prolonged duration. Repetitive contractions are also noted. These abnormalities are intermittent and the oesophagus has not lost its ability to produce normal peristalsis and normal lower oesophageal sphincter relaxation.

The barium cineradiography appearance of DES is described as 'curling', segmental spasm, rosary bead oesophagus and corkscrew oesophagus. These terms refer to the segmental, non-peristaltic contractions which may trap the barium and push it back and forth. This is best demonstrated when the patient is recumbent. A second but less common picture is a tight contraction of the oesophagus over a length of several centimetres or a slight diffuse narrowing of the lower half of the oesophagus with a slightly dilated upper segment. The extent and severity of the radiological abnormalities may vary widely from patient to patient and from one time to another in the same patient, and this may explain the normal barium swallow examinations in some of these patients. Barium swallow is not quantitative, relies entirely on subjective interpretation by the observer, the radiation dosage to the subject being significantly higher than with a radionuclide study and the physical properties of barium (eg density and viscosity) appear to influence its behaviour within the oesophagus.

Accurate characterisation of a motility disorder requires manometry. Although it is the most sensitive method, it is uncomfortable, invasive and requires experience and expertise in its performance and interpretation. However manometry which records intraluminal pressure changes provides only an indirect measure of peristalsis; it does not reflect bulk transit or correlate with the force applied in an aboral direction to move a bolus. Radionuclide transit measurement is an excellent alternative method to manometry for the detection and assessment of oesophageal dysmotility when manometry is not available or with poor patient compliance, and provides useful additional information about oesophageal function[1,3,5]. It is a simple, quick, non-invasive and safe test that involves very little radiation and discomfort to the patient. The overall sensitivity of radionuclide transit test in detecting oesophageal dysmotility was 75%, while the sensitivity of manometry was 83%, and that of barium swallow 30%[4]. The concordance between RT and manometry was 84%[5]. There are also significant number of patients with intermittent dysphagia and normal manometry but abnormal 'incoordinate' RT pattern[3]. However Mughal et al found an overall sensitivity of RT in detecting oesophageal dysmotility to be only 44%[7]. Discrepancy between these results are possibly due to factors such as patient selection and interpretation of manometry[7]. DES is frequently intermittent and may be present during one examination but not the other.

It is stressed that because radionuclide oesophageal scintigraphy lacks precise anatomical definition, abnormal radionuclide screening studies should be followed by endoscopy and/or barium cineradiography to exclude anatomic lesions (eg a tumour) as the cause of dysmotility or pain if these tests had not been performed earlier. The transit of liquid bolus through the oesophagus in the supine position is determined by the production or propagation of a peristaltic contraction in response to a swallow. The variation in the amplitude, duration and form of peristaltic contraction and the pressure of the lower oesophagus do not have significant influence on RT time, provided that the wave front after a swallow is peristaltic and the lower oesophageal sphincter relaxed normally[6]. Adynamic pattern is typically seen in conditions of 'aperistalsis' such as achalasia, scleroderma and occasionally in diabetes[1,3,7]. Regional analysis helps to differentiate achalasia from scleroderma. Although there is delayed transit through the distal two-thirds of oesphagus in scleroderma, most of the bolus enters the stomach. In patients with achalasia, the oesophageal retention is more marked, involving the entire length of the oesophagus and only a little portion of bolus enters the stomach. In diabctes or 'presbyesophagus', the delayed transit occurs in the entire length of the oesophagus but a significant portion of radioactive bolus enters the stomach. Mughal et al reported the 'adynamic' pattern to be demonstrated in a significant number of patients with non-specific motility disorders (NSMD), in patients with manometric diagnosis of diffuse oesophageal spasm (DES), in patients with achalasia, and in patients with 'nutcracker' oesophagus and the 'incoordinate' pattern to be seen in majority of diffuse oesophageal spasm and in a high proportion of non-specific motility disorders[7]. De Caestecker et al found a low but significant number with achalasia to have both an 'incoordinate' and an 'adynamic' patterns RT[1]. The repetitive intermittent non-progressive/ non-peristaltic contractions of high amplitude and prolonged duration interspersed among normal peristaltic contractions trap and push the radionuclide bolus or barium back (retrograde movement) and forth (or 'to and fro') giving rise to the 'incoordinate' pattern seen in diffuse oesophageal spasm[1]. The 'incoordinate' RT pattern, although more commonly seen in DES, is not pathognomonic of it.

RT and manometry are complementary investigations, each detecting abnormalities not detected by the other because they assess different aspect of oesophageal motor function. RT not only measures the oesophageal transit time but also the actual dynamics (pattern) of bolus progression by the graphic analysis of radioactivity in the three thirds of the oesophagus. Manometry examines only some aspect of peristalsis and in particular it does not measure the actual force acting in an aboral direction on a bolus[1,3]. Russel et al found abnormal RT in all 15 patients with known manometric abnormalities and an incidence of 64% of RT

abnormality in a group of patients with dysphagia and normal manometry[3]. Also Kjellen et al have shown that at least 50% of patients with dysphagia, but normal results of manometry, acid perfusion test, acid clearing test pH reflux studies and barium studies, have objective evidence of oesophageal dysmotility with radionuclide transit scintigraphy[8]. Whether RT abnormalities in asymptomatic patients or in symptomatic patients with normal manometry represent true 'false positive' result or whether they are significant abnormalities is a conjecture. The fact that they are seen also in some patients with a manometric abnormality suggest they may indeed be of clinical relevance. False positive RT results may occur from technical reasons such as from a hesitant double swallow, pooling proximal to a tight stricture, in diverticulum or in herniated part of the stomach[9] and from failure of swallow to generate propagated peristalsis[10]. However, it does not necessarily mean the failure of a swallow to generate propagated peristalsis as described by Nelson et al[10] in an asymptomatic patient is normal. It is to be expected that in some patients with DES, RT studies will be abnormal in the face of normal manometry and vice versa, as oesophageal spasm is frequently intermittent and may be present during one examination but not the other[1]. Thus, in symptomatic patients with normal manometry, a prolonged RT time with an 'incoordinate', 'to and fro' pattern can be regarded as evidence of DES[1].

Current interest in oesophageal motility disorders has arisen because of the role these disorders may play in non-cardiac chest pains. A significant proportion of patients with angiographically normal coronary arteries have oesophageal dysmotility[2]. These include diffuse oesophageal spasm, nutcracker oesophagus and hypertensive lower oesophageal sphincter. These abnormalities appear to be present in higher prevalence compared to normal subjects and it is highly suggestive that oesophageal dysmotility is the cause of the chest pain. With the advent of continuous ambulatory oesophageal motility recording where correlation of chest pains with abnormal motility tracings is possible, the aetiological role of oesophageal motility disorders in non-cardiac chest pains may be better established.

CONCLUSION

The ten cases illustrate that patients who complain of obscure central chest pain and/or unexplained dysphagia can be due to oesophageal motility disorders. There should be awareness of the potential of the oesophagus to cause chest pain that mimic cardiac pain in view of the proximity of the two organs, and a shared innervation.

The investigation of patients with central chest pain and/or dysphagia that may be due to oesophageal motility disorders should commence with endoscopy to exclude mechanical obstruction especially of malignant aetiology. If no abnormality is detected manometry is required, and if not available, RT study is the best alternative. Barium swallow is too insensitive to be recommended except when endoscopy is not available to exclude mechanical obstruction. If manometry is normal, RT studies should still be done as it may detect abnormality not detected by the former. In patients with central angina-like chest pain, exclusion of coronary artery disease should begin with non-invasive tests like stress electrocardiography, exercise 201 Thallium myocardial scintigraphy and exercise radionuclide ventriculography. A negative thallium study excludes functionally significant coronary artery disease and coronary angiography need not be done. RT instead of manometry can be used to follow up and in assessing the response to medical or surgical therapy of dysmotility disorders non-invasively.

ACKNOWLEDGEMENTS

The authors wish to thank Puan Latifah for secretarial help.

REFERENCES

1. DE Caestecker JS, Blackwell JN, Adam RD, Hannan WJ, Brown J, Heading RC. Clinical value of radionuclide oesophageal transit measurement. Gut 1986; 27:659-6
2. Davies HA, Rhodes J. How often does the gut cause anginal pain? Acta Med Scand 1981; Suppl 644:62-5
3. Russell COH, Hilt LD, Holmes ER, Hull DA, Gannon R, Pope CE. Radionuclide transit: A sensitive screening test for oesophageal dysfunction. Gastroenterology 1981; 80: 887-92
4. Holloway RH, Krosin G, Lange RC, Bahe AE, McCallum RW. Radionuclide oesophageal emptying of a solid meal to quantitate results of therapy in achalasia. Gastroenterology 1983; 84:771-6
5. Blackwell JN, Hannan WJ, Adam RD, Heading RC. Radionuclide transit studies in the detection of oesophageal dysmotility. Gut 1983; 24:421-6
6. Richter HE, Blackwell JN, Wu WC, Cowan RJ, Johns DN, Castell DO. Assessment of liquid bolus transit (LET) by simultaneous radionuclide transit and oesophageal manometry. Gastroenterology 1983; 84:1285
7. Mughal MM, Marples M, Bancewicz J. Scintigraphic assessment of oesophageal motility: What does it show and how reliable is it? Gut 1986; 27:946-53
8. Kjellen G, Svedberg JB, Tibbling L. Solid bolus transit by oesophageal scintigraphy in patients with dysphagia and normal manometry and radiography. Dig Dis Sci 1984; 29:1-5
9. Blackwell JN, Richter JE, Wu WC, Cowan RJ, Castell DO. Oesophageal radionuclide transit test: potential false positive results. Gastroenterology 1983; 84:1108
10. Nelson JL, Wu WC, Richter JE, Blackwell JN, Johns DN, Castell DO. What is normal oesophageal motility? Gastroenterology 1983; 84:1258