Today's Treatment

Diseases of the alimentary system

Dysphagia

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British Medical Journal, 1977, 1, 91-93

Dysphagia must always be taken seriously, not only because it usually indicates serious disease but because in itself it often has dire consequences. As a symptom, at the best it is an embarrassment that detracts from the pleasures of the table and may disrupt social life for fear of eating in public; at the worst it threatens existence by causing starvation or respiratory complications. Although life may be maintained for varying periods by feeding either intravenously or through a gastrostomy or jejunostomy, in the long term these methods are poor substitutes for being able to swallow one's food. The rational management of dysphagia must be based on a proper understanding of the nature of the derangement that is present and of the possible consequences of being unable to swallow.

Mechanism of dysphagia

Swallowing is a complex reflex, the initial stages of which are under voluntary control; hence it is absent in the unconscious patient. The reflex is initiated by compressing the bolus between the tongue and the hard palate, thus pushing solids into the pharynx and squirting liquid down into the lower oesophagus. Difficulty in initiating swallowing may result from paralysis of the tongue muscles, palatal damage, and painful lesions in the mouth and throat including those after traumas or surgery in this area.

Weakness of the pharyngeal constrictor muscles retards the rate of passage of the bolus through the throat, and ineffective closing off of the larynx and nasopharynx allows some to enter the respiratory tree causing coughing and choking or regurgitation of fluids through the nose. The constrictor muscles receive their motor supply from the nucleus ambiguus, damage to which from brain stem ischaemia, poliomyelitis, motor neurone disease, or syringobulbia causes pharyngeal dysphagia.

Upper oesophagitis, which may or may not be associated with web formation, occurs because iron deficiency causes atrophic changes in the mucosa, which make it vulnerable to minor trauma from food. As with peptic oesophagitis in the lower oesophagus, mucosal irritation leads to spasm of the underlying oesophageal muscle that obstructs the passage of the bolus.

Loss of oesophageal peristalsis occurs with advancing years and in systemic sclerosis, but dysphagia from this cause is

usually mild provided the patient swallows when sitting or standing, when gravity helps transit. Under these conditions liquids pass readily down the oesophagus and when peristalsis is weak or absent dysphagia usually occurs only with solid foods. Radiological examination shows that swallowed contrast medium remains in the oesophagus for minutes or even hours when the patient is examined in the recumbent or 15° head down position. Effective peristalsis may also be disrupted by neuromuscular incoordination, as in oesophageal spasm or achalasia. Here there is no organic obstruction, but the failure of the lower oesophageal sphincter or of the oesophagus to relax when the bolus arrives coupled with the absence of co-ordinated propulsion higher in the gullet causes dysphagia. Fortunately the upper oesophageal sphincter (cricopharyngeus) is unaffected in these disorders and protects against regurgitation of retained oesophageal contents into the pharynx and respiratory tree.

Mechanically obstructive lesions such as neoplasm or peptic stricture occurring anywhere between the throat and the upper part of the stomach cause dysphagia. The degree of hold-up depends not only on the diameter of the residual lumen but also on the force and co-ordination of peristalsis higher in the oesophagus. In disorders such as systemic sclerosis and in old age peristalsis may be weak or lacking and so a peptic stricture or neoplasm may cause severe dysphagia for solids before luminal narrowing is gross. The storage capacity of the oesophagus above an obstructing lesion in its lower part is usually sufficient to prevent choking on the first swallow and a sense of hold-up deters the patient from taking more food.

Consequences of dysphagia

STARVATION

The body needs a constant source of energy to support its metabolic processes and to maintain the life of its cells. Food intake provides this energy source and in its absence body fat reserves are used to provide metabolic fuel in the form of free fatty acids and ketones. Glucose for metabolism, however, is mainly provided by the breakdown of body protein, particularly that of muscle, and administered glucose has a considerable protein sparing effect. Surgical operations and radiotherapy increase body protein breakdown and thus accentuate the effects of starvation. On the other hand, correction of under-nutrition enables the patient to tolerate more easily these procedures that may be necessary to deal with the cause of his dysphagia. Starvation impairs wound healing, reduces resistance to infections, and depresses immune responsiveness, all of which increase morbidity and mortality.

If immediate relief of dysphagia is impossible, measures to correct under-nutrition should be instituted promptly. If a

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nasogastric tube can be passed feeding by this means is preferable to intravenous feeding. Homogenised ward food may be used, but more accurate control of the patient's intake of calories, nitrogen, and electrolytes may be obtained by using a specially made feed consisting of a glucose polymer (Caloreen) 600 g, a beef serum hydrolysate (Albumaid) 60 g, 1 egg yolk to provide essential fatty acids, plus vitamins and minerals made up to 1500 ml of water and given over 24 hours. Appropriate adjustment to the composition of the feed must be made in the presence of hypercatabolism or electrolyte imbalance. Intravenous feeding combining an amino-acid solution, a fat emulsion, and fructose, glucose, sorbitol, or ethanol to provide calories is needed only in patients in whom nasogastric feeding is impracticable because of difficulty in passing the tube or associated disturbance of gastrointestinal function. If the problem persists, such patients in the longer term need a feeding gastrotomy or jejunostomy, but fortunately these procedures are now rarely used.

RESPIRATORY COMPLICATIONS

Disorder of the pharyngeal phase of swallowing often causes food and liquids to enter the respiratory tract; when the hold-up is in the middle or lower oesophagus regurgitation of oesophageal content is particularly likely to occur at night causing staining of the pillow or during the induction of anaesthesia. In the elderly patient after mild regurgitation has continued for some time the larynx becomes less sensitive to foreign material, and at radiological examination barium contrast medium may enter the larynx and trachea without causing undue distress.

Acute massive aspiration of oesophageal contents is often fatal, although lacking gastric juice and bile the aspirated material is less irritant than vomit. The risk of aspiration during anaesthesia may be reduced by allowing only clear fluids by mouth for several days beforehand, by aspirating the oesophageal contents while the patient is still conscious, and by the rapid insertion of a cuffed endotracheal tube after induction.

Chronic aspiration is much commoner and leads to aspiration pneumonitis, bronchiectasis, and pulmonary fibrosis. Chronic cough, wheezing, and increased breathlessness on exertion are the presenting symptoms and on occasion may overshadow those of the underlying lesion. Aspiration pneumonitis appears in the chest x-ray picture as a patchy shadowing usually in the mid-zones, and it is more frequent in the right lung. Mycobacteria derived from food may appear in the sputum and lead to a mistaken diagnosis of tuberculosis. Lung abscess is a serious but fortunately now uncommon complication. The best preventative for aspiration pneumonitis is effective treatment of the underlying cause of dysphagia, but the risk may be diminished by avoiding meals for three hours before retiring and by sleeping with the head of the bed raised on 10-cm blocks.

CARCINOMA OF THE OESOPHAGUS

Carcinoma of the oesophagus occurs seven times more often in patients with longstanding achalasia than in the general population. Probably stasis and consequent oesophagitis are the major factors accounting for this association. There is an increased incidence of postcricoid and upper oesophageal carcinoma in sideropenic dysphagia.

Management of dysphagia

UNCONSCIOUSNESS

Injudicious attempts at feeding the comatose patient by mouth may lead to respiratory complications, and those who remain unconscious for long period, as for example after brain damage, should be fed by a nasogastric route.

OROPHARYNGEAL DISEASE

Dryness of the mouth or oropharyngeal infections make swallowing difficult. Lack of saliva in Sjögren's syndrome (keratoconjunctivitis sicca) may cause mild difficulty in swallowing and can usually be helped by taking plenty of liquids with meals and by sucking acid drops. Stomatitis caused by monilial infections, herpes simplex, Stevens-Johnson syndrome, and throat infections may cause severe dysphagia sufficient to result in fluid imbalance. The treatment here is that of the specific infection. Extensive aphthous ulceration can make eating painful, and the topical application of a local anaesthetic (benzocaine lozenges *BPC*) or triamcinolone in oral paste (Adcortyl in Orabase) before meals is of value. Dysphagia due to pharyngeal pouch or postcricoid carcinoma is usually relieved by appropriate treatment of its cause, but if the resulting nutritional disturbance is great preliminary nasogastric feeding is worth while.

NEUROMUSCULAR DISORDERS OF THE PHARYNX

Although nasogastric feeding is sometimes needed in the early stages of the lateral medullary syndrome, sufficient recovery usually ensues for this to be discontinued after a week or two. Dysphagia in chronic bulbar palsy and dystrophia myotonica is progressive and difficult to manage; aspiration into the respiratory tract is common in the later stages and may necessitate tracheostomy. Dysphagia in myasthenia gravis usually responds dramatically to neostigmine, which should be given before meals.

SIDEROPENIC DYSPHAGIA

Repletion of the body's iron stores alleviates sideropenic dysphagia, but added anxiety is often an aggravating factor and reassurance and tranquillising drugs are then helpful in improving swallowing.

ACHALASIA

There is no way in which normal function can be restored to the achalasic oesophagus, and treatment aims at disrupting the circular muscle fibres of the lower oesophageal sphincter by either cardiomyotomy or bag dilatation. Bouginage has no place in managing this disorder because its effects are transient. Effective treatment of achalasia not only restores swallowing to near normal and so improves nutrition but by abolishing oesophageal stasis removes the risk of respiratory complications. Whether effective treatment diminishes the likelihood of carcinoma developing is uncertain, and this complication may arise after successful cardiomyotomy.

Cardiomyotomy is the treatment of choice for achalasia, and most surgeons prefer a transthoracic approach and an incision through the muscle coat to expose the mucosa carried upwards for 8 to 10 cm from just distal to the cardia. A good result is obtained in 80 to 90% of patients, and complications are few, although in a minority gastro-oesophageal reflux is a troublesome sequel. The operation is advisable even in patients with mild or moderate dysphagia and minimal weight loss, because of the likelihood of the condition progressing and the risks of respiratory complications.

Bag dilatation gives a less permanent cure and is best reserved for those patients who are unfit for operation on grounds of age or other disability. If the disease has progressed to the stage of causing advance starvation then bag dilatation may be useful as a temporary measure to restore satisfactory swallowing and so enable nutrition to be improved before undertaking cardiomyotomy. A mechanical dilator (Starck), or hydrostatic (Plummer) or pneumatic (Rieder Moeller) bags, may be used

and they are best introduced along a guidewire threaded through the cardia under direct fibre-endoscopic vision checking the final position of the bag radiologically. Distension to a pressure of 250 mm Hg for two minutes is adequate to rupture many of the circular muscle fibres of the lower oesophageal sphincter. Good results may be expected in 60% of patients, but the procedure often has to be repeated several times.

Although these procedures open up the cardia, they do not restore normal oesophageal peristalsis. Transit through the oesophagus is largely dependent upon gravity and swallowing in recumbency causes oesophageal stasis.

PEPTIC OESOPHAGEAL STRICTURE

Treatment of peptic oesophageal stricture has two objectives: the first to dilate the stricture and so relieve dysphagia, and the second to control gastro-oesophageal reflux and so prevent the stricture reforming.

Dilatation is best done using either solid bougies pushed down the lumen of the rigid oesophagoscope or olive dilators mounted on a flexible carrier and passed over a guiding string or wire that has been inserted through the stricture. Peptic strictures can conveniently be dilated using fiberoptic endoscopy and the Eder-Puestow olive dilators. Under direct vision a guidewire is inserted through the stricture into the stomach. The wire may be difficult to pass in patients with much distortion and angulation of the strictured area, and also it may be held up at diaphragmatic level in those with hiatal hernias. Radiological screening using a mobile image intensifier is helpful in these cases.

When the wire is in position the scope is removed, and olive dilators of increasing size mounted on a flexible carrier are passed over it and through the stricture up to a No 45 French (14.5 mm diameter) olive. The wire is then removed and the scope reinserted, when it will usually pass through the dilated stricture allowing full visualisation of its whole extent and allowing adequate biopsy specimens to be taken. This is important because neoplastic strictures may closely resemble peptic strictures in their radiological and endoscopic appearances. A careful endoscopic search of the stomach, pylorus, and first and second parts of the duodenum is made to detect associated peptic ulcer or fibrotic narrowing that may be aggravating gastro-oesophageal reflux. The examination can be done under local anaesthesia using intravenous diazepam supplemented by pethidine if the patient develops pain. General anaesthesia using a cuffed endotracheal tube is preferable in children, uncooperative patients, and those with tight strictures or considerable food residues in the oesophagus. When this method is used perforation and mediastinitis occur in under 2% of dilatations.

The frequency with which oesophageal dilatation has to be repeated varies greatly from patient to patient and is largely determined by the efficacy of treatment to control reflux. Since most peptic strictures occur in the elderly, the reflux is usually treated medically and this comprises sleeping with the head of the bed raised on 10-cm blocks, regular antacids combined with dimethicone (Asilone Gel) and metoclopramide to improve oesophageal clearance together with getting the patient to lose weight and stop smoking. Control of gastric secretion with the H_2 antagonist cimetidine offers hope of reducing oesophagitis and restructuring. Careful mastication is important in the presence of oesophageal narrowing, and some elderly edentulous patients prefer to homogenise their food.

Surgical measures to control reflux should be considered in younger patients with symptoms that have not responded to medical measures. Repair of reducible hiatal hernia with tightening of the hiatus, or a gastropasty type of operation for a fixed hiatal hernia, often substantially diminishes the need for further dilatation.

NEOPLASTIC DYSPHAGIA

Curative resection is the best means of treating carcinoma of the oesophagus or upper stomach causing dysphagia. In many patients, however, this is contraindicated by the presence of metastases or by the patient's poor general health, and palliative intubation with the Mousseau-Barbin or Celestin tube offers the only means of relieving distressing dysphagia and improving nutrition. In the past such intubation has been done by laparotomy and pull-through. This carries an immediate mortality of 25%, and oral intubation under endoscopic control offers the advantages of relative safety and simplicity. The tube can be slid down the rigid oesophagoscope over a filiform bougie passed through the stricture. Today, however, fiberoptic endoscopic placement offers the most satisfactory method. After dilatation of the narrowing using the Eder-Puestow dilators in the same way as for peptic stricture, a Celestin tube cut to appropriate length and mounted on an introducer is passed along the Puestow guidewire until its position is satisfactory as judged by radiological screening. Most patients can take a semisolid diet within two days and can be discharged from hospital often on the third day. Particular attention must be given to careful mastication, and meals should be taken with fizzy drinks that help to prevent blockage of the tube.

Nutritional improvement often follows successful intubation, and squamous growths can be treated by radiotherapy with the tube in position. Rarely the improvement in their general condition may be such that surgical resection of the growth may be reconsidered in patients previously too weak to withstand operation.

What are the criteria for diagnosing diabetes mellitus?

Diabetes mellitus is a syndrome, not a single entity, the central feature of which is a raised blood sugar concentration much as a low haemoglobin concentration characterises anaemia. The diagnosis is self-evident in those patients with typical symptoms—heavy glycosuria and a random blood sugar of more than 200 mg/dl. It is at the other end of the diabetic spectrum that diagnostic difficulties arise—for instance, asymptomatic patients with a trace of glycosuria or minor rises in blood sugar, often discovered on routine biochemical screening. In these patients the oral glucose tolerance test is still the cornerstone of diagnosis. The oral glucose load normally given to adults in Europe is 50 g, but 75 g, 100 g, and 1.75 g/kg body weight are used elsewhere. Blood sugar levels are measured at timed intervals after ingestion. The establishment of diagnostic cut-off points is entirely arbitrary, as there is no clear dividing line between normal and abnormal values. In Britain the criteria recommended by the Medical and Scientific Section of the British Diabetic Association are used. The test is considered diabetic if, after a 50 g glucose load, the venous

blood glucose concentration is 110 mg/dl or more at two hours and 160 mg/dl or more at some other time point in the test. In the USA an oral load of 1.75 g glucose/kg ideal body weight is commonly given. The test is defined as abnormal if the one-hour venous blood glucose value was over 160 mg/dl, the 90-minute value over 140 mg/dl, and the two-hour value over 120 mg/dl. The use of plasma or serum rather than whole blood and the selection of the biochemical method to measure glucose alters the above values slightly. The oral glucose tolerance test is a relatively crude investigation but arguably still the best available. More complex tests have failed to give consistently better diagnostic discrimination. Finally, testing urine for sugar is a procedure commonly used to screen large populations for diabetes. Yet, dependence on glycosuria alone for diagnosis is notoriously unreliable as it may be absent in mild disease and diabetic nephropathy. Patients with minor degrees of glucose intolerance are just as liable to long-term diabetic sequelae as those with overt disease.

Keen, H, *Hospital Medicine*, 1968, 2, 760.