THE NATURE AND TREATMENT OF PLUMMER-VINSON DYSPHAGIA

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This syndrome of dysphagia of upper oesophageal type associated with present or past anaemia is relatively common in the North of Ireland. Treatment by dilatation is very satisfactory, but the condition is unfortunately often misdiagnosed as hysteria or "nervous swallow." Indeed, Vinson in 1922 put forward the view that the dysphagia was hysterical. We have been impressed by the diagnostic difficulty the condition evidently offers and by the consequent prolonged physical and social distress the patient suffers. That the condition is due to an organic stricture and that relief may be obtained by dilatation have been pointed out by McGibbon (1935), but these observations have not become widely known. This account is based on 16 cases observed in the last five years. Details of these cases are given in the Table.

Clinical Features

The clinical features have often been described, among others by McKenzie (1918), Brown Kelly (1919, 1927), Paterson (1919), and Munro Cameron (1928, 1929). The onset is insidious and the course lasts many years. There is no tendency to spontaneous cure. The social class and

the level of intelligence are below average. Their lack of understanding often makes it difficult to persuade these patients to be treated (19% of our patients offered dilatation refused). The condition occurs only in women, and usually begins in the child-bearing years. There always is or has been in the past an iron-deficiency anaemia of long standing; and associated with this in varying degree are the usual features of smooth tongue, splenomegaly, spoon-nails, and angular stomatitis. Very acute orificial stomatitis is not common. The pallor often has a yellowish tinge, and is due as much to change in skin pigmentation as to low haemoglobin. Courses of iron produce no permanent improvement in the anaemia if not repeated twice a year. If the patient has been treated energetically with iron the anaemia may be mild and there may be no glossitis or stomatitis.

Some cases may be seen after the menopause, when improvement in the anaemia has occurred and only the dysphagia remains. The E.S.R. is sometimes inexplicably high (Cases 2, 5, and 6). Emaciation is rare, though some patients put on considerable weight after dilatation. Since all usual foods except meat can be reduced to a pulp by cooking processes or by chewing, these patients can usually take enough calories. It is almost invariable that these people have not been able to eat meat for years and that their diet shows a selective meat deficiency.

Morbid Anatomy and Oesophagoscopic Appearances

The dysphagia is due to organic stenosis in the upper oesophagus at or just distal to the introitus of the oesophagus. The site of the obstruction is either at the level of the crico-pharyngeal sphincter or immediately beyond it. When it is at the level of the sphincter the mouth of the oesophagus does not open in front of the advancing oesophagoscope enough to allow it to pass, the lumen at the level of stricture being sometimes only a few millimetres in diameter. Sometimes the lumen appears in the middle of a diaphragm-like obstruction. Often some degree of stenosis extends for

Details of Cases

Case	Age	Haemoglobin %	E.S.R.	Spleen	Oesophagus		Duration of Dysphagia	Menses	Cancer	Stricture Dilated	Result	Remarks
					Endoscopy	X-ray						
1	40	100	—	+	Tight introital stricture	Cuffing and web	20 years	Hysterectomy at 38	Nil	+	Good	Mother said to have died of pernicious anaemia. Sister said to have P.A.
2	56	100	34	—	Introital stricture and web	Cuffing and two webs	6 "	Ceased	"	+	"	Fat absorption normal. Needs dilating yearly
3	60	58	10	+	Web	Web	Many "	"	"	+	"	Gastro-enterostomy years ago
4	37	87	0	+	Stricture at 15 cm. from upper teeth	Unsatisfactory because of choking	10 "	No information	Squamous epithelioma 1 year after dilatation	+	Died	Benign stricture at first examination. 1 year later cauliflower carcinoma as well
5	64	100	24	+	Refused	" "	Many "	Ceased	Nil	Refused	No change	Lives on semi-fluids
6	46	64	20	—	Diaphragm	Web "	5 "	Regular	"	+	Good	Hypertension. Haemoglobin remains low
7	65	74	—	—	Introital stricture	"	"Always"	Ceased	"	+	"	"
8	49	95	10	—	Introital stricture. Squamous carcinoma at 22 cm.	Carcinoma	Uncertain	"	+	+	Died	Clinical typical Plummer-Vinson disease. Exacerbation of dysphagia and x-ray diagnosis of carcinoma
9	77	86	2	—	Refused	No study	"	"	Nil	Refused	No change	Grossly deficient diet. Stated to have P.A.; on vitamin B ₁₂ . Acute orificial stomatitis
10	48	56	17	—	Web	" "	8 years	Regular	"	+	Good	Haemoglobin remains low
11	41	70	7	+	Introital stenosis	Web "	1 year	"	"	+	"	Has also a papilloma of a vocal cord
12	48	60	8	—	Refused	No study	9 months	Nil	"	Refused	No change	Always bloodless
13	65	Normal	—	—	Not offered	Web	Years	"	"	Not offered. Can eat meat with care	" "	Anxiety state. Atheromatous vascular disease
14	58	75	—	—	" "	"	"	"	"	" "	" "	Angina of effort. Gastro-enterostomy in 1914. Severe iron-deficiency anaemia from 1932. Glossitis + +. Occult blood test negative
15	73	46	13	+	" "	No study	"	Uterine bleeding	"	Not offered	" "	Bleeding uterine fibroid for many years. Hysterectomy. Pathological diagnosis carcinoma of body of uterus
16	42	49	17	—	Awaiting dilatation	" "	2 years	Regular. Heavy	"	Awaiting	" "	Active mucosal changes in mouth and on lips. Chokes on meat and bacon

a varying distance, up to a centimetre or so, along the oesophagus. In a few cases one is able to introduce the oesophagoscope through the sphincter and then almost immediately beyond this is seen a web of mucosa, occluding perhaps two-thirds of the lumen and ending in a free crescentic edge. There may be two webs (Case 2). However, it is our impression from endoscopic examination that web formation is less often the cause of the obstruction than would appear from x-ray examination. We think that the x-ray appearance of a web may often be due to viewing from the lateral aspect a diaphragm-like obstruction with an eccentric opening in it. No oesophagitis or areas of ulceration have been seen in these cases.

It is emphasized again that there is an anatomical narrowing to cause the dysphagia. There is no muscular spasm or achalasia. It should be noted that a narrowing which lower in the oesophagus would be asymptomatic will at the introitus cause severe dysphagia.

The Dysphagia

Because of the site of the narrowing at or just beyond the introitus the dysphagic difficulty consists in the patient not being able to move food from the hypopharynx into the oesophagus. It is this feature that differentiates this dysphagia from lower oesophageal dysphagia. The bolus mills about in the hypopharynx on the very verge of the glottis. The patient, anxious, even desperate, makes repeated and ineffective attempts to swallow—ineffective because to make an effective attempt would drive the food into the larynx. The sensation of being about to choke, of something about to go the wrong way, is instant. Drinking something to help is impossible—and useless. Eventually the patient, unable to swallow and about to choke, with much unpleasant hawking and spitting, brings up the food mouthful. This spitting out at the table is as characteristic of Plummer-Vinson dysphagia as vomiting or the emptying of a full oesophagus after a meal is typical of a low oesophageal obstruction. Note that in Plummer-Vinson disease there is no accumulation or retention of food or fluid. The anxiety, the violent hawking up, the complaint of choking, the unwillingness of the patient to swallow effectively enough to satisfy the onlookers, lend themselves to a diagnosis of hysteria—as readily made by the physician as by the family. Further features of the dysphagia are the slowness of eating, and the occasional completion of the obstruction—for example, by a pea (Case 6). This is not a painful dysphagia, unless carcinoma has supervened.

There should be little difficulty in distinguishing this disease on clinical grounds from neurological, hysterical, or anxiety dysphagia. Endoscopy clinches the diagnosis. A negative barium swallow is valueless in excluding it.

Complications

The most important complication is the onset of carcinoma, usually in the upper oesophagus but sometimes in the lower oesophagus or in the stomach. The syndrome is the precursor of post-cricoid carcinoma (Simpson, 1939), and the prophylaxis of the syndrome should prevent these cancers occurring. The onset of carcinoma is marked by hoarseness, painful swallowing, and increasing obstruction. Inability to swallow saliva may be the first sign of cancerous change.

Rejection by the family may be serious. Sometimes (Cases 1 and 10) the patient has eaten for years in a separate corner because her family could not bear to have her at the table with them. In Case 1 there was marital estrangement and delinquency in the children.

Lung complications are rare: Case 2 had a lung collapse some years before diagnosis, presumably due to spill-over.

Management

The basic and only essential requirement in treatment of this dysphagia is dilatation of the stenosis. This is easily and satisfactorily accomplished. It can be done, if neces-

sary, with only local analgesia and without admission to the ward. It should be done under direct vision through an oesophagoscope and not by blind dilatation, both because this would be difficult to carry out in cases of stricture at the entrance to the oesophagus and because it is advisable to exclude the presence of a carcinoma. Hurst's mercury bougies are unsuitable.

Before the oesophagoscope can be passed through the stenosed area the stricture almost always has to be dilated with gum-elastic bougies, beginning with one of the smaller sizes and working up to as large a size as can be passed down the instrument. It is then possible to use the oesophagoscope itself as a dilator by passing it through the stricture. The rest of the oesophagus is then examined to exclude carcinoma lower down. An oesophagoscope 4.6 cm. in circumference has been used in this condition. It would in most cases be difficult and probably dangerous to attempt to dilate the stricture enough to allow a large instrument to pass.

The role of radiology is to exclude cancer lower down and in the stomach. Demonstration of the stenosis is possible with special care, but will often be missed at a routine barium swallow. There are some important points in visualizing the stricture with the x-ray screen. Barium suspensions of creamy consistency are used. For obvious reasons, thick barium cannot be used in these patients, and thin barium passes too rapidly for visualization. The patient should be turned into the left oblique position till the air-filled pharynx and trachea and the retrotracheal space clear the spine. The observer must fix his gaze on the upper inch (2.5 cm.) of the oesophagus. This is roughly $1\frac{1}{2}$ in. (3.8 cm.) below the point where the pharynx ceases to be air-containing—that is, below the entrance to the larynx. If the patient then swallows a mouthful of barium the abnormality will easily be seen for as long as three to four seconds, but no more. Most cases show a general narrowing at the beginning of the oesophagus which is pronounced enough in some

FIG. 1.—Case 2. Showing two "webs" at either end of a cuff-like stenosis about 1 cm. in length, and hypopharyngeal bulging above the obstruction.

FIG. 2.—Case 1. Showing a web appearance anteriorly at the level of the crico-pharyngeal bulge, which is well seen posteriorly. The "web" is at the level of the fifth cervical vertebral body.

FIG. 3.—Case 1. Showing web appearance particularly well.

severe cases to produce a cuff-like appearance (Fig. 1). In addition most cases when viewed laterally show the well-known web appearance (Figs. 2 and 3).

For reasons of safety, gastroscopy is not undertaken, though the condition of the gastric mucosa is of great theoretical interest. Gastroscopists should beware of women in this age group, since they may have an undeclared stenosis of this kind.

Follow-up should be directed to securing a permanently normal haemoglobin, and redilatation should the stenosis recur. In the worst cases a surgical or radium menopause may be necessary if oral iron cannot keep the blood normal. Frequent endoscopy to detect early cancerous change is impractical and of doubtful value.

If the anaemia is gross it should be corrected before dilatation. However, no quantity of iron will correct the dysphagia. If in doubt whether to dilate or not it is useful to remember that inability to eat meat is an absolute indication for the procedure.

Prognosis and Causation

Redilatation may be needed as often as every year (Case 2). Repeated courses of iron will be necessary till the menopause occurs. Two of our 16 cases developed carcinoma (Cases 4 and 8). Simpson (1939) reported that 10 of his 18 cases developed carcinoma.

The cause of mucosal atrophy in the upper gastrointestinal tract, the relationship of this to hypochromic anaemia, and the relationship of both to the oesophageal stenosis are quite unknown. Whatever the condition of the gastro-intestinal tract, it seems clear that the dysphagia does not occur unless iron-deficiency anaemia has been present. Despite superficial resemblances there seems to be no relationship to ariboflavinosis. The exclusive incidence in women is sufficiently accounted for by their peculiar liability to menstrual blood-loss anaemia. The incidence of hypochromic anaemia in Northern Ireland is considerable, and is to be attributed to the inadequate nutrition common in middle-aged women in Northern Ireland and to uncorrected menorrhagia. The implications for prophylaxis of post-cricoid carcinoma by improved nutrition and effective treatment of menorrhagia are obvious.

Summary

The clinical diagnosis of Plummer-Vinson disease is described. The dysphagia is due to organic stenosis. Dilatation of the stricture is the essential treatment. Plummer-Vinson disease is a pre-cancerous condition.

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A guide to epileptic colonies, the accommodation they offer, their charges, training, etc., has been published by the British Epilepsy Association (price 1s.). Although only sane epileptics are admitted to the colonies, the standards of subnormality for exclusion vary. The handbook gives details for each colony of the method of admission and administration, conditions for discharge of patients, and the length of the waiting-list. The booklet points out that, while the provision of a permanent sheltered home remains an important function of these colonies, the main object of care is to stabilize the patient sufficiently to permit return to work.

ANTI-SIALOGOGUE AND OTHER EFFECTS OF ATROPINE MUCATE

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Although it is barely 50 years since atropine began to be used to inhibit salivary secretion before the administration of an anaesthetic, this drug has come to be regarded as an essential requirement in these circumstances. Pre-operative administration of atropine is so important that if it has been omitted most anaesthetists would either postpone the operation or administer atropine intravenously.

In big hospitals with long operating lists it is usually impossible to estimate the correct time for the administration of atropine. Any preparation of this drug whose effect would be so prolonged that accurate timing was unnecessary would be an important and welcome innovation. We regarded it as of some clinical importance, therefore, to investigate further the claim made by Asquith and Thomas (1948) that hypodermic injections of atropine mucate ("hyperduric atropine") lead to a more prolonged inhibition of salivary and mucus secretion than do the more commonly used atropine sulphate or tartrate.

Asquith and Thomas in their paper relied on clinical observation and apparently used no criterion for assessing the anti-sialogogue effect, other than their own or their patients' opinion. We decided to use a more objective and quantitative method of estimation, in volunteers in whom many environmental factors could be controlled.

The only published method we found for studying salivary secretion was that of Lashley (1916), and its modification by Richter and Wada (1923). In this method the parotid duct is cannulated and the saliva allowed to flow through a long glass tube. The passage of a bubble of air introduced into the stream of saliva is timed over a measured distance. This method was deemed unsuitable for an experiment requiring frequent observations over a period of perhaps six hours. We considered and discarded the suitability of other methods of cannulation of one or more salivary ducts. Electrical measurement of the wetness of the mucosa of the mouth was unsatisfactory, and so was weighing pledgets of wool allowed to soak saliva for a fixed time.

Method

We finally devised the following method for measuring the salivary activity at any particular moment. The subject swallows repeatedly, to get rid of any saliva present in the mouth. Four drops of lemon juice (a powerful if not a specific stimulus) are then placed on the tongue and allowed to act for 30 seconds. The mouth is then rinsed for 15 seconds with 5 ml. of water, the subject then spitting into a funnel and measuring cylinder. For a further 15 seconds the mouth is emptied by repeatedly spitting into the measur-