

of delivery was calculated from the two readings available. The cases were then grouped according to the number of weeks that elapsed between examination and delivery, and an average of each group was calculated. These growth figures are given in Table III. The standard error of the means of these groups is ± 0.02 in.

TABLE III.—Average Growth of Biparietal Diameter from Date of Examination to Date of Delivery

Week of Pregnancy	Days Before Delivery	No. of Cases	Average Growth
40	Same day	13	-0.008 in. (-0.203 mm.)
39	1-7	71	-0.022 in. (-0.559 mm.)
38	8-14	45	-0.024 in. (-0.609 mm.)
37	15-21	25	-0.024 in. (-0.609 mm.)
36	22-28	36	-0.005 in. (-0.127 mm.)
35	29-35	20	+0.042 in. (+0.067 mm.)
34	36-42	9	+0.078 in. (+1.981 mm.)
33	43-49	1	+0.150 in. (+3.810 mm.)
32	50-56	1	+0.250 in. (+6.350 mm.)
31	—	—	—
30	64-70	1	+0.300 in. (+7.620 mm.)
29	71-77	1	+0.400 in. (+10.160 mm.)

The notable features of this table are: (1) The radiological method overestimates the size of the head by approximately 0.02 in. Consequently there is an apparent slight negative growth in the later stages (see Fig. 5). (2) There is no sign of a growth curve between the 36th and 40th weeks of pregnancy. (3) The growth curve between the 30th and 36th weeks cannot yet be accurately assessed, owing to scanty data, but even at this stage the biparietal diameter appears to grow at a rate of less than 0.1 in. a week.

We have now collected 34 cases in which radiological cephalometry was performed twice, with an interval of 21 days or more between the examinations. In 13 cases both radiological examinations were performed during the last 28 days of pregnancy, and the apparent growth between the two radiological examinations was 0.02 in. This figure is within the limits of our experimental error and could not therefore be definitely attributed to growth. Further details of these 34 cases are given in Table IV.

TABLE IV.—Results of Two Radiological Examinations in Same Patient

Time from First Radiological Examination to Date of Delivery	No. of Cases	Average Time between Two Radiological Examinations	Average Growth between Two Radiological Examinations
28 days or less	13	24 days	0.02 in. (0.508 mm.)
29-35 days	13	26 "	0.04 in. (1.016 mm.)
36 days or more	8	30 "	0.12 in. (3.048 mm.)

For reasons stated above we have estimated radiologically only the biparietal diameters, and have no observations to offer on the growth, or lack of growth, of the other diameters of the foetal skull during this period. In the absence of craniostenosis, however, it seems illogical to suppose that one diameter of the foetal skull should remain stationary while the others increase.

The investigation continues, and we are trying to estimate the foetal biparietal diameters at 32 weeks, again at 36 weeks, and then at term. Thereby we hope to observe the rate of growth between the 32nd and 36th weeks of pregnancy and to obtain further confirmation of the absence of growth between the 36th and 40th weeks.

Summary

Two hundred and twenty-three estimations of intrauterine foetal biparietal diameters, performed on 189 patients, are compared with calliper measurements obtained on the third day after birth.

Eighty-four cases were estimated radiologically within seven days of delivery.

In 34 cases radiological cephalometry was performed twice, with a minimum of 21 days between the examinations.

For individual cases the predicted measurement will be within 0.1 in. of the calliper measurement in 75% of cases, and within 0.15 in. in 95% of cases.

For groups of over 16 cases, accuracy of 0.02 in. may be claimed for the mean reading.

Radiological cephalometry overestimates the biparietal diameters by approximately 0.02 in.

There is no sign of a growth curve between the 36th and 40th weeks of pregnancy.

Grateful thanks are due to the staffs of the obstetrical and radiological departments of the Newcastle General Hospital; to Mr. F. Pearse, senior radiographer, who produced many of the radiographs of these cases; to Dr. Mary Buchanan and Dr. W. Ingham, who assisted Dr. Fawcett in her measurements; to Dr. Whately Davidson, radiologist in charge, Newcastle General Hospital, Mr. Linton Snaithe, obstetrician in charge, Newcastle General Hospital, and Dr. Blair Hartley, honorary radiologist to the Christie Hospital and Holt Radium Institute, Manchester, for their criticism and encouragement in this work.

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THE SHORT OESOPHAGUS

A REVIEW OF 31 CASES

BY

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Although the association of gastric hernia with shortening of the oesophagus is no new conception, it is far from widely appreciated that it can produce a variety of symptoms simulating carcinoma of the oesophagus, peptic ulceration of stomach or duodenum, cholelithiasis, and even disease of the coronary arteries. In this communication, therefore, we propose to review the literature briefly, to discuss the incidence of the condition relative to other lesions of the lower third of the oesophagus, and to point out associations with pregnancy and post-operative oesophageal stricture which we believe have not been previously recognized.

Isolated references to patients described as having a short oesophagus were recorded years ago by physicians from findings at necropsy (Bright, 1836; Bund, 1918; Bailey, 1919). The radiologist Akerlund (1926) seems to have been the first to differentiate hernia of the stomach through the oesophageal hiatus into three types according to the x-ray appearances (Fig. 1).

In the oesophago-gastric variety (a) the oesophagus is redundant and tortuous, in the para-oesophageal (b) the oesophagus enters the stomach below the diaphragm and the gastric hernia passes up beside it, while in hiatal hernia with short oesophagus (c) the gastro-oesophageal junction is above the diaphragm but the oesophagus is not redundant.

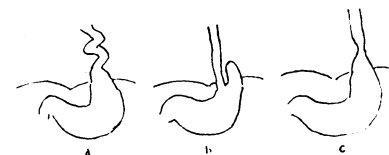


FIG. 1.—Varieties of gastric hernia through the oesophageal hiatus. a, Oesophago-gastric; b, para-oesophageal; c, with short oesophagus.

The first clinical records of a series of cases were published by Findlay and Brown Kelly (1931). Their nine patients were young children in whom regurgitation developed shortly after birth or when solid food was first taken. With barium swallowed in the horizontal position, oesophageal stenosis was found well above the diaphragm, and below it a pouch in which rugae were seen passing through the oesophageal hiatus and continuous with those of the stomach. Confirmation that this pouch was in fact part of the stomach was obtained from oesophagoscopy and biopsy. In the opinion of Findlay and Brown Kelly, this shortening of the oesophagus was congenital, although later Brown Kelly (1936) appears to have thought that in some instances the shortening was cicatricial and secondary to oesophagitis.

Shortly after the paper by Findlay and Brown Kelly, Monkhouse and Montgomery (1933) reported seven instances in adults. In the same year peptic ulceration in the lower end of the short oesophagus was conclusively demonstrated by Clerf and Manges (1934). They described 14 patients, children as well as adults, who had a short oesophagus and thoracic stomach, an ulcer being found in four. Biopsy in eight cases showed gastric mucosa above the level of the diaphragm. The presenting symptom in those with ulcer was burning pain at the lower end of the sternum, coming on immediately after taking solid food, and tending to radiate through to the back between the shoulder-blades or up to the root of the neck and sometimes down the left arm. It was relieved by alkalis, by regurgitation, or by drinking bland fluids, and was made worse by lying down. Dysphagia often did not appear until pain had been present for months or years. In those in whom no ulcer was demonstrated dysphagia was the presenting symptom, and in one patient in whom complete oesophageal obstruction was precipitated by impaction of a piece of apple it had been present from birth.

Clerf and Manges emphasized the importance of the observation made originally by Soresi (1919) in regard to diaphragmatic hernia in general, that to demonstrate part of the stomach above the diaphragm it was essential to lower the head of the table to allow the barium to flow back through the cardia. They found that the best view was obtained with the patient semi-supine in the right oblique position. Failure to use this technique meant failure to reach a correct diagnosis, as only the oesophageal stenosis was revealed by barium swallowed in the upright position, while the thoracic stomach, essential for the diagnosis, was missed. They also held that the lesion was a congenital shortness of the oesophagus accompanied by thoracic stomach—i.e., the cardiac part of the stomach had never descended below the diaphragm in course of development. A predisposition to peptic ulceration of the oesophagus existed because of reflux of gastric juice through the cardia, which was incompetent in its thoracic position. Increase or onset of pain on lying down was held to support this hypothesis.

The occurrence of short oesophagus with thoracic stomach and its association with oesophageal ulcer has been noted by many other observers (Dunhill, 1935, 1937; Chamberlin, 1938; Feldman, 1939; Cleaver, 1943). Among the later workers, Johnstone (1941) recorded seven examples in all of which a chronic peptic ulcer of the oesophagus was present. He held that shortening of the oesophagus occurred as a sequel to the ulceration and that the cardia was as a result drawn above the diaphragm. In support of this view he pointed out that all his cases were at or above middle age. On the other hand Dick and Hurst (1942) believed that a chronic ulcer of the oesophagus

seldom occurred save as a sequel to congenital shortening of the oesophagus and thoracic stomach. In the few cases in which this abnormality was not demonstrated the ulcer was ascribed to the secretion of gastric juice by heterotopic gastric mucosa in the oesophageal wall. They considered that the symptoms of which a patient with a short oesophagus complained were as a rule those due to the ulcer, and if there were no ulcer the condition was latent and was found fortuitously on barium examination. It was also noted that pain was made worse not only on lying down but on bending, whereby intra-abdominal pressure was increased and gastric juice regurgitated into the oesophagus.

Polley (1941) had noted that in some cases pain had radiated into the neck and down the left arm. As a rule dysphagia occurred only after days or even weeks of pain. Intermission of symptoms was often observed, suggesting that spasm from the ulcer was the initial cause of dysphagia, while organic stricture gave rise to permanent dysphagia and required dilatation for its relief. In a recent review of the subject Allison (1948) discussed 78 examples of oesophageal ulcer or stricture all but two of which were associated with hiatal hernia and short oesophagus.

From the point of view of the radiologist the literature has been well reviewed by Smithers (1945).

In the present paper are described the clinical features of 31 patients with hiatal hernia and short oesophagus who were admitted to the Western Infirmary, Glasgow, over a period of 10 years.

Incidence

The frequency of short oesophagus relative to other common lesions in the lower third of the oesophagus is shown in Table I. Thus of 139 patients seen during a

TABLE I.—Frequency of Lesions of the Lower Third of the Oesophagus with Dysphagia (1937-46)

Carcinoma	80 (all sites 203)
Achalasia	31
Hiatal hernia with short oesophagus	26 (+5 without dysphagia)
Fibrous stricture (caustics)	2

period of 10 years complaining of dysphagia due to obstruction in that site, 80 had carcinoma; achalasia was the cause in 31, and short oesophagus with benign stricture or active oesophageal ulcer in 26. Excluding the two cases due to swallowing of caustic, benign stricture was not seen in the lower third except in association with short oesophagus.

Short oesophagus was discovered during the barium examination of five patients whose symptoms of haematemesis or severe flatulence were unrelated to the oesophagus. Since this survey was made largely in retrospect, it is probable that some cases were missed. An attempt was made to re-examine all those in whom there was doubt about the cause of dysphagia in the lower third of the oesophagus, but three patients could not be traced.

It is certain that small sliding gastric hernia with shortening of the oesophagus occurs without symptoms, as it can be demonstrated when the appropriate radiological technique is employed. Kemp Harper (1946) put the incidence at 2% of all patients undergoing routine barium-meal examination. One of us (S. D. S. P.) confirmed this observation in 100 consecutive barium investigations.

Symptomatology

The age-and-sex incidence is shown in Table II, and is contrasted with that of achalasia and carcinoma of the lower third of the oesophagus. The age is that at the onset of symptoms in each group.

TABLE II.—Age and Sex of Patients with a Lesion in the Lower Third of the Oesophagus

Age at Onset	Short Oesophagus		Achalasia		Carcinoma	
	M	F	M	F	M	F
0-19 years ..	2	2	1	1	0	0
20-39 " ..	2	6	2	11	1	0
40-59 " ..	5	8	9	5	23	11
60-80 " ..	1	5	1	1	38	7
	10	21	13	18	62	18

Earlier workers found that symptoms associated with short oesophagus might appear at any age. In our series three patients had dysphagia from childhood, but in the majority no complaint was made before middle or late adult life, the oldest being 74. Only one case of cancer of the lower third of the oesophagus was observed in a person under 40 years of age, in a man of 26.

The sex incidence of short oesophagus (Table II), found to be about equal by Dick and Hurst (1942) and by Allison (1948), showed a preponderance of females in the ratio of 2 to 1 in our series. In achalasia this difference was less marked, while in carcinoma the ratio of males to females was 3.4 to 1.

The presenting symptom was pain in three cases, dysphagia in eight, and pain and dysphagia together in 13. This does not accord with the observations of Dick and Hurst (1942), who found that often pain alone was present, sometimes for years, before the onset of dysphagia. Two patients, without previous complaint related to the alimentary system, were admitted to hospital because of massive haematemesis. Analysis of the symptoms in our cases agrees for the most part with the clear descriptions by Clerf and Manges (1934) and later by Dick and Hurst (1942). Pain was complained of by 18 patients. In 13 instances it was felt at the lower end of the sternum, three times high in the epigastrium, and once each in right and left hypochondriac regions. It tended to radiate upwards behind the sternum and through to the back. Two patients complained of radiation to the neck and down the outer aspect of the arm—the left in one, the right in the other. In character it was burning, often coming on immediately after swallowing solid food, particularly meat or fibrous vegetables. It lasted for several minutes and was relieved either when the food was felt "to pass on into the stomach" or was regurgitated. In others the onset of pain was delayed for about 30 minutes and was relieved by regurgitation or by taking bland fluid. Alkali was often helpful. In addition to pain associated with food eight patients experienced pain on lying down or on bending forward. Five said that posture had no influence.

Dysphagia and Regurgitation.—Difficulty in swallowing solid food was felt by 26 patients, and 21 regurgitated food in varying degree. Complete obstruction to the passage of food was precipitated in two patients with partial stricture—in one by impaction of a piece of wood in a sausage, in the other by a piece of orange and again, two years later, by a raisin.

Haematemesis.—This occurred in nine cases. In five instances it was slight and recurrent, and in some consisted of altered blood. The other four patients were admitted to hospital as cases of emergency because of gross vomiting of blood; in two of them bleeding occurred without warning or any symptom of alimentary disorder.

There was one example of chronic haemorrhage with anaemia in the series.

Case 1.—A woman aged 36 had for over a year felt dull pain in the epigastrium relieved by food. No regurgitation. Stools

were occasionally dark. Breathlessness, fatigue, and pallor had been present for some months, due to anaemia. Hb, 47%; R.B.C., 3.7 millions; occult blood was present in the faeces. A radiograph showed a short oesophagus and thoracic stomach but no stricture. No ulcer was seen in the oesophagus, stomach, or duodenum.

In this group a definite ulcer was located twice—once in the thoracic pouch of stomach, and once in the lower oesophagus. In the others an associated acute peptic ulcer of stomach or duodenum may have been responsible, as these lesions may be undetectable by radiology within a few weeks of the bleeding. The other possibility is haemorrhage from erosions in the thoracic pouch which are prone to occur because of congestion of the mucosa in that site. In two of this group dysphagia did occur later.

Flatulence.—This was a prominent complaint in four patients. Dysphagia was absent in two and minimal in two.

Case 2.—A woman of 56 had for at least 15 years complained of pain under the left costal margin and very severe flatulence after meals. In 1934 cholecystectomy had been performed without relief, no stones having been found. Peptic ulcer and spastic colon were diagnosed on subsequent occasions, but no proof of either of these lesions was found. In 1942 the pain was gnawing in character and was relieved by vomiting and by alkaline powder. No complaint of dysphagia was made. On her last admission in 1942 a radiograph showed a short oesophagus and a thoracic stomach. No oesophageal stricture or ulcer was seen. This was an x-ray diagnosis and was reached quite by chance so far as the clinician was concerned.

Intermission of the Symptoms.—This was characteristic, 21 patients agreeing that the severity of pain and difficulty in swallowing varied. Some lost their pain after months or years but were left with dysphagia, because of which they had to eat only food which could be very thoroughly masticated.

X-ray and Oesophagosopic Appearances

In the earlier part of the period covered by this survey the actual barium swallow was as a rule given to patients in the standing position only. A typical picture of the result thus obtained is given in Fig. 2, which shows a partial stricture with irregular wall some distance above the diaphragm, the appearance resembling that of carcinoma. If, however, the patient is tilted in order to allow the barium to flow back into the oesophagus the existence of a portion of the stomach above the diaphragm becomes apparent (Fig. 3). It is also sometimes necessary to increase the intra-abdominal pressure by compressing the abdomen or by instructing the patient to strain. It is interesting to note, however, that in two cases in our series it was found impossible to reproduce these appearances on every occasion a barium examination was made, no evidence of hiatal hernia or short oesophagus being obtained.

In this series of 31 cases of short oesophagus and thoracic stomach a partial stricture of the oesophagus was present in 26, and 15 patients showed also an oesophageal ulcer. Thus five patients had a short oesophagus and a thoracic stomach without stricture, though subsequently a stricture did develop in two. Carcinoma was found in the thoracic part of the stomach once, and simple ulcer once.

Oesophagitis was found in varying degree over a distance of about an inch (2.5 cm.) proximal to the constriction in the lower oesophagus. The mucosa was generally engorged with superficial erosions, and radiating streaks of greyish exudate were conspicuous. In some cases there were granulations simulating malignant fungations. Active peptic

ulcers had a flat yellowish-white base with a narrow bright-red margin, and greyish-white scars marked the sites of healed ulcers. Where pronounced spasm or secondary

fibrosis was absent the constriction was readily distended, revealing the typical rugose mucosa in the supradiaphragmatic gastric pouch; in others spasm or fibrosis produced varying degrees of resistance to dilatation.

The Short Oesophagus in Association with Other Conditions

Carcinoma in Thoracic Part of Stomach.—According to Dick and Hurst (1942) there is no unequivocal evidence that oesophageal ulcer undergoes malignant change. In 1934 Clerf and Manges reported an instance of cancer occurring not in the short oesophagus but in the thoracic stomach. Smithers (1945), reviewing the literature, quoted such a case recorded by Christiansen (1941) and another by Raven (1941), and added two of his own.

Case 3.—For three months before admission a woman aged 67 had progressive dysphagia until only fluids could be swallowed. Food was felt to stick at the lower end of the sternum and was accompanied by a burning sensation. Regurgitation was frequent, with blood present on several occasions. Appetite was very poor from early in the illness. A radiograph showed a short oesophagus and a stricture. A thoracic stomach was present in which there was a persistent ragged outline at one point. Oesophagoscopy revealed a stricture 27 cm. from the teeth, and a specimen taken for biopsy showed gastric mucosa infiltrated with adenocarcinoma. This patient died in another hospital, and no necropsy was made.

Peptic Ulcer in Stomach or Duodenum.—Seven out of 31 patients had a history of peptic ulceration of the stomach or duodenum.

Case 4.—A man aged 54 was admitted in February, 1941, because of haematemesis from a duodenal ulcer which had given rise to symptoms for three years. Between February, 1941, and July, 1943, he was readmitted on three occasions because of haematemesis. At the time of admission in 1943 there was much vomiting due to pylorospasm. Within a few days he began to complain of burning pain behind the sternum and to regurgitate solid food. A radiograph showed a thoracic stomach and a short oesophagus to be present along with an oesophageal ulcer and a tight stricture. For about four weeks all solids and much fluid were regurgitated. Thereafter he improved rapidly, and, while occasional dysphagia occurred, he remained well without dilatation until readmitted in December, 1945, with a haematemesis which proved fatal. Post-mortem examination showed that the fatal bleeding had taken place from a duodenal ulcer, while the scar of a healed ulcer was observed in the oesophagus 5 cm. above the cardia. There was no stricture, and shortening of the oesophagus was not noted. As the pathologist had unfortunately not been informed of the radiological findings the oesophageal hiatus was not measured.

This case is of interest in that it demonstrated that extreme dysphagia lasting for several weeks was caused by spasm without organic stricture.

Dysphagia after Abdominal Operation.—In three of the cases in this series persistent dysphagia which had lasted for years developed within a few days of an abdominal operation.

Case 5.—A man aged 51 was operated on in July, 1943, for perforation of a duodenal ulcer. After operation continuous vomiting took place, for which gastro-enterostomy was performed six days later. Within a few days he felt food sticking at the lower end of the sternum. Pain was slight, of a burning character, and always at the same spot. These symptoms were at first intermittent, but dysphagia became very marked in October, 1943, and a barium swallow showed a short oesophagus, a thoracic stomach, an oesophageal ulcer, and stricture. Oesophagoscopy revealed a partial stricture 28 cm. from the teeth. No break of mucosa was seen and no biopsy was made. Apparently because it was felt that carcinoma could not be excluded, it was decided to give a course of deep therapy, which was soon discontinued at the radiologist's request.

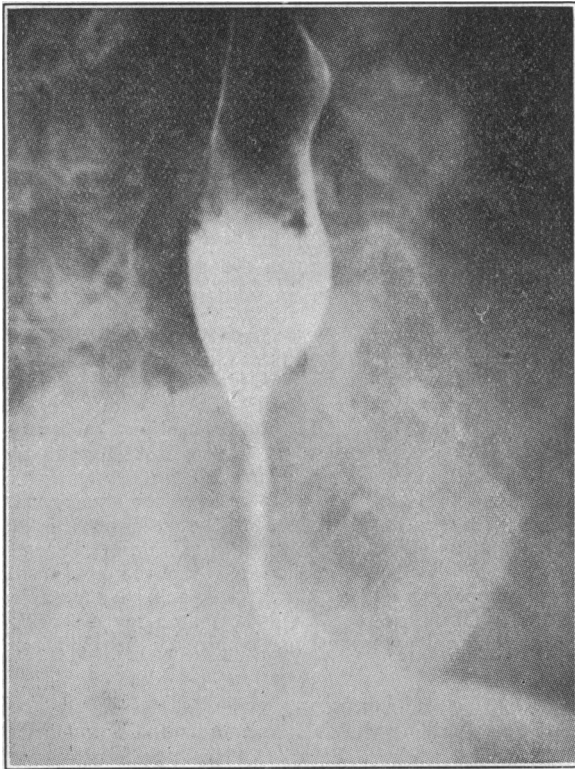


FIG. 2.—Radiograph taken in standing position showing partial stricture with irregular wall some distance above diaphragm, resembling carcinoma.



FIG. 3.—Radiograph taken while patient was tilted in order to allow the barium to flow into the oesophagus. A portion of stomach above the diaphragm is apparent.

When seen in 1946 he had no pain, but still had to chew solid food very carefully. A radiograph again showed a short oesophagus, a thoracic stomach, an oesophageal ulcer, and stricture.

Case 6.—A woman aged 58 developed dysphagia three days after an operation for gall-stones in 1936. Food stuck at the lower end of the sternum, associated with pain in the epigastrium and left hypochondrium. It was worst about half an hour after food and was relieved by regurgitation, which occurred frequently. These symptoms were notably intermittent, even from meal to meal. X-ray examination in the upright position only revealed a stricture in the lower third of the oesophagus, while oesophagoscopy showed a "fungating growth" starting 30 cm. from the teeth and becoming obstructive near the cardia. No biopsy was made, and x-ray treatment for carcinoma was given. She was seen by us in 1946, when she was fairly well but required the passage of bougies at frequent intervals. Her pain, which was still present, was relieved by sodium bicarbonate. She had had slight haematemesis in 1945. A radiograph in 1946 disclosed a short oesophagus with ulcer, stricture, and thoracic stomach. Oesophagoscopy showed an ulcer proximal to the stricture 25 cm. from the teeth, and gastric mucosa was obtained on biopsy from the supradiaphragmatic pouch.

Case 7.—A man aged 37 was operated on for a perforated duodenal ulcer in 1940. Within a week of this he felt discomfort at the lower end of the sternum on swallowing solid food, which stuck momentarily at that site, but there was no regurgitation. A barium swallow in the upright position was stated to indicate nervous spasm. The symptoms continued intermittently till September, 1945, when he had a bout of severe substernal pain and marked dysphagia with regurgitation of solid food. A radiograph at this time showed a short oesophagus, a thoracic stomach, an oesophageal ulcer, and stricture. Oesophagoscopy revealed a stricture 32 cm. from the teeth and a short oesophagus. Biopsy was not made. Bougies were passed with good effect, and in April, 1946, he stated that he was able to swallow most things if well masticated.

Only three instances of persistent dysphagia appearing first after operation were found in the hospital records over 10 years. Oesophageal ulcer and benign stricture were present, associated in every case with a short oesophagus and a thoracic stomach.

Pregnancy

The occurrence of dysphagia in the last three months of pregnancy has been described by Vinson (1923, 1924). The initial symptoms were "vomiting" and pain behind the lower end of the sternum, followed within a day or two by dysphagia, for which the passage of bougies was required. Haematemesis was common and even proved fatal in some cases. Vinson believed that these patients developed an oesophageal stricture because of frequent vomiting. From his records dysphagia was still present months after the onset.

In five of the present series the onset of oesophageal symptoms occurred in the later months of pregnancy.

Case 8.—A woman of 36 developed dysphagia in the sixth month of pregnancy. It was accompanied by a burning pain at the lower end of the sternum which passed through to the back. Symptoms intermitted with almost complete relief for several weeks at a time. In 1933 radium treatment was given for carcinoma of the oesophagus, the diagnosis being based on an oesophagoscopy examination which showed an "ulcerated growth" 30 cm. from the teeth and an obstruction to barium at that level. No biopsy was made. After treatment symptoms continued as before, but the patient's general condition remained quite good. In 1946 x-ray examination revealed a short oesophagus with ulcer, and oesophagoscopy showed the oesophagus to be dilated, with a conical approach to a fibrous stricture 26 cm. from the teeth. A whitish scar was visible on the posterior wall at the entrance to the stricture.

Case 9.—Just before the birth of her baby in 1940 a woman aged 36 felt that food stuck at the lower end of the sternum; there was no pain. Regurgitation was frequent at first. In 1940 x-ray examination in the upright position revealed stricture of the lower third of the oesophagus, and oesophagoscopy showed an annular obstruction 31 cm. from the teeth without breach of mucosa. No biopsy was made. Cancer was diagnosed and x-ray therapy was given. Some months later the stricture was dilated by bougies with complete relief. When seen in 1946 she was very well and could eat anything without dysphagia provided she chewed it thoroughly and had no emotional disturbance. A radiograph revealed a short oesophagus, a thoracic stomach, an oesophageal stricture, and an ulcer.

Case 10.—A woman aged 38, in the seventh month of pregnancy in 1942, quite suddenly felt solid food stick at the lower end of the sternum. At the same time she was conscious of a burning pain at this site immediately after taking food. Symptoms were rather worse after delivery and she lost weight. Regurgitation was frequent and gave relief. X-ray examination showed that barium was held up 7 cm. above the diaphragm, where there was a smooth stricture. Below this there was a globular enlargement in which mucosal markings were present, continuous with those of the main body of the stomach below the diaphragm, but no ulcer was seen. Oesophagoscopy was not done. In 1946 she was fairly well, but had to be careful what she ate. X-ray findings were similar to those in 1942.

Case 11.—This patient, aged 39, developed difficulty in swallowing solid food during the last weeks of pregnancy in 1944. Regurgitation was frequent but there was no pain. X-ray examination revealed a short oesophagus, a thoracic stomach, a small ulcer of the oesophagus, and a stricture. In April, 1946, she still had difficulty in swallowing.

Case 12.—This woman, at the age of 41 and during the last two months of pregnancy, felt a burning pain in the mid-sternal region which radiated to the back. It started 10 minutes after taking solid food, but was also present at night, when it was so severe that she could not lie flat. There was no regurgitation, but food tended to stick at the site of the pain. Nineteen years after the onset of symptoms, which were intermittent, she suddenly began to regurgitate all food and drink. A barium swallow showed complete obstruction in the lower third of the oesophagus. The diagnosis of foreign body was made and a piece of orange was removed from the lower end of the gullet with immediate relief. When seen in April, 1946, she said that some pain and dysphagia were still present. X-ray examination revealed a short oesophagus and a thoracic stomach, with partial stricture of the oesophagus, but no ulcer could be demonstrated.

The findings in these five patients are summarized in Table III. All the patients were over 35 and symptoms started between the fifth and last months of pregnancy.

TABLE III.—*Dysphagia in Pregnancy*

Case No.	Dysphagia	Pain	Month of Pregnancy	Age at Onset	Duration of Symptoms (Years)	X-ray Findings in 1946
8	+	+	6	36	13	S.O., T.S., Str., U.
9	+	0	9	36	6	S.O., T.S., Str., U.
10	+	+	7	38	4	S.O., T.S., Str.
11	+	0	9	39	2	S.O., T.S., Str., U.
12	+	+	7	41	19	S.O., T.S., Str.

S.O. = Short oesophagus. T.S. = Thoracic stomach. Str. = Stricture. U. = Ulcer.

The duration of symptoms ranged from 2 to 19 years. Dysphagia was present in all, and pain behind the lower end of sternum in three. X-ray examination revealed short oesophagus, thoracic stomach, and oesophageal stricture in all, with ulcer also present in three.

Vinson attributed the stricture to the presence of gastric juice in the oesophagus resulting from excessive vomiting, but the development of dysphagia and regurgitation in our

series was in no instance preceded by vomiting. On the facts it seems reasonable to argue that the development of the stricture during pregnancy is due to increased intra-abdominal pressure at that time causing reflux of gastric juice into the oesophagus through a cardia incompetent because of its position in the thorax. The age of the patients in relation to pregnancy, the youngest being 36, is possibly another factor predisposing to laxity of the oesophageal hiatus. Allison, Johnstone, and Royce (1943) describe a similar case in which dysphagia began during pregnancy and a short oesophagus was found to be present, but they make no comment on the association.

Pathology

Since the earlier reports, summarized by Findlay and Brown Kelly (1931), there has been singularly little addition to the pathological knowledge. Morison (1930) described a further clear-cut case.

In this series, two patients died (one of haemorrhage from a duodenal ulcer and the other of cancer of the colon) and post-mortem examinations were made. The description of the findings in Case 4 has already been given.

Case 13.—A woman aged 62 was admitted to hospital because of bleeding from the rectum due to carcinoma of the pelvic colon. She gave a 40-year history of regurgitation of solid food which stuck at the lower end of the sternum. No other information was available from records. X-ray examination showed a short oesophagus, a large thoracic stomach, and a partial oesophageal stricture, but no ulcer was seen. This patient subsequently died, and at necropsy the oesophagus was not short, since the measurement from the top of the thyroid cartilage to the cardia was 30 cm. The hiatus, however, measured 2 in. (5 cm.) in diameter, the oesophagus being placed centrally and attached loosely to it by a fibrous diaphragm.

Unfortunately in Case 4 no special search was made and the oesophageal hiatus was not measured, but there was at least no pronounced shortening of the oesophagus and no part of the stomach was found in the thorax. The findings in Case 13 were also unexpected in that here again none of the stomach was in the thorax and there was no shortening of the oesophagus—this in spite of the unequivocal radiological evidence to the contrary (Harrington, 1940). One is therefore forced to the conclusion that even after years of dysphagia and regurgitation the hiatal hernia may remain unfixated or sliding, while the shortening of the oesophagus must be attributed to spasm. It should be noted, however, that in neither of these cases was there a fibrous stricture.

In an exhaustive discussion Smithers (1945) reached the conclusion that the initial lesion is a congenital or acquired weakness of the oesophageal hiatal ring with herniation of the cardiac end of the stomach followed by passage of gastric juice into the oesophagus. Contraction of the longitudinal muscle fibres is thereby provoked with production of "shortening" of the oesophagus. Support for this view is drawn from the work of Gilbert, Dey, and Rall (1946), who stimulated the vagi of cats electrically and observed contraction of the oesophagus, with drawing upwards of the cardia through the oesophageal orifice. We have ourselves noted in two very recent patients (not included in this series) at one time radiological appearances typical of large para-oesophageal hernia and a few weeks later those of hiatal hernia with short oesophagus. This would make it very difficult to accept the hypothesis of congenital shortening of the oesophagus.

On the other hand the post-mortem observations of the earlier workers and recent work of Allison (1948), who excised the fibrous oesophageal strictures, show that in

some patients, presumably after ulceration, the oesophagus becomes permanently shortened by cicatricial contraction.

Prognosis and Treatment

The majority of patients in this series were able to lead a reasonably comfortable and normal life in spite of persistence of oesophageal symptoms, provided they exercised care in the choice of diet and masticated their food thoroughly. From time to time a number required the passage of bougies. The upright position in bed to minimize the reflux of acid, and the use of alkaline powders, were often helpful. One patient seemed to have made a complete recovery, but refused to have a re-examination with barium. One patient was found to have adenocarcinoma of the thoracic stomach and died in another hospital. The other two deaths in this series were due to a coexistent duodenal ulcer (haemorrhage) and to carcinoma of the colon respectively.

It is of interest that we were able, after an interval of 17 years, to re-examine two of the patients originally reported on by Findlay and Brown Kelly in the Royal Hospital for Sick Children, Glasgow, when one of us was house-physician to Professor Findlay. One was a woman now aged 25, small and slightly built, who was in good health and had served in the A.T.S. She confessed that at times she had dysphagia and regurgitation, but apparently had had no difficulty in concealing this from the Army authorities. Barium examination revealed a short oesophagus, a thoracic stomach, and a partial stricture. The other patient was a sturdy youth of 18 who occasionally felt food stick at the lower end of the sternum. X-ray examination gave a similar picture.

Summary

Over a period of 10 years dysphagia in 26 patients was found to be due to simple ulcer or stricture of the lower end of the gullet, accompanied by x-ray appearances of gastric hiatal hernia and shortening of the oesophagus. Over the same period cancer in the lower third and achalasia caused dysphagia in 80 and 31 instances respectively.

The diagnosis was not often considered, and proof was obtained only by radiology, using a special technique with, on occasion, oesophagoscopy and biopsy.

Dysphagia and ulcer pain developed during the later months of pregnancy in five patients and immediately following abdominal operation in three.

In five patients there were no oesophageal symptoms—three were admitted because of massive haematemesis, one because of anaemia, and the fifth gave a long history of pain and flatulence for which her gall-bladder had been removed without relief.

Congenital or "acquired" weakness of the oesophageal ring is common, allowing of a small sliding hiatal hernia without symptoms. It is considered that only in a minority of people does oesophageal ulcer develop. A rise of intra-abdominal pressure in pregnancy would seem to be one factor concerned. Oesophageal ulcer was not encountered in the absence of hiatal hernia and short oesophagus.

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TRILENE ANALGESIA IN PAEDIATRIC PRACTICE

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Various minor diagnostic procedures in paediatrics, such as venepuncture, lumbar puncture, marrow puncture, testing electrical reactions of muscles, abdominal palpation, and chest exploration, cause apprehension and discomfort, often sufficient to render a child uncooperative or even actively resistant. Many other painful and uncomfortable undertakings occur in the course of treatment, such as intravenous injections and burn and wound dressings.

It is possible to diminish apprehension with such sedatives as chloral hydrate or "seconal" and still leave the patient co-operative; but often at the first appreciation of pain the child becomes resistant. The use of anaesthesia or local analgesia would seem to be justified where there is likely to be more than trivial discomfort or where it is essential that the patient should remain still and not resist. General anaesthesia eventually eliminates the patient's resistance but deprives us of his co-operation, and is not without a certain minimal risk. Local analgesia, with its attendant first stab of pain, often renders the child apprehensive, so that he becomes restless and uncooperative.

It occurred to one of us (J. T.) that analgesia produced by a simple inhaler might overcome the disadvantages of sedatives and of anaesthesia. In this communication the apparatus, indications for its use, the technique of administration, and the results obtained in a series of 60 consecutive administrations are described.

Apparatus

The apparatus used was the Blease inhaler. This is a simple draw-over type of inhaler, which can give three concentrations of "trilene" (trichlorethylene) suitable for analgesia and an additional one for anaesthesia by adjusting

a simple screw setting. On occasions when induction was slow it was necessary to increase the concentration to "anaesthesia." The only modification required in the standard equipment was the provision of a small face-mask. We used the smallest obtainable (B.O.C. type, small size). At times an even smaller size would have been an advantage.

Indications.—We define analgesia as the loss of sensation of pain without loss of consciousness. The apparatus was used when a diagnostic or therapeutic measure was likely to cause pain, discomfort, or undue apprehension, and where formerly we would have employed sedatives or local or general anaesthesia. When analgesia is desirable, trilene would seem to have no special contraindication.

Our series of 60 cases represents a cross-section of patients admitted to a children's medical ward for investigation and treatment. Analgesia was given for lumbar puncture (34 cases), cisternal puncture (3 cases), venepuncture (7 cases), chest aspiration (8 cases), marrow puncture (3 cases), burn and wound dressings (3 cases); and in one case each for abdominal palpation and electrical reactions.

Age.—The ages ranged from 8 months to 11 years. In infants under 9 months old inhalational analgesia has little to recommend it. We found its greatest use in children over 1 year old. In those in the group aged 1 to 5 years it prevents resistance; and in those over 5 years it will also often secure actual co-operation.

State of Health.—The state of health varied from the acutely ill newly admitted child to the chronically ill case from the waiting-list for investigation or the comparatively healthy convalescent.

Procedure to be Undertaken.—The predominant symptom, or the suspected lesion, along with the diagnostic procedure undertaken in the various cases, is shown in the Table. In the course of treatment, intravenous injections, toilet of burns, suture of lacerations, and burn and wound re-dressings were all carried out under analgesia.

Administration

No premedication was given, and analgesia was induced without special regard to the interval since the last meal. One patient received trilene as early as five minutes after a meal without untoward effect. Several have been given trilene soon after meals, because of urgency in getting samples for laboratory examination. It is not suggested that a recent meal should be ignored, but when circumstances warrant haste trilene analgesia can justifiably be employed soon after a meal. The child, if old enough to understand, is told the purpose of the inhaler and asked to take extra deep breaths if he should feel anything hurt. The mask is applied and a conversation is kept up with the child to allay fear and encourage regular breathing. The older children, aged 5 to 11, were more or less co-operative during the administration and responded to the instruction to take regular deep breaths, usually becoming analgesic in two or three minutes. The younger children struggled and cried in some instances, but after a few deep breaths, associated with the effort of crying, they quieted down, became analgesic, and were usually drowsy. Coughing and spluttering were unusual. When we had become more conversant with the signs of analgesia (see below), if induction seemed a little slow the control screw was turned to "anaesthesia" and returned to "maximum analgesia" when analgesia was obtained. No increase in concentration of trilene was required for older children. Indeed, the younger ones were usually more resistant; but