THE GASTRO-OESOPHAGEAL REGION IN INFANTS

OBSERVATIONS ON THE ANATOMY, WITH SPECIAL REFERENCE TO THE CLOSING MECHANISM AND PARTIAL THORACIC STOMACH

BY

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The nature of the closing mechanism between the oesophagus and the stomach has attracted considerable anatomical study in the past 300 years. Main interest has centred on the adult; the anatomy of the infant, which does not necessarily correspond, has not received the same attention. The present investigation is therefore concerned with infants. It was undertaken with two objects: first, from the anatomical study, both macroscopic and microscopic, to assess the importance of the various components of the normal closing mechanism; secondly, to consider the abnormal infant, in whom this mechanism is incompetent, in the light of clinical experience and radiological observation.

Material

The diaphragm, lower oesophagus, upper stomach, aorta and surrounding tissue were removed *en bloc* from 115 human subjects and fixed in 10% formol saline. Every specimen was carefully dissected and drawings were made of the essential features in all. Histological confirmation was obtained in doubtful cases. In age, the patients varied from a 32-week foetus to 8 years. In the great majority the ages ranged from a few hours to 1 month (average weight of 5½ lb.). One foetus of 1 lb. 2 oz. and one girl of 13 years were also studied. The sexes were about equally represented. In addition to the dissected specimens, numerous studies were carried out in the necropsy room where the organs were examined *in situ*. The following observations were made.

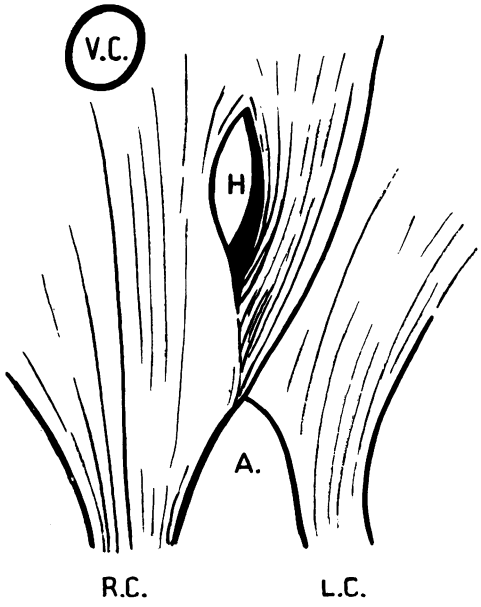
Diaphragm

Anatomy textbooks describe, and interesting pictures convincingly show, decussation of the crural fibres dorsal to the oesophagus. This is rare. Only a few specimens were found where the overlap of muscle fibres at their origin from the median arcuate ligament in the standard type could possibly be mistaken for decussation. Low (1907) and Collis, Kelly and Wiley (1954) found no decussation in a series of 25 and 50 diaphragms respectively. Considerable normal variation occurs, the functional significance of which has not been fully appreciated.

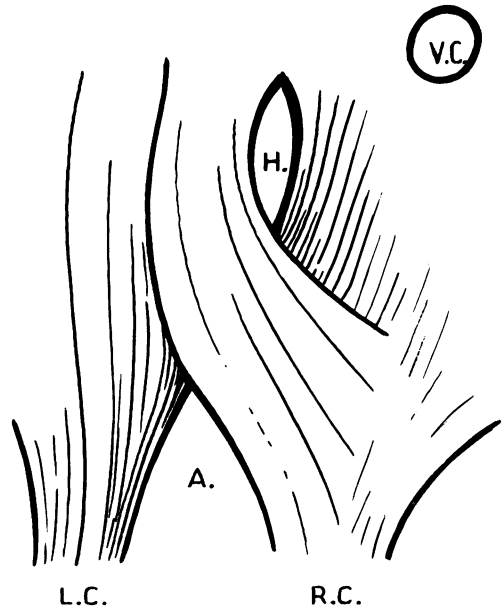
The Standard Pattern. The aorta, a midline structure, divides the crura into a right and left crus (Fig. 1a, 1b, 4). The right crus is better developed, more fleshy and wider than the left one. It arises from the bodies and discs of the upper three or four lumbar vertebrae (sometimes five); it passes cranially and divides into a large right and a smaller left limb which embrace the gullet and join again anteriorly. The fibres of the right limb arise from the vertebral column and right border of the median arcuate ligament and run straight upwards to insert into the central tendon. The right limb is always anterior. The spinal fibres of the left limb run upwards behind the right limb, arch obliquely over the aorta and then ascend immediately to the left of the gullet to insert into the central tendon. The lateral fibres of the left limb are short and arise from the central portion of the median arcuate ligament. The medial fibres sometimes curve acutely to the right, caudal to the oesophagus to gain origin from aponeurotic fibres to the right of the hiatus. The right limb is therefore visualized from the ventral aspect whereas the left limb can only be displayed fully from the back (Fig. 1b, 4). This arrangement results in a perfect overlap. The twisted figure of eight which is so well depicted in most drawings was not seen in a single instance; one must therefore conclude that it is not an anatomical fact. On the contrary, the 'straight' arrangement of the fibres is usually conspicuous.

The left crus is small and almost separate from the lateral fibres of the left limb; it takes no part in the formation of the hiatus. Decussation of muscle

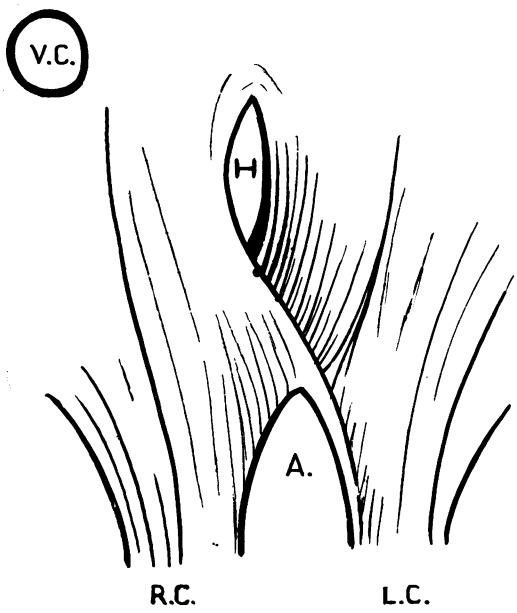
(a) The standard pattern seen from the abdomen.



(b) The standard pattern seen from the chest.



(e) Stage 1 shift to the left.



(f) The broad dorsal band consists of almost half the fibres of the left crus.

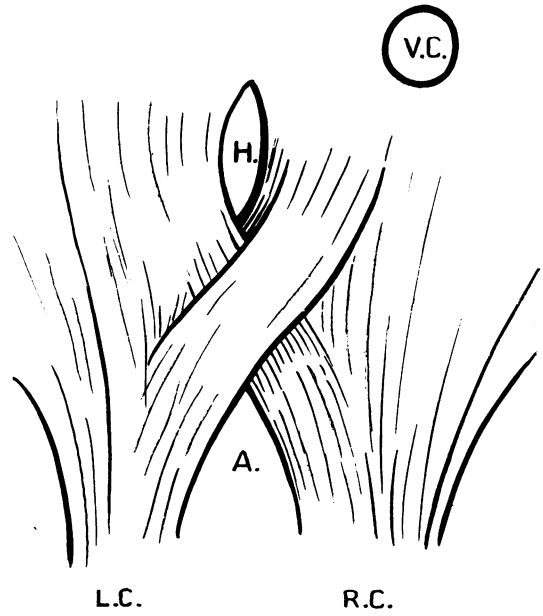
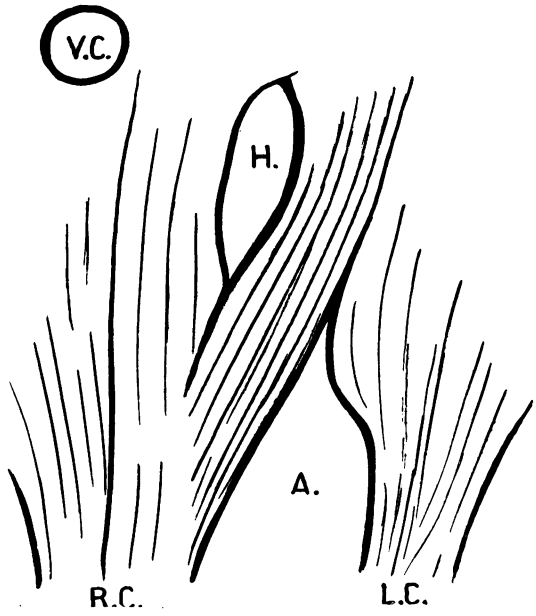


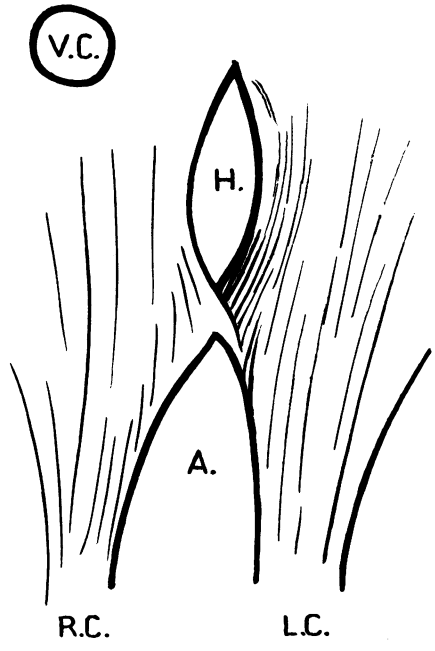
FIG. 1.—Variations in crural anatomy

R.C.=right crus L.C.=left crus A.=aortic opening H.=hiatus V.C.=vena cava

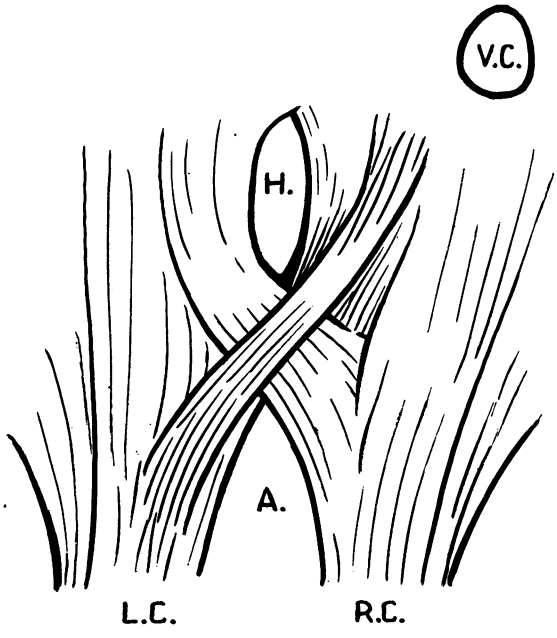
(c) Shift to the right.



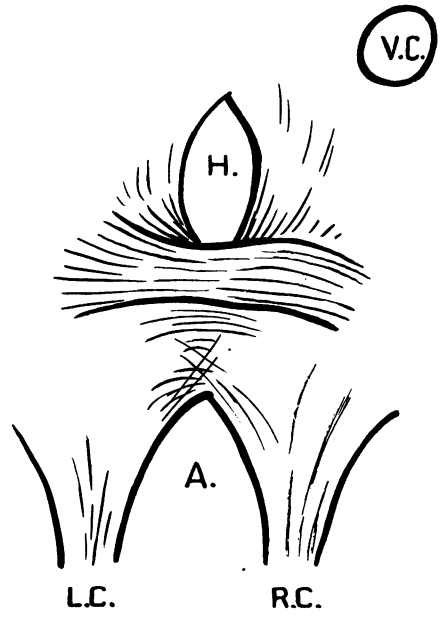
(d) A large hiatus approximates the aortic opening.



(g) A narrow dorsal band.



(h) A transverse band.



fibres does not occur dorsal to the hiatus, but is often present where the fibres arise from the median arcuate ligament.

Deviations from the standard pattern are very common.

Shift to the Right. In this variation, instead of the ventral and dorsal 'waistcoat effect' of the standard pattern, the right crus merely splits into two limbs so that a pyriform hiatal opening is formed (Fig. 1c). There is little or no overlap. The left limb does not arise from the median arcuate ligament, which is non-existent, but itself forms the right border of the aortic opening. The left crus is small and takes no part in the hiatus.

Shift to the Left: Stage 1. The medial fibres of the right crus sometimes transgress the midline to gain origin from the left border of the arcuate ligament (Fig. 1e). This encroachment on the left side may be slight or so complete as to extend down to the tendinous origin of the left crus, but essentially it remains part of the right crus. The left crus still does not partake in the formation of the hiatus.

The Scissors Overlap: Stage 2. The right and left crura may be equally well developed, but commonly the right one is still bigger. The left crus, which is for the first time taking part in the formation of the hiatus, forms a perfect overlap by criss-crossing with the right crus. The most common variation is a broad band from the left crus which crosses anteriorly to form the right hiatal margin, supplemented by a posterior band from the right crus which forms the left hiatal margin. These bands constitute the 'scissors'. They are easily separated and run antero-medially on each side of the aorta as the right and left borders of the median arcuate ligament which, as such, is non-existent. The treble or quadruple overlap when three or more bands criss-cross, is more uncommon (Fig. 2). The relative size and origin of the bands vary tremendously and no two are quite alike.

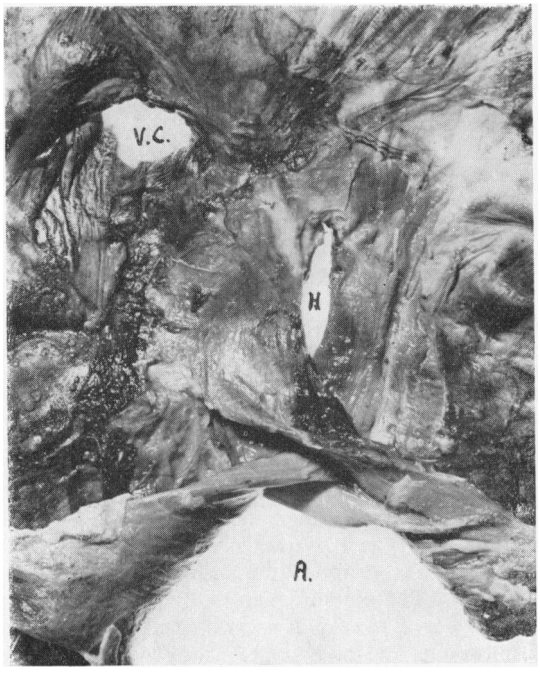


FIG. 2.—A triple 'scissors' overlap.

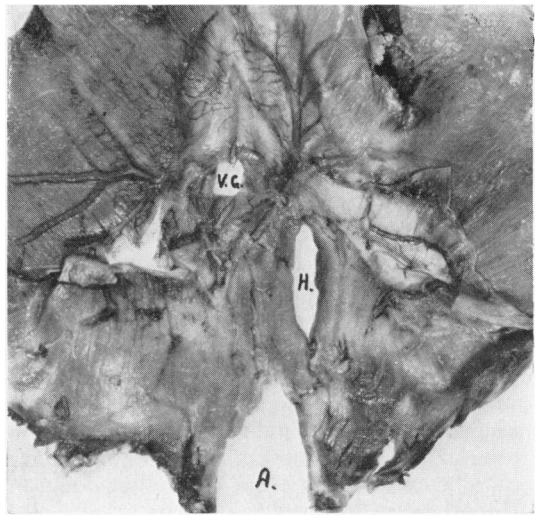


FIG. 3.—Stage 3 to the left. Diaphragm seen from the abdomen.

The Hiatus Formed Completely by Left Crus: Stage 3. This variation must be relatively uncommon. It was encountered in only one specimen and no previous descriptions of it have been seen. The left crus formed the entire hiatus and presented a mirror image of the 'shift to the right' (Fig. 3). There was no overlap in this case.

Accessory Bands. Low (1907) first described an accessory muscle band, 4 mm. wide, that extends from the superior aspect of the left crus, in front of the aorta and within the fibres of the right crus, to the caval orifice. He found four of these bands in 25 specimens. In an excellent paper Collis and his colleagues (1954) described a variant of Low's band as well as a transverse inter-tendinous bundle which they displayed in dissections of 50 diaphragms. Examples of all three types were found in this study, as well as other variants (Figs. 1f, 1g, 1h). The Low bands varied from a few muscle strands to a

TABLE 1
NORMAL VARIATIONS IN THE PATTERN OF THE CRURA

Crural Pattern	Standard Type	Shift to the Right	Shift to the Left: Stage 1	Shift to the Left: Stage 2	Shift to the Left: Stage 3	Accessory Bands	
						Small	Large
Total 115	67	12	28	7	1	20	19
Percentage ..	58·6	10·3	24·2	6	0·9	34	

bundle as thick as the left limb of the right crus and then formed a powerful 'scissors overlap'. The fibres either passed to the right, superficial to the right crus (the great majority), or through the substance of the left limb or lay in front of it. The obliquity varied from almost vertical to almost transverse. Some bands were long, thin and insignificant; other bundles were short, flat and powerful. Whereas most of the small bands are of academic interest only, there can be little doubt that on contraction they tend to decrease the size of the hiatal opening. When well developed, they may become an important factor in, first, enhancing the supportive function of the crura to maintain gastro-oesophageal competence and secondly, to reinforce the posterior weak spot in the hiatus.

The postero-medial branches of the phrenic nerves are the sole motor supply to the crura of the diaphragm (Botha, 1957a). Each nerve innervates the muscle on the ipsilateral side of the hiatus irrespective of its origin.

Table 1 indicates the wide variation that exists in crural pattern.

The Median Arcuate Ligament. In the standard type it is tendinous, well developed and gives origin to crural fibres from both sides. It does not always form the arching 'ligament' so well figured in text-books. It is weakest in the 'scissors' type (Fig. 2) or when the hiatus has shifted to the right and may sometimes be completely absent. In a few dissections the tendinous crural fibres joined in the midline, posterior to the aorta, and thus formed a fibrous ring for the passage of this big vessel.

Overlap. This is one of the most characteristic features of crural architecture and is by far most marked in the standard type. It obviously serves three purposes:

(1) TO STRENGTHEN A POTENTIALLY WEAK AREA. Anteriorly the crura are inserted into the central tendon by short, decussating, musculo-tendinous fibres. Clinical weakness never occurs at this site and therefore no anatomical overlap is indicated. Posteriorly, however, there is little stability and the criss-cross fibres might be easily separated by a force that acts on the hiatus. This is only too well demonstrated by the frequent occurrence of hiatus hernia in late adult life. Overlap is still Nature's best way of providing the greatest protection for the weakest area with the minimum available tissue.

(2) RIGHT LIMB ALWAYS OVERLAPS ANTERIORLY. This creates an oblique hiatus which is essential to accommodate the lower oesophagus with its marked deviation to the left. Overlap is not the cause of the obliquity of the gullet, as this is present if there is poor or no overlap (as found in some types), but it maintains its position more firmly and embraces the oesophagus more snugly during diaphragmatic or swallowing movements. A secure fixation decreases the chances of herniation.

(3) OVERLAP CHANGES ALIGNMENT OF MUSCLE FIBRES. This is specially noticeable in the 'scissors' type where not only the longitudinal, but also the transverse diameter of the hiatus is decreased when the diaphragm contracts. In the standard type, the right limb mainly pulls caudally, but the left limb pulls down and to the right, thus narrowing the hiatus from side to side. This tendency to constrict the hiatus is least marked in stage 3 and 'shift to the right', which on theoretical grounds should therefore be inferior functionally.

The Hiatus. Unlike the adult, the hiatus in the infant is of very uniform size. In all the above dissections, it was small, so small that the narrowest portion of the oesophagus could just pass through (Fig. 4). The oesophagus was gripped firmly.

The presence of a hiatal tunnel in the adult is doubtful. In the infant it is a fact. The crural muscle is round and 'heaped up' at the hiatal margins especially on the right side. In the 7 cm. embryo the length of the right hiatal wall is twice the diameter of the oesophageal opening (Botha, 1958). This length gradually decreases as the foetus develops, but it is still well marked at birth. The length of the canal depends on the degree of obliquity. Where no overlap is present, the hiatus consists of a straight hole through the diaphragm (Fig. 3). The obliquity can easily be seen when the diaphragm is looked at from the front. The hiatus appears as a small, ovoid slit, which sometimes almost disappears (Fig. 4). When viewed obliquely

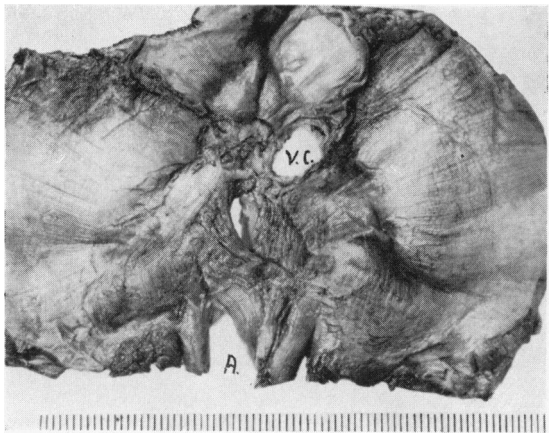


FIG. 4.—The hiatus in the standard pattern is slit-like when seen directly from above.

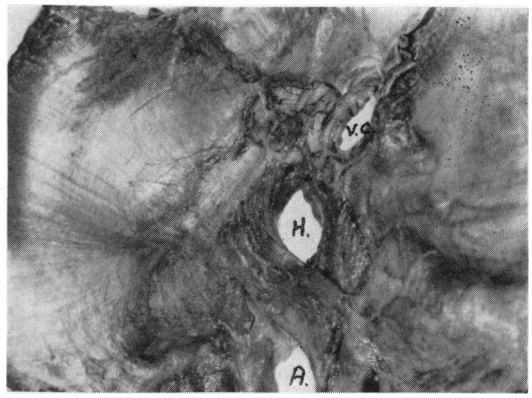


FIG. 5.—The hiatus in Fig. 4 appears large and ovoid or round when viewed obliquely.

in the line of the tunnel, the true size becomes apparent (Fig. 5). As in the adult, but less marked, the anterior wall of the canal is much shorter than the posterior one. This is primarily due to the wide overlap behind and the oblique upward slope of the diaphragm. In the older subjects in this series the canal was relatively shorter, wider and the oesophagus less securely fixed. In infants it was mostly from 3 to 8 mm. long, and proportionately much longer and firmer than in the adult.

The relative distance from the aorta to the caudal margin of the hiatus is also much greater in the infant than in the adult, suggesting that with age the oesophagus gradually approximates to the aorta. This is most certainly due to opening of the overlap and attenuation of the muscle fibres. In one specimen the median arcuate ligament and the inferior hiatal margin were very close together (which is most unusual in infants) and no overlap was present. In addition, the hiatus was relatively big (Fig. 1d). Although the oesophagus was firmly attached in the crural tunnel, this type of diaphragm with short fibres, lack of overlap and a wide hiatus may in future, when the necessary strain is present, give way to herniation.

Phreno-oesophageal Membrane. Any doubt as to the existence of this structure would be quickly dispelled by an examination of the infant diaphragm. There is no basic difference in its formation, position and attachments from that in the adult. It is, however, a much more definite structure that could be seen in every specimen as a thick, closely woven, creamy-white layer that bridged the potential gap between diaphragm and oesophagus. The main component is formed by the well-developed subdiaphragmatic fascia that divides at the level of the hiatus into a short, stout inferior limb and a thinner, long superior limb. The lower portion is usually stronger and is easily seen after removal of the fat and peritoneum as it splays out to a broad insertion into the cardia and adjacent stomach. The superior limb passes upwards through the hiatus and is joined above by the supra-diaphragmatic fascia which is a very definite layer but less well developed than its counterpart below. This strengthened limb then ascends in a circular fashion to insert into the terminal 5-10 mm. of thoracic oesophagus. The insertion of the membrane is never abrupt but in successive concentric layers of elastic strands with small areolar spaces in between. Individual strands are tough and can withstand considerable pressure. Some appear to be attached to the outer connective adventitia of the oesophagus, fascia propria, whereas others penetrate the external lomgitudinal muscle coat and can be traced down to the submucosa. The greatest concentration of fibres is inserted immediately above the diaphragm. Fat is absent in this structure.

The importance of this structure, which has been called a figment of the imagination, cannot be overestimated. It is the sole fixation of the oesophagus in the diaphragmatic hiatus, as the supportive value of the serosal layers above and below is negligible. The stomach and oesophagus were soundly secured in every specimen that I dissected, yet sufficient resiliency was present to allow adequate physiological movement. Fixation was much more firm in infants and movement was less than in children, and even more noticeable when compared with the adult. This difference is essentially due to the anchoring ability of an unattenuated phreno-oesophageal membrane and secondly, to a narrow, tight-fitting hiatus.

This structure is not a ligament. It is a fibro-elastic layer which is specially adapted to stand the strain of incessant movement at the hiatus during diaphragmatic contraction for 70 or 80 years. The histological appearance of the elastic fibres as well as their ratio to collagen is the same as that of a normal adult. Commonly 40-60% of elastic fibres occur but the proportion varies from 10 to 90%. The fibres vary in size, shape and form. Short, stout, blunt-ended strands appear next to thin, long filamentous fibrils. They vary from place to place and are mostly single. Branching does occur but is not marked. Resiliency and strength are obtained by wavy intertwining of the strands. As the fibres run in lamellae or bundles, different layers may be recognized on cross section.

Lower Oesophagus

Anatomically the lower oesophagus can be divided into a supra-diaphragmatic portion, a narrowed hiatal area and the abdominal oesophagus (Fig. 6).

The Supra-diaphragmatic Portion. This part of the oesophagus was generally wide and in the majority the diameter gradually decreased towards the diaphragm. In the older children it appeared wide right up to the hiatus. In only one specimen (a stillborn female of 9 lb.) was there a definite saccular dilatation above the diaphragm. As in the adult, the 'phrenic ampulla' is not an anatomical entity. It must be regarded as a radiological concept for a physiological phenomenon.

The Area of Constriction. In almost all the specimens a constricted area on the oesophagus coincided with the diaphragmatic hiatus which is always terminal in infants. The crura fit tightly around the oesophagus immediately above the cardia so that the abdominal part of this organ is very short. As the infant grows, the abdominal oesophagus increases in length so that the area of constriction shifts proximally. A narrowing was also observed in specimens where the oesophagus was removed from the diaphragm before fixation in formalin, but it was never so pronounced as when the specimen was fixed with the organs *in situ*. This clearly suggests that the diaphragm is mainly responsible for the localized area of constriction, but that another factor is present as well. This factor is the inferior oesophageal sphincter. Evidence of an anatomical sphincter was found in a small proportion of these subjects (Fig. 7). The fibres are thick, coarse and more irregular. Interlacing and cross bundles are common. The width depended on the age, but it was mostly 5 mm. wide

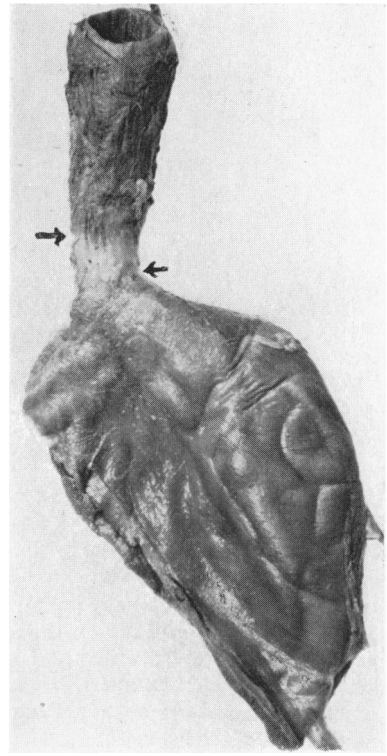


FIG. 6.—The lower oesophagus: the arrows indicate the crural tunnel.

in infants, and could only be inspected properly if the mucosa was removed. It was more difficult to see this in small specimens, but in the older age groups it became more evident. Sometimes it began at the oblique fibres, but in most cases it was above and separate from this bundle. In one specimen, a definite ring-like muscle band of 3 mm. wide was found a few millimetres above the cardia.

The Abdominal Oesophagus. In the 18 mm. embryo the abdominal oesophagus is half as wide as the stomach and almost as long as the lesser curve of this organ (Botha, 1958a). When the embryo is 9·5 cm. long, it is still very well marked. Gradually the abdominal oesophagus gets shorter. At birth it is so short as to be almost non-existent in some cases. After a few months it is usually a few millimetres long and within a few years it measures from 5 to 15 mm.

The left wall is short or non-existent. The right wall is long and curves towards the left in a characteristic fashion to join the lesser curve at the cardia. The distal widening before the oesophagus joins the stomach is slight in the infant, but becomes more apparent in the first five to 10 years.

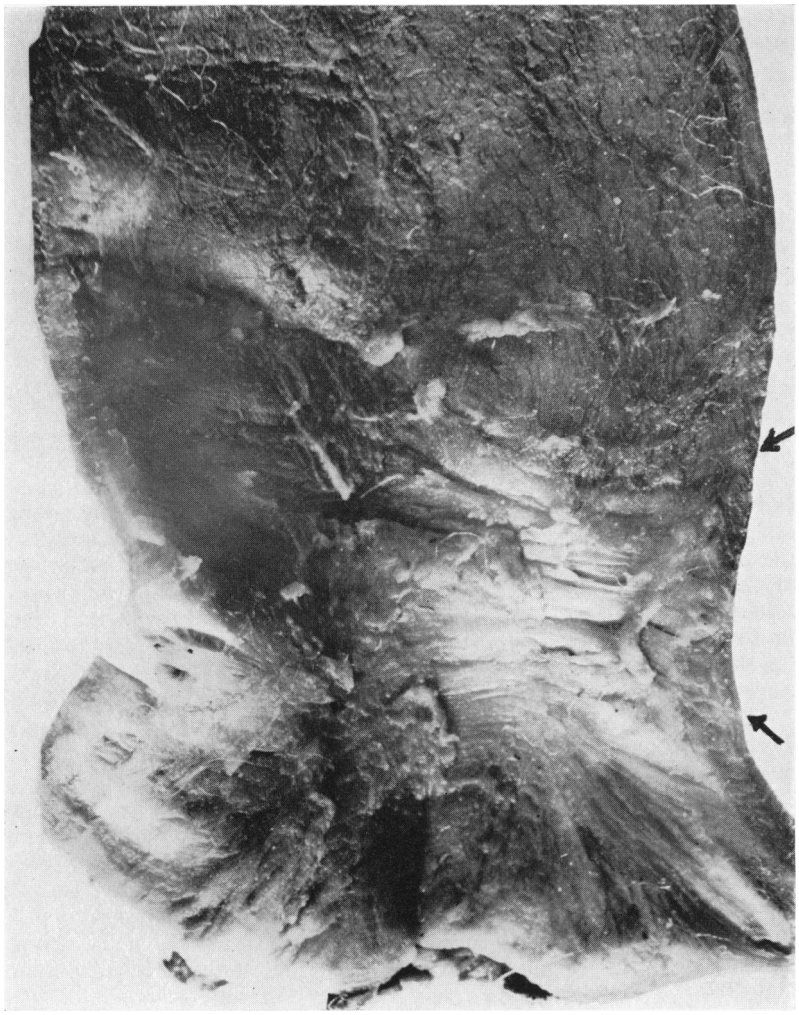


FIG. 7.—The inferior oesophageal sphincter after removal of the mucous membrane.

Cardia

The site where the oesophagus joins the stomach is called the cardia, and in infants it is always well defined. It is narrow in the infant but wider in older children. Internally, the sling fibres are the only guide as to the level of the cardia. They were present in every specimen, but better developed in some. These fibres are already macroscopically evident in the 8 cm. foetus, and form a much sharper ridge in the infant than in the adult.

Cardiac Angle

Several angles are formed by the foregut in the region of the diaphragm. The most widely discussed of these is the gastro-oesophageal angle (cardiac angle or incisura of His, 1903) which is formed on the left side between the abdominal oesophagus and the gastric fundus. Unfortunately this is often confused with the lower oesophageal angle which is formed at the hiatus as the abdominal oesophagus deviates to the left. The other angle is formed by the almost vertical oesophagus entering the stomach which lies at an oblique, horizontal plane.

The first angle is very variable. Its acuteness mainly depends on the fullness of the stomach. In the empty organ it is mostly more than 90°. In the full stomach, especially when a large gastric air bubble is present, the fundus rises higher and bulges medially so that this angle becomes more acute; but even in a full stomach it may still be a blunt angle.

Serosa

The peritoneal reflections in the infant are similar to those in the adult.

Fat Pads. Subperitoneal fatty deposits around the cardia are commonly present in the adult and especially large and lobular in obese individuals with a lax hiatus. These fat pads are either very small or absent in children and infants.

Gastro-phrenic Band. This was present, at least to some extent, in all the specimens. It is formed by a double fold of peritoneum that extends to the left from the abdominal oesophagus and fixes the superior border of the fundus to the under surface of the diaphragm. Sometimes this fold is narrow and almost non-existent. Commonly it forms an appreciable layer and may even extend as far laterally as the spleen. Loose, areolar connective tissue and elastic fibres are present, especially near the hiatus. Histologically the structure resembles

closely the phreno-oesophageal membrane, except that elastic fibres are not nearly so abundant.

Infracardiac Bursa. Persistence above the diaphragm of the pneumato-enteric recess on the right side is common (Fig. 8). In this series 16 mesothelial-lined cavities were found of which seven were quite big. Histological sections proved the serosal lining. In two of these the cavity extended downwards on the right of the oesophagus, through the hiatus into the lesser omentum, where it ended blindly. In none of the present series was any freely communicative sac with the peritoneal cavity found, but this was demonstrated in adult dissections. These serosal pockets or bursae are uncommon in adults, which indicates that most are obliterated after birth.

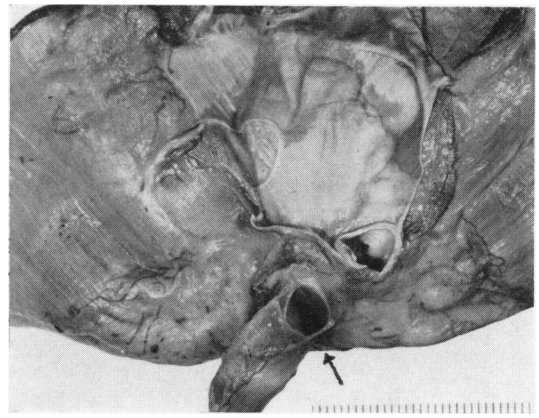


FIG. 8.—Infracardiac bursa on the right of the oesophagus seen from above.

Mucosa

Great variation occurred in the level at which the squamous epithelium changed to gastric mucous membrane. In the great majority the change took place near the gastro-oesophageal junction. In 63% the change took place at the cardia. Although the squamous epithelium was never found to extend well down into the stomach (as in the horse, rat or pig), it did in some cases descend below the cardia for a few millimetres. In 33% the change was above the cardia and in 4% it was well above the cardia. In 69% it occurred below the diaphragm, in 27% at and in 5% above.

The junction was usually clearly visible as a serrated, irregular line where the smooth, pale, stratified squamous epithelium changed to the darker, more granular, glandular mucosa. In some cases, however, it was almost impossible to define the junction on macroscopic examination. The junction lies obliquely in the line of the cardia and is therefore always more distal on the right side.

Longitudinal oesophageal folds are tightly pressed together and no change occurs up to the cardia. There the folds terminate in small irregular triangular pads that radiate away from the cardiac orifice. The gastric folds begin separately at this level, and sometimes appear puckered and bunched up at the level of the hiatus. The folds bear no relation to the level of mucosal change. Even in those cases where this level does appear above the cardia, no alteration can be seen in the longitudinal arrangement of the oesophageal folds. It is quite certain that the site of mucosal change (as distinct from the cardia) cannot be located by *x*-ray examination.

Discussion

The Closing Mechanism. The exact role of the diaphragm, as a factor in preventing abnormal reflux of gastric contents in the adult, is still a matter of dispute. From the present dissections, however, there is little personal doubt as to the important function of the diaphragm in assisting the gastro-oesophageal closing mechanism of the infant. The crural muscle fibres are long, thick and well developed. The excellent overlap provides a small hiatus which is very oblique and relatively long. This, together with a tough, unstrained phreno-oeosophageal membrane ensures firm fixation. The short, almost non-existent abdominal oesophagus minimizes movement and rests snugly in the crural canal. The diaphragm thus forms a most important accessory mechanism which acts during all phases of respiration, although the marked overlap and criss-cross of the broad, stout bundles must result in a maximum constriction when it contracts. Compared with the adult, this special adaptation appears very necessary at the beginning of life. The infant is almost always supine. The stomach is relatively small, and filled every three or four hours with fluid and excess gas. Furthermore, the closing mechanism is frequently subjected to sudden bouts of raised intra-abdominal pressure during crying, hiccoughing or defaecation. Despite this, competence is excellent. In fact, competence is so remarkable that even the conventional posture and traditional tap on the back might sometimes fail to dislodge the gas bubble for five or 10 minutes after a feed. When the gas bubble eventually escapes, it does so with a characteristic burst which is easily perceptible by the flat of the hand on the abdomen; and deflation is as rapid as pricking a balloon. Practically speaking, cardiac incompetence in the normal infant is very uncommon. Thomsen (1955) examined 145 children (7 months to 7 years) radiologically, who were

admitted to hospital for diseases other than gastro-intestinal. Despite steep Trendelenburg and manual compression of the abdomen, reflux was never demonstrated. However, cardiac incompetence in infants with partial thoracic stomach (where support of the diaphragm is lost) is much worse than in adults with hiatus hernia. The excellent competence in infancy is therefore not primarily due to a superior closing mechanism, but to the more efficient function of a less attenuated hiatus.

The existence of a physiological inferior oesophageal sphincter is now well accepted (Lyons, Ellis and Olsen, 1956). In a combined cine-radiographic and manometric study of the gastro-oesophageal junction, Botha, Astley and Carré (1957) proved that this sphincter extends over a considerable segment of the lower oesophagus: it is not confined to the cardia alone. The present study supports these views. In a few specimens, definite macroscopical thickening was present in the circular muscle coat, but this was never confined only to the cardia. These anatomical sphincters should not be compared with the pyloric sphincter as a standard. It is furthermore essential to remove the mucous membrane carefully before inspection of the muscle. Lerche (1950), who conducted a similar investigation in adults, concluded that anatomical sphincters do exist. Histology is notoriously unhelpful in the localization of anatomical sphincters and their presence cannot be refuted on microscopy alone.

The cardiac mucosal folds in the infant appeared without exception to be of a rosette pattern. Minor variations were frequent but no special folds, as seen in the adult, occurred. There were no 'curtains' on the greater curve side and no evidence to suggest a valve mechanism. The cardiac orifice was mostly round: the centre of symmetrically radiating mucosal folds. These folds are bunched together by the inferior sphincter so that a watertight seal is formed at the cardia. Together, these two factors act in perfect harmony and constitute the normal closing mechanism between stomach and oesophagus (Botha, 1958b).

Partial Thoracic Stomach

It has been shown that cardiac competence and oesophageal fixation are proportionately much better in infancy than in the adult. A partial thoracic stomach which causes vomiting immediately after birth is therefore not merely a 'slight herniation', but a major derangement.

The occurrence of part of the stomach in the posterior mediastinum was before considered to be rare in children. Thomsen (1949), who reviewed 70 reported cases up to 1947, published 58 cases of his own in 1955. Carré, Astley and Smellie (1952) found reference to just over 100 cases, yet they found an annual incidence of 18 infants with a partial thoracic stomach at the Children's Hospital in Birmingham. As there is no reason to suspect any recent alteration in the incidence of the condition, it is reasonable to assume that most cases have remained undiagnosed in the past. There are several reasons for this; sliding hiatal hernia has only become a recognized clinical entity over the last 20 or 30 years, and then mainly in adults. Many centres have lacked diagnostic facilities as well as experienced radiologists in this field. Recent advances in thoracic surgery have made this region readily accessible, when once the disorder is diagnosed. Another important reason for improved understanding of this condition is that a widespread interest in the oesophagus and closing mechanism has developed in the last decade, referred to in the American literature as 'esophagology'.

A considerable difference of opinion exists as to the aetiology of this abnormality. The mechanism of sliding hiatal hernia in adults has been admirably described by Allison and other workers in numerous publications. Partial thoracic stomach in infants has subsequently been regarded, without reservation, as a similar condition that developed in the same way. Husfeldt, Thomsen and Wamberg pointed out in 1951 that this explanation does not directly apply to children. The findings now recorded strongly support their view. The present confusion is considerable, partly because the evolution of the disorder is not fully appreciated. It is essential, therefore, first to point out why the mechanism of partial thoracic stomach in infants and that of sliding hiatal hernia in adults is not the same.

Evolution of Sliding Hiatal Hernia. The outstanding feature of the above anatomical observation, is the small, powerful hiatus with secure oesophageal fixation in marked contrast to the findings in the elderly. It is therefore not justified to discuss 'hiatal hernia' in infancy by referring to adult anatomy (Thomsen, 1955). It cannot be accepted that a normally developed and situated stomach with normal attachments would herniate through a normal hiatus, either shortly before or after birth, as a result of extrinsic forces that might act on the viscus.

Within the first few years of life, however, certain changes become manifest in the anatomical relationships of the cardiac region. The hiatus enlarges and the tunnel becomes shorter and less oblique. The abdominal oesophagus lengthens and becomes less securely fixed so that freer movement takes place.

These changes are even more exaggerated in the adult, due to the continuous strain on one of the potentially weak spots of the abdominal wall. The crural muscle becomes thin, stretched and weak. The force acting on the hiatus continually tends to enlarge the opening, separate the crural limbs and diminish the posterior overlap. The hiatal tunnel looses its obliquity, becomes shorter and wider until ultimately it constitutes a direct hole through the diaphragm. The phreno-oesophageal membrane stretches to a thin, attenuated layer, located only with difficulty. The elastic fibres are fragmented, dissociated and less wavy. This encourages greater mobility, so that on straining the cardia is forced into the hiatus and the aperture is further stretched, thus setting up a vicious circle. Small additional strains in the form of obesity, posture, constipation, bronchitis, corsets, etc., are sufficient to force the cardia progressively through the weakened hiatus until it lies in the posterior mediastinum. A benign situation then rapidly becomes significant. Displaced into an abnormal position, deprived of the important support of the diaphragm, the closing mechanism acts at a functional disadvantage; and when subjected to a sudden, forceful jet of gastric contents, it must give way in one direction only—into the oesophagus. As the sphincter weakens, reflux occurs more readily, until the sensitive squamous epithelium is continuously exposed to the relentless activity of gastric juice.

Oesophagitis follows. The acute inflammatory reaction is gradually followed by chronic fibrosis. The hernia increases in size, partly due to pressure from below and partly due to pull from above (the negative pressure, suction force and the remarkable longitudinal retraction of the outer muscle coat). As the cardia ascends the oesophagus maintains its straight contour, but gradually the lower portion is fixed in this position by ulceration and scarring; thus the patient ultimately ends up with a short oesophagus complicated by a stricture. The many variations on this theme need no elaboration.

In the vast majority of adults, sliding hiatal hernia is an acquired condition that usually develops in the later decades of life without any obvious congenital abnormality. It signifies a degenerative change in a normal hiatal region which is closely analogous to certain types of herniation of the anterior abdominal wall or pelvic floor and subject to the same predisposing causes.

Partial thoracic stomach in infants, I believe, is a congenital abnormality in the great majority of cases. The following facts substantiate this view.

Age Incidence. In the vast majority of infants with partial thoracic stomach (proved radiologically), symptoms date from birth or the early neonatal period: this in itself is highly suggestive of a congenital origin. In a series of 58 children with 'hiatus hernia', Thomsen (1955) found only three cases which presented after the age of 7 years. Two of these had para-oesophageal herniae (14 and 15 years) and therefore fall into a separate group and in the other child symptoms were present from birth. More than half of the infants in this series were admitted to hospital within the first year of life.

On the other hand, the incidence of sliding hiatal hernia in the 10-20 year age group without any previous symptoms is extremely small. If partial thoracic stomach is the result of simple herniation as in the adult, the incidence must increase, or at least some cases must present during this time, as the hiatus gets weaker with advancing age; the condition should not become rare.

Sex Incidence. Although Astley (1956) gives the sex incidence as equal, Thomsen (1955), Greenwald and Steiner (1929) and Truesdale (1935) found a much higher incidence amongst boys. In adult herniae, females predominate.

Familial Incidence. 'Enough familial instances occur to indicate a hereditary factor; in 10% there are one or more other cases in the family' (Astley, 1956). There may be as many as three infants in one family. The condition also occurs in twins (Wamberg, 1947) and in mother and daughter (Myles, 1939). No familial tendency has been noted in adults with herniation.

Radiology. The hiatus is sometimes unexpectedly small in adult sliding herniae. Where the hiatus is wide, a large portion of the stomach usually lies in the chest. As small herniae get bigger, reflux occurs more easily and stricture may develop.

The hiatus in infants with partial thoracic stomach is on the whole surprisingly wide; it is out of proportion to that in the adult. This lax, wide, and at times conical or tented hiatus contrasts very sharply with the normal crural pattern. With this wide gap, it is reasonable to expect further deterioration and increase in the size of the hernia as the infant gets older, if in fact, thoracic stomach is due to simple herniation. Instead, the supradiaphragmatic loculus remains small and insignificant in the majority of cases; it may even get smaller—or disappear completely. Regurgitation, however, is very free and competence relatively poor; the mechanism at the cardia is disturbed out of proportion to its radiological appearance. The fact that this wide hiatus later assists cardiac control with

greater efficiency suggests that a congenitally deranged or maldeveloped diaphragm might with advancing age become more normal either by further development or by accommodation.

Clinical Course. In adults symptoms often have no relation to the radiological appearance of hiatus hernia; the tendency, however, is to deteriorate. Just the opposite happens in infants. The great majority follow a benign course and become completely asymptomatic by the age of 5 years with no subsequent relapse in childhood. The incidence of stricture varies in different centres. Untreated it is less than 10%; with adequate medical treatment it should be much less (Carré *et al.*, 1952).

Symptomatic cure does not necessarily imply restoration to normal anatomical relationships. Astley (1956) found that symptoms usually disappeared at the time of weaning to solids, although often gastro-oesophageal incompetence could still be demonstrated. At first reflux might occur from the abdominal to the thoracic portion of the stomach, although little barium passed back into the oesophagus. Later reflux ceased at the level of the diaphragm. This suggested to him an initial improvement in the sphincteric power of the oesophagus, followed later by improvement in the 'pinchcock' mechanism.

Surgery. The outcome of surgical treatment in partial thoracic stomach in infants is not nearly so good as in adults. This is the opposite to what might be expected. In at least a proportion of infants it means that either the oesophagus is congenitally short so that the tendency for the stomach to herniate is too great; or that the diaphragmatic crura are abnormal and therefore unable to maintain the stomach in its normal position after surgical repair.

Associated Abnormalities. The simultaneous occurrence of partial thoracic stomach and other malformations have been reported (Williams, 1945; Radloff and King, 1947; Thomsen, 1955; Astley, 1956; Kohler, 1957). The significance of this association is doubtful but it has been suggested as corroborative evidence in favour of a congenital origin of thoracic stomach. There is no indication that infectious diseases during pregnancy, complicated labours or prematurity have any relation to this condition. The interesting association of hypertrophic pyloric stenosis and partial thoracic stomach is too common to be disregarded. Roviralta (1952) described this combination as the 'phrenopyloric syndrome'. Astley and Carré (1954) found five infants with pyloric stenosis as well as three other children with 'infantile pylorospasm' in a series of 115 cases with thoracic stomach. Forshall (1955) reported a series of 93 cases of 'cardio-oesophageal syndrome' in childhood in which eight infants were subjected to Rammstedt's operation. Four were irrefutable cases of pyloric stenosis. It has been suggested that herniation follows as a result of the stenosis. However, this is not true in most cases: vomiting is often present from birth; the hiatus is normal in cases of pyloric stenosis alone; and symptoms as well as the radiological appearance persist after division of the pyloric sphincter.

It is reasonable to conclude that sliding hiatal hernia in the adult is an acquired, degenerative condition that affects a normal hiatal region. Partial thoracic stomach in the infant is either due to a congenital short oesophagus or to herniation of the stomach through a congenitally malformed or undeveloped diaphragm. The relative frequency of each type is unknown. Despite statements to the contrary, it is impossible in the great majority of these cases to identify, beyond doubt, the 'congenital short oesophagus' or the 'hiatal hernia'; therefore the non-committal term 'partial thoracic stomach' is preferred.

Congenital Short Oesophagus

Tonndorf in 1923 postulated that thoracic stomach is the result of incomplete descent of the gastric organ due to failure of the oesophagus to elongate. During the fourth to seventh weeks of embryonic life, the oesophagus lengthens rapidly to keep up with the extension of the pharynx and the development of the lung buds (Botha, 1958a). If this elongation fails, the oesophagus will be partly or completely shortened. The stomach will lie somewhere in the posterior mediastinum, not because it has herniated to that position but because it has never descended into the abdomen, and it may therefore occur anywhere from the pharynx to the diaphragm. A case of extreme short oesophagus is to be published elsewhere (1958c) where the entire oesophagus consisted of a constriction of 2 or 3 mm. (Figs. 9 and 10). The stomach was in the neck, covered by peritoneum that stretched upwards like a funnel. The cardiac angle was obliterated. The poorly developed membranous diaphragm was tented up over the stomach and spleen. In the place of the hiatus was a huge, wide defect. Iniencephaly and cleft palate were also present. This maldevelopment of the diaphragm is secondary to insufficient lengthening of the oesophagus. This extreme degree of shortening, of which a previous description has not been found, must be relatively rare. More commonly the

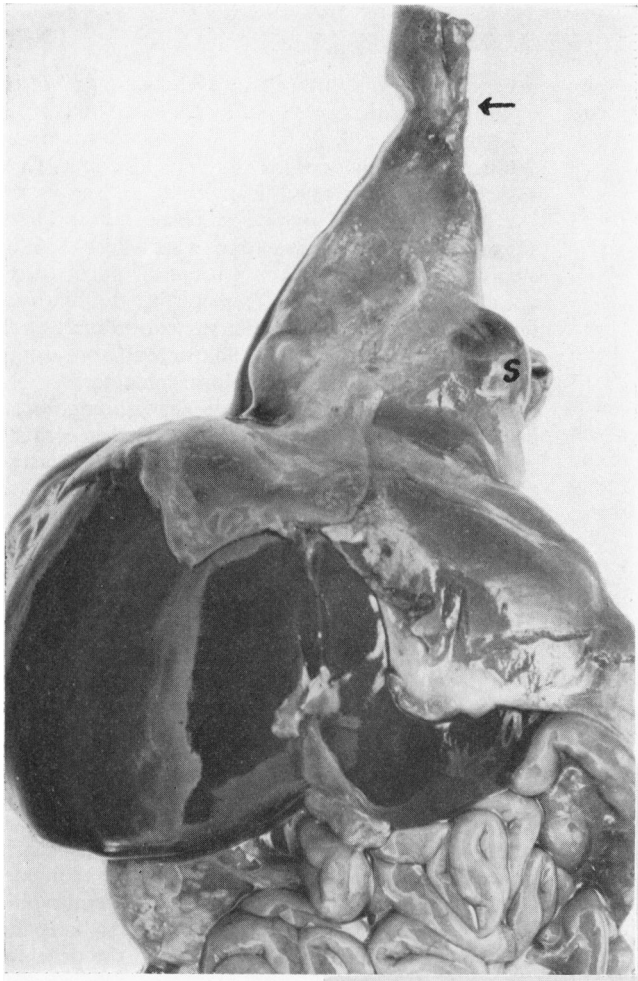


FIG. 9.—The mediastinal organs and abdominal viscera of an iniencephaly which shows complete failure of the oesophagus to elongate. The spleen (S) lies in the chest enveloped by a thin serosal membrane.

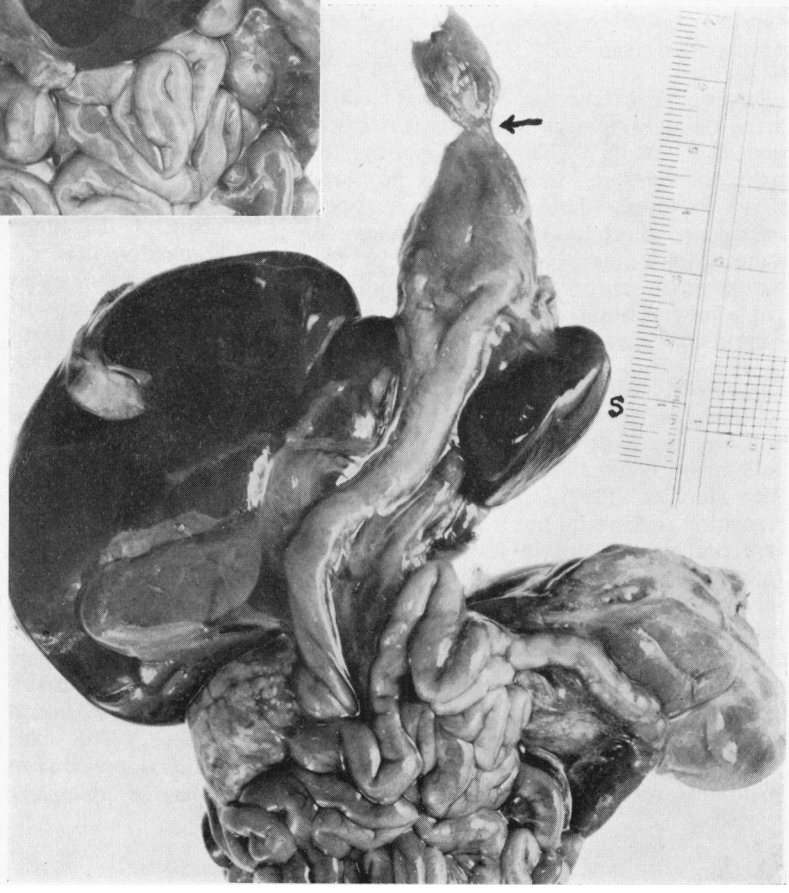


FIG. 10.—The serosal membrane in the specimen in Fig. 9 is removed. The arrow indicates the oesophagus, a mere constriction between stomach and pharynx.

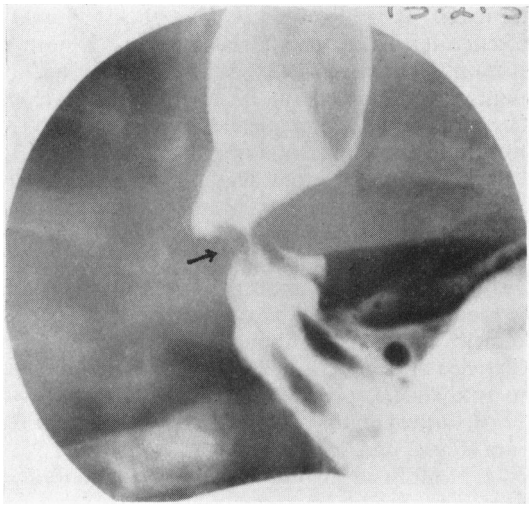


FIG. 11.—Partial thoracic stomach in an infant. The arrow indicates the 'empty segment', which is due to the contraction of the inferior oesophageal sphincter.

oesophagus extends to or below the level of tracheal bifurcation.

The oesophagus has remarkable powers of retraction. The organ may therefore shorten, after herniation of the stomach (as in a simple hiatus hernia), without any signs which might indicate its true length. It may become secondarily fixed in this position due to inflammatory changes which follow oesophagitis and ulceration; the shortening may be further aggravated by chronic fibrosis and scar contraction. Gastric mucosa is then present above the hiatus and the supradiaphragmatic loculus of stomach may in the course of years elongate to resemble the lower third of the oesophagus very closely. This well known clinical entity, which is not uncommon, is truly called 'short oesophagus' although it is not a 'congenital short oesophagus', a confusion of identity which has led to considerable misunderstanding. For this reason Tonndorf's conception fell into disrepute, especially after the papers of Johnstone (1943), Allison (1948) and others, so that later authors regarded congenital short oesophagus as a rare anomaly (Belsey, 1954; Thomsen, 1955 and Forshall, 1955). Certainly, many of the cases reported as congenital short oesophagus were really due to acquired shortening with associated hiatus hernia—described at a stage when the mechanism of hiatal herniation was imperfectly understood; but the diagnosis of congenital short oesophagus cannot be ruled out in the infant who vomits from birth and shows the following radiological features (Fig. 11): (1) A small, tubular, gastric loculus above the diaphragm; (2) a relatively fixed cardia above the diaphragm as evidenced by the distal border of the 'empty segment'. (This is due to the constriction of the inferior oesophageal sphincter and has been wrongly attributed to 'spasm' or 'stricture'). (3) A straight oesophagus; (4) obliteration of the cardiac angle; (5) a very wide, lax hiatus which may be tented.

The complete absence of oesophagitis in many infants with partial thoracic stomach entirely excludes any retraction or fixation due to chronic inflammatory scarring. Because oesophageal changes developed after one or two years in 'sliding hernia' where the gullet was normal before, Thomsen (1955) concluded that 'as is the case in adults, the shortening is due to reflux oesophagitis'. However, congenital short oesophagus necessarily implies that part of the stomach lies above the hiatus. As this deranged, insecurely fixed viscus is subject to all the forces that normally predispose to herniation, the loculus of stomach above the diaphragm may slowly increase in size. The normal closing mechanism is further disturbed, displaced and deprived of the important support of the crura. The reflux that follows leads to oesophagitis, fibrosis and more shortening. Thus 'congenital short oesophagus' and 'sliding hiatal hernia' may produce an indistinguishable end-result, and may co-exist.

Sliding Hiatal Hernia Due to a Malformed Hiatus

Instead of ascribing a primary inhibition of growth in the lower oesophagus (Tonndorf, 1923; Kelly, 1936; Lelong, 1947) as a cause for thoracic stomach, Bund (1918), Bailey (1919) and Åkerlund (1926) blamed the lack of fixation of the cardia on a persistence of the pneumato-enteric canal. This is considered unlikely because a relatively big thoracic stomach is sometimes seen at birth and even the biggest bursa should not allow this degree of mobility. Furthermore, little evidence of this peritoneal recess has been found at operation and the hiatus, which should be normally developed if the peritoneal sac is the only factor, is often abnormal. Harrington's (1940) theory that descent of the stomach is disturbed, with poor subsequent fixation to a wide hiatus, cannot be substantiated embryologically (Botha, 1958a). Shanks (1948) felt that the hiatus was congenitally enlarged, but the crural muscle was normal. Dunhill (1935 and 1948) described hiatal hernia with congenital absence of the left crus—the aorta bowed far to the left. Although this type of anomaly occurs it must be rare, and, relatively speaking, not an aetiological factor of clinical importance.

Bremer (1943) stated that hernia in the region of the tendinous antrum on the left side near the

oesophagus is less common than herniation through the trigonum lumbocostale. He thought it 'due to failure of the last step in the closure of the pleural passage, the growth above the descending stomach of the thin strip of liver and septal extension to join the mediastinum. Except for the large opening the diaphragm in this type is normal'. This writer's view on the development of the diaphragm cannot be accepted (Botha, 1958a) and no clinical or experimental evidence has been found to support his theory.

Numerous vague references to 'hiatal insufficiency' appear in the literature (Allison, 1948; Johnstone, 1943), but no satisfactory explanation has been advanced as to the cause of herniation in children. Forshall (1955) found at operation in 30 cases of partial thoracic stomach that the right crus invariably appeared poorly developed, especially the left limb and the posterior fibres. The diaphragm as a whole was often unduly thin, almost flaccid to the touch. She thought that a poorly developed hiatus is the congenital element of this sliding hernia in which the cardia is drawn through the hiatus by the difference in intra-thoracic and intra-abdominal pressures. Her operative findings, supported by the findings at operation of other workers, contrast so sharply with the normal anatomy of the crura that one must accept some congenital maldevelopment at the hiatus.

The nature of this abnormality is not yet known, but the following suggestions may be considered:

(1) Congenital absence of one or both crura is rare.

(2) Failure of muscularization of hiatal muscle so that a membrane forms at the site of the crura which envelops the cranial portion of the stomach (Christiansen, 1941). However, muscularization of the diaphragm begins in the 12 mm. embryo (Botha, 1957b), when the oesophagus has already become fully elongated and the stomach has reached its normal position. There is, furthermore, no selective failure of the muscle cells to invade the septum transversum so that the rest of the diaphragm must show signs of such an anomaly if it does occur. In any case, muscle fibres are easily demonstrated in the hiatal margins in cases of partial thoracic stomach at operation and necropsy.

(3) The wide hiatus, with no muscular defect. *X*-ray appearances certainly confirm the wide hiatus. It is difficult to understand though, why infants should be born with such a wide opening in the diaphragm and the rest of the anatomy normal. A study of the embryology and development definitely points against this theory. If the oesophagus is of normal length and such a big hole exists, one would expect a progressive, large, freely movable hernia; partial thoracic stomach is small and fixed. It is also unlikely that such a primary defective lax hiatus should later resume a normal function. Thomsen (1955) stated that 'hernia' in infants is not present at birth but only develops afterwards as a consequence of the change in pressure in the thorax and abdomen, and is due to a 'lacking or abnormal fixation of the cardiac region'. From what has been said before, this statement cannot be accepted.

Very large, mobile herniae are sometimes found where the oesophagus appears of normal length and they are quite unlike the usual case of partial thoracic stomach. Although it may only be a difference of degree, it is thought that these herniae are true sliders, probably in part due to either a poorly formed hiatus or atypical arrangement of the crura (Fig. 3 and 1d).

(4) Malformed hiatus secondary to congenital short oesophagus. When the oesophagus fails to elongate, the cardia is retained in the chest. The serosa is drawn out to this level and may form a membranous sac around the upper stomach. Crural development has been interfered with; the fibres are attenuated and underdeveloped; the crura are thin and the wide hiatus is tented upwards. In the very short oesophagus (Figs. 9 and 10; Christiansen, 1941, and many others) the phreno-oesophageal elastic and serosal attachment to the high cardia has attenuated the crura to an insignificant membrane-like covering. When the abnormality is minimal, a wide hiatus and poorly developed crura, as found at operation by Forshall, may be the only anatomical signs of abnormality.

Smithers (1945) found it 'difficult to believe that short oesophagus has nearly always been overlooked at post-mortem'. However, after spending some time in the necropsy room examining hiatuses of normal subjects as well as others with hiatus hernia, I fully sympathize and agree with Anders and Bahrmann (1932) and other authors on this subject; the degree of herniation is difficult if not impossible to assess after death.

It has been said that reduction of a partial thoracic stomach at operation refutes the diagnosis of a congenital short oesophagus. This is not necessarily true. The gullet not only possesses amazing qualities of retraction but also of extension. The hiatus in the adult may move as much as two and a half vertebral bodies during respiration (Botha, 1957b). At operation, with the patient in the supine position, the oesophagus is mobilized to 'above the aortic arch level' and pulled down while the diaphragm is completely paralysed by relaxants. The cardia, after reduction, is maintained in the sub-diaphragmatic position by approximating the crura and fixing the

oesophagus to the hiatal margin. This procedure might be impossible on inspiration or in the erect posture !

In conclusion, partial thoracic stomach is a relatively common disorder in infancy that might be due to congenital short oesophagus or a sliding hernia (or a combination of these conditions).

The two abnormalities cannot be distinguished in the majority of patients either by radiography or by endoscopy. I believe that a large proportion of instances of partial thoracic stomach are due to a congenital short oesophagus—a far greater percentage than has hitherto been recognized.

Para-oesophageal Hernia

Para-oesophageal herniae are uncommon. A portion of the stomach, usually the fundus, herniates through a relatively wide but otherwise normal hiatus while the cardia maintains its normal position. Cases in which the gastro-oesophageal junction has been displaced upwards, whether primary or secondary, cannot be regarded as true para-oesophageal types, but of a mixed variety. Para-oesophageal herniae are seldom present in infancy but usually occur later in childhood, adolescence or in the adult. They are always on the right side: left-sided ones are either of the mixed type or diaphragmatic. As the closing mechanism is acting in its normal position with optimum function, reflux and oesophagitis are unusual. However, this type of distortion frequently leads to ulceration, haemorrhage and even perforation.

The peritoneal sac which is commonly found is undoubtedly derived from the right pneumato-enteric recess (Åkerlund, 1926; Barrett, 1954; Botha, 1958a). Examples of these small empty sacs have been found on the right side as well as posteriorly, without any sign of herniation or any other abnormality around the hiatus. A large sac clearly invites early herniation, whereas the small ones remain as potential spaces which might later give way under strain. It then leads to a para-oesophageal hernia, a mixed hernia or it might weaken the attachment sufficiently to predispose to an ordinary sliding hernia with a sac. Harrington (1940, 1948) and Olsen and Harrington's (1948) views that these herniae, as with the sliding varieties, are due to congenital dilatation of the hiatus have not been verified.

Oesophagus Lined with Glandular Epithelium

Barrett (1950) first aroused interest in a condition which he called 'oesophagus lined by gastric mucous membrane'. Unfortunately this has been confused with congenital short oesophagus.

I have already indicated that some variation exists as to the level at which the squamous epithelium changes to glandular mucosa. In the adult, this variation is more marked and the level may occur anywhere from just below the cardia to the cricoid (Barrett, 1954). The epithelium is, however, not gastric epithelium, but glandular epithelium that closely resembles the 'zone of cardiac glands' in animals and man (Botha, 1958d). The tubules are short and tortuous; mucus-secreting alveoli are in abundance and oxyntic cells are either absent or very scanty. It is suggested that these glands represent a more primitive epithelial cell type, which, for one or other reason, has not fully differentiated into squamous epithelium (Botha, 1958d). There is evidence that these aberrations might predispose to oesophagitis and ulceration (Allison and Johnstone, 1953). Normally this condition has no relation whatsoever to short oesophagus or hiatus hernia. It might, however, be present in the lower oesophagus of a patient who later develops a hernia or it might line the lower portion of an oesophagus that is congenitally short, but it is then unusual and purely coincidental. Some of the cases reported as of gastric-lined oesophagi are actually cases of hiatal hernia or congenital short oesophagus where the cardia lies well above the diaphragm. One of the main reasons for this misconception is the view held by some workers that the squamo-glandular junction is the dividing line between stomach and oesophagus. If this is used as a standard, some animals will either possess an oesophagus and no stomach or others will only have a stomach without a gullet (Botha, 1958d). The site at which squamous and glandular epithelium meet cannot be regarded as the dividing line between stomach and oesophagus in man; it so happens that in the vast majority of people it takes place at or near the cardia.

Cardio-oesophageal Relaxation or 'Chalasia'

Neuhauser and Berenberg in 1947 reported 12 infants with 'persistent vomiting evidently due to relaxation or dysfunction of the hiatus portion of the esophagus with failure of the normal "sphincter" action of the cardia'. They called this 'cardio-esophageal relaxation or chalasia'. With conservative treatment symptoms disappeared after a few months and the infants showed no physical or fluoroscopic abnormalities. It was thought to be a temporary neuromuscular dysfunction of the hiatus portion of the oesophagus and diaphragm (Berenberg and Neuhauser, 1950). Forshall (1955) preferred the term 'lax oesophagus', because 'it is not only the cardia which is relaxed but also the whole lower two-thirds of the oesophagus'. However, it has been shown that the lower oesophagus in normal

infants is characteristically wide, readily distensible and even sac-like on occasions, with irregular contractions and contours (Lelong and Aimé, 1934; Bakwin, Galenson and Le Vine, 1944; Thomsen, 1955; Henderson, 1942; Malenchini, Roca and Banzas, 1948).

There are other reasons why this condition is not likely to be a neuromuscular imbalance: the hiatus is usually wide and lax though there is no obvious autonomic connexion with the phrenic nerves or the diaphragmatic crura (Botha, 1957a). The cardiac angle is lost (Forshall) and the cardia itself appears on a higher level than normal, as if it has been ' drawn up ' into the hiatus. Drugs have no effect on this condition but recovery is remarkable in the great majority by nursing in the sitting posture. In adults, the level of the diaphragm as seen radiologically does not necessarily correspond to the level of the hiatus (Botha, 1957b). This is probably true in children, to a lesser degree, which makes it impossible to say with certainty that the cardia is below, at, or above the hiatus. One knows that in at least some of these cases, partial thoracic stomach later develops (and not necessarily associated with oesophagitis). Post-mortem and operative findings cannot be regarded as reliable. The radiological appearances are often extremely difficult to interpret and one may still be uncertain as to the diagnosis even after several examinations (Astley and Carré, 1954).

I believe that patients with 'chalasia' really suffer from mild degrees of partial thoracic stomach, but instead of the cardia being clearly in the chest, it is lying in or just above the hiatus. This slight distortion is sufficient to render the normal closing mechanism incompetent. Some deteriorate into sliding herniae. The great majority are treated medically in the sitting posture with permanent cure. Gravity assists the descent of the cardia to its normal position and thus normal competence is re-established.

Summary

The anatomy of the gastro-oesophageal region in more than 115 infants and children has been studied. The outstanding features were: a small powerful hiatus with marked overlap and an oblique lengthy tunnel; a formidable phreno-oesophageal membrane and secure fixation of a narrow terminal oesophagus. These features were considered to be mainly responsible for the superior gastro-oesophageal competence in infancy. In contrast, hiatus hernia in the elderly is a terminal degenerative manifestation in the evolution of the cardiac region.

Differences in age, sex and familial incidence, clinical course, radiological features, associated abnormalities and in the outcome of surgery support the view that the mechanism of partial thoracic stomach in infancy and hiatus hernia in adults are not identical conditions. The noncommittal term 'partial thoracic stomach' is preferred as it is impossible in the great majority of cases to distinguish between sliding hiatal hernia and congenital short oesophagus.

Evidence suggests that a large proportion of cases of partial thoracic stomach are due to congenital short oesophagus.

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References

Åkerlund, A. (1926). *Acta radiol. (Stockh.)*, **6**, 3.
Allison, P. R. (1948). *Thorax*, **3**, 20.
—— and Johnstone, A. S. (1953). *Ibid.*, **8**, 87.
Anders, H. E. and Bahrmann, E. (1932). *Z. klin. Med.* **122**, 736.
Astley, R. (1956). *Radiology of the Alimentary Tract in Infancy*. London.
—— and Carré, I. J. (1954). *Radiology*, **62**, 351.
Bailey, P. (1919). *Anat. Rec.*, **17**, 107.
Bakwin, H. Galenson, E. and Le Vine, B. E. (1944). *Amer. J. Dis. Child.*, **68**, 243.
Barrett, N. R. (1950). *Brit. J. Surg.*, **38**, 175.
—— (1954). *Ibid.*, **42**, 231.
Belsey, R. (1954). *Ann. roy. Coll. Surg. Engl.*, **14**, 303.
Berenberg, W. and Neuhauser, E. B. D. (1950). *Pediatrics*, **5**, 414.
Botha, G. S. Muller (1957a). *Thorax*, **12**, 50.
—— (1957b). *Lancet*, **1**, 662.
—— (1958a). *Anat. Rec.* In the press.
—— (1958b). *Brit. J. Surg.* In the press.
—— (1958c). To be published.
—— (1958d). *Acta Anat. (Basel*, In the press.
——, Astley, R. and Carré, I. J. (1957). *Lancet*, **1**, 659.
Bremer, J. L. (1943). *Arch. Path. (Chicago)*, **36**, 539.
Bund, R. (1918). *Frank. z. Path.*, **21**, 243.
Carré, I. J., Astley, R. and Smellie, J. M. (1952). *Lancet*, **2**, 1150.
Christiansen, H. (1941). *Acta radiol. (Stockh.)*, **22**, 360.
Collis, J. L., Kelly, T. D. and Wiley, A. M. (1954). *Thorax*, **9**, 175.
Dunhill, T. (1935). *Brit. J. Surg.*, **22**, 475.
—— (1948). In *British Surgical Practice*, ed. E. Rock Carling and J. Paterson Ross, Vol. 4, p. 451. London.
Forshall, I. (1955). *Arch. Dis. Childh.*, **30**, 46.
Greenwald, H. M. and Steiner, M. (1929). *Amer. J. Dis. Child.*, **38**, 361.
Harrington, S. W. (1940). *Amer. J. Surg.*, **50**, 381.
—— (1948). *Surg. Gynec. Obstet.*, **86**, 735.
Henderson, S. G. (1942). *Amer. J. Roentgenol.*, **48**, 302.
His, W. (1903). *Arch. Anat. Physiol. [Anat. Abt.]* (Lpz.), p. 345.
Husfeldt, E., Thomsen, G. and Wamberg, E. (1951). *Thorax*, **6**, 56
Johnstone, A. S. (1943). *Brit. J. Radiol.*, **16**, 357.
Kelly, A. B. (1936). *J. Laryng.*, **51**, 78.
Kohler, H. G. (1957). Personal communication.
Lelong, M. (1947). *Paediat. danub.*, **2**, 65.
—— and Aimé, P. (1934). *Arch. Mal. Appar. dig.*, **24**, 305.
Lerche, W. (1950). *The Esophagus and Pharynx in Action*. Springfield, Illinois.
Low, A. (1907). *J. Anat. (Lond.)*. **42**, 93.
Lyons, W. S., Ellis, F. H. and Olsen, A. M. (1956). *Proc. Mayo Clin.*, **31**, 605.
Malenchini, M., Roca, J. and Banzas, T. (1948). *Pren. méd. argent.*, **35**, 1698.
Myles, R. B. (1939). *Brit. J. Radiol.*, **12**, 645.
Neuhauser, E. B. D. and Berenberg, W. (1947). *Radiology*, **48**, 480.
Olsen, A. M. and Harrington, S. W. (1948). *J. thorac. Surg.*, **17**, 189.
Radloff, F. F. and King, R. L. (1947). *Gastroenterology*, **9**, 249.
Roviralta, E. (1952). *Les Vomissements du Nourrisson*. Paris.
Shanks, S. C. (1948). *Brit. J. Radiol.*, **21**, 55.
Smithers, D. W. (1945). *Ibid.*, **18**, 199.
Thomsen, G. (1949). *Acta radiol. (Stockh.)*, **32**, 193.
—— (1955). *Ibid.*, Suppl. 129.
Tonndorf, W. (1923). *Dtsch. Z. Chir.*, **179**, 259.
Truesdale, P. E. (1935). *New Engl. J. Med.*, **213**, 1159.
Wamberg, E. (1947). *Acta paediat. (Uppsala)*, **34**, 293.
Williams, E. R. (1945). *Brit. J. Radiol.*, **18**, 323.