There would seem to be little doubt that a toxin or an allied substance acting on the marrow is at the root of the production of the disease. Here, by its irritative or stimulating action, it produces a specific type of marrow change. There is no evidence for the view that the lesions in the various organs in pernicious anaemia are produced directly by the same toxin or by a variety of different toxins. Everything suggests that the conditions in the nervous system, stomach, and liver (in the later stages) are produced secondarily by the anoxaemic state brought about by the primary marrow lesion. Any other view would seem to be incapable of explaining how such diverse causes as those producing idiopathic pernicious anaemia, sprue, and the pernicious anaemia of pregnancy, syphilis, tapeworm infestation, malaria, etc., can so uniformly bring about what Vaughan calls these "oddly associated lesions."

Summary.

The essential and primary factor in the production of pernicious anaemia is the involvement of the bone marrow in an irritative process, which causes it to revert to embryonic cell formation.

The resulting anoxaemia causes, secondarily, the complications which are to be found in the central nervous system, the stomach, and the liver (later stages).

There is no evidence for the existence of a "pernicious anaemia diathesis" either in relation to the disease as a whole or to the organ complications.

I wish to express my indebtedness to Mr. Archibald Macdonald, M.A., B.Sc., for supplying the fowls; to Dr. Marion B. Richards and Mr. Kenneth McKenzie, M.Sc., for preparing the photographs; and to the Carnegie Trust for the Universities of Scotland for a grant towards their reproduction.

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THORACIC STOMACHS:

A STUDY OF PHARYNGEAL POUCH AND CARDIOSPASM, WITH REPORT OF THREE CASES.

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(With Special Plate.)

For several decades occasional observers have reported cases of simple dilatations of the oesophagus resembling adventitious stomachs, but they are only now becoming readily recognized. For this recent facility in diagnosis we have to thank Roentgen. Radiography, after the ingestion of an opaque meal, makes the diagnosis of such

FIG. A.—Showing upper and lower thoracic stomachs in profile in the chest.

intrathoracic enlargements one of easy and convincing clarity instead of recondite conjecture, as when the doctor depends wholly on clinical acumen.

These expansions are found at the upper and lower ends of the gullet. In shape and in their relationship to the parent gullet they resemble aneurysms, such as occur in the adjacent aorta. The upper is saccular and the lower fusiform in outline. A diagram of them in profile indicates their deep dwellings in the chest—and the reason for the picturesque title of these remarks (Fig. A). As a matter of fact, the title is not an abuse of the word "stomach," but a reversion to its proper use, for "stomach" is derived from the Greek word meaning "gullet," and Galen, among other anatomists, preferred the name "ventricle" for the subdiaphragmatic stomach. They are familiar as "pharyngeal pouch" or "diver-

ticulum," the lower one occurring in the condition known as cardiospasm. Diaphragmatic hernia of the normal stomach into the thorax is not included in this paper.

It is hoped that reasons other than the mere infrequency and pathological curiosity of these conditions will make this communication helpful. A doctor's duty to his patients makes it necessary to recall these disorders to mind from time to time. Their remoteness in the hidden depths of the chest hides them from simple view, and the old saying still holds good—"Out of sight, out of mind." While failure to recognize some lesions makes little practical difference to the patient, this is not true in the case of thoracic stomachs. They disable the victim, progressively and gravely, if left to nature, but, despite their inaccessibility, they are amenable to sure relief by surgery. Reports on such cases, rare but vital, keep the doctor on the look out for them.

A few notes of illustrative cases will serve as a basis for consideration of the clinical features of these alimentary pouches.

CASE I.

W. H. C., aged 62, was a monumental mason, but had only been a supervisor for the last fifteen years. He came to me in August, 1929. His past health had been good. He stated that for ten years queer audible noises occurred during swallowing. Three years ago vomiting—as from a pump—started and brought relief. For nine months there had been increasing dysphagia and vomiting. This was lately so severe that life had become unbearable; he was rapidly losing weight, and had increasing constipation for five months. He sought urgent relief in order to nourish himself. He had been disposed to heart attacks, having fainted three times in the street, and he had swelling of the feet.

On examination he was somewhat pale and spare in flesh. His teeth were offensive. There was no swelling in his neck, but there was a feeling of food stoppage behind the upper ribs. A vomit of six ounces by his bed looked like creamy tomato soup. It was acid to litmus, due to decomposition, not, presumably, to admixture with gastric juices, and inoffensive in odour. Vomiting occurred within, at the most, a few minutes of ingestion. Pharyngoscopy by Mr. Angell James revealed the walls of the pouch, but no exit.

He was radiographed after the ingestion of an opaque meal, when the barium was seen to occupy a substernal pouch the shape of half a small orange, with the flat surface upwards (Figs. 1 and 2). It was median, with a slight bulge to the right, and its smooth, convex lower aspect was at the level of the fourth dorsal vertebra.

Similar cases reported in the literature occur mostly in patients of the fifth decade, and are of several years' duration. Sensations of strangling and of troublesome coughing on recumbency are noted, as well as regurgitation of food on pressure of the left side of the neck. The patient referred to above showed diminished nutrition, for thoracic stomachs, unlike their abdominal congener, boast no digestive attributes, but rather impede the process.

A. WILFRID ADAMS: THORACIC STOMACHS.

FIG. 1.—Skigram of W. H. C., showing pharyngeal pouch, antero-posterior view.

FIG. 2.—Skigram of W. H. C., showing pharyngeal pouch, lateral view.

FIG. 3.—Photograph of W. H. C., showing pharyngeal pouch stitched to the surface of the skin.

FIG. 4.—Dilated lower gullet of W. S.; case of cardiospasm. (1923.)

FIG. 5.—Mercury bougie entering stomach of W. S. (1930.)

FIG. 6.—Lower thoracic stomach of A. F.; case of cardiospasm.

Operative Treatment.

Following the method used by Wilkie,¹ diverticulectomy was commenced on September 4th, 1929, and the second stage of the operation was completed on the 18th. The pouch was exposed by an incision along the anterior border of the left sterno-mastoid muscle between the thyroid gland and larynx medially and the carotid sheath laterally. Resting on the prevertebral muscles was a smooth, bluish-yellow, flappy, membranous structure. It was like a thick-walled hernial sac, disclosing boundaries and coming to resemble a bag as it was gently dissected free of the vertebrae at the back and the viscera in front. The recurrent laryngeal nerve was guarded. To help to define the neck of the sac at this stage, an oesophageal bougie was passed by Dr. Stock with ease, demonstrating the absence of stricture. Here and there a few vessels coursed over the sac and were ligated, but bleeding was not troublesome. The sac was pulled on to the neck, and maintained there by stitching it to the skin margins (Fig. 3). The patient swallowed solids joyfully after this operation, and suffered only mild cellulitis. The second stage consisted in dissection of the pouch to its neck, which was ligated; the fundus was cut off, and the stump cauterized.

The patient was able to eat anything after this, and returned home and was about for a short time. During this period the neck healed soundly. He then had an attack of influenza followed by pyrexia, and died in four months. When he was radiographed in November an opaque meal dropped straight to his stomach; no trace remained in his neck. Professor Nixon saw him on November 12th, and stated that his poor health had nothing to do with the operation.

A rival procedure for diverticulectomy is diverticulopexy—that is, fixation of the inverted sac—so successfully carried out by Hill² in a series of fifty cases.

Before studying the nature and cause of the upper thoracic stomach a few notes on two examples of the lower thoracic stomach of cardiospasm will be given.

CASE II.

W. S., a man aged 50, complained of dysphagia of only two years' duration. His past history included four years in the trenches in the late war, and was good, apart from a tendency to a winter cough. He felt food arrested on a level with the sternum, and usually vomited his food after taking it. Cough aggravated the condition. Fluids were swallowed better than solids. He was a big man, and had lost much weight. His teeth were carious, but he had a fair colour. Nothing abnormal was found, except that auscultation revealed an interval of ten seconds between swallowing a drink and the entry of the fluid into the stomach. An opaque meal showed a fusiform swelling of the lower oesophagus (Fig. 4). A diagnosis of cardiospasm was made.

Operative Treatment.

Mikulicz's method, recently revived by Walton,³ was followed. Walton gives a timely warning on the need for lavage and emptying of the stagnant oesophagus prior to operation. The stomach was opened and the index finger passed into the contracted cardiac orifice. The remaining digits were also introduced one by one till the resistance yielded. Thereafter he was able to swallow any kind of food; he put on weight, and worked. In April this year the symptoms recurred in association with bronchial trouble, and he vomits if he takes his food too quickly. Radiography revealed food passing, undelayed, through the lower oesophagus. A mercury bougie was used for the relief of his symptoms, and a radiogram shows it *in situ* (Fig. 5).

CASE III.

A. F., aged 48, whose trouble started gradually in 1924 with an occasional meal causing a block in the gullet, had a good past history, apart from nervous debility in 1912-14. He was specially affected by greasy foods and cold liquids. Severe retrosternal pain, often rising to a crisis, disappeared when his food passed a certain part, leaving him exhausted. On the morning after, he often vomited unaltered the food he had eaten the day before. Blood was never present. His radiogram, after an opaque meal, is reproduced as typical (Fig. 6). It shows a large lower thoracic stomach, with its epiphrenic contour characteristically smooth. The fine, oblique trickle of barium that alone escapes into the stomach is a constant feature of these cases. It is notable that the constriction is a tunnel about 1 inch long, and not a mere ring at the cardiac orifice. It corresponds to the normal so-called cardiac sphincter of the oesophagus (Hurst),⁴ where that tube passes through the diaphragm. This appearance led Chevalier Jackson to apply the term "phrenospasm" in preference to "cardiospasm." With such a picture and history endoscopy seems as superfluous as with the one illustrating pharyngeal pouch.

Operative Treatment.

As in the former case, Mikulicz's digital dilatation was adopted, and the fingers were introduced up to the proximal interphalangeal joints, as advised by Walton. Convalescence was smooth, except

for a slight messy discharge from an upper stitch hole, which lasted a few weeks and was possibly caused by a slight leakage from the gastrotomy. He is a stone heavier, and is still very pleased to swallow normally, not having had a decent meal for years prior to the operation.

The best alternative treatment is the use of mercury bougies of increasing diameter, as prescribed by Hurst; but for inveterate cases, such as the above, I am in favour of Mikulicz's measure, so ably endorsed by the skilful practice of Walton.³

Dilatation with the hydrostatic bag of Plummer⁵ is open to the grave objection that during its use accidental rupture of the oesophagus can be less easily guarded against than with digital dilatation, and if rupture occurs it cannot be repaired at the time, as in the open method.

In the diagnosis of these maladies a detailed discussion of symptomatology is no longer necessary, since the radiogram taken routinely in dysphagia reveals a shadow characteristic both in site and in level. Thus it readily distinguishes the mild, transient, oesophageal spasm of functional troubles, thoracic aneurysm, and other mediastinal swellings. With the comparatively rapid formation of cancerous obstruction in the oesophagus little dilatation develops, and with this type of stricture the lower end of the barium column is uneven where it rests on the cancer.

NATURE AND ETIOLOGY.

A comparative study of the upper and lower types of thoracic stomach reveals more about both than when each is considered separately. Common to both is this important feature—namely, that the site of origin of each corresponds with a transition stage in alimentary transport. At the upper end is the change over from voluntary to automatic action, and at the lower, from the simple tubular oesophagus to the specialized function of the normal stomach.

It is notable, on examining the structure of their walls, that the epiphrenic stomach consists of the hypertrophied muscular coats of the oesophagus, and shows all the usual characteristics of muscular tubes proximal to an obstruction, while the pharyngeal pouch is merely a mucosal hernia with a thin areolar investment which hangs, as a blind bag, at the back of the main alimentary tube. For the lower stomach, dilatation of the oesophageal exit through the diaphragm is needed and adequate; in the upper pouch there is no constriction of the normal passage nor, if there were, could its dilatation remove the pouch that hangs outside the tube. These differences arise from the different structures and mechanisms of the entry and exit of the gullet. During deglutition the food bolus is squeezed from the mouth into the pharyngeal funnel. The longitudinal muscles—for example, the stylo-pharyngeus and the palato-pharyngeus—then raise this funnel at its upper end, and circular contraction squeezes the bolus into the narrower hypopharynx. Here the bolus is handed over to the involuntary peristalsis of the gullet. It is a region of stress, and also of inherent weakness. The signet of the cricoid ring forms a projecting hump anteriorly (Fig. A), while the rest of the circumference is encircled by the crico-pharyngeal portion of that compound muscle known as the inferior constrictor. Little longitudinal support is present, and the osseo-muscular ring, above described, receives the assault of the bolus descending into its smaller circumference from the roomier upper pharynx. It is a segment of strain owing to the narrowing lumen and to transition from voluntary to automatic transport of food. The subtle mechanism of adjustment may easily be thrown out of gear. Oral sepsis, pharyngitis, and oesophagitis, or even subphrenic ulceration, may raise the sensibility of the part and so set up protective hypertonus at the oesophageal doorway. Just outside the resistant door lies the inherently weak posterior area of the pharyngeal walls (Negus⁶) between the crico-pharyngeal and thyropharyngeal elements of the inferior constrictor. No actual congenital pouch exists. When the crico-pharyngeus fails to relax smoothly the pent-up ingesta press on the posterior pharyngeal wall, and the weak area yields. The bulge is directed down behind the oesophagus by gravity and mutual pressure of the thyroid gland and the bulkier

pharynx above. When large enough a descending bolus will drop more readily into it than into the mouth of the oesophagus, which is displaced forwards and comes to look backwards. The oesophageal orifice is like the slit of a letter-box, past which the descending bolus readily drops, and falls instead into the open mouth of the pouch below (Fig. B). They are shown respectively as X and Y in the diagram.

FIG. B.—Diagram showing pharyngeal pouch as a mucosal hernia.

That the diversion of the food is not caused by stricture of the oesophagus is proved, as in the case of W. H. C., by the immediate ease with which food passes as soon as the pouch is elevated from the thorax by operation and pulled out on the neck. The relation of X and Y is simply reversed by this manoeuvre, and the orifice of the pouch now presents a narrow slit on the side of the pharyngeal wall, while the mouth below leads straight down the red lane to the stomach (Fig. C). Hence the rationale of the operations previously mentioned.

FIG. C.—Diagram showing rationale on which diverticulectomy and diverticulopexy are based.

Development of the Lower Thoracic Stomach.

Just above the oesophageal exit arises the epiphrenic stomach involving, to varying heights, the lumen of the gullet above. Below the dilatation the lumen of the terminal inch of the gullet, when examined radiographically or by the sense of touch at operation, is but a tiny tunnel through which only a trickle of barium escapes. Valuable necropsies, such as that of Morley⁷ on such a case, confirm the truth of this, and establish the absence of muscular hypertrophy at the constriction.

Numerous causes for this shrunken calibre have been advanced. Among them a defect in the neuro-muscular mechanism from birth has been suggested, but it is hard to attribute to a congenital cause a disease first manifesting itself in maturity. Walton³ supports this view, citing rare cases that have occurred in the early years of life. He regards the phenomenon as parallel with the faulty co-ordination of neuro-muscular mechanism responsible for the megacolon of Hirschsprung's disease and infantile dilatation of the urinary system, which occur without mechanical blocks. Harold Brunn⁸ supports the theory, and explains the late onset of the condition by the analogous latent period in cases of cervical rib.

The opinion of Mikulicz, to whom we owe the cure by digital dilatation, is expressed by the historic title given

to the disease by him—namely, "cardiospasm." That spasm is a potent element in these cases, at least in the earlier stages, is evident from the sudden relief of obstruction, onward passage of food, and relief of pain observed in my case (A. F.) and in so many of the histories reported.

Plummer,⁵ after his vast experience, supports the neuro-genic theory. If accepted, the condition falls into line with that of congenital pyloric stenosis; but in that, if there is overaction of the muscle, it is certainly accompanied by hypertrophy. However, it is open to question whether an accommodative diminution in the size of the muscular opening in the diaphragm occurs *pari passu* with the diminished calibre of the gullet, and so inhibits the expected hyperplasia.

Hurst⁴ remarks on the easy passage of the gentle mercury bougie, and affirms that there can be no positive spasm but mere non-relaxation of the sphincter ("achalasia"). This he attributes to degeneration of Auerbach's plexus, and has seen this in sections from such cases. If the theory is accepted, it involves the belief in some peculiar susceptibility of this segment of the alimentary tract to a poison apparently impotent elsewhere, for there is actual hypertrophy of the circular muscle immediately above the constriction. Certainly, in the inveterate case on which I operated, resistance was inelastic and caused by organic contracture. This may well be the late outcome of an achalasia of the incipient stages by accommodative contracture of the surrounding tissues and the fibrosis following chronic congestion induced by the superincumbent pseudo-stomach.

Soper and Cassidy⁹ found, in 29 out of 60 cases, pathological lesions elsewhere—for example, gastro-duodenal ulcers, gall-stones, pleural contractures, and exophthalmic goitre. The last were improved by dilatation of the gullet. In my patient (W. S.) associated bronchial catarrh is noted. This conjunction affords a source for possible reflex dysfunctions of the cardiac sphincter. Greenwood,¹⁰ from his own case, forcibly argues that dysfunction is due to imprudent feeding when hurried; unmasticated meals provoke irritability and hypertonus of the stomach inlet.

The title "phrenospasm" (Chevalier Jackson) lays the blame on the encircling diaphragm. Two criticisms of this explanation are suggested. The grip of a thin sheet of muscle would make a narrow groove on the gullet, but the findings are those of a tunnel obstruction an inch or more long. Morley remarks that phrenicotomy fails to relieve the impasse.

Finally, in the cadaver, I am impressed with the resistance offered by the half-encircling edge of the liver when passing a finger into the oesophagus. Clark¹¹ refers to this relationship. It is not, in my opinion, an adequate etiological factor, or signs of an impeded cardiac orifice would be manifested more often in conditions of hepatomegaly.

The theories mentioned broadly encourage the belief that there is defective neuro-muscular action at the oesophageal exit, but whether this is inherited, brought on by the irritation of imprudent feeding, reflex from distant foci of disease, or the result of local neural degeneration, further research must prove.

Summary.

It is very probable that a neuro-muscular dysfunction at susceptible transition points in the gullet raises the pressure of ingesta. This effect expresses itself by different forms of dilatations. The inherent weakness in the posterior aspect of the hypopharynx produces a hernia of the mucosa. By contrast, in the epiphrenic region, the oesophageal walls respond to the hypertension within by a typical uniform dilatation and hypertrophy. From such origins spring the substernal and epiphrenic stomachs. They are likely to continue to vex the human race, evoke the discernment of the doctor, elude prophylaxis, and require most skilful surgery to relieve them.

In pharyngeal pouch two-stage diverticulectomy is the best remedy, while cardiospasm, in the early stages, will respond to mere dilatation with bougies, which is best done with the aid of the radiographic screen. Where this fails,

Mikulicz's digital dilatation, by means of a gastrotomy, is necessary and effective. By reason of the comparative infrequency of these conditions, their deep situation, and the vital structures around them, their treatment calls for special care and skill.

I am indebted to Dr. F. J. Mayes for all the radiological work contributed to the diagnosis of my cases.

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TWO CASES OF BONE TUMOUR FROM ANCIENT EGYPT.

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(With Special Plate.)

Of the nature of the diseases which were present among the ancient Egyptians but little is known. Such specimens as have been found and examined have been generally derived from the Roman period of occupation, which is, comparatively speaking, late in the history of Egypt. Few pathological specimens have been obtained from the earlier dynastic periods.

Those so far described are mostly associated with the hard and resistant portions of the body—namely, the bony skeleton—as these parts, unless some form of mummification has been undertaken, best resist the destructive influence of time and climatic conditions. There have thus been preserved for us specimens which enable us to diagnose conditions which were then associated with the osseous system of the body. The supply of these interesting specimens, which reach us only too rarely, is mostly derived from the archaeologists, who in the course of their work observe abnormalities in the occupants of the graves and tombs which are being investigated from the archaeological point of view. To them, therefore, our thanks are due for the interest they take in this side issue of their work. We have to thank Mr. Guy Brunton for both the specimens described in these notes.

Osteosarcoma of the Femur.

The first of these specimens, that of a tumour of the femur, was found by Mr. Brunton in a grave at Mostagadda, south of Assiut, on the eastern side of the Nile, on the edge of the desert. The specimen belongs to the Ptolemaic period.

The bone is the right femur of a young person; the head of the bone and the trochanteric epiphyses have not become united, and are therefore absent. The photograph in the centre of the illustration (Fig. 1) gives a good impression of the condition of the bone; the neck of the femur is seen at the upper end of the specimen. The bone

itself is 10 in. long, and the surrounding bony growth involves the greater part of it. The growth at its widest part is $3\frac{1}{2}$ in. in diameter. In the fresh state the growth would have been considerably larger, because the delicate spicules of bone, which form the osseous portion of the tumour, are in large part broken off, while time and wear have produced a relatively smooth surface on the mass. The soft parts of the tumour have completely disappeared. In certain areas, however, the typical bone spicule formation of osteosarcoma is evident, while the skiagram on the left of the photograph in the illustration shows the typical architecture of the growth. The skiagram is obviously that of a long bone, of which the cortex, while rarefied, is unbroken and unchanged in width. The medullary space shows no expansion.

Extending outwards in a direction at right angles to the shaft are shadows arranged in the form of parallel striae. It will be observed that the extent is largest about the mid-point of the bone. We should suggest, from the radiographic appearance, it is a justifiable inference that a bone-forming sarcoma has originated about the mid-point of the femur. One can therefore definitely exclude such a condition as chronic osteomyelitis, which might perhaps be suggested by the gross naked-eye appearance of the specimen.

For comparison, a skiagram of a similar osteosarcoma of the humerus, occurring in a young Egyptian patient (Kasr-el-Aini Hospital, in-patient No. 449 of 1930) is reproduced. If allowance is made for the fact that in this skiagram the soft parts are shown, the similarity between it and the skiagram of the Ptolemaic bone is evident. We therefore venture to diagnose this tumour of the Ptolemaic period as an osteosarcoma, and infer that some 2,000 years ago this type of malignant growth was already present in the population of Egypt.

A search of the literature for records of similar cases shows that Ruffer,¹ in his "Studies in palaeopathology," described a tumour of the pelvis (dating from the Roman period) found in the catacombs of Kom-el-Shongafa in Alexandria. He observed that this tumour had expanded the iliac bone and consisted of a solid bony tumour containing cyst-like spaces. Although he suggests that it may be an osteosarcoma from which all soft parts have disappeared, it seems more probable to us, from the illustration, that it is an ossifying chondroma.

Tumour of the Mandible.

The second specimen, also sent to us by Mr. Brunton, was found by him in the same district in a grave of the pre-dynastic period. In all probability, therefore, its age is not less than 5,500 years. This tumour presumably is one of the earliest, if not the earliest, yet found amongst the former dwellers in the Nile Valley.

The tumour occurs in the left side of the mandible, and involves portions of both the horizontal and ascending rami. It extends from the region of the canine tooth to the angle of the jaw, the bone of which is expanded and reduced to a shell. The soft parts of the tumour have disappeared, leaving the cavity in which it lay widely open at the alveolar surface. This cavity is $3\frac{1}{2}$ in. in length and $\frac{3}{4}$ in. in depth, while the widest diameter is $1\frac{1}{4}$ in. From the floor of the cavity, and projecting from its sides, the remnants of some bony septa can still be seen. The teeth and soft parts are absent. The illustration (Fig. 2) shows on the left side a photograph, and on the right side a skiagram of the condition. In the latter may be seen the widening and thinning of the bone, while the cortex along the inferior border of the mandible is intact. For the production of the skiagram the rays were directed, relatively to the bone, at approximately the same angle as would be employed in the living subject.

The diagnosis of this condition cannot be definitely established, but the possibilities of a myeloid epulis, a dentigerous cyst, a simple cyst, or a fibroma should be considered; of these the first appears to be the most probable.

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