

with such widespread glandular infection that a radical operation was impossible. A year later she had gained nearly 3 st. in weight, and was so well that when the American Society of Clinical Surgery was in this country I showed her to the members as a case in which I had been mistaken as to the nature of the disease. The patient remained well for two years, then exhibited signs of sudden, rapid growth of the disease, and died within a few months.

A more remarkable case is that of a man, aged 52 years, upon whom I performed gastro-jejunostomy in 1908. He had cancer of the pylorus, which had spread in the sub-mucous tissue almost up to the cardiac end of the stomach. Radical operation was impracticable. I removed a portion of the growth, and microscopically it proved to be a spheroidal-celled carcinoma. It is now more than four and a half years since the operation, and the patient states that "he is as well as he has ever been in his life." Until we know more of the natural history of gastric cancer we are not justified in drawing conclusions as to whether it is or is not, greatly prolonged in some instances.

The alternative to the conclusion that carcinoma is grafted on ulcer is that simple ulcer and carcinoma may occur independently in the same patient. That sometimes this should happen is highly probable, for the frequency of the incidence of cancer and ulcer is very similar. Further, it is a hypothesis based on fact. On several occasions, when operating for cancer, I have been able to demonstrate to those present either an open ulcer or the scar of an ulcer quite distinct from the malignant growth. In several, at least, of the cases recorded as instances of *ulcus carcinomatosum* two ulcers were observed—one simple and the other malignant.

A perusal of the records of cases of *ulcus carcinomatosum* reveals one striking feature—namely, the frequency with which there is an interval between the symptoms of supposed ulcer and the onset of the symptoms of gastric cancer. With regard to such cases as these, the assumption that cancer has been grafted on an ulcer is less reasonable than the hypothesis that an ulcer has been present and has become healed, and subsequently cancer has occurred independently of the ulcer.

(b) *Pathological evidence.*—According to Dr. Wilson and Dr. MacCarty, in 71 per cent. of their series of cases there was "a large ulcer with scar tissue centres and overhanging borders, deep in the bases of which cancer is present, which ulcer in almost every instance has unmistakably originated in the lesser curvature of the stomach, the usual site of gastric cancer. Further, almost every case gives a clinical history suggesting gastric ulcer for a long period of years preceding the relatively short period when the history became that of gastric carcinoma." Apart from the assumption that a previous history of gastric trouble is evidence of gastric ulcer—an assumption which I have criticised already—the weak point of this argument appears to me to be absence of proof that an ulcer in which cancer cells are present has ever been other than malignant. The presence of scar tissue is not necessarily evidence of non-malignancy. We know that in cancer of the breast there may be formation of scar tissue, nature's attempt at arrest of the disease. Further, the border line between a benign and a malignant growth is a narrow one, and a given tumour may be inherently malignant before clinically or pathologically we can recognise its malignant character, with the means at present at our disposal.

There are two truths which appear to me to make it very difficult to accept the view that the grafting of cancer on ulcer is of frequent occurrence. First, simple ulcer of the duodenum is, at least, as common as gastric ulcer, and yet cancer of the duodenum is a rare disease. Surely, if simple ulcer be a frequent precursor of cancer, then cancer of the duodenum should be at least as frequent as gastric cancer. Then, again, cancer is commoner in the second than in the first portion of the duodenum. Ulcer in the second portion is the rare exception. If ulcer be the precursor of cancer, then cancer should be far commoner in the first than in the second portion. Exactly the contrary is the case.

Secondly, death from cancer after gastro-jejunostomy for supposed simple ulcer is a rare event. If the hypothesis as to the frequency with which simple ulcers become malignant be correct, then many of the ulcers supposed to be simple should prove later to be malignant, and consequently many of the patients on whom gastro-jejunostomy is performed

should die subsequently from cancer. As a matter of fact, the mistake when made is more often in the opposite direction—it is the supposed malignant growth which proves to be innocent, not the supposed simple ulcer which proves to be malignant. My own experience is that 1 per cent. of the patients on whom gastro-jejunostomy is performed for simple ulcer die later from carcinoma. Professor Kocher has published recently a review of 50 cases of gastro-jejunostomy. The period which had elapsed since operation varied from two to twelve years. In no case was there a recurrence of symptoms, or any suggestion of malignancy. Dr. Gressot states that malignant degeneration of surgical ulcers occurs in only 2.3 per cent. of all cases after gastro-jejunostomy.

In view of such observations it is difficult to accept the view that grafting of cancer on simple ulcer is a frequent event. Of course, I do not deny that a simple ulcer may become malignant. My point is as regards the frequency of such a happening. That it is anything like so common as some authorities teach, in my opinion, is doubtful. The subject requires much further investigation. In the meantime the verdict should be the Scotch verdict of "Not proven."

[Upper Wimpole-street, W.]

ON THE SECRETORY ACTIVITY OF THE STOMACH IN CHRONIC APPENDICITIS WITH GASTRIC SYMPTOMS.

WITH AN APPENDIX CONTAINING CLINICAL DESCRIPTIONS OF 19 CASES.

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Brompton.)

SINCE W. J. Mayo drew attention to the association of chronic appendicular disease with gastric ulcer, the combination of these two conditions has attracted the notice of a number of observers, and is now generally admitted (1).¹ Moynihan (2) especially has shown beyond all reasonable doubt that there is a definite relationship between these two pathological states, and has laid emphasis on a constriction of the base of the appendix that is liable to occur in such cases. Of late years, also, many other writers have been inclined to attribute gastric symptoms to chronic appendicular disease, and Fenwick (3) and Paterson (4) have found that a large proportion of cases presenting obscure gastric symptoms are the subjects of chronic appendicitis and in many they have demonstrated hyperacidity.

I have recently had the opportunity of making very detailed analyses of a series of 300 cases presenting gastric symptoms. Most of these cases were subjected to laparotomy. Among them some 19 presented unequivocal signs of appendicular disturbance, and analyses of the gastric contents of these cases are set out in the accompanying table, being divided into two groups according as a peptic ulcer was or was not present. A few lines of explanation must be given of the actual measurements and readings in the table.

The phenolphthalein and dimethylaminoazobenzene readings must not be taken to indicate total acidity and free hydrochloric acid. These readings, as explained elsewhere (6), are dependent on hydrogen ion concentration, and their difference (i.e., phenol minus dimethyl) is a function of the amount of nitrogen present and of the degree of digestion. In an active stomach, therefore, in which all the dissolved nitrogen is present as peptones or similar products of digestion, the fraction $\frac{\text{phenol minus dimethyl}}{\text{nitrogen}}$ is a constant,

provided that a standard test-meal be adopted and removed always at a fixed time after ingestion. A certain multiple of this constant has been designated the nitrogen factor and has been found to nearly approach 2.4 in normal cases. A rise above this number indicates either stasis or impairment of the digestive process.

¹The numbers in brackets refer to the bibliography at the end of the paper.

In all cases the peptic activity has been estimated by the digestive power of the juice on edestin, and this capacity has been designated the "peptic index" (5). This method and its application have been explained elsewhere (5 and 6), and it is here only necessary to point out that a normal peptic index is within the range 40-50, and that a variation of more than 10 on either side is probably always pathological.

The total chloride and secreted chloride which occupy the fifth and sixth columns of the table have been estimated by the Volhard-Lüttke process (7) recommended by Willcox (8).

The nitrogen estimated by the Kjeldahl process is expressed in the seventh column as the number of cubic centimetres of decinormal sulphuric acid required to neutralise the ammonia formed from 10 c.c. of the juice. For the complicated question of the "nitrogen factor" the reader may be referred to another work (6).

Lastly, all cases have been examined for a peptolytic ferment. Ferments similar to that here investigated have been claimed as specific to gastric carcinoma, but we have shown that a peptolytic ferment is by no means rarely

chronic variety. In nearly all cases the "nitrogen factor" was raised, and the three exceptions were all ulcers of the lesser curvature. The presence of a peptolytic ferment was demonstrated in three cases.

Of late years there has been an increasing tendency to regard gastric ulcer as of toxic origin, and since the proof of the existence of a gastrototoxic serum by Bolton (9) this toxic source may be sought in the blood stream. The recent work of Poynton and Paine on the appendix (10) suggests that a vascular origin has also to be considered for appendicular disease, and it seems not unlikely that the two conditions may in some cases have a common septicæmic origin. Experiments on this point are now in progress.

Bolton has shown that for the production of a chronic gastric ulcer stasis, or hyperchlorhydria or both must be added to the action of the gastrot toxin. The existence, however, of a gastric hormone (11) the specific reaction of the pyloro-duodenal canal (12) in causing the secretion of hydrochloric acid, and our own experiments and analyses (5 and 6) make it appear likely that the situation of an ulcer in the

TABLE OF 19 CASES PRESENTING APPENDICULAR DISTURBANCE, WITH ANALYSES OF THE GASTRIC CONTENTS.

Case No.	Phenolphthalein acidity.	Dimethylaminoozobenzene acidity.	Günzburg's test.	Peptic index.	Total chloride.	Secreted chloride.	Nitrogen.	"Nitrogen factor."	Peptolytic ferment.	Remarks.
With Definite Gastric or Duodenal Lesion.										
1	4.8	1.8	+	100	7.3	6.3	9.2	3.3	-	Duodenal ulcer. Chronic appendicitis with concretions.
2	5.6	3.8	+	100	6.1	4.6	7.5	3.8	-	Old duodenal ulcer (healed). Appendix bound down by adhesion behind cæcum and full of fæces.
3	5.4	1.9	+	95	8.3	5.7	12.0	2.9	-	Large duodenal ulcer. Appendix densely adherent and chronically inflamed.
4	8.4	5.6	+	91	9.5	7.4	9.4	3.0	-	Scar of duodenal ulcer. Appendix long, indurated, and peritoneal surface injected.
5	6.8	4.8	+	73	8.7	6.8	7.2	2.8	-	Very extensive duodenal ulcer. Appendix fibrous for part of its length.
6	7.5	5.5	+	60	9.6	6.5	7.7	2.6	-	Duodenal ulcer. Retrocæcal appendix full of concretions and with many adhesions.
7	6.9	4.2	+	67	8.9	7.4	10.8	2.5	-	Gastric ulcer on lesser curvature. Appendix acutely kinked on itself and bound down behind cæcum.
8	3.1	-0.2	-	20	5.5	3.6	9.6	3.5	-	Gastric ulcer on lesser curvature. Appendix long, thin, full of fæcal matter, and markedly constricted at base.
9	3.9	1.9	+	19	6.6	3.2	7.9	2.5	-	Ulcer of lesser curvature. Appendix with many old adhesions.
10	4.4	1.4	+	18	8.0	5.0	12.2	2.4	-	Large ulcer on lesser curvature near cardiac end. Chronic fibrosed appendix.
Without Definite Gastric Lesion.										
11	7.5	3.6	+	120	9.2	8.2	12.7	3.1	-	Club-shaped appendix, greatly thickened.
12	5.1	2.0	+	80	7.4	6.2	12.2	2.5	-	Appendix extensively adherent, very long and pointing upwards towards right kidney and to right of colon. Seed calculi in gall-bladder.
13	5.7	3.6	+	67	7.2	6.4			-	Very long full appendix with adhesions.
14	7.7	5.9	+	66	8.5	7.2	4.5	4.0	-	Most of lumen of appendix obliterated by fibrosis.
15	4.4	0.0	-	16	5.3	3.3	12.8	3.5	+	Stomach dilated. Appendix with many adhesions.
16	2.1	0.0	-	<10	5.4	3.7	9.4		+	Appendix removed 18 months previously in a condition of subacute inflammation.
17	2.3	0.0	-	<10	4.6	2.8	8.2	>2.9	-	Appendix chronically inflamed. Dilated pelvis of right kidney.
18	1.2	0.0	-	<10	2.8	2.0	3.6	>3.3	-	Appendix removed 4 years previously in a state of chronic inflammation. No improvement.
19	0.5	0.0	-	<10	2.3	1.2	3.7		+	Long appendix with adhesions. Stomach dilated.

encountered in the juices of stomachs dilated from various causes (5).

The standard test-meal has been a pint of weak tea with milk and sugar to taste and two rounds of buttered toast. It has been removed in all cases one hour after ingestion.

From the table it will be seen that these 19 cases agree with one another only in two things—namely, that both the appendix and the peptic power were in all abnormal. This accords with the conclusions reached on other grounds (6) that the peptic index is more easily disturbed than the chloride secretion. The chloride secretion varies from a high to a very low limit, and the same may be said of the phenolphthalein and dimethyl acidity, but these readings, like the peptic index, were in nearly all the 19 cases either abnormally high or abnormally low.

Ulcers when present were found both on the lesser curvature and in the duodenum, and were all of the

pyloric region or duodenum may be itself a factor in the increase of hydrochloric acid secretion. A gastric or duodenal ulcer regarded in this light thus becomes not only the result but also a cause of hyperchlorhydria. Prolonged hyperchlorhydria may thus be of mechanical origin, and the existence of a duodenal or pyloric ulcer may account for the persistence of hypersecretion of hydrochloric acid over a long period of time. On the other hand, there is probably an original tendency to hyperchlorhydria acting along with the toxic element in the production of a chronic ulcer. It appears not unlikely that the cause of this element is to be found among the etiological factors of appendicitis, for the writer has observed cases in which there was well-marked hypersecretion of chloride and of pepsin with no other demonstrable lesion than a chronic appendicitis.

Lastly, it may be pointed out that in the cases in which no gastric lesion has been present the removal of the appendix has not always relieved the gastric symptoms.

I have to thank members of the staff of the Dreadnought Hospital and the London Hospital whose material has been utilised in the course of this research, and especially Dr. P. N. Panton, clinical pathologist to the London Hospital, who provided a series of samples of gastric juice.

Conclusions.

1. Chronic appendicitis has been frequently found in association with gastric symptoms.
2. When this association occurs, a gastric or duodenal ulcer may or may not be present, but in all cases the gastric juice has yielded abnormal analytical results.
3. The abnormalities may consist of hypersecretion or of hyposecretion both of chloride and pepsin, of the presence of a peptolytic ferment, and of the elevation of the "nitrogen factor."
4. These abnormalities can best be explained as due to toxic substances which act both on the stomach and on the appendix.
5. Removal of the appendix does not always relieve or improve the symptoms.

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APPENDIX.

Clinical Descriptions of 19 Cases in which a Diseased Condition of the Appendix was Associated with Gastric Symptoms.

CASE 1.—Male, aged 46. Symptoms for seven years. Commenced with epigastric pain not at first related to food. The pain at first came on at long intervals and lasted a few days, but the intervals have gradually become shorter and more definitely related to food and about one hour after it. For six months previous to admission the pain was very severe. The pain is gnawing in character, and especially severe about 2 A.M. Food, especially hot milk, relieves it, as does also vomiting. Definite hunger pain. Very constipated. Prolapsed hæmorrhoids. Tenderness over appendix and in epigastrium.

CASE 2.—Male, aged 24. "Indigestion" as long as patient could remember, and some pain or discomfort every day for years. Burning, sinking sensation in the epigastrium, with a shooting pain in the iliac fossa. Pain about half an hour after food. Took proteins fairly well, but starchy foods were liable to upset him. Constipated. Hæmorrhoids.

CASE 3.—Male, aged 46. Twenty-six years' history of dyspepsia, and for last seven years liable to attacks of vomiting. Pain always three hours after food and on the right side at level of umbilicus, lasting about an hour and relieved by milk and vomiting. Teeth all artificial.

CASE 4.—Female, aged 22. Eight years' history of attacks of abdominal pain and sickness. The attacks lasted a few days, and consisted of vomiting and epigastric pain not definitely related to food and not relieved by it. Pain often very severe and colicky in character.

CASE 5.—Male, aged 33. A history of attacks of flatulence, dark motions, constipation, and vomiting, and pyrosis for 13 years. The pain came on about 1½ hours after food and was occasionally colicky. Was brought into hospital with a very severe attack of pain simulating gall-stone colic.

CASE 6.—Male, aged 38. Three years' history, during which time patient had been twice previously in hospital with symptoms of duodenal ulcer that had yielded to medical treatment. Pain always worst at 9.30 P.M., and not relieved by food. Had had enteric fever. Marked wasting. Melæna observed several times in hospital. No tenderness over appendix.

CASE 7.—Male, aged 44. A history of attacks lasting a few days for seven years. Quite well between times. Attacks consisted of constipation, acid eructations, and sometimes vomiting and tarry motions. Teeth good. Epigastric tenderness to right of middle line. No tenderness over appendix. Marked wasting.

CASE 8.—Male, aged 57. Had been ill for three years with numerous attacks. Worse in summer than in winter. Dragging pain in epigastrium 1½ hours after food. Pain lasted for one or two hours unless he vomited, which relieved it immediately. Had several times brought up considerable quantities of blood. Had lost 2 st. 3 lb. in a few months. Definite history of gout beginning in big toe.

CASE 9.—Male, aged 38. Three and a half years' history of attacks lasting some weeks, but was well nourished between such times. Pain in left gastric region commencing one hour after food and lasting till next meal, which gave relief. Warmth also relieved pain. Never got pain at night. Had had several severe attacks of hæmatemesis, but had not vomited otherwise. Melæna. Much wasting. Teeth fair. Hæmatemesis after operation.

CASE 10.—Male, aged 40. Ill on and off for 12 years. Pain from three to four hours after eating, which would be relieved by next meal. Vomiting, nausea, and loss of weight. Had had three attacks of hæmatemesis. Ill-defined epigastric tenderness.

CASE 11.—Male, aged 58. Had had "indigestion" for 15 years. Pain directly after food, gripping in character, and felt in epigastrium and abdomen. The pain had been getting steadily worse, and he had of late never been free from it. Had lost a stone in weight in six weeks. Had a yellow skin with numerous telangectases. Teeth all artificial. Tongue furred.

CASE 12.—Male, aged 24. Ill on and off for four years, with intervals which had gradually been getting shorter. Pain from half an hour to one hour after food, lasting half an hour at first, but gradually lasting a longer time until he was now seldom free from it. Tenderness in epigastrium, but not over appendix. Teeth good. Some loss of weight.

CASE 13.—Male, aged 55. "Indigestion" for 30 years, with numerous severe attacks. During attacks epigastric pain and heartburn about half an hour after food, and sometimes while taking food. Often felt distended, giddy, and faint, a condition which was followed by shivering. Pain relieved by bicarbonate of soda or peppermint. Mouth and teeth septic. Always constipated.

CASE 14.—Male, aged 34. Had had gastric symptoms for 14 years. Attacks consisted of pain, vomiting, and loss of weight. Pain would start in sternal region one hour after food, and would last about an hour and a half, and gradually settle down into the right iliac fossa. It was relieved by sodium bicarbonate and hot flannels. Vomiting was frequent, often eight times a day—i.e., after each meal. Flatulence was very troublesome and painful. Hæmatemesis and melæna on one occasion.

CASE 15.—Male, aged 32. Symptoms for two years, greatly accentuated during last two months. Loss of appetite and marked wasting with attacks of epigastric pain, occasionally becoming very severe and exhausting. Vomiting occasional. One severe hæmatemesis. Pain half an hour after food and lasting an hour; also at about 2 A.M.

CASE 16.—Female, aged 46, married. For two years had had pain across lower abdomen indefinitely related to food. The pain had been worse for the last year. A year ago the abdomen was opened and a subacutely inflamed appendix was removed. The pain, however, has become worse instead of better. The patient has lost a good deal of flesh. Radiographically the stomach appears to be normal. The right kidney is mobile.

CASE 17.—Female, aged 46. For the last year the patient had suffered with a shooting pain directly after food, beginning in the epigastric or sternal region and gradually settling to the lower abdomen. Much wasting. At operation, besides a chronically inflamed appendix, the pelvis of the right kidney was found to be dilated.

CASE 18.—Female, aged 43, married. For many years had had pain about half an hour after food, relieved by occasional vomiting. The pain became more troublesome about four years ago, when the abdomen was opened and a chronically inflamed appendix was found. She was better for a time, but for the last three months the symptoms had returned. A laparotomy revealed no abnormal condition of stomach or duodenum.

CASE 19.—Male, aged 59. Thirty years' history of attacks of pain occurring about an hour or an hour and a half after food. These attacks were more frequent and severe in summer than in winter. Lately they had had less and less relation to food and had become almost continuous. The pain began in the chest or shoulder, and extended later into the lower abdomen. Mouth septic. Constipated. Resistance all over right rectus abdominis.

ASSOCIATION OF THE FELLOWS OF THE ROYAL COLLEGE OF SURGEONS IN IRELAND.—A dinner, followed by a meeting, of this association was held recently at the Hotel Cecil, London. Lieutenant-Colonel F. G. Adye-Curran, I.M.S. (retired), president of the association, was in the chair, and he explained the objects of the society, viz:—(a) to further the welfare of the College, and to safeguard the interests of its graduates; (b) to ensure that members of the association may take an active part in the proceedings of the annual and other meetings of the College; (c) to approach the Council upon all matters affecting the material interests of the College and its graduates; and (d) to give the Council of the College all the support possible in all matters of professional interest when the association considers such support advisable. It was unanimously resolved to form a council in London and to take immediate steps to initiate an active propaganda. Any Fellow of the College wishing to join the association should communicate with the secretary, Mr. Andrew Charles, 64, Harcourt-street, Dublin, or with the London secretary, Mr. Frederick Spicer, 142, Harley-street, London, W.

EARLY SIGNS OF MEDIASTINAL TUMOURS.¹

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MEDIASTINAL tumours are usually classed among the uncommon diseases, but their occurrence is far from being rare. Under this title are included malignant growths involving any of the mediastinal structures, enlargement of the intrathoracic lymphatic glands from any other cause, and inflammatory swellings. Closely allied to them in the resulting symptoms are many cases of thoracic aneurysms, to which, however, I do not propose to allude in this communication, and also tumours originating in the substance of the lungs. All these tumours manifest themselves by symptoms due to pressure on adjacent structures. The rate at which symptoms develop varies considerably, for in some rapidly growing glandular tumours and in some inflammatory affections evidence of pressure on several organs may manifest themselves in the course of a few days, whereas in slowly growing tumours symptoms may be limited to irritation of a single organ for many weeks. By "early signs" I therefore mean those which are "early" in time, and not in degree.

For the purpose of this paper I have based my remarks on 36 cases with evidence of intrathoracic pressure which I have met with during recent years. Of these, 14 were cases of malignant growths of glands or lung, 8 were cases of malignant disease of the œsophagus subsequently affecting the surrounding structures, 3 were cases of enlarged tubercular glands, and 4 of non-tubercular lymphatic affections, 3 were cases of mediastinitis, and 4 were syphilitic in character. The symptoms met with in these cases were pain, dyspnoea, cough, dysphagia, wasting, anasarca, vomiting, hiccough, and palpitation. Wasting was, of course, common to all the malignant cases, and dysphagia was almost, but not altogether, limited to cases of disease of the œsophagus. The relative frequency of the remaining symptoms may be inferred from the fact that pain was the most prominent symptom in 11 cases, cough in 8, dyspnoea in 7, anasarca in 6, vomiting in 2, and hiccough in 1.

If the relative frequency of the causes in my cases is a fair average, seeing that 22 out of my 36 cases were malignant in character, it follows that the prognosis of mediastinal tumour, speaking generally, must be very bad. Of my remaining cases, one died from mediastino-pericarditis and two from lymphadenoma, but the remaining 11 cases recovered more or less completely from their symptoms, and the degree of recovery seemed to vary inversely with the length of time during which the patient had been affected. It follows, therefore, that early diagnosis of the presence and cause of intrathoracic pressure is particularly important in those cases where therapeutic treatment may be possible—i.e., when the cause is specific, inflammatory, or tubercular in nature. For this reason it seemed to me that a consideration of the signs of the disease—as far as can be undertaken within the limits of a short paper—as they were discovered in my cases on their first examination might not be without interest. In order to economise time I propose to confine my remarks to a few points bearing upon (1) obstruction to the venous circulation; (2) obstruction in the respiratory tract; (3) referred pains; and (4) effect upon the pericardium.

1. *Venous obstruction.*—This is, of course, one of the classical symptoms of mediastinal tumour. The dilated veins of the skin of the thorax, upper arms, and abdominal walls, as well as the œdema often associated with them, are familiar to all. But under this heading I wish to refer only to one particular vein which is less frequently affected and is apt to be overlooked in this connexion—viz., the vena azygos major. Pressure upon this vein is interesting for more than one reason. I was first led to take an interest in it by a case of mediastino-pericarditis which I met with some years ago in a young boy. This boy was sent

into hospital as a case of acute Bright's disease, and upon a superficial inspection the diagnosis seemed fully justified. The patient had marked general anasarca—over his abdomen, loins, legs, thorax, arms, and face—as well as effusion into his pleural and peritoneal cavities. But examination of the urine showed it to be perfectly normal, without albumin, deposit, or casts, so that it was clear that his dropsy was not due to renal trouble. That being so, the heart was examined as the next most probable seat of trouble. This organ, however, showed no sign of abnormality, and was beating slowly and regularly, without any suspicion of a murmur. From these facts it became possible to deduce that the dropsy was due to a mechanical obstruction to the circulation, and by consideration of the veins obstructed it was possible to locate the trouble in the thorax, pressing upon the superior vena cava and the right auricle.

There was, however, one symptom about this boy which impressed me—viz., that the effusion into the peritoneal cavity was quite insignificant in comparison with the amount of effusion into the abdominal walls and lumbar region. Now these parts are drained by the intercostal and lumbar veins, which empty themselves into the azygos veins, and it would seem a reasonable deduction that the trouble in the chest in addition to pressing on the great veins pressed also on the vena azygos major. The subsequent post-mortem examination showed this to be the case. It struck me that this great disproportion between the œdema into the peritoneal cavity and that into the subcutaneous tissue of the abdominal walls and loins might be a useful diagnostic point in distinguishing between anasarca due to cardiac or renal disease on one hand, and that due to mechanical obstruction in the chest on the other. And I have since then several times found this point of much help in diagnosis. This œdema is usually bilateral, but in one case of malignant mediastinal growth that I met with it was unilateral and limited almost entirely to the right half of the abdominal wall and the right loin. This was due to the fact that the tumour obstructed the right lower azygos vein before it was joined by the vein from the left side. It was thus possible to localise the tumour more exactly, as being below the level of the eighth dorsal vertebra. In another case to which I shall refer the œdema was limited to the left side.

One character of such œdema sometimes met with, when the inferior vena cava is obstructed as well as the azygos vein, is brawny hardness of the skin of the legs and abdomen, with almost complete absence of pitting. Not long since I was asked to see a patient with such a condition of the lower half of the body, and the appearance at once suggested the diagnosis of posterior mediastinal tumour occluding the azygos major in addition to other veins. This was not an early case, and upon examination other physical signs were found demonstrating a growth occluding the lower division of the right bronchus, thus supporting the above suggestion, which was subsequently verified.

A case of brawny œdema of this distribution was reported to this Institution a few years back by Dr. W. Carter, which recovered completely on the administration of mercury. This may have been specific in origin. At any rate, I have learned that tumours occluding this vein are not rarely gummatous in nature, and are therefore capable of being benefited by treatment. The following case is an interesting example.

A large-looking man was admitted into the Royal Southern Hospital with anasarca of all parts except the face, neck, and arms, with fluid in the pleural and peritoneal cavities, and considerable enlargement of the liver. These symptoms had all come on in the course of three weeks without any previous illness to account for them, the heart and kidneys being found healthy. On examination the œdema was found to be far more marked in the subcutaneous tissue of the loins and abdominal wall than elsewhere. These facts at once pointed to mediastinal obstruction, and other physical signs were found showing evidence of pressure on the left lower bronchus. Owing to the rapidity of development a diagnosis was made of gummata of the posterior mediastinum, and the patient was treated with mercury and iodide of potassium. Improvement was immediate. Almost in a day the œdema began to diminish, and a week later the note reads: "The œdema is entirely absent, the back and thighs being clear." The signs of pleural and peritoneal effusion had also disappeared. I kept the patient under observation in the ward for three weeks longer, but there was no return of symptoms, and he went out feeling quite well. There was, however, distinctly less air-entry into his left lower lobe than into the right, so that although the supposed gummata had diminished sufficiently to relieve the venous obstruction, there remained its effects causing some obstruction to the bronchial tract.

This patient kept well for seven months, and then was readmitted with a recurrence of all his symptoms. The

¹ A paper read before the Liverpool Medical Institution.