in Chelsea, a licentiate of the Apothecaries' Hall, and "one of the livery." Mr. Knight presented it to the Hall in 1857. Now, in her researches into the history of Reynolds's great picture, Miss Tildesley was puzzled by this curious circumstance: From Reynolds's notebook she found that Hunter gave his first sittings, four in number, early in 1786; the sketch then done was shown in the Academy exhibition of 1786 as "the portrait of a gentleman." On its return from the exhibition Hunter gave Reynolds another sitting—in September. Then something happened—probably a dissatisfaction with the result, both on the part of artist and of sitter. Nothing was done in 1787, but in 1788 Hunter again sat three times to Reynolds; then, in 1789, he sat five times—in the afternoon from 2 o'clock to 4 each time. The Apothecaries' picture represents Hunter with a thin, straggling beard and moustache, but the poise of the sitter is exactly that of the acknowledged portrait. The Apothecaries' portrait cannot be a copy of the finished one; the rough materials for the final picture are there, but the inspiration and the finish, in a multitude of details, are altogether lacking. How are we to explain all of these facts—the two quite separate sets of sittings, a sketch by Reynolds, which Mrs. Hunter disliked; "the portrait of a gentleman" exhibited at the Royal Academy in the summer of 1786; its coming home and an attempt to mend it? And, then, why should Reynolds want a plaster cast of Hunter's face? All these things can be explained if we suppose that the Apothecaries' portrait is the picture done in 1786—that it was an admitted failure; that Mrs. Hunter disliked it; that a new one, the final portrait, was painted in 1788-89; that Mrs. Hunter was glad to get rid of the original picture, and gave it to Mr. Weatherall; and that Reynolds succeeded in getting Hunter to shave off his beard and become a fit subject for his art—by the subterfuge of proposing a plaster cast of his sitter's face. It is a painful process for a bearded man to submit his face to the cast-maker; it is easier and better to have the beard removed. That seems the most feasible explanation of a curious sequence of events.

Such, then, is the story of Hunter's portraits, so far as our present evidence will permit us to tell it. Our study has had the advantage of bringing us into close touch with the personality of a man whose like the world will see but seldom.

Lastly, as evidence of the authenticity of the new portrait, I must cite the testimony of one who is better qualified than any of us to recognize a Hunter—Miss Helen Hunter Baillie, the granddaughter of Matthew Baillie, Hunter's nephew, and now the chief living representative of John Hunter's family. She herself reproduces her great-grand-uncle's features and mental outlook with remarkable fidelity, and has been familiar with Hunterian portraits all her life. She has no doubt that the new portrait is that of John Hunter.

[Sir Arthur Keith informs us that there is to be an exhibition of Hunterian portraits and relics in the Museum of the Royal College of Surgeons, Lincoln's Inn Fields, from February 13th to 19th, open to all medical men and students of portraiture. Visitors will have an opportunity of comparing the "new" portrait of Hunter with other well known portraits. A series of specimens have been fitted up to represent the background of Reynolds's portrait. Hunter's chief discoveries in surgery, pathology, anatomy, physiology, zoology, botany, and geology will be exemplified by preparations chosen from the original Hunterian collection. Students of Hunter's history will be interested by an exhibit prepared at the request of the President of the College—Sir Berkeley Moynihan—by Dr. George C. Peachey. By means of maps, sketches, and photographs, Dr. Peachey has succeeded in giving a vivid picture of London in the latter half of the eighteenth century, the "homes" of the Hunters during that period, and what these "homes" look like to-day. Specimens of Hunterian interest have been loaned by the Wellcome Historical Medical Museum, University of Glasgow, St. George's Hospital, Apothecaries' Society, and other institutions, to make the present exhibition as complete as possible.]

REFERENCE.

¹ Dr. Matthew Young: A Contribution to the Study of the Scottish Skull, *Trans. Roy. Soc. Edin.*, 1916, vol. 51, p. 347.

An Address

ON

THE ACUTE ABDOMEN.

GIVEN BEFORE THE DEWSBURY DIVISION OF THE BRITISH MEDICAL ASSOCIATION

BY

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DURING the years 1924 and 1925 almost one-fifth of the patients admitted to the surgical wards of the Leeds General Infirmary were cases of one kind or another of the acute abdomen, and the total mortality rate of these two years is mainly attributable to these lesions.

The Commoner Conditions occurring in 1924-25 in Order of Frequency (excluding Subacute Types).

	Cases.	Deaths.
Acute appendicitis ...	1,080	58 = 5.7 per cent.
Strangulated herniae of all types ...	190	43 = 22.6 "
Perforated gastric and duodenal ulcers ...	150	34 = 22.6 "
Acute obstructions ...	139	55 = 40 "
Acute cholecystitis ...	196	6 = 5.6 "
Acute salpingitis ...	48	2 = 4.1 "
Ruptured viscera ...	29	16 = 55 "
Ruptured ectopic gestation ...	16	0 = 0 "
Acute pancreatitis ...	13	9 = 69.2 "

Though these statistics are not sufficiently large to form an accurate index of the order of frequency of these diseases as a whole, yet they illustrate the common types of acute abdomen in an approximately correct order. In some cases the acute lesion is a complication of a disease which has existed it may be for months or years, and has, therefore, given ample warning of the impending catastrophe. Perforated gastric and duodenal ulcers afford a good example, but the same applies to many of the acute obstructions and the strangulated herniae. Certainly over 90 per cent. of gastric and duodenal ulcers have shown their existence by characteristic indigestion over many years.

Further examination of these statistics brings to light another unsatisfactory feature, and to illustrate it I will analyse the acute appendicitis figures. In 1925 the cases of acute appendicitis admitted numbered 612, and of these 301 (almost half) were complicated by abscess, or diffuse or general peritonitis. When appendicectomy was performed before these complications arose there were no deaths, so that the mortality is confined to one-half of the cases, and these all late ones. It is this "too late" factor which is responsible almost entirely for the high mortality of the acute abdomen; I wish to emphasize this most strongly, and at the same time to say that the time is brief between the onset of the attack and the "too late" period; it does not as a rule exceed forty-eight hours, and after twelve to twenty-four hours the margin of safety is rapidly passing.

The surgeon may make his contribution to the mortality by performing unsuitable operations, but I think it is fair to say that he has little or no share in the "too late" episode. There can be no doubt the responsibility lies between the medical practitioner and the patient, or perhaps, more correctly, the patient's friends. The more I see of acute abdominal conditions the more convinced I become that it is the friends who waste the time. In no disease is this better exemplified than in acute appendicitis. There is too often a meddling household therapy as well. The patient, often a child and so unable to fend for itself, complains of bad stomach-ache and is promptly given a dose of castor oil. There is no more certain way of spreading infection to the peritoneum than by stirring up the intestines with aperients at the beginning of a local inflammation such as appendicitis; when it is seen that the pain is worse the doctor is summoned, and by this time probably the appendix has perforated, and, of course, the doctor or the surgeon receives the blame if the result of an operation is unsatisfactory. I recently advocated a campaign of public education, properly controlled, in

certain medical matters. I feel convinced that without it progress will be terribly slow. Even if only one statement were thoroughly driven home—namely, that it is dangerous to give an aperient for acute abdominal pain without the sanction of a doctor—I feel sure we should see an immense and immediate improvement in the outlook for the acute abdomen.

I do not intend to say much about operative technique; I shall confine my remarks to diagnosis, in the hope that what I have to say may be of use in focusing attention on the all-important twelve hours following the onset of the acute attack. The surgeon is really of quite secondary importance in this connexion; the man who has the greatest opportunity of reducing the operative mortality is the physician. It is true that operative skill and a knowledge of the best pre- and post-operative treatment are of great importance in such a condition as acute intestinal obstruction, but in early cases of acute appendicitis—and these make up the bulk of the acute abdominal lesions—any surgeon of average competence can show a death rate of *nil*, even over a long series. The credit for this lies entirely with the physician, and it is he who should receive it, though he by no means always does. In the later cases operative skill is of great moment, but even the most accomplished surgeon cannot put back the clock, and he will often be unable to repair what lost time has effected.

It will be seen from the table that acute lesions can roughly be classified into four groups: (1) inflammations; (2) perforations; (3) obstructions and strangulations; and (4) haemorrhages. This is not a scientific classification, for many of the perforations are the result of inflammatory diseases—for example, gastric and duodenal ulcers. But the perforation, which calls so urgently for treatment, is not necessarily immediately preceded by an obviously acute inflammatory stage. On the other hand, acute inflammatory diseases, such as appendicitis, frequently go on to perforation, but here there is, as a rule, a definitely recognizable inflammatory period before the perforation occurs, and it is with this early stage I am concerned. It is for this reason I have made this differentiation between groups 1 and 2.

Each of the four groups has its definite signs and symptoms, and once a particular case has been firmly placed in its proper group it is not difficult to find out the individual organ concerned, if a thorough examination be made and a clear history be elicited.

The signs and symptoms of an acute inflammatory lesion are pain, localized tenderness and localized rigidity, vomiting, quickened pulse, and raised temperature. Those of the second group are pain, more widespread tenderness and an equally widespread rigidity, possibly vomiting, rather quickened pulse, normal, or possibly subnormal, temperature. Those of the third group are pain, very slight, if any, tenderness, no rigidity, persistent vomiting, rather quickened pulse, temperature around normal, and absolute constipation. Those of the fourth group are pain, tenderness, slight rigidity, a distended, rather doughy, abdomen, possibly shifting dullness, usually some vomiting, rapid weak pulse, subnormal temperature, pale face and mucous membranes, syncope, and shallow respirations. The more advanced the condition the more the groups fade into one another in a common peritonitis.

On paper there does not seem to be much to distinguish groups 1 and 2, but a detailed examination of the signs and symptoms will soon dispel this opinion. Let us take acute appendicitis as a typical instance of the first group, and a perforated duodenal ulcer as one of the second. An acute inflammatory lesion of the appendix, in common with a similar lesion in all parts of the small intestine, gives rise to pain referred in the first place to the upper abdomen and umbilical region. The intestine and its visceral peritoneum, unlike the parietal peritoneum, has no appreciation of ordinary tactile sensation; pain occurs when the walls are stretched either by some obstruction to the onflow of the contents, or to exudation of inflammatory products into the wall, and the reason pain is felt in the upper abdomen is that the splanchnic nerves converge on the solar plexus situated in this area. This pain ceases when tension is relieved by the pouring out of the

exudate into the peritoneal cavity or intestinal lumen. As soon as the parietal peritoneum over the inflamed organ becomes involved the pain is localized to the region in which the organ is situated, and spreads outwards from it as the infection continues to involve a wider area of peritoneum. It is coextensive with the tenderness and muscular rigidity. Pain in the first instance—that is, when the intestinal wall only is involved—is of sudden onset, increasing in intensity as the tension from oedema increases, and of a colicky nature, due to intestinal contractions which, while they last, exaggerate this tension. The peritoneal pain, besides being constant, is of a stabbing nature.

Tenderness is due to inflamed peritoneal surfaces rubbing together, or to pulling on light adhesions either by pressure or by muscular movements, such as occur in appendicitis, from contractions of the ilio-psoas when the leg is shifted from one position to another. Rigidity is a protective mechanism, an attempt to limit movement of all kinds, and it is situated over the inflamed site through interaction between the peritoneal nerves and those supplying the muscles concerned. The extent of the rigidity is an index of the area of peritoneum involved, as no rigidity is present unless the corresponding part of the parietal peritoneum is being irritated. There is one exception to this—namely, when the inflammatory trouble is confined to the pelvis, for here the muscular wall is replaced by the pelvic bones, and so no rigidity is found. This fact has often accounted for a wrong diagnosis in appendicitis. The segmented nature of the rectus makes it possible for rigidity to be confined to limited areas of the abdomen; obviously there would be no use in a protective muscular action at any other point than over the diseased part.

In a few hours the referred pain tends to give place to pain localized over the involved organ, and as the appendix is an organ which occupies different places in different patients the pain is not always felt in the same situation. It may be in or near the gall bladder and kidney regions, or on the left side of the abdomen, or in the pelvis. In the latter situation it may cause frequent and painful micturition, and the need for a rectal examination is obvious. There are few inflammatory lesions in the abdomen which appendicitis may not simulate, and a differential diagnosis will depend to a large extent on the history; though, applying the rule of probabilities, it will usually be found that the appendix is the offender in children and young adults.

The pain of a perforated duodenal ulcer is quite different. It is terribly sudden, and excruciating from the outset, and accompanied by a degree of rigidity seldom found in any other condition. The extravasated contents seem to be particularly irritating to the peritoneum (probably it is the hydrochloric acid), and they trickle down in relation to the ascending colon, so that the tenderness is usually most marked here—again bringing appendicitis into the picture. But the initial pain is always worse, the pulse rate is frequently only 80 or thereabouts when the patient is first seen—that is to say, one of the cardinal signs of shock is absent, a feature not generally appreciated, I think—and there is no rise of temperature. It is as well to remember here that there is one acute abdominal condition which gives rise to as excruciating a pain as a perforated duodenal ulcer, and that is acute pancreatitis; but in this condition there is usually profound collapse with a very rapid and weak pulse and a well marked cyanosis, and the maximum tenderness and rigidity are found in the epigastrium. The adrenaline eye test and increased diastase content of the urine may help.

Traumatic perforations (or ruptures) of the hollow viscera do not as a rule give rise to such agonizing pain in the first instance as does a perforated duodenal or gastric ulcer; this is probably because the tear is usually small, and may become temporarily plugged by pouting mucosa. The tenderness and rigidity are at first localized, and, of course, there is the history of accident. These cases are very confusing at times because they are often associated with shock, and not infrequently with internal haemorrhage. It is in this class of case that a definite diagnosis frequently cannot be made, and a laparotomy will have to be done on suspicion of serious internal abdominal injury.

This attitude is quite justified, for, if one awaits a certain diagnosis, peritonitis or haemorrhage, or both, are usually so advanced that a fatal result is inevitable. The primary shock may be severe, and an operation have to be delayed on this account; but if the patient rallies in two or three hours and still the pulse rate does not drop, it is almost certain that there is some condition needing operative relief. These cases require repeated examinations, and at short intervals.

Obstructions.

An early diagnosis in intestinal obstruction is, perhaps, even more important than in the inflammatory conditions. It may happen that an inflammatory condition will get well of itself, but it is extremely rare for an acute obstruction to do so, and after the obstruction has lasted forty-eight hours the operative mortality becomes very heavy; in the table it will be seen that it is about 40 per cent., excluding strangulated herniae. Moreover, it is often difficult to differentiate acute obstruction from spreading peritonitis after the second or third day, when absolute constipation, incessant vomiting, rapid pulse, distension, and great prostration may be found in both conditions.

The three cardinal symptoms of acute obstruction in the early stage are pain, absolute constipation, and persistent vomiting. Pain is most marked when there is strangulation as well—for example, in volvulus and the various kinds of hernia. It is at first sharp and colicky, later more or less continuous, until it ceases with the onset of paralysis. In obstructions of the large intestine it is felt in the lower abdomen. Gradually increasing distension is a prominent sign of obstruction. In the earliest stages it may be confined to particular regions of the abdomen—for example, in obstruction of the small intestine it will be central, whereas in obstruction of the large intestine it may be most marked in the right side, across the upper abdomen, or along the whole course of the colon, according to the site of the lesion. Visible peristalsis may be seen at this stage, if the patient be not too fat, and periodic hardening of the caecum may be felt; this is a very useful sign of colon obstruction at the beginning of the attack. When the acute attack follows chronic stenosis of some duration a tumour may be felt, and it should be a rule always to examine the rectum.

Constipation.—This is absolute for both faeces and flatus, but there are one or two points to note. Both may be passed in small quantities with the first enema, owing to the bowel below the obstruction being cleared of its contents. Subsequent enemata will produce no result. Also, to be a dependable sign the enema must be a high one, given with the patient's hips raised. In acute intussusception frequent liquid stools may be passed, as there may be a sufficient lumen in the invaginated gut to allow of this, but other signs clinch the diagnosis, such as haemorrhage from the bowel, a sausage-shaped tumour in the abdomen, and a tumour felt in the rectum, and also tenesmus; and, of course, it is most often seen in children under 2 years of age.

Vomiting.—Faecal vomiting only occurs after the third or fourth day, and therefore has no useful place in diagnosis. The first vomit is stomach contents, and has a more or less innocent appearance; later it becomes bilious, and frequently repeated, much more so than in acute gastro-enteritis. The oft-repeated vomiting is in strong contrast to the vomiting of appendicitis, which occurs once or twice soon after the onset of pain, but is not frequently repeated until peritonitis supervenes.

In arriving at an early diagnosis the first question to be settled is whether or no an acute obstruction exists. If present, the earlier an operation is performed the better. The exact position of the actual lesion is not such an important matter; of course, an attempt should be made to ascertain this, and a good history will be most useful in this respect. If there is a scar of a previous operation, it should be kept in mind that adhesions associated with this may be the cause of the obstruction. The hernial apertures should always be examined, and also the rectum. A history of comparatively recent increasing difficulty with the bowels, slight colicky pain at times, possibly a little loss of weight and general well-being, and occasional distension of the abdomen in a patient at, or over, middle

age, would suggest a growth as the underlying cause. A previous history of gall stones should make one suspect that a stone may be impacted in the small intestine. In this condition the symptoms may appear rather insidiously, and visible peristalsis may be well marked. I have been able to diagnose the condition before operation in the last four cases I have seen.

In the early stages of acute obstruction repeated examinations are necessary. It may be impossible to be sure of the condition in the first few hours; if the bowels have not acted or enemata are unsuccessful, if vomiting continues, accompanied by an increasing pulse rate and abdominal distension, even if a definite diagnosis cannot be made, it will be in the best interests of the patient to have a laparotomy performed. A delay of forty-eight hours, when obstruction is present, may clinch the diagnosis, but will usually prove fatal from toxic absorption and the shock of the obstruction.

Haemorrhages.

The haemorrhages associated with gastric and duodenal ulcers, cholecystitis, appendicitis, and the like, though acute abdominal conditions, very seldom indeed require urgent operation, and I shall not deal with them here. The pathological lesions responsible for the type of haemorrhage in group 4 of my classification are practically confined to two—namely, those associated with injury, in which case there is a history of accident to guide one, and ruptured ectopic gestation, which I will deal with later.

In the presence of shock which the accident initiates it is almost impossible to say whether bleeding is going on or not. The symptoms are very similar, though with haemorrhage alone there is usually an absence of unconsciousness, cold clammy sweating, and dilated pupils. Sometimes shifting dullness may be found, and is suggestive of haemorrhage; it should be looked for particularly in the flanks and above the pubis, and when found in the early hours before the onset of distension it is a very valuable sign. Bleeding may stop as a result of the low blood pressure associated with the shock, only to begin again as the shock passes off, and one notices an increasing pulse rate and pallor, and the respirations become more shallow. It is essential to chart the pulse rate every half-hour. A slight degree of rigidity is often present, and also localized or diffuse pain. There is also tenderness coextensive with the rigidity. Increasing pulse rate, distension, and vomiting indicate an advancing peritonitis. If pain is due to injury of the abdominal wall alone it may be quite severe, and may be associated with rigidity too, but in this case the rigidity is never so marked as when there is visceral injury, and it yields to light pressure.

If a hollow viscus has been torn, vomiting, perhaps mixed with blood, is a prominent feature, and haemorrhage, tarry or bright red, may be passed by the bowel.

Injury to the kidney is followed by haematuria, possibly a swelling in the loin, and pain radiating into the testis or thigh. A diagnosis of rupture of the bladder depends upon the history and location of the injury, small quantities of bloody urine recovered on catheterization, pain in the bladder region, and constant ineffectual desire to micturate. The injection of fluid into the bladder as a test is unreliable.

Rupture of solid viscera gives the signs and symptoms of internal haemorrhage, but the maximum of rigidity and tenderness will usually be found over the injured organ. Fracture of the lower ribs often accompanies injury to the liver and spleen, and pain may radiate to the shoulders when the injury is to the liver.

In ruptured ectopic gestation there is sudden severe abdominal pain, with collapse and the signs of internal haemorrhage; the face and mucous membranes are blanched, there is great restlessness and thirst, and attacks of syncope, with a rapid, weak pulse. The abdomen is uniformly tender and there is a degree of rigidity, but not so marked as is found in the inflammatory conditions. The abdomen is distended and gives a peculiar doughy sensation to touch, and there is tenderness and fullness in Douglas's pouch. These are diagnostic indications of a

ruptured ectopic gestation if they occur in a woman who has had a long period of sterility, or a history of pelvic disease with irregular menstruation, and especially if menstruation has ceased for six weeks or so, during which the early signs of pregnancy may be seen, and at the end of about six weeks there is a discharge of clots from the vagina, with colicky pain in the lower abdomen.

The treatment is immediate operation, and it is surprising how well these patients do afterwards, even though they may be, and usually are, terribly blanched at the time. I have occasionally given a transfusion of blood, and it seems to help. Blood transfusion may be of the greatest value in those cases where the bleeding is due to injury.

I have not gone into a detailed differential diagnosis of all the surgical lesions included under the heading of the acute abdomen; it would make the paper much too long. In the early stages there is usually little difficulty in deciding which organ is responsible. Again, I have refrained from discussing abscesses and peritonitis. These are complications, and the acute abdomen should be operated on before they appear. If a patient suddenly experiences acute abdominal pain, followed by nausea or vomiting, or both, and this is accompanied by tenderness and rigidity, possibly in some degree over the whole abdomen, but most pronounced over the most painful area (which is suggestive of the site of the lesion), with or without shock, or if with the pain and vomiting there is absolute constipation, there is an acute abdominal condition present which is almost certainly of a surgical nature. If the lesion can be accurately placed so much the better; but this does not matter so much to the surgeon as getting the case early, for he should not tackle the job at all if he is not able to deal with any surgical catastrophe which may be exposed on opening the abdomen.

TWO CASES OF CARDIAC INFARCTION:

ONE FOLLOWED BY CALCIFICATION OF THE HEART,
THE OTHER BY RUPTURE.

BY

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DURING the past two years there has been a considerable increase in the literature on coronary occlusion, cardiac infarction, and rupture of the heart. The two cases to be described have been put on record owing to certain unusual and interesting features.

CASE I.

A man, aged 55 (shoe hand), was admitted in June, 1901, in a state of acute excitement with hallucinations. He slowly became demented, but led an active and useful life as messroom worker, vanman, and hospital messenger. In August, 1925, at the annual physical examination, the heart was found to be enlarged to the left nipple line; there was a soft mitral systolic murmur and occasional extra-systoles. The lungs were clear. On November 13th, 1925, he was confined to bed for a few days with bronchitis and slight oedema of feet and legs; there was no albuminuria. He was soon up and about again, working as usual. On the evening of December 12th, 1925, he returned from afternoon leave and complained of not feeling well. He went to bed and slept for an hour; at 10 p.m. he went to the ward commode, collapsed, and died at 10.3. There was no record of the Wassermann reaction.

Post-mortem Examination.

The heart was greatly hypertrophied and weighed 809 grams (28½ oz.); length from aortic ring to apex, 14 cm.; mid-ventricular diameter, 12 cm.; left ventricular wall, 2.5 cm. thick; right ventricular wall, 0.9 cm. thick. The pericardium was much thickened, and was adherent around the apex over an area 8 cm. wide; a plentiful supply of blood vessels passed from the adherent pericardium to the apex. There was a small quantity of straw-coloured fluid in the pericardial sac. The lower half of the left ventricle was ballooned out sufficiently to contain a golf ball. The anterior wall of this dilatation was fibrous, with scattered patches of calcification and extensive areas of recent sub-endocardial haemorrhage. The apex and the left lateral, medial, and posterior walls were fibrous, with marked calcareous deposit which extended 7 cm. along the interventricular septum. There was formation of fibrin on the calcareous nodules that projected here and there through the endocardium, which otherwise was intact, and lined the irregular inner aspect of the dilatation. The myocardium was brownish and soft, and the papillary muscles much hypertrophied. All the chambers were dilated and hyper-

trophied. The aortic cusps were thickened, and the mitral thickened and incompetent. There were a few small patches of atheroma in the ascending aorta.

The left coronary artery was healthy at its origin, but just inside the mouth there was atheroma with early calcareous deposit; 3 cm. from the origin of the main vessel there was complete occlusion of the anterior descending branch for a distance of 2 cm., and beyond that the walls were greatly thickened, with narrowing of the lumen. The right coronary artery showed slight atheroma.

Microscopical Examination.—The left coronary artery was atheromatous and occluded by organized thrombus. There was great proliferation of the intima, and fibrous tissue was arranged in whorls around small vessels formed during the canalization of the thrombus. Yellow granules of haemosiderin lay amongst the fibrous tissue. The muscle fibres of the myocardium were irregular

FIG. 1.—Case I. Section through calcified cup in left ventricle: (a) adherent pericardium; (b) calcareous nodules; (c) recent haemorrhage.

in size, stained indifferently, and many were dotted with black granules; fibrous tissue was increased between the muscle fibres. At the apex the muscle fibres were very scanty, and there was abundant white fibrous tissue overgrowth. The lungs showed signs of chronic bronchitis and old pleural adhesions. The kidneys were in a state of chronic interstitial nephritis.

Close inquiry revealed no history of any illness nor complaint on the patient's part to point to occlusion of the coronary artery, yet the anterior descending branch had been blocked and extensive infarction of the inferior half of the left ventricle, including the septum, had occurred. The infarct had undergone softening and been replaced by fibrous tissue which had stretched, producing aneurysm of the heart. Extensive calcareous deposit had occurred in the dead heart muscle, and judging from this the patient must have carried on with a damaged heart for a considerable time, possibly years, until he presented signs of heart failure in November, 1925, from which he apparently recovered, but which was followed by sudden death, possibly whilst straining at stool. Goodall and Weir¹ report three cases of coronary disease and rupture of the heart in patients who died suddenly in or near lavatories. Crawshaw² reports another case of ruptured ventricle in similar circumstances.

Hamman³ considers that occlusion may occur so slowly that no symptoms mark the final occlusion. Thayer⁴ quotes cases of recovery for periods of three years and thirteen years after infarction, and states that ultimately death may be sudden, or from gradual heart failure. The case described certainly bears out these points.

Calcification of the heart is rare. In Gibson's⁵ collection of forty-three cases of infarction from English, American, and Continental literature there is no example of calcification of the infarct. Scholz⁶ describes a case of calcification of an infarct following coronary occlusion in a male, aged 74, demonstrated radiographically *intra vitam* and confirmed *post mortem*. He remarks that where calcium salts are deposited in a cardiac infarct "the calcifying process gradually takes on a massive solid form involving a large area and all the elements of the heart wall. It may be worth noting that this apparent elimination of so large a portion of the heart muscle causes very little distress to the patient unless the conducting system of the heart happens to be interfered with by the calcifying process." Scholz also quotes a case of coronary occlusion, followed by calcification of the infarct, described by Burns in 1809, and a case described by Askanazy in 1901.