than one observer in this country is making use of it upon clinical material.

Observations in Various Conditions.

The only published observations of a considerable variety of conditions are those of Michaelis and Davidoff, who use the electrical method. Most of the results they obtained lie near to normal values, but they represent apparently single observations mostly made upon only one case of each disease. In diabetic coma they found a definite diminution of effective alkalinity. This finding we have, of course, long been led to expect, though the occurrence of any change of blood reaction in this condition has been denied. Some of the denials have been based upon results got by unreliable methods, and the question cannot be considered as settled. Kennaway, Pembrey, and Poulton⁷ have lately pointed out that the alveolar tension of carbonic acid is the best guide to prognosis and treatment in diabetes. A value below 25 mm. is, according to these authors, of grave significance, and one below 20 mm. indicates that coma is imminent. One would think that these low values are associated with the establishment of a definite rise in the hydrogen ion. Dr. E. P. Poulton is engaged upon investigations which deal with the blood dissociation curve in diabetes. Evidence so obtained will, I have no doubt, settle the question for us.

In conditions such as those of cyclical vomiting, the vomiting of pregnancy, eclampsia, and the like, in which a possible intrusion of acidosis as a causal factor is suspected, we want clearly to know whether an actual change in the blood reaction has occurred before we can be clear as to the exact relations which exist. Michaelis and Davidoff give no data respecting these conditions. They found nearly normal values in chronic nephritis, though we have no information as to the type of the disease or of the stage at which it was studied. The older literature teems with observations showing a diminished titration alkalinity in uræmic conditions, and, as I have already mentioned, Poulton and Ryffel have discovered a real increase in the hydrogen ion concentration. The case of cancer is interesting, as while so many have found diminished alkalinity in malignant disease, the investigations of Benjamin Moore, described in 1907, pointed to a small but measurable increase of alkalinity as measured by titration. To these changes the author attached a causal significance. Michaelis and Davidoff found in carcinoma a normal hydrogen ion concentration, but it is clear that much more research will be required to establish the facts. The case of cancer blood may prove to be specially difficult to investigate from the point of view of hydrogen ion concentration. About a year ago I was engaged upon a study of the effect of certain diets upon cancer, and Barcroft examined the blood of some of my carcinoma-bearing rats. To all appearance there was a marked diminution of alkalinity, but Barcroft's scientific acumen prevented him from making a wrong conclusion on these lines. He found that the blood, owing to the existing anæmia and probably to the existence of newly formed corpuscles, showed a tendency to rapid self-reduction, and this accounted for the apparently meionectic dissociation curve. With any blood exhibiting within itself considerable metabolic change like this, an estimation of the

ionic reaction may be difficult by any process. Michaelis and Davidoff found an appreciable diminution in the hydrogen ion concentration in cases with fever. This is a point of interest. It can be shown theoretically that a fluid such as blood should become more alkaline with a rise of temperature, and if, as seems to be the case, no complete adjustment due to increased acid production occurs in fever, we have good reason to believe that a more alkaline condition of the blood in fever plays its part in determining the increased metabolism. Oxidation processes are known to be increased in velocity by an increase in solution of the hydroxyl ion.

ACUTE PANCREATITIS:

A CLINICAL STUDY OF SEVEN CASES; FIVE TREATED BY OPERATION SUCCESSFULLY.

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No apology is needed for recording even a single case of acute pancreatitis completely arrested by immediate operation on definite lines, but the one now first described is the second which I have recently seen and treated in the same way with recovery. And had it not been for the experience gained in the previous case¹ I hardly think the action taken in the last would have been so definite. Besides my own two cases, I am permitted by my colleagues Dr. Batty Shaw, Mr. R. Johnson, Mr. W. Trotter, and Mr. H. M. Davies to refer to further cases, five in number, which have been under treatment in University College Hospital since my first—three being equally successfully treated by operation, one fatal after operation, and one not operated on.

I am particularly obliged for this permission as a group of cases occurring close together as to time and treated by men closely associated is of special value. For few surgeons can claim to have had any very large experience of the disease in question as a field for operation, and fewer still to have had much success in dealing with it. This is a matter of some note in view of the countless abdominal catastrophes which we have all to treat nowadays. It may be best explained by the great difficulty in diagnosing pancreatic affections owing to the variety of clinical features they present in their several forms and the suddenness of the attacks. Here and there we read of individual cases met with in the course of abdominal explorations for obscure conditions, and some of these have recovered. But we are still in need of a clear clinical picture of the symptoms of acute pancreatitis and of definite guiding principles in operating for it. This may be seen from the classical and exhaustive monograph on the pancreas published in 1898 by Körte.² In this he reviews the whole literature of the subject up to that date, and states that as to any characteristic symptoms and signs of acute pancreatitis no advance had been made since Claessen in 1842 had expressed the opinion that "there were none." Only deep-seated pain in the epigastrium and possibly a swelling above the umbilicus, if present, were of value from a diagnostic point of view.

Since 1898 much more attention has been given

¹ Vide THE LANCET, 1910, vol. ii., p. 1264.

⁷ Proceedings of the Physiological Society, Journal of Physiology, vol. xlvii., 1913.

² Körte: Die Chirurgischen Krankheiten des Pankreas, Deutsche Chirurgie, 1898.

to these affections, especially as Körte had shown by successful operation in one case that they were curable by surgical interference. Their causation, morbid anatomy, and clinical history in uncomplicated cases have since been more accurately studied and recorded, and some advance in our general knowledge of them has been made. Nevertheless, the obscurity which hangs over these sudden attacks is very great, and definite characteristic diagnostic signs are still wanting.

That there are several different forms of the disease is now well known to all morbid anatomists. But whether these (most probably, if not positively, all directly or indirectly infective) differ simply in the virulence or duration of infection with the same organism, or whether they depend upon inoculation with totally different bacteria alone or combined, displaced secretions such as bile, or the succus entericus from the duodenum we do not know in spite of much study, both pathological and experimental. We do know that there is a chronic pancreatitis lasting a long time without immediately threatening life and presenting few, if any, marked symptoms until the organ is gravely compromised by interstitial fibrosis; also that there is an acute inflammation of the organ associated with the escape of much blood into its substance and around it (= acute hæmorrhagic pancreatitis); that there is a more localised suppurative form producing one or more distinct abscesses in the organ; and, finally, that there is a fulminating form in which, if the patient survive the severe shock of the onset, a very extensive and rapid sloughing of the organ and surrounding tissues gives a peculiar character to the process.

What relations these several varieties have to one another is not yet clear, and space forbids me to speculate about them here or to allude to the various views held in regard to them. But it is very desirable to keep them in mind when we have much general abdominal work to do, and in operating to note every morbid change *in vivo* in and around the pancreas which may help to explain them. Now that the patients suffering from these affections have begun to be saved by surgery, much that is obscure about the earlier morbid changes in and around the organ ought to be cleared up while the patient is living in a way impossible on the post-mortem table when the disease has much later proved fatal. Moreover, there is a practical value in acknowledging the above-mentioned varieties. A surgeon even suspecting that he has before him a case of acute pancreatitis will, when he has explored it, be able without loss of valuable time and a minimum of cutting and manipulation to modify his procedure to suit the particular variety. For no stereotyped practice appears to be suitable for all. It is one thing, for instance, to deal with a limited acute inflammation of the head or tail of the pancreas which has reached the omental sac and can be approached and drained through a median incision into this; it is quite another thing to attack a destructive process *early* which lies *behind* the peritoneum, is spreading in the retroperitoneal and mesenteric tissues, and has either already resulted in sloughing there or may be expected to do so ultimately, as we shall see. In the latter case an anterior incision and draining of the omental sac will *not* be adequate and a lateral one in the flank should be made to give access to the retroperitoneal area around the pancreas by burrowing behind the serous membrane, as was done in the first and last of these

cases recorded below, and was suggested by Körte long ago. Had this been done in my earlier case (No. 2), at the outset the inflammatory process might have been arrested sooner and perhaps the second operation avoided. At all events, the serious adhesions which followed the second transperitoneal operation and which caused bad intestinal obstruction ten months later might have been prevented. This third operation, though very trying, was happily successful.

From these two cases in which it has fallen to my lot to operate for acute pancreatitis successfully I received the impression that possibly in some of its earlier stages the inflammation may be benefited, at all events for a time, by drainage of the omental sac. But where there has been much extravasation of blood or pancreatic juice at the onset, subsequent so-called "fat necrosis" and sloughing are sure to follow, and will require sooner or later to be drained, and this is most easily and safely done from behind by a tube introduced through the retroperitoneal tissues. In my earliest case (No. 2) simple drainage of the bursa omentalis was all that was done *at first* and with much temporary benefit, all the symptoms having subsided by the ninth day, when the stitches were removed from the healed abdominal median wound. Then after this deceptive pause the trouble recurred, and at the end of four days a swelling was found in the splenic region and reached by a transperitoneal incision in the left flank. This revealed advanced *subperitoneal* fat necrosis, as well as a large collection of blood-stained fluid in the peritoneum near the spleen. Drainage of both areas was followed by recovery, and the patient remains well. It seems obvious that had retroperitoneal drainage been provided in the first operation the second might have been unnecessary, and the intestinal adhesions requiring a third might have been prevented.

It seems also clear that it takes some time after the initial lesion for a necrotic process to develop an abscess (in this case nine or ten days), and that the drainage of the omental sac, though immediately beneficial, was not adequate to arrest it. But in the other two cases, Nos. 3 and 4, successfully operated on by my colleagues, the collection had actually burst into the omental sac and drainage of this cavity was sufficient, sloughs and pus coming away in large amount. Sloughs and altered blood clot also came away ultimately in my last case (No. 1) through the retroperitoneal opening in the flank, *but much later*, and I assume that the disintegrating process had only just started when I operated. As a matter of fact, the retroperitoneal drainage was interrupted by the premature shortening of the tube, which we thought was indicated by a temporary general and local improvement of conditions, and it was only when a fresh and larger one was reinserted right up to boggy tissue behind the pancreas in the middle of the fifth week that the products of disintegration came away freely with sloughs and altered blood clots and with rapid and permanent improvement. Still, the first tube as long as it worked was beneficial. Had it been left unshortened the process might have been cut much shorter. But feeling one's way on almost untrodden ground such as this is no easy matter. My idea in putting in a retroperitoneal drain at once was to anticipate changes which had been seen to follow even where the omental sac had little or nothing in it, and to minimise the spread of the process in the retroperitoneal tissue as far as

possible, as free drainage even of serum will do in other conditions.

It is worth emphasising once more that in my own two cases the destructive process in the subserous fat followed its chemical splitting up by the pancreatic juices at some considerable interval of time: as far as I can judge by symptoms and temperature chart at about the end of the second week. In Case 3 extensive sloughing was found four weeks after the onset. In Case 4 a grumous collection of broken-down blood clot 57 hours after the onset, which was very clearly indicated as to time. Fig. 1 (Case 5) shows the appearance of the pancreas five days after a well-defined onset; so far no sloughing is to be seen. Fig. 2 (Case 6) shows the condition 24 to 30 hours after the onset, beginning at 2 P.M. the previous day. Intense congestion and some fat necrosis are the only changes seen. It seems probable also from what was seen in my own two cases that every lobule of fat affected in this way does not necessarily lead up to suppuration. Many outlying lobules of whitened fat were seen in the omentum far from the pancreas. They gave rise to no sign during convalescence which could indicate that they had become the foci of inflammation. Probably they were absorbed in course of time. It may be that outlying lobules escape bacterial invasion, and that this is necessary for the sloughing usually associated with pancreatitis. But that the fat-splitting plays a part in this sloughing, and that this is more marked close around the organ where the bacteria would be more numerous appears more than likely.

An interesting observation bearing on this point was made by Körte (*loc. cit.*) in operating for acute cholecystitis. Here he noticed a "typical fat necrosis" with much peritoneal irritation, but without any ascertainable disease of the pancreas. A year later, in operating on the same patient for an impacted stone in the common duct, he found that all trace of fat necrosis had disappeared. It seems possible, however, that at the first operation a mild pancreatitis may have been overlooked. This would have accounted for the peritoneal irritation which is described as very marked. We have still much to learn both as to the nature and exact mode of production of fat necrosis in and around the pancreas, in the mesentery and omentum. But as a practical point, when it is recognised on exploring the abdomen in an acute case, it is the most valuable guide that we possess as to the nature of the ailment. In my own two cases it led almost at once to the right conclusion and to the right steps in treatment. When attention was first drawn to "fat necrosis" by Balser³ in 1879-82 it was wrongly interpreted. Langerhans appears to have been the first to point out that the change in the fat cells was due to the fat-splitting effect of the pancreatic secretion, after which the fatty acids combine with calcium to produce the white material which is so characteristic and which leads to the destruction of the cells.

Later Hildebrandt proved experimentally that either an engorgement of the organ with its own secretion or with blood, or the escape of its own secretion into the peritoneum will produce typical fat necrosis in the pancreas itself, in the mesentery or omentum. If this be so there may be several ways in which the change may be produced. Injury as in some recorded cases; anything which arrests the passage of the pancreatic juices into the

common bile-duct, whether it be a gall-stone or pancreatic concretion; inflammatory swelling due to infection in its duct, or, as suggested at the last International Medical Congress by Dr. Archibald, of Montreal,⁴ a spasmodic contraction of the muscle around the outlet of the common duct into the duodenum due to irritating material in the latter may cause retention and ultimate extravasation of the fat-splitting pancreatic secretion; and those first changes noticed in early cases. Bacterial infection will do the rest, and in due time lead to extensive sloughing.

If this broad conception of the process be correct immediate and prolonged retroperitoneal drainage of the fat around the organ would appear to be the rational treatment. At all events this was the conception in my mind in the first of the cases in the list given below, and the result of the drainage was success in arresting the process, although the drainage was interrupted for some days. In another seventh similar case operated on during my absence last Easter in one of my beds by Mr. H. M. Davies success followed the same *retroperitoneal* drainage through the right flank. The details will be published later.

The following notes are abstracted from the more voluminous clinical reports of the cases, but contain all the essential facts and observations made at the time.

CASE 1.—On Nov. 12th, 1913, I was hurriedly called by Dr. A. Binning, of Hatfield, to see a patient who had been suddenly seized with very severe abdominal symptoms the previous evening; and I may say at once that it was to Dr. Binning's promptness in recognising the gravity of the condition as much as to anything done by operation that the patient owes his life.

The patient had been in the army and had served abroad. Health always excellent until recently. Physique splendid but inclined to stoutness. Family history good. At 12 years of age had typhoid; was ill 13 weeks. Had fever for four days in Malta in 1904 but none since. Has had colicky pains at times, relieved by wearing a belt. Earlier in 1913 had a severe attack of abdominal pain and shivering, which caused him to roll on the floor; this passed off the same night. No syphilis or gonorrhoea. Lived very generously, drinking about 4 pints of beer and occasional spirits daily and eating much. Before the present attack he had for 12 months no energy and frequently vomited greenish fluid in the morning. Smoked cigarettes by preference.

The present attack began on Nov. 8th. The patient had felt tired and drowsy all the afternoon and evening; no pain. Had had usual four pints of ale and one glass of port. Nov. 9th: Better, but tired and drowsy as before; vomited phlegm at 7 P.M., and again at 10 P.M., when there was a sharp pain across the abdomen "as if something had given way," and which kept getting worse until the operation. Nov. 10th: Had had no sleep and could not stand up for the pain; felt sick all day but did not vomit. Nov. 11th: Saw Dr. Binning and was sent to bed. Bowels acted after calomel. Nov. 12th: Was much worse in the morning; pulse 116. I saw the patient at 2 o'clock.

The appearance of the patient indicated a very grave intra-abdominal catastrophe. The pain was exceedingly severe and vomiting had taken place. Pulse feeble, 120, temperature 100.2° F. Face wore a look of great distress and was pale and cyanosed. The abdomen distended, and this was increasing. It was soft below, harder above the navel, where the pain was most intense. Tenderness was extreme above and on each side of the umbilicus, but most over the head of the pancreas. Tumour could not be made out on account of rigidity. No specially distended coils. No particular tenderness or dulness over the appendix. Flanks clear on percussion. The vomited matter was bilious and contained no blood. My diagnosis was provisionally perforated gastric or duodenal ulcer, but pancreatitis was considered.

³ Balser: Virchow's Archiv, Band xc., p. 520.

⁴ E. Archibald: Transactions of International Medical Congress, Section VII., Surgery, p. 21.

Operation on Nov. 12th at 8 P.M., assisted by Dr. Binning. Median incision above the navel showed much intraperitoneal turbid fluid as in my earlier case No. 2 and in No. 4. This lay mostly in the upper abdomen over the right kidney. The stomach was carefully examined anteriorly and through the omental sac, opened for the purpose. It was quite sound. The omental sac contained no fluid. Great congestion and general shagginess of the peritoneum over the head of the pancreas was very striking. The organ was swollen to the size of a large fist at its right end, and showed fresh exudation of lymph. When the depression to the right of the duodenum was sponged dry the peritoneum was seen to be bulged forwards with chalk-white subperitoneal fat in which were a few hæmorrhages. This white material extended on the right of the spinal column from the head of the pancreas upwards to the liver border. It had not broken through its serous covering. Its appearance was so characteristic taken with the swollen pancreas as at once to clear up the nature of the disorder. It was obviously very acute pancreatitis in a fairly early stage.

The problem now was what to do. It was no use to drain the sac of the omentum, as was done in my earlier case, as it was empty and there were no signs of the process advancing into it. On the other hand, it seemed risky to incise the swollen area and allow the inflammatory products to escape into the peritoneal cavity and drain through this, in view of the serious adhesions from similar drainage which nearly cost my earlier patient her life. I therefore made a two-inch incision below the last rib on the right side and deepened it down to the subperitoneal fat. With my left hand within the abdomen between the duodenum and the kidney I got the direction and then tunnelled with the right forefinger through the fat behind the peritoneum up to the head of the pancreas. This was not so difficult as expected. A long large drainage-tube was introduced along this track to the head of the organ to provide for the escape of serum in the first place and the products of the inflammation as they were formed. As an extra possible drain for the pancreas the gall-bladder was drawn into the upper angle of the abdominal wound and stitched there. It showed no signs of disease, contained no calculi, and could be emptied by gentle pressure into the duodenum, showing there was no obstruction of the cystic or common duct. When opened it was found to contain perfectly normal bile (*vide* note of Nov. 27th for contrast), which was drained by a rubber tube into a bottle outside the dressings. The median wound was then stitched up in layers. The intestines had been seen to be a little distended and hyperæmic, but not paralysed; there was much turbid fluid to be mopped away in the upper part of the abdominal cavity.

The patient's condition during the operation was generally good, as reported by Dr. Dudley Buxton, who gave the anæsthetic (chloroform plus oxygen). Pulse at the end 120. The next day the condition was improved; pulse 110 to 116. During the first week the chief features of the case were reduction of pulse-rate and temperature, profuse sweating, sleeplessness, restlessness, and tendency to wandering at night; cyanosis still notable at times. There was also an attack of foul diarrhœa on the fourth day. In the second week general improvement; pulse 88 to 98; but temperature 97° to 101°. Profuse sweating continued for first four weeks. After the diarrhœa on fourth day there was always constipation and flatulence, only relieved by enemata. At no time was fat detected in the motions, though looked for carefully. The stitches in the anterior wound were removed on the twelfth day (Nov. 23rd); perfect primary union. Gall-bladder in upper angle draining well.

Third week much general improvement; food taken well. The gall-bladder yielded abundance of pure clear bile. The tube in the flank drained so little and the progress generally seemed so favourable that it had been shortened by degrees to about two inches. On Nov. 27th (sixteenth day) temperature rose to 101° for the whole day, and at this time Dr. Binning noticed a deposit in the bile bottle, which was proved by microscopic examination to be pus. It amounted to about an inch deep in a six-ounce bottle. This pus came away in the bile freely for about a week from the 27th, the temperature at the same time falling steadily to normal on the 31st. On Dec. 3rd the temperature rose once to 102°, then dropped to absolute normal for 4th, 5th, and 6th, on the 7th it again began to oscillate about 100° to 101°, and on the

10th it once touched 102°. This fairly regular oscillation lasted for the next two weeks each day and then fell permanently to normal with the re-establishment of free drainage from the track in the flank which had become partly closed.

During the week of highest temperature (Dec. 9th to 16th) the patient had two nervous explosions of a very startling kind with a day of very good condition in between them. They had been preceded by the disappearance of the pus in the bile, which had been abundant for 12 days, and by the fall in temperature mentioned above. On some of the previous days severe headache had been complained of and vomiting had occurred, but on the 8th, 9th, and 10th he was noted as having had "very comfortable" days without headache or sickness. Then on Dec. 11th, at 5 P.M., he had the first of these strange attacks, which I can only suppose were of the same nature as the "tetany" seen sometimes with pyloric obstruction and in cases of severe diarrhœa in children, and described by Dr. F. J. Poynton and others. The nurse's notes, which were admirable all through the case, may be given in abstract as checked by Dr. Binning. "At 5 P.M. patient felt very faint; complained of not being able to see or breathe properly; pulse quick and small. 5.30, suddenly became rigid for a few seconds and unable to speak; remained so for nearly 30 minutes; convulsed at intervals." At 6.30 saline rectal injections were attempted, but excited the patient, who then commenced to scream and get violent; tried to vomit. Violent and convulsed off and on until 7.10, when he was seen by Dr. Binning, who described this condition to me at once over the house telephone, and suggested putting the patient under chloroform, to which I agreed. This was done at 7.30 with immediate effect. At 8 o'clock he was again awake, quite sensible and quiet; temperature 101°, pulse 114. He was soon inclined to sleep and had a quiet night; passed 8 oz. of clear normal urine. A sample about this time sent to University College Hospital for analysis. Dr. F. H. Thiele reported that the Cammidge test gave a positive result. The following day and night were very comfortable, the sweating was gone, urine free, normal.

On the 13th a similar attack to that on the 11th occurred, but less severe. He was "wandering" frequently during the evening, but slept well the following night and seemed to have no recollection of the attack. I saw the patient the next day, and feeling that the shortening of the tube in the flank might have contributed to the fever, &c., as very little discharge came from it now, I enlarged it with my finger as far as I could reach. At a depth which must have been close to the duodenum the finger came upon a quantity of pulpy material behind the peritoneum. Into this I pushed a longer and larger drain-tube. This was all done under light chloroform anæsthesia. The fomentations were continued as before.

From this on there was no further serious trouble. Very free discharge was soon established through the tube, and several sloughs, large and small, came away during the next 12 days, which on examination were believed to be bits of the pancreas. Small portions of broken-down old blood clots were frequently noted, and also from the 22nd to 29th that "small white spots" were present in the discharge, which during that time was very offensive and varying in amount. After the reinsertion of the tube on Dec. 14th the temperature varied between 98° and 101°, and on the 24th it touched 102°. After this, as the discharge from the flank became free and offensive, the temperature fell to 99°, and a couple of days later to normal. After Dec. 25th the retroperitoneal discharge became less and less in amount and in foulness, and the tube could be gradually shortened. The last slough came away on Jan. 5th. After this convalescence was rapid and the patient was able to go to the seaside at the beginning of February. The relation of the appearance of much pus in the bile, and the tetany, to defective drainage from behind seems clear, especially as these symptoms disappeared and convalescence was rapid when the tube had been effectively reinserted and acted well.

CASE 2.—Mrs. A, aged 40, was admitted into University College Hospital on Feb. 21st, 1910. Two days earlier severe griping pain set in above the navel. She vomited and trembled with pain, but there was no definite rigor. The pain was paroxysmal. Bowels confined since Feb. 13th, no flatus; no result from two enemata on 21st. Past history: Six years ago a similar attack after birth of first

child, accompanied by vomiting and constipation. Describes another lasting 48 hours soon after the birth of the last child six months ago; no jaundice. Present state: Well nourished; no facies suggesting acute peritonitis. Temperature 101.4° F.; pulse 124. Abdomen soft and not distended. Pain in epigastrium, left hypochondrium, and left lumbar region. No dulness in flanks; liver dulness normal. No blood, sugar, or albumin in urine. No shock, but patient looked very ill. Diagnosis doubtful; exploration necessary.

Operation on Feb. 21st at 9 P.M. Median incision below umbilicus. There was a considerable amount of thin, slightly turbid fluid in the general peritoneal cavity. The small intestine wherever seen was markedly injected, slightly dilated, but not paralysed. Search throughout its length for anything abnormal was negative except for a few flakes of lymph above. The colon was then drawn down and spots of "fat necrosis" were seen in the omentum, which at once suggested to me the true nature of the case as one of acute pancreatitis. The omental sac was opened between the stomach and colon. There was no fluid in it, but more patches of fat necrosis were seen, in greater number as the pancreas was approached. The organ itself was hardened especially about its head, but there was no evidence of breaking-down tissue or hæmorrhagic change around it. Nothing more than drainage of the omental sac was done. The wound was closed after a piece of omentum containing fat necrosis had been excised for examination. The change in the fat lobules was typical and showed up grass-green with Benda's stain. (Contrast Fig. 1 from Case 5.)

The patient rapidly improved and the stitches were removed on the tenth day; primary union. On March 3rd (eleventh day after operation, thirteenth after onset) severe pain set in "from nipples to navel." There was tenderness over the whole abdomen, but most at the tip of the right ninth costal cartilage. Pulse 124; temperature 98°. At 10 A.M. morphia was given, but pain continued all day. Vomiting began at 4 P.M. up to 2 oz. in three acts. March 4th: Temperature 100° in the morning and 101° in evening. Vomited in all 12 oz. Seemed better in spite of pain; abdomen not distended any more. Pain had shifted to tip of left tenth costal cartilage.

On March 5th the pain and tenderness had increased; temperature 101°. In the evening under chloroform a swelling was now felt in the left hypochondrium, and I made an incision commencing 2 inches to the left of the umbilicus and running backwards and upwards for some 4 to 5 inches. Fat necrosis was at once recognised in the subperitoneal tissues, and portions were removed for analysis. The peritoneum was then opened far back, and about 6 oz. of blood-stained fluid mixed with pus escaped. This was followed by very free venous bleeding, which ceased on the introduction of a large drain-tube. The mass felt before the operation was now found to be the enlarged body of the pancreas. The spleen was not enlarged. For the next five days there was steady improvement, with much discharge. Temperature generally about 100°. There was a return of pain in the right hypochondrium on March 10th, with rise of temperature to 102° and vomiting; no rigor. This severe pain continued over a palpable mass below the navel; no jaundice. The pain then subsided and steady convalescence followed.

The patient was discharged well on March 31st, 1910. She remained well until the following October, when she was re-admitted for bad intestinal obstruction by band and volvulus in the left flank. This I released by a third operation through the old scar in the flank. She has been well ever since. On this occasion I examined the conditions in and around the pancreas and found nothing abnormal remained from the former attack. Had retroperitoneal drainage been carried out somewhat earlier it seems probable that the peritoneal adhesions requiring this third operation would not have been produced. The notable improvement for 11 days which followed the drainage of the omental sac was deceptive. Around the pancreas the disintegrating process was steadily advancing and could only be approached from behind.

CASE 3.—The patient, a widow aged 42, was admitted into University College Hospital on April 3rd, 1911, complaining of "pain in the stomach soon after food for the last four weeks." It was stated to go through to the back and be relieved by vomiting. It sometimes occurred immediately,

sometimes two or three hours after food. The vomit at first was dark, then in last few days greenish. She described a similar attack in previous August; was subject to "biliousness." On admission there were rigidity of abdominal wall and general tenderness, most severe in right epigastrium. Frequent vomiting occurred, and rectal feeding was adopted. On April 11th, under an anæsthetic, a mass of the size of a coconut was found in the umbilical region. It was distinct from the liver and well defined below; movement with respiration doubtful.

On April 12th Mr. H. Morriston Davies made a 3½ inch vertical incision through the upper part of the right rectus muscle half an inch to the right of the middle line. An elastic mass was found behind the stomach and transverse colon. Above, it reached to the liver, below to 3 inches below the navel, and on each side 3 inches from the latter. The small intestines were found adherent to the cystic mass below the colon, and on separating them an opening was made into it accidentally, dark grumous fluid escaping. The parts had been previously packed with gauze. More fluid followed by a large sloughy mass was squeezed out. It proved to be part of the pancreas. The upper part of the swelling was firmly adherent to the transverse colon and pressed upon the duodenum and bile ducts. Gall-bladder distended but healthy. The cavity was further opened between the stomach and colon, and about a pint more of the grumous fluid evacuated. Both openings above and below the colon were drained with large rubber tubes brought out of the abdominal wound, which was stitched up. Convalescence progressed steadily, some irritation of the skin from the discharge being the only check. On May 1st another large slough came through the upper opening. All well by May 18th and patient left for home on 19th. The temperature was never very high, 100.6° F. being the highest on April 20th; after this the average was 99°.

CASE 4.—The patient, a man aged 39, was admitted to University College Hospital on Sunday, August 6th, 1911, at 12 noon, for pain, vomiting, and absence of stools for 48 hours. Until recently had been in usual health and only returned from a week's holiday in the country the previous Monday. Violent pain in abdomen set in 48 hours ago sufficient to make him rest, but it passed off and he went to work. At midnight the pain returned intensely and he vomited at first food, then bile and slime. No sickness since noon on Saturday. But the pain had persisted always in the umbilical region without radiating. No flatus or stools since Friday in spite of enemata.

Previous history: Described a similar attack in July, 1906, with intense pain in the abdomen for two days with vomiting and constipation. He remained in bed then for 11 weeks with symptoms apparently of gastroenteritis. In 1907 he had a similar attack and was in bed for three weeks. In 1908 a third attack like it; in bed one week. Since then quite well on careful dieting. Never jaundiced; never lived abroad. Influenza 20 years ago.

Present state: Looked ill and anxious and complained of pain in abdomen; resented being moved. Temperature 100° F.; pulse 108. The abdomen moved fairly to respiration; was distended generally; umbilical region specially prominent, more on left than right. No bulging in flanks, but slight shifting dulness; otherwise resonant. Liver dulness normal. There was tenderness, not very marked, below the navel more to left than right, but no hyperæsthesia of the skin. Owing to rigidity no definite tumour could be felt anywhere but the resistance suggested distended gut. No visible peristalsis. Organs sound; rectum empty. At 4 P.M. bowels still not moved; no vomiting. Temperature 99.4°; pulse 112. At 6 P.M. pain and distension more marked; pulse 120.

Operation by Mr. Wilfred Trotter on August 6th at 9 P.M. Median incision below navel. Much yellowish peritoneal fluid escaped; no obstruction of bowel; appendix normal. Small bowel in middle of its length distended and hyperæmic, but no peritonitis. Mesentery dotted with fat necrosis. Incision prolonged upwards; gastrocolic omentum bluish in colour, bulged forward by fluid in omental sac. On incision dark, blood-stained fluid welled out quite unlike that in the general cavity of the peritoneum. This was explained by the fact that the foramen of Winslow was closed by lymph. About 1 to 1½ pints of liquid escaped from the sac of the omentum, which was sponged dry. The pancreas was now seen to be greatly swollen, covered with lymph, and its

central part very dark, much enlarged, and infiltrated with blood. No incision into the organ was made, reliance being placed on drainage of the omental sac. This was provided for by a rubber tube and gauze enveloped in oil silk going down to the pancreas. Wound stitched up round these.

Condition after operation fair; pulse 120; respiration 32; some cyanosis. At midnight shock was marked, but gave way to restoratives. August 7th: General improvement; temperature 98°; pulse 120; flatus passed; no vomit; urine normal, no sugar. From this on to August 27th steady

CASE 5.—The patient, a well-nourished man aged 52, was admitted into University College Hospital on June 17th, 1913, under Dr. Batty Shaw's care, with a history of "pains below the navel and reaching round the back, scanty urine and retching" for three days. Bowels not moved since the 15th. Temperature 99° F.; pulse 60; respiration 30. His habits appeared to have been regular; internal organs fairly healthy; occasional headaches; appetite good up to the 15th. Nothing notable in family history on past illnesses.

Present state: Pain and tenderness at umbilicus, mostly

FIG. 1.

CASE 5.—Acute hæmorrhagic pancreatitis. A, Omentum showing fat necrosis stained green by Benda's copper method (the green stain comes out black in the illustration). B, Pancreas showing a, hæmorrhagic patch; b, fat necrosis unstained.

convalescence; discharge less and less. The brownish particles seen in the pus of Case 1 are noted as present here too. There was no irritation of the skin from the secretions as in Case 3. The urine tested by Cammidge's method gave negative result. With the exception of some sickness due to error in diet on August 27th, there is nothing further to report before the patient left hospital on Sept. 9th quite well. During convalescence there was a tendency to constipation.

just below it; some rigidity in this region. Abdomen did not move with respiration; flanks bulging; no dyspnoea. Rectal examination negative. Washing out brought away practically no motion, but much membranous material suggesting colitis. Temperature 100.4°.

June 18th: Had been sick twice to 6 oz. At 3 P.M. vomited 16 oz. of dark brownish material. At 4 P.M. feeling worse; marked hiccoughing frequently. Temperature 98.4°; pulse had risen to 120, and was weaker. Abdomen now

slightly distended; no marked increase in tenderness, which was now most evident in the epigastrium. Intestinal obstruction diagnosed. Operation at 11 P.M. same day by Mr. Trotter; median incision. Patient died the next morning, a few hours after operation.

Post-mortem notes, by Pathologist, June 19th, 1913:—"Lungs: Both very oedematous and congested; extensive adhesions in pleuræ. Peritoneum: numbers of small nodules of fat necrosis are present in the great omentum, root of mesentery, fatty capsule of kidneys, and fat about the pancreas. Pancreas: greatly swollen, the head measuring 2 inches from before backwards; extensively hæmorrhagic and in places broken down and showing islands of fat necrosis. The duodenal end of the duct is distended with a number of small calculi; no pus present. (Fig. 1.) Liver: shows cloudy changes; gall-bladder, ducts, and bile papillæ are normal. Stomach and intestines: stomach lining shows scattered submucous hæmorrhages; these are also present in the upper part of the ileum, and some of them are ulcerated.

In view of the general condition of the patient it was felt that any operation was out of the question. Mr. Johnson's diagnosis was acute pancreatitis. He thought nothing else would account for the condition of severe pain, extreme cyanosis, and collapse after sudden onset, so well, though no tumour was discoverable. The patient died a few hours after admission, and the post-mortem examination showed that he was right.

Pathologist's report:—Abdomen: "About half a pin of opaque, blood-stained fluid in the abdominal cavity a few flakes of fibrinous exudation in places. Scattered areas of fat necrosis of small size in the omentum larger areas in the retroperitoneal tissue below the pancreas, where also there is considerable extravasation into the areolar tissue. The pancreas is greatly swollen with much extravasation of blood on its surface and into the septa between its lobules. There are numerous white areas of fat necrosis on the surface of the organ. There is no sloughing or breaking down of the pancreatic

FIG. 2.

CASE 6.—Acute hæmorrhagic pancreatitis. Mr. Johnson's case, Feb. 22nd, 1914 (not operated on). *a*, Wirsung's duct inflamed in middle portion. *b*, Head of pancreas much swollen and hæmorrhagic. *c*, Fat necrosis stained green by Benda's copper method (the green stain comes out black in the illustration). *d*, Unaltered fat tissue.

The intestines contain brown fluid. Spleen soft, follicles enlarged. Kidneys normal."

CASE 6.—The patient, a single man aged 65, was admitted into University College Hospital on Feb. 22nd, 1914, for severe abdominal pain beginning at 2 P.M. on the day before and lasting ever since. The pain was not localised. Vomiting began soon after the onset and occurred 12 times before admission. Flatus but no motion passed last night. Had been in good health until the 21st. The attack came on after a good meal. When seen by Mr. Raymond Johnson cyanosis was very marked even in the fingers. The man appeared desperately ill and collapsed. Pulse 130, intermittent; respiration 38; temperature 97.1° F. Tongue dry. Abdomen slightly distended towards the left iliac region; did not move with respiration. Tenderness and rigidity were general. No definite tumour could be felt in the abdomen. Nothing abnormal could be detected in the chest.

tissue, except at one spot, which is somewhat softened and of a grey red colour. (Fig. 2.) Stomach: dilated; scattered petechiæ on its mucous membrane and swelling of its follicles. Spleen: small and diffuent. Liver: soft; no gall-stones; lining of gall-bladder and ducts normal. Kidneys soft; heart normal. Lungs both extremely congested."

Looking at this group of cases and endeavouring to construct from them a clinical picture which may be of use in the future, one is first struck with the fact that in three the patients described similar attacks at long intervals antecedent to that for which they sought surgical help. In Case 2 there were two, one six years and one six months before the last; both after normal childbirth. In Case 3 there was one attack nine months previously described as quite similar to the last. It was

not related to labour. In Case 4 the patient, a male, described three illnesses of the same kind, the first five years before (in bed 11 weeks), the second four years before (in bed for three weeks), and the third three years before (for which he remained in bed one week). In my own last case (No. 1) the attack for which I operated may have been his first. But there is a history of very severe abdominal pain and shivering earlier in 1913, causing him to roll on the floor, and lasting some hours, which may have been an illness of the same nature but not so deadly. If the present attack was the first, that and the fact that it was taken in hand early may account for the more limited area involved around the much swollen and inflamed pancreas. It may be recalled that the sac of the omentum was empty, but that there was extensive fat necrosis in the retroperitoneal tissues but no sloughing at the moment of operation. This took place later.

Another point noted as regards past history was that in two cases, Nos. 1 and 4, there was much gastric trouble for some time previous to the attacks; and in No. 2 the teeth were in very bad condition indeed, which, as we know, is usually associated with stomach derangement. In No. 3 the patient stated that she was subject to "frequent biliousness" before her attack.

In none of the cases was there anything in the history to suggest derangement of the biliary apparatus except doubtful jaundice in a previous attack in No. 2. Nor at the operations was anything wrong found in the gall-bladder. In No. 1 I opened it simply as a possible means of relief for the inflamed pancreas. The bile, which was drained through a tube into a bottle, was perfectly normal for 24 days. It was, however, of very great interest to note that at about the time when the exacerbation of symptoms followed by "fits" (tetany) came on (thirtieth day) the previously clear bile became turbid, and a considerable sediment of pus was found in the bottle for about a week. From the improvement which followed on this both in the general condition and the bile it seems clear that this pus found its way from the inflamed pancreas into its duct, then into the common duct, and thus into the gall-bladder. In Case 6 there was no time for actual destructive change in or around the organ. The patient was killed outright by the shock of the onset in a few hours, and before any surgical relief could be undertaken. Case 5, too, had not advanced to sloughing. Case 7 was precisely like No. 1, but not so severe.

The onset of the attack in all these cases was strikingly *sudden* except in No. 3, in which four weeks of "pain after food" elapsed before the condition was thought grave enough to call for admission into hospital. In the others, except for indefinite gastric disturbance, *sudden* severe pain in the epigastrium was the first and most prominent symptom. This was always described as very intense, and in my own last case (No. 1), where I had the opportunity of seeing the patient very early, the powerful man appeared to be suffering horribly and was pallid and markedly cyanosed. This cyanosis lasted some days after operation. In Mr. Johnson's case (No. 6) the suffering was very severe and accompanied by extreme cyanosis and collapse which rapidly ended in death.

The temperature in all cases was relatively low at the onset. In Case 6 it was 97.1° F., indicating

severe shock from which the patient died before any operation was done. The pulse was rapid and gradually increasing while losing tension. Vomiting was usual at the onset in these cases, not so prominent a feature later. The material brought up was usually bilious with mucus. In no case was a tumour found in the region of the pancreas without an anæsthetic. In one case only was one discovered just before the operation when the anæsthetic had been given.

On opening the abdomen turbid peritoneal fluid was found in three cases at operation (and in one, Case 6, not operated on) in considerable amount. The intestines were injected and dilated in four out of the five cases operated on, but not paralysed. They had not the appearance of being actually inflamed, their lustre being unimpaired, and showing no lymph upon them. But lymph was found in three cases in the neighbourhood of the pancreas.

From the character of the discharges coming from the inflamed gland there was only evidence of hæmorrhage into or around it in three (Cases 1, 2, and 4). Perhaps the grumous fluid which came away with sloughs in No. 3 indicated broken-down clot, but it is hard to say, as the operation was performed four weeks after the onset; the attack was probably less acute than in the other instances. The omental sac was opened in all the six cases operated on, but only in three had the destructive process made its way into it.

Six of the cases here reported are the only instances of acute pancreatitis which have occurred in our hospital in the last five years. With my own private case there are six operated on by members of the staff; and it is encouraging to find that five have been saved. One was too far gone to admit of any operation, and one died the morning after an attempt had been made to arrest the destructive process by surgical interference. As the disease becomes better understood and is dealt with in its earlier stages better things may be expected.

I have dealt with this series as a purely clinical study in spite of the temptation to speculate at any length as to its pathology.

Harley-street, W.

THE annual general meeting of the National Society of Day Nurseries will be held at 35, Chesham-place, S.W., on Thursday, June 11th, at 4 P.M.

LIVINGSTONE COLLEGE: COMMEMORATION DAY.—Saturday, June 13th, Commemoration Day at Livingstone College, marks an important stage in the history of this missionary auxiliary, inasmuch as it is 21 years since the college was founded. From 3 to 3.20 in the afternoon there will be a reception by the Principal and Mrs. C. F. Harford, after which a meeting will be held in the grounds of the college under the presidency of the Bishop of Chelmsford. The speakers will include Dr. Ernest N. Cook, one of the staff of the Mengo Mission Hospital in Uganda. Dr. Loftus E. Wigram, the vice-principal, who is to succeed Dr. Harford as principal, will also take part in the proceedings. After the meeting an opportunity will be given for visitors to view the college. It will be remembered that an appeal was made last year for a sum of £10,000 to form a Livingstone Centenary Fund in order that the college might be placed on a sound financial footing. Only £640 have been received in answer to this appeal. It is hoped that before Dr. and Mrs. Harford leave the college in July next the mortgage on the property, amounting to £3500, may be cleared off. Applications for tickets should be made to the Principal, Livingstone College, Leyton, E., who will gladly receive any contributions towards liquidating the mortgage or towards the special endowment fund.