the hepatic duct and the upper end of the common bile-duct might easily have been mistaken for an enlarged gall bladder containing numerous gall stones.

5. *The Liver*.—The appearances presented by this organ are represented in the accompanying photographs.

Chemical Analysis of the Intrahepatic Calculi.

For this we are indebted to our colleague, Dr. Schölberg, who carried out a careful analysis on 20 grams of the calculi after the method described by Salkowski, with the following result:

Cholesterin	...	...	18.0 per cent.
Calcium bilirubin	...	...	38.9 "
Iron	...	...	0.01 "
Phosphates	...	...	A faint trace.
Water	...	...	43.09 per cent.

From the small abscesses on the surface of the liver Dr. Schölberg isolated the *Bacillus coli*. The typhoid bacillus was not discovered.

Fig. 4.—Calculi removed from the intrahepatic ducts.

Literature on Intrahepatic Calculi.

1. Mayo Robson, *Diseases of Gall Bladder and Bile Ducts*, p. 216: "Gall stones formed almost entirely of bile pigment may be seen. On two occasions there were found soft concretions of this nature in large numbers in the hepatic ducts within the liver."

2. Rolleston, *Diseases of the Liver, Gall Bladder, and Bile Ducts*, p. 721: "Large intrahepatic calculi occupying the hepatic ducts and distending them are very rare. I met with one such case in a man who died with diabetes from secondary chronic pancreatitis (vide Fig. 95). Murchison (*Diseases of Liver*, second edition, p. 545) refers to a plate of Cruveilhier showing large intrahepatic calculi like coral. There is a good specimen of multiple intrahepatic calculi in the Westminster Hospital Museum (No. 581). These calculi almost necessarily set up jaundice and a good deal of pericholangitis, which may be suppurative. The calculi are chiefly composed of bilirubin-calcium. Small black bilirubin-calcium calculi are not uncommon. Occasionally masses of calculous material are found embedded in the substance of the liver in dilated ampulla-like terminations of bile ducts which have lost their continuity with the bile ducts."

3. Naunyn, *Cholelithiasis* (Trans. A. E. Garrod, 1896, N. S. S.), p. 29: "Bilirubin-calcium gravel in the bile ducts within the liver are in size from finest gravel to nodular and stalked masses in thick, greasy, brownish-black bile." "They consist of calcium compounds of the bile pigments." "Contain large amounts of the higher oxidation products of bile pigments and bilirubin." "One finds in such concretions biliverdin, bilicyanin, and bilifuscin—the transition substances between bilirubin and humin." "... found ... in the large bile ducts ... calculi of cholesterin tightly enclosed by the mucous membrane of the duct." "... exceptional circumstances, calculi can even originate in the large bile ducts ... increase in size ... due to bilirubin-calcium and cholesterin."

4. Frerichs, *Clinical Treatise on Diseases of the Liver*, vol. ii, p. 512—Gall stones in the interior of the liver, in the branches of the hepatic duct: "Gall stones are rare in this locality. Morgagni, however, long since collected a series of observations from the works of Plater, Fallopius, Dodonoeus, Columbus, Paysch, and others,

showing that concretions had been found in the interior of the liver. Biliary concretions in this locality most frequently present the form of brown or black grains. Sometimes, also, we meet with larger round stones, or, more rarely, with branched, coral-like concretions which form casts of the ducts and are sometimes solid, but at other times hollow. The branches of the hepatic duct are dilated by these deposits, either uniformly or in a saccular manner. The concretions may give rise to inflammation and ulceration of the bile ducts, and also to hepatic abscesses and pylephlebitis."

OBSERVATIONS ON PANCREATIC NECROSIS, WITH A REPORT ON TWO CASES.*

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TOGETHER WITH A NOTE

By C. LOVATT EVANS

ON CAMMIDGE'S PANCREATIC REACTION IN THE URINE.

THE two following cases of pancreatic necrosis form together an interesting study. In a paper in the **BRITISH MEDICAL JOURNAL** of December 22nd, 1900, I drew attention to infection spreading up the common bile duct and setting up various inflammations of the pancreas. Probably this is the channel of infection in the majority of such, and in the second case here described it is very probable that this was the channel of infection. This is in marked contrast to the conditions found in the first case; here there was no clinical evidence of infection, and the removed fluids proved to be quite sterile. It seems probable, therefore, that the death of pancreatic tissue was due rather to deprivation of blood supply, possibly through thrombosis of some of the arterial supply to the organ. In acute infective lesions of the pancreas haemorrhages into and around the organ are common, but haemorrhages may be found altogether apart from infection, and for these there is often no adequate explanation.

In a short paper on pancreatic cysts¹ I drew attention to the presence of large quantities of blood in some of these cysts. Injury may no doubt account for some of the bleedings, but in one of my cases there was no evidence of any traumatism, and the explanation of the bleeding remains obscure. Is there any relation between these and the first case here described?

The notes by Mr. C. Lovatt Evans on Cammidge's test are of considerable interest, as Mr. Lovatt Evans is a skilled chemist, constantly engaged in physiological chemistry. The utility of this test is considerably restricted by the fact that it requires an expert to carry out the examination, and that even then the result may be vitiated if suitable precautions are not taken.

CASE I.—*Pancreatic Necrosis, with Cyst Formation, but apparently without Infection. Operation.*

A woman, aged 43, was admitted to the General Hospital under the care of Mr. Barling on July 1st, 1904, with a history that she was taken ill on June 12th with severe abdominal pain and vomiting, followed by diffuse tenderness over the whole abdomen; the patient was so ill that perforation of a gastric ulcer was suspected. For a week she improved, but on June 20th she had another acute attack of pain in the epigastrium, going through to the back, with incessant vomiting, which continued until the day of admission, and soon after this second attack of pain a fullness was noticed in the upper part of the abdomen. Morphine had been required on several days to subdue the pain.

State on Admission.

The patient was a well-developed woman, with a swelling in the epigastric and upper part of the umbilical region, which was firm, but rather elastic, and lay with its long axis transversely; stomach resonance appeared to be entirely above it. At this time the chief pain complained of was in the dorsal region about the level of the last dorsal vertebra, and the eleventh dorsal vertebra was very hyperaesthetic on pressure. The pulse was 124, temperature 99.6°, respirations 24; the urine was free from albumen and sugar.

* Read at a meeting of the Pathological Section of the Birmingham Branch of the British Medical Association.

First Operation.

On July 2nd the abdomen was opened in the middle line above the umbilicus, and some ounces of free serous fluid of a greenish-brown colour escaped. It was found that the stomach was pushed forward and upwards, and the great omentum was rolled up on itself, and was very dense. An exploring needle was passed between the stomach and transverse colon, and withdrew a dark fluid like altered blood; the needle puncture was enlarged, and some ounces of fluid escaped. The cavity was packed with gauze, and drained. The exact locality of this cavity was doubtful, but it was thought to be over the pancreas. This operation gave relief to pain, and the pulse dropped, the temperature became normal, and vomiting ceased for a few days.

On July 10th the note is that the wound was practically healed, but the pain had returned and there was occasional vomiting.

July 18th. The pain and the sickness had practically disappeared.

July 21st. Both these symptoms returned with intensity, and the patient had to be fed with nutrient enemata.

July 22nd. There was a distinct rounded tumour again present in the epigastric region, dull on percussion, tender, and doubtfully fluctuating. The patient now complained of severe pain in the back.

July 24th. The pain in the back was still severe and the tumour was extending.

Second Operation.

On August 2nd a 4-in. incision was made 2 in. to the right of the middle line, above the umbilicus. A large cyst was found pressing the duodenum to the right, and bulging forward between stomach above, the colon below, and the duodenum to the right. Through an aspirating needle $1\frac{1}{2}$ pints of thin brownish fluid, without odour, was withdrawn. The opening in the cyst was enlarged, the finger introduced, and two necrotic pieces of pancreas were removed, the one $1\frac{1}{2}$ in. by $\frac{3}{4}$ in., the other $\frac{3}{4}$ in. by $\frac{1}{4}$ in. The edges of the opening in the cyst were stitched to the peritoneal and fascial edges and the cavity lightly packed with gauze. Following this second operation there was complete relief to all the symptoms, the only inconvenience being from the excoriated skin around the opening, and a good recovery followed.

Note by the Pathologist, Dr. Wynn.

The fluid is quite sterile; there is no growth in broth or agar after twenty-four hours. The specimen examined is a light brownish-yellow turbid fluid with a slight reddish tinge from admixture of blood. On standing the fluid becomes clear and colourless, and deposits a reddish-brown flocculent material.

Specific gravity, 1007. *Reaction*, decidedly alkaline. There is a dense cloud of albumen on boiling and a flocculent precipitate on adding alcohol. It also reduces slightly Fehling's solution and gives tests for albumoses and peptones.

Digestive Actions.—It rapidly converts starch into sugar. It digests egg albumen and converts it into peptone. It emulsifies fat but only slightly converts it into fatty acids. All three pancreatic ferments (amylase, trypsin, steapsin) are therefore present, the first being the most active, the last the least active. There is a trace of urea, 0.05.

Microscopical Examination of Deposit.—This shows red and white blood cells, large swollen granular and fatty cells, flat squamous epithelial cells often in small sheets, and crystals of fatty acids and amorphous, darkly-pigmented masses.

The fluid is undoubtedly that from a pancreatic cyst, but rather more dilute than usual.

At the present time (November, 1905) the patient is in good health and has since the operation grown fat.

REPORTS BY MR. LOVATT EVANS.

July 11th, 1904.

I have examined the urine of Mrs. A., Ward XI, and the results are as follows:

Gravity.—1009.

Microscopic.—Micrococci. No crystals.

Reaction.—Acid.

Bile
Albumen } Absent.
Sugar

Cambridge's Reaction A.:

1. No crystals.
2. No crystals.

Reaction B.:

1. Few crystals } These cannot be crystals of glycerosazone,
2. Few crystals } as their rate of solution is too slow.

In this case, therefore, there is no indication (from the urine, at least) of any pancreatic disorder.

July 23rd, 1904.

The urine is a very different sample this time as compared with the first of Mrs. A.'s examined. I was informed at the hospital that the diet at the time of collection was mainly peptonized milk with its full contents of fat, whereas before it had not contained the fat in such amount or in such a readily assimilable form.

Possibly the question of diet may have great influence upon the presence of glycerine in the urine, upon which the reaction entirely depends.

Analysis of Urine.

Reaction.—Acid speedily becoming alkaline.

Deposit (in moderate amount).—Phosphates and few urates, etc.

Sugar appears to be present in traces, though the presence of urates and a high amount of creatinin may account for the reducing power of the urine. Moreover, no crystallizable osazone was obtainable in the phenyl-hydrazin test for sugar.

As the presence of sugar was doubtful, the urine was fermented with yeast for twelve hours to remove possible traces of glucose, and the reaction performed.

Reaction A yields a very few crystals, which adhere to the bottom of the tube. They are quite typical, and very different in appearance from crystals of the "lead compound." Soluble in $\frac{1}{2}$ H₂SO₄ in 1 min. 10 sec.

Reaction B yields a much more abundant crop of crystals.

The diagnosis is therefore, according to Camidge, either (b) "a damaged pancreas due to past pancreatitis," or (c) "some disease not connected with the pancreas."

October 23rd, 1905.

Specific Gravity.—1025.

Reaction.—Very faintly acid.

Deposit.—Very copious. Calcium oxalates, envelope variety. Amorphous urates abundant (murexide test with great ease). Vaginal and bladder squames, etc.

Albumen and Bile absent.

Sugar.—A minute trace present, removed by yeast before proceeding to test.

A Reaction.—Abundant crystals, soluble in one to three minutes.

B Reaction.—Abundant crystals, together with a heavy black deposit.

Diagnosis.—Doubtful, but probably slight persistent inflammation (or former pancreatitis).

General Note on Camidge's Reaction.

From various experiments made in July of last year and from the consideration of this case, I am able to arrive at the following brief summary of the rationale and value of Camidge's reaction:

1. Provided that the patient has a certain amount of neutral fat in the diet, glycerine is undoubtedly present in the urine.

2. Camidge's reaction performed on a 2 per cent. aqueous solution of glycerine fails, or at most yields only a few crystals.

3. The same test with a 2 per cent. solution of glycerine in normal urine yields an abundant crop of crystals of phenyl-glycerosazone hydrochloride, soluble in 33 per cent. H₂O₂ in a half to one minute.

4. The crystals of the compound of phenyl-hydrazine with lead salts are entirely different in appearance to the glycerosazone crystals, and are altered, but not dissolved, by sulphuric acid.

5. Lead is usually present in traces in the filtrate after neutralization with PbCO₃, but in the final stage there is more lead in the mother liquor than in the crystals deposited on cooling.

6. Glycerol, when in the urine, can be oxidized by boiling with hydrochloric acid thus:

but if the boiling be too prolonged, or if from any cause the oxidation be too vigorous, then:

Since neither of these bodies contain a $\text{C} \begin{array}{l} \diagup \text{H} \\ \diagdown \text{O} \end{array}$ group they will not form an osazone.