A Lecture ON THE SURGICAL TREATMENT OF CERTAIN CASES OF GLYCOSURIA.

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Nothing was more impressed on my mind by my surgical teachers in the early Seventies than the desirability, nay, the absolute necessity, of avoiding surgical treatment in any patient whose urine was found to contain sugar; and I have a distinct recollection in those pre-antiseptic days of seeing several simple operations to go wrong in which glycosuria received the blame that, as one can now recognize it, should have been awarded to sepsis.

Although glycosuria patients are not subjects that a surgeon would choose to operate on if relief could be afforded in any other way, yet I have found by considerable experience that patients whose urine contains even a considerable amount of sugar may usually be safely treated surgically if due precaution be taken to secure as far as possible an aseptic condition of the wound, and if care be taken in the preparation of the patient by securing free evacuation of the bowels before operation, and obtaining by means of carbonate of soda or some other alkali a neutral, or almost neutral, reaction of the urine so as to avoid acetonæmia.

My purpose now is, however, not simply to prove what is already known, but to show that certain cases of diabetes may be materially benefited, and some even may be cured if the glycosuria be of pancreatic origin; and furthermore, to show that a fairly accurate prognosis of the onset of diabetes may be made by a careful examination of the urine and faeces in certain cases at a period long antecedent to the appearance of glycosuria, and that in this early stage the onset of diabetes may be averted by timely surgical treatment.

While the literature of diabetes so far as its pathology is concerned is almost overwhelming in its amount, the student soon discovers that the subject of treatment is confined within very narrow bounds, and in fact until quite recently it has been limited to certain modifications of diet and other general palliative measures.

Although it had been noticed by several observers that diabetes was occasionally associated with disease of the pancreas, no definite causal relationship between the two was surmised until 1889, when Mering and Minkowski, as the result of experimental extirpation of the gland, demonstrated the direct relationship of the pancreas to carbohydrate metabolism and its independence both of the nervous connexions and of the secretory functions of the organ. As the result of numerous observations since 1889, it seems to be fairly established that the pancreatic origin holds in from 70 to 80 per cent. of cases of diabetes.

My colleague, Dr. Cammidge, and I have observed together 31 cases of pancreatic diabetes in which the question of surgical treatment has arisen or has actually been carried out, and each of us has carried out observations on other cases independently. In all these 31 cases the pancreatic reaction in the urine has been found associated with glycosuria, and in the greater number of them the accuracy of the diagnosis has been verified by operation or at autopsy. Dr. Cammidge tells me that he has examined 83 specimens of urine from 48 cases of diabetes by his special method, and obtained the pancreatic reaction in 36, or 75 per cent. of the patients. Of the 12 that gave no reaction, the opportunity for *post-mortem* examination occurred in 3, in all of which there was no evidence of disease of the pancreas.

The suggestion that the islands of Langerhans are concerned in the production of the "internal secretion" of the pancreas was first made by Laguesse. This view was subsequently adopted by Schäfer, Diamere, and others. The theory that such a relationship exists is

based partly on histological grounds and partly upon the results of experimental work; but the most important evidence in support of it has been furnished by pathological observations, which suggest that pancreatic diabetes is due to a disturbance of the functions of the cell islets. The peculiarity of their structure, their independence of the duct system of the gland, and their comparative resistance to certain morbid changes by which the secreting acini are destroyed, point to their being independent organs with an independent function, while their rich blood supply may be taken to indicate that they are possibly vascular glands engaged in the elaboration of some internal secretion which is poured into the blood stream.

Opie considers that diabetes is peculiarly related to interacinar pancreatitis, and that in the interlobular form it is rare. The difference, he considers, depends upon the relations of the fibrous tissue overgrowth to the islands of Langerhans, which he in common with many observers believes are responsible for the elaboration of the internal secretion by means of which the pancreas exerts its influence upon carbohydrate metabolism. The newly-formed fibrous tissue in interacinar pancreatitis is diffusely distributed within the lobules and between the individual acini, so that the islands are affected at the same time as the other elements of the gland; but in the interlobular form, which is the type following duct irritation or obstruction, the proliferation of fibrous tissue takes place between the lobules and invades them from the periphery, so that the cell islets suffer only when the process is far advanced, and the secreting parenchyma has been replaced by masses of scar-like tissue.

Opie found that diabetes was present in 7 out of 9 cases of interacinar pancreatitis, but only in 1 out of 21 cases of chronic interlobular inflammation, in which case the induration of the gland was far advanced and the islands of Langerhans were fibroid. The two cases of interacinar pancreatitis in which diabetes was absent, were both, he found, in an early stage of the disease.

The pathological changes that have been met with in the interacinar islets in cases of diabetes are hyaline degeneration, necrosis, atrophy with vacuolization and liquefaction of the cell protoplasm, acute and chronic inflammation with hæmorrhage, sclerosis, or calcification, and diminution of the number of islets. In 19 cases of diabetes in which Opie investigated the condition of the islands of Langerhans he found hyaline degeneration in 7 (35 per cent.). When I was in Baltimore as a guest of the American Surgical Association I had the opportunity, through Dr. Opie's kindness, of seeing his specimens, which were most convincing.

Fortunately interacinar pancreatitis, which gives rise to diabetes at an early stage, is a much less common disease than the interlobular form.

Opie's description of hyaline degeneration in the islands of Langerhans alone, in diabetes, furnished the most convincing evidence of the association of these structures with carbohydrate metabolism up to that time available, and from the publication of his paper in 1901 may be dated a revival of interest in the pancreatic theory of diabetes, and a more searching and minute inquiry on the part of other observers into the condition of the cell islets in fatal cases coming to *post-mortem* examination.

Chronic interstitial pancreatitis has been frequently observed in association with diabetes, but in the majority of cases where interstitial changes in the pancreas arise from irritation of the ducts by gall stones, or from other causes, glycosuria is not met with as a symptom. In a communication made some time ago I referred to 65 consecutive cases of my own where biliary calculi were found in the common duct at operation, and in which the pancreas was enlarged and hard from the effect of interstitial pancreatitis; sugar was detected in the urine only of 4 (6.1 per cent.). In 3 of these the amount was under 0.2 per cent., and in the fourth 0.4 per cent. After operation the sugar disappeared from the urine in all but the last, in which it slowly increased in amount, the patient dying from diabetic coma ten months subsequently.

Since the interstitial changes arising from the presence of gall stones in the common duct principally affect the head of the gland in the first instance, and the results of experiments upon animals have shown that even a small

portion of healthy pancreatic tissue will prevent the onset of diabetes, or at least delay the appearance of the symptoms, so long as it remains undestroyed by fibrotic changes, the comparative rarity of glycosuria in these cases is not difficult to explain. When, however, a great part of the parenchyma has been destroyed, or is functionally impaired by the progressive changes consequent on repeated or long-continued irritation, either from gall stones or an unrelieved duodenal catarrh, or from the presence of pancreatic concretions, sugar will make its appearance in the urine, first as an alimentary glycosuria, and later as a permanent diabetes.

Although the cause of glycosuria in a considerable percentage of cases is due to pancreatic disease, yet very obvious disease of the pancreas may be unaccompanied by glycosuria; this is particularly noticeable in cancer, which is seldom associated with diabetes. A very striking example of this has recently been investigated and described by Dr. S. G. Scott:

The patient was a man suffering from cancer of the head of the pancreas with occlusion of the duct and secondary atrophy and fibrosis of the body of the gland. There was no trace of sugar in the urine during life, and although *post-mortem* microscopic examination showed that the whole of the secreting parenchyma had been replaced by fibrous tissue, the islands of Langerhans were unaffected.

On the other hand, in the case recorded by Dr. Churton, in which there was complete fibrosis of the gland due to pancreatic lithiasis, the islets of Langerhans were destroyed and a large amount of sugar was present in the urine.

Dr. Cammidge only met with sugar in the urine in 2 out of 48 cases of primary malignant disease, and both of these gave a modified pancreatic reaction, due to the presence of associated chronic pancreatitis. In 2 cases in which the disease invaded the pancreas secondarily sugar was present, and at autopsy there was found to be interacinar pancreatitis in one and advanced fibrosis and calculi in the ducts of the other.

My own experience in over a hundred cases of cancer of the pancreas has been that glycosuria is a very rare complication, not more frequent than 3 per cent., and, needless to say, no treatment can be of any permanent avail, though in the following case of cancer of the common duct the operation of cholecystenterostomy led to a disappearance of the glycosuria.

Cancer of Common Bile Duct Involving Head of Pancreas: Deep Jaundice: Glycosuria: Boils: Cholecystenterostomy.

On April 30th, 1909, I was asked by Dr. C. to see Mrs. M., aged 60, who had been subject to attacks of indigestion since influenza a year ago. For nine weeks before my seeing her she had had a severe pain at the epigastrium. Jaundice had been present for five weeks. On examination I found an enlargement of the right lobe of the liver, with some enlargement of the gall bladder. Three weeks before my seeing her Dr. C. had found 3 per cent. of sugar in her urine, and during this time she had been suffering from numerous boils on various parts of the body.

On May 17th, 1909, I operated and found a hard tumour of the head of the pancreas, which felt like a gall stone. The common duct was opened and the finger passed down the duct, but no gall stone could be felt. The common duct was therefore closed, and the gall bladder was connected to the duodenum, so as to short-circuit the obstruction. The abdomen was closed, and then the boils on various parts of the body were laid open and scraped. The patient made a good recovery from her operation, and Dr. C. reported in July that the sugar had entirely disappeared from the urine. From a report that I received later in the year the growth had evidently extended to the hepatic duct, but at that time there was no recurrence of the glycosuria.

The evidence I have thus far brought forward shows that we cannot hope to cure or even materially relieve, except temporarily, pancreatic diabetes depending on extensive destruction of the gland by organic disease, whether that be dependent on growth or on chronic inflammation, if either class of disease has extensively involved the islands of Langerhans; nor can we hope to benefit that form of inflammation that singles out the cell islets, which is known as interacinar pancreatitis. But, fortunately, by far the greater number of cases of glycosuria due to pancreatitis are dependent on inflammation of the catarrhal type in which the gland is invaded by way of the ducts from varying causes, such as gall stones, pancreatic calculi, duodenal catarrh (often associated with duodenal ulcer), typhoid fever, etc.

In this class of cases the disease probably exists for a period as a catarrhal inflammation simply, and if caught in this stage it is, I believe, curable by surgical measures in a considerable proportion of cases. At a later stage the inflammation produces an interstitial inflammation, which may be arrested and decided relief given by surgical means, though the damage already done to the pancreas cannot be repaired. At a still later stage cirrhosis of the gland takes place, and if the cell islets become involved extensively, surgical means are quite useless.

The difficulties that have until recently attended the rational treatment of diabetes have not only been due to the obscurity of the etiology of the disease, but even since diseases of the pancreas have been recognized as the cause of glycosuria in a large percentage of cases, the difficulties attending their diagnosis in an early stage, that is at a stage when curative treatment can be adopted, have been insuperable.

These difficulties have fortunately been overcome, for it is now possible to diagnose the catarrhal and inflammatory diseases of the pancreas even in their earliest stages by a careful examination of the urine and faeces, and by a consideration of the clinical history and symptoms.

As the diagnosis of pancreatic disease giving rise to diabetes can be made in an early stage before the onset of glycosuria, it seems to me that there can be no reasonable excuse for allowing the condition to progress untreated until the more serious disease supervenes. The results that may follow the neglect of a warning given by the pancreatic reaction are well shown in a case that I saw in May, 1904, in which I removed the appendix for recurring appendicitis.

The urine of the patient was examined on June 2nd, and found by Dr. Cammidge to give a well-marked pancreatic reaction, which was confirmed by another examination five days later.

As an operation had recently been performed, the patient was averse to further surgical treatment, and it was hoped that general treatment at home might bring about relief to the affected pancreas.

Two years later (August 30th, 1906) a specimen of urine was again sent to Dr. Cammidge, when he found not only the pancreatic reaction but also 1.25 per cent. of sugar. On April 11th, 1908, the sugar had increased to 4.5 per cent., and within a short time the diabetes terminated fatally.

I firmly believe that a cholecystenterostomy performed as soon as the pancreatic affection was recognized would have prevented the onset of glycosuria and that an operation undertaken in 1906 would have arrested the progress of diabetes.

The following case, out of several that I could relate, affords an example demonstrating that pancreatitis if taken early, when only catarrhal, can be completely cured.

A lady, aged 50, had for several years suffered from attacks of distinct biliary colic, which during the past two months had been followed by jaundice, fever, and collapse. There had recently been loss of flesh. On examining the urine the pancreatic reaction was discovered, and at the operation on April 30th, 1903, 40 gall stones were removed from the gall bladder and cystic duct. None were found in the common duct, though the pancreas, especially the head of the gland, was distinctly swollen and harder than normal. The gall bladder was drained. The patient made a good recovery and is now well. Normal weight has been regained, and there is no longer any evidence of disturbed metabolism.

This is a good example out of many that I could relate of the so-called catarrhal jaundice and catarrhal pancreatitis so frequently associated with gall stones, though the concretions are in the gall bladder or cystic duct, and can therefore not be directly responsible for obstructing the flow of bile into the intestines. The pancreatic ducts become affected at the same time as the bile ducts by the catarrhal process extending from the duodenum, and this leads to swelling of the head of the pancreas and so to pressure on the common bile duct, and to slight or in some cases to serious obstructive jaundice. Had this condition been allowed to persist the prognosis of advancing interstitial pancreatitis ending in glycosuria would have been probably correct. It may be asked, Why are not all cases of gall stones in the gall bladder associated with jaundice and pancreatitis?

The explanation, so far as the pancreatic condition is concerned, is probably dependent on the power of resistance of the individual to *coli* infection, but, with regard to the jaundice, it is an anatomical one; in 62 per cent. of all cases the pancreas embraces the third portion of the common bile duct—hence, although pancreatic catarrh or interstitial pancreatitis may be a frequent concomitant of cholelithiasis, in only about two-thirds of such cases will it be likely to lead to jaundice by pressure on the main bile channel, for if the choledochus passes behind and not through the head of the pancreas, the duct may escape pressure when the pancreas is congested or otherwise swollen, whereas, if it pass through the gland, any compression or swelling of the pancreas will, by pressing on the common duct, bring on jaundice with its various sequelae. Thus are also explained many of the cases of so-called catarrhal jaundice.

An early stage of catarrhal pancreatitis, if not associated with organic disease, may possibly be cured by general treatment, but it is most important that medical treatment, if not giving relief, should not be continued too long, or it may be too late to secure a favourable issue.

It is most desirable that the pancreatic disease should not be too far advanced before surgical treatment is adopted, otherwise relief and not complete cure is all that can be hoped for, and for this reason I strongly recommend that all cases of cholelithiasis and of duodenal ulcer, if associated with pancreatitis, as shown by the pancreatic reaction in the urine, should be operated on with as little delay as possible, though even in the more advanced conditions operation may be undertaken with the hope of arresting the disease of the pancreas, and so preventing the otherwise inevitable progress of the diabetes.

In some cases in which operation has been delayed or drainage of the bile ducts not performed, or not long enough continued, the original interstitial pancreatitis may persist, as in the following two cases, and lead to an alimentary glycosuria or to a slight permanent diabetes.

Mrs. C., aged 51, who was suffering from persistent jaundice with periodical pains and ague-like seizures that had extended over a long period, was operated on by the author in July, 1895, when several gall stones were removed and others crushed and removed from the common duct. A tumour of the pancreas was felt, which it was feared at the time might be malignant. The gall bladder was therefore drained into the duodenum by a cholecystenterostomy. The patient recovered and has remained apparently well since the operation fifteen years ago, but an examination of the urine by Dr. Cammidge showed a well-marked pancreatic reaction and the presence of dextrose on November 11th, 1902. This shows that although the patient had been relieved by the operation and had apparently enjoyed good health, yet that she is living with a damaged pancreas, the operation having apparently arrested the progress of the pancreatic disease and averted a fatal diabetes.

Mr. D., aged 42, had an attack of pain in the right hypochondrium in August, 1888, but no jaundice. He had been free from pain up to six weeks before seeing me, when he had a severe attack in the right hypochondrium, radiating to the right shoulder, accompanied by rigors and vomiting, followed by jaundice, which had persisted. No swelling could be felt. An exploratory operation was performed on October 27th, 1898, when a mass, thought to be growth in the head of the pancreas, was discovered. The tumour was freely handled, to try to ascertain if any gall stone was impacted in the common duct, but no concretion could be felt and the abdomen was closed. The enlargement of the head of the pancreas was clearly not cancer, and must have been chronic pancreatitis, as the patient made a good recovery, and gradually lost his jaundice. The manipulation may have dislodged an obstruction, leading to the relief of the jaundice.

A specimen of his urine was obtained in 1904, and although he was reported to be quite well, this was found to give the pancreatic reaction and to contain sugar in fair quantity.

These cases, along with others that I know of, lead me to think it is advisable to drain the bile ducts thoroughly in all such conditions, and I consider that drainage ought to be continued until the bile becomes free from organisms and its normal route free from obstruction. Either long-continued drainage through a cholecystotomy opening or permanent drainage by a cholecystenterostomy will meet these conditions.

In certain cases, even of well-marked glycosuria, recovery doubtless occurs under general treatment without operation, and I have notes of one case where a gentleman of advanced age had deep jaundice associated with glycosuria and with a well-marked pancreatic reaction in

the urine, pointing to the case being one of pancreatic diabetes. Under general treatment, combined with massage, he not only lost his jaundice and glycosuria, but regained his health, and is now said to be quite well. In this case it is quite possible that the massage dislodged a concretion which was blocking both the common bile duct and the pancreatic duct, or a concretion may have been discharged into the duodenum by ulceration, but as no search was made in the faeces this cannot be proved. As the patient lives abroad, I have not been able to get a specimen of the urine to see if the glycosuria is still absent.

That glycosuria dependent on an early stage of catarrhal pancreatitis, if not associated with organic disease, may be relieved or possibly cured by general means, the following case proves, but it is important that, unless decided relief is being given, radical treatment should not be deferred until the disease is too far advanced for surgical treatment to be adopted.

Catarrhal Pancreatitis: Glycosuria.

Mr. K., aged 62, was sent to me on May 16th, 1908, from South Africa, there being a question as to whether he was not suffering from some internal malignant disease, as for some time he had been steadily losing flesh, having lost a stone in weight within a few months, during which time he had suffered from indigestion and severe headaches. I could find no evidence of malignant disease, but an examination of his excreta showed that he was passing 0.24 per cent. of sugar in the urine, which also gave a distinct pancreatic reaction, and contained indican and a well-marked reaction for urobilin. The faeces contained a slight excess of fat, but no occult blood. The condition therefore pointed to pancreatic catarrh, which was probably an extension from a duodenal catarrh. I advised that unless he was shortly relieved by general treatment he should undergo an operation.

On September 17th I received a letter from him, written off Tenerife, to say that he was much better as the result of general treatment, and that an examination of his urine showed it to be quite free from sugar, though there was still a slight pancreatic reaction.

As bearing on the importance of the early diagnosis and treatment of interstitial pancreatitis—which is the form that complicates cholelithiasis and duodenal ulcer—it is clearly established that for some considerable time, varying doubtless in different cases, the disease of the pancreas is local, and confined to the neighbourhood of the head of the gland or of the large ducts, and, as shown by microscopic examination, it does not invade the whole of the organ or the islands of Langerhans until a later stage of the disease.

In these cases which are associated with glycosuria the cause of the glycosuria is not merely the local chronic pancreatitis, but a general catarrhal inflammation of the pancreas involving the whole of the gland, and inhibiting the function of the cell islets. Thus, I believe, can be explained the appearance of glycosuria in some cases of acute pancreatitis and its disappearance after operation, as in Mr. Giffard Nash's case reported in the *Lancet*, November 1st, 1902.

The following is a brief abstract of the case:

History.

Man aged 60. Influenza 1891 and 1892. For seven years "bilious attacks," pain and discomfort at pit of stomach; very severe attacks of pain one and two years previously; no sign of jaundice; urine increased, average 5 pints.

November 5th, 1891. Sugar 8.75 grains per oz.

October 27th, 1901. Acute abdominal symptoms suggesting acute intestinal obstruction, or perforation of stomach or duodenum.

Operation November 17th, 1901. Much fat in omentum, and mesentery unusually hard. Pancreas enlarged. Numerous spots fat necrosis in fat in neighbourhood of pancreas. Large calculus in gall bladder. Gall bladder drained.

December 28th, 1901. Urine 4½ grains sugar per oz.

March 1st, 1902. Urine 4½ grains sugar per oz.

May 17th, 1902. Urine free from sugar.

Urine examined later by Dr. Cammidge showing permanent damage to the pancreas, but progress of disease arrested.

November 11th, 1902. Sugar 0.9 per cent.; pancreatic reaction positive.

February 16th, 1904. Sugar 1.0 per cent.; pancreatic reaction positive.

January 8th, 1906. Sugar 1.0 per cent.; pancreatic reaction positive.

The cases I am about to relate in which pancreatic glycosuria was associated with gall stones or with duodenal

catarrh, and in which the sugar disappeared from the urine after operation at the same time as the pancreatic reaction, can, I think, be explained in the same way by inhibition of the islands of Langerhans, owing to irritation or to a general catarrhal pancreatitis.

Common Duct Cholelithiasis: Pancreatic Catarrh: Glycosuria.

I was asked to see Mr. P. by Dr. K. on March 19th, 1904. He gave me the history that he had suffered from gall stone attacks for ten years, and had passed many concretions the size of millet seeds. He had been slightly jaundiced for some months, and had had repeated pains and rigors. He had lost flesh very considerably. An examination of the urine showed a well-marked pancreatic reaction and 0.2 per cent. of sugar. At operation on March 20th the gall bladder was found to be surrounded by adhesions, after the separation of which seventy-three small gall stones were removed from it. The common duct was exposed and opened, and a large gall stone the size of a pigeon's egg was removed, together with two others from the pancreatic portion of the duct. The common duct was closed and the gall bladder drained. The head of the pancreas was enlarged and hard. The patient made a good recovery, and though I have not seen him since, I have had a report to say that he is quite well, and that the glycosuria has completely disappeared.

Duodenal Ulcer: Cholangitis: Interstitial Pancreatitis: Glycosuria.

On April 15th, 1905, I was consulted by a military officer, aged 35, who gave me a history that he had had typhoid fever in 1890 and had since suffered from malarial fever. For the past three years he had had regular attacks of pain resembling biliary colic together with symptoms of indigestion, pointing to duodenal ulcer, and slight jaundice with occasional attacks of fever. He had lost nearly a stone in weight. There was a tender spot an inch above and to the right of the umbilicus, where an enlargement of the right lobe of the liver could be felt. During the attacks of pain his medical man told me that the projection became more distinct, as if the gall bladder was enlarged. An examination of the urine by Dr. Cammidge showed a well-marked pancreatic reaction with the presence of sugar. There were also traces of urobilin and indican. An operation was performed on April 20th, when the gall bladder was found to be distended, but no gall stones could be discovered either in it or in the ducts. There were numerous adhesions between the gall bladder and duodenum and colon. On separating these the duodenum was seen to be scarred opposite to the site of the ulcer. The head of the pancreas was enlarged and hard. Cholecystenterostomy together with gastro-enterostomy was performed. The patient made a good recovery. On March 26th, 1907, there was 0.5 per cent. of sugar in the urine with a well-marked pancreatic reaction, though his general condition had considerably improved and he was no longer having the attacks of pain. On March 2nd, 1909, there was a mere trace of sugar hardly discoverable, though there was still a slight pancreatic reaction. A later report showed a disappearance of the sugar, and the patient was said to be well.

Gall Stones: Pancreatic Catarrh: Glycosuria.

On March 27th, 1906, Mr. B., aged 62, was sent to me by Dr. K., with a history that he had been suffering from biliary colic for three years, though he had had slight jaundice four years before the colic began. He had lost flesh considerably, and showed a number of small calculi the size of No. 6 to No. 2 shot that he had passed, though one the size of a swan shot had also passed. There was no special tenderness nor any enlargement of the gall bladder. The urine was examined by Dr. Cammidge, and found to contain 0.4 per cent. of sugar, together with a quantity of bile and urobilin. The pancreatic reaction was well marked. At the operation, on April 2nd, 1906, a number of calculi were removed from the gall bladder and cystic duct. The head of the pancreas was felt to be enlarged and hard, but no stones could be felt in the common duct. The gall bladder was drained. He made a good recovery from the operation, and an examination of the urine by Dr. Cammidge on May 5th, 1906, showed that the sugar had entirely disappeared, together with the pancreatic reaction. When heard of two years later, the patient was in good health and had had no recurrence of his trouble.

Mr. P. was sent to me in November, 1907, with a history that he had been subject to flatulency and dyspepsia for years. He also had had pain off and on for five years below and to the right of the umbilicus, not affected by food or relieved by taking a meal. He had a good appetite, and did not suffer from excess of flatulency. He had lost considerably in flesh, and was very depressed. An examination showed rigidity of the upper part of the right rectus, with tenderness above and to the right of the umbilicus, but no tumour could be felt, and there was no dilatation of the stomach. A report received from his medical man stated that sugar had been present in his urine in January, and that it had increased in March. An examination by Dr. Cammidge showed a well-marked pancreatic reaction. At operation on April, 6th, 1908, the pancreas was found to be hard and inflamed throughout its whole length, but there were no gall stones to be found, nor was there any evidence of duodenal ulcer, though there were firm adhesions between the gall

bladder, duodenum, pylorus, colon, and liver. These were all separated, and as the gall bladder was too small to anastomose to the duodenum, it was opened and drained, the drainage being continued for some weeks. An examination of the urine on April 15th showed that though the pancreatic reaction continued, there was no trace of sugar, acetone, or diacetic acid.

Gall Stones: Glycosuria: Cholecystotomy.

On June 29th, 1909, I received a letter from a medical man, asking me if I would see a Mrs. D., aged 58, who was suffering from painful attacks of gall stones, but that she also had diabetes of four years' duration. On examination I found a marked swelling of the right lobe of the liver with dilatation of the gall bladder. Though she had never been jaundiced, there could be no doubt about the diagnosis of gall stones. An examination of the urine by Dr. Cammidge showed 4.8 per cent. of sugar with traces of acetone and aceto-acetic acid, a well-marked reaction for indican, and traces of urobilin. There was also a marked pancreatic reaction. She could not make up her mind for operation, having been told that any surgical treatment in her condition would be extremely dangerous. As her sufferings were continuing, she returned for treatment, and I operated on July 21st, when I found the gall bladder and cystic duct filled with gall stones, some of which were as large as small walnuts, others the size of haricot beans; the lowest was impacted at the lower end of the cystic duct, impinging the common duct. The pylorus was firmly adherent to the gall bladder, and the head of the pancreas was hard, evidently the seat of interstitial pancreatitis. I removed the gall stones, detached the adhesions, and drained the gall bladder. Recovery from operation was uninterrupted, and on July 28th Dr. Cammidge reported that there was no evidence of sugar in the urine, and that the pancreatic reaction had entirely disappeared. On October 27th I heard that the patient was quite well, and that there had been no recurrence of the glycosuria.

I could relate other cases, but these, I think, will be sufficient to show that my propositions are not merely theoretical or the results accidental.

The practical outcome of my experience is:

1. That the early recognition and treatment of interstitial pancreatitis, or of pancreatic catarrh, by drainage of the bile ducts, and thus indirectly of the pancreatic ducts, and the removal of the cause, whether that be gall stones, duodenal ulcer, or other conditions, may be the means of averting diabetes.
2. That in certain diseases of the pancreas, even after the appearance of glycosuria, surgical treatment is well worth considering, as in a number of cases it has led to a complete disappearance of sugar from the urine, and in others to an arrest of the disease causing glycosuria.
3. That every case of diabetes should be considered from its etiological point of view, seeing that certain cases of glycosuria of pancreatic origin are curable, and in others the progress of the disease may be arrested by suitable surgical methods that can be carried out with very small risk.

THE Bombay Vaccine-Therapy Institute is a private venture started by Mr. S. P. Shiydas, L.M. and S., in October, 1908. It appears from the first annual report that the founder and director, recognizing the importance of bacterial methods of diagnosing and treating disease, proceeded to London in 1908 and studied the subject practically in the laboratory of St. Mary's Hospital, under Sir A. E. Wright. On his return to Bombay he opened a laboratory on a small scale for the practice of the methods which he had learnt, and subsequently transferred his operations to a larger building. The number of cases treated by serum method in the year 1908-9 was 118, and included tuberculous diseases, staphylococcus, streptococcus, acne, and gonococcus infection, and three cases of cancer. Some clinical observations are recorded regarding the various diseases dealt with, and the success or otherwise of the proceedings. These remarks, though somewhat inconclusive, give an impression of honesty. Considerable success appears to have been achieved in treating tuberculous glands, early cases of phthisis, scrofula, and various forms of staphylococcus and streptococcus infections. The laboratory is utilized for clinical and diagnostic purposes. Mr. Shiydas is entitled to commendation for his energy in placing advanced methods of pathological and therapeutical practice at the disposal of the profession in Bombay, and we shall watch with interest the further development and results of his undertakings. As the project is a novel and very important one, we would suggest that it would be well for him to submit his proceedings to the inspection of competent critics, such as the scientific gentlemen whose names he mentions as having taken a warm interest in his work, and to publish in future reports the opinions of these observers regarding the value and success of his practice.