DISCUSSION.

DR. ROBERT PREBLE, Chicago, (exhibiting a boy with a cardiac aneurism): This patient was shot a few months ago, the bullet entering to the left side of the sternum and at the time giving no special cardiac disturbances. Later the patient developed a characteristic aneurism at the upper and outer portion of the left ventricle.

DR. JAMES B. HERRICK, Chicago: Dr McElroy's paper recalled the case, seen by me five or six years ago, of a colored man, about 30 years old. At autopsy, in addition to some valvular trouble, extensive adhesions and a mediastinopericarditis were found, as well as a chronic fibrous myocarditis. In one of the areas of cardiac softening, near the tip of the left ventricle, a rupture of the myocardium, evidently preceded by an aneurismal dilatation, had occurred; this rupture, instead of occurring directly into the open pericardium, occurred in a weakened spot between the two adherent surfaces of the pericardium that were apparently dissected apart by the blood. This little sac of pericardium had, at the time of death, reached the size of a goose's egg. The interesting clinical feature in this case was that, high up in the epigastrium beneath the xiphoid cartilage, was a visible, palpable, pulsating tumor, and over this could be heard a systolic bruit. The case presented many perplexing clinical features which made the diagnosis somewhat difficult. It was only at autopsy that the real nature of the trouble was learned; then the little sac communicating with the left ventricle was clearly disclosed.

DR. WILLIAM J. BUTLER, Chicago: I had a patient, a boy, 10 years of age, who had primarily a congenital stenosis of the pulmonary arterial tract. He acquired a septic endocarditis involving the pulmonary orifice. In addition to this, which had been diagnosed clinically, there was found at autopsy an aneurism, the size of a walnut, of the right ventricular wall. It had apparently resulted both from an involvement of the mural endocardium and underlying myocardium in the inflammatory changes, more or less fatty degeneration of the myocardium in general and the increased pressure in right ventricle.

DR. JAMES B. MCELROY, Memphis: The diagnosis of these conditions is possible only when the aneurism is favorably located. A most favorable and valuable diagnostic point is the heaving character, the apex-impulse and expansile character, which is only possible when the aneurism is favorably located; then and only then, in my judgment, is a diagnosis possible. In contrast to ninety cases diagnosed postmortem, I have met but one case that was diagnosed antemortem.

CHRONIC PANCREATITIS.*

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Perhaps the most important advances in abdominal surgery of recent years have been those made toward the elimination of digestive disorders, that is, the recognition that by surgery we are able to attack successfully not only the acute and obvious lesions, such as acute appendicitis or intestinal obstruction, but also those dependent on more obscure lesions of the stomach, biliary system and pancreas.

As the importance of the pancreas in the process of digestion was recognized by pathologists long after painstaking investigations had served to bring us to an understanding of gastric and intestinal physiology, so also the surgery of pancreatic conditions has been considerably behind that of the other abdominal viscera.

This is due to a number of reasons. The pancreas is deeply situated and almost inaccessible for direct surgical interference. Its functions being but little understood, their derangements naturally were not recognized, and those finer tests in physiology were as yet unknown.

The work of Fitz brought the attention of the profession to acute pancreatitis, but it was many years later that the chronic form of inflammation of this organ was clinically recognized, even though it had been pathologically studied by many observers.

PATHOLOGY AND ETIOLOGY.

As to pathology, chronic pancreatitis may be subdivided into the interlobular and the interacinar forms. For the purpose of the surgeon it is sufficient to state that the most frequent form is the interlobular, and that it is this form with which we have to deal in the pancreatitis complicating disease of the biliary passages. The interacinar form is due to systemic conditions not yet understood and is therefore correspondingly unfavorable to direct or local attack.

The etiology of chronic pancreatitis is of the utmost importance. There is no doubt that its most frequent cause is some interference with the free discharge of the pancreatic secretion, either associated or unassociated with an ascending infection. As by far the most common cause of such obstruction is the lodgment of a gallstone in the ampulla of Vater, or other part of the common duct, the frequent association of the conditions is at once explained. The occurrence under such circumstances of a pancreatitis, either acute or chronic, is not inevitable, but it is very common. The damming back of the pancreatic secretion with dilatation of the ducts and interference with function is in itself sufficient to cause a chronic inflammation of the gland. Yet in most instances we have no doubt to deal also with an added infection ascending the duct of Wirsung and joining its action to the other cause of inflammation. If the infection be virulent, acute pancreatitis may supervene—if of diminished virulence, we have the chronic form of pancreatitis as a result.

When the duct of Santorini is so situated that it can take the place of the duct of Wirsung, the main duct of the pancreas, as an avenue of discharge, pressure on the duct of Wirsung does not bring about such dire results, yet an infection at the same time active may nullify this action and lead to a pancreatitis even in the absence of a marked stasis in the secreting ducts.

The feature of infection would account for those cases of chronic pancreatitis which we find in those cases of gallstone disease in which the stone does not actually occlude the common duct. The very presence of the stones presupposes an infection of the biliary passages at one time or another, and when this is present there is little doubt that the whole biliary tract is involved. In a certain proportion of these cases the pancreatic duct would, of course, become infected also, and as the infection causing gallstones is known to be a subacute one—by some organism of diminished virulence—we have in the condition causing the gallstones one which also will cause a pancreatitis.

Indeed, most of my cases of chronic pancreatitis have, contrary to the general rule, occurred in patients in whom no gallstones were present. Yet in almost every case I could discern either the presence of an infection as shown by the bile, the congestion and an inflammation of the gall bladder, or the fact that there had been some cholecystitis, as evidenced in a shrunken and distorted gall bladder, or the presence of pericholecystic adhesions.

So while duct obstruction plus infection furnishes the most likely cause of chronic pancreatitis, either factor

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alone is sufficient to cause this condition. The rôle of infection alone is also demonstrable in those cases in which a chronic pancreatitis follows on a long-standing gastroduodenal catarrh. Several cases have come under my notice and have been reported by other surgeons. I have in mind particularly one case in which this was so, and in which the occurrence of the pancreatitis was directly traceable to a chronic gastroenteritis. It is important in these cases to bear in mind the fact that the supposed gastroenteritis thought to precede the pancreatitis may in reality have been its first manifestation instead of its precursor.

Blocking of the pancreatic ducts by calculi in them or by new growths of the common duct or the ampulla of Vater may be mentioned as among the rarer causes of chronic pancreatitis.

In some of my operations I have noted the association of a chronic pancreatitis with cirrhotic conditions of the liver, and in a number of cases which have come to autopsy from the medical wards of the German Hospital the two conditions have existed together.

Many other causes of chronic pancreatitis have been claimed. At the German Hospital it has been found at autopsy in cases of cardiorenal disease, tuberculosis, pernicious anemia, syphilis and other conditions.

There is no doubt that were microscopic examinations made of the pancreas many instances of beginning change would be found in cases where the organ feels and looks entirely normal. It is a well-known fact among surgeons that in quite a few instances in which in life the pancreas is found to be markedly indurated and congested, at autopsy, held some hours after death, the organ presents no macroscopic evidence of disease and is entirely normal to the touch.

The diagnosis of chronic pancreatitis in life was thought some years ago to be impossible, except in those cases in which the metabolism was so greatly disturbed that evidences of this were most markedly shown in both urine and feces. As surgeons have had opportunity of studying the organ on the living subject at operation, we have come to associate certain clinical symptoms with a condition of chronic pancreatic disease, and it may even be asserted that a somewhat definite syndrome has been established. In spite of this, I can not agree with Mayo Robson when he characterizes it as being easy to diagnosticate. At the present time by far the majority of cases are unrecognized except at the operation or necropsy.

SYMPTOMATOLOGY OF THE DISEASE.

In considering the symptomatology we must remember that the symptoms are caused in three ways, and may thus be divided into three groups:

1. Those which depend on the local lesion, i. e., the local manifestations of disease in the upper abdomen.
2. Those which come as a result of the interference with pancreatic secretion, i. e., a form of indigestion.
3. Those depending on interference with the internal secretion of the pancreas—shown principally in the occurrence of diabetes and in the pancreatic reaction in the urine.

1. *Symptoms Depending on a Local Lesion.*—The local symptoms are essentially unimportant as compared to the systemic ones. The patient will at times complain of some epigastric pain, occasionally localized somewhat to either side of the median line. Tenderness in this locality may also be present. The presence of a tumor has been described by some, but I have never been able to satisfy myself of the presence of this sign in chronic pancreatitis.

A general symptom, dependent often on local mechanical interference, is jaundice. We would naturally expect to find this in those cases in which the pancreatitis is associated with gallstone disease, but it occurs also when this is not the case. This in some instances is due to the partial obstruction of the common duct by swelling of the head of the pancreas and would be particularly liable to occur when the duct runs within the substance of the pancreas, as it does in about two-thirds of all cases. Occasionally the jaundice appears to have been a coincidence, i. e., to have been caused by cholangitis resulting from the gastroduodenal catarrh responsible for the onset of the pancreatic lesion.

Mayo Robson has lately offered the suggestion that many instances of catarrhal jaundice, especially of the chronic form, are due to a pancreatic lesion, i. e., that the engorged pancreas by pressure on the choledochus is responsible for the jaundice. This contention he has supported by the report of a number of cases in which the pancreatic disease was discovered by other tests; operation in all cases brought a permanent cure. I am not prepared to admit that all cases of catarrhal jaundice are pancreatic in origin. But the possibility should be borne in mind when we have to deal with forms of the disease which are resistant to the ordinary medical and dietetic treatment.

At times the damming back of the bile will give rise to distention of the gall bladder, which organ may even become palpable. In these instances the symptom is more apt to confuse than to help us, as it would lead us to believe the condition to be primarily biliary. This, however, would not be of such moment, as surgical interference is indicated in either case.

The local signs, as I have stated, are in themselves not significant and gain importance only when associated with other signs of pancreatic trouble. The jaundice, of course, would give us a clue, but as often as not it, as well as the local signs, leads us to suspect disease of the bile ducts or cholelithiasis, when the lesion is in reality pancreatic.

2. *Symptoms Due to Interference with Pancreatic Symptoms.*—The digestive symptoms due to interference with the pancreatic secretion are of the utmost importance. The patients often have anorexia, fulness in the epigastrium and eructations of gas. Associated with these we often have diarrhea of an intermittent or continuous form, in which the stools are large and often grayish in color and contain an excess of free fat.

This combination of such an indigestion with the local signs of a lesion in the upper abdomen should lead us to suspect a pancreatic condition at once.

Occasionally we become aware of the presence of a chronic pancreatitis by an acute exacerbation. Several instances have come to my notice in which this was so, with history about as follows: The patient, previously in fairly good health, was taken ill with symptoms of a lesion in the upper abdomen; pain here was preceded by a chill and followed by marked prostration and cyanosis. These in turn were succeeded by fever with the development of a dulness in the upper left abdomen and lower part of the chest. The Cammidge tests, A and B, were both positive.

It is true that many other portions of the gastrointestinal tract when diseased give us symptoms of indigestion, but each lesion will be found on close study to give rise to a more or less characteristic form. Thus,

in chronic gastric ulcer we have pain after eating, vomiting, hyperacidity and a tendency to constipation. The stools show us no excess of fats and undigested muscle fiber as in pancreatitis, but often give us signs of hemorrhage—such as the occult blood test. The associated epigastric signs are also often more severe, and inflation of the stomach may at times show us a considerable degree of dilatation. Jaundice also is not found.

Chronic gastroenteritis or colitis may give us persistent or intermittent diarrhea. Yet the localizing signs are all to be found in the lower abdominal segment. The stools are often watery and they show us no evidence of impaired digestion of fats.

In chronic appendicitis, constipation and not diarrhea is the rule. The indigestion is usually of a less marked grade than in either pancreatic or gastric lesions and does not exercise such a deteriorating effect on the patient's health in general. Then again we often find a history of acute attacks of appendicitis, or else of chronic soreness and tenderness in the right iliac fossa. The stools also show nothing characteristic.

The emaciation and weakening which the indigestion of pancreatic disease brings with it is insidious in onset, but in late cases most marked and intractable and furnishes another symptom, if it may be so called. But when the disease has progressed to this stage the diagnosis as a rule interests us more as a curiosity preceding autopsy than as an indication for treatment.

In spite of the apparent distinctness of the symptomatology of chronic pancreatitis, its differentiation is not always easy. Its onset is often most insidious, and one or another point in the symptom-complex is not present. Jaundice may not occur; the local signs may be practically absent. The examination of the feces is at times inconclusive and often demands skill in examination not easy to obtain.

Then again, in those cases in which the pancreatitis is associated with gallstone disease, the latter in almost all cases overshadows the often more dangerous pancreatic condition. And so well known are the symptoms of disease of the gall ducts compared to those of the pancreas that physicians are prone to consider the symptoms of a chronic pancreatitis as being the aberrant ones of cholelithiasis, even when the distinction is comparatively plain.

3. *Symptoms Due to Interference with the Internal Secretion.*—The third group of symptoms—those depending on disorders of metabolism, due to interference with the internal secretion of the pancreas—are really the most distinctive of all and give us the most definite basis for a diagnosis of pancreatic disease.

Diabetes has been long recognized as being often associated with pancreatic disease. Unfortunately, when this condition has supervened, the pancreatic lesion is often too far advanced for any marked improvement by any method of treatment, either medical or surgical. In some cases it must be remembered that the glycosuria is not due to a true diabetes resulting from interference with the internal secretion of the pancreas, but is due to the absence in the alimentary canal of its secreted ferments, and in some instances may be a pure alimentary glycosuria associated with pancreatic disease. It may be noted here that the interacinar form of chronic pancreatitis, affecting as it does the islands of Langerhans earlier than the interlobular form, is more apt to give us an early and intractable diabetes.

The reaction discovered by Mr. Cammidge I believe to be an aid in the diagnosis of pancreatic disease. It is true that the originator and his collaborators have been able to get more positive results from its use than others, but it has been found of value in many cases. I am inclined to regard it for the present, in the hands of most investigators and laboratory workers at least, as a fairly constant sign of pancreatic disease, rather than of great value in the differential diagnosis.

It will be seen, then, that the majority of cases of chronic pancreatitis, either associated or unassociated with gallstone disease, may be diagnosed with a fair amount of certainty if sufficient care be taken. They are cases in which the history must be most carefully taken and the patient's memory for details at times taxed to the utmost. In conjunction with this the study of the urine and feces should be undertaken by one who is an expert. The results in this line of work by the unpracticed are more likely to be misleading and confusing than of any real aid to the diagnostician.

TREATMENT.

Granted, then, that the diagnosis of chronic pancreatitis has been made, or that chronic pancreatitis is discovered on the operating table, what should be our line of treatment?

It can not be denied that the efforts to benefit patients suffering from chronic pancreatitis by the use of pancreatic extracts, derivatives or substitutes have been futile in all but a few instances. In these the results have at times been remarkable, but the treatment is, as a rule, irksome, especially when a strict diet must be followed. Not only this, but as no effort is really made to restore the pancreas itself to function, its action by its internal secretion is lost, and in spite of the substitution of its ferments metabolic disturbances generally increase.

Medical treatment also is entirely unable to really reach and remove the cause of the pancreatic lesion—to give the pancreas a chance to functionate again the normal way. Surgery in many of these instances will enable us to attain a radical cure. It does this in two ways:

1. By removing the underlying cause of the pancreatitis when this is to be found in an obstructed choledochus.

2. By enabling us to overcome the infection in the biliary and pancreatic ducts.

When the pancreatitis is secondary to gallstone disease, as is the case in so many instances, removal of the calculi, together with free drainage of the bile ducts, in most cases leads to a complete subsidence of pancreatic symptoms. The obstruction being removed, the pancreatic ducts have an opportunity of again emptying themselves and the irritating bile is no longer forced into them by back-flow. The coexisting infection of the biliary passages is cured or rendered harmless by the drainage instituted at the time of operation, and thus the pancreas is enabled to take its place as the main factor in the whole cycle of digestion. The benefit to the chronic pancreatitis in many cases of gallstone disease doubtless accounts for the wonderful restoration to complete health of those patients who are found to be weakened and emaciated to a degree not explainable by the mere biliary condition.

When the gall ducts are found clear, and the infection which has given rise to the chronic pancreatitis has subsided, drainage of the organ by way of the biliary passages nevertheless exerts a remarkable curative effect on the lesion. This is especially true when the pan-

creatic condition is diagnosed and the patient operated on while it is in its incipiency, before the so-called catarrhal pancreatitis has really become a chronic interstitial interacinar lesion.

The choice of operation in dealing with chronic pancreatitis resolves itself into a decision between simple drainage of the biliary ducts or a cholecystenterostomy. I have used both methods, and each has its advantages in special cases. When gallstones are found in conjunction with pancreatic disease, or when the latter is found during a gallstone operation, I consider the drainage indicated by the biliary condition to be sufficient. Thus, if we have stones in the choledochus, a choledochostomy should be performed in the usual manner. The operation of cholecystostomy is to be preferred when we find a pancreatitis the result of a still active infection of the bile ducts or when the pancreatitis is discovered in its incipient or catarrhal stage.

Cholecystenterostomy is indicated when the pancreatic condition is well advanced and we wish to procure permanent drainage. This operation is not my choice when much biliary infection is present, as I always prefer surface drainage when marked infection is manifest. This operation also I consider to be more grave than ordinary cholecystostomy, and I have found it to be attended by a higher mortality in my own operative work. Especially does this become true when we are dealing with a gall bladder not well suited for the procedure.

The indications for surgical interference in chronic pancreatitis I consider to be found in the diagnosis itself, unless some circumstance prohibits it. Thus, I would not operate in the presence of marked organic disease of other organs, nor would I be prone to advise operation in patients who are *in extremis*.

Moderate anemia and glycosuria are not contraindications to operation in pancreatic cases, as both are often greatly benefited when the metabolism of the body is restored to its normal status. My results in chronic pancreatitis have been such as to encourage me to further operative work in this direction. The immediate mortality is still quite high, due in large part to the extremely weakened condition of the patients, the grave associated conditions, and especially the tendency in those patients in whom we find both jaundice and a pancreatic lesion, to uncontrollable hemorrhage.

The details of the operative technic are not matters of extreme difficulty, and improvement in results must come from earlier diagnosis. In gallstone cases the patients should be operated on before chronic pancreatitis supervenes, and other cases of pancreatitis unassociated with lesions of the biliary passages should be recognized sooner than they now are.

PANCREATITIS IN ITS RELATION TO GALLSTONE DISEASE.*

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The pancreas is the most sequestered organ in the body and has only recently yielded up, in part, the secrets of its incorrigible inflammations. The clinical knowledge of its pathology has been obtained in operations on the gall tracts. Its natural history has been unraveled and, while in the past its recognition has been uncertain, it can now be identified during life in a considerable number of instances, both in its acute and chronic form. It is important in all operations on the upper abdomen that the surgeon should examine with detailed care the head of the pancreas, not only to determine the existence of pathologic changes, when present, but also to appreciate the "feel" of the normal gland. The fact that the Mayos found the pancreas to be involved in 6 per cent. of all the operations on the gall tracts is sufficient indication of its importance, as well as of the causal relation between gallstones and diseases of this neighboring gland. They further found 81 per cent. of pancreatic diseases to be the result of, or coincident with, gallstones. Egdahl regards biliary lithiasis the most frequent single cause. Osler says that forty-five out of one hundred and five cases were associated with gallstones. Robson found pancreatic implication in 60 per cent. of cases in which gallstones were in the common duct. Out of 118 cases Quénu and Duval in 46 cases found stones in the gall bladder or cystic duct; in 20 cases in the common duct; in 8 cases in the ampulla of Vater; in 2 cases in the duodenum, near the ampulla; in 28 cases in the entire tract; in 10 cases undetermined, and in 3 cases in the stools. Mayo observed pancreatitis to be four times as frequent when the stones are in the ducts as when they exist in the gall bladder (18.6 to 4.45). The head of the gland is involved seven times as often as the entire gland (124 to 17). Nearly two-thirds of common ducts are surrounded by the head of the pancreas, which, if swollen, causes obstructive jaundice. In the other one-third an independent opening of the duct of Santorini may act as a safety valve.

Opie discovered the rôle which a small (pea-sized) stone lodging in the ampulla of Vater plays in converting the common and pancreatic ducts into a through channel, allowing bile to be injected directly into the pancreas and thus produce the acute type of infective and hemorrhagic pancreatitis.

Robson has established on an indefensible basis the relationship of gallstones in the common duct and chronic interstitial pancreatitis. It is probable that stones simply render the organ more vulnerable to bacterial invasion.

Flexner asserts that modified bile, with diminished salts and increase in colloid, sets up chronic pancreatitis. Fresh and unaltered bile gaining entrance to the pancreas begets acute changes. If an impacted gallstone does not cause bile to enter the pancreas direct, simple obstruction and retention of the pancreatic secretions, if infected, will cause inflammation.

Desjardins thinks that micro-organisms find their way through the duct of Santorini into the Wirsungian duct and back into the duodenum, thus causing infection in the "triangle of inflammation" when there is obstruction. This could not occur in one-third of the cases for in 21 per cent. the duct of Santorini is impervious and in 10 per cent. it does not communicate with the duct of Wirsung.

Regurgitation of fatty materials from the duodenum into the duct where it had been dilated from the previous passage of a gallstone has been suggested by Hess as a causative factor.

Robson operated in fifty-two cases of the chronic interstitial type which were due to gallstones and in forty-six cases in which there were no gallstones. Of this latter group it has been said that gastrointestinal disor-

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