the expense of diastole. Very important is it, therefore, to reduce the heart rate where this is excessive. A chief cause of rapid heart action where heart failure threatens or has come is fibrillation of the auricles, and it is in this condition that digitalis acts so beneficially; it reduces and holds the rate within normal bounds.

The reduction of accelerated ventricular rate is the only important action of the drug upon the human heart of which we have knowledge. There are few, if any, instances of which we know with certainty in which digitalis acts beneficially, except cases of accelerated action; there are few instances of acceleration in which the drug produces unquestionable benefit apart from those provoked by fibrillation of the auricles. The principle of digitalis therapy—and when I speak of digitalis I include the allied drugs, strophanthus and squills—is that, administered to suitable cases, the heart, by means of it, obtains rest. The giving of this drug to unselected cardiac cases is much to be deplored. Those who regard digitalis as a cardiac stimulant mistake its character; its chief action is to rest the heart. *To the heart foreglove is not tonic, but powerfully hypnotic.* It extends the diastoles of the heart; it extends the period of sleep.

There is a direction in which medical men are much hampered in dealing with chronic heart disease. It is in trying to order the future lives of working class men to suit the heart's power of reserve. Many cardiac patients leave our hospital wards after a short period of rest, a period during which there has been much improvement of the heart's capacity for work. Yet these patients are but too often totally unfit to take up again those daily tasks in and by which they have already suffered. To tell a working class man that his former work suits him ill, to tell him that he must find new work of lighter grade, is good enough advice; but how often is it within the man's power to act on that advice? how often is it within the doctor's power to see that his advice is followed? We greatly need in this country a system by which disabled workers are placed and cared for in suitable employment. Cardiac cases are for the most part conspicuously willing workers, frequently too willing; there are many men doing heavy work in this country to-day, and at the same time surely, but may be slowly, dying of heart disease. It is a shameful thing that these unfortunates are put by hard necessity to tasks far beyond their endurance.

Work is good for cardiac cases, as it is good for men in whom the voluntary muscles of the body are ill conditioned, but only such work as falls well within the bounds of the heart's reserve is beneficial. It is a most important matter, and a matter very little attended to to-day in hospital practice, to test a man's capacity for work before he goes to his home; it is not sufficiently understood that discharge from hospital is read by many as a sanction to return to the old work.

Infection.

As infection is the chief cause of heart disease, in the stage of its inception, and in the stages of its advance, so, when we come to treatment, to stay the advance by removing the cause is a chief principle. The treatment of early syphilitic lesions of the heart by appropriate measures is a field as yet little explored, but one which is in promise fertile. To treat drastically local invasions by pyogenic organisms is clearly indicated, and of proved benefit in many cases. Even more important is it to guard against infection. Cardiac cases run very serious risks, risks which but too often cannot be avoided, by contact with those suffering from some infection of the respiratory tract. Nevertheless, much harm may be avoided by timely warnings of dangers, met in the family circle, in crowded buildings and in ill ventilated rooms, by the prescription of the open air régime, and, in bed cases, by rigorous separation from febrile cases.

REFERENCE.

¹ *The Soldier's Heart and the Effort Syndrome.* London: Shaw and Sons. 1918.

THE forty-third annual report of the Boston Medical Library recently issued states that on December 31st, 1918, there were 98,097 volumes catalogued and on the shelves, besides several thousands of others not yet catalogued. A number of current German periodicals were received after an interval of nearly three years. The number of foreign books received was small.

REMARKS ON THE FREQUENCY, DIAGNOSIS, AND TREATMENT OF CHRONIC PANCREATITIS.

By JAMES H. NICOLL, M.B.,

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For some time it has been generally accepted that in many cases of disease in the abdomen a pathological tripod stands in that cavity with feet implanted in the appendix, biliary apparatus, and gastro-duodenal tract. The coexistence, or the sequential development in any order, of appendicitis, cholecystitis or cholangitis, and gastric or duodenal ulcer, is familiar to clinicians and surgeons. Opinions vary as to whether one or other of the three stands in causal relation to the others, or all three derive from a common and central source in the form of catarrhal inflammation of stomach and bowel. Much work has been done, and remains to be done, on the question.

Meantime, as the records of cases accumulate, it is becoming evident that the tripod is being gradually replaced in the problem by a quadrupedal figure. Pancreatitis is claiming increasing attention.

In the issue of the JOURNAL for July 12th last Sir Berkeley Moynihan publishes a very instructive lecture on "Disappointments after Gastro-enterostomy," in which he quotes me on one of them. In my experience chronic or subacute pancreatitis ranks next in order to chronic appendicitis and disorders of the biliary apparatus on the list of affections which mimic gastric and duodenal ulcer, and for which gastro-enterostomy is erroneously performed, with resulting disappointment.

Clinically pancreatitis occurs in two widely differing types—acute and chronic—which often prove equally elusive of diagnosis.

Acute pancreatitis (necrotic, haemorrhagic, and suppurative) is rarely suspected until discovered at an autopsy or in course of a laparotomy undertaken for a supposed perforated gastric or duodenal ulcer, a fulminating appendicitis, or other similar acute abdominal affection calling for operation. The case commonly goes on to the operating table a surgical puzzle and leaves the hospital by way of the mortuary a surgical disaster, though prompt operative treatment is claiming an increasing number of successful results.

Chronic pancreatitis (catarrhal and, later, cirrhotic) may, especially in its later phases, present suggestive diagnostic signs. Many cases, however, more especially of the earlier and slighter types, present no such signs, and the disease closely mimics other abdominal disorders, particularly duodenal and gastric dyspepsia and ulceration and affections of the biliary apparatus, for which supposed diseases the appropriate operative measures are apt to be erroneously carried out. The error in the case of gastro-enterostomy is unfortunate, for, while gastro-enterostomy may do the patient no harm in some cases, in others it aggravates his distress. In either event the operation results in disappointment. In the case of the biliary apparatus the error may ultimately be harmful or helpful; and this raises the whole question of the technique of cholecystectomy. If in a case of supposed cholecystitis which is really one of pancreatitis, or in a case in which pancreatitis complicates cholecystitis, the gall bladder is only partially removed and the cystic duct, and possibly also the common duct, drained, the pancreatic patient may benefit, though not to the degree possible had his pancreatitis been diagnosed and suitable dietetic and medicinal treatment prescribed. If, on the other hand, complete cholecystectomy with closure of the duct is carried out, all chance of surgical treatment of the pancreatitis is, for the time being at least, lost. Pancreatic drainage can only be carried out later by a much more serious operation and after valuable time has been wasted. It should be an unvarying rule not to remove the entire gall bladder without drainage of the ducts unless the surgeon is satisfied that pancreatitis is absent.

Diagnosis of Chronic Pancreatitis.

Chronic pancreatitis is of more frequent occurrence than has generally been supposed. It commonly complicates

catarrhal affections of the duodenum, of the biliary apparatus (whether the seat of cholelithiasis or not), and of the vermiform appendix and colon, but apparently it may exist as an affection standing alone. Its diagnosis is nearly always a matter of difficulty. Frequently the difficulties prove insuperable until an excised portion of the gland is examined microscopically. Even after the surgeon has the organ between his fingers and thumb during an exploratory laparotomy uncertainty often remains, though in many cases such palpation goes far to settle the diagnosis.

Two recent contrasting cases may be quoted. Both were operated on for supposed duodenal ulcer, in the expressed diagnosis of which I had concurred. On operation no lesion of the duodenum was found, nor was any other explanation of the symptoms discovered till the pancreas was examined. In one case the gland to touch was clearly cirrhotic, in the other it felt normal. A small wedge of the gland was removed in each case, and the specimens were microscopically examined by Dr. G. Haswell Wilson. The hard gland proved to be the seat of "cirrhotic changes with small areas of necrosis." The apparently normal gland showed "typical haemorrhagic pancreatitis with areas of necrosis in the substance of the gland."

During the past eleven months I have operated on seven cases of supposed gastric ulcer, duodenal ulcer, or cholecystitis in which neither ulcer nor cholecystitis was found, but in all of which the microscope revealed the presence of pancreatitis. In all of these cases the diagnosis of ulcer or of cholecystitis, with which I had agreed, rested on the investigations of medical colleagues well qualified to form an opinion. In none of the seven was there anything to suggest pancreatitis until operative exploration revealed its existence. In one of them gastro-enterostomy had previously been performed without, of course, relief of the symptoms.

In the diagnosis of pancreatitis the following points may prove helpful:

Glycosuria (islands of Langerhans affected?). This characteristic symptom is, unfortunately, present in comparatively few of the cases.

Large light coloured greasy stools, or pale fatty diarrhoea. These equally suggestive symptoms are also found in a minority only of the cases.

Excess of fats and fatty acids in the stools as demonstrated microscopically. Except in advanced cases this test is apt to fail in definiteness. Much depends on the patient's diet.

Jaundice is not uncommon. Its presence naturally directs attention to the biliary apparatus rather than to the pancreas. It is, however, a reliable indication of the need for operation, and, if this is carried out thoroughly, pancreatitis if present, either alone or as a complication of cholecystitis or cholangitis, is discovered.

Schmidt's test for undigested tissue nuclei (consumed as food) in the stools.

Wohlgemuth's test for starch-splitting ferments in the stools. In a case at present convalescing after operation for confusing, though pronounced, abdominal symptoms, Dr. Hugh Morton had in April last, by employing Wohlgemuth's test, discovered that the pancreatic secretion was defective. The only lesion I found in the exploration was a cirrhotic pancreas, on an excised portion of which Dr. Haswell Wilson reports as showing "advanced chronic interstitial pancreatitis."

The two last-mentioned tests may prove valuable. As yet their reliability in the early or slighter cases remains to be proved, and it is the slighter cases which so closely mimic other abdominal affections.

"*Fat Necrosis*."—The discovery during exploratory abdominal section of the characteristic patches on the pancreas, peritoneum, and other abdominal structures leads at once to a diagnosis of pancreatic disease. Some years ago, assisted by Dr. Chas. Cutlbert, I opened the abdomen of a patient who had been ill for five days and whose case had been diagnosed as intestinal obstruction by one physician and perforation of gastric or duodenal ulcer by another. An exhaustive search failed to discover any lesion whatever (at that time I was not so alive to the necessity of a routine examination of the pancreas in obscure abdominal cases), and I prepared to close the abdomen. On eversion of the parietal peritoneum with forceps, preparatory to suture, patches of fat necrosis were at once discovered

limited to the inner aspect of the anterior parietes, and therefore not visible during the exploration. "Fat necrosis," which is pathognomonic of pancreatic disease, is, unfortunately, rarely present, except in cases in which the outflow of the pancreatic secretion is obstructed. It is absent in the larger number of cases of pancreatitis.

Palpation of the Pancreas.—This may reveal a cirrhosis so hard as to simulate carcinoma. Such a case occurred recently in my practice. In this case Dr. Roy Young, who co-operated with me, concurred in the view that the pancreas was the seat of cancer. Yet microscopical examination of an excised portion proved the presence of pancreatic cirrhosis, and not cancer. In other cases palpation discovers the bulky soft outlines and consistence of a gland the seat of catarrhal pancreatitis. In many cases in which the gland to palpation seems normal microscopic examination reveals the existence of pancreatitis.

Resection of a Small Wedge of the Gland for Microscopic Examination.—This has of late become in my practice almost a routine procedure in cases in which there is any doubt whatever as to the actual cause of the abdominal symptoms present. The results have convinced me that chronic pancreatitis is a common disease, and that meantime its diagnosis by other means is not infrequently impossible. Failure to diagnose it, whether it exists alone or as a complication of other diseases, entails various misfortunes. It may lead the surgeon during an abdominal exploration to do nothing, whereas drainage, by way of cholecystostomy, choledochostomy, or, preferably, cholecystenterostomy, would constitute the first step in effective treatment. It may lead the surgeon to do an inefficient operation—namely, a complete cholecystectomy without drainage. And it may lead the surgeon to perform an entirely erroneous operation, such as removal of the appendix or performance of gastro-enterostomy. Further, it deprives the physician, in the subsequent course of the case, of that precise knowledge on which medicinal and dietetic treatment could be successfully based, and of the opportunity of testing definitely the value of such tests as Schmidt's and Wohlgemuth's. Lastly, it deprives the patient of the mental comfort of knowing that, though his convalescence may be long (and chronic pancreatitis is a tedious trouble) his case is clear, and cure to be looked forward to.

Operation.

The pancreas may be reached for exploration in the course of a laparotomy by the gastro-hepatic, the gastro-colic, or the trans-mesocolic route, of which the gastro-colic is probably the most generally useful. The small gap left in the gland after excision of the wedge is closed by sutures inserted by an intestinal needle, and covered over by omentum tacked on by further sutures. Accurate closure in this way secures all haemorrhage, and prevents leakage into the peritoneum of the glandular secretion, which, in many of the cases, is not only infective, but in itself capable of necrosing effects.

Treatment of Chronic Pancreatitis.

Pancreatitis apparently often forms one of the four feet of the pathological intra-abdominal figure referred to above. Treatment, therefore, of any accompanying, and possibly causative, mischief in biliary apparatus, gastro-duodenal area or appendix, is essential. Pancreatitis itself, whether existing alone or accompanying other of the associated affections, is a persistent trouble, and its treatment is best carried out by the combined efforts of surgeon and physician. On the surgeon rests in many of the cases the ultimate diagnosis by means of excision of a portion of the gland for microscopic examination. On him also rests the initiation of the treatment, by drainage of the inflamed gland. With the physician remains the direction of the later stages of treatment on dietetic and medicinal lines, a much more definitely purposive effort after the diagnosis has been cleared up, the gland drained, and associated diseases dealt with.

Drainage of the pancreas is most often carried out by way of the biliary apparatus—cholecystostomy, choledochostomy, or preferably, when the conditions permit, cholecystenterostomy. Direct drainage of the duct of Wirsung is adopted in cases in which pancreatic calculi are present or obstruction of the duct suspected. Where such drainage is established the tube should be fixed as accurately as possible in the duct or up against the

opening made in the duct, and the junction reinforced by drainage wicks of gauze brought out through the abdominal incision, the whole arrangement being surrounded and isolated by omentum carefully fixed by sutures. The gauze acts as a drain for any leaking pancreatic secretion, and further sets up firm omental adhesions before its withdrawal at the end of two or three days. Neglect to isolate the drainage aperture in the way indicated may lead to serious or fatal results, for the secretion is not only in itself capable of necrosing effects on the peritoneum, but is in addition germ-infected in many cases of pancreatitis. Drainage of the pancreas by incisions into the gland itself, while necessary in desperate cases of acute pancreatitis, is of doubtful expediency in chronic cases. If employed, it should be carried out only by means of the freest combination of large rubber tubes and ample gauze packing carefully surrounded by omentum, and, if at all possible, by the gastro-colic route, which best lends itself to such an arrangement.

THE CAUSATION AND TREATMENT OF RICKETS.*

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It is to my mind open to question whether the time has not now arrived for a reconsideration of the whole subject of rickets—its definition, its etiology, and its treatment.

The following may be taken as a typical definition of the disease:

Rickets is a chronic general disorder of nutrition occurring during infancy from faulty diet and hygienic deficiencies. It involves the whole body, but its most obvious effects are recorded in the skeletal system.

This definition, and the majority of those with which I am acquainted, appear to me to confuse cause and effect. If the term is not confined to the bony changes, but covers also all the various forms of malnutrition which ultimately terminate in rickets, the name means little else than a generic one for ill health in infants and young children. Some authorities classify such symptoms as sweating, muscular weakness, laxity of ligaments, delayed teething, spasmophilic manifestations, and tendencies to catarrhs as rachitic. Francis Glisson included in his famous monograph on rickets every symptom and childish ailment which was known in his day.

The view which I propose to develop in this lecture is that practically all varieties of malnutrition occurring during infancy and early childhood tend to terminate in rickets, provided they are sufficiently severe or long enough continued. They should not, however, be regarded as evidence of rickets unless they are actually accompanied by the typical changes in bone which are characteristic of the disease.

Each variety of malnutrition has its own particular etiology and requires special lines of treatment, and cannot be dealt with rationally on the cut-and-dried lines that are usually advocated for rickets itself.

Shortly stated, the theory which I wish to propound is that the essential and central feature of rickets is the want of calcification or mineralization of developing bone, and that this in its turn is due to the existence of requirements for calcium, which for the time being are more urgent than those of developing bone. These urgent requirements are the necessity for neutralizing acid bodies in the blood; in other words, to neutralize or compensate an existing acidosis. My argument is that all chronic conditions of malnutrition, of whatever kind or from whatever cause arising, finally terminate in an acidosis, and that all claims on alkaline bases arising in connexion with the neutralization of this acidosis must be satisfied before those of developing bone are attended to.

It is not difficult to understand why all forms of malnutrition tend to end in a condition of acidosis, for in all cases, though in various ways, metabolic or oxidation processes are interfered with and curtailed. Any interference with the due oxidation of food elements to their normal

end-products, carbonic acid, urea and water, will conduce to the formation of semi-oxidized bodies of large molecular size and acid in reaction, which must be neutralized as quickly as they are formed to prevent a dangerous rise in the hydrogen ion concentration of the blood. It is in the satisfaction of these claims for alkaline bases that the injury is done to growing bone. One of the direct causes of an acidosis is a disturbance of the relationship between the caloric value of the food consumed and the actual output of energy, either in the form of heat or of work. If more food is consumed than is required for the physiological purposes of the body there will be an attempt to adjust this want of balance by excreting part of the food in an unoxidized, or semi-oxidized, condition. Any condition, therefore, which tends to diminish the efficiency of the human body as a working machine will tend to reduce the physiological demands for food, and so renders a pre-existing correct diet thereby excessive, or relatively excessive.

Most of the recognized etiological factors of rickets are factors which interfere in some way with the metabolic or oxidizing processes of the body—in other words, interfere with the proper utilization of food. Any condition, therefore, which interferes with the supply of oxygen will tend in the long run to be a factor in the production of rickets. So also will be any condition which interferes with the output of work—as, for instance, the poisoning or degeneration of nerve cells. Anything also which unduly interferes with the dissipation of heat—as, for instance, warm rooms or excessive clothing. The withholding of surface stimulation, such as is afforded by sunshine and currents of fresh air, will also have the same effect. Most of the etiological factors of rickets are included in this category of causative factors of an acidosis; and, in the light of these explanations, I propose now to review some of the theories on the pathogenesis of rickets which have been formulated from time to time, and which for the most part have been unwarrantably rejected as unsatisfactory, because, although possibly applying to certain cases, fail to do so to all. I shall hope to show that while there are different theories required to explain the various forms of malnutrition which are fore-stages in the common terminal result—namely, the production of an acidosis—there is only one explanation of the want of calcification of bone, and that is the urgent requirement of the body to have the acidosis neutralized at the expense of the alkaline reserves of the blood, reserves from which growing bone would otherwise draw its mineral material.

The first theory to be considered is that of a *deficiency of lime in the food*. It is clear, apart from any experimental proof, that without lime bones cannot be formed, but since there is an adequate supply of calcium in breast milk, and an excess of this base in cow's milk, it is obvious that want of this element cannot *per se* be the explanation of rickets in the majority of cases. None the less, a deficiency of lime is quite probably the true explanation of a small minority of cases in which certain patent foods are exclusively employed.

Defective absorption of lime is a more reasonable explanation, and colour is lent to this theory because in rickets an increased output of lime in the faeces can practically always be proved.

It is easy to understand that any available base such as calcium existing in the intestinal contents will be readily seized to neutralize any acid bodies formed as the result of putrefactive or fermentative changes in the bowel. It has been denied that this is the true explanation of rickets, for the reason that the administration of supplementary supplies of calcium salts in rachitic conditions is followed by an equivalent output of calcium in the urine. This to my mind proves nothing, for if under such conditions excess of calcium is absorbed into the blood, it may well be utilized for the neutralization of acid bodies coexisting in the blood. It is interesting to note that soap stools are very common in rickets, and these soaps consist largely of fatty acids in combination with calcium, a fact which is very suggestive of the occurrence of an excess of fatty acids in the bowel in the conditions which predispose to rickets. But whether calcium is eliminated in excess or not in the bowel, there is abundant evidence to show that it is eliminated in excess either in the faeces or in the urine, or in both.

* A lecture delivered before the Fellowship of Medicine at the Royal Society of Medicine, October 1st, 1919.