Surgical lesions of the pancreas are not sufficiently common for any one surgeon to see a large enough number and so to form definite conclusions from his personal experience. Such lesions do occur, however, with enough frequency to make it necessary for the general practitioner at least to suspect that the pancreas may be the cause of the symptoms, particularly in the presence of an acute abdominal catastrophe.

In order to make up for the lack of a large personal experience, a study of the literature and an examination of the histories of a group of cases from a large hospital help to clarify one's ideas. But, in the literature, one meets with many differences of opinion, and, in the case histories, a study of the pathological condition found at the time of operation compared with that found in subsequent autopsy reports further may confuse one's ideas. Even the nomenclature of the disease is a subject of confusion. The lesions variously are reported as “acute pancreatitis”, “acute pancreatic necrosis”, “acute hemorrhagic pancreatitis”, “subacute pancreatitis”, “edema of the pancreas” or even “chronic pancreatitis”. No one diagnosis covers all cases, and chronic pancreatitis is included here because what appears to be a minor or chronic lesion of the pancreas may be found at an operation for cholecystitis or cholelithiasis. Then, after what is believed to be the proper surgical operation, should the patient die within a few days, a most severe form of necrosis or disintegration of the pancreas may be found at autopsy.

Data from the Literature and Hospital Records

On examining the literature, one finds very little reduction in the mortality over a period of years. In 1911, Korte reported one hundred and three cases with 60 per cent mortality. In 1927, Schmieden and Sebening tabulated one thousand two hundred and seventy-eight cases with fifty-one and two-tenths per cent mortality. A statistical study of this condition, however, would appear to be of somewhat questionable value because of the various types of lesion included in such a group of cases. In a review of the histories of the cases operated on for acute surgical lesions of the pancreas at St. Luke’s Hospital, this difficulty was made most apparent. Many of the cases were operated upon with the diagnosis and in the presence of acute gall bladder lesions and a certain degree of pathology of the pancreas was found, frequently no more than a thickening of the head of the pancreas with some fat necrosis about the gland. Several of these patients died, and at autopsy it was found that the pancreatic lesion had extended even to a degree of almost complete pancreatic destruction; this had been the cause of death. Thus, the mortality rate would depend on the type of case on which operation was performed. In a series of thirty cases operated on in St. Luke’s Hospital during fifteen years previous to 1933, there was a 50 per cent mortality. Since that time, there have been six operative cases with only one death, but the fatal case was the only one in which an acute fulminating lesion was present.

Etiological Considerations

In the study of the etiology, conflicting facts, or observations which have been interpreted as facts, should be considered with relation to each other. Such consideration may demonstrate the reasons for the conflicting theories. Seventy-eight years ago (1856), Claude Bernard proved, experimentally, that, in animals, the injection of certain substances into the pancreatic duct would cause death from, or various degrees of, acute pancreatic necrosis. Bernard used a mixture of bile and olive oil. Since that time, these and similar experiments have been repeated; gastric juice, duodenal contents, solutions of acids, alkalies, formalin, bile and bile salts were found to have the like effect, while less irritating substances had little or none.
The work of Opie (1901), in which he produced acute pancreatic necrosis in animals by the injection of bile into the duct and his publication of an autopsy finding, in which a small gallstone obstructed the ampulla of Vater in a patient dying of acute pancreatic necrosis, linked up much of this experimental work with the findings of the disease in human beings. Five years later, Flexner proved that the essential constituents of the bile, necessary to cause pancreatic necrosis, were the bile salts, of which the taurocholate was the more active. He also showed that the mixture of mucin, due to its action as a colloid, with the injected material rendered it less active. It had been observed that the passage of a gallstone through the common duct, resulted in a dilatation or paralysis of the papilla (Vater's). This was responsible for the theory, advanced by Hess, and by Williams and Buck, that acute pancreatic necrosis was caused by a damming back of the duodenal contents into the pancreatic duct.

By his experimental work, Archibald has done much to explain an important factor in the production of the lesions of acute pancreatic necrosis. Working on cats, he found that the small sphincter at the common duct orifice, first described by Oddie (1887), furnished this link. Oddie had demonstrated that irritation of the duodenum or even stomach mucosa, either mechanically or with dilute hydrochloric acid, or stimulation of the vagus would cause spasm of the sphincter and that the sphincter, in dogs, would resist pressure of fifty millimeters of mercury. By the introduction of infected bile under just sufficient pressure, so as not to overcome this sphincteric action, Archibald was able to cause the death of a cat from acute pancreatic necrosis in twenty minutes. He enumerated a group of factors, the necessity of the presence of which makes understandable certain conditions in the causation of the disease which otherwise would be hard to explain. These three factors were: first, changes in the composition of the bile due to infection, which increases the proportion of bile salts; second, undue resistance, perhaps amounting even to spasm of the common duct sphincter; third, abnormal rise of pressure in the biliary system, either in the gall bladder or the common duct.

These experimental observations, linked up with observations on what is known of the occurrence of attacks of acute pancreatic necrosis in human beings, would seem to make a case for the usually accepted "biliary reflux" theory as to its etiology. The association of the attacks with gallstones and biliary infection accounts for the necessary change in the bile. The occurrence of an acute attack after a hearty meal would account for the necessary spasm of the sphincter of Oddie, as would also gastric hyperacidity or duodenal inflammation. The occasional finding of a gallstone impacted in the ampulla suggests that, in certain cases, a stone may have been impacted and then passed; this would explain the previous damming back of infected bile.

But, having enumerated these various biliary conditions, which would, apparently, be causes of the acute lesions of the pancreas, one must consider certain observations which would seem absolutely to refute the possibility of the disease being caused by a reflux of infected bile in other cases. These are, that anatomical conditions in some cases make it impossible that a reflux of bile could have been possible etiologically. Opie, in his book on diseases of the pancreas, quotes various case reports in which reflux of bile could not have been the cause of the pancreatic necrosis. In one case report by Eliot, the duct of Santorini was the larger and entered the duodenum about one and five-tenths centimeters above the papilla, through which emptied the smaller duct of Wirsung and the common duct. The necrosis, in this case, was most marked in the portion of the gland drained by the larger duct of Santorini. Johnstone reported two cases of pancreatic necrosis in which the pancreatic duct opened into the duodenum one or two centimeters above the common bile duct opening. Bassett reports one case in which the necrosis involved the area drained by the small duct of Santorini, which entered the duodenum through a small diverticulum above the papilla. It is also known that, at autopsy, the pancreas may be found to be extensively stained with bile, but exhibit no necrosis.

In these cases, reflux of bile could not have occurred. The theory that the duodenal contents might be forced back into the pancreatic duct and would cause pancreatic necrosis, would have to be the explanation of the etiological factor in these latter instances, admitting the reflux theory. Many years ago, Polya showed that duodenal contents injected into the duct would cause pancreatic necrosis. But, experimentally, this could occur only when the injection was made under pressure. Attempts to cause necrosis of the pancreas by experimental duodenal obstruction below the duct have not been successful. White and Owen have reported a case in which a carcinoma extending from the stomach, so dilated the duodenum and the sphincter of Oddie that it allowed free flow of the duodenal contents, bile and pancreatic juice into the pancreatic duct without causing any particular damage to the pancreatic tissue.

To add to the confusion of thought, the theory of infection of the pancreas by means of retrograde infection through the lymphatics from the gall bladder, appendix or a duodenal ulcer, has been advocated by Bartels, Arnsperger, Franke, Deaver, Pfeiffer and Sweet and must be considered. It would seem to me that the theory of infectious origin of pancreatic necrosis would appear to be contrary to the fact that, although in some cases, one finds enlarged lymph nodes about the head of the pancreas, in the series of 32 cases examined in St. Luke's Hospital only one showed