In 90 specimens the two ducts are united; in 10 two wholly independent ducts enter the intestine.

1. Of the ducts in anastomosis: (1) Duct of Wirsung, larger in 84—(*a*) duct of Santorini patent in 63, (*b*) duct of Santorini not patent in 21. (2) Duct of Santorini larger in 6—(*a*) duct of Wirsung patent in 6, (*b*) duct of Wirsung not patent, 0.

2. Ducts not in anastomosis, in 10: (*a*) Duct of Wirsung, larger in 5, (*b*) duct of Santorini, larger in 5.

In 89 per cent. the duct of Wirsung was larger than the duct of Santorini. In 21 per cent. the duct of Santorini was apparently obliterated near its termination. In 6 cases the duct of Santorini was larger than the duct of Wirsung. In all cases where the duct of Santorini is patent it diminishes in size towards the duodenum. Thus the duct of Santorini cannot be relied on in many cases to supplement the duct of Wirsung, if it be obstructed; moreover, the duct of Santorini, even if patent and communicating with the duodenum, may itself be compressed by a moderate sized gall-stone passing down the pancreatic portion of the common duct. Now it might be argued that, if the two ducts communicate, why should not the duct of Santorini act as a safety valve to the duct of Wirsung when it is compressed, and thus free the pancreas from the retained secretion which is in danger of becoming septic?

It will be seen that in only half or less than half of all cases will the duct of Santorini act as a safety valve if the duct of Wirsung is obstructed, for, although in 63 per cent. of cases the duct opens at the same time into the main channel and into the intestine, yet in probably less than half of these is the anastomosis efficient as a through channel.

The reasons why gall-stones in the common bile duct do not always produce pancreatic inflammation are:

1. Some gall-stones are so large that they never reach the pancreatic portion of the duct, but remain in the supra-duodenal portions of the common duct, producing jaundice, but no pancreatitis. The following is an example:

Mr. S., aged sixty-five, had for two years been subject to occasional attacks of epigastric pain. In January, 1903, a severe attack was followed by jaundice, since which time he had rapidly lost weight, and the jaundice had never disappeared. Pain after food had been a marked feature. He had neither vomited blood nor had malena. There was no dilatation of the stomach, and no evidence of tumor. The recti were rigid. He was seen by a well-known physician, who diagnosed cancer of