- 37. Mozes, M. and H. Bogokowsky: Les Cystadénocarcinomes Papillaires du Pancréas. Lyon Chir., **59**:499, 1963.
- Murray, J. B. and J. J. Spier: Cystadenoma of the Pancreas. Amer. J. Surg., 101:500, 1961.
 Patel, J. and J. Naulleau: Une Observation
- D'épithélioma Kystique du Pancréas. Ann.
- Anat. Path., 12:175, 1935. 40. Peltokallio, P.: Cystadenoma of the Pancreas. Ann. Chir. Gynaec. Fenn., 52:74, 1963.
- 41. Piper, C. E., W. H. ReMine and J. T. Priestley: Pancreatic Cystadenomata. J.A.M.A., 180:648, 1962.
- 42. Probstein, J. G. and H. T. Blumenthal: Progressive Malignant Degeneration of a Cystadenoma of the Pancreas. Arch. Surg., 81: 683, 1960.
- 43. Pyrah, L. N. and J. W. Cowie: Two Unusual Aortograms. J. Fac. Radiol., 8:416, 1957.
- 44. Rey, A. M. and A. S. Introzzi: Cistadenoma de Pancreas; Pancreatectomia Caudal, Esplenectomia y Colectomia Segmentaria; Curacion. Bol. Acad. Argent. Chir., 35:635, 1951.
- 45. Rosenbaum, H., P. J. Connolly and A. R. W. Climie: Pancreatic Cystadenoma with Intestinal Hemorrhage. Amer. J. Roentgenol., **90**:735, 1963.
- Rosman, N. P.: A Case for Diagnosis. McGill Med. J., 27:169, 1958.
 Rowe, P. G.: Papillary Cystadenocarcinoma of the Pancreas. Canad. Med. Ass. J., 74:724, 1956.
- 48. Rutledge, R. and C. E. Lischer: Papillary Cystadenocarcinoma of the Pancreas. Texas J. Med., **54**:89, 1958.
- Saphir, O.: A Text on Systemic Pathology, Vol. II. New York, Grune and Stratton, 1959. p. 1387.

- Sawyer, K. C., J. R. Spencer and A. E. Lubchenco: Proliferative Cysts of the Pan-creas. Ann. Surg., 135:549, 1952.
- 51. Scalvini, L.: A Rare Tumor of the Pancreas: Cystocarcinoma. Minerva Chir., 15:1207, 1960.
- Shulman, A. G., R. W. Lippman and W. Miller: Internal Drainage of Malignant Pancreatic Cysts as an Effective Palliative Procedure. Amer. J. Surg., 102:470, 1961.
 53. Sommers, S. C. and W. A. Meissner: Unusual
- Carcinomas of the Pancreas. Arch. Path., **58**:101, 1954.
- 54. Swanson, G. E.: A Case of Cystadenoma of the Pancreas Studied by Selective Angiography. Radiology, 81:592, 1963.
- Tagariello, P.: L'adenoma Cistico del Pan-creas. Arch. Ital. Mal. Appar. Dig., 17:325, 1951.
- 56. Trapnell, D. H.: Cystadenoma of the Pancreas. Brit. J. Surg., 41:574, 1954. 57. Trasino, M. and C. Laudenzi: Contributo allo
- Studio del Cistoadenoma Pancreatico. Minerva Gastroenterol., 9:83, 1963.
- 58. Vernhet, J.: Cystadenocarcinome Pancreatique. Mem. Acad. Chir. (Paris), 89:821, Nov.,
- Warren, K. W., W. M. McDonald and M. C. Veidenheimer: Trends in Pancreatic Surgery. Surg. Clin. N. Amer., 44:743, 1964.
- 60. Willis, R. A.: Pathology of Tumours, 2 ed. St. Louis, C. V. Mosby Co., 1953. p. 710.
 61. Young, E. L., Jr.: Pancreatic Cyst. New Engl. J. Med., 216:334, 1937.
- 62. Zintel, H. A., H. T. Enterline and J. E. Rhoads: Benign Cystadenoma of Pancreas: Report of Four Treated Cases, One by Whipple Type of Resection. Surgery, 35: 612, 1954.

DISCUSSION

DR. KENNETH WARREN (Boston): I think Dr. Priestley has pointed out all the important features of the far advanced chronic relapse in pancreatitis, and he has especially emphasized that there is no single operation to accommodate all of these people. Regardless of how you individualize these operations, the long-term results will probably still leave much to be desired.

(Slide) For many years we have been interested in the direct approach, such as Dr. Priestley described, to the treatment of chronic relapse in pancreatitis, believing that all of these patients have partially complete obstruction of the duct of Wirsung. This is the type of procedure which I assume he had in mind. It shows the sacculation. This is a specimen (slide) removed 18 months later from a patient who had a lateral anastamosis and then developed an obstruction in the distal part of the gland with recurrent symp-

(Slide) I would like to emphasize that patients may have spontaneous pancreatitis confined to the

left segment of the pancreas. Here you see a simple stone to the left of the vertebral column, many satellite stones distal to that, and in the next specimen (slide) you will see the pathology represented by distal pancreatectomy. One might assume from this roentgenogram that this patient has the distal part of the pancreas involved in the far advanced pancreatitis, while the head and neck would be normal.

The cavity you see to your left, near the center of the screen, is the cavity from which the simple stone was removed. There are innumerable stones in the duct beyond that. The duct has been almost destroyed, and there was a spontaneous fistula. This is the type procedure we think indicated in distal pancreatitis of this nature.

We do not believe that internal drainage has a place (and I am sure most of us would agree the same applies to the situation here) in the patient with almost total destruction of the distal part of the pancreas, having had numerous previous attempts to drain the cyst.

(Slide) I put this on the screen to show something Dr. Priestley has not discussed, but it certainly is pertinent to the problem. This is even more far advanced chronic pancreatitis with obstruction of the proximal portion of the pancreatic duct, with cystic formation, and the accumulation of stones. In these particular individuals we think the resection of the head of the pancreas is indicated. We do this with some reluctance, and certainly never in early pancreatitis.

(Slide) A specimen removed from a total pancreatectomy, where the gland is destroyed with stones; you see the innumerable septa there, multiple points of destruction which render this gland certainly unsusceptible to this type of repair.

I think this was an excellent presentation by Dr. Priestley, and that direct attempts to relieve obstruction may be the key.

In discussing Dr. Becker's presentation (he is an old friend and has already told me what to say) I hope I remember what he has told me. Actually, we agree so much on this subject. The important thing is that these patients should be explored when they have a history suggestive of pancreatic disease and x-ray evidence of pancreatic disease, despite what may appear to be a hopeless tumor. At operation they may have a tremendous tumor, and if there is no evidence of metastasis or invasion of vital structures, one should be extremely aggressive in removing such tumors.

(Slide) This is a large cyst, having been drained four years prior to the distal pancreatectomy, and involving the distal part of the pancreas. The patient with long-standing symptoms may still have a resectable tumor very likely to be a cystadenoma or a cystadenocarcinoma. At the bottom of the screen you see the malignant portion of this tumor, which was removed about 8 years ago, and the patient is completely well.

(Slide) Here is a large cystadenoma involving the head of the pancreas, removed by the Whipple procedure. I think some of the differences of opinion about this relate to the interpretation the pathologist gives. In a tumor this size one would suspect—if a sufficient number of slides were made—that malignant elements might be found.

(Slide) A more typical example of cystadenocarcinoma (is shown) where the tumor is essentially solid, having smaller cystic elements, and again removed by Whipple procedure. This operation was done about six years ago, and the patient is doing well.

(Slide) Finally, a total pancreatectomy done for an extremely large cystadenocarcinoma in a male. This was done about 11 or 13 years ago by the late Dr. Cappel. You will see how this man's nutrition has been maintained and that he has a jejunal ulcer, but aside from that has had no evidence of recurrence.

DR. ROBERT J. COFFEY (Washington, D. C.): Dr. Priestly's excellent paper provides encouragement for those interested in treating this extremely difficult condition. I would like to sound one note of warning concerning those patients with chronic pancreatitis who are chronic alco-

holics. It has been my experience that any type of surgical treatment is doomed to failure if the alcoholism continues. Conversely, if one can by some means convince the victim to refrain from the use of alcohol, surgical intervention may be avoided not infrequently.

On analyzing our cases of chronic pancreatitis it was clearly evident that the best results of surgical treatment were those in whom associated calculous disease of the biliary tract was eliminated. Furthermore, in our cases of calcific pancreatitis, surgical treatment of those with intraductal calculi of an obstructing character was very satisfying. The results following sphincterotomy have been anything but favorable. And finally, in four cases in which hyperparathyroidism and chronic pancreatitis co-existed, removal of the primary parathyroid pathology was followed, in our opinion, by a favorable course of the pancreatitis.

DR. KEITH S. GRIMSON (Durham): It has been my privilege to try two new treatments for pancreatitis. One, was an operation, ganglionectomy in 1945, the other a drug, Banthine, in 1949. Fortunately, Banthine has been accepted as an aid in the treatment of pancreatitis. Nobody has extensively tried the operation.

In 1945, amylase levels were not routinely determined in patients with chronic pancreatitis. Nevertheless, we did have a group of patients with a good diagnosis. One had diabetes, three calcification. One was a child, his father and a brother had recurrent episodes. Each patient at operation had an indurated pancreas; each had consistent recurring episodes of pain.

Results were reported in 1947 and 1950. The operation was bilateral celiac and superior mesenteric ganglionectomy. The rationale, you may question. What was done to relieve the dilated ducts, small cysts, calculi and extensive inflammatory pathology? Is this the cause of the pancreatitis or the result of it? May these results of repeated inflammation be left alone, would the vagotomy and sympathectomy of the pancreas effected by ganglionectomy protect the gland, remove it as a target organ from some nervous influence, put it at rest and reduce inflammation? The answer seems to be yes for the ganglion ectomy as evidenced by follow-up observations.

Much is known about the hormonal influences that apply to the pancreas, effects of alcohol, etc. Obviously these are present after pancreatic denervation. It is known that the pancreas is an organ which is under the influence of the autonomic nervous system. It was on this basis and not on the basis of sensory denervation that celiac and superior mesentery ganglionectomy was originally tried.

I noticed in one of the slides that the discussor had two of four successes with celiac ganglionectomy. Denervation must be complete to effect consistent results. Operative mortality in my group was nil, and it seems encouraging that none of the original 13 patients is troubling me today.

DR. WILLIAM S. McCune (Washington, D. C.): I would like to say, first, a word about Dr. Becker's paper. I think it is extremely important when anyone encounters a cystic lesion of the pancreas, that—before he attempts some sort of internal drainage—he get a thorough biopsy to be sure he is not dealing with carcinoma. I know of one patient who had internal drainage of a cystic lesion which turned out to be carcinoma, perhaps one that might have been removed.

Particularly, I would like to urge you to read again Dr. Priestley's fine discussion of the question of chronic relapsing pancreatitis. This disease, I think, is still one of the challenges of abdominal surgery. Our results are good in some cases but we have many failures. Of course, as has already been expressed, chronic pancreatitis, in general, is due to two different causes: First of all, gallstones as distinct from noncalculus gallbladder disease. The treatment of this type of pancreatitis is relatively simple—usually removal of the gallbladder and stones with or without drainage of the common duct is successful. The second cause, of course, is alcoholism, but of chronic relapsing pancreatitis is much more difficult.

In regard to sphincterotomy, which had a phase of popularity, Dr. Thistlethwaite reported 30 patients in whom he had done a sphincterotomy because of alcoholic relapsing pancreatitis. He studied these by leaving a polyethylene tube in the pancreatic duct and in the common duct after doing the sphincterotomy. Much to his surprise and dismay, he found that within 2 or 3 weeks the pressures had returned to normal and the effect of morphine on the sphincter had also returned to normal.

We have in one case, a very recalcitrant patient upon whom we had operated several times, we performed a total pancreatectomy for chronic relapsing pancreatitis with calcification, and with very good results.

I believe, and we all do at our hospital, that the method of caudal drainage with or without slitting up the pancreatic duct (in the method described by Drs. Puestow and Priestley) is most effective, and I think in many cases it produces satisfactory results. However, we have also had a number of failures. I should say that our good results do not constitute more than 65% of cases as a result of this type of drainage, particularly in patients with calcification, steatorrhea, so-called cirrhosis of the pancreas and diabetes.

The question which I should like to ask Dr. Priestley, after this long preamble, is if he thinks that in a patient who has had a satisfactory drainage procedure for relapsing pancreatitis without success, is there any place for total pancreatectomy?

DR. WILLIAM H. REMINE (Rochester, Minn.): Dr. Becker very kindly invited me to discuss his paper, and he generously allowed me an opportunity to look over the manuscript. I have been interested in pancreatic diseases for a number of

years, and recently I had the opportunity to review our experience in these two fields—cystadenomas and cystadenocarcinomas—or the neoplastic cystic lesions of the pancreas.

I would like just briefly to show the results of our studies in support, really, of what Dr. Becker has already said. In reviewing all our cystic lesions of the pancreas (slide), we find this distribution: 242 pseudocysts, 36 cystadenocarcinomas, and 20 cystadenomas. Not all of the cystadenocarcinomas were satisfactory for study, because there was not enough tissue available for pathologic examination, but for those that were available, I will give you the results in a moment.

(Slide) Let me consider the cystadenomas first. I think the most important factor revealed by this slide is the high incidence of abdominal pain of an aching nature and of distress in the upper abdomen associated with loss of weight. Other problems may be associated, but these may at times be difficult to separate from functional disorders that the patient may have or from such factors as narcotic addiction and alcoholism that we often encounter.

(Slide) Here we see the vast variation in the size of these lesions, ranging from less than 5 cm. all the way up to more than 20 cm. in diameter.

The location of the lesions, as Dr. Becker described, is mainly in the body and tail, but may be found anywhere along the entire length of the pancreas; and as you note in the next-to-last line, there may also be multiple lesions, as we found in one case.

(Slide) Here we have a résumé of our study of cystadenomas, showing the procedures that were performed. Just as Dr. Becker demonstrated, marsupialization definitely is not the operation of choice. Excision of a wide rim of pancreatic tissue around the lesion should be performed if possible. The last line shows a case of ours in which the lesion was so large, and involved so many structures, that only biopsy was done. Because of jaundice and duodenal obstruction, an anastomosis was made to bypass the biliary tract, and a gastroenterostomy was also performed to bypass the duodenum on the opposite side. The results of excision of this type of lesion have been outstandingly good.

(Slide) Just a brief comment about cystadenocarcinomas: The location, the symptoms, and many of the other findings that are associated with cystadenomas are essentially those also of cystadenocarcinomas. Here we see one that we had the opportunity of removing in 1962. This lesion was producing a large mass in the upper part of the abdomen, and it was this finding that precipitated the procedure. When one operates on cystic lesions of the pancreas in the absence of findings of alcoholism, there are no signs or symptoms of pancreatitis ,and one is not able to elicit any laboratory evidence by some of the more sophisticated laboratory studies that we now have, one should first of all think of a neoplastic lesion when a cyst is encountered at laparotomy. In our experience, from the results of the procedure, if the lesion can be excised with a generous portion of the pancreas, there has been a 5-year survival rate of about 63 per cent.

DR. HAMP PRATT (New Orleans): We are impressed that there are two definite categories of pancreatic cystadenomas pathologically, which have very different prognostic implications.

(Slide) This first category of cystadenoma is the highly characteristic, classic type, usually described in text books, which we have termed the centro-acinar type. It is made up of multiple small cysts lying in a variable amount of interstitial fibrous connective tissue stroma.

(Slide) Microscopically these cystic spaces are lined with highly characteristic cuboidal or low cuboidal epithelial cells resembling those of the terminal pancreatic ducts of centro-acinar cells. This form constitutes 45% of our series and is easily recognizable, having an identical gross and histologic appearance from patient to patient.

The other category of cystadenomas is a heterogenous one of varying histogenic types, all lined by different forms of tall columnar cells. (Slide) This is a photograph of one such cyst which has been opened, showing several daughter cysts on the wall. (Slide) The lining is composed of tall columnar cells, which appear to be derived from large pancreatic ducts. In other cases they seem to be derived from intestinal-like epithelium or may resemble that seen in cystadenoma of the ovary.

It is important to distinguish the centro-acinar type from the heterogenous tall columnar group, for we have found no evidence of the centro-acinar ever having malignant potential. In fact one of our cases of this type was biopsied twice at a six-year interval and showed no evidence of malignant progression.

Contrariwise, the heterogenous group is implicated as a possible origin of cystadenocarcinoma. In our cases of cystadenocarcinoma, and in most reported cases, there were residual benign areas, lined by tall columnar-type epithelium, present in the cyst wall.

These impressions are clinically important to the surgeon dealing with a cystadenoma in the heterogenous group. He may believe he is compelled to treat it more aggressively as a pre-malignant lesion, when a more conservative approach may be acceptable in dealing with the centroacinar type in a poor-risk patient.

DR. JAMES T. PRIESTLEY (closing): I am sure there are, as far as I am concerned anyway, more questions than there are answers about pancreatitis. The question that Dr. McCune raised is certainly an interesting one. An unfavorable result after one or more conservative types of operation on the patient with chronic pancreatitis certainly raises the question of total pancreatectomy. This of course is an extensive and difficult operation under

these circumstances and involves a significant operative risk. Not many such cases have been reported in the literature, and, unfortunately, those which have been reported have not always been followed by favorable long range results. Personally, I am inclined to think that total pancreatectomy has a limited place in the treatment of chronic pancreatitis.

Our own experience in efforts to relieve pain due to pancreatitis by various neurosurgical procedures has not been very rewarding. Some patients have been partially or completely relieved for a time, but then the pain has returned. I suppose it is possible that we have not employed the most effective neurosurgical procedure.

I certainly agree with Dr. Coffey about the use of alcohol and the remarks made about the biliary tract. I think Dr. Warren made a very important point, and that is that not all of these patients have exactly the same type of lesion, so one must select the procedure which, in his opinion, seems best suited to the individual patient.

Among the many questions regarding chronic pancreatitis there are two which I would like to leave with you for your further consideration. One is: how sure can we be when we see a patient with chronic relapsing pancreatitis and there is obstruction in the pancreatic duct (in the absence of calculi) that this obstruction is cause rather than effect? Further, how much drainage do we really give the pancreas when we drain the main pancreatic duct into the intestine? It strikes me as being something like draining the trunk of a tree. There may be obstruction in the branches, and it seems to me, therefore, that we are really not sure how effectively the entire pancreas is drained when we only drain the main pancreatic duct into the jejunum.

One hears so often and sees laboratory evidence that reflux of bile in the pancreatic duct can result in pancreatitis, and I think the laboratory evidence is irrefutable. I do not think I can recall one patient in whom I have opened the pancreatic duct to find bile there. Juice was always clear or a little turbid—but not green. If regurgitation of bile to the pancreatic duct is important in the development of pancreatitis in these patients, why do we not see bile in the pancreatic duct more often?

DR. WALTER BECKER (closing): The vast majority of pancreatic cysts are either post inflammatory or post traumatic pseudocysts. Ordinarily the diagnosis is not difficult, and usually the treatment of choice is some type of drainage procedure. The principal purpose of our paper is to emphasize that about 10% of pancreatic cysts are either cystadenomas or cystadenocarcinomas, and that they require total excision for cure. The surgeon must be capable of recognizing these neoplastic cysts at laparotomy, and he must be willing to make a determined effort to excise them.