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#### DISCUSSION

DR. ROBERT ZEPPA (Miami, Florida): Dr. Polk, Dr. Jones, first I would like to thank Dr. Townsend for the honor of his request to discuss this paper, especially because we have no scientific data in support of this, but merely anecdotal information that does lead us to be in almost complete agreement.

The problem of pseudocyst, as is indicated in their paper, suggests that ultrasound and CT scans were useful in identifying the correct differential diagnosis between acute pancreatitis and chronic pancreatitis with ductal dilatation.

On the basis of your studies, how many of the patients had already been identified as having dilated ducts before ERCP was undertaken?

Second, were any of the patients drained percutaneously in this series? That was notably missing in the manuscript, and I must say that in our institution, most pancreatic pseudocysts are drained percutaneously and quite satisfactorily. The result is that we are doing fewer operative procedures.

The problem of recurrence in this population of patients needs to be addressed. I don't know how it is in Galveston, but I can tell you that in Miami those patients who have pancreatic disease secondary to alcohol abuse and who develop pseudocysts are extraordinarily difficult to find in terms of the follow-up, so I don't know what the recurrence rate for our group is with the percutaneous drainage. But so far, we have not identified more than a small handful of patients who have come back and those have been fairly soon after the percutaneous drainage.

I suspect that Courtney asked us to comment on this because some years ago Dr. Duane Hudson and I had presented a paper before the Southern on the operation drainage of pseudocysts, particularly cystogastrostomy. We presented a modest series of patients with cystogastrostomies from which data we tried to influence the Association into believing that when you have sutured the edges of the cystogastrostomy the anastomosis will be incompetent, and that the bleeding would occur from the pancreatic side because of the reflux of acid, something that was determined by Dr. Warren years before in some elegant studies he did while he was at Virginia.

I would like to close this brief discussion by apologizing to Jim Thompson for not providing rapt adulation for the paper, but we do not do ERCP on all patients, but we do believe that it is an extraordinarily useful procedure in patients who have complex cysts where you can't quite identify a track for simple percutaneous drainage.

DR. GEORGE L. JORDAN, JR. (Houston, Texas): Dr. Polk, Dr. Scott: I also would like to thank Dr. Townsend for the privilege of reviewing his manuscript that includes the report of a very interesting group of patients.

He is certainly to be congratulated on his low rates of morbidity and mortality in what often is a very complex group of individuals.

If I understand his manuscript correctly, all but three of his patients had alcoholic pancreatitis, and, therefore, the importance of ERCP becomes immediately important in that particular group.

Many other series have many patients who have pseudocysts following

acute attack of pancreatitis due to gallstones. They almost never have ductal abnormalities, and in that group ERCP is much less important. Consequently, we would use a selective approach rather than routine use of ERCP because we think that there are groups of patients in whom the probability of ductal abnormalities is so low that ERCP is not likely to be of benefit.

Another interesting fact, to me, in his paper is the small size of the pseudocysts. Back before we had ultrasound and CAT scans and ERCPs, we didn't diagnose pseudocysts unless we could palpate them and see them on displacement of the stomach in an upper GI series, so we were dealing with an entirely different group of people at that time. They were large pseudocysts, and when we have a pseudocyst that fills the entire lesser sac, I am not sure how I would do a Puestow procedure and drain that pseudocyst at the same time. Again, I feel that there needs to be some selection.

I would like to ask about the pathology because some of the slides that I saw suggested that some were true cysts, intrapancreatic cysts rather than what I call a pseudocyst, which is usually external to the pancreas per se.

The last thing I would like to comment on is the very high incidence of common-duct obstruction. That has certainly not been my experience. Certainly stricture of the common duct occurs in chronic pancreatitis, and I have combined the Puestow procedure with common-duct drainage in many instances, but once one relieves the pseudocyst and drains the pancreatic duct, the likelihood of progression to a severe-enough stricture for which subsequent reoperation is necessary or from which permanent damage to the liver occurs is very, very small in my experience.

Consequently, I would like to ask what their follow-up data are in patients before they did this study, which would indicate the incidence of hepatic damage in those who did not have common-duct drainage—patients who were not jaundiced—because that is the group I am talking about, and, again my question about pathology.

DR. JOAQUIN S. ALDRETE (Birmingham, Alabama): I congratulate Drs. Nealon, Townsend, and Thompson for their carefully planned, skillfully executed, and elegantly presented analysis of the usefulness of ERCP in the operative treatment of patients with pseudocysts of the pancreas. Their contribution is important to the field of pancreatic surgery because it clarifies several aspects of pancreatic pseudocysts that have great relevance to their appropriate operative treatment. I have a few comments and some questions that I hope will be pertinent.

Because of the use of ERCP, the authors modified their categorization of acute to chronic pancreatitis in nine of 24 patients (38%); however, the criteria they used for the pre-ERCP categorization into chronic pancreatitis was the presence of pancreatic calcification, steatorrhea, or glucose intolerance. We know that some patients with chronic pancreatitis do not have any of these three manifestations. In fact, in a group of nearly 150 cases of chronic pancreatitis that we have analyzed over the last few years, we found that 16% of them do not have pain at all, 20% never drank alcohol, and many do not have dilated pancreatic ducts as assessed by ERCP or CT. At any rate, the authors established that 63% of their patients treated for PPSC had chronic pancreatitis and 37%

had acute pancreatitis. This is important because, although in the past most analyses of pancreatic pseudocysts have been without differentiation as to whether they had chronic or acute pancreatitis, I suspect that if we were to elucidate one day how these cysts develop, we would find that their respective pathogenesis is very different from each other. My first question is, did the endoscopic criteria used for CP correlate with your operative findings?

Another comment pertains to the finding that in over one half of the patients studied, the ERCP did not show a communication between the cyst and the MPD. If one thinks carefully how a pancreatic pseudocyst is formed, one has to suspect that at one time or another in all pseudocysts there was a communication with the MPD, but as the cyst becomes more chronic this communication closes and therefore cannot be demonstrated by ERCP. My second question is, do you agree with this hypothesis or, why do you think in over one half of your cases the ERCP did not show a communication between the cyst and the MPD?

A third comment relates to the size of the pancreatic pseudocysts that were treated by operation. The mean diameters were 6.6 cm  $\pm$  2.1 cm for CP and 5.1 cm  $\pm$  for acute pancreatitis. I wonder if some of the pseudocysts studied that measured less than 5 cm in diameter, if followed for longer periods of time, would have disappeared spontaneously as was the case in three patients are noted by the authors in their manuscript. I postulate this premise because I have suspected for some time that the majority of the pancreatic pseudocysts that measure less than 4 cm or 5 cms in diameter disappear without operation. Two years ago we analyzed 75 patients seen in our institution with pancreatic pseudocysts; one half of them measured less than 5 cm in diameter, and all but one disappeared spontaneously. So, my third question is, exactly how many of your cases measured less than 5 cms in diameter, and would you be willing to observe these patients with pancreatic pseudocysts that are less than 5 cms in diameter for a longer period of time?

My fourth and last comment relates to the usefulness of CT primarily to evaluate the status of the MPD in patients with CP. It is my impression that in most cases of pancreatic pseudocyst the information that you need for the proper decisions regarding surgical treatment can be made with the use of CT and US that are less invasive than ERCP.

A final question is did you correlate your ERCP findings with the CT and US findings in these patients? I agree that a prospective study using ERCP in all cases was indicated, but with the information obtained from your excellent study, could we look for the same information in less invasive studies like the CT and US?

Their paper greatly contributes to heightening our awareness that obstruction of the MPD and the CBD often coexists with the presence of a pancreatic pseudocyst, and that the preoperative assessment of these two ductal structures allows a more effective operative treatment of these complications of chronic and acute pancreatitis. I wish to express to the authors my gratitude for inviting me to discuss their work, and for providing me with the manuscript ahead of time. I am sure we all learned from this work.

DR. ROBERT E. HERMANN (Cleveland, Ohio): Dr. Polk, Dr. Jones, Members of the Association: I would first like to take this opportunity to thank Dr. Bob Sparkman and the Southern Surgical Association for the opportunity to be here as a guest of the Association at this meeting and to have the opportunity to discuss this excellent paper by Drs. Townsend, Thompson, and Nealon.

The controversy as to the role of ERCP in preoperative decision making for the treatment of pancreatic pseudocyst is one that concerns all of us who treat these patients. And as you can well see, the controversy goes on.

My own experience agrees with that of some of the other discussants: I use it selectively. I use it most often in patients with a history of chronic pancreatitis who develop pseudocysts and rarely in patients with a pseudocyst after acute pancreatitis.

I think the patient with chronic pancreatitis is more likely to have fixed ductal changes in both the pancreatic duct system and in the biliary system than is the patient who has acute pancreatitis.

Secondly, I would use it more often if there is more than one pseudocyst on a CT scan, or if I see a dilated duct system on CT scan, or in the patient who has small pseudocysts rather than large ones as has been mentioned previously.

I use it rarely or never when a patient has a single large pseudocyst, the old-fashioned kind that we used to see so frequently.

Finally, I would use it if I wanted primarily biliary information, as in the patient with a dilated bile duct on a CT scan or an elevated bilirubin or alkaline phosphatase level.

To reiterate then, my own indications would be: the patient with chronic pancreatitis, small pseudocysts, multiple pseudocysts on CT scan, or evidence of biliary disease.

Finally, I would like to add that you can do an intraoperative pancreatogram study frequently through the pseudocyst if you find chronic pancreatitis or fibrosis of the gland. You can either inject the cyst directly and get a picture, and this will sometimes show you the pancreatic duct system, or occasionally you can do an intraoperative pancreatogram if the pancreatic duct is dilated and you have small pseudocysts. I have no questions, but I wonder if Dr. Townsend would care to comment on these indications in light of their study.

I enjoyed this paper very much.

DR. HUNTER H. MCGUIRE, JR. (Richmond, Virginia): Preoperative ERCP is clearly needed for pseudocysts that are recurrent or complicated by jaundice or ascites, but I have until now thought that uncomplicated new cysts in patients with chronic pancreatitis were so quickly, easily, and safely cured by cyst-enterostomies that when I read Dr. Townsend's abstract, I felt he was overstudying and overtreating these patients.

So I looked up results in our Veterans Hospital of the last 36 cyst-enterostomies that I have been able to follow for 6 months to 11 years (mean 3.3 years), and I was surprised to find that our outcomes seemed better when ERCP altered operations.

In all five cases in which ERCP altered operations, patients are so far cured. In four cases in which ERCP was done but did not alter operation, only two are cured. In 27 patients with no preoperative ERCP, only 18 were cured: 9 have had subsequent problems and 4 needed reoperation within a year.

These results seem to support Dr. Townsend's conclusions, but in spite of them I still think that ERCP should be reserved for cysts that are recurrent or complicated. Most uncomplicated chronic pseudocysts are in uninsured patients whose subsequent problems are governed not by surgical techniques but by alcohol, and whose immediate problems can be instantly solved by simple operations that will have them home in a week. I doubt if we should prolong their stays and add to their debts with ERCP and duct-enterostomies for anomalies that can't be seen on ultrasound or CT scan, or for which there is no clinical evidence.

The real question is not whether ERCP can alter operations, but whether the altered operations will make patients better. To prove that patients will be better, we need larger numbers and longer follow-up than either I or Dr. Townsend have been able to show you.

DR. W. H. NEALON (Closing discussion): Dr. Polk, Dr. Jones, Members, and Guests: I would like to thank all the discussants for their kind words and interesting questions.

I will start with Dr. Zeppa. The CT scan will not provide identical information to that obtained from ERCP. The yield is not great, in our hands, for CT scan telling us even the size of the pancreatic duct. Certainly, the nuances of the actual course of the pancreatic duct cannot be displayed by CT scan, and stones within the duct or communication between the duct and the pseudocyst cannot be delineated.

Dr. Zeppa, you mentioned percutaneous drainage of pseudocysts, and that is actually one of the issues that prompted this study. The interventional radiologist who performs simple percutaneous drainage is likely to totally ignore any associated abnormalities in the pancreas when he is decompressing the pseudocyst. One of our pleas with this study is to urge a thorough evaluation of the pancreas and the biliary tree prior to any measure aimed at draining the pseudocyst.

We have speculated that perhaps noncommunicating pseudocysts in an acute pancreatitis patient would be well served by percutaneous drain. The recurrence rates have been quite high in percutaneously drained patients.

We acknowledge that our follow-up is relatively short, but we, thus far, have had no recurrences of pseudocysts. It is quite clear that to evaluate the impact of duct decompression on the recurrence rates of

pseudocysts in patients with chronic pancreatitis requires a longer follow-up period.

Dr. Jordan observed the preponderance of ethanol-induced chronic pancreatitis and acute pancreatitis in our study. Chronic pancreatitis is rarely caused by biliary disease, but, even in our institution, biliary disease is the most common cause of acute pancreatitis. We have speculated on a different frequency of pseudocyst formation in biliary-induced compared to ethanol-induced acute pancreatitis. You are correct in suggesting that gallstone pancreatitis patients are not likely to have unsuspected chronic pancreatitis. You might forego ERCP in patients whose ethanol history is reliably noncontributory.

The one observation provided by ERCP that might assist you, even in the gallstone pancreatitis patients, is communication between the pseudocyst and the duct. As I have mentioned, noncommunicating pseudocysts may be the best candidates for percutaneous drainage.

You mentioned pseudocyst size, and past studies, when diagnosis depended on palpation of the pseudocyst, had included large pseudocysts only. In more recent studies the average sizes of even 4 cm have been reported, and our mean diameter of 6 cm is consistent with recent reports. I do agree with you that a very large cyst would be technically challenging. We would still advise evaluating the pancreatic duct, but combined drainage of the pseudocyst and the duct may not be possible. One thought might be to shave away some of the cyst, and even a partial incision into the duct, through the pseudocyst, might be possible.

We have pathologic evaluations of the majority of these pseudocysts, primarily to rule out any cystadenomas or cystadenocarcinomas. They have all been pseudocysts. We have had no carcinomas in this group and we have had no true cysts in this group.

The frequency of biliary drainage, as you suggested, is relatively high, although our incidence is almost precisely that reported by Prinz and

his group from Chicago in their 1986 series of chronic pancreatitis patients. We do not have preoperative evaluation of liver histology. There are a number of studies suggesting that there is a risk of progression to primary biliary cirrhosis in patients with long-standing subacute stenosis of the biliary tree.

We are currently taking liver biopsies on our patients and we plan to follow our patients who have been so treated, but we don't have any data of our own to tell you that we are avoiding this outcome.

Dr. Aldrete, you mentioned some of your patients who had no ductal changes. We would say that recurrent attacks of acute pancreatitis are not the same as chronic pancreatitis, and for our diagnosis of chronic pancreatitis we depend on the findings of ductal changes, functional impairment, or gland calcification to document that diagnosis. Our operative findings were consistent with ERCP findings in all patients. Our frequency of pseudocyst communication is similar to other studies. The mechanism of obliteration of the communication has not been defined, but this is a nonanatomic, nonepithelialized tract and spontaneous closure is not unexpected.

Dr. Hermann, you described selective ERCP and your system is very sound. Once again, I would simply mention the usefulness of recognizing communication between the cyst and the duct. Clearly, someone with your experience in surgery of the pancreas needs no prompting to consider unsuspected pancreatic duct abnormalities. If others choose to perform ERCP, I think you are going to find a patient with a remarkably dilated pancreatic duct that you had not suspected. Our data suggest that it is not possible to predict, on the basis of other clinical findings, that certain patients do or do not have chronic pancreatitis.

Finally, Dr. McGuire mentioned the length of hospital stay. We have not prolonged our patients' hospital stay in any way. Their average stay is between 5 and 7 days as well.