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# Management of Infected Pancreatic Necrosis by Open Drainage

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Twenty-eight consecutive patients with infected pancreatic necrosis were managed by extensive unroofing of the superior retroperitoneum, blunt pancreatic sequestrectomy, laparotomy pad packing of the lesser sac over a layer of *Adaptic*<sup>®</sup> gauze, and scheduled re-explorations at intervals of 2–3 days (open drainage). Wounds were permitted to heal by secondary intention. All patients were maintained on intravenous hyperalimentation. Three of the 28 patients died (11%); none died of sepsis. Procedure-specific complications included: pancreatic fistula (10 patients), incisional hernia (8 patients), persistent functional gastric outlet obstruction (2 patients), retroperitoneal venous hemorrhage (2 patients), and intestinal fistula (1 patient). Limited initial experience with dynamic pancreatography and serial monitoring of acute phase reactants as indicators of pancreatic necrosis is promising. Compared with historic controls, open drainage of infected pancreatic necrosis represents a significant advance over more conventional surgical approaches. Controlled studies and more widespread experience are necessary for further evaluation of this procedure.

**D**ESPITE SIGNIFICANT recent advances in supportive therapy and intensive care, the mortality rate from acute pancreatitis has remained constant for more than 40 years.<sup>1–3</sup> Today, few patients die of the well-recognized consequences of hypovolemia induced by acute pancreatitis. Rather, the development of sepsis has emerged as the principal determinant for survival in patient with acute pancreatitis surviving longer than 7 days.<sup>2–4</sup> In this regard, it would seem that our previous efforts to reduce mortality in acute pancreatitis have served to change the mode of death from fluid deficit to septicemia.

Pancreatic abscess is a particular case in point. In 1963, Altemeier and Alexander recommended anterior celiotomy, identification and debridement of necrosis,

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and closed drainage for patients with pancreatic abscess.<sup>5</sup> In the 25 years that this conventional surgical approach to pancreatic sepsis has been used, mortality rates have remained unacceptably high, ranging from 30–60%.<sup>6–11</sup> Furthermore, as many as three fourths of the deaths *after* conventional surgical debridement and drainage for pancreatic abscess have been due to sepsis.<sup>12</sup> These observations cast serious doubt on the efficacy of the conventional surgical approach to the management of these patients. It is becoming increasingly clear that if we are to reduce mortality in acute pancreatitis, additional approaches to the various forms of pancreatic sepsis must be sought.

Recently, infected pancreatic necrosis has become recognized as the most severe form of pancreatic sepsis, with surgical mortality rates greater than twice those of classical pancreatic abscess.<sup>13</sup> The current report details a 10-year personal experience using an alternative surgical procedure (open drainage) in the management of these difficult patients with infected pancreatic necrosis.

## Patients and Methods

The patient population consists of 28 consecutive patients with surgically proved infected pancreatic necrosis who were seen at Grady Memorial Hospital and the Atlanta Veterans Administration Hospital during 1976–1986. Previous communications have described our earlier experiences with these patients.<sup>14,15</sup> Each patient had an antecedent episode of *severe* acute pancreatitis as stratified by two independent clinical classification systems (Table 1). The anticipated overall mortality rate for acute pancreatitis of this severity exceeds 50% in both systems.<sup>9,16</sup>

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The cause of the acute pancreatitis was believed to be alcohol in 19 patients, gallstones in three patients, Type IV hyperlipidemia in two patients, hyperparathyroidism in one patient, and three cases of acute pancreatitis were classified as idiopathic. There were 20 men and eight women with an average age of 46.1 years (range: 36–85 years).

In the initial phase of the study, the diagnosis of infected pancreatic necrosis was primarily one of suspicion; *i.e.*, progressive deterioration in a septic patient with antecedent pancreatitis. Later, computed tomography (CT) became of considerable assistance to the clinical diagnosis. Most recently, bolus contrast enhanced CT scans (dynamic pancreatography), guided needle aspiration bacteriology, and serial serum monitoring of acute phase reactants have materially assisted the clinical diagnosis of infected pancreatic necrosis. Bolus contrast enhanced CT scans were performed by the administration of iodine contrast material (400 mg/kg) by pressure injection through a large central vein catheter over 10–20 seconds. Serum levels of C-reactive protein,  $\alpha$ -1 antitrypsin, and  $\alpha$ -2 macroglobulin were monitored every other day in the last four patients.

Surgical exploration was begun in each patient by a left subcostal incision. Pancreatic necrosis was considered to be present when the typical grey-black necrotic material of putty-like consistency with surrounding turbid fluid was found in the lesser sac. Considerable effort was spent in determining whether the necrotic material was pancreatic or peripancreatic tissue. It was estimated at the operative table that pancreatic necrosis exceeded 30% of the gland in each of these 28 patients. Exposure of the splenic or superior mesenteric veins after debridement of the necrotic tissue greatly assisted this determination. When pancreatic necrosis was established as being present, the incision was extended and extensive unroofing of the retroperitoneum, sequestrectomy, and open packing of the lesser sac over Adaptic® gauze was carried out as previously described.<sup>15</sup> Pancreatic necrosis was considered infected only when smears or cultures of the necrotic material were positive for bacteria. After operation, additional debridement was carried out and dressings were changed in the operating room under light inhalation anesthesia at 2–3-day intervals. After three to five such re-explorations, dressing changes were done at the bedside. Wounds were permitted to heal by secondary intention. All patients were managed by intravenous hyperalimentation until oral feedings were resumed. Needle catheter jejunostomy for enteral feeding access has been added in recent patients.

The duration of hospitalization in these 28 patients ranged from 31 to 96 days (mean: 46 days). This time is not appreciably different from the 50–70 days required for drainage by the conventional surgical approach.<sup>7,17,18</sup>

TABLE 1. *Stratification of Severity in 28 Patients with Infected Pancreatic Necrosis*

Staging System	Average # Criteria (Range)	Expected Surgical Mortality Rate
Ranson and Spencer <sup>9</sup>	5.3 (3–8)	65%
Bank et al. <sup>16</sup>	1.6 (1–4)	56%

In the 10 most recent patients who had open drainage, the average length of postoperative recovery was 39 days.

## Results

Twenty-five of 28 patients treated by open drainage survived (11%). A 75-year-old woman died of autopsy-proved myocardial infarction 21 days after surgery. Massive aspiration after attempts at enteral feeding for persistent gastric outlet obstruction was responsible for the death of a 65-year-old man 87 days after surgery. Sudden death in a 47-year-old man 60 days after surgery was attributed to air embolism from a disconnected hyperalimentation catheter. In none of the three deaths was persistent or recurrent sepsis a factor.

Particularly impressive was the amount of additional necrotic and infected material that was found at the scheduled re-explorations. Reaccumulation of such materials, and the discovery of additional pockets of infected necrosis at scheduled dressing changes, occurred in each patient in this series. In general, the discovery of additional necrosis became less frequent in the individual patients as granulation tissue began to appear in the base of the lesser sac. In some patients, granulation tissue began to occur as early as the third dressing change. The predominant bacteria cultured from the necrotic tissue are shown in Table 2. Anaerobic bacteria (principally bacteroides) were also cultured in seven of these patients.

Despite favorable mortality, considerable morbidity attended the hospital courses of these patients. In addition to the known pulmonary, renal, metabolic, hematologic, and regional consequences of severe acute pancreatitis, a number of complications seemingly specific

TABLE 2. *Bacteriology of Infected Pancreatic Necrosis*

Organisms	No. of Patients
Escherichia coli	12
Aerobacter	7
Klebsiella	6
Pseudomonas	2
Enterococcus	1
Total	28

TABLE 3. Specific Complications Associated with Infected Pancreatic Necrosis and Open Drainage

Complications	No. of Patients	Percentage
Pancreatic fistula	10	36
Incisional hernia	8	29
Gastric outlet obstruction	6	21
Temporary	4	
Persistent	2	
Retroperitoneal hemorrhage	2	7
Intestinal fistula	1	4

for open drainage occurred (Table 3). Pancreatic fistula occurred in 10 patients but could be managed by conservative means in each patient. Somewhat surprisingly, incisional hernia developed in only eight patients, despite extension of the initial left subcostal incision to a bilateral subcostal incision in 26 of 28 patients. Gastric outlet obstruction was noted in six patients, and was persistent in two patients. Unfortunately, both of the latter patients died as a direct result of attempts to find alternate methods of nutrition. Two additional patients had major retroperitoneal venous hemorrhage from the portal system (one splenic vein and one superior mesenteric vein). These episodes were controlled with great difficulty by a combination of pressure and clotting agents. Early in our experience, we attempted to accelerate wound healing by sewing synthetic mesh to the abdominal wall. In one of the two patients in whom this technique was used, a jejunal fistula developed from contact with the mesh. This approach is no longer used and wounds now are permitted to heal entirely by second intention.

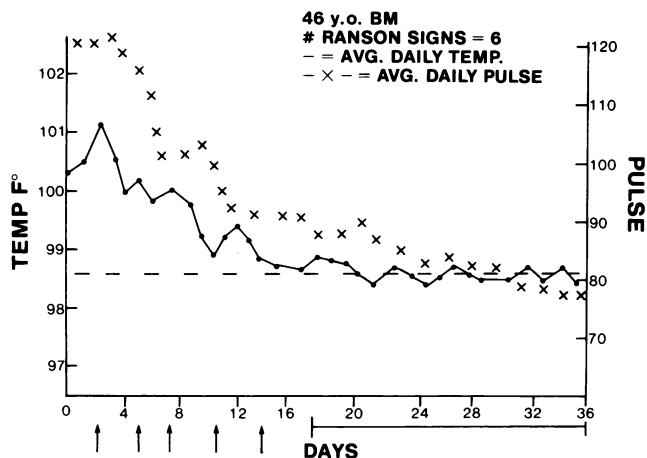


FIG. 1. Hospital course of a recent patient with infected pancreatic necrosis treated by open drainage. Arrows represent re-exploration in operating room; daily dressing changes on ward are shown by solid line.

Most importantly, none of the 28 patients subjected to open packing and scheduled re-exploration had persistent or recurrent sepsis. A representative hospital course from a recent patient is seen in Figure 1.

Dynamic CT pancreatography was correctly predictive of pancreatic necrosis in the four patients in whom it was used (Figs. 2 and 3).

Severely elevated serum levels of C-reactive protein ( $205 \pm 30$  mg/L SEM; NL  $< 40$  mg/L) and  $\alpha$ -1 antitrypsin ( $6.1 \pm 1.8$  g/L SEM; NL 1.5–3 g/L) along with depressed values for  $\alpha$ -2 macroglobulin ( $1.5 \pm 0.6$  g/L SEM; NL 2.3–2.7 g/L) were found before operation in the last four patients with infected pancreatic necrosis. Values tended to normalize as the necrotic process was controlled.

### Discussion

In recent years, the clinical course of patients with severe acute pancreatitis has changed. Formerly, the majority of deaths occurred early in the course of the disease, and were principally due to the consequences of hypovolemia.<sup>1-2</sup> More recently, however, advances in resuscitative and supportive management have all but eliminated dehydration as a mechanism of death in these patients. As a consequence of an increase in the length of patient survival, the later complications of severe acute pancreatitis (*i.e.*, necrosis and infection) are being recognized with increasing frequency. Sepsis has now become the most common cause of death in patients with acute pancreatitis who survive the first week of illness.<sup>2-4</sup> However, since the *overall* mortality rate in acute pancreatitis has not changed during this same period,<sup>1-3</sup> it seems clear that we must shift our attention to the management of sepsis if we are to make further progress in reducing mortality.

After the original morphologic classification of "hemorrhagic, suppurative, and gangrenous pancreatitis" by Fitz in 1889,<sup>19</sup> over 30 years passed before Moynihan recommended anterior celiotomy, debridement, and external drainage for patients with pancreatic abscess.<sup>20</sup> However, it was not until the landmark report of Altemeier and Alexander in 1963,<sup>5</sup> that these principles were widely adopted. In the 25 years that this conventional approach has been followed, operative mortality rates have remained in the range of 30–60%.<sup>6-11</sup> Periodic dissatisfaction with such results seemed to have been centered around the number and the types of drains used; the majority opinion being that sump suction was superior to other forms of drainage.<sup>6,7,9,21</sup> However, three of the largest surgical series of pancreatic abscess using the conventional approach failed to demonstrate any difference in survival regardless of the type or number of drains used.<sup>8,10,22</sup>

Why should the results of surgical drainage in pancreatic abscess be so greatly inferior to abscesses of other tissues? It has been claimed that 40% of patients who have conventional surgical drainage for pancreatic abscess will require re-exploration for persistent or recurrent abscesses.<sup>23</sup> Other workers have also noted a disturbingly high incidence of recurrent postoperative sepsis.<sup>6,7,10,22,24,25</sup> In an extensive review of the reported cases of pancreatic abscess in which the cause of death could be determined, we found that 76% of the postoperative deaths after conventional surgical drainage were due to sepsis.<sup>12</sup> These observations are sufficient to indict the conventional surgical approach. Fry and co-workers have also concluded that ineffective surgical drainage is primarily responsible for postoperative mortality in patients with intra-abdominal abscess.<sup>26</sup>

Why should the rate of persistent or recurrent sepsis be so high after conventional surgical drainage for pancreatic abscess? In addition to the observations that the retroperitoneum has few anatomic limitations to extensive tracking, and seems limited in its protective mechanisms against infection,<sup>27</sup> necrosis is a predominate feature of pancreatic abscess, and such particulate material cannot be drained by small caliber drains.<sup>10,15,22,28</sup> Recognition of pancreatic necrosis as a significant feature of pancreatic abscess has important clinical implications. Patients with infected pancreatic necrosis have a significantly higher mortality rate, more severe pancreatitis, increased renal insufficiency, and more profound metabolic aberrations than do patients with the more classical purulent forms of pancreatic abscess.<sup>13</sup> In view of these observations, a precise morphologic definition of any septic retroperitoneal process is clearly required.

Survival figures for "pancreatic abscess" may therefore be influenced either favorably (by inclusion of infected pseudocysts, suppurative peripancreatic abscess,

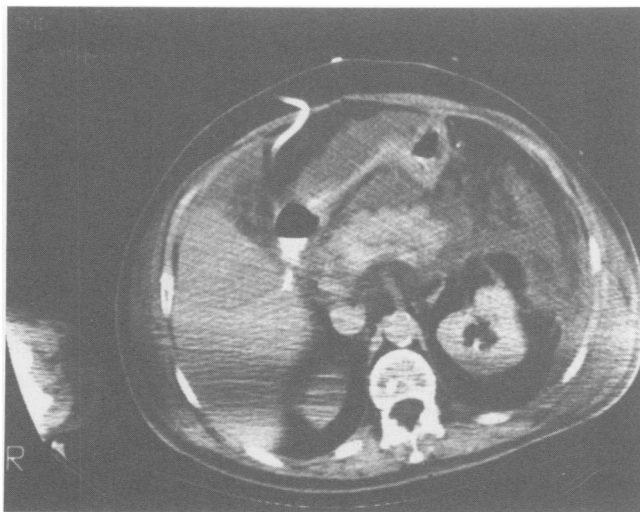


FIG. 2. Dynamic pancreatogram depicting absence of pancreatic parenchymal opacification in tail of pancreas. Necrosis of the tail of the pancreas was confirmed at surgery. Pancreatitis developed after common duct exploration (note T-tube).

and possibly even nonpancreatic lesser sac abscesses) or unfavorably (by including infected pancreatic necrosis). Many examples of such favorable definitions appear in the literature. In a recent review of 112 articles dealing with pancreatic abscess, we found that *only ten* authors even offered operational definitions of pancreatic abscess.<sup>29</sup> No two definitions were the same, and only two groups defined "pancreatic abscess" as necrosis. In view of the significantly greater mortality and morbidity of infected pancreatic necrosis, it is important that future reports distinguish between these conditions and recognize infected pancreatic necrosis as a separate clinical entity.

The low mortality rate in the current series of patients with infected pancreatic necrosis treated by open drain-

FIG. 3. Differential diagnosis of pancreatic necrosis by dynamic pancreatography. Process shown by conventional CT on left could represent either phlegmon or necrosis. After intravenous bolus of contrast material (note contrast in aorta), no pancreatic tissue could be opacified. Surgical exploration revealed extensive pancreatic necrosis.



age compares favorably with the 30–60% mortality rates from previous reports of “pancreatic abscess” using conventional drainage.<sup>6–11</sup> The comparison becomes even more favorable when it is appreciated that in many of the previous series pancreatic abscess was not defined as adversely as in the current report.

Dismayed by a 55% surgical mortality rate for patients with infected pancreatic necrosis treated by conventional surgical drainage in our own institution, in 1976, we devised the concept of open drainage to combat the problem of continuing necrosis and reaccumulation of purulent material after conventional drainage.<sup>14</sup> At that time we were unaware of previous efforts in the use of this principle. In 1928, Schmeiden and Sebening recommended that after exploration for pancreatic necrosis, “the incision should be kept wide open for many weeks because of the persistent wound and pancreatic enzyme secretion and also because of the extremely long time required for the discharge of the gland sequestra.”<sup>30</sup> In 1968, Bolooki and co-workers reported suturing the wall of pancreatic abscesses to the abdominal skin (marsupialization) with successful results.<sup>28</sup> The current technique of open drainage differs from these historic precedents by extensive unroofing of the retroperitoneum, the use of nonadherent *Adaptic*<sup>®</sup> gauze, and scheduled re-explorations with additional debridement and dressing changes.

Favorable experiences with open drainage are also being accumulated by other workers. Pemberton and his colleagues reported a 20-year longitudinal comparison of open drainage (17 patients) with conventional closed drainage (64 patients) in which they found a significant decrease in mortality rate when open drainage was used (18% vs. 44%;  $p < 0.05$ ).<sup>31</sup> Wertheimer and Norris salvaged 8 of 10 deteriorating patients with necrotizing pancreatitis and persistent sepsis after conventional surgical drainage by converting each case to open drainage.<sup>32</sup> Vogel and his associates have successfully managed 11 consecutive patients with infected pancreatic necrosis by open drainage (Vogel S, personal communication, 1987). Waclawiczek et al. reduced their surgical mortality rate in infected pancreatic necrosis from 70% using conventional surgical drainage to 17% in a group of 17 patients treated by open drainage.<sup>33</sup> Each of these workers have also attributed the reduction in mortality rate with open drainage to the removal of reaccumulated necrotic and infected material by the continued debridement of scheduled re-exploration and dressing changes. We continue to believe that reaccumulation of necrosis and infection accounts for the high incidence of postoperative sepsis after conventional debridement and drainage, and ultimately results in increased mortality.<sup>14</sup>

Warshaw and Jin have taken issue with the necessity for open drainage in patients with pancreatic sepsis, cit-

ing a 5% mortality rate for conventional closed drainage in 45 patients with “pancreatic abscess.”<sup>34</sup> Compared with previous studies using similar conventional surgical drainage, they attribute their greatly improved survival rates to earlier diagnosis and surgical intervention, extensive debridement and drainage, and improved postoperative care. Unfortunately, they did not define “pancreatic abscess” as adversely as infected pancreatic necrosis, nor did they stratify the severity of the underlying pancreatitis. Each of these factors is known to significantly affect mortality rate.<sup>2,13</sup> Furthermore, Pemberton and co-workers separated their data on 81 patients with pancreatic abscess into three time periods and could not demonstrate any recent increase in survival, which if present, might have been attributable to earlier diagnosis by computed tomography or to recent improvements in intensive care.<sup>31</sup> These observations suggest that apples and oranges remain incomparable.

Three complications of open drainage that could be considered procedure-specific require further comment. In the Mayo Clinic series, an enteric fistula developed in 31% of patients, presumably resulting from the frequent dressing changes.<sup>31</sup> We have continually advocated placing a porous nonadherent petrolatum gauze (*Adaptic*<sup>®</sup>) next to the intestine to prevent inadvertent intestinal debridement during frequent dressing changes,<sup>14</sup> an important step omitted in the Rochester series. Using this approach, only one of our 28 patients had an enteric fistula. Similarly, it is recommended that *Adaptic*<sup>®</sup> be used to cover any major veins exposed by the process of debridement to limit the frequency of major venous hemorrhage. Less major hemorrhage is easily controlled by the packing process. Finally, functional gastric outlet obstruction has been seen in six of our 28 patients. In each of the six patients, barium studies failed to demonstrate a mechanical cause. Since prolonged delays in gastric emptying have also been noted after conventional closed drainage,<sup>6,8</sup> it is possible that the observed functional obstruction in our patients represents a defect in gastric or duodenal motility caused by severe pancreatitis, rather than being due to the technique of open drainage. Nevertheless, since gastric emptying did not improve in two of the six patients despite intense medical management, and ultimately led to the death of both patients, needle catheter jejunostomy for subsequent enteral feeding is now placed in an uninvolved segment of jejunum at the original exploration.

Recognition of infected pancreatic necrosis can be difficult even using currently available diagnostic modalities. Bolus contrast enhanced CT scanning (dynamic pancreatography) appears to be particularly attractive in its ability to distinguish pancreatic necrosis from the less severe edematous pancreatitis. Kivisaari and his associates described significantly decreased contrast en-

hancement after a rapid bolus injection of contrast material (dynamic pancreatography) in a group of nine patients who had surgery for pancreatic necrosis.<sup>35</sup> In a prospective study using dynamic pancreatography, Block et al. correctly predicted the presence of pancreatic necrosis in 50 of 59 patients.<sup>36</sup> In the four patients in the current study in whom dynamic pancreatography was used, it was of material value in both the diagnosis of pancreatic necrosis and the timing of surgery.

Favorable experiences with serum monitoring of the acute phase reactants C-reactive protein,  $\alpha$ -1 antitrypsin, and  $\alpha$ -2 macroglobulin to indicate the presence of pancreatic necrosis are being accumulated by several groups.<sup>37,38</sup> Overall detection rates have been as high as 85–90% in small groups of patients. If these serum tests maintain similar accuracy in controls and larger groups of patients with acute pancreatitis, they will represent a significant diagnostic addition. We are continuing to collect data on these tests.

In agreement with others<sup>39–41</sup> it has been our experience that CT-guided fine needle aspiration for smear and culture can be a valuable adjunct in determining whether a given pancreatic process is sterile or infected. This technique may prove to be particularly helpful in evaluating whether pancreatic necrosis indicated by dynamic pancreatography or serum acute phase reactants has become infected.

Open drainage of infected pancreatic necrosis appears to represent a significant advance over previous conventional techniques. Although it is likely that the final form of open drainage has not yet been described,<sup>42</sup> the principles of management described in this report continue to be promising and merit further study.

### Acknowledgments

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#### DISCUSSION

DR. CHARLES F. FREY (Sacramento, California): Dr. Bradley a number of years ago brought to our attention the open packing method of treating patients with infected necrosis of the pancreas and peripancreatic tissues. He has now accumulated a large experience and has achieved the lowest mortality reported for this otherwise lethal complication of pancreatitis. I believe Dr. Bradley's technique of dealing with infected necrosis represents a significant advance.

It is important to emphasize one semantic point that bears on this. That is, a well-loculated walled-off collection, whether it is called an abscess or an infected pseudocyst, is a different entity from infected pancreatic necrosis. Reports lumping the two together are mixing apples and oranges. Infected pancreatic necrosis occurs earlier after the onset of symptoms than an abscess. It consists of large segments of intact or infected particulate matter involving the pancreas and/or pancreatic tissues. It is usually associated with signs of gram-negative sepsis and can only be effectively managed by debridement or excision of particulate matter, and as Dr. Bradley is teaching us, by open packing and repeated debridement. The latter is made necessary by the fact that the combination of bacteria and enzymatic destruction of the pancreas and peripancreatic tissue continues after the initial debridement, creating new areas of necrosis. Abscesses, on the other hand, are easily managed by operative or percutaneous drainage as no new necrosis occurs after drainage.

I support the use of the open packing technique, which we use in our patients, as we believe it reduces the mortality and the length of hospitalization.

I would like to show two slides, and I would like comments from Dr. Bradley, which are little variations in technique.

Before we put in the open packing technique, we use a large Davol drainage catheter from one side of the abdomen to the other for irrigation purposes. (Slide) Then we place the adaptic gauze and packed over it.

The other thing I would like to ask Dr. Bradley about is that we have encountered patients in whom there has been hemorrhage at the time of the debridement with extension of infection into the spleen. We have found splenectomy should be performed under these circumstances to avoid further bleeding.

I would also like to ask the question of Dr. Bradley as to whether he has follow-up information on his patients after they have been discharged from the hospital. Dr. Braasch, a number of years ago, pointed out that many of the patients who had recovered from their pancreatic infections returned with complications of chronic pancreatitis.

Finally, I do not believe we can attribute all of the reductions in mortality to the open packing technique. The importance of other factors such as improved surgical intensive unit care, including monitoring, ventilator care, fluids and electrolytes, antibiotics and TPN, cannot be discounted and are, in my opinion, significant contributors to the reduction in mortality we are seeing in this disease.

DR. ANDREW L. WARSHAW (Boston, Massachusetts): I rise in admiration of Dr. Bradley's continuing efforts to deal with these very ill patients, but I must say that I have to disagree with him on a number of points.

He and Dr. Frey have made the point that infected necrosis is different from an abscess. There is an element of difference in that some of these patients are more ill than others, but I submit that infection is

not the primary difference, but whether or not there is ongoing necrotizing pancreatitis.

The infected necrosis patients do present earlier, at an average of about 10 days. The so-called pure pus collections are several weeks later, and they are often less ill but not always. At the time of operation the difference between infected necrosis and noninfected necrosis may be absolutely indistinguishable to the naked eye. It, therefore, may make little difference in terms of the treatment.

In terms of toxicity, Beger, whom Dr. Bradley quoted, has shown that the hemodynamic changes of necrotic tissue, whether or not infected, are virtually identical, and therefore, the toxic effects on the organism as a whole may be indistinguishable.

On the contrary, the patient with infected necrosis may be completely nontoxic. Percutaneous needle aspiration studies have shown in fact that a patient may have no signs of toxicity: no fever, leucocytosis or hemodynamic instability, and yet have bacteria present in the pancreatic necrosis. I remind you that Ranson's criteria are prognostic signs developed in the first 2 days of illness. They are not signs of what goes on 2 weeks later at the time of pancreatic abscess or infected necrosis.

The infection can set in as early as the fourth or fifth day, much earlier than we had previously suspected. This indicates that there may be a long indolent phase before it is clinically apparent. It would seem that the effects of infection and the ongoing enzymatic and necrotizing effects of pancreatitis combine early in some patients to generate a particularly fulminant course.

I find it difficult as well to accept the bland statement of how much of the pancreas is involved. Much of the lucent areas seen in these CAT scans is not pancreatic but peripancreatic fat. Since the tissue that is debrided is unidentifiable necrotic debris, I find it difficult to know how much of the pancreas is involved no matter how big the glob of swamp muck you pick out. In fact, as Bradley's figures show, few of these patients turn out to be diabetic in the long run. Although up to 80 or 90% has to be lost before producing diabetes, long-term studies do not show much pancreatic insufficiency after severe necrotizing pancreatitis.

The use of contrast-enhanced CT scanning is being suggested. This is a bandwagon that many are jumping on now. As far as I am aware, it has yet not been validated in any long-term study in Europe or here.

Finally, the statement that this is the best series in terms of reduction of mortality from this very difficult problem is a slight overstatement. Beger's own large study is reported as achieving about a 5% mortality rate with closed debridement and drainage. Although he does add local lavage catheters into the pancreatic bed, it is closed drainage, not packing. In our own series presented before this society 2 years ago, now up to 60 patients, the mortality over the past 7 years, and about 40 patients is also 5%. Our historical controls like Dr. Bradley's had a 40% mortality rate in the previous 5 years.

Therefore, what we are seeing in a number of different centers is a much improved survival rate resulting from a variety of different techniques: open packing, closed debridement and drainage (which is what we use), and Beger's closed debridement and drainage with addition of local lavage. Since all are accomplishing the same thing, it is probable that the common element is adequate debridement. Whatever else you do is probably less important and camouflages the basic issue.

I would like to ask Dr. Bradley at what point would he use needle aspiration techniques to determine whether or not there is infection, and would he use that information once he had it to decide whether or