Parenteral Nutrition in the Treatment of Acute Pancreatitis:

Effect on Complications and Mortality

JOHN T. GOODGAME, M.D.,* JOSEF E. FISCHER, M.D.

From the Hyperalimentation Unit, Department of Surgery, Massachusetts General Hospital, Boston, Massachusetts

Clinical characteristics of 46 cases of acute pancreatitis treated with total parenteral nutrition were examined. Hyperalimentation may be used in these severely ill patients with minimal technical or metabolic morbidity. This method of nutritional support can maintain patients with nonfunctional gastrointestinal tracts for several months. Catheter-related sepsis was more common than expected early in the course of acute pancreatitis but caused minimal morbidity. The incidence of catheter-related sepsis late in disease was minor. Hyperalimentation had little if any effect on the pathophysiology of acute pancreatitis as judged by the overall mortality and the incidence and severity of the complications of acute respiratory failure and acute renal failure. It is not clear that parenteral hyperalimentation alters the course of acute pancreatitis but it is a useful adjunct for nutritional support in this illness.

THE THERAPY OF PANCREATITIS is basically symptomatic and empiric as the fundamental etiology of the disease is unknown. Suggestions of potentially beneficial modalities are made as new features of the disease or of pancreatic excretory function are elucidated. Specifically, glucagon,[14] trasylol,[26] diamox,[19] atropine,[16] antibiotics[16] and steroids[4] have all been acclaimed as helpful, or even life-saving, in acute pancreatitis. Subsequently, most have been questioned as non-contributory to the ultimate outcome of the disease.[18,24]

Emphasis on adequate nutrition as a cornerstone of adequate surgical therapy is relatively recent. As methods of nutritional support in severe illness have become more readily available, evaluation of the usefulness in various conditions has become essential. The alternatives for nutritional support are oral feedings, feedings via indwelling intestinal conduits such as gastrostomy or jejunostomy tubes, and parenteral nutrition. In pancreatitis, because of the usually associated ileus and the desire to keep the G.I. tract and pancreatic exocrine system in a state of "rest," nutritional maintenance by means of the G.I. tract is difficult. The concomitant problems of sepsis, metabolic abnormalities, and potential cardiovascular instability mitigate against plans for nutritional supplementation with glucose-rich hyperosmolar fluids administered via indwelling polyethylene catheters. Attention has recently been focused by Feller et al.[6] on the value of nutritional support in the treatment of "severe pancreatitis". They partially attribute a reduction of overall mortality from 22% to 14% to their ability to correct nutritional depletion via hyperalimentation.

This report reviews our experience and discusses the relationship of hyperalimentation to the complications of pancreatitis. It is to be emphasized that the retrospective nature of this analysis and the absence of any true control group makes definitive conclusions difficult. Qualitative assessment of the risk-benefit ratio is our present objective. Table 1 summarizes some of the published experiences of other groups with acute pancreatitis, with specific reference to the complications and mortality, and will serve as a baseline for discussion.

Materials and Methods

The clinical records of patients with the diagnosis of acute pancreatitis who were treated with parenteral nutrition during the 36 month period between January 1972 and December 1974 were reviewed. The 44 patients required 46 admissions for acute pancreatitis and represent 11% of all patients admitted with the diagnosis of acute pancreatitis during that time period. Criteria for inclusion in the study were as follows: (1.) All patients were hyperalimented during the period of time in which they had clinical pancreatitis. (2.) Significant oral intake had been omitted for at least

* Current address: Surgery Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland, 20014.

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TABLE 1. *Complications of Acute Pancreatitis in Selected Published Series*

Author	Total Number of Patients and Selection Factor	Overall Mortality	Renal Failure		Respiratory Failure		Surgical Mortality
			Incidence	Mortality	Incidence	Mortality	
Present study	46 (severe pancreatitis)	9/46 (20%)	7/46 (15%)	4/7 (57%)	13/46 (28%)	8/13 (62%)	8/36 (22%)
Gordon[10]	41 (unselected)	6/41 (15%)	6/41 (15%)	3/6 (50%)	—	—	—
Interiano[13]	50 (unselected)	5/50 (10%)	—	—	9/50 (18%)	5/9 (55%)	—
Gleidman[11]	26 (malignant pancreatitis)	9/26 (35%)	—	—	—	—	9/16 (56%)
Feller[6]	83 (severe pancreatitis)	12/83 (14%)	—	—	18/83 (22%)	8/18 (44%)	—
Frey[8]	306 (unselected)	78/306 (25%)	19/306 (6%)	18/19 (95%)	—	—	36/211 (17%)
Ranson[21]	31 (severe pancreatitis)	15/31 (48%)	10/31 (32%)	6/10 (60%)	12/31 (39%)	9/12 (75%)	10/21 (48%)
Lawson[15]	15 (severe pancreatitis)	4/15 (26%)	—	—	—	—	4/16 (26%)

4–5 days prior to use of parenteral nutrition. The diagnosis was established by each patient meeting at least two of the following three criteria: (1.) A clinical syndrome of abdominal pain and/or vomiting and epigastric tenderness (46 out of 46 patients). (2.) Increased serum level of amylase greater than 25 Russell units or lipase greater than one unit/ml (43 out of 46 patients) and/or increased ratio of amylase clearance to creatinine clearance (17 out of 29 patients).[27] (3.) Operative or pathologic findings consistent with the diagnosis of acute pancreatitis (37 out of 46 patients).

Of the 44 patients studied, 21 were male and 23 were female. Their age range was from 8 to 90 years. There were 10 patients treated in 1972, 17 in 1973, and 17 in 1974.

The details of the composition of the parenteral hyperalimentation solutions and administration have been described elsewhere.[1,7,22] Six patients with renal failure complicating their hospital course were treated with essential amino acids and hypertonic dextrose.[1]

Studies

Study Population

The study population is heterogeneous. The statistical profile of etiologies, underlying medical diseases, and management modalities places it on the "severe" end of the spectrum of acute pancreatitis.

For many reasons the group of patients studied is heavily weighted toward that sub-group of acute pancreatitis which is handled surgically, and does not illustrate a representative cross-section of the patients who present with pancreatitis. Our experience overall has been that approximately 70% of those with acute pancreatitis have alcohol ingestion as the principal underlying etiologic agent and 80% have no operative intervention during their hospital course. The large percentage of patients in this series treated operatively does not suggest differences in indications for operative intervention as much as selection of patients for hyperalimentation because of the relative severity of their illness. Biliary tract disease was the etiology of pancreatitis in 39% (18 of 46 of our patients), alcohol in less than 20% (6 of 46). Postoperative pancreatitis (three of 46) followed caesarian section, transduodenal sphincteroplasty for recurrent common duct stones, and excision and grafting of an abdominal aortic aneurysm. Carcinoma of the pancreas (two of 46) presented with the syndrome of recurrent epigastric pain, elevated pancreatic enzymes and volume requirement. Laparotomy was required in both cases to establish the diagnosis. The category of traumatic pancreatitis (6 of 46) might well be considered with the postoperative group as all required emergency surgery after their primary injury. Pancreatitis persisted in the postoperative course but in all 6, was thought etiologically related to the trauma. All 6 initial procedures were for control of intraabdominal bleeding but two involved repair of a pancreatic laceration and one distal pancreatectomy. The contribution of etiology of pancreatitis to ultimate mortality is illustrated in Table 2.

TABLE 2. *Mortality by Etiology of Pancreatitis*

	Survived	Died	Total
Biliary	15	3	18 (39%)
Alcohol	8	1	9 (20%)
Traumatic	5	1	6 (13%)
Hyperlipoproteinemia (Type IV)	2	—	2 (4%)
Postoperative	3	—	3 (7%)
Carcinoma of Pancreas	1	1	2 (4%)
Idiopathic	3	3	6 (13%)

Four of the 46 patients in this group had uncomplicated past medical histories. The vast majority had complicating underlying illnesses which further jeopardized their survival capacity when faced with the stress of acute pancreatitis. These concomittant medical problems are summarized in Table 3.

Management

Non-Operative

The multiplicity of modalities which exist for the therapy of pancreatitis was reflected in the various

regimens used in these patients. All but two had nasogastric tubes placed (44 out of 46). Seventy per cent of the patients were placed on "prophylactic" antibiotics. The choices of specific agents reflected an attempt at broad spectrum coverage and included various regimens. Ampicillin or cephalothin was used alone in 18 patients. In 8 additional cases the combination of penicillin and chloramphenicol or clindamycin and an aminoglycoside was chosen. No statement can be made as to the incidence of septic complications with these regimens but certainly none of the antibiotic umbrellas completely prevented wound infection or septicemia in these patients.

Volume requirement was dealt with appropriately with heavy reliance on colloid. Seventy-four per cent of these patients required blood transfusions during the course of their illness. Forty-one per cent received two to 10 units while 20% received greater than 10 units of blood. Salt poor albumin and furosemide were used in 6 patients with pulmonary edema and pancreatitis but results were equivocal. Two patients seemed to respond rapidly to this regimen but two resolved their pulmonary problems over 5–7 days and two clearly deteriorated on this therapy. Cardioactive drugs were used in 25% of all patients for complications of low cardiac output. Digitalization was carried out acutely in 8 patients and 7 required vasoactive amines to maintain their cardiac output. One patient was treated for a brief period with an intra-aortic balloon pump with initial success in dealing with cardiac dysfunction only to fall victim to late cardiac arrest. Large doses of corticosteroids were given to three patients whose initial course suggested a septic component to their presenting shock (two of the three patients survived). Peritoneal dialysis was used in the therapy of 6 patients and atropine was used in only one. Glucagon and trasylol were not used in any of the patients in this series.

There were 10 patients (22%) who were treated completely nonoperatively. In this sub-group there was only one death. The immediate cause of death of this patient was acute renal failure and intractable cerebral edema.

TABLE 3. *Associated Medical Problems at Time of Presentation with Pancreatitis (46 Patients)*

Cardiovascular Disease (including angina, valvular disease and arrythmias requiring outpatient medication)			16
Pulmonary Disease			
COPD			6
Pneumonia			3
Pleural effusion			2
Active TB			1
Acute/Chronic Alcoholism			12
Delirium Tremens			2
Significant Liver Disease			
Biopsy confirmed cirrhosis			4
Viral hepatitis			1
Status–post portocaval shunt			1
Significant Renal Disease (BUN > 36, Creatinine > 2.0)			3
Anuria			1
Other			
Duodenal ulcer disease	5	Cerebrovascular accident	2
Diabetes Mellitus	5	Sjogren's Syndrome with cryoglobulinemia	1
GI bleeding	1	Porphyria	1
		Multiple Sclerosis	1

TABLE 4. *Operative Procedures During Hospital Course for Pancreatitis*

	No. of Patients	Survival by Procedure	
		Survived	Died
GU reconstruction and small bowel resection	1	1	0
3-Tube placement (15)	9	4	5
External drainage of pancreatic pseudocyst or abscess	8	4	4
Distal pancreatectomy	3	3	0
Cholecystectomy	11	9	2
Cholecystostomy	3	2	1
CDE	11	9	2
Pancreatic cystogastrostomy	6	1	5
Splenectomy	1	1	0
Puestow procedure	2	2	0
Transduodenal sphincterotomy and sphincteroplasty	0	7	1
Dilatation of papilla	1	1	0
Repair of pancreatic laceration	1	0	1
By-pass procedure for pancreatic carcinoma	2	2	0

Operative Treatment

Indications for operation in these cases of acute pancreatitis were: (1) Deterioration or failure to improve on an "optimal medical program". (2) Clinical suspicion of biliary disease, pancreatic carcinoma or pancreatic pseudocyst/abscess as the etiology of the acute pancreatitis. (3) Complication of the course of acute pancreatitis with sepsis from an apparent intra-abdominal source or intra-abdominal bleeding. (4) Therapy of relapsing pancreatitis.

Because of the variability of intra-abdominal pathology, the clinical setting and individual experience, multiple procedures were often required at a single operative venture. The variety and frequency of these procedures is illustrated in Table 4. Six patients had two operations during the course of their hospital stay with four requiring redrainage of intra-abdominal abscesses, one requiring re-exploration for bleeding

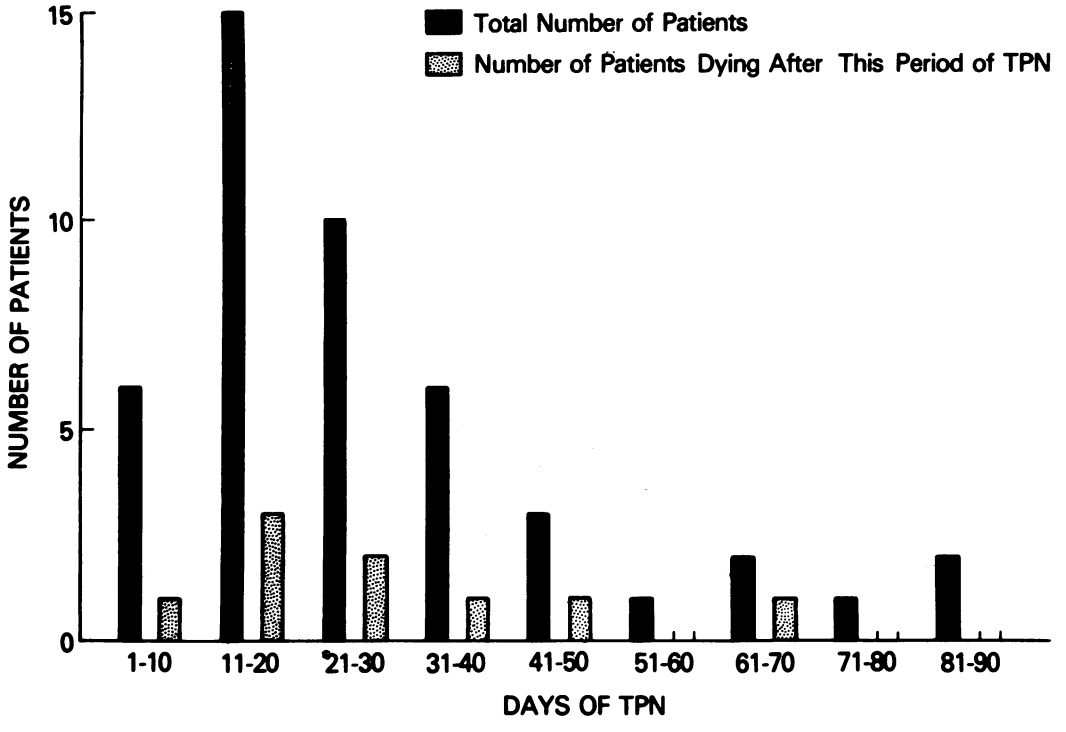

FIG. 1. Duration of G.I. dysfunction and inability to take oral nourishment as indicated by duration of parenteral nutrition had no effect on the mortality of the illness.

immediately postoperatively, and one requiring a gastrojejunostomy for chronic duodenal obstruction secondary to pancreatitis. Of these 6 patients, two died and four were discharged. Of the total operative group of 36 patients, eight (22%) died and 28 (77%) were discharged. Of the 8 patients who died, two died in intractable septic shock, two died of combined cardiac, pulmonary and renal failure, and one succumbed to an acute myocardial infarction secondary to septic hypotension following a T-tube cholangiogram. The other three patients died of pulmonary embolism, pulmonary insufficiency, and acute renal failure with terminal candida sepsis.

Hyperalimentation and Hospital Course

Despite the overall severity of acute pancreatitis and the multiplicity of underlying medical problems in this group, the technical features of hyperalimentation offered few problems. Ninety-nine catheters were placed with only three mechanical complications. There was one episode of subclavian artery puncture which required no specific therapy and one episode of subcutaneous infiltration of hyperalimentation solution secondary to inadequate fixation of the administration catheter. Neither of these complications added to in-hospital time. One patient developed subclavian vein thrombosis noted two weeks after discharge after complaints of arm swelling prompted a venogram. This required 6 weeks of anticoagulation for resolution.

Duration of hyperalimentation varied from five to 90 days with an average of 28 days per patient. When the 46 patients are divided into two groups according to the length of time they required parenteral nutrition (Fig. 1), those who were unable to eat for more than one month did no worse in terms of survival than those whose disease resolved in less than 30 days. Much of this similarity may be explainable on the basis of more severe disease present in those who died early in their course, but nutritional support certainly contributed to the progress of the group of severely ill patients unable to take oral alimentation for 50 to 90 days.

Parenteral nutrition was discontinued after oral intake was adequate in 33 of the 46 patients studied. Five patients were hyperalimented until their death and two patients had their I.V. nutrition discontinued when hope for their recovery was lost. Six patients had their hyperalimentation discontinued because of complications directly related to that modality of therapy. Episodes of catheter-related sepsis prompted discontinuation in five patients while inability to control rising serum potassium was given as the reason for stopping parenteral nutrition in one other.

Despite the relative metabolic chaos resulting from pancreatitis itself and the 15% incidence of acute renal failure, there were only four episodes of metabolic abnormalities which required discontinuation of hyperalimentation. Initial glucose intolerance was common (25%) but was usually controlled by temporarily reducing the infusion rate or by incorporating small amounts (10–20 units) of regular insulin to the infusion mixture. There was only one patient in whom glucose

intolerance posed a significant problem. This eventually required 60 units of regular insulin per liter of hyperalimentation solution to maintain a blood glucose of less than 200 mg per 100 milliliters. There were two episodes of protein intolerance with elevation of serum ammonia. One episode occurred in a patient with a previous portacaval shunt. The second occurred in a patient with persistent bacteremia secondary to undrained intra-abdominal abscesses who finally succumbed to cardiovascular collapse related to her prolonged sepsis. Electrolyte abnormalities were common in this population but usually responded to manipulation of the content of the hyperalimentation mixtures. Of particular interest was hypocalcemia (<7 mg/100 ml) which occurred in 7 of the 46 patients. One of these 7 died. Calcium replacement normalized serum calcium in 6 of the 7 patients but one of the 7 required 160 mEq of calcium every four hours for a period of 12 hours to arrest the continued decline of serum calcium. Eventually, peritoneal dialysis was instituted because of the continued progression of her pancreatitis and her calcium requirement ceased after the second run of peritoneal dialysis. She eventually survived.

Twenty-two of the 46 patients experienced no complications after definitive therapy of their acute attack of pancreatitis. The pancreatitis resolved and they were discharged. Four of these 22 patients required no operation during their courses of therapy. Eighteen, however, required surgical intervention but had a benign postoperative course. None of the patients whose hospital course was without complications was over 64 years of age.

Twenty-four of the 46 patients had one or more of a host of medical and surgical complications. These are summarized in Table 5. Cardiovascular dysfunction was the most frequently noted complication with 16 patients exhibiting cardiovascular problems. Respiratory insufficiency requiring intubation was the next most frequent complication with 13 patients requiring ventilatory assistance for variable lengths of time. Fluid overload and congestive heart failure were the most frequent etiologic agents (5 of 13) but pneumonia, septicemia, and the idiopathic ventilatory insufficiency syndrome of pancreatitis, played a role in more than half of the patients requiring respiratory support. Hyperalimentation per se made no discernible contribution to either cardiovascular or respiratory complications. Only two of our patients who required ventilatory support for more than 8 days survived. There was no statistically relevant relationship between the age of the patient and the development of ventilatory insufficiency.

In the series of acute pancreatitis summarized in Table 1, the incidence of respiratory failure varies from 18% to 39%. The mortality associated with this complication ranges from 44% to 75%. There is no improvement in these parameters in this series of hyperalimented patients with an incidence of respiratory failure of 28% and a mortality associated with this complication of 62%.

TABLE 5. *In-Hospital Complications (46 Patients)*

	Affected	Died
Cardiovascular	26	9
Low cardiac output	13	8
Arrhythmias	9	8
PAT	4	1
Ventricular Tachycardia	3	2
PVC	3	1
Junctional rhythm (digitalis toxicity)	1	1
Atrial fibrillation	1	—
Myocardial infarction	2	2
Pulmonary embolus	2	1
Cerebro-vascular accident	1	1
Respiratory Insufficiency (requiring intubation)	13	8
Fluid overload/CHF	5	3
Idiopathic	4	2
Pneumonia	2	2
Septicemia	9	4
Renal		
Acute renal failure (BUN > 50, Creatinine > 3.5, urine output <400c/d)	7	4
Significant pre-renal azotemia (BUN > 75 with normal creatinine & urine output)	2	—
Metabolic		
New onset glucose intolerance requiring insulin	7	3
Hyperammonemia	2	1
Hypokalemia	1	1
Hypocalcemia	7	1
Encephalopathy	1	—
Surgical Complications (36 patients required operation)	16	4
Wound infections	13	3
Wound dehiscence	2	—
Recurrent intrabdominal abscess	4	3
Fistulas	3	1
Re-exploration for bleeding	1	—
Duodenal obstruction requiring re-operation post-sphincteroplasty	1	—
Bleeding Dyscrasia	7	2

Renal abnormalities affected 9 patients and ranged from simple pre-renal azotemia in two patients, to acute renal failure (BUN >50, creatinine >3.5, urine output <400 cc per 24 hours) in 7 others. Advancing age was positively correlated with this complication. Five of the seven patients received Renal Failure Fluid[1] as part of the therapy of their acute renal failure and three required peritoneal dialysis. Four of the 7 patients who developed acute renal failure died. There does not appear to be a beneficial effect of hyperalimentation upon this complication of pancreatitis (Table 1). The similarity between previous large series and our own hyperalimented patients in overall mortality, and

incidence and outcome of these selected complications suggest little effect of total parenteral nutrition on the underlying pathologic mechanisms of acute pancreatitis.

Sixteen of the 36 patients who required operation as part of their therapy for acute pancreatitis developed complications directly related to their operation. Thirteen of the 36 developed wound infections with two of these resulting in frank dehiscence. Four of the 36 developed recurrent intra-abdominal abscesses and three of the 36 developed enterocutaneous fistulas. Four of the 16 patients who developed surgical complications died.

The mortality of operative procedures in this group was 22%. This compares favorably to the published experiences listed in Table 1. Lawson et al.[15] stated that all of their patients were alimented via jejunostomy tubes as soon as bowel motility returned. Their experience with prolonged convalescence (mean 49 days) and few late deaths (none after 35 days) mirrors our own and serves to underline the importance of nutritional support rather than hyperalimentation itself for these potentially salvageable patients.

The major hyperalimentation-related morbidity in this series principally relates to episodes of blood stream microbial invasion involving the polyvinyl catheters. In this series of 46 admissions there were 8 episodes of documented catheter-related sepsis. Documentation required either positive blood cultures coincident with catheter cultures positive for the same organism or positive blood cultures with disappearance of the clinical syndrome of septicemia after removal of the hyperalimentation catheter. In 46 patients there were 8 episodes (17%) but those 8 episodes were spread over 99 intravenous catheters. All patients in this series with septicemia documented by at least one positive blood culture are summarized in Table 6. Two patients had two episodes of catheter-related sepsis. Four out of the eight episodes of catheter-related septicemia occurred in catheters which had been in place less than 10 days. Five of the episodes of septicemia associated with the parenteral nutrition lines were with *Staphylococcus aureus*, two were with *Staphylococcous epidermidis* and one was with an *Enterococcus*. In none of these cases of catheter-sepsis could seeding from other areas of infection be implicated. The incidence of septicemia with catheters which had been in place less than two weeks (7 of 73 catheters) is greater than the incidence in those catheters in place more than two weeks (one of 26 catheters). All but one of the episodes of catheter-related sepsis in these 7 "young" catheters occurred with the first catheter placed in these patients implying an increased morbidity to hyperalimentation in the early phase of acute pancreatitis. Our institution has previously reported incidences of catheter-related sepsis of three or 7% depending on observance of strict protocol.[21] The incidence of septic catheter complications in this series of 8% (catheters) or 17% (patients) is considerably higher than our past[21] and present (one septicemia/380 patient days, 1975) overall hyperalimentation experience but 6/8 (75%) of those problems arose during the early, acute phase of the illness. It seems possible that the early, acute phase of pancreatitis carries increased risks of septicemia when hyperalimentation is used as a therapeutic modality. The only potential consolation to the high incidence of catheter-related septicemia is the relatively low morbidity associated with this event relative to other episodes of septicemia. Removal of the hyperalimentation catheter for any fever of unknown origin in the patient who is being hyperalimented is standard practice. As a result, damage sustained by catheter-related sepsis has been held to a minimum. Of the 6 patients who had positive blood cultures related to their hyperalimentation catheters, five survived their pancreatitis. The single death was secondary to pulmonary insufficiency and the course was apparently unaffected by the episode of catheter-related septicemia. In this same group of patients, septicemia secondary to other sources was seen in eight patients. Five of these 8 patients died and, in four of these five, the septicemia was a significant contributor to death. Sources of these episodes are summarized in Table 6.

Discussion

The rationale for TPN in acute pancreatitis rests on several clinical assumptions: (1) The maintenance of nutritional integrity in the absence of completely functional G.I. tract and in the presence of severe illness is essential, particularly if the course is prolonged. (2) TPN may represent active intervention in the pathological processes that exacerbate the acute disease (reversal of malnutrition, prevention of complications, decreasing pancreatic exocrine secretion). (3) TPN may be carried out in this group of patients without prohibitive morbidity. The first of these assumptions is suggested, but not proven, by the eventual survival of 12 of 15 patients critically ill with non-functioning G.I. tracts for periods of time ranging from one to three months. The overall 80% survival compares favorably with the experience with severe pancreatitis reported by other clinical centers (Table 1). Feller, et al.[6] have suggested that aggressive correction of nutritional depletion has had significant impact on the survival of their patients with severe acute pancreatitis. In the patient with prolonged illness, initial malnutrition and

TABLE 6. *Analysis of Episodes of Septicemia in Patients with Acute Pancreatitis Receiving Hyperalimentation*

Age	Etiology of Pancreatitis	Operation	Organism	Catheter Related	TPN Related	Associated Infective Sources	Antibiotics at Time of Septicemia	Outcome	Comments
79	Biliary	+	*Staph epi.*	+	+	1. UTI-gram negative rods 2. Positive bile culture for *Klebsiella & Enterocci*	Penicillin Chloramphenicol	Discharged	2 separate episodes of septicemia with the same organism traced to 2 different TPN catheters
67	Idiopathic	+	*Staph epi.*	+	–	None	Ampicillin	Died	Septicemia traced to pulmonary artery catheter inserted prior to institution of TPN
66	Trauma	+	*Staph. aureus*	+	+	UTI-*E. coli*	Cephalothin	Died	Death 2° to progressive pulmonary insufficiency unrelated to this episode of sepsis
63	Idiopathic	–	*Staph. aureus*	+	+	UTI-*E. coli*	None	Discharged	
62	Idiopathic	–	*Staph. aureus*	+	–	UTI-*E. coli*	None	Discharged	Septicemia traced to CVP line inserted prior to institution of TPN
42	Biliary	+	*Staph. aureus*	+	+	None	None	Discharged	1 episode protocol violation 7 days prior to septicemia
40	Hyperlipoproteinemia	+	*Enterocci*	+	+	None	Clindamycin	Discharged	Deviations from TPN dressing protocol noted
8	Trauma	+	*Staph. aureus*	+	+	UTI-*Candida albicans* ad *pseudomonas*	Cephalothin	Discharged	2 separate episodes involving different catheters with the same organism 15 days apart
90	Biliary	+	*Candida albicans*	–	–	1. Pyelonephritis-*Candida albicans* 2. Wound infection-*E. coli*	Penicillin Chloramphenicol	Died	
50	Biliary	+	*E. coli*	–	–	Biliary cultures grew *E. coli* and *Klebsiella*	None	Died	Septicemia followed T-tube cholangiogram
36	Traumatic	+	*E. coli*	–	–	Wound infection-*E. coli* and *Enterocci*	Cephalothin	Discharged	
25	Idiopathic	+	*E. coli*, *Candida albicans*	–	–	1. UTI-*Candida albicans* 2. Wound infection-4 gram negative rods	Penicillin Chloramphenicol	Died	
17	Post-op	+	*Proteus*, *Herellea*, *Pseudomonas*	–	–	Pancreatic abscess	Oxacillin Gentamycin Chloramphenicol	Discharged	

no need for surgical intervention, parenteral hyperalimentation can correct nutritional abnormalities. Support can be provided without the possibly increased risk of catheter sepsis by jejunostomy feedings in those patients who require surgical intervention for their underlying disease, provided the GI tract becomes functional.

We are unable to confirm the second assumption. The lack of effect of intravenous nutritional support on the complications of pancreatitis such as renal and respiratory failure suggests that this therapeutic tool is simply sophisticated support rather than active intervention directed against the poorly understood pathophysiology. Some experimental data does exist

indicating that hyperglycemia will reduce pancreatic exocrine secretion both in animal preparations[12] and in man.[17] It has also been demonstrated that malnutrition and specific amino acid deprivation can cause or exacerbate pancreatic inflammatory disease.[5,9,25] It must be reaffirmed, however, that no clear relationship exists between these experimental observations and the therapeutic options available or eventual clinical outcome in acute pancreatitis.

It is the third assumption which provokes pause. Clearly, the technical or metabolic considerations in hyperalimentation pose relatively minor problems which are easily met with experience and attention to detail. What is less clear and more disturbing are the implications of a very high incidence of catheter-related sepsis in patients with pancreatitis who are hyperalimented early in their course. Copeland *et al.*[3] have cast significant doubt on the concept that there are disease categories which have particular susceptibility to catheter complications with their report of a 2% incidence of catheter-related sepsis in patients hyperalimented during cancer chemotherapy with peripheral leukocyte counts of less than 2500 per cubic mm. However, the observations that 75% of the septic catheter complications occurred during the first two weeks of illness in our population suggests that the risk of catheter-related sepsis is higher in this period than in the later phases of this disease. Scrupulous attention to detail is essential in limiting the incidence of catheter sepsis.[2,20,23]

The results of this analysis of our experience in hyperalimentation of patients with acute pancreatitis prompt these conclusions: (1) In the treatment of acute pancreatitis, hyperalimentation should be seen as a method of support and not primary therapy. (2) Hyperalimentation in the early phase of acute pancreatitis is associated with a higher incidence of catheter-associated septicemia than normally seen. (3) In those patients with acute pancreatitis who require laparotomy for diagnosis or therapy, jejunostomy should be performed as a potential means of long-term nutritional management. (4) Intravenous nutritional support can be offered to patients with severe acute pancreatitis with a minimum of technical or metabolic morbidity. (5) Total parenteral nutrition may help salvage patients with prolonged GI tract dysfunction secondary to the complications of acute pancreatitis.

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