Acute Pancreatitis

April 2000

Pirmin Schmid
ps31415@gmx.net

Acute Pancreatitis

1 Introduction

There are about 185'000 new cases of acute pancreatitis per year in the United States [2]. Acute pancreatitis accounts for 3% of all cases of abdominal pain admitted to hospital in the United Kingdom [5]. True incidence is difficult to calculate and is estimated to be 2-28/100'000/y [5]. Although the disease is self-limited in 85-90%, it can become life threatening. Its overall mortality is estimated to be 10-15% [2, 5].

Acute pancreatitis can histologically be classified as interstitial edematous or as necrotizing. Severe pancreatitis is usually a result of pancreatic glandular necrosis. Mortality is estimated to be < 2% in interstitial edematous pancreatitis. Necrosis can be found in about 20%. In this case, mortality is increased to 20-30%. Mortality is directly related to the amount of necrosis. Infected necrosis develops in 40-60% of necrotizing pancreatitis and accounts for > 80% of deaths from acute pancreatitis. The risk of infected necrosis increases with the amount of pancreatic necrosis and the time from the onset of the disease, peaking at three weeks. Infected necrosis has a mortality of 20-65% with treatment, 100% without appropriate intervention [2, 3, 5, 11].

Gastrointestinal bleeding, infected necrosis, adjacent bowel necrosis, and development of pancreatic abscesses or pseudocysts are local complications of acute pancreatitis. Systemic complications include ARDS, acute renal failure, shock, coagulopathy, hyperglycemia, and hypocalcemia [2]. Late complications include endo- and exocrine glandular insufficiency. The mean quality-of-life outcome up to two years after treatment of necrotizing pancreatitis are similar to those obtained with coronary-artery bypass grafting [2].

Causes of acute pancreatitis are listed in table 1. Gallstones and alcohol abuse are the most common causes in the United States and Europe.

Table 1: Causes of Acute Pancreatitis

Common

- Biliary tract disease (gallstones) 45%
- Alcohol ingestion 35%
- Idiopathic 15%
- Drugs (diuretics, β-blockers, ACE-inhibitors, estrogens, glucosteroids, antibiotics, virostatics, NSAID, salicylates, cytostatics, contrast material,…)

Uncommon

- Trauma, postoperative state (abdominal or nonabdominal operations), ERCP
- Infections (viral, bacterial, Ascariasis)
- Metabolic causes (Hypertriglyceridemia, Hypercalcemia)
- Renal failure, after renal transplantation
- Acute fatty liver of pregnancy
- Hereditary pancreatitis
- Penetrating peptic ulcer
- Obstruction of the ampulla of Vater
- Regional enteritis
- Duodenal diverticulum
- Pancreas divisum

Causes to be considered in patients having recurrent bouts of acute pancreatitis without an obvious cause

• Occult disease of the biliary tree or pancreatic ducts, • Drugs, • Hypertriglyceridemia, • Pancreas divisum, • Pancreatic cancer, • Sphincter of Oddi dysfunction, • Cystic fibrosis, • Truly idiopathic

2 Diagnosis

2.1 Symptoms and Signs

Acute pancreatitis usually has a rapid onset of upper abdominal pain, nausea, vomiting, and fever. Abdominal pain is the major symptom, which characteristically is steady and boring in character. Classically, it is located in the epigastrium and periumbilical region and often radiates to the back as well as to the chest, flanks, and lower abdomen. The pain is frequently more intense when the patient is supine, and patients often obtain relief by sitting with the trunk flexed and knees drawn up.
Physical examination frequently reveals a distressed and anxious patient. Low-grade fever, tachycardia, and hypotension are fairly common, shock is not unusual. Jaundice occurs infrequently. Erythematous skin nodules due to subcutaneous fat necrosis may occur. In 10-20% of patients, there are pulmonary findings, including basilar rales, atelectasis, and pleural effusion. Abdominal tenderness and muscle rigidity may be present. Bowel sounds are usually diminished or absent. A pancreatic pseudocyst may be palpable in the upper abdomen.
Uncommonly, a faint blue discoloration around the umbilicus (Cullen's sign), and a blue-red-purple or green-brown discoloration of the flanks (Grey-Turner's sign) can be seen. Both of them indicate the presence of a severe necrotizing pancreatitis.
ST-T wave changes may be seen on electrocardiographs, but they usually differ from those of myocardial infarction. Abnormal Q waves do not occur as a result of pancreatitis.

2.2 Radiological Findings

Plain radiographs of the abdomen and thorax may show gallstones, a "sentinel loop" (a segment of air-filled small intestine most commonly in the left upper quadrant), the "colon cutoff sign" (a gas-filled segment of transverse colon abruptly ending at the area of pancreatic inflammation) or linear focal atelectasis of the lower lobe of the lungs with or without pleural effusion.
Ultrasound cannot be used for definitive diagnosis of pancreatitis since the gland is poorly visualized in 25-50% of cases. But it is a valuable tool in diagnosing free peritoneal fluid, gallstones, dilatation of the common bile duct and occasional other pathology such as abdominal aneurysm [5].
Computed tomography (CT) is useful in demonstrating an enlarged pancreas if diagnosis of pancreatitis is uncertain, in detecting pseudocysts and in differentiating pancreatitis from other possible intra-abdominal catastrophes. It is the most sensitive imaging modality in evaluation of acute pancreatitis.
CT scans are indicated in patients with acute pancreatitis (1) who clinically appear to have severe disease (e.g. shock), (2) whose prognostic indicators (e.g. Ranson's score) predict severe disease, (3) who do not improve after 3 to 4 days of conservative therapy, and (4) whose condition detoriates after treatment [2, 3].
Contrast-enhanced dynamic CT (CECT) of the abdomen is the gold standard for the noninvasive diagnosis of pancreatic necrosis, with an accuracy of more than 90% when there is more than 30% glandular necrosis [2].
CECT is of particular value after the first 3 days of severe acute pancreatitis to identify areas of pancreatic necrosis, although the use of intravenous contrast may increase the risk of renal failure. Contrast should be avoided when the serum creatinine level is greater than 1.5 mg/dL (130 µmol/L).

CT findings reflect the presence and extent of the retroperitoneal inflammatory process. In most cases the gland is enlarged, has an irregular contour, and the parenchyma appears heterogeneous. The peripancreatic fat is hazy if it is involved in the inflammatory process. The fat will show an increase in density accompanied by thickening of adjacent fascia.
In more severe cases, more extensive peripancreatic exudate can be seen. Exudate, necrotic tissue, and blood will be seen as poorly defined, irregularly contoured solid and fluid elements in the

peripancreatic regions. The exudates may extend along the pararenal spaces continuing inferiorly along the psoas muscle into the pelvis and rarely into the thighs.
Pancreatic necrosis can be seen as a definite, focal area of decreased enhancement compared with the normally enhancing pancreatic parenchyma. This lack of enhancement is due to destruction or thrombosis of vessels in the area of necrosis. In few patients, gas bubbles are evident on the CT study in the area of pancreas. These should be assumed to be product of bacterial fermentation from infection [3]. The presence of pancreatic calculi in addition to findings of acute pancreatitis signifies underlying chronic pancreatitis.
The extent of devitalized parenchyma appears to remain stable during an average follow-up of 4.5 months in two thirds of the patients [11]. Patients with increase in necrosis consequentially are more likely to require a surgical necrosectomy. There is no restoration of normal parenchymal enhancement in the previously necrotic area. A complete resorption of the necrosis with formation of a focal, fat-replaced cleft reminiscent of a scar can be observed 3 to 9 months after necrotizing pancreatitis in conservatively treated patients [11].

2.3 Diagnosis and Severity Stratification

The differential diagnosis of any severe acute pain in the abdomen or back should include acute pancreatitis. The diagnosis is usually entertained when a patient with a possible predisposition to pancreatitis presents with the clinical features mentioned above. Laboratory studies frequently reveal leukocytosis, hypocalcemia, and hyperglycemia. An elevated level of serum amylase (four times above normal) and/or lipase (twice above normal) usually confirms the diagnosis [5]. Obviously, not all features above have to be present to establish the diagnosis.
It is important to evaluate illness severity as soon as possible since mild and severe acute pancreatitis have completely different risks of complications, morbidity and mortality. Different scores are used in recent studies: Ranson's score (table 2) [2], the modified Glasgow score [5, 7] or the Acute Physiology and Chronic Health Evaluation score (APACHE II or III) [12, 13].
Severe acute pancreatitis is diagnosed if three or more of Ranson's criteria are present, if the APACHE II score is 8 or more, *or* if one or more of the following are present: shock, renal insufficiency, and pulmonary insufficiency [2].

Table 2: Ranson's score – severe pancreatitis, if ≥ 3 criterias fulfilled

At admission	
• Age	> 55 yr
• White-cell count	> 16'000/mm^3
• Blood glucose	> 200 mg/dL (11.1 mmol/L)
• Serum LDH	> 350 IU/L
• Serum AST	> 250 IU/L
During initial 48 hr	
• *Absolute decrease* in hematocrit	> 10 %
• USA: *Increase* in blood urea nitrogen (BUN)	> 5 mg/dL (1.8 mmol/L)
Europe: *Increase* in blood urea	> 11 mg/dL (1.8 mmol/L)
• Serum calcium	< 8 mg/dL (2 mmol/L)
• Arterial PaO_2	< 60 mm Hg
• Base deficit	> 4 mmol/L
• Fluid sequestration	> 6 liters

Number of criteria	**Estimated mortality rate**
0 – 2	1 %
3 – 4	16 %
5 – 6	40 %
7 – 8	100 %

The patient's clinical state has to be followed up very closely. Especially the complications mentioned above or signs of inflammation have to be sought actively. An elevated CRP level suggests the development of pancreatic necrosis [3].
Severe acute pancreatitis is usually a result of pancreatic glandular necrosis, which is present in about 20% of all cases of acute pancreatitis. Infected and sterile necrotizing pancreatitis can be difficult to distinguish clinically, since both may produce fever, leukocytosis, and severe abdominal pain. Nevertheless, this distinction is very important, because mortality among patients with infected acute necrotizing pancreatitis without intervention is nearly 100% [2, 3]. CT-guided fine-needle aspiration of pancreatic and peripancreatic tissue or fluid is safe and accurate with a sensitivity of 96% and a specifity of 99% [2]. It is recommended for patients with acute necrotizing pancreatitis whose clinical condition detoriates or fails to improve despite aggressive supportive care. Surveillance aspiration may be repeated weekly, as clinically indicated [2]. Evidently, the rules of asepsis must vigorously be observed to prevent iatrogenic contamination of necrotic tissue.
Pancreatic infections usually are caused by gram-negative enteric bacteria. The most frequently isolated pathogens are E. coli (35%), Klebsiella pneumoniae (24%), and Enterococcus spp. (24%). Less common are Staphylococcus spp. (14%), Pseudomonas (11%) and others. Anaerobic pathogens occur in about 6% of cases, fungal infections are seen frequently, especially in patients with antecedent antibiotic therapy [3].
The cause of an attack of acute pancreatitis should be sought as the aetiology will affect decision making and further therapeutic options [5]. Biliary origin can be assumed, if gallstones can be seen on ultrasonographic or computed tomographic scans or if two of the following are raised: Alkaline phosphatase, AST, bilirubin [4].

Complication rate and mortality seem not to be related to the initial enzyme increase (amylase/lipase). Therefore, it is important to recognize that the severity of the acute pancreatitis does not depend on the enzyme level elevation at admission. In particular, patients with alcohol induced acute pancreatitis have lower amylase levels on admission than patients with other causes of pancreatitis [6]. Severity of pancreatitis must be evaluated by close clinical assessment and by using one of the score systems mentioned above.

2.4 Differential Diagnosis

The differential diagnosis should include the following disorders: (1) perforated viscus, especially peptic ulcer, (2) acute cholecystitis and biliary colic, (3) acute intestinal obstruction, (4) mesenteric vascular occlusion, (5) renal colic, (6) myocardial infarction, (7) dissecting aortic aneurysm, (8) connective tissue disorders with vasculitis, (9) pneumonia, and (10) diabetic ketoacidosis.
Sometimes it may become a formidable task to elaborate the right diagnosis in time. It must be considered that serum amylase levels may also be elevated in high intestinal obstruction, in gut perforation or infarction [!], after abdominal surgery, in salivary gland diseases not involving the pancreas, in (ectopic) pregnancy, in renal insufficiency [!], certain tumors, after administration of narcotics, and other causes.

3 Treatment

In most patients (85-90%) with acute pancreatitis, the disease is self-limited and subsides spontaneously, usually within 3 to 7 days. Conventional measures include (1) analgesics, (2) intravenous fluids and colloids to maintain normal intravascular volume, (3) no oral alimentation, and (4) elective nasogastric suction to decrease gastrin release from the stomach and prevent gastric contents from entering the duodenum. A clear liquid diet can frequently be started on the third to sixth day, and a regular diet by the fifth to seventh day.

Drugs to block pancreatic secretion in acute pancreatitis have failed to have any therapeutic benefit. For this and other reasons, anticholinergic drugs are not indicated in acute pancreatitis. Several other drugs (including glucagon, H_2 blockers, protease inhibitors such as aprotinin, glucocorticoids, calcitonin, somatostatin analogues such as octreotide, and NSAID) have been evaluated by prospective, controlled trials and found ineffective in the treatment of acute pancreatitis.

The patient with *severe pancreatitis* needs the diagnostical, therapeutical and nursing advantages of an intensive care unit in addition. Close attention must be spent to complications mentioned on page 1.
Since the development of infected necrosis substantially increases mortality among patients with acute necrotizing pancreatitis, prevention of infection is critical. Imipenem-Cilastatin is the drug of choice as prophylactic antibiotic in radiographically proven necrosis. It should be continued for at least two to four weeks [2, 3]. Treatment of necrosis shifted away from early surgical débridement to aggressive intensive medical care with specific criteria for operative and nonoperative intervention [2, 3].
The current thinking is that primarily those patients with infection of the necrotic tissue benefit form surgical débridement and drainage. Viable pancreas should not be removed. The role of surgery in critically ill patients with *sterile* necrosis is still controversial [3].
In general, outcome is better when surgery can be postponed at least until the second week or later. Except in the unusual situation of fulminating acute pancreatitis, most patients should not be considered for surgery during the first week of illness *unless* pancreatic infection is present. Proven infection is an absolute indication for surgical intervention [3]. After detection of infected necrosis, the patient's condition should be optimized, and surgery should be undertaken within 24 to 48 hours in most cases [3].
The most common local postoperative complications are hemorrhage and intestinal fistulas. Cardiopulmonary complications accompany nearly 50% of the cases. Overall morbidity rates average about 80%. The best reported results in patients who have undergone surgery for infected necrosis are mortality rates of about 15% [3].
Alternative methods of débridement of necrosis (percutaneous drainage by interventional radiologists or endoscopic drainages) have recently been described. They require considerable technical expertise, and potential complications (sepsis, bleeding) may be life threatening. More experimental data is necessary to define the precise role of these techniques in management of necrotizing pancreatitis [2]. They cannot be recommended in general at the moment.

Nutritional support is important in long term therapies of critically ill patients. In absence of substantial ileus, enteral feeding by nasojejunal tube (placed beyond the ligament of Treitz) can be used as well as total parenteral nutrition (TPN). There are fewer total and infectious complications with enteral feeding than with the more expensive TPN [2]. But nasojejunal tubes have the risk to dislocate with following stimulation of the pancreas by gastric or duodenal feeding.

There is good evidence that *early endoscopic intervention* (ERCP with papillotomy) is the procedure of choice in patients with stone impaction and cholangitis. In contrast, a recent controlled, randomized trial of early endoscopic retrograde cholangiopancreatography (ERCP) and papillotomy in patients with acute biliary pancreatitis demonstrated that patients without biliary obstruction or biliary sepsis did not benefit from these interventions [4]. This suggests that this intervention is a reduction of biliary sepsis, rather than an improvement in pancreatitis [2].

Future medical therapies of severe acute pancreatitis might be improved by antagonists of proinflammatory cytokines. Antagonists of platelet-activating factor (PAF) have showed to improve survival in animal models and preliminary human studies [2].

4 Discussion

4.1 General Considerations

There are a few important key points in the management of acute pancreatitis:

- It is essential to pick the diagnosis early although it is not always easy to differentiate pancreatitis from other severe diseases.
- Clinically severe acute pancreatitis must be recognized as soon as possible by close clinical assessment, using one of the scoring systems, and imaging methods.
- Intensive care unit management for clinically severe acute pancreatitis includes supportive care, prophylactic use of antibiotics for radiographically documented pancreatic necrosis, and prevention of pancreas-activation by nutritional support with nasojejunal tube or TPN.
- Strong consideration of urgent ERCP with papillotomy for gallstone pancreatitis when jaundice or cholangitis is present.
- Identification of infected necrosis by CT or sonographically guided fine-needle aspiration.
- Débridement of infected necrosis (according to the old but wise rule *"ubi pus, ibi evacua"*).

4.2 Remarks About the Value of Imaging Methods in Acute Pancreatitis

The reported abdominal plain film findings (page 2) in acute pancreatitis are unreliable and non-specific. They cannot be recommended for use in diagnosis [5]. Even gallstones cannot be detected on plain films in 90%. Chest films are useful in documentation of pleural effusions and signs of ARDS. Quality of ultrasound examination is very user dependent. Ultrasound has its restrictions mentioned on page 2. Nevertheless, it may be helpful in confirming the diagnosis. All of these imaging methods are useful in ruling out differential diagnoses of acute pancreatitis.

New MRI-technologies (e.g. [8]) can be used to evaluate presence and extent of necrosis in acute pancreatitis [9]. They eliminate the radiation burden of multiple CT scans. Computed tomography, however, retains several significant advantages: (1) It is more widely accessible and less costly. (2) The environment of CT is more favorable for dealing with severely ill patients. (3) CT is more sensitive than MRI in detecting small gas bubbles and calcifications. (4) The insertion and monitoring of punction and drainage devices is more readily accomplished with CT than with MRI, although emerging technology (open-magnet systems) reverse this difference [9].

Current magnetic resonance cholangiopancreatography (MRCP) techniques [1] have reached the stage where they may be considered as primary imaging approach for suspected biliary and pancreatic ductal disease [1, 9]. The lower cost, absence of ionizing radiation, and avoidance of complications of ERCP (e.g. acute pancreatitis) make MRCP an attractive diagnostic method. ERCP retains diagnostic superiority and is essential for therapeutic manoeuvres [9]. Therefore, MRCP cannot replace ERCP in cases of acute pancreatitis.

4.3 Remarks About Disease Severity Assessment Scores

There are different general and disease-specific scoring systems to assess severity of acute pancreatitis [2, 3, 5, 7, 12, 13]. They are used in clinical trials and practical work. But there are only a few studies that compare these scores [12, 13]. This makes it more difficult to compare scientific trials since they do not use the same criterias to assess patient's state.

Some characteristics of these methods in predicting severity of acute pancreatitis are summarized in table 3. Clinical assessment has an excellent specificity. There is no doubt that patients, who clinically appear to be very ill, *are* very ill. But many patients with severe pancreatitis are missed by clinical assessment alone. Scores have been developed for this, but all of them have limited sensitivity and specificity. Furthermore, severity of acute pancreatitis as judged by imaging criteria (e.g. Balthazar

score) does not correlate well with clinical severity (e.g. using Ranson's score or APACHE) [9]. But it should be noted that a close correlation between early CT grades and risk factor scores is not necessarily expected, because CT grades primarily local complications, whereas scoring systems evaluate systemic complications.

Table 3: Characteristics of different methods in predicting severe acute pancreatitis

	Sensitivity [%]	Specificity [%]	PPV [%]	NPV [%]	Accuracy [%]
Clinical assessment on admission	34	98	87	83	83
Clinical assessment at 24h	47	100	100	86	87
APACHE II (>7) on admission	68	67	40	87	68
APACHE II (>9) peak in day 1 to 3	82	74	50	93	76
Modified Glasgow score	71	88	66	91	84
Ranson's score	87	71	49	94	75
CECT detects glandular necrosis [2]					90
CRP > 210 mg/L (peak in day 1 to 4) [5]					80
CRP & LDH [3]					84
IL-6 during the first 24h [3]	100	86			91

Data from [13], unless indicated otherwise. PPV = positive predictive value, NPV = negative predictive value.
CECT: this accuracy has been measured in cases where necrosis is > 30% of gland. CRP & LDH was considered positive if CRP > 120mg/L and LDH > 270mg/L. Interleukin-6 (IL-6) tests are not currently available for routine laboratory use.

Although each score is useful, none of them is completely perfect. Both Ranson's score and the modified Glasgow score may need up to 48 hours to be calculated. They are much more sensitive than clinical assessment and can separate mild and severe disease quite well (accuracy). Both of them may help exclude severe pancreatitis since they have a high negative predictive value.
APACHE is much more complicated to calculate (needs a computer) but has a few advantages: It can be calculated earlier, and it can be a monitoring tool during the whole course of pancretitis. Consequently, it is more important to integrate one of these scores into a prudent and comprehensive framework than which score is used in detail.
Patients are at risk to die because of local complications (e.g. infection) *or* systemic complications (e.g. ARDS, acute renal failure, shock, coagulopathy). Therefore, this management framework should at least consist of repeated close clinical assessment, one of the scores, parameters of inflammation (fever, CRP), imaging methods, and elective invasive diagnostic procedures.

4.4 Conclusion

Büchler and Reber [3] wrote: "The decision about whether and when to operate on these patients [with necrotizing pancreatitis] is often difficult, and *it requires mature clinical judgment*." In my opinion, this statement can be generalized to the entire management of patients with suspected acute pancreatitis – it requires mature clinical judgment. A book founded by Sir Zachary Cope [10] must be mentioned in this context.

5 References

References 2, 3, and 5 are state of the art review articles, which give quite a comprehensive overview of most important facts and ideas in management of acute pancreatitis. References 1, 8, 9, and 11 discuss special radiological topics.

1. Barish MA, Kent Yucel E, Ferrucci JT: Magnetic resonance cholangiopancreatography. N Engl J Med 1999;341:258-64
2. Baron TH, Morgan DE: Acute necrotizing pancreatitis. N Engl J Med 1999;340:1412-7
3. Büchler P, Reber HA: Surgical approach in patients with acute pancreatitis. Gastroenterology Clinics of North America 1999;28:661-71

4. Fölsch UR, Nitsche R, Lüdtke R, Hilders RA, Creutzfeldt W and the German Study Group on Acute Biliary Pancreatitis: Early ERCP and papillotomy compared with conservative treatment for acute biliary pancreatitis. N Engl J Med 1997;336:237-42
This randomized controlled trial (RCT) showed that early ERCP is no benefit for patient with acute pancreatitis without signs of cholangitis or persistent stone obstruction.
5. Glazer G, Mann DV et al.: United Kingdom guidelines for the management of acute pancreatitis. Gut 1998;42(Suppl 2):S1-13
Evidence based medicine: The strength of evidence is indicated for all citations and for each recommendation in this paper.
6. Lankisch PG, Burchard-Reckert S, Lehnick D: Underestimation of acute pancreatitis: patients with only a small increase in amylase/lipase levels can also have or develop severe acute pancreatitis. Gut 1999;44:542-4
Amylase and lipase levels at admission are no prognostic signs. Severity of pancreatitis must be assessed otherwise.
7. Moore EM: A useful mnemonic for severity stratification in acute pancreatitis. Ann R Coll Surg Engl 2000;82:16-7
A useful mnemonic for the modified Glasgow score is presented in this paper:

Mnemonic letter	***Criteria***	***Positive if***
P	*PaO_2*	*< 60 mm Hg*
A	*Age*	*> 55 yr*
N	*Neutrophils, i.e. WBC*	*> 15'000/mm^3*
C	*Calcium*	*< 2 mmol/L (8 mg/dL)*
R	*Raised urea*	
	- absolute urea	*> 16 mmol/L (96 mg/dL)*
	- absolute blood urea nitrogen (BUN)	*> 16 mmol/L (45 mg/dL)*
E	*Enzyme - LDH*	*>600 IU/L*
A	*Albumin*	*< 32 g/L*
S	*Sugar - glucose*	*> 10 mmol/L (180 mg/dL)*

Severe pancreatitis, if ≥ 3 criterias positive on admission or in subsequent repeated tests over 48h.

8. Piironen A, Kivisaari R, Kemppainen E, Laippala P, Koivisto AM, Poutanen VP, Kivisaari L: Detection of severe acute pancreatitis by contrast-enhanced magnetic resonance imaging. Eur Radiol 2000;10:354-61
A "breath-hold multislice rapid gradient-echo turbo fast low-angle shot (FLASH) sequence" can be used to detect pancreas necrosis.
9. Robinson PJA, Sheridan MB: Pancreatitis: computed tomography and magnetic resonance imaging. Eur Radiol 2000;10:401-8
This review compares MRI and CT, MRCP and ERCP, and discusses actual knowledge and clinical conclusions of the experimental finding that CT contrast material may impair pancreatic micro-circulation.
10. Silen W: Cope's early diagnosis of the acute abdomen. 19th ed., Oxford University Press, New York, 1996
"If only one book about surgery could be made available to physicians from all specialties, it should probably be [this book]" (review of the 18th edition in N Engl J Med). What shall I write more…
11. Vitellas KM, Paulson EK, Enns RA, Keogan MT, Pappas TN: Pancreatitis complicated by gland necrosis: evolution of findings on contrast-enhanced CT. J Comput Assist Tomogr 1999;23:898-905
Long-term evolutions of CT findings have been studied in the rare cases of necrotizing pancreatitis, in which the patient did not need surgery and did not die.
12. Williams M, Simms HH: Prognostic usefulness of scoring systems in critically ill patients with severe acute pancreatitis. Crit Care Med 1999;27:901-7
Three different scoring systems are compared in this retrospective study of patients admitted to an ICU with severe pancreatitis: Ranson's score, the modified Glasgow score, and APACHE III. In my opinion, the terms "GCS" and "modified Glasgow Coma Scale", which are used by the authors of this study, should be read as "modified Glasgow score", ranking from 0 to 8 [5, 7]. The "Glasgow Coma Scale", generally known as GCS and ranking from 3 to 15,

may be used in the evaluation of patients with pancreatitis, but it obviously has a different meaning than the "modified Glasgow score". By the way, GCS is part of APACHE.

13. Wilson C, Heath DI, Imrie CW: Prediction of outcome in acute pancreatitis: a comparative study of APACHE II, clinical assessment and multiple factor scoring systems. Br J Surg 1990;77:1260-4
A general ICU scoring system (APACHE II), two disease specific systems (Ranson's and modified Glasgow score), and clinical assessment are compared in this prospective study with patients admitted to a hospital with pancreatitis of unknown severity.
A comprehensive documentation of the latest version of the "Acute Physiology and Chronic Health Evaluation" score – APACHE III – can be found in the following supplement: Crit Care Med 1989;17(12 Pt 2):S168-221.