Pancreatitis

Organ specific problems

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Module Author (Update 2010)

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FACULTY DISCLOSURES

The authors of this module have not reported any disclosures.

DURATION

7 hours

Learning Objectives

After studying this module on Pancreatitis, you should be able to:

1. Recognise the patient at risk. Diagnose acute pancreatitis and determine the severity, aetiological factors and complications.
2. Manage severe acute pancreatitis with appropriate use of supportive therapy for organ function, antibiotics and surgery.
3. Feed the patient with acute pancreatitis. Determine the nutritional needs of patients with acute pancreatitis and the optimum mode of delivery.
4. Identify and manage local and systemic complications of acute pancreatitis.
INTRODUCTION

Epidemiology of acute pancreatitis

Severe acute pancreatitis is still an alarming condition, which presents many challenges for Critical Care staff. Relevant epidemiological data are:

- Reported incidence ranges from 21 to 900 cases per million, per year. Although fluctuating due to environmental and diagnostic differences the true incidence of acute pancreatitis is steadily increasing. This reflects increased alcohol intake amongst the young but also an increase in gallstone disease in some areas.
- Overall mortality rate ranges from 2 to 10% 10.7–14% (see Eland reference, below) but reaches 10 to 40% in necrotising forms. The fatality rate has decreased during the past two decades. This is ascribed in part to an increase in the number of mild attacks, but also reflects advances in patient care, particularly in the area of intensive care.
- Those greater than 60 years old are at the highest risk of death as a consequence of comorbidity.
- The male/female ratio ranges from 1/1.2 to 1/1.5. There is a definitive prevalence amongst females for biliary pancreatitis, and amongst males for acute pancreatitis secondary to alcohol abuse.


Acute pancreatitis covers a wide clinical spectrum that includes the systemic inflammatory response syndrome (SIRS), sepsis, multiple organ failure (MOF) and death. Patients with acute pancreatitis are amongst the most labour and resource intensive in intensive care medicine. They are typically ill for an extended period and require numerous medical, surgical, radiological and nutritional interventions if they are to survive.

Diagnosis may at times be difficult. There is no other abdominal problem where patient outcome is so unpredictable at onset, as severe local and subsequent remote complications may arise unexpectedly. In addition the pathogenesis is incompletely understood so that specific treatment is still lacking.

Communication

All through the course of an attack it is essential that one adopts a multidisciplinary approach. Throughout the diagnostic work-up, severity stratification and subsequent management, intensivists, surgeons, gastroenterologists skilled in endoscopy and interventional radiologists work as a team to ensure the best possible patient outcome.
This disease is so variable that it cannot be effectively managed by blindly following any given set of recommendations. Nevertheless, in your daily practice, you should refer to the consensus statements that focus on the management of the critically ill patient with severe acute pancreatitis.


1. HOW TO RECOGNISE THE AT RISK PATIENT


You should first be able to recognise that the patient has acute pancreatitis, then make an early assessment of its severity, recognise the aetiological factors and finally detect and anticipate complications by monitoring the course of the attack.

**Note** It is important to consider this global approach in the management of these patients. Each step of this complex process will be covered in this and subsequent Tasks.

**Management steps in acute pancreatitis**
Diagnosis of acute pancreatitis

Diagnosis can be straightforward when the following key signs are present:

- Acute upper abdominal pain and tenderness
- Nausea and vomiting
- Increase of serum amylase or lipase more than three times the upper limit of normal.

These clinical and biochemical signs are however non-specific. Several abdominal problems, such as hollow viscus perforation or mesenteric infarction may present with similar diagnostic features. The difficulty in making a diagnosis is illustrated by the finding that, in some patients, the diagnosis is not established until autopsy. For further information see the following reference.


Be aware of the clinical and biochemical pitfalls in making a diagnosis of acute pancreatitis and be familiar with biochemical assays and imaging procedures that help you to overcome diagnostic uncertainties.

Diagnostic pitfalls

Pancreatitis without pain is particularly misleading. Lack of a major symptom is usually attributed to a postoperative situation where analgesics/sedatives are in use. Diabetic coma, severe hypothermia, or remote organ failures such as shock, severe gastrointestinal bleeding and respiratory distress may however at times be the presenting feature of acute pancreatitis and conceal abdominal pain.

Diagnostic clues

- Use the lipase assay alone or in combination with amylase. It is more sensitive and specific and remains elevated longer than the serum amylase. (Note: in patients with renal failure, an elevated amylase may be a false-positive.)
- A plain abdominal radiograph may aid diagnosis in the case of hollow viscus perforation, and may in addition show local ileus in cases of pancreatitis.
- When the diagnosis is in doubt, use ultrasound-guided peritoneal aspiration if fluid is present in the peritoneal cavity: Gram staining may
suggest peritoneal infection which is unusual in acute pancreatitis and necessitates urgent laparotomy. Fluid rich in amylase is of diagnostic value in acute pancreatitis.

**Imaging**

A computed tomography (CT) scan is the most accurate diagnostic tool and should be done whenever there is doubt and in any case when a patient fails to improve after three days. Before interpreting abdominal CT images, consider the normal anatomy of the retroperitoneal area.

Useful websites; in the third you will find images of the retroperitoneal area.

http://www.instantanatomy.net/
http://www.medicdirect.co.uk/virtual_body/default.ihtml
http://rad.usuhs.mil/medpix/

On a CT (preferably with nonionic intra-venous radiocontrast) the normal pancreas is seen as an area of homogeneous density with smooth contours and with regular contrast enhancement of the gland.

**Diagnostic clues to acute pancreatitis on a CT scan include:**

- Pancreatic swelling (arrow 1)
- Areas of spontaneous attenuation in the gland
- Areas which lack enhancement during bolus IV contrast administration (arrow 2)
- (Peri)pancreatic fluid collection(s) (arrow 3)
These signs usually persist for weeks and are almost 100% diagnostic. Areas of necrosis (recognisable as lack of contrast enhancement) may increase with time. In addition CT may provide diagnostic assistance where enzyme levels are misleadingly elevated or coexistent extra-pancreatic disease is present.

If you want to obtain more information on the use of CT in acute pancreatitis refer to:


In your daily practice identify those patients who present with an abdominal emergency. Using clinical history and physical examination, see how many have symptoms that are consistent with acute pancreatitis both clinically and biochemically. Build a differential diagnosis based on clinical, biochemical and radiological findings.

**Note**

**Have a high index of suspicion for the diagnosis** of acute pancreatitis whenever there is unexplained remote organ failure especially in unconscious patients and in patients with a history of gallstones or alcoholism.

You will find useful reviews of diagnostic tools in:


**Note**

A timely diagnosis of acute pancreatitis is essential in order to ensure appropriate therapeutic intervention.
Early assessment of severity

Together with early diagnosis, assessment of disease severity at admission is central to appropriate clinical management. Prognostic criteria can be classified into four categories: clinical signs, biochemical indicators, multi-factor grading systems and imaging procedures.

Clinical assessment

During the early phase of acute pancreatitis many patients look clinically better than they actually are. Clinical assessment of severity is therefore unreliable on admission and misclassifies around 50% of patients. However in most cases severity becomes clinically obvious within the first 48h.

Within 48h of admission, clinical assessment is as accurate, if not better than any other means of assessing the prognosis of acute pancreatitis. Never underestimate the importance of clinical evaluation. Keep returning to assess the patient every few hours and look systematically for the clinical signs shown in the table below.
Most of these clinical signs are a manifestation of an exaggerated local and systemic inflammatory response, which typically manifests in the first few days and potentially have a high mortality. A careful and continuing clinical assessment therefore remains invaluable to implement preventive and therapeutic measures.

A thorough discussion on how to integrate clinical assessment with other prognostic indicators can be found in:

Flank Ecchymosis

**Multiple prognostic criteria**

Several multi-factor grading systems have been devised to identify patients at higher risk. The two most popular, both of which were specifically developed for acute pancreatitis, are the Ranson and Glasgow (Imrie) scoring systems.

**Glasgow (Imrie) scoring system**

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial PO2</td>
<td>&lt;60 mmHg (8.0 kPa)</td>
</tr>
<tr>
<td>Serum albumin</td>
<td>&lt;32 g/l</td>
</tr>
<tr>
<td>Serum calcium</td>
<td>&lt;8 mg/dl (2 mmol/l)</td>
</tr>
<tr>
<td>White cell count</td>
<td>&gt;15 x 10⁹/l</td>
</tr>
<tr>
<td>AST</td>
<td>&gt;200 iu/l</td>
</tr>
<tr>
<td>LDH</td>
<td>&gt;600 iu/l</td>
</tr>
<tr>
<td>Blood glucose</td>
<td>&gt;180 mg/dl (10 mmol/l)</td>
</tr>
<tr>
<td>Plasma urea</td>
<td>&gt;45 mg/dl (16 mmol/l)</td>
</tr>
</tbody>
</table>

Consider the worst value during the first 48h and if >2 adverse prognostic criteria: Severe attack

The basic principles of the Ranson score are similar to the Glasgow scoring system. These disease specific scores are cumbersome to use, as they need 48 hours to be determined and are flawed by a high false-positive rate. You can find further details in the following references.


See also the PACT module on Clinical outcome

**Q. Why are the Ranson and Glasgow scores of limited value in the later stages of the disease?**
A. The Ranson and Glasgow scores only include clinical and laboratory data from the first 48 hours. They primarily determine the risk of early complications and death. Monitoring of pancreatic necrosis and accurate prediction of late septic complications are impossible. In addition they do not take account of the effects of treatment nor the influence of inappropriate therapy upon measured parameters.

**Note** It may be better to use the Acute Physiological and Chronic Health Evaluation (APACHE II) scoring system. Although not originally designed for acute pancreatitis, a score of at least 8 points classifies patients as being in the severe category. Unlike Ranson and Glasgow scores, an APACHE II score can be obtained at the outset.

*Individual outcome relates closely to the severity of remote organ dysfunctions: early (on admission), multiple, persistent or evolving organ dysfunctions carry the worst prognosis*

Be aware that the scoring systems are not 100% accurate. It is easy to identify the extremely sick patient, and also the very mild attack, but the majority between the two ends of the spectrum are far more difficult to classify.

See the following for a review of the scoring systems in acute pancreatitis:


PACT module on Clinical outcome
**Single biochemical indicators**

Several biochemical assays of products released by the pancreas, or resulting from the inflammatory reaction, have been developed to identify severe disease and/or pancreatic necrosis before the occurrence of multiple organ dysfunction.

None are regularly used in clinical practice, except the serum CRP (C-reactive protein): a value >150 mg/dl indicates severe disease and necrosis, but the rise in CRP is often delayed for 48-72 hours after onset of disease.


**Imaging procedures**

Contrast-enhanced CT is the most popular imaging method for quantifying (peri)pancreatic necrosis. Necrosis can be detected by CT as a focal or diffuse area of diminished pancreatic parenchymal contrast enhancement (<30 Hounsfield units) with an overall accuracy of 90%. However it may take four days for necrosis to become fully established.

**THINK** about the diagnostic value of CT scans throughout the course of the illness for assessment of severity.

Several scoring systems based on contrast-enhanced CT scans can be used to assess the severity of acute pancreatitis. You should be aware of the Balthazar CT Severity Index which takes into account both the extent of pancreatic necrosis and the number of abdominal fluid collections, denoting the intensity of the local inflammatory process. The score ranges from 0 to 10 points and is obtained by adding the points attributed to the extent of the inflammatory process to the volume of necrosis (see table below). Note: These patients are at risk for contrast nephropathy.

When contrast dye is contraindicated, magnetic resonance imaging (MRI) with gadolinium enhancement is as accurate as CT in documenting the degree of pancreatic necrosis and staging the severity of acute pancreatitis, but accessibility for the critically ill is difficult.
**CT Severity index of acute pancreatitis**

<table>
<thead>
<tr>
<th>Inflammatory Process</th>
<th>Score</th>
<th>Sub-totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>A</td>
<td>0</td>
</tr>
<tr>
<td>Focal or diffuse enlargement</td>
<td>B</td>
<td>1</td>
</tr>
<tr>
<td>Contour irregularity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inhomogeneous attenuation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B+ peripancreatic haziness/mottled densities</td>
<td>C</td>
<td>2</td>
</tr>
<tr>
<td>B, C + 1 ill-defined peripancreatic fluid collection</td>
<td>D</td>
<td>3</td>
</tr>
<tr>
<td>B, C + 2 ill-defined peripancreatic fluid collections</td>
<td>E</td>
<td>4</td>
</tr>
<tr>
<td><strong>Necrosis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 30%</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>50%</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>&gt; 50%</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Look at the CT scans that are presented with the first two 'patient challenges' and evaluate the CT Severity Index according to the table above.


Balthazar EJ. Acute pancreatitis: assessment of severity with clinical and CT evaluation Radiology. 2002; 223(3): 603-613. Review. PMID 12034923


There is no clear relationship between the development of multiple organ failure and the extent of (peri)pancreatic necrosis. However, the latter is closely related to the incidence of local complications, particularly infection, and outcome.

*Severity = necrosis ... but the reverse does not hold true!*

*CT is not an early (first 3 days) indicator of severity unless you focus on extrapancreatic signs of inflammation*
Task 1. How to recognise the at risk patient

Unless there is doubt about the diagnosis in a critically ill patient, postpone the initial CT scan until 72h after onset of symptoms in order to facilitate identification of necrotic areas and avoid underestimating severity. On the other hand, you may use CT within 24h for prognostic purposes if you restrict your evaluation to the extent of extrapancreatic inflammatory fluid collections.

You will find the description of the EPIC score in:


Q. What are the two main indications for the early use of a CT scan in patients with acute pancreatitis?

A. Patients with clinically persistent severe acute pancreatitis after 72 hours of conservative medical treatment.
Patients who demonstrate clinical improvement initially but then manifest an acute change in clinical status suggesting the development of a complication.

Prognostic stratification

Early prognostic assessment is best achieved by a combination of repeated clinical examination, APACHE II score and CT. The primary goal of this approach is to identify those patients who are likely to need intensive or high dependency care given the risk for early organ dysfunction(s), life-threatening local complications and the need to prevent or reverse organ injury (see Tasks 2 and 4). Accurate and prompt stratification will become more important when specific agents or strategies are introduced for treatment of the inflammatory necrotising process.

In the next five patients admitted to the emergency unit with acute pancreatitis, assess the severity of the attack using the prognostic tools discussed. Follow up the patient until discharge from hospital and reflect on the accuracy of each prognostic marker.
The presence of organ dysfunction is not truly a predictive system but compared to the extent of necrosis, it is a reliable marker of severe disease. Once established, use dynamic scores of organ dysfunction to provide a daily assessment as changes in organ function are a valuable predictor of outcome. For further information on how to assess the dynamics of organ dysfunction and its prognostic importance in acute pancreatitis, see:


Johnson CD, Abu-Hilal M. Persistent organ failure during the first week as a marker of fatal outcome in acute pancreatitis Gut 2004; 53(9): 1340-1344. PMID 15306596

PACT module on Clinical outcome

Aetiological assessment

In Western industrialised societies, alcohol abuse and choledocolithiasis account for approximately 45% and 35% of cases, respectively. Numerous other aetiological factors, albeit rare, have been identified. Some are obvious (post trauma, surgery, endoscopic retrograde cholangiopancreatography (ERCP) and antiretroviral therapy) but sometimes a detailed history and work-up is required to identify the cause.

A biliary aetiology should be suspected in females over 40 years of age with a serum alanine aminotransferase level >3 times the upper reference limit. The most sensitive diagnostic tool is an ultrasound study, which should be performed in all patients on admission. Gallstones can be detected with 70-90% accuracy. Bile duct stones are more difficult to see, and an invasive procedure is usually required: endoscopic ultrasound, ERCP or Magnetic resonance cholangiopancreatography (MRCP) (see below).

You should be aware of these aetiological factors as some of them impact significantly either on the severity of the attack or on the risk of recurrence. Rapid identification of the cause is required so as not to delay specific therapeutic interventions.

You will find details of aetiological factors and the associated diagnostic work-up in the following reference.


Q. What are the commonest causes of acute pancreatitis?

A. In Western industrialised societies, choledocolithiasis and alcohol abuse, account for approximately 45% and 35% of cases, respectively. However these percentages will vary from place to place according to economic and cultural factors.

Q. Do you think aetiological factors impact on prognosis?

A. There may be a weak association. Postoperative, idiopathic, ERCP and antiretroviral agent-induced acute pancreatitis have been associated with a higher mortality rate, in part due to co-morbidity and delay before diagnosis and institution of specific therapeutic interventions. However once acute pancreatitis has been initiated, it is unlikely that the underlying aetiological factor has a further influence on the disease itself, provided specific therapy is timely.

Q. Which operative procedures carry the greatest risk for postoperative acute pancreatitis?

A. Operations on the upper part of the abdomen, particularly those involving the gallbladder and stomach. Among operations remote from the pancreatic area cardiac surgery is a recognised precipitating cause. These patients are notoriously difficult to assess.

Management of biliary pancreatitis

The ducts

Acute biliary pancreatitis is due to the impaction – albeit often transient – of bile duct stones in the sphincter of Oddi. This has led endoscopists to propose early decompression of the common bile duct by endoscopic sphincterotomy (ES), in patients with clear evidence of cholestasis.
Sharma VK, Howden CW. Metaanalysis of randomized controlled trials of endoscopic retrograde cholangiography and endoscopic sphincterotomy for the treatment of acute biliary pancreatitis Am J Gastroenterol 1999; 94(11): 3211-4. PMID 10566716

**Raised pressure in the pancreatic duct triggers acute pancreatitis at the cellular level**

**Early (within 24–48h after admission): Consider endoscopic sphincterotomy (ES) in severe biliary pancreatitis associated with either cholangitis or persistent distant organ dysfunction. Endoscopic clearance of bile duct stones has benefits in terms of morbidity/mortality in these cases. Broad-spectrum antibiotic prophylaxis should be prescribed, assuming the presence of cholangitis, otherwise pancreatic infection may result from biliary reflux.**

**In patients with biliary pancreatitis:** within the first 48 hours 75% show stones in the common bile duct ... but 90% pass spontaneously into the duodenum

**Complications of ES are rare when performed by an of experienced operators**

**ERCP and ES may aggravate the inflammatory process and carry the risk of bacterial contamination of necrosis, duodenal perforation and haemorrhage. In the patient with remote organ failures including coagulopathy, ES is a technical challenge. It may be wise to withhold such a procedure as an alternative drainage mechanism may be possible either via endoscopically inserted biliary catheter which bypasses (or even dislodges) the impacted stone by percutaneous hepato-biliary drainage.**

Investigations directed at the common bile duct should be expeditiously carried out in patients with persistent organ dysfunction in the setting of acute biliary pancreatitis and in those with increasing jaundice, persistent dilatation of the common bile duct, biliary pain and high fever. Due to the transient nature of stone
impaction and the potential morbidity of endoscopic interventions, persistent impaction or at least the presence of retained common bile duct stone(s) should be confirmed prior to ES. Endoscopic ultrasound is highly accurate and safer than ERCP to detect stones in the common bile duct as it does not require injection of dye into the pancreatic duct. Magnetic resonance cholangiopancreatography (MRCP) offers comparable accuracy and also has the potential to limit the use of invasive transpapillary imaging for interventional use. Limited availability, absence of immediate curative possibility and practical difficulties are serious limitations to the wider use of MRCP in severe, acute, biliary pancreatitis.

**Stone impaction**

![Stone impaction](image)

**The gallbladder**

Removal of the gallbladder should be scheduled early to avoid recurrence (of biliary pancreatitis). Recurrence rates are around 30% after 3 months. In severe attacks, it is recommended to wait 4–6 weeks in order to allow inflammation to subside. The laparoscopic approach has proved to be feasible and safe, even in cases where surgical debridement is required.

**Q. Is sphincterotomy a safe procedure that should be performed in all cases of biliary pancreatitis?**

**A.** Endoscopic sphincterotomy is a difficult and risky procedure when performed in severe acute pancreatitis, due to the local inflammation. Only expert interventional endoscopists should undertake this. The procedure has proven beneficial only in severe attacks with persistent common bile duct stones and in those associated with frank cholangitis.

**Q. After endoscopic sphincterotomy, is cholecystectomy still necessary?**

**A.** Endoscopic sphincterotomy avoids recurrence of biliary pancreatitis but predisposes to cholecystitis or biliary colic. Therefore (interval) cholecystectomy is recommended, except in patients with a short life expectancy or in those who are not fit for surgery.
Q. If surgical debridement is required in biliary pancreatitis, should a biliary procedure be associated?

A. Usually, surgical debridement is carried out 21–40 days after the onset of acute pancreatitis, and where there is no remaining problem with retained bile duct stones. At surgery, the gallbladder should be removed if technically possible and intraoperative cholangiography performed if there is any doubt about the patency of the bile duct.

You may wish to extend your knowledge on this subject, see:

PMID 10503139


Ongoing assessment

The clinical course of severe acute pancreatitis can be divided into an early 'toxaemic' phase (0-15 days) characterised by the emergence of remote organ dysfunction and a later 'necrotic' phase when local complications in particular infection, prevail. What sets acute pancreatitis apart from other non-infective diseases of the gastrointestinal tract is its propensity to amplify the local necrotising process through the induction of a systemic inflammatory response. It is this amplification that dictates most of the remote organ injury. Release into the systemic circulation of activated enzymes, vasoactive substances, and proinflammatory mediators generated retroperitoneally, as well as secondary activation of immune effector cells in distant organs, lead to early widespread organ damage.

The concepts of the systemic inflammatory response (SIRS) and multiple organ dysfunction syndrome (MODS) are covered in:


PACT modules on Sepsis and MODS and Pyrexia

All patients with severe acute pancreatitis require ongoing assessment at least on a daily basis to monitor progress during the course of the attack, to allow early diagnosis of local and/or systemic complications and to guide appropriate therapy.
This ongoing assessment is complementary to the early classification of severity and consists of clinical, biochemical and radiological investigations.

**Clinical evaluation**

The most useful signals of impending remote organ dysfunction are the early clinical signs used to assess severity. The ICU environment offers additional monitoring tools. Local complications are dominated by infection. Clinical features alone cannot differentiate between sterile and infected necrosis but you should suspect infection in the presence of persisting or worsening distant organ failure, high fever, a prolonged ileus, abdominal distension and tenderness. Appropriate investigations e.g. repeat CT scan should then be organised.

**Biochemical assessment**

There are limitations to biochemical assessment of remote organ dysfunction. An increase in the leukocyte count, the APACHE II score and CRP are indicators of both sepsis and inflammation and are therefore *non-specific*.

**Radiological assessment**

CT scanning is the most useful investigation for detecting and monitoring local complications and in particular pancreatic infection. In severe acute pancreatitis, this investigation should be repeated on a regular basis, usually every week or at shorter intervals if dictated by a sudden deterioration in clinical or biochemical status. Beyond its diagnostic value, CT scanning provides invaluable guidance for interventional (either percutaneous or surgical) procedures directed at necrosis.

**Early diagnosis of pancreatic infection is crucial:** antimicrobial therapy (after blood and other cultures taken) and resuscitative measures may be required and urgent operative intervention or drainage may be warranted. Refer to Task 4.

**Q. What are the three major determinants of outcome from acute pancreatitis?** You may find the following reference helpful.

A. The intensity of the early amplification of the inflammatory response
The development and extent of local necrosis
Secondary microbial contamination/infection of necrosis.

2. Management of severe acute pancreatitis

Over the last two decades advances in supportive therapy of failing organs have enabled most patients to survive the early toxaemic phase of pancreatitis and have decreased the overall mortality rate associated with this disease. Death from single or multiple organ failure, such as cardiovascular collapse, is now rare except in those with co-morbidities. Although early multiple organ dysfunction syndrome (MODS) still carries significant morbidity, patients' outcome is now primarily influenced by the volume of necrotic areas and their secondary infection.


Implement supportive therapy early for remote organ dysfunction and start interventions aimed at modulating SIRS, e.g. nutritional therapy (see Task 3). Early diagnosis and treatment of pancreatic infection is key. Indications for surgery are usually restricted to local complications and in particular infected pancreatic necrosis.

So far there is no specific therapy for acute pancreatitis that interferes with the inflammatory necrotising process and hence lessens the emergence of remote organ failure and the volume of necrotic areas.

In this task we will cover general intensive care and various therapeutic modalities and conclude with a brief discussion on the indications for surgery.

General intensive care

Supportive therapy of vital organs

Cardiovascular system

You should establish sufficient oxygen delivery as soon as possible. Tissue perfusion, in particular in the splanchnic area, may be drastically diminished in these patients. Rapid restoration of intravascular fluid volume is the priority as most of these patients...
are severely hypovolaemic. Use inotropes or vasoactive agents after ensuring adequate intravascular volume and allowing for ongoing continuing losses which may be substantial – a characteristic incorporated in the Ranson score.

**Note** Local splanchnic perfusion may be worsened by abdominal compartment syndrome - increased pressure due to intra-abdominal oedema, fluid sequestration and excessive fluid resuscitation.


Unfortunately the cytotoxic-related mechanisms responsible for the impaired pancreatic microcirculation are usually not addressed by manipulation of the systemic circulation aimed at increasing global oxygen delivery.

**THINK** why gastric intramucosal pH may be a better marker of adequacy of resuscitation than the commonly used haemodynamic parameters, in critically ill patients. Why is this of particular concern in severe acute pancreatitis?

For further information:


See also the PACT modules on Hypotension, Haemodynamic Monitoring and Sepsis and MODS

**Respiratory system**

Consider:

- Prevention/correction of hypoxia.
- Early physiotherapy and adequate analgesia (perhaps using epidural analgesia) to ensure free airways and to prevent atelectasis, prevent pulmonary aspiration by nasogastric decompression.
- Evacuation of pleural effusion by fine needle puncture ultrasound guided drainage.
• Continuous positive airway pressure (CPAP) ventilation and/or bilevel positive airway pressure (BiPAP) ventilation by face or nasal mask in cooperative patients.
• Early tracheal intubation and mechanical ventilation, particularly in those with acute lung injury. Apply the general principles of mechanical ventilation as in other causes of acute respiratory distress syndrome (ARDS).

Further information on the therapeutic strategy for acute lung injury can be found in:


See PACT module on Mechanical ventilation Respiratory failure

Renal system

• Prevent and/or minimise renal injury by rapid correction of hypovolaemia.
• If acute renal failure develops, start renal replacement therapy without delay to ensure optimal fluid and metabolic control and to enable nutritional support without haemodynamic instability.
• Although not evidence based, consider Continuous veno-venous haemofiltration (CVVH) as it tends to cause less haemodynamic instability compared with intermittent haemodialysis with ultrafiltration.

For further information, see


PACT modules on Acute renal failure and Oliguria and anuria

Acute Kidney Injury Initiative: http://www.akinet.org

Gastrointestinal system

Beware of intra-abdominal hypertension and assess the patient for this complication regularly. If abdominal compartment syndrome occurs, consider decompression either surgically or in cases of colonic distension with a wide bore tube inserted via the rectum. Abdominal compartment syndrome should be suspected whenever there is evidence of new or worsening organ dysfunction.
You will find more information on abdominal compartment syndrome in the following references.


See the PACT module on Abdominal problems

Q. What are the most important consequences of abdominal compartment syndrome in acute pancreatitis?

A. Increased airway pressures during mechanical ventilation due to elevated diaphragm associated with haemodynamic compromise associated with decreased venous return to the heart, hypotension, renal compromise and possible exacerbation of splanchnic ischaemia.

**Supportive therapy** may appear basic. Nonetheless it is an essential part of the management of these patients: delayed diagnosis and insufficient resuscitation may exacerbate MODS and the risk of mortality.

**Pain relief**

Provide effective pain relief. First try conventional analgesics by the i.v. route. Morphine is not contraindicated but the use of continuous thoracic epidural analgesia with a mixture of diluted local anaesthetic solution (bupivacaine) and opiates offers advantages. You can find further information about epidural analgesia and pancreatitis in the following reference.


Q. What are the possible advantages and potential complications of epidural analgesia in acute pancreatitis?
Epidural analgesia exposes patients to the risk of epidural infection and haematoma, and of circulatory instability, particularly in hypovolaemic patients and is not universally used in this circumstance. The main advantage of this technique is that effective pain relief is almost guaranteed. The dose of systemic opiates can therefore be reduced so that the patient is able to breathe spontaneously or in an assist-mode if mechanically ventilated. The consequent avoidance of systemic narcotics may improve bowel motility, facilitate enteral nutrition and may reduce the risk of secondary pancreatic infection.

**Miscellaneous**

In this high-risk population, routine stress ulcer prophylaxis as well as thrombo-embolic prevention, usually by low-molecular weight heparin, would be standard.

**Specific therapeutic modalities**

**Antibiotics**

Prevention of secondary bacterial contamination of necrotic areas is of the utmost importance in view of the mortality/morbidity attributed to infected pancreatic necrosis. As the nidus of infection, e.g. the volume of necrosis, cannot be influenced by any therapeutic means except for prompt restoration of splanchnic perfusion you have to rely on alternative methods of prevention.

**Randomised controlled trials of intravenous prophylactic antimicrobial therapy** have failed to demonstrate a benefit in terms of incidence of pancreatic infection, surgical intervention and outcome.


Another option is the topical administration of prophylaxis by using selective decontamination of the digestive system (SDD). Non-absorbed antibiotics are used to reduce the number of aerobic Gram-negative bacteria and yeasts in the gastrointestinal tract, whilst retaining the normally predominant anaerobic flora and so modulating colonisation in the digestive tract. Current evidence is not conclusive enough to allow a firm recommendation in Severe Pancreatitis patients.

Unlike i.v. prophylaxis, Selective Decontamination of the Digestive tract (SDD) addresses bacterial translocation from the gut to the pancreas.
Antibiotic therapy

Patients with severe acute pancreatitis are prone to infections early in the course of the disease. Antibiotics are required in those with infection and empirical therapy should be started without delay once infection in the pancreas or elsewhere is suspected on clinical grounds. Continuation of antibiotics and the ultimate choice of drugs should be based on a thorough investigation for a source of infection and on the results of specimen culture obtained by fine needle aspiration, initial aspirate taken at time of wide bore drainage or during surgery in cases of suspected pancreatic infection. Once the presence of infection is established and the patient suffers from severe sepsis or septic shock, additional interventions should be considered according to current sepsis therapeutic guidelines.


Nutritional therapy

This aspect of management is discussed extensively in Task 3.
Indications for surgery

Improved intensive care in regionalised, multidisciplinary centres, advances in interventional radiology and endoscopic techniques as well as implementation of specific therapeutic modalities that address the pathophysiology of the disease have reduced the indications for surgery, during the toxaemic phase of acute pancreatitis and later. Early complications that might prompt the surgeon to intervene urgently are outlined below.

**Undisputed indications**

- Infected pancreatic necrosis when percutaneous/other techniques not indicated
- Severe retroperitoneal haemorrhage
- Acute abdomen – peritonitis
- Biliary obstruction in case of failure of Endoscopic Sphincterotomy
- Abdominal compartment syndrome where percutaneous/other drainage techniques not successful.

**Controversial indications: The case for sterile necrosis**

**Extensive (>50%) sterile pancreatic necrosis:** Early ‘routine’ debridement of necrosis irrespective of its bacteriological status in order to prevent remote organ dysfunction and pancreatic infection fails to achieve these goals and may even be harmful. The development of local complications, postoperative infection of necrosis, problems related to poor demarcation of necrotic areas and long-term sequelae in survivors have led most surgeons to abandon this indication.

**Persisting multiple organ failure despite intensive care therapy:** early and repeated removal of necrotic tissue combined with continuous drainage/lavage have been advocated to overcome systemic effects. This is based on the assumption that ongoing pancreatic inflammation is the sole culprit and that removal of toxic mediators released by the gland will abort the process of distant organ injury once it has been initiated. A prohibitive mortality has led most surgeons to also abandon this indication.

**Note** Neither the extent of sterile pancreatic necrosis, the clinical severity of the disease or the duration of intensive supportive therapy should be regarded as indications for surgery.
3. **Feeding the Patient with Acute Pancreatitis**

Artificial nutrition is increasingly considered to be a key aspect of the specific management of pancreatitis patients and not only as an adjuvant therapeutic modality. In this task we shall discuss the indications, modalities and potential complications of nutritional therapy during acute pancreatitis.

**Artificial nutrition: Why?**

Although no prospective randomised controlled trials have demonstrated that nutritional support lessens the severity of acute pancreatitis or improves outcome, there are several theoretical reasons to provide some of these patients with nutritional therapy. You will find details of controlled trials into the effects of nutrition in acute pancreatitis in the following references.


- Acute pancreatitis is a catabolic, hypermetabolic disease process that increases protein and calorie requirements. The following may be exacerbating factors:
- Oral intake may be impossible for prolonged periods due to the presence of pain, gastric atony, ileus, or partial duodenal obstruction from pancreatic enlargement.
- There are increased protein losses across inflamed retroperitoneal surfaces and through pancreatic fistulas.
- In a subset of patients there are pre-existing protein-calorie malnutrition and micronutrient deficiencies, usually as a result of alcohol abuse.

Recovery results from the successful application of a combination of therapeutic measures, including nutritional care/therapy

Feeding during severe acute pancreatitis may be challenging... See the Patient Challenges in the 'Nutrition' module
The risk for a net negative energy and protein balance and hence for deterioration of nutritional status is very high in severe cases.

**Early assessment of severity dictates the need for nutritional therapy:** the patients at risk of developing organ failure and pancreatic infection and those with preexisting malnutrition should be considered for early nutritional therapy.

For further information on the rationale for feeding the critically ill patient see the PACT module on Nutrition.

**Q. List the metabolic hallmarks of severe acute pancreatitis**

**A.** Patients with severe acute pancreatitis have, among other features, a number of metabolic similarities with septic patients. They share many of the same inflammatory mediators and the subsequent hormonal response to injury is virtually identical in both groups. Metabolic manifestations are:

- Increased energy expenditure, oxygen consumption and a hyperdynamic circulation
- High protein catabolism and ureagenesis
- Increased amino acid oxidation
- Increased and unsuppressible gluconeogenesis while glucose clearance and oxidation are diminished (insulin resistance)
- Increased rate of lipolysis and free fatty-acid oxidation

These metabolic alterations account for some of the potential complications that may be exacerbated by nutritional therapy in acute pancreatitis. For further information on the stress-related metabolic disturbances in acute pancreatitis and in the critically ill patient see:


**Q. Indicate the main consequences of malnutrition in acute pancreatitis**

**A.** Protein-calorie malnutrition may result in:
Task 3. Feeding the patient with acute pancreatitis p 29

- Poor pancreatic healing
- Impaired immune, gut, and lung function
- Increased risk for nosocomial and pancreatic infection
- Worsened outcome

There is some evidence that cell-mediated immunity is already compromised in severe acute pancreatitis, regardless of the nutritional status.

**Nutritional therapy: How, what and when?**

**Route of nutrient delivery: Enteral versus parenteral**

The concept of ‘resting’ the pancreas should not be applied too strictly. This approach implies strict avoidance of all stimuli to exocrine secretion from the pancreas in order to negate the perpetuation of premature enzymatic activation through which pancreatitis is initiated. This principle explains why starvation was previously regarded as the most physiologically appropriate response to pancreatic injury. In the past, if nutritional support was deemed necessary, parenterally infused nutrients were the preferred option as they had the least likelihood of stimulating an inflamed pancreatic gland.

**THINK** about how you could combine early enteral feeding therapy with the therapeutic principle of pancreatic rest? Refer to the regulation of exocrine secretion at the gut level in the following references.


The efficacy of pancreatic rest has never been validated - mechanical, pharmacological and hormonal interventions devised to block exocrine pancreatic secretion have all failed to influence the local inflammatory process and patient outcome. Moreover, although introduction of oral feeding may be associated at times with resurgence of pancreatitis, nutrients infused distally into the jejunum have minimal effect on exocrine secretion.

**Q.** What is the level of basal exocrine pancreatic secretion early after onset of acute pancreatitis?
A. Baseline exocrine secretory capacity is spontaneously diminished during acute pancreatitis.

**Note** The preferred route of nutritional support is no longer controversial: the benefits of enteral delivery in terms of outcome depend mainly on the avoidance of CVC-related infections, better glycaemic control, improvement in gut blood flow, maintenance of gut structural and immune barrier function, reduction in microbial translocation and pancreatic infection and possibly on immunomodulation.

A detailed discussion of the advantages of the enteral over the parenteral route of feeding can be found in the PACT module on Nutrition and in the following references.


**Note** In order to maximise clinical benefit, enteral feeding should be initiated as soon as possible after admission in all attacks predicted to be severe. Patients in whom enteral access cannot be achieved or in whom clear-cut contraindications (intestinal rupture, obstruction, or necrosis), intolerance, or exacerbation of the disease occurs should be considered for partial or total parenteral nutrition (TPN).

**Enteral access**

**Repeated plain abdominal X-ray enables detection of tube migration**

Early feeding is usually undertaken through a silicone or polyurethane tube with an inner stylet that is positioned (under fluoroscopic guidance) beyond the first jejunal loop.
Right-lateral positioning of the patient as well as the use of a prokinetic (e.g. erythromycin 250 mg IV bolus) may assist the passage of the tube through the pylorus so that fluoroscopic guidance is unnecessary in >50% of the cases. An alternative method is to pass the feeding tube over an endoscopically placed guide wire. The correct positioning of the tube should be ascertained regularly by radiography.

A blind bedside insertion technique should be used with caution as the duodenum may be distorted and injured by the retroperitoneal inflammatory process. Continuous gastric decompression by gravity should be achieved by a separate tube in patients with gastro-paresis or post-pyloric obstruction. This prevents the risk of massive pulmonary aspiration and may also help to detect proximal migration of the feeding tube.

Preliminary data indicate that early nasogastric tube feeding is safe and well tolerated in patients with predicted severe acute pancreatitis and the ease of insertion facilitates the earlier commencement of enteral nutrition. Although duodenal secretion of pancreatic enzymes is inversely related to the severity of acute pancreatitis, this route of feeding may be associated with a risk of exacerbating the disease process and of pulmonary aspiration, in particular in those with gastric outlet compression.

Patients who undergo surgery for local complications later in the course of the attack can have a catheter jejunostomy performed. This offers an alternative to the nasojejunal tube.

**Energy and protein requirements**

Resting energy expenditure, as measured by indirect calorimetry, varies widely in acute pancreatitis, depending upon the magnitude of the regional inflammatory
process and the presence of superimposed infection. The latter raises energy expenditure by 5 to 20% above resting values, but overfeeding should be avoided.

Further information on the use of indirect calorimetry in nutrition and on energy, protein, and micronutrient requirements during acute pancreatitis can be found in the PACT 'Nutrition' module as well as in the following references.


**Composition of the diet**

Parenteral nutrition should be administered as indicated in other critically ill patients and should be supplemented with glutamine. Plasma lipid clearance should be regularly monitored to avoid hypertriglyceridaemia. Infused nutrients do not exacerbate the disease.

For further information see:

PACT module on Nutrition


There are no data on the optimal enteral formula in severe acute pancreatitis. Polymeric solutions are cheaper, well tolerated and maintain gut structure/function. Immune-enhancing diets have not been specifically tested in this disease.

**Q. Indicate the theoretical advantages of medium-chain triglycerides in acute pancreatitis in both enteral (EN) and parenteral nutrition?**

**A.**

- If used enterally: better assimilation by direct absorption into the portal vein in an environment deficient in lipase.
- If administered parenterally as a lipid emulsion: rapid clearance and more complete mitochondrial oxidation so that the risk of hypertriglyceridaemia is theoretically reduced.
**Prescription and timing of nutrient administration**

You should administer enteral solutions as a continuous 24 hours pump driven infusion. Increase the diet gradually (250-500 ml/day), starting with 500 ml/day until the patient's targeted calorie needs are tolerated. Jejunal residual volumes should be <10-20 ml/4h with feeding via the small bowel. If the nutritional target cannot be met exclusively by the enteral route after a 5-to 7-day trial you should consider combined nutritional support. (TPN plus EN).

There are no clear cut data on the optimal timing of nutrient administration in severe acute pancreatitis. Although priority is given to resuscitation, a prolonged and/or repeated period of cardiorespiratory compromise should be anticipated in most of these patients and should not unduly delay nutritional therapy. Even small amounts (250–500 ml/day) of enterally-infused nutrients started within 48 hours after onset of pancreatitis may be beneficial.

Most of these patients suffer from functional ileus and are at risk of gut failure. Overzealous prescription of enteral nutrition may result in intestinal distension and worsen gut and pancreatic ischaemia.

**Oral refeeding**

No precise guidelines exist as to when and what to feed orally after an episode of severe acute pancreatitis. Empirical decisions are usually based on resolution of ileus, the alleviation of the retroperitoneal inflammatory process and of remote organ dysfunctions as well as on the absence of digestive fistulae. A low-fat diet should be introduced gradually as the risk of relapse (pain, rise in serum amylase) may theoretically be influenced by the stimulatory effect of the diet on the enzymatic secretion.

**Complications of nutritional therapy**

During artificial nutrition in acute pancreatitis, watch for these complications:

- Hyperglycaemia due to the combination of beta cell necrosis/dysfunction with peripheral insulin resistance commonly complicates nutritional support. Whatever the route of feeding monitor serum glucose level and titrate i.v. short acting insulin to ensure glycaemic control. Avoid
overfeeding and improve glucose tolerance by supplying some calories as lipids.

- Hypertriglyceridaemia is usually attributed to overfeeding with inadequate supply of carbohydrate/triglycerides, insulin resistance and poor nutrient utilisation. Monitor serum turbidity and triglyceride level, and titrate fat content to keep the serum triglyceride level below 400 mg/dl (4.5 mmol/l).

- Exacerbation of the disease process: Relevant causes are hypertriglyceridaemia, nasogastric feeding, proximal dislodging of the jejunal feeding tube or too early return to oral diet. Monitor serum amylase level and perform abdominal CT.

- Gut intolerance: monitor bowel distension, abdominal pressure, residual jejunal volume, and diarrhoea. Titrate enteral infusion rate accordingly and/or use prokinetics and enemas.

**Note** Significant intolerance to appropriate nutritional therapy by either the enteral or the parenteral route usually denotes ongoing local complications (e.g. infection, mesenteric ischaemia) and requires appropriate investigation (refer to Task 1 and Task 4).
Algorithm of nutritional therapy in acute pancreatitis

Useful review articles on the issues covered in this Task are:


In the next five patients you see, assess the timing and success of enteral feeding.
4. **How to identify and manage local complications of acute pancreatitis**

Approximately 50% of patients with severe acute pancreatitis progress to a spontaneous and uneventful resolution of the regional inflammatory process. In the remainder local complications will emerge usually weeks or even months after the onset of the attack. Therefore these patients must be kept under review for deterioration since timely operative, endoscopic or radiological interventions are essential.

**Pathophysiology of regional necrosis**

**Three mechanisms act in concert to promote regional necrosis and widespread organ damage**

Premature activation of lipolytic and proteolytic enzymes, in particular trypsinogen, is the key trigger of acinar cell necrosis. As the natural safeguards that prevent autodigestion are overwhelmed in acute pancreatitis, trypsin is able to activate the cascade of proteases.

Secondary overactivation of immune effector cells results in the local accumulation of various proinflammatory and cytotoxic substances including proteases, reactive oxygen species, cytokines and lipid mediators. These cellular components and humoral mediators play a pivotal role in acinar cell injury.
Local microcirculatory disturbances soon amplify regional necrosis. They are primarily ascribed to endothelial cell damage resulting from the combined effect of activated leucocytes and proteases.

The pancreas is the source of damage and surrounding tissues are the primary target

![Image of a scan showing the pancreas]

The enzyme-rich exudates diffuse from the pancreas into the surrounding tissues and often form fluid collections. Tissue necrosis involves primarily the peri-pancreatic tissue; the core of the gland being more resistant. However at times necrosis may affect the main pancreatic duct, often in the isthmic area, causing rupture.

A deeper insight into this topic can be found in:


**THINK** about how biochemical markers relate to the severity of necrotising pancreatitis.
Pancreatic infection

Pathophysiology

Necrosis creates an excellent culture medium for micro-organisms. Accordingly the risk of infection is proportional to the extent and duration of necrosis. The incidence of infection peaks in the third week after onset, but in up to 25% of these patients infection is documented in the first seven days. The earlier infection occurs, the higher the mortality, as the combination of infection with the inflammatory process generates a highly toxaemic course. Cultures yield predominantly common enteric bacteria. Although not proven in humans, bacterial translocation from the gut lumen either transmurally, via lymphatics, ascites or blood is probably the leading mechanism of infection.

THINK On the basis of the previous information, think about how we can influence the risk of infection.

Q. What are the other potential routes resulting in infection of necrosis during acute pancreatitis?

A.
- Intra-abdominal spread: digestive fistula – hollow viscus perforation (colon)
- Biliary reflux (cholangitis)
- Duodenal reflux (post-ERCP)
- Haematogenous: beware of secondary bacterial contamination of necrosis by extra-pancreatic infectious foci (nosocomial infection)

Q. List the factors that may promote bacterial translocation during acute pancreatitis

A.
- Ileus and secondary aerobic Gram-negative bacterial overgrowth
- Disruption of the normal gut microflora with loss of colonisation resistance (antibiotics)
- Gut barrier failure (both structural and immunologic) as a result of splanchnic ischaemia and the spread of the nearby inflammatory process
- Bowel rest (Total Parenteral Nutrition)

**Types of infection: infected necrosis or abscess?**

Because of differences in anatomy, prognosis and management it is crucial that you differentiate between infected pancreatic necrosis and pancreatic abscess.

**Infected pancreatic necrosis**

![Image of infected pancreatic necrosis](image_url)

**Differentiation between the two forms of pancreatic infection**

<table>
<thead>
<tr>
<th></th>
<th>Infected Pancreatic Necrosis</th>
<th>Pancreatic Abscess</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence</td>
<td>15-50% of necrotising forms</td>
<td>2-3%</td>
</tr>
<tr>
<td>Onset</td>
<td>Maximal in the 3rd week</td>
<td>&gt;4 weeks</td>
</tr>
<tr>
<td></td>
<td>25% within 7 days after onset</td>
<td></td>
</tr>
<tr>
<td>Pathologic finding</td>
<td>Predominance of necrosis</td>
<td>Collection of pus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Little or no necrosis</td>
</tr>
<tr>
<td>Clinical expression</td>
<td>Severe sepsis</td>
<td>Fever</td>
</tr>
<tr>
<td></td>
<td>Multiple organ failure</td>
<td>Abdominal pain</td>
</tr>
<tr>
<td>Bacteriology</td>
<td>Mono-microbial 60-80%</td>
<td>Poly-microbial 50-60%</td>
</tr>
<tr>
<td></td>
<td>&gt;50% aerobic Gram-negative</td>
<td>anaerobes/fungi/Gram-negative</td>
</tr>
<tr>
<td></td>
<td>microorganisms</td>
<td>bacilli/Gram-positive cocci</td>
</tr>
<tr>
<td>Mortality</td>
<td>15-60%</td>
<td>&lt;10%</td>
</tr>
</tbody>
</table>
Pancreatic abscess results from the secondary infection of an acute fluid collection or a liquefied necrotic area

Q. Do you think drainage of the pancreatic abscess should be percutaneously attempted in this particular patient and why?

A. Percutaneous drainage is likely to fail as the collection is large, central and multi-loculated: given the appearances on CT scan, surgery may be the preferred option in this case.

Pancreatic infection may be suspected from clinical signs of sepsis and/or general deterioration, but confirmation of the diagnosis requires CT imaging and appropriate culture (aerobes, anaerobes, fungi) of material obtained by ultrasound- or CT-guided fine needle aspiration (FNA).

Occasionally infection may be diagnosed by CT alone when gas bubbles are present within a necrotic area. Otherwise, FNA should be directed to all sites of necrosis or at least to those showing the most obvious changes on repeated CT. Monitoring of serum procalcitonin, the propeptide of calcitonin, is a potential marker for the non-invasive identification of infected pancreatic necrosis as well as for the selection of patients with persisting sepsis after drainage.

Microbiological analysis of material harvested by FNA is not 100% accurate: all necrotic areas may not be sampled or antibiotic prophylaxis, if used, may affect culture results. FNA (if negative) should be repeated every 5–7 days in patients with persistent signs of sepsis. Gram-staining should be used to guide early treatment.

Use empirical antimicrobial therapy while awaiting the results of culture, deescalate or discontinue based on the results

Negative sampling should not delay surgical exploration and/or drainage when the clinical course is strongly suggestive of infection.


Treatment of pancreatic infection

Antimicrobial therapy alone is usually ineffective

Endoscopic transgastric or duodenal drainage is an alternative to the percutaneous route in specialised centres

While pancreatic abscess is usually easily drained, often by CT guided aspiration, infected necrosis is difficult to evacuate as it is not a well-circumscribed collection of pus but rather a diffuse area of bacterial proliferation within ill defined necrotic tissues. Therefore drainage is not straightforward and the surgeon can only remove pus and solid debris of necrosis easily detached by suction and manual debridement. Surgical intervention will leave in place adherent areas of necrotic tissue that allow proliferating bacteria to persist. Moreover production of necrosis is a dynamic process and continues after the surgical debridement, thus perpetuating infection. This accounts for the high incidence of sepsis recurrence after surgery and the development of specific techniques aimed at evacuating pus and necrotic tissues throughout the evolution of the attack, e.g. an average of eight weeks.

A percutaneous approach is tempting as it does not injure disease-free areas. It is particularly attractive in pancreatic abscess where the infected material is mostly liquefied or as a temporising measure in the desperately ill patient until more formal surgical debridement may be tolerated.
Percutaneous drainage has been associated with a high incidence of sepsis recurrence, even in the case of well-circumscribed abscesses and despite insertion of multiple catheters and prolonged drainage. These results may be due to the inability of percutaneous drainage to remove solid debris lying at the periphery of the abscess or inside infected necrosis.

**Note**  
**Percutaneous drainage** is classically reserved for non-loculated, peripheral, and well-defined collections that usually appear late in the course of the attack. Remember that it is a lengthy and resource-intensive approach.

Observe the placement of drainage catheters with the radiologist and assist the nursing staff in maintaining their patency and the drainage.

The **surgical approach** remains the gold standard. Exploration of the abdomen and dissection are guided by the CT scan that provides a 'roadmap' to the surgeon. All collections and all pockets of tissue necrosis and infection are opened and evacuated as completely as possible. Overall morbidity averages 70%. Patients require re-operation in 25% of cases, mainly for bleeding, hollow viscus perforation or necrosis, fistula and recurrent sepsis.

The ongoing inflammatory, necrotising process necessitates a prolonged continuous drainage procedure postoperatively in order to prevent sepsis recurrence.

The precise nature of the operative procedure varies according to the surgeons' expertise. Whatever the surgical technique (see illustration below), thorough debridement and continuous postoperative drainage are the key features of infection management. Depending on the circumstances some surgeons prefer a transverse sub-costal or flank incision for exploration.

**Note**  
If the **clinical situation permits**, operative necrosectomy and/or drainage should be postponed at least until four weeks after onset. Over time, infected necrotic areas demarcate from viable tissues, undergo liquefaction and coalesce into an encapsulated entity. This **organised necrosis** leads to an easier and safer debridement, with sparing of viable pancreatic tissue. It might be even more amenable to percutaneous, endoscopic or minimally-invasive (laparoscopic) operative drainage alone or in combination. Thus, the optimal type of intervention depends on the clinical course and the timing of the procedure.
You will find details on surgical indications in the first of the following references and details on surgical techniques and other types of drainage in the other references.


**Haemorrhage and perforation**

Significant *spontaneous haemorrhage* is rare but carries a high mortality. It is usually due to erosion of the vessel wall by proteases and pseudoaneurysm formation of the pancreatic arteries within a pseudocyst (see below) or an area of necrosis. In case of massive retroperitoneal bleeding the CT scan can identify the approximate location of the haemorrhage. However angiography is often necessary to localise precisely the source of bleeding and at times to achieve haemostasis with embolisation. Surgery is required if angiographic occlusive techniques fail.
Massive haemorrhage often heralds infection: surgical drainage is mandatory in these cases

Spontaneous perforation occurs usually at the level of the left and transverse colon. The mechanism is predominantly ischaemic as a consequence of extension of necrosis in the mesocolon and secondary vessel thrombosis. The diagnosis should be suspected in the presence of septic signs, diarrhoea, rectal bleeding, or high-pitched bowel sounds. CT scan shows thickening of the colonic wall and extra luminal gas bubbles or contrast media. At times colonic necrosis is an incidental operative finding.

During surgery, it is difficult to evaluate colonic viability. Colectomy is indicated in patients with perforation or obvious irreversible necrosis. If there is doubt regarding the viability of the colon, it is safer to perform a loop ileostomy and to leave the colon in situ. This procedure prevents further deterioration of the colonic lesions, improves tolerance to enteral feeding and is easily reversed.

Pseudocysts and pancreatic fistulas

A pseudocyst is a collection of pancreatic juice enclosed by a wall of granulation tissue and is formed near an area of tissue necrosis associated with rupture of a pancreatic duct (see illustration below).
Pseudocyst

It can result from the rupture of the main pancreatic duct itself, but in the majority of instances, the leaking duct is located at the periphery of the gland. Formation of pseudocysts takes at least four weeks. Fluid collections apparent at an earlier stage lack a defined wall and are named 'acute fluid collections'. Drainage, either percutaneous, surgical, or endoscopic, is required in those with large and/or symptomatic pseudocysts. Endoscopic treatment, utilising endoscopic ultrasound guidance, is the primary procedure for pseudocysts with amenable anatomy.

Complications for which drainage should be considered:

- **Compression** of adjacent structures.
- **Rupture** of pseudocysts is observed in less than 5% of cases and may present either as an acute episode or a silent and progressive process. It may lead to a reactive effusion (ascites, pleural effusion or pericardial effusion) or rupture into a hollow viscus, usually the stomach or the colon.
- **Bleeding** is seen in 5% of pseudocysts and is the most serious complication.
- **Infection:** approximately 10% of all large pseudocysts become infected, either spontaneously or after an inappropriate attempt at drainage, usually percutaneous.

**Note** Do not mistake collections of organised necrosis as a pseudocyst. Infection is facilitated by the presence in the cavity of solid necrotic debris, which hinders drainage. The internal consistency of the collections is best determined by ultrasound or MRI.

Pancreatic fistulas result from an unsealed rupture of the pancreatic duct. A minor leak is treated conservatively with drainage and somatostatin or octreotide. Major rupture may require resection, jejunal anastomosis or endoscopic stenting.

Baron TH, Harewood GC, Morgan DE, Yates MR. Outcome differences after endoscopic drainage of pancreatic necrosis, acute pancreatic pseudocysts, and chronic pancreatic pseudocysts Gastrointest Endosc 2002; 56(1): 7-17. PMID 12085029

Keep a record of the differences in timing, diagnosis and treatment of acute fluid collections, pseudocysts and pancreatic abscess. Refer to the following reference:


**CONCLUSION**

Severe acute pancreatitis is a challenging disease for the ICU clinician: the pathophysiology at the cellular level remains obscure, the outcome is unpredictable, and the long-lasting course of the attack is characterised by the emergence of life threatening local complications and multiple distant organ failures.

So far, specific treatment is lacking and the modern management of these patients requires treatment in, or referral to, a specialist centre. Here, clinical experience, technical skills and a comprehensive implementation of all facets of Intensive Care Medicine are effected. A consistent and concerted approach, close cooperation as well as detailed communication between the intensivist, gastroenterologist skilled in endoscopy, surgeon, interventional radiologist and the microbiology service is required to optimise patient outcome.
SELF-ASSESSMENT

EDIC-style Type K

1. Type K. Usual imaging techniques to aid in the diagnosis of acute pancreatitis in the initial phase (first 1–2 days after hospitalisation) include:
   A. Plain abdominal X-ray
   B. Endoscopic retrograde cholangiopancreatography (ERCP)
   C. Abdominal CT with or without contrast
   D. Abdominal ultrasound

2. Type K. Prognostic scores often used in the assessment of acute pancreatitis include:
   A. APACHE II score
   B. Ranson score
   C. Murray score
   D. Imrie score

3. Type K. Regarding infectious complications related to severe acute pancreatitis, which of the following statements are correct:
   A. Infection is usually present from the start
   B. A common complication is infected pancreatic necrosis
   C. Infections most often come from haematogenous spread of bacteria
   D. Anaerobic bacteria is the cause of most frequent clinical infection

4. Type K. Systemic inflammatory response syndrome (SIRS) is frequently found in patients with acute pancreatitis. Which of the following is/are true regarding the development of SIRS in this condition?
   A. SIRS is only present in the severe forms of acute pancreatitis
   B. SIRS seldom develops prior to the second week of the disease
   C. Includes CRP rise >180 mg/dl
   D. Includes LPK elevation >12.000/ l

5. Type K. Symptomatic treatment of acute pancreatitis include the following options:
   A. Analgesia with opioids intravenously
   B. Epidural analgesia
   C. Fluid restriction
   D. Peritoneal lavage

6. Type K. Usual complications in severe acute pancreatitis includes:
   A. Intra-abdominal hypertension
   B. Intestinal ischaemia
   C. Acute respiratory failure
   D. Increased intracranial pressure
7. Type K. Which of the following statements is/are true regarding nutritional therapies in severe acute pancreatitis?
   A. Enteral nutrition is associated with fewer complications than parenteral nutrition
   B. A probiotic should usually be included in the nutritional regimen
   C. Specific immune-enhancing diets are superior to standard enteral nutrition
   D. Nutrition direct into the duodenum is not advocated

8. Type K. Hyperglycaemia is common in acute severe pancreatitis and can be a consequence of:
   A. Alpha cell necrosis in the pancreatic tissue
   B. Beta cell necrosis in the pancreatic tissue
   C. Insulin resistance in the CNS
   D. The use of parenteral rather than enteral nutrition

EDIC-style Type A

9. Type A. The severity of acute pancreatitis may be evaluated by the following tests and signs EXCEPT
   A. Development of new organ dysfunctions(s)
   B. C-Reactive Protein (CRP)
   C. S-amylase
   D. Sequential Organ Dysfunction Score (SOFA score)
   E. Procalcitonin

10. Type A. Which of the following is NOT a cause of acute pancreatitis:
   A. Hyperthyreosis
   B. Abdominal trauma
   C. Endoscopic retrograde cholangiopancreatography (ERCP)
   D. Hyperlipidaemia
   E. Mumps

11. Type A. Prognostic criteria in patients with acute severe pancreatitis include the following EXCEPT:
   A. Age >80
   B. The extent of pancreatic necrosis
   C. Co-morbidity
   D. Circulatory failure
   E. Development of acute renal failure
Self-assessment Answers

1.
A. T 
B. F 
C. T 
D. T 

2.
A. T 
B. T 
C. F 
D. T 

3.
A. F 
B. T 
C. F 
D. F 

4.
A. F 
B. F 
C. F 
D. T 

5.
A. T 
B. T 
C. F 
D. F 

6.
A. T 
B. F 
C. T 
D. F 

7. Answer: TFFF
A. T 
B. F 
C. F 
D. F 

8. Answer: FTFT
A. F 
B. T 
C. F 
D. T 

9. Answer C is correct

10. Answer A is correct

11. Answer D is correct
**Pancreatitis**

**PATIENT CHALLENGES**

You are called to the Gastroenterology unit to attend to a 56-year-old obese man who is in cardio-respiratory distress. While reviewing the patient's record you see that he has a four-year history of alcohol abuse and that he was admitted to the hospital via the emergency room 36h previously with a two-day history of epigastric pain and vomiting. On admission vital signs indicated a blood pressure of 95/30 mmHg, a pulse rate of 110 beats/min, a respiratory rate of 28 breaths/min and an axillary temperature of 38.6 ºC. The abdomen was distended and diffusely tender on palpation without guarding. No bowel sounds were heard.

**Learning Issues**

Epidemiology of acute pancreatitis  
Aetiology of acute pancreatitis  
Diagnosis of acute pancreatitis  
Diagnostic clues  
Diagnostic pitfalls

**Laboratory data included:**  
Haematocrit 51%  
White blood cell count 18 000/ml  
Blood glucose 11.6 mmol/l  
Calcium 1.95 mmol/l  
Creatinine 195 µmol/l  
Lactate dehydrogenase 980 IU/l  
C-reactive protein 15 mg/dl  
Amylase 180 IU/l  
Lipase 1540 IU/l  
The serum was lipaemic.

**Learning Issues**

Early assessment of severity  
Clinical assessment  
Scoring systems  
Biochemical indicators  
Role of CT
Arterial blood gas analysis without supplementary oxygen showed a PaO$_2$ of 7.7 kPa and lactate 2.4 mmol/l. A chest X-ray demonstrated a moderate right pleural effusion and elevation of the diaphragm. Abdominal ultrasound examination showed a slightly dilated common bile duct without evidence of stones. Incremental dynamic bolus abdominal CT revealed a moderate enlargement of the pancreas without enhancement defect and small fluid collections around the head and the tail of the gland.

The patient was on the ward, having ‘nil by mouth’ and had received non-opioid analgesics, oxygen by nasal cannulae and crystalloids 3000 ml/day.

**Learning Issues**

**Imaging techniques in pancreatitis**

**Q.** Considering the data on admission would you have promptly referred the patient to the intensive care unit (ICU) and if so why?

**A.** Immediate referral to ICU is appropriate. There are signs of hypovolaemia, renal dysfunction, major risks of respiratory failure as well as biochemical indicators of an ongoing inflammatory and necrotising process in the pancreatic area. Early multiple organ dysfunction syndrome is a likely event and this warrants at least close observation and preventive measures in a Critical Care area.

**Learning Issues**

**Task 1 Early identification of severe and/or necrotising forms of pancreatitis is the priority.**
Q. What therapeutic measures would you have taken from the outset?

A. Firstly, aggressive fluid resuscitation in an effort to protect or restore the microcirculation, especially in the splanchnic area and at the same time respiratory support. The latter should include chest physiotherapy, relief of pain, nasogastric suction and supplemental oxygen. Consideration could be given to non-invasive positive pressure ventilation. Aspiration and atelectasis with secondary bacterial infection are the main causes of early respiratory failure in acute pancreatitis, especially in an alcoholic patient.

**Learning Issues**

*Task 2 General intensive care*

The patient's respiratory condition deteriorates and you decide to initiate mechanical ventilation and circulatory support before moving him to the ICU. During the next 72h, his condition remains precarious.

A chest X-ray indicates widespread and bilateral pulmonary infiltrates.

**Learning Issues**

*Pathophysiology of pancreatitis*
*Remote organ dysfunctions*
*Clinical features of pancreatitis*
*Toxaemic phase*
*Necrotic phase*
*Monitoring the course of pancreatitis*

Central venous cannulation, titrated fluid infusion and a high dose of norepinephrine are required to maintain a mean arterial pressure of 70 mmHg. Lactate levels range between 1.5 and 3 mmol/l. Haemodynamic assessment demonstrates a hyperdynamic circulation...
and increased extravascular lung water. Mechanical ventilation is carried out with low tidal volume and an adjusted positive end-expiratory pressure (PEEP) between 10–15 cm H₂O. The PaO₂/FiO₂ ratio is 60–100 mmHg.

The patient is anuric and is started on continuous veno-venous haemodiafiltration (CVVH). Core temperature ranges from 37.4 °C to 39.5 °C. Ecchymotic spots appear on the flanks. There is 1000 ml/day of bile-stained nasogastric reflux.

See PACT modules on Oliguria and anuria and Acute renal failure.

**Learning Issues**

*Initial management of pancreatitis*

*Supportive therapy*

**Relevant laboratory data on ICU admission are:**

- White blood-cell count 23 000/ml
- Platelets 40 000/ml
- C-reactive protein 30 mg/dl
- Creatinine 362 µmol/l
- Total bilirubin 68 µmol/l

On admission to the ICU Ranson and APACHE II scores are 9 and 24, respectively. On the fourth day FiO₂ was decreased to 0.6.

**Learning Issues**

*Scoring systems*

**Q.** Which procedure is now urgently required for this patient?

**A.** You should recommend a contrast-enhanced abdominal CT scan in order to identify and localise the presence of (peri)pancreatic necrosis, to appreciate its extent and to assess bacteriologic status by CT-guided percutaneous sampling, Gram stain and appropriate cultures.

**Q.** Assuming sterile necrosis would you now consider surgery?

**A.** Surgeons are increasingly reluctant to operate on these patients early after onset of disease because of the poor demarcation between viable and necrotic tissues and the prohibitive mortality/morbidity associated with early debridement and drainage. There is also fair evidence of an improved overall prognosis with conservative (critical care) management, even in the face of multiple organ dysfunction, and delayed (necrosectomy) surgery.
Learning Issues

Task 2 Indications for surgery

This policy of conservative therapy is now less controversial. However, on site practice may still vary from centre to centre depending on available surgical and other expertise and management policy.

Q. Assuming sterile necrosis which specific therapeutic measures would you implement other than continuing intensive support of failing organs?

A. You should consider nutritional support and refrain from systemic antibiotic prophylaxis.

Learning Issues

Task 2 Indications for antibiotic prophylaxis in acute pancreatitis

You order an abdominal CT which indicates the presence of necrosis of more than 50% of the pancreas as well as widespread fluid collections.

The Balthazar CT severity index is 10. No gas is detected outside the digestive tract and cultures of percutaneous aspirates yield no micro-organisms. Selective digestive
decontamination is undertaken via the feeding tube which was endoscopically placed beyond the first jejunal loop and also by daily enema.

**Learning Issues**

Pathophysiology of pancreatitis  
Pancreatic necrosis  
Early identification of local complications  
Non operative treatment  
Specific therapeutic issues  
Balthazar CT severity index

**Q.** Would you have recommended an alternative approach to the patient's management given that multiple organ failure persisted in spite of intensive therapy?

**A.** Surgical debridement and drainage of devitalised areas can be an option in an attempt to limit the release of mediators of remote organ failures and to prevent regional infection. This remains highly controversial considering the natural prognosis of sterile necrosis and the morbidity associated with surgery.

**Learning Issues**

Determinants of outcome  
Surgical treatment  
Indications and timing  
Associated morbidity

**Q.** Why was the bacteriologic status of both the colon and the necrotic areas regularly assessed?

**A.** Bacterial translocation from the colon is thought to be the predominant mechanism of pancreatic infection. Pancreatic infection is an indication for antibiotics and debridement and drainage and is usually preceded by colonisation by aerobic Gram-negative bacteria.

**Learning Issues**

Pathophysiology of pancreatitis  
Local complications
The patient is started on an isocaloric semi-elemental diet administered as a continuous 24h pump driven infusion. Resting energy expenditure as measured by indirect calorimetry averages 120% of predicted values. Within four days the target of 2000 kcal and 14 g nitrogen/day is reached exclusively by the enteral route without any clinical, biochemical or radiological exacerbation of the inflammatory process nor signs of digestive intolerance. The serum is no longer lipaemic.

**Learning issues**

*Nutritional support in pancreatitis*

**Q.** What are the two main metabolic issues relating to early feeding in this kind of patient?

**A.** The patient should be regularly assessed for hyperglycaemia and hypertriglyceridaemia.

**Learning issues**

*Task 3 Nutritional support*

*PACT module on Nutrition (Patient Challenges)*

Intensive support of failing organs is continued. Multiple organ dysfunction syndrome persists for the next two weeks. As serial CT-guided samples of necrotic areas fail to grow significant micro-organisms, conservative management is continued. The patient makes a slow but uneventful recovery: vasopressor support and mechanical ventilation were withdrawn 3 and 4 weeks respectively after ICU admission. On day 36 the patient was discharged to the ward while resuming a low-fat oral diet. Selective digestive tract decontamination, which had been utilised in the management, was discontinued at that time.

**Q.** On the ward and during the following months which complications should you look for?

**A.** The emergence of pancreatic endocrine insufficiency and local complications including acute pseudocyst and late infection (pancreatic abscess).

**Learning issues**

*Task 4 Abscess v infected pancreatic necrosis*
The patient left the hospital on day 50. At the follow-up visit one year later abdominal CT indicated complete resolution of fluid collections and atrophy of the pancreas.

However the patient had developed insulin dependent diabetes and clinical signs of malabsorption suggestive of pancreatic exocrine insufficiency.

A 61-year-old male, with a history of severe Parkinson's disease and hypertension, is admitted to the local hospital with acute pancreatitis of moderate severity.

Management of biliary pancreatitis
Role of early sphincterotomy
Diagnosing the biliary origin of acute pancreatitis
Detection of choledocholithiasis in acute pancreatitis

Ultrasound shows gallbladder stones. On day 2, endoscopic ultrasound is performed, showing a stone in the main bile duct.

On day 4, endoscopic sphincterotomy removes the stone, but this is followed by clinical deterioration with fever, chills and blood culture positive for E. Coli. Antibiotic
treatment is ineffective and the patient is referred to the University Hospital on day 13.

The appearances on the initial CT scan are dramatic, with necrosis of the peripheral areas of the pancreas, associated with a heterogeneous collection, while the core of the gland remains well perfused.

**Note** Bile and pancreatic ducts are pathways for bacterial contamination of pancreatic necrosis

Fine needle aspiration under CT guidance produces a purulent exudate containing Gram-negative rods (E. Coli). The patient undergoes surgery on day 30. Debridement of the necrotic tissue situated around the gland results in large cavities, which are then packed. A jejunostomy and an ileostomy (with a view to preventing colonic complications) are undertaken. No biliary drainage is possible. Packing is subsequently replaced by drains.

**Learning Issues**

*Pancreatic infection: pathophysiology, diagnosis, and management of pancreatic infection*
Despite the development of a pancreatic fistula the general condition of the patient improves and he is discharged at day 75. One year later the patient is admitted for recurring episodes of abdominal pain, vomiting and increased pancreatic enzymes. CT scan shows a large pseudocyst.

**Learning Issues**

_Hollow viscus perforation during acute pancreatitis_
_Pseudocysts: symptoms, diagnosis and management_

Surgical drainage (Roux-en-Y cystojejunostomy) is performed, leading to resolution of the cyst with no resulting diabetes.

Rupture of the pancreatic duct: a frequent event in a case of severe necrosis

Q. Can you indicate two possible mechanisms of infected pancreatic necrosis in this patient?

A. Cholangitis and endoscopic transpapillary interventions. Immediate decompression of the common bile duct is warranted in case of associated cholangitis, but this procedure carries the risk of introducing micro-organisms into necrotic areas via the pancreatic duct.

Note

Ultrasound is the best diagnostic test for gallstones. Endoscopic retrograde cholangiopancreatography (ERCP) and endoscopic sphincterotomy: useful but potentially dangerous. Peri-procedure antibiotic coverage is required.

Q. Why was antibiotic treatment ineffective in spite of extraction of the stone?

A. Assuming adequate drainage of the common bile duct it is likely that the necrotic pancreas was already infected at this stage. Surgical debridement and drainage were necessary as antibiotics alone usually cannot cure this kind of infection. Retrospectively percutaneous sampling of necrotic areas was unduly delayed in this patient. As the patient’s condition remained stable on antibiotics, surgery was delayed for 4 weeks to allow a clear demarcation between necrotic and viable tissues and facilitate complete evacuation of infected necrosis.

A 45-year-old alcoholic male, is admitted to a local hospital with acute pancreatitis. Severity criteria are not available, therefore the Ranson score is unknown. The general symptoms recede but pain recurs following every attempt at feeding. A CT scan is performed on day 19 after onset, showing a rupture of the pancreas and a fluid collection at the level of the neck of the gland.

Note

Food intolerance is a sign of complications or of persisting inflammation. CT-scan allows direct observation of the local evolution of the disease. Organisation of a diffuse infiltrate into an organised collection is frequent

Learning Issues

Pancreatic fistulas: pathophysiology and management
Role of percutaneous/endoscopic drainage in acute pancreatitis
The postoperative course in acute pancreatitis
The patient is referred to the University Hospital on day 23. A CT scan is performed on day 27 showing that the fluid collection is thick walled.

The patient is in a good general condition, but still unable to tolerate any food.

Q. What approach would you suggest at this stage: Operate, continue medical treatment, or establish percutaneous or endoscopic drainage?

A. You should establish percutaneous, endoscopic, or surgical drainage. Local expertise may play a decisive role in the choice of the therapeutic approach.

NOTE Persistent illness is the only remaining (late) surgical indication for sterile ‘organised’ necrosis

Q. Name two major complications associated with percutaneous drainage of acute pseudocysts?
A. Secondary infection, particularly when organised necrosis is mistaken as a pseudocyst and cutaneous fistula, especially if the pseudocyst communicates with the pancreatic duct.

**A percutaneous drainage of the collection is performed.** The fluid is sterile. Despite effective drainage (200 ml/24h) and treatment with octreotide (300 mcg/24h iv), intolerance of enteral feeding persists, leading to a decision to operate.

On day 50, the patient finally undergoes surgery. Operation confirms rupture of the gland and shows the presence of solid necrotic debris in the collection. A drain is inserted, together with a feeding jejunostomy and a cholecystotomy is performed.

The post-operative course is unremarkable. There is a persisting fistula (50 ml/24h) from the rupture site. The patient is discharged one month after operation with a well-fitted appliance to collect the fistula fluid.

Three months after operation, the pancreatic fistula is still open. An ERCP shows the duct in the head of the gland and a fistulography outlines the remaining, glandular duct.


A pancreatic prosthesis is inserted endoscopically in an attempt to bridge the ductal defect, but this fails. Surgery is then performed and a Roux-en-Y pancreatico-jejunostomy is constructed leading to cure of the fistula. The patient is seen one year after the operation and is in good condition, with no diabetes.
Fistula is the expected sequel of a rupture of the gland treated by surgical drainage

On reflection, you were presented with three challenging cases of acute pancreatitis that highlighted the often complex and protracted nature of the condition.

Q. Do you, as an intensivist, envisage having a continuing role in the long-term care of such patients? Give your reasons.

A. As a general rule in Intensive Care Medicine patients’ outcome should be assessed not only on the basis of survival at discharge from the unit. Quality of life is an increasingly important issue. This applies particularly to patients with severe acute pancreatitis. Even if they survive the early phase of the attack and are transferred to the ward a substantial number have a protracted course.

Even after discharge from hospital it is useful to keep up to date with your patients’ conditions since many of them may face long-term sequelae, particularly diabetes mellitus, that may be a late consequence of the disease and your management of the initial necrotising process. Malabsorption and chronic or relapsing pancreatitis are also possible. This awareness may influence further therapeutic interventions and is an integral part of quality enhancement.
