Management in Acute Pancreatitis

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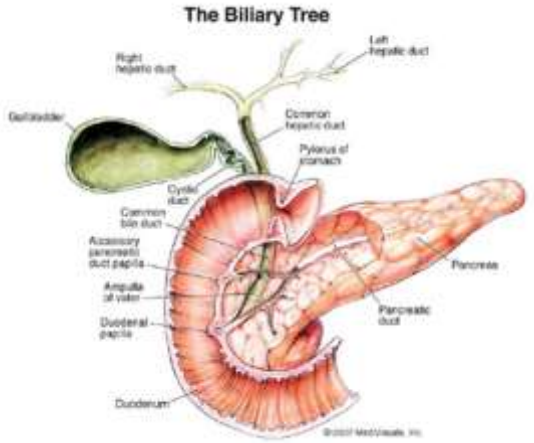
Acute pancreatitis

Definition

Acute pancreatitis - definition

- Inflammatory disease caused by activation, interstitial liberation and autodigestion of the pancreas by its own enzymes
- A group of reversible lesions characterised by inflammation of the pancreas



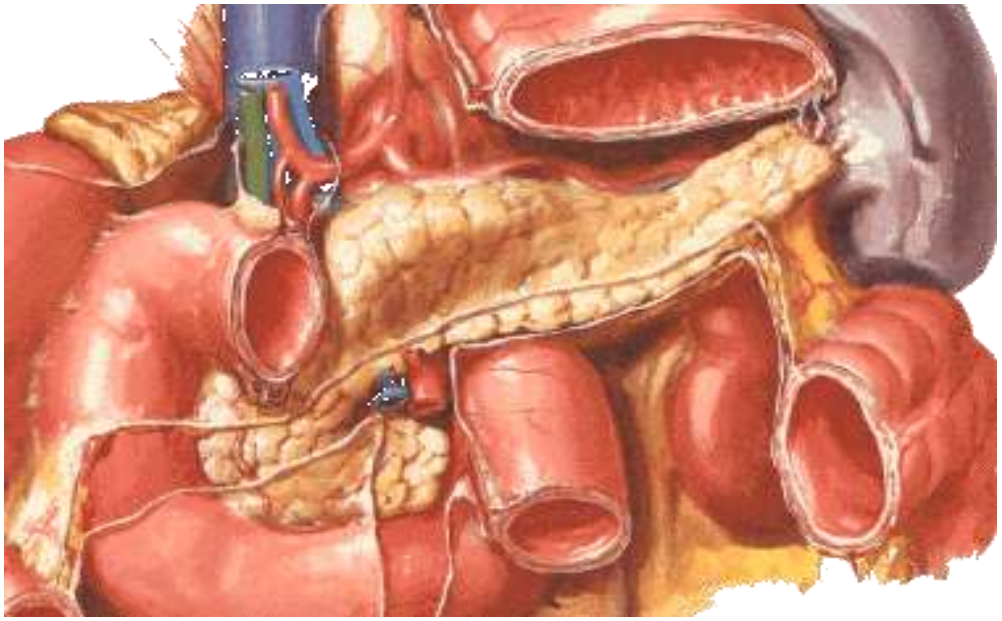
Bradley EL III. Atlanta, Ga, September 11-13, 1992. Arch Surg 1993,128, 586-590
Cavallini G, Uomo G, Pezilli R et al – Pancreatology 2001, 1: 129-199

Acute pancreatitis

Anatomy

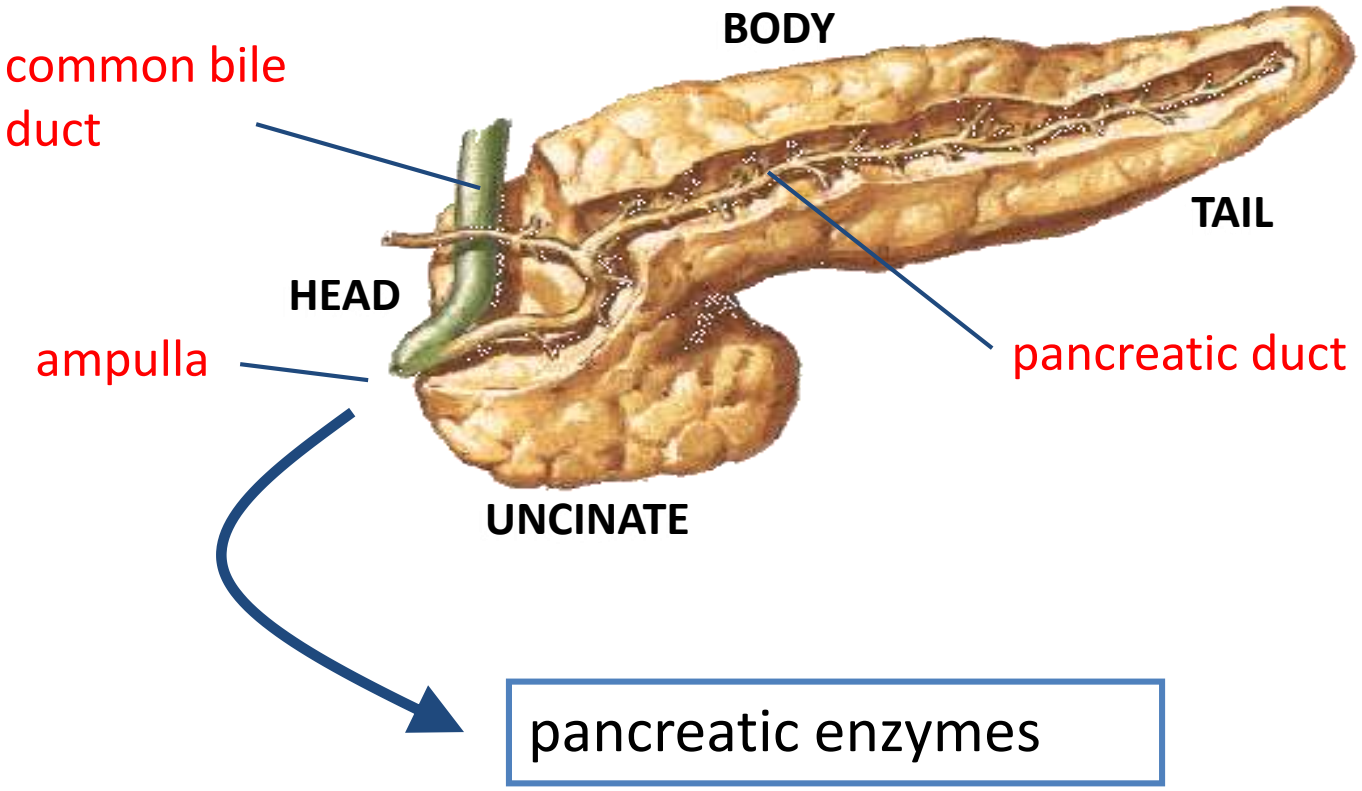
Physiology

Normal Anatomy & Physiology

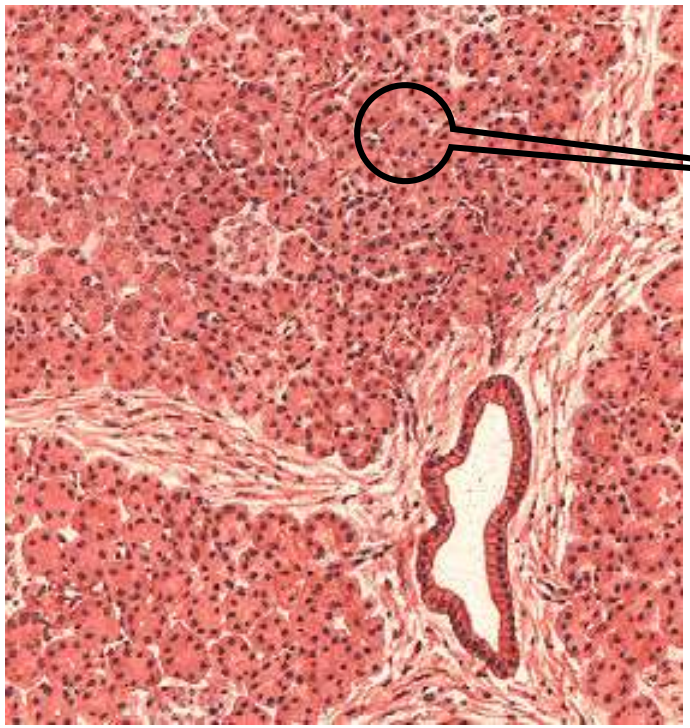


- neutralize chyme
- digestive enzymes
- hormones

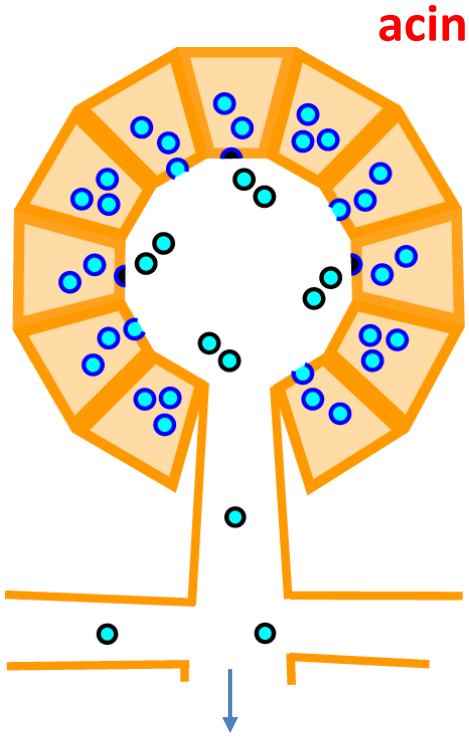
Exocrine Function



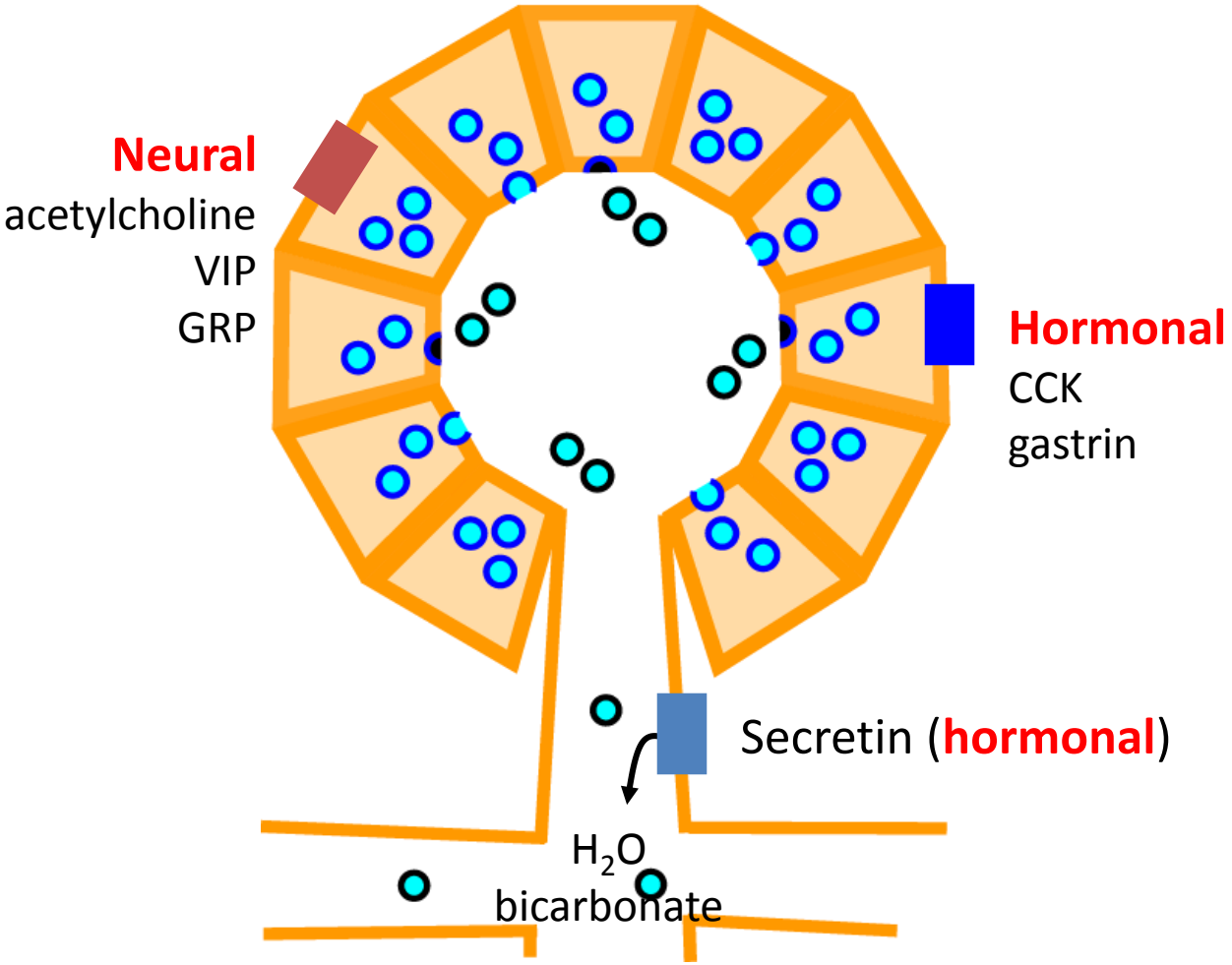
Enzyme Secretion



microscopic view
of pancreatic acini



Enzyme Secretion



Digestive Enzymes in the Pancreatic Acinar Cell

PROTEOLYTIC ENZYMES

Trypsinogen
Chymotrypsinogen
Proelastase
Procarboxypeptidase A
Procarboxypeptidase B

AMYOLYTIC ENZYMES

Amylase

LIPOLYTIC ENZYMES

Lipase
Prophospholipase A2
Carboxylesterase lipase

NUCLEASES

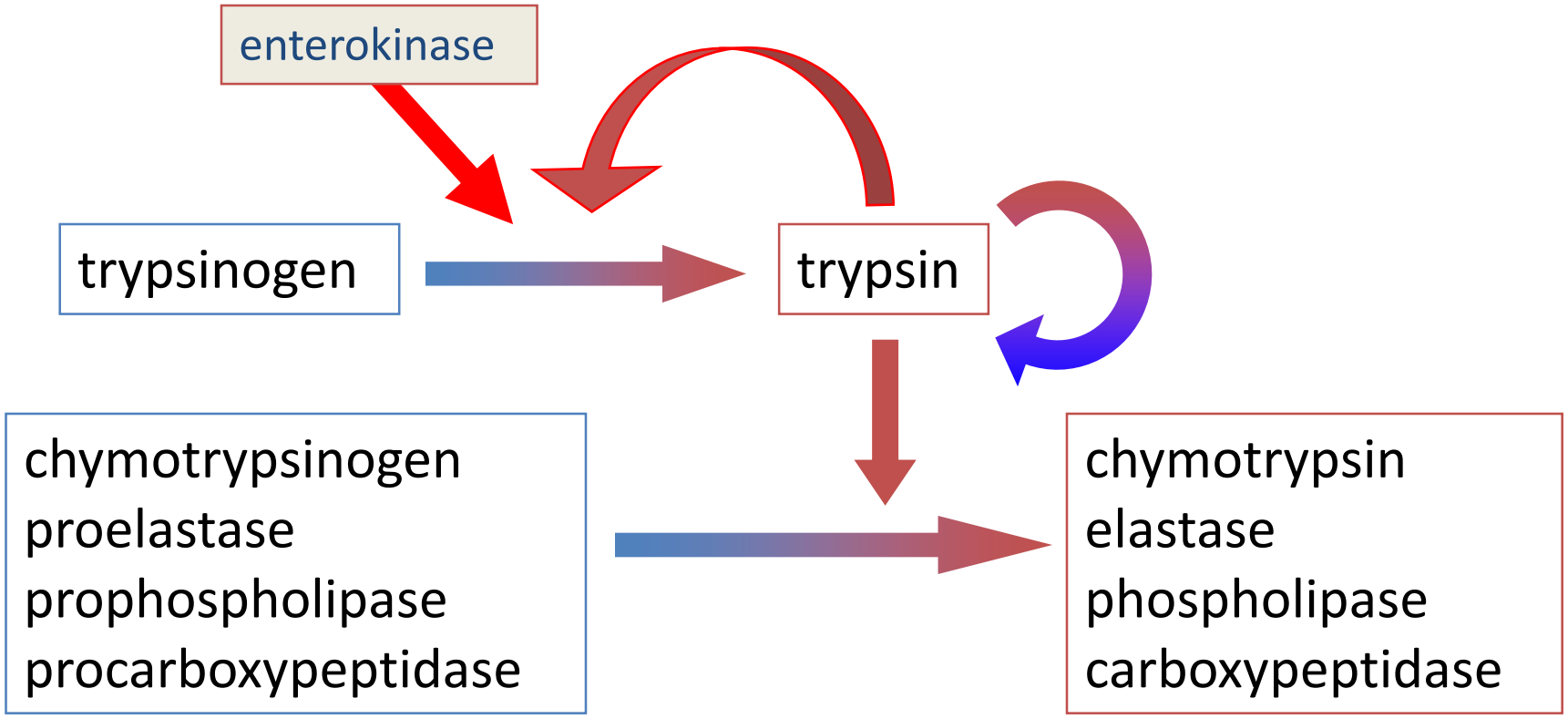
Deoxyribonuclease (DNAse)
Ribonuclease (RNAse)

OTHERS

Procolipase
Trypsin inhibitor

Normal Enzyme Activation

duodenal lumen



Exocrine Stimulation

- The more proximal the nutrient infusion…the greater the pancreatic stimulation *(dog studies)*
  - stomach – maximal stimulation
  - duodenum – intermediate stimulation
  - jejunum – minimal / negligible stimulation
- Elemental formulas tend to cause less stimulation than standard intact formulas
  - intact protein > oligopeptides > free amino acids
- Intravenous nutrients (even lipids) do not appear to stimulate the pancreas

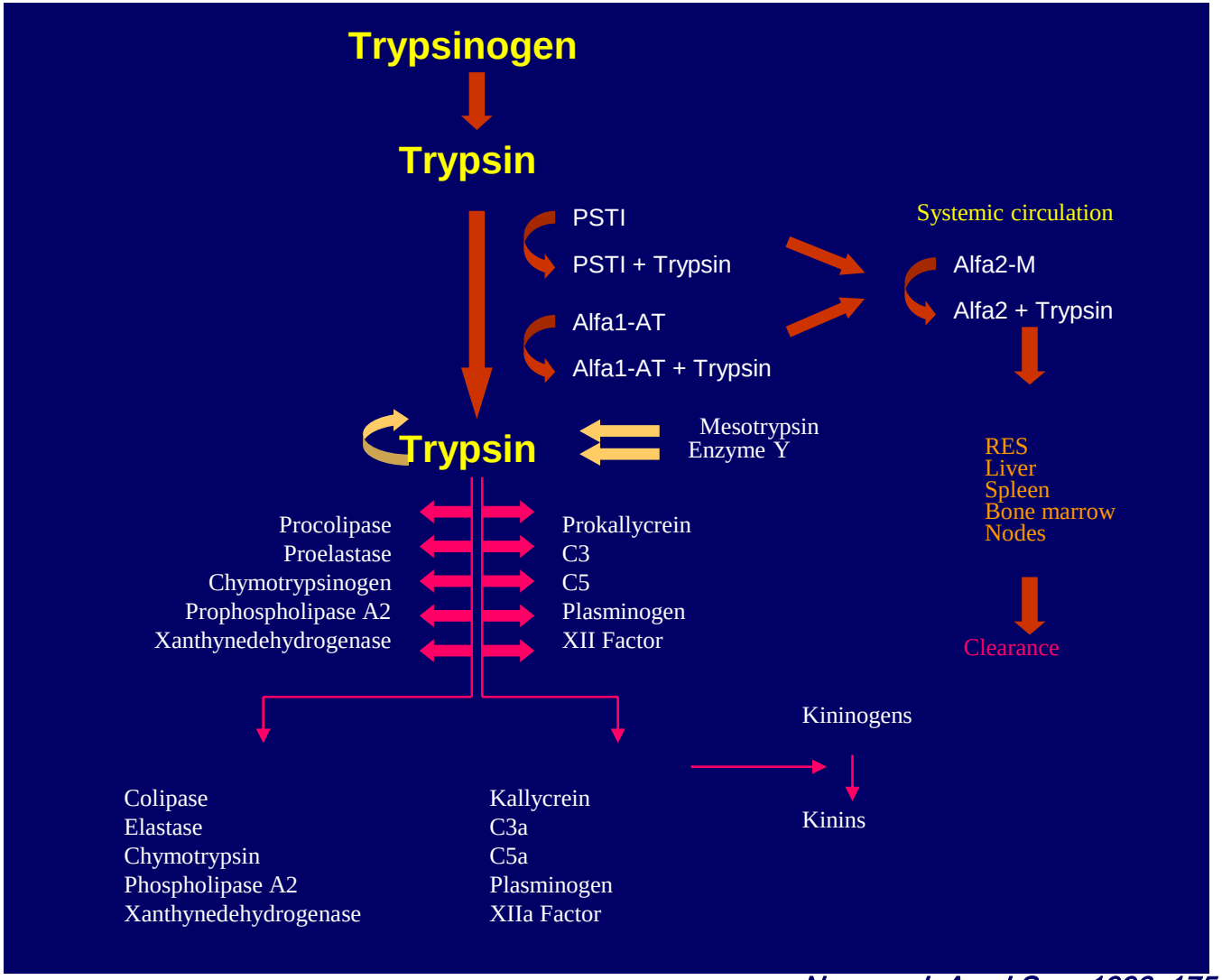
Protective Measures

- COMPARTMENTALIZATION - digestive enzymes are contained within *zymogen granules* in acinar cells
- REMOTE ACTIVATION - digestive enzymes are secreted as *inactive proenzymes* within the pancreas
- PROTEASE INHIBITORS – *trypsin inhibitor* is secreted along with the proenzymes to *suppress* any premature enzyme activation
- AUTO "SHUT-OFF" – trypsin *destroys* trypsin in high concentrations

Acute pancreatitis

Pathogenesis

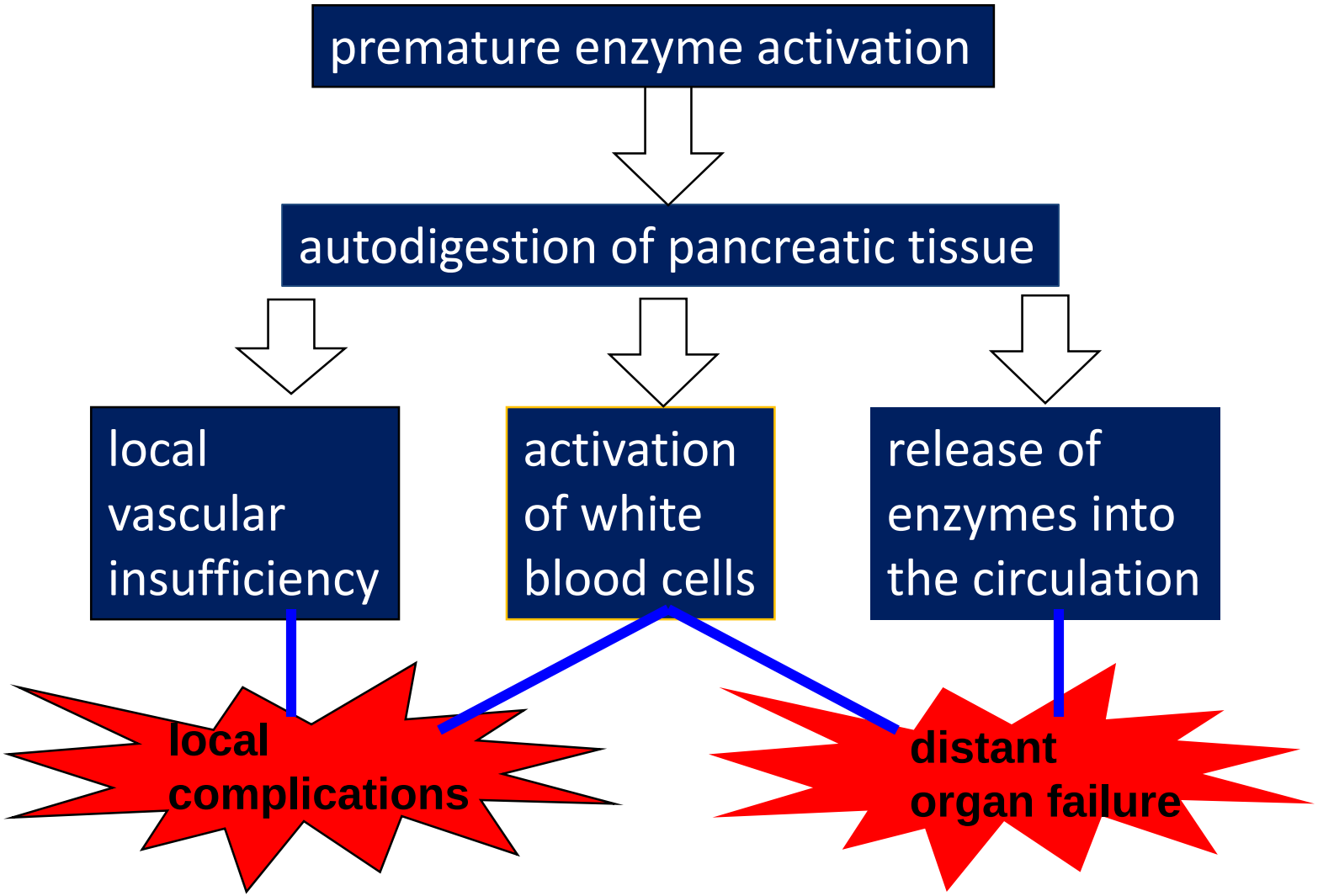
Pathogenesis



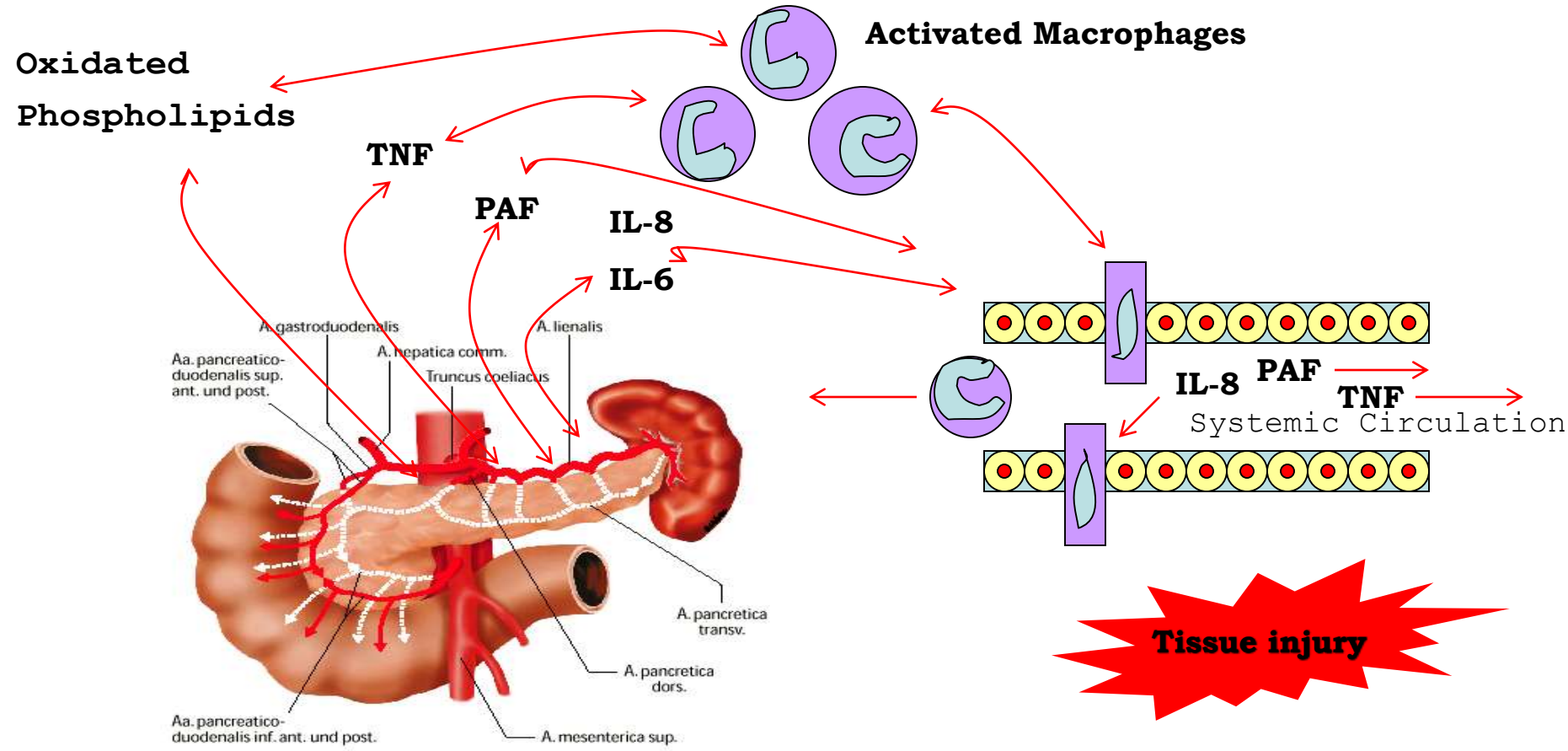
Norman J. Am J Surg 1998; 175:76-83. (modified)

Acute Pancreatitis

Pathogenesis



Pathophysiology



Link between pankreatic inflammation and sytemic tissue damage

(from R.Stocker – "Acute pancreatitis nutritional simposium" 2000

Relationship Between the Systemic and Cellular Immune Responses

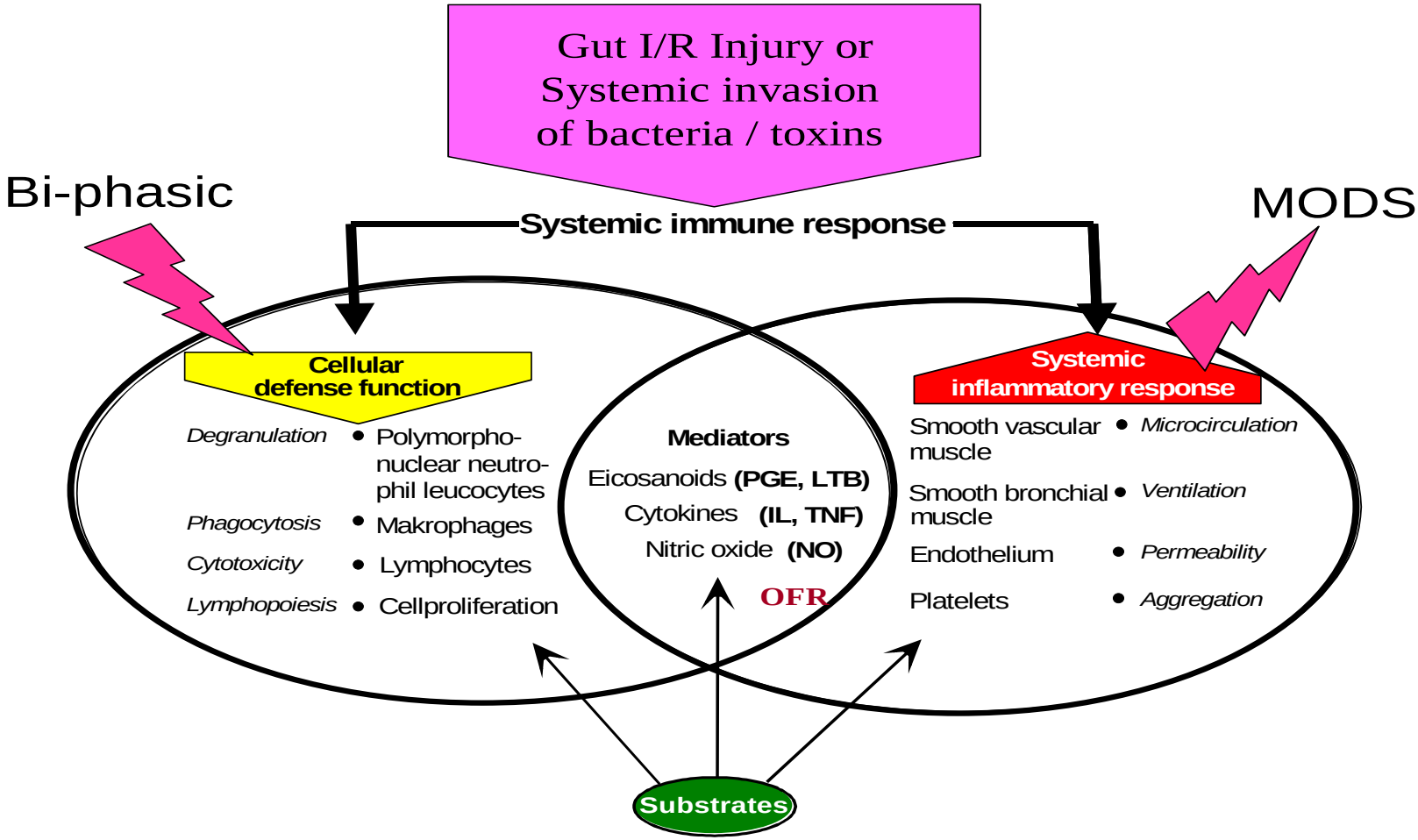
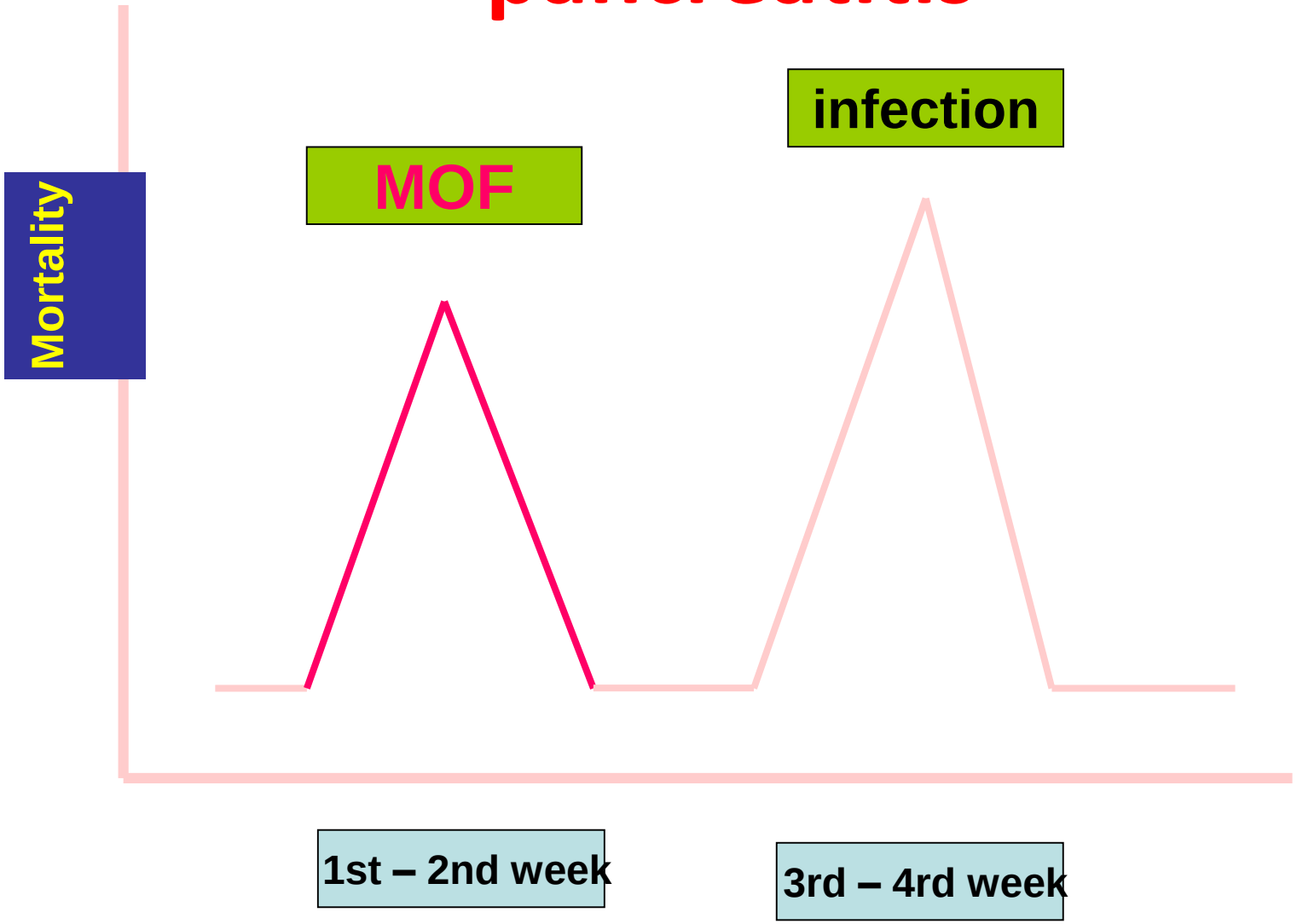
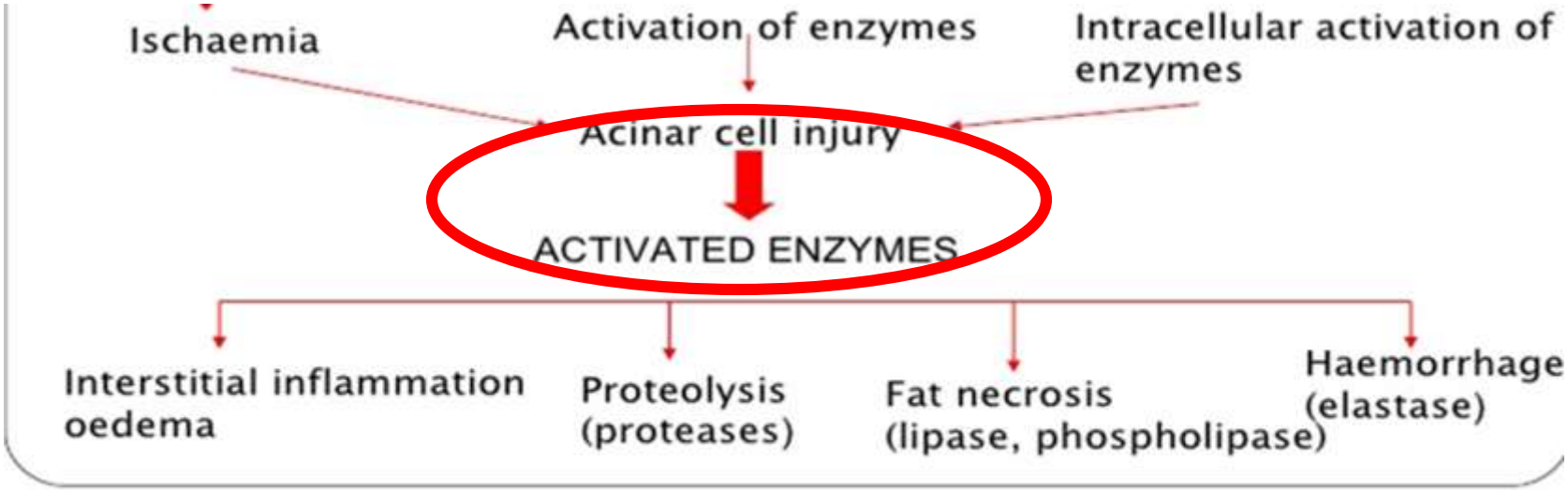


Fig.: 1

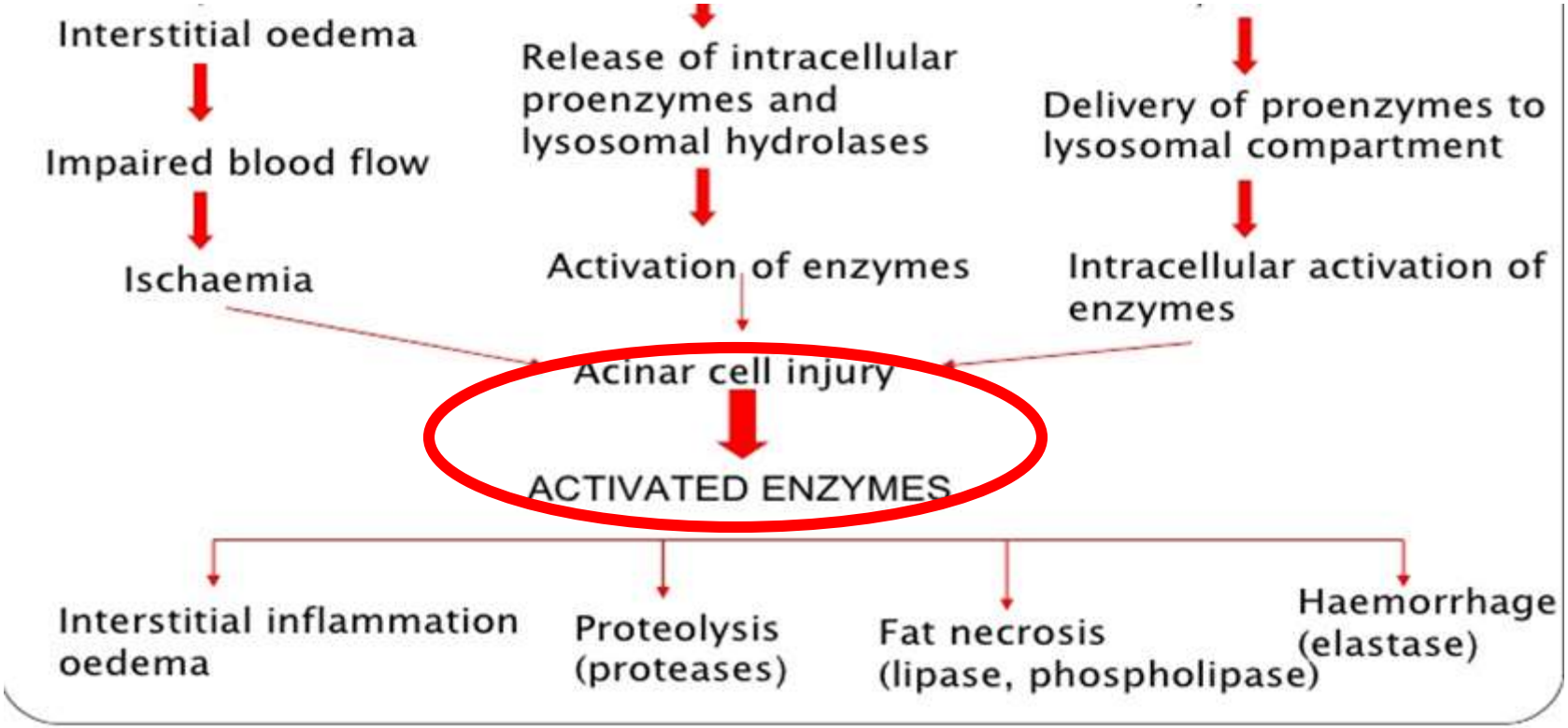
Two mortality peaks of severe acute pancreatitis



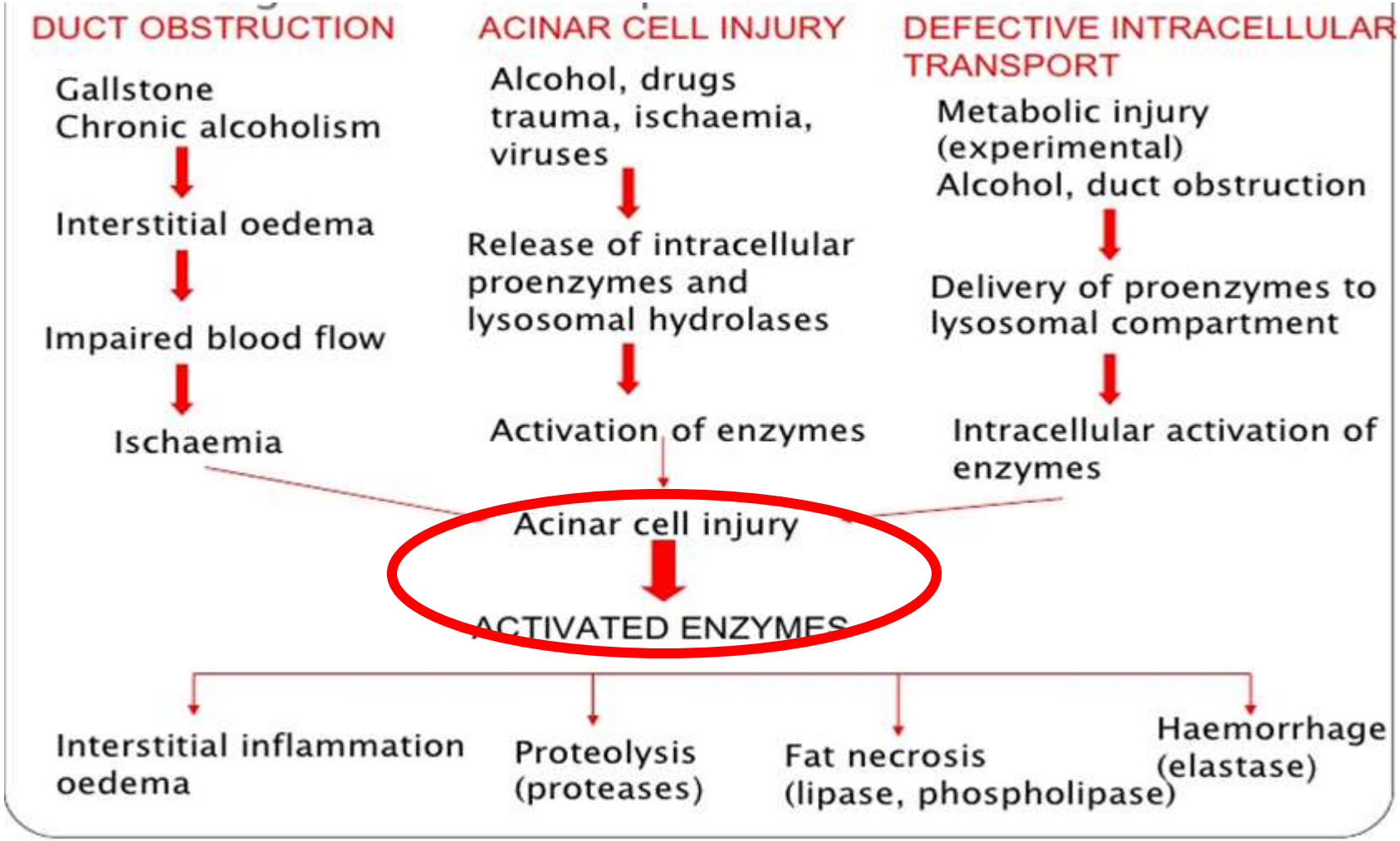
Pathophysiology



Pathophysiology



Pathophysiology



Acute pancreatitis

Etiology

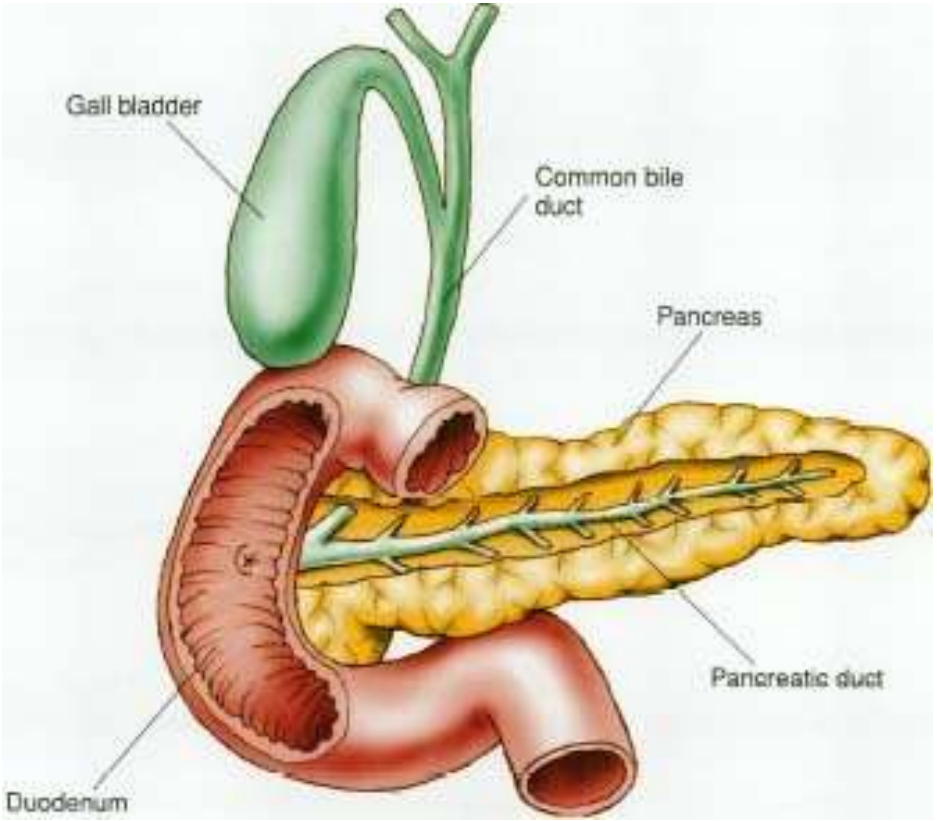
Etiology

- Non-traumatic (75%)
  - **Biliary tract disease**
  - **Alcohol**
  - Viral infection (EBV, CMV, mumps)
  - Drugs (steroid, thiazide, furosemide)
  - Scorpion bites
  - Hyperlipidemia
  - Hyperparathyroidism
- Traumatic (5%)
  - Operative trauma
  - Blunt/penetrating trauma
  - Lab test ERCP/angiography
- Idiopathic (20%)

Etiology

Gallstones (35%-60%)

- Gallstone pancreatitis risk is highest among patients with small GS < 5mm and with microlithiasis
- GS pancreatitis risk is also increased in women > 60 yrs



Etiology

Alcohol (30-40%)

- Mechanism not fully understood
- Not all alcoholics get pancreatitis (only about 15%)
- This suggests a subset of the population predisposed to pancreatitis, with alcohol acting more as a co-precipitant

Etiology – Trauma

Blunt Trauma

- Automobile
- Bicycle handlebar injuries
- Abuse

Iatrogenic – ERCP (1-7%)

- Likely secondary to contrast but also very operator dependant
- Risk is also increased with Sphincter of Oddi manometry

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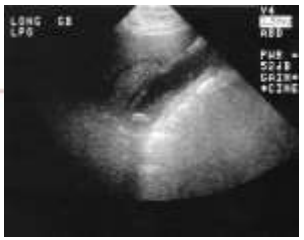
CME

American College of Gastroenterology Guideline: Management of Acute Pancreatitis

Scott Tenner, MD, MPH, FACG[1], John Baillie, MB, ChB, FRCP, FACG[2], John DeWitt, MD, FACG[3] and Santhi Swaroop Vege, MD, FACG[4]



Etiology - 1

Transabdominal ultrasound should be performed in all patients with acute pancreatitis (strong recommendation, low quality of evidence)

In the absence of gallstones and/or history of alcohol use serum triglyceride should be obtained and considered the etiology if > 1000 mg/dL (conditional recommendation, moderate quality of evidence)

In a patient older than 40 years, a pancreatic tumor should be considered as a possible cause of AP (conditional recommendation, low quality of evidence)

Am J Gastroenterol 2013; 108:1400–1415; doi:10.1038/ajg.2013.218; published online 30 July 2013

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Etiology - 2

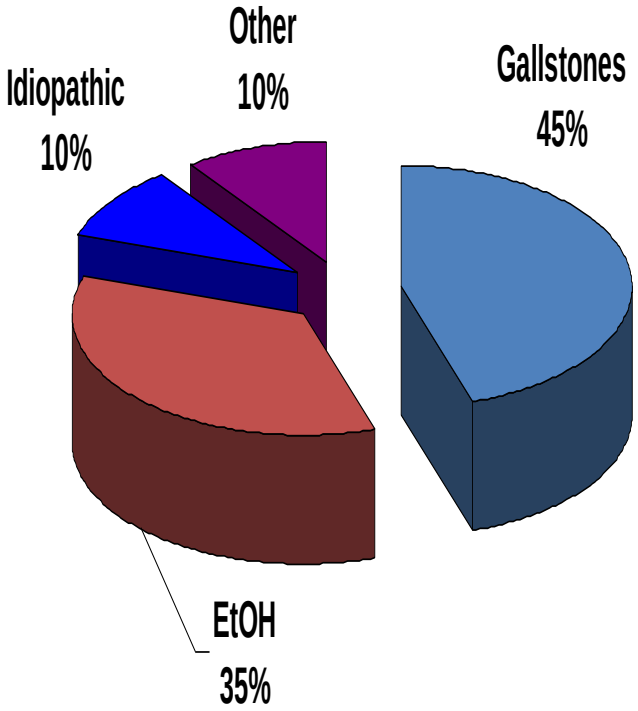
Endoscopic investigation in patients with acute idiopathic pancreatitis should be limited, as the risks and benefits of investigation in these patients are unclear (conditional recommendation, low quality of evidence)

Patients with idiopathic pancreatitis should be referred to centers of expertise (conditional recommendation, low quality of evidence)

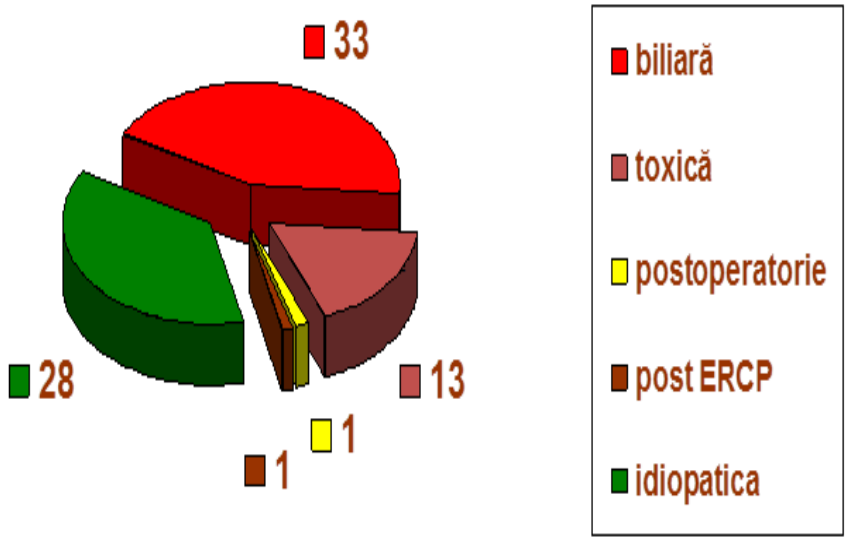
Genetic testing may be considered in young patients (< 30 years old) if no cause is evident and a family history of pancreatic disease is present (conditional recommendation, low quality of evidence)

Am J Gastroenterol 2013; 108:1400–1415; doi:10.1038/ajg.2013.218; published online 30 July 2013

Acute Pancreatitis Etiology



Fagenholz H et al. AEP 2007;17: 491-49

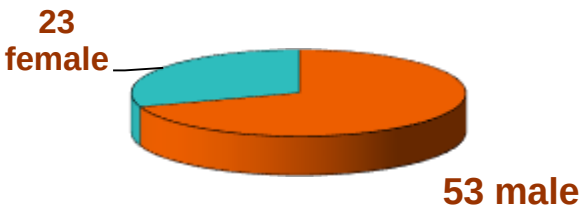


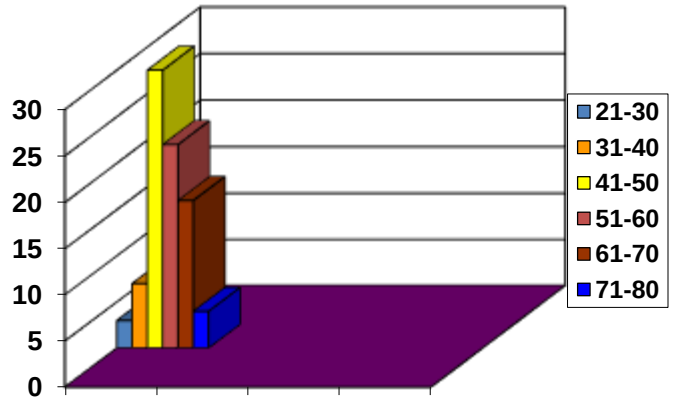
Clinical Emergency Hospital of Bucharest, 2009

Incidence

Male:female ratio is

- 1:3– in those with gallstone and
- 6:1 in those with alcoholism





Sex and age distribution of AP

Clinical Emergency Hospital

Famous people who have had pancreatitis

Alexander the Great

Ludwig von Beethoven

Dizzie Gillespie

Maximilian Schell

John Ashcroft

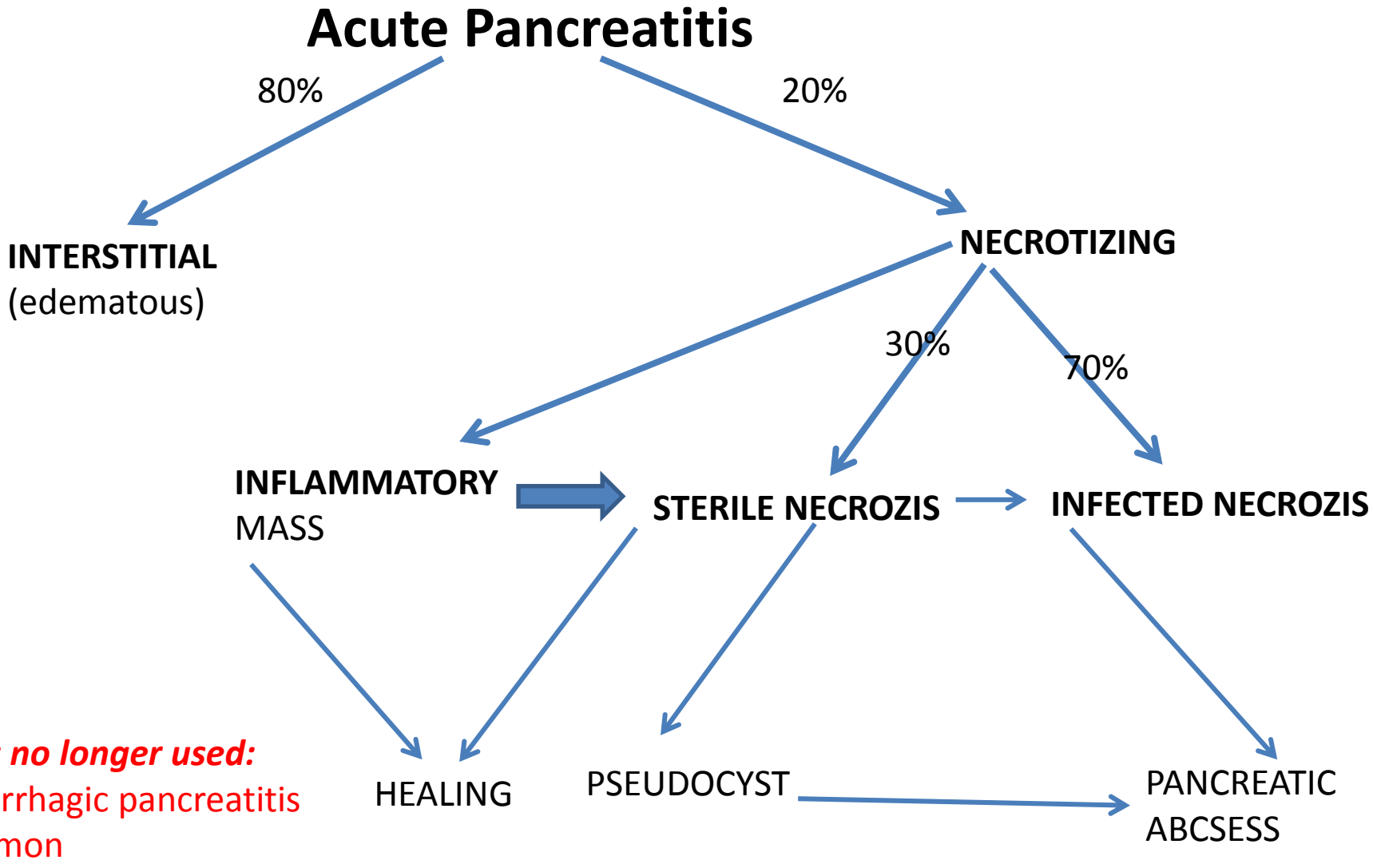
Acute pancreatitis

Terminology

Classification

Epidemiology

Terminology of Acute Pancreatitis



Terms no longer used:

Hemorrhagic pancreatitis

Phlegmon

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Definitions of severity in acute pancreatitis: comparison of Atlanta and recent revision

Atlanta criteria (1993)
Mild acute pancreatitis
Absence of organ failure
Absence of local complications
Severe acute pancreatitis
1. Local complications **AND/OR**
2. Organ failure
GI bleeding (>500 cc/24 hr)
Shock – SBP ≤90 mm Hg
PaO 2 ≤60%
Creatinine ≥2 mg/dl

Atlanta Revision (2013)
Mild acute pancreatitis
Absence of organ failure
Absence of local complications
Moderately severe acute pancreatitis
1. Local complications **AND/OR**
2. Transient organ failure (<48 h)
Severe acute pancreatitis
Persistent organ failure >48 h[a]

Am J Gastroenterol 2013; 108:1400–1415; doi:10.1038/ajg.2013.218; published online 30 July 2013

Determinat- based Classification

Based on the factors that are causally associated with severity of acute pancreatitis

	Mild AP	Moderate AP	Severe AP	Critical AP
(Peri)pancreatic necrosis	NO	***Sterile***	***Infected***	***Infected***
	AND	AND/OR	OR	AND
Organ failure	NO	***Transient***	***Persistent***	***Persistent***

Determinant-Based Classification of Acute Pancreatitis Severity

An International Multidisciplinary Consultation

*E. Patchen Dellinger, MD,† Christopher E. Forsmark, MD,‡ Peter Layer, MD, PhD,§ Philippe Lévy, MD,‖ Enrique Maravi-Poma, MD, PhD,¶ Maxim S. Petrov, MD, MPH, PhD,# Tooru Shimosegawa, MD, PhD,** Ajith K. Siriwardena, MD,†† Generoso Uomo, MD,‡‡ David C. Whitcomb, MD, PhD,§§ and John A. Windsor, MBChB, MD, FRACS##; on behalf of the Pancreatitis Across Nations Clinical Research and Education Alliance (PANCREA)*

Determinat- based Classification

Based on the factors that are causally associated with severity of acute pancreatitis

Definition

Local determinanat

(Peri)pancreatic necrosis

- Pancreatic, peiripancreatic, both
- Solid, semisolid, without a radiologically wall

Sterile (peri)pancreatic necrosis

- Absence of proven infection in necresis

Infected (peri)pancreatic necrosis

- Gas bubbles within (CT)
- Positive culture obtained
  - by image guiidet fine needle aspiration
  - During the first drainage or necrosectomy

Systemic determinanat

Organ failure

- *Cardiovascular*:
  need for inotropic agent
- *Renal*:

creatinine≥ 171μmol/L(≥2.0 mg/dL)

- *Respuratory*:

$PaO_2/ FiO_2 \leq 300$ mmHg (≤40kPa)

Persistent organ failure

For 48 hours or more

Transient organ failure

For less than 48 hours

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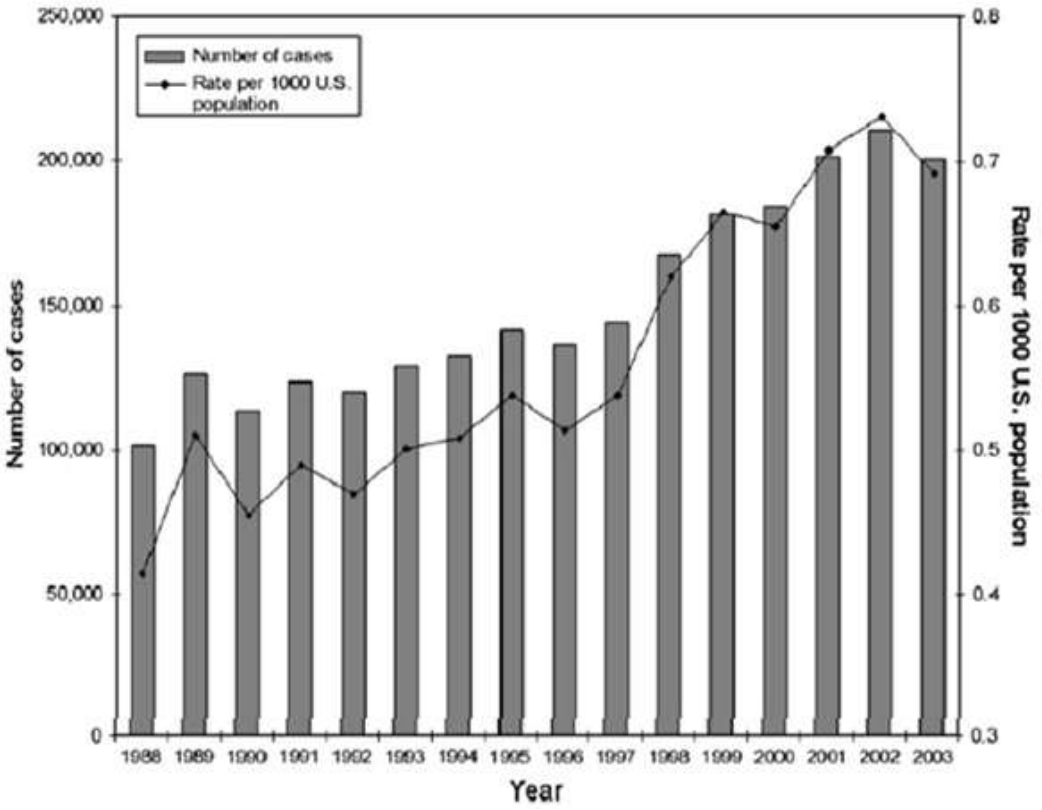
Acute Pancreatitis – Epidemiology



Increases in total hospitalisation for acute pancreatitis and in the population rate of hospitalisation for acute pancreatitis during the study period (p for trend = 0.001 for both)

Fagenholz PJ1, Castillo CF, Harris NS, Pelletier AJ, Camargo CA Jr Increasing United States hospital admissions for acute pancreatitis, 1988-2003.Ann Epidemiol. 2007 Jul;17(7):491-7. Epub 2007 apr 19.

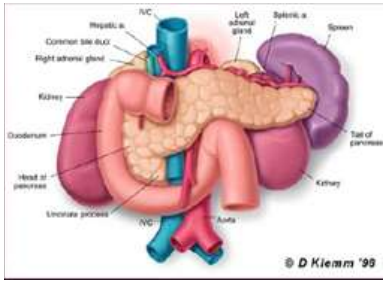
Acute pancreatitis

Diagnosis

Clinical Presentation

Pain (95%)

- Acute onset
  - Mid-abdominal or mid-epigastric
  - Radiates to the back (50%)
- Peak intensity in 30 minutes
  - Lasts for several hours



Nausea and vomiting (80%)

Abdominal distention (75%)

Abdominal guarding and tenderness (50%)

Restlessness and agitation

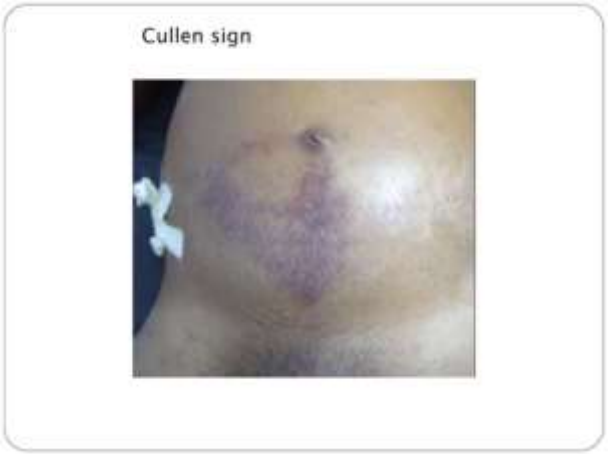
Differential Diagnosis

- Choleledocholithoasis
- Perforated ulcer
- Mesenteric ischemia
- Intestinal obstructuion
- Ectopic pregnancy

Clinical signs

More severe cases

- Jaundice
- Ascites
- Pleural effusions – generally left-sided
- *Cullen's sign – bluish peri-umbilical discoloration*
- *Grey Turner's sign – bluish discoloration of the flanks*



Labs

Amylase

- Elevates within HOURS and can remain elevated for 4-5 days
- High specificity when using levels >3x normal
- Most specific = pancreatic isoamylase (fractionated amylase)
- **Many false positives**

Diferential diagnosis – Amylase Elevation

Pancreatic Source

- Biliary obstruction
- Bowel obstruction
- Perforated ulcer
- Appendicitis
- Mesenteric ischemia
- Peritonitis

Salivary

- Parotitis
- DKA
- Anorexia
- Fallopian tube
- Malignancies

Unknown Source

- Renal failure
- Head trauma
- Burns
- Postoperative

Labs

Lipase

- The preferred test for diagnosis
- Begins to increase 4-8H after onset of symptoms and peaks at 24H
- Remains elevated for days
- Sensitivity 86-100% and Specificity 60-99%
- >3X normal S&S ~100%

Lab Investigations

- Full blood count:neutrophil, *leucocytosis*
- Electrolyte abnormalities include *hypokaemia,hipocalcemia*
- Elevated LDH in biliary desease
- *Glycosuria* (10% of cases)
- Blood sugar: hyperglycaemiain severe cases
- Ultrasound look for stones diseases

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Diagnosis

The diagnosis of AP is most often established by the presence of two of the three following criteria: (I) abdominal pain; (II) serum amylase and/or lipase greater than three times the upper limit of normal, and/or (III) characteristic findings from abdominal imaging (strong recommendation, moderate quality of evidence)

Contrast-enhanced CT and/or MRI of the pancreas should be reserved for patients in whom the diagnosis is unclear or who fail to improve clinically within the first 48-72 h after hospital admission (strong recommendation, low quality of evidence)

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Diagnosis – Imaging

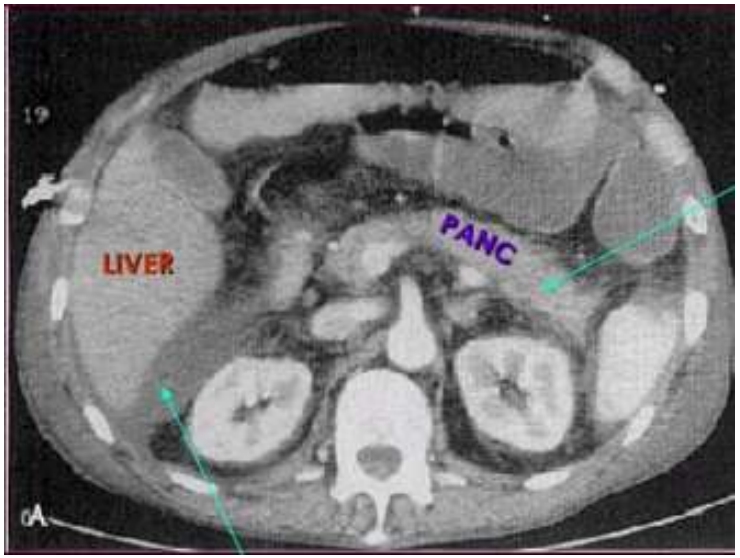
CT

- Excellent pancreas imaging
- Recommended in all patients with persisting organ failure, sepsis or deterioration in clinical status (6-10 days after admission)
- Search for necrosis – will be present at least 4 days after onset of symptoms; if ordered too early it will underestimate severity
- Follow-up months after presentation as clinically warranted for CT severity index of >3

CT Findings

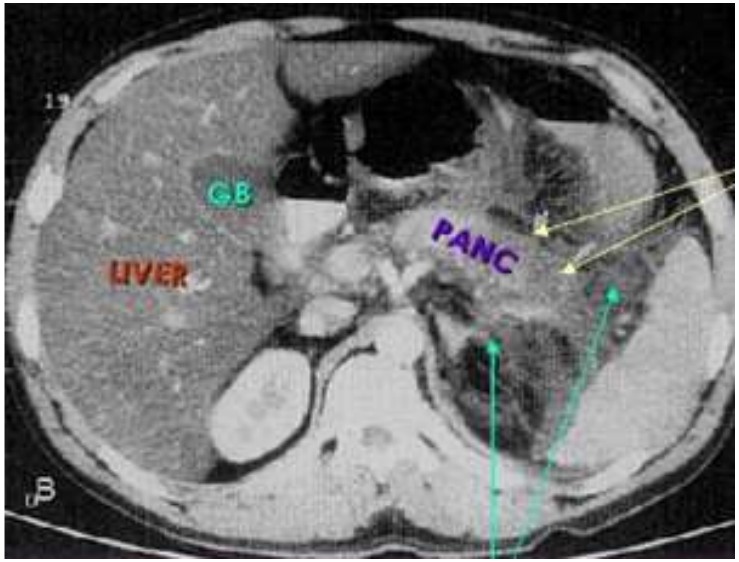
Severe Pancreatitis

72 H



Tail Indistinct

Intraperitoneal fluid



Unenhancing Necrosis

Peripancreatic edema and inflammation

Balthazar's score

Severe = Score > 6 (CT Grade + Necrosis)

CT Grade

– Normal	A	0 points
– Focal or diffuse enlargement	B	1 point
– Intrinsic change or fat stranding	C	2 points
– Single ill-defined fluid collection	D	3 points
– Multiple collections of fluid or gas	E	4 points

Necrosis Score

– None	0 points
– 1/3 of pancreas	2 points
– 1/2 of pancreas	4 points
– > 1/2 of pancrease	6 points

Balthazar EJ, Robinson DL, Megibow AJ. Radiology 1990; 174(2): 331- 336

CT Severity Index

appearance	normal	enlarged	inflamed	1 fluid collection	2 or more collections
grade	A	B	C	D	E
score	0	1	2	3	4

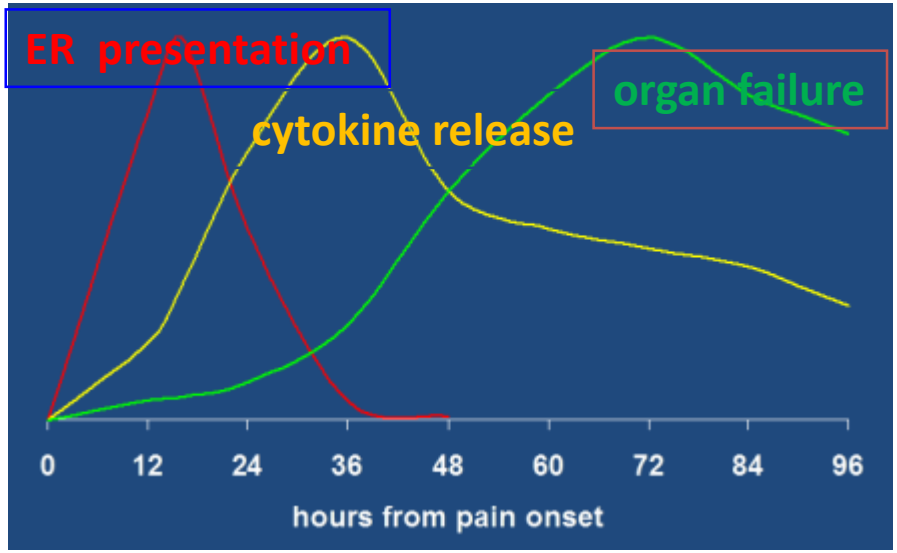
necrosis	none	< 33%	33-50%	> 50%
score	0	2	4	6

score	morbidity	mortality
1-2	4%	0%
7-10	92%	17%

Balthazar et al. Radiology 1990

Acute pancreatitis

Predictors of Severity



Predictors of Severity

Why are they needed?

- appropriate patient triage & therapy
- compare results of studies of the impact of therapy

When are they needed?

- optimally, within first 24 hours (damage control must begin *early*)

Which is best?

Determining severity

- Clinical criteria
    - Early development/persistence of organ dysfunction
  - Ranson criteria
  - Atlanta criteria
  - POP score
  - BISAP
- Clinical assessment
    - Frequent VS, fluid status/UOP, pulse oximetry
- Radiographic criteria
  - CT severity index
    - Necrosis may not be evident until 48-72h

Ranson Criteria

At admission

1. Age>55 years
2. WBC >16.000/mm3
3. Glucose >200 mg/dl
4. LDH >350 UI/l
5. AST >250 U/l

Number	<2	3-4	5-6	7-8
Mortality	1%	16%	40%	100%

Wthin 48 Hours

1. Hct decrease of >10mg/dl
2. BUN increase of >5mg/dl
3. Base deficit >4 mEq/l
4. Fluid sequestration >6L
5. Ca++ < 8mg/dl
6. PaO2< 60 mmHg

- Directly related to fluid resuscitation
- Independent predictors of mortality

Ranson JH et all. Surgery Gynecology and Obstetrics-1974; 139(1):69-84

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American College of Gastroenterology Guideline: Management of Acute Pancreatitis

Scott Tenner, MD, MPH, FACG[1], John Baillie, MB, ChB, FRCP, FACG[2], John DeWitt, MD, FACG[3] and Santhi Swaroop Vege, MD, FACG[4]

Clinical findings associated with a severe course for initial risk assessment – Intrinsic patient-related risk factors for developing of severe disease

Patient characteristics
Age >55 years (53,57)
Obesity (BMI >30 kg/m^2) (68)
Altered mental status (69)
Comorbid disease (53)

The systemic inflammatory response syndrome (SIRS) (6,53,54,70,71) Presence of >2 of the following criteria:
– pulse >90 beats/min
– respirations >20/min or $PaCO_2$ >32 mm Hg
– temperature >38 °C or <36 °C
–WBC count >12,000 or <4,000 cells/mm^3 or >10% immature neutrophils (bands)

Laboratory findings
BUN >20 mg/dl (63)
Rising BUN (63)
HCT >44% (62)
Rising HCT (62)
Elevated creatinine (72)

Radiology findings
Pleural effusions (73)
Pulmonary infiltrates (53)
Multiple or extensive extrapancreatic collections (67)

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Acute pancreatitis

Treatment

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Initial assessment and risk stratification

Risk assessment should be performed to stratify patients in to higher and lower risk categories to assist triage, such as admission to ICU (conditional recommendation, moderate quality of evidence)

Patients with organ failure should be admitted to ICU or intermediary care setting whenever possible (strong recommendation, low quality of evidence)

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When Do I Transfer to the Intensive Care?

- Severe pancreatitis
- Multi-organ failure
  - Pulmonary
  - Renal
- Consider it if you are placing the patient on antibiotics and/or ordering a CT to evaluate non-improvement

When Do I Transfer to the Intensive Care?

- Cardiovascular
  - Hypotension
  - ↑HR, ↑ CO and ↓SVR
- Respiratory
  - Hypoxemia
  - Pleural effusion
- Renal
  - ATN
  - Oliguria
- Haematologic
  - DIC
  - Thrombocytosis
- Hepatic
  - Encephalopathy
  - ↑T bili (3mg/dl)
  - ↑ AST/ALT 2x nl
- GI
  - Stress ulcer
  - Acalculous cholecystitis

Therapeutical goals

- Ventilatory support
- Fluid resuscitation
- Haemodynamic support (vasopresors, inotropes)
- Antibiotherapy
- Sedation
- Analgesia
- Early enteral nutrition
- Glycemic and triglycerides control
- Prokinetics
- Stress ulcer prophylaxis
- Thromboprophylaxis

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Fluids initial management

Aggressive hydration, defined as 250-500 ml/h of isotonic crystalloid solution should be provided to all patients, unless cardiovascular and/or renal comorbidites exist. Early aggressive iv hydration is most beneficial the first 12-24 h, and may have little benefit beyond (strong recommendation, moderate quality of evidence)

In patients with severe volume depletion (hypotension and tachycardia), more rapid repletion (bolus) may be needed (conditional recommendation, moderate quality of evidence)

Lactated Ringer's solution may be preferred (conditional recommendation, moderate quality of evidence)

Fluid requirements should be reassessed every 6h for the next 24-48 h. The goal of aggressive hydration should be to decrease the blood urea nitrogen (strong recommendation, moderate quality of evidence)

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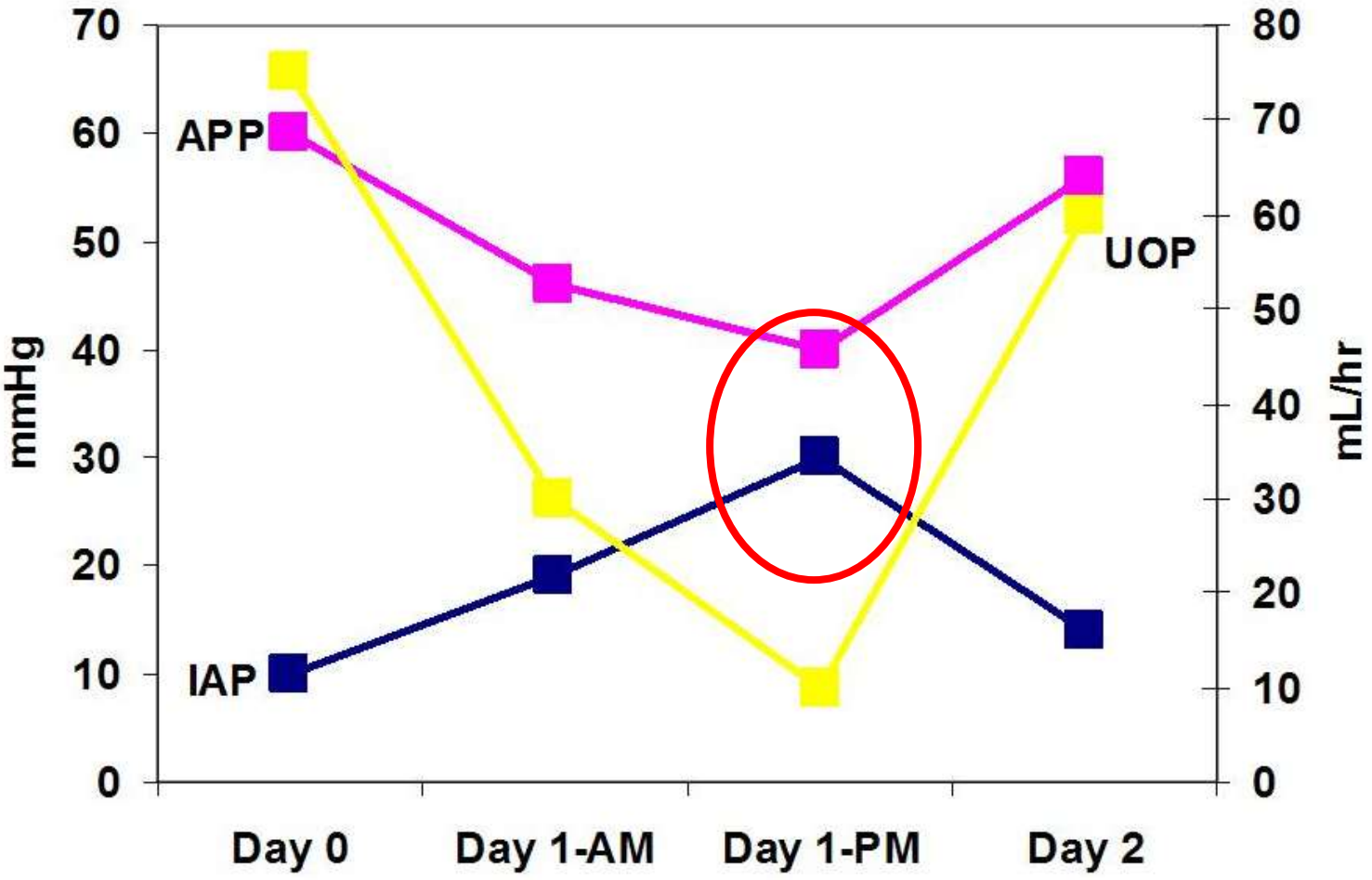
Fluid Resuscitation

- Fluid resuscitation volume should be carefully monitored to avoid over-resuscitation in patients at risk for IAH/ACS (Grade 1B)
- Hypertonic crystalloid and colloid-based resuscitation should be considered in patients with IAH to decrease the progression to secondary ACS (Grade 1C)
- Fluid resuscitation and “early goal-directed therapy” are cornerstones of critical care management
- Excessive fluid resuscitation is an independent predictor of IAH/ACS and should be avoided
- The use of goal-directed hemodynamic monitoring should be considered to achieve appropriate fluid resuscitation

Balogh Z et al.(2003) Arch Surg 138:637–642; McNelis J et al (2002) Arch Surg 137:133–136; Oda J et al (2006) J Trauma 60:64–71; O’Mara MS et al. (2005) J Trauma 58:1011–1018

Graphic Display Of IAP, APP, UOP



How IAP Should Be Measured?

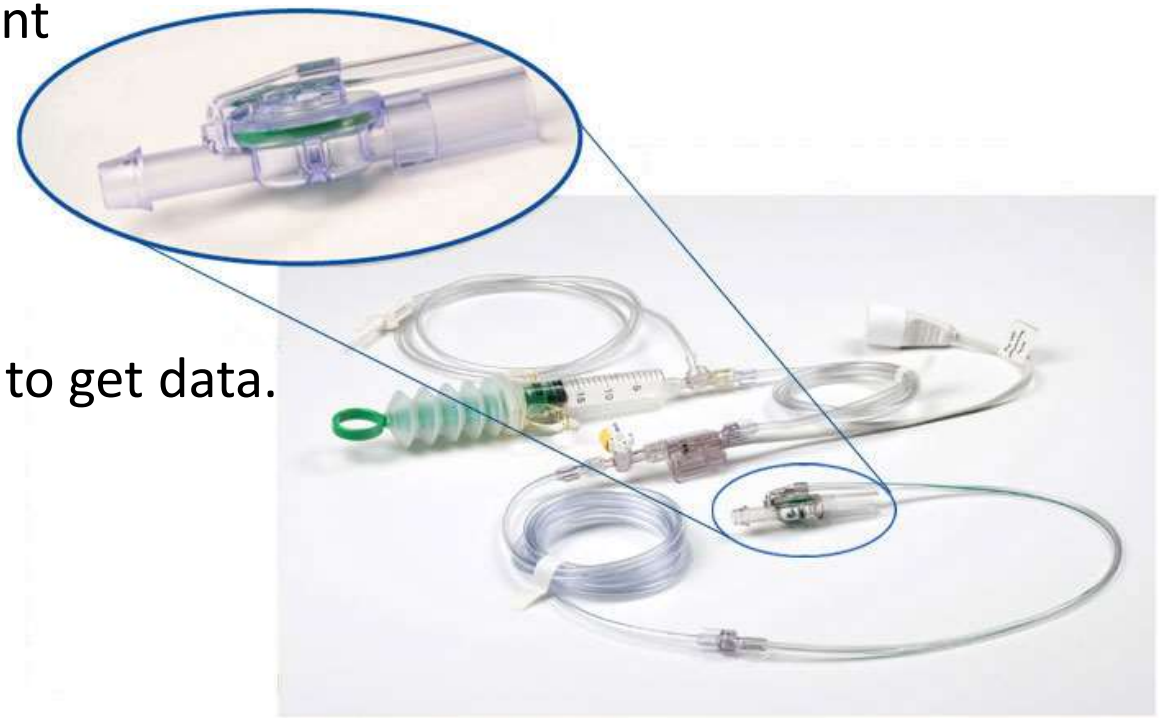
- If two or more risk factors for IAH / ACS are present, a baseline IAP measurement should be obtained (Grade 1B)
- If IAH is present, serial IAP measurements should be performed throughout the patient's critical illness (Grade1C)

- Physical examination is insensitive in detecting IAH
- IAP monitoring is a cost-effective, safe, and accurate tool for identifying the presence of IAH and guiding resuscitative therapy for ACS
- Serial IAP measurements are necessary to guide resuscitation of patients with IAH / ACS

Malbrain ML et al. (2005) Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. • Crit Care Med 33:315–322 Ivatury RR et al (1998) Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: prophylaxis, incidence, and clinical relevance to gastric mucosal pH and abdominal compartment syndrome. • J Trauma 44:1016–1021. Balogh Z et al. (2003) Both primary and secondary abdominal compartment syndrome can be predicted early and are harbingers of multiple organ failure. J Trauma 54:848–859

Intra-Abdominal Pressure
Monitoring Kit

- Kit contains everything you need
- Standardized measurement
- No reproducibility errors
- Ease & simplicity of use
- Time savings: 30 seconds to get data.
- Closed system
- No needles
- No contamination risks



AbViser Kit

When Do I Start Antibiotics?

- Acute pan atitis - inf n ~10%
  - 30-40% of t inf
- Pro ylactic a
  - Co
    - N
    - Abx
- General r
  - Biliary pancreatiti th s
  - >30% necrosis on CT scan

Fever early in the disease process is almost universally secondary to the **inflammatory response** and **NOT an infectious process**

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The role of antibiotics in acute pancreatitis - 1

Routine use of prophylactic antibiotics in patients with severe AP is not recommended (strong recommendation, moderate quality of evidence)

The use of antibiotics in patients with sterile necrosis to prevent the development of infected necrosis is not recommended (strong recommendation, moderate quality of evidence)

Infected necrosis should be considered in patients with pancreatic or extrapancreatic necrosis who deteriorate or fail to improve after 7-10 days of hospitalization.
In these patients, either (I) initial CT-guided fine needle aspiration (FNA) for Gram stain and culture to guide use of appropriate antibiotics or (II) empiric use of antibiotics without CT FNA should be given (strong recommendation, low quality of evidence)

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The role of antibiotics in acute pancreatitis - 2

In patients with infected necrosis, antibiotics known to penetrate pancreatic necrosis, such as carbapenems, quinolones, and metronidazole, may be useful (conditional recommendation, low quality of evidence)

Antibiotics should be given for an extrapancreatic infection, such as cholangitis, catheter-aquired infections, bacteremia, urinary tract infections, pneumonia (strong recommendation, high quality of evidence)

Routine administration of antifungal agents is not recommended (conditional recommendation, low quality of evidence)

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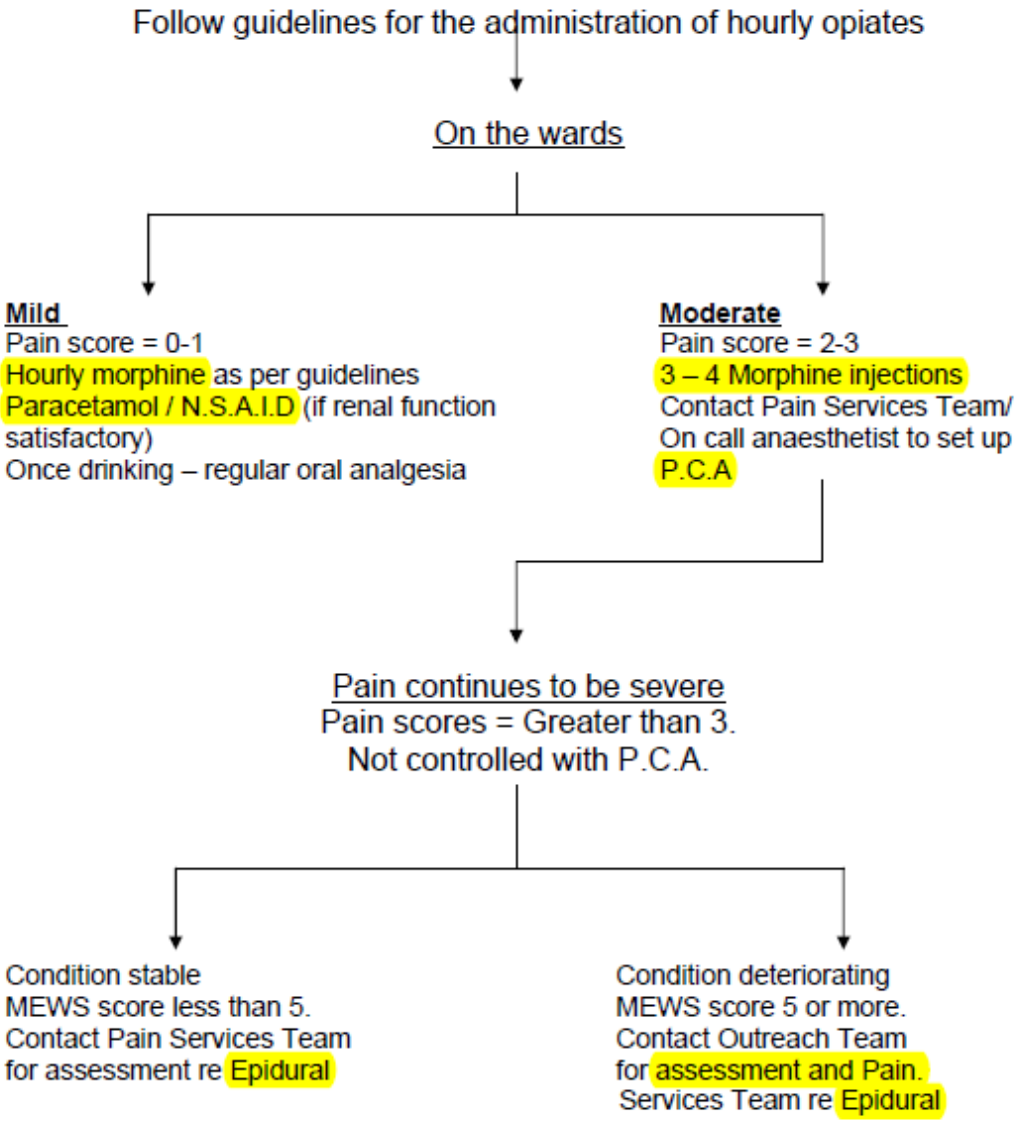
Guidelines for managing pain



Epidural analgesia

- Thoracic trauma

*(Bulger EM et al. Surgery 2004; **136:**426-430)*

- Cardiac surgery

*(Liu SS et al. Anesthesiology 2004, **101:**153-161)*

- Acute pancreatitis
- The effectiveness and safety of epidural analgesia has also been demonstrated in critically ill patients with severe acute pancreatitis

*(Bernhardt A et al. Anaesthesiol Reanim 2002, **27:**16-22)*

Epidural analgesia

- ↓ time to extubation
- ↓ ICU stay
- ↓ incidence of renal failure
- ↓ morphine consumption during the first 24 hours
- ↓ maximal glucose and cortisol blood concentrations
- improves forced vital capacity

(Guay J. *J Anesth* 2006, **20:**335-340)

- Gold standard - thoracic epidural analgesia (TEA) with a local anaesthetic/opioid infusion

Thoracic epidural analgesia

- ↓sympathetic activity and the stress response
  (A segmental temporary sympathetic block)
- Improved mucosal capillary perfusion

Daudel F, Freise H, Westphal M, et al. Shock 2007; 28: 610–4
Freise H, Lauer S, Anthonsen S, et al. Anesthesiology 2006; 105: 354–9

- Accelerated recovery of intestinal function

(Jorgensen H, Wetterslev J, Moiniche S, Dahl JB. Cochrane Database Syst Rev 2000; CD001893)

- The faster resolution of postoperative ileus after major open surgery has been attributed to superior pain therapy, reduced opioid consumption, and sympathetic block

Epidural analgesia – adverse effects

Hypotension

- 3.0% to 10.2%
- Corelate with hypovolemia

(Wheatley RG, Schug SA & Watson D *Br J Anaesth 2001;***87**(1): 47–61)

Treatment failure

- 22% premature termination of postoperative epidural infusions
  - dislodgement (10%)
  - inadequate analgesia (3.5%)
  - sensory or motor deficit (2.2%)

(Ballantyne JC, McKenna JM & Ryder E .*Acute Pain 2003;***4**: 89–97)

Neurological injury

Epidural abscess

Nutrition

Mild - moderate pancreatitis

- Calories from IVF (D5W) are sufficient
- No benefit from additional nutritional support
- Oral intake advancing to low fat diet once pain/anorexia resolve
- NGT decompression
  - If frequent emesis or evidence of ileus on plain films
  - Tube feed if anticipate NPO > 1 week

DO NOT follow amylase and lipase levels

Nutrition

Severe AP

Enteral nutrition is preferred

- Begin nutritional support as early as possible
  - NJ tube preferred
- however nasogastric feeds have been shown to be effective in 80% of cases
- NGTs should be used with caution in patients with ACS

TPN only if

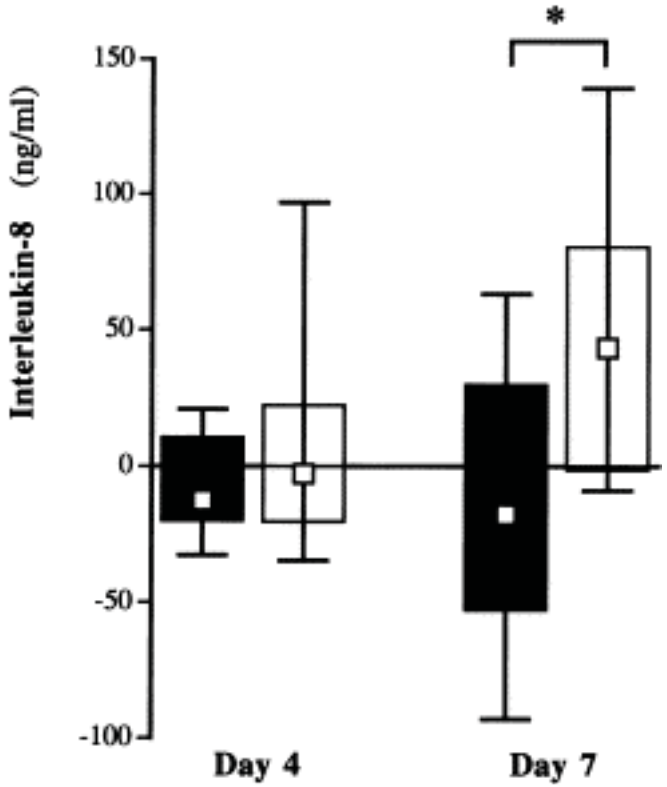
- Can't maintain adequate jejunal access
- Unable to meet caloric demands enterally for > 5 days

TPN + Glutamine in severe acute pancreatitis

double-blind study

Gln reduces the severity of acute-phase response

Gln supports lymphocyte proliferation



Glutamine-TPN in acute pancreatitis

- reduced acute-phase response and better lymphocyte proliferation

De Beaux, Nutrition 1998

- reduced length of TPN (10 vs 16 days, $p < 0.05$)
- reduced length of hospital stay (21 vs 25 days)

Ockenga et al, Clin Nutr 2002

Glutamine-TPN in acute pancreatitis : other RCTs

- less infections and reinterventions

Fuentes-Orozco et al, JPEN 2008

- less patients with complications

Sahin et al , Eur J Clin Nutr 2007

- lower incidence of complications, prevention of pancreatic infections

He et al , Clin Nutr Suppl 2004

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Nutrition in acute pancreatitis

In mild AP oral feeding can be started immediately if there is no nausea and vomiting and no abdominal pain (conditional recommendation, moderate quality of evidence)
In mild AP initiation of feeding with a low-fat solid diet appears as safe as clear liquid diet (conditional recommendation, moderate quality of evidence)

In severe AP enteral nutrition is recommended to prevent infectious complications .
Parenteral nutrition should be avoided unless the enteral route is not available, not tolerated or not meeting caloric requirement (strong recommendation, high quality of evidence)

Nasogastric and nasojejunal delivery of enteral feeding appear comparable (strong recommendation, moderate quality of evidence)

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ERCP in acute pancreatitis

Patients with acute pancreatitis and concurrent acute cholangitis should undergo ERCP within 24 h of admission (strong recommendation, moderate quality of evidence).

ERCP is not needed in most patients with gallstone pancreatitis who lack laboratory or clinical evidence of ongoing biliary obstruction (strong recommendation, low quality of evidence).

In the absence of cholangitis and / or jaundice, MRCP or endoscopic ultrasound (EUS) rather than diagnostic ERCP should be used to screen for choledocholithiasis if highly suspected (conditional recommendation, low quality of evidence).

Pancreatic duct stents and / or postprocedure rectal nonsteroidal anti-inflammatory drug (NSAID) suppositories should be utilized to prevent severe post-ERCP pancreatitis in high-risk patients (conditional recommendation, moderate quality of evidence).

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The role of surgery in acute pancreatitis - 1

In patients with mild AP, found to have gallstones in the gallbladder, a cholecystectomy should be performed before discharge to prevent a recurrence of AP (strong recommendation, moderate quality of evidence).

In a patient with necrotizing biliary AP, in order to prevent infection, cholecystectomy is to be deferred until active inflammation subsides and fluid collections resolve or stabilize (strong recommendation, moderate quality of evidence).

The presence of asymptomatic pseudocysts and pancreatic and / or extrapancreatic necrosis do not warrant intervention, regardless of size, location, and / or extension (strong recommendation, moderate quality of evidence).

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The role of surgery in acute pancreatitis - 2

In stable patients with infected necrosis, surgical, radiologic, and / or endoscopic drainage should be delayed preferably for more than 4 weeks to allow liquefication of the contents and the development of a fibrous wall around the necrosis (walled-off necrosis) (strong recommendation, low quality of evidence).

In symptomatic patients with infected necrosis, minimally invasive methods of necrosectomy are preferred to open necrosectomy (strong recommendation, low quality of evidence).

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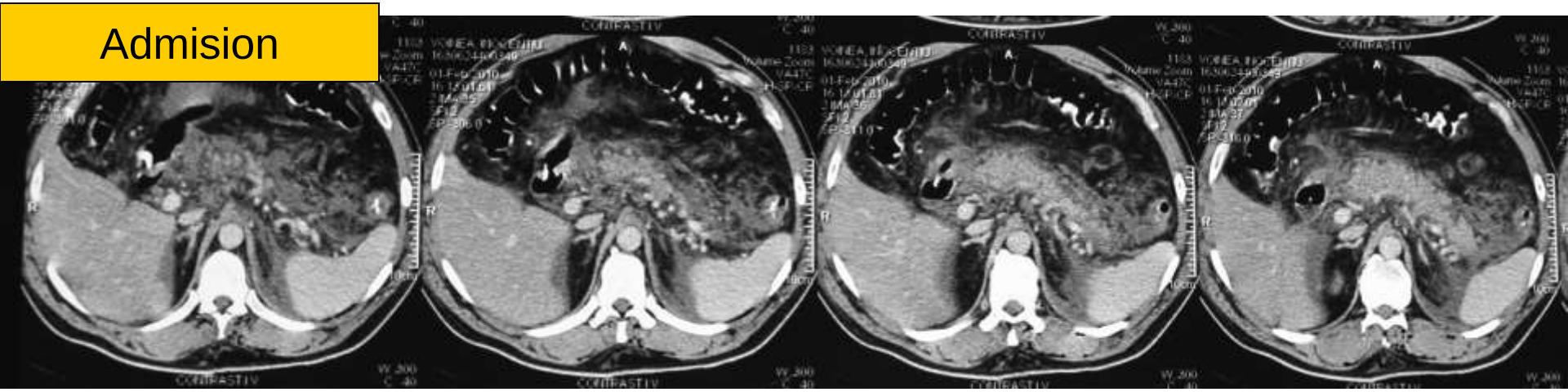
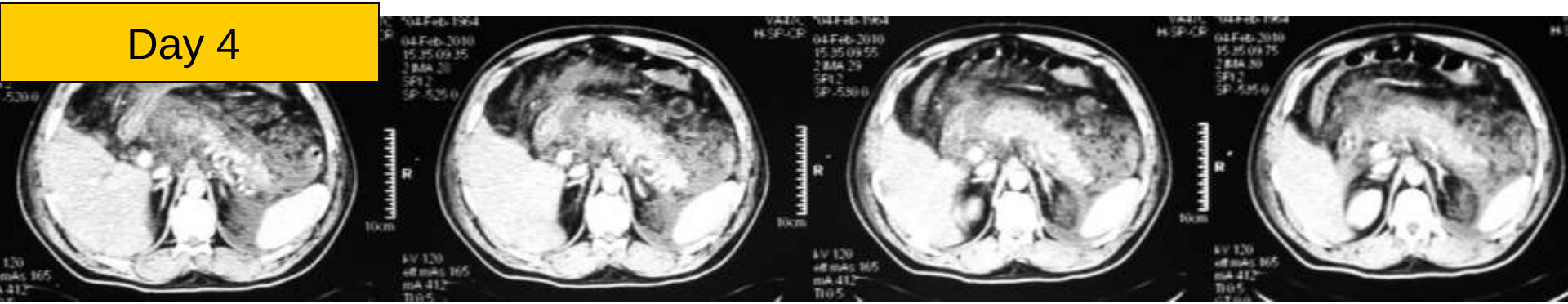
Conclusions

- Severe acute pancreatitis should be managed in ICU by a **multidisciplinary team** (surgeon, intensive care, gastroenterology, radiologist, nutritionist etc.)
- Infected necrosis carries a high mortality
- Antibiotics for suspected infected necrosis
- Tube feedings preferred, post ligament of Treiz
- Always look for the myriad of complications
- Guidelines are useful but not enough

Admision



Day 4



Day 24