Intra-Abdominal Catastrophe

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Disclosure

No financial interests in any biomedical or other healthcare-related entities

Overview

- Most Common Abdominal Catastrophes
  - Mesenteric Ischemia
  - Colitis
  - Bowel Perforation
  - Cholecystitis
  - Necrotizing Pancreatitis
  - Abdominal Compartment Syndrome

Pneumatosis Intestinalis

Abdominal Catastrophes in The ICU

- Often misdiagnosed or diagnosed late
  - Atypical presentation
  - Altered sensorium
  - Altered physiology
- Respiratory failure can often be the first sign
- Often require surgical treatment
- Increase mortality, length of stay, resource utilization, financial costs

Pneumatosis Intestinalis

- First described in 1754 by Du Vernoi
  - During dissection of cadavers
- Presence of extraluminal bowel gas within bowel wall
  - 0.03% in general population (autopsy series)
  - Incidence rising with increasing CT use
- Most asymptomatic, incidentally detected
- Breakdown of mucosal and immunological barrier of intestine, especially in the setting of increased intraluminal pressure

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Diseases associated with pneumatosis intestinalis

Intraabdominal catastrophe:
Intestinal ischemia
Intestinal infarction
Intestinal perforation
Intestinal obstruction
Necrotizing enterocolitis
Typhlitis
Mucosal disruption:
Peptic ulcer disease
Crohn's disease
Ulcerative colitis
Feeding jejunostomy tube
Caustic ingestions
Ruptured diverticulum
Infections:
Clostridium difficile
Tuberculosis
Whipples disease
AIDS entrocolitides
Cryptosporidium
Mycobacterium avium
Intracellulare
Cytomegalovirus (CMV)

Pulmonary disorders:
Chronic obstructive pulmonary disease
Asthma
Cystic fibrosis
Mechanical ventilation
Endoscopic procedures:
Esophagogastroduodenoscopy
Colonoscopy
Sclerotherapy
Endoscopic retrograde cholangiopancreatography (ERCP)
Diseases affecting gastrointestinal motility:
Diabetes
Scleroderma
Hirschprung's disease
Intestinal pseudoobstruction
Jejunoileal bypass
Pyloric stenosis/obstruction
Immunological disturbances:
AIDS
Steroids
Chemotherapy
Lymphoproliferative disorders
Bone marrow transplantation
Solid organ transplantation
Graft versus host disease
Amyloidosis
Collagen vascular disease

Pathogenesis

- Mechanical Theory
  - Gas dissects into the wall of the bowel from either the luminal surface through break in the mucosa or by tracking along mesenteric blood vessels.
    - Can be experimentally reproduced
    - Accounts for PI with condition that disrupt mucosal integrity (necrotizing enterocolitis, intestinal ischemia, caustic ingestion)

Pathogenesis

- Bacterial theory
  - Results from gas-forming bacteria gaining access to the submucosa through breaches in the mucosa
    - PI can be reproduced by Injecting *Clostridium perfringens* into the bowel wall
    - PI may resolve following treatment of antibiotics
    - Elemental diets have been reported to improve PI

Pathogenesis

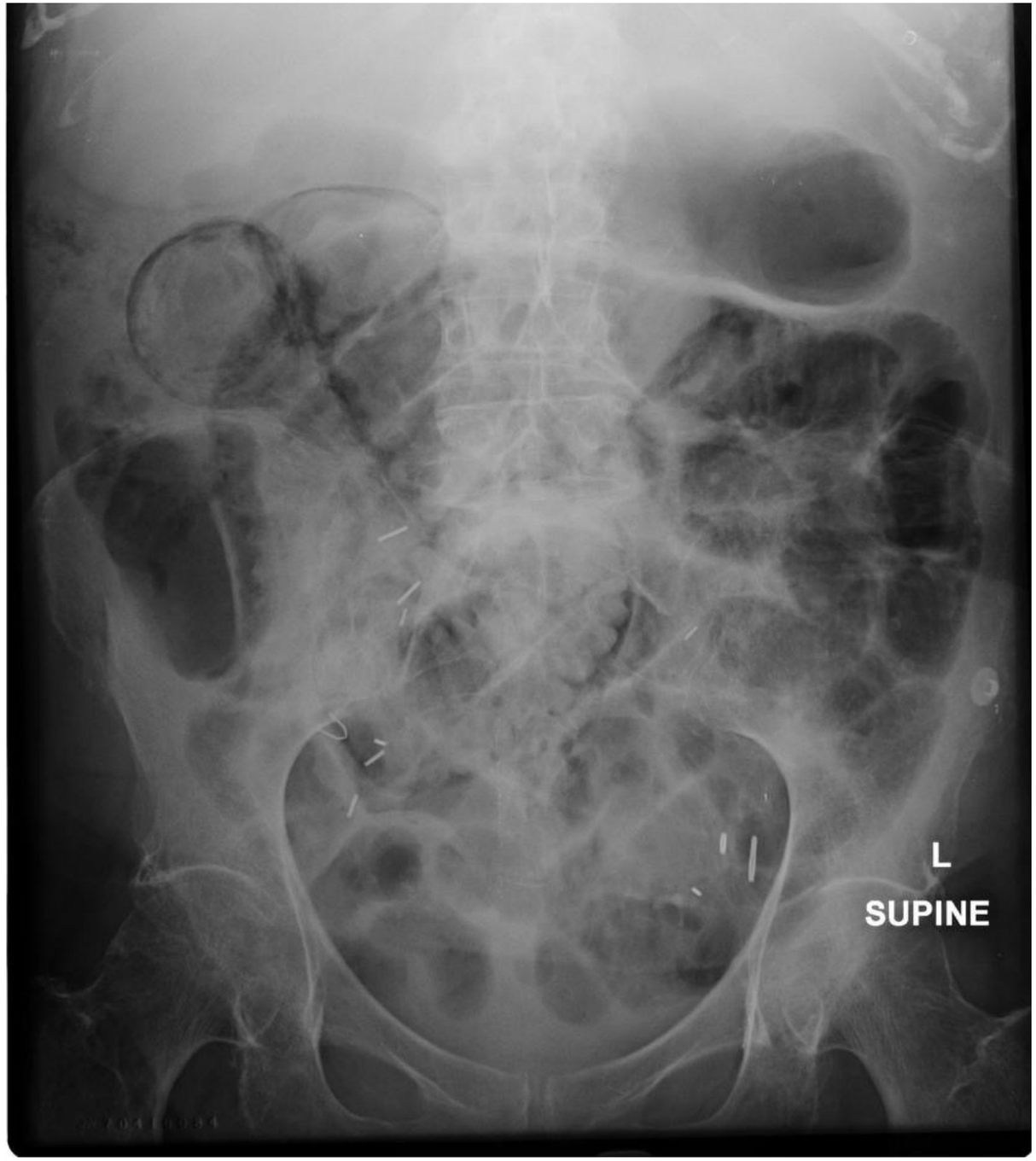
- Biochemical theory
  - Luminal bacteria produce excessive amount of hydrogen gas through fermentation of carbohydrates and other food.
    - As the pressure of the gas within the lumen increase, gas may be forced directly though the mucosa

Clinical Presentation

- Many are asymptomatic
- Symptoms will depend upon the region of intestine involved
  - Stomach
    - Nausea, vomiting
  - Small Intestine
    - Vomiting, abdominal distention, abdominal pain, diarrhea
  - Colon
    - Diarrhea, hematochezia, abdominal pain, distention

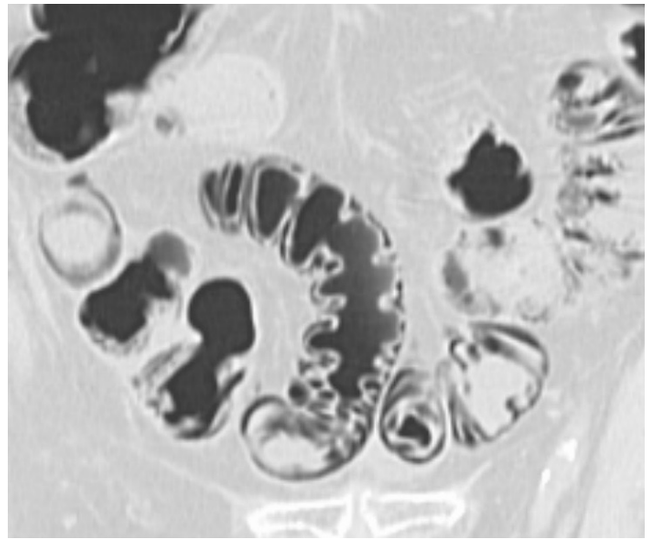
Imaging

- Abdominal Radiograph
  - Positive in ~ 2/3 of patients
  - Intramural gas can be linear, curvilinear or circular
  - Pneumoperitoneum detected in ~ 9%



Imaging

- CT SCAN
  - Circumferential collections of air adjacent to the lumen of the bowel that run in parallel with the wall of the bowel
  - Decreased mural contrast-enhancement
  - Portal venous gas (PVG)
  - Often suggestive of ischemia
  - ~30% of PI and PVG have a benign idiopathic cause



Management

- Based upon severity of symptoms
  - Asymptomatic and those with mild symptoms
    - Treat underlying cause of PI
    - Antibiotics
    - Bowel rest/Elemental diet
  - Severe Symptoms
    - Antibiotics
    - Bowel rest/TPN/Elemental diet
    - Inhalation oxygen therapy
    - Consider hyperbaric oxygen therapy if continued symptoms after 10 days
- Follow up imaging should be performed once symptoms resolve or until there is radiographic resolution

Surgical Management

Pneumoperitoneum may be managed medically if there are no clinical features suggesting underlying acute abdominal emergency

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Patients with PI and any *ONE* of the following should strongly consider emergent surgery

- Signs of peritonitis
- Metabolic acidosis (pH<7.3)
- Lactic acidosis (>2)
- PVG

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Remain symptomatic despite medical therapy or who develop complications from PI

The Fearsome Four

- Mesenteric Ischemia
- Colitis
- Cholecystitis
- Bowel Perforation

Mesenteric Ischemia

- Most Frequently missed diagnosis of the five
- Highest Mortality
- Can occur in the absence of arterial occlusive disease (non-occlusive mesenteric ischemia – NOMI)

Occlusive Disease

- In the ICU occlusive ischemia and infarction of the bowel is most frequently due to embolism in the setting of arterial fibrillation
- Can be sudden
  - “Pain out of proportion”
- Can be insidious
  - Non-specific abdominal complaints, constipation, melena, distension, feeding intolerance
- Can progress to fatal sepsis

NOMI

- Usually occurs as a consequence of a “low flow state”
  - Heart failure, abdominal compartment syndrome, sepsis of other cause
- Never Sudden
- Worsening hypotension, progressive organ failure

Common Features

- High Mortality (up to 88%)
- Circulating markers non-diagnostic (lactate, WBC, BE, CK)
- Require High Index of Suspicion
- Most effective treatment is surgery

Diagnosis

- Contrast CT and CTA
  - Greater than 90% sensitive and specific for occlusive MI
  - Nondiagnostic for NOMI
  - Excludes other diagnoses
- Bedside Laparoscopy
  - Cumbersome, not easily tolerated
- Peritoneal Lavage
  - Nonspecific, not as sensitive as CT

Treatment

- Occlusive disease almost always requires surgery
- NOMI survival depends on:
  - Correction of underlying illness
  - Resection of necrotic bowel
- To date there is no reliable non-invasive test to exclude bowel necrosis

Colitis

- *C.diff Colitis*
  - Exudative inflammatory colitis resulting from *Clostridium Difficile*
  - Spectrum of Presentation

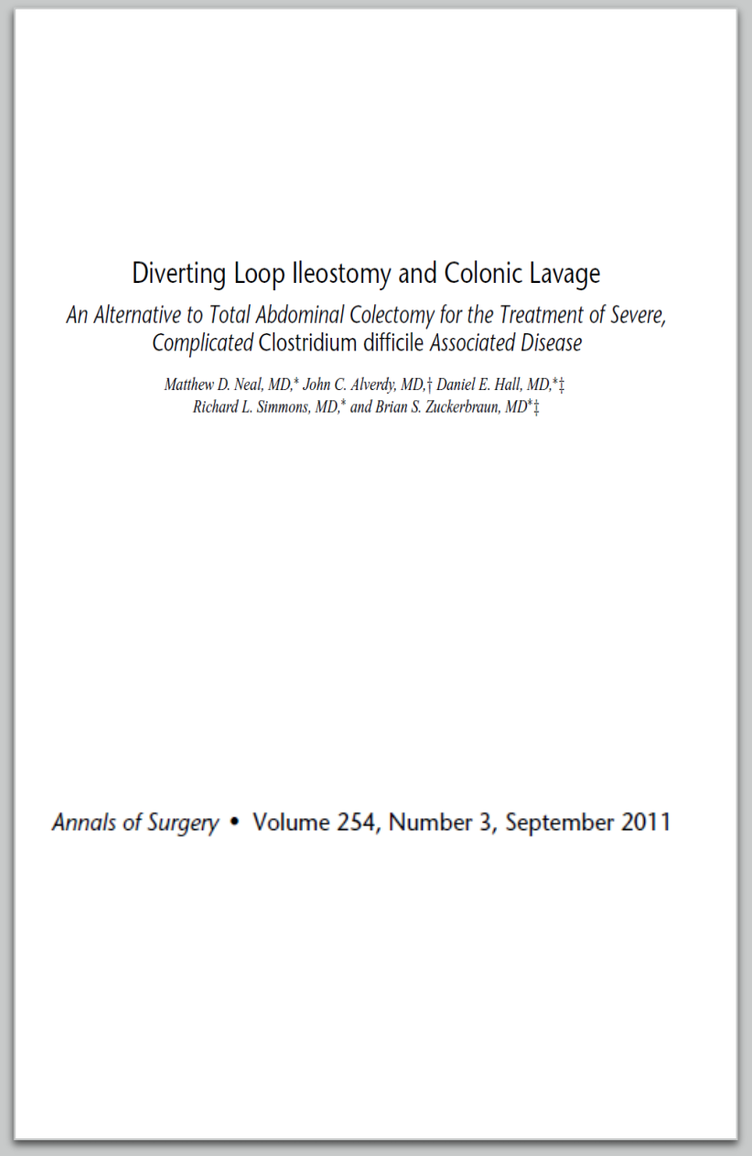
- Fulminant Pseudomembranous Colitis
  - *C.diff* colitis with significant systemic toxicity and shock
  - Only 3-5% of all C.diff colitis
  - Mortality > 30%

Diagnosis

- Nonspecific abdominal pain and tenderness
- Diarrhea absent in up to 20% of patients
- Leukocytosis
- CT sensitive but not specific
- Stool toxin assay
  - 70% sensitive
- Stool culture
  - Very sensitive, time consuming
- Colonoscopy

Treatment

- Medical therapy for non-fulminant
  - Oral Vancomycin, oral Metronidazole, Vancomycin Enemas
  - Fidoxamicin a relatively new bactericidal antibiotic with promising results
  - Intravenous Metronidazole
- Total abdominal colectomy for fulminant
  - Timing is crucial
  - Mortality increase with delay



Diverting Loop Ileostomy and Colonic Lavage

An Alternative to Total Abdominal Colectomy for the Treatment of Severe, Complicated Clostridium difficile *Associated Disease*

Matthew D. Neal, MD, John C. Alverdy, MD,† Daniel E. Hall, MD,*‡ Richard L. Simmons, MD,* and Brian S. Zuckerbraun, MD*‡*

Annals of Surgery • Volume 254, Number 3, September 2011

Diversion and Lavage

- Small series
- Diagnostic laparoscopy to rule out perforation and construct loop ileostomy
- Lavage colon with PEG
- Vancomycin per stoma, metronidazole IV for 10 days
- Colonic Preservation was achieved in 39 of 42 patients
- Reduced mortality as compared to historical data

- **TO DATE STANDARD OF CARE REMAINS TOTAL ABDOMINAL COLECTOMY**

Acalculous Cholecystitis

- 1 in 100 patients, depending on ICU population
- Mortality 20-50%
- Results from biliary stasis and chronic distension or poor perfusion
  - Sepsis, cardiogenic shock, hemorrhagic shock
  - Associated with already established organ failure
- Gallbladder wall ischemia
- Necrosis, gangrene present in 44% at diagnosis

Diagnosis

- Few reliable signs or symptoms
  - >90% of patients are already on ventilator
- Fever, leukocytosis, elevation in transaminases not reliable
- Rising bilirubin without ductal dilation in 60% of cases
- Feeding Intolerance, worsening organ failure without other causes

Diagnosis

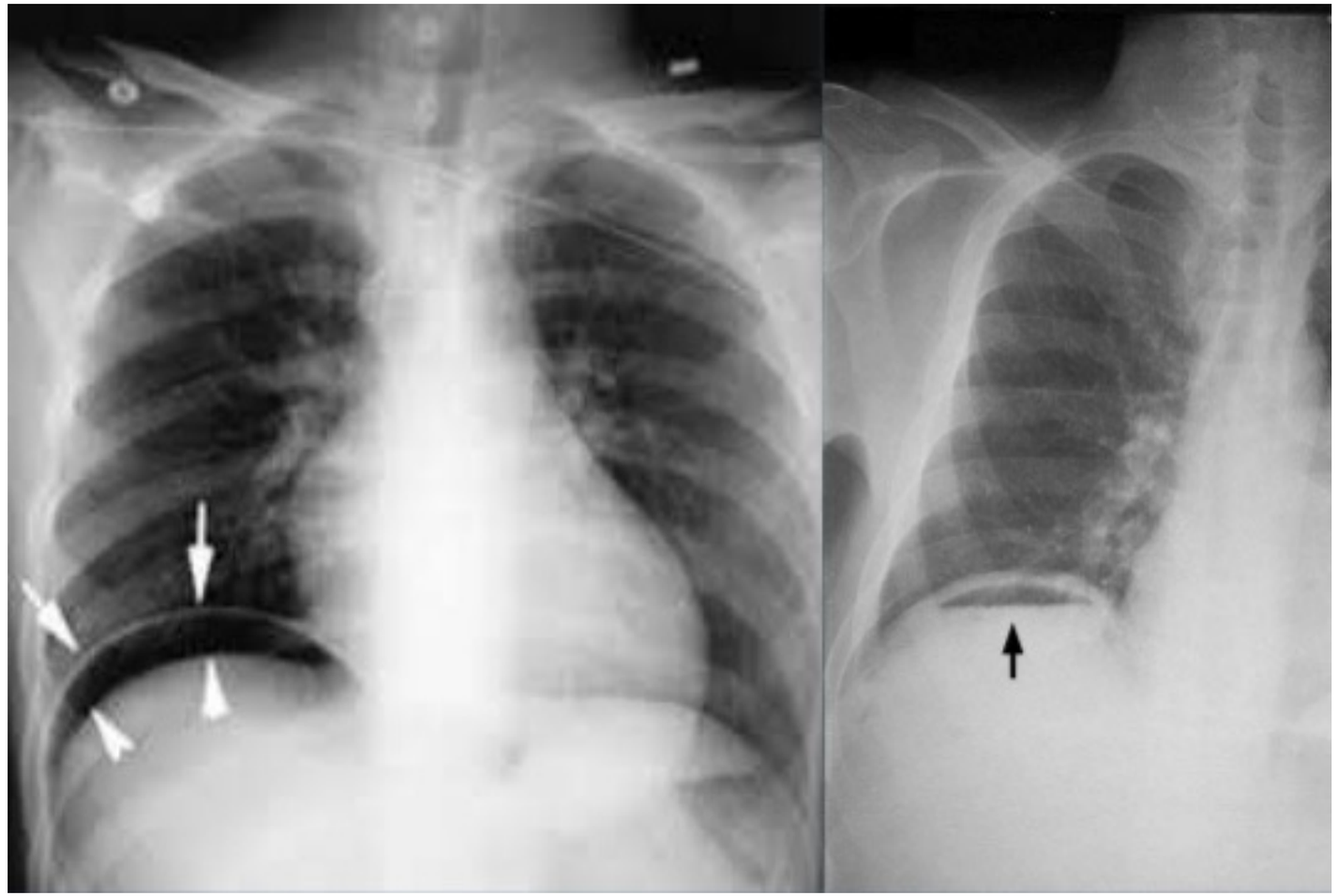
- Ultrasound
  - Gallbladder distension, wall thickening
  - Sludge, rarely calculi
  - Less than 90% sensitivity in most series
- Scintigraphy
  - Often nondiagnostic due to fasting or elevated bilirubin
- CT with contrast
  - Facilitates diagnosis of infarction and gangrene
  - May reveal or exclude other diagnoses (e.g. mesenteric ischemia)
  - Thickened wall, pericholecystic fluid, sludge, air in gallbladder wall

Treatment

- Cholecystectomy
  - Usually open procedure
  - Higher risk of bleeding and biliary injury

- Percutaneous cholecystostomy
  - Less invasive, less complicated
  - Mortality still high
  - Does not treat gangrene
  - Reasonable option for complicated patients, if detected early

Bowel Perforation



Bowel Perforation

- Can occur as a further complication of ischemia, obstruction, or colitis
- Iatrogenic or traumatic
- Does not always cause typical peritonitis, can be insidious
- High mortality
- May present as incidental finding of pneumoperitoneum

Diagnosis

- Diffuse peritonitis (mild or severe)
- New ascites
- Abdominal distension
- Constipation
- Feeding intolerance

Imaging

- Plain radiography is insufficient to exclude perforation
  - Benign pneumoperitoneum

- CT with intravenous contrast
  - High sensitivity
  - Oral contrast not necessary

Treatment

- Almost always surgical
  - Laparotomy, resection, diversion
- Rare cases can be managed without laparotomy
  - Selective endoscopic perforation
  - Contained perforated diverticulitis
- Delay in surgical treatment increases risk of mortality

Necrotizing Pancreatitis

Necrotizing Pancreatitis

- ~275K admissions per year in the US for acute pancreatitis, 5-10% of which are for necrotizing pancreatitis

- Mortality rate:
  - Sterile necrosis: 10-15%
  - Infected necrosis: 20-30%
  - Multiorgan failure: 40-50%

- Infected pancreatic necrosis = 30-45% of all pancreatic necrosis

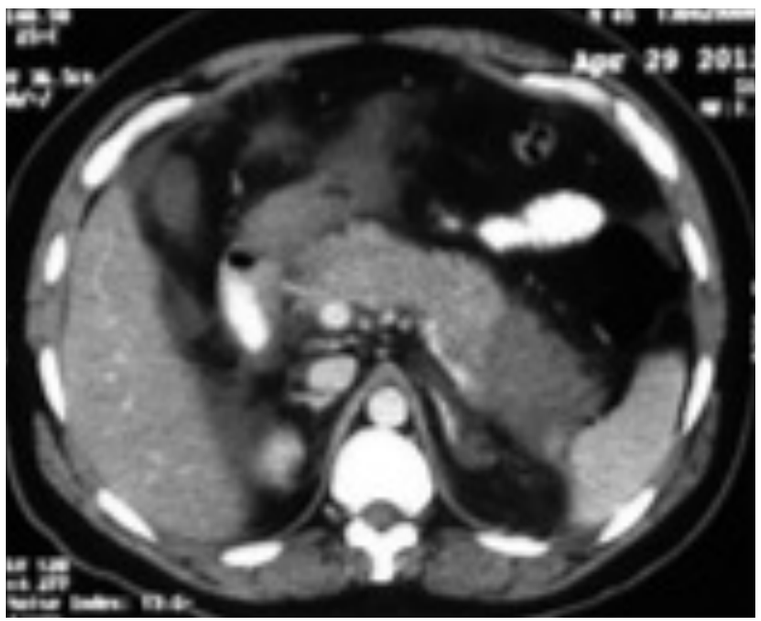
2012 Atlanta Classification

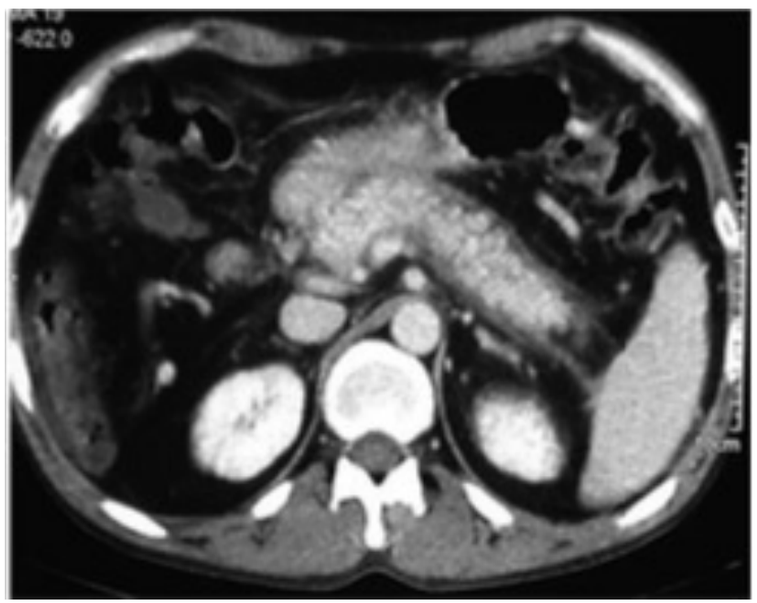
- ***Necrotizing Pancreatitis***
  - Lack of pancreatic parenchymal enhancement by intravenous contrast agent OR
  - Presence of findings of peripancreatic necrosis (acute necrotic collection or walled off necrosis)

- ***Acute Necrotic Collection (ANC)***
  - Necrotic collection without a well defined inflammatory wall
  - Typically less than 4 weeks from onset of necrotizing pancreatitis

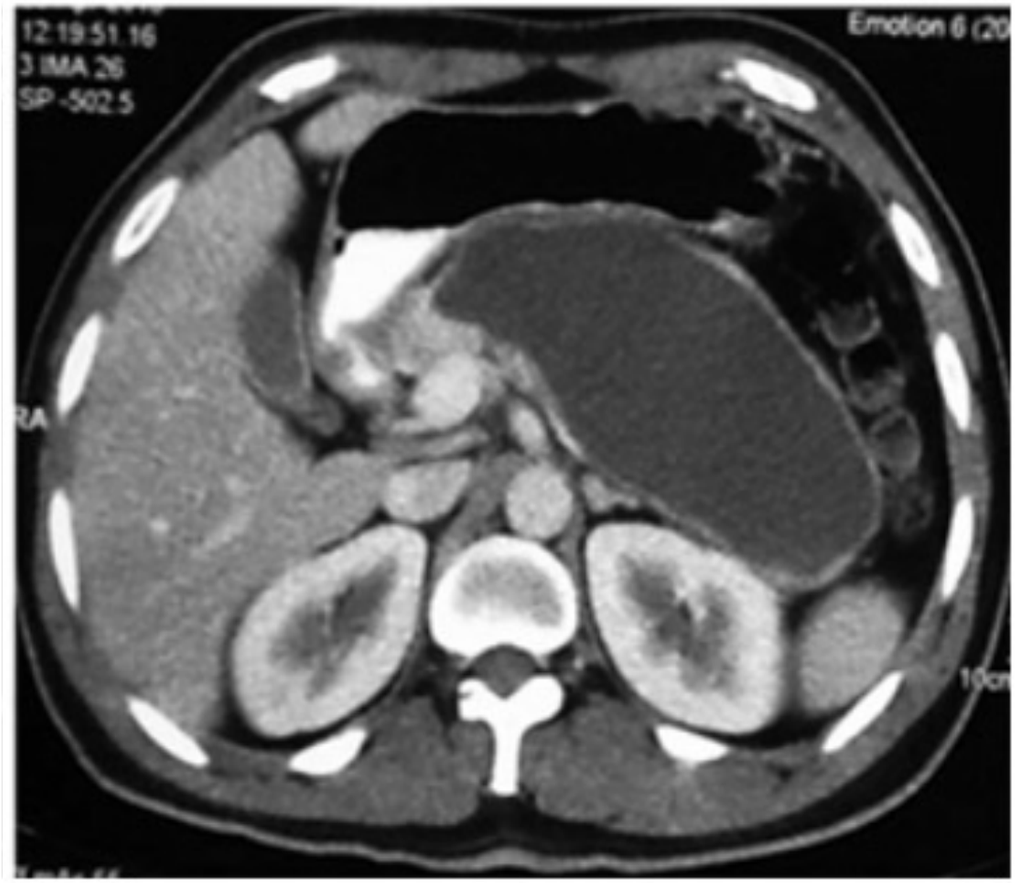
- ***Walled Off Necrosis (WON)***
  - An encapsulated collection of pancreatic or peri-pancreatic necrosis that has developed a well defined inflammatory wall.
  - Typically more than 4 weeks since onset of necrotizing pancreatitis

Diagnosis: Contrast Enhanced CT





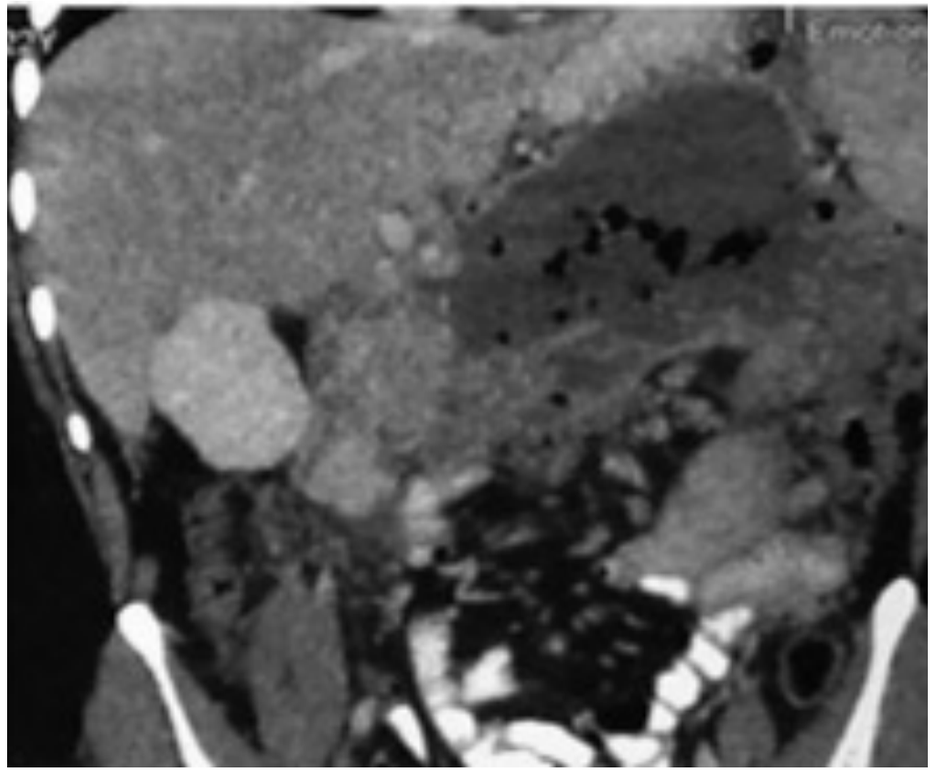
Acute Necrotic Collection vs. Walled Off Necrosis

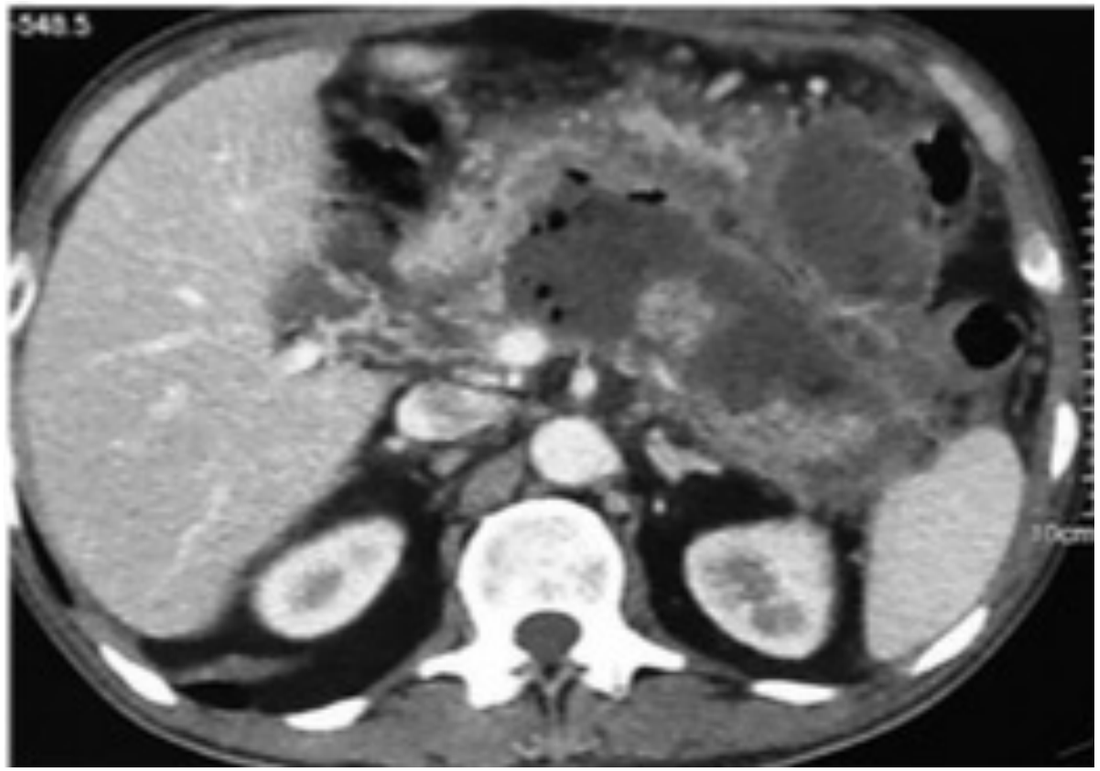


Infected Pancreatic Necrosis

- Median onset = 26 days after diagnosis of necrotizing pancreatitis
- Affects 30-45% of all necrotizing pancreatitis patients
- Thought to be the result of increased intestinal permeability and decreased immunity that results from florid inflammatory response to necrotizing pancreatitis
- Bacteremia is a risk factor: 65% incidence of infected necrosis vs. 37.9% in those without bacteremia
  - ~40% match between organism isolated in blood culture and pancreas culture
- Direct correlation between extent of necrosis and risk of infected necrosis
  - Risk particularly elevates once necrosis >30% of pancreas

Infected Pancreatic Necrosis: CT findings





Diagnosis of Infected Necrotizing Pancreatitis

- 2013 American College of Gastroenterology guidelines: maintain high index of suspicion in patients who clinically decline or fail to improve after 7-10 days of hospitalization.

- Confirm diagnosis by one of the following:
  - Positive culture of pancreatic or peripancreatic necrosis obtained by FNA
  - Presence of air in the collections on CT

- FNA can be inaccurate in detecting infection:
  - Sensitivity: 88%
  - Specificity: 90%

Effect of antibiotic prophylaxis on acute necrotizing pancreatitis: Results of a randomized controlled trial, J Gastro Hepatology, 2009

- Randomized control trial of 56 patients with necrotizing pancreatitis who were randomized to one of two groups:
  - A: imipenem-cilastatin FOR 7-14 days (within 3 days of onset of symptoms)
  - B: no antibiotics

	Study group ($n = 29$)	Control group ($n = 27$)
Mortality, *n* (%)*	3 (10.3)	4 (14.8)
Organ complication, *n* (%)*		
ARDS	10 (34.5)	9 (33.3)
ARF	4 (13.8)	3 (11.1)
Hepatic insufficiency	13 (44.8)	10 (37.0)
Shock	1 (3.6)	1 (3.8)
Pancreatic pseudocyst	6 (20.7)	5 (18.5)
Use of respirator, *n* (%)*	9 (32.1)	8 (30.8)
Operative necrosectomy, *n* (%)*	8 (29.6)	9 (34.6)
Duration of CVC, days (median, range)†	23 (14–27)	21 (15–29)
Hospital stay, days (median, range)†	28.3 (23–71)	30.7 (25–60)

Differences between the study group and the control group regarding mortality, the incidence of organ complication, usage rate of respirator, the incidence of operative necrosectomy, duration of CVC and hospital stay were not statistically significant ($P > 0.05$, *χ^2-test, †unpaired Student's *t*-test). ARDS, acute respiratory distress syndrome; ARF, acute renal failure; CVC, central venous catheterization.

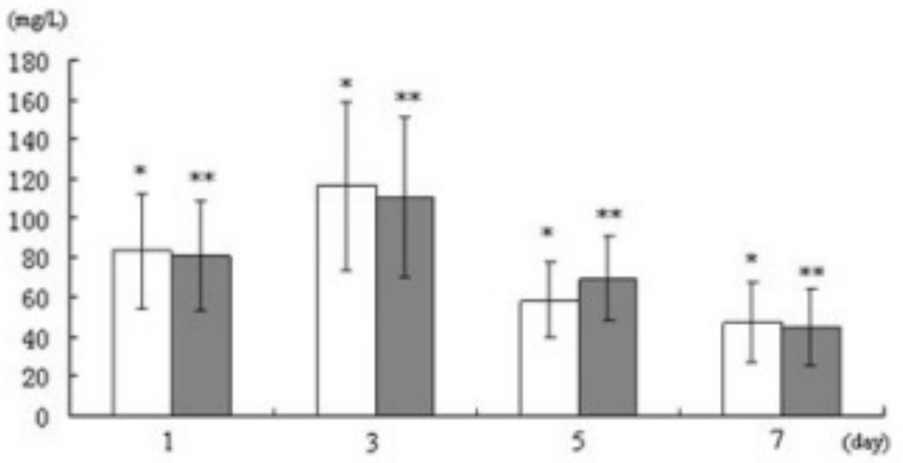
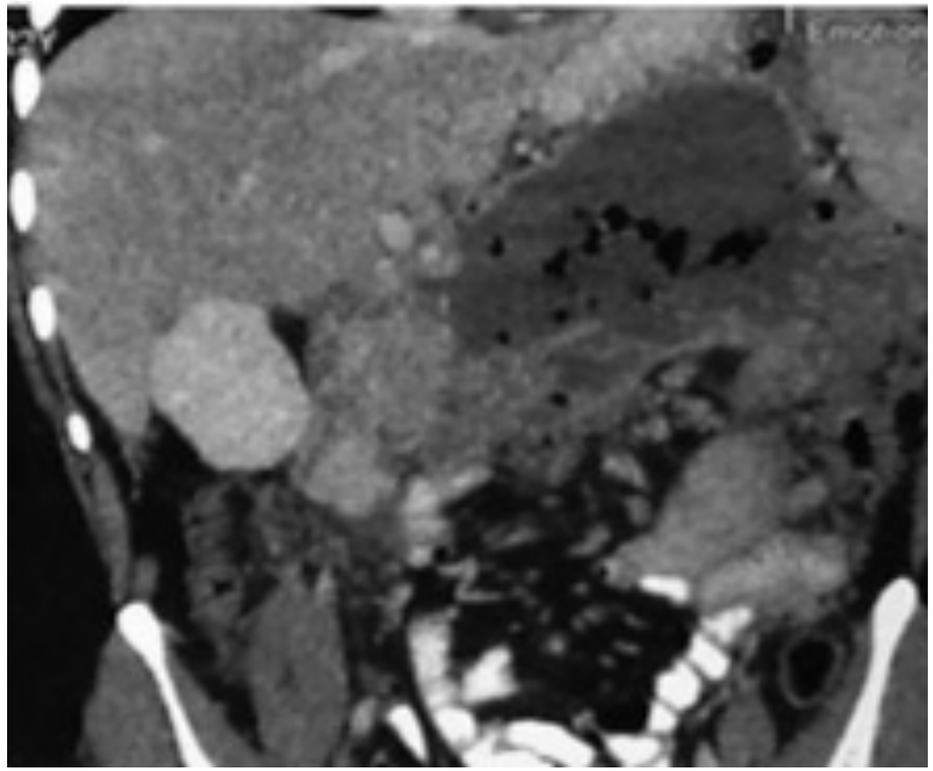
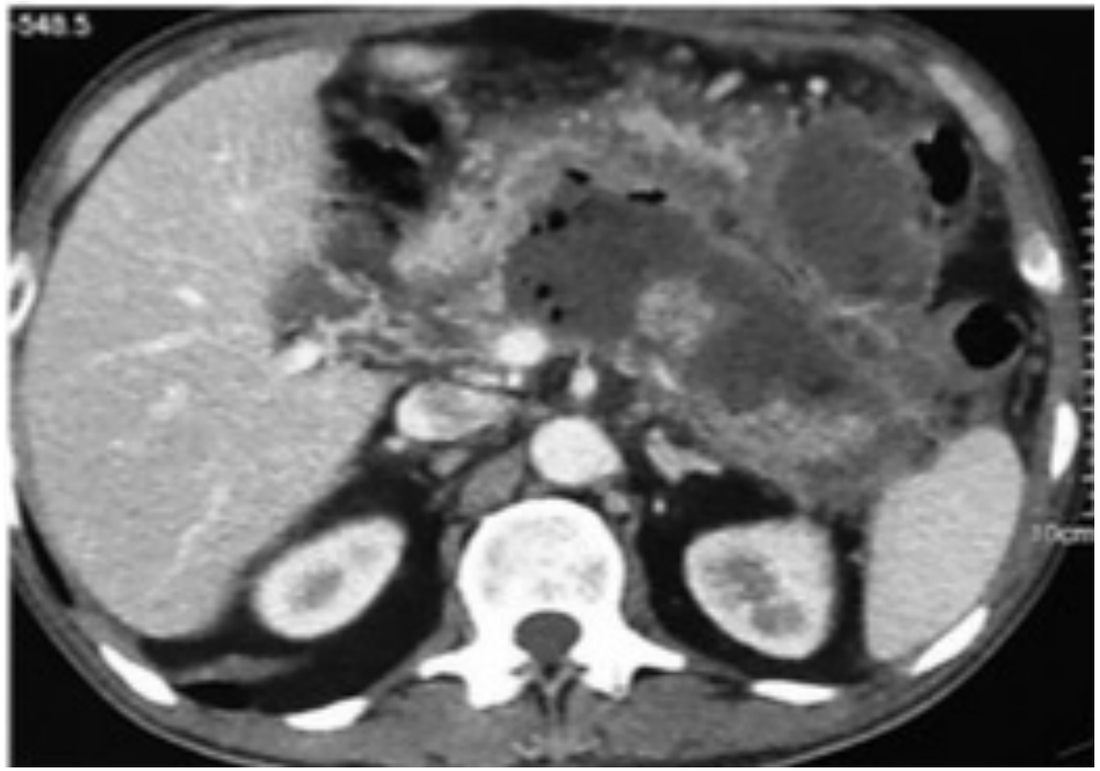


Figure 2 Comparison of the level of serum C-reactive protein (CRP) between the study group and the control group. *Control group; **study group. Difference in the level of serum CRP at each time point between the study group and the control group was not statistically significant ($P > 0.05$, unpaired Student's *t*-test).

Infected Pancreatic Necrosis: Management





Timing of Surgical Intervention in Necrotizing Pancreatitis, Arch Surg, 2007

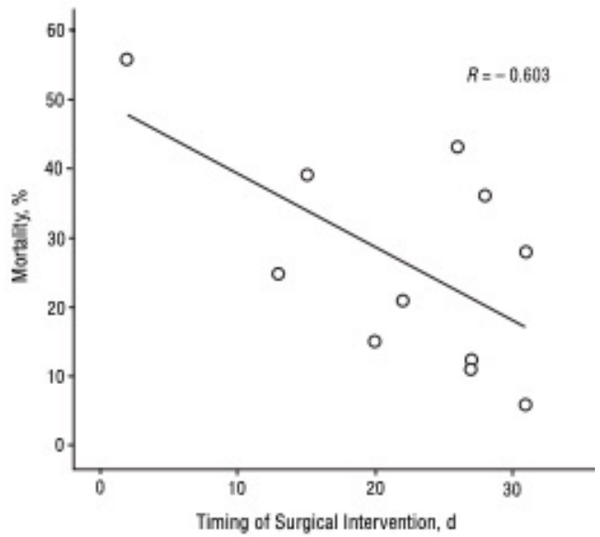
- Retrospective analysis of 53 patients with necrotizing pancreatitis at a single center
- Delayed surgical intervention correlated with decreased mortality for patients with necrotizing pancreatitis

Hospital Mortality	Time From Initial Admission to Operation, No. (%)			*P* Value
	Day 1-14 (n=16)	Day 15-29 (n=11)	Day ≥30 (n=26)	
Patients without organ failure	3/5 (60)	0/4 (0)	2/15 (13)	.045
Patients with organ failure	9/11 (82)	5/7 (71)	0/11 (0)	< .001
Patients with multiple organ failure	6/6 (100)	4/5 (80)	0/6 (0)	.001
Total	12/16 (75)	5/11 (45)	2/26 (8)	< .001

Timing of Surgical Intervention in Necrotizing Pancreatitis, Arch Surg, 2007

- Also completed meta-analysis of 11 total studies which also revealed negative correlation between time to surgical intervention and mortality

Source	Design	No. of Patients	No. of Patients Operated On/y	Patients With Infection, %	Median Time From Initial Admission to Operation, d	Mortality, %
Mier,[2] 1997	Randomized controlled	25	8.3	60	2	56
Fernandez-del Castillo,[10] 1998	Retrospective	64	9.1	56	31	6
Branum,[11] 1998	Retrospective	50	8.3	84	27	12
Farkas,[12] 1998	Retrospective	203	11.3	100	20	15
Büchler,[13] 2000	Prospective	28	5.6	96	22	21
Ashley,[14] 2001	Retrospective	36	7.2	92	27	11
Beattie,[15] 2002	Retrospective	54	6.8	68	26	43
Götzinger,[16] 2003	Prospective	250	15.6	74	15	39
Connor,[17] 2005	Prospective	88	14.7	77	31	28
Rau,[18] 2005	Retrospective/prospective	285	15	49	13	25
Present study	Retrospective	53	5.3	83	28	36
Total, median		54	8.3	77	26	25



Pancreatitis Necrosectomy vs. sTEp up approach (PANTER, 2005-2008)

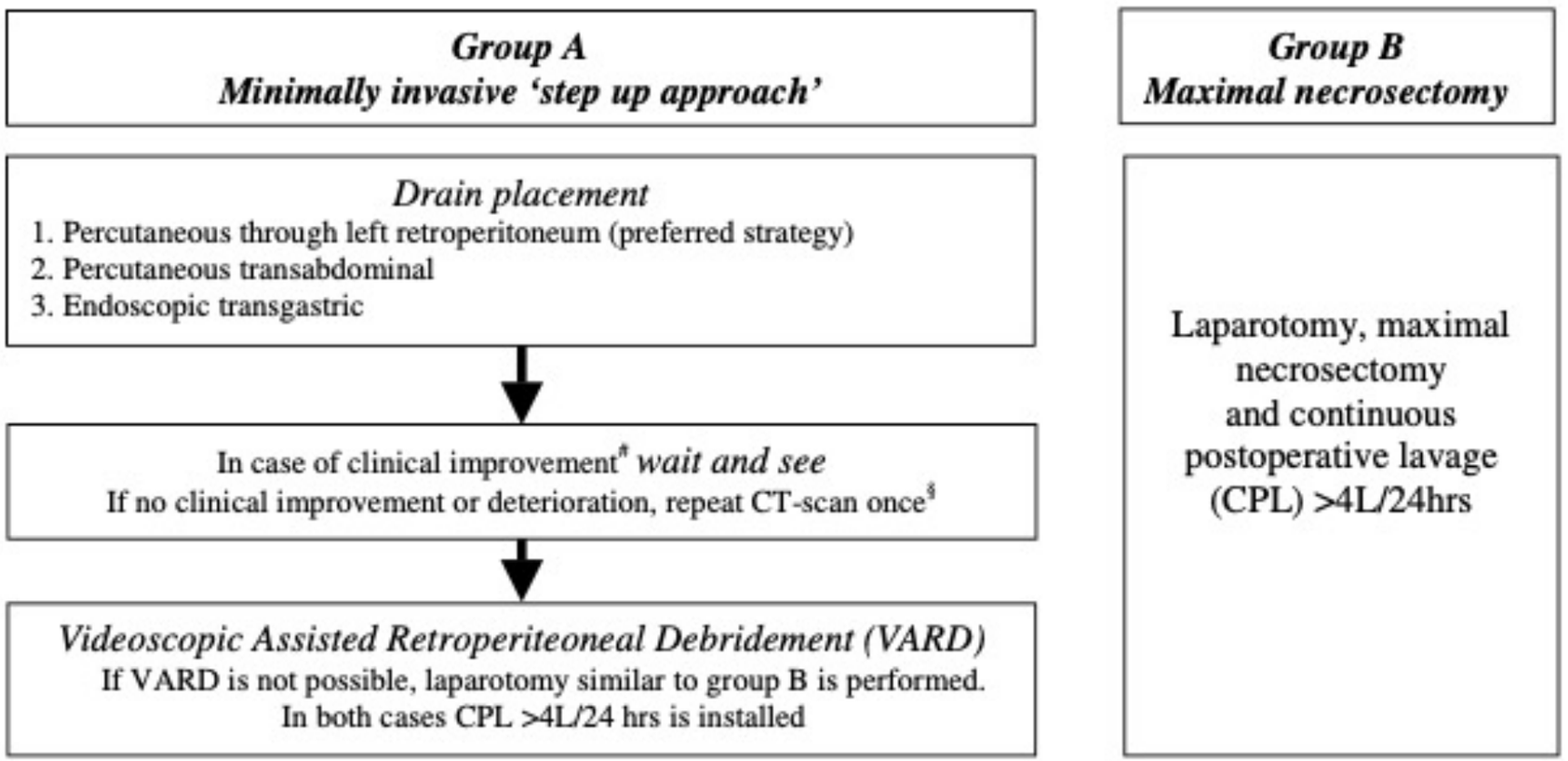
- 88 patients with suspected/confirmed infected necrotizing pancreatitis randomized to one of two groups:
  - Group A: Laparotomy + necrosectomy + continuous postoperative lavage
  - Group B: Step-Up Approach
    - Drainage of necrotic collection followed by video-assisted retroperitoneal debridement (if needed)

- Primary endpoint: proportion of patients with major morbidity or mortality

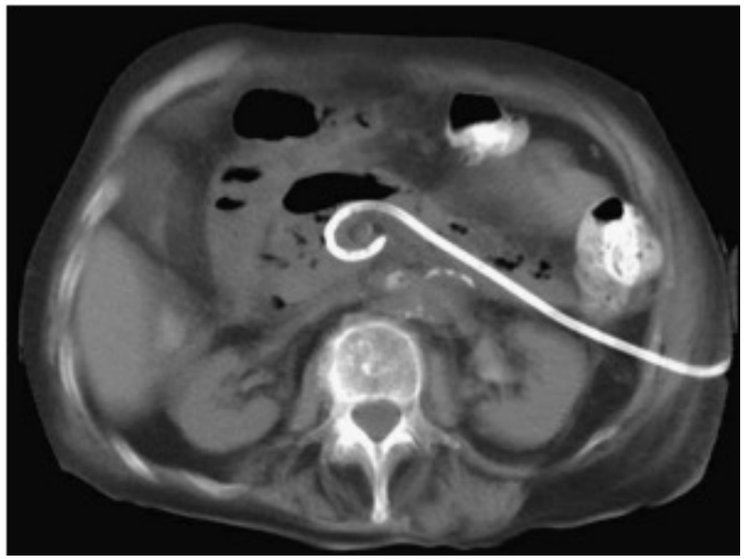
- Secondary endpoint: minor complications (fistula, abscess, strictures, pseudocyst)

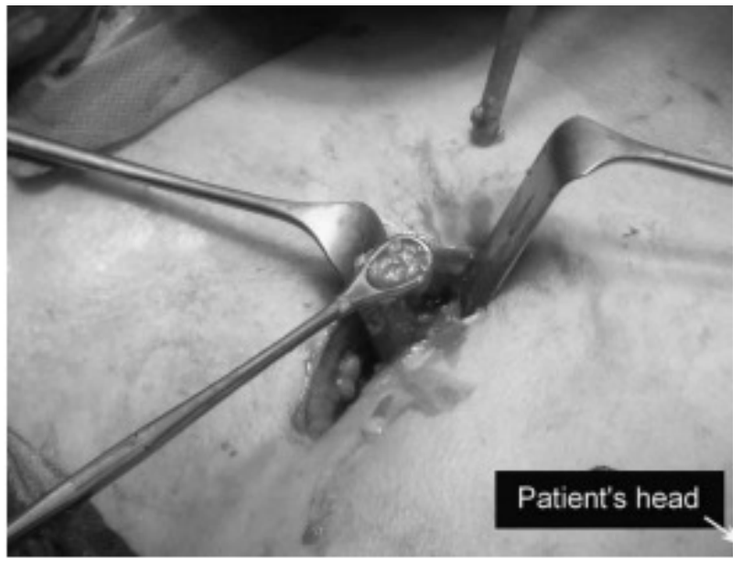
- Multicenter study- 20 hospitals part of Dutch Acute Pancreatitis Study Group

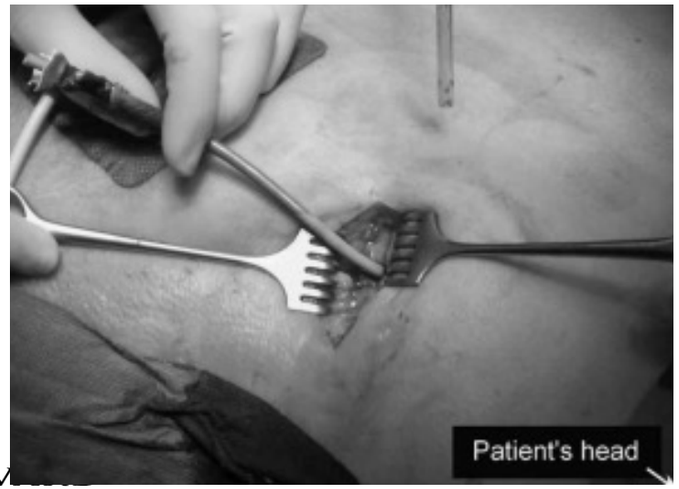
PANTER: Study Design

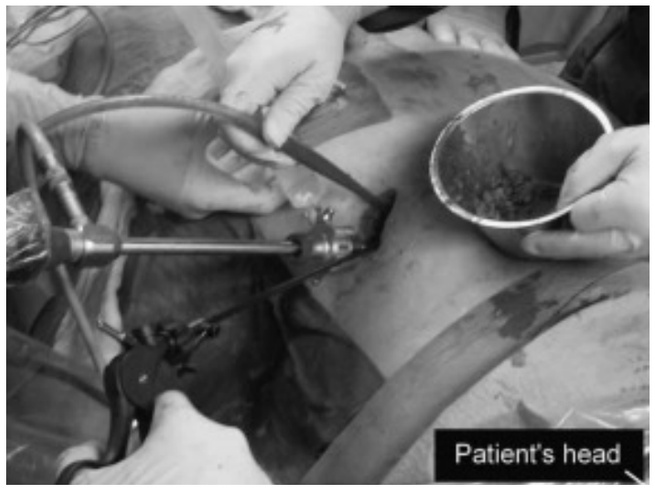


Video Assisted Retroperitoneal Debridement









PANTER Results (NEJM, 2010)

Table 3. Primary and Secondary End Points.*

Outcome	Minimally Invasive Step-up Approach (N=43)	Primary Open Necrosectomy (N=45)	Risk Ratio (95% CI)	P Value
Primary composite end point: major complications or death — no. (%)†	17 (40)	31 (69)	0.57 (0.38–0.87)	0.006
Secondary end points				
Major complication — no. (%)				
New-onset multiple-organ failure or systemic complications‡	5 (12)	19 (42)	0.28 (0.11–0.67)	0.001
Multiple-organ failure	5 (12)	18 (40)		
Multiple systemic complications	0	1 (2)		
Intraabdominal bleeding requiring intervention	7 (16)	10 (22)	0.73 (0.31–1.75)	0.48
Enterocutaneous fistula or perforation of a visceral organ requiring intervention	6 (14)	10 (22)	0.63 (0.25–1.58)	0.32
Death — no. (%)	8 (19)	7 (16)	1.20 (0.48–3.01)	0.70
Other outcome — no. (%)				
Pancreatic fistula	12 (28)	17 (38)	0.74 (0.40–1.36)	0.33
Incisional hernia§	3 (7)	11 (24)	0.29 (0.09–0.95)	0.03
New-onset diabetes§	7 (16)	17 (38)	0.43 (0.20–0.94)	0.02
Use of pancreatic enzymes§	3 (7)	15 (33)	0.21 (0.07–0.67)	0.002
Health care resource utilization				
Necrosectomies (laparotomy or VARD) — no. (%)				<0.001
0	17 (40)	0		
1	19 (44)	31 (69)		
2	6 (14)	8 (18)		
≥3	1 (2)	6 (13)		
Total no. of operations¶				0.004
Per study group	53	91		
Range per patient	0–6	1–7		
Total no. of drainage procedures‖				<0.001
Per study group	82	32		
Range per patient	1–7	0–6		
New ICU admission at any time after first intervention — no. (%)**	7 (16)	18 (40)	0.41 (0.19–0.88)	0.01
Days in ICU				0.26
Median	9	11		
Range	0–281	0–111		
Days in hospital				0.53
Median	50	60		
Range	1–287	1–247		

Minimal Access Retroperitoneal Pancreatic Necrosectomy Improvement in Morbidity and Mortality With a Less Invasive Approach, Annals Surgery, 2010.

- Retrospective analysis of outcomes of 189 patients with infected pancreatic necrosis or non-resolving pancreatic necrosis undergoing VARD vs. open necrosectomy.

TABLE 3. Preoperative Data

	MARPN n = 137	Open n = 52	*P*
Culture-proven pancreatic infection at first operation	74/116 (64%)	33/46 (72%)	0.336
Fungal infection	14/121 (12%)	4/45 (9%)	0.337
Prophylactic antibiotics	75 (55%)	36 (69%)	0.101
Median (range) maximum CT score	9 (4–10)	9 (3–10)	0.199
>50% necrosis	79 (58%)	31 (60%)	0.938
ICU preoperatively	53 (39%)	22 (42%)	0.773
Median (range) initial APACHE II score	10 (2–31)	9 (1–17)	0.341
Median (range) preoperative APACHE II score	8 (1–29)	10 (2–20)	0.038
Preoperative MOF present	36 (26%)	20 (38%)	0.144
Median (range) initial CRP, mg/L	268 (31–577)	234 (17–508)	0.152
Median (range) preoperative CRP, mg/L	162 (5–490)	216 (5–526)	0.682
Median (range) time to surgery, d	32 (1–181)	34 (1–95)	0.745

TABLE 5. Complications

	MARPN n = 137	Open n = 52	*P*
Myocardial infarct	4 (2.9%)	5 (9.6%)	0.122
Cerebrovascular event	2 (1.5%)	2 (3.8%)	0.652
Pseudocyst	3 (2.2%)	5 (9.6%)	0.063
Bleeding	16 (11.7%)	9 (17.3%)	0.436
Biliary stricture	3 (2.2%)	5 (9.6%)	0.253
Pancreatic fistula	5 (3.6%)	4 (7.7%)	0.434
Enteric fistula	10 (7.3%)	5 (9.6%)	0.865
Pulmonary embolus	1 (0.7%)	1 (1.9%)	0.621
Colonic necrosis	4 (2.9%)	3 (5.8%)	0.621
Hepatic portal/superior mesenteric/splenic vein thrombosis	7 (5.1%)	4 (7.7%)	0.741
Clostridium difficile infection	4 (2.9%)	1 (1.9%)	0.893
Miscellaneous	10 (7.3%)	8 (15.4%)	0.313
Total	69 in 75 patients (55% of patients)	52 in 43 patients (83% of patients)	

Minimal Access Retroperitoneal Pancreatic Necrosectomy Improvement in Morbidity and Mortality With a Less Invasive Approach, Annals Surgery, 2010.

- Decrease in mortality, complications, organ failure and ICU requirement in VARD vs. open necrosectomy.
- Undergoing VARD = independent predictor of reduced mortality

TABLE 4. Postoperative Findings

	MARPN n = 137	Open n = 52	*P*
Postoperative MOF present	34 (31%)	39 (56%)	<0.0001
Median (range) postoperative APACHE II score	8 (1–22)	11 (2–24)	0.0006
Median (range) postoperative CRP, mg/L	173 (8–443)	216 (33–378)	0.142
Number of patients with one or more complications	75 (54.7%)	42 (80.8%)	0.001
Mortality	26 (19%)	20 (38%)	0.009
Median (range) time to death, d	77 (16–252)	47.5 (13–222)	0.099
Median (range) time to death postsurgery, d	51 (5–220)	16 (1–164)	0.091
Median (range) time to discharge, d	96 (29–300)	89 (8–180)	0.139
Median (range) time to discharge postsurgery, d	64 (15–272)	43 (5–158)	0.004
Median (range) length of stay—all patients, d	94.5 (16–300)	85 (8–222)	0.011
Median (range) stay postsurgery—all patients, d	60 (5–272)	36 (1–164)	0.0002
Number of patients needing ICU postoperatively	59 (43%)	40 (77%)	<0.0001
Median (range) ICU stay for those needing ICU only	15 (1–118)	10 (1–82)	0.299
Median (range) no. procedures	3 (1–9)	1 (1–9)	<0.0001

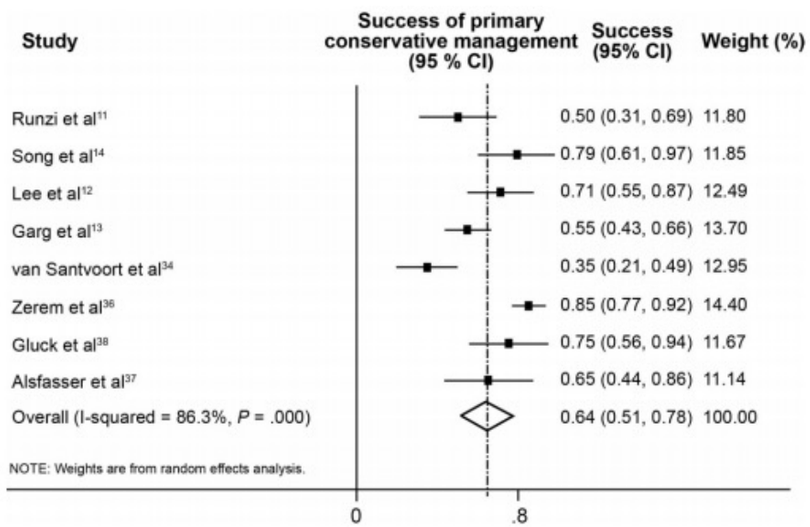
TABLE 1. Large Published Series of Open Pancreatic Necrosectomy

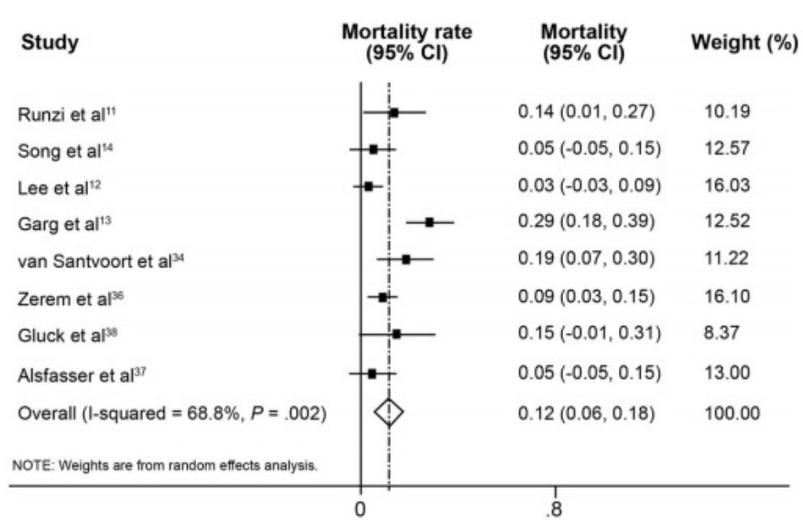
Author	Year	No. Patients	No. Deaths	Mortality (%)
Borie et al[38]	1994	157	28	17.8
Armbruster et al[39]	1998	108	30	27.8
Bradley[40]	1999	176	23	13.1
Kriwanek et al[41]	1999	100	19	19.0
Gotzinger et al[19]	2002	340	133	39.1
Rau et al[27]	2005	285	71	24.9
Farkas et al[42–45]	2006	281	39	13.9
Olakowski et al[45]	2006	126	26	20.6
Reddy et al[46]	2006	118	45	38.1
Howard et al[47]	2007	102	12	11.8
Rodriguez et al[48]	2008	167	19	11.4

TABLE 7. Multivariable Logistic Regression: Independent Risk Factors for Death (With Forced Entry of Variables Imbalanced Between Groups at Baseline)

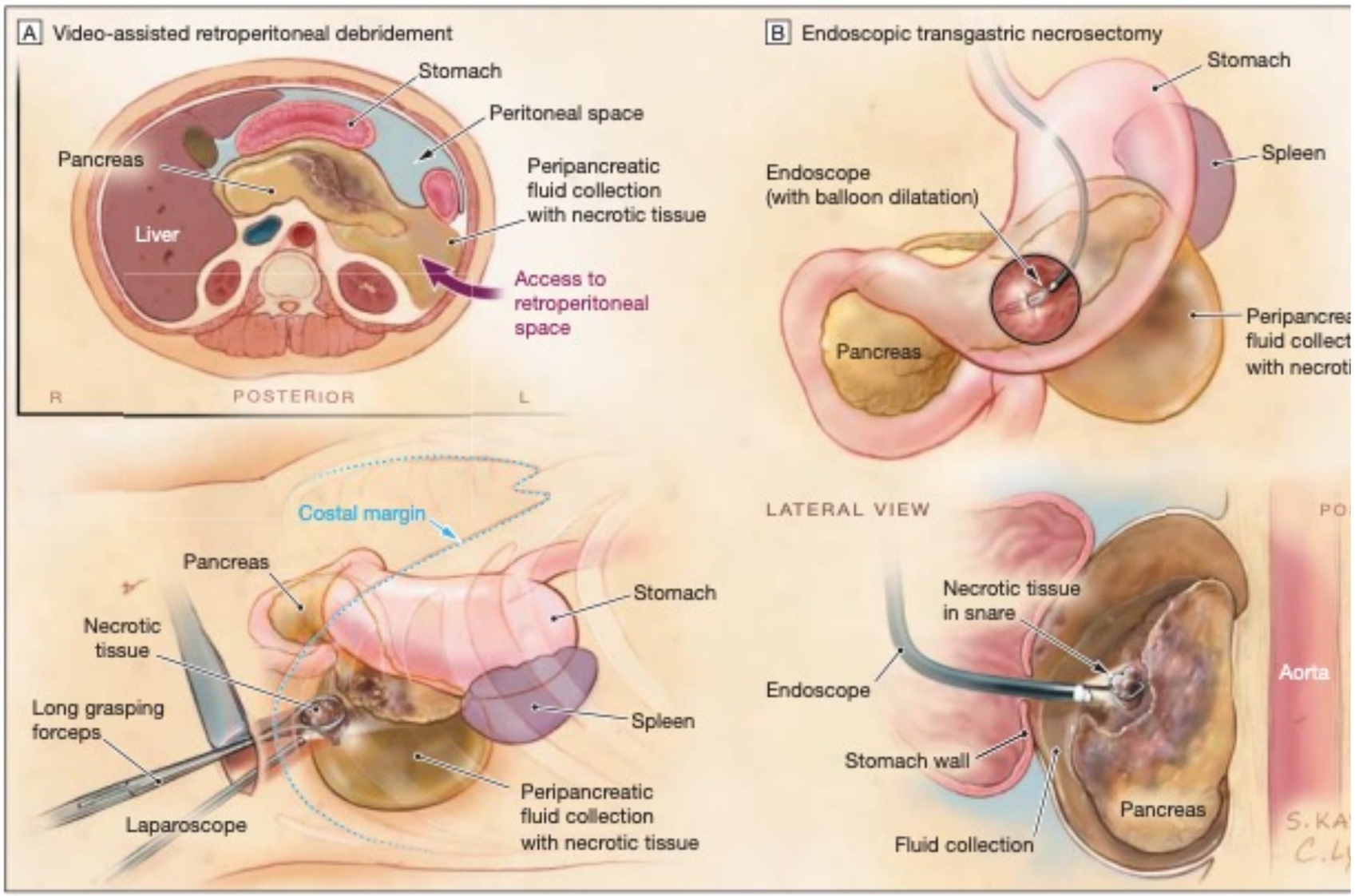
Variable	Odds Ratio (95% Confidence Interval)	*P*
Age (yr)	1.07 (1.04, 1.11)	<0.0001
Preoperative multiorgan failure	8.67 (3.01, 25.0)	<0.0001
MARPN	0.31 (0.12, 0.81)	0.016
Preoperative APACHE II score	1.00 (0.90, 1.10)	0.939

- ***Efficacy of Conservative Treatment, Without Necrosectomy, for Infected Pancreatic Necrosis: A Systematic Review and Meta-analysis (2013)***
- 64% who are treated with antibiotics and/or percutaneous drainage do not need necrosectomy
- 12% mortality rate for those who are treated with abx and/or percutaneous drainage
  - Compared to ~45% who undergo open necrosectomy





VARD vs. Transgastric Necrosectomy

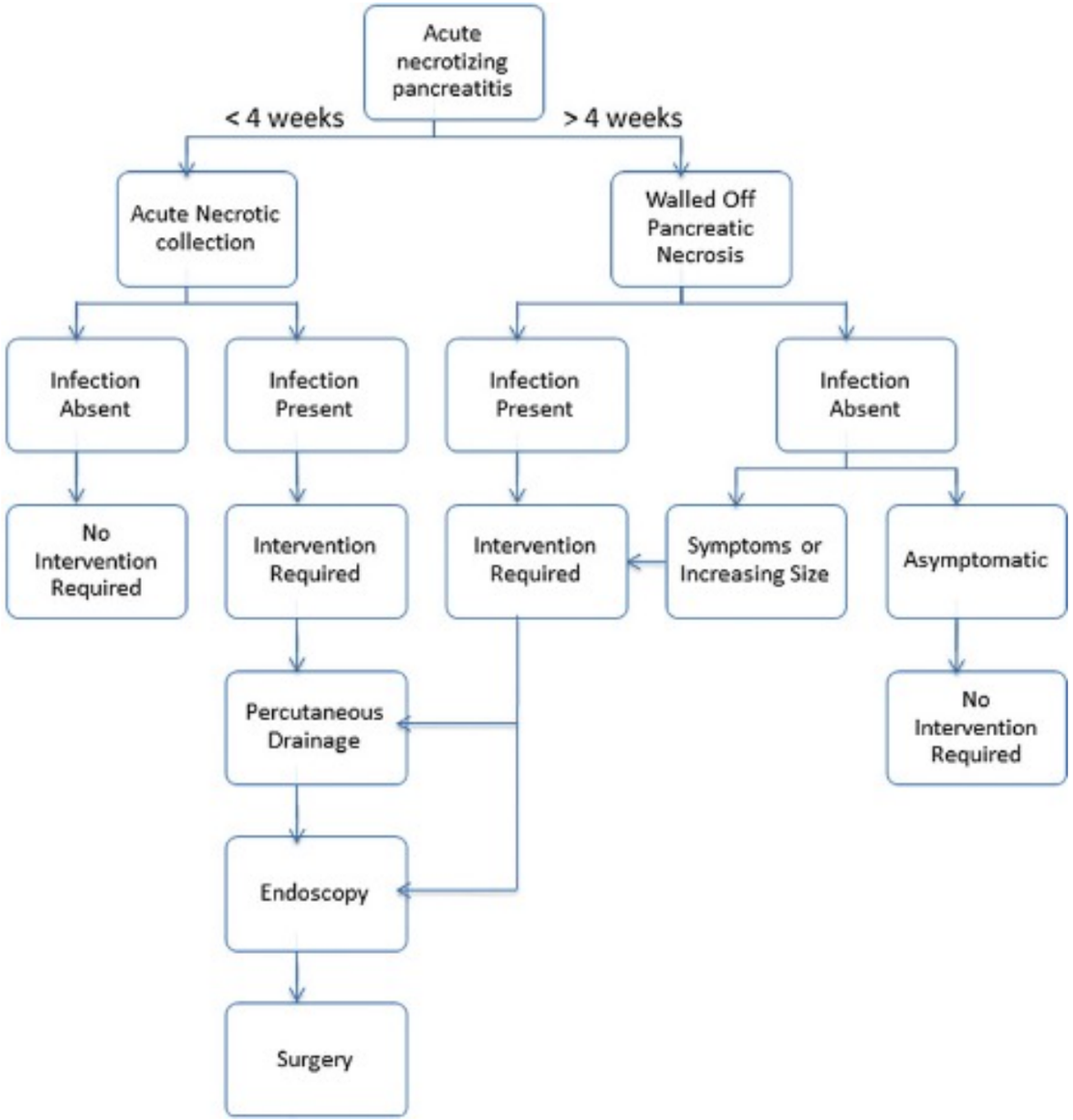


Endoscopic transgastric vs surgical necrosectomy for infected necrotizing pancreatitis: a randomized trial, JAMA, 2012.

- Decreased major complications (fistula, organ failure), number of necrosectomies and need for pancreatic enzymes when necrosectomy is completed via transgastric route rather than via VARD/laparotomy.
  - **Less dissection and no general anesthesia required during transgastric route**

	Surgical Necrosectomy (n = 10)	Endoscopic Transgastric Necrosectomy (n = 10)	Risk Difference (95% CI)	*P* Value
Major complications or death, No. (%)[b]	8 (80)	2 (20)	0.60 (0.16 to 0.80)	.03
Death, No. (%)	4 (40)	1 (10)	0.30 (–0.08 to 0.60)	.30
Major complications, No. (%)				
New-onset multiple organ failure[c]	5 (50)	0 (0)	0.50 (0.12 to 0.76)	.03
Intra-abdominal bleeding requiring intervention	0 (0)	0 (0)		
Enterocutaneous fistula or perforation of a visceral organ requiring intervention	2 (20)	0 (0)	0.20 (–0.11 to 0.51)	.47
Pancreatic fistula	7 (70)	1 (10)	0.60 (0.17 to 0.81)	.02
Long-term complications, No. (%)[d]	(n = 6)	(n = 9)		
New-onset diabetes	3 (50)	2 (22)	0.28 (–0.17 to 0.63)	.33
Use of pancreatic enzymes	3 (50)	0 (0)	0.50 (0.07 to 0.81)	.04
Persisting fluid collections[e]	3 (50)	2 (22)	0.28 (–0.17 to 0.63)	.33
Health care utilization, No.	(n = 10)	(n = 10)		
No. of necrosectomies, endoscopic or surgical	1 (1 to 2)	3 (2 to 6)		.007
New ICU admission anytime after randomization, No. (%)	5 (50)	1 (10)	0.4 (–0.002 to 0.68)	.14
Days in hospital after randomization[f]	36 (17 to 74)	45 (12 to 69)		.91

Treatment of necrotizing pancreatitis



Abdominal Compartment Syndrome

- Intra-abdominal hypertension has profound effects on both local and systemic tissues and organ systems that if untreated can lead to lethal organ failure

- The clinical condition that results from the organ dysfunction that occurs with IAH is termed abdominal compartment syndrome (ACS)

Abdominal Compartment Syndrome

- Local and systemic tissue and organ effects
  - Cardiovascular
  - Pulmonary
  - Renal
  - Gastrointestinal
  - Central nervous system

Intra-abdominal Hypertension and Abdominal Compartment Syndrome

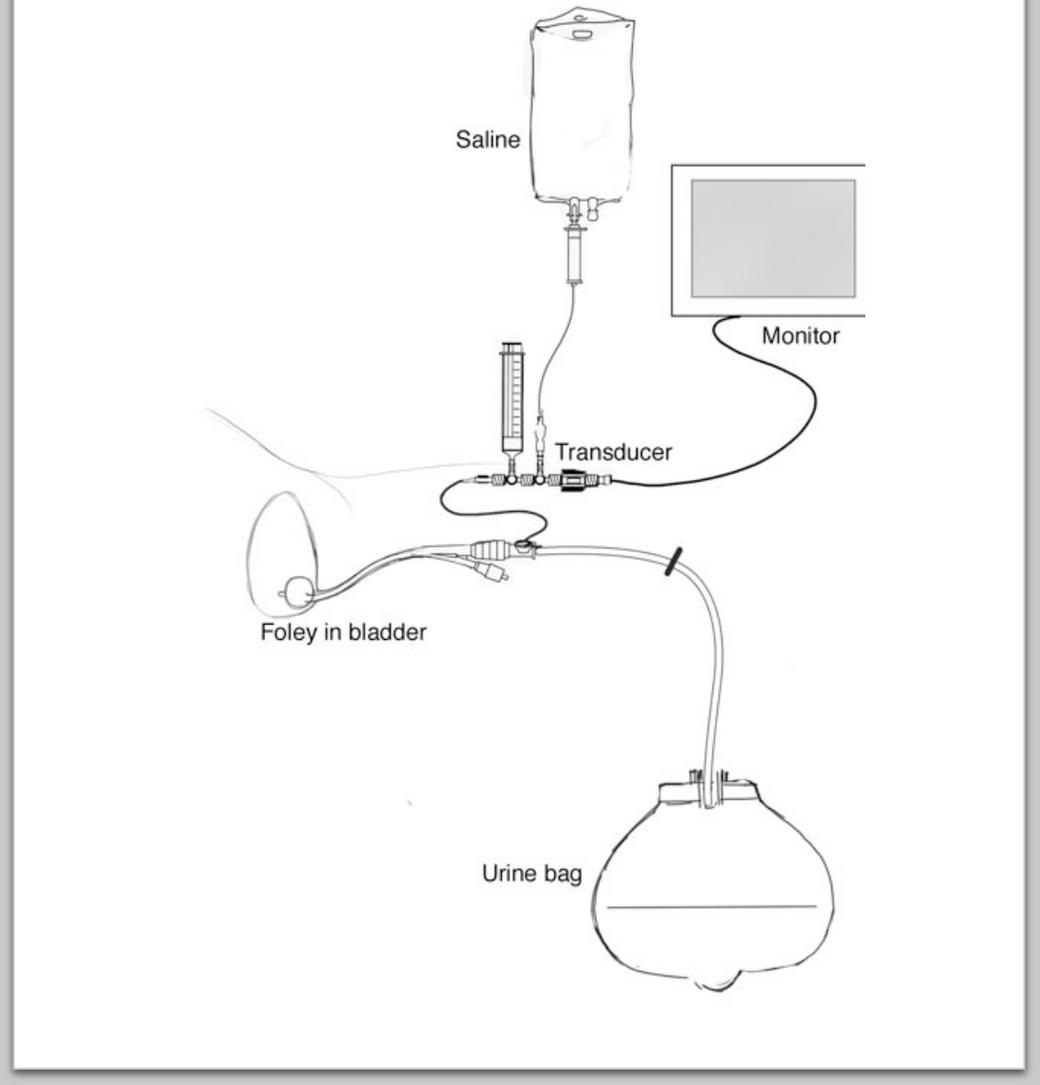
- Intra-abdominal Hypertension
  - Bladder Pressure >12mmHg (15cmH20)

- Abdominal compartment syndrome
  - Bladder pressure>20 mmHg (29cmH2O)
  - with evidence of pressure related organ dysfunction

2004 International ACS Consensus Definitions Conference

Bladder Pressure

- Instill 50cc saline into bladder via Foley
- Tubing at collecting bag clamped
- Transducer introduced at sampling port
- Transducer zeroed at level of symphysis pubis



Clinical Diagnosis

- Tense distended abdomen
- Elevated intra-abdominal pressures
- Decreased cardiac output
- Inadequate ventilation
- Elevated peak airway pressures
- Hypoxia and hypercarbia
- Renal dysfunction

Measurement of Intra-abdominal Pressure

- Prospective blinded trial of staff physician ability to detect intra-abdominal hypertension
- < 50% of the time was the clinician able to determine when IAP was elevated.

Primary Abdominal Compartment Syndrome

- Primary
  - associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical intervention

Secondary Abdominal Compartment Syndrome

- Secondary
  - conditions that do not originate from the abdomino-pelvic region

Burns

Sepsis/
Capillary Leak

Massive
Resuscitation

Burch Grading Scale

Grade	Pressure (mmHg)	Possible Organ Dysfunction
I	7-11	Splanchnic hypoperfusion
II	11-18	Elevated airway pressure, reduced cardiac output, oliguria
III	18-25	Hypotension, hypercarbia, hypoxia, anuria, increased Intracranial pressure
IV	>25	Multiple organ failure

Burch JM, Moore EE, Moore FA, Franciose R. The abdominal compartment syndrome. *Surg Clin North Am*. 1996; 76:833

Abdominal Compartment Syndrome

- Local and systemic tissue and organ effects
  - Cardiovascular
  - Pulmonary
  - Renal
  - Gastrointestinal
  - Central nervous system

Cardiovascular

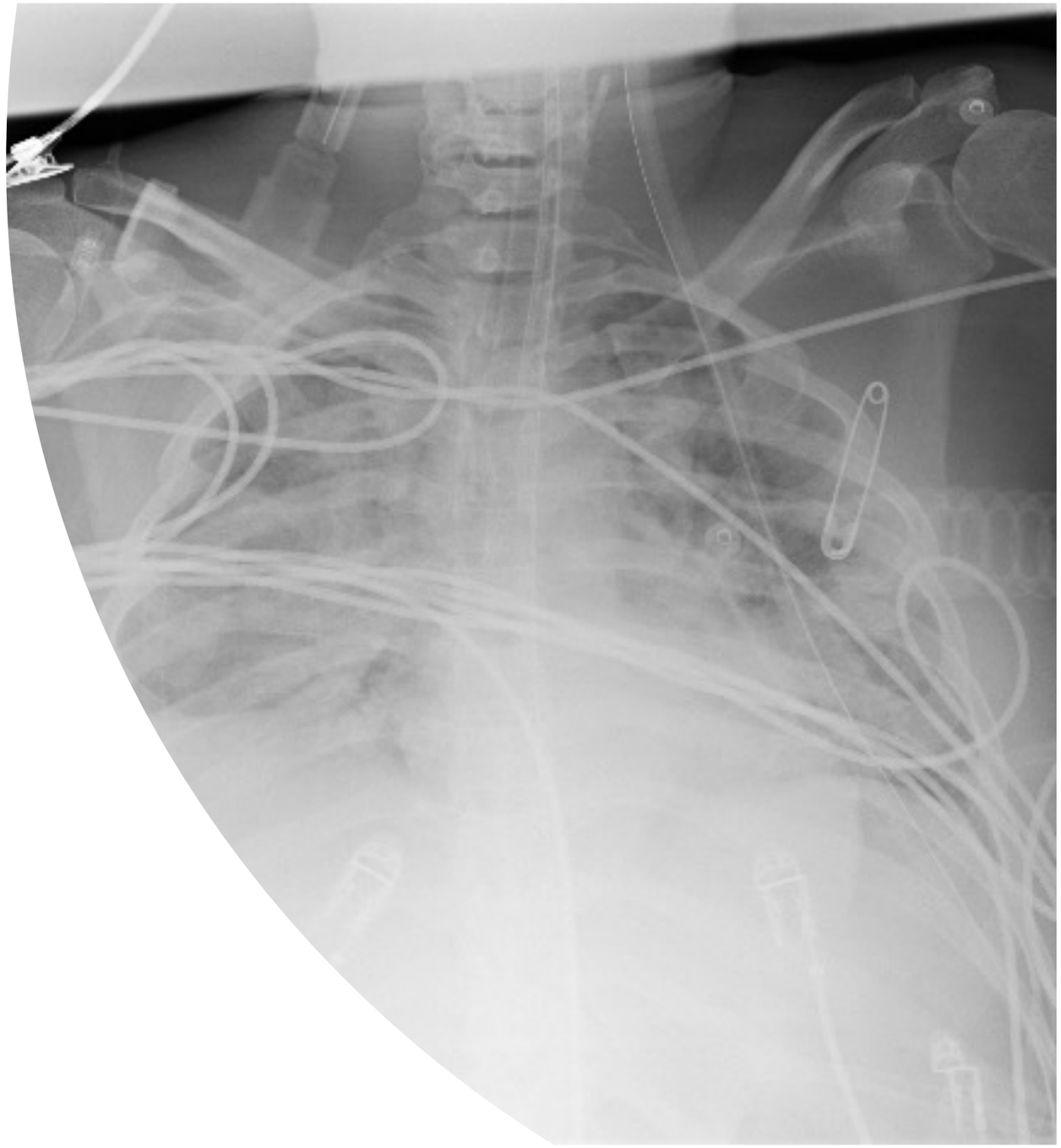
- The Causes
  - Compression of IVC decreases venous return
  - Direct compression of heart via diaphragm causes decreased cardiac compliance

- The Consequences
  - Decreased cardiac output
  - Increased SVR
  - Increased cardiac workload
  - Decreased tissue perfusion
  - Falsely elevated CVP
  - Cardiac insufficiency leading to cardiac arrest

Abdominal Compartment Syndrome

- Local and systemic tissue and organ effects
  - Cardiovascular
  - **Pulmonary**
  - Renal
  - Gastrointestinal
  - Central nervous system

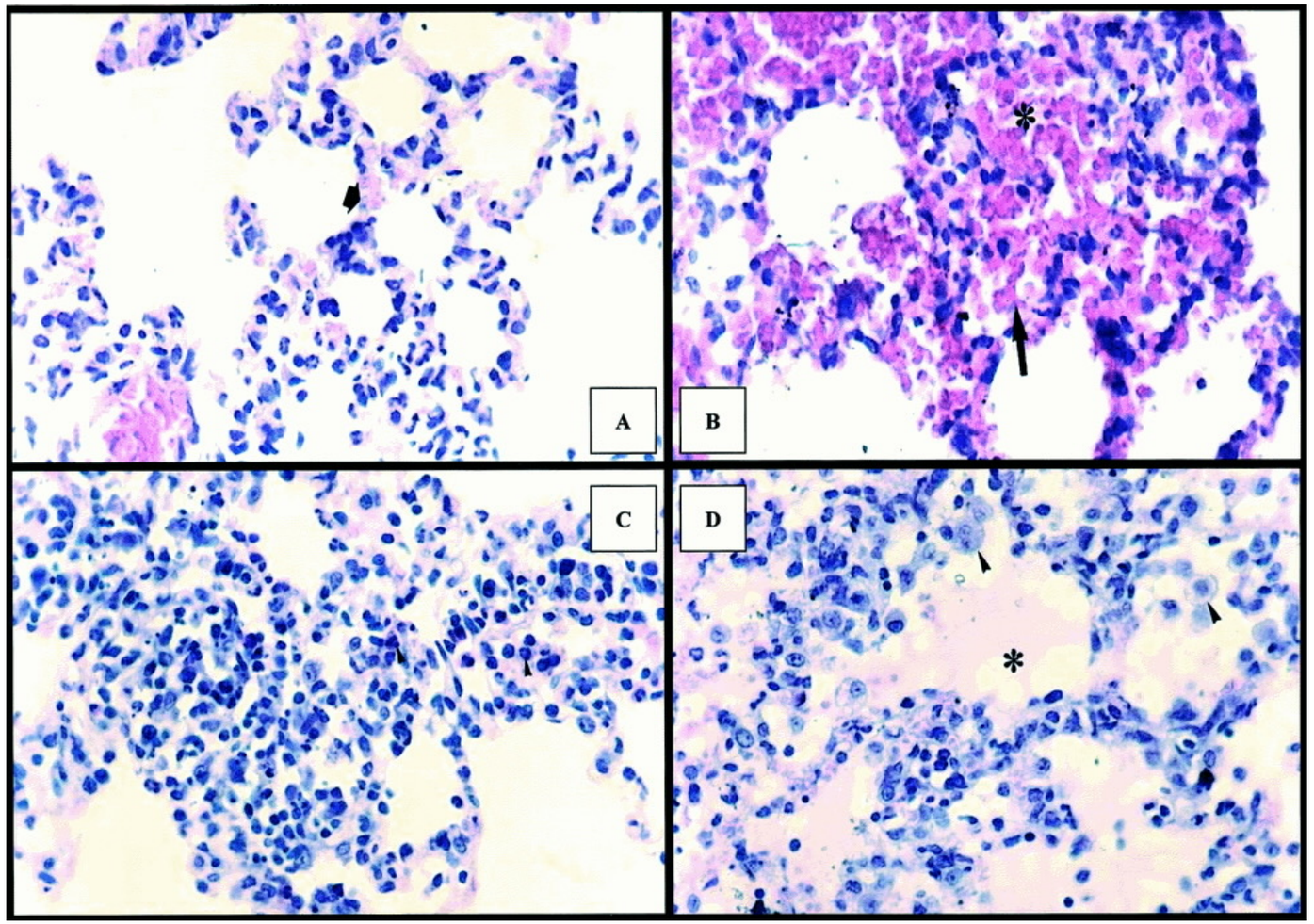
Pulmonary



- Pressure transmitted to the thorax via the diaphragm
  - Decreased compliance
  - Increased atelectasis
  - Pulmonary edema
  - Decreased O2 transport
  - Increased shunt
  - Increased dead space
- Further impairment
  - Capillary leak
  - Worsening pulmonary compliance
  - Systemic inflammatory response
  - Acute lung injury

Systemic Inflammatory Response Secondary to Abdominal Compartment Syndrome: Stage for Multiple Organ Failure

Joao B. Rezende-Neto, MD, Ernest E. Moore, MD, Marcus Vinicius Melo de Andrade, MD, Mauro Martins Teixeira, MD, PhD, Felipe Assis Lisboa, Rosa Maria Esteves Arantes, MD, PhD, Danielle Gloria de Souza, MSc, and Jose Rennan da Cunha-Melo, MD, PhD



Rezende Neto: J Trauma, Volume 53(6).December 2002.1121-1128

Abdominal Compartment Syndrome

- Local and systemic tissue and organ effects
  - Cardiovascular
  - Pulmonary
  - **Renal**
  - Gastrointestinal
  - Central nervous system

The Measurement of Intra-abdominal Pressure as a Criterion for Abdominal Re-exploration

IRVING L. KRON, M.D., P. KENT HARMAN, M.D., STANTON P. NOLAN, M.D.

TABLE 2. *Correlation of Intra-abdominal Pressure (IAP) and Renal Insufficiency*

Pt.	Procedure	Age	C.I.	PCW	Mean BP	Urine Output	IAP	Result
Patients Re-explored								
1	Repair aortic stab wound	64	3.3	19	120	0/hr	45	late death from sepsis
	(after re-exploration and decompression)		4.2	20	130	150/hr	23	
2	Gastrectomy	55	3.5	25	105	5/hr	34	
	(after re-exploration and decompression)		3.3	20	100	200/hr	15	excellent
3	Ruptured AAA	72	3.0	18	100	5/hr	40	
	(after re-exploration and decompression)		3.2	18	100	150/hr	10	excellent
4	Resection pelvic tumor	29	—	22	100	5/hr	30	late death from sepsis
	(after re-exploration and decompression)		—	15	90	100/hr	10	
Patients not Re-explored								
5	Hemobilia repair	55	2.0	24	100	5/hr	77	death
6	Portacaval shunt	68	3.0	24	100	0/hr	60	death
7	Splenorenal shunt	42	—	20	100	5/hr	40	death

Kron et al. The measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. Annals of Surgery. 1983; 199:28-30.

Abdominal Compartment Syndrome

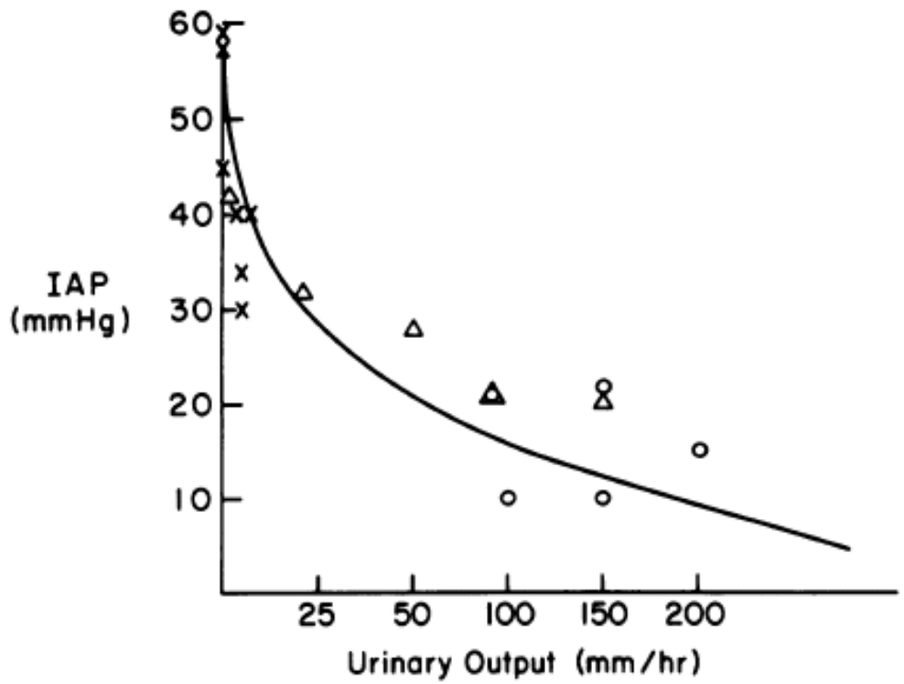


FIG. 1. The correlation of intra-abdominal pressure (IAP) and urinary output. △ = patient #1; × = without re-exploration; and ○ = after re-exploration.

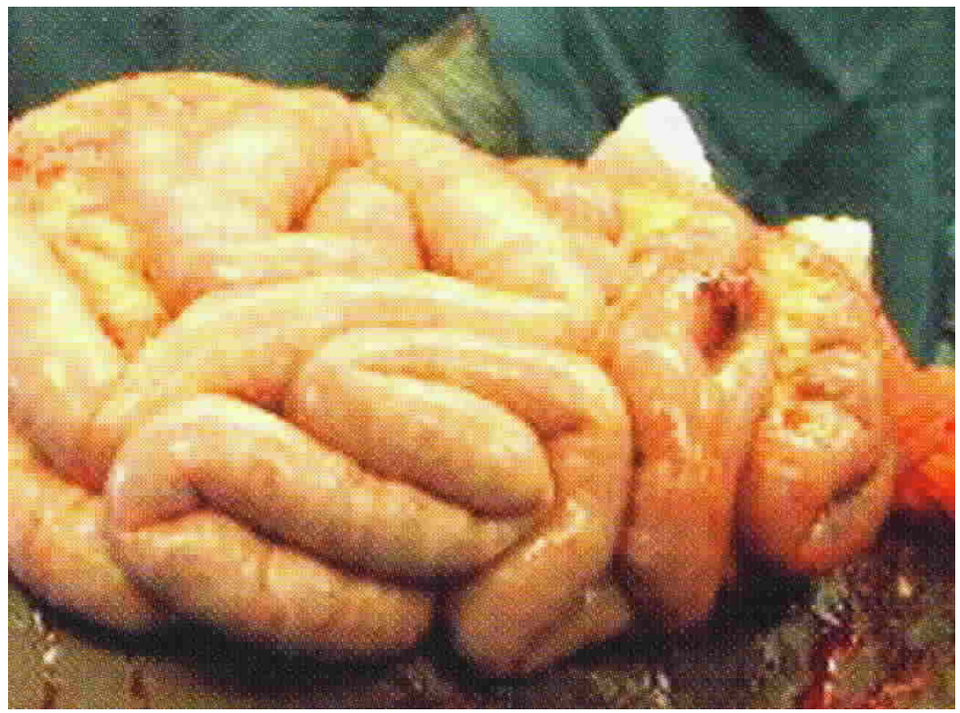
Kron et al. The measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. Annals of Surgery. 1983; 199:28-30.

Abdominal Compartment Syndrome

- Local and systemic tissue and organ effects
  - Cardiovascular
  - Pulmonary
  - Renal
  - Gastrointestinal
  - Central nervous system

Gastrointestinal

- Splanchnic hypoperfusion
  - Edema
  - Acidosis
  - Bacterial translocation
- Hepatic hypoperfusion
  - Impaired clearance of lactate



Abdominal Compartment Syndrome

- Local and systemic tissue and organ effects
  - Cardiovascular
  - Pulmonary
  - Renal
  - Gastrointestinal
  - Central nervous system

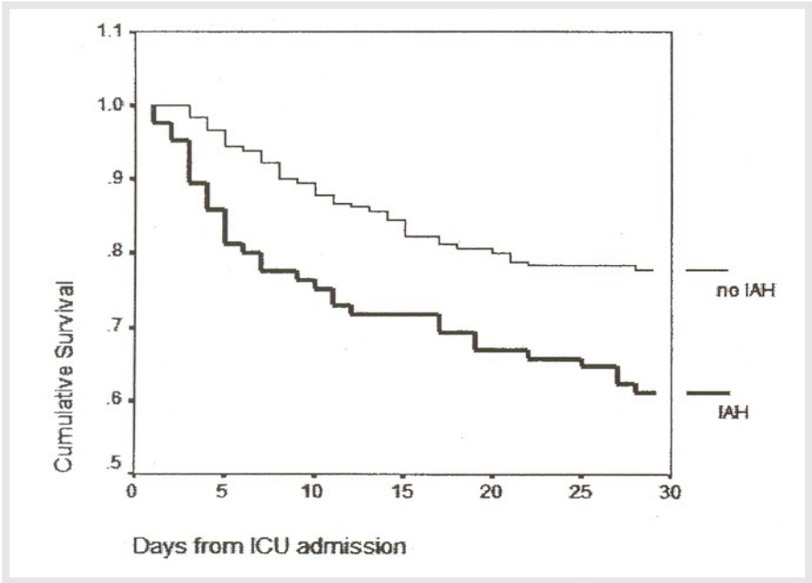
Central Nervous System

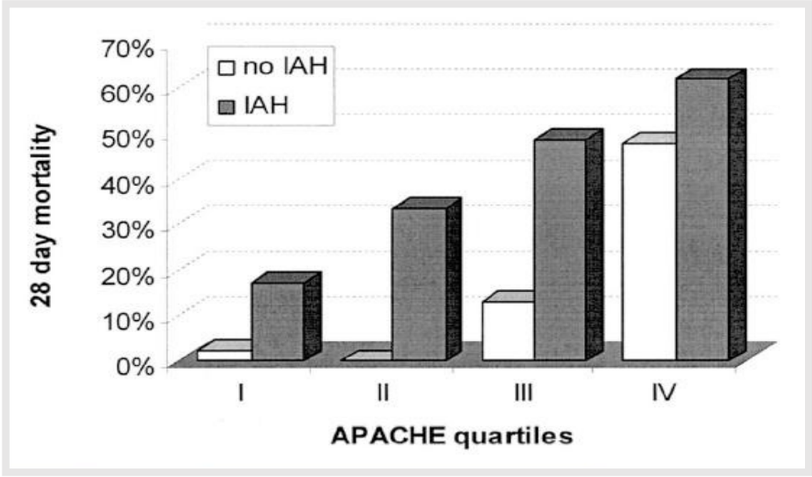
- Impaired venous drainage
- Increased intracranial pressure (ICP)
- Decreased cerebral perfusion pressure (CPP)
- Catecholamine surge

Decompressive Laparotomy to Treat Intractable Intracranial Hypertension after Traumatic Brain Injury

D'Andrea K. Joseph, MD, Richard P. Dutton, MD, Bizhan Aarabi, MD, and Thomas M. Scalea, MD

Intra-abdominal Hypertension Independent Predictor for Mortality





- Multicenter, prospective epidemiologic study
- 14 mixed medical and surgical intensive care units
- IAP>12 was an independent risk factor for organ failure and mortality

Malbrain, Crit Care Med, 33(2):315-22. 2005

Management of Intra-abdominal Hypertension

- Evacuate intraluminal contents
- Evacuate intra-abdominal space occupying lesions
- Improve abdominal wall compliance
- Optimized fluid administration
- Optimized systemic/regional perfusion

Management of Intra-abdominal Hypertension

- Evacuate intra-abdominal space occupying lesions
  - Surgical evacuation of lesion
  - In certain cases consider catheter drainage

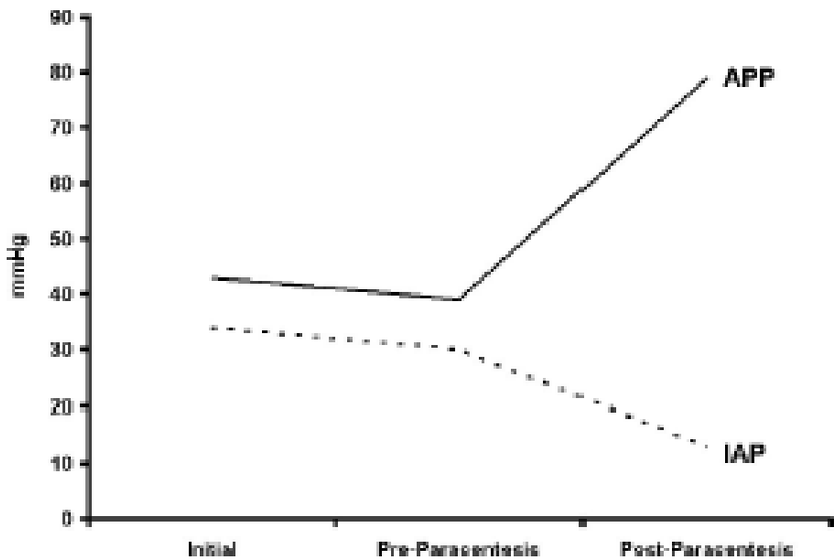
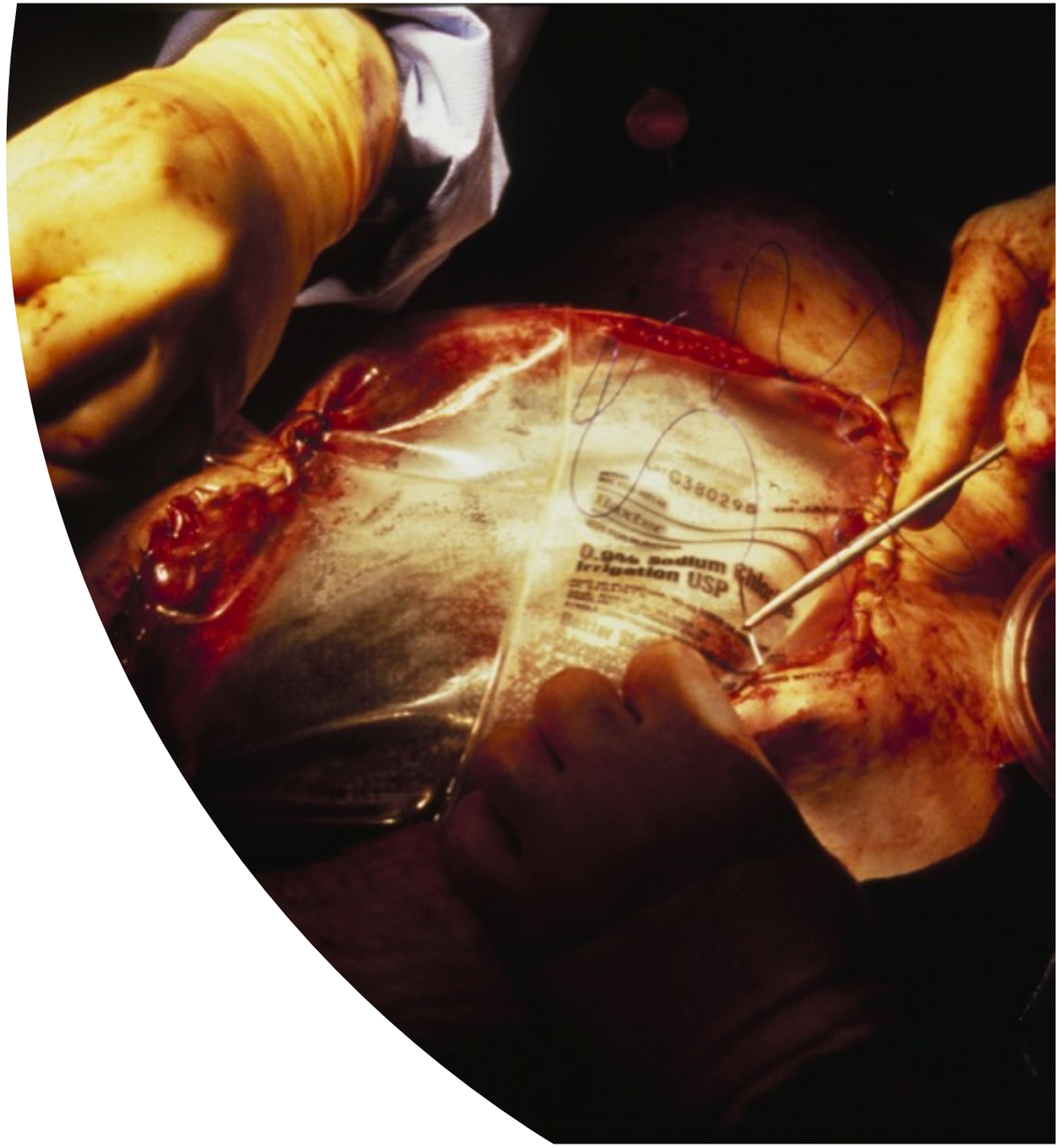


Fig. 1. *Response to paracentesis in the burn patient with abdominal compartment syndrome. APP, abdominal perfusion pressure; IAP, intra-abdominal pressure.*

Parra MW, Haitham A, Smith HG, Cheatham ML. Paracentesis for resuscitation-induced abdominal compartment syndrome: an alternative to decompressive laparotomy in the burn patient. 2006. *J Trauma*. 2006; 60:1119-1121.

Management of Abdominal Compartment Syndrome

- Decompressive laparotomy
  - Placement of temporary abdominal closure
    - Bogotá bag
    - Silo/Chimney
    - Vacuum dressing



HARVARD MEDICAL SCHOOL

Summary

- Don’t forget the Fearsome Four
  - Mesenteric Ischemia, colitis, cholecystitis, Bowel Perforation
- Pneumatosis Intestinalis – Can be commonly seen on imaging of critically ill patients
  - Etiology is varied, doesn’t always require surgical intervention.
- Necrotizing Pancreatitis has very high mortality
  - Surgical Necrosectomy carries high morbidity and mortality
- ACS is a clinical syndrome of organ dysfunction that results from intra-abdominal hypertension
  - Characterized by tense abdomen, respiratory, cardiovascular, renal, GI and CNS dysfunction
  - Local and systemic effects
  - ACS should be anticipated and avoided
  - Early intervention for IAH prevents ACS
  - Early decompression for ACS saves lives


Thank You
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Question 1

- A 48-year-old man undergoes successful renal transplantation for ESRD. He is maintained on prednisone and tacrolimus for immunosuppression. 2 weeks post-transplant he presents to the ED with severe diarrhea and increasing abdominal pain. He denies nausea or vomiting. He is found to have a Temp 39.0 degree Celsius. Pulse of 120 and BP of 80/50. His abdomen is markedly distended and diffusely tender to palpation. A CT of the abdomen and pelvis reveals diffuse colonic thickening. His WBC is 40,000. His stool studies are positive for *C.diff.* He is admitted to the Surgical ICU and started on oral Vancomycin. Over the first few hours the patient requires the start of pressor despite fluid resuscitation. The next best step in the management of this patient is?

  A) Continue with IV hydration, oral vancomycin and add IV metronidazole
  B) Exploratory laparoscopy
  C) Exploratory laparotomy and diverting ileostomy
  D) Exploratory laparotomy and subtotal colectomy with end-ileostomy
  E) Place rectal tube and add Vancomycin enemas

Answer Question 1

- Answer - D

  - This patient shows evidence of severe C diff Colitis and worsening clinical picture in the setting of the addition of pressor despite fluid resuscitation. The recommended procedure for *C.diff* colitis is a subtotal colectomy and end ileostomy. A described procedure of diverting loop ileostomy and colonic lavage (with polyethylene glycol) followed by postoperative antegrade instillation of Vancomycin flushes via the ileostomy has been described as an alternative procedure. However there has not been any randomized controlled studies to validate this approach.

Question 2

- A 42 year-old male remains in the SICU on post injury day 2 after a fall from 30 feet. He sustained multiple spinal fractures, a grade III splenic laceration (managed non-op) and a SDH. Since admission he has received 10L crystalloids and two units of PRBC. He is sedated and mechanically ventilated on AC mode and you are called for slowly rising peak airway pressures without a change in other paraments or SaO2. He is suctioned with scant amount of bloody secretions. His Urine output has decreased from 70cc/hr to 15cc/hr. The next most appropriate step in the management is?

  A) Magnesium sulfate 4 mg IVP

  B) Neuromuscular blockade

  C) Change to pressure control ventilation

  D) Bladder Pressure Measurement

  E) Fluid bolus with 1L crystalloid

Answer Question 2

- D – Bladder pressure measurement

  - In this scenario, slowly rising pressures indicate a different process than those that rise acutely. The patient received large-volume fluid resuscitation due to his multiple injuries. This can lead to an increase in intra-abdominal pressure from visceral edema, hemorrhage and well as acute ascites formation. Measuring the intra-abdominal pressure using the bladder pressure to assess for intra-abdominal HTN and abdominal compartment syndrome would readily assess for this possibility.