

Surgical Decompression for Abdominal Compartment Syndrome in Severe Acute Pancreatitis

Panu Mentula, MD, PhD; Piia Hienonen, MD; Esko Kemppainen, MD, PhD;
Pauli Puolakkainen, MD, PhD; Ari Leppäniemi, MD, PhD

Hypothesis: In patients with severe acute pancreatitis and abdominal compartment syndrome, establishment of the indications and optimal time for surgical decompression may avoid exacerbation of multiple-organ dysfunction syndrome.

Design: Retrospective study.

Setting: Tertiary care university teaching hospital.

Patients: Twenty-six consecutive patients with severe acute pancreatitis and abdominal compartment syndrome treated by surgical decompression between January 1, 2002, and December 31, 2007.

Intervention: Surgical decompression of the abdomen.

Main Outcome Measures: Morbidity, mortality, and organ dysfunction before and after surgical decompression.

Results: At the time of surgical decompression, the median sequential organ failure assessment score among patients was 12 (interquartile range, 10-15), and the median intra-abdominal pressure was 31.5 (interquartile range, 27-35) mm Hg. After surgical decompression, renal or respiratory function was improved in 14 patients (54%). The overall hospital mortality was 46%, but mortality was 18% among 17 patients in whom surgical decompression was performed within the first 4 days after disease onset.

Conclusions: Patients with severe acute pancreatitis and abdominal compartment syndrome managed by surgical decompression had severe multiple-organ dysfunction syndrome and high mortality. Surgical decompression may improve renal or respiratory function. Early surgical decompression is associated with reduced mortality in patients with severe acute pancreatitis, early multiple-organ dysfunction syndrome, and abdominal compartment syndrome.

Arch Surg. 2010;145(8):764-769

SEVERE ACUTE PANCREATITIS (SAP) is characterized by the development of local pancreatic complications, organ failure, or both.¹ The development of persistent multiple-organ dysfunction syndrome (MODS) has been recognized as a major determinant of mortality among patients with SAP²; therefore, it is thought to have a major role in defining SAP.³ Systemic release of inflammatory mediators in conjunction with systemic activation of leukocytes and microvascular endothelial cells is considered to have a detrimental role in the progression of MODS, which develops early in the course of SAP.⁴ Up to one-half of deaths from acute pancreatitis occur during the first week of hospital admission and are related to MODS.⁵ In the later course of the disease, death results from persisting MODS accompanied by secondary infection.⁶

In SAP, several inflammatory mediators contribute to increased capillary per-

meability in various organs,⁷ which together with aggressive fluid resuscitation may result in visceral edema and the development of intra-abdominal hypertension (IAH).^{8,9} High intra-abdominal pressure (IAP) levels can significantly reduce perfusion of abdominal viscera, rendering tissues susceptible to hypoxic injury that may aggravate systemic inflammatory response syndrome. Reduced renal blood flow can impair renal function¹⁰; furthermore, IAH can result in reduced pulmonary compliance and decreased venous return to the heart.^{11,12} Abdominal compartment syndrome (ACS) is a clinical condition that consists of IAH (IAP >20 mm Hg) and new organ dysfunction.¹³ The best treatment for ACS is its prevention by intensive monitoring to avoid excess fluid administration.¹² The first-line management of ACS in SAP should be conservative, but surgical decompression is needed when conservative methods are insufficient.¹⁴

Author Affiliations:
Department of
Gastroenterologic and
General Surgery, Helsinki
University Central Hospital,
Helsinki, Finland.

Table 1. Characteristics of 26 Patients

Variable	Value
Sex, No.	
Male	23
Female	3
Age, median (interquartile range), y	42 (35-49)
Causes of acute pancreatitis, No. (%)	
Alcohol	21 (81)
After ERCP	1 (4)
Medication	2 (8)
Idiopathic	2 (8)
Body mass index, median (interquartile range) ^a	31.5 (29-36)
Time to hospital admission after disease onset, median (interquartile range), d	1.5 (1-3)
Time to ICU admission after hospital admission, median (interquartile range), d	0.5 (0-1)
Preoperative organ failure, SOFA score \geq 3 per organ system, No. (%)	
Respiratory	24 (92)
Cardiovascular	23 (88)
Renal	14 (54)
Coagulation	4 (15)
Hepatic	2 (8)
Central nervous system	1 (4)

Abbreviations: ERCP, endoscopic retrograde cholangiopancreatography; ICU, intensive care unit; SOFA, sequential organ failure assessment.

^aCalculated as weight in kilograms divided by height in meters squared.

The fastest and the most effective treatment of ACS is surgical decompression by midline laparostomy, but bilateral subcostal laparostomy¹⁵ or subcutaneous linea alba fasciotomy¹⁶ may be used as an alternative. Although surgical treatment of ACS results in immediate decrease of IAP, the effects on organ dysfunction and mortality have been inconclusive.¹⁷ Patients unsuitable for early closure of an open abdomen may encounter substantial morbidity, including bowel fistulas and giant ventral hernias.¹⁸ Although ACS may exacerbate MODS in patients with SAP, indications and the optimal time for surgical decompression need to be established in this entity. Knowledge about surgical decompression for ACS in patients with SAP is limited and is mainly based on published case series with high mortality.^{8,19,20}

METHODS

This study is a retrospective analysis of 26 consecutive patients with SAP undergoing surgical decompression for ACS during a 6-year period (January 1, 2002, to December 31, 2007). Five of these patients have been described earlier.^{9,15,16} During 6 years, 1620 patients with 2345 episodes of acute pancreatitis were treated at Helsinki University Central Hospital, Helsinki, Finland. Of these, 226 episodes (9.6%) involved admission to an intensive care unit (ICU) because of SAP and organ dysfunction. Characteristics of the patients are summarized in **Table 1**. All patients were treated in the ICU, and admission to the ICU preceded surgical decompression. Intra-abdominal pressure was measured using a Foley bladder catheter (Foley Manometer LV; Holtech Medical, Charlottenlund, Denmark), and IAP values presented herein represent the highest measured IAP of the day. Patients' sequential organ failure assessment (SOFA) scores²¹ were calculated daily during the ICU stay. Organ failure was defined as an organ-specific SOFA score of

Table 2. Risk, Injury, and Failure From the RIFLE Criteria²² for Acute Renal Failure

Variable	Increased Serum Creatinine Level or GFR Criterion	Urine Output Criterion
Risk	Increased serum creatinine level 1.5-fold from baseline or GFR decrease >25%	<0.5 mL/kg/h for 6 h
Injury	Increased serum creatinine level 2-fold from baseline or GFR decrease >50%	<0.5 mL/kg/h for 12 h
Failure	Increased serum creatinine level 3-fold from baseline or GFR decrease >75% or serum creatinine level \geq 4.0 mg/dL and acute rise \geq 0.5 mg/dL	<0.3 mL/kg/h for 24 h or anuria for 12 h

Abbreviations: GFR, glomerular filtration rate; RIFLE, risk, injury, failure, loss, and end-stage kidney failure.

SI conversion factor: To convert creatinine level to micromoles per liter, multiply by 88.4.

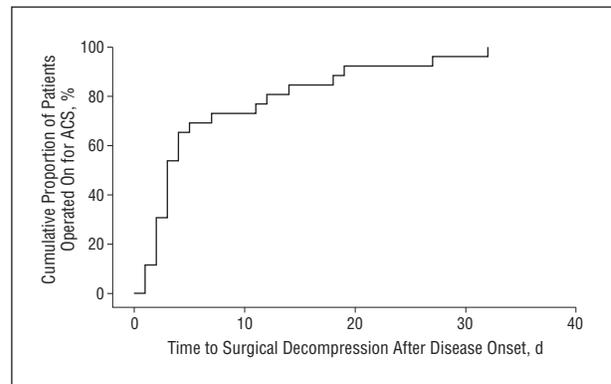


Figure 1. Cumulative proportion of patients with severe acute pancreatitis (n=26) undergoing surgical decompression for abdominal compartment syndrome (ACS).

3 or 4. Acute renal failure was retrospectively classified according to the risk, injury, failure, loss, and end-stage kidney failure (RIFLE) criteria²² (**Table 2**).

Statistical analysis was performed using commercially available software (SPSS 15.0 for Windows; SPSS Inc, Chicago, Illinois). The Mann-Whitney test was used in comparisons of continuous data among independent groups. When appropriate, Wilcoxon signed rank test or Friedman test was used in comparisons of IAP at different time points. χ^2 Test or Fisher exact test was used in comparisons of proportions. Correlations were tested using Pearson product moment correlation.

RESULTS

SURGICAL DECOMPRESSION

Surgical decompression was performed a median of 1 day (range, 0-29 days) after hospitalization. The time of surgical decompression after disease onset is shown in **Figure 1**. The method of surgical decompression was full-thickness midline laparostomy in 18 patients (69%), full-thickness transverse bilateral subcostal laparostomy in 1 patient, and subcutaneous linea alba fasciotomy in 7 patients (27%), 2 of whom underwent completion full-thickness midline laparostomy on postoperative day 1. The

Table 3. Renal Function Outcome Among 26 Patients

Preoperative Renal Function ^a	No. (%) of Patients			
	All Patients	Preoperative RRT	Need for RRT	No RRT
No acute kidney injury	4 (15)	0	1 (25)	3 (75)
Risk	2 (8)	0	0	2 (100)
Injury	6 (23)	0	4 (67)	2 (33)
Failure	14 (54)	7 (50)	13 (93)	1 (7)
Total	26	7 (27)	18 (69)	8 (31)

Abbreviations: RIFLE, risk, injury, failure, loss, and end-stage kidney failure; RRT, renal replacement therapy.

^aAccording to risk, injury, and failure from the RIFLE criteria.²²

Table 4. Risk Factors for Fatal Outcome

Variable	Survivors (n=14)	Nonsurvivors (n=12)	P Value
Preoperative SOFA score, median (interquartile range)	10 (9-15)	13 (12-15)	.06
Preoperative renal failure, No. (%) ^a	5 (36)	9 (75)	.045 ^b
Preoperative intra-abdominal pressure, median (interquartile range), mm Hg	33 (30-36)	28 (20-34)	.04 ^b
Time to surgical decompression after disease onset, median (interquartile range), d	1 (1-2)	7 (1-17)	.005 ^b
Improved respiratory or renal function, No. (%)	7 (50)	7 (58)	.67
Need for renal replacement therapy, No. (%)	10 (71)	8 (67)	.79
Other intra-abdominal surgery, No. (%) ^c	7 (50)	10 (83)	.11

Abbreviation: SOFA, sequential organ failure assessment.

^aAccording to the risk, injury, failure, loss, and end-stage kidney failure (RIFLE) criteria.²²

^bStatistically significant.

^cNecrosectomy, cholecystectomy, splenectomy, or bowel resection.

primary coverage of abdominal viscera in 21 patients with an open abdomen was a Bogota bag (a self-made plastic silo that was cut from a sterile 3-L genitourinary irrigation bag) in 14 patients and vacuum-assisted closure in 7 patients.

INTRA-ABDOMINAL PRESSURE

The median preoperative IAP was 31.5 (interquartile range [IQR], 27-35) mm Hg. The IAP decreased 16 (IQR, 9-21) mm Hg after full-thickness laparostomy (n=19) and 12 (10-13) mm Hg after subcutaneous linea alba fasciotomy (n=7) (P=.31). The decrease in IAP showed strong positive correlation with preoperative IAP levels after full-thickness laparostomy (r=0.943, P<.01) but not after subcutaneous linea alba fasciotomy. The decrease in IAP (median, 18 to 15.5 mm Hg; P=.05) continued for 5 subsequent days.

ORGAN DYSFUNCTION

At the time of surgical decompression, the median SOFA score among patients was 12 (IQR, 10-15). Twenty-five patients (96%) had MODS, defined by at least 2 organ failures, and 13 patients (50%) had 3 or more organ failures.

As summarized in Table 1, respiratory and cardiovascular systems were the most frequently failed organs. After surgical decompression, 14 patients (54%) demonstrated improved renal function (n=7), as indicated by increased urine output exceeding 200 mL/d, or showed improved respiratory function (n=10), defined as a greater than 20% increase in the ratio of PaO₂ to fraction of inspired oxygen (FIO₂). Despite this, surgical decompression did not result in any significant improvement to organ dysfunction according to the SOFA score during 5 postoperative days.

Patients who increased their ratio of PaO₂ to FIO₂ as a result of surgical decompression had significantly lower ratios of PaO₂ to FIO₂ before decompression (median, 68 [IQR, 62-115] mm Hg; n=13) than patients who did not (median, 132 [115-182] mm Hg; n=13) (P=.01). To find optimal cutoff values for the ratio of PaO₂ to FIO₂ for patients with acute pancreatitis and ACS who would benefit from surgical decompression, a receiver operating characteristic curve analysis was performed. Results showed that 10 patients (77%) with a ratio of PaO₂ to FIO₂ below 108 mm Hg improved their respiratory dysfunction, whereas only 3 patients (23%) with a ratio above 108 mm Hg demonstrated an increased ratio of PaO₂ to FIO₂ (P=.01).

Preoperative renal function and renal function prognosis are summarized in **Table 3**. Fifteen of 19 patients without preoperative renal replacement therapy had renal dysfunction (risk, injury, or failure according to the RIFLE criteria). Five of these 15 patients showed improved renal function (increased urine output >200 mL/d) after surgical decompression, but only 3 of 5 avoided renal replacement therapy.

MORTALITY

The overall hospital mortality was 12 of 26 patients (46%) at a median of 25 (IQR, 18-56; range, 11-129) days after admission. Preoperative renal failure according to the RIFLE criteria, lower preoperative IAP, and longer time to surgical decompression after disease onset were significantly associated with mortality (**Table 4**). As shown in **Figure 2**, none of 9 patients in whom surgical decompression was performed 5 or more days after disease onset survived, whereas 14 of 17 patients in whom surgical decompression was performed within the first 4 days after disease onset survived.

MORBIDITY

The median number of reoperations was 4 (IQR, 2-9; range, 0-17) per patient. Patients with an open abdomen had significantly more reoperations (median, 7 per patient; $n=21$) than patients with subcutaneous linea alba fasciotomy (median, 2 per patient; $n=5$) ($P=.03$). Subsequent intra-abdominal operations were performed in 17 patients, including necrosectomy in 16 patients, cholecystectomy in 1 patient, splenectomy in 1 patient, and colon resection in 3 patients. In 2 patients, necrosectomy was performed at the same time as surgical decompression 18 and 27 days after disease onset. In both patients, the indication for simultaneous necrosectomy was clinical suspicion of infected pancreatic necrosis because of deterioration of MODS. In 4 patients, subsequent necrosectomy was performed 1 to 5 days after surgical decompression because of deterioration of MODS and suspected or verified infection of pancreatic necrosis 13 to 37 days after disease onset. Four patients developed fistulas (1 biliary, 2 small bowel, and 1 colon). All fistulas developed after necrosectomy or bowel resection. Intra-abdominal infection as determined by microbiologic culture was present in 18 of 21 patients with an open abdomen, whereas intra-abdominal infection was present in 1 of 5 patients with subcutaneous linea alba fasciotomy ($P=.01$). Infected pancreatic necrosis was diagnosed in 12 patients. Of 17 patients surviving to abdominal closure after full-thickness laparostomy (midline, transverse, or completion), 7 patients underwent primary (one time or gradual) fascial closure a median of 13 (range, 5-31) days after subcutaneous linea alba fasciotomy, and 10 patients underwent split-thickness skin grafting a median of 32 (range, 22-40) days after subcutaneous linea alba fasciotomy.

COMMENT

In the present study, all patients (except 1) with SAP and ACS experienced severe MODS before surgical decompression. An earlier study²³ from our institution showed that patients with SAP and MODS have greater than 50% mortality. Furthermore, patients with SAP and IAP exceeding 25 mm Hg within the first 14 days in the ICU have been shown to have a 50% mortality in our hospital.⁹ Considering these earlier findings, the 46% hospital mortality in the present series of patients is comparable to the historical control values. Remarkably, we found that in the subgroup of patients with early surgical decompression the mortality was only 18%, which is considerably lower than the 65% mortality in patients with SAP and early (≤ 72 hours after admission) MODS reported by Halonen²⁴ and is lower than the 35% mortality in patients with SAP and persistent early-organ failure reported by Johnson and Abu-Hilal.²⁵ Also notable is that no deaths in the present study occurred during the first week of hospitalization, in contrast to other SAP studies.^{5,25} Although several confounding factors may explain these variations among studies, one explanation may be that ACS has a critical role in early SAP mortality and that surgical management of ACS can save patients' lives

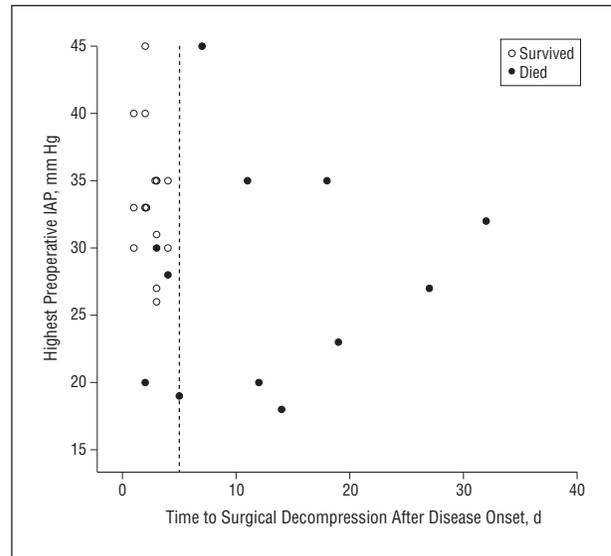


Figure 2. Highest preoperative intra-abdominal pressure (IAP) and the time to surgical decompression after disease onset. The dotted line denotes day 5.

during the early course of SAP. In contrast, mortality in the present study was related to deterioration of MODS longer than 2 weeks after disease onset, and surgical decompression for ACS at this later stage of SAP did not show any survival benefit.

Although increasing evidence indicates that patients with SAP can develop ACS,^{8,9,26} little is known about its management in this setting. Abdominal compartment syndrome can be categorized as primary or secondary based on its etiology.²⁷ According to consensus definitions,¹³ patients with SAP who develop ACS have primary ACS because of their abdominal origin of the disease. However, patients with SAP also have some features of secondary ACS, such as primary nonoperative management and high-volume fluid resuscitation.²⁷ Recommended treatment in patients with primary ACS is prompt surgical decompression, whereas nonoperative methods should be attempted first in patients with secondary ACS.¹³ Considering the clinical features of SAP, the logical first-line approach would be conservative, as in secondary ACS.¹⁴ Nonoperative methods for the treatment of IAH are sedation and neuromuscular blockade, nasogastric decompression, evacuation of excess peritoneal fluid, and correction of positive fluid balance, which may include continuous hemodiafiltration.²⁸ Indeed, our treatment policy in these patients has been that surgical decompression is performed only if conservative management fails.

Indications for surgical decompression in patients with SAP and ACS are unclear. In patients with SAP and deteriorating organ function, the degree to which IAH contributes to the progression of MODS is unknown. Although IAP has been shown to correlate with Acute Physiology and Chronic Health Evaluation II score in patients with SAP,⁸ a causal relationship between ACS and MODS in SAP has not been proven. According to current knowledge, the development of MODS is multifactorial, and inflammatory response has a major role in the pathogenesis.²⁹ In experimental models, IAH has been shown to increase the release of proinflammatory cyto-

kines into circulation and to cause acute lung and liver injury in the presence of circulating activated neutrophils.^{30,31} In patients with SAP, the activation of circulating neutrophils and monocytes is an early event and precedes ICU admission.⁴ Considering these factors, the development of ACS could further increase a proinflammatory stimulus that contributes to the development of organ failure in SAP. Therefore, early recognition of ACS and its adequate and timely treatment might prevent the progression of MODS to patient death. Unfortunately, the optimal threshold of IAP that would mandate surgical decompression is unknown. Furthermore, the optimal time for intervention is unclear. According to our mortality data herein, patients with IAP exceeding 25 mm Hg within the first 4 days after disease onset might be good candidates for surgical decompression.

The effect of surgical decompression on MODS was variable. There was no improvement in the daily SOFA score, and few patients showed increased urine output after surgical decompression. Furthermore, respiratory system changes were inconsistent, although patients with a lower ratio of PaO₂ to FIO₂ were more likely to improve their respiratory function. These results are in accord with earlier data reviewed by De Waele et al.¹⁷ Renal function may already be affected when the IAP exceeds 12 mm Hg, and risk for acute renal failure increases with increasing IAP.³² Restoration of renal function may be sensitive to the duration of ACS. According to the present data, the optimal time until surgical decompression to avoid renal replacement therapy is when the patient is at risk for acute renal failure or renal injury but not failure according to the RIFLE criteria. Although some patients showed no improvement in organ function, their deterioration may have been worse if ACS was left untreated. However, a controlled randomized trial would be needed to prove this.

Substantial morbidity was associated with the need for surgical decompression. Management of an open abdomen is a risk factor for fistulas, and management of fistulas in patients with an open abdomen is challenging.^{33,34} Four patients in the present study developed fistulas, although all of them developed after additional abdominal operations. Most patients with an open abdomen and temporary abdominal closure developed intra-abdominal infection. Furthermore, the proportion of patients who developed infected pancreatic necrosis was high at 46%. Although MODS is a known risk factor for infected pancreatic necrosis,⁶ an open abdomen may also serve as a route for contamination. Some patients needed necrosectomy, which probably reflected a high rate of infected pancreatic necrosis and MODS. The low rate of primary fascial closure resulted in several patients with planned hernia requiring subsequent abdominal wall reconstruction.³⁵ During the study period, a vacuum-assisted closure (V.A.C.; Kinetic Concepts, Inc, San Antonio, Texas) system³⁶ was introduced, but this did not dramatically change the rate of primary fascial closure (data not shown). A 2007 study³⁷ described a method of vacuum-assisted wound closure combined with mesh-mediated fascial traction, which might reduce the planned ventral hernia rate.

Because midline laparostomy and an open abdomen are associated with significant morbidity, alternative surgical methods have been developed. Subcutaneous linea alba

fasciotomy¹⁶ was initially performed in 7 patients, and bilateral subcostal transverse laparostomy¹⁵ was subsequently performed in 1 patient. The rationale was that primary closure of the abdominal wall would be more likely in patients with transverse laparostomy than in patients with midline laparostomy. Although successful closure was achieved in the patient with transverse laparostomy, additional patient experience is needed to draw any conclusions. Subcutaneous linea alba fasciotomy resulted in significantly decreased IAP levels among patients in the study by Leppäniemi et al.,¹⁶ although 2 patients required an additional laparostomy for recurrent ACS on the first postoperative day. Because the decrease in IAP did not correlate with preoperative IAP among the patients undergoing subcutaneous linea alba fasciotomy, we do not recommend this method in patients with IAP exceeding 35 mm Hg, as it may insufficiently treat ACS. Furthermore, patients with subcutaneous linea alba fasciotomy should be closely monitored because they may be at risk of developing recurrent ACS. Potential benefits of the subcutaneous linea alba approach may be lower rates of intra-abdominal infections and fistulas. Patients treated by this approach also need fewer operating room resources because there is no temporary abdominal coverage that needs to be changed. However, surviving patients may develop ventral hernia that requires subsequent surgery. For the time being, few data support subcutaneous linea alba fasciotomy or bilateral subcostal transverse laparostomy over midline laparostomy.

In conclusion, this study showed that early surgical decompression for ACS in patients with SAP is associated with reduced mortality. Surgical decompression is effective in decreasing IAP and improves respiratory and renal functions in more than half the patients. An open abdomen in patients with SAP is associated with significant morbidity, high reoperation rate, and low rate of primary fascial closure. Among patients with SAP, those having early MODS and ACS are good candidates for surgical decompression.

Accepted for Publication: June 25, 2009.

Correspondence: Panu Mentula, MD, PhD, Department of Gastroenterologic and General Surgery, Helsinki University Central Hospital, PO Box 340, 00029 HUS, Helsinki, Finland (panu.mentula@hus.fi).

Author Contributions: *Study concept and design:* Mentula, Kempainen, and Leppäniemi. *Acquisition of data:* Mentula and Hienonen. *Analysis and interpretation of data:* Mentula, Puolakkainen, and Leppäniemi. *Drafting of the manuscript:* Mentula. *Critical revision of the manuscript for important intellectual content:* Hienonen, Kempainen, Puolakkainen, and Leppäniemi. *Statistical analysis:* Mentula. *Obtained funding:* Puolakkainen. *Administrative, technical, and material support:* Hienonen and Kempainen. *Study supervision:* Leppäniemi.

Financial Disclosure: None reported.

Funding/Support: This study was supported by Helsinki University Research Funds (Dr Puolakkainen).

Previous Presentation: The abstract of this article was presented as a scientific poster at the 67th Annual Meeting of the American Association for the Surgery of Trauma; September 25, 2008; Maui, Hawaii.

1. Bradley EL III. A clinically based classification system for acute pancreatitis: summary of the International Symposium on Acute Pancreatitis, Atlanta, Ga, September 11 through 13, 1992. *Arch Surg*. 1993;128(5):586-590.
2. Mofidi R, Duff M, Wigmore S, Madhavan K, Garden O, Parks R. Association between early systemic inflammatory response, severity of multiorgan dysfunction and death in acute pancreatitis. *Br J Surg*. 2006;93(6):738-744.
3. Bollen TL, van Santvoort H, Besselink M, et al; Dutch Acute Pancreatitis Study Group. The Atlanta Classification of acute pancreatitis revisited. *Br J Surg*. 2008;95(1):6-21.
4. Mentula P, Kylänpää ML, Kempainen E, et al. Early prediction of organ failure by combined markers in patients with acute pancreatitis. *Br J Surg*. 2005;92(1):68-75.
5. McKay CJ, Evans S, Sinclair M, Carter CR, Imrie CW. High early mortality rate from acute pancreatitis in Scotland, 1984-1995. *Br J Surg*. 1999;86(10):1302-1305.
6. Büchler MW, Gloor B, Müller CA, Friess H, Seiler C, Uhl W. Acute necrotizing pancreatitis: treatment strategy according to the status of infection. *Ann Surg*. 2000;232(5):619-626.
7. Eibl G, Buhr H, Foitzik T. Therapy of microcirculatory disorders in severe acute pancreatitis: what mediators should we block? *Intensive Care Med*. 2002;28(2):139-146.
8. De Waele JJ, Hoste E, Blot SI, Decruyenaere J, Colardyn F. Intra-abdominal hypertension in patients with severe acute pancreatitis. *Crit Care*. 2005;9(4):R452-R457.
9. Keskinen P, Leppäniemi A, Pettilä V, Piilonen A, Kempainen E, Hynninen M. Intra-abdominal pressure in severe acute pancreatitis. *World J Emerg Surg*. 2007;2:e2. doi:10.1186/1749-7922-2-2. <http://www.wjes.org/content/2/1/2>. Accessed June 1, 2010.
10. Doty JM, Saggi BH, Sugerman HJ, et al. Effect of increased renal venous pressure on renal function. *J Trauma*. 1999;47(6):1000-1003.
11. McNelis J, Marini CP, Simms HH. Abdominal compartment syndrome: clinical manifestations and predictive factors. *Curr Opin Crit Care*. 2003;9(2):133-136.
12. An G, West M. Abdominal compartment syndrome: a concise clinical review. *Crit Care Med*. 2008;36(4):1304-1310.
13. Cheatham ML, Malbrain ML, Kirkpatrick A, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome, II: recommendations. *Intensive Care Med*. 2007;33(6):951-962.
14. Leppäniemi A, Johansson K, De Waele JJ. Abdominal compartment syndrome and acute pancreatitis. *Acta Clin Belg Suppl*. 2007;(1):131-135.
15. Leppäniemi A, Mentula P, Hienonen P, Kempainen E. Transverse laparostomy is feasible and effective in the treatment of abdominal compartment syndrome in severe acute pancreatitis. *World J Emerg Surg*. 2008;3(1):e6. <http://www.wjes.org/content/3/1/6>. Accessed June 1, 2010.
16. Leppäniemi AK, Hienonen PA, Siren JE, Kuitunen AH, Lindström OK, Kempainen EA. Treatment of abdominal compartment syndrome with subcutaneous anterior abdominal fasciotomy in severe acute pancreatitis. *World J Surg*. 2006;30(10):1922-1924.
17. De Waele JJ, Hoste EA, Malbrain ML. Decompressive laparotomy for abdominal compartment syndrome—a critical analysis. *Crit Care*. 2006;10(2):R51 <http://ccforum.com/content/10/2/R51>. Accessed June 1, 2010.
18. Miller RS, Morris J Jr, Diaz J Jr, Herring M, May A. Complications after 344 damage-control open celiotomies. *J Trauma*. 2005;59(6):1371-1374.
19. Siebig S, Iesalnieks I, Bruennler T, et al. Recovery from respiratory failure after decompression laparotomy for severe acute pancreatitis. *World J Gastroenterol*. 2008;14(35):5467-5470.
20. Chen H, Li F, Sun JB, Jia JG. Abdominal compartment syndrome in patients with severe acute pancreatitis in early stage. *World J Gastroenterol*. 2008;14(22):3541-3548.
21. Vincent JL, de Mendonça A, Cantraine F, et al; Working Group on "Sepsis-Related Problems" of the European Society of Intensive Care Medicine. Use of the SOFA score to assess the incidence of organ dysfunction/failure in intensive care units: results of a multicenter, prospective study. *Crit Care Med*. 1998;26(11):1793-1800.
22. Bellomo R, Ronco C, Kellum J, Mehta R, Palevsky P; Acute Dialysis Quality Initiative Workgroup. Acute renal failure: definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. *Crit Care*. 2004;8(4):R204-R212.
23. Halonen KI, Pettilä V, Leppäniemi AK, Kempainen EA, Puolakkainen PA, Haapiainen RK. Multiple organ dysfunction associated with severe acute pancreatitis. *Crit Care Med*. 2002;30(6):1274-1279.
24. Halonen K. *Outcome Prediction and Quality of Life in Severe Acute Pancreatitis* [dissertation]. Helsinki, Finland: Institute of Clinical Medicine, Faculty of Medicine, University of Helsinki; 2004. <http://urn.fi/URN:ISBN:952-10-1831-3>. Accessed May 26, 2010.
25. 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.
26. Geceelter G, Fahoum B, Gardezi S, Schein M. Abdominal compartment syndrome in severe acute pancreatitis: an indication for a decompressing laparotomy? *Dig Surg*. 2002;19(5):402-405.
27. Balogh Z, McKinley BA, Holcomb JB, et al. Both primary and secondary abdominal compartment syndrome can be predicted early and are harbingers of multiple organ failure. *J Trauma*. 2003;54(5):848-861.
28. Oda S, Hirasawa H, Shiga H, et al. Management of intra-abdominal hypertension in patients with severe acute pancreatitis with continuous hemodiafiltration using a polymethyl methacrylate membrane hemofilter. *Ther Apher Dial*. 2005;9(4):355-361.
29. Marshall JC. Inflammation, coagulopathy, and the pathogenesis of multiple organ dysfunction syndrome. *Crit Care Med*. 2001;29(7)(suppl):S99-S106.
30. Rezende-Neto JB, Moore EE, Melo de Andrade MV, et al. Systemic inflammatory response secondary to abdominal compartment syndrome: stage for multiple organ failure. *J Trauma*. 2002;53(6):1121-1128.
31. Rezende-Neto JB, Moore EE, Masuno T, et al. The abdominal compartment syndrome as a second insult during systemic neutrophil priming provokes multiple organ injury. *Shock*. 2003;20(4):303-308.
32. Dalfino L, Tullo L, Donadio I, Malcangi V, Brienza N. Intra-abdominal hypertension and acute renal failure in critically ill patients. *Intensive Care Med*. 2008;34(4):707-713.
33. Goverman J, Yelon JA, Platz JJ, Singson RC, Turcinovic M. The "fistula VAC," a technique for management of enterocutaneous fistulae arising within the open abdomen: report of 5 cases. *J Trauma*. 2006;60(2):428-431.
34. Connolly PT, Teubner A, Lees NP, Anderson ID, Scott NA, Carlson GL. Outcome of reconstructive surgery for intestinal fistula in the open abdomen. *Ann Surg*. 2008;247(3):440-444.
35. Hultman CS, Pratt B, Cairns BA, et al. Multidisciplinary approach to abdominal wall reconstruction after decompressive laparotomy for abdominal compartment syndrome. *Ann Plast Surg*. 2005;54(3):269-275.
36. Perez D, Wildi S, Demartines N, Bramkamp M, Koehler C, Clavien PA. Prospective evaluation of vacuum-assisted closure in abdominal compartment syndrome and severe abdominal sepsis. *J Am Coll Surg*. 2007;205(4):586-592.
37. Petersson U, Acosta S, Björck M. Vacuum-assisted wound closure and mesh-mediated fascial traction: a novel technique for late closure of the open abdomen. *World J Surg*. 2007;31(11):2133-2137.