

Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: A multiple-center epidemiological study*

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LEARNING OBJECTIVES

On completion of this article, the reader should be able to:

1. Define intraabdominal hypertension.
2. Describe intraabdominal compartment syndrome.
3. Use this information in a clinical environment.

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Objective: Intraabdominal hypertension is associated with significant morbidity and mortality in surgical and trauma patients. The aim of this study was to assess, in a mixed population of critically ill patients, whether intraabdominal pressure at admission was an independent predictor for mortality and to evaluate the effects of intraabdominal hypertension on organ functions.

Design: Multiple-center, prospective epidemiologic study.

Setting: Fourteen intensive care units in six countries.

Patients: A total of 265 consecutive patients admitted for >24 hrs during the 4-wk study period.

Interventions: None.

Measurements and Main Results: Intraabdominal pressure was measured twice daily via the bladder. Data recorded on admission were the patient demographics with Simplified Acute Physiology Score II, Acute Physiology and Chronic Health Evaluation II score, and type of admission; during intensive care stay, Sepsis-Related Organ Failure Assessment score and intraabdominal pressure were measured daily together with fluid balance. Nonsurvivors had a significantly higher mean intraabdominal pressure on admission than survivors: 11.4 ± 4.8 vs. 9.5 ± 4.8 mm Hg. Independent predictors for mortality were age (odds ratio, 1.04; 95% confidence interval, 1.01–1.06; $p = .003$), Acute Phys-

iology and Chronic Health Evaluation II score (odds ratio, 1.1; 95% confidence interval, 1.05–1.15; $p < .0001$), type of intensive care unit admission (odds ratio, 2.5 medical vs. surgical; 95% confidence interval, 1.24–5.16; $p = .01$), and the presence of liver dysfunction (odds ratio, 2.5; 95% confidence interval, 1.06–5.8; $p = .04$). The occurrence of intraabdominal hypertension during the intensive care unit stay was also an independent predictor of mortality (relative risk, 1.85; 95% confidence interval, 1.12–3.06; $p = .01$). Patients with intraabdominal hypertension at admission had significantly higher Sepsis-Related Organ Failure Assessment scores during the intensive care unit stay than patients without intraabdominal hypertension.

Conclusions: Intraabdominal hypertension on admission was associated with severe organ dysfunction during the intensive care unit stay. The mean intraabdominal pressure on admission was not an independent risk factor for mortality; however, the occurrence of intraabdominal hypertension during the intensive care unit stay was an independent outcome predictor. (*Crit Care Med* 2005; 33:315–322)

KEY WORDS: intraabdominal pressure; intraabdominal hypertension; abdominal compartment syndrome; surgery; trauma; critically ill patients; intensive care

*See also p. 147.

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Intraabdominal pressure (IAP) is the pressure generated inside the abdominal cavity and is influenced by body weight (1–5), body position (6), and abdominal muscle activity (7).

A transient increase in IAP, such as with pneumoperitoneum during laparoscopic surgery, causes only minimal adverse effects (8, 9). Several clinical conditions such as blood or ascites accumulation in the abdominal cavity (10–13), bowel distension (14, 15), packing after damage control laparotomy, and closure of a swollen and noncompliant abdominal wall can cause a persistent increase in IAP (16, 17). Experimental data in animals and short-term physiologic studies in a relatively small number of patients reported that a persistent increase in IAP causes impairment of respiratory, hemodynamic, liver, and renal functions (16, 18–25).

The presence of intraabdominal hypertension (IAH) is variable, ranging from 18% to 81% of the patients depending on the threshold used to define it (12, 15, 18, 20, 25 mm Hg) and on the type of population studied such as in trauma, surgical, or medical patients (19, 26–28).

Several clinical scores not including IAP measurements have been proposed as independent predictors of mortality in critically ill patients (29, 30). However, in liver transplant recipients an IAP >25 mm Hg and in trauma patients an IAP >25 mm Hg with at least one organ failure (i.e., defined as the abdominal compartment syndrome, ACS) were associated with a higher rate of multiple organ failure and mortality (31–34).

Cheatham et al. (36) showed that abdominal perfusion pressure (APP) (i.e., the mean arterial blood pressure minus the IAP) was a better resuscitation end point compared with the only single measurement of the arterial blood pressure (35).

The aims of this study were a) to establish the cumulative incidence of IAH and ACS in a large mixed population of critically ill patients by measuring IAP during intensive care stay; b) to assess whether the mean IAP at admission or the occurrence of IAH during intensive care stay was an independent predictor for mortality; and c) to evaluate possible independent predictors for IAH development at intensive care admission.

METHODS

All new consecutive patients admitted in 14 intensive care units (ICUs) who stayed >24

hrs, from six countries (Belgium, Australia, Austria, Brazil, Israel, and Italy) during a 4-wk period (from January 1, 2001, to January 31, 2001) were prospectively enrolled. For an ICU to be included in the study, it had to have six or more beds and the physicians had to have experience in measuring IAP. General and specialized ICUs for adults were included. Patients were excluded from the study if they had contraindications for intravesical pressure measurement (pelvic fracture, hematuria, neurogenic bladder).

The study was conducted in accordance with the study protocol, the Declaration of Helsinki, and applicable regulatory requirements. The institutional review board and the local institutional ethics committee of each participating center approved the protocol before data collection.

Data Collection

The following data were collected for each patient admitted to intensive care during the 1-month study period.

Demographic Data. Age, gender, weight, height, date of enrollment, and diagnostic category for ICU admission (medical or surgical) were recorded. Patients were followed until death, until hospital discharge, or for a maximum of 28 days, whichever came first, irrespective of whether they remained in the ICU for the entire observation period or were discharged from the ICU to another department within the same hospital. Patients discharged during the study to an ICU or ward of another hospital were not followed after this transfer, but if possible 28-day outcome was recorded.

Measurement of Organ Dysfunction. The worst Acute Physiology and Chronic Health Evaluation (APACHE) II score and Simplified Acute Physiology Score (SAPS) II during the first 24 hrs of intensive care stay were recorded (29, 30).

The Sepsis-Related Organ Failure Assessment (SOFA) score was computed daily and for the entire duration of the intensive care stay (35). For each patient, the worst value for each organ system (respiratory, cardiovascular, renal, coagulation, liver, and neurologic) in each 24-hr period was considered.

Fluid Balance. The daily fluid balance was calculated by subtracting the output from the intake. Insensible losses were calculated with the formula of Dubois: 550 mL/body surface area, where body surface area = $71.84 \times (\text{body weight in kg})^{0.425} \times (\text{height in cm})^{0.725}$. In case of mechanical ventilation or active humidification, this value was divided by 2. Temperature corrections were made (for each 1°C increase of temperature above 37°C, a 13% increase in insensible losses) (37).

Clinical Concomitant Factors and Conditions. Clinical concomitant factors and conditions potentially associated with increased IAP at intensive care admission were recorded for each patient.

We defined the following as clinical concomitant factors: a) abdominal surgery (with or without laparoscopy, reduction of hernia, tight closure, or abdominal banding with postoperative Velcro belt to prevent incisional hernia); b) hemoperitoneum caused by either intra- or retroperitoneal bleeding; c) abdominal infection (pancreatitis, peritonitis, abscess, etc.); d) massive fluid resuscitation arbitrarily defined as >3.5 L of colloids or crystalloids in the 24 hrs before the study; and e) ileus, whether paralytic, mechanical, or pseudo-obstructive defined as abdominal distention or absence of bowel sounds or failure of enteral feeding evidenced by gastric dilation or gastroparesis with a gastric residual >1000 mL in the 24 hrs before the study.

We defined the following as concomitant conditions: a) acidosis defined as an arterial pH <7.2; b) hypothermia defined as a core temperature <33°C (38–40); c) polytransfusion defined as the transfusion of >6 units of packed red cells in the 24 hrs before the study; d) coagulopathy defined as a platelet count <55,000/mm³ or an activated partial thromboplastin time more than two times normal or a prothrombin time <50% or an international standardized ratio >1.5; e) sepsis defined according to the American-European consensus conference definitions (41); and f) liver dysfunction (defined as decompensated or compensated cirrhosis or other liver failure with ascites (paraneoplastic, cardiac failure, portal vein thrombosis, ischemic hepatitis).

IAP

IAP was measured via a Foley bladder catheter, according to the modified Kron technique described by Cheatham and Safcsak (42) and others (43, 44). IAP was always measured in the complete supine position and in stable conditions twice daily (morning and evening) according to a standardized protocol.

Definitions. The mean IAP was the mean of the two daily measurements. APP was defined as the mean arterial pressure minus the IAP.

IAH was defined as a mean IAP >12 mm Hg (20, 26, 27), whereas ACS was defined as an IAP >20 mm Hg with at least one organ failure (19, 20, 26, 27, 45, 46).

Statistical Analysis. For statistical purposes only, the data obtained during the first week were used or less if discharge or death occurred before day 7.

Continuous variables are presented as the mean (\pm SD) or median in case of skewed distribution; categorical variables are expressed as numbers and percentages for the group from which they were derived. Continuous variables were compared with the Student's *t*-test for nor-

mally distributed variables and the Mann-Whitney test for nonnormally distributed variables. The chi-square test or Fisher's exact test was used to compare ordinal variables.

The prognostic relevance of the recorded variables in the prediction of 28-day survival was analyzed by backward multiple logistic regression models. In the multiple regression logistic model have been included all the variables statistically significant at the univariate analysis and the variables with a *p* value at a borderline significance value (*p* < .10). Odds ratios (ORs) are given with 95% confidence intervals (CIs). The prognostic role of the occurrence of IAH on mortality during ICU stay was assessed by means of Cox's proportional hazard model for time-dependent variables.

All *p* values were two-tailed, and *p* < .05 was considered statistically significant. Statistical analysis was done with SAS (version 8.2, SAS Institute, Cary, NC) and SPSS (Windows version 10.0, SPSS, Chicago, IL).

RESULTS

Cumulative Incidence of Intraabdominal Hypertension, Mortality Rate, and Prognostic Factors

We enrolled 265 consecutive patients fulfilling inclusion/exclusion criteria, and there were 31 missing data (1.2% of the expected) of IAP measurements. None of patients admitted to the ICUs was missed. Mean IAP was 10 ± 4.8 mm Hg for all measurements. Figure 1 shows the distribution of mean IAP at admission. On admission, 140 patients (67.9%) had a normal IAP (<12 mm Hg), 85 (32.1%) had IAH >12 mm Hg, and 11 (4.2%) had ACS. The prevalence of ACS in patients with IAH was 12.9%. Two patients had an IAP >20 mm Hg without any organ failure. Only one patient with ACS underwent decompressive laparotomy.

Seventy-three patients (27.5%) died; 14 patients of 28 discharged to the ward died within 28 days. Table 1 shows the admission characteristics of the patients subdivided as survivors and nonsurvivors. Nonsurvivors were significantly older and had significantly higher mean IAP, SAPS II, APACHE II, and SOFA score at admission. They had more medical admissions and a higher rate of ileus, acidosis, coagulopathy, sepsis, and liver dysfunction (Table 2).

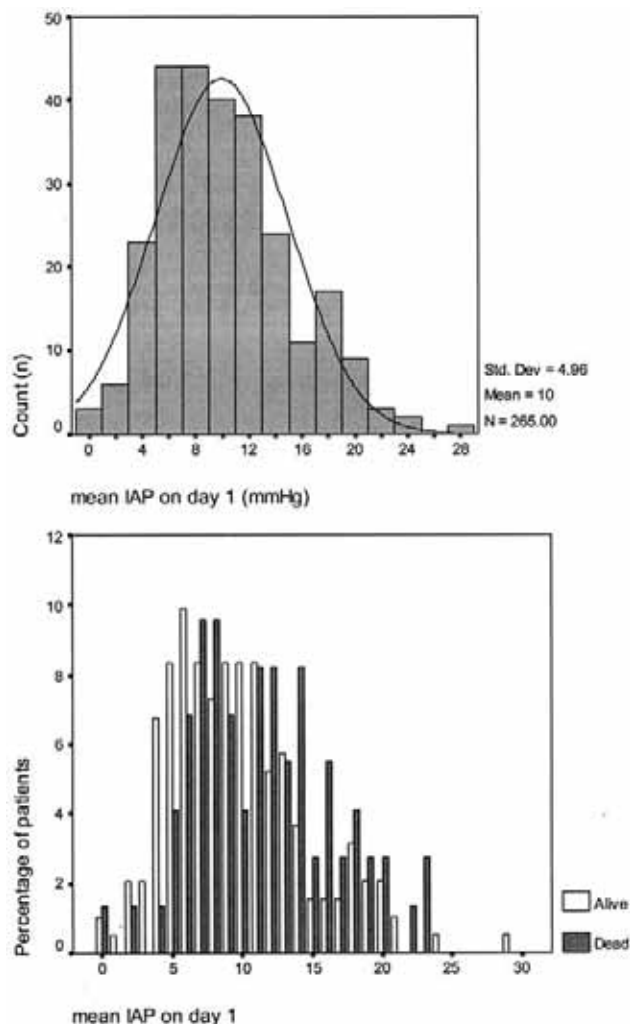


Figure 1. Top, distribution of mean intraabdominal pressure (IAP) on admission. Bottom, distribution of mean IAP split according to outcome at 28 days.

From a logistic regression model, the only independent predictors for mortality were age (OR, 1.04 for each year increase; 95% CI, 1.01–1.06; *p* = .003), APACHE II score (OR, 1.1 for each unity increase; 95% CI, 1.05–1.15; *p* < .0001), type of ICU admission (OR, 2.5 medical vs. surgical; 95% CI, 1.24–5.16; *p* = .01), and the presence of liver dysfunction (OR, 2.5; 95% CI, 1.06–5.8; *p* = .04). Mean IAP ≥ 12 mm Hg at day 1 was at borderline statistical significance value (OR, 1.8; 95% CI, 0.88–3.86; *p* > .01). In a subgroup of patients (*n* = 235), APP was an independent predictor for outcome (OR, 1.03; 95% CI, 1.01–1.06; *p* = .003).

During ICU stay except for day 5, nonsurvivors had a significantly higher IAP (Fig. 2) and SOFA compared with survivors. The occurrence of IAH during the ICU stay was an independent predictor of mortality (relative risk, 1.85; 95% CI,

1.12–3.06; *p* = .01) in addition to the type of admission and liver disease. IAH remained associated with mortality after stratification in quartiles of admission APACHE II scores (Fig. 3).

IAH at Day 1 and Clinical Concomitant Factors—Conditions

At admission, 85 patients (32.1%) had IAH. The mean IAP in patients with IAH was 15.8 ± 3.4 vs. 7.2 ± 2.7 mm Hg compared with patients without IAH. The mortality rate was significantly higher in the group with IAH group compared with the non-IAH group (38.8% vs. 22.2%, *p* = .005, Fig. 4).

Considering the clinical concomitant factors and conditions, patients with IAH had a higher rate of abdominal surgery, hemoperitoneum, fluid resuscitation, il-

Table 1. Patient characteristics at intensive care admission

	Total Group	Survivors	Nonsurvivors	<i>p</i> Value
No. of patients	265	192	73	
Age, yrs	61.5 ± 18.1	59.4 ± 19.3	67.1 ± 12.8	.002
Male gender, n (%)	165 (62.3)	123 (64.0)	42 (57.5)	NS
BMI, kg/cm ²	25.7 ± 5.2	25.4 ± 4.9	25.4 ± 6.0	NS
APACHE II	17.4 ± 8.3	15.3 ± 7.2	23.0 ± 8.5	<.0001
SAPS II	39.1 ± 17.2	34.5 ± 14.9	50.9 ± 17.1	<.0001
SOFA day 1	6.2 ± 3.7	5.3 ± 3.4	8.5 ± 3.6	<.0001
Organ failures day 1	1.1 ± 1	0.8 ± 0.8	1.8 ± 1.1	<.0001
ICU stay, days	10.3 ± 12	10.8 ± 13.5	8.8 ± 6.5	NS
ICU stay, median (range)	5 (1–77)	5 (1–77)	7 (1–28)	
IAP mean day 1	9.9 ± 4.9	9.5 ± 4.8	11.4 ± 4.8	.002
IAP median (range)	9.0 (0–28.5)	8.5 (0–28.5)	11.0 (1.5–23)	
IAP minimal day 1	9.4 ± 5	8.9 ± 4.8	10.8 ± 5	.005
IAP maximal day 1	10.5 ± 5.2	10 ± 5.1	11.8 ± 5.1	.01
Medical, %	124 (46.8)	74 (38.5)	50 (68.5)	<.0001
Surgical, %	141 (53.2)	118 (61.5)	23 (31.5)	
Elective, %	74 (27.9)	69 (35.9)	5 (6.8)	
Emergency, %	44 (16.6)	28 (14.6)	16 (21.9)	
Trauma, %	23 (8.7)	21 (10.9)	2 (2.7)	

NS, not significant; BMI, body mass index; APACHE, Acute Physiology and Chronic Health Evaluation; SAPS, Simplified Acute Physiology score; SOFA, Sepsis-Related Organ Failure Assessment; ICU, intensive care unit; IAP, intraabdominal pressure, expressed in mm Hg.

Table 2. Cumulative incidence of clinical etiologic factors and predisposing conditions for intra-abdominal hypertension in survivors and nonsurvivors

	Total Group n = 265	Survivors n = 192	Nonsurvivors n = 73	<i>p</i> Value
Etiologic factors				
Abdominal surgery	80 (30.2)	63 (32.8)	17 (23.3)	NS
Hemoperitoneum	14 (5.2)	12 (6.2)	2 (2.8)	NS
Abdominal infection	36 (13.5)	22 (11.5)	14 (19.2)	NS
Fluid resuscitation	123 (46.4)	83 (43.2)	40 (54.8)	.09
Ileus	113 (42.6)	75 (39.0)	38 (52.0)	.056
>1	117 (44.2)	86 (44.8)	31 (42.5)	NS
Predisposing conditions				
Acidosis	43 (6.2)	24 (12.5)	19 (26.0)	.008
Hypothermia	28 (10.6)	16 (8.3)	12 (16.4)	.055
Polytransfusion	47 (17.7)	31 (16.1)	16 (21.9)	NS
Coagulopathy	74 (27.9)	46 (24.0)	28 (38.4)	.02
Sepsis	87 (32.8)	51 (26.5)	36 (49.3)	<.0001
Liver dysfunction	47 (17.7)	23 (12.0)	24 (32.9)	<.0001
>1	99 (37.4)	57 (29.7)	42 (57.5)	<.0001

NS, not significant.
Values are n (%).

eous, acidosis, polytransfusion, coagulopathy, sepsis, and liver dysfunction compared with patients without IAH (Table 3).

Independent predictors for IAH were liver dysfunction (OR, 2.25; 95% CI, 1.1–4.58; *p* = .03), abdominal surgery (OR, 1.96; 95% CI, 1.05–3.64; *p* = .03), fluid resuscitation (OR, 1.88; 95% CI, 1.04–3.42; *p* = .04), and ileus (OR, 2.07; 95% CI, 1.15–3.72; *p* = .02).

During the ICU stay, the mean total SOFA score was significantly higher in patients with IAH on admission compared with patients without IAH (Fig. 5). Patients with IAH on admission had a significantly higher respiratory, liver, coagulation, and renal SOFA organ sub-

scores compared with patients without IAH during the following days.

We did not find any significant correlation between the daily changes of IAP and the daily changes of total or partitioned SOFA scores during intensive care stay.

DISCUSSION

We found that in a general population of critically ill patients, a) IAH at intensive care admission was not an independent risk factor for mortality, whereas the occurrence of IAH during intensive care stay was an independent predictor for mortality; b) independent predictors for

IAH development at admission were abdominal surgery, fluid resuscitation, ileus, and liver dysfunction; and c) patients with IAH on admission had a higher SOFA score during the following days of ICU stay.

Prognostic Implications

Several organ dysfunction scores such as APACHE II (29), SAPS II (30), and the multiple organ dysfunction score (47) using measurements taken during the first 24 hrs of ICU stay have been developed to predict outcome in critically ill patients. However, none of them includes the measurement of IAP. Previous studies in sur-

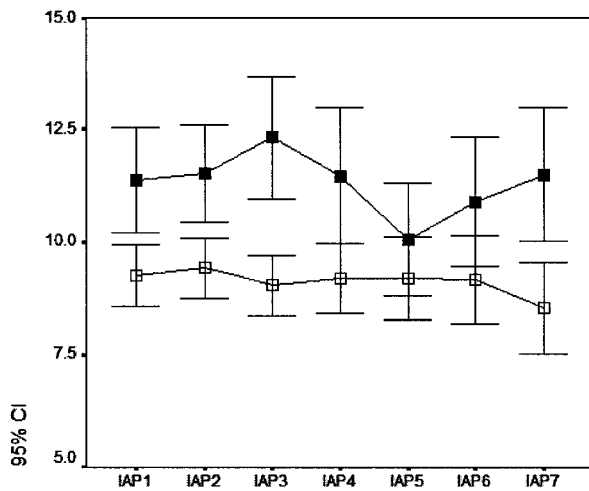


Figure 2. Evolution of mean intraabdominal pressure (IAP) during the first week of intensive care unit stay in survivors (open squares) and nonsurvivors (filled squares), $p < .05$ vs. survivors except on day 5. CI, confidence interval.

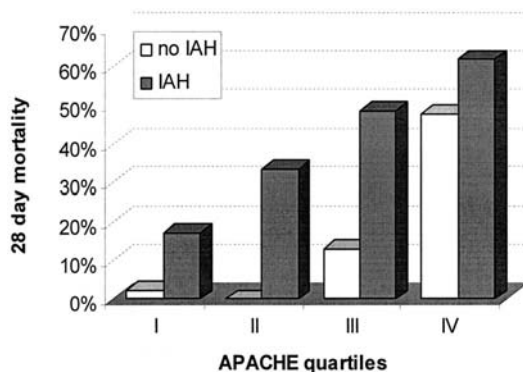


Figure 3. Relative mortality according to admission Acute Physiology and Chronic Health Evaluation (APACHE) II quartiles, subdivided according to maximal intraabdominal pressure (IAP) value (gray bars, patients with $IAP \geq 12$ mm Hg; white bars, patients with normal IAP). Odds ratios for patients with vs. patients without intraabdominal hypertension (IAH) were 9.5 (95% confidence interval [CI], 1.1–83.1; $p = .016$) for the first quartile (APACHE II 0–12), 1.5 (95% CI, 1.2–1.9; $p = .001$) for the second quartile (APACHE II 12–16), 6.3 (95% CI, 1.8–22.4; $p = .002$) for the third quartile (APACHE II 16–22), and 1.8 (95% CI, 0.6–5.2; $p =$ nonsignificant) for the fourth quartile (APACHE II >22). $p < .05$ for all comparisons.

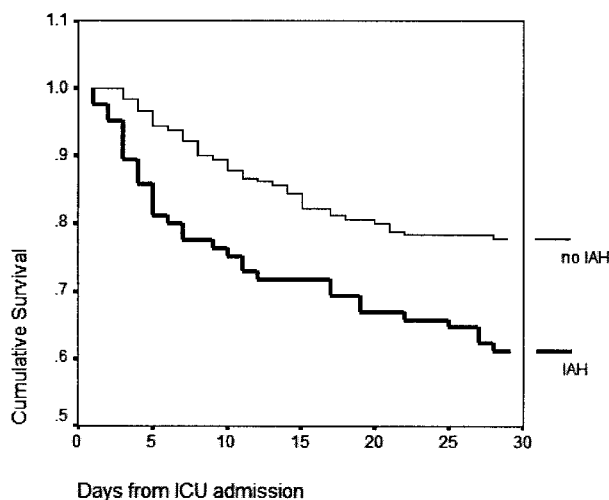


Figure 4. Kaplan Meier cumulative survival curve split according to patients with or without intraabdominal hypertension (IAH). ICU, intensive care unit.

gical and trauma patients found that an increase in IAP was associated with an increase in mortality (45, 48–50). In the present large mixed population of critically ill patients, IAH at day 1 was not an independent risk factor for mortality, whereas the occurrence of IAH during intensive care stay was an independent predictor for mortality. This is in agreement with Balogh et al. (34, 51) (torso trauma patients) and Biancofiore et al. (32, 33) (liver transplant patients), who found that increased IAP, based on at least one or two consecutive measurements within the first 3 days, was a significant predictor for mortality. However, these authors investigated surgical patients with extremely high IAP, consistent with ACS.

Etiological Factors and Associated Conditions

We found that among the clinical associated factors and conditions, abdominal surgery, fluid resuscitation, ileus, and liver dysfunction were independent predictors for IAH. Conversely, we did not find any difference regarding the diagnostic category for intensive care admission (medical or surgical) and the presence of IAH. Massive fluid resuscitation may indicate an inflammatory process with an alteration in vascular permeability that triggers a vicious cycle of fluid sequestration, cardiovascular failure, splanchnic hypoperfusion, bowel edema, and increase in the IAP. Liver dysfunction may lead to the formation of ascites and increase the risk of bleeding because of coagulopathy, thus affecting the IAP (10).

Ileus with intestinal dilation, increased luminal pressure, and gut wall ischemia can easily increase the IAP by a direct mechanical effect (52, 53). In a previous study of critically ill patients with similar characteristics, we found that the only independent risk factor for IAH development was the body mass index, although the amount of fluid resuscitation was close to the limit of statistical significance (27). However, that study had fewer patients than the current one (97 vs. 265, respectively) and IAP was measured not on admission and during the following days but only during one “snapshot” day of the intensive care stay. In trauma and surgical patients, the 24-hr net fluid balance (50, 54) and the crystalloid and red blood cell infusion rate (55) were found to be risk factors for the development of abdominal compart-

Table 3. Incidence of clinical etiologic factors and predisposing conditions for intraabdominal hypertension (IAH) in patients with and without intraabdominal hypertension

	No IAH n = 180	IAH n = 85	p Value
Etiologic factors			
Abdominal surgery	42 (23.3)	38 (44.7)	<.0001
Hemoperitoneum	6 (3.3)	8 (9.4)	.04
Abdominal infection	23 (12.8)	13 (15.3)	NS
Fluid resuscitation	68 (37.8)	55 (64.7)	<.0001
Ileus	61 (33.9)	52 (61.2)	<.0001
>1	64 (35.6)	53 (62.4)	<.0001
Predisposing conditions			
Acidosis	23 (12.8)	20 (23.5)	.03
Hypothermia	16 (8.9)	12 (14.1)	NS
Polytransfusion	25 (13.9)	22 (25.9)	.02
Coagulopathy	43 (23.9)	31 (36.5)	.03
Sepsis	52 (28.9)	35 (41.2)	.05
Liver dysfunction	22 (12.2)	25 (29.4)	.001
>1	59 (32.8)	40 (47.1)	.03

NS, not significant.
Values are n (%).

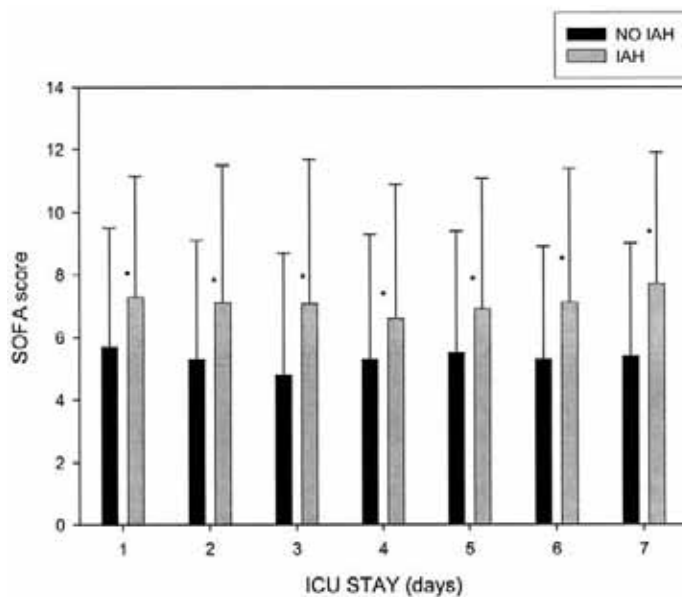


Figure 5. Evolution of mean total Sepsis-Related Organ Failure Assessment (SOFA) score during the first week of intensive care unit (ICU) stay in patients with and without intraabdominal hypertension (IAH). * $p < .05$ vs. no IAH.

ment syndrome. Similarly, in the present study, >60% of patients with IAH (43% medical, 23% elective surgery, 27% emergency surgery, 7% trauma) received massive fluid resuscitation.

IAP

The abdominal cavity can be considered as a semiclosed compartment, so any changes in its content may affect the IAP (18, 19, 26, 36). When a critical volume is reached, the compliance of the abdomen wall abruptly drops, causing a progressive increase in IAP (18).

An abnormal IAP increase can induce moderate to severe organ failure, mainly through a direct mechanical effect and, if untreated, multiple organ failure (46). Several studies showed that marked increase in IAP above 20 mm Hg negatively affected respiratory, cardiovascular, splanchnic, neurologic, and renal function (21, 31–34, 48, 49, 51, 56, 57). We found that patients with IAH on admission had a greater impairment of pulmonary, renal, coagulation, and liver function, evaluated with the daily SOFA score (58) during intensive care stay (Fig. 5).

However, we used a threshold of 12 mm Hg for the definition of IAH, lower than that previously reported. This suggests that even relatively low values of IAP can markedly affect organ function. This was already suggested in an animal model by Diebel et al. (59, 60), who found that starting from an IAP of 10 mm Hg, the arterial and microcirculatory hepatic blood flow was decreased. Others also found a decrease in renal blood flow (61) and submucosal tissue oxygenation (62).

In addition, the risk of organ dysfunction is related to the magnitude of the IAP and the APP: The higher the IAP or the lower the APP, the higher the risk for organ failure. Kron et al. (63) showed that the higher the IAP, the lower the urine output. Meldrum et al. (46) found that 40% of abdominal trauma patients with IAP between 15 and 25 mm Hg had pulmonary failure and 20% had cardiovascular failure, whereas all patients with an IAP >35 mm Hg presented simultaneous pulmonary, cardiovascular, and renal dysfunction. Decompressive laparotomy in selected trauma/surgical patients was able to decrease the number of organ failures (16, 25, 46, 48, 64).

Limitations and Clinical Consequences

Although this study does not prove whether an increase of IAP is a phenomenon or epiphenomenon (i.e., directly or indirectly related to mortality), the presence of IAH should be considered in the presence of renal or hepatic failure in patients with multiple organ failure (20, 27, 57).

Due to the possible impairment of every organ system and the high prevalence and incidence of IAH either in generally critically ill patients (10–40% of the patients) or in surgical patients (30–80% of the patients), we suggest that clinicians measure the IAP in all patients in the first days after intensive care admission. There are no conclusive data on the frequency of IAP monitoring, but to adapt treatment appropriately, and in analogy with the brain, continuous monitoring of IAP and APP could be a good option in selected patients (27, 65). Finally, we suggest that clinicians continue IAP measurement in the case of clinical deterioration. The impact of IAP in our therapeutic decision making is still not clear; however, the presence of IAH should be considered as an “organ dysfunction” for which specific diagnostic

The cumulative incidence of intraabdominal hypertension (defined as an intraabdominal pressure >12 mm Hg) in a mixed population of critically ill patients was high, and the occurrence of intraabdominal hypertension during intensive care stay was an independent risk factor for organ failure and mortality.

and therapeutic (medical or surgical) interventions are needed.

CONCLUSIONS

The cumulative incidence of IAH (defined as an IAP >12 mm Hg) in a mixed population of critically ill patients was high, and the occurrence of IAH during intensive care stay was an independent risk factor for organ failure and mortality.

APPENDIX

List of Participating Centers and Patients Enrolled Per Country

Belgium (80)—Manu Malbrain (Europe Hospitals, campus Ste-Elisabeth, Brussels and ZiekenhuisNetwerk Antwerpen, campus Stuivenberg, Antwerpen), Yves Debaveye (Europe Hospitals, campus Ste-Elisabeth, Brussels), Didier Delmarcelle (Europe Hospitals, campus Ste-Elisabeth, Brussels), Alexander Wilmer (University Hospital UZ Gasthuisberg, Leuven), Luc Jacquet (University Hospital St-Luc, Brussels), Pierre-François Larterre (University Hospital St-Luc, Brussels), Bart De Keulenaere (ZiekenhuisNetwerk Antwerpen, campus Stuivenberg, Antwerpen), Ronny Daelemans (ZiekenhuisNetwerk Antwerpen, campus Stuivenberg,

Antwerpen), Dirk Denie (ZiekenhuisNetwerk Antwerpen, campus Stuivenberg, Antwerpen), Anita Jans (ZiekenhuisNetwerk Antwerpen, campus Stuivenberg, Antwerpen); **Italy** (82)—Monica Del Turco (University of Pisa, S. Chiara Hospital, Pisa), P. Cosimini (University of Pisa, S. Chiara Hospital, Pisa), Marco Ranieri (University of Pisa, S. Chiara Hospital, Pisa), F. Giunta (University of Pisa, S. Chiara Hospital, Pisa), Paolo Pelosi (Ospedale di Circolo, Varese), Davide Chiumello (Ospedale Maggiore Policlinico, Milano), Luciano Gattinoni (Ospedale Maggiore Policlinico, Milano), Nicola Brienza (Policlinico University of Bari, Bari), Vincenzo Malcangi (Policlinico University of Bari, Bari); **Israel** (20)—Jonathan Cohen (Rabin Medical Centre, Petah Tikva), Ora Ben Shimon (Rabin Medical Centre, Petah Tikva), Pierre Singer (Rabin Medical Centre, Petah Tikva); **Austria** (12)—Guenther Frank (Hospital Wiener-Neustadt, Wiener-Neustadt), Helmut Trimmel (Hospital Wiener-Neustadt, Wiener-Neustadt); **Brazil** (28)—Andre Japiassu (Hospital Universitario Clementino, Federal University, Rio de Janeiro), Fernando Bozza (Federal University of Rio de Janeiro, Rio de Janeiro), Paulo César P Souza (Hospital de Clinicas Niteroi, Rio de Janeiro, Rio de Janeiro); **Australia** (33)—David Bihari (Prince of Wales Hospital, Sydney), Richard Innes (Prince of Wales Hospital, Sydney), Elizabeth Kurttop (Prince of Wales Hospital, Sydney)

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