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Mission to Eliminate Postinjury Abdominal Compartment Syndrome

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Objectives: To determine the current incidence of postinjury abdominal compartment syndrome (ACS), the effect of intra-abdominal hypertension (IAH) on trauma outcomes, and the independent predictors of postinjury IAH.

Design: Prospective cohort study.

Setting: University-affiliated level 1 trauma center.

Patients: Eighty-one consecutive shock/trauma patients admitted to the intensive care unit (mean [SD] values: age, 41 [2] years; 70% male; injury severity score, 29 [1]; base deficit, 6 [0.5] mmol/L; lactate level, 29.73 [4.5] mg/dL; transfusions of packed red blood cells, 5 [0.5] U in first 24 hours; mortality rate, 2.5%; and multiple organ failure [MOF], 6%) had second hourly intra-abdominal pressure (IAP) monitoring.

Main Outcome Measures: Intensive care unit length of stay, ACS, IAH, MOF, mortality.

Results: The mean (SD) IAP was 14 (1) mm Hg. No patients developed ACS. Sixty-one patients (75%) had sustained IAH. Both patients with IAH and those without had similar demographics and injury severity. Patients with IAH had worse metabolic acidosis ($P=.02$), re-

ceived more crystalloids ($P=.03$), and underwent laparotomy more frequently ($P=.005$). One patient with IAH and one without died. MOF occurred in 1 patient without IAH (5%) vs 4 with IAH (7%). The mean (SD) intensive care unit length of stay was 11 (3) days in patients without IAH vs 8 (1) days in those with IAH. Intra-abdominal hypertension was poorly predictive of MOF (odds ratio, 1.17; 95% confidence interval, 0.96-1.43; $P=.13$). Of the 30 variables in multiple logistic regression analysis, only base deficit, laparotomy, and emergency department crystalloids were identified as weak predictors of IAP greater than 12 mm Hg. No predictors were found for the clinically more relevant IAP greater than 15 mm Hg and IAP greater than 18 mm Hg.

Conclusions: Most of the severe shock/trauma patients developed sustained IAH. Based on univariate and multivariate analyses, there was no difference in outcomes between the trauma patients with IAH and those without. Multiple logistic regression analysis failed to show IAH as a predictor of MOF. The attenuation of the deadly ACS to a less deleterious IAH could be considered a success of the last decade in trauma and critical care.

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POSTINJURY ABDOMINAL COMPARTMENT SYNDROME (ACS) was originally described as severe abdominal distension with raised peak airway pressures, carbon dioxide retention, and oliguria culminating in unplanned re-exploration following damage-control¹⁻⁶

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laparotomy. The seminal publications on ACS reported high mortality and incidence of multiple organ failure (MOF), with considerable intensive care re-

source use.⁷⁻¹³ Owing to recent advances in trauma and critical care (hemostatic resuscitation, open abdomen strategy, and advanced wound management options), the incidence and mortality rate of postinjury ACS is declining.¹⁴⁻¹⁶ Abdominal compartment syndrome in nontrauma populations (medical, mixed intensive care unit [ICU] populations) has always been less frequent but epidemiological studies of these populations have included intra-abdominal hypertension (IAH) as a predictor of poor outcome.^{17,18} The clinical relevance of sub-ACS IAH (elevated intra-abdominal pressure without organ dysfunction) in the shock/trauma population is largely unknown. We hypothesized

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that postinjury IAH is associated with poor outcome and is a predictor of MOF and death. The specific aims of this study were to determine (1) the incidence of IAH and ACS in the same high-risk population in which the predictors were originally described; (2) the effects of IAH on postinjury outcomes; and (3) the potential independent predictors of postinjury IAH.

METHODS

A prospective observational study was performed at the John Hunter Hospital (University of Newcastle affiliated level I trauma center), New South Wales, Australia. The John Hunter Hospital is the busiest trauma center in the state of New South Wales, with 4500 trauma admissions annually, including more than 400 patients with an Injury Severity Score (ISS) greater than 15. Second hourly intra-abdominal pressure (IAP) measurements via Foley manometry are standard procedure for all major trauma patients admitted to the John Hunter Hospital 16-bed ICU.^{19,20} The study protocol was approved by the John Hunter Hospital institutional review board and the need for informed consent waived, as no intervention in addition to standard treatment was required owing to the observational nature of the study.

Consecutive severe shock/trauma patients who met the inclusion criteria (>18 years; ISS > 15; and pre-ICU transfusion requirement or emergency department base deficit >6 mmol/L; no severe head injury (AIS < 3), pregnancy, or bladder injury) were included during the 24-month study period that ended February 28, 2009. Isolated head injuries were also excluded. These inclusion and exclusion criteria were selected to be comparable with our previous studies describing the epidemiology and modifiable predictors of postinjury ACS.^{11,21-23}

PROSPECTIVE DATA COLLECTION

Demographics (age and sex), mechanism of injury (blunt or penetrating), ISS, established independent predictors of ACS (resuscitation fluids in ED [crystalloid and blood]), resuscitation fluids until ICU admission, initial urine output on ICU admission, systolic blood pressure at admission, temperature on ICU admission, lactate level, base deficit, hemoglobin level, specific injuries (abdominal, pelvic fractures, retroperitoneal bleeding), and laparotomy (yes/no).

DEFINITION OF OUTCOMES

The primary outcomes (IAH, ACS) were defined according to the World Society of ACS criteria.^{24,25} Abdominal perfusion pressure (APP) was determined by mean arterial pressure minus IAP. Abdominal compartment syndrome was defined as a sustained IAP greater than 20 mm Hg (with or without an APP < 60 mm Hg) that is associated with new organ dysfunction or failure. It is important to note that, while we have previously defined ACS as only IAP greater than 25 mm Hg, these studies were published before the establishment of the World Society of ACS consensus definitions and, as such, the World Society of ACS definition is now the standard. Intra-abdominal hypertension levels were defined as grade 1, IAP 12 to 15 mm Hg; grade 2, IAP 16 to 20 mm Hg; grade 3, IAP 21 to 25 mm Hg; and grade 4, IAP greater than 25 mm Hg.

Secondary outcomes were mortality, ICU length of stay (ICU-LOS), and MOF (defined by the Denver MOF score, calculated according to the latest modification).²⁶

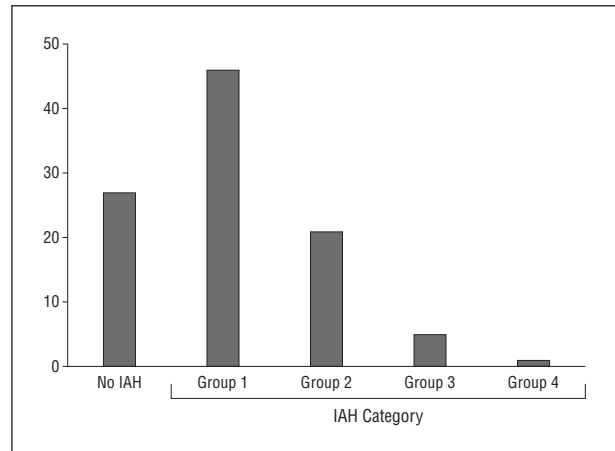


Figure. Incidence of different intra-abdominal hypertension (IAH) categories.

STATISTICAL ANALYSIS

From the physiological parameters, values until ICU admission were considered for the IAH prediction model. This was based on our previous research, which showed that both primary and secondary ACS developed within 12 hours after ICU admission in trauma patients. Physiological parameters required for outcome measures (such as MOF) were collected daily until ICU discharge or death. The IAP was recorded using both the highest reading (IAP_{max}) and average reading (IAP_{mean}). Cutoff values of greater than 12 mm Hg and greater than 15 mm Hg were used for univariate analysis (patients with vs without IAH, respectively). For multivariate analysis, a cutoff IAP value greater than 18 mm Hg was also considered. Abdominal perfusion pressure was recorded as the lowest (APP_{min}) and average (APP_{mean}). A cutoff APP value of less than 60 mm Hg was used as a definition of IAH for both univariate and multivariate analyses. Univariate analysis was performed using the *t* test for continuous variables and the Fisher exact test for noncategorical data. Multivariate analysis was performed by using multiple logistic regression analysis. Predictors were determined for MOF and ICU-LOS. Because ICU-LOS represents a continuous variable, univariate analyses of variables were performed, followed by a parsimonious multiple regression model. Data are presented as an absolute figure, percentage, or mean (standard error of mean); *P* < .05 was considered significant.

RESULTS

Eighty-one shock/trauma patients (mean [SD] values: age, 41 [2] years; 70% male; 91% blunt injury; ISS, 29 [1]; base deficit, 6 [0.5] mmol/L; lactate level, 29.73 [4.5] mg/dL (to convert to millimoles per liter, multiply by 0.111); transfusion, 5 [0.5] U per 24 hours; ICU-LOS, 9 [1] days; mortality rate, 2.5%; MOF, 6%) met the inclusion criteria. The mean (SD) IAP was 14 (1) mm Hg based on IAP measurements in the first 24 hours of all patients. No patients developed ACS. Fifty-nine patients (73%) were diagnosed with IAH based on an IAP_{mean} greater than 12 mm Hg; 70 (86%) based on IAP_{max} greater than 12 mm Hg; 13 (16%) based on APP_{mean} less than 60 mm Hg; and 48 (59%) based on APP_{min} less than 60 mm Hg. As depicted (Figure), most of the cohort developed IAH; however, grade III-IV IAH (IAP_{mean} > 20 mm Hg) was present in only 5 patients (6%).

Table 1. Univariate Comparison of Demographics, Injury Severity, Shock Severity, Resuscitation Fluid Requirements, and Outcomes of the Cohort

Characteristic	Mean (SE)		P Value
	No IAH (n=22)	IAH (n=59)	
Age, y	39 (7)	43 (2)	.22
Male sex, %	64	73	.86
ISS	27 (2)	30 (2)	.17
Laparotomy, No. (%)	3 (9)	28 (47)	.005
Open abdomen, No. (%)	1 (5)	12 (20)	.09
Retroperitoneal hematoma, No. (%)	10 (45)	20 (34)	>.99
Pelvic fracture, No. (%)	9 (41)	17 (29)	.42
Base deficit, mmol/L	4.1 (1)	6.8 (0.6)	.02
Lactate level, mg/dL	17.1 (1.8)	30.6 (3.6)	.02
Pre-ICU PRBC, U	5 (0.6)	4 (0.6)	.34
Pre-ICU crystalloids given, L	3 (0.3)	3.8 (0.2)	.03
First ICU hour urine output, mL	133 (21)	149 (15)	.56
First ICU temperature, °C	35.7 (0.3)	35.7 (0.15)	>.99
First ICU hemoglobin, g/dL	10.5 (0.4)	10.4 (0.2)	>.99
Time to ICU, min	267 (29)	327 (29)	.24
Mortality, No. (%)	1 (5)	1 (2)	.40
MOF, No. (%)	1 (5)	4 (7)	.59
ICU-LOS, d	11 (4)	8 (1)	.36

Abbreviations: IAH, intra-abdominal hemorrhage; ICU, intensive care unit; ISS, injury severity score; LOS, length of stay; MOF, multiple organ failure; PRBC, packed red blood cells.

SI conversion factors: To convert lactate level to millimoles per liter, multiply by 0.111; hemoglobin to grams per liter, 10.0.

The univariate comparison between patients with and without IAH did not show any demographic or injury severity differences (**Table 1**). Patients with IAH underwent laparotomy more frequently and had a worse initial base deficit and lactate levels. The only other marginal difference was found in mean (SD) pre-ICU crystalloid administration rates, which were 3.8 (0.2) L in patients with IAH and 3.0 (0.3) L in those without ($P=.03$). There was no difference in outcomes found between patients with and without IAH on univariate comparison. One patient with IAH and one without died. One patient from the non-IAH group developed MOF (5%) as well as 4 (7%) in the IAH group. The ICU-LOS did not reveal a statistically significant difference (mean [SD], 11 [3] days in patients without IAH vs 8 [1] days in those with). Results were similar comparing patients with and without IAH, when IAP greater than 15 mm Hg; IAP greater than 18 mm Hg; or APP less than 60 mm Hg were used for categorization (data presented only for the IAP > 12 mm Hg cutoff as definition of IAH in Table 1).

To determine potential predictors of intra-abdominal hypertension using 3 potential definitions (IAP > 12 mm Hg; IAP > 15 mm Hg; and IAP > 18 mm Hg), multiple logistic regression analysis was performed. The only predictors of IAH at IAP greater than 12 mm Hg were base deficit (odds ratio, 1.15; 95% confidence interval, 1.01-1.33; $P=.04$), postlaparotomy state (odds ratio, 5.72; 95% confidence interval, 1.5-21.43; $P=.01$), and pre-ICU crystalloid administration (odds ratio, 1.4; 95% confidence interval, 1.00-1.96; $P=.05$) were predictive. At the more clinically relevant IAPs of

greater than 15 mm Hg and IAP greater than 18 mm Hg, no predictors were found.

The logistic regression analysis aiming to identify potential predictors of MOF found age and ISS to be predictors. Neither IAP nor APP were significant predictors of MOF (**Table 2**). The low mortality rate in the cohort prevented further analysis of potential predictors of death.

The parsimonious multiple regression model for LOS identified ISS, first-hour ICU admission, urine output, and MOF as predictors. There were no associations between IAP and LOS or APP and LOS (**Table 3**).

COMMENT

One major finding of this prospective observational study on severely injured trauma patients who required shock resuscitation is that none of the 81 consecutive shock/trauma patients developed ACS. Just a decade earlier, the incidence of postinjury ACS in a cohort of trauma patients with severe shock who had similar demographics and injury severity (mean age, 39 years; 76% male; mean ISS, 28) was 15%, with more than 50% ACS-associated MOF and mortality.^{27,28}

Trauma surgeons have typically paid less attention to IAH over the years because ACS has been considered the lethal complication to prevent and treat.^{29,30} However, single-center and multicenter prospective studies have described the incidence of IAH in the general ICU population and have identified it as a predictor of poor outcome.^{31,32} Recent studies focusing on hemostatic resuscitation suggested that the incidence of postinjury ACS is decreasing compared with historic controls.¹⁴⁻¹⁶ While ACS is less frequent in trauma ICUs, injured patients continue to develop IAH during early resuscitation. This transient (approximately 24-hour) IAH does not cause organ dysfunction or require surgical intervention. Theoretically, sustained IAH compromises intestinal perfusion and could cause secondary local (intestinal) and remote organ dysfunctions. Similar to observations in general ICU populations, this could lead to an extended LOS and increased incidence of MOF and mortality in trauma patients who develop IAH.

The incidence of IAH was 73% in our high-risk cohort (Figure), which is much higher than in nontrauma series. We defined IAH (IAP > 12 mm Hg) as the average of the measurements taken in the first 24 hours (12), which means that all patients with IAH had sustained elevation of IAP. In our earlier description (University of Texas-Houston), patients with ACS had fundamentally different resuscitation volumes, times to ICU, hemoglobin concentrations, urine outputs, and shock parameters from those without ACS.¹¹ It is important to note that the Houston cohort had more severe shock parameters and required more pre-ICU transfusions. In our current cohort, the univariate comparison of patients with and without IAH showed similar characteristics; even the significant differences were clinically almost negligible (Table 1). Patients with IAH had 0.8 L more crystalloids before ICU admission (5.5 hours) compared with those without IAH. This shows a

Table 2. Results of Multivariate Analysis for Multiple Organ Failure Prediction^a

Predictor	Mean (SE)		OR (95% CI)	P Value
	No MOF (n=76)	MOF (n=5)		
Age, y	40 (2)	62 (5)	1.06 (1.01-1.12)	.03
ISS	28 (1)	44 (7)	1.09 (1.02-1.17)	.01
IAP _{mean} , mm Hg	14 (0.5)	17 (2)	1.17 (0.96-1.43)	.13
APP _{mean} , mm Hg	70 (1)	67 (3)	0.97 (0.88-1.07)	.55

Abbreviations: APP_{mean}, 24-hour average abdominal perfusion pressure; CI, confidence interval; IAP_{mean}, 24-hour average intra-abdominal pressure; ISS, injury severity score; MOF, multiple organ failure; OR, odds ratio.

^aOnly significant predictors and IAP/APP are presented.

Table 3. Results of Univariate and Parsimonious Multiple Regression Model for Association Between Potential Predictors and Length of Stay^a

Variable	Univariate		Forward Selection		Backward Selection	
	Estimate	P Value	Estimate	P Value	Estimate	P Value
ISS	0.2	.07	0.24	<.001	0.24	<.001
Urine output, mL/h	0.01	.10	0.02	.001	0.02	.001
MOF	16.36	.002	14.7	<.001	14.7	<.001
IAP _{max}	-0.16	.53				
IAP _{mean}	-0.39	.23				
APP _{min}	-0.08	.54				
APP _{mean}	-0.15	.27				

Abbreviations: APP_{mean}, 24-hour average abdominal perfusion pressure; AAP_{min}, lowest abdominal perfusion pressure during the first 24 hours; IAP_{max}, highest intra-abdominal pressure during the first 24 hours; IAP_{mean}, 24-hour average intra-abdominal pressure; ISS, injury severity score; MOF, multiple organ failure.

^aOnly the significant parameters and IAP/APP are listed.

fundamental change in clinical practice because historically our patients with ACS received more than 10 L of crystalloids before ICU admission; the current groups received less than 4 L.¹¹ The changes in resuscitation protocols were driven by the identified independent predictors of ACS, among which crystalloid resuscitation volumes were consistently strong.^{11,21-23}

Previously we established that crystalloid resuscitation (>3 L in emergency department and >7 L before ICU) is an independent predictor of ACS.¹¹ Based on this, we included crystalloid limits in our resuscitation strategy. Currently, patients with traumatic shock receive a maximum of 2 L crystalloids in the emergency department and a total of less than 4 L of crystalloids before ICU admission. These “crystalloid caps” are monitored by our trauma surgeons and fellows during resuscitation. Our study was not designed to answer the question of why the incidence of ACS decreased. The positive result has to be multifactorial (open abdomen management, crystalloid limits, hemostatic resuscitation, earlier hemorrhage control, IAP monitoring, awareness of predictors).²⁸ Because excessive crystalloid resuscitation was one of the strongest independent predictors of ACS, we believe that controlling this element of trauma resuscitation has the biggest effect on elimination of postinjury ACS.

While the outcomes of patients with postinjury ACS were shown to be markedly different from those with similar injury and shock severity who did not develop ACS,⁸⁻¹² the outcomes of patients with and without IAH were not

different in our current prospective cohort. Low mortality and low MOF incidence were characteristics of both groups. The ICU-LOS was not adversely affected by a sustained IAH (patients without IAH had a mean of 11 days in the ICU compared with 8 days for patients with IAH). Our findings highlight the need to reassess the validity of the World Society of ACS grading for IAH because at least grades I and II appear to have little effect on outcome.^{24,25}

The predictors of IAH were the need for laparotomy, metabolic acidosis, and pre-ICU crystalloid resuscitation volumes. Apart from the postlaparotomy status, the other 2 predictors were weak considering that, in the past, shock parameters and shock resuscitation parameters were strong predictors of ACS.¹¹ Most previously described independent predictors of ACS (time to ICU, first ICU hour urinary output, ICU admission temperature, hemoglobin concentration, and transfusion volumes) were not relevant to IAH prediction. Our data supports the notion that IAH during the first 24 hours is unlikely to represent an important confounder of shock/trauma patients' ultimate outcome.

Previously we showed that ACS was an independent predictor of postinjury MOF.^{11,22,23,27} Our campaign to reduce the incidence of MOF included elimination of postinjury ACS as a modifiable predictor of organ failure.²⁷ Our current cohort shows that IAP and APP are not predictors of MOF (Table 2). The identified predictors such as age and ISS are well-described harbingers of postinjury MOF.³³ We believe that the lack of statistical associa-

tion found between IAH and MOF is a major achievement in the process of overcoming ACS as a lethal disease. Patients with ACS in our previous cohorts had more than 50% mortality and MOF incidence; both outcomes were predicted by the presence of ACS.^{11,22}

In mixed ICU populations, IAH is shown to be a major cause of extended ICU-LOS.¹⁸ In our high-risk trauma cohort only MOF, ISS, and admission urine output were predictors of extended ICU-LOS; IAP and APP were not significant predictors of LOS (Table 3).

Abdominal perfusion pressure was proposed to be a superior measure to IAP in describing intestinal perfusion and predicting outcomes from retrospective studies.³⁴ It is interesting that our prospective results do not support the idea of APP having any superior predictive value over IAP in either univariate or multivariate comparisons.

These findings add to the efforts of trauma and critical care physicians during the last 15 years, which began with the recognition of the detrimental effects of acute postinjury ACS.¹⁻⁶ The journey continued, with continuously better characterization of the syndrome and identification of its independent predictors.⁷⁻¹³ The prospective awareness of the independent predictors, along with the liberal open-abdomen strategy, gradually decreased the incidence of and mortality associated with ACS.²⁸ Recognition of the catastrophic consequences of uncontrolled crystalloid resuscitation in the prevention of ACS influenced the fundamental paradigm shift toward hemostatic resuscitation during the last 5 years.^{22,35}

We determined that postinjury IAH is a frequent phenomenon during traumatic shock resuscitation but it is a relatively benign condition compared with its predecessor, the extremely morbid ACS. Our study failed to find any association between IAH and unfavorable outcomes such as MOF, mortality, or extended ICU-LOS.

The limitations of our study are the relatively small population, which we attempted to outweigh by our prospective design, and the focus on the already-identified high-risk group from our previous research. Owing to the relative paucity of the critically injured patients, any comparisons with historic cohorts should be interpreted critically. We are confident that, contrary to this, our improved outcomes are valid because similar trends were mentioned by researchers independent of our group,^{15,16} and the study populations between Houston and Newcastle were comparable at least in terms of demographics and injury severity.¹¹

In conclusion, our prospective cohort study on high-risk multiple-trauma patients failed to support our hypothesis that postinjury IAH is associated with poor outcome. Most of the severe shock/trauma patients develop sustained IAH. Based on univariate and multivariate analyses, there is no difference in outcomes of shock/trauma patients with and without IAH. Multivariate analysis failed to show IAH as a predictor of MOF. Attenuation of ACS to less deleterious IAH could be considered a success of the last decade in trauma and critical care.

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Abdominal Compartment Syndrome: Going, Going, Gone

The article by Balogh et al is important in that it shows that intra-abdominal pressure following significant resuscitation seems to be decreasing in incidence. It is rarely associated with abdominal compartment syndrome. Although severe shock and trauma are still occasionally accompanied by intra-abdominal hypertension, there is little to distinguish between patients that develop intra-abdominal hypertension and those who do not. Although all of this is true, it is most likely owing to the change in resuscitation strategy that this group and others have adopted.

Historically, shock resuscitation was based on a practice of giving 3 times crystalloid resuscitation to blood volume loss. This was the most common form of resuscitation during the 1960s and 1970s. During the 1980s, supranormal oxygen delivery as a target for resuscitation led to excessive crystalloid administration. This was never shown to improve mortality, but excessive crystalloid resuscitation lead to visceral edema and abdominal compartment syndrome. With trauma systems, sicker patients survived to undergo massive re-

suscitation. The use of decompression and the open abdomen followed.

The most significant thing that has occurred during the time course of the present study is that resuscitation has switched away from massive crystalloid resuscitation to a "crystalloid cap." This, as much as anything, accounts for the observed decrease in intra-abdominal hypertension and the apparent disappearance of the abdominal compartment syndrome. The authors have documented an important evolution in trauma management.

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