

Prospective Study of the Incidence and Outcome of Intra-Abdominal Hypertension and the Abdominal Compartment Syndrome.

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Prospective study of the incidence and outcome of intra-abdominal hypertension and the abdominal compartment syndrome

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Background: Intra-abdominal hypertension has been recognized as a source of morbidity and mortality in the traumatized patient following laparotomy. Multiple organ dysfunction attributable to intra-abdominal hypertension has been called the abdominal compartment syndrome. The epidemiology and characteristics of these processes remain poorly defined.

Methods: Intra-abdominal pressure was measured prospectively in all patients admitted to a trauma intensive care unit over 9 months. Data were gathered on all patients with intra-abdominal hypertension.

Results: Some 706 patients were evaluated. Fifteen (2 per cent) of 706 patients had intra-abdominal hypertension. Six of the 15 patients with intra-abdominal hypertension had abdominal compartment syndrome. Half of the patients with abdominal compartment syndrome died, as did two of the remaining nine patients with intra-abdominal hypertension. Patients with abdominal compartment syndrome had a mean intra-abdominal pressure of 42 mmHg compared with 26 mmHg in patients with intra-abdominal hypertension only ($P < 0.05$).

Conclusion: The incidence of intra-abdominal hypertension and abdominal compartment syndrome was 2 and 1 per cent respectively. Intra-abdominal hypertension did not necessarily lead to abdominal compartment syndrome, and often resolved without clinical sequelae. Abdominal compartment syndrome did not occur in the absence of earlier laparotomy. Abdominal compartment syndrome was associated with a marked increase in intra-abdominal pressure (above 40 mmHg).

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Introduction

Intra-abdominal hypertension, defined as a sustained increase in intra-abdominal pressure, can cause direct mechanical impairment of respiratory, haemodynamic, renal and splanchnic function. Organ dysfunction attributable to increased intra-abdominal pressure has been termed the abdominal compartment syndrome. The abdominal compartment syndrome has been increasingly recognized as a source of morbidity and mortality in the traumatized patient following laparotomy, typically as the result of massive visceral oedema, retroperitoneal haemorrhage or intra-abdominal packing^{1–5}. Abdominal compartment syndrome has also been reported in non-traumatized patients^{6–11}.

Despite several recent reviews^{12–14}, fundamental questions about intra-abdominal hypertension and abdominal

compartment syndrome remain unanswered. The overall incidence of intra-abdominal hypertension and abdominal compartment syndrome, the importance of an isolated finding of raised intra-abdominal pressure, the clinical progression of intra-abdominal hypertension, and the level of increased pressure that is associated with clinically significant impairment of respiratory, haemodynamic, renal and splanchnic function are all unknown. This study measured intra-abdominal pressure prospectively in a cohort of patients to characterize better the clinical significance of abdominal compartment syndrome and intra-abdominal hypertension in critically ill trauma patients. The ultimate goal of the study was to define a specific set of risk factors that would allow early identification of patients at high risk of developing abdominal compartment syndrome, ideally before overt manifestation of multiple organ dysfunction.

Patients and methods

From April 1998 to January 1999, all patients admitted to the intensive care unit (ICU) at a level I trauma centre underwent serial indirect measurement of intra-abdominal pressure via an indwelling urinary catheter. The standard procedure used was that first popularized by Kron *et al.*⁶ and subsequently validated clinically^{15–17}. Measurements were obtained initially at 6-h intervals. The study protocol was approved by the Institutional Review Board at the University of Miami/Jackson Memorial Hospital.

If intra-abdominal pressure remained below 20 mmHg without evidence of organ system dysfunction, measurements were discontinued after 24 h. If it was 20 mmHg or higher and was sustained for two reproducible measurements, the patient was given a diagnosis of intra-abdominal hypertension, and additional data were collected: haemodynamic variables (heart rate, blood pressure, central venous pressure and right heart catheter data if available); respiratory variables (respiratory rate, ventilator settings if applicable); renal variables (urine output, serum creatinine and blood urea nitrogen); Injury Severity Score (ISS); and the amount and composition of fluid resuscitation before and after the increased intra-abdominal pressure was initially recorded. Intra-abdominal pressure measurements were also increased in frequency from every 6 h to every 1–4 h. If intra-abdominal hypertension was associated with multiple organ dysfunction syndrome (MODS), abdominal compartment syndrome was suspected. If clinical suspicion was high, a laparotomy for abdominal decompression was performed. The diagnosis of abdominal compartment syndrome was ultimately confirmed when physiological variables improved with decompressive laparotomy.

Statistical analysis was performed using Fisher's exact test.

Results

The findings of the study are summarized in *Fig. 1*. A total of 706 patients was evaluated during the study period. The characteristics of these patients are summarized in *Table 1*. Fifteen (2 per cent) of 706 patients had intra-abdominal hypertension, defined as sustained intra-abdominal pressure of 20 mmHg or more. Six of these 15 patients had abdominal compartment syndrome confirmed at decompressive laparotomy (1 per cent of total admissions). The characteristics of these 15 patients are summarized in *Table 2*.

Table 3 summarizes notable differences between patients with abdominal compartment syndrome and those with intra-abdominal hypertension only. The six patients with abdominal compartment syndrome had a mean(s.d.) ISS of

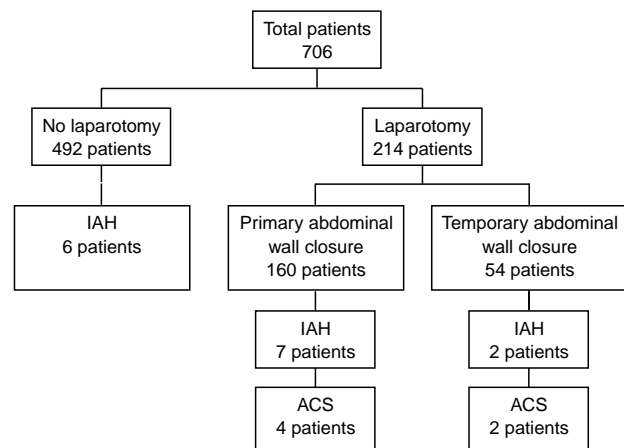


Fig. 1 Intra-abdominal hypertension (IAH) and compartment syndrome (ACS) in patients admitted to a trauma intensive care unit

Table 1 Characteristics of trauma intensive care unit patient population

Mean (range) age (years)	42 (14–90)
Sex ratio (M : F)	508 : 198
Mean (range) Injury Severity Score	18 (1–75)
Deaths*	88 (12)
Mechanism of injury*	
Motor vehicle crash	311 (44)
Gunshot wound	155 (22)
Pedestrian/cyclist struck	107 (15)
Stab wound	49 (7)
Fall	42 (6)
Assault	21 (3)
Other†	21 (3)

*Values in parentheses are percentages. †For example, boating accident, mauling, train injury

34(12) and an intra-abdominal pressure of 42(11) mmHg. Three of the six patients died from progressive multiple organ failure despite abdominal decompression. Notably, two of the six patients developed abdominal compartment syndrome despite 'prophylactic' temporary abdominal closure.

The nine patients with intra-abdominal hypertension who did not undergo abdominal decompression had a mean(s.d.) ISS of 27(12) and a mean(s.d.) intra-abdominal pressure of 26(4) mmHg. Two of these nine patients died, one from a late pulmonary embolus during rehabilitation after discharge from the ICU and one from complications of a severe closed head injury. Four of the nine patients had intra-abdominal hypertension without laparotomy before admission. Two of these patients had resolution of intra-

Table 2 Characteristics of patients with intra-abdominal hypertension and abdominal compartment syndrome

	Intra-abdominal hypertension only	Abdominal compartment syndrome
Sex ratio (M : F)	9 : 0	4 : 2
Mean (range) age (years)	42 (23–76)	44 (21–88)
Cause of injury		
Gunshot wound	4	2
Motor vehicle crash	4	2
Pedestrian struck	1	1
Fall		1
Type of injury		
Isolated vascular injury	1	
Retroperitoneal haematoma, amputation	1	
Multiple thoracic injuries	1	
Multiple abdominal injuries	2	1
Multiple abdominal and orthopaedic injuries	2	3
Multiple abdominal and thoracic injuries		2
Multiple abdominal, thoracic and head injuries	1	
Multiple abdominal, thoracic and orthopaedic injuries	1	
Clinical course		
Abdominal decompression		6
Discharged from hospital	7	3
Deaths	2	3
Head injury	1	
Pulmonary embolus	1	
Multiple organ dysfunction syndrome		3

Table 3 Comparison of intra-abdominal pressure, Injury Severity Score and mortality rate in patients with intra-abdominal hypertension and abdominal compartment syndrome

	Intra-abdominal hypertension only	Abdominal compartment syndrome	<i>P</i> *
Intra-abdominal pressure (mmHg)	26(4)	42(11)	< 0.05
Injury Severity Score	27(12)	34(12)	0.08
Mortality rate	2 of 9	3 of 6	0.07

Values are mean(s.d.). *Fisher's exact test

abdominal hypertension following 'large' bowel movements.

There were trends toward increased ISS and increased mortality rate in the patients with abdominal compartment syndrome. However, only the difference in intra-abdominal pressure between the two groups reached statistical significance: 26(4) *versus* 42(11) mmHg ($P < 0.05$).

Discussion

Although MODS has been described for over 30 years, its treatment remains primarily supportive. The abdominal compartment syndrome – like tension pneumothorax, its better known analogue in the chest – is a potentially treatable cause of early MODS in the severely injured patient. Like tension pneumothorax, abdominal compart-

ment syndrome causes organ dysfunction by direct mechanical effects. Respiratory derangement occurs as the result of elevation of the diaphragm, causing decreased functional residual capacity and increases in airway pressures in patients on positive pressure ventilation¹⁸. Haemodynamic derangements are the result of decreased cardiac preload from compression of the inferior vena cava with resultant decreased cardiac output¹⁹. Compression of the renal vein results in impairment in kidney function²⁰. Increased intra-abdominal pressure has also been shown to decrease splanchnic perfusion^{21,22}. Once diagnosed, abdominal compartment syndrome requires and responds immediately to invasive therapeutic intervention.

That this diagnosis may be a difficult one to make further complicates an already complex clinical problem. Among trauma surgeons abdominal compartment syndrome is a

well established diagnosis²³. However, despite numerous series in the literature, much about abdominal compartment syndrome remains unknown and therefore controversial, in part because of confusion regarding terminology. There are, as yet, no universally accepted definitions. This study used a broad definition of intra-abdominal hypertension to ensure capture of all patients with a sustained increase in intra-abdominal pressure. The threshold for intra-abdominal hypertension was defined arbitrarily as a sustained intra-abdominal pressure of 20 mmHg and above.

Normal intra-abdominal pressure is highly variable in healthy individuals, depending on body position, body habitus and activity. Transient increases in intra-abdominal pressure to 80 mmHg have been described in normal subjects^{24–26}. Sustained pneumoperitoneum to 15 mmHg is routinely used without apparent adverse effects during laparoscopic surgery²⁷. In the present study 20 mmHg was chosen so that all individuals with even mild, occult intra-abdominal hypertension were included and followed, with the goal of characterizing the spectrum of disease, from a mild asymptomatic pressure increase to frank abdominal compartment syndrome, and to ensure that all cases of intra-abdominal hypertension were captured. In the absence of baseline data of this kind, no true estimate of the overall incidence of intra-abdominal hypertension and abdominal compartment syndrome may be made.

For the purposes of this study, abdominal compartment syndrome was narrowly defined as intra-abdominal hypertension with associated MODS that improved following abdominal decompression. This very strict definition of abdominal compartment syndrome was used to ensure that the diagnosis of abdominal compartment syndrome was accurate. Some patients with intra-abdominal hypertension associated with relatively mild organ dysfunction may not require decompressive laparotomy, but will respond with non-operative observation; according to the present definitions, such patients had intra-abdominal hypertension and not true abdominal compartment syndrome.

The ultimate goal of this study was to identify specific risk factors that would allow early diagnosis of intra-abdominal hypertension before the onset of frank MODS, for example the amount and composition of fluid resuscitation, specific injuries such as pelvic fracture or hepatic injury, the use of perihepatic packing, etc. However, the incidence of intra-abdominal hypertension and abdominal compartment syndrome in this cohort of patients was surprisingly low, and did not allow a statistically meaningful analysis of risk factors.

The data show that intra-abdominal hypertension and abdominal compartment syndrome are rare (2 and 1 per cent respectively). This differs markedly from two previous series, which reported incidences of 14 per cent²⁸ and 33 per

cent²⁹. There are two plausible explanations for this discrepancy. First, by measuring intra-abdominal pressure in all patients admitted to the ICU, this study included low-risk as well as high-risk patients, whereas the previous studies confined data collection to high-risk patients. While the latter approach ensures a good yield of patients with abdominal compartment syndrome, it may result in a very high incidence compared with that seen clinically in the ICU population overall. Furthermore, such an approach potentially misses patients who are not at high risk, and yet may have MODS falsely attributed to sepsis or irreversible shock when in fact they have unrecognized abdominal compartment syndrome. By measuring intra-abdominal pressure prospectively in all patients, this study obtained a true overall incidence. By using this methodology, the study also identified all cases of occult intra-abdominal hypertension, regardless of whether they were associated with clinical sequelae.

The second reason for such a low incidence is the large number of temporary abdominal closures performed; 54 (25 per cent) of 214 patients who underwent laparotomy had temporary abdominal closure before arrival in the ICU, and before any measurement of intra-abdominal pressure. Some characteristics of these patients are shown in *Table 4*. In most cases temporary abdominal closure was performed using a prosthetic mesh device or a 'Bogota' bag to expand the abdominal wall, as prophylaxis against the development of abdominal compartment syndrome. If even a small number of these patients had undergone primary fascial closure, the incidence of intra-abdominal hypertension and abdominal compartment syndrome may have been substantially higher.

Table 4 Characteristics of 54 patients who had temporary abdominal closure

Type of closure†	
Prosthetic mesh/'Bogota' bag‡	49 (2)
Primary closure of skin only§	5 (0)
Mean (range) age (years)	33 (19–67)
Sex ratio (M : F)	40 : 14
Mean (range) Injury Severity Score	31 (16–59)
Deaths*	21 (39)
Mechanism of injury*	
Motor vehicle crash	17 (31)
Gunshot wound	33 (61)
Pedestrian/cyclist struck	2 (4)
Stab wound	2 (4)

Values in parentheses are *percentages or †number of patients with abdominal compartment syndrome. ‡Allows expansion of abdominal wall for prophylaxis against abdominal compartment syndrome; §fascia left open, for planned re-exploration

The presence of intra-abdominal hypertension following temporary abdominal closure was an ominous finding. Two patients developed intra-abdominal hypertension despite prophylactic temporary abdominal closure, both of whom had frank abdominal compartment syndrome. Both patients required abdominal decompression, with the use of larger prosthetic materials to expand the abdominal cavity.

Similarly, the presence of intra-abdominal hypertension following laparotomy with primary fascial closure may be an ominous finding, suggestive of abdominal compartment syndrome. In this study intra-abdominal hypertension occurred in only 4 per cent (nine of 160) of patients following laparotomy and primary fascial closure. However, four of these patients had abdominal compartment syndrome as a result of intra-abdominal hypertension.

Abdominal compartment syndrome did not occur in the absence of an earlier laparotomy in the present study. There are increasing reports of abdominal compartment syndrome occurring without laparotomy in the literature, in burn patients (especially those with circumferential full-thickness torso burns) and in patients without intra-abdominal injury^{30,31}.

Intra-abdominal hypertension does not inevitably progress to abdominal compartment syndrome, and may be associated with relatively benign conditions, such as constipation. Two patients had resolution of intra-abdominal hypertension following 'large' bowel movements. An isolated finding of intra-abdominal hypertension in an otherwise asymptomatic patient is clearly different from frank abdominal compartment syndrome in a patient with severe abdominal trauma and worsening MODS.

In general, asymptomatic intra-abdominal hypertension had a benign clinical course. True abdominal compartment syndrome, by comparison, is associated with significant morbidity and mortality. Three of six patients with abdominal compartment syndrome died from progressive MODS, despite early recognition and abdominal decompression.

Abdominal compartment syndrome was also associated with a significantly higher intra-abdominal pressure than asymptomatic intra-abdominal hypertension (42 *versus* 26 mmHg). Although not unexpected, this is a meaningful finding; an intra-abdominal pressure of 40 mmHg clearly is more worrisome than one of 20 mmHg, especially when associated with MODS.

Based on the findings of this study, routine measurement of intra-abdominal pressure is not necessary in all patients, given its rarity and given the benign nature of asymptomatic intra-abdominal hypertension. However, routine measurement of intra-abdominal pressure is warranted in patients at high risk, certainly in those with early MODS following trauma, even if they have undergone prophylactic temporary

abdominal closure previously. Because of the small number of patients with intra-abdominal hypertension and abdominal compartment syndrome, this study was not able to elucidate definitively variables that would characterize low or high risk.

Acknowledgements

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European Colorectal Congress

28 November – 1 December 2022, St.Gallen, Switzerland

Monday, 28 November 2022

09.50

Opening and welcome

Jochen Lange, St.Gallen, CH

10.00

It is leaking! Approaches to salvaging an anastomosis

Willem Bemelman, Amsterdam, NL

10.30

Predictive and diagnostic markers of anastomotic leak

Andre D'Hoore, Leuven, BE

11.00

SATELLITE SYMPOSIUM

ETHICON

PART OF THE Johnson & Johnson FAMILY OF COMPANIES

11.45

Of microbes and men – the unspoken story of anastomotic leakage

James Kinross, London, UK

12.15

LUNCH

13.45

Operative techniques to reduce anastomotic recurrence in Crohn's disease

Laura Hancock, Manchester, UK

14.15

Innovative approaches in the treatment of complex Crohn Diseases perianal fistula

Christianne Buskens, Amsterdam, NL

14.45

To divert or not to divert in Crohn surgery – technical aspects and patient factors

Pär Myrelid, Linköping, SE

15.15

COFFEE BREAK

15.45

Appendiceal neoplasia – when to opt for a minimal approach, when and how to go for a maximal treatment

Tom Cecil, Basingstoke, Hampshire, UK

16.15

SATELLITE SYMPOSIUM

Medtronic

Further Together

17.00

Outcomes of modern induction therapies and Wait and Watch strategies, Hope or Hype

Antonino Spinelli, Milano, IT

17.30

EAES Presidential Lecture - Use of ICG in colorectal surgery: beyond bowel perfusion

Salvador Morales-Conde, Sevilla, ES



18.00

Get-Together with your colleagues

Industrial Exhibition

Tuesday, 29 November 2022

9.00

CONSULTANT'S CORNER

Michel Adamina, Winterthur, CH

10.30

COFFEE BREAK

11.00

SATELLITE SYMPOSIUM

INTUITIVE

11.45

Trends in colorectal oncology and clinical insights for the near future

Rob Glynn-Jones, London, UK

12.15

LUNCH

13.45

VIDEO SESSION

14.15

SATELLITE SYMPOSIUM



15.00

COFFEE BREAK

15.30

The unsolved issue of TME: open, robotic, transanal, or laparoscopic – shining light on evidence and practice

Des Winter, Dublin, IE

Jim Khan, London, UK

Brendan Moran, Basingstoke, UK

16.30

SATELLITE SYMPOSIUM



17.15

Lars Pahlman lecture

Søren Laurberg, Aarhus, DK

Thursday, 1 December 2022
Masterclass in Colorectal Surgery
Proctology Day

Wednesday, 30 November 2022

9.00

Advanced risk stratification in colorectal cancer – choosing wisely surgery and adjuvant therapy

Philip Quirke, Leeds, UK

09.30

Predictors for Postoperative Complications and Mortality

Ronan O'Connell, Dublin, IE

10.00

Segmental colectomy versus extended colectomy for complex cancer

Quentin Denost, Bordeaux, FR

10.30

COFFEE BREAK

11.00

Incidental cancer in polyp - completion surgery or endoscopy treatment alone?

Laura Beyer-Berjot, Marseille, FR

11.30

SATELLITE SYMPOSIUM



12.00

Less is more – pushing the boundaries of full-thickness rectal resection

Xavier Serra-Aracil, Barcelona, ES

12.30

LUNCH

14.00

Management of intestinal neuroendocrine neoplasia

Frédéric Ris, Geneva, CH

14.30

Poster Presentation & Best Poster Award

Michel Adamina, Winterthur, CH

15.00

SATELLITE SYMPOSIUM

OLYMPUS

15.45

COFFEE BREAK

16.15

Reoperative pelvic floor surgery – dealing with perineal hernia, reoperations, and complex reconstructions

Guillaume Meurette, Nantes, FR

16.45

Salvage strategies for rectal neoplasia

Roel Hompes, Amsterdam, NL

17.15

Beyond TME – technique and results of pelvic exenteration and sacrectomy

Paris Tekkis, London, UK

19.30

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