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TRAUMA Grapevine

Introduction

Principles of trauma care are increasingly evolving into a global approach with increasing consistency in terms of level of agreement and delivery of care. Many developed and most disadvantaged countries have significant challenges facing them in trauma care. The opportunity is there for improvements in trauma education, trauma system and the development of clinical practice guidelines suitable for the variety of levels of trauma care we currently provide. Recent advances within Australia have seen the increased designation of trauma services, a focus on education through the definitive surgical trauma care course, the early management of severe trauma, TNCC, pre-hospital trauma course and many other innovations. The conceptual development of a national trauma registry, a trauma research institute in Melbourne and an Australia-Pacific foundation for trauma standards in Liverpool will herald a new era.

In this issue, advances in our understanding of the Abdominal Compartment Syndrome are identified and it is increasingly recognized as a major problem in trauma patients. Dr. Jo Ollerton discusses the challenges of incomplete spinal cord syndrome.

Post-injury Abdominal Compartment Syndrome:

Summary of the Recent Advancements

Zsolt Balogh, MD
Trauma Fellow
Trauma Department, Liverpool Hospital, Sydney

Frederick A. Moore, MD
Professor and Vice-Chairman, Department of Surgery
University of Texas – Houston Medical School

The objective of this article is to provide an overview of the recent advances in the characterisation of post-injury abdominal compartment syndrome (ACS) including (1) epidemiology, (2) factors related to outcome, (3) the association between ACS and multiple organ failure, (4) the association between shock resuscitation and ACS, (5) the potential problems of standard resuscitation, (6) predictors and (7) new strategies in the management of open abdomens.

Definitions (recapitulation):

Abdominal compartment syndrome (ACS) is defined as intra-abdominal pressure (IAP) higher than 25 mmHg with organ dysfunction (cardiac, respiratory, renal) if the organ dysfunction improves after abdominal decompression[1,2]. ACS is the imbalance between abdominal volume and abdominal content where the abdominal volume is defined by the less tensile component of the abdominal compartment, by the fascia. Increasing abdominal content or decreasing volume causes ACS with impaired abdominal organ perfusion also seen in the well recognized compartment scenarios of increased intra-cranial pressure, pericardial tamponade, tension pneumothorax or extremity compartment syndromes.

ACS was described earlier in non-trauma clinical scenarios such as closure of large abdominal defects ("visceroabdominal disproportion") in infants[3] and abdominal aortic aneurysm surgery[4]. Post-injury ACS (used as ACS from here) became a common lethal complication of the damage control surgery[5]. Trauma patients entering into the "bloody vicious cycle" of bleeding, resuscitation, hypothermia, coagulopathy and acidosis can benefit from abbreviated laparotomy (damage control) with quick hemorrhage control (ligatures and packing) and the prevention of further contamination/spillage from hollow viscus perforations (stapled resections without obligatory restoration of the contibuity of the intestines)[6]. Although damage control has saved many severely injured patients who would have previously died on the operating table, it has however caused new challenges, in the next phase of damage control, during the restoration of physiology on the ICU including ACS, open abdomens and early multiple organ failure (MOF).

Postinjury ACS has two clinical manifestations with distinct predictors and hospital course.

Primary ACS: A recognized complication of damage control laparotomy. The space occupying nature of the abdominal packs together with ongoing bleeding and the progressive edema of the reperfused bowel all contribute to increased abdominal content. If the fascia is closed the volume of

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the abdominal cavity is returned to its original uninjured volume and, therefore, the increase in abdominal contents increases the IAP. Primary ACS can also occur in patients who fail non-operative management of abdominal organ injuries because of ongoing bleeding[7].

Secondary ACS: This typically occurs in the setting of severe shock requiring massive resuscitation (whole body ischaemia reperfusion injury)[8-11]. As there are no abdominal injuries, which would draw clinicians' attention to the abdomen, secondary ACS is more elusive and recognition is often delayed[8,9]. Here the abdominal content is increased in volume by bowel edema and ascites, and the volume of the abdominal cavity can be decreased by a retroperitoneal hematoma originating from pelvic fractures[8,9,11].

The pathophysiology of the full blown ACS manifested as consequences of increased IAP are similar. The consequences of intra-abdominal hypertension (IAH) on cerebral, respiratory, cardiac, intestinal and renal function are well described and already published in this journal (Sugrue 1999).

Early seminal reports convinced clinicians that ACS is a real entity that is often delayed in diagnosis and associated with significant mortality. These first generation studies described a new syndrome and not surprisingly lack the study design and statistical power to answer important questions concerning potential pathogenesis and prediction. Most are retrospective studies with either very high or very low incidence and data were not subjected to multivariate analysis. The definition of ACS was not uniform among the studies, and management and monitoring were not standardized.

The actual incidence of ACS is impossible to determine because of the very different study populations (denominator) that are not consistent with the definition of ACS (numerator). As a consequence of this and the consistently high mortality rates, we aimed to provide better characterisation of the Abdominal Compartment Syndrome. We aimed to identify key clinical intervention opportunities and to develop prediction models to enable earlier interventions to improve the outcome.

The prospective computerized shock trauma resuscitation database of the Memorial Hermann Hospital was utilized12. The resuscitation protocol has strict inclusion criteria. The need for shock resuscitation is objectively defined by (1) specific injuries, (2) early arterial base deficit (BD) ≥ 6 mEq/L, and (3) need for transfusion of ≥6 units/first 12 hours; or a trauma victim of age ≥ 65 years with any two of the other criteria. Patients who have incurred significant brain injury are not resuscitated by protocol. The identified patients have a pulmonary artery catheter and gastric tonometer placed and are resuscitated by a hierarchy of five sequentially applied therapies with intervention thresholds to achieve a specified oxygen delivery index (DO2I) goal for 24 hours. These interventions are: (1) blood (PRBC) transfusion to maintain hemoglobin [Hb] concentration, (2) lactated Ringer's infusion to maintain pulmonary capillary wedge pressure [PCWP], (3) Starling curve generation with successive 500 ml crystalloid boluses to optimize cardiac index (CI)-PCWP relationship if Hb ≥ 10 g/dL, PCWP ≥ 15 mmHg, and DO2I < goal, (4) inotrope if CI-PCWP has been optimized and DO2I < goal, and (5) vasopressor if MAP < 65 mmHg. At the inception of the protocol, DO2I ≥ 600 mL/min/m2 was the goal of the protocol process. After 2 years we decreased the DO2I goal to 500 mL/min/m2 because randomized clinical trials failed to prove the superiority of supra-normal resuscitation[13].

(1) Epidemiology

In the above-described cohort primary and secondary ACS has occurred with a similar incidence 6% vs 8%. For comparison this 14% overall ACS incidence in the shock resuscitation protocol group means 1.15% (26/2,258) incidence if the denominator is all shock trauma ICU admissions and 0.16% (26/16,376) incidence among all trauma admissions[12]. The use of the damage control population as a denominator is not feasible to study since it does not take the secondary ACS into account.

The demographics, ISS, GCS, and initial base deficit are not different among the non-ACS, ACS, and the ACS subgroups in the shock resuscitation protocol cohort. Primary ACS and non-ACS patients have a similar injury pattern, but secondary ACS patients have no abdominal injuries (by definition), but had more extremity injuries, especially major pelvic fractures and multiple long bone fractures. In the emergency department, the ACS patients present with lower systolic blood pressure, which prompts more aggressive fluid and blood resuscitation. The hemorrhage control of primary ACS and non-ACS patients is achieved predominantly in the operating room while the secondary ACS patients have significantly more frequent pelvic arterial embolization at interventional radiology. Pre-ICU resuscitation differs between the groups. The ratio of liters of crystalloid to units of blood transfusion is higher in secondary ACS (1.8 ± 0.2) compared to the primary ACS (1.2 ± 0.2) and non-ACS patients (1.4 ± 0.1). Both primary and secondary ACS patients were decompressed at a similar time point after hospital admission (mean of 12-13 hours). Given their longer pre-ICU course, secondary ACS patients were decompressed earlier after admission to the ICU (1° ACS=10 hrs versus 2° ACS=6 hrs).

(2) Factors related to outcome

Despite initial good physiologic responses to decompression, the outcomes (vent days, ICU days, MOF, mortality) of both ACS groups were uniformly poor compared to the non-ACS group. As formerly published in the literature, the decompression improved all the previously deranged parameters2,8,14,15, but only the improved cardiac index and urinary output are associated with better outcome12. Other improved parameters like increased pulmonary compliance, decreased airway pressures, systemic vascular resistance, CVP and PCWP are simple physical consequences of the decreased IAP and not related to the outcome.

(3) The association between ACS and multiple organ failure

There is convincing evidence that both ACS and MOF are preceded by hemorrhagic shock and significant tissue injury. ACS occurs very early during shock resuscitation and is frequently followed by the development of MOF. The poor outcome of ACS is frequently published in the literature but the statistical relationship was never explored. In our cohort ACS]is a predictor of both MOF (odds ratio= 9.2, 95% confidence intervals: 3.8 - 22.8, p< 0.0001) and mortality (odds ratio= 8.4, 95% confidence intervals: 3.5 - 20.6, p< 0.0001)[12].

Trauma patients are resuscitated into a state of early systemic hyper-inflammation, now known as the systemic inflammatory response syndrome (SIRS). This response is presumed to be beneficial and resolves in most patients as they recover. However, if exaggerated or perpetuated, severe SIRS evolves into overt early MOF. Early MOF can occur because of the overwhelming initial insult, which leads to severe SIRS (one-hit model) or with a less severe initial insult with moderate SIRS amplified by secondary insults (two-hit model).

It is known that the neutrophil is the key effector cell in early MOF[16,17]. In vitro studies of PMN functions demonstrate that PMNs can be "primed" by previous exposure to inflammatory mediators such that they have exaggerated response to subsequent activating stimuli[16]. Moreover, in trauma patients, "priming" of circulating PMNs has been demonstrated to start as early as 3 to 6 hours postinjury and last for 24 hours. It is believed that this represents an "early vulnerable window" to secondary insults (e.g. aspiration, fat embolism, recurrent shock, massive transfusion)[16]. In the laboratory, "two-hit" models of MOF have been created to simulate common clinical scenarios and have shown that exacerbated PMN mediated tissue injury is a central pathogenic

mechanism[18,19]. Most recently, investigators have shown that abdominal decompression of ACS releases pro-inflammatory cytokines and, when sequenced with prior hemorrhagic shock/resuscitation (HS/R), ACS decompression amplifies cytokine release and causes more severe lung injury than with HS/R or ACS decompression alone[20,21]. Moreover, the timing of ACS decompression was shown to be important. With decompression 2 hrs or 18 hrs after HS/R, ACS had much less adverse effect than when performed at 8 hrs[22]. This time interval effect of decompression is consistent with the 'early vulnerable window' of PMN priming observed in patients (3 to 24 hrs). Moreover, we have observed that gastric tonometry CO2 gap is an independent predictor of ACS and, after decompressive laparotomy, the CO2 gap measured by gastric tonometry decreases significantly[12]. The effectiveness of gastric tonometry in the detection of IAH/ACS related intestinal hypoperfusion has been alluded in earlier clinical studies on damage control patients and general surgical patients[14,23]. Thus, abdominal decompression of established ACS appears to cause a fulminant reperfusion syndrome that does not respond to post decompression resuscitation[1,24]. Taken together, these data supports the hypothesis that postinjury ACS is a 'second hit' in the two-hit model of postinjury MOF.

(4) The relationship between ACS and the resuscitation endpoints:

Anecdotally many authors connected ACS with vigorous resuscitation, but no study showed the cause-effect relationship[8-11]. The above described traumatic shock resuscitation protocol with the two different DO2I endpoints provided a unique opportunity to compare the supra-normal and normal resuscitation endpoints effect on the incidence of ACS[25].

The change was instituted January 1, 2001. During the 16 months prior to the change, 85 patients were resuscitated to the supra-normal goal of 600 ml/min/m2 compared to 71 patients who were resuscitated to a goal of 500 ml/min/m2 in the 16 months after the change. No other changes were implemented in the protocol. Demographics, ISS, and initial severity of shock were the same. The two cohorts received the same amount of crystalloids and blood in pre-ICU resuscitation. Their response to the ICU protocol was quite similar in regards to increasing CI and mixed venous oxygen saturation and decreasing base deficit and lactate elevations. By the protocol, the supra-normal resuscitated cohort, received significantly more crystalloid [13 $\pm$ 2* vs 7 $\pm$ 1 liters (*denotes $p<0.05$)] and there was a trend for more blood transfusions (6 $\pm$ 1 vs 4 $\pm$ 1 units, $p = 0.07$) per protocol. The supra-normal group had a higher CO2 GAP (gastric mucosal PCO2 minus end-tidal CO2), a higher incidence of IAP > 20 mmHg (42%* vs 20%), ACS (16%* vs 8%), MOF (22%* vs 9%) and mortality (27%* vs 11%). The only difference between the groups was the significantly more LR administration in the supra-normal group. Supra-normal resuscitation was associated with more crystalloid infusion during the protocol and worse outcome.

(5) The potential problems of preload driven resuscitation in patients with impending ACS:

The early descriptive studies recommended hypervolaemic resuscitation in patients with elevated IAP to overcome or prevent organ dysfunction[1,2]. According to our experience the resuscitation response of patients with impending ACS upon ICU admission differed from those patients who did not develop ACS in their response to our standardized ICU resuscitation[26]. During the first 8 hours of ICU resuscitation, ACS patients received more PRBC's (11 $\pm$ 2 vs 2 $\pm$ 1 units*) and crystalloid (13 $\pm$ 2 vs 4 $\pm$ 1 L*). Both groups started at a similarly low cardiac index (2.8 $\pm$ 0.4 vs 3.1 $\pm$ 0.2 L/min/m2). The non-ACS patients had a good cardiac response to the resuscitation protocol while the ACS did not respond well to volume loading (at 8 hr ICU resuscitation: ACS CI = 3.2 $\pm$ 0.2 vs non-ACS CI = 4.2 $\pm$ 0.1* L/min/m2). Significantly more of the ACS group progressed in the protocol to have a Starling curve performed (56% vs 23%*) and because of their continued poor response, the performance of the Starling curve tended to require more crystalloids (3 $\pm$ 1 vs 5 $\pm$ 1 L). As a result, PCWP increased to a higher level in the ACS patients (at 8 hr: 20 $\pm$ 2 vs 15 $\pm$ 1 mmHg*). At the beginning of ICU resuscitation, ACS and non-ACS patients had similar elevations in IAP and gastric regional CO2 (PgCO2) levels. By 8 hrs of ICU resuscitation, IAP had risen significantly in the ACS patient, but not in non-ACS patients (26 $\pm$ 4 vs 14 $\pm$ 2mmHg*). Similarly, ACS patients developed pathologic elevations in PgCO2 (70 $\pm$ 7 vs 48 $\pm$ 1 mmHg*). Contrary to earlier recommendations our data suggests that preload driven crystalloid based resuscitation is not effective and potentially detrimental in patients with impending ACS ("futile crystalloid preloading")[25,26].

(6) Independent risk factors and prediction model for post-injury ACS

The IAP measurement is a widely accepted, inexpensive and simple monitoring tool for ACS[4,27]. However, it is an intermittent measurement and the organ dysfunction that typifies ACS can occur at IAP <25 mmHg, while some patients with IAP > 25 mmHg do not develop any symptoms. Not surprisingly, trauma surgeons are less likely to make their decision for decompression based only on IAP[28]. The other problem with IAP is that it is an indicator of the syndrome (defined by IAP) not a real predictor. When IAP is pathologically elevated and organ dysfunction is imminent, the prevention of the syndrome is a faint hope. Numerous clinical case reports, reviews, retrospective studies, and some prospective studies describe the potential not statistically identified risk factors based on univariate analysis and expert opinion[14,29,30]. Case series on secondary ACS empirically recommended crystalloid volume thresholds above which IAP monitoring is recommended[8,10].

Recently a predictive equation31 was created based on 22 case controlled general surgical patients: $P = 1/(1+ e-z)$, where $z = -18.6763 + 0.1671$ (peak airway pressure) + 0.0009 (24 hours fluid balance). In our experience, postinjury ACS occurs most frequently during the first 12 hours after injury, and waiting for the 1st 24 hour fluid balance is much too late. By this time, most patients who are susceptible already exhibit the full blown syndrome[11,12], and studies have shown that postinjury ACS recognized after 24 hours has a lethal outcome[8,9]. Additionally, two prospective trauma studies have failed to identify predictors for ACS in their cohorts. The failure can be explained by the very heterogeneous (all ICU admissions, 1% incidence) or too homogeneous (damage control patients, 36% incidence) nature of the selected study populations[32,33].

Given the early occurrence of postinjury ACS, our prediction models and preventive efforts should focus on the first 6 hours after hospital admission. We developed two prediction models: Emergency Department model (0 - 3 hours, i.e. all patients have initial diagnostic work-up, clinical laboratory results and discharge from the emergency department completed) and Intensive Care Unit model (0 - 6 hours, i.e. all patients have been admitted to ICU and their first physiologic monitor and clinical laboratory measurements on the resuscitation protocol are available)[12].

As we described above, postinjury ACS is not a homogenous group. Primary and secondary ACS patients develop the same symptoms and pre-decompression physiology, but their injury pattern, resuscitation and hospital times are different. We hypothesized, therefore, that their predictors would be different. The variables that were used in the multivariate prediction models are: demographics, shock severity, injury severity, interventions, hospital times, crystalloid and blood volumes, vital signs, and, in the ICU, initial pulmonary artery catheter readings, mechanical ventilatory settings and response parameters, gastric tonometry data, and blood gas, clinical chemistry and coagulation results. From these variables, those listed in Table 1 were found to be

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independent risk factors for ACS12. The primary ACS predictors at ICU admission (low temperature, low hemoglobin concentration and high base deficit) are all indicators of the "bloody vicious cycle" physiology, the reason that damage control surgery is elected. The secondary ACS predictors (high crystalloid infusion volume and impaired renal function compared to non ACS resuscitation patients) suggest that the process is strongly related to the current standard of care in the United States, i.e. crystalloid resuscitation. The receiver operator characteristic analysis showed that ACS can be predicted with 0.88 accuracy at the time of emergency department discharge and surprisingly with 0.99 accuracy 1 hour after ICU admission with adequate monitoring. The use of these predictors together (even excluding IAP measurements) permits very early detection of the impaired physiology characteristic of ACS. Because the predictors of ACS include both physiologic measurements and resuscitative interventions, this model should perform better in clinical situations during ongoing resuscitation than arbitrary IAP and organ dysfunction thresholds[12].

(7) Vacuum Assisted Wound Closure of Open Abdomen

ACS can be prevented by leaving the abdomen open after damage control and can be treated with abdominal decompression (opening the midline fascia in full length). Both approaches lead to open abdomen management with significant morbidity and mortality. Those abdomens which fail to have fascial closure within 5-7 days, are unlikely to be closed primarily later. These patients end up with large fascial defects (hernias) correctable after 6-12 months with extensive, lengthy plastic surgical interventions.

With current resuscitation strategies, ACS and the open abdomen have become a new challenge. A vacuum assisted wound closure (VAWC) system has been developed to assist with obtaining early fascial closure. After damage control or decompressive laparotomy, we routinely close the abdomen with a 'Bogota bag.' Our standard care is to remove the 'Bogota bag' 2 days after the first laparotomy and, if fascial closure is not feasible, VAWC is used[34,35]. A non-adherent perforated dressing is placed over the bowel, followed by a polyurethane sponge and an overlying occlusive barrier. The airtight dressing is then placed at –175mmHg using the VAWC system (V.A.C. Therapy,, Kinetic Concepts Inc, San Antonio TX). This dressing is changed at 2 - 3 day intervals, the fascia closed as feasible by placing interrupted suture superiorly and inferiorly, and the dressing replaced with a downsized sponge. This is repeated until fascia is closed. In our ongoing experience, we obtain complete fascial closure in 80% of patients in an average of 7 days.[35,12]

Conclusions, recommendations and future directions:

Post-injury ACS is relatively rare (1.15%) phenomenon among trauma ICU admissions but more frequent (14%) in the high-risk multiply injured shock resuscitation patients. In centers with aggressive resuscitation the incidence of primary and secondary ACS can be similar. Primary and secondary ACS patients' demographics, injury and shock severity are not different from each other and from non-ACS severely injured patients with traumatic shock. Primary and secondary ACS has distinct injury pattern, pre-ICU course and pre-ICU resuscitation with distinct independent predictors. Both syndromes can be predicted within 3 hours of admission with a very sensitive predictive model (over-inclusive) and within 6 hours with a very specific predictive model. Early definitive haemorrhage control is warranted in both groups to avoid uncontrolled resuscitation. Goal oriented resuscitation and abundant monitoring is helpful in the management of potential ACS patients. Supra-normal resuscitation goals and excessive preload to improve cardiac function is harmful in patients with already elevated IAP and excessive risk for ACS. In these patients alternative resuscitation with hypertonic saline or colloids are may be helpful and should be tested. With prospective awareness (temporary abdominal closure after damage control, early haemostasis and controlled resuscitation with crystalloid limits, abundant monitoring) the incidence of ACS could be minimized. The management of the open abdomen related morbidity and mortality is going to be a more difficult challenge to solve, but the preliminary results with new temporary closure techniques are very promising.

Table 1. Independent Predictors Identified by Multiple Logistic Regression

	ED Model			ICU Model		
	Independent Predictors	**OR**	**95% CI**	**Independent Predictors**	**OR**	**95% CI**
All ACS	Crystalloids ≥ 3L SBP < 86 mmHg	23.0 5.0	6.4 - 83.1 1.8 - 14.0	GAP_{CO2} Crystalloids ≥ 2.5L UO ≤ 150 ml Hb ≤ 8 g/dl CI < 2.6	>999 166 90 252 13	22 - > 999 4.7 - >999 4.5 - >999 10 - >999 1 - 154
1º ACS	To OR < 75 min Crystalloids ≥ 3L	103 70	10 - >999 10 - 478	Temp ≤ 34ºC GAP_{CO2} ≥ 16 Hb ≤ 8 g/dl BD ≥ 12 mEq/L	23 54 206 4	1.4 – 378 2.2 - >999 7.4 - >999 1.4 - 840
2º ACS	Crystalloids ≥ 3L No Urgent OR PRBC's ≥ 3 Units	16 0.3 5.6	1.7 – 144 0.07 – 0.9 1.0 - 31	GAP_{CO2} ≥ 16 Crystalloids ≥ 7.5 UO ≤ 150 ml	>999 39 64	>999 - >999 3 – 470 6 - 750

References:

1. Burch JM, Moore EE, Moore FA, Franciose R. The abdominal compartment syndrome. Surg Clin North Am. 1996;76:833-42.
2. Meldrum DR, Moore FA, Moore EE, Franciose RJ, Sauaia A, Burch JM. Prospective characterization and selective management of the abdominal compartment syndrome. Am J Surg. 1997;174:667-72
3. Swartz KR, Harrison MW, Campbell JR, Campbell TJ. Selective management of gastroschisis. Ann Surg. 1986;203:214-8.
4. Kron IL, Harman PK, Nolan SP. The measurement of intra-abdominal pressure as a criterion for exploration. Ann Surg. 1984;199:28-30.
5. Burch JM, Ortiz VB, Richardson RJ et al. Abbreviated laparotomy and planned reoperation for critically injured patients. Ann Surg.1992; 215:476-484.
6. Moore EE. Thomas G Orr Memorial Lecture: Staged laparotomy for the hypothermia, acidosis and coagulopathy syndrome. Am J Surg.1996;172:405-410.
7. Chen RJ, Fang JF, Chen MF: Intra-abdominal pressure monitoring as a guideline in the nonoperative management of blunt hepatic trauma. J Trauma 51:44-50, 2001.
8. Maxwell RA, Fabian TC, Croce MA, Davis KA. Secondary abdominal compartment syndrome: an underappreciated manifestation of severe hemorrhagic shock. J Trauma.1999; 47:995-999.
9. Kopelman T, Harris C, Miller R, Arrillaga A. Abdominal compartment syndrome in patients with isolated extraperitoneal injuries. J Trauma. 2000; 49:744-749.
10. Biffl WL, Moore EE, Burch JM, et al. Secondary abdominal compartment syndrome is a highly lethal event. Am J Surgery.2001;182:645-648.
11. Balogh Z, McKinley BA, Cocanour CS et al. Secondary Abdominal compartment syndrome: An elusive complication of traumatic shock resuscitation. Am J Surg 2002;184:538-543.
12. Balogh Z, McKinley BA, Holcomb JB, Miller CC, Cocanour CS, Kozar RA, Valdivia A, Ware DN, Moore FA. Both primary and secondary abdominal compartment syndrome can be predicted early and are harbingers of multiple organ failure. J Trauma. 2003;54:848-59.
13. Velmahos GC, Demetriades D, Shoemaker WC, et al: Endpoints of resuscitation of critically ill patients: Normal or supranormal? A prospective randomized trial. Ann Surg 232:409-418, 2000.
14. Ivatury RR, Porter JM, Simon RJ, et al. Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: prophylaxis, incidence, and clinical relevance of gastric mucosal pH and abdominal compartment syndrome. J Trauma 44:1016-1023, 1998
15. Sugrue M, D'Amours S. The problems with positive end expiratory pressure (PEEP) in association with abdominal compartment syndrome (ACS). J Trauma. 2001;51:419-20.
16. Botha AJ, Moore FA, Moore EE, Kim FJ, Banerjee A, Peterson VM. Postinjury neutrophil priming and activation: an early vulnerable window. Surgery 118:358-364, 1995.
17. Botha AJ, Moore FA, Moore EE, Sauaia A, Banerjee A, Peterson VM. Early neutrophil sequestration after injury: a pathogenic mechanism for multiple organ failure. J Trauma 39:411-417, 1995.
18. Partrick DA, Moore FA, Moore EE, Barnett CC Jr, Silliman CC. Neutrophil priming and activation in the pathogenesis of postinjury multiple organ failure. New Horiz 4:194-210, 1996.
19. Moore EE, Moore FA, Franciose RJ, Kim FJ, Biffl WL, Banerjee A. The postischemic gut serves as a priming bed for circulating neutrophils that provoke multiple organ failure. J Trauma 37:881-887, 1994.
20. Oda J, Ivatury RR, Blocher CR, et al. Amplified cytokine response and lung injury by sequential hemorrhagic shock and abdominal compartment syndrome in a laboratory model of ischemia-reperfusion. J Trauma 52:625-631, 2002.
21. Rezende-Neto J, Moore EE, Melo de Andrade MV, et al. Systemic inflammatory response secondary to abdominal compartment syndrome: stage for multiple organ failure. J Trauma 53:1121-1128, 2002.
22. 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, in press.
23. Sugrue M, Jones F, Lee A, et al. Intraabdominal pressure and gastric intramucosal pH: Is there an association? World J Surg 20:988-991, 1996.
24. Shelly MP, Robinson AA, Gensford JW, et al. Haemodynamic effects following surgical release of increased intra-abdominal pressure. Br J Anaesth 59:800-805, 1987.
25. Balogh Z, McKinley BA, Cocanour CS, Kozar RA, Valdivia A, Sailors RM, Moore FA. Supranormal trauma resuscitation causes more cases of abdominal compartment syndrome. Arch Surg. 2003;138:637-43.
26. Balogh Z, McKinley BA, Kozar RA, Cocanour CS, Moore FA. Patients with impending abdominal compartment syndrome do not respond to early volume loading. Am J Surg (in press)
27. Sugrue M, Bauman A, Jones F, Bishop G, Flabouris A, Parr M, Stewart A, Hillman K, Deane SA. Clinical examination is an inaccurate predictor of intraabdominal pressure. World J Surg 26:1428-1431, 2002.
28. Mayberry JC, Goldman RK, Mullins RJ, Brand DM, Crass RA, Trunkey DD. Surveyed opinion of American trauma surgeons on the prevention of the abdominal compartment syndrome. J Trauma 47:509-513, 1999.
29. Ertel W, Oberholzer A, Platz A, et al. Incidence and clinical pattern of the abdominal compartment syndrome after "damage control" laparotomy in 311 patients with severe abdominal and/or pelvic trauma. Crit Care Med 28:1747-1753, 2000.
30. Offner PJ, de Souza AL, Moore EE et al. Avoidance of abdominal compartment syndrome in damage-control laparotomy after trauma. Arch Surg 136:676-681, 2001.
31. Mcnelis J, Soffer S, Marini CP, Jurkiewicz A, Ritter G, Simms HH, Nathan I. Abdominal compartment syndrome in the surgical intensive care unit. Am Surg 68:18-23, 2002.
32. Hong JJ, Cohn SM, Perez JM et al. Prospective study of the incidence and outcome of the abdominal compartment syndrome. Br J Surg 89:591-596, 2002.
33. Raeburn CD, Moore EE, Biffl WL et al. The abdominal compartment syndrome is a morbid complication of postinjury damage control surgery. Am J Surg 182:542:546, 2001.
34. Garner GB, Ware DN, Cocanour CS, et al. Vacuum-assisted wound closure provides early fascial reapproximation in trauma patients with open abdomens. Am J Surg 2001;182(6):630-638.
35. Suliburk JW, Ware DN, Balogh Z, Moore FA, McKinley BA, Cocanour CS, Kozar RA. Vacuum Assisted Wound Closure allows for Early Abdominal Fascial Closure in Severely Injured Trauma Patients after Damage Control Laparotomy. J Trauma (in press)

Pathophysiology and early management of incomplete spinal cord injury

Jo Ollerton, Honorary Trauma Fellow, TraumaTrauma Department,
Liverpool Hospital, Liverpool, NSW 1871

Background

Spinal cord injury (SCI) is a devastating condition. Patients with a complete cord injury have a less than 5% chance of recovery in contrast to the incomplete cord syndromes where ultimately 90% of patients will return to their homes with some degree of independence. Currently the 5 year survival rate for patients with traumatic quadriplegia exceeds 90% (1,2).

Men account for three quarters of SCI patients with 80% between the ages of 18 and 25 years. Motor vehicle accidents and violence each account for around a third of all cord injuries with falls responsible for 20% and sporting incidents most of the remainder. Over half of all injuries are in the cervical spine and a third are thoracic spine, particularly at the thoraco-lumbar junction. This area is prone to trauma in deceleration injuries due to forming a pivot point between the relatively fixed thoracic cage and pelvic girdle.

SCI may be complete, in which case there is absolutely no function below the level of injury accounting for almost half of all SCI, or incomplete where a variable amount of function remains. The incomplete cord injuries are divided into syndromes based on the clinical findings as outlined in table one.

Damage to the cord may also be classified as primary or secondary. Primary injury to the spinal cord occurs as a direct result of the initial insult. Secondary causes of SCI include vascular compromise, hypoperfusion due to shock, and hypoxia which result in cascades of biochemical substances such as cytokines, glutamate and free- radicals acting as mediators of secondary cord injury.

Pathophysiology

Each level of the spinal cord has two anterior (motor) and two posterior (sensory) spinal nerve roots (figure 1). On either side the anterior and posterior nerve roots join to form the spinal nerve as it exits through the neuroforamina of the vertebral column. The cord extends from the base of the skull to the level of the L1 vertebral body where the spinal nerves continue distally as the Cauda Equina. An injury at this level is therefore not formally a SCI but a nerve or nerve root injury with lower motor neurone features as seen in the Cauda Equina Sydrome typified by areflexic bowel and bladder, with variable motor and sensory loss in the lower limbs and lower limb areflexia.

The spinal cord is organised into longitudinal tracts which carry sensory (ascending tracts) or motor (descending tracts) information between the peripheries and the cerebral cortex. The Corticospinal tracts are descending motor pathways which carry axons from the cerebral cortex through the Medulla where they decussate before continuing down to the relevant segment of the cord. Here they synapse in the anterior horn cells with the motor neurone, which exits in the anterior nerve root. The dorsal columns are ascending sensory pathways transmitting vibration, light touch and proprioception modalities along a similar path to the Corticospinal tracts. The Spinothalamic tracts are also sensory pathways transmitting pain and temperature through the lateral pathways and light touch in the anterior Spinothalamic tracts. Unlike other tracts these decussate within 3 segments of entering the cord before continuing up to the cerebral cortex. Autonomic fibres travel in centrally placed anterior tracts with the sympathetic nerves exiting the spinal column above T6 – relevant when considering the possibility of neurogenic shock in a trauma patient (injuries below T6 cannot cause neurogenic shock).

The blood supply to the cord is via the anterior spinal artery supplying the anterior 2/3 of the cord, and two posterior spinal arteries supplying primarily the dorsal columns. Both arise from the vertebral arteries at the base of the skull, branches from the aorta provide a collateral blood supply. Occlusion of the single anterior spinal artery at any level will result in loss of pain and temperature sensation, variable degree of paraplegia and autonomic dysfunction.

Neurogenic shock results when there is disruption of the cord above T6 affecting the autonomic pathways. The triad of hypotension, bradycardia and peripheral vasodilation occurs but may be masked by other concurrent injuries in the trauma patient. Hypotension must be assumed haemorrhagic until proven otherwise. There may be no tachycardia or peripheral vasoconstriction associated with massive haemorrhage as would usually be seen with intact spinal cord tracts. Search for occult haemorrhage in the abdomen, thoracic cavity and retroperitoneum with pelvic and long bone fractures excluded must be the first priority. Only when haemorrhage has been excluded should the hypotension be attributed to spinal cord injury.

Early Management

The immediate management of these patients

Table 1. Incomplete Spinal Cord Syndromes

Central Cord Syndrome	Associated with hyperextension injury in the elderly with degenerative changes in the cervical spine. It is the most common of the incomplete spinal cord syndromes. Antero-posterior compression of the spine by osteophytes causes contusion and ischaemic insult to the central cord. It may occur in all ages after a variety of mechanisms of injury. There is frequently a transient quadriplegia with the legs recovering first followed by bladder and bowel control then proximal muscle groups of the upper limbs. Residual motor impairment is worse in the upper limbs and more so distally. Sensory loss is variable.
Anterior Cord Syndrome	Anterior cord syndrome is characterised by bilateral symptoms with variable loss of sensation to pain, temperature and light touch, paralysis and autonomic dysfunction depending on the level of injury. The level of injury is described as the lowest level with intact neurology. The Dorsal Columns tend to be preserved with sensation to light touch, vibration and proprioception.
Brown-Sequard Syndrome	Brown-Sequard Syndrome is the clinical picture of injury to one side of the spinal cord. Frequently due to penetrating trauma. Results in ipsilateral paralysis / weakness with loss of light touch, vibration and proprioception. As the spinothalamic tracts cross on entering the spinal cord there is contralateral loss of sensation to pain and temperature – a dissociated sensory loss.
Conus Medullaris Syndrome	Conus Medullaris Syndrome describes injury to the sacral cord. There is a variable degree of inclusion of the lumbar nerve roots. Motor and sensory loss is therefore variable in the lower limbs with areflexia of the bladder and bowel.

*Cauda Equina Syndrome is sometimes included in this group though involves spinal nerves rather than the spinal cord

follows the accepted approach to all trauma patients as per ATLS and EMST courses (3,4). Prior to arrival of the patient the trauma team should be activated and the neurosurgeons informed. Radiology should be made aware of the likely need for investigation by CT scan and possibly MRI when available. Transfer of the patient pre-hospital should include the use of in- line stabilisation of the spine, hard cervical collar, and a long spinal board, oxygen must be administered en route to hospital. On arrival in the Emergency Department the airway is assessed with meticulous attention to in-line stabilisation of the spine, followed by breathing and circulation assessment with haemorrhage control (5) (figure 2).

Co-existing injuries will often warrant investigation and management before the spinal injury, of note 25% of acute SCI patients also have a serious head injury. Hypotension must be assumed due to haemorrhage until this is excluded. As mentioned above, the normal parameters of shock may be masked in the patient with autonomic dysfunction due to spinal injury.

Prevention of secondary injury to the head or spine must be attended by maintaining an adequate systolic blood pressure and preventing hypoxia. Haemodynamically significant bradycardia may be treated with Atropine and rarely needs Isoprenaline or cardiac pacing. Inotropes such as Dopamine may be required for hypotension once haemorrhage has been excluded as a cause.

Ileus is common in SCI so early placement of a nasogastric tube is imperative to prevent aspiration of gastric contents. The spinal board must be removed before or during the secondary survey as denervated skin is particularly prone to ulceration. All patient transfers thereafter must be by log-rolling the patient or use of the Jordan frame.

Methylprednisolone has been suggested as advantageous in SCI when used within 8 hours and probably more so when used within 3 hours (6,7). Until definitive evidence becomes available it is recommended to consult with local neurosurgical advice and to follow local hospital guidelines. A recent position statement based on a systematic review conducted by the Canadian Spine Society and the Canadian Neurosurgical Society concluded there was insufficient evidence to make methylprednisolone a standard of care. They recommended it should be a treatment option for use within 8 hours of acute closed spinal cord injury based on level II and III evidence (8). Methylprednisolone is recommended as a bolus of 30 mg/kg over 15 minutes followed by an infusion of 5.4 mg/kg /h over the next 23 hours.

A thorough neurological examination is mandatory and this must be repeated to assess for progress with time. SCI usually deteriorate over the first few hours or days so careful documentation is invaluable. Examination should include motor function with grading of power in each muscle group and sensory changes testing for light touch, pain, vibration and proprioception including examination of the perineum. Assessment of sacral sensation and anal tone with recognition of urinary or faecal incontinence is important. Clinical findings allow one of the spinal cord syndromes to be attributed to the patient. Improvement in clinical findings over time will be encouraging but deterioration may indicate pathology warranting surgical intervention and consideration of repeat imaging will be needed.

Plain radiographs in the acute stages of management will highlight fractures but do not exclude cord injury. CT scan will provide information on associated injuries and CT reconstruction will provide important detail on associated bony injury. MRI is the optimal investigation for imaging ligamentous and cord injury. MRI may show focal oedema, haemorrhage, necrosis, ischaemia or in some cases no abnormality despite clear clinical

Fig 1 Cross-section of the spinal cord to demonstrate the positions of the corticospinal, spinothalamic tracts and the dorsal columns.

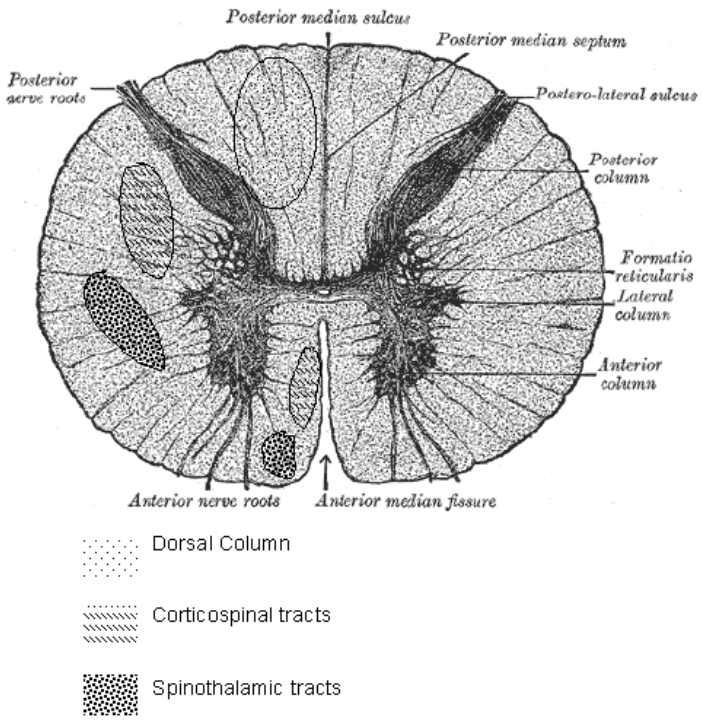
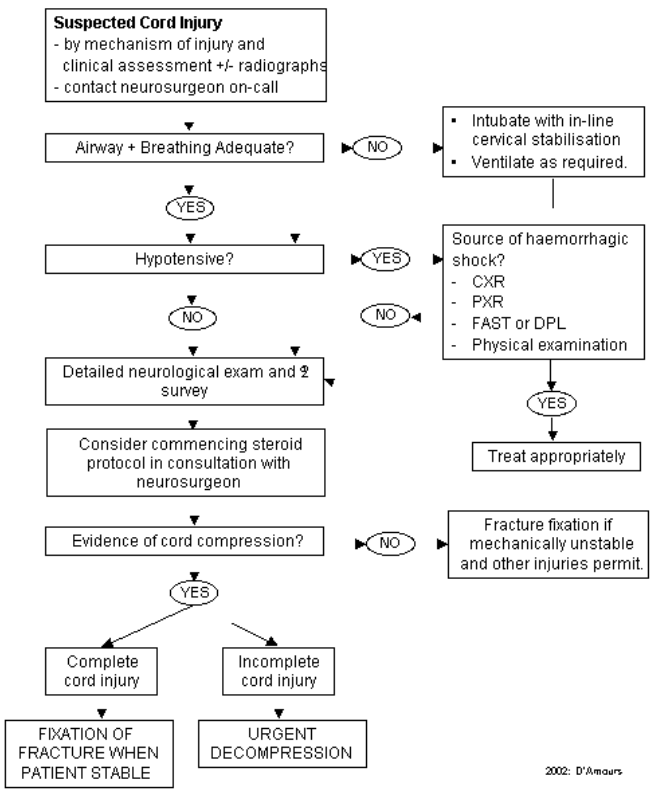


Fig 2. Management algorithm for suspected spinal cord injury



symptoms.
Radiological investigation will help to determine if surgical intervention is required. This may be evacuation of haematoma for spinal decompression and prevention of further damage, or immobilisation of the spine by internal or external fixation.
Depending on local policy regional spinal centres are usually best placed for ongoing treatment and rehabilitation which is often prolonged.

Prognosis
There is no cure for the incomplete spinal cord injury syndromes although aggressive rehabilitation currently enables 90% of patients to return to their homes with some degree of independence. Brown-Sequard has perhaps the best prognosis in this respect with 90% of patients able to walk independently and regain bowel and bladder function. Central cord syndrome has a variable recovery with 50% able to walk independently, usually with a degree of spasticity. Bowel and bladder control usually recovers but functional recovery of the upper limbs is often limited and fine motor control is usually poor. Anterior cord syndrome has the worst prognosis with only 10-20% of patients recovering functional motor control. Ongoing research includes exploring techniques to prevent localised cell damage after the initial injury by blocking apoptosis and efforts to repair the spinal cord based on embryonic stem cell propagation (2). Neural prostheses amplify power in weakened limbs in order to facilitate standing or enhance grip which promote independence.

Conclusion
Spinal cord injury is not common but frequently results in devastating disability requiring prolonged rehabilitation with limited potential for returning to the pre-morbid state. This is particularly relevant when it is considered that most of the victims are under 25 years old. A high level of suspicion and thorough clinical examination enables the diagnosis of spinal cord injury. Early recognition in the Emergency Department may prevent secondary injuries to the spinal cord and will allow rapid assessment with optimal ongoing management.

PITFALLS IN MANAGEMENT OF PATIENTS WITH SPINAL CORD INJURY

1. Missing subtle signs of SCI because of co-existing injuries. Assume there is spinal injury and immobilise the whole spine until SCI can be excluded.
2. Assuming hypotension is due to cord injury. SCI with autonomic dysfunction may obscure the usual signs of bleeding therefore first exclude haemorrhage as a cause of hypotension in trauma.
3. Assuming normal xrays indicate no spinal cord injury. Look for evidence of soft tissue injury as an indicator of significant spinal injury and request further imaging if SCI suspected.
4. Excluding spinal injuries in patients unable to give a history nor allow a full neurological examination. Unconscious, confused, agitated patients, or those under the influence of drugs and alcohol must be assumed to have a SCI until a complete examination is possible.

References

1. Burney RE, Maio RF, Maynard F, Karuna R. Incidence, characteristics and outcome of spinal cord injury at trauma centres in North America. Arch Surg 1993; 128:596-599

2. Schreiber D. eMedicine - Spinal Cord Injuries. http://www.emedicine.com/emerg/topic553.htm

3. American College of Surgeons Committee on Trauma. Advanced Trauma Life
Support (ATLS) Course Manual. Chicago: American College of Surgeons;1997.

4. Trauma Committee. Royal Australasian College of Surgeons. Early Management of Severe Trauma (EMST) Course Manual. Melbourne: Royal Australasian College of Surgeons;1992

5. D'Amours SK, Sugrue M, Russell R, Nocera N. Handbook of Trauma Care: The Liverpool Hospital Trauma Manual. 6th Edition. Sydney 2002.

6. Bracken MB, Shepard MJ, Collins WF et al. A randomised controlled trial of methylprednisolone or naloxone in the treatment of acute spinal cord injury. Results of the second National Spinal Cord Injury Study. N Eng J Med 1990;322:1405-1411

7. Nesathurai S. Steroids and spinal cord injury: revisiting the NASCIS 2 and NASCIS 3 trials. J Trauma 1998; 45: 1088-1093

8. Hugenholtz H, Cass DE, Dvorak MF, et al. High dose methylprednisolone for acute closed spinal cord injury: only a treatment option. Can J Neurol Sci 2002:29:227-235

Meetings

Definitive Surgical Trauma Care Course (DSTC)
Melbourne November 25th and 26th 2003
Sydney 28th and 29th July 2004
For both courses Contact: Michael Sugrue or Charmaine Miranda (61 2) 9828 3928
Email: charmaine.miranda@swsahs.nsw.gov.au

Austrauma 2004
February 20-21, 2004 Parramatta Sydney
Contact Greg Weir (61 2) 9845 7458

SWAN 12
SWAN 12 will be held on 30 and 31 July 2004
Bringing you nine world leaders in trauma care from overseas. Registration is limited, so get in early!
Contact: Thelma Allen
Email: thelma.allen@swsahs.nsw.gov.au
Phone: (61 2) 9828 3927
http:// www.swsahs.nsw.gov.au/livtrauma

World Congress on Abdominal Compartment Syndrome
December 6-8th Noosa Queensland 2004
Contact: Michael Sugrue or Charmaine Miranda (61 2) 9828 3928
Email:
charmaine.miranda@swsahs.nsw.gov.au

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