

ABRIDGED PRODUCT INFORMATION. INDICATIONS: 1. Prevention or treatment of shock associated with reduction in effective circulating blood volume due to: (a) haemorrhage (visible, concealed); (b) loss of plasma (burns, peritonitis, pancreatitis, crush injuries); (c) loss of water and electrolytes from persistent vomiting and diarrhoea. 2. As a plasma substitute in surgery where controlled haemodilution is employed. 3. Procedures involving extracorporeal circulation. 4. Carrier for insulin infusion. 5. Isolated organ perfusion. CONTRAINDICATIONS: Known hypersensitivity to constituents of Haemacel. Existing anaphylactoid reactions. WARNINGS: Infuse clear solutions only. Once the bottle is opened, the solution should be used immediately. Any unused contents should be discarded. There is a residual air volume in the container and bottle must be carried out under supervision. Administration of red cell intravascular fluid volume may be replaced by Haemacel alone. Haemacel may be given only after the prophylactic use of H1 and H2 receptor antagonists to the following patients. Patients with known allergic conditions such as asthma, a history of histamine response or patients who have received a histamine-releasing drug (such as anaesthetics, muscle relaxants, analgesics, anticholinergic ganglion blockers) within 7 days prior to Haemacel administration as they increase the risk of histamine release. USE IN PREGNANCY AND LACTATION: Haemacel for its usual indications, is not contraindicated in pregnancy. However, particular care should be exercised when fluid or volume replacements are administered during labour and no harmful effects on the newborn have been reported. It is not known whether poly-gelins is excreted in breast milk. ADVERSE EFFECTS: Transient skin reactions (urticaria, wheezing, bradycardia, nausea, rises in temperature and/or chills may occasionally occur. Rare cases of anaphylactoid reactions have been reported with bronchospasm, tachycardia and severe hypotension. Quincke's oedema has also been reported in such instances. These reactions are due to histamine release and may be result of the cumulative effect of not true anaphylactic reactions on an immunological basis. If side effects occur, the infusion should be discontinued immediately. Mild reactions: administer be given as follows: Mild reactions: administer reactions: if appropriate, immediately inject adrenaline (slowly I.V.), high doses of corticosteroid (slowly I.V.), volume replacement (e.g. human albumin, Ringer's lactate solution), oxygen and, if necessary, resuscitation. DOSAGE AND ADMINISTRATION: Haemacel is administered intravenously, and can be infused immediately. Adults and children: 1. Prevention or treatment of shock associated with reduction in effective circulating blood volume due to: (i) haemorrhage, blood loss up to 1500 mL - correct by use of Haemacel alone, blood loss in the range 1500-4000 mL - recommended ratio, Haemacel/whole blood is 1:1. blood losses above 4000 mL - recommended ratio Haemacel/whole blood is 1:2. The rate of infusion and total dose employed will be governed by clinical assessment. In acute situations of severe rapid blood loss, large volumes and rapid infusion may be required. The haematocrit should not be permitted to fall below 25 to 30 volume % during therapy. (ii) relative hypovolaemia. Normovolaemia and a high speed of Haemacel infusion are considered as factors contributing to anaphylactoid reactions in susceptible individuals. Where Haemacel is used to restore circulating blood volume in the absence of loss of intravascular fluid, the patient should be carefully observed for skin reactions, difficulty in breathing or precipitous fall in blood pressure. (iii) burns. The management of extensive burns should be undertaken by specialised units. The volume of Haemacel and crystalloid given should be varied according to the clinical response of the patient and the assessment of renal function. (iv) water and electrolytes. Haemacel may be used to restore deficiencies in circulating blood volume in conditions such as persistent vomiting and diarrhoea. 2. As a plasma substitute in controlled haemodilution. Autologous blood transfusion and haemodilution techniques involve the collection of 2-3 units of blood from the patient after the induction of anaesthesia, each unit being simultaneously replaced by 500 mL of Haemacel. During the operation, blood losses are immediately replaced with an equal volume of Haemacel, as long as the haematocrit falls above 0.25-0.30, or with blood below this level. 3. Procedures involving extracorporeal circulation machine, pump oxygenator, plasmapheresis and plasma exchange has been documented (see full Pre-cribing Information). 4. Carrier solution for insulin. Haemacel, added before the addition of insulin to the infusion fluid, minimises adsorption of insulin onto glassware and plastic delivery rate to be maintained. Concentrations as low as 0.5X (100 mL: Haemacel with 500 mL infusion solution) are effective. Iso-lated organ perfusion models has been documented. (Product Information). With impaired hepatic function - no modification necessary. With impaired renal function, Haemacel has a beneficial effect on renal function and no exacerbation of pre-existing renal disease need be expected. COMPATIBILITY: Provided sterile precautions are observed, Haemacel may be mixed with ordinary infusion fluids (saline, glucose, Ringer's solution, etc.) and with drugs acting on the cardiovascular system, corticosteroids, muscle relaxants, barbiturates, vitamins, streptokinase and antibiotics of the penicillin series, provided they are water soluble. Citrated blood (stored blood for transfusion) must not be mixed with Haemacel. Haemacel would cause reclassification if mixed immediately before or after an infusion of Haemacel. There is no objection to mixing heparinized blood with Haemacel. In common with all infusion fluids, Haemacel - for physiological reasons - should not be administered at a low temperature. PRESENTATION: Flexible plastic infusion bottle in 500 mL. REFERENCES: 1. Haemacel Product Information. 2. Silvey, J et al Journal of Thoracic and Cardiovascular Surgery (1968), 55:350-358. 3. Merikallio, E. Annales Chirurgiae et Gynaecologiae (1976), 65: 138-144. 4. Donahue, J.G., et al NEJM (1992), 327:367. 5. Dax, E., et al, MJA (1992), 157:69. Hoechst Marion Roussel Australia Pty. Ltd. ACN 008 558 807, Private Mail Bag 2067, Lane Cove 2066. Further information available on request. Haemacel® is a registered trademark of Hoechst AG.

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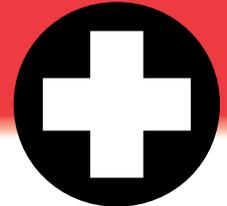
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TRAUMA

Grapevine



Introduction

This edition deals with two common sequelae of trauma, which often receive less attention than they should. These are the abdominal compartment syndrome and predicting patient's prognosis following brain injury. Dr Sheridan provides editorial comments on Head Injury prognosis on page 5.

Patients with serious injuries who undergo major surgery, to limb or abdominal cavity, are at risk of developing compartment syndromes. Compartment syndromes in the limbs are well recognised, as are their potentially serious sequelae with tissue, limb and even loss of life. The

abdominal compartment syndrome has, until recently, not attracted as much interest due to the hidden nature of the abdominal viscera and the potential multi-factorial nature of renal and visceral failure. We need to be more aware of the abdominal compartment syndrome, prevent where possible and treat in an early aggressive fashion.

Michael Sugrue

The Trauma Department at Liverpool Hospital would like to wish our colleagues and friends a very happy Christmas and a peaceful non-traumatic new century!



Abdominal Compartment Syndrome

M Sugrue FRCSI, FRACS

Summary

- ACS common especially after major abdominal surgery for trauma
- Diagnosis -Clinical supplemented by IAP>20mmHg and PaCo2>45mmHg
- Recognise major physiological sequelae of ACS
- Need to consider early abdominal decompression
- Abdominal decompression must be "aggressive"
- Not all patients are "cured" by decompression

INTRODUCTION

The Abdominal Compartment Syndrome (ACS) has become a topic of interest to surgeons and intensivists in the last few years. This article will provide an overview of the ACS, its frequency and implications in the management of trauma patients.

DEFINITION

The concept of the ACS was first reported by Fiestman in 1989, in four patients bleeding following aortic surgery.

To make the diagnosis you need at least three of the following:

- Appropriate clinical scenario (liver packing or large pelvic haematoma)
- Increased IAP (usually >20mmHg - often > 25mmHg)
- Increase in PaCo2> 45 mmHg
- Decrease in tidal volume and rise in airway pressure



Abdominal Compartment Syndrome

Continued

HOW DO YOU MEASURE IAP

The gold standard for IAP measurement involves using a urinary catheter. The patient is positioned flat in the bed. A standard Foley catheter is used with a T piece bladder pressure device attached between the urinary catheter and the drainage tubing. This piece is then connected to a pressure transducer, on-line to the monitoring system. The pressure transducer is placed in the mid axillary line and the urinary tubing is clamped. Approximately 50 mls of isotonic saline is inserted into the bladder via a three way stopcock. After zeroing, the pressure on the monitor is recorded.

TIPS FOR IAP MEASUREMENT

- A strict protocol and staff education on the technique and interpretation of IAP is essential.
- Very high pressure (especially unexpected ones) are usually caused by a blocked urinary catheter
- The size of the urinary catheter does not matter
- The volume of saline instilled into the bladder is not critical.
- A central venous pressure (CVP) manometer system can be used but it is more cumbersome than on-line monitoring.
- Elevation of the catheter and measuring the urine column provides a rough guide and is simple to perform.
- If the patient is not lying flat, IAP can be measured from the pubic symphysis.

PATHOPHYSIOLOGY

The incidence of increased IAP (which we take as >18mmHg) is 30% of post operative general surgery patients in ICU. After emergency surgery the incidence is even higher. The causes of acutely increased IAP are usually multifactorial. The first clinical postoperative reported cases of increased IAP were often after aortic surgery with postoperative haemorrhage from the graft suture line. In patients with peritonitis and intra-abdominal sepsis, tissue oedema and ileus is the predominant cause of increased IAP. Raised IAP in trauma patients is often due to a combination of both blood loss and tissue oedema. Trauma and the surgery of trauma are one of the commonest subsets of patients to develop intra-abdominal hypertension and the ACS.

The common causes of increased IAP are shown below.

CAUSES OF INCREASED IAP

- Tissue oedema secondary to insults such as ischaemia and sepsis
- Ileus
- Intraperitoneal or retroperitoneal haematoma
- Ascites
- Pneumoperitoneum

EFFECT OF RAISED IAP ON INDIVIDUAL ORGAN FUNCTION

Renal

In 1945, Bradley in a study of 17 volunteers demonstrated that there was a reduction in renal plasma flow and glomerular filtration rate (GFR) in association with increased IAP. In 1982, Harman showed as IAP increased from 0 to 20 mmHg in dogs, the GFR decreased by 25%. At 40 mmHg, the dogs were resuscitated and their cardiac output returned to normal. However their GFR and renal blood flow did not improve, indicating a local effect on renal blood flow. The situation in seriously ill patients may, however, be different and the exact cause of renal dysfunction in the ICU is not clear, due to the complexity of

critically ill patients. In a recent study, we found that out of 20 patients with increased IAP and renal impairment, 13 already had impairment before the IAP increased.

The most likely direct effect of increased IAP is an increase in the renal vascular resistance, coupled with a moderate reduction in cardiac output. Pressure on the ureter has been ruled out as a cause, as investigators have placed ureteric stents with no improvement in function. Other factors which may contribute to renal dysfunction include humeral factors and intra-parenchymal renal pressures.

The absolute value of IAP that is required to cause renal impairment is probably in the region of 20mmHg. Maintaining adequate cardiovascular filling pressures in the presence of increased IAP also seems to be important.

Cardiovascular

Increased IAP reduces cardiac output as well as, increasing central venous pressure (CVP), systemic vascular resistance, pulmonary artery pressure and pulmonary artery wedge pressure. Cardiac output is affected mainly by a reduction in stroke volume, secondary to a reduction in preload and an increase in afterload. This is further aggravated by hypovolemia. Paradoxically, in the presence of hypovolemia an increase in IAP can be temporarily associated with an increase in cardiac output. It has been identified that venous stasis occurs in the legs of patients with abdominal pressures above 12 mmHg. In addition, recent studies of patients undergoing laparoscopic cholecystectomy show up to a four-fold increase in renin and aldosterone levels.

Respiratory

In association with increased IAP, there is diaphragmatic stenting, exerting a restrictive effect on the lungs with reduction in ventilation, decreased lung compliance, increase in airway pressures, and reduction in tidal volumes.

In critically ill ventilated patients the effect on the respiratory system can be significant, resulting in reduced lung volumes, impaired gas exchange and high ventilatory pressures. Hypercarbia can occur and the resulting acidosis can be exacerbated by simultaneous cardiovascular depression as a result of raised IAP. The effects of raised IAP on the respiratory system in ICU can sometimes be life-threatening, requiring urgent abdominal decompression. Patients with true ACS undergoing abdominal decompression demonstrate a remarkable change in their intra-operative vital signs.

Visceral Perfusion

Interest in visceral perfusion has increased with the popularisation of gastric tonometry and there is an association between IAP and visceral perfusion as measured by gastric pH. This has been confirmed recently in 18 patients undergoing laparoscopy where reduction of between 11% and 54% in blood flow was seen in the duodenum and stomach respectively at an IAP of 15 mmHg. Animals studies suggest that reduction in visceral perfusion is selective, affecting intestinal blood flow before, for example, adrenal blood flow. We have demonstrated in a study of 73 post-laparotomy patients that IAP and pH are strongly associated, suggesting that early decreases in visceral perfusion is related to levels of IAP as low as 15 mmHg.

Intracranial Contents

Raised IAP can have a marked effect on intracranial pathophysiology and cause severe rises in intracranial pressure (ICP).

Review of last issue's case of the Month

Remember - the 28 year old driver of a truck trapped at scene?

Pre-Hospital information

Trapped Hypotensive and received 2.2L Colloid

Resus Room:

Primary Survey

- A Airway at risk GCS (6)
- B Breathing - RR 38/m
Sats 92%
- C Pulse 160/m
BP not palpable
Bleeding from compound right femur
No other external blood loss
- D Disability GCS falling to 4.

As part of the primary survey, the patient was simultaneously intubated and bilateral large IV cannulas inserted. These were converted to rapid infusion devices with administration of "O" Negative blood through a rapid infusion

blood administrator on the left arm and warm fluids on the right arm. In the initial 15 minutes, x-rays of chest, c-spine and pelvis have been taken. A diagnostic peritoneal lavage was in the process of being done. A urinary catheter was passed. There is obvious bleeding (but not torrential) from the right compound femur fracture. The abdomen looked a little bit distended. The chest x-ray, which has returned, revealed no gross haemothorax on either side. Blood pressure improved to 75 systolic, Pulse 140/m after receiving resuscitation fluids: 3 units of blood, 2.7 litres of crystalloid and colloid coupled with 2.2 litres of colloid pre-hospital.

What would you do now?

Organise an urgent laparotomy aiming to have the patient in theatre in 8-10 minutes proceeding with the DPL to confirm your suspicions of an intra-abdominal injury. A check on the pelvic xray is essential, to ensure that there is not a significant fracture.

What are the key factors that are going to effect the patient's outcome? What are your time frames to definitive care?

Stop the bleeding

Amount of Blood Loss

Hypothermia

Appropriately swift surgery

Intra-operative monitoring blood gases, potassium and temperature

Patient's outcome

The patient proceeded to urgent laparotomy and major mesenteric tear 6 in all were actively bleeding. In 20 minutes these were controlled, the patient had a BP 90, temperature of 32.8°C and pH of 7.02 having received 12 units of blood. The bowel that was not viable was resected and the end stapled without anastomosis. With no major site of blood loss in the abdomen, temporary abdominal closure was performed with some abdominal packing. The patient left hospital 4 weeks later having had 2 further abdominal operations prior to discharge.

Case of the Month

62 year Sheet Metal Worker

Pre-Hospital information

- (M) Mechanism Crushed 500kg press for 20 seconds
- (I) Injury Chest, Abdomen,
- (S) Signs RR 28/m, P 100/m, BP 80 mmHg, GCS 15
- (T) Treatment Oxygen, C Collar, Spinal Protection, IV Fluids 1 L Haemaccel

Resus Room:

Primary Survey

- A Talking
- B Breathing - RR 28/m
Sats 95%
- C Pulse 60/m
BP 140/90
No external blood loss. Looked awful!
- D Alert GCS 15

Secondary Survey

Laceration to nose
Tender lower right and left rib cage
Upper abdomen mild tenderness

CXR fractured 7th, 8th, 9th right ribs

The plan was to observe and organise a CT abdomen. The patient was a known diabetic and had a previous CABG, At 27 minutes he dropped BP to 100mmHg and Pulse remained at 68/m. A further 500 of Haemaccel was administered and a second episode of hypotension (BP to 98mmHg) occurred at 43min. A FAST examination revealed free fluid in Morrison Pouch and suprapubically. He looked well and had significant pain in his lower ribs

Where to from here? CT scan? Laparotomy? Find out next time.

Meetings

Humanitarian Aspects of Trauma: A Worldwide Perspective
December 7th 17.00-18.00.
Speakers Dr Adam Kushner, San Antonio Texas, Drs John Crozier and Kevin Baker Liverpool Hospital
Education Centre Liverpool Hospital. Admission is free but booking via Thelma, 9828 3928.

Trauma Education Evening
February 3rd 16.00-20.00. Education Centre Liverpool Hospital



Definitive Surgical Trauma Care Course (DSTC) Sydney
(For Surgeons Only)
1st and 2nd August, 2000
Email: michael.sugrue@swsahs.nsw.gov.au

SWAN 8

SWAN 8 will be held on the 4th and 5th of August 2000, bringing to you a number of world leaders in trauma care from overseas. Sorry registration is limited, so get in early! Contact: Thelma Allen
Email: thelma.allen@swsahs.nsw.gov.au
Phone: 02 9828 3927
<http://www.swsahs.nsw.gov.au/livtrauma>

BACKCHAT

Congratulations to Dr Silent Tovosia on becoming the first EMST Instructor for the Solomon Islands and to Dr Eddie McCaig on becoming the first EMST Instructor for Fiji.



What's New in Trauma

Alex Perez, visiting trauma student from Bogoto Colombia reviews 3 recent articles.

Adult versus paediatric prehospital trauma care: Is there a difference?
Tim R. Paul and colleagues
Denver Health Medical Center, Denver, Colorado
The Journal of Trauma 47; 3: 455-459 1999

Dr. Paul and colleagues confront the topic of paediatric trauma care performed at adult trauma care centers. They wish to analyse data that indicate a trend toward raised mortality in paediatric trauma. To accomplish this the authors focus on:

- Intravenous access (I.A.)
- Endotracheal Intubation (E.I.)
- Survival

This study reveals that the paediatric population in trauma care differs with that in adult trauma care in that:

Paediatric vs Adult

Male	59%	71%
Blunt	94%	80%
Penetrating	6%	20%
MVA	25%	41%
Falls	16%	10%
Pedestrian	32%	7%
<u>Injury Severity Score</u>		
(1 - 15)	92%	86%
(16 - 25)	6%	10%
(> 25)	2%	4%
<u>Ambulance</u>		
Response Time	6.6 min	6.4 min
Scene Time	10.5 min	10.3 min
Return Time	8.2 min	7.4 min
<u>Survival</u>		
Blunt	99%	97%
Penetrating	93%	87%

There were no significant differences between prehospital procedures such as endotracheal intubation and intravenous access times.

Contrary to previous reports no substantial difference could be detected when comparing mortality in minor and moderate injuries in the paediatric population and this same group subjected to severe trauma were more likely to survive when compared to their adult counterparts.

Comment

Paediatric patients comprise 10 % of all paramedical calls. Trauma care providers can be reassured by this analysis that the service that they are providing is optimal.

Control of splenic bleeding by using high intensity ultrasound

Shahram Vaezy and colleagues
University of Washington, Seattle, Washington
The Journal of Trauma 47; 3: 521-525, 1999

Dr. Vaezy and colleagues describe an innovative technique where high intensity focused ultrasound (H.I.F.U.) is use to cease blood loss.

The H.I.F.U. system implements a sharply focused ultrasound beam that when delivered to a bleeding site, splenic tissue in this case, achieves cauterisation through two pathways:

- Thermal
- Mechanical

The first entails an increase in tissue temperature up to values of 70°C at the focused area which produces haemostasis through coagulation necrosis and local accumulation of fibrin. The second method uses a mechanical plug effect through the homogenisation of the desired tissue. The porcine population under surveillance maintained stable vital signs throughout the entire experiment.

The following data is obtained comparing the time required to achieve total haemostasis with H.I.F.U., conventional surgery (a combination of suturing, electrocautery, digital pressure and application of oxidised regenerated cellulose) (C.S.) and argon beam coagulators (A.B.C.):

System	Time
H.I.F.U.	55 +/- 22 sec
C.S.	546 +/- 85 sec
A.B.C.	77 +/- 16 sec

This system showed that there was a complete cessation of haemorrhage within seconds and there was no rebleeding evidenced 2-4 hours after initial use.

Comment

This is a must-read article for those who are on the look-out for what the future might possibly look like in operative and non-operative management of not only splenic trauma but also surgery in general.

Staged physiologic restoration and damage control surgery

Ernest E. Moore and colleagues, Denver Health Medical Center, Colorado
World Journal of Surgery, 1998; 22: 1184-1191

Dr. Moore and colleagues analyse definitive operative management of patients with massive acute abdominal injuries and patients

undergoing elective operative procedures. The article centres itself on staged laparotomy and compartmentalise this procedure into five phases of management which include:

- Patient selection
- Intraoperative reassessment
- Physiologic restoration in the surgical intensive care unit
- Return to the operating room for definitive procedures
- Abdominal wall closure and reconstruction.

The indications for abbreviated laparotomy include:

- Unachieved haemostasis
- Inaccessible major venous injury
- Time consuming procedures
- Extraabdominal life-threatening injury
- Reassessment of intraabdominal contents
- Inability to reapproximate abdominal fascia

The coagulopathic risk grading system uses:

- Injury severity score (I.S.S.)
 - Systolic blood pressure (S.B.P.)
 - Arterial blood pH
 - Core body temperature (C.B.T.)
- With an I.S.S. > 25 + C.B.T. < 34 (C there is a 49% probable risk of developing a life-threatening coagulopathy while the former + S.B.P. <70 mmHg + pH < 7.10 has a 98% probability.

A system for grading Abdominal Compartment Syndrome (A.C.S.) is described demonstrating that a grade I is equivalent to a urinary bladder pressure (U.B.P.) of 10-15 mmHg and requires maintenance of normovolemia while a grade III with a U.B.P. of 26-35 mmHg requires decompression.

Organ dysfunction as seen in terms of:

- Urinary output of < 0.5 ml/kg/hr
- Pulmonary artery pressure > 45
- Systemic vascular resistance > 1000
- Oxygen delivery index of < 600

These are not frequent in grade I while it is common to see 65%, 78%, 65% and 57% compromise respectively in grade III A.C.S.

Comment

Dr. Moore and colleagues venture into the realm of damage control surgery and produce a thought provoking article with clear practical implications for everyone who is involved in trauma and also a vast array of practitioners who have critical patients in their care.



TREATMENT

General Support

The precise management of IAP remains somewhat clouded by many published anecdotal reports and uncontrolled series. Aggressive non-operative intensive care support is critical to prevent the complications of ACS. This involves careful monitoring of the cardiorespiratory system and aggressive intravascular fluid replacement.

Reversible Factors

The second aspect of management is to correct any reversible cause of ACS, such as intra abdominal bleeding. Massive retroperitoneal hemorrhage is often associated with a fractured pelvis and consideration should be given to measures which would control hemorrhage such as pelvic fixation or vessel embolization. In some cases, severe gaseous distension or acute colonic pseudo-obstruction can occur in ICU patients. This may respond to drugs such as neostigmine but if it is severe, surgical decompression may be necessary. A common cause of raised IAP in ICU is related to the ileus. There is little that can be actively done in these circumstances apart from optimizing the patient's cardiorespiratory status and serum electrolytes.

Remember the ACS is often only a symptom of an underlying problem. In a prospective review of 88 post-laparotomy patients, we found those with an IAP of 18 mmHg had odds increased ratio for intra-abdominal sepsis of 3.9 (95%CI 0.7-22.7). Abdominal evaluation for sepsis is a priority and this obviously should include a rectal examination as well as investigations such as ultrasound and CT scan. Surgery is the obviously mainstay of treatment in patients whose rise in IAP is due to postoperative bleeding.

Surgery for raised IAP

As yet, there are few guidelines for exactly when surgical decompression is required in the presence of raised IAP. Some studies have stated that abdominal decompression is the only treatment and that it should be performed early in order to prevent ACS. This is an overstatement and not supported by level 1 evidence.

The indications for abdominal decompression are related to correcting pathophysiological abnormalities as much as achieving a precise and optimum IAP.

Wittman, in two separate studies in 1990 and 1994 prospectively evaluated outcomes in 117 and 95 patients respectively. A multi-institutional study of 95 patients concluded that a staged approach to abdominal repair, with Temporary Abdominal Closure (TAC) was superior to conventional techniques for dealing with intra-abdominal sepsis. Torrie and colleagues from Auckland reported their experience with 64 patients (median APACHE II score 21) undergoing TAC and found the mortality to be 49%.

Indications for performing TAC include:

- Abdominal Decompression
- Facilitate Re-exploration in abdominal sepsis
- Inability to close the abdomen
- Prevention of abdominal compartment syndrome

A large number of different techniques have been used to facilitate a temporary abdominal closure, including IV bags, velcro, silicone and zips. Whatever technique, it is important that effective decompression be achieved with adequate incisions.

TIPS FOR SURGICAL DECOMPRESSION FOR RAISED IAP

- Early investigation and correction of the cause of raised IAP
- On-going abdominal bleeding with raised IAP requires urgent operative intervention
- Reduction in urinary output is a late sign of renal impairment. Gastric tonometry may provide earlier information on visceral perfusion
- Abdominal decompression requires a full length abdominal incision
- The surgical dressing should be closed using a sandwich technique using 2 suction drains placed laterally to facilitate fluid removal from the wound
- If the abdomen is very tight, pre-closure with a silo should be considered

Unfortunately, clinical infection is common in the open abdomen and the infection is usually polymicrobial. Particular care needs to be taken in patients post-aortic surgery as the aortic graft may become colonized. The mesh in this situation should be removed and the abdomen left open. It is desirable to close the abdominal defect as soon as possible. This is often not possible due to persistent tissue edema.

Figure 1:

Shows the stages in the fashioning of a silo. In this case PTFE is the material, but any sterile material is adequate. Porous material is better.



FUTURE

The concept of IAP measurement and its significance is increasingly important in the ICU and is rapidly becoming part of routine care. Patients with raised IAP require close and careful monitoring, aggressive resuscitation and a low index of suspicion for requirement of surgical abdominal decompression.



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SEVERE BRAIN INJURY "WHAT HAPPENS TO THEM?"

Influences on prognosis

Karen Woods Nurse Educator

SCENARIO

A 20 year old male arrives in the emergency department following a motor vehicle accident. He is not obeying commands, there is nil eye opening, he is moaning and groaning and extending to painful stimuli.

A C-T reveals a 2 cm right extradural haematoma with midline shift, right frontal skull fracture extending into the roof of the right orbit. He has also sustained a right rib fracture with pulmonary contusions and a fractured left pubic rami. He is transferred to operating theatre for an emergency burr hole and evacuation of the extradural haematoma. The nature and circumstances of his injury also suggests diffuse axonal injury.

Following surgery, he is transferred to the ICU where he spends the next 17 days.

At some stage in the weeks following this trauma, the emergency department staff are bound to ponder "I wonder what happened to that young guy who...?" Assumptions and prediction may be made as to his outcome. These will probably be based on his age, the location and apparent severity of his injury, the timeframe from injury to medical intervention and his initial Glasgow Coma Score.

Accurate prediction of physical and cognitive outcomes for a severely traumatic brain injured person in the days immediately following injury is difficult. Prognosis made in those early days are sometimes contradicted by positive functional outcomes seen a number of months later.

The challenges to the prediction of outcome in this group arises from the fact that the brain injury population is not a homogeneous group. Observation of a widespread variation in disability and function amongst the severely brain injured population demonstrates this and highlights the need for refinement of how we define severity and make judgements about these patients' outcome.

The way we define severity in brain injury varies considerably. A review of the literature reveals a lack of consensus as to a definition of 'severe' traumatic brain injury. Definitions range from: a period of coma lasting 6 hours to a period of greater than one hundred days; an initial Glasgow coma score between 3-8; or a length of post-traumatic amnesia greater than 7 days.

The indicators used in brain injury rehabilitation vary from those used in the acute areas. Length of Post-Traumatic Amnesia (PTA) is used to define severity and Functional Independence Measure (FIM) scores are used to categorise physical and cognitive function at various stages of recovery.

Some researchers have suggested that length of post-traumatic amnesia is the most reliable predictor of severity in brain injury. Haslam et al research demonstrates that speed of information processing was best predicted by the duration of PTA.

When patients make slow or very little improvement in the first few weeks post injury, it is often believed that the patient will make no further recovery. On occasions, personal judgements can lead to errors in placement of severely brain injured patients in nursing homes without the opportunity for brain injury rehabilitation referral or assessment.

With anticipation of no further recovery, persons experiencing prolonged coma are occasionally sent to a nursing home. Sazbon and Grosswater (1990) examined 134 patients with prolonged post-traumatic unawareness. Fifty-four percent showed a return to consciousness. Seventy-two percent of those who regained consciousness returned home and 48% regained self-care. A review of the Liverpool Brain Injury unit database revealed approximately 3 % of patients admitted to the Liverpool Brain Injury Unit are discharged to nursing homes. Leading to the conclusion that only a small percentage experienced prolonged unawareness and require this type of placement.

So, if the outcome of the severely brain injured population is not a life in a state of prolonged coma or unawareness then what is it?

The functional outcomes of the severe traumatic brain can be categorised into cognitive, physical, emotional and behavioural types. Cognitive problems are characterised by memory disturbances, perception problems and impaired judgement. All of these can impact on work performance, family harmony and social interactions. Post-traumatic Amnesia (PTA) refers to a period following a traumatic brain

*Fifty-four percent showed a return to consciousness.
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injury where the person is confused, disorientated and unable to lay down new memory.

Functional Independence Measure (FIMs) is a 'measure of disability... (which) assesses the need for assistance... and the amount of assistance required for a person with a disability to perform basic life activities effectively'.

Mobility and physical dexterity are affected by the presence of a

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iction of outcome

Brain Injury Unit Liverpool Hospital

hemiplegia, limb spasticity and dysfunction of balance. Communication problems can manifest as dysfunction in the expression and the

Staff in the Trauma, Intensive Care, acute neurosurgical and brain injury settings are often pleasantly surprised and heartened by the improvements made by individuals who have suffered severe traumatic brain injury.

interpretation of speech. Aphasia or dysarthria may be present.

Psychiatric and emotional problems can also appear following severe traumatic brain injury. A retrospective random sample of 119 Liverpool Brain Injury Unit community clients revealed 16% had developed a mental illness following brain injury which was not present prior to brain injury. Psychiatric illnesses following brain injury can range from manifestations of depression to psychotic disorders. Behavioural changes affecting anger management and the ability to curtail socially inappropriate behaviour can also be present in the severely brain injured population.

The above mentioned problems may occur in isolation of one another or in a variety of combinations. They can therefore impact with varying degrees on the functioning of the individual in their work, family and social environments.

"So what did happen to that guy...?"

Thirty-three days following his injury he was transferred to the Liverpool Brain Injury Unit. On arrival he was disorientated to person, place and time but was following one stage commands. He was moving all limbs but not mobilising due to drowsiness and ataxia. He was dysarthric and dysphasic.

He spent 61 days in post-traumatic amnesia which is indicative of a severe traumatic brain injury.

Comment on Severe Brain Injury

This article is an important reminder to everyone caring for patients with severe head injuries that outcome, especially during the early stages of the disease process, is extremely difficult to predict. Too often a feeling of pessimism prevails when dealing with head injuries in the acute phase because the trauma team only deal with the patient when they are at their worst. The weeks and months of intensive rehabilitation therapy that often leads to a functional outcome takes place out of sight of the acute care setting. Because of this, it becomes all too easy to lose sight of the long term successes we achieve thanks

By his discharge on day 90 he was alert and orientated, mobile and self-caring. At home he meets all his ADL's but has been experiencing problems with word finding and lack of motivation. He occasionally gets irritable if the environment becomes too noisy. He is about to undergo driving assessment for return of drivers' licence and hopes to return to his previous employment as a truck driver.

Staff in the trauma, intensive care, acute neurosurgical and brain injury settings are often pleasantly surprised and heartened by the improvements made by individuals who have suffered severe traumatic brain injury. It is recommended that all these individuals are referred to a brain injury rehabilitation service. These peoples lives and those of their families will never be the same again but the provision of rehabilitation can impact on the degree of services they receive which can improve their quality of life.



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to the early and aggressive treatment of these injuries. The temptation to prognosticate too soon and make judgements on possible quality of life issues after only days or even a few weeks post injury must be avoided. We must remember that the contribution of rehabilitation in achieving the progressive improvement in outcome from head trauma is as important as the application of early surgical intervention and intensive care treatment.

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