Vol 3 No 1 April - June 2001

TRAUMA Grapevine

Contents:

- C Spine Injury
- Blunt Cardiac Injury
- Survey of Quality Trauma Care
- Case of the Month
- Meetings

Introduction

The recent passing of Dr. David Sloane after a battle with cardiovascular disease, has resulted in the loss of a foundation stone in established trauma care systems in New South Wales. David established the Department of Trauma at Liverpool Hospital. As a Trauma Director, he drove forward trauma care in Australasia. He will be sadly missed by us all, his wife Avril and his family.

It gives us an opportunity to reflect on Trauma Director positions, their performance and achievements. Who should be a Trauma Director, what should the goals of trauma leadership be within the current Australian system? Should it be to provide leadership with better practice guidelines, effective QA, establish research, education and ensure that our patients outcomes are optimised. A formal program for the development of trauma care must include structured Trauma Directorships within our Major Trauma Services. Trauma Directors working fulltime towards optimising delivery of care within a framework that encourages achievement of key performance indicators with empowerment to achieve, like David Sloane did. The issues of Trauma Directorships and their career path need to be clearly addressed by the Colleges and Department of Health. It is worth reflecting on the results of a survey of the perception of quality in trauma care undertaken at the Injury 2000 meeting in Canberra, reported in this issue.

In recent months there have been a number of important developments relating to trauma care in NSW. The Trauma Systems Advisory Committee have recommended the reduction in number of Major Trauma Services, recognising an over supply. This has been supported by the independent consultative evaluation of critical care services. In addition, it has been recommended that a Trauma Institute be established in NSW. The Ambulance Service has undergone a major restructuring aimed to improve quality assurance and delivery of care.

The Trauma Grapevine has now become a peer review journal and would like to welcome our new editorial board: Scott D'Amours, Trauma Surgeon Liverpool Hospital, Tim Hodgetts, Professor Emergency Medicine Birmingham; Kenth Johannson, General and Trauma Surgeon Vastervik Sweden; Karel Kolkman, Trauma Surgeon Holland; Martin Jones, Rural Surgeon Nowra NSW; Kathy Martin, Trauma Co-ordinator Philadelphia USA; Michael Sugrue, Trauma Surgeon Liverpool; Don Trunkey, Chair of Trauma, Oregon USA.
Papers can be submitted for consideration for publication, in Vancouver style at the recommendation of the International Committee of Medical Journal Editors, http://www.icmje.org.

Michael Sugrue

Upper Cervical Spine Trauma

Cervical spine fractures are not uncommon, in the US the incidence ranges from 1.5% - 12% with up to 2-3% of all trauma patients sustaining spinal cord injury.[1,2] The majority of patients are male, between 15-24 years old and have been involved in a vehicular or a sporting accident such as rugby or diving. Rather worryingly, of those with C.spine injury, 3-25% suffer an extension of those injuries from delays in diagnosis or unwarranted manipulation in the emergency department.[3,4]

One should suspect a C.spine injury in the following circumstances:

1. Bony pain, bruising, step deformity at the midline posteriorly
2. Injury above the clavicle
3. Unconscious patient/altered GCS
4. Multisystem injured patient/distracting injuries
5. Patient intoxicated with drugs/alcohol
6. Signs/symptoms of neurogenic shock
7. Neurological symptoms/signs
8. Mechanism: Gunshot wounds above the clavicle, hanging, falls, diving or horse riding accidents, MVA's with ejection or rapid deceleration.

Assessment and Resuscitation

The pathophysiology of spinal cord injury that follows cervical spine trauma is analogous to CNS injury, in that one can divide the injury into a primary and secondary insult. Although the primary insult due to

Continued next page

Upper Cervical Spine Trauma

Continued

energy transfer down the spine can't be reversed, the aim of accurate assessment and resuscitation is to prevent a secondary injury due to hypoxia, swelling or mechanical injury. Therefore according to the ATLS algorithm, the airway should be maintained with high flow oxygen delivery, the spine immobilised with a collar, log rolling and perfusion optimised.

Spinal cord injury can have a significant effect on the physiological status of the patient, whether it be from ensuing neurogenic shock or as a result of the spinal lesion. As such, initial management focuses on the consequences of spinal cord injury first and addresses the bony abnormalities once the patient has been stabilised.

Acute management

Respiratory complications:

Patients with lesions below C5 are generally able to ventilate adequately, unless they have superimposed chest problems. Higher lesions produce intercostal muscle paralysis and diaphragmatic breathing. This reduced vital capacity together with pulmonary insults such as lung contusion or pneumothorax can easily tip the balance into respiratory failure and so high flow oxygen (+/- artificial ventilation) with close monitoring of the respiratory rate, oxygen saturation and vital capacity are vital.

Circulatory complications:

Neurogenic shock creates a hypotensive, hypothermic, bradycardic picture due to the loss of sympathetic drive below the lesion and an increased parasympathetic stimulation of the vagus nerve. Hypotensive management includes fluid, vassopressor and ionotrope resuscitation with early screening for a haemorrhagic cause for the hypotension. Atropine should be at hand during oropharyngeal suction and endotracheal intubation and administered if the BP<80 systolic or the HR<35[5]. Careful fluid replacement with close urinary output monitoring is necessary to avoid pulmonary oedema and respiratory failure.

Abdominal complications:

Major abdominal trauma may be difficult to detect in complete cord injuries because of the absence of pain or guarding. However, mechanism of injury, abdominal bruising and haematuria together with a FAST/DPL or abdominal CT may indicate visceral trauma or alternative reasons for hypotension. Bowel sounds persist into the early stages of cord injury although ileus almost always develops eventually. As such the patient should remain nil by mouth and with a nasogastric tube if they become distended, as this can hinder diaphragmatic breathing.

Neurological complications:

After the initial resuscitation, the spinal column and the neurological status of the patient need careful assessment. The secondary survey should incorporate a complete inspection and palpation of the entire spine to identify areas of bony tenderness, swelling, bruising, step deformity and interspinous gap widening. The importance of the PR examination should be stressed as preserved anal sensation and tone may be the only indication that the spinal cord lesion is incomplete and potentially recoverable with urgent decompression. Following this, motor, sensory and reflex testing is essential, however, with the unconscious patient, the response of each limb to deep pain should be recorded as a gross estimate to limb power and sensation.

Major abdominal trauma may be difficult to detect in complete cord injuries because of the absence of pain or guarding.

Uncertainty surrounding the efficacy of high dose methylprednisolone in acute spinal injury persists. It is believed that corticosteroids mop up free radicals and provide membrane stabilisation to partially damaged nerves. However, the protocol outlined by the NASCIS-2 trial of 30mg/kg over 30 mins IV within 8 hours of the injury, followed by 5.4mg/kg/hour IV over 23 hours saw an improved outcome in patients but couldn't translate these results into improvements in functional status.[6]

Radiological Examination

This component of cervical spine management is also hotly debated. ATLS guidelines state that a lateral C.spine film showing the C7-T1 junction should be routinely taken of "*every patient sustaining an injury above the clavicle, especially head injury...*" in order to identify abnormalities in spinal structure, alignment and stability.[7] However, several studies have questioned its accuracy.[8,9,10] For example, Fischer et al[1] found that the cross-table lateral film had a maximum sensitivity of 82% with a specificity of 50%. Zabel et al[11] found that the absence of C.spine symptoms in fully alert, high risk, blunt trauma patients was a better screening tool than lateral C.spine radiology. Furthermore, it is generally believed that provided the patient has a normal level of consciousness, virtually all cervical spine pathology will have symptoms/signs referable to the C.spine.[12] Interestingly though, Jacobs and Swchwartz found that the diagnostic accuracy of clinical judgement fell to 50% when the patients had distracting injuries or were not fully alert.[13]

Based on the results from extensive studies, including the Steitweiser study which found that the sensitivity rose to 92% once an AP and an open mouth view was incorporated in to the C.spine work-up,[14] the American College of Radiology has recently recommended that a minimum of three views are needed to adequately evaluate cervical injury.[15] CT and MRI scans can then be taken to fully examine potentially injured areas.

Once the spine has been radiologically cleared, the collar can be removed on careful tertiary examination, providing the patient is fully conscious with no persisting distracting injuries. Clinically clearing the spine without radiology is possible providing the patient sustained a low energy injury and is fully alert with no neck pain or distracting injuries.

Fractures associated with upper C spine trauma

The unique anatomical and functional properties of the atlanto-axial vertebrae predispose them to a variety of unique fractures and dislocations.[22]

Fractures of the Atlas ring are as a result of vertical compressive force through the skull and often occur with other c. spine fractures, especially those of the dens. C1 fractures can occur in the posterior arch, the lateral mass or as a Jefferson 'burst' fracture but neurological sequalae are rare as the spinal canal is large and fracture fragments usually displace away from the spinal canal. C1 fractures are best visualised on the lateral and AP views.

Three types of dens fracture exist, usually resulting from sustaining extreme shearing forces and subaxial loading. Type I involves an avulsion of the tip, Type II is an unstable transverse fracture through the body of the dens and a Type III fracture extends into the body of C2. Dens fractures are best visualised on the open mouth view.

The Hangman's fracture of the Axial ring was first described by Haughton in 1866,[16] at post mortem after judicial hangings. Nowadays the lesion is more commonly associated with vehicular accidents. They classically result from hyperextension and axial loading of the occiput onto the posterior pedicles and laminae of C2.

Effendi et al[17] drew up a classification system -

Review of last issue's case of the Month

M 22 year old male driver
I Upper Abdominal pain
S P 110/m, BP 90, RR 22/m GCS 15
T Cervical collar, oxygen

Primary Survey
A Intact
B RR 22/m, SaO2 97% on 10L,
C P118/m, BP 95/82, all pulses intact and symmetrical
D GCS 15, pupils equal and reactive

Secondary survey
Abdomen - Tender RUQ
- Marked guarding in the Upper Abdomen
- BS reduced
- PR NAD

Summary of Investigations

C Spine	Normal
CXR	Normal
Pelvic X-ray	Normal
FAST	Positive in all 3 views Small Amount of free fluid

What is your patient management?
(He has received 3L of fluid, his BP has fluctuated to 80mmHg on 2 occasions, his abdomen is not distended).

Comment
Our 22 year old male driver has a problem with circulation and is haemodynamically unstable. His instability is not a "critical" instability and it is one that may become critically unstable or may stabilise. Given that he has received 3L of fluid, the question arises in relation to a management plan, should he go to CT scan to define what is probably a liver injury or should he go to the operating theatre? In terms of outcome either is acceptable so long as a decision is made to stop the bleeding if it is visible on CT scan. A vascular blush will mandate hepatic angiogram and embolisation or surgery. It is possible there is an associated injury to the spleen and this needs to be ruled out. An approach, therefore, would be to select potential non-operative management with interventional radiology, if that is available in your hospital with the expertise that is required. If it is not, then operative management is going to become likely once he has received another 1-2L of Gelofusin. If he becomes unstable in CT, with a further drop in BP, he needs to be in the operating theatre. The outcome for surgery in liver trauma is varied. For moderate injuries embolisation may be associated with less transfusion requirements and post treatment sepsis than an operative approach. In this case we opted for operative management bringing the patient in the operating theatre where he had active bleeding from a grade IV right hepatic laceration through segments V and VIII requiring hepatic artery branches to be oversewn and ligation of a major branch of the right portal vein. His liver was packed and he was returned to ICU after a transfusion of 8 units of blood, 5L of Saline and 2L of Gelofusin. On removal of his pack 24 hours later his gallbladder was necrotic requiring a cholecystectomy. After a prolonged course in hospital (over three weeks) he made a full recovery without significant sequelae.

This patient demonstrates the use of one of two options, in this case a safe operative approach with haemostasis was achieved, remembering the alternative hepatic angiography with embolisation. It is important to remember, however, that embolisation will not control venous bleeding. Also unless you are clinically quite sure that a liver lesion is most likely (from MIST and Secondary Survey) it would be very unwise to bring such a patient to a Radiology Department.

Victoria C Banks Michael Sugrue

Case of the Month

33 year old male self presented to an Urban Hospital Emergency Department with neck pain. He was at a buck's party and dived into a swimming pool after 11 stubbies.

Emergency Cubicle
Primary Survey
A Airway fine Pain in the neck
B OK
C OK
D OK

Secondary survey
revealed 10cm vertex laceration with a haematoma and a tender upper cervical spine with no swelling, bruising or step deformities. Both the upper and lower limbs were neurologically intact, with no abnormality of tone, power, reflex or sensory function. Anal tone was intact and there was no priaprism.

What do you do now, can you clear his spine clinically?

How many X-rays does he need?
Should he have a CT if his X-rays are normal?
What is the role of MRI?
If he did have a fracture how would you transfer him?
If he had a fracture should he go to a dedicated spinal centre or a major trauma service?

Meetings

Trauma 2001
Well done to ATS and CAT Great Meeting.

Definitive Surgical Trauma Care Course (DSTC) Sydney
(Taking Bookings now for 2002)
2nd and 3rd August, 2001
Email: charmaine.miranda@swsahs.nsw.gov.au
For Melbourne DSTC course in August contact Peter Danne

SWAN 9
SWAN 9 will be held on the 4th and 5th of August, 2001, bringing to you a number of world icons 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

Inaugural International Conference on Abdominal Compartment Syndrome
What an opportunity to be at the FIRST international ACS conference with world leaders on intra-abdominal pressure. Limited registration
Contact: Charmaine Miranda
Email: charmaine.miranda@swsahs.nsw.gov.au
Phone: 02 9828 3927
http:// www.swsahs.nsw.gov.au/livtrauma

Controversies in Civilian and Military trauma 2001
Promises to be a great meeting Brisbane 25-27 May Brisbane 61 7 33958989

Remember if your are not a member of ATS you could be! Contact Dr Bill Griggs, President Elect of ATS

Quality Trauma Health Care

The delivery of optimum trauma care requires an established trauma care delivery system. The NSW Department of Health have issued their guidelines for the delivery of quality health care service and embraced the following principles as shown in Table 1.

At the recent Injury 2000 meeting, organised by the Trauma Department of Canberra Hospital, in conjunction with the Australasian Trauma Society, I undertook a survey of those in attendance at the Trauma session on Quality Trauma Systems. Those surveyed were multidisciplinary providers of trauma care throughout Australia, predominantly from New South Wales. They were asked whether the quality guidelines were currently achieved in their own institution with the items as listed in Table 1. These questions were chosen verbatim from a quality assurance document from the NSW Department of Health.

Table 1. As A Quality Health Care Service, The NSW Health System Embraces the Following Principles

Question Number		Currently Achieved	
1.	The health consumer as the primary focus of any model of health care quality management.	Yes	No
2.	Consumers being enabled and encouraged to participate effectively both in their own care and treatment, and in the planning, delivery and evaluation of health services.	Yes	No
3.	Consumers having ready access to effective systems of complaint and compliment.	Yes	No
4.	The Area Health Service Board accepting responsibility for the quality of the health care provided to the consumers of its services.	Yes	No
5.	An Area Health Service Executive taking responsibility for creating and maintaining a structure and policies for managing the quality of health care.	Yes	No
6.	Those practicing within the system taking responsibility for the standard of their own practice and sharing responsibility for creating and maintaining a system which provides safe, high quality health care.	Yes	No
7.	Health treatment and care being based on the best available evidence with Area Health Services facilitating and monitoring the application and evaluation of best practice.	Yes	No
8.	A systematic and system-wide approach to continuous improvement of the quality of care delivered.	Yes	No
9.	A robust advisory and reporting structure designed to promote the quality improvement of health services and to provide regular information to the Area Health Service Board on the quality of services provided.	Yes	No
10.	An emphasis on preventing adverse outcomes through simplifying and improving the processes of care.	Yes	No
11.	The quality of health care being measured systematically with a focus on the minimisation of inappropriate variation in practice.	Yes	No
12.	A system driven by performance in the six primary dimensions of quality health care.	Yes	No
13.	Useful information relating to the above mentioned performance areas being readily available to all those who want it.	Yes	No
14.	All health care providers having access to systems which produce information about the outcomes of the care they provide.	Yes	No
15.	Quality information being used in planning and resource allocation decisions within health services.	Yes	No
16.	An emphasis on the development of partnerships of care most especially with health workers in the community including general practitioners.	Yes	No
17.	The quality framework being supported by high quality organisational structures that have been evaluated by a recognised external accrediting body.	Yes	No

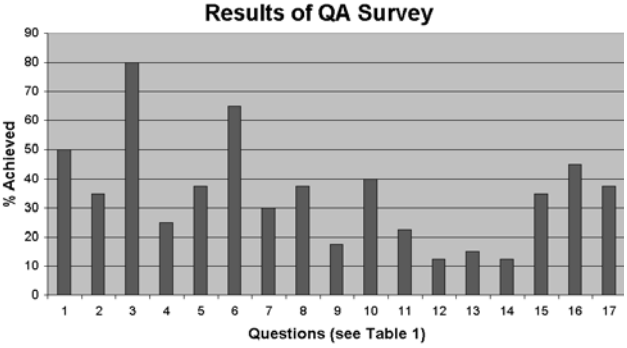
Results

There were 40 respondents. There were only two key areas where more than 50% of respondents felt that quality trauma care was being delivered. These were the concepts that the health consumer was the primary focus of care and that patients and consumers have ready access to effective systems of complaint and compliment. Less than 50% felt than any of the other criteria were being met. A distribution of the results in relation to each question is shown in Figure 1.

The results of this survey of committed trauma care providers indicates the perception of a significant lack of delivery of effective trauma care according to the Department of Health's own guidelines.

Michael Sugrue

Figure 1



Type I injuries are minimally displaced and considered stable although they are commonly associated with atlas burst fractures and odontiod injuries. Type II injuries are similar but extend into the C2/3 disc. Displacement and angulation are common although often only seen on flexion/extension views. Type III are bilateral pars fractures with facet dislocation, which although unstable rarely incur neurological damage.

Cervical spine fracture management

Treatment is based on the basic fracture management principles of reduction and immobilisation. The decision to treat conservatively or surgically depends on the type of fracture, spinal stability and neurological status.

Conservative management:

Stable injuries which by definition are less likely to change position or deform if subjected to physiological loading may not need immobilisation from the bony point of view, but may require it to reduce pain. Traction is the mainstay of cervical immobilisation, but it can be used to reduce Hangman's and odontoid peg fractures and dislocations. Gardener Wells tongs or Halo crowns are commonly used, however should this traction fail, muscle relaxants, anaesthetics, or open reduction under anaesthesia may be employed. Bony healing usually takes 6-8 weeks after which the patient can mobilise using an external orthosis such as a Halo vest or a cervicothoracic brace for 2-3 months.

Surgical Management

Unstable fractures require surgery to prevent deformity on physiological loading although, controversy surrounds the timing of such intervention. It is still unknown if the late spinal deformity that results from delayed surgical fixation causes neurological deterioration. Several studies have been completed but data obtained supports the efficacy of both immediate and delayed surgery.[18,19,20]

Many approaches and methods of internal fracture stabilisation exist and thus the procedure performed depends on the particular injury and the surgeon's expertise. Fixation involves the insertion of metal work to initially stabilise followed by bone grafting to obtain permanent fusion. Stabilising implants include screws, plates, Kirschner wires and rods.[21]

Long term management of spinal injury

The long term care of the patient has two main goals - to minimise the neurological sequelae and to prevent the complications associated with prolonged immobilisation. Chest physio and bladder retraining should be initiated early on, with regular passive movement of limbs to prevent contractures and strengthen the non-paralysed muscles.

Pressure sores and DVTs are a big concern with the immobile, and several strategies are implemented to prevent these complications. Namely, the patient is put on sub-cut heparin or calf compressors, and nursed on a Stoke-Egerton bed with a rolling cycle and regular position changes whilst maintaining cervical spine immobilisation.

EDITORIAL COMMENTS

Thanks for the useful review of upper cervical trauma.
The clinical examination is more important than the plain cervical X-rays for co-operative patients without neurological signs. Patients can usually tell you whether they have a serious cervical injury or not. In the emergency department, take the firm collar off and ask if the patient can lift the head off the bed. If not, or if it is very painful then put the collar back on and assume a fracture. If the patient can lift the head off the bed then palpate the spine and gently examine the range of motion. By this time you have a feeling for the degree of injury and the stability. This information will help you interpret the plain X-ray findings. Patients often have neck pain that can be greatly relieved by using a pillow or folded towel under the head. Even for patients with a cervical fracture, a support under the head will prevent painful hyper-extension and pressure sores or hair loss on the occiput. Patients with cervical injuries, particularly with neurological signs can frequently have disc or ligamentous injuries so urgent MRI scans can be very helpful. Immobilisation with a Halo device is more versatile than the Gardener Wells or tongs. Regular position changes are necessary in the first day for patients immobilised because of a cervical injury. The debate on the results of the NASCIS trials is not resolved and high dose methylprednisolone can not be recommended at present[1]. In the modern surgical era with spinal instrumentation for upper cervical fractures such as long term immobilisation with traction is very uncommon, furthermore halo-vest immobilisation is becoming less frequent. In elderly patients unstable fractures with good alignment can be treated with a cervical collar surprisingly often.

James van Gelder MMed Stats, MD, FRACS.
Neurosurgeon Liverpool Hospital

1. Hulbert, RJ. Methylprednisolone for acute spinal cord injury: an inappropriate standard of care. J. Neurosurg. 2000; 93: 1-7.

References:

1. Fischer RP. Cervical radiographic evaluation of alert patients following blunt trauma. Ann Emerg Med.1984; 13: 905-907
2. MacDonald RL, Schwartz MD, et al. Diagnosis of cervical spine injury in motor vehicle crash victims: how many x-rays are enough? J Trauma.1990; 30: 392-397
3. Young JS, Northrup NE. Statistical information pertaining to some of the most commonly asked questions about SCI. Sci Dig.1979; Spring: 111
4. Bohlam HH. Acute fractures and dislocations of the cervical spine: an analysis of 300 hospitalised patients and review of the literature. J Bone Joint Surg Am.1979; 61: 1119-1142
5. Trauma Handbook, Liverpool Hospital. 5th Edition; July 1994
6. McCelland MR, Tharion G. Acute Spinal Cord Injury: Surgery.1999;17:2,25
7. Advanced Trauma Life Support Manual. Chicago, III: American Royal College of Surgeons; 1993: 27 & 196
8. Woodring JH, Lee C. Limitations of cervical radiography in the evaluation of acute cervical trauma. J Trauma.1993; 34:32
9. Shaffer MA, Doris PE. Limitations of the cross table lateral view in detecting cervical spine injuries: a retrospective analysis. Ann Emerg Med. 1981;10: 508
10. Ross SE, Schwab LW.et al. Clearing the cervical spine injuries: initial radiographic evaluation. J Trauma. 1987;27:1055-1060
11. Zabel DD, Tinkoff G. et al. Adequacy and efficacy of lateral cervical spine radiography in alert, high risk blunt trauma patients. J Trauma. 1997;43:6: 952-958.
12. Gatterell CB. Asymptomatic cervical spine injury: a myth? Am J Emerg Med. 1985:3: 264.
13. Jacobs LM, Schwartz R. Prospective analysis of acute cervical spine injury: a methodology to predict injury. Ann Emerg Med. 1986:15: 44-49
14. Steitweiser DR, Knopp R. et al. Accuracy of standard radiographic views in detecting cervical spine fractures. Ann Emerg Med. 1983; 12:538-542
15. The American College of Radiology Task Force on Appropriateness criteria. Appropriateness Criteria for Imaging and Treatment Decisions. Reston. Va: American College of Radiology: 1995
16. Haughton S. On hanging, considering from a mechanical and physiological point of view. London, Edinburgh and Dublin Philosophical Magazine and Journal of Science. 1866; 32: 23-34
17. Effendi B, Roy D et al. Fractures of the ring of the axis. A classification based on the analysis of 131 cases. J Bone Joint Surgery. 1981:63:3:319-327
18. Duh MS, Shepard MJ et al. The effectiveness of surgery on the treatment of acute spinal cord injury and its relation to pharmacological treatment. Neurosurgery. 1994;35: 240-249
19. Marshall LF, Knowlton S,et al. Deterioration following spinal cord injury: a multicenter study. J Neurosurg. 1987; 66: 400-4
20. Vaccaro AR, Daugherty RJ et al. Neurologic outcome of early versus later surgery for cervical spinal cord injury. Spine. 1997;22:2609-13
21. Howard SA. Cervical Spine Trauma. Spine. 1998; 23:24:2713-2729
22. Cass M, Hughes S. Injuries to the Cervical Spine, Surgery. 1999; 17:2

NON-INVASIVE TESTS FOR CARDIAC

The search for

Blunt cardiac injury, also called cardiac contusion, is defined as myocardial cellular damage that can result from non-penetrating chest trauma. The heart may receive a direct blow from a precordial impact, or it may be compressed between the sternum and vertebral column. The frequency and clinical significance of this diagnosis is controversial. The reported incidence of cardiac involvement in blunt chest trauma varies from 0% to 76%[1,2] depending on the criteria used for establishing the diagnosis. Some studies indicate that cardiac contusion is rare and has little prognostic significance[3,4] whilst others show that the presence of cardiac contusion does influence patient outcome.[5] It is an elusive clinical entity as lesions from blunt cardiac trauma may vary from asymptomatic myocardial injury to cardiac rupture. Within this spectrum lies a wide range of clinical presentations, including arrhythmias, hypotension and valvular dysfunction, which have variable physiological consequences. However, perhaps the greatest source of contention is how best to diagnose the lesion. The goal of diagnosis in blunt cardiac injury is to identify patients who need treatment. Despite the multitude of tests introduced over the last 20 years, there is still no universally accepted diagnostic algorithm. An overview of the current thinking in this evolving area of trauma care is given below.

I. ECG

Many patients with chest trauma have an abnormal ECG.[6] Trauma is frequently associated with pain and fear (high catecholamine states), intoxication and electrolyte abnormalities, all of which may cause ECG changes. Some patients may have pre-existing heart disease, and atrial fibrillation has been identified as an independent predictor of increased mortality.[7] Arrhythmias, especially premature ventricular contractions, are common after chest trauma, but reported data suggests no relation between the complexity of the arrhythmia and the degree of cardiac contusion6. When present, ST segment change of at least 1mm or T wave inversion in two contiguous leads are more specific than arrhythmias in cardiac injury.[8] Hossack et al. used either echocardiography or radionuclide angiography to define the presence of contusion in 75 patients with non-penetrating chest trauma. ECG ST or T wave changes were present in 25 patients, yielding a sensitivity of 47% and a specificity of 79%. The negative predictive value for cardiac complications was 90%.[9] Therefore, although the ECG is useful in indicating myocardial injury by ST-T changes and has a good negative predictive value for cardiac complications, this modality is limited by a lack of sensitivity.

The reported incidence of cardiac involvement in blunt chest trauma varies from 0% to 76%[1,2] depending on the criteria used for establishing the diagnosis.

II. Cardiac Enzymes

The most widely used enzyme for the diagnosis of acute myocardial infarction is the CK-MB isoenzyme. Many investigators have questioned its specificity for cardiac contusion.[6,10,11] The heart has a high proportion of CK-MB, but it is also present in skeletal muscle, lung, pancreas, stomach, colon, small intestine and liver leading to false positives in multiply injured patients.[12] Furthermore, the MB fraction (CK-MB/total CK) may be falsely negative if the total CK is elevated by multiple non-cardiac injuries. The absolute CK-MB level may therefore be more reflective of cardiac injury than the percentage CK-MB. Nevertheless, Healey et al. found that a CK-MB concentration of 200mg/dL or greater had a 100% positive predictive value for cardiac complications, and conversely the CK-MB concentration of 5% or less together with a normal ECG had a 100% negative value for treated arrhythmias.[13] More recently the cardiac markers troponin I (cTn-I) and troponin T (cTn-T) have become available. Both cardiac troponins are regulatory proteins of the thin filament of striated muscle involved in the calcium-sensitive switch that regulates the interaction of actin and myosin. Antibodies raised against these cardiac isoforms have no or only minor cross-reactivity with skeletal muscle isoforms,[14] and are therefore considered as highly sensitive and specific markers of myocardial cell injury. Bertinchant et al. evaluated the value of circulating cTn-I and cTn-T compared with conventional markers in suspected myocardial injury after blunt chest trauma in haemodynamically stable patients.[15]

Myocardial contusion was diagnosed in 27.6% of 94 patients with blunt chest trauma from electrocardiographic and/or electrocardiographic abnormalities. The percentage of patients with elevated CK indices did not significantly differ between patients with or without myocardial contusion. The percentage of patients with elevated circulating cardiac troponins was significantly higher in those with myocardial contusion. However, although specificity of cTn-I and cTn-T (97% and 100%) compared well with conventional markers, the sensitivity of cTn-I and cTn-T (23% and 12%) was low. Furthermore, the cardiac troponins did not efficiently identify trauma patients at risk for the development of cardiac complications. This data supports some earlier studies assessing these enzymes[16,17] and has tempered the initial optimism that the cardiac troponins will represent a marked improvement over traditional biochemical markers in the diagnosis of blunt cardiac injury.

III. Cardiac Imaging Studies

The cardiac imaging techniques most often used in chest trauma are transthoracic echocardiography (TTE) and radionuclide angiography (RNA). Transthoracic echocardiography is useful in visualising wall motion abnormalities, intracardiac structures and pericardial effusions.[18] The study by Karalis et al. reported a significantly greater rate of adverse cardiac events in patients with echocardiographic myocardial contusion (26% of 31 patients) compared with those without echocardiographic myocardial contusion (3% of 74 patients).[19] However other investigators have not shown a correlation between abnormal echocardiography and cardiac events[9,20] and have concluded that it adds little to patient management. Furthermore, echocardiographic studies can be suboptimal in the presence of chest tubes or significant chest wall brusing.[9] Some studies have shown that echocardiography helps identify which of those patients with positive enzymes will have cardiac complications.[19,21] Transoesophageal echocardiography (TOE) has the advantage of allowing better visualisation of the thoracic aorta and right heart where the majority of cardiac contusion probably occur. As a diagnostic modality, TOE more accurately detects cardiac contusion than TTE.[22] However it is a difficult procedure to perform on an awake patient or a patient with an unstable cervical spine. Transoesophageal echocardiography is therefore of most value in intensive care unit patients when the TTE examination results are suboptimal and when aortic injury is suspected.[19] Radionuclide angiography has also been used to visualise

DAMAGE IN BLUNT CARDIAC INJURY
a gold standard!

wall motion and estimate ejection fraction. This modality may be more sensitive than TTE in evaluating right ventricular injuries. In a series of 77 blunt chest trauma patients, 55% had segmental wall motion abnormalities by RNA. Mortality was significantly higher in patients who had changes in wall motion (29%) compared to those without RNA changes (6%).[23] However, RNA is suboptimal when arrhythmias are present and does not visualise intracardiac structures. Its use has been limited in the trauma setting. Technetium-99m pyrophosphate scanning can detect myocardial necrosis. However, studies assessing this test have been disappointing as there are high rates of false negatives.[6] Furthermore, since there is a higher likelihood of injury to the right ventricle and the right ventricle has a lower mass relative to the left ventricle, the scintigraphic detection of right ventricular necrosis is difficult. False positive may also be expected as technetium-99m pyrophosphate can be taken up by bone, such as in sternal fractures. Several investigators have evaluated the role of thallium-201 combined with single-photon emission CT (SPECT).[24,25] Thallium studies identify segments of decreased myocardial perfusion as a consequence of blunt cardiac trauma. In a prospective series of 40 patients sustaining non-penetrating chest trauma,[26] 12 patients developed cardiac complications (arrhythmias, ventricular conduction defects and pericarditis). The sensitivity of thallium scanning was 55.6% and the specificity was 32.1%, with an accuracy of 37.8%.

The sensitivity of the initial electrocardiogram in this series was 91.7% and the specificity was 82.1%, with an accuracy of 85%. In another study of 123 patients, 75 had positive thallium scans. All 11 patients with serious arrhythmias had abnormal scans. This technique does not normally image the right ventricle, and the presence of a thallium defect does not distinguish between a recent injury or a prior myocardial infarct. One small study has tested indium-111 antimyosin scintigraphy in patients with blunt chest trauma. Antimyosin is a Fab fragment of the monoclonal antibody to cardiac myosin. Further studies are required to enable comment on such a test.[27]

The controversies in the management of trauma victims with significant blunt cardiac injuries remain unresolved partly due to the lack of a diagnostic gold standard that can predict subsequent cardiac morbidity. Although early studies suggested that myocardial contusion was associated with significant morbidity and mortality, it now appears that patients who survive until admission to the hospital and are haemodynamically stable have a favourable prognosis.[28,29] Of the currently available diagnostic modalities, the ECG has a good negative predictive value. The absolute CK-MB measurement has a good positive predictive value. Therefore it appears that at present these represent the two best screening tests. The future role of the cardiac troponins is still unclear but due to their greater sensitivity they may still supercede the conventional enzyme markers. Several distinguished authors have argued against the use of extensive and expensive work-ups with cardiac imaging following blunt trauma.[30] Until further data indicates otherwise, it is probably reasonable to reserve imaging studies for patients with an abnormal ECG and/or elevated cardiac enzyme levels.

The controversies in the management of trauma victims with significant blunt cardiac injuries remain unresolved...

Anthony Manes

EDITORIAL COMMENT

Blunt cardiac trauma refers to a spectrum of injuries ranging from simple ECG changes to free wall rupture. Since it was first described by Borch in 1676 it has been the subject of much controversy. Cardiac troponin I has a role in identifying blunt cardiac injury. There is, however, only a modest increase for individual negative predictive values of ECG and cTnI from 95% and 93% to 100% when the two tests are combined. One could argue that the expense of an additional test is not justified for such a small increase. This increase becomes important, however, if patients are to be discharged on the basis of these tests. The negative predictive value of 100% when these tests are combined allows for such a decision to be made.[1]

M Sugrue

Reference: 1. Clinically Significant Blunt Cardiac Trauma: Role of Serum Troponin Levels Combined with Electrocardiographic Findings. Salim A, Velmahos G, Jindal A et al. J Trauma 2001;50:237-243.

REFERENCES:

1. Parmley L, Manion W, Mattingly T. Non-penetrating traumatic injury to the heart. Circulation. 1958; 18:371.
2. Wisner D, Reed W, Riddick R. Suspected myocardial contusion. Ann Surg. 1990; 212:82.
3. Beresky R, Klingler R, Peake J. Myocardial contusion: when does it have clinical significance? J Trauma. 1988; 28:64.
4. Cachecho R, Grindlinger G, Leev V. The clinical significance of myocardial contusion. J Trauma. 1992; 33:68.
5. Baum V. Anaesthetic Complications during emergency non-cardiac surgery in patients with documented cardiac contusion. J Cardiothorac Vasc Anesth. 1991; 5:57.
6. Potkin R, Werner J, Trobaugh G et al. Evaluation of non-invasive tests of cardiac damage in suspected cardiac contusion. Circulation. 1982; 66:627.
7. McLean R, Devitt J, McLellan B et al. Significance of myocardial contusion following blunt chest trauma. J Trauma 1992; 33:240.
8. Sava J, Demetrios D. Penetrating and blunt cardiac trauma: Diagnosis and management. Em Med. 2000; 12:95.
9. Hossack K, Moreno C, Vanway C, Burdick D. Frequency of cardiac contusion in non-penetrating chest injury. Am J Cardiol. 1988; 61:391.
10. Keller K, Shatney C. Creatine phophokinase-MB assay in patients with suspected myocardial contusion: diagnostic test or test of diagnosis? J Trauma. 1988; 28:58.
11. Fabian T, Cicala R, Croce M et al. A prospective evaluation of myocardial contusion: correlation of significant arrhythmias and cardiac output with CPK-MB measurements. J Trauma. 1991; 31:653.
12. Tsung S. Creatine kinase isoenzyme patterns in human tissue obtained at surgery. Clin Chem. 1976; 22:173.
13. Healey M, Brown R, Fleiszer D. Blunt cardiac trauma: is this diagnosis necessary? J Trauma. 1990; 30:137.
14. Baum H, Braun S, Gerhardt W et al. Multicentre evaluation of a second-generation assay for cardiac troponin T. Clin Chem. 1997; 43:1877.
15. Bertinchant J, Polge A, Mohty D et al. Evaluation of incidence, clinical signficance, and prognostic value of circulating cardiac troponin I and T elevation in haemodynamically stable patients with suspected myocardial contusion after blunt chest trauma. J Trauma. 2000; 48:924.
16. Helm M, Hauke J, Weiss A, Lampl L. Cardiac troponin T as a biochemical marker of myocardial injury early after trauma. Diagnostic value of a qualitative bedside test. Chirurg. 1999; 70:1347.
17. Ferjani M, Droc G, Dreux S. Circulating cardiac troponin T in myocardial contusion. Chest. 1997; 111:427.
18. Feghali N, Prisant L. Blunt myocardial injury. Chest. 1995; 108:1673.
19. Karalis D, Victor M, Davis G et al. The role of echocardiography in blunt chest trauma: a transthoracic and transoesophageal echocardiographic study. J Trauma. 1994; 36:53.
20. Fabian T, Mangiante E, Patterson C et al. Myocardial contusion in blunt trauma. Clinical characteristics, means of diagnosis, and implications for patient management. J Trauma. 1988; 28:50.
21. Frazee R, Mucha P, Farnell M et al. Objective evaluation of blunt cardiac trauma. J Trauma. 1986; 26:510.
22. Brooks S, Young J, Cmolik B et al. The use of tranoesophageal echocardiography in the evaluation of chest trauma. J Trauma. 1992; 32:761.
23. Sutherland G, Driedger A, Holliday R et al. Frequency of myocardial injury after blunt chest trauma as evaluated by radionuclide angiography. Am J Cardiol. 1983; 52:1099.
24. Holness R, Waxman K. Diagnosis of traumatic cardiac contusion utilising single photon-emission computed tomography. Crit Care Med. 1990; 18:1.
25. Godbe D, Waxman K, Wang F et al. Diagnosis of myocardial contusion: qualitative analysis of single photon-emission computed tomographic scans. Arch Surg. 1992; 127:888.
26. McCarthy M, Pavlina P, Evans D et al. The value of SPECT-thallium scanning in screening for myocardial contusion. Cardiovasc Intervent Radiol. 1991; 14:238.
27. Hendel R, Cohn S, Aurigemma G et al. Focal myocardial injury following blunt chest trauma: a comparison of indium-111 antimyosin scintigraphy with other non-invasive methods. Am Heart J. 1992; 123:1208.

©1996 Trauma Department Liverpool Letters to Dr Michael Sugrue
Trauma Department, Liverpool Hospital,
Elizabeth Street NSW 2170 Fax 9828 3922
www.swsahs.nsw.gov/livtrauma Email:michael.sugrue@swsahs.nsw.gov.au

The Trauma Grapevine is owned, published, copyrighted © 2001 It is sponsored by B Braun All rights reserved.
Editorial Offices: Trauma department Liverpool Hospital Locked bag 7103 Liverpool BC 1871 Australia Phone: (61) 2 98283927 FAX: (61) 2 98283926

ISSN 1329-2846