CLINICAL PAPER

The incidence of neurogenic shock in patients with isolated spinal cord injury in the emergency department☆

H.R. Guly, O. Bouamra, F.E. Lecky, on behalf of the Trauma Audit and Research Network

a Derriford Hospital, Plymouth, PL6 8DH, UK
b Trauma Audit and Research Network, University of Manchester, UK
c Salford Royal Hospitals NHS Trust and Trauma Audit and Research Network, University of Manchester, UK

Received 20 March 2007; received in revised form 5 June 2007; accepted 11 June 2007

KEYWORDS
Spinal cord injury; Hypotension; Bradycardia; Neurogenic shock

Summary

Background: Spinal cord injury (SCI) is recognised to cause hypotension and bradycardia (neurogenic shock). Previous studies have shown that the incidence of this in the emergency department (ED) may be low. However these studies are relatively small and have included a mix of blunt and penetrating injuries with measurements taken over different time frames. The aim was to use a large database to determine the incidence of neurogenic shock in patients with isolated spinal cord injuries.

Methods: The Trauma Audit and Research Network (TARN) collects data on patients attending participating hospitals in England and Wales.

The database between 1989 and 2003 was searched for patients aged over 16 who had sustained an isolated spinal cord injury.

The heart rate (HR) and systolic blood pressure (SBP) on arrival at the ED were determined as was the number and percentage of patients who had both a SBP <100 mm Hg and a HR < 80 beats per minute (BPM) (the classic appearance of neurogenic shock).

Results: Four hundred and ninety patients had sustained an isolated spinal cord injury (SCI) with no other injury with an abbreviated injury scale (AIS) of greater than 2.

The incidence of neurogenic shock in cervical cord injuries was 19.3% (95% CI 14.8–23.7%). The incidence in thoracic and lumbar cord injuries was 7% (3–11.1%) and 3% (0–8.85%).

☆ A Spanish translated version of the summary of this article appears as Appendix in the final online version at 10.1016/j.resuscitation.2007.06.008.

* Corresponding author. Tel.: +44 1752 777111; fax: +44 1752 778101.
E-mail addresses: henry.guly@phnt.swest.nhs.uk (H.R. Guly), omar.bouamra@hope.man.ac.uk (O. Bouamra), Fiona.E.Lecky@manchester.ac.uk (F.E. Lecky).
Conclusions: Fewer than 20% of patients with a cervical cord injury have the classical appearance of neurogenic shock when they arrive in the emergency department. It is uncommon in patients with lower cord injuries. The heart rate and blood pressure changes in patients with a SCI may develop over time and we hypothesise that patients arrive in the ED before neurogenic shock has become manifest.

© 2007 Elsevier Ireland Ltd. All rights reserved.

Introduction

The cardiovascular effects of spinal cord injury (SCI) are well described. It is recognised that SCI may cause hypotension due to loss of sympathetic tone and decreased peripheral vascular resistance. Bradycardia may occur due to unopposed vagal activity in a high cord lesion that disrupts the sympathetic supply to the heart. This bradycardia is exacerbated by hypoxia and endobronchial suction. Piepmeyer et al. observed 45 patients with a cervical SCI for 2 weeks and found that of the 23 with Frankel grade A SCI, 87% had an average daily heart rate (HR) of <55 BPM and in 96% the heart rate dropped below 50 BPM. Patients were given fluid replacement to maintain their blood pressure but nine required vasopressors to maintain their systolic blood pressure above 100 mm Hg. In another series of 31 patients with a cervical cord injury, all had persistent bradycardia (mean HR < 60 BPM for 1 day) and 68% had hypotension (SBP <90 mm Hg on two consecutive occasions). There is also decreased pulmonary vascular resistance. Hypotension with bradycardia caused by a spinal cord injury is given the name "neurogenic shock." The literature indicates that there is no universally accepted definition of neurogenic shock but one paper has defined it as a systolic BP < 100 mm Hg and a heart rate <80 BPM in a patient without other obvious cause. Another paper has defined hypotension in SCI as a systolic BP < 90 mm Hg.

Most of these studies have been performed in spinal injuries units and neurological intensive care units. Many studies include patients with other injuries and there may be a component of haemorrhage in addition to the neurogenic shock. There are inevitable delays before patients receive care in a specialised neurological centre, consequently studies reporting the incidence of cardiovascular abnormalities therein are describing patho-physiology hours (and sometimes days) post-injury. In addition these studies have often ignored the cardiovascular effects of treatments such as fluids and inotropes given in an attempt to correct abnormal physiology.

There is much less research on the cardiovascular effects of SCI on presentation to the emergency department (ED) where the patho-physiological picture cannot (for reasons stated above) be assumed to be the same as that in the spinal injuries unit.

On arrival in the ED there is often diagnostic uncertainty as to the precise nature of the patient’s injuries. SCI occurs in less than 5% of major trauma cases, therefore the resuscitation team may have little experience of SCI. However, the advanced trauma life support (ATLS) system has become an international “gold standard” for the initial management of the traumatised patient and gives advice on the immediate recognition of SCI. It states (without referencing): "the classic picture of neurogenic shock is hypotension without tachycardia or cutaneous vasoconstriction. A narrowed pulse pressure is not seen in neurogenic shock." Other textbooks describe: "the result [of a high cord lesion] is vasodilatation, hypotension, bradycardia…" and "the pulse rate will be slow, but the blood pressure will be low." Another book describes hypotension and the failure to mount a sympathetic response with the possibility of a bradycardia.

There are some studies relating to the early cardiovascular picture in SCI patients. In a study of 75 patients with penetrating SCI only 24% were hypotensive in the field and only 7% of patients had neurogenic shock. Only 22% of patients who were hypotensive had neurogenic shock as a cause. A further study of 408 patients with both blunt and penetrating trauma, gives the incidence of hypotension (<100 mm Hg) as 4.5% of 107 neurologically intact spinal injuries (most had associated injuries to account for the shock), 20.7% of 111 with incomplete SCIs and 31.6% of 190 with complete cord lesions.

In another study, 23.6% of 50 patients with a complete quadriplegia had hypotension (BP <90 mm Hg) at the time of admission but in some of these patients, haemodynamic monitoring did not start for 11 h and some patients had been treated with up to 3 l of fluid at the scene. There is no mention of associated injuries.

While some patients with cervical SCI have hypotension when first seen, it would appear from the above references that these patients are a minority. However these studies are relatively small samples including a mix of blunt and penetrating injuries with measurements taken over different time frames. The incidence of neurogenic shock in lower level cord injury does not appear to have been quantified in the literature; although a lesser insult to the sympathetic nervous system would lead one to expect a lower incidence than that found in cervical cord injuries.

The aim of this research is to employ a large European trauma registry to determine the incidence of neurogenic shock in patients with isolated SCI (at any level) at first presentation. This should indicate to clinicians the sensitivity of admission cardiovascular variables for the early diagnosis of SCI.

Materials and methods

Data and inclusion criteria

The Trauma Audit and Research Network (TARN; which includes 50% of trauma receiving hospitals in England and
Incidence of neurogenic shock

Table 1 Isolated complete SCI (Epidemiological data, adult 1989–2003)

<table>
<thead>
<tr>
<th>Level</th>
<th>Cervical</th>
<th>Thoracic</th>
<th>Lumbar</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>301</td>
<td>155</td>
<td>34</td>
<td>490</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median age</td>
<td>41</td>
<td>33.1</td>
<td>37</td>
<td>37.1</td>
</tr>
<tr>
<td>ISS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median ISS</td>
<td>25</td>
<td>25</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>Outcome</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% (number) died</td>
<td>18.9 (57)</td>
<td>3.2 (5)</td>
<td>0 (0)</td>
<td>12.7 (62)</td>
</tr>
<tr>
<td>95% confidence intervals (CI)</td>
<td>14.5–23.4</td>
<td>0.4–6.0</td>
<td>0.0</td>
<td>9.7–15.6</td>
</tr>
</tbody>
</table>

Table 2 Isolated complete cord injuries (Physiological data, adult 1989–2003)

<table>
<thead>
<tr>
<th>Level</th>
<th>Cervical</th>
<th>Thoracic</th>
<th>Lumbar</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>301</td>
<td>155</td>
<td>34</td>
<td>490</td>
</tr>
<tr>
<td>Systolic BP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median systolic BP (mm Hg)</td>
<td>110</td>
<td>130</td>
<td>130</td>
<td>120</td>
</tr>
<tr>
<td>IQR (25th–75th percentile)</td>
<td>98–130</td>
<td>110–140</td>
<td>120–150</td>
<td>100–140</td>
</tr>
<tr>
<td>SBP % (N) &lt; 100 mm Hg</td>
<td>25.8 (70)</td>
<td>11.8 (16)</td>
<td>3.1 (1)</td>
<td>19.8 (87)</td>
</tr>
<tr>
<td>95% CI</td>
<td>20.9–30.8</td>
<td>6.7–16.8</td>
<td>0–9.0</td>
<td>16.3–23.3</td>
</tr>
<tr>
<td>Heart rate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median HR (BPM)</td>
<td>70</td>
<td>80</td>
<td>83</td>
<td>74</td>
</tr>
<tr>
<td>IQR (25th–75th percentile)</td>
<td>60–80</td>
<td>70–88</td>
<td>73–96</td>
<td>62–83</td>
</tr>
<tr>
<td>HR % (N) &lt; 80 BPM</td>
<td>67.0 (187)</td>
<td>45.8 (66)</td>
<td>36.4 (12)</td>
<td>58.1 (265)</td>
</tr>
<tr>
<td>95% CI</td>
<td>61.7–72.3</td>
<td>38.0–53.7</td>
<td>20.2–52.5</td>
<td>53.7–62.5</td>
</tr>
<tr>
<td>Systolic BP &lt; 100 mm Hg and heart rate &lt; 80 BPM</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% (N)</td>
<td>19.3 (53)</td>
<td>7.0 (10)</td>
<td>3.0 (1)</td>
<td>14.2 (64)</td>
</tr>
<tr>
<td>95% CI</td>
<td>14.8–23.7</td>
<td>3.0–11.1</td>
<td>0–8.8</td>
<td>11.1–17.3</td>
</tr>
</tbody>
</table>

Wales) collects data on patients attending participating hospitals who sustain an injury resulting in immediate admission to hospital for 3 days or longer, admission to an intensive care or a high dependency unit, or death within 93 days. TARN excludes patients over 65 years with isolated fracture of the femoral neck or pubic ramus and those with single uncomplicated limb injuries. Details of TARN have been described elsewhere.10

The database between 1989 and 2003 was searched for patients aged over 16 who had sustained an isolated SCI. This included patients with a SCI with another injury with an abbreviated injury score (AIS) of greater than 2. Details of these patients, their ages, injury severity score (ISS) and mortality are shown in Table 1.

Table 2 shows details of the systolic blood pressure and heart rate in these patients and also shows the number and percentage of patients who have hypotension, a low heart rate or both.

Prehospital times (ambulance arrival on scene to arrival in hospital) were recorded in 33.3% of cases. The median time was 75 min (IQR 48.25–301 min). Prehospital fluids (type not stated) were recorded in 22.4% of cases. The median volume of fluid infused was 500 ml.

All physiological data used was that identified as measured on arrival in the Emergency Department.

Results

Nine hundred and twenty six patients had sustained a SCI. Four hundred and ninety (53%) of these had sustained an isolated SCI with no other injury with an Abbreviated Injury Scale (AIS) of greater than 2. The vast majority of these, 481 (98.2%) were injured by blunt trauma.

Details of these patients, their ages, injury severity score (ISS) and mortality are shown in Table 1.

Table 2 shows details of the systolic blood pressure and heart rate in these patients and also shows the number and percentage of patients who have hypotension, a low heart rate or both.

Prehospital times (ambulance arrival on scene to arrival in hospital) were recorded in 33.3% of cases. The median time was 75 min (IQR 48.25–301 min). Prehospital fluids (type not stated) were recorded in 22.4% of cases. The median volume of fluid infused was 500 ml.
Discussion

In this relatively large study of patients with an isolated complete cervical SCI, we found an incidence of hypotension (systolic BP < 100 mm Hg) at arrival to the emergency department of 25.8% (95% CI 20.9–30.8%). The incidence of typical neurogenic shock was only 19.3% (95% CI 14.8–23.7%). We used a heart rate of <80 BPM as a definition of bradycardia because that had been previously used in another study. Bradycardia is usually defined as a heart rate <60 BPM: had we used that as a cut-off, the incidence of neurogenic shock would have been even lower. The interquartile range of heart rate in patients with cervical cord injuries was 60–80 BPM and so only 25% of patients had a bradycardia according to this definition. We found typical neurogenic shock and hypotension to be uncommon in patients with lower level cord injuries. This is not unexpected in injuries below the level of the sympathetic chain.

Our incidence of hypotension (systolic BP < 100 mm Hg) of 25.8% compares with an incidence of 31.6% found by Soderstrom. Levi found that 23.6% of quadriplegic patients had a systolic BP < 90 mm Hg on arrival in the emergency room but some of these patients had been treated with up to 3 l of fluid at the scene and had had inotropic drugs to correct shock, so the incidence of true hypotension could be higher. There is no mention of associated injuries. Zipnick found a systolic BP < 100 mm Hg in 24% of patients with penetrating cervical cord injury (but these were not isolated injuries). This study is therefore not really comparable with our study as only 1.8% of our samples were injured by penetrating trauma. However this prevalence of hypotension is similar to that of our sample. In Zipnick’s study only 7% of patients had neurogenic shock and only 22% of patients who were hypotensive, had neurogenic shock as a cause.

The cardiovascular effects of SCI vary with time and patients are seen in the emergency department before they reach the intensive care unit. There is very little information about the very early effects of SCI in humans and so it is necessary to look at the cardiovascular effects of SCI in animals. Tibbs et al. investigated the effects of spinal cord transaction in anaesthetised dogs. The majority developed an immediate bradycardia with many developing cardiac dysrhythmias. This phase lasted between 45 s and 3 min. The systolic and diastolic blood pressures in all animals rose within 3–6 s, reaching a peak within 45 s. Following this initial bradycardia, the heart rate rebounded briefly to pre-injury levels before falling again. Within 2 h the animals had developed a sustained bradycardia. The mean arterial pressure also fell to 71% of the control value. In anaesthetised rats with a cervical spinal cord injury, there was also a transient rise in SBP over 2–3 min with a fall over the next 5 min with persisting hypotension. The HR reduced but this was not significant until 45 min post-injury and continued to decline over 135 min. Compression of the spinal cord in anaesthetised cats also produces hypertension starting within 2–6 s and persisting for 4–5 min before gradually fading away. The authors of this paper found that the amplitude and duration of this pressor response was greater with cervical cord compression than with thoracic or lumbar cord compression.

This study cannot say why patients on arrival in the emergency department with SCI do not show the classical signs of neurogenic shock as seen later in the intensive care unit or spinal injuries unit though we believe that this is due to time. The first phase of hypertension and bradycardia in animals only lasts a few minutes but the profound persisting bradycardia may take 2 h to develop. In a human study the greatest prevalence of bradycardia was found at 4 days. We hypothesise that many patients are reaching the ED before the maximum bradycardia and hypotension have developed. This is supported by a case report of an 18-year-old man with transection of the cord at C1/2. He was resuscitated early and maintained his blood pressure and heart rate for about 40 min before both suddenly fell. Unfortunately we do not have full details of the prehospital times in our study but the median prehospital time for those in whom it was recorded was 75.5 min.

The results of our study would be consistent with a suggestion that SCI patients presenting to the ED do so between the initial pressor response and the full bradycardia, which takes 2 h to develop.

In addition, it is known that the blood pressure is raised by pain and anxiety. All our patients would be conscious and, in addition to the pain of the injury, would have discomfort due to spinal immobilisation. They would also be very anxious. By the time they reach a spinal injuries unit these problems would have been addressed. A previous study from TARN has shown that the blood pressure in traumatised children is consistently higher than the quoted normal values of blood pressure in children.

This study looked at patients with isolated spinal cord injuries. However, only 53% of patients with spinal cord injury in the TARN database had isolated injuries. The other 47% had other injuries in addition. This study does not examine how neurogenic shock interacts with other causes of shock such as blood loss. TARN does not collect information on the diastolic blood pressure so we are unable to comment on the pulse pressure in patients with neurogenic shock.

Some patients had prehospital fluid but this was a minority and even in these patients, we do not believe that the volumes of fluid infused (median volume 500 ml) would have been sufficient to cause a major change in blood pressure.

In conclusion, neurogenic shock is not a sensitive maker for the early diagnosis of SCI in adult blunt trauma patients presenting to the emergency department. Further research is needed to show the haemodynamic changes that occur early after SCI in humans and to show how spinal cord injury influences the pulse rate and blood pressure of patients with hypovolaemia.

Conflict of interest statement

There has been no external funding for this research and we are not aware of any conflicts of interest.

Appendix A. Participating hospitals since 1989

Addenbrooke’s Hospital, Cambridge; Heatherwood & Wexham Park Hospital, Slough; Rotherham District General
Hospital; The Princess Royal Hospital, Shropshire; Airedale General Hospital, Yorkshire; Hillingdon Hospital, Middlesex; Royal Albert Edward Infirmary, Wigan; Torbay Hospital, Devon; Arrowe Park Hospital, Merseyside; Hinchingbrooke Hospital, Cambridgeshire; Royal Berkshire Hospital, Reading; Trafford General Hospital, Manchester; Ashford General Hospital, London; Homerton Hospital, London; Royal Bolton Hospital; University Hospital Lewisham, London; Atkinson Morley’s Hospital, London; Hope Hospital, Salford; Royal Cornwall Hospital, Truro; University Hospital of Hartlepool; Barnsley District General Hospital, Yorkshire; Huddersfield Royal Infirmary; Royal Devon & Exeter Hospital; University Hospital of North Staffordshire; Basildon Hospital, Essex; Hull Royal Infirmary, North Humberside; Royal Gwent Hospital, Newport; University Hospital of North Tees, Cleveland; Bassetlaw Hospital, Nottinghamshire; Powis Hospital, Suffolk; Royal Hallamshire Hospital, Sheffield; University Hospital of Wales, Cardiff; Bedford Hospital; James Cook University Hospital, Cleveland; Royal Hampshire County Hospital, Winchester; University Hospital, Aintree, Liverpool; Birmingham Heartlands Hospital; James Paget Hospital, Norfolk; Royal Lancaster Infirmary; Walton Centre for Neurology, Liverpool; Blackburn Royal Infirmary, Lancashire; Jersey General Hospital; Royal Liverpool Children’s Hospital (Alder Hey); Wansbeck General Hospital, Northumberland; Blackpool Victoria Hospital; John Coupland Hospital; Royal Liverpool University Hospital; Warrington Hospital, Cheshire; Booth Hall Children’s Hospital, Manchester; John Radcliffe Hospital, Oxford; Royal London Hospital; Warwick Hospital, Warwick; Bradford Royal Infirmary, Yorkshire; Kent & Canterbury Hospital; Royal Manchester Children’s Hospital, Pendlebury; Waterford Regional Hospital, Ireland; Bristol Royal Infirmary; Kent & Sussex Hospital; Royal Oldham Hospital; Watford General Hospital, Herts; Bromley Hospital, Kent; Kettering General Hospital, Northamptonshire; Royal Preston Hospital; West Cumberland Hospital, Cumbria; Broomfield Hospital, Essex; Kings College Hospital, London; Royal Shrewsbury Hospital, Shropshire; West Middlesex University Hospital; Burnley General Hospital; Kings Mill Hospital, Nottinghamshire; Royal Surrey County Hospital; West Wales General Hospital, Dyfed; Calderdale Royal Hospital, Halifax; Leeds General Infirmary, Royal Sussex County Hospital, Brighton; Weston General Hospital, Avon; Cheltenham General Hospital; Leicester Royal Infirmary; Royal United Hospital, Bath; Weymouth & District Hospital, Dorset; Chesterfield & Nth Derbyshire Royal Hospital; Leigh Infirmary; Royal Victoria Hospital, Belfast, N Ireland; Whips Cross Hospital, London; Chorley District General Hospital; Leighton Hospital, Cheshire; Royal Victoria Infirmary, Newcastle Upon Tyne; Whiston Hospital, Liverpool; City Hospital, Birmingham; Lincoln County Hospital; Sandwell District General Hospital, West Midlands; William Harvey Hospital, Kent; Colchester General Hospital, Essex; Maidstone General Hospital, Kent; Scarborough Hospital, North Yorkshire; Withington Hospital, Manchester; Conquest Hospital, East Sussex; Manchester Royal Infirmary; Scunthorpe General Hospital, South Humberside; Withybush General Hospital, Dyfed; Countess of Chester Hospital; Medway Hospital, Kent; Selly Oak Hospital, Birmingham; Worcester Royal Infirmary; County Hospital, Hereford; Milton Keynes Hospital; Sheffield Children’s Hospital; Worthing Hospital, West Sussex; Coventry & Warwickshire Hospital; Morriston Hospital, Swansea; Skegness & District Hospital, Lincolnshire; Wrexham Maelor Hospital, Clwyd; Craigavon Area Hospital, Co. Armagh; Nevill Hall Hospital, Wales; South Tyneside District Hospital, Tyne & Wear; Wycombe Hospital, High Bucks; Crawley Hospital, West Sussex; Newcastle General Hospital; Southampton General Hospital; Wythenshawe Hospital, Manchester; Cumberland Infirmary, Cumbria; Nor- folk & Norwich General Hospital; Southend Hospital, Essex; York District Hospital; Daisy Hill Hospital, County Down, Northern Ireland; North Manchester General Hospital; Southmead Hospital, Bristol; Ysbyty Gwynedd District General; Darrent Valley Hospital, Kent; North Tyneside General Hospital, Tyne & Wear; Southport & Formby District General Hospital; Derbyshire Royal Infirmary; Northampton General Hospital; St. Bartholomew’s Hospital, London; Derriford Hospital, Plymouth; Northern General Hospital, Sheffield; St. George’s Hospital, London; Dewsbury District Hospital, Yorkshire; Northwick Park Hospital, Middlesex; St. Helier Hospital, Surrey; Diana, Princess of Wales Children’s Hospital, Birmingham; Nottingham University Hospital, St. James’ University Hospital, Leeds; Diana, Princess of Wales Hospital, South Humberside; Ormskirk & District Hospital; St. Mary’s Hospital, London; Doncaster Royal Infirmary; Peterborough District Hospital; St. Peters Hospital, Surrey; Ealing Hospital, Middlesex; Pilgrim Hospital, Lincs; St. Thomas’ Hospital, London; East Surrey Hospital, Redhill, Surrey; Pinderfields General Hospital, Wakefield; Stepping Hill Hospital, Stockport; Eastbourne District General Hospital, East Sussex; Pontefract General Infirmary; Stoke Mandeville Hospital, Buckinghamshire; Epsom Hospital, Surrey; Queen Elizabeth Hospital, Kings Lynn; Sunderland Royal Hospital; Fairfield General Hospital, Bury; Queen Elizabeth, Queen Mother Hospital, Kent; Tameside General Hospital, Ashton Under Lyne; Hammersmith Hospital, London; Regional Spinal Injuries Unit, Southport, Merseyside; Taunton & Somerset Hospital; Harrogate District Hospital, Yorkshire; Rochdale Infirmary, Lancashire; The Horton Hospital, Oxfordshire.

References