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ACUTE SPINAL CORD INJURY: THE 4-6 HOUR WINDOW DEBATE

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INTRODUCTION

Rugby, as a collision contact sport, has an associated injury risk. Although infrequent, spinal cord injuries remain a concern as they are devastating to the players involved. In South Africa it is unclear what the exact incidence is. Dunn[1] reports on the Western Cape spinal column injuries over a 5-year period until 2008. On average, 5 spinal column injuries were managed per annum with 1.8 per annum representing catastrophic injuries, i.e. quadriplegia. Hermanus[2] provided a 27-year review on national rugby-related spine injuries. They identified 264 cases with a national average of 9 per annum, 61% being catastrophic.

The timing of intervention and the rapidity of access to care is controversial. A spinal cord injury in a young, fit adolescent is an emotionally charged scenario and places tremendous stress on the carer or carers. In the past, a 4-hour window has been promulgated. This has never been proven to be of ultimate benefit but largely based on anecdotal experience and intuition. Podium presentations to this effect have been given locally but there has been no specific published data. In a large country with rugby players injured in rural areas outside the metropole, 4 hours from field to hospital requires a huge resource to be ever-ready. Of course, this comes at a cost.

THIS DOCUMENT DEALS WITH THE ISSUES SURROUNDING THE "4-6 HOUR WINDOW".

DEFINITION

The concept of the 4-6 hour window implies that early intervention within this period will have a superior outcome, as compared to medical care occurring after this period. The time would start from the moment of injury, i.e. on the field.

MECHANISM

The most common anatomical region for spine injury in rugby is the cervical spine. Injuries occur during different phases of play. In years gone by, the set scrum posed the highest risk. This has been addressed with many law changes reducing the number of scrums as well as 'de-powering' it. This has been successful in reducing injury. However, as the pace of the game and the athleticism of players has increased, loose-play injuries have increased proportionally. Currently the tackle phase is the risk area – both for the tackler and the tackled player[1,2,3].

Cervical injuries occur from a combination of forces. These are either flexion or extension – forward or backward bend combined with compression or distraction. The forces are translated to the spine as the injured player collides with another player or the ground. In the example of a high tackle, the head and neck will be pulled (distracted) backwards (extension). The most common rugby injuries are those of flexion. This typically occurs when the player hits the ground. However, a flexion or extension force results depending on the manner he strikes. Should he be rolling forward, a distractive force may be generated that dislocates the facet joints. Equally, a compression force can occur if there is more of an axial load. This fractures one or more of the vertebral bodies.

If a distractive flexion injury occurs, the spinal cord is stretched and may be injured. In addition, the resultant dislocation narrows the space available for the spinal cord and causes ongoing compression. In the compression flexion injury, the vertebral body fractures and compresses the spinal cord from the front.

Once the spinal cord is injured by an indirect mechanism such as described above, a relentless physiological process occurs. There is an inflammatory process which further injures the cord. This is referred to as the secondary injury. The inflammatory process further damages the cells and thus the spinal cord function. There is associated swelling and cell death. In addition, the damaged cord loses the ability to maintain basic bodily functions such as pulse and blood pressure control. This reduced blood pressure has a further negative effect on the cord. Thus, a self-perpetuating downward spiral occurs.

As one cannot alter the initial injury other than through prevention, much medical research has been concentrated on the secondary insult. For many years, high-dose intravenous prednisone (steroid) was advocated. This was based on limited trials promoting better spinal cord functional outcome with these massive doses[4,5,6]. In recent times, this data has been questioned. There are many negative effects from high-dose steroids including death from respiratory infections, with very little evidence of benefit[10]. Most centres outside the USA no longer promote steroid use. The papers that support it, limit it to an 8-hour window[6]. This may well be where the concept of the 4-6 hour window originated.

PRESENTATION

A distractive flexion injury tears the posterior ligaments, i.e. at the back of the neck. This creates some instability but a low incidence of neurological injury. Further force causes one of the two facet joints to dislocate. This is termed a unifacet dislocation. This frequently does not cause neurological injury but depending on the amount of force dissipated may well injure the cord. The next step is a bifacet dislocation when both facets dislocate. This allows the spine to move forward on itself, producing traction and compression of the spinal cord. This is associated with a high incidence of neurological injury. In addition, there is ongoing spinal cord compression, which may cause deterioration in function in the incomplete injury (see later).

The compressive flexion injuries cause the vertebrae above to compress the one below. This causes the inferior (lower) vertebral body to deform and then split as the compression increases. This creates a teardrop-type fracture. As the force continues, the posterior (behind) ligaments tear and the superior (above) vertebrae moves back into the spinal canal and compresses the spinal cord. This causes neurological deterioration. Again there will be ongoing spinal cord compression with possible deterioration.

Spinal cord injuries are classified as complete or incomplete. This is based on whether there is any residual neurological function below the level of the injury. The spinal cord is responsible for relaying sensation, power (muscle control) and pain modalities. If a SCI causes total disruption and no sensation or voluntary muscle activity is present below the lesion, it is termed complete. As an example, if a C5 injured player can still shrug his shoulders but has no hand or leg sensation or movement, he is complete. This assessment is complex as the only remaining sensation may be peri-anal sensation. This is the last to go, and may be present even if the limbs are totally involved. This is extremely important to note as the ultimate outcome is vastly different in a complete or incomplete injury.

Another issue is the timing of the assessment. In the initial phase, spinal shock occurs. This simply means that the spinal cord is 'stunned' and that all reflexes are suppressed. During this period (24-72 hours) subtle areas of neurological preservation may be masked. Thus, final assessment can only be made once spinal shock resolves and the patient's reflexes have returned.

Carers need to be cautious about prematurely labelling a patient as 'complete', as this may alter interventions provided. Complete patients injured in low-velocity accidents, such as sport, need to be afforded optimal early management in case they fall into the above category.

LITERATURE REVIEW

The literature was reviewed with a focus on the timing of intervention after SCI. There is very little available on this, as it is difficult to study in a controlled fashion. Many of the drivers for early intervention are based on animal models. The problem with this research is that it frequently has little application to clinical practice. Firstly, injuries in clinical practice are not controllable and the force dissipated is often much higher than that of the animal models. Secondly, animal studies allow extremely early intervention – within minutes – which will never occur in the real world. However, the animal models do indicate that in the incomplete spinal cord injury, there appears to be better functional outcome and smaller volume of cord tissue injury with earlier decompression. Of course, this simply means lifting of the piston or weight, unlike the challenges related to bony fractures in often awkward locations.

Fehlings[7] identified 10 prospective studies dealing with the issue of surgical decompression. Of these, Papadopoulos's paper reports improvement in 39/66 decompressed patients versus 6/25 controls. La Rosa performed a literature review of available studies between 1966 and 2000. They concluded that those operated on within 24 hours had a better neurological outcome than those operated on after 24 hours. However, Fehlings identified many papers to the contrary where no benefit was shown. A criticism he makes of this literature is the variation and prolonged period which was regarded as early intervention, some as long as 72 hours. This however highlights the difference between reality and the animal studies.

Both the La Rosa study and Fehlings' literature review concluded that early decompression can only be considered as a practice option.

The issue of early closed reduction is also reviewed. This is the application of traction in an effort to relocate the dislocated spine. There are many proponents of early reduction (within 6-10 hours). The literature is difficult to interpret due to the lack of randomisation and controls. There is of course anecdotal evidence of neurological improvement with early reduction. Moreover, a number of studies did not find any neurological benefit due to reduction, with the possible exception of patients with bilateral facet dislocation[8].

There does not appear to be a downside to early reduction in terms of safety. Cotler[9] performed a prospective study of early reduction by traction in 24 patients and recorded no neurological deterioration in any of the patients, most of whom had successful reduction with closed techniques within 24 hours of injury.

Fehlings concludes that, based on the available literature, a recommendation for urgent reduction of bilateral locked facets in a patient with incomplete neurological status could be supported.

There are those that promote delay to allow the medical / physiological situation to stabilise. With a high cervical lesion the patient can be autonomically unstable, i.e. volatile blood pressure. They might require inotropic (adrenaline) support. This may make anaesthesia more difficult and add risk to the patient. The physician needs to take the individual situation into consideration. In cases where it is quite clear that surgery will not improve neurological function (complete cord disruption on MRI) and the aim is mechanical stability to allow early mobilisation, a delayed surgical intervention may be far safer. However, if there is any reason to believe decompression will improve neurological outcome (such as in an incomplete injury with slow deterioration), surgery should be expedited.

The length of admission is no different whether operated upon early or late. This is understandable, as the underlying neurological status will dictate the management (such as ventilation) and complications expected.

DISCUSSION

In summary, the literature is not clear on the issue of timing. Most injuries are from motor vehicle accidents while studies on sports injuries are even more limited. Rugby injuries differ from traffic accidents in that they are low velocity. This implies that less energy is dissipated when the injury occurs. This may well reduce the amount of injury the cord sustains.

It is of course impossible to know exactly the extent of SCI when the player is lying on the field. Even though the scrum simply collapses, the forces on the player's neck may be extremely large. However, as a group, one would expect the forces to be lower and thus the likelihood of incomplete SCI higher. In the immediate post-injury period, accurate assessment is difficult. The player is on the field. The parent or paramedic is unlikely to be confident about a full neurological assessment and may easily miss retained peri-anal sensation, even if re-examined in the ambulance. In addition, the spinal shock may mask it, as discussed above.

Therefore it would be reasonable to treat all rugby spinal injuries as potentially reversible.
Having said that, one needs to ask what is going to happen at the hospital on arrival to justify the efforts of rapid transfer. As steroids are no longer universally administered (never at the Groote Schuur Hospital ASCI unit), the need for the infusion before 8 and preferably 4 hours is no longer a reason, as it was in the past. Urgent reduction is only indicated in the dislocations which account for around 60% of the injuries seen[1]. It is unusual for these patients to undergo surgery immediately unless they are deteriorating neurologically.

The problem is that the information to make these decisions and decide which injured player would potentially benefit from a 4-6 window is not available on the field. The risk of taking the injured patient to a local hospital without a spine surgeon for an X-ray assessment, risks further delays of repeated transfer.

In my opinion, a rugby player sustaining a cervical dislocation should undergo reduction as soon as possible (whether closed or surgical), irrespective of immediate neurological status. Those with persistent compression from a fracture and deteriorating neurological status should undergo expedited surgical decompression. I do not believe that there is a specific window but that these patients should be transferred to an appropriate hospital (spinal surgeon available) as soon as possible.

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