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Review

The impact of the 21st century on rugby injuries

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Study Design: This is a review article concerning rugby injuries resulting in tetraplegia between the years of 1965 and 2000. It is based on a review of worldwide literature.
Objectives: The objective is to evaluate research that has taken place in the last 10 years.
Setting: The author first became interested in this problem in 1965 when there were few papers on the subject. Understanding of these injuries has changed decade by decade and the number of spinal injuries has increased dramatically. For the first three decades the author was actively engaged in gathering material particularly at the National Spinal Injuries Centre. During the last 10 years there has been a greater understanding in the mechanism of these injuries.
Methods: The following subjects were reviewed in the literature: (1) mechanism of injury; (2) the diameter of the spinal canal; (3) arthritis; (4) treatment; (5) fitness and (6) medico-legal aspects.
Results: These injuries are no longer regarded as an Act of God or bad luck but mechanisms of injury have been clearly defined.
Conclusion: To prevent these injuries occurring there is a need for: (1) better statistics; (2) enforcement of the laws; (3) improved standards of refereeing; (4) higher standards of fitness and training in particular to de-power the set scrum; (5) study of the cervical spine to look for abnormalities which would predispose the player to tetraplegia; (6) the use of MRI and CT scans to monitor the spinal cord anatomy; (7) the importance of pathology and the status of the cervical spine; (8) the awareness of the dangers to the cord of congenital and acquired abnormalities that could prejudice spinal cord function and (9) the awareness of the necessity for comprehensive insurance.
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Introduction

Rugby football is a popular game and is played in over 100 countries. As there is physical contact, injuries inevitably occur. The most catastrophic are to the cervical spine causing tetraplegia.

I became concerned when I began to see players with tetraplegia as a result of rugby accidents at the Liverpool Regional Paraplegic Centre between 1965 and 1970 and subsequently at the National Spinal Injuries Unit (NSIC) from 1970 to 1992. In September 1972[1] I described five cases. The number had increased to 18 by 1975.[2] There were few papers on the subject.

At the Liverpool Centre over a 20 year period, of 626 patients admitted, only three had rugby injuries.[3] At Stoke Mandeville, Frankel *et al*[4] described five rugby cases among 259 traumatic injuries over a 17 year period. In the early years there was only one serious spinal injury from rugby every 4 years. When confronted with the occasional injury, they were regarded as being 'the luck of the game' or 'an act of God'. Their rarity and this macho philosophy made it difficult to see any pattern or carry out any research.

There was a dramatic increase in the injuries from 1970 onwards, reaching a peak of nine players admitted to the NSIC with broken necks in the 1980/1981 season. Some players were still present in the Unit when the following season began. In March 1981 there were 13 players with broken necks at the NSIC. This constituted the commonest cause of admission from organised sport in the NSIC.[5] The patients were so concerned about their plight that they wrote to all the former patients at Stoke Mandeville who had broken their necks playing rugby and a

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meeting of patients and players was held at the NSIC (Figure 1).

In 1984[6] (first decade) the injuries to 67 rugby players were described and various factors were identified responsible for causing injuries, in particular, the head being driven into the ground. This had commonly occurred in the set scrums but, owing to a change in the tackle law in an attempt to make the game faster and more open, the loose scrums and the mauls were allowed to continue. As a result, a new mechanism of injury was identified: that was injuries occurring in the maul and the ruck when powerful players were wrestling, fighting for the ball, and their heads were driven into the ground breaking their necks. Greater skill did not protect. There was a higher incidence of injury in the first class skilled players than in the unskilled since they played more vigorously.

These findings were presented at a further meeting with administrators and rugby players at the Chiltern Hospital in 1983[5] and resulted in changes in the laws, the emphasis being to protect the head and neck by stopping the scrums from collapsing and preventing rucks and mauls from carrying on for too long.

In 1994[5] (second decade) my personal series had increased to 100 cases. There had been some improvement as a result of the law changes in that:

- the ruck and maul injuries had almost ceased;
- the overall number of admissions to the NSIC with tetraplegia as a result of rugby injuries had fallen;
- Injuries due to stupidity had ceased;
- Injuries due to the mismatch of strength and skill had ceased.

At that stage 226 papers had been published.

This is now the third decade since I began my research on the subject and I would like to address what the situation is at the beginning of the 21st century.

Figure 1 Meeting held in March 1981 at the NSIC. There are many paralysed players. Phil Blakeway and Roger Uttley (national coach) can be seen along with Leon Walkden

The game and the laws have changed. Players are bigger, stronger and fitter. Professionalism was adopted by the International Rugby Board (IRB) after the second World Cup in South Africa in 1995 resulting in increased publicity, commercial pressures and very substantial earnings. The advent of the European and World Cups has led to professional players travelling the world, playing in multiple competitions throughout the year.

Injuries, particularly of the cervical spine, are no longer regarded as bad luck or an Act of God. The mechanism of injury and their prevention are better understood. The aspects of the game that I wish to address are:

(1) Mechanism of injury.
(2) The diameter of the spinal canal.
(3) Arthritis.
(4) Treatment.
(5) Fitness.
(6) Medico-legal aspects.

Rapid transfer by helicopter

Players can be transported directly from the rugby field within hours of injury to a trauma unit. It is mandatory that there is availability of CT and MRI scans and a dedicated surgeon trained to operate upon the spinal column.

Mechanism of injury

In rugby, players lead with their heads and when the head strikes the ground the force is absorbed by the skull and the higher vertebrae and diminishes progressively lower down the vertebral column. The likelihood of damage occurring to the cervical vertebrae is enhanced by the disparity in the movements of the unsupported skull on the cervical spine. The danger of dislocation occurring is further increased by the alignment of the facets, which in the upper cervical spine do not present such resistance to dislocation as the vertebrae lower down because their facets are aligned less obliquely.

The production of a bifacetal dislocation is dependent upon the speed and the force which is exerted upon the skull. Diving accidents when the head strikes the bottom never seem to produce such a dislocation but produce a crush fracture whereas the slow collapse of the scrum is a mechanism par excellence for producing a dislocation.

Flexion

When a force is exerted through the crown of the head it is transmitted through the skull to the cervical vertebrae, resulting in crushing of the vertebrae and extrusion of the vertebral body and disc material posteriorly into the cervical cord. Dislocation may occur without any fracture of the vertebrae. Roaf,[7]

using cadaveric spines, was unable to produce dislocation without a fracture by hyperflexion alone and found that some rotation must be present. Bauze and Ardran,[8] in a critical and fundamental study, solved this problem by showing that when the vertex is fixed (being locked on the ground, a common occurrence in rugby) far less force is required to dislocate the vertebrae and dislocation can occur without fracture (a mechanism of injury reproduced and repeated on the rugby field). The association of disc lesions with dislocations was recognised by Bohlman[9] and the advent of the non-invasive investigation of MRI, which enables us to visualise the bones, the canal, the discs, the ligaments and the spinal cord, has shown that this occurs in up to 50% of cases.[10]

Extension

The anterior posterior diameter of the spinal cord is reduced during extension of the cervical spine so that when the neck is forcibly extended the spinal cord is compressed between the discs and vertebral bodies anteriorly and the lamina and ligaments posteriorly (tetraplegia). The cord can thus be injured without there being any overt fracture or dislocation – the so-called 'extension' injury (though in many cases the name is spurious since there is a tear through the disc which is not visible on conventional X-rays).

These have been identified either on the postulated mechanism of injury – extension (from a high short arm tackle) or so-called 'whiplash' injury. This was first described by Thorburn[11] but there is controversy as to the mechanism of spinal cord injury. Braakman and Penning[12] believe that nipping of the cord could take place by a pincer movement without there being dislocation.

The diameter of the spinal canal

An alternative or additional suggestion is that the reduction in size of the spinal canal could be an important factor in causing acute tetraplegia in patients with injuries since Payne and Spillane,[13] Adams and Logue,[14–16] Murone[17] and McMillan and Silver[18] demonstrated that in patients with chronic myelopathy the size of the spinal canal was reduced.

Cervical stenosis

Torg *et al*[19] described a distinct clinical entity of neurapraxia of the cervical spinal cord with transient quadriplegia in USA footballers. The X-rays were negative for fractures or dislocations but they did show developmental spinal stenosis in 17 patients, congenital fusion in five patients, cervical instability in four patients, and intervertebral disc disease in six patients. They postulated that neurapraxia resulted from a decrease in the anterior-posterior diameter of the spinal canal. Neurapraxia was produced by forced hyperextension, hyperflexion or axial loading of the cervical spine.

The absolute minimum sagittal diameter of the cervical spinal canal that can accommodate the spinal cord without cord compression is somewhere between 11 and 13 mm, depending on the relative diameter of the spinal cord. The cervical spinal canal can narrow up to 2 mm in full extension.

Measurement

Torg *et al*[19] tried to refine this concept by introducing a ratio of the size of the canal to the size of the vertebral body.

Ratio

Torg and Pavlov[20] described a sagittal diameter ratio of the central canal to the vertebral body and determined that a ratio of 0.8 or less was indicative of cervical stenosis. Odor *et al*[21] compared results of the ratio formula with diameters obtained from CT scans of the central canal in patients with myelopathy and agreed with the 0.8 ratio but Herzog *et al*[22] found the ratio to be of no value because the footballers had big vertebrae.

The presence of a narrow canal significantly influences the morbidity and prognosis of an injury. Patients with the lowest AP diameters have the most severe myelopathy after trauma. A person with symptoms of myelopathy over a 6-month period is obviously at greater risk for return of those symptoms than someone who had the episodes for 5 s. The hemiparetic symptoms are different from unilateral arm dysaesthesia.

To assess the severity of the episode, the following should be considered:

(1) The extent of the neurologic deficit.
(2) The duration of the paralysis.
(3) The narrowing of the central canal diameter.

Controversy

There are professional footballers who have a narrowed canal and yet have no signs or symptoms of spinal cord compression. Torg *et al*[19] were initially of the opinion that a patient with congenital stenosis was not at greater risk of developing permanent damage to the cord.

Cantu,[23] in contrast, believed that permanent tetraplegia was more likely after episodes of transient quadriparesis. He introduced the concept of functional stenosis. Torg *et al*[24] followed up Cantu's work and described four cases in his own series. Each of these four cases, Torg *et al*[24] believed, had some evidence of structural injury: two had an old compression fracture, a third showed ligamentous laxity and the fourth sustained injury of the C5 limbus vertebra.

The implications of these findings for Rugby Union players are that a player with a narrow canal, whether it be congenital, developmental or acquired, and who has an episode of tetraplegia as a result of a blow to his head, is at risk of developing serious neurological deficit subsequently. Albright *et al*[25] showed that the re-injury rate after all neck injuries was 17.2%. The twice injured players at all levels of play had an 87% chance of future injury. Therefore there is a significant recurrent injury rate.

Arthritis

Scher[26] studied three groups of 50 players aged 20–25, 25–30 and 30–35 years (Table 1). Each group consisted of 25 forwards and 25 backs and all were asymptomatic. They were matched up with 150 men who were also asymptomatic and films were taken of the cervical spine at a fixed focus distance of 180 cm.

Forty players showed degenerative changes. The highest incidence of degenerative disease was present in the front row and the locks and to a lesser extent in the loose forwards but we do not know how well they were represented. Interestingly eight of the players showed fractures: six compression, and two of the spinous processes. In the control group only one showed evidence of a fracture. In the general population it was only in the 40–50 year old age group that 25% to 29% showed degenerative changes. Scher[26] postulates that forces of up to 1.5 tons are generated in the front rows of the scrums. He says rugby players are likely to get pain and nerve root compression and concludes that the risk of spinal cord injury is increased in the presence of degenerative disease.

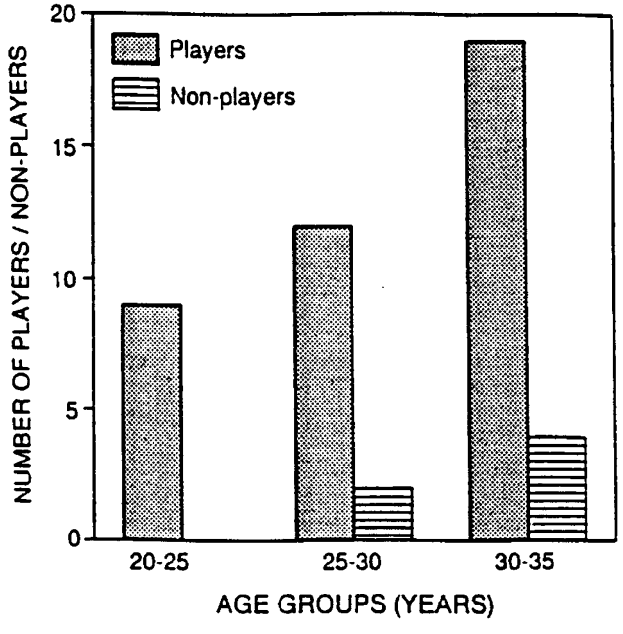
This work has been repeated by Berge *et al*[27] in France by MRI scanning. They compared 47 front row forwards with 48 matching controls. There were more early degenerative changes in the front row forwards and they attributed this to repetitive cervical trauma. They also found that the diameter of the cervical canal was significantly decreased in all the players. They considered that this reduction in diameter was directly related to the trauma. There was a significant loss of the vertebral body height resulting in significant acquired stenosis of the cervical spine in more than one in two senior players and three in four veteran players.

In my own series[6] four players had nerve root injuries secondary to degenerative changes of the vertebra and there are currently several international players who have had to have disc surgery because of degenerative disease of the cervical spine.

Rugby causes degenerative disease of the spine, which will give rise to long term morbidity. The question of whether such individuals should carry on playing rugby is a matter of judgement. These players may be earning £50–100 000 a year, or more, and arbitrarily removing them from the field of play may be hard, but Albright *et al*[25] showed that the re-injury rate after all neck injuries was 17.2%. The twice injured players at all levels of play had an 87% chance of future injury. Therefore there is a significant recurrent injury rate.

In view of the work of Scher[26] it is clear that arthritis is not restricted to one level.

Table 1 Scher.[26] Radiological changes of degenerative disease of the cervical spine in 150 rugby players and in a control group of 150 non-rugby players of different ages



Treatment

The patient must be transported as quickly as possible to a centre for investigation and treatment. This means the anatomy of the lesion should be determined by CT and MRI scanning. If the patient presents with a unifacetal or a bifacetal dislocation then disc injury is almost certainly present. It may be that, following a transient episode of tetraplegia, the cord injury will subside if there is no anatomical compromise of the canal, but, if disc prolapse is present, over the ensuing 24–48 h swelling of the cord will occur and lead to irreversible damage from secondary impairment of the blood supply.

It is necessary to restore the normal anatomy as soon as possible. Ideally, following CT/MRI scanning, reduction should be achieved. The contraindications to a closed reduction are that the manoeuvre to disengage the facets by flexion will inevitably lead to some further compromise of the spinal canal and thus put cord function at further risk. In South Africa where MRI and CT scanning are not freely available there has been a school of thought[28] which advocates immediate closed reduction whereas the thinking in the UK favours an open reduction.

Figure 2 Unfit players are likely to get injured

Late disc surgery
There are players who have developed chronic prolapse of a disc giving rise to neurological impairment. The treatment of these players by removal of the disc and their restoration to full play at international standard has given rise to controversy. Some doctors (myself included) have arbitrarily banned them from ever playing again whereas others think that the patient should be appraised of the risk and given advice accordingly.

Fitness

The purpose of my study of 1984[6] was to determine the reason why rugby players broke their necks. Was it due to lack of technique? Were players injured because they were not fit enough to be playing any kind of sport (Figure 2)?

Three players were injured because they had not received sufficient specific training. A wing three quarter was made to play as a front row forward because of his strength and general ability but was unable to cope with the particular skills required; a schoolboy who had played only three or four games of rugby was put in the first team and was injured in a maul and ruck in a match between schools; and another player had played for only half a season.

These problems have been specifically addressed by the governing body. Efforts have been made to de-power the scrum and to prevent it twisting. In the front row of the scrum, only players who are used to playing there can participate in scrums. Complicated arrangements are made so that if a front row forward is sent off, a replacement front row forward can play in his position. If a replacement is not available, uncontested scrums are awarded to prevent the scrums collapsing. My limited figures suggest that greater skill does not protect as six of the players injured were first class players and there were only about 2000 such players in England at that time compared with a total of 400 000 players at all standards. The large number of injuries sustained on the various Lions touring sides support the view that the stronger and fitter the player the greater the likelihood of an injury occurring.

Schneider[29] made a separate study of injuries to the cervical spine among American football players (Table 2). He found that 141 serious injuries occurred among 780 000 high school football players, 34 among 70 000 university footballers, and 14 among 4500 professional players; whereas in Sandlot football, an unskilled form of the game, 26 injuries occurred among 1 645 000 players. He concluded that unskilled players do not play as hard as highly skilled or professional athletes and that the greater degree of force and skill exaggerates the likelihood of injury. Injuries among schoolboys in my study would support this view. Sixteen of the schoolboys injured were team players, compared with four non-team players; most of the injuries occurred in competitive games. Again it is impossible to establish the total numbers of team and non-team players in schools, but in two public schools the ratio of team players to non-team players was 1 : 3.

This work has now been substantiated in detail by Garraway *et al*[30] who studied rugby injuries of all varieties (not broken necks). Garraway *et al*[30] studied players from senior rugby clubs in the Scottish Borders in 1993–94 when rugby union was entirely amateur and again in 1997–98 when the game had turned professional in the same clubs in the borders of Scotland (Table 3).

The proportion of players who were injured almost doubled from 1993–94 to 1997–98 despite an overall reduction of 7% of the playing strength of participating clubs. There was an injury episode every 3.4 matches in 1993–94 rising to one in every two matches in 1997–98. An injury episode occurred in a professional team for every 59 min of competitive play, the equivalent of 1.4 injuries per match.

The number of hours of competitive rugby was lower in 1997–98 yet the overall proportion of players injured almost doubled from 27% to 47%. The rise was particularly high in teenage players.

No spinal injuries were recorded in 25 224 playing hours in 1993–94. Two neck dislocations occurred in 23 487 playing hours in 1997–98, one of which resulted in permanent neurological damage.

The highest proportion (48%) of injury episodes occurred in the tackle in professional and amateur players (so it has changed from the scrum but these are not injuries to the spine). Thirty per cent of professionals in 1997–98 had not returned to playing/training by the end of the season.

Professionalism was adopted by the international rugby board (IRB) after the second World Cup in South Africa in 1995. The introduction of professionalism in rugby union has coincided with an increase in injuries to both professional and amateur players. The penalties for accepting the financial and other rewards accompanying professionalism in rugby union appear to include a major increase in player morbidity.

Table 2 Schneider.[29] Injuries to the cervical spine in USA footballers

	Total no of players	*Serious injuries*
High school footballers	780 000	141
University footballers	70 000	34
Professional players	4500	14
Sandlot football (unskilled)	1 645 000	26

Table 3 Garraway.[30] Injuries of all varieties

Type of player	*Date*	*Injury episode*	*Spinal injuries*
Amateurs	1993–94	1 in 3.4 matches	None in 25 224 playing hours
Amateurs & Professionals	1997–98	1 in 2 matches	Two neck dislocations in 23 487 : 1 resulted in permanent neurological damage

An injury epidose occurred in a professional team for every 59 min of competitive play, the equivalent of 1.4 injuries per match

Garraway *et al*[30] were of the opinion that professional rugby union produced higher injury rates than professional rugby league because tackles come in at greater force. This is not in accordance with my own figures and those derived from the Rugby League. The advent of professionalism has resulted in more emphasis being placed on strength, speed and stamina in all players.

Medico-legal aspects

Rugby is a high risk sport and insurance must be carried. The accident victim may be entitled to take proceedings for compensation for the tort of assault against the other player. Such an entitlement arises if the injury was sustained as a result of a deliberate foul as opposed to the 'rough and tumble' inherent in a contact sport. Someone voluntarily playing the game of rugby is regarded as having consented to the risk of such bumps and bruises (and even more serious injuries) which can arise through the fault of no one.

Additionally, if the injury arose from a deliberate foul, there may be a criminal element involved whereby the accident victim finds himself as a witness for the prosecution in proceedings taken by the police or against the assailant. Such prosecutions are rare but not rare enough or, looked at another way, do not occur often enough. There are too many deliberate fouls in the game. The penalty from the court against a convicted player will depend on the circumstances ranging from a fine through to imprisonment.

If a player is injured as a result of a deliberate and unprovoked assault causing serious substantial injury then there is likely to be an entitlement on the part of that player to make a claim against the Criminal Injuries Compensation Board which pays compensation to victims of criminal offences (Figure 3). There is no double compensation. If the player can get redress by way of compensation as a result of his own law suit, then one sum is used to counterbalance the other.

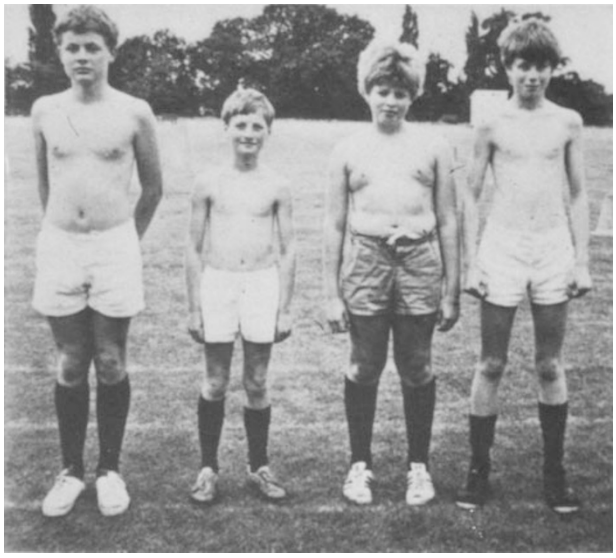
The injured player may have an entitlement to take proceedings against a number of different persons or bodies depending upon the circumstances. These can include (a) his school; (b) the opposing team's school; (c) the referee; (d) the team coach; (e) his rugby club; (f) the opposing team's rugby club; (g) the emergency services (or others) if the first aid is carried out in an improper manner thereby aggravating the injury and (h) the owners or controllers of the playing area (if the injury was caused needlessly by an unsafe pitch).

Civil liability of schools and school authorities

Schoolboys are in a different position to adults as the school has a duty of care. A player at school is entitled to take proceedings against his school or the opposing school if the injury arose through negligence. This means proving a duty of care, a breach of duty and damage resulting. If a schoolboy can demonstrate, on the balance of probabilities, the type of factors listed below, then the seeds of a successful claim for compensation can develop but this list is not exhaustive:

- Inadequate coaching: has the standard of coaching fallen below acceptable levels as customarily played? This leads to debate as to what are acceptable standards;
- Unevenly matched teams with regard to size, weight and experience (Figure 4);
- Lack of proper warm up;
- Lack of proper pre-match fitness;
- Unsuitable playing conditions;

Figure 3 This picture drawn in 1925, although humorous, does get the flavour of rugby played at that time. The players jumped on top of each other and subsequently had their necks broken



These boys are all 11 years of age!

Figure 4 Difference in height and weight in 11-year old boys. At puberty this discrepancy increases

- Unsuitable first-aid arrangements;
- Lack of teaching of the laws of the game;
- Inadequate refereeing;
- Over-psyching up.

The highest profile case was that of *Van Oppen* vs *The Trustees of Bedford School* (1989).[5] The pupil was seriously injured in 1980 when 16 years old. The claim was pursued on two alternatives: firstly with regard to the duties of the school to make sure that the injury itself was avoided and secondly with regard to the failure of the school to advise the boy's parents of the need to take out insurance protection against such accidents.

The case was lost but nevertheless the decision and the legal principles involved gave encouragement for another lawsuit (*Quinn* vs *Devon County Council*[5]) to be pursued by a different judge. This was on behalf of a pupil at college who sued the local authority responsible for management of the game when he fractured his spine in two places. Again similar allegations regarding lack of coaching, over-psyching and lack of a warm-up were paraded before the judge who, after about 5 days of strong evidence about the way in which the team had been coached, gave very strong hints that he wanted the claim settled. Negotiations then took place and a case which could have been a landmark decision against a local authority was buried on terms which remain confidential.

Based on these two cases, and particularly the case of *Quinn* v *Devon County Council*,[5] there have been a number of other claims pursued which have not proceeded to trial but where negotiated settlements have been achieved in favour of the injured person. In the case of *Smoldon* vs *Whitworth and Nolan*, the judge relied on the defendants' referee's evidence that it was improper to allow the set scrum to collapse six times and this was negligent.

The evidence from my research work and the conclusions reached resulted in changes in the laws. I gave evidence to the court in all three cases, in the first case in person.

Conclusion

The most dangerous situations are when the player's head is driven into the ground or hyperextended. The increase in serious injuries has led to a search for their causes and how they could be prevented. This has led to:

- endeavours to produce proper statistics;
- enforcement of strict laws to stop the scrums collapsing;
- the use of the touch judge as an extra referee to observe what is going on in the set scrums;
- improved standard of refereeing;
- better standards of fitness and training, in particular, efforts to de-power the set scrum and monitor it;
- study of the cervical spine to look for abnormalities which would predispose the player to tetraplegia;
- the use of MRI and CT scans to monitor the spinal cord anatomy;
- the importance of pathology and the status of the cervical spine;
- an awareness of the dangers to the cord of congenital and acquired abnormalities that could prejudice spinal cord function;
- the awareness of the necessity for comprehensive insurance.

As a result of several lawsuits these recommendations are being enforced.

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