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# The need to make rugby safer

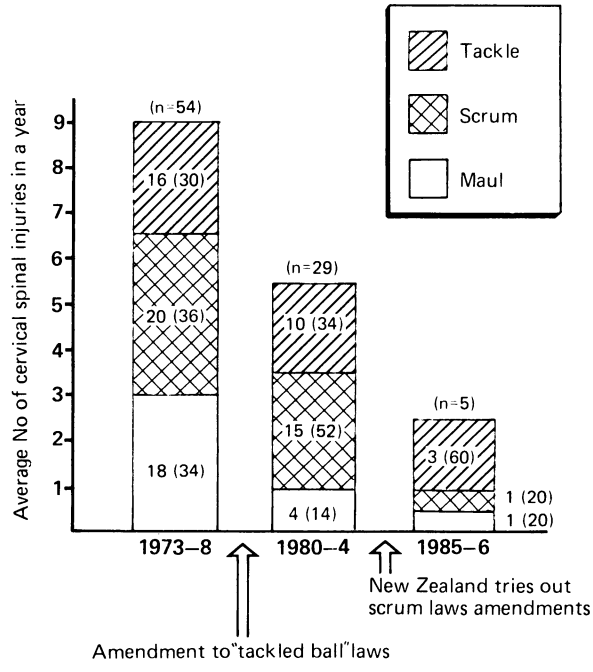
Cervical spinal cord damage is a known hazard of rugby, and changes in the rules of the game have been accompanied by a dramatic fall in the number of such injuries in New Zealand. The risk is now estimated to be less than one in a million appearances. Yet the International Rugby Football Board has declined to adopt these changes. Perhaps as the home internationals begin in the British Isles and France it is time for the board to think again, not least because the law may find rugby administrators negligent if they do not.

Most cervical spinal cord injuries occur in scrums, mauls, or tackles.[1-3] The unexpected finding that mauls after tackles were particularly hazardous caused a revision of the laws in 1980. Only five cervical spinal cord injuries have occurred in mauls in New Zealand since 1980, although on previous experience 21 would have been expected.

Most cervical spinal cord injuries occurring in scrums happen when the front rows collapse on to the ground or as the two opposing front rows collide, but others are due to the deliberate ploy of forcing the opponents upwards—"popping." This tactic may leave the head of the middle player (hooker) trapped and forced into flexion and rotation with distraction. The New Zealand Rugby Football Union in 1984 obtained dispensation from the International Rugby Football Union to try modifying the laws of rugby to make the scrum safer. These changes sought to lessen the chance of a scrum collapsing by altering the players' binding methods, by minimising its duration, and by preventing the scrum moving more than 1·5 m or wheeling. These laws have been used to control all domestic matches below senior level in 1985, 1986, and 1987 seasons. Other modifications sought to decrease the force of impact as the opposing scrums came together. The changes in the laws do not affect the fundamental nature of the game.

Whereas nine cervical spinal cord injuries occurring in scrums could have been predicted in this period, only one occurred. This case was the result of a completely inexperienced player being deliberately "popped" out of the scrum by a stronger, well organised opposition. It illustrated the folly of allowing a player to compete in the front row of a scrum without adequate training. It was also evidence that disruption of a scrum upward may be as hazardous as a collapse downward.

The reduction in the number of cervical spinal cord injuries after the two law changes is shown in the figure. The decline cannot be attributed to there being fewer players at risk or fewer games in a season. The extensive campaigns to introduce safer techniques and increase awareness of the hazard may have had some influence, but the fact that the number of cervical spinal cord injuries occurring in tackles has not declined sharply like those occurring in scrums and mauls does not support that view. The changes in the laws have probably been the major reason for the improved safety.




Cervical spine injuries in rugby games in New Zealand. Figures within each bar are average numbers (and percentages) of injuries occurring each year in tackles, scrums, and mauls in each period.

Although since 1985 there have been no cervical spinal cord injuries in New Zealand occurring in the collision phase of scrums, there continue to be such accidents in other rugby playing countries and the danger has not been eliminated in New Zealand by the modifications. Such accidents could be prevented by requiring the front rows to join before the

remainder of the players apply their weight. The amount of pressure generated by three players of each side coming together would not be sufficient to cause a fracture or dislocation of the cervical spine. We have substantial experience of playing and coaching in international and provincial rugby and believe that the proposal has considerable merit. [Dr Burry was an All Black and Mr Calcinai played in the front row of the Otago provincial scrum.—ED *BMJ*.]

A new dimension has been added by the decision in April 1987 of an Australian court to award more than $2m damages to a youth who became tetraplegic after a rugby league scrum. The judge castigated the state government for failing to make known to the player and his coach the fact that players with long necks were much more vulnerable to cervical injury and should not be allowed to play in the front row of a scrum. Since the administration were known to be aware of this fact (or opinion) they were found to be negligent in not disseminating warnings.

A recent comprehensive study of cervical spinal cord injuries in various football codes in Australia found that in rugby union most injuries occurred in the collision phase of scrum formation.[4] As this danger can be eliminated by requiring the opposing front rows to engage and stabilise themselves before the remainder of the players take up their positions, legal action could be taken against the administration of rugby union by any player who damaged his cervical spine during the formation or collision phase of a scrum. Failing to alter the procedures of a game despite the knowledge that existing practices were hazardous and a safe alternative existed could well be held by a court to constitute culpable negligence.

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# Tardive dyskinesia

Tardive dyskinesia is a disorder of movement caused by dopamine antagonist drugs—mainly antipsychotic drugs but also antiemetics such as metoclopramide and prochlorperazine. The core feature is orofacial dyskinesia, although abnormal involuntary movements of the trunk and limbs, usually described as choreiform, are commonly included in definitions of the syndrome. The condition was first reported within five years of the introduction of antipsychotic drugs.[1] The prevalence of tardive dyskinesia among schizophrenic patients receiving antipsychotic drugs varies widely from 0·5% to over 50% with a mean of around 20%.[2] In most cases the condition is mild and not progressive and tends to wax and wane.[3 4]

The most popular pathophysiological explanation of the condition is that patients develop more postsynaptic dopamine receptors in the striatum in response to blockade by dopamine antagonist drugs.[5] Flaws and inconsistencies in this theory have, however, been identified.[6] For example, though this denervation supersensitivity would seem to be an inevitable consequence of giving antipsychotic drugs, tardive dyskinesia develops in only a few patients. Other variables must be important. Further, the term "tardive" referred to the assumption that the condition was a late complication of drug treatment, but a clear association has not been shown between developing tardive dyskinesia and the length of antipsychotic drug treatment or the type or the class of the drug.[2 3 7]

Tardive dyskinesia is both more common and more severe in the elderly, and usually the abnormal movements appear during or after the sixth decade. This pattern may be partly caused by age related changes in the brain, possibly degenerative changes in the nigrostriatal system.[8] Tardive dyskinesia is more likely to remit in younger patients whether or not their drugs are stopped.[3 8]

Limited evidence suggests that vulnerability to tardive dyskinesia may be predicted by the patient developing parkinsonism, acute akathisia, or acute dystonia early in drug treatment,[9 10] and in schizophrenic patients having long term antipsychotic drugs akathisia and tardive dyskinesia commonly coexist.[11] Patients with affective disorder, both unipolar and bipolar, who receive antipsychotic drugs may be at a high risk of developing early tardive dyskinesia—that is, within two years of starting drug treatment.[10 12 13] Among patients with schizophrenia those with mainly negative symptoms, such as poverty of speech and flattening of affect, may be particularly susceptible to the condition.[9 14] In addition, the balance of evidence suggests that schizophrenic patients with tardive dyskinesia show more signs of both organic brain damage and cognitive impairment.

The seemingly endless list of potential treatments for tardive dyskinesia that have been tested is testimony to the failure to identify a specific drug treatment.[15] The latest putative remedies for which some success has been reported include calcium channel blockers (such as verapamil and diltiazem[16 17]) and α tocopherol (vitamin E),[18] but double blind placebo controlled trials are needed to substantiate these claims. The first response to tardive dyskinesia must be to prescribe antipsychotic drugs only when they are clearly indicated. Their use in patients with affective disorder should be tempered with the knowledge that they may be particularly at risk of early dyskinesia. There is no convincing evidence that any antipsychotic drug is less likely to produce tardive dyskinesia. Claims have been made that sulpiride is less likely to cause tardive dyskinesia, but the clinical importance of this remains to be established.[19] Secondly, drug treatment should be continued only when there is evidence of benefit. Thirdly, the daily dosage should be maintained at the minimum effective dose. Fourthly, intermittent treatment should be avoided. Drug holidays have been recommended for reducing the risk of tardive dyskinesia, but the limited evidence suggests that interruptions in the drug treatment may increase the risk of both persistent dyskinesia and psychotic relapse.[20] Fifthly, any concomitant anticholinergic drugs should be stopped if this is possible without precipitating severe parkinsonism. Though concurrent anticholinergic medication worsens tardive dyskinesia there is no convincing support for the popular idea that long term anticholinergic drugs increase the risk of