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CAUSATION OF INJURY

One of the major roles a sports medicine doctor plays is advising on prevention of injury. To understand how injury can be prevented one must understand how injury is caused.

Except for the overuse injuries which to a large extent occur in endurance sports most injuries are due to overloading of a body part. This may be due to intrinsic factors e.g. contraction or stretching of a muscle beyond which that muscle cannot adapt.

The second factor is extrinsic which means that an external force is applied to a body part which cannot withstand it. Tearing of knee ligaments following a rugby tackle is a good example of this.

The overloading may be singular or repetitive, in the latter giving rise to chronic injuries such as tennis elbow.

Thus there are two basic factors in the causation of injury

- the force;
- the adaptability of the body to resist the force.

Let us consider the second factor first.

A large number of variables are associated with ones ability to adapt to force.

Age

In youth the epiphyses are more liable to intrinsic overloading than any other part of the muscle-skeletal system. Severe disease of the heel (9-12 years), Osgood-Schlatter disease of the upper tibial tuberosity, (13-15 years) and apophyseal avulsion injuries of the pelvis (16-18 years) are common growth injuries.

From 18 years to 30 years the epiphyses are usually fused and muscle tears are more common. After 30 years of age injuries to tendons are more common as these become progressively weaker with ageing.

Extrinsic forces are much more variable injuries occurring to all tissues depending on the site of application. Muscles tend to protect other tissues. In adolescence muscles are relatively weak and protection of the spine may be inadequate resulting in spinal injuries.

In older athletes there is progressive deterioration of tissue with a loss of tissue elasticity. Over loading forces are more likely to result in injury to weakened tissue.

Ligamentous Laxity

This is somewhat controversial but it would appear that people with lax ligaments are more likely to injure ligaments resulting in dislocations. Palettar subluxation and dislocations are typical examples of this where minimal violence may cause injury.

Fitness

Good fitness implies that the sportsman has adapted to the stresses that the specific forces have placed on his body. Fitness is specific; a long distance runner will require muscle endurance and a weight-lifter muscle strength. To obtain fitness progressive loading is mandatory. Failure of adaptation occurs when the training programme has been too rapid. If the forces are too great no amount of fitness will prevent injury.

Warm up

An adequate warm-up means that the tissues have not been put into the functional mode i.e. sudden overloading is liable to cause injury. At least one degree increase in body temperature and a full range of joint movements are the minimum requirements.

Technique

Incorrect technique causes overloading and is a major source of injury.

Force

If the force exceeds the maximum loading a tissue can tolerate tearing will occur no matter how great the fitness of the athlete.

Thus in a sport like rugby a tackle of excessive force will cause injury. If the tackle is high or late, injury is more likely to occur. Excessive forces in rugby may occur in a number of situations. The scrum, especially the loose scrum, and the tackle are the two danger areas.

In conclusion before one can offer advice on prevention of injuries it is essential to understand all the factors which cause injury.

Dr. Clive Noble MBBCh, FCS(SA)
Editor-in-chief

ALTITUDE FAILS TO INCREASE SUSCEPTIBILITY OF ULTRAMARATHON RUNNERS TO POST-RACE UPPER RESPIRATORY TRACT INFECTIONS

Distance runners are more pre-disposed to URT infections than non-runners

EM Peters

INTRODUCTION

Reduced host resistance to infection following participation in an ultramarathon event, has been attributed to damage to the local mucosal membranes of the upper respiratory tract (URT). Physical properties of the mucous membranes have been reported to change and to result in impaired lymphocyte, neutrophil and macrophage function (Solomon and Armkraut, 1981).

It was hypothesized that the amount of damage to the mucous membranes should be greater at altitude due to:

- the reduced relative humidity and greater "dryness" of the air inhaled by runners; and
- the increased pulmonary ventilation at a given running speed.

This study compared the percentage incidence of URT infections following a 56 km ultramarathon run in Pretoria (reported relative humidity: 52%; altitude ±160m) to the incidence obtained amongst runners who had completed a 56 km at sea level (reported relative humidity: 79%) in Cape Town (Peters and Bateman, 1983).

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METHOD

The officially recorded maximum and minimum temperature and relative humidity readings in Cape Town on 10 April 1982 were obtained from the weather bureau in Pretoria.

Questionnaires were completed by ±130 entrants for the Milo Korkie Ultramarathon, a 56 km race from Pretoria (±1600 m above sea level) to Johannesburg (1800 m above sea level) during the seven days prior to the race. Information requested included running history, state of health and training record prior to participation in the race. Each athlete nominated a control of similar age and with whom they resided or were in frequent contact before the race.

Two weeks after the race, 116 runners were questioned with regard to URT symptoms experienced during the post-race period and their running performance in the 56 km race. The incidence of symptoms among the control group during this period was also recorded.

Data with regard to environmental conditions on the day of the race were obtained from the Weather Bureau in Pretoria (Table 1).

Statistical analysis of the results included the use of chi-square analysis and calculation of co-efficients of correlation.

RESULTS

Of the 117 subjects who were questioned, 106 (90,6%) were male and 11 (9,5%) were female. Five (4,3%) of the subjects were less than 25 years old; 84 (72,4%) were aged between 25 and 40 years and 28 (23,9%) were over the age of 40.

Thirty-one (28,7%) of the 108 subjects who completed the race reported non-allergy derived URT symptoms during the two weeks following the race as compared to 14 (12,9%) among the control group. This is a significantly higher percentage incidence ($x^2=22,8$; $p<0,005$). Only seven (23%) of the symptomatic runners were matched with symptomatic controls. Seven of the runners remained symptom-free despite being in contact with a symptomatic control.

The greatest majority (57%) of the total sample of runners who completed the race (n=108) had completed less than 85 km.wk^{-1} in training. Thirty-five percent of this subsample who had completed less than 85 km.wk^{-1} became symptomatic. Only eight percent of the symptomatic group (n=108) had completed more than 120 km.wk^{-1} in

Table 1: Environmental Conditions at the time of 56 km races in Cape Town (sea level) and Pretoria (moderate altitude)

Venues of 56 km races	Relative Humidity	Maximum Temp.	Minimum Temp.	Mean Temp.
Pretoria	52 %	25°C	14,8°C	20°C
Cape Town	87 %	24°C	14,0°C	16°C

Table 2: Incidence of URT symptoms amongst runners and their matched controls (n=108)

	Asymptomatic Control	Symptomatic Control
Symptomatic Runners	24	7
Asymptomatic Runners	68	7

Table 3: Time taken to complete the race in symptomatic and asymptomatic groups

Time taken to complete race	<4h30	4h30 – 5h00	5h00 – 5h30	5h30 – 6h00	>6h00
Symptomatic subjects	7	10	6	6	2
Asymptomatic subjects	15	18	22	18	2
Percentage Symptomatic	31,8	35,6	21,4	25	50
n	22	28	28	24	4

Table 4: Nature and Duration of symptoms (n=46)

	<1 day	1 3 days	4 – 7 days	>7 days	Total
– Running nose	2	5	2	6	15
– Sneezing	–	3	–	4	7
– Sinusitis	2	1	–	1	4
Nasal	4	9	2	11	26
Sore throat	–	9	2	3	14
Cough	–	1	1	2	4
Fever	–	–	–	2	2
Total number of symptoms	**4**	**19**	**5**	**18**	**46**

training.

Only 12 of the 108 who finished the race, did not compete at maximal effort, indicating dissatisfaction with the times in which they completed the race. Of the 31 runners who completed the race and developed URT symptoms thereafter, 29 (94%) indicated that they were satisfied with their performance in the event.

The greatest number of symptomatic runners (n=10) finished the race between 4h30 and 5 hours. However, of the total subsample which finished the race in less than 5 hours (n=50) 34 percent (17) were symptomatic (Table 2). With increasing time taken to complete the race, the percentage symptomatic decreased to a 24 percent incidence amongst the group completing the 56 km between 5h30 and 6 hours. It increased again in the sample taking more than 6 hours to complete the race (n=28).

The most prevalent symptoms after the race were nasal (57%) followed by sore throats (30%). Thirty-nine percent of the symptoms lasted for more than seven days and only two reported fever accompanying the URT symptoms. Four (9%) of the symptoms reported were trivial lasting less than one day. Eleven (33,3%) of the symptomatic subjects treated their symptoms with various cold cures and/or anti-pyretics, whereas one subject reported taking anti-biotics.

In an attempt to include only patients who presented with symptoms of an infective origin, persons with a recent history of allergic rhinitis (n=5) were excluded. As sinusitis is usually infective in origin, this was included in the nasal symptoms category and was not isolated as a separate category. Lower respiratory tract conditions (e.g. bronchial, tracheal) were not included in this survey.

DISCUSSION

This epidemiological study confirms previous findings of a significantly higher incidence of URT infections amongst runners than their matched controls following participation in an

ultradistance marathon event (Peters and Bateman, 1983; Douglas and Hanson, 1978). The higher incidence of URT symptoms amongst runners following participation in ultradistance running events has been attributed to:

- the impairment of host resistance to infection resulting from the prolonged stress; and/or
- local damage to the mucosal surfaces resulting from their drying and cooling during mouthbreathing (Peters and Bateman, 1983).

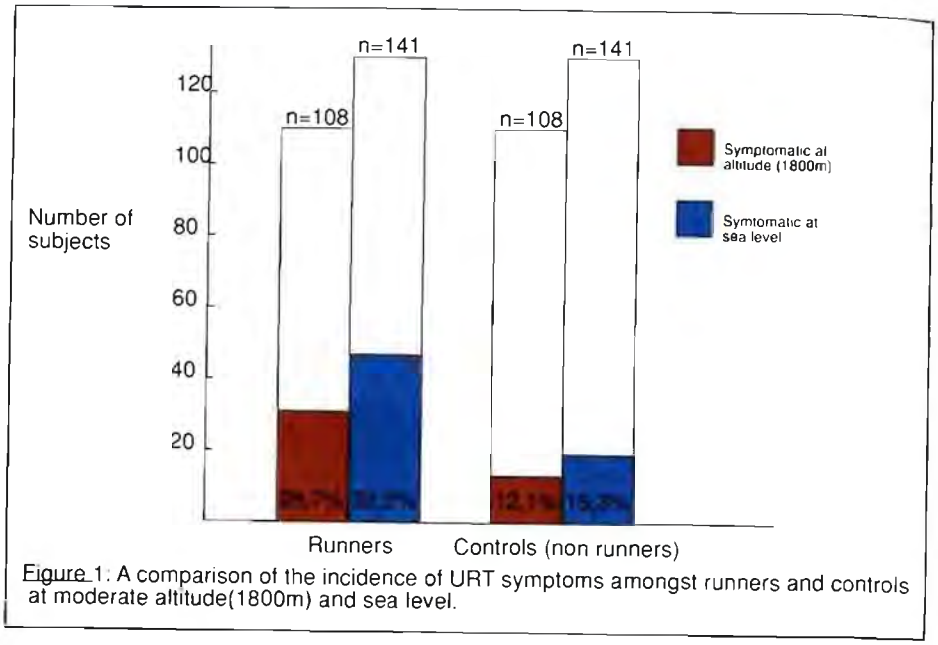

Figure 1: A comparison of the incidence of URT symptoms amongst runners and controls at moderate altitude(1800m) and sea level.

In this study, the exposure to moderate altitude with the concomitant increase in pulmonary ventilation, higher mean temperature and greater dryness of the air (Table 1) did not result in an increased incidence of URT infections among a sample of runners who completed a 56 km race when compared to the incidence among a similar sample who ran a race of the same length at sea level. These results thus appear to indicate greater contribution of impaired systemic immune mechanisms than reduced local mucosal function to the reduced resistance to infection experienced by the athletes.

It is well documented that the physiological and biomechanical stress of exercise results in elevated blood catecholamine and corticosteroid levels, which, in turn, influence immuno-competence. Adrenaline concentrations in the blood have been reported to rise as much as 8 times during prolonged exercise (Galbo *et al*, 1977) thereby possessing substantial potential to modulate immune function. Elevated adrenaline levels are known to reduce the number and function of circulating lymphocytes, resulting in both lymphocytosis and neutrophilia (Gary *et al*, 1983). Suppression of immune function has also been reported following elevation of blood cortisol levels (Solomon and Armkraut, 1981) and manifests with:

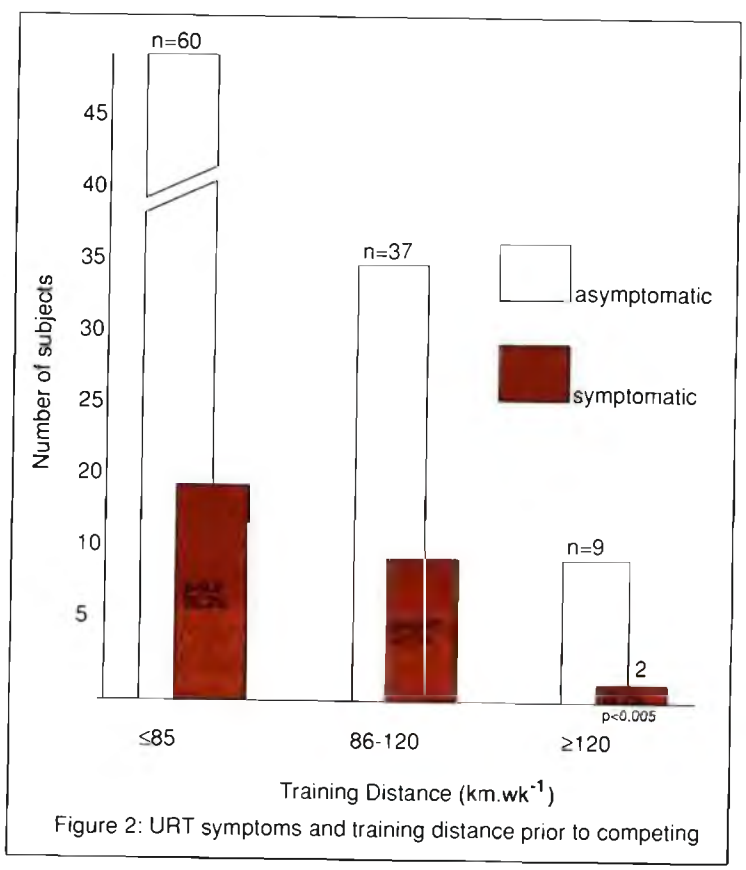

Figure 2: URT symptoms and training distance prior to competing

- selective depletion of T lymphocytes and reduced T cell cytotoxicity (McCarthy and Dale, 1988);
- macrophage receptor is the blockage and chemotaxis (Lewicki *et al*, 1987);
- reduced distribution and activity of natural killer (NK) cells. (MacKinnon, 1989).

Eskola *et al* (1976) have reported reduced lymphocytosis at the end of marathon races while Edwards *et al* (1984) reported significant changes in the B cell/T cell lymphocyte ratio. Recently, research workers have been able to discriminate changes in the subsets of T lymphocyte compartment showing a reduction in the helper to suppressor ratio following exercise (Berk *et al*,

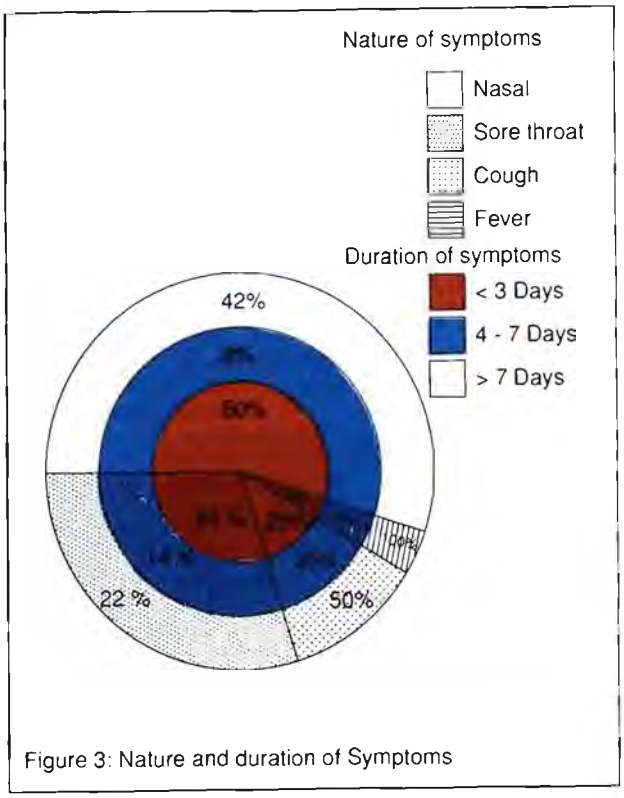

Figure 3: Nature and duration of Symptoms

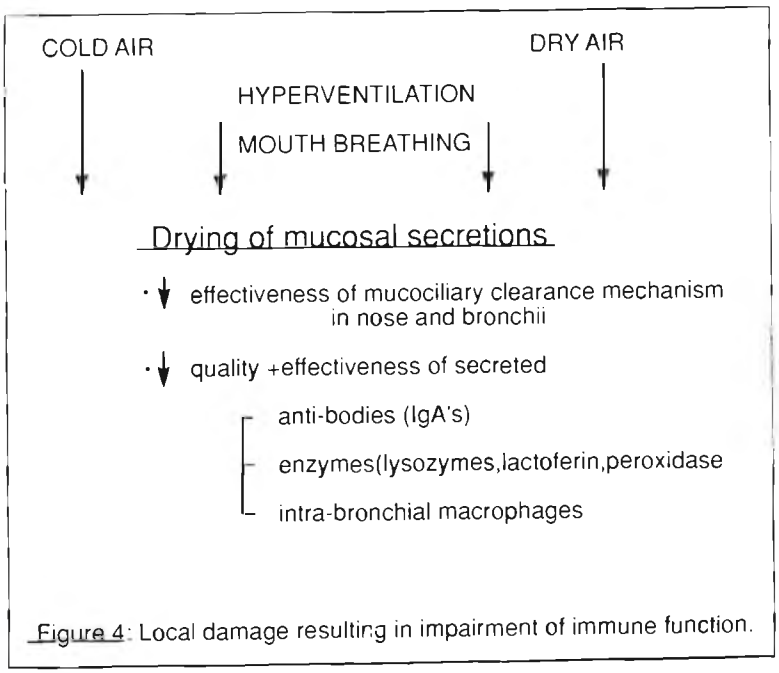

Figure 4: Local damage resulting in impairment of immune function.

1985). Further, neutrophilia (Petrova *et al*, 1983) eosinophilia and eosinpenia (Keast *et al*, 1988) have been recorded following exercise.

A recent study by Lewicki *et al* (1987) suggests that non-specific immunity may be the greatest contributor to increased susceptibility of suppression of resistance to infection. This is supported by the studies of Tomasi *et al* (1982) tested eight nationally ranked Nordic skiers before and after completion of a 50 km race. When compared to a control group, lower levels of salivary IgA were found in the skiers before the race and antibody levels dropped even lower during the race. Petrova *et al* (1983) produced evidence of reduction of both serum and secretory immunoglobin which was related to increasing intensity of exercise workload.

Smith *et al* (1988) have described that with greater oxygen turn-over during exercise, greater production of oxygen derived free radicals can play a substantial role in depressing immune function. It is therefore possible that the athlete who is deficient in the anti-oxidant vitamins (C, E and A) may have a greater susceptibility to infection than the runner who possesses more positive vitamin status. This is a factor which we are at present investigating and which may well have increased the results of this and previous epidermiological surveys.

This study showed that the less trained runners, i.e. runners who have completed less than 84 km.wk^{-1}, had the highest incidence of infection. It is possible that the combination of little training with less oxygen radicals available to activate the neutrophils (less respiratory burst) and the greater degree of acute stress experienced by the unadapted organism with greater impairment of host resistance to infection, may have contributed to this result. It was not possible to address the "overtraining" phenomenon as so few of our sample had been completing more than 160 km.wk^{-1} in training and no record was kept of the intensity of the training.

As found in our previous study, there was a high incidence of infection (± 32%) amongst the faster athletes. Those athletes who completed the race in less than 5 hours had encountered a greater stress situation than the slower athletes. On the other end of the spectrum, the athletes who were on the road for more than 6 hours, also showed a greater incidence of infection. It appears that prolonging the stress situation may also result in greater impairment of host resistance to infection.

Three factors thus point to decreased general host resistance to infection and not local mucosal being the primary cause of the increased incidence among ultra-distance runners. Firstly, despite the environmental conditions being more conductive to local mucosal dam-

age than in our previous study, this was not reflected by a greater incidence of infection. Secondly, the greater stress experienced by the less trained athletes correlated with a higher incidence of infection and thirdly, the fact that in both studies, it has been in the faster group of athletes who also experienced greater impairment of their immune response at systemic level, that the greater incidence of infections was recorded.

It is however, incumbent upon researchers to continue with biochemical investigation into the exact mechanisms underlying this apparent reduced resistance to infections following prolonged exercise.

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REFERENCES

1. Berk LS, Tan SA, Nieman DC, Eby WC: The suppressive effect of stress from acute exhaustive exercise on T lymphocyte helper/suppressor cell ratio in athletes and non-athletes. Abstract. *Medicine and Science in Sports and Exercise*, **17**: 706 (1985).
2. Crary B, Hauser SL, Borysenko M, Kutz I, Hoban C: Epinephine-induced changes in the distribution of lymphocyte subsets in the peripheral blood of humans. *Journal of Immunology*, **131**: 1178-1181, (1983).
3. Douglas DJ and Hanson PJ: Upper respiratory infections in the conditioned athlete. *Medicines and Science in Sports and Exercise*, **10**: 55 (1978).
4. Edwards AJ, Bacon RH, Elms CA, Veradi R, Felder M: Changes in the populations of lymphoid cells in human peripheral blood following physical exercise. *Clinical and Experimental Immunology*, **58**: 420-427 (1984).
5. Eskola J, Ruuskanen O, Soppi E, Viljanen MK, Jarvinen M: Effect of sport stress on lymphocyte transformation and anti-body formation. *Clinical and Experimental Immunology*, **3**: 339-345 (1978).
6. Galbo H, Christensen NJ, Horbst JJ: Glucose-induced decrease in glycogon and epinephrine responses to exercise in man. *Journal of Applied Physiology*, **42**: 525-530 (1977).
7. Keast D, Cameron K. and Morton AR: Exercise and the immune response. *Sports Medicine*, **5**: 248-267 (1988).
8. Lewicki R, Tchorzewski H, Denys A, Golinska A: Effects of physical exercise on some parameters of immunity in conditioned sportsmen. *International Journal of Sports Medicine*, **8**: 309-314 (1987).
9. MacKinnon LT: Exercise and natural killer cells. What is the relationship? *Sports Medicine*, **7**: 141-149 (1989).
10. McCarthy DA and Dale MM: The Leucocytosis of exercise. A review and model. *Sports Medicine*, **6**: 333-363 (1988).
11. Nicholl PJ, Williams BT: Medical problems before and after a popular marathon. *British Medical Journal*, **285**, (1982).
12. Peters EM, Bateman ED: Ultramarathon running and upper respiratory tract infections, *SA Medical Journal*, **64**: 1 582-584, (October 1983).
13. Solomon GF, Armkraut AA: Psycho-neuro-endocrinological effects in immune response, *Annual Review Microbiology*, **35**: 178-179, (1981).
14. Smith JA, Telford RD, Hahn AG, Mason IB, Weideman MJ: Training, Oxygen radicals and the immune response, *Excel*, **4**: 3-6, (1988).
15. Tomasi TB, Trudeau FB, Czerwinski D, Erredge S: Immune parameters in athletes before and after strenuous exercise. *Journal of Clinical Immunology*, **2**: 173-178 (1982).

RUGBY INJURIES OF THE CERVICAL SPINE AND SPINAL CORD – HAS THE SITUATION IMPROVED?

AT Scher

ABSTRACT

Paralysis due to injury of the cervical spinal cord in rugby players, is a subject of great concern. Since the publication of previous reports on these injuries, considerable attention has been paid to amending the rules governing rugby and improving the standard of refereeing. In an attempt to assess whether these measures have reduced the incidence of injury or altered the type of injury sustained, an analysis of injured rugby players admitted to the Spinal Cord Injury Centre at Conradie Hospital during the period 1981 to 1987 has been made. Results of this study indicate that there has been no decrease in the incidence of these serious injuries. The scrum and tackle remain the most important cause of injury. Flexion violence applied to the head and neck is still the major injuring force. Foul play, in particular the high tackle remains an important and avoidable cause of injury. A new finding is the identification of a group of rugby players who have sustained temporary paralysis without evidence of orthopaedic injury.

INTRODUCTION

In 2 previous papers[1,2] I have reported on patients admitted to the Spinal Cord Injury Centre at Conradie Hospital in Cape Town who have sustained paralysis due to rugby injuries to the cervical spinal cord. These reports dealt with a total of 50 players admitted during the period 1964 to 1980. In a series of papers, I identified the important phases of the game responsible for the majority of injuries, specifically the tight scrum,[1] the tackle,[3] rucks and mauls.[4]

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Since the first paper,[1] published in 1976 there has been much discussion as regards reducing the incidence of these serious injuries and various changes to the rules have been introduced.

In an attempt to ascertain whether there has been a decrease in the incidence of, or change in the mechanism of, these injuries following on changes in the rules, an analysis of rugby players admitted to the Spinal Cord Injury Centre with injuries of the cervical spinal cord during the 7 year period 1981 to 1987 has been made.

Method and materials

The case histories and radiographs of all patients admitted during the period 1981 to 1987 with spinal cord injury due to rugby, have been analyzed. Specific attention has been paid to the circumstances of injury, orthopaedic injuries as reflected on X-ray and the neurological deficit present on clinical examination. The cases were subdivided into groups according to the phase of the game during which the injuries were sustained. These cases were further subdivided into groups using the admission radiographs of the cervical spine as indicators of the mechanism of injury.

Results

A total of 38 players with cervical spinal cord injury either permanent or temporary, have been identified.

Age of patients

The age distribution is shown in Table 1. Nine (24%) of the players were 17 years of age or under at the time of injury.

Orthopaedic levels of injury

Table 2 shows the distribution of the levels of injury. Noteable is the high percentage (32%) of players who sustain injury at the C4/C5 level. Two players injured at the C2 level and 10 players

Table 1: Age distribution

Age	No. of players	Age	No. of players
14	1	15	1
16	3	17	4
18	–	19	3
20	–	21	1
22	4	23	5
24	3	25	2
26	1	27	2
28	1	29	1
30	1	31	3
32	–	33	–
34	–	35	–
36	–	37	–
38	–	39	1
40	–	41	–
42	1p		
Total: 38			

Table 2: Orthopaedic levels of injury

Level	Number	Level	Number
C2	2	C2/3	–
C3	–	C3/4	1
C4	1	C4/5	12
C5	2	C5/6	5
C6	3	C6/7	2
C7	–	C7/T1	–
No. Orthopaedic injury: 10			

Table 3: Phases of the game in which injury occurred

	No.	%
Scrums	15	39%
Tackles: (A) Tackler	3	42%
(B) Tackled	13	
Rucks/mauls	4	10%
Collision	1	2,6%
Unknown	2	5%

Table 4: Degree of paralysis due to injury in different phases of the game

	Incomplete	Complete
Scrum	9	6
Tackle	12	4
Ruck	3	1
Other	1	1
Collision	–	1

showed no evidence of orthopaedic injury on X-ray examination. This is in contrast to the large series reported by Silver[5] in which no high cervical spinal injuries were found nor any case reported without evidence of injury on X-ray.

Phases of the game in which injury occurred (Table 3)

The majority of injuries were sustained either in the scrum (39%) or during tackles (42%). This is in keeping with the findings in previous analysis.[1,2]

Degree of paralysis due to injury in different phases of the game (Table 4)

It can be seen in Table 4 that the majority of players (61%) sustained incomplete paralysis. 39% of players were completely paralysed. Notable is the high incidence of scrum injuries amongst this group.

Type of orthopaedic injuries sustained in different phases of the game (Table 5)

Reference to the table shows that there is a wide spectrum of orthopaedic injuries. Notable is the high number of flexion-dislocations sustained consequent upon collapse of the scrum.

Mechanism of injury as reflected on admission radiograph (Table 6)

Reference to the table shows that flexion was the most frequent important injury force. Twenty-five players (66%) sustained their injuries as a result of pure flexion violence or a combination of flexion and rotation.

DISCUSSION

Comments on players injured in scrums

Fifteen (39%) players were injured in scrums. This figure correlates well with the 40% incidence of scrum injuries noted in my previous analyses[1,2] and also with international experience. Burry and Gowland[6] reporting on a series of New Zealand rugby players with cervical spinal cord injuries, noted a 35% incidence of scrum injuries. Williams and Mackibben[7] in a similar analysis in Wales reported a 44% incidence.

Thirteen of the 15 players were injured as a result of collapse of the scrum. One player was injured by crashing of the two packs of forwards with the injured player being caught unaware. Details of the position of all the players injured are not available, but the majority of scrum injuries involved players in the front row, either props or hookers. (Figure 1) As shown in previous studies,[1,2] scrum injuries often result in severe neurological deficit. Six of the players sustained complete, permanent quadriplegia. Analysis of the type of orthopaedic injury sustained in the scrum, Table 5, reveals that the majority of injuries were anterior dislocations, either bilateral or unilateral. This is in keeping with the findings in the previous study.[1] The common mechanism of injury is flexion trauma as the majority force with a rotational component in those cases with unilateral facet dislocation. In cervical spinal cord injury due to causes other than rugby, the percentage of patients with bilateral locking of facets is much lower. In a series of 335 unselected cervical spinal cord injuries, only 15,5% (52) cases of bilateral locking of facets were recorded.[8] The mechanism of these injuries was for some time, controversial. Roaf,[9] using cadaveric spines, was unable to produce dislocation unaccompanied by fracture by hyperflexion alone and found that some degree of rotation must also be present. This was not in keeping with clinical observations, in particular as regards scrum injuries. Clear histories were obtainable from players in the front row who stated that as the scrum collapsed, their foreheads struck the ground and while the rest of the pack kept on pushing, their necks were progressively flexed and paralysis suddenly ensued. In 1978 Bauze and Ardran,[10] in an experimental study showed that pure dislocation unaccompanied by fracture

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Table 5: Type of orthopaedic injury sustained in different phases of the game

	B.L.F	U.L.F.	Subluxation	"Tear drop" Fracture	Compression Fracture	Hangman's Fracture	No Orthopaedic Injury
Scrum	7	3	2	–	–	–	5
Tackle	–	2	–	1	3	2	6
Ruck	1	2	1	–	–	–	–
Other	2	–	–	1	–	–	–

Table 6: Classification of cases in relation to mechanism of injury as reflected on radiological examination

Flexion	Subluxation	3
	Dislocation with bilateral locking of facets	10
	Wedge fracture	3
	"Tear-drop" fracture	2
Flexion/rotation	Unilateral locking of facets	7
Vertical compression	"Burst" fracture	–
Hyperextension	Avulsion fracture of the vertebral body	–
	Fractures of the spinous processes and laminae	–
	Posterior vertebral displacement	–
	Fracture dislocation	–
	Haemorrhage into the retrotracheal soft tissue	–
	Hyperextension injury in the elderly without radiographic evidence	1
	Hangman's fracture	2
No orthopaedic injury		10

could occur with flexion force. With the neck slightly flexed, and the vertex fixed on the ground, the normal cervical lordosis is lost and the spine straightened. In this position, the force is transmitted down the long axis of the spine. When the force exceeds the energy-absorbing capacities of the involved structures, far less force is required to dislocate the vertebrae and dislocation can occur without fracture.

Comment on tackle injuries

Sixteen (42%) of players were injured during a tackle. Three players were injured attempting to tackle an opponent, while the remaining 13 were tackled. Neurological deficit was less severe and only 4 players sustained complete paralysis. The spectrum of orthopaedic injury is shown in Table V and varies considerably from the orthopaedic injuries due to scrumming. Noteable are the large group of players who showed no orthopaedic injury on X-ray and also the two players with high (C2) fractures. The wider variation of orthopaedic injury and lesser degrees of neurological deficit in tackle injuries as compared to scrums is in keeping with the more varied mechanism. Nevertheless, reference to Table VI shows that flexion injuries predominate. In these cases the element of restraint of the vertex is often absent and the amount of damage to the spinal cord often less severe. One player was injured as a result of a double tackle. (Figure 2) Six players were injured as a result of high tackles around the neck. The mechanism of injury in double tackles[11] and high tackles[12] has been analyzed in previous studies.

The overall percentage of injuries sustained in tackles has increased, which is in keeping with the findings of Silver.[5] Of particular note and cause for concern, is the finding that 6 players were injured by a high tackle. In Silver's series,[5] only one is recorded as having been injured due to a high tackle. Silver also reported 4 players injured as a result of double tackles.

Comment on injuries sustained in rucks and mauls

Only 4 (10%) of the players were injured in these phases of the game. One player reported that he was injured while trying to break loose, 2 players jumped on him and while he was lying on the ground trampled and kicked him. The other players appear to have been injured by players falling on top of them while they were lying on the ground.

There is a surprising discrepancy between the figures found in the survey of Silver[5] and ours in this respect. He records a marked increase in the number of players injured in these phases of the game. A similar finding has been made in the 20 year survey of cervical spinal injuries in Wales.[7] The reason for this marked decrease in the percentage of players injured in rucks and mauls is not clear.

Figure 1: Anterior dislocation at the C4/C5 level with unilateral facet dislocation. This player was a 14 year old hooker who sustained complete quadriplegia and died some weeks after injury.

Figure 2: Typical double tackle with one player attempting to grasp the head of the tackled player.

Players with spinal cord injury without radiological evidence of orthopaedic injury

Five of these players were injured in tackles and the rest in collapsing scrums. Although spinal cord injury without evidence of orthopaedic injury X-ray is found commonly in patients admitted to spinal cord injury units, these are generally elderly patients with spondylosis. The observation of this large group of young rugby players suffering this type of injury is new and has not been recorded in other surveys. A recent paper has however reported on similar findings in rugby players and is the subject of another paper in preparation.

CONCLUSION

During the period 1964 to 1980, 50 rugby players with cervical spinal cord injury were admitted to the Spinal Cord

Figure 3: Frustrated opponents applying force to the neck while trying to gain possession of the ball.

Injury Centre at Conradie Hospital,[2] an average of 3 players a year. During the period under review in the present paper (1981 to 1987) 38 players were admitted, an average of 5,4 players a year. This indicates that despite increased public awareness of serious rugby spinal injuries and considerable amendments to the rules, the incidence of rugby spinal cord injuries in the Cape Province has not decreased. Silver and Gill[14] comment on the gratifying decrease of serious cervical spinal cord injuries in rugby players in the United Kingdom, Wales and New Zealand, but note that the only country where there has not been a decrease in the incidence is South Africa.

As commented on in previous papers, [1,2] and also in reports from overseas,[7] flexion remains the most important mechanism of injury (Figure 3). The two phases of the game where most serious injuries occur are again identified to be the scrum and the tackle. In the scrum, flexion usually combined with restraint of the vertex or combined with rotation produces most injuries. The mechanism of injuries in tackles is more varied, but flexion violence again predominates.

Foul play was responsible for a significant number of injuries. Apart from the single instance, where a player was injured by crashing of the scrum, a disturbing number of players were injured by the high tackle. Despite previous comment on the danger of this illegal play,[12] no decrease in these avoidable injuries appears to have taken place.

REFERENCES

1. Scher AT. Rugby injuries to the cervical spinal cord. *S Afr Med J* 1977; **51**: 473-475.
2. Scher AT. Rugby injuries of the spine and spinal cord. *Clin Sports Med* 1987; **6(1)**: 87-99, 12.
3. Scher AT. The high rugby tackle – an avoidable cause of cervical spinal injury? *S Afr med J* 1978; **53**; 1015-1018.
4. Scher AT. Rugby injuries to the cervical spine sustained during rucks and mauls. *S Afr Med J* 1983; **64**: 592-595.
5. Silver JR. Injuries of the spine sustained in rugby. *Br Med J* 1984; **288**: 37-43.
6. Burry HC, Gowland H. Cervical injury in football – a New Zealand survey. *Br J Sports Med* 1981; **15**: 56-60.
7. Williams P and McKibbin B. Unstable cervical spine injuries in rugby – a 20-year review. *Injury* 1987; **18(5)**: 319-332.
8. Braakman R, Penning L. Injuries of the cervical spine. Excerpta Medica. Amsterdam 1971.
9. Roaf R. A study of the mechanics of spinal injuries. *J Bone Jt Surg* 1960; **42B (4)**: 810-823.
10. Bauze RJ, Ardan GM. Experimental production of forward dislocation in the human cervical spine. *J Bone Jt Surg* 1978; **60B (2)**: 239-245.
11. Scher AT. The "double tackle" – another cause of serious cervical spinal injury in rugby players. *S Afr Med J* 1983; **64**: 595-596.
12. Scher AT. The high rugby tackle – a continuing menace. *S Afr Sports Med 1981;* **11**: 3.
13. Torg JS, Pavlov H, Genuario SE *et al.* Neurapraxia of the cervical spinal cord with transient quadriplegia. *J Bone Jt Surg* 1986; **68A (9)**: 1354-1370.
14. Silver JR, Gill S. Injuries of the spine sustained during rugby. *Sports Med* 1988; **5**: 328-334.

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A SURVEY OF ALL SPORT INJURIES
EXAMINED AT THE BUREAU FOR STUDENT HEALTH OF THE UNIVERSITY OF THE ORANGE FREE STATE DURING THE PERIOD JANUARY 1985 – DECEMBER 1985

EAM Prinsloo

AIM

The incidence of sport injuries was examined. The type of injury sustained in various sport events was examined. Special attention was given to rugby and athletic injuries. The study was done to determine the need for sport medicine education in South Africa.

METHODS

The clinical records of the doctors and orthopedic surgeons were used. All persons included in the study were sportsmen of the University of the Orange Free State who sustained injuries during the period January 1985 – December 1985.

Attention was given to the type of sport, type of injury, level of competition, recurrent injuries, referral to orthopedic surgeons, sex of sportsmen and special investigations done.

RESULTS

(Table 1)
There was a total of 438 injuries; 5% of consultations in 1985.

There were 145 referrals to orthopedic surgeons.

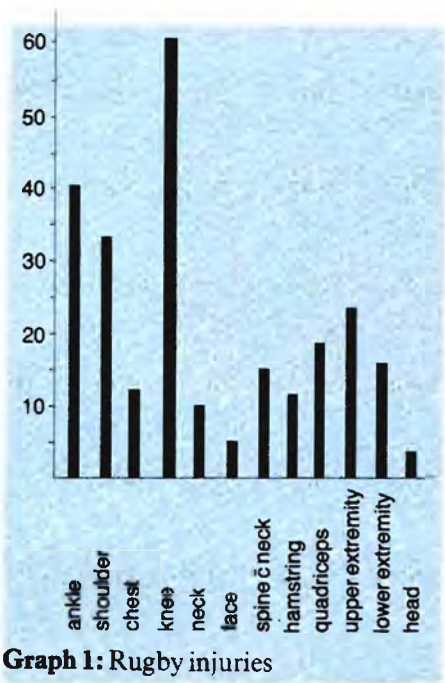
Table 1: Injuries in various sporting events

Sport	Number	%
Rugby	246	56,2
Athletics	60	13,7
Hockey	23	5,3
Marathon	19	4,3
Netball	14	3,2
Soccer	12	2,7
Squash	9	2,0
Gymnastics	8	1,8
Judo/Boxing/Karate	8	1,8
Cricket	7	1,6
Jogging	7	1,6
Swimming/Diving	5	1,1
Volleyball	4	0,9
Tennis	4	0,9
Water-skiing	3	0,7
Weightlifting	3	0,7
Cycling	1	0,2
Biathlon	1	0,2
Parachuting	1	0,2
Rowing	1	0,2
Tug at the rope	1	0,2
Fencing	1	0,2
Total	**438**	**100%**

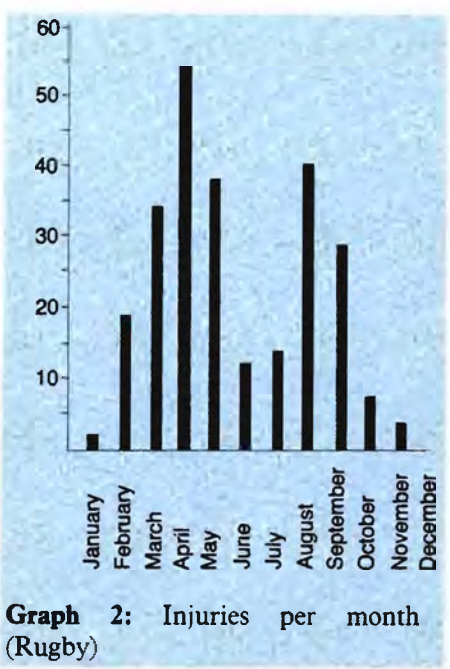
RUGBY INJURIES

(Graph 1)
193 Injuries were sustained during hostel league: 25 at Club level, 25 at Provincial level, 1 Springbok player.

The high incidence at hostel level may be due to different factors. More players participate at this level. The players are usually not very fit. Players play in unusual positions. The motive to win is very high in hostel league.



Graph 1: Rugby injuries



Graph 2: Injuries per month (Rugby)

Dr EAM Prinsloo

Causes of injuries

Graph 2 indicates the number of injuries per month. 24% (58/246) were recurrent injuries. Rugby players tend to play with injuries.

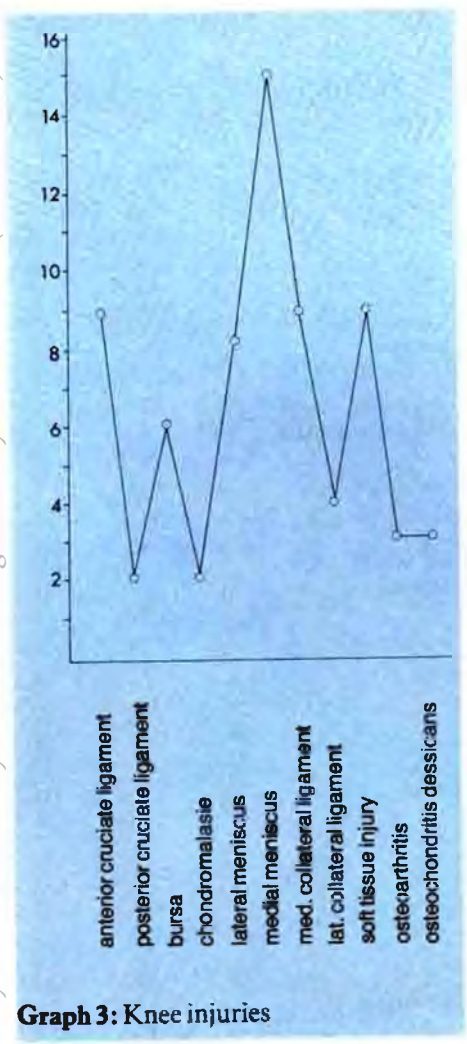
The doctor should put the health of his patient first and stand up to coaches who want to force injured players to play.

The player, team doctor and coach should work together as a team. They should have confidence in the doctor and he has to show enough interest and knowledge of the game.

Knee injuries

(Graph 3)
24% (59/246) were knee injuries.

Examination
See Figures 1-7.



Graph 3: Knee injuries

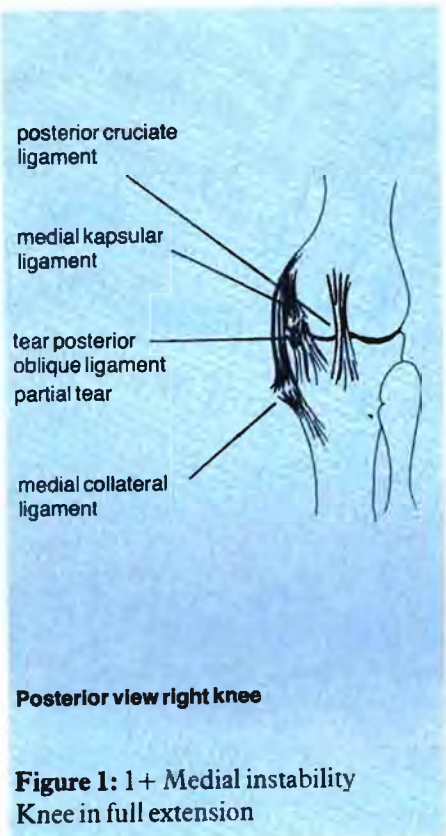


Figure 1: 1+ Medial instability
Knee in full extension

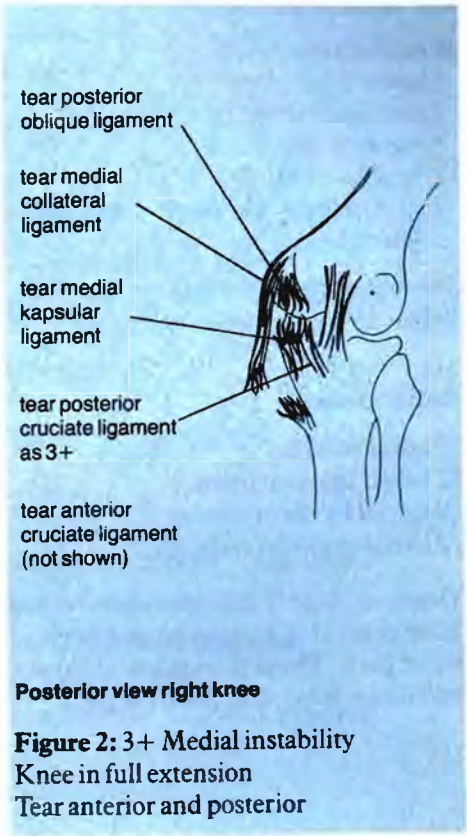


Figure 2: 3+ Medial instability
Knee in full extension
Tear anterior and posterior

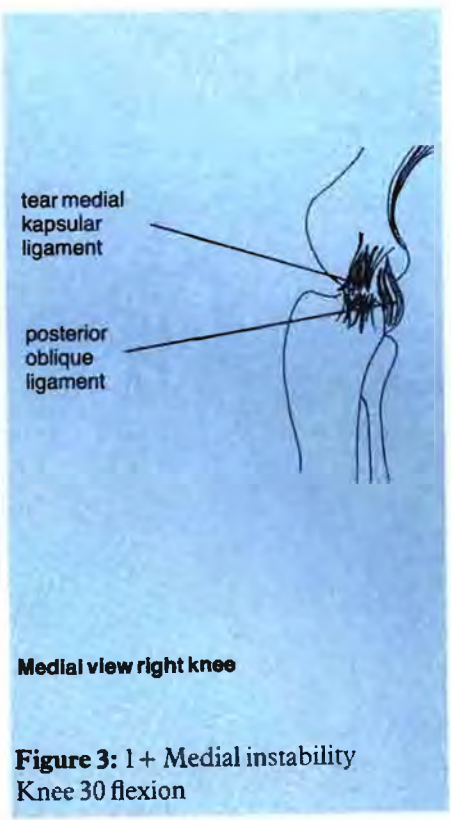


Figure 3: 1+ Medial instability
Knee 30 flexion

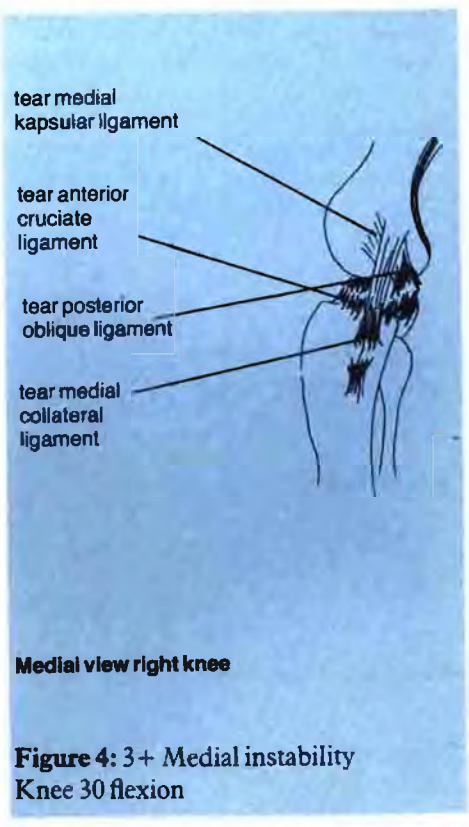


Figure 4: 3+ Medial instability
Knee 30 flexion

Referrals
42 knee injuries were referred to orthopedic surgeons. 27 patients were operated upon.

10 medial menisectomy
5 lateral menisectomy
7 anterior cruciate reconstruction
1 lateral collateral tear
1 iliotibial band
2 arthroscopy for loose fragment – knee

Post-operative physiotherapy plays an important part in treatment.

Ankle injuries

39 ankle injuries
32 lateral ligament injuries
9 medial ligament injuries
2 lateral malleolar fractures

Treatment
Most grade II + injuries are put in plaster of paris. Physiotherapeutic rehabilitation after removal of plaster of paris.

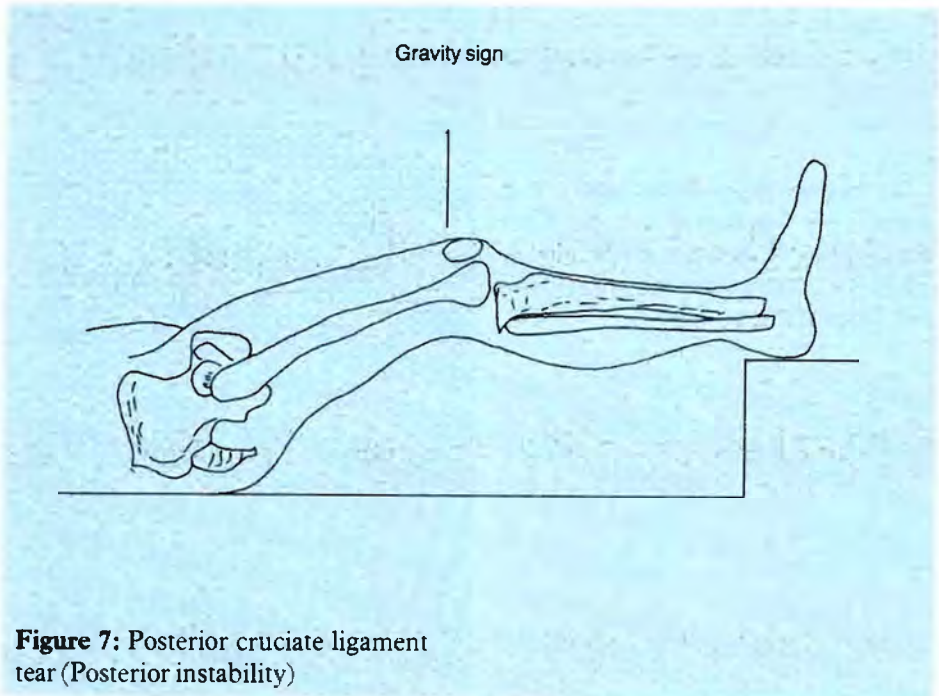


Figure 7: Posterior cruciate ligament tear (Posterior instability)

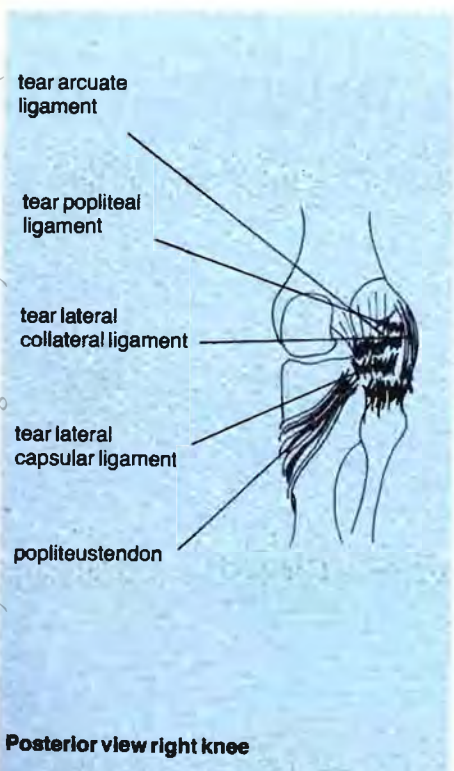


Figure 5: Lateral instability with knee in full extension (2+)

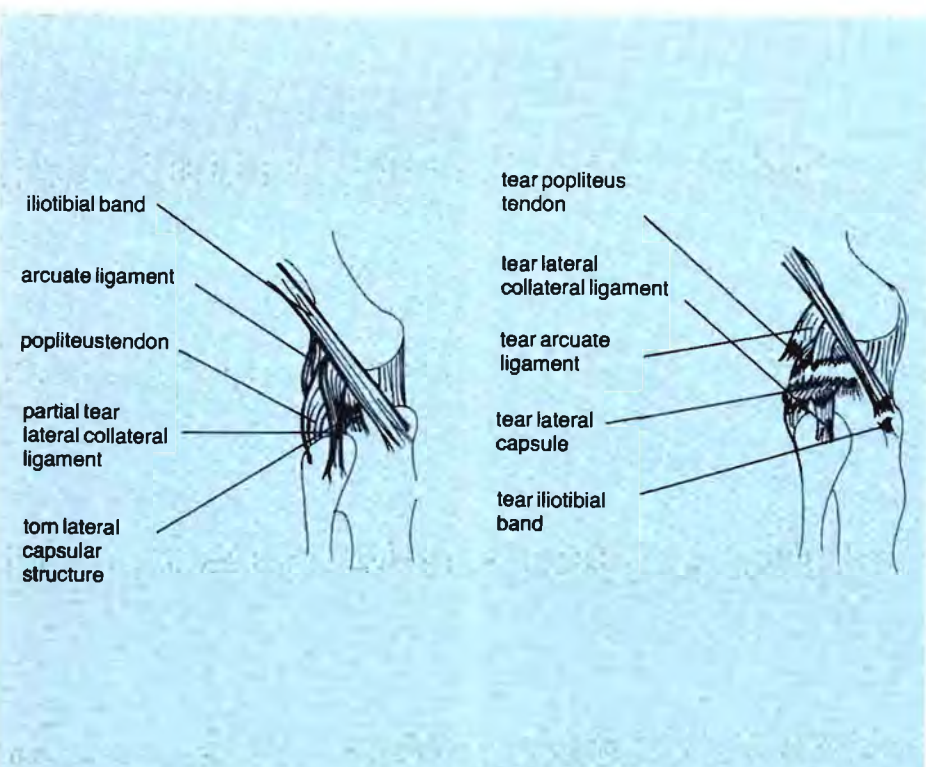


Figure 6
Lateral view right knee
(1+ instability knee 30 flexion)

Lateral view right knee
(3 + instability)

Table 2: Marathon injuries

Injury	Number	%
Stress fracture	6	31,6
Iliotibial band	5	26,3
Achillestendonitis	4	21,0
"Shins"	2	10,5
Sports anemia	1	5,3
Chondromalasia	1	5,3
Total	**19**	**100%**

Table 3: Type of athletic injury

Injury	No.	%
Hamstring	14	23,3
Knee	11	18,3
chondromalasie	(5)	
bursa	(3)	
osteochondritis dissecans	(1)	
anterior cruciate ligament	(1)	
tibiofibular ligament	(1)	
"Shins"	6	10,0
Iliotibial band	4	6,7
Adductor Hip	4	6,7
Ankle	4	6,7
ligament	(3)	
osteofyte	(1)	
Back	3	5,0
Foot	3	5,0
Stress fracture	3	5,0
Quadriceps	2	3,3
Calf	2	3,3
Hand/elbow	2	3,3
Achillestendonitis	1	1,7
Compartment syndrome	1	1,7
Total	**60**	**100%**

Table 4: Injuries in various items

Item	Number	%
Middle/Longdistance	22	36,7
Longjump	12	20,0
Sprints	12	20,0
Javelin	6	10,0
High jump	4	6,7
Steeplechase	2	3,3
Hurdles	1	1,7
Shotput	1	1,7

Table 5: Injuries in track events

Item	Injury	Number
Sprints	Hamstring	6
	Quadriceps	2
	Knee/bursa	2
	Back	1
	"Shins"	1
Middle/Long distance	Hamstring	4
	Iliotibial band	4
	Knee	4
	"Shins"	3
	Stress fracture	2
	Calf	1
	Back	1
	Compartment sindrome	1
	Ankle ligament	1
	Gout	1
Steeplechase	Chondromalasia	1
	Adductor hip	1

Shoulder injury

Total of 33 injuries:

15 acromioclavicular ligament tears
5 sternoclavicular injuries
6 rotator cuff injuries
5 anterior dislocations
2 capsular injuries

Neck injuries

Only 9 injuries:
8 soft tissue
1 cervical disk

ATHLETIC INJURIES

Analysis

60 athletic injuries
19 marathon injuries (See Table 2)

Anatomical analysis See Table 3.

Type of injury in various items

See Table 4, 5 and 6.

Stress fractures Tibiotalor pinching.

CONCLUSION

There is much room for research into sport injuries and there is a need for post-graduate education in sport medicine in South Africa.

There should be better co-operation between sportman, coach and team physician.

PHYSICAL EXAMINATION OF THE ANKLE AND FOOT

RF Spencer

INTRODUCTION

Adequate physical examination of any part of the musculoskeletal system should be preceded, whenever possible, by comprehensive history and general examination. One must bear in mind that pain is often caused by compensatory stresses in response to malalignement.[1] It is therefore essential, when assessing an ankle or foot problem, to exclude the possibility of pathology in the spine, hip or knee.

Routine physical examination should follow traditional guidelines. These can be summarised under the following headings:

- Look
- Feel
- Move
- Test stability

The process of looking should commence as early as possible and should embrace all aspects of the athlete's general demeanour and gait. It should then focus on the limb in question and assess discrepancies of contour, difference in girth when comparing left and right, and the posture when standing observed from both front and back. At this stage it may be appropriate to ask the patient to stand on tiptoe to assess possible problems in the tendo-Achilles mechanism.

One should then proceed to look at the particular area in question, paying attention to previous scars, sinuses and areas of erythema or bruising. Palpation can then be carried out for differences in skin temperature, areas of tenderness, and prominent bony landmarks. During the palpation process the foot may be gently moved both passively and actively to assess crepitus within the tendon sheaths and joints. Particular attention should then be paid to localized areas of tenderness. Movements should be assessed both actively and passively. Active movements should be assessed first in order to delineate the range of relatively painfree movement. Passive movements may then be carried out to assess the range more fully. Once again, crepitus both in the joints and tendon sheaths should be noted. Finally, stability may be tested. It is important when assessing the medial and lateral ligamentous structures of the ankle to hold the heel and not the forefoot when doing this. Varus and valgus stresses can be applied to assess any possible instability. At this stage special investigation appropriate to the diagnosis may be required.

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TENDO ACHILLES PATHOLOGY

The patient with an Achilles tendon problem may have difficulty standing on tiptoe and contracting the calf muscles. More than one pathology may be present in this region[2]. The mesotenon may be diffusely inflamed and tender and give rise to clearly visible swelling in the region when viewed from behind and compared with the other side. This condition is referred to as Achilles peritendonitis.

At the same time, or in isolation, either of the bursae in the region may be inflamed. There is a bursa deep to the Achilles tendon and one superficial to it. The latter may become inflamed as a result of wearing tight running shoes. Focal areas of mucoid degeneration or partial rupture of the tendo-Achilles may be assessed by palpation. Where a complete rupture has occurred the patient will commonly complain of a sudden pain in the region not unlike the feeling of having been kicked in the back of the heel. The most useful test for assessing this situation is the Thompson's (or Simmonds') test. This is best performed with the patient prone with the feet overhanging the end of the examination couch. It may immediately become obvious that there is a gap in the tendinous mechanism. The foot on the affected side does not naturally assume a slight equinus position. The diagnosis is confirmed by squeezing the calf muscles on both sides. On the affected side the foot will not move into a position of plantar flexion, whereas it will do so on the unaffected side. It is pertinent to note that the patient may be able to actively plantar flex the foot in the presence of a complete rupture of the tendon.

Other tendons in the region of the ankle joint may become affected by inflammatory processes and tenosynovitis. Similarly, the peroneal tendons may be subject to subluxation and dislocation.

SEVER'S DISEASE

This condition frequently affects young and adolescent athletic individuals, and gives rise to pain and local tenderness in the region of the insertion of the tendo-Achilles into the calcaneus. The condition may be a traction apophysitis of the tendon insertion. The radiological features include flattening and sclerosis of the posterior calcaneal apophysis.

NERVE ENTRAPMENT SYNDROMES

Two nerve entrapment syndromes may become troublesome to the athlete. The superficial peroneal nerve emerges from the deep fascia on the anterolateral aspect of the leg. It may become constricted when it passes through the fascia giving rise to pain on the dorsolateral aspect of the foot and over it's site of emergence. Similarly, the posterior tibial nerve may be constricted within the flexor retinaculum on the posteromedial aspect of the ankle. This gives rise to pain and paraesthesiae on the plantar and medial aspects of the foot. Palpation may reveal tenderness and sometimes a positive Tinel sign over the area of constriction.

The diagnosis may be confirmed by inflating a blood pressure cuff over the calf to a pressure between the systolic and diastolic levels for a minute or so, thus providing the onset of typical symptoms.

FRACTURES AND SPRAINS OF THE ANKLE

The typical fractures and fracture dislocations around the ankle joint are extensively covered in standard text books. The clinical findings are usually quite characteristic with extensive ecchymoses, crepitus and severe bony tenderness in the region. Attention must be paid to the neurovascular status of the foot.

Sprains may involve either the medial or lateral structures at the ankle joint. The internal or medial type of strain is less common and involves the superficial part of the deltoid ligament. This is most easily assessed by examining the patient from behind and comparing the two sides, a clear swelling being visible over and inferior to the medial malleolus on the affected side. On the lateral side, the ligament is divided into three parts; the anterior and posterior talofibular and the calcaneofibular ligaments. Frequently, only the anterior talo-fibular ligament is damaged giving rise to the common "sprained ankle". In this condition swelling over and anterolateral to the lateral mallolus is visible and pain is elicited by inverting the foot.

However in more severe injuries all three bands may be ruptured giving rise to possible instability. An injury of this kind is thought by many to be an indication for surgical treatment. The diagnosis is best made by taking stress views in both the AP and lateral projections.

The AP stress view is obtained by applying a varus strain to the heel while taking the film. Instability is demonstrated by an opening of the lateral aspect of the ankle mortice. (Figure 1).

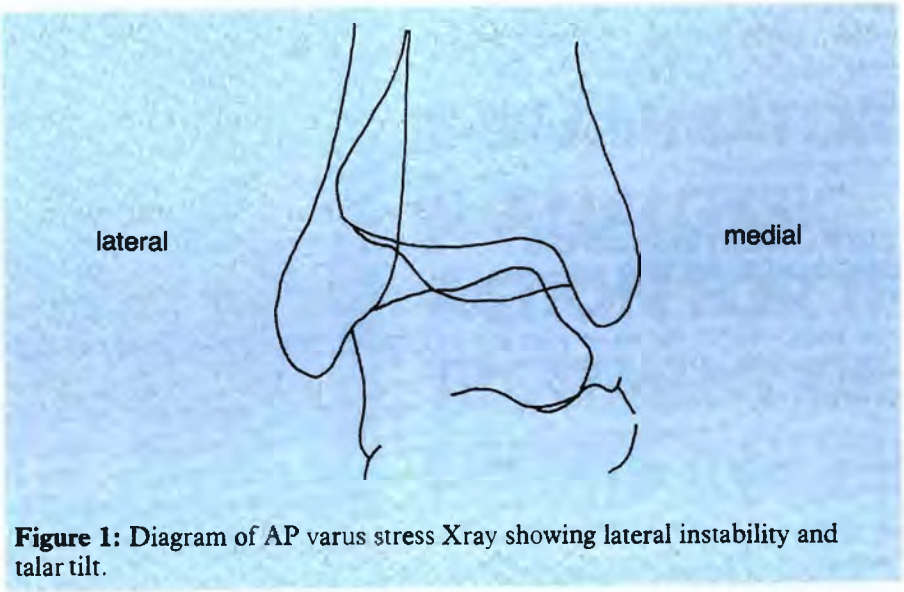


Figure 1: Diagram of AP varus stress Xray showing lateral instability and talar tilt.

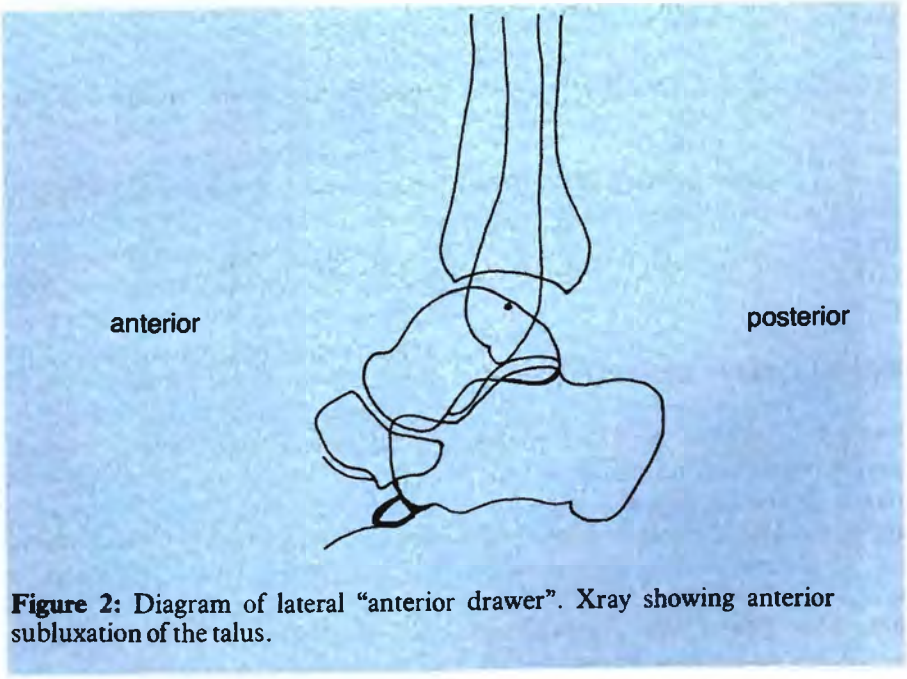


Figure 2: Diagram of lateral "anterior drawer". Xray showing anterior subluxation of the talus.

The lateral view, or so called anterior drawer test, is best obtained by placing the patient's heel on a sandbag and pushing downwards on the tibia. If instability exists, the tallus can be seen to subluxate forward in the ankle mortise. (Figure 2). Some Surgeons believe that the AP views should be augmented using an arthrogram, which will show dye leaking extensively into the lateral soft tissues.

It should be remembered that following rupture of the lateral ligamentous structures, whether these have been managed by operative or non-operative means, the normal proprioceptive structures within the ligament have been damaged. Therefore, although no residual instability may be demonstrable clinically, the patient may complain of a feeling of instability on the lateral side. This condition is referred to as "stable instability".

OTHER CONDITIONS GIVING RISE TO INSTABILITY

Loose bodies within the ankle joint are occasionally a cause of instability. These may arise from anterior traction spurs in the so called "Footballer's Ankle", or from osteochondritic fragments which have become detached. The most common cause of the latter is the condition of osteochondritis dissecans, which frequently affects the superior weight bearing surface of the talus. Another lesser known condition is the so called meniscoid lesion. This consists of a synovial and fibrous frond of tissue which grows into the ankle mortice as a result of repeated trauma to the area.

It may impinge between the weight bearing surfaces and give rise to symptoms.

DEGENERATIVE DISORDERS

"Footballer's Ankle" may affect the younger patient, giving rise to traction spurs from the capsule and decreased joint space with sclerosis, stiffness and pain. In older athletes, post traumatic osteoarthritis becomes a factor to consider. The patient will complain of mechanical pain, clinical examination may reveal diffuse synovial swelling, tender osteophytes, and the range of motion may be decreased. The radiological findings are characteristic.

FOOT PRONATION

This may give rise to considerable pain and disability in the runner. Several conditions may predispose to the development of the pronated foot.[3]

- Joint laxity
- Heet valgus
- Genuvarus
- Tight tendo-Achilles
- Subtalar varus
- Forefoot supination

It is worth re-iterating the importance of assessing the whole limb before concentrating on the foot problem. Examine the running shoes for uneven wear and observe the patient from behind, as the position of the heels can only be accurately assessed from this view.

CONGENITAL ABNORMALITIES

A number of congenital anomalies within the foot may be important to the athlete. Perhaps the most common of these is the condition of metatarsus primus varus which has a strong hereditary influence and may rise to hallux valgus.

Assessory ossicles may also occur. The "os trigonum" is a frequently found radiological anomaly. Most common in causing clinical symptoms is the so called "os naviculare" on the dorsomedial aspect of the foot. This is simply an accessory navicular bone.

Tarsal coalition is an unusual but important cause of pain in the adolescent. This gives rise to "peroneal spastic flat foot". In this condition the patient complains of pain on the posterolateral aspect of the ankle with exercise. This pain is related to the peroneal tendons which on clinical examination appear to be tender and the muscles in spasm. The medial arch of the foot is frequently dropped, hence the "flat foot" description. It is important to take adequate Xrays, including Harris oblique views of both heels in order to establish the possible presence of a tarsal coalition. CT Scanning may also be helpful.

ISELIN'S DISEASE

This condition, which affects the adolescent, is another example of a chronic traction injury. In this instance, this involves the insertion of the peroneus brevus tendon into the base of the 5th metatarsal. Local tenderness may be elected in this area and radiological features usually consist of a presence of a small flake of bone displaced from the apophysis, near the insertion of the tendon.

STRESS FRACTURES

Nearly 50% of stress fractures which occur in athletes involve the foot.[4]

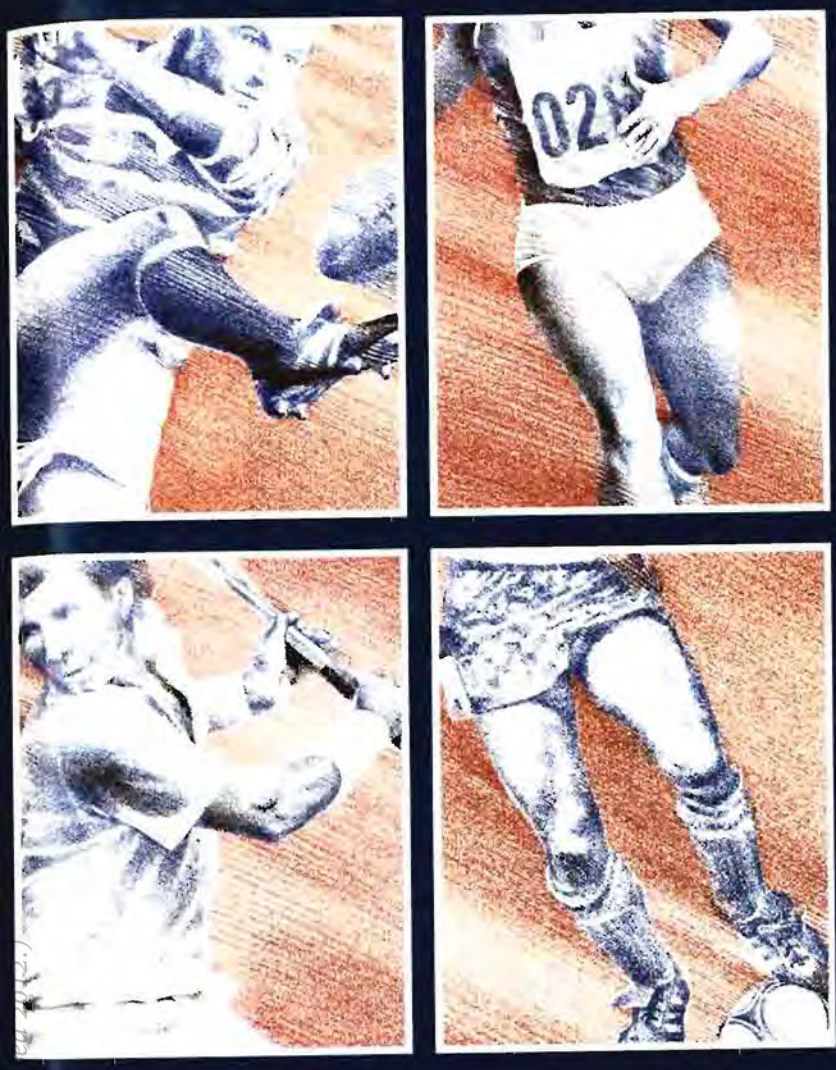
The important clinical features consist of gradually increasing and chronic pain which eventually make it impossible to continue with athletic activity.

Local tenderness is invariably present, and the radiological features include a periosteal reaction, and sometimes a fracture line. It is important to differentiate these conditions from malignancies, and therefore a precise history is essential. In the case of the "Jones fracture", which occurs about 2cms from the base of the 5th metatarsal the injury consists of a traction type of stress fracture, rather than one due to repeated compressive loading. This injury should be differentiated from the avulsion fracture of the base of the 5th metatarsal, as the treatment is quite different and the prognosis without bone grafting much worse in the case of the Jones fracture.

FRACTURES AND DISLOCATIONS OF THE FOOT

These may follow many patterns, the clinical history and examination usually being revealing. The important feature is the development of considerable swelling within the first 24-48 hours.

Common areas of involvement include the subaltar joint, the talonavicular and calcaneocuboid joints (the so-called Chopart dislocation) and the tarsometatarsal joint (Lisfranc dislocation). Deformity may be obvious on clinical examination. The metatarsal bones may be fractured as a result of



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crushing injuries.

In all cases, early diagnosis is important in order to allow treatment (including early reduction of any dislocation) to begin.

OSTEOCHONDROSES

These have been described involving many of the tarsal and metatarsal bones. The most commonly observed examples include Kohler's Disease of the navicular and Frieberg's infraction of the head of the 2nd metatarsal. Repeated trauma has been thought to be an important aetiological factor, but this is not certain. Localised tenderness will be present and may sometimes suggest a stress fracture. However, the radiological features are characteristic. The areas affected become flattened and sclerotic.

NEUROMAS

Morton's Neuroma, usually in the third interdigital cleft on the plantar aspect of the foot, is the most common condition of this kind to affect the athlete. However, a less frequent condition known as Joplin's Neuroma may involve the cutaneous nerve on the dorsal aspect of the hallux. This is frequently due to repeated trauma from tight running shoes. Local exquisite tenderness is characteristic of these conditions. In the case of the Morton's neuroma transverse compression of the metatarsal heads may elicit pain on clinical examination. In addition there may be parasthesiae distal in the area of supply, and a local positive Tinel sign.

HALLUX VALGUS

Hallux valgus is a problem which more often affects the female runner. The aetiological factors include the wearing of tight fashionable shoes and the inherited condition of metatarsus primus varus. Clinical examination frequently reveals a tender bunion overlying the head of the 1st metatarsal with the well known lateral angulation of the hallux. The sesamoids may become involved and be locally tender on the plantar aspect.

HALLUX RIGIDUS

This commonly affects long distance runners and is a condition of degenerative arthritis of the 1st metatarsophalangeal joint. The history is important in that the patient will complain bitterly of pain and stiffness in the joint. Local tenderness may be present. The range of movement is grossly decreased and is painful. The Xray findings are those of a degenerative arthritis of the joint.

OTHER CAUSES OF FOREFOOT PAIN

The sesamoid bones may become inflammed or fractured in the absence of other surrounding pathology. They are, on occasion, subject to stress fracture. However, the simple Verruca vulgaris may give rise to pain and local tenderness in the plantar skin and should be considered.

It is worth remembering that ingrowing toenails and athletes foot are not uncommon in runners and may be difficult to treat.

SUMMARY

The above account of the physical examination of the foot and ankle is by no means comprehensive with respect to all the conditions which may be found. However the problems which commonly affect the athlete have been detailed. It is most important to reiterate that full assessment of the patient is necessary in all cases, and that malalignment problems may give rise to ankle and foot pain.

REFERENCES

1. Maron BR. "Orthopaedic aspects of Sports Medicine" in *Sports Medicine*, Appenzeller O, and Atkinson R. Ed. Urban and Schwarzenberg, Ballimore-Munich, 1983.
2. Schepsis AA and Leach RE. "Surgical management of Achilles tendinitis". *The American Journal of Sports Medicine* Vol **15**(4), 308-315, 1987.
3. Noble CA. "Excessive distances are major causes of injury". *SA Sports Medicine*, No **3**, 4-5, 1978.
4. Matheson GO, Clement DB, McKenzie DC. "Stress fractures in athletes. A study of 320 cases". *The American Journal of Sports Medicine*. Vol **15**(1). 46-58, 1987.