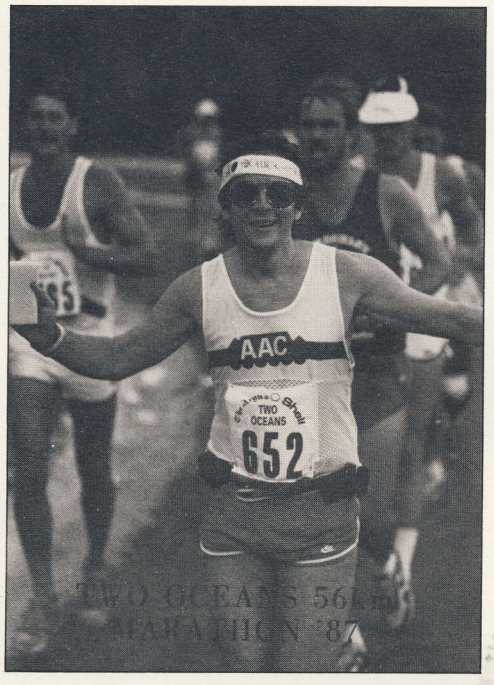
"Sick" runners - how much is in the mind?

— S Furman



Saville Furman MB ChB, MFGP (SA)
302 Centrepoint, Loxton Road
Milnerton 7441 CP

Summary

Runners represent a healthy segment of our population. They are well-informed about their bodies, yet are susceptible to misrepresentations. There are a variety of illnesses induced by sports activities and also pseudo-syndromes and abnormal test results in otherwise healthy athletes. This often represents a dilemma for the doctor treating them. Four cases are presented, two from the author's practice and two with whom he runs, illustrating these dilemmas.

S Afr Fam Pract 1987; **8:** 391-6

KEYWORDS: Sports medicine; Physicians, family; Jogging

Curriculum vitae

Saville Furman graduated from UCT in 1973 and entered general practice in December 1974. He obtained the MFGP (SA) in 1977. He has a wide field of interest in medicine, the main being 'Doctor-Patient Relationship' and 'Sports Medicine'. He is Chairman of the Western Cape Region of the SA Academy of Family Practice/Primary Care, a member of the Academy Council, part-time lecturer in the Dept of Community Med (UCT) and is very active on the Editorial Board of *SA Family Practice,* where he is, inter alia, responsible for the section 'Through My Lens'. Dr Furman received the Louis Leipoldt Award for the best GP paper published in the *S Afr Med J* in 1980, has run many marathons and a few ultra-marathons since 1982. Saville is also a keen photographer, an avid reader and very busy at the moment as Academic Chairman of the 6th GP Congress, with the organisation of the Congress in Cape Town in March '88.

He is married to Shelly and they have 2 children: Donna and Graham.

This article was presented at the 2nd SA Sports Medicine Association Congress, Cape Town, 15 April 1987. The Boots Gold Award for the best local presentation was won by Dr Furman for this paper. Congratulations!

Runners represent a unique group of patients.[1] They comprise a very healthy segment of our population. Many doctors, even those interested in sports, hate to see a runner or jogger enter their consulting room.[2] Why? Because their injuries appear difficult to diagnose and even more difficult to treat. Often they find it difficult to communicate with the runner. Runners are addicted. It is part of their life. The physician must remember this. If he appears indecisive, he will lose the runner's confidence, and even the correct treatment programme will go unheeded.

Physicians who want to treat recreational athletes should be prepared for patients who are well-informed about fitness, sports medicine and who know their bodies.[3] They present excellent histories of their injuries. They are one step ahead of the (physicians's) diagnosis. At the same time physicians will also see athletes who have questions about pseudo-sophisticated anecdotal information about sports medicine, body wraps, vitamin, electrical muscle stimulators and electrolyte replacement drinks. From one point of view, this is encouraging because it indicates that people are

really interested in improving their health. But athletes are susceptible to misrepresentations, and physicians can only help if they can differentiate accurate sports medicine information from the "hype".

Physicians who previously had no interest in sports medicine are now facing a variety of illnesses induced by sports activity, such as exercise-induced asthma, urticaria and anaphylaxis.[3] Other disorders such as exercise-related hematuria and secondary amenorrhoea requires specific workups

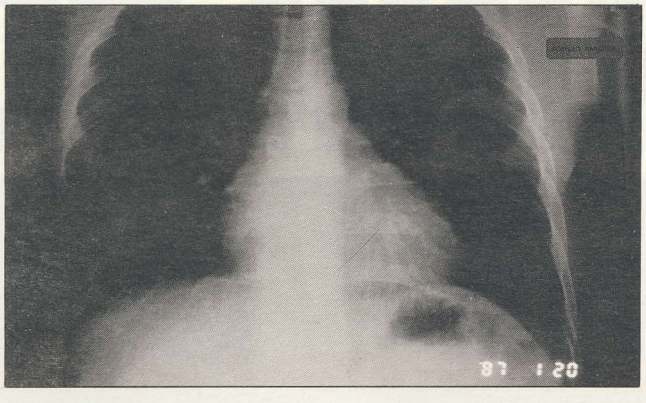
Many GPs hate to see a jogger enter their consulting rooms

to exclude conditions or causes independent of exercise. The sports physician should be able to identify the pseudo-syndromes or abnormal test results in otherwise healthy athletes. These include pseudo-anaemia, pseudo-nephritis (transient proteinuria and haematuria, curing within 24 hours), pseudo-hepatitis (increased serum LDH, SGOT from muscular injury) and even pseudo-myocardial infarction (increased serum MB Isoenzyme of creatine kinase after prolonged strenuous exercise such as that of marathon runners). The physician must exclude the possibility of specific organ system dysfunction or pathology such as bona fide iron deficiency, hepatic or renal dysfunction, and even myocardial injury and explain his abnormalities to the athlete.

Four patient studies are presented illustrating the above anomalies.

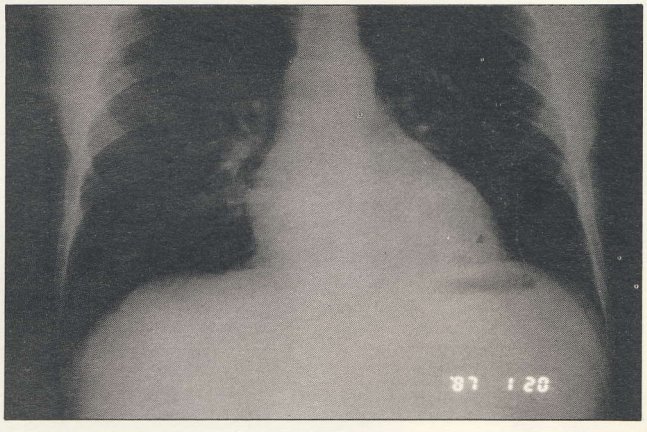
Patient 1

Mr FS, aged 35, a sub-three-hour marathon runner presented to me in December 1986 with a high temperature and body aches. He had no other symptoms or signs at initial presentation. A viral infection was diagnosed and he was advised to stop running, go to bed for a few days, and take paracetamol.

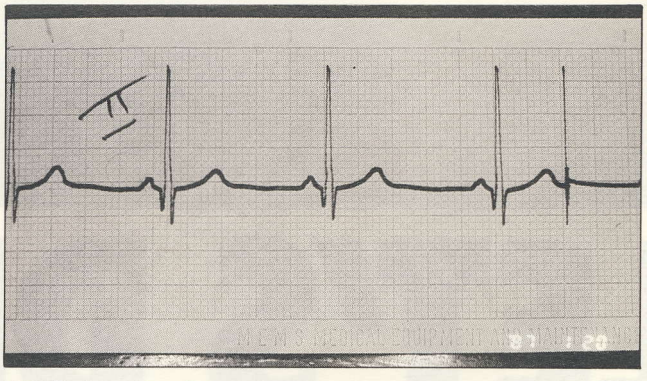


Chest X-ray revealing right lower lobe pneumonia.

Over the weekend, when I was off-duty, he developed a dry, unproductive cough; his temperature went up to 40 °C and he was given a course of amoxycillin by another doctor. Two days later he presented with a cough, productive of green mucous, swinging fevers and body aches. A chest X-ray revealed a right lower lobe pneumonia. The antibiotic was changed to erythromycin. The patient made an uneventful recovery and was feeling very well. I cautioned him not to exercise too vigorously, but to wait at least two weeks in view of the high temperature and X-ray changes before starting to run hard again. As he was going on holiday, I asked him to have an X-ray as soon as he returned. He felt very much better while on holiday, so much so, that he started running again. In January, he decided to run the Atlantic 30 kilometre race in Cape Town. During the race he developed chest pain, became short of breath, but nevertheless still did a very good time. He came to visit me the following day with his new X-rays,

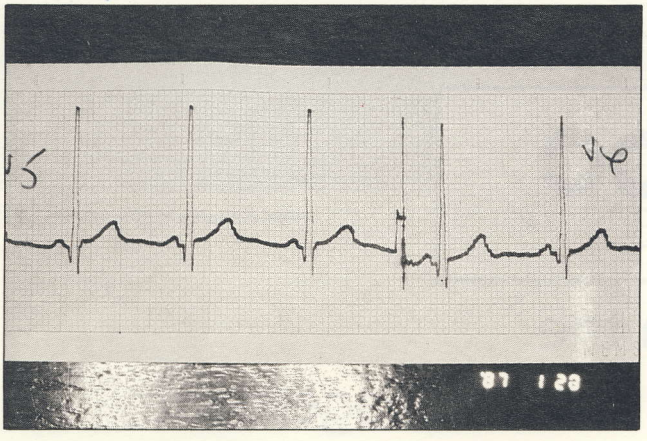


Resolution of pneumonia with increased heart size.



ECG showing Q waves in Std lead II.

which showed resolution of his pneumonia, but also an apparent increase in his heart size. A murmur was noted over the aortic area. An ECG showed some inferior and lateral Q waves. These features induced much anxiety in the doctor. A full blood count and enzymes were requested. The FBC and ESR were normal. The AST was slightly raised. The LD was normal. The CK was twice normal, but the CKMB was within the normal limits.



ECG of patient 1 showing Q waves in lead V5.

(Of note is that over the last three-and-half years, this patient had 4 episodes of documented respiratory infection. On two occasions the bronchitis-like illness had progressed to pneumonia. On each occasion it had begun with a flu-like illness with fever, chest pain, tightness of the chest and dry unproductive cough. Usually he had produced yellow-brownish sputum and once had sputum which was slightly blood-stained. On each occasion he had been treated with antibiotics. In 1985, while he was laid-off running, following a cartilage operation on his knee, he became over-weight to the extent of increasing his weight by 12 kilograms and was noted to be hypertensive with a blood pressure as high as 170/110. He was treated with Atenolol and Prazosin and gradually, after weight reduction and resumption of his normal training programme, his weight decreased and his blood pressure returned to normal. He had not been on any medication for the last year).

GPs should obtain special skills in the area of sports injuries

He was referred to a cardiologist to exclude myocardial pathology and I quote from the report: "He is in regular sinus rhythm with a normal amplitude pulse. Blood pressure 140/90, heart size is normal and on auscultation, apart from a soft short ejection murmur across the aortic valve, there are no abnormalities. The chest is clear. I am sure the murmur is an innocent systolic murmur due to high stroke volume. The chest X-ray of December 1986 shows a normal sized heart with an area of patchy consolidation in the right lower lobe. The follow-up X-ray in January shows very slight increase in heart rate but his CTR is still within normal limits (15,5/32) and the increase in size is within the acceptable range of less than 1 cm. The ECG of 1985 that he brought along with him is not calibrated but within normal limits. The ECG of Janaury this year shows an apparent increase in voltage, however the calibration signal of 1,4 MV is bigger than standard and this explains the change in voltage and also the prominence of the inferior and lateral Q wave. They are narrow and I am quite confident that they are non-pathological. The ECG is otherwise perfectly normal. Echo-cardiology confirms a structurally normal heart. In summary then, I cannot find any evidence of myocarditis or other pathology in this young man. He is very fit and I re-assured him about his heart and encouraged him to exercise as much as he likes."

GPs must be aware of pseudo-syndromes

Because of the history of repeated upper respiratory tract infections going on to pneumonia, he was referred to the respiratory clinic at Groote Schuur Hospital. The attending physician, himself a well-known marathon runner, suspected that his problem lay in the category of altered host defenses. There are not one but several factors involved: Extracts from his report:

"The patient is a facultative mouth breather and although he does not have nasal obstruction, he has nasal speech and his tonsillar crypts contain inspissated excudate. Secondly, he runs between 60-90 kilometres per week and this involves a considerable cold-air challenge to his upper and lower respiratory tract with ensuing drying effect and lowering of mucosal barriers. Thirdly, on all 4 occasions his respiratory infection had followed within weeks of a fairly strenuous marathon or ultra-marathon. The physician felt that although it has yet to be proved, that runners who take part in strenuous running while incubating or mildly symptomatic from upper respiratory infections, frequently develop lower respiratory complications. These, when they occur, take longer to clear." In his experience he frequently sees this type of patient. A repeat of his FBC and ESR was normal as were his

Joggers are usually well-informed about fitness and sports medicine – and they know their bodies well

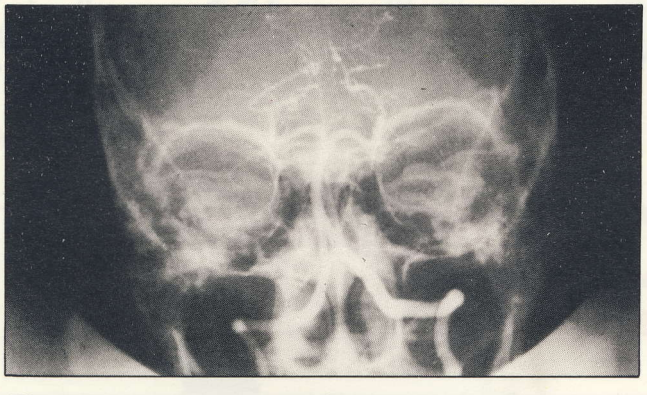
Serum Bio-Chemistry and Immunoglobulin levels. Spirometry was within normal limits but a saw-tooth appearance on the expiratory and inspiratory limbs were shown, which is within keeping with the large uvular which was clinically observed.

It was suggested that the patient use an antiseptic

mouthwash and gargle after running and once again at night. He was cautioned not to run when he had any upper respiratory symptoms.

Patient 2

Mr IS, a travelling sales representative, aged 36, a novice marathon runner only started running in 1985. His big aim was to run the Peninsula Marathon in 1986. He did not train very hard, running a maximum of about 70-80 kilometres per week with the author. As he was so frequently away from home, he could not conform to a regular running schedule. About 10 days before the Peninsula Marathon in 1986, he developed a mild upper respiratory tract infection. He vacillated between the author, the pharmacist and his regular general practitioner. Six days before the Peninsula Marathon he started an antibiotic and decided not to run. His sights were still set on the Two Oceans Marathon (56 kilometres), but as he had not previously run a marathon, he did not qualify. A special run for runners in Cape Town was held one week after the Peninsula Marathon and he managed to complete the distance within the required time to qualify for the Two Oceans. He ran the Two Oceans Marathon in a modest time of 5 hrs 32 minutes and admitted as to not having ever felt better in his life. However, two weeks later, after a short run, he started developing headaches. The headaches were bi-temporal with a tight-like band across his head. He consulted his general practitioner and tension headaches were diagnosed. Two days later he developed a severe headache which got worse. The next morning he woke up and could not move his head. He was admitted to a private hospital and a neuro-surgeon was consulted. A lumbar puncture revealed Xanthochromic fluid. A brain-scan showed a suspicion of an aneurysm on his anterior communication artery. However, an angiogram was completely normal. The patient



The angiogram was normal.

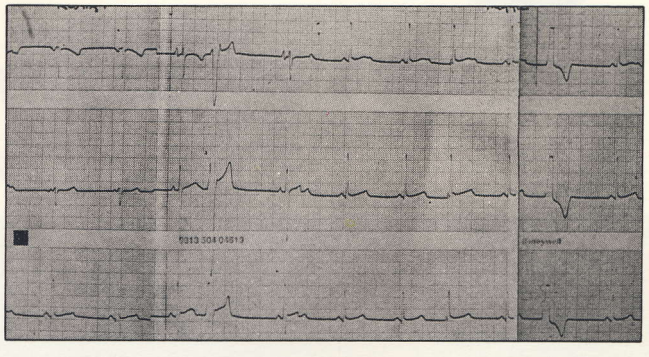
was re-assured, discharged after a few days and told not to exercise for a while and then gradually to start exercising again. However, the patient and his family blamed this unfortunate episode on marathon running and he undertook never to run a marathon ever again. He was told to report back to the neuro-surgeon a month later for a repeat angiogram. However, as he was feeling very much better, he decided not to have a repeat procedure. It is now one year since this episode and the patient has not run further than 15 kilometres.

Athletes are usually one step ahead of the physician's diagnosis

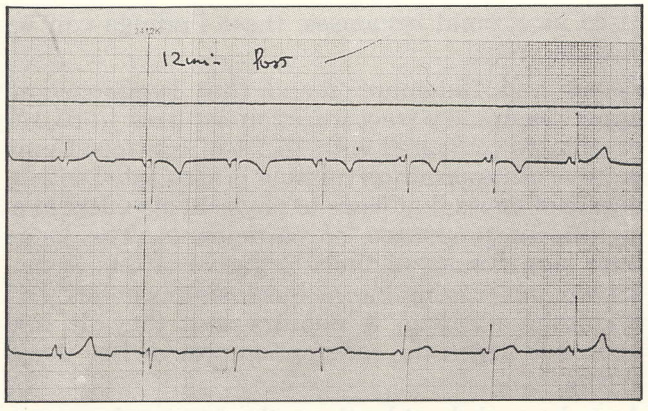
This case was discussed with the neuro-surgeon. He was asked the significance of the Xanthochromic fluid, as this was definitely not a "bloody-tap". He explained that in 20% of cases when seen after the initial bleed, an aneurysm can seal-off and not show up on angiogram. To be 100% sure in excluding the presence of an aneurysm, a follow-up angiogram should be performed. The patient is quite adamant about not having a repeat of his procedure so we are left not knowing the actual cause of his headaches.

Patient 3

Dr SJ, aged 35, a keen runner who runs 60 kilometres per week and plays squash every lunchtime, presented to his physician on 15 January 1987 after a fairly strenuous game of squash. The reason for his presentation was that he developed a rather non-specific pain in his chest which did not radiate and had no associated features. However, in view of the persistance of the pain, he decided to go to his physician. The physical examination was completely normal. His resting ECG showed him to be in sinus rhythm with numerous unifocal ventricular premature beats. His PR interval was normal and he had a normal resting ECG. In view of the atypical chest pain and normal resting ECG, the physician decided to go ahead and do a multi-stage treadmill test according to the Bruce Protocol. The physician was rather concerned during the exercise as the patient achieved a heart-rate of 150 after 2 minutes and 10 seconds in stage 1 of the Bruce Protocol. This is a rather rapid heart response for a



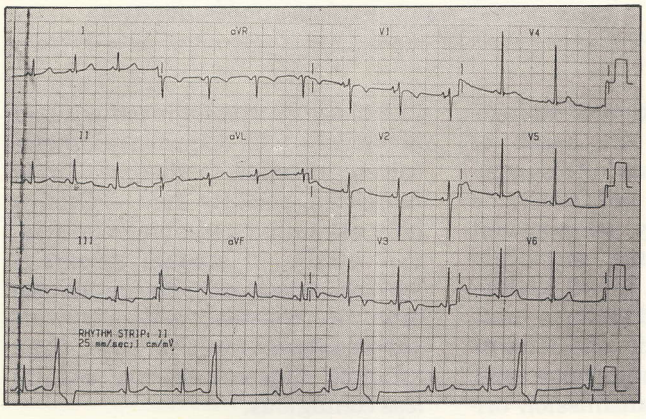
ECG - no change in spite of rapid heart response to exercise.



Post-exercise ECG showed bi-phasic T-wave changes across the anterior chest leads.

person who is supposedly fit and athletic. However, as he was completely asymptomatic and his blood pressure responses adequate, the exercise was continued. However, the physician decided to stop it early in stage 2 in view of the very rapid heart rate response to exercise. At this stage his ECG showed no change at all. However, during the post-exercise period he then developed bi-phasic T-wave changes across the anterior chest leads and these persisted 11 minutes into the post-exercise period. The patient experienced no chest pain whatsoever during this time. In the physician's words: "I was therefore left with a rather awkward position of a person who has atypical chest pain, has a rather rapid heart rate response to exercise and then developed these nasty looking T-wave changes in the post-exercise period. Although T-wave changes during exercise have very little value in diagnosing ischaemic disease, I felt in view of the fact that the patient was keen to go for a run, we should admit him to hospital for a 24-hour period of observation."

On arrival at the Coronary Care Unit, his ECG persisted in showing these changes and what com-



Patient 3 - ECG in the Coronary Care Unit.

plicated the issue further was the fact that his cardiac enzymes were elevated. The CK of 581 (normal up to 130). This however did not concern the physician as he did have a very strenuous game of squash and skeletal muscle CK could be responsible for this abnormality. In any event, he decided to treat him as ischaemic at that stage, although there were a lot of features to suggest non-cardiac pain.

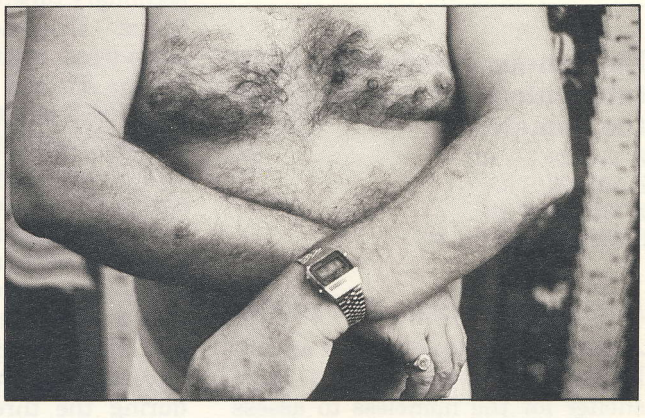
The next morning his iso-enzymes were done and this showed the level was 9 (upper limit of normal being 8). This therefore excluded a myocardial infarct.

The dilemma of the physician is that he had a patient who had atypical chest pain, an abnormal heart rate response to exercise, uninterpretable T-wave changes in the post-exercise period and thus he could still not rule out ischaemic heart disease completely. In view of the complexity of the situation, he elected to proceed with angiography. This was completely normal and the patient discharged himself from hospital and went for a run that very same day!

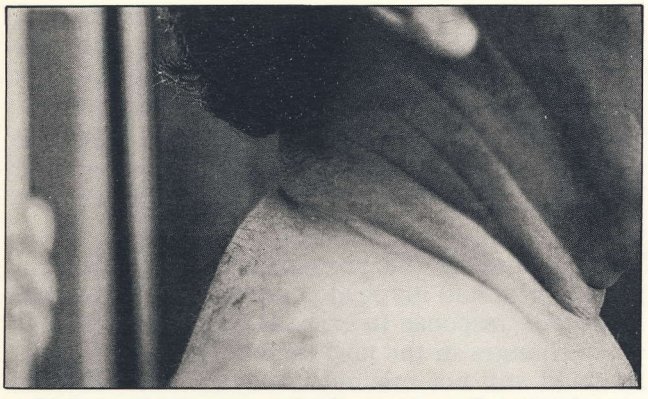
Runners are well-informed about their bodies, yet are susceptible to misrepresentations

Patient 4

Mr FD, aged 54, consulted me in November 1985. He is the Regional Service Manager for a large motor company in the Western Cape. He complained of tiredness, water-retention and palpitations. He had attempted to run the Peninsula Marathon for the first time in that year and found that he kept running into "the wall". (A year previously he had been to a physician. He performed well on a treadmill and was told there was nothing wrong with him. Blood tests showed that his cholesterol was raised and he was advised to go on diet and lose some weight.) He came back to consult me saying that his weight was varying; he could lose up to as much as 3 kilograms in one day and put it on the next day. He had to get up a few times at night to pass urine; he was getting short of breath going up steps; he also complained of



Patient 4 - Bruising on the arms.



Patient 4 showing thickened neck.

palpitations and anxiety. His fasting Lipogramme, uric acid, thyroid function test, glucose, full blood count and ESR were repeated and all found to be normal. His potassium was on the lower side of normal. The physician he was referred to felt that he had symptoms highly suggestive of Cushing's Disease. This was confirmed by doing urinary cortisoles and a dexamethazone suppression test. He was referred to Groote Schuur Hospital where the diagnosis was confirmed. He was referred to a neuro-surgeon for trans-sphenoidal hypophysectomy. The patient was subsequently successfully operated on in May 1986.

He was seen again in October 1986 when he was suffering from the side-effects of cortisone. He has subsequently done very well and has now started running again.

Discussion

It is not always appreciated that "abnormal" test results can be found transiently after vigorous exercise. They may also occur on a more long term basis in athletically fit individuals such as joggers. These changes may be relatively mild, but at times may be more pronounced in degree. Unless recognised as normal responses, these findings can be misinterpreted.

Peters and Bateman[4] found that symptoms of upper respiratory tract infection occurred in 33,3% of runners compared with 15,3% of controls. Competitive ultramarathon running is associated with a degree of stress that leads to physical disorders in a significant proportion of participants. The long-term significance of these negative effects is not known, but judging by the increasing popularity of marathon running, it appears that they do not appear to lessen the satisfaction provided by these events.

A study carried out by the author[5] on road-runners revealed that most runners do not initially use general practitioners (GPs) for sports injuries. One of the reasons given was that the GP was not perceived to have the knowledge, expertise or appropriate attitude to treat their injuries.

The above cases illustrate that not only must the doctor obtain skills in the area of sports injuries, but he/she must also be aware of "pseudo-syndromes", acceptable ECG changes in fit athletes and also bear in mind that their illness may have no relation at all to the sports in which they participate.

References

1. James SL, Bates BT, Ostering LR. Injuries to Runners. *Am J Sports Med* 1978; **6:** 40-9
2. Clancy W, Jun. Injuries to Runners (Editorial Comment). *Am J Sports Med* 1978; **6:** 49-50
3. Legwold G, Moore M, Hage P. Sports Medicine: The Momentum Continues. *The Phys & Sports Med* 1983; **11**(6): 152-67
4. Peters EM, Bateman ED. Ultramarathon Running and Upper Respiratory Tract Infections. *S Afr Med J* 1983; **64:** 582-4
5. Furman S. Sports Injuries and Family Practice - Who do Roadrunners Consult? *S Afr Med J* 1986; **69:** 108-10

From the Journals

Exogenous reinfection with tuberculosis in a shelter for the homeless

E NARDELL MD
B McINNIS RN
B THOMAS RN
S WEIDHAAS RN, MS

N Engl J Med 1986; 315: 1570-5

Abstract We investigated an outbreak of tuberculosis in a large shelter for the homeless to assess the role of exogenous reinfection as opposed to reactivation of endogenous infection as the cause of secondary tuberculosis in this population. Exogenous reinfection is considered relatively unimportant in the United States and other developed countries. Of 49 shelter-related cases, 22 had cultures resistant to both isoniazid and streptomycin and of the same phage type, indicating recent transmission originating with a single index patient. The probable index patient had a 10-year history of isoniazid and streptomycin resistance - an uncommon pattern at the shelter during the three years preceding the outbreak.

In 4 of the 22 cases, the patient had previously had documented tuberculosis infection or disease. These reinfected patients had extensive lung cavitation and numerous acid-fast bacilli on sputum smears - features associated with contagiousness. In contrast, patients with tuberculosis for the first time (primary tuberculosis) are usually less contagious.

We conclude that exogenous reinfection may have been an important factor leading to highly contagious secondary cases and an acceleration of the usual pattern of tuberculosis transmission in this highly susceptible population.