morphologically and with the help of immunocytochemistry. There was no staining by antibodies CK 1 (Dako) or CAM 5.2 (Becton-Dickinson) to intracellular cytokeratin antigens. All the tumours stained positively for vimentin but not for desmin or leucocyte common antigen. The main differential diagnosis of spindle cell squamous carcinoma was excluded in each case.

Comment
Squamous cell carcinoma is a well recognised complication of venous ulceration, and basal cell carcinoma has also been reported.1 Sarcomatous change has previously been considered exceptionally rare,2 and malignant fibrous histiocytoma has never been reported.

Predisposing factors to malignant fibrous histiocytoma include radiotherapy and trauma. None of our patients had a history of radiotherapy, and only one

Is severe bradycardia in veteran athletes an indication for a permanent pacemaker?

Robin J Northcote, Andrew C Rankin, Regina Scullion, William Logan

Severe bradycardia may be present in athletes without symptoms who wish to continue training, and the correct way to manage these patients is uncertain.

Case reports

CASE 1
A 66 year old man with 50 years’ running experience presented with a non-specific anaemia. He ran between 40 and 129 km a week. Apart from a resting bradycardia he had no clinically detectable cardiovascular abnormality. A resting 12 lead electrocardiogram showed sinus bradycardia, first degree heart block (PR interval=0.31 seconds), normal ventricular axis, and voltage criteria for left ventricular hypertrophy (S wave in V1+R wave in V6=39 mm). He exercised on a treadmill (Bruce protocol) for 16.5 minutes. By stage II he had developed ST segment depression of 1 mV in the inferolateral leads. Ambulatory electrocardiography showed a mean heart rate over 24 hours of 49 beats/minute. His minimum heart rate was 17 beats/minute, at 0300 (figure). He had first and second degree heart block (Mobitz type II), and complete heart block occurred nocturnally. Pauses of greater than 2 seconds occurred on 846 occasions, the longest being 8.9 seconds. Subsequent monitoring in hospital showed recurrent nocturnal ventricular standstill lasting 10-12 seconds.

We implanted a dual chamber permanent pacemaker (Pacesetter AFP 283), and subsequent coronary angiograms were normal. He felt generally more energetic and noticed a considerable improvement in his racing performances.

CASE 2
A 52 year old former professional boxer who ran 11 km daily had risen from bed and lost consciousness. When admitted to hospital he lost consciousness again; this was associated with ventricular standstill lasting 30 seconds. He denied cardiovascular symptoms but admitted to having had episodes of lightheadedness or “muzziness” that were relieved by brisk exertion. We implanted a permanent pacemaker (Vitatron Quintech TX 915 rate responsive generator) on the basis of his history and asystolic pauses. Subsequently his symptoms stopped.

Comment
Athletes’ bradycardia is ascribed to an increase in vagal tone,1 though pharmacological denervation has shown that they have a slower intrinsic heart rate independent of vagal tone. High degree atrioventricular block or prolonged ventricular pauses are rare.2 Veteran athletes may be more susceptible to bradyarrhythmias because of the decrease in heart rate that occurs with increasing age combined with the effects of many years of physical training.

The absence of underlying cardiac disease in these two patients makes it likely that the profound bradycardia with prolonged cardiac pauses was the result of lifelong endurance training. Whether such secondary bradyarrhythmias require implantation of a permanent pacemaker is uncertain. Nocturnal high degree atrioventricular block, with pauses of up to six seconds, has been reported to resolve after the intensity of physical training has been reduced.3 Pacemakers have been implanted in three young adults with prolonged sinus pauses during rapid eye movement sleep because of
Dietary maladvice as a cause of hypothyroidism and short stature

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Restrictive diets for children can be dangerous.1 We describe a child who was given a restrictive diet for purported food sensitivity and presented with short stature and hypothyroidism due to dietary iodine deficiency.

Case report

A 4 year old boy was investigated for short stature. He had weighed 3200 g at birth (>25th centile) and had been fed cows' milk formula for six months, when solid food was introduced. When he was 2 his mother sought advice because of his poor appetite and constipation. His height and weight were below the third centile, but his rate of growth was normal. Both parents were short (father 152 cm, mother 149 cm). Despite treatment with lactulose he remained constipated, and a dietitian advised the mother to withdraw cows' milk and give him soya milk. During the next six months he developed diarrhoea and was referred by his general practitioner to a centre for “alternative therapies,” where sensitivity to cows' milk, dairy products, goats' milk, eggs, chocolate, sugar, food additives, fish, beef, lamb, and pork was diagnosed. On advice his mother withdrew these items from his diet. During the next year his rate of growth dropped below the third centile. The table summarises the investigations over time. On examination at 4 years 3 months his weight and height were below the third centile. Plasma electrolyte concentrations were normal, albumin concentration was 36 g/l, and radiological assessment gave a bone age of 3 years. Six weeks later results of pituitary function tests showed suboptimal growth hormone response but normal responses of cortisol, gonadotrophin, and prolactin. Basal plasma thyroid stimulating hormone concentration was raised but the response to thyrotrophin releasing hormone was normal. Reassessment six months later showed an adequate response of growth hormone to glucagon. Plasma total and free thyroxine concentrations were low, and because the basal plasma thyroid stimulating hormone concentration was raised and the response to thyrotrophin releasing hormone exaggerated hypothyroidism was diagnosed. His autoimmune profile was normal, and thyroidal uptake of iodine-131 was 68% at four hours (normal 11-30%). Dietary assessment showed an average daily iodine intake of 40 μg/day (recommended daily allowance 120 μg), and 24 hour urine iodine excretion was low on two occasions (6·2 and 30·2 μg/g creatinine). A normal diet including cows' milk supplemented with iodine (40 μg/day) was reintroduced. After four

Results of biochemical investigations, weight, and height related to changes in diet

<table>
<thead>
<tr>
<th>Age</th>
<th>Treatment</th>
<th>Results of investigations</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 Years 2 months</td>
<td>Cows' milk substituted with soya milk</td>
<td>Plasma growth hormone 2-5 μU/l basally, 4-5 μU/l after exercise test. Plasma insulin-like growth factor 1-100 U/I. Plasma total thyroxine 94 nmo/l</td>
<td>77-5</td>
<td>9-0</td>
</tr>
<tr>
<td>3 Years 0 months</td>
<td>Restrictive diet introduced</td>
<td>Plasma growth hormone 2-5 μU/l basally, 4-5 μU/l after exercise test. Plasma insulin-like growth factor 1-100 U/I. Plasma total thyroxine 94 nmo/l</td>
<td>81-3</td>
<td>9-9</td>
</tr>
<tr>
<td>3 Years 6 months</td>
<td>Restrictive diet introduced</td>
<td>Plasma growth hormone 2-5 μU/l basally, 4-5 μU/l after exercise test. Plasma insulin-like growth factor 1-100 U/I. Plasma total thyroxine 94 nmo/l</td>
<td>87-5</td>
<td>12-0</td>
</tr>
<tr>
<td>4 Years 3 months</td>
<td>Restrictive diet introduced</td>
<td>Plasma growth hormone 2-5 μU/l basally, 4-5 μU/l after exercise test. Plasma insulin-like growth factor 1-100 U/I. Plasma total thyroxine 94 nmo/l</td>
<td>87-6</td>
<td>12-4</td>
</tr>
<tr>
<td>4 Years 5 months</td>
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<td>91-0</td>
<td>12-5</td>
</tr>
<tr>
<td>5 Years 0 months</td>
<td>Normal diet reintroduced, with iodine supplementation 40 μg daily for 4 weeks</td>
<td>Plasma growth hormone 2-5 μU/l basally, 4-5 μU/l after exercise test. Plasma insulin-like growth factor 1-100 U/I. Plasma total thyroxine 94 nmo/l</td>
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</tr>
</tbody>
</table>

1 Insulin 0-15 U/kg and gonadotrophin releasing hormone 100 μg and thyrotrophin releasing hormone 200 μg all intravenously.

2 Glucagon 0.5 mg intramuscularly and propranolol 20 mg orally and gonadotrophin releasing hormone 100 μg and thyrotrophin releasing hormone 200 μg intravenously.

Reference ranges: total thyroxine 60-160 nmol/l; free thyroxine 9-28 pmol/l; triiodothyronine 1-2.5 μmol/l; basal thyroid stimulating hormone <5 μU/mL; insulin-like growth factor 1-120-900 U/I.