morphologically and with the help of immunocyto-chemistry. There was no staining by antibodies CK 1 (Dako) or CAM 5.2 (Becton-Dickinson) to intra-cellular 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 histio-cytoma include radiotherapy and trauma. None of our patients had a history of radiotherapy, and only one

(case 3) had had a minimal injury. Malignant fibrous histiocytoma is a highly malignant neoplasm with a pronounced tendency to recur locally after excision.[3] Distal and superficial tumours, however, carry a better prognosis than those in proximal sites or deeper structures.[4] This complication of venous ulceration is therefore potentially curable if diagnosed early. All three of our patients were probably cured. It therefore becomes even more important that malignancy should be considered by all doctors managing chronic venous ulcers and that a biopsy should be performed promptly when necessary.

1 Burns DA, Calnan CD. Basal cell epithelioma in a chronic leg ulcer. Clin Exp Dermatol 1978;3:443-5.
2 Nunnery EW, Lipper S, Reddick R, Kahn LB. Leiomyosarcoma arising in a chronic venous stasis ulcer. Hum Pathol 1981;12:951-3.
3 Weiss SW, Enzinger FM. Malignant fibrous histiocytoma, an analysis of 200 cases. Cancer 1978;41:2250-66.
4 Kearney MM, Soule EH, Ivins JC. Malignant fibrous histiocytoma, a retrospective study of 167 cases. Cancer 1980;45:167-78.

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

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

University Department of Medical Cardiology, Queen Elizabeth Building, Glasgow G12 8QQ
Robin J Northcote, MD, senior registrar
Andrew C Rankin, MD, lecturer

Department of Medical Cardiology, Victoria Infirmary, Glasgow G42 9TY
Regina Scullion, MB, registrar

Department of Cardiology, Victoria Hospital, Blackpool FY3 8NR
William Logan, MD, consultant

Correspondence to: Dr Northcote.

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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 brady-cardia 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 V_1+R wave in V_5=39 mm). He exercised on a treadmill (Bruce protocol) for 16·5 minutes. By stage II he had developed ST segment depression of 1 mm

in the inferolateral leads. Ambulatory electrocardio-graphy 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 pace-maker (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 2] though pharmacological denervation has shown that they have a slower intrinsic heart rate independent of vagal tone. High degree atrioventri-cular block or prolonged ventricular pauses are rare.[2 3] Veteran athletes may be more susceptible to brady-arrhythmias 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 brady-cardia 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 atrio-ventricular 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

Response of heart rate over 24 hours and frequency of cardiac asystole (longer than two seconds). Note maintenance of circadian rhythm

concern about possible insidious loss of neural or other bodily functions as a result of repeated nocturnal asystole.[4]

The frequent prolonged asystolic pauses in case 1 were considered sufficient indication for a pacemaker because of concern that the arrhythmia might progress, with the possibility of sudden death and adverse effects of chronic recurrent ventricular standstill. Whether stopping the patient from running would have successfully managed his condition is unknown, but he would have considered this to be an important deterioration in his quality of life.

Lifelong physical endurance training may result in an adaptive bradycardia and prolonged atrioventricular nodal conduction, which cannot be regarded as a purely benign physiological adaptation but may represent a pathological process causing symptoms, syncope, systemic embolism, or sudden death.[2] We chose to manage both patients with permanent pacemakers to maintain cardiac rhythm and allow them to continue their sporting activities. The improvement in performance in both patients would seem to support this policy.

1 Talan DA, Bauernfeind RA, Ashley WW, Kanakis C, Rosen KM. Twenty-four hour continuous ECG recordings in long distance runners. Chest 1982; 82:19-24.
2 Ector H, Verlinden M, Vanden-Eynde E, et al. Bradycardia, ventricular pauses, syncope and sports. Lancet 1984;ii:591-4.
3 Di Nardo-Ekery D, Abedin Z. High degree atrioventricular block in a marathoner with 5-year follow-up. Am Heart J 1987;113:834-7.
4 Guilleminault C, Pool P, Moota J, Gillis AM. Sinus arrest during REM sleep in young adults. N Engl J Med 1984;311:1006-10.

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Dietary maladvice as a cause of hypothyroidism and short stature

M Labib, R Gama, J Wright, V Marks, D Robins

Departments of Clinical Biochemistry and Clinical Nutrition and Paediatrics, St Luke's Hospital, Guildford, Surrey GU1 3NT
M Labib, MRCPATH, senior registrar
R Gama, MRCP, registrar
J Wright, MRCPATH, consultant
V Marks, FRCPATH, professor
D Robins, FRCP, consultant paediatrician

Correspondence to: Dr Labib, department of clinical biochemistry and clinical nutrition.

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 cow's 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 urinary 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

Age	Treatment	Results of investigations	Height (cm)	Weight (kg)
2 Years 2 months			77·5	9·0
3 Years 0 months	Cows' milk substituted with soya milk		81·3	9·9
3 Years 6 months	Restrictive diet introduced			
4 Years 3 months		Plasma growth hormone 2·5 mU/l basally, 4·5 mU/l after exercise test. Plasma insulin-like growth factor-1 100 U/l. Plasma total thyroxine 94 nmol/l	87·5	12·0
4 Years 5 months		Insulin stress test* showed cortisol 215 (basal) and 872 (peak) nmol/l, growth hormone 4·4 and 12·5 mU/l, prolactin 248 and 1365 mU/l, luteinising hormone 1 and 4 IU/l, follicle stimulating hormone 1 and 6 IU/l, thyroid stimulating hormone 6·4 and 20·7 mU/l. Plasma total thyroxine 106 nmol/l	87·6	12·4
5 Years 0 months		Glucagon-propranolol test† showed cortisol 554 (basal) and 1010 (peak) nmol/l, growth hormone 8·6 and 39·0 mU/l, prolactin 254 and 648 mU/l, luteinising hormone 1 and 6 IU/l, follicle stimulating hormone 1 and 5 IU/l, thyroid stimulating hormone 12·4 and 62·6 mU/l. Plasma total thyroxine 63 nmol/l. Plasma free thyroxine 7·8 pmol/l	91·0	12·5
5 Years 1 month	Normal diet reintroduced, with iodine supplementation 40 µg daily for 4 weeks		91·0	12·5
5 Years 2 months		Thyroid function tests‡ showed total thyroxine 102 nmol/l, triiodothyronine 2·4 nmol/l, free thyroxine 12·7 pmol/l, and thyroid stimulating hormone 4·8 mU/l (0 minutes), 24·8 mU/l (20 minutes), and 17·9 mU/l (60 minutes)		
5 Years 3 months		Thyroid function tests‡ showed total thyroxine 102 nmol/l, triiodothyronine 1·8 nmol/l, free thyroxine 15·8 pmol/l, and thyroid stimulating hormone 2·2 mU/l (0 minutes), 14·4 mU/l (20 minutes), and 11·1 mU/l (60 minutes)	93·0	14·0

*Insulin 0·15 U/kg and gonadotrophin releasing hormone 100 µg and thyrotrophin releasing hormone 200 µg all intravenously.
†Glucagon 0·5 mg intramuscularly and propranolol 20 mg orally and gonadotrophin releasing hormone 100 µg and thyrotrophin releasing hormone 200 µg intravenously.
‡Thyrotrophin releasing hormone 200 µg intravenously.
Reference ranges: total thyroxine 60-160 nmol/l; free thyroxine 9-28 pmol/l; triiodothyronine 1·2-3·0 nmol/l; basal thyroid stimulating hormone <5·0 mU/l; insulin-like growth factor-1 120-900 U/l.