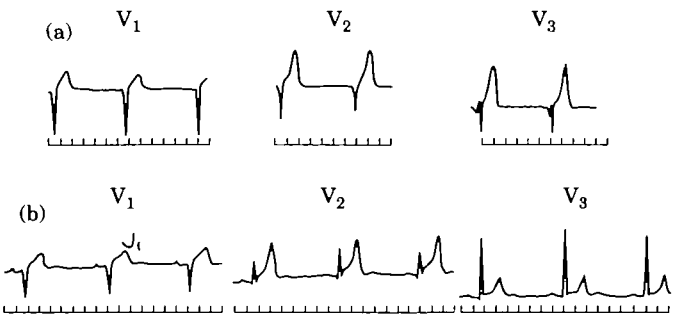

Figure 1 **Electrocardiogram leads V_1–V_3. A — before the removal of the parathyroid adenoma. B — 1 year after the removal of the adenoma.**

had shown a gradual deepening of the Q waves in leads V_2 and V_3, and 2 years later a QS pattern was fully established (Fig. 1a). The patient represented with episodes of oligoarthritis and treatment with indomethacin was started. Since 1993 he has complained of bone pain and nephrolithiasis. Serum calcium showed a value of 12 mg . dl^{-1}, serum phosphorus 1·3 mg . dl^{-1} and alkaline phosphatase a level of 884 U. He had 24-h urine calcium of 456 mg and hydroxyproline of 602 mg. An ultrasound scan of the parathyroids revealed an adenoma. One year after the removal of the adenoma (1995) the systolic murmur decreased in intensity to 2/6, the presystolic sound was not heard, but the carotid impulse was brisk. On the ECG, the QS pattern on leads V_2 and V_3 disappeared (Fig. 1b) and the former qR pattern reappeared. The echocardiogram demonstrated a decrease in the thickness of the interventricular septum, and absence of systolic anterior motion of the mitral valve.

Massive intracardiac calcific deposits are rare in persons under 65 years of age[1]. In recent years chronic hypercalcaemia has been linked with deposition of calcium in the cardiac valves, in the media and the intima of the coronary arteries and in individual myocardial fibres[2]. It has been suggested that parathyroid hormone rather than a rise in extracelular calcium concentration is associated with a spectrum of left ventricular hypertrophy and hypertrophic cardiomyopathy[3]. In our patient the clinical signs of hypertrophic subaortic stenosis were fully established before the clinical evidence of hyperparathyroidism. Slight increases of parathyroid hormone in serum may act on the heart, inducing disarray of myofibrils and myofilaments common to asymmetrical and symmetrical hypertrophy. The regression of the electrocardiographic QS pattern to qR pattern in leads V_2 and V_3 after the removal of the parathyroid adenoma suggests an effect of the parathyroid hormone on myocardial structural changes.

Katoh *et al.*[4], studying the effect of parathyroid hormone on the isolated papillary muscle of the rat heart, demonstrated that parathyroid hormone acts directly on the myocardium and increases the contractility. The inotropic effect of parathyroid hormone was suppressed by the addition of propanolol and by depletion of endogenous myocardial norepinephrine by reserpine. Complete suppression of the inotropic effect of the hormone by moethoxyverapamil and its action attenuation by increasing Ca^{++} concentration in the perfusate demonstrated that parathyroid hormone action on the myocardium is also mediated by an increase in Ca^{++} influx across the plasma membranes. As it has been shown that excessive inotropic stimulation of the myocardium can produce a haemodynamic picture similar to hypertrophic obstructive cardiomyopathy[5], prolonged inotropic action of the parathyroid hormone on the myocardium, may cause the combination of primary hyperparathyroidism with hypertrophic cardiomyopathy.

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Atypical elevation of creatine phosphokinase and isoenzyme (CK-MB) levels in a patient with mitral valve disease

Plasma creatine phosphokinase (CPK) and creatine kinase isoenzyme (CK-MB) levels are of great value in diagnosing acute myocardial infarction[1]. However, rare instances of atypical elevations of CPK and CK-MB have been reported in patients with asthma[2,3], in marathon runners[4], or during hyperventilation in healthy subjects[5]. It has been proposed that in these conditions the diaphragm may be the source of elevated CPK and CK-MB plasma levels.

A 70-year-old female with severe mitral valve disease was admitted because of severe dyspnoea and right heart failure with hepatosplenomegaly and peripheral oedema. After clinical recompensation with intravenous furosemide and spironolactone, echocardiography revealed normal systolic left ventricular function, severe calcification of the mitral valve with an orfice area 1·7 to 2 cm^2, significant mitral regurgitation with enlarged left and right atria as well as severe tricuspid regurgitation. Spirometry showed reduced vital capacity of 2 l, and an FEV_1 indicating mild bronchial obstruction. Right heart catheterization disclosed pulmonary hypertension (pulmonary artery pressure: 80/45 mmHg, mean: 60 mmHg); left ventricular function and coronary angiography were normal. Mitral valve replacement was recommended.

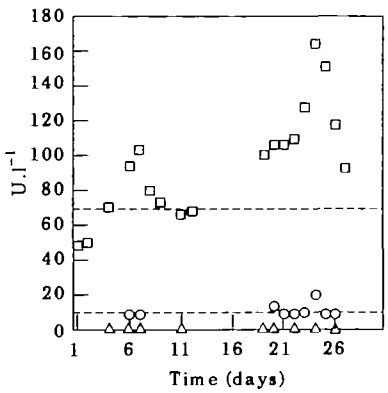

Figure 1 **Changes of creatine phosphokinase (CPK; normal range: 0–70 U . l^{-1}; □) and creatine kinase isoenzyme (CK-MB; normal range: 0–10 U . l^{-1}; ○) during hospital stay; △ marks the additional treatment with i.v. furosemide.**

After initial stabilization, the patient experienced repeated episodes of severe dyspnoea with clinical and radiological evidence of pulmonary congestion. During these episodes the respiratory rate increased to 30 to 40 per min, and additional i.v. furosemide (40–125 mg) was administered. In those instances there were no ECG changes and the patient denied any angina symptoms, but transient increases in CPK and CK-MB were observed (Fig. 1).

We describe a patient with mitral valve disease, who suffered from repeated episodes of severe dyspnoea and hyperventilation in the course of pulmonary congestion, followed by transient elevations of CPK and CK-MB. The increase in the plasma levels of these parameters is unlikely to have been caused by myocardial ischaemia, because the patient denied anginal symptoms, the ECG remained unchanged and the coronary angiogram showed normal coronary arteries. In the given scenario, a likely explanation is creatine phosphokinase release from the diaphragm in the course of hyperventilation episodes, as previously observed in some patients with primary pulmonary diseases[2–5].

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Streptococci strike twice

We wish to report an interesting case of infective endocarditis. A 75-year-old Ugandan Asian retired businessman was first noted to have a pan systolic murmur when he presented with a parotid abscess, which was drained with appropriate antibiotic cover. He had no history of rheumatic fever and had suffered no cardiac symptoms. An echocardiogram revealed mitral regurgitation. Eighteen months later he presented with a short history of fever, night sweats and shortness of breath: six sets of blood cultures yielded penicillin sensitive *Streptococcus bovis*, and he responded rapidly to intravenous benzylpenicillin and gentamicin for 10 days. Echocardiogram confirmed mitral regurgitation and thickened aortic valve cusps. Transoesophageal echocardiography was not performed.

Four months later, he represented with a 5 week history of fever, night sweats, declining exercise tolerance and orthopnoea. Splinter haemorrhages were noted. Ausculation and echocardiogram were unchanged. Three sets of blood cultures yielded *Streptococcus sanguis*, fully sensitive to penicillin. He received intravenous benzylpenicillin and gentamicin for 2 weeks followed by 5 weeks oral penicillin therapy. A barium enema showed scattered colonic diverticulae.

He experienced an episode of slurred speech and four episodes of facial weakness whilst on antibiotics, and these were felt to be embolic in origin. A CT scan of his brain was normal; and blood cultures were repeatedly sterile.

Six months later he underwent dental clearance for widespread periodontal disease, with co-amoxyclav and gentamicin prophylaxis. He has remained well since.

The prevalence of infective endocarditis is estimated to be between 0·3 and 3·0 per thousand hospital admissions[1]. This equals about a thousand cases per year in England and Wales[2]. The oral streptococci are the commonest cause of bacterial endocarditis, and of these *Streptococcus sanguis* is one of the most frequent[3]. However, a recent review confirmed the traditional association between dental disease or dental treatment and the causative organism of endocarditis in only 15% of cases[2].

Simonson *et al.*[4] reported a case of an intravenous drug abuser who suffered nine separate episodes of endocarditis over a 17-year period. Our patient, however, illustrates the traditional association of endocarditis and dental disease. His episode of bacterial endocarditis with *Streptococcus bovis*, although treated for a shorter period than is recommended[5], has shown no signs of recurrence. His case reminds us that dental examination and appropriate dental treatment is warranted after an episode of endocarditis from whatever source, to help reduce the future risk of infection to an already damaged valve.

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I would like to thank Dr Mark Dancy for permission to report this case.

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