19 Freedom RM, Dische MR, Rowe RD. Pathologic anatomy of subaortic stenosis and atresia in the first year of life. *Am J Cardiol* 1977;**39**:1035–44.
20 Vogel M, Freedom RM, Brand A, Trusler GA, Williams WG, Rowe RD. Ventricular septal defect and subaortic stenosis: an analysis of 41 patients. *Am J Cardiol* 1983;**52**:1258–63.
21 Minich LL, Snider R, Bove EL, Lupinetti FM. Echocardiographic predictors of the need for infundibular wedge resection in infants with aortic arch obstruction, ventricular septal defect and subaortic stenosis. *Am J Cardiol* 1992;**70**:1626–7.
22 Cooley DA, Garrett JR. Septoplasty for left ventricular outflow obstruction—without aortic valve replacement: a new technique. *Ann Thorac Surg* 1986;**42**:445–8.
23 DeLeon SY, Ilbawi MN, Arcilla RA, Thilenius OG, Quinones JA, Duffy EC, Sulayman RF. Transatrial relief of diffuse subaortic stenosis after ventricular septal defect closure. *Ann Thorac Surg* 1990;**49**:429–34.
24 Anderson RH, Becker AE, Robertson WB. The cardiovascular system (part A) In: Robertson WB, ed. *Systemic pathology*. 3rd ed. London: Churchill Livingstone, 1993: 130.
25 Van Praagh R, Bernhard WF, Rosenthal A, Parisi LF, Fyler DC. Interrupted aortic arch: surgical treatment. *Am J Cardiol* 1971;**27**:200–11.
26 Freedom RM, Bain HH, Esplugas E, Dische R, Rowe RD. Ventricular septal defect in interruption of aortic arch. *Am J Cardiol* 1977;**39**:572–82.
27 Cassidy SC, Van Hare GF, Silverman NH. The probability of detection of a subaortic ridge in children with ventricular septal defect or coarctation of the aorta. *Am J Cardiol* 1990;**66**:505–8.
28 Gewillig M, Daenen W, Dumoulin M, van der Hauwaert L. Rheologic genesis of discrete subvalvular aortic stenosis: a Doppler echocardiographic study. *J Am Coll Cardiol* 1992;**19**:818–24.
29 Menahem S, Brawn WJ, Mee RB. Severe subaortic stenosis in interrupted aortic arch in infancy and childhood. *J Card Surg* 1991;**6**:373–80.

IMAGES IN CARDIOLOGY

ECG changes of severe hyperkalaemia

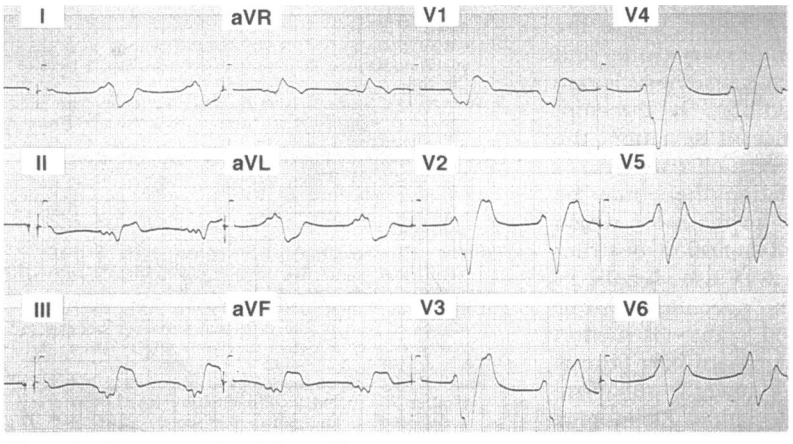

Figure 1 Serum potassium 8·0 mmol/l.

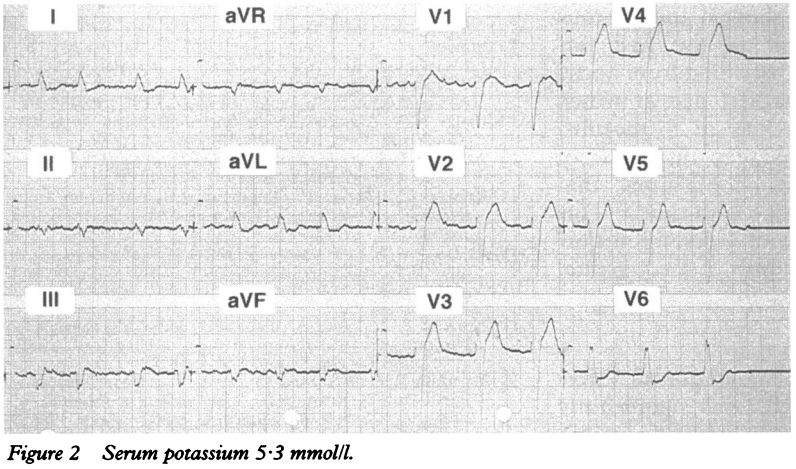

Figure 2 Serum potassium 5·3 mmol/l.

A 71 year old man was admitted after being found collapsed at home. Clinically he was in cardiogenic shock with severely impaired cerebration, gross evidence of cardiac failure, a systolic blood pressure of 50 mm Hg, and a regular pulse of only 42 beats per minute. His initial electrocardiogram is shown (fig 1). As soon as the electrocardiogram was available he was given 10 ml calcium gluconate together with dextrose and insulin. A repeat electrocardiogram performed after these measures was dramatically improved (fig 2). Subsequently, his admission potassium was found to have been greater than 8·0 mmol/l. It had declined to 5·3 mmol/l at the time of the second electrocardiogram. Figure 1 shows extremely broad, bizarre QRS complexes with markedly raised ST segments that can be mistaken for the acute injury current of myocardial infarction. In addition there is no evidence of P wave activity. Such electrocardiographic changes may be the harbinger of ventricular tachycardia, fibrillation, or asystole. It is rare to find such gross electrocardiographic changes. More frequently with lesser degrees of hyperkalaemia there are tall, peaked T waves. Increasing hyperkalaemia, however, leads to a progressive reduction in P wave amplitude, PR prolongation, loss of R waves, and progressive widening of the QRS complex.

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