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IMAGES IN CARDIOLOGY

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Hyperkalaemia causing profound bradycardia

Two elderly patients presented with profound bradycardia. Both were taking angiotensin converting enzyme (ACE) inhibitors for widespread vascular disease. One had diarrhoea and the other recently started spironolactone. The first patient's ECG showed regular broad QRS complexes with no P waves (panel A). She was paced without improvement. The second patient had a ventricular rate of 20/min with irregular broad QRS complexes and absent atrial activity, consistent with slow atrial fibrillation (ECG not shown). Initial treatment was with atropine and an external pacemaker. Serum potassium in the first patient was 9.4 mmol/l with urea 42.6 mmol/l and creatinine 598 µmol/l. Corresponding values for the second patient were potassium 7.7 mmol/l, urea 20.2 mmol/l and creatinine 21 µmol/l. The heart rate in both cases responded to intravenous calcium chloride (shown for first patient in panel B).

When patients collapse and are found to be bradycardic it is likely that an ECG will be available before the serum biochemistry. Clinicians should be aware that life threatening hyperkalaemia may cause profound bradycardia and bear a superficial resemblance to complete heart block. The clue to the correct diagnosis is the broad QRS complex with absence of P waves. True second and third degree atrioventricular (AV) block have been described in hyperkalaemia but are uncommon because the P wave usually disappears before such advanced AV block occurs. If the ECG is available before the serum potassium and is consistent with life threatening hyperkalaemia, then it would seem sensible to give calcium chloride speculatively while waiting for the biochemistry results.

K Noble
C Isles
chris.isles@nhs.net