SHORT REPORTS

Effect of dual chamber pacing on raised plasma atrial natriuretic peptide concentrations in complete atrioventricular block

Atrial natriuretic peptide is released into the circulation in normal humans in situations of increased central blood volume and hence atrial pressure, such as head out immersion in water.[1] The same stimulus of raised atrial pressure is also responsible for the increased plasma concentrations of atrial natriuretic peptide observed in pathological states of fluid overload, including cardiac and renal failure.[2] Raised atrial pressures also occur in patients with complete atrioventricular block because of the frequent occurrence of atrial systole during ventricular systole. In theory this could provide an adequate stimulus for a sustained rise in plasma atrial natriuretic peptide concentrations. Dual chamber pacing restores the normal sequence of atrial and ventricular contraction, whereas ventricular pacing leaves atrial and ventricular systole uncoordinated. We used these pacing modes to determine the relative effects on plasma atrial natriuretic peptide concentration of coordinated and dissociated atrial and ventricular chamber contraction.

Patients, methods, and results

Thirty two patients (17 men and 15 women, aged 44-85) with complete atrioventricular block were studied. All had normal atrial activity and none had retrograde atrioventricular conduction, cardiac failure, or renal failure. Six of these patients (aged 72 (SEM 14)), who presented acutely with complete atrioventricular block, had a dual chamber mode programmable pacemaker implanted. Plasma atrial natriuretic peptide concentrations (determined by radioimmunoassay[3]) were measured before pacing and at the end of each of two consecutive 30 day periods. During these periods patients were paced in ventricular demand mode at 70 beats/min and in dual chamber mode (back up 70 beats/min, upper rate 125-150 beats/min, and atrioventricular delay of 150 ms) in randomised order. Plasma atrial natriuretic peptide concentration was also estimated in 13 patients (aged 71 (2), atrial rate 78 (2) beats/min) paced in ventricular demand mode at 70 beats/min for 43 (6) months and in 13 patients (aged 63 (4), atrial rate 72 (3) beats/min) paced in dual chamber mode (programmed as above) for 40 (4) months. Thirteen of the patients were paced with ventricular demand pacing rather than the preferred dual chamber mode, either because dual chamber pacing had not yet become available (seven patients) or because of disease in other systems (six patients). Atrial natriuretic peptide values were compared with those from 13 age and sex matched convalescent hospital patients with no cardiac or renal disease.

The mean plasma atrial natriuretic peptide concentration in patients presenting with complete atrioventricular block was 141 (28) pmol/l compared with 58 (7) pmol/l in the controls. These raised concentrations were essentially unchanged after ventricular demand pacing for 30 days, whereas pacing in dual chamber mode for 30 days returned plasma atrial natriuretic peptide values to normal values (table). In the two groups long term ventricular demand pacing was again associated with raised plasma atrial natriuretic peptide concentrations whereas those paced in dual chamber mode had normal plasma concentrations.

Comment

The frequent simultaneous occurrence of atrial and ventricular systoles in complete atrioventricular block causes episodic raised atrial pressure, which can be observed clinically as cannon waves in the jugular venous pulse.[4] We tested, and confirmed, the hypothesis that these raised atrial pressures increase plasma atrial natriuretic peptide concentrations.

Plasma atrial natriuretic peptide concentration is also raised during paroxysmal tachycardia.[5] The stimulus for atrial natriuretic peptide secretion during these episodes might be either increased atrial pressure or increased heart rate. The occurrence of raised plasma concentrations of atrial natriuretic peptide in the presence of reduced heart rate in complete atrioventricular block suggests, however, that heart rate or even atrial rate per se is not an important independent factor promoting atrial natriuretic peptide secretion. Atrial natriuretic peptide possesses natriuretic and vasodilator properties and also inhibits renin and aldosterone release. Its secretion in conditions of volume overload is therefore clearly appropriate. In complete atrioventricular block, which in these patients was not accompanied by fluid overload, raised plasma atrial natriuretic peptide concentrations may protect against the development of overt cardiac failure.

Dual chamber pacing restores the normal atrioventricular sequence, whereas ventricular demand pacing does not. The restoration of plasma atrial natriuretic peptide concentrations to control values by dual chamber pacing serves to emphasise the physiological nature of this mode.

Plasma atrial natriuretic peptide (ANP) concentrations in controls, patients with complete atrioventricular block unpaced and paced acutely in ventricular demand and dual chamber modes, and patients paced long term in ventricular demand and dual chamber modes

		Study group (n=6)				
	Controls (n=13)	Unpaced	Short term ventricular demand pacing	Short term dual chamber pacing	Long term ventricular demand pacing (n=13)	Long term dual chamber pacing (n=13)
ANP (pmol/l)	58 (7)	141 (28)*	139 (30)	53 (7)	112 (13)**	43 (7)

*p<0·05; **p<0·01 compared with controls (unpaired *t* tests).

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Early diagnosis of chronic fetal hypoxia in a diabetic pregnancy

Late intrauterine fetal death continues to be a problem in diabetic women, and studies of cord blood have implicated hypoxia as a possible cause.[1] The fact that these deaths may occur without warning has led to the practice of early delivery. We describe a diabetic pregnancy in which hypoxia was suspected from Doppler studies of blood velocity in the fetus and was confirmed by cordocentesis.[2]

Case report

A 27 year old primigravid, insulin dependent diabetic (White class B) was found to have a high resistance index in the uteroplacental circulation (>95th centile for our normal range) on routine Doppler screening at 23 weeks' gestation. As she had already requested cordocentesis for fetal karyotyping the fetal blood gas tensions were also measured and compared with our normal range[3]: oxygen tension was 4·9 kPa and pH 7·33 (figure). Fetal growth was normal, with a good volume of amniotic fluid. A series of blood glucose tests performed by the mother showed good control of her diabetes, but her glycosylated haemoglobin concentration was raised (mean 13%; range 9·4-17·2%) throughout pregnancy.

At 29 weeks' gestation the Doppler studies showed no end diastolic frequencies in the umbilical artery. Because of the association between this characteristic and hypoxia[4] cordocentesis was repeated. Blood gas tensions confirmed that the fetus was hypoxic but not acidotic (oxygen tension 3·4 kPa, pH 7·35).

Daily inpatient monitoring by Doppler measurement of fetal blood flow showed persistent abnormalities, although cardiotocograms showed reactivity, fetal activity was good, and fetal growth was within the normal range. At 31 weeks