Table 1. Subject characteristics

Subject	Sex	Age (years)	Pacemaker	Indication	IHD	Heart failure	Drugs
1	Female	72	AAI	Bradycardia	No	No	Nifedipine, bumetanide
2	Male	73	AAI	Stokes Adams attacks*	Yes	No	Aspirin
3	Female	62	AAI	Stokes Adams attacks*	Yes	No	Aspirin
4	Female	64	DDD	Second degree heart block	No	No	—
5	Male	74	DDD	Second degree heart block	No	No	—
6	Female	54	DDD	Stokes Adams attacks*	Yes	No	Aspirin
7	Male	77	DDD	Second degree heart block	Yes	No	Aspirin, frusemide, atenolol
8	Female	71	AAI	Stokes Adams attacks*	No	No	—
9	Female	84	AAI	Stokes Adams attacks	No	Yes	Lisinopril, bendrofluazide
10	Female	77	AAI	Stokes Adams attacks*	No	No	—
11	Male	83	AAI	Stokes Adams attacks*	Yes	No	Aspirin
12	Male	77	DDD	Syncope	Yes	Yes	Aspirin, enalapril
13	Male	73	DDD	Stokes Adams attacks*	No	No	—
14	Male	73	DDD	Stokes Adams attacks*	No	No	Bendrofluazide
15	Female	65	AAI	Stokes Adams attacks*	No	No	Bendrofluazide
16	Male	54	DDD	Complete heart block	No	No	—
17	Male	51	DDD	Syncope	No	No	—
18	Male	59	AAI	Stokes Adams attacks*	No	No	—
19	Female	36	DDD	Complete heart block	No	No	—
20	Male	37	AAI	Syncope	No	No	—
21	Male	44	DDD	Complete heart block	No	No	—
22	Male	21	AAI	Syncope	No	No	—

AAI, atrial sensing and atrial pacing pacemaker. DDD, dual chamber pacemaker. IHD, ischaemic heart disease. * Documented sinus arrest.

Haemodynamic changes during pacing

The mode of pacing, atrial *versus* sequential atrioventricular, did not significantly affect the haemodynamic responses during incremental pacing (data not shown). Therefore, the data were treated as homogeneous, and analysed as a single group.

Incremental pacing from 60 to 110 beats min^{-1} resulted in a significant increase in peripheral systolic, peripheral diastolic and central diastolic blood pressure; but no change in central systolic pressure occurred (Table 2). AIx declined linearly with increasing heart rate (linear regression analysis: $r = -0{\cdot}76$; $P < 0{\cdot}001$). The slope of the regression line was $-0{\cdot}39$, indicating a reduction in AIx of 3·9% for each 10 beats min^{-1} increase in heart rate (Fig. 2*A*). Defining negative values of AIx as zero did not significantly affect the relationship between AIx and heart rate. Ejection duration also fell linearly with increasing heart rate (Fig. 2*B*), but there was no significant change in the timing of the reflected wave (Table 2).

The responses of peripheral and central diastolic pressure to pacing did not differ significantly ($P = 0{\cdot}50$), but the effect on systolic pressures did ($P < 0{\cdot}001$) as can be seen in Fig. 3*A* and *B*. There was an increase in arterial pressure amplification from the aorta to the brachial artery, as defined by the ratio of peripheral to central pulse pressure, during pacing (Table 2). However, when the ratio of peripheral pulse pressure to *non-augmented* central pulse pressure (P_1 − central diastolic pressure) was calculated, there was no significant change.

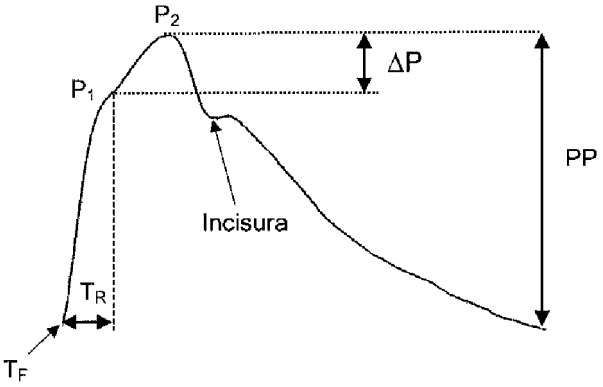

Figure 1. A central aortic pressure waveform

Ascending aortic pressure waveform from a 60-year-old man. Two systolic peaks are seen, P_1 and P_2, the latter becoming more prominent after the age of 35 years. The augmentation index is calculated as the difference between P_2 and P_1 (ΔP), expressed as a percentage of the pulse pressure (PP). Ejection duration was calculated as the time between the foot of the wave (T_F) and the incisura, and T_R is defined as the time between T_F and the inflection point.

Table 2. Effect of increasing heart rate on peripheral and central haemodynamics

HR (beats min^{-1})	AIx (%)	PSBP (mmHg)	PDBP (mmHg)	PMAP (mmHg)	CSBP (mmHg)	CDBP (mmHg)	CMAP (mmHg)	PPP:CPP ratio	P_1 (mmHg)	P_1 − CDBP (mmHg)	PPP:(P_1 − CDBP) ratio	T_R (ms)	ED (ms)
60	24·1 ± 2·9	135 ± 6	80 ± 2	98 ± 3	126 ± 6	82 ± 3	96 ± 3	1·28 ± 0·03	114 ± 4	30 ± 2	1·71 ± 0·03	137 ± 4	323 ± 5
70	21·3 ± 2·9	137 ± 6	84 ± 3	102 ± 4	128 ± 6	86 ± 3	100 ± 4	1·35 ± 0·04	117 ± 4	35 ± 3	1·73 ± 0·03	140 ± 4	307 ± 4
80	15·2 ± 3·1	141 ± 5	88 ± 2	106 ± 3	128 ± 5	90 ± 2	102 ± 3	1·42 ± 0·04	120 ± 4	34 ± 2	1·69 ± 0·03	138 ± 3	295 ± 4
90	12·1 ± 3·1	142 ± 6	90 ± 3	108 ± 4	128 ± 6	92 ± 3	104 ± 4	1·50 ± 0·04	122 ± 4	32 ± 2	1·72 ± 0·03	137 ± 2	284 ± 4
100	8·8 ± 3·3	144 ± 6	93 ± 3	110 ± 3	129 ± 5	96 ± 3	107 ± 3	1·59 ± 0·04	124 ± 4	32 ± 2	1·76 ± 0·03	135 ± 3	272 ± 3
110	5·0 ± 3·2	145 ± 5	96 ± 3	112 ± 3	129 ± 5	99 ± 3	109 ± 3	1·73 ± 0·05	126 ± 4	30 ± 2	1·83 ± 0·03	134 ± 2	257 ± 2
P	<0·001	0·001	<0·001	<0·001	0·89	<0·001	<0·001	<0·001	<0·001	0·52	0·34	0·09	<0·001

HR, heart rate; AIx, augmentation index; PSBP, peripheral systolic blood pressure; PDBP, peripheral diastolic blood pressure; PMAP, peripheral mean arterial pressure; CSBP, central systolic blood pressure; CDBP, central diastolic blood pressure; CMAP, central mean arterial pressure; PPP, peripheral pulse pressure; CPP, central pulse pressure; P_1, height of the first systolic peak of the central waveform; T_R, time to inflection point; ED, ejection duration. All values are quoted as means ± S.E.M. Significance was determined using ANOVA, and data were analysed as the change from values at a heart rate of 60 beats min^{-1}.

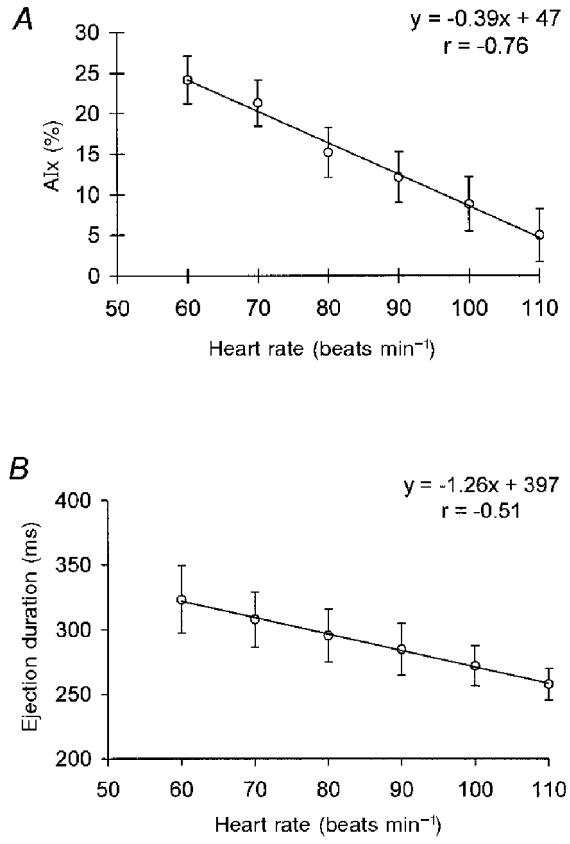

Figure 2. The effect of heart rate on augmentation index and ejection duration

A, the relationship between augmentation index (AIx) and heart rate. *B*, the relationship between ejection duration and heart rate. Values represent means ± S.E.M. ($n = 22$). A linear regression line is shown ($P < 0·001$, ANOVA).

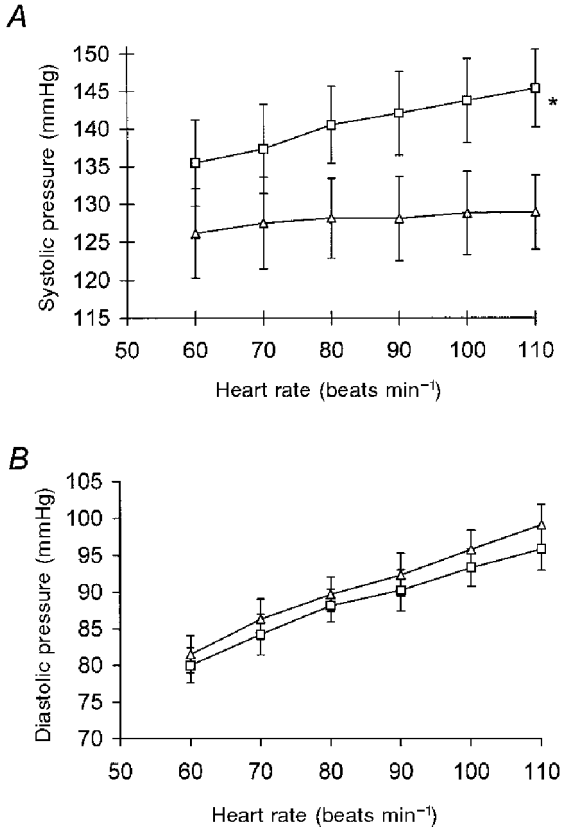

Figure 3. The effect of heart rate on peripheral and central blood pressure

A, changes in systolic blood pressure with increasing heart rate. *B*, changes in diastolic blood pressure with increasing heart rate. □, peripheral (brachial) arterial pressure; △, central (ascending aortic) arterial pressure. Values represent means ± S.E.M. ($n = 22$). *$P = 0·001$ (ANOVA).