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Prolonged atrio-ventricular conduction and aortic insufficiency

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The haemodynamic and electrocardiographic data of 10 patients with aortic insufficiency were reviewed. None of them had received a digitalis preparation, and all had a significantly longer P–R interval than 10 similarly studied normal patients (mean P–R 0·213 and 0·153 second respectively, $P<0.001$) thus corroborating previous reports of the association of atrio-ventricular conduction delay and aortic insufficiency.

Left ventricular end-diastolic pressure was approximately the same or higher than pulmonary artery systolic pressure in three patients and higher than pulmonary artery end-diastolic pressure in seven patients.

An analysis of the timing of atrio-ventricular events suggested that an earlier atrial systole (due to P–R prolongation) enabled an increment of forward flow which would otherwise be precluded by the premature closure of the mitral valve associated with aortic insufficiency. Due to the advantageous timing, left atrial and therefore right heart pressures were lower, thus tending to protect the pulmonary vascular bed.

Patients with aortic insufficiency (AI) frequently have gross elevations of left ventricular end-diastolic pressure (LVEDP). They also experience premature closure of the mitral valve attributable to the aortic reflux (Welch, Braunwald, and Sarnoff, 1957; Colvez, Alhomme, Samson, and Guedon, 1959; Meadows, Van Praagh, Indreika, and Sharp, 1963; Rees, Epstein, Criley, and Ross, 1964). It has been observed that patients with AI may have a prolongation of the P–R interval (Segal, Harvey, and Hufnagel, 1956; Colvez *et al.*, 1959; Gordon, Kirschner, and Moscovitz, 1961). However, in spite of what is frequently a severe distortion of left heart dynamics, it is not uncommon to find normal or near normal left atrial and pulmonary artery pressures (Welch *et al.*, 1957). These observations suggest that there may be a protective or compensatory mechanism related to atrio-ventricular (A-V) conduction delay. The records of patients with AI were studied to determine the significance of premature mitral valve closure and its association, if any, with P–R interval prolongation.

METHODS

All patients in the study underwent simultaneous right and left heart catheterization with cineangiocardiography.

Group I comprised 10 patients with AI, 7 men and 3 women, their ages ranging from 26 to 52 years. The diagnosis of AI was based on gross regurgitation of contrast media into the left ventricle following injection into the aorta. None of the patients had ever received a digitalis preparation.

The patients in group II were the 10 normal studies obtained in the last 500 catheterizations in this laboratory. All had undergone evaluation because of a murmur or a radiographical abnormality. Following normal pressure-flow studies and normal angiography, these patients were adjudged to have no heart disease.

All pressures were measured with Statham p23db gauges and monitored with a 12-channel Electronics for Medicine recorder. The gauges were levelled to a common zero by means of a fluid-filled manifold to which they were all connected. The level of the fluid in the manifold was set to 5 cm. below the angle of Louis. All gauges were adjusted to equal sensitivity by imposing several known pressures into the common manifold.

All pressures reported were obtained by averaging the actual measurements over two respiratory cycles.

RESULTS

Table I presents the haemodynamic and electrocardiographic data of the AI patients. Patients 3, 4 and 5 had pulmonary artery–left ventricular end-diastolic pressure relationships (PA-LVEDP),

TABLE I

AORTIC INSUFFICIENCY PATIENTS (GROUP I)

Patient	Age	Sex	P–R Interval (sec.)	Heart Rate Beats/min.	Pulmonary Artery Pressure (mm. Hg)	Left Ventricular Pressure (mm. Hg)
1	45	M	0·20	88	48/30	136/54
2	38	M	0·28	83	52/27	152/56
3	36	M	0·19	68	24/11	137/11
4	26	F	0·22	78	29/10	135/10
5	27	F	0·20	88	30/13	156/13
6	41	M	0·22	80	21/8	150/15
7	41	F	0·20	78	33/17	154/21
8	41	M	0·20	60	30/13	114/20
9	52	M	0·20	72	44/24	184/40
10	42	M	0·22	77	22/12	157/17
	Mean		0·213	77·2		

as normally expected. That is, there was some degree of equilibration of these pressures at end-diastole (Hamilton, Woodbury, and Vogt, 1939; Shaw, 1963; Kaltman, Herbert, Conroy, and Kossmann, 1966). The remaining patients had PAEDP considerably lower than the LVEDP (Wigle and Labrosse, 1965). Three patients (1, 2 and 9) had pulmonary artery systolic pressures which were approximately the same or even below the LVEDP. The comparisons of the pulmonary artery systolic and diastolic pressures and the LVEDP are shown in Figure 1. The simultaneous

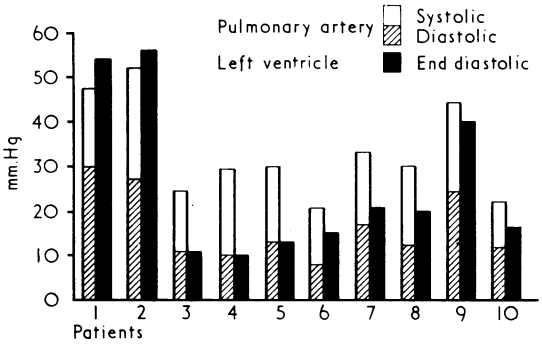

FIG. 1. *Relationships of pulmonary artery systolic and diastolic pressures to left ventricular end-diastolic pressure in each of the group I (AI) patients.*

pulmonary artery and left ventricular pressure trace, as recorded in patient 1, is seen in Figure 2. The solid arrow indicates the point at which the EDP is measured in the left ventricle. The similarity of the PAEDP and wedge EDP is indicated by the open arrow. The P–R interval of 0·20 second is seen. The left atrial and right heart pressures are maintained at levels well below the LVEDP (Welch *et al.*, 1957).

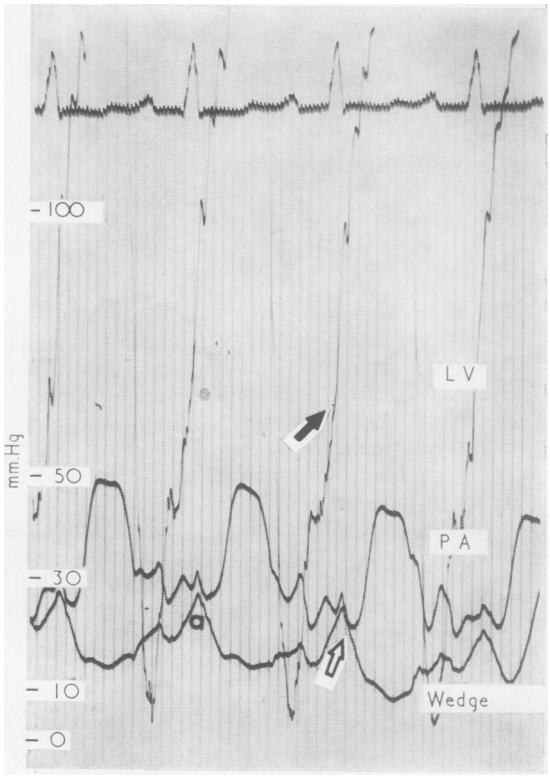

FIG. 2. *Simultaneous pressure traces recorded from patient* 1. *The left ventricular end-diastolic pressure is indicated by the solid arrow. The similarity of the end-diastolic point on the 'a' wave in the wedge reflection of the left atrium and that in the pulmonary artery (PA) is shown with the open arrow. LV=left ventricle; a=a wave. Time lines are* 0·04 *second.*

Table II lists the pertinent data of the normal group (II).

Figure 3 is a bar graph of the P–R intervals of the two groups. Those of group I are significantly

TABLE II

GROUP II PATIENTS WITHOUT HEART DISEASE

Patients	Age	Sex	Heart Rate Beats/min.	P–R Interval (sec.)
1	14	M	57	0·14
2	18	M	78	0·17
3	22	M	78	0·15
4	72	M	88	0·17
5	42	F	71	0·14
6	21	M	48	0·16
7	19	F	75	0·18
8	18	F	75	0·14
9	15	F	78	0·12
10	28	F	78	0·16
	Mean		72·6	0·153

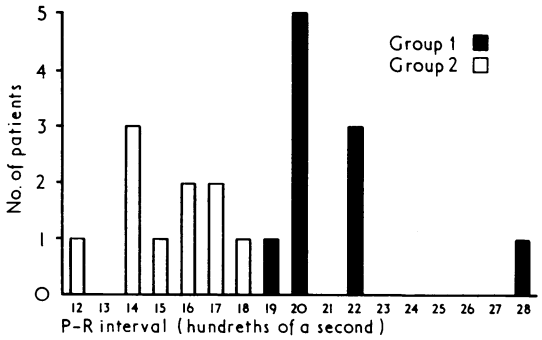

FIG. 3. *Comparison of the P–R intervals. Group I (AI) intervals are significantly longer than those of group II (normals) ($P<0.001$).*

longer than those of group II (mean values 0·213 and 0·153 second respectively; $P<0.001$).

The heart rates of the two groups did not differ significantly. Mean values for groups I and II were 77·2 and 72·6 beats/minute respectively ($P=0.4$–0.3).

DISCUSSION

Six of the 10 AI patients had P–R intervals within the 0·12–0·20 second range generally considered to be normal although these patients proved to be statistically different from the normal group ($P<0.001$; Fig. 3). The finding that these patients clustered at one extreme of the 'normal' range was the indication that there was some alteration in their atrio-ventricular conduction time. Further, it has recently been shown that the criteria used to establish the 'normal' range for the P–R interval are suspect and that an upper value of 0·18 second is a more realistic figure (Herbert and Sobol, 1970).

Many additional patients with AI were studied who also manifested the A–V conduction delay and the haemodynamic abnormalities above described. They were, however, receiving digitalis and were therefore excluded because of the known pharmacological effect of the drug on A–V conduction. This study then confirms previous reports of P–R prolongation in patients with AI (Segal *et al.*, 1956; Colvez *et al.*, 1959; Gordon *et al.*, 1961).

Since it was the primary purpose of this report to evaluate the haemodynamics, no attempt to determine the aetiology of the P–R prolongation was made, though we must emphasize that none of our patients had been given digitalis.

In an effort to evaluate the possible effect of A–V conduction delay, the PA–LV pressure relationships shown in Fig. 2 are replotted and magnified in Figure 4. In order to plot Fig. 4, it was necessary to consider corrective factors for two types of transmission delay. The first is the transmission delay of the wedge pressure trace through the pulmonary vascular bed. Braunwald, Fishman, and Cournand (1956), by direct puncture, determined that mechanical atrial systole commences 0·08 second after the onset of electrical systole. Line A (Fig. 4), representing the pressure generated by atrial systole, was therefore

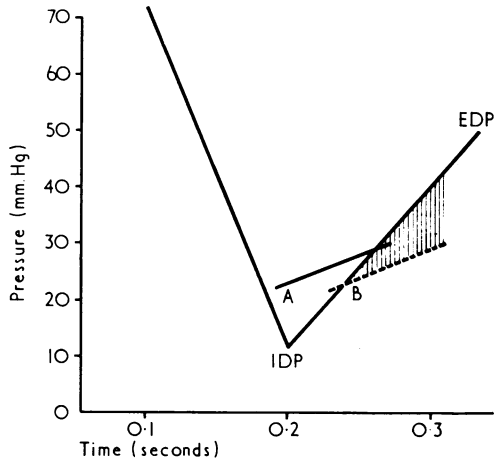

FIG. 4. *A graphic display of the pressure relationships seen in Figure 2. The ventricular pressure falls early in diastole to the initial diastolic pressure (IDP) and then rises rapidly to the end-diastolic pressure (EDP). Line A represents a left atrial pressure rise due to atrial systole from 22 to 30 mm. Hg when the P–R interval is 0·20 second. Line B demonstrates the relationship of this same atrial systole to the left ventricular pressure level were it to occur just 0·04 second later. The stippled area represents the increasing reverse gradient which would obstruct atrio-ventricular flow.*

corrected to reflect this timing. Its duration of 0·08 second (onset to peak) and elevation of 22 to 30 mm. Hg is unchanged. The second type of transmission delay is that of the catheter-manometer system itself. Laurens (1966) reports that, in the absence of air bubbles or fibrin, the delay is negligible, being only one or two milliseconds for a 125-cm. catheter. He further suggests that it is possible to superimpose the pressure trace from a standard external manometer over that recorded with a catheter-tip micromanometer. After checking the integrity of the recording system we therefore disregarded the negligible delay in

the recording of the left ventricular pressure curves but did correct the wedge reflection of left atrial events. We think that any residual error related to the recording system is of insufficient magnitude significantly to affect our observations.

The pressure course in the left ventricle (Fig. 4) from approximately the end of systole through diastole is represented. The pressure falls, as usual, precipitously to the point labelled IDP (initial diastolic pressure) but then rapidly rises due to the aortic reflux to the EDP (end-diastolic pressure). The initial atrial pressure rise (line A) occurs at a most opportune time: that point just preceding the lowest ventricular pressure. Dotted line B depicts the relationships which would obtain if the P–R interval were 0·16 second instead of 0·20 second. The rapid left ventricular pressure rise is already well advanced. The shaded area demonstrates the increasing pressure differential. Thus, an atrial systole occurring just 0·04 second later would be either ineffective, or, in order to produce an increment of forward flow, would require almost a doubling of the existing pressures. The figures are correct only for one particular heart rate. It should be noted, however, that the 0·04 second prolongation of the P–R interval would undoubtedly lose much of its advantage if the heart rate were significantly lower.

The left ventricle with an insufficient aortic valve dilates. End-diastolic volumes well in excess of 400 ml. have been reported (Dodge, Sandler, and Evans, 1960). The first defence is to provide a huge ejection fraction so that even after the volume of the regurgitant flow is subtracted, an adequate net forward flow remains. Since the reflux fills the left ventricle early, the mitral valve is prematurely closed (Welch *et al.*, 1957; Colvez *et al.*, 1959; Meadows *et al.*, 1963; Rees *et al.*, 1964). This severely limits diastole, and, with a normal P–R interval, precludes the atrial systolic flow as well (Figs 2 and 4). Diastole will in any event be foreshortened, but the prolonged P–R interval enables the left atrium to deliver its volume contribution earlier and thus at a time (at least in part) before left ventricular pressure has risen so precipitously.

The dual advantage, due to the early atrial contraction described above, occurs only in those AI patients whose LVEDP exceeds that in the left atrium. Firstly, it permits an increment of forward flow which otherwise would be precluded by premature mitral valve closure. Secondly, by occurring early and therefore at a lower pressure level, it appears to be a factor in enabling the left atrium and thus the right heart to operate at normal or relatively near normal pressure (Welch *et al.*, 1957). The intervening structures, primarily the pulmonary capillary bed, are protected from an increased hydrostatic pressure.

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