Atrial Transport and Aortic Insufficiency*

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The study of patients with aortic insufficiency has revealed that many demonstrate a first degree A-V block (Segal, Harvey, and Hufnagel, 1956; Colvez *et al.*, 1959; Gordon, Kirschner, and Moscovitz, 1961; Herbert, 1967). The possible advantages of this P-R prolongation have only recently been described (Herbert, 1967). It appears that such a mechanism enables the left atrium to contract earlier (relative to ventricular systole) and thus deliver its volume contribution to forward flow before the premature closure of the mitral valve (Colvez *et al.*, 1959; Meadows *et al.*, 1963; Rees *et al.*, 1964; Herbert, 1967). This also enables the left atrium, and thus the right heart as well, to function at a normal or nearly normal pressure level. In short, atrial transport (whose major components are the early rapid filling phase and atrial systole) occupies an abbreviated diastolic period due to the aortic reflux which prematurely closes the mitral valve.

It is the purpose of this paper to describe further, (a) the components of the prolonged P-R interval in aortic insufficiency, and (b) the manner in which atrial transport is affected.

SUBJECTS AND METHODS

Twenty-two patients were submitted to right and left heart catheterization with cine-angiocardiology.

The diagnosis of aortic insufficiency in 12 patients was based on angiographic demonstration of a grossly incompetent aortic valve. The other 10 patients were referred for evaluation of either an unusual murmur or a possible cardiac aberration suggested by routine chest films. Following a normal right and left heart catheterization these patients were adjudged to have no heart disease.

Lead II was selected for the electrocardiographic measurements as it proved to reflect in almost every instance the true P wave and P-R interval duration (Schlamowitz, 1946; Macruz, Perloff, and Case, 1958).

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Two of the patients with aortic insufficiency were receiving digitalis preparation. Neither was considered to be digitalis toxic. Early records in one of these patients (Case 5, *see* Table II), before any drug therapy, also demonstrated the high degree of A-V block.

The heart rates varied from 48 to 93 beats a minute, and were considered to be within a range which would not significantly affect the P-R interval (Schlamowitz, 1946; Macruz *et al.*, 1958).

RESULTS

The electrocardiographic data of the 10 normal patients appear in Table I. The mean P wave duration is 0.094 sec., conforming to that reported by Macruz *et al.* (1958) of 0.09 sec. The mean P-R interval is 0.153 sec. and does not vary significantly from the 0.143 value reported by Schlamowitz (1946).

The comparable data of the aortic insufficiency group appear in Table II. The P-R intervals and P waves are both considerably longer than in the control group. In Fig. 1, the P waves (shaded portions) and the P-R intervals (total bar height) of the aortic insufficiency group are compared to their counterparts of the normal group. The p value is > 0.001 in each instance.

TABLE I
NO HEART DISEASE

Patient No.	Age (yr.)	Sex	Heart rate	P-R interval	P wave
1	14	M	57	0.14	0.09
2	18	M	78	0.17	0.09
3	22	M	78	0.15	0.10
4	72	M	88	0.17	0.10
5	42	F	71	0.14	0.08
6	21	M	48	0.16	0.10
7	19	F	75	0.18	0.11
8	18	F	75	0.14	0.10
9	15	F	78	0.12	0.08
10	28	F	78	0.16	0.09
Total				1.53	0.94
Mean				0.153	0.094

TABLE II
AORTIC INSUFFICIENCY

Patient No.	Age (yr.)	Sex	Heart rate	P-R interval	P wave
1	51	M	75	0.19	0.15
2	27	F	88	0.22	0.13
3	26	F	88	0.20	0.14
4	36	M	75	0.17	0.11
5	38	M	83	0.28	0.12
6	45	M	93	0.22	0.14
7	41	M	88	0.24	0.14
8	41	M	75	0.23	0.12
9	39	F	65	0.18	0.12
10	22	M	68	0.20	0.14
11	41	F	78	0.20	0.13
12	54	M	83	0.22	0.15
Total				2.55	1.59
Mean				0.212	0.132

DISCUSSION

The P-R interval in patients with aortic insufficiency is not only prolonged, but contains within its length a broader P wave. The increase in P duration is apparently due to enlargement of the left atrium. As noted above, one of the effects of the prolonged P-R interval is that it enables the left atrium to function at a lower or even normal pressure level, thus eliminating an increased left atrial pressure as an aetiological factor in its enlargement. The reason for the increased left atrial chamber size

appears to be related to the unusual volume demands placed upon it. Fig. 2 represents the electrocardiograms which might be recorded from two hearts with equal rates, left atrial pressures, and outputs. The upper trace (designated AI) is drawn with a prolonged P-R interval. It is representative of the tracings of the aortic insufficiency group, with premature closure of the mitral valve. The lower (labelled normal) represents normal intervals and dynamics. The hatched area superimposed on each designates that period in the cardiac cycle during which the left atrium functions as a reservoir only, i.e., the period during which the mitral valve is closed. The stippled area incorporates the time lapse during which atrial transport takes place. The components of the latter are atrial systole and passive flow. When the mitral valve is open, the atrium, then open in both directions, acts as a conduit enabling blood returning from the lungs to pass through it without significantly altering its volume. Fig. 2 thus demonstrates why these two hearts with equal rates, left atrial pressures, and outputs *must* have different left atrial volumes. The left atrium of the patient with aortic insufficiency has a shortened conduit time, as compared to the normal, and therefore by definition its period of reservoir function is increased.

In summary, the period of collection or pooling

FIG. 1.—A bar graph representation of the comparison of the P-R intervals (total height) and the P waves (shaded areas) between the patients with aortic insufficiency and the normal groups. The patients are represented in the same order as they appear in the Tables. The time scale is in hundredths of a second. The p value is > 0.001 for both comparisons.

FIG. 2.—The scalar tracings as they might appear when recorded from two hearts with equal rates, outputs, and left atrial pressures. The upper trace represents the patient with aortic insufficiency, premature closure of the mitral valve, and a prolonged P-R interval. The lower represents the normal. The hatched areas designate that period during which the left atrium functions as a reservoir only, that is while the mitral valve is closed. The stippled area covers that portion of the cycle which includes atrial transport and during which the atrium is in effect a conduit. The patient with aortic insufficiency has the longer reservoir period and the shorter transport and conduit time.

in the chamber is increased, thus causing the left atrium to accumulate an increment of blood which the normal heart would allow to pass. The period during which this chamber can discharge the volume of forward flow is shortened, and, therefore, if this volume is to be the same as the normal heart the rate of flow must be increased. The increased fibre length secondary to the dilated chamber would provide such an acceleration of atrial ejection (Braunwald and Frahm, 1961).

The above suggests that a vital role is played by atrial systole in maintaining an adequate cardiac output in patients with atrial insufficiency, and that all efforts should be made to maintain these patients in sinus rhythm and first degree A-V block.

As described by Macruz *et al.* (1958) the A-V node is activated before the inscription of the terminal forces of the left atrium. Therefore, the P-R prolongation cannot, even in part, be attributed to the broadened P wave. Instead, the P wave must mask a portion of the prolonged P-R segment. Rather than invoke obscure changes in the decremental conduction (Hoffman and Cranefield, 1960) of the A-V node and bundle, it is certainly more attractive to consider that the delayed A-V conduction is due to nothing more than the massive dilatation and elongation of the left ventricle and the intraventricular septum, thus attributing the delay to the distance which the impulse must travel.

SUMMARY

Twelve patients with aortic insufficiency and 10 normal patients were studied by right and left heart

catheterization. Both the P waves and the P-R intervals of the group with aortic insufficiency were demonstrated to be significantly longer. The delayed A-V conduction in this group was attributed to the massive dilatation of the left ventricle. The broadened P wave may be explained on the basis of an enlarged left atrium due to the greater volume demands placed upon this chamber. These volume requirements stem from the premature closure of the mitral valve, which foreshortens the period during which the atrium functions as a conduit and prolongs the phase during which it acts as a reservoir. These studies suggest that with a P-R interval prolongation, atrial systole plays an important role in atrial transport in the presence of an insufficient aortic valve.

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