

# Atrioventricular Pressure Half-Time

## Measure of Mitral Valve Orifice Area

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### SUMMARY

In 20 patients studied by cardiac catheterization, the time required for the diastolic left atrioventricular pressure difference to fall to half its initial value (half-time) varied with the anatomic severity of the valvular stenosis. In mild mitral stenosis, the half-time was approximately 100 msec; in moderate stenosis, it was about 200 msec; and it was 300 msec or longer in severe stenosis. The half-time at the nonstenotic mitral valve was less than 25 msec.

Although exercise increased the atrioventricular pressure gradient, heart rate, and cardiac output significantly, the half-time was affected only slightly. Mitral regurgitation of various severities did not affect the half-time. The half-time appears to vary inversely with the anatomic orifice area of the mitral valve. The determination of the half-time obviates the need for the commonly used exercise test for the evaluation of mitral stenosis.

### Additional Indexing Words:

Starr-Edwards prosthesis      Cineangiography      Mitral regurgitation      Exercise

**I**N THE resting subject, the rate of decline of the diastolic atrioventricular pressure gradient varies with the anatomic area of the mitral valve orifice.<sup>1</sup> A measure of the rate of decline of this pressure gradient is the half-time interval, during which the pressure difference falls to half of its initial value. We have shown that the severity of mitral valve stenosis varies with the half-time. The present study examines the usefulness of the half-time as a measure of the severity of mitral valve stenosis in the presence of exercise and of mitral regurgitation.

### Materials

Mitral valve disease was diagnosed in the 20 patients of this study by clinical examination, electrocardiogram, chest x-ray, and cardiac catheterization findings. The diagnosis was confirmed at surgery in six patients. Two patients were catheterized 1 year after replacement of the

mitral valve with a Starr-Edwards prosthesis (table 1).

### Methods

Following intramuscular meperidine (50 mg) and promethazine (25 mg), left atrial pressure was recorded by transeptal cardiac catheterization.<sup>2</sup> Retrograde catheterization through the right brachial artery provided simultaneous recordings of the left ventricular pressure. The recording system in the 0 to 200 mm Hg range was flat, up to 10 cycles per second. Cardiac output at rest was measured by the Fick principle and by the dye-dilution forward-triangle method.<sup>3</sup> Exercise, performed by left leg pumping against an air pump, was continued until the heart rate increased by 20 or more beats per minute. During exercise the cardiac output was determined by dye dilution.

Left atrial and left ventricular pressures were recorded simultaneously. The half-time, that is, the time interval during which the atrioventricular pressure gradient fell to half its initial value, was calculated from these tracings (figs. 1 and 2). An alternative method was a plot of the transmitral pressure gradient on a logarithmic scale against time; this semilogarithmic plot produced a nearly straight line (fig. 3). The half-times found by the two methods were almost identical.

The severity of mitral stenosis, determined on the basis of clinical, catheterization, or surgical

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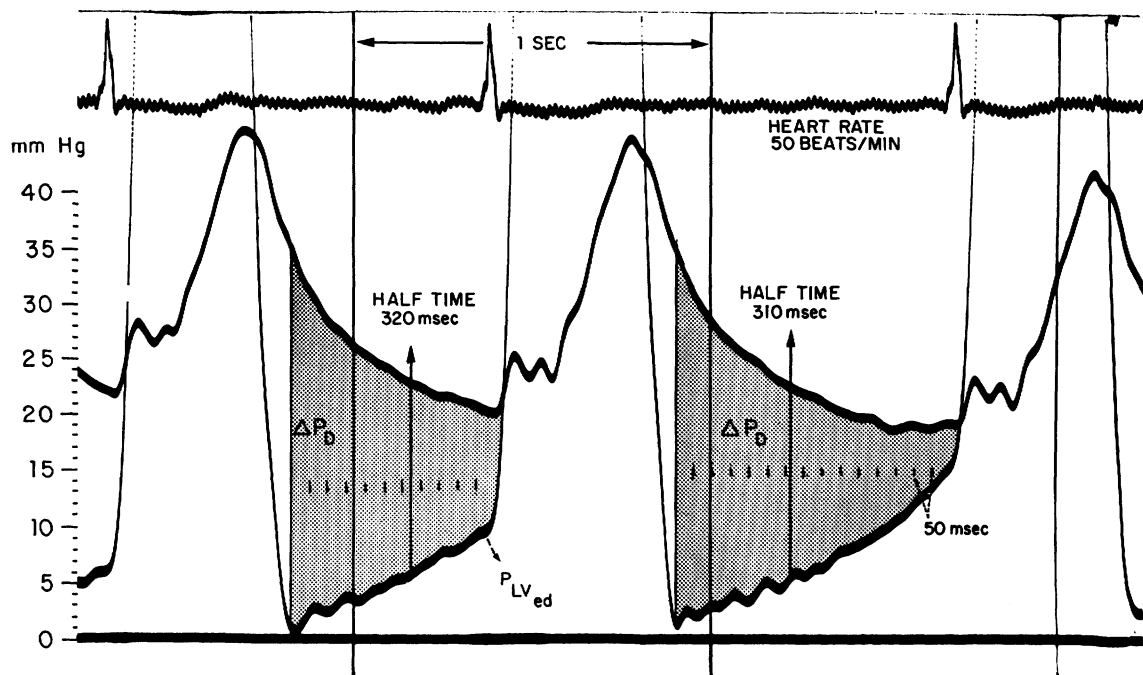


Figure 1

Simultaneous left atrial and ventricular diastolic pressures in a patient with severe mitral stenosis at rest. The horizontal axis is time. The original recording speed was 100 mm/second. The diastolic pressure difference starting at the onset of the rise in ventricular pressure is measured at 50-msec intervals (short vertical lines). The shaded area shows the changing atrioventricular pressure gradient. Symbols:  $P_{LV_{ed}}$ , end-diastolic left ventricular pressure;  $\Delta P_D$ , atrioventricular diastolic pressure difference.

findings, was classified as zero when no stenosis was present and as four, when the stenosis was considered to be severe (table 1).

Mitral regurgitation was evaluated after injection of radiopaque dye into the left ventricle, on the basis of the increase and persistence in the cineangiographic radiodensity of the left atrium. In grade-I regurgitation, dye passing from the left ventricle slightly increased the radiopacity of the left atrium, but the atrium emptied within a beat or two. Grade-II regurgitation was characterized by a mild to moderate increase in left atrial density, which was dissipated after a few beats. A moderate to marked increase in left atrial density, which was washed away only after a series of beats, was classified as grade III regurgitation. Severe regurgitation (grade IV) was diagnosed when the regurgitant increase in left atrial density was marked and persisted for several beats.

### Results

At rest, the atrioventricular pressure half-

time varied from 20 msec when no stenosis was present to 340 msec in a patient with severe mitral stenosis (table 1).

Exercise was used to increase the heart rate by 20 or more beats per minute (table 1). The resultant shortening of the diastolic filling interval was usually associated with lowering of the end-diastolic ventricular pressure as compared to that recorded while the patient was at rest. The left atrial pressure and the transmitral pressure gradient increased significantly during exercise in patients with dominant mitral stenosis, but remained unchanged in three of the five patients with dominant mitral regurgitation. Statistical analysis showed the half-times obtained during exercise to be slightly shorter than those obtained at rest. The mean difference was  $6.5\% \pm 7.7\%$  (SD). These results

Table 1

Hemodynamic Data of the Patients in the Present Study

Case	Mitral lesion*		Left atrial pressure (mm Hg)		Mean	Left ventricular diastolic pressure (mm Hg)		Cardiac output (L/min)		A-V pressure half-time (msec)	
	Stenosis	Regurg.	Rest	Exercise		Rest	Exercise	Rest	Exercise	Rest	Exercise
1	0	2	10/0	4	4	0-5	0-5	5.0	7.0	20	20
2	0	4	20/5	10	15	0-5	2-8	3.4	4.0	25	25
3	1	0	19/5	9	13	4-10	4-10	4.1	6.0	70	60
4	1	3	55/16	25	25	10-14	8-13	3.7	5.2	80	70
P <sub>5</sub>	1	1	14/6	9	14	0-8	0-8	6.0	9.0	85	85
P <sub>6</sub>	1	1	18/10	12	21	6-11	10-14	5.5	8.5	100	90
S <sub>7</sub>	1	4	50/18	25	27	5-11	2-10	3.5	4.2	110	110
8	2	1	20/13	16	25	0-14	0-12	4.2	5.0	140	150
9	2	0	22/13	16	26	2-13	5-10	4.5	6.9	150	120
10	2	1	30/15	20	26	5-10	5-10	3.0	4.3	160	140
11	2	0	20/14	16	24	5-12	4-10	4.0	4.8	160	150
12	2	1	26/11	16	23	2-10	2-7	4.7	7.5	180	150
S <sub>13</sub>	2	1	25/12	16	22	0-10	0-5	4.0	4.6	180	180
14	3	3	15/8	10	21	0-5	0-5	4.5	5.5	190	150

15	3	2	$\frac{6}{9}$	9	$\frac{17}{15}$	17	0-5	0-5	3.8	4.7	200	190
16	3	1	$\frac{20}{14}$	16	$\frac{30}{15}$	21	4-10	0-8	3.6	5.6	200	200
S <sub>17</sub>	3	1	$\frac{30}{15}$	20	$\frac{36}{18}$	34	0-6	0-6	3.0	4.6	290	280
S <sub>18</sub>	4	1	$\frac{43}{20}$	27	$\frac{58}{28}$	37	0-12	0-7	3.8	5.7	315	300
S <sub>19</sub>	4	0	$\frac{35}{15}$	21	$\frac{40}{20}$	27	0-5	0-5	3.4	4.3	330	280
S <sub>20</sub>	4	1	$\frac{45}{22}$	30	$\frac{53}{32}$	40	0-6	0-5	3.0	4.0	330	340

P = Postoperative Starr valve prosthesis.

S = Diagnosis confirmed at surgery.

\*Severity of stenosis or regurgitation: 0 = none; 1 = barely noticeable; 2 = mild; 3 = moderate; and 4 = severe.

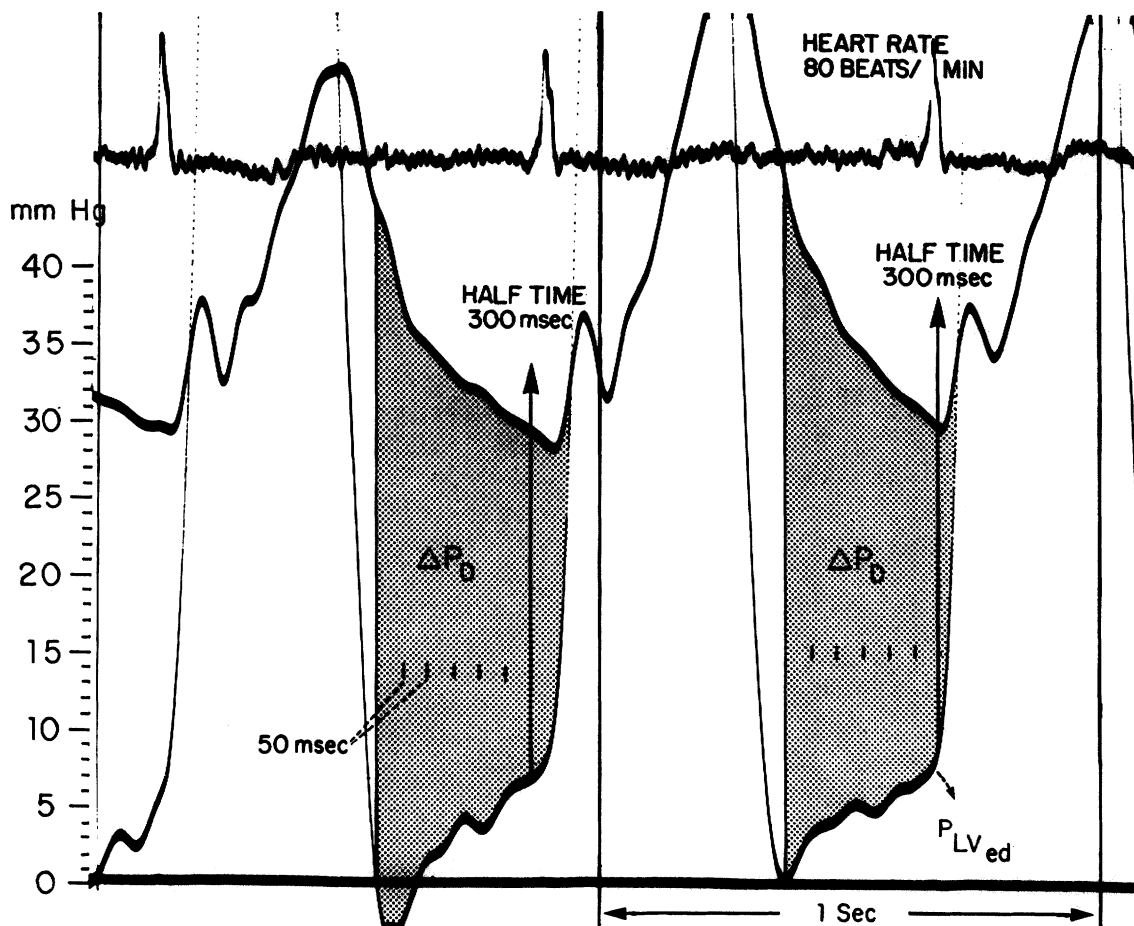
did not differ significantly from those expected on the basis of chance alone (probability less than 0.5). The largest absolute change observed in the half-time was from 330 msec at rest to 280 msec during exercise, a variation of 15% (table 1, case 19). The largest percentage variation was 18% (table 1, case 14).

The simultaneous resting diastolic left atrial and ventricular pressures in case 18 of table 1 are shown in figure 1, and during exercise in figure 2. The left atrial mean pressure increased 37%, from 27 mm Hg at rest to 37 mm Hg during exercise. Cardiac output increased 58%, from 3.8 to 5.7 L per minute, and the heart rate increased 60%, from 50 to 80 beats per minute, indicating a decrease in stroke volume from 76 to 71 ml. Although the measurements given above changed significantly on exercise, the average half-time decreased only 5%, from 315 msec at rest to 300 msec during exercise.

A semilogarithmic plot of temporal changes in the A-V pressure difference at rest and during exercise in case 18 is shown in figure 3. At rest, the initial diastolic pressure difference in two successive beats was 33 and 34 mm Hg; the half-time was 300 msec in the first beat at a pressure difference of 16.5 mm Hg and 330 msec in the second beat at a pressure difference of 17 mm Hg. During exercise the A-V pressure difference at the onset of diastole increased to 44 and 42 mm Hg, but the half-time of these values remained at 300 msec.

Figure 4 compares the half-times at rest and during exercise. Each patient is represented on the graph according to the severity of the regurgitation as indicated by values in the section on "Methods," ranging between 0 (no regurgitation) to 4 (marked regurgitation). The values for 15 of the 20 patients fall on or very near the line of equality, showing the half-time to be relatively unaffected by exercise.

A plot of the varying R-R intervals and heart rate due to atrial fibrillation against the half-time in 11 consecutive beats during



**Figure 2**

*Simultaneous left atrial and ventricular diastolic pressures during exercise in the same patient as in figure 1.*

rest and 14 consecutive beats during exercise (table 1, case 10) is given in figure 5. At rest, the R-R intervals varied from 0.7 to 2.0 sec; the half-time remained relatively unaffected, averaging  $160 \pm 10$  msec. During exercise, the R-R interval shortened as the heart rate increased, but the average half-time was decreased 13%, to an average of  $140 \pm 25$  msec.

#### Discussion

The rate at which the falling left atrial pressure and the rising left ventricular pressure approach a common value is a function of the anatomic area of the mitral orifice. The normal adult mitral valve orifice of about 4

cm<sup>2</sup> permits atrioventricular flow to reduce the transmitral pressure difference to negligible values in less than 50 msec after the onset of the diastolic rise in left ventricular pressure.<sup>1</sup> In mitral stenosis the atrial pressure falls slowly and an atrioventricular pressure gradient may be evident even at the onset of the succeeding beat. However, since the gradient may persist, even in mild stenosis when the filling interval is brief, or may disappear in severe mitral stenosis when the diastolic interval is prolonged, the end diastolic atrioventricular pressure gradient cannot be used as a consistent guide for the evaluation of the severity of mitral stenosis.

The Gorlin formula<sup>4</sup> is commonly reported

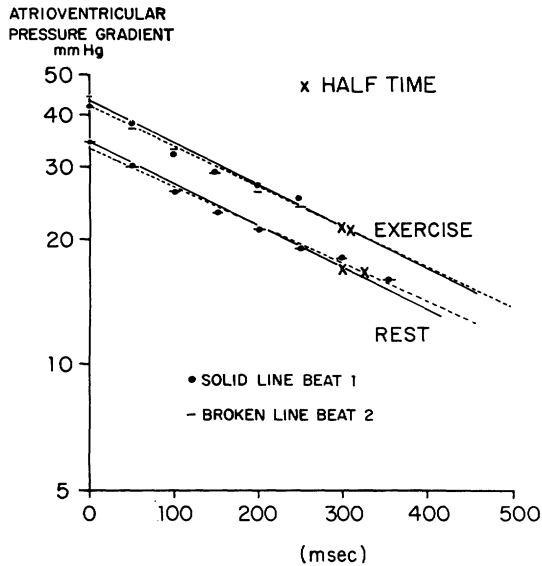


Figure 3

The half-time at rest and at exercise for the beats shown in figures 1 and 2. The vertical axis gives the atrioventricular pressure difference in mm Hg on a logarithmic scale. The horizontal axis is time beginning with the diastolic ventricular pressure rise.

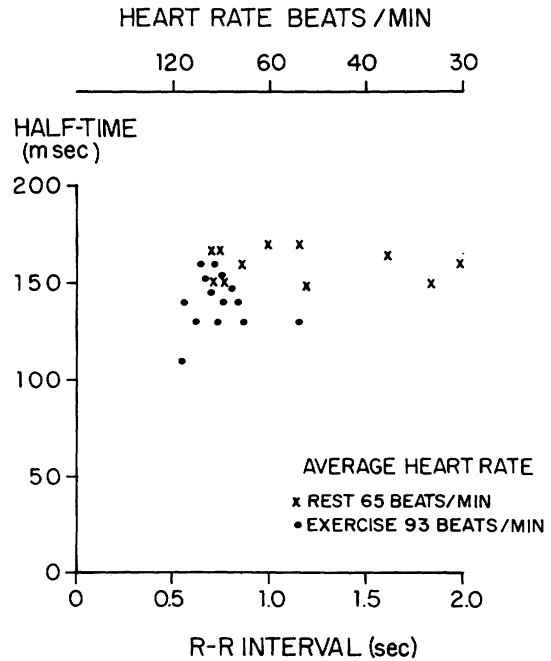


Figure 5

A plot of 11 beats during rest (solid circles) and 14 beats during exercise (+) in a patient with moderate mitral stenosis, mild mitral regurgitation, and atrial fibrillation. Horizontal axis shows the R-R interval in seconds and the heart rate. The vertical axis is half-time in milliseconds.

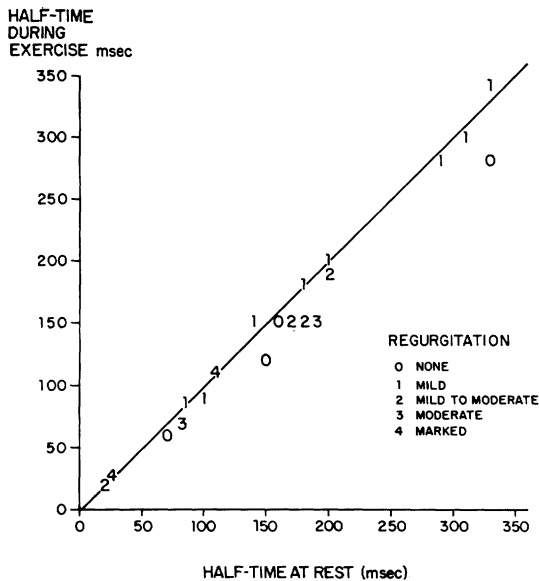


Figure 4

A comparison of the half-time at rest and at exercise. The horizontal axis is the half-time at rest; the vertical axis shows the half-time during exercise. The symbols 0 to 4 represent the severity of mitral regurgitation as determined by left ventricular cineangiography in each case. The line of equality is shown.

to reflect the degree of mitral valve stenosis; exercise or mitral regurgitation complicate the analysis.<sup>5</sup>

The present results show that the half-time of the decline of the atrioventricular pressure difference is a consistent estimator of the severity of valvular narrowing, even when exercise or mitral regurgitation change other factors significantly. Thus, the left atrioventricular pressure difference can vary greatly with changes in heart rate and cardiac output. Exercise lowered the diastolic left ventricular pressure while increasing the heart rate, cardiac output, left atrial pressure, and the atrioventricular pressure difference. Despite these large changes, the atrioventricular half-time is changed to a much smaller degree. In normal subjects, the atrioventricular pressure declined to half its initial value in less than 25 msec.<sup>1</sup> In mild mitral stenosis, the average half-time was prolonged to about



100 msec. In moderate mitral stenosis, the half-time was about 200 msec, and in severe stenosis, the half-time was 300 msec or more. It would thus appear that the data obtained at rest are sufficient for the evaluation of the severity of mitral stenosis and that the additional data obtained during exercise add little further information regarding the valve area. It is also noted that the half-time utilizes only the dimensions of pressure and time, and does not require the measurement of cardiac or stroke output.

In a previous study we showed that the extent of the physical disability produced by a stenotic lesion varied with the ratio of the half-time of the A-V pressure decline to the cardiac index.<sup>1</sup> This ratio evaluates the adequacy of the valve orifice with respect to the metabolic and vascular demands of the individual. Thus, a given absolute orifice area adequate for a small child may be inadequate for a large active man.

The half-times in the two patients with Starr-Edwards mitral prosthesis were somewhat prolonged, indicating that these prosthetic valves may offer a slight obstruction to atrioventricular flow<sup>6</sup> as in mild mitral stenosis, even though the apparent valve area of the prosthesis is of normal value. The energy losses at the valve may result from the impedance to flow of the axial stream, that is, the streamlines that normally would flow at the greatest velocity. Energy losses, which are stenosis equivalents, also result from vibrations of the ball valves during filling, and by turbulence induced at those

regions of separation of the stream on the downstream surface of the balls.

Although mitral regurgitation may produce an elevation of the initial diastolic atrioventricular pressure gradient, the half-time is relatively unaffected and therefore the severity of the stenosis is not masked.

It is concluded that the half-time of the atrioventricular pressure gradient offers a direct, easily determined means for the evaluation of mitral stenosis, even in the presence of complicating mitral regurgitation, or of a hypermetabolic state such as exercise.

### References

1. LIBANOFF, A. J., AND RODBARD, S.: Evaluation of the severity of mitral stenosis and regurgitation. *Circulation* 33: 218, 1966.
2. ROSS, J., JR.: Catheterization of the left heart through the interatrial septum: A new technique and its experimental evaluation. *Surg Forum* 9: 297, 1958.
3. WARNER, H. R., AND WOOD, E. H.: Simplified calculation of cardiac output from dye dilution curves recorded by oximeter. *J Appl Physiol* 5: 111, 1952.
4. GORLIN, R., AND GORLIN, M. E.: Hydraulic formula for calculation of the area of the stenotic mitral valve, other cardiac valves, and central circulatory shunts. *Amer Heart J* 41: 1, 1951.
5. WERKO, L.: Dynamics and consequences of stenosis or insufficiency of the cardiac valves. *In Handbook of Physiology, Section 2: Circulation, vol. 1*, edited by W. F. Hamilton and P. Dow. Washington, D. C., American Physiological Society, 1962, p. 645.
6. MORROW, A. G., HARRISON, D. C., ROSS, J., JR., BRAUNWALD, N. S., AND CLARK, W. D.: Surgical management of mitral valve disease: Symposium on diagnostic methods, operative techniques and results. *Ann Intern Med* 60: 1073, 1964.



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