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Duration of cardiac systole in children with femoral arteriovenous fistulas

James B. Bassingthwaighe, M.D., Ph.D.* and **Howard B. Burchell, M.D., Ph.D.****
Rochester, Minn.

Femoral arteriovenous fistulas have been produced surgically in children in attempts to increase leg length.¹ This results in decreased peripheral resistance and increased cardiac output²—a biventricular volume overload. Some cardiac enlargement was observed in these children, but it always disappeared when the fistula was closed.³ The volume overload might affect the two ventricles differently because the left ventricular myocardial fiber arrangement and chamber shape are best designed for pressure work while the pocket-shaped right ventricle should be better equipped to handle increased volumes.⁴ Clinical observations by one of us (H.B.B.) led to the suspicion that the splitting of the second sound might be abnormally wide, which might occur if right ventricular systole was more prolonged by the overload than was left ventricular systole. Estimation of the duration of systole for each ventricle was undertaken in these children to assess differences either between the two ventricles or between these and normal. The wide range of heart rates occurring within a few minutes in a child at rest could be extended by changing the flow through the fistula,^{2, 5, 6} and thereby presented an opportunity for observing the effects of heart rate on systolic duration.

Methods

We studied six children in whom femoral arteriovenous fistulas had been created in order to increase leg length (Table I). Although minor electrocardiographic changes were seen,³ in none was there a definite electrocardiographic abnormality.

Heart sounds were recorded from the left second or third intercostal space at the left sternal border, whichever position provided optimal observations of the two components of the second sound. A simultaneous recording was also made at the apex when the recording from the pulmonary area did not show the first heart sounds sufficiently well. Recording was by means of a direct-writing phonocardiograph (Elema Mingograph) having a frequency response flat to approximately 600 cycles per second (Hz). A medium-frequency band-pass filter selected frequencies of 100 to 300 Hz; higher frequencies (200 to 500 Hz) also could be recorded selectively from the same microphone. Paper speed was 100 mm. per second. The electrocardiogram was recorded simultaneously from the lead (usually Lead I or Lead II) in which the onset of the QRS was observed to be the earliest. Recordings were made while the patient was lying, sitting, and standing and, in one case, while the patient was at various angles of tilt from 0 to 80 degrees head up. Recordings were made not earlier than

Reprint requests to: J. B. Bassingthwaighe, M.D., Mayo Clinic, Rochester, Minn. 55901..

*Consultant, Department of Physiology, Mayo Clinic; Associate Professor of Physiology, Mayo Graduate School of Medicine (University of Minnesota), Rochester, Minn. Recipient of National Institutes of Health Career Development Award.

**Emeritus Consultant, Department of Internal Medicine, Mayo Clinic and Mayo Foundation; Professor of Medicine, University of Minnesota, Minneapolis, Minn.

With the technical assistance of Elizabeth Brekke.

60 seconds after a change in position. The effect of closure of the fistula in each of these positions was studied.

The following intervals were measured: (1) R-R, the time between the peaks of the R waves, to represent the cycle length; (2) Q-S₁, the time from the onset of the QRS complex to the first major high-frequency deflection of the first heart sound (we did not attempt to distinguish mitral and tricuspid components of the first sound); (3) Q-A₂, the time from the onset of the QRS complex to the first major high-frequency component of the second heart sound; (4) S₁-A₂, measured directly and also obtained as the difference between Q-A₂ and Q-S₁ to check the accuracy of measurement; and (5) A₂-P₂, the time from the first major high-frequency deflection of the first component to the similar deflection of the second component of the second heart sound.

S₁-A₂ may be considered as the duration of left ventricular systole, that is, the isometric contraction period plus the left ventricular ejection period, ending with aortic valve closure. S₁-P₂, obtained by addition of S₁-A₂ and A₂-P₂, can be defined as right ventricular systole although less accurately, because, in addition to the isometric and ejection phases ending with pulmonary valve closure, it also includes the time between mitral closure and tricuspid closure. This error should be fairly constant and quite small, approximately 0.01 second.

Respiration was recorded by means of a thermocouple in front of the nose and mouth.

The arteriovenous fistula was closed by digital pressure on the femoral artery over the head of the femur as gently as possible so as not to cause pain. Fistula flow was thought to be stopped when either no murmur was recorded from a microphone at the site of the fistula or no thrill or pulse could be detected.

Results

A typical recording is shown in Fig. 1; this patient (Patient 1) had slightly longer A₂-P₂ than did some of the others. This clarity of tracing with absence of base-line noise was usually attainable. Estimates of the various intervals for this same patient are shown in Fig. 2. With closure of the fistula, there was a dramatic increase in the cycle length followed by slower increases in left ventricular systole (S₁-A₂), right ventricular systole (S₁-P₂), and Q-A₂. There were no consistent changes in Q-S₁. The duration of splitting of the second sound during respiration was unchanged in the supine position but was decreased by closure of the fistula when the patient was sitting or standing (Table II). There were 198 cycles measured on this patient, and 167, 141, 177, 134, and 269 measured on the others, for a total of 1,076 cycles and 5,380 observations in this phase of the study.

In both the control situation and when the fistula was closed, a respiratory effect on duration of cycle length was seen. The changes of S₁-A₂ and Q-A₂ were in phase with those seen in R-R, while changes in A₂-P₂ were in the opposite phase and of low amplitude. The respiratory rate remained constant, an apparent increase in frequency (top two lines, Fig. 2) with closure of the fistula being due entirely to the decrease in heart rate and the fact that the abscissa is the number of cardiac contractions from the beginning of the control period.

Systole increased in duration within a few heart beats after the fistula was closed, remained approximately steady during the period of closure, and returned to control levels several seconds after the fistula was reopened in all the patients studied. The change in systolic duration was much less abrupt than was the onset of bradycardia with closure and was comparable to the rate of recovery of R-R when the fistula was reopened. When the patients were supine, A₂-P₂ were broad, and their constancy with opening or closing of the fistula showed that the changes in right and left ventricular systolic durations were essentially

similar. With one exception, the decrease in heart rate with closure was most marked when the subject was supine.

The relationship between systole and cycle length during open-fistula and closed-fistula measurements is shown in Fig. 3 for Patient 5. The relationships of $Q-A_2$, S_1-P_2 , and S_1-A_2 to the duration of the preceding R-R interval were apparently linear. Closure of the fistula extended the relationship to a region of longer cycle length without inducing a change in either slope or intercept. The cycle lengths were longer during expiration, as one might expect in young persons with sinus arrhythmia. The longest R-R immediately followed closure of the fistula. The duration of right ventricular systole (S_1-P_2) was significantly prolonged during inspiration ($p < 0.001$ by the chi square test). There was no statistically demonstrable respiratory influence on left ventricular systole, although the plot of S_1-A_2 in Fig. 2 suggests that changes directionally similar to but smaller than those in S_1-P_2 may be present. The regression lines for $Q-A_2$, S_1-P_2 , and S_1-A_2 are approximately parallel and indicate that the durations of electrical and mechanical and of right and left ventricular systoles were affected more or less equally; as a result, there was little change in either $Q-S_1$ or A_2-P_2 in all six patients in the supine position.

Linear relationships similar to those of Fig. 3 were found whether the patient was lying, sitting, standing, or at various angles on a tilt table. However, in all patients in the sitting and standing positions and in some (as in Fig. 3) in the supine position, the slope of the S_1-A_2 regression line was slightly steeper than that of the S_1-P_2 . This suggests that A_2-P_2 shortens at longer cycle lengths. This tendency was seen in each subject; it is likely that the effects of respiratory phase on ventricular filling and systolic intervals outweigh the effects of cycle length. The mean A_2-P_2 interval was slightly diminished also by closure of the fistula. When the data from all patients were used (Table II), the shortening of A_2-P_2 was statistically significant and occurred in every patient in the sitting and standing positions but was not consistent in the supine position.

The regression equations of all the data on S_1-A_2 of the six patients are presented in Table III and Fig. 4. For each patient, the regression line for the sitting position was between the lines for the other two positions; the slope and intercept values were approximately the average of those for supine and standing. Certain trends can be seen in the data. In each patient, and in the combined data from all patients, the intercept of the regression lines decreased and the slope increased as the patient was tested in a more upright position. The differences in systolic duration with position were large at all heart rates but were less at slow than at fast heart rates.

The data for S_1-P_2 and $Q-A_2$ were similar, the same trends existing as for S_1-A_2 .

The differences in systolic duration with body position were observed at all heart rates; in Fig. 5 they are shown for a specific rate. These data points are the intersections of the regression lines of Table III with a vertical line at $R-R = 0.60$ second. This rate (100 beats per minute) was chosen for illustration because R-R intervals of about 0.6 second were observed at all body positions. Therefore, an average S_1-A_2 at an R-R of 0.6 second could be calculated for each subject without extrapolating from the data represented by the regression equations of Table III. On the shift from supine to standing, systole shortened by 0.029 to 0.047 second (mean, 0.037 second), a highly significant change.

The relationship between body position and duration of systole was studied in more detail in one patient (Patient 3). The data were recorded with the patient supine on a table tilted head up from 0 to 80 degrees in 10 degree steps. For each level of tilt, a regression line. obtained to represent the relationship between S_1-A_2 and cycle length. The value of S_1-A_2 at a heart rate of 120 beats per minute was obtained from this line. The results (Fig. 6) show a more or

less linear relationship between the duration of systole and the angle of tilt. The linearity of the relationship was not improved by plotting the interval against the Sine of the angle of tilt.

Discussion

The earliest observations on the cardiovascular responses to the closure of an arteriovenous fistula were made by Nicoladoni⁵ in Vienna. His kymographic records of the carotid pulse showed that, as the fistulous arm was compressed, the heart slowed, the blood pressure increased, and systole became longer. Detailed observations on the duration of systole have been lacking although data concerning cardiac output, stroke volume, and blood volume have been accumulated over the years.^{2, 7}

The method of measurement of the duration of systole used in this study was recommended 57 years ago by Lewis.⁸ Among the first to take advantage of it were Wiggers and Katz⁹ who used it to study the effects of cardiac sympathetic nerve stimulation in dogs. Despite the simplicity of the measurements, we have found only one reported set of observations on the relationship between systolic duration and cycle length in an individual subject, that by White and Mudd.¹⁰ Previous workers, such as Lombard and Cope,¹¹ Cheer and Li,¹² Fridericia,¹³ Katz and Feil,¹⁴ Adams,¹⁵ and Weissler and associates,¹⁶ reported the average duration of systole and the average cycle length for a number of beats in each subject rather than relating systolic duration to cycle length of each beat.

In our study, systolic duration was related to the duration of the preceding R-R because it was during that interval that ventricular filling occurred. It logically might be argued that one should relate systole not to R-R but to the S₂-S₂ during which systole occurred, as was done by Katz and Feil.¹⁴ This would account for the filling period, would avoid including the previous systole which might have less direct influence on the present systolic length, and would relate systolic length to the cycle which is subject to the same influences of neural impulses, peripheral resistance, or homeometric regulation as the ventricular contraction itself. This was not done because R-R can be measured slightly more accurately than S₂-S₂ and the influences of variation in ejection time and of emptying during the preceding beat would be partially compensated for.

The relationship of systolic length to cycle length in our individual patients was little different from the relationships observed by Lombard and Cope¹¹ in groups of people at different average heart rates; their results for the supine position are illustrated in Fig. 7. They obtained the length of systole from the carotid sphygmogram as the time from the beginning of the rise of the pulse to the nadir of the dicrotic notch; systolic and cycle lengths were averaged over 15 cycles in each of 252 male and female subjects. At heart rates higher than 120 beats per minute, systole was shorter than indicated by their regression lines. The consistently slightly greater length of systole for our subjects compared with those of Lombard and Cope¹¹ may be partly explained by the tendency of estimates of systolic length from the peripheral pulse tracing to be less than those from sound tracings, which may be due to the slower transmission velocity of the foot of the peripheral arterial pulse than the peak.²¹ Even so, they found, as we did, that linear regressions are more appropriate than functions of the square root of cycle length.^{12, 14, 19, 20} In Fig. 7, Cheer and Li's equations¹² have been translated to linear form. However, for the data acquired by these and other observers and summarized by the plots of Fig. 7, as well as for our data, there is so much scatter that, over the range of cycle lengths from 0.5 to 1.0 second, the choice of type of equation is arbitrary.

The lack of respiratory influence on the systole-cycle length relationship is interesting. It suggests that sinus arrhythmia occurs wholly by influence on the atrial pacemaker (mediated principally via the right vagus nerve) and is unaccompanied by changes in sympathetic outflow that might be expected to shorten systole more than was observed. In fact, if there were a direct inotropic effect accompanying each respiratory increase in rate, then the slope of systole versus cycle length should be steeper than that seen for the average of groups of subjects, but the actual observation is that the slope is much less. The inotropic effects of a rate increase per se begin within a few beats but are not fully evident for six to eight beats.²²

On the other hand, the extent of the influence of increased sympathetic response to elevation of the upper part of the body is shown by the clear-cut shortening of systole with sitting or standing (Table III; Figs. 4 and 5). The slope of systole versus cycle length in each individual and for the group is also steeper in the standing than in the supine position. Cheer and Li¹² observed no difference in the slope between supine and sitting positions, but Lombard and Cope¹¹ noted increases at least as large as ours (for women standing they found $S = 0.157C + 0.137$ and for men standing, $S = 0.150C + 0.124$). In our subjects there was a decrease of about 0.02 second in S_1-A_2 on shifting from supine to sitting and the same for shifting from sitting to standing. Lombard and Cope¹¹ observed the difference to be about 0.025 second, which may be explained by a diminution of mean blood pressure in the upright position and relatively slower transmission of the foot of the carotid pulse compared to the incisura. However, the difference between the studies is practically insignificant. In our patient who was tilted (Fig. 6) the decrease at a cycle length of 0.50 second was greater than 0.05 second in tilting from horizontal to 80 degrees.

The splitting of the second heart sound provides a fairly precise estimate of the time delay between aortic and pulmonic valve closures and some indication of relative length of systole of the two ventricles. With closure of the fistula there are increases in both aortic pulse pressure and mean pressure² which tend to decrease the systolic ejection fraction and promote early aortic closure, but apparently, with diminution of venous return, the right ventricular systole is shortened even more, resulting in the observed diminution of the splitting. In the horizontal position, the cycle length is already prolonged and A_2-P_2 does not shorten significantly.

The mean A_2-P_2 intervals in our patients were only slightly longer than in normal adults²³ but the variation was wide and the splitting usually remained detectable throughout the cardiac cycle. Persistent splitting is common in children, and we conclude that the dynamics of cardiac ejection in these children were quite similar to those of normal children. The decreased peripheral resistance and the continuously increased cardiac output had the same cardiac effect as daily athletic training—increased vagal tone, decreased resting heart rate for the same cardiac output, and mild cardiac enlargement which diminished on termination of the daily exercise or closure of the fistula. Fistulas of even several years' duration apparently had no detrimental effects on the cardiovascular system although slight cardiac enlargement was apparent in some of these children. The increase in A_2-P_2 with sitting and standing is contrary to the observations in normal subjects by Romeo and colleagues²⁴ and Breen and Reke²⁵ and to our usual clinical experience. The former group observed diminution in 12 subjects with change from lying to sitting and no change in 10 subjects. The latter investigators observed persistent splitting in subjects lying down but not in those sitting or standing, except in patients with complete right or left bundle-branch block, pulmonary stenosis, or ventricular septal defect. In our subjects this was evident only with the fistula open, suggesting that the unusually high venous return caused relative prolongation of right ventricular systole. This factor would presumably play a role at all levels of sympathetic tone.

The proposed mechanism of the variation in splitting is the same in these patients as in normal subjects and follows the arguments of Boyer and Chisholm²⁶: With expiration, venous return to the right ventricle is reduced and S_1 - P_2 is shortened, left atrial and left ventricular filling is promoted by increased alveolar pressure, and the enlarged stroke volume is accompanied by lengthening of S_1 - A_2 . Thus, both the lengthening of S_1 - A_2 and the relative shortening of S_1 - P_2 , which occur in spite of the lengthening of the R-R intervals, contribute to a shorter A_2 - P_2 (Table II). With inspiration, the volume load of the right ventricle exceeds that of the left ventricle, and A_2 - P_2 broadens.

S_1 - A_2 appears to be more sensitive to changes in posture than Q - A_2 . Stafford and associates²⁷ observed Q - S_1 to increase by over 30 msec. with tilting to vertical of 15 normal subjects but, nevertheless, Q - A_2 diminished slightly.

Increased sympathetic tone is a possible cause of the shortening of both right and left systolic durations in upright posture.⁹ Lombard and Cope¹¹ observed systole to be 0.049 second shorter at a 40 degree (head up) tilt than in the supine position and 0.022 second longer at a -15 degree (head down) tilt. In our patients the shortening was only 0.04 second on changing from supine to standing at R-R = 0.60 second and the difference would be less at slower heart rates. Our observations were that tilting from 0 to 40 degrees head up produced little more change than simply sitting up. At slow heart rates the effect of position is small compared to that at high rates. Although Lombard and Cope's data for the sitting and standing positions showed shorter systoles and slightly steeper regression lines than ours, the degree of change of the systole-cycle-length relationship on going from supine to more upright was similar in the two studies, despite the difference in methods and despite the important fact that their data were from groups of adults and ours from individual children.

Summary

Intervals related to right and left ventricular systoles were measured from phonocardiograms and electrocardiograms in six children who had surgically induced femoral arteriovenous fistulas but otherwise were normal. Heart rates varied widely because of respiratory variation; the range was further extended to slow rates by manual closure of the fistula. In each subject, whether supine, sitting, or standing, the relationship between the systolic interval and cycle length was approximately linear. The relationship between systole and cycle length observed over a wide range of heart rates for each child (or for the whole group) was remarkably similar to the corresponding relationships obtained in studies, by other investigators, of large numbers of subjects each of whom was observed over only a narrow span of heart rates. At a given heart rate, the systole intervals in each subject decreased with change from supine to sitting to standing or with increasing degrees of tilt from horizontal to upright.

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Fig. 1. Recording from Patient 1 while fistula was open. Respiration (line 2) was determined by thermocouple. Sounds were recorded from second left intercostal space with medium-frequency filter (line 3) and high-frequency filter (line 4). Splitting of second sound (A_2-P_2) increased with inspiration as in normal subjects.

Fig. 2. Interval durations before, during, and after release of digital compression of femoral artery in Patient 1. Closure reduced cardiac output and resulted in sudden cardiac slowing (prolonged R-R) and also in definite increases in systolic intervals ($Q-A_2$, S_1-P_2 , and S_1-A_2). Right ventricular systole was prolonged as much as left ventricular systole. A_2-P_2 did not change.

Fig. 3. Duration of systolic intervals over wide range of heart rates in Patient 5 in supine position. Broken lines separate data points representing different intervals. In regression equation, y is systolic duration and x is R-R.

Fig. 4.
Influence of cycle length on S_1 - A_2 in six patients in three body positions. Thin lines represent regression equations for individual patients and are comparable to lowest line of Fig. 3. Thick lines represent regression equations for all six patients together in each position.

Fig. 5.
Effect of body position on left ventricular systolic duration at heart rate of 100 beats per minute ($R-R = 0.60$ sec).

Fig. 6.
Influence of head-up body tilting on duration of left ventricular systole at a constant heart rate of 120 beats per minute.

Fig. 7.

Relationships of systolic length to cycle length in humans in supine position as reported by several investigators. Slopes of QT interval versus cycle length, C , tended to be steeper than slopes of mechanical systole, S , versus cycle length, whether these were obtained for wide range of heart rates in individual patients (present study) or at constant heart rates in large groups (all other studies). Data are: 1, Adams, 15 $QT = 0.146C + 0.257$; 2, Kuhns, 17 average QT intervals of groups; 3, Fridericia, 13 $QT = 0.0822 \sqrt[3]{100C}$; 4, Ashman, 18 $QT = 0.375 \log_{16}[10(C + 0.07)]$; 5, Cheer and Li, 12 women, $QT = 0.208C + 0.18$ and men, $QT = 0.16C + 0.207$; 6, Weissler and associates 16 ($S = S_1 - S_2$), women, $S = 0.16C + 0.205$ and men, $S = 0.18C + 0.168$; 7, Bazett 19 ($S = QT$), $S = 0.363 \sqrt{C}$; 8, Graser and Mehl, 20 $QT = 0.356 \sqrt{C}$; 9, Lombard and Cope 11 (S from carotid pulse), women, $S = 0.065C + 0.248$ and men, $S = 0.105C + 0.201$; 10, Katz and Feil 14 (S from sounds and subclavian pulse), $S = 0.31 \sqrt{C}$.

Table 1

Patients studied

Patients	Age; sex	Weight (lb.)	Height (in.)	Duration of fistula (mo.)	Cardiac enlargement (grade [*])
1	15; M	122	65	71	1
2	10; M	69	54	30	1
3	12; F	75	59	25	1-2
4	14; M	94	62	84	2
5	11; M	82	59	63	2
6	9; F			64	1 [†]

^{*} On basis of 1 to 6.

[†] Left ventricle.

$$A_2\text{-}P_2 \text{ (and R-R) intervals (sec.} \times 10^{-2}\text{) in six children with arteriovenous fistulas}$$

Position of patient	Fistula open				Fistula closed			
	Insp.* Mean $\pm$ S.D.	N	Exp. [†] Mean $\pm$ S.D.	Both Mean $\pm$ S.D.	Insp.* Mean $\pm$ S.D.	N	Exp. [†] Mean $\pm$ S.D.	Both Mean $\pm$ S.D.
Supine	3.98 $\pm$ 0.78 (3.8)	104	2.81 $\pm$ 0.78 [‡] (71.2 $\pm$ 5.6)	3.38 $\pm$ 0.77 (70.0 $\pm$ 5.0)	3.93 $\pm$ 0.96 (87.1 $\pm$ 6.0)	62	2.84 $\pm$ 0.83 [‡] (93.0 $\pm$ 9.4)	3.38 $\pm$ 1.0 (90.1 $\pm$ 9.4)
Sitting	4.58 $\pm$ 0.84 (5.2)	106	3.22 $\pm$ 0.91 [‡] (64.5 $\pm$ 6.8)	3.86 $\pm$ 1.11 (63.5 $\pm$ 6.1)	3.93 $\pm$ 1.17 [§] (78.9 $\pm$ 8.2)	62	2.44 $\pm$ 0.82 [‡] (85.9 $\pm$ 17.8)	3.18 $\pm$ 1.22 [§] (82.5 $\pm$ 14.5)
Standing	4.00 $\pm$ 0.96 (57.7 $\pm$ 4.8)	111	3.45 $\pm$ 0.92 [‡] (57.2 $\pm$ 6.3)	3.72 $\pm$ 0.98 (57.0 $\pm$ 5.3)	3.54 $\pm$ 1.0 [§] (72.8 $\pm$ 7.5)	66	3.01 $\pm$ 0.92 [‡] (75.2 $\pm$ 13.9)	3.28 $\pm$ 1.00 [§] (74.0 $\pm$ 8.9)

§ For difference from open fistula (same phase of respiration), $p < 0.005$.

Table III

Regression equations for data from six patients: $S_1 - A_2 = a(R - R) + b$

Patient	Supine			Sitting			Standing		
	a	b*	r	a	b*	r	a	b*	r
1	0.065	25.9	0.71	0.103	21.6	0.68	0.114	18.7	0.85
2	0.056	25.9	0.70	0.082	21.0	0.59	0.097	20.0	0.72
3	0.064	24.4	0.61	0.120	19.4	0.71	0.140	15.1	0.84
4	0.051	28.0	0.56	0.094	22.7	0.80	0.097	21.3	0.84
5	0.093	23.3	0.73	0.083	22.9	0.70	0.101	19.6	0.77
6	0.070	26.0	0.78	0.077	24.8	0.69	0.074	22.9	0.53
Mean	0.068	25.3	0.77	0.093	21.9	0.75	0.104	19.3	0.79
N		348			376			406	

* In seconds $\times 10^{-2}$.