Ventricular Dynamics in Atrial Fibrillation

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During atrial fibrillation there are beat-to-beat changes of ventricular rate, end-diastolic volume, end-diastolic pressure, and arterial pressure or resistance to ventricular ejection. The effect of these variables on left ventricular dynamics in man has been approached by studying single sequences of beats in patients with atrial fibrillation. An index of changes of left ventricular volume has been determined from the electromyogram of left cardiac border. Left ventricular pressures have been measured at surgery or by the transbranchial technic. These studies provide a better understanding of the hemodynamics of the left ventricle during atrial fibrillation and demonstrate another approach to the study of left ventricular function in man.

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R-R interval and duration of ventricular diastole, show considerable beat-to-beat variation for any given R-R interval or period of diastole within a single sequence of beats. A number of mechanisms have been suggested to explain why variations in the duration of just the preceding R-R interval or period of ventricular diastole do not adequately explain the beat-to-beat hemodynamic variations in this arrhythmia. Einthoven and Korteweg observed that the amplitude of a beat was determined not only by the preceding R-R interval, but also by the amplitude of the preceding beat. Lewis, using the cardiometer in experimental animals with atrial fibrillation, showed that the variations of ventricular end-diastolic volume were not determined solely by the variations of the immediately preceding R-R interval. Wiggers in studies of left ventricular pressures in experimental animals with this arrhythmia has shown that the rate of ejection and the ejection period do not depend on initial tension, or ventricular end-diastolic pressure, as in the heart with normal rhythm.

As a consequence of the ventricular arrhythmia of atrial fibrillation, there are beat-to-beat variations of ventricular rate, volume of ventricular filling and ejection, and arterial pressure or resistance to ventricular ejection. The influence of these variables on beat-to-beat ventricular function has been studied. The study forms a basis for a better understanding of the arterial pressure variations as observed in this arrhythmia. In addition, such studies make it possible to utilize the beat-to-beat changes of ventricular end-diastolic volume and resistance to ejection secondary to an ar-
rhythmia as another approach to the study of ventricular function in man.

**METHODS**

Studies have been performed on a total of 25 patients with atrial fibrillation, both with and without heart failure. The majority of patients had rheumatic heart disease with mitral stenosis and some had mitral insufficiency as well. One patient had myocarditis and severe left ventricular failure. Another had atrial fibrillation of undetermined etiology without other evidence of heart disease. Some subjects were and some were not receiving digitalis at the time of these studies.

Observations were recorded on a Sanborn Polyvisor direct-writing, 4-channel recorder. The observations included left heart border electrokymograms (EKY), carotid pulse, arterial pressure, and heart sounds. Durations of systole and diastole were determined from the heart sound recordings. Arterial pressures were measured directly from a brachial artery through a no. 18 arterial needle connected to strain gage (Statham P-23-A). Carotid pulses were recorded by means of a cup placed over a carotid artery as described by Henny, Boone, and Chamberlain, but modified for the direct-writing instrument by connecting the carotid cup to a crystal pickup. The carotid pulse recording was used for purposes of timing the EKY.

An index of changes in left ventricular volume was determined from the left heart border EKY in 6 patients. Studies on 4 patients were performed with an EKY that was a resistor-condensor coupling network with an approximately 1-second time constant. In all currently available EKY units, the curves are modified by the decay characteristics of the unit, particularly when the heart rate is slow. Because of this difficulty a new type of direct-coupled EKY that does not have these decay problems was developed by Boone and his associates. Studies on 2 patients with atrial fibrillation were performed with this unit. The results with these 2 types were qualitatively similar for the purposes of this study.

The resemblance of the ventricular border EKY curves to ventricular volume curves obtained on experimental animals has been noted by others. However, it is well recognized that the ventricular EKY is influenced not only by changes in ventricular volume, but also by shifts in heart position and changes in the shape of the heart. Ring and associates have used the EKY as a densogram to quantitate heart volume changes in experimental animals and man and have noted that the human heart has much smaller positional changes than the canine heart. In this present study EKY's were recorded from the left heart border and hence record border excursion rather than changes in density as in previous studies because patients with cardiac enlargement show such small changes of ventricular density during the cardiac cycle that the EKY requires additional amplification, which introduces serious technical problems, and because many of the patients had an enlarged left atrium that was superimposed on the left ventricle in the A-P projection, hence introducing an error in the ventricular densograms. The EKY curves in these studies were not used to determine absolute volumes, but to determine a relative index of ventricular volume change.

EKY's were recorded from the left heart border with the patient facing directly anterior and tilted head-up 45° to 60°. The left heart border was explored by the EKY to find an area that showed little or no inward or outward motion during isometric contraction or isometric relaxation, as such excursions are a result of changes of heart shape or position and do not reflect volume changes. Such an area was usually found in the region of the mid left heart border. In a few patients, especially those with marked cardiac enlargement, no area could be discovered that showed little or no inward or outward motion during isometric contraction or relaxation. Curves from such patients and curves showing paradoxical motion were not suitable for the type of analysis used in this study. The patients suspended respirations during the recording period. EKY curves that satisfied these criteria were used to indicate the beat-to-beat changes in left ventricular end-diastolic and end-systolic volumes. An arbitrary baseline was selected, and the beat-to-beat variations of end-diastolic and end-systolic volume

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**FIG. 1.** An example of data recorded. On the electrokymographic record an arbitrary baseline is drawn. The beat-to-beat variation in amplitude of the ventricular electrokymographic excursion above or below this baseline during end-diastole and end-systole is used as an index of the beat to beat changes of left ventricular end-diastolic and end-systolic volumes. Amplitudes are expressed as mm. of deflection.
were expressed in terms of arbitrary units above or below the baseline (fig. 1).

To test to what extent these changes in the EKY excursions reflect changes in left ventricular volume, an independent method for measuring ventricular volume is necessary. Such is not available in man or even in the experimental animal. However, there is an indirect way to test the validity of the method: other workers have demonstrated that there is a relation between stroke volume and pulse pressure. 34-38 This relationship is also influenced by the distensibility of the components of the arterial tree and the volume of the arterial tree. 39 However, these other factors influencing the relationship between pulse pressure and stroke volume probably become less important under the conditions of this study where observations are limited to single sequences of beats and where, in the majority of cases, the arterial diastolic pressure variations did not exceed 10 to 15 mm Hg. Accordingly, the beat-to-beat arterial pulse pressure changes have been considered to reflect stroke volume changes. If the systolic EKY excursions are related to stroke volume, there should be a relation between the amplitude of these excursions and the variations of arterial pulse pressure. In the EKY curves that satisfied the previous criteria, the variations in amplitude of the systolic EKY excursions prove to have a linear relation to the arterial pulse pressure variations as can be seen in figure 2. It was then concluded that in these records the beat-to-beat variations of the EKY excursions have a quantitative relation to beat-to-beat variations of left ventricular end-diastolic volume, end-systolic volume, and stroke volume. No attempt has been made to quantitate the data in terms of absolute volumes or absolute volume changes.

Pressure work per beat or stroke work was calculated in the following manner. Stroke work is the product of the stroke volume and mean arterial pressure during ventricular ejection. The beat-to-beat changes of systolic EKY excursions were used as quantitative indices of stroke volume changes. Mean arterial pressures during ventricular ejection were calculated from the brachial artery pressure curves. Stroke work is then a relative value and equal to the product of the systolic EKY excursion and the mean arterial pressure during ventricular ejection. Analyses were made from observations on single sequences of beats. It is not possible to compare separate observations on the same subject or different subjects by this method.

Left ventricular pressures were obtained through the cooperation of Dr. A. G. Morrow in 1 of 2 ways, directly from the left ventricle at surgery by means of a needle connection to a strain gage (Statham P-23-D) through a length of rigid plastic catheter and recorded directly on a Sanborn Polyviso direct-writing recorder (this system has a damping ratio of 0.3 and is flat to within 10 per cent at 3 c.p.s.), and by a polythene catheter inserted into the left ventricle by the bronchoscopic technic with pressures recorded by means of a strain gage (Statham P-23-D) connected to a direct-writing Sanborn Polyviso recorder. 39 (This system has a resonant peak at 17 c.p.s. and is flat to within 10 per cent at 8 c.p.s.) The frequency response characteristics of both these recording systems is not of a high order. Accordingly, the results of this portion of the study were confirmed in a limited number of observations in which left ventricular pressures were measured with a no. 18 thin-walled needle connected directly to a Statham P-23-D strain gage. This recording system has a flat frequency response to 60 c.p.s. 40

RESULTS

Arterial Pressure

One of the features of atrial fibrillation is the irregular ventricular rhythm, which is associated with irregularity of both rate and amplitude of the arterial pulse. The relation of the beat-to-beat variations of arterial pulse pressure and systolic pressure to the variations in the duration of the immediately preceding R-R interval has been determined in 10 cases. In each case 50 to 100 successive beats were analyzed and plotted as in figure 3. As can be seen in the figure, the amplitude of both pulse pressure and systolic pressure bears a direct relation to the duration of the preceding R-R interval. However, there is a wide scatter of both for a given R-R interval. This scatter is most marked in the range of the shorter R-R intervals, in particular shorter than 0.4 to 0.6 second, and was found whether or not the patient had evidence of heart disease other than atrial fibrillation.

One of the factors accounting for the varying

![Fig. 2. Systolic electrokymographic excursions (ordinate) related to brachial artery systolic pulse pressures (abscissa) during a single sequence of beats.](image-url)
amplitude of pulse pressure or systolic pressure for a given R-R interval is the nature of the preceding beat. This is also illustrated in figure 3 where for each point the pulse pressure amplitude of the preceding beat is indicated by a symbol. It was found that for a given R-R interval, the amplitude of both pulse pressure and systolic pressure was inversely related to the amplitude of pulse pressure of the preceding beat, as has been noted previously by both Einthoven and Lewis.\textsuperscript{14, 22}

In 5 subjects left ventricular pressures were determined either by catheterizing the left ventricle by the bronchoscopic technic or with a needle inserted directly into the left ventricle at surgery. The left ventricular systolic pressures showed considerable beat-to-beat variation for a given R-R interval similar to that observed in the brachial arterial pressures. In 1 subject simultaneous aortic and left intraventricular pressures were recorded. Left ventricular systolic pressure for a given R-R interval was shown to be inversely related to the pulse pressure of the immediately preceding beat as measured in the aorta. From these 2 types of studies it is concluded that the peripheral arterial pressures as recorded in the brachial artery reflect the central pressure variations. In summary, the beat-to-beat arterial systolic pressure and pulse pressure variations in atrial fibrillation not only bear a direct relation to duration of the preceding R-R interval, but also an inverse relation to the pulse pressure of the preceding beat. The role of the preceding beat in determining the arterial pressure variations for a given R-R interval is clarified by the study of ventricular dynamics in this arrhythmia.

**Ventricular Dynamics**

Ventricular dynamics in atrial fibrillation have been studied by determining the beat-to-beat variations of the durations of systole and diastole, left ventricular end-diastolic volume, end-systolic volume, and stroke volume, and left ventricular systolic and end-diastolic pressures.

**Durations of Systole and Diastole.** One of the mechanisms by which the preceding beat could influence arterial pressure for a given R-R interval is through its effect on the duration of ventricular diastole or the ventricular filling period. It has been shown by Katz and Feil,\textsuperscript{23} and confirmed in this study, that there are beat-to-beat changes in the duration of ventricular systole in this arrhythmia. This is illustrated in figure 4, where it can be seen that the beats with the larger arterial pulse pressures have systoles of longer duration than do those beats with the smaller arterial pulse pressures. Because of this, beats with the same R-R interval may have varying periods of diastole depending on the duration of systole of the immediately preceding beat, and the R-R interval changes do not accurately reflect the variations in the duration of ventricular diastole. For the same R-R interval, the duration of diastole will be shorter if the immediately preceding beat has a large pulse pressure. In 6 subjects the beat-to-beat arterial pulse pressure and arterial systolic pressure were plotted against the duration of the immediately preceding diastole as determined from simulta-
neously recorded heart sounds. In each case a series of more than 50 consecutive beats was studied. As can be seen in figure 5, the results were similar to those in which the R-R intervals were plotted against arterial pressure. The beat-to-beat arterial pressure variations still bear a direct relation to the duration of the preceding diastole and an inverse relation to the pulse pressure amplitude of the preceding beat. It was concluded that the preceding beat has an effect in addition to its effect on the duration of diastole. Further clarification of the mechanism whereby the preceding beat influences the arterial pressure variations for a given R-R interval depends upon an examination of the ventricular volume changes in this arrhythmia.

Ventricular "Volume" Studies. Left heart border electrokymogram curves that were suitable for the type of analysis used in these studies were obtained from 6 subjects: 2 with mitral stenosis, 2 with postoperative mitral stenosis, 1 with mitral insufficiency, and 1 with atrial fibrillation without other evidence of heart disease. The results were qualitatively similar regardless of the underlying heart disease. The limitations of the methods do not permit quantitative comparison of results in these groups of patients.

An example of the type of data recorded is illustrated in figure 1, where the striking

![Figure 4](image-url)  
**Fig. 4.** Duration of ventricular systole (abscissa) as determined from heart sounds related to the arterial systolic pulse pressure (ordinate).

![Figure 5](image-url)  
**Fig. 5.** Brachial arterial pulse pressure and systolic pressure related to the duration of the immediately preceding diastole determined from heart sounds. For each point the arterial pulse pressure of the immediately preceding beat is indicated by a symbol.

beat-to-beat variation in the amplitude of the ventricular excursions in atrial fibrillation can be seen. The variations in amplitude of these excursions were used to indicate the beat-to-beat changes of left ventricular end-diastolic volume, end-systolic volume, and stroke volume as described earlier. Those beats with the larger arterial pulse pressures arose from the larger left ventricular end-diastolic volumes. The ineffective or small pulse pressure beats
arose from the smaller left ventricular end-diastolic volumes. This relation can be seen in figure 6, where the arterial pressure changes are related to changes of left ventricular end-diastolic volume. In addition, the left ventricle does not empty to the same degree with each beat in atrial fibrillation. Indeed, the beat-to-beat variations of ventricular end-systolic volume may be as marked as the variations of ventricular end-diastolic volume. Those beats having larger end-diastolic volumes have larger stroke volumes and more complete ejection than in the case of beats with the smaller end-diastolic volumes. This can be seen in the electrokymograph curve illustrated in figure 1.

These beat-to-beat variations of ventricular end-systolic volume become important in a consideration of the factors that determine the beat-to-beat variations of ventricular end-diastolic volume. The ventricular end-diastolic volume of any 1 beat is a function of the volume of ventricular inflow during diastole and the ventricular volume from which filling was initiated, which of course is the end-systolic volume of the preceding beat. The relative volume of ventricular inflow during diastole is to a large extent determined by the duration of diastole. As was shown earlier, the duration of diastole is a function of both the duration of the R-R interval and the duration of systole of the immediately preceding beat. As a result of the beat-to-beat variations of ventricular end-systolic volume in this arrhythmia, ventricular filling is initiated at varying ventricular volumes. Consequently, the ventricular end-diastolic volume shows considerable beat-to-beat variation for a given volume of ventricular inflow. Because of these considerations there may be wide variations of ventricular end-diastolic volume for a given R-R interval within a single sequence of beats in this arrhythmia. This can be seen in figure 7 where the beat-to-beat changes of ventricular end-diastolic volume are plotted against the duration of the immediately preceding R-R interval.

This analysis of factors that control the beat-to-beat variations of ventricular end-diastolic volume helps to explain the inverse effect of pulse pressure amplitude of the immediately preceding beat on the beat-to-beat arterial pressure changes in this arrhythmia. It was shown that the arterial pressure changes are closely related to the changes of ventricular end-diastolic volume. When the preceding beat generates a large pulse pressure, it is a beat initiated from a relatively large ventricular end-diastolic volume, has more complete emptying or a smaller ventricular end-systolic volume, and has a longer duration of systole. Following these beats ventricular filling is initiated at relatively smaller ventricular volumes. The longer duration of systole of these large beats relatively shortens the duration of ventricular filling for any given R-R interval. As a result of these factors ventricular end-diastolic volume for a given R-R interval is relatively smaller following the large pulse pressure beats.
**Left Ventricular Pressures.** Others have shown that during a regular ventricular rhythm changes of ventricular end-diastolic volume are associated with changes of ventricular end-diastolic pressure. Because of the problems inherent in ventricular volume studies some investigators have used changes of ventricular end-diastolic pressure as an index of ventricular end-diastolic volume changes. In the experimental animal with a regular ventricular rhythm, Wiggers has shown that increases of ventricular end-diastolic pressure and volume are associated with an increase in the rate of ventricular ejection and an increase in the maximum ventricular systolic pressure. If these relationships exist in the human subject with atrial fibrillation, the left ventricular systolic pressures and rates of left ventricular pressure rise should be directly related to the left ventricular end-diastolic pressure variations.

Left ventricular pressures have been recorded in 5 cases, and the systolic pressure of each beat compared with the ventricular end-diastolic pressure that immediately preceded. In atrial fibrillation the left ventricular systolic pressure variations are not directly related to the left ventricular end-diastolic pressure variations (fig. 8). Beat number 7 of W.V. and beats number 5 and 6 of G.S. show slower rates of ventricular pressure rise and relatively low ventricular systolic pressures, although these beats are initiated from relatively high ventricular end-diastolic pressures when compared with the other beats. Each of these beats is preceded by a short R-R interval and by a beat with a relatively high intraventricular systolic pressure. This indicates that the preceding beat had a relatively large stroke volume and a relatively small end-systolic volume. Accordingly, the ventricular volume prior to ventricular filling was relatively small. Because of the short R-R interval there was little time for ventricular filling; therefore, ventricular end-diastolic volume was relatively small. Yet these beats are associated with relatively elevated ventricular end-diastolic pressures. From this it is concluded that in atrial fibrillation, left ventricular end-diastolic pressure changes are not a measure of left ventricular end-diastolic volume changes. In other words there are beat-to-beat changes in the relationship between the so-called initial tension and initial volume.

**Ventricular Function**

Ventricular function in this arrhythmia has been studied by expressing ventricular function in terms of pressure work per beat or stroke work. An index of the beat-to-beat changes of stroke work was calculated in 6 patients by the method described earlier. This is an expression of the beat-to-beat changes of both stroke volume and arterial pressure or resistance to ejection that are a consequence of the arrhythmia. In each of the subjects stroke work
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was directly related to the ventricular end-diastolic volume and not closely related to the duration of the preceding R-R interval. Figure 9 shows the relation of the duration of the preceding R-R interval to stroke work. A wide scatter can be seen, particularly in the range of the shorter R-R intervals. Figure 10 shows the close relationship between ventricular end-diastolic volume and stroke work. These studies indicate that the left ventricular stroke work in human subjects with atrial fibrillation is determined by the left ventricular end-diastolic volume.

**DISCUSSION**

In subjects with atrial fibrillation the beat-to-beat changes of left ventricular end-diastolic volume and arterial pressure, although related to the duration of the immediately preceding R-R interval, show considerable scatter for any given R-R interval within a single sequence of beats. These studies have made it possible to explain this scatter, which in effect represents a dissociation between the immediate rate changes and these hemodynamic phenomena.

The ventricular end-diastolic volume for any given R-R in atrial fibrillation is a function of the ventricular volume from which filling is initiated and the volume of blood that flows into the ventricle during diastole. The ventricular volume from which filling is initiated is determined by the end-systolic volume of the preceding beat. The beat-to-beat variations of ventricular end-systolic volume may be as

![Figure 9](image)

**Fig. 9.** Duration of the immediately preceding R-R interval *(ordinate)* related to “stroke work” *(abscissa)*.

![Figure 10](image)

**Fig. 10.** Left ventricular end-diastolic “volume” *(ordinate)* related to “stroke work” *(abscissa)*.
marked as the variations of ventricular end-diastolic volume in this arrhythmia. Thus for a given R-R interval ventricular filling may be initiated at differing ventricular volumes. The volume of blood that flows into the ventricle during diastole is determined by the duration of diastole and the rate of ventricular filling. The duration of diastole is a function of both the duration of the R-R interval and the duration of systole of the immediately preceding beat. It has been shown by others and confirmed in this study that there are beat-to-beat changes in the duration of systole in this arrhythmia. Therefore, the beat-to-beat changes in the duration of ventricular diastole are not accurately reflected by changes in the duration of the immediately preceding R-R intervals.

The ventricular filling curves as recorded by the electrokymograph in these studies are characterized by rapid filling early in diastole and slower filling late in diastole as illustrated schematically in figure 11 and are similar to filling curves recorded with the cardiometer in the experimental animal. The rate of ventricular filling is indicated by the slope of the curve. Because of the flattening of the curves as diastole lengthens, changes in the duration of the diastolic filling period or the volume from which ventricular filling is initiated have little effect on ventricular end-diastolic volume when the ventricular filling period is long. However, when the period of diastole is short, small changes in the duration of diastole or in the volume from which ventricular filling begins have a marked effect on ventricular end-diastolic volume. For this reason the largest beat-to-beat variation of ventricular end-diastolic volume for a given R-R interval occurs when the R-R intervals are short. Another feature of the ventricular filling curves in atrial fibrillation is that the rate and duration of the rapid phase of ventricular filling differ from beat-to-beat as evidenced by the slopes of the ventricular filling curves. However, the slopes of these filling curves are similar if they are related to the ventricular volume during which filling occurs rather than to a portion of diastole with respect to time. Hence, the relative rate of ventricular filling at a given instant during diastole in this arrhythmia is determined by

![Graph](image)

**Fig. 11.** Schematic illustration of ventricular filling curve.

the relative ventricular volume at that instant of diastole.

The beat-to-beat dissociation of ventricular rate and ventricular end-diastolic volume makes it possible to test the effect of these 2 variables on ventricular function in atrial fibrillation. Left ventricular function was calculated in terms of an index of pressure work per beat or stroke work. This index of stroke work is determined by the beat-to-beat changes of both stroke “volume” and mean arterial pressure during ventricular ejection. These studies show that the beat-to-beat variations of stroke work in atrial fibrillation bear a linear relation to the beat-to-beat variations of ventricular end-diastolic volume. There was considerable scatter when stroke work was related to the duration of the preceding R-R interval. From the analysis of the relation of R-R interval to ventricular end-diastolic volume a dissociation between the immediate ventricular rate changes and ventricular end-diastolic volume was demonstrated. In spite of this, left ventricular stroke work was closely related to ventricular end-diastolic volume without any correction for rate. Thus these data give no evidence to indicate that ventricular rate changes per se alter ventricular function in man. This differs from what has been observed
in the experimental animal by Sarnoff with a different experimental approach. Furthermore, in vitro observations on heart muscle strips by others have shown that changes in the rate of contraction are associated with changes in the force of contraction. To explain this phenomenon, the presence of a poststimulation potentiating substance had been postulated. The results of these studies showed the variations of stroke work to be dependent on the variations of ventricular end-diastolic volume and revealed no evidence of beat-to-beat changes in the mechanical properties of left ventricular muscle as a consequence of the rate changes.

These variations of ventricular end-diastolic volume, stroke volume, and arterial pressure as a result of the ventricular arrhythmia have made it possible to test Starling’s “Law of the Heart” in man by studying a single sequence of beats. These studies indicate that the human heart behaves according to Starling’s “law.” Figure 10 shows portions of Starling curves from the human heart calculated from the data in this study. Here the index of stroke work is plotted against end-diastolic volume rather than stroke volume against end-diastolic volume as was done by Starling. In no instance was there seen evidence of a descending limb of the curves as described by Starling although several of the patients in this study had left ventricular enlargement and elevation of the left ventricular end-diastolic pressure. This is manifest clinically by the fact that the beats with the longer R-R intervals always have the largest arterial pulse pressures and highest arterial systolic pressures. If the heart with greater filling operated on the descending limb of the Starling curve, one might expect beats with lower arterial pulse pressure and systolic pressure occasionally to follow the longer R-R intervals. Such has not been observed, even in patients with severe left ventricular failure.

Studies of the left ventricular pressure curves demonstrate other interesting aspects of ventricular dynamics in this arrhythmia. First, ventricular end-diastolic volume and ventricular end-diastolic pressure may vary independently when the ventricular rate is rapid and irregular. In other words, left ventricular end-diastolic pressure and end-diastolic volume are not proportional to each other from beat to beat under these conditions. This phenomenon has been described by Wiggers and by Katz in the experimental animal with atrial fibrillation and is similar to that described under conditions of ventricular premature contractions in the experimental animal. It has also been described in man by Richards. The mechanism of the dissociation of the ventricular pressure-volume relationships during diastole as seen in this arrhythmia remain unexplained. It seems likely that it is due to 1 of 2 mechanisms, either the rate of ventricular relaxation varies from beat to beat, or what seems more likely, the elastic properties of the left ventricle differ during the various portions of diastole. The latter is in part supported by studies reported by Johnson and Katz. More recent studies of left ventricular compliance during diastole, by Buckley and associates also support the latter. Finally, these studies indicate that during ventricular irregularity the force of left ventricular contraction is determined by the ventricular end-diastolic volume and not by the end-diastolic pressure. This has also been shown to be the case in the experimental animal. It also points out one of the shortcomings of the use of pressure technics to study cardiac function and the need for volumetric technics.

This approach to the study of cardiac function by the application of the volumetric technic used in this study is admittedly a crude one with many shortcomings. There is at present no method that enables one to calculate absolute ventricular volumes in the intact human subject, nor is it possible to relate these electokymograph observations to absolute changes of volume or absolute volumes. Therefore, it is not possible to compare in any quantitative sense separate observations on the same subject or observations on different subjects. Furthermore, this method is applicable only when there are hemodynamic changes from beat to beat. Its usefulness is limited to the study of the relative characteristics of beats within a single sequence of beats.

There are certain hemodynamic features of atrial fibrillation that resemble those of another cardiac abnormality, namely, pulsus alternans.
In figure 3 can be seen a wide scatter of arterial systolic pressures and pulse pressures for the same R-R intervals within a single sequence of beats. This beat-to-beat variation of arterial systolic pressure and pulse pressure for the same R-R interval is similar to that observed in pulsus alternans. The similarity between atrial fibrillation and pulsus alternans in this regard is not a new observation, as it was noted by Lewis many years ago. Furthermore, this phenomenon resembling pulsus alternans as observed in subjects with atrial fibrillation is not a manifestation of left ventricular failure because several of the subjects in this study had left ventricles of normal size and normal left ventricular end-diastolic pressures. Studies of pulsus alternans both in the experimental animal by means of a cardiomere and in man by means of the left cardiac border electrokymogram have demonstrated that the larger beats arise from relatively larger ventricular end-diastolic volumes and the smaller beats from relatively smaller end-diastolic volumes. It seems clear from these published observations that in pulsus alternans there are beat-to-beat variations of ventricular filling, ventricular ejection, and resistance to ejection, much as in atrial fibrillation. In addition to these hemodynamic factors, studies by Green have indicated that in pulsus alternans there is also a defect in contraction of groups of myocardial fibers from beat to beat. No evidence of the latter was found in these studies of ventricular function in atrial fibrillation inasmuch as the beat-to-beat variations of ventricular work were explainable on the basis of the beat-to-beat variations of ventricular end-diastolic volume. There are no published observations of the relationship, if any, between the variations of stroke work and end-diastolic volume during pulsus alternans.

**Summary**

The beat-to-beat changes of ventricular rate, durations of systole and diastole, arterial pressure, left ventricular pressure, and an index of left ventricular volume have been studied in human subjects with atrial fibrillation.

Arterial pulse pressure, systolic pressure, and ventricular end-diastolic volume may show considerable scatter for a given R-R interval within a single sequence of beats in this arrhythmia. Mechanisms involved in determining this scatter have been studied and are related to certain features of the preceding beat; notably, the arterial pressure, end-systolic volume and duration of systole of the preceding beat.

There are beat-to-beat changes in the relationship between left ventricular end-diastolic pressure and end-diastolic volume when the ventricular rate is rapid and irregular in atrial fibrillation. Thus left ventricular end-diastolic pressure changes do not necessarily reflect volume changes under these conditions.

A method for calculating an index of ventricular stroke work has been described.

The beat-to-beat dissociation of the changes of ventricular rate, ventricular end-diastolic volume, and ventricular end-diastolic pressure has made it possible to study the effect of these variables on left ventricular work in man. The results indicate that left ventricular work is determined by the end-diastolic volume.

**Summario in Interlingua**

Le variationes del velocitate ventricular ab un pulso al proxime, le duration del systole e del diastole, le pression arterial, le pression sinistro-ventricular, e un indice del volumine sinistro-ventricular eseva studiate in subjectos human con fibrillation atrial.

Le pression arterio-pulsatile, le pression systolic, e le volumine ventricular diastolico-terminal monstra a vices in iste arrhythmia un grado considereble de dispersion pro un specific intervallo R-R intra un sol sequentia de pulsos. Le mechanismos participate in le determination de iste dispersion eseva investigate. Illos es relationate a certe characteristics del pulso precedente, specialmente su pression arterial, su volumine systolico-terminal, e su duration systolic.

Existe variationes ab un pulso al proxime in le relation inter le pression sinistro-ventricular diastolico-terminal e le volumine diastolico-terminal quando le velocitate ventricular es alte e irregular in le presentia de fibrillation atrial. Assi, alterationes del pression sinistro-ventricular diastolico-terminal non reflecte necessarimente alterationes de volumine sub le conditiones hie discutite.
Es describete un metodo pro calcular un indice del labor ventrículo-pulsatil.

Le dissociation del alterationes ab un pulso al proxime in le velocitate ventricular, le volume ventricular diastolico-terminal, e le pression ventricular diastolico-terminal ha rendite possibile le studio del efecto de iste variabels super le labor sinistro-ventricular in homines. Le resultatos indica que le labor sinistro-ventricular es determinate per le volume diastolico-terminal.

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Circulation. 1957;15:335-347
doi: 10.1161/01.CIR.15.3.335

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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