J Appl Physiol
90: 2238–2244, 2001.

Phase plane analysis of left ventricular hemodynamics

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Received 25 July 2000; accepted in final form 10 January 2001

Eucker, Stephanie A., Jennifer B. Lisauskas, Jasvindar Singh, and Sándor J. Kovács. Phase plane analysis of left ventricular hemodynamics. *J Appl Physiol* 90: 2238–2244, 2001.—We sought to extract additional physiological information from the time-dependent left ventricular (LV) pressure contour and thereby gain new insights into ventricular function. We used phase plane analysis to characterize high-fidelity pressure data in selected subjects undergoing elective cardiac catheterization. The standard hemodynamic indexes of LV systolic and diastolic function derived from the time-dependent LV pressure contour could be easily obtained using the phase plane method. Additional novel attributes of the phase plane pressure loop, such as phase plane pressure loop area, graphical representation of the isovolumic relaxation time constant, and quantitative measures of beat-to-beat systolic-diastolic coupling were characterized. The asymmetry between the pressures at which maximum isovolumic pressure rise and pressure fall occur, as well as their load dependence, were also easily quantitated. These results indicate that the phase plane method provides a novel window for physiological discovery and has theoretical and applied advantages in quantitative ventricular function characterization.

nonlinear dynamics; isovolumic relaxation; systolic-diastolic coupling

IN THE ABSENCE OF VALVULAR disease, left ventricular (LV) pressures obtained during cardiac catheterization are rarely used for more than the measurement of peak systolic and end-diastolic pressures. In pathophysiological studies of selected subjects, additional assessment of systolic and diastolic function may be performed on the basis of the hemodynamic waveform. In some settings, the shape of the waveform conveys diagnostic information (e.g., the "square-root" sign in constrictive-restrictive physiology). Usual hemodynamic assessment involves the measurement of selected points on the LV pressure (P) vs. time (t) contour and/or its derivative (dP/dt). These selected points obtained from catheterization data usually include maximum LV pressure (P_{max}), minimum LV pressure (P_{min}), pressure at diastasis (P_{diasta}), LV end-diastolic pressure (LVEDP), peak positive dP/dt ($\dot{p}_{+max}$), and peak negative dP/dt ($\dot{p}_{-min}$) (3, 13). Other parameters such as the time constant of isovolumic relaxation (τ) are computed by performing a least-mean-square fit of an assumed mathematical expression (exponential decay) to a selected portion of the hemodynamic contour. The physiological and clinical significance of these parameters and the indexes derived or constructed from them is well established and forms a firm basis for ventricular function analysis, clinical decision making, and patient management (3, 13). The LV and aortic root pressure waveforms, i.e., pressure as a function of time, are the standard contours through which cardiovascular physiology is taught to graduate students, medical students, and clinicians. Recognition of the features of these waveforms and their physiological and pathophysiological causes by cardiologists, surgeons, and others constitutes an important clinical diagnostic skill (3, 13).

In an effort to maximize the amount of useful physiological information extracted from hemodynamic waveforms, we have found it useful to view the heart not only from the perspective of a physiologist or cardiologist but also from the perspective of a physical scientist or an applied mathematician. In this paradigm, the heart is viewed as a nonlinear oscillator that generates an output (pressure) as a function of time. The precise mathematical rules governing the oscillator (i.e., differential equations with solutions that correctly predict pressure as a function of time) cannot be written down, because it is not really understood how all the cellular and subcellular components of the heart interact with each other and with the load against which the heart works on a beat-by-beat basis. However, we can use the methods of nonlinear dynamics, which are familiar to mathematicians, physicists, and biomedical engineers, to record and analyze the output of the oscillator (pressure as a function of time). Our goal is to learn more about the heart with the use of this paradigm and to reexpress the results of our analysis in terms that are comprehensible and of practical use to physiologists and cardiologists.

In analogy with the characterization of nonbiological, nonlinear oscillators for which equations of motion are known (7), we have selected the phase plane as the arena in which to carry out our analysis of ventricular hemodynamic data. Phase plane analysis is carried out on graphs of precisely periodic or nearly periodic func-

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tions plotted such that the function is the abscissa and its time derivative is the ordinate (7). In our application, pressure (P) is plotted on the *x*-axis and its derivative dP/d*t* on the *y*-axis. Because the ventricular pressure completes one oscillation for each cardiac cycle, phase plane plots of dP/d*t* vs. P form closed trajectories. Physical scientists refer to these loops as limit cycles.

Phase plane display of physiological signals has been previously performed to characterize selected aspects of the electrical activity of the heart. In this analysis, the myocardium was viewed as an excitable medium, thereby allowing the analytic methods of nonlinear dynamics to be used to characterize the heart's propensity to certain arrhythmias (12).

The isovolumic relaxation portion of LV pressure (rather than the entire cardiac cycle) has been plotted in the phase plane for canine hearts to compare the appropriateness of a logistic model with the conventional exponential model of pressure decay (6). This analysis has also been used in human hearts to assess the effects of human heart failure on consistency of fit for several different time constants derived from exponential and logistic models of pressure decay (9). Phase plane analysis allowed for greater robustness of results than conventional P vs. *t* fits, because pressure and its time derivative were accounted for in the dP/d*t* vs. P fits. Phase plane analysis has also been used to discern respiratory-cardiac coupling and to characterize LV function (4). More recently, the phase plane method has been employed to characterize the relationship between $\dot{p}_{-min}$ and ejected aortic blood momentum (10).

We used the phase plane method to characterize LV pressure contours in selected subjects. New features related to ventricular function that are not easily recognizable when the data are viewed in the usual P vs. *t* format (Fig. 1*A*) can be deduced by analysis of phase plane limit cycle attributes (Fig. 1*B*). We have selected five of these attributes for analysis: *1*) correspondence between phase plane area (PPA) and ejection fraction (EF), *2*) validity of the exponential pressure decay assumption during isovolumic relaxation, *3*) symmetry of $\dot{p}_{+max}$ and $\dot{p}_{-min}$, *4*) relationship between the pressures at which $\dot{p}_{+max}$ (P_C) and $\dot{p}_{-min}$ (P_R) occur, and *5*) comparison of P_C and P_R to P_{max}.

Information about diastolic and systolic function, global ventricular function, and systolic-diastolic coupling that could not be discerned from the usual P vs. *t* display format could be easily visualized and quantitated using the phase plane format. Our method naturally lends itself to the derivation of phase plane-based indexes of systolic and diastolic ventricular physiology and function. Our approach is also well suited for the development of mathematical modeling strategies of the heart's action. Further application to gain insight into novel physiological relationships and for assessment of clinical utility is in progress.

Glossary

LV	Left ventricle; left ventricular
P	Pressure
t	Time
dP/d*t*	Time derivative of pressure (change in pressure with respect to time)
P_{max}	Maximum systolic pressure
P_{min}	Minimum diastolic pressure
P_{diasta}	Pressure at diastasis
LVEDP	LV end-diastolic pressure
$\dot{p}_{+max}$	Peak positive dP/d*t*
$\dot{p}_{-min}$	Peak negative dP/d*t*
P_C	Pressure at $\dot{p}_{+max}$
P_R	Pressure at $\dot{p}_{-min}$
EF	Ejection fraction
PPA	Phase plane area
τ	Time constant of isovolumic relaxation
P_{ex}	External mechanical power of the heart
IVRT	Isovolumic relaxation time

METHODS

Patients undergoing elective cardiac catheterization at the request of their referring physicians were recruited, and informed consent according to Barnes-Jewish Hospital/Washington University Medical Center criteria was obtained. Single transducer-tipped 7-F pigtail micromanometric catheters (model SP-747, Millar Instruments, Houston,

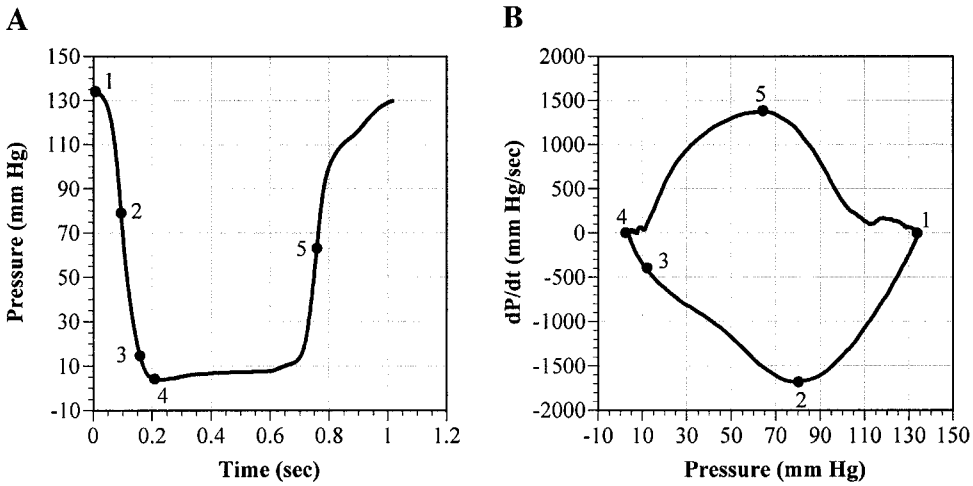

Fig. 1. *A*: pressure vs. time contour. *B*: phase plane plot of time derivative of pressure (dP/d*t*) vs. pressure. *1*, Maximum systolic pressure; *2*, peak −dP/d*t*; *3*, mitral valve opening; *4*, minimum diastolic pressure; *5*, peak +dP/d*t*.

TX) were used to record high-fidelity LV and subsequent aortic root pressure data at the commencement of cardiac catheterization. The acquired data were stored on a Macintosh Power PC 7200 computer system using LabVIEW (National Instruments). Pressure data were acquired at a programmable sample rate of 1 kHz at 16-bit accuracy at the commencement of the procedure before the use of any contrast agent. Along with the simultaneous electrocardiogram and pressure data, calibration, patient identification, and demographics were recorded in the header of each data file. Analysis of the data files was done off-line in the Cardiovascular Biophysics Laboratory. The format of LabVIEW facilitates rapid, interactive plotting of the pressure contours and their analysis in the phase plane.

LVEF was calculated for each patient by two methods. For *method 1*, LVEF was calculated from ventriculographic data obtained in the 30° right anterior oblique position with the injection of 35 to 40 ml of Optiray (Mallinckrodt, St. Louis, MO) by a power injector (model Mark V, Medrad, Pittsburgh, PA). EFs for each subject were calculated by two independent observers and averaged. Well-opacified end-systolic and end-diastolic LV contours in sinus rhythm were traced using the electronic joystick feature of the laboratory's imaging system (Siemens Hi-Cor). Beats associated with catheter- or injection-induced ectopy were rejected. The area-length method (8) was selected from the option menu of the imaging system, and the EFs were automatically displayed. Discrepancies of >10% were adjudicated by consensus. For *method 2*, 35-mm cineangiographic film and the beat selection criteria described for *method 1* were used. LV contours were traced and digitized by hand using a commercially available computer and associated digitizing tablet (Sony SMC-70G). EFs were computed using the area-length method of Dodge et al. (1).

RESULTS

Data from 51 subjects were analyzed. Clinical attributes of the sample studied were as follows: slight elevation of LVEDP ($12 < \text{LVEDP} < 18$ mmHg) but no valvulopathy (11 subjects), trace to 1+ mitral valve prolapse (4 subjects), trace aortic insufficiency (2 subjects), 1+ mitral valve prolapse and trace aortic insufficiency (1 subject), and diminished LVEF (≤50%) but no valvulopathy (3 subjects). The remaining 30 subjects had normal cardiac physiology. Figure 1 shows the relationship between a typical LV pressure vs. time plot and the corresponding LV pressure phase plane plot. Five points during the cardiac cycle with physiological significance have been selected and labeled. As one follows *points 1–5* on the P vs. *t* format (Fig. 1*A*), one inscribes a clockwise loop, *points 1–5*, in the phase plane (Fig. 1*B*). Note that the LV pressure phase plane plot is bounded on the left by P_{min}, on the right by P_{max}, on the top by $\dot{p}_{+max}$, and on the bottom by $\dot{p}_{-min}$. The isovolumic relaxation segment, *portion 2–3*, is easily perceived.

Figure 2 shows a three-dimensional phase plane/time embedding diagram, with time on the *x*-axis, pressure on the *y*-axis, and dP/d*t* on the *z*-axis. When a single cardiac cycle of the continuous, spiral-like, three-dimensional plot is projected onto the three orthogonal planes, the standard P vs. *t*, dP/d*t* vs. *t*, and dP/d*t* vs. P (phase plane plot) are recovered. Using this projection format, the standard characteristics of the LV pressure contour, such as R-R interval, diastole, systole, and isovolumic contraction and relaxation, are easily discerned. Figure 2 also illustrates that the progress of time is not explicitly observable in the phase plane (i.e., time is orthogonal to the dP/d*t* vs. P plane).

Correspondence of PPA to EF. LVEF is defined as the ratio of stroke volume to LV end-diastolic volume. The range of volumes is determined in part by the range of operating pressures for the pressure-volume (P-V) loop of a given ventricle. Similarly, the area of the P-V loop for one cycle is related to the external work ($W = \int PdV$)

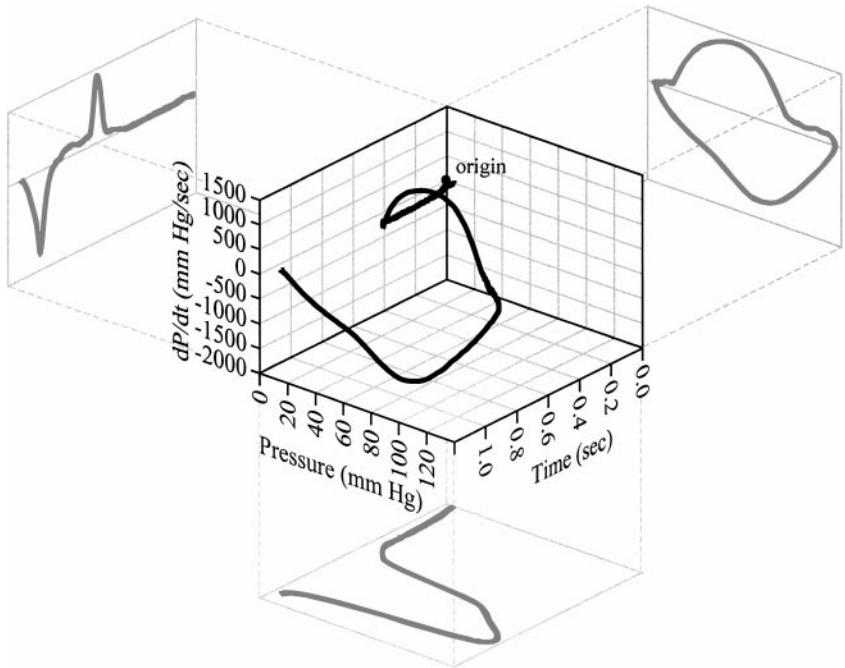

Fig. 2. Three-dimensional contour of time (*x*-axis) vs. pressure (*y*-axis) vs. dP/d*t* (*z*-axis; *center*, black plot) and 2-dimensional projections of the contour onto the phase plane (dP/d*t* vs. pressure), the pressure vs. time plane, and the dP/d*t* vs. time plane (gray plots, clockwise from *top right*).

performed by the heart. The area of the limit cycle inscribed in the phase plane is bounded by the pressure differential ($P_{max} - P_{min}$) and the dP/dt differential ($\dot{p}_{+max} - \dot{p}_{-min}$). Therefore, the hypothesis that PPA and EF are related to the extent to which the range of operating pressures is common to both can be tested. We tested this hypothesis through experimental determination of the correlation between PPA and LVEF.

The general (differential) expression for external mechanical power (P_{ex}) is the time rate of change of work, PdV (1, 2)

$$P_{ex} = P\frac{dV}{dt} \quad (1)$$

which can be rewritten as

$$P_{ex} = P\frac{dV}{dP}\frac{dP}{dt} \quad (2)$$

In *Eq. 2*, power (P_{ex}) is expressed as a function of the variables that define the phase plane (P, dP/dt) and the expression dV/dP = 1/(dP/dV), where V is volume. Note that dP/dV is (variable) chamber stiffness.

Figure 3 illustrates limit cycle area vs. EF for all 51 subjects; the linear regression relationship for EF as a function of PPA is given by

$$EF = (2.66 \times 10^{-5})PPA + 69.3 \quad (r = 0.17) \quad (3)$$

Similar results were obtained when EF vs. PPA was considered for only the 30 subjects having normal LV function (defined by LVEF ≥60%) and normal blood pressure. Thus the relationship between EF and PPA cannot be accurately described by a line. Further theoretical and experimental work is needed to more fully characterize the relationship between power or work and (variable) stiffness, dP/dV, for the entire cardiac cycle to allow application of *Eq. 2*. One line of inquiry suggesting that this relationship may be further characterized involves consideration of a three-dimensional plot of dP/dt, P, and V. The projection of the three-dimensional loop in this space onto the P-V plane yields the classic P-V loop, and projection onto the dP/dt vs. P plane yields the LV pressure phase plane plot.

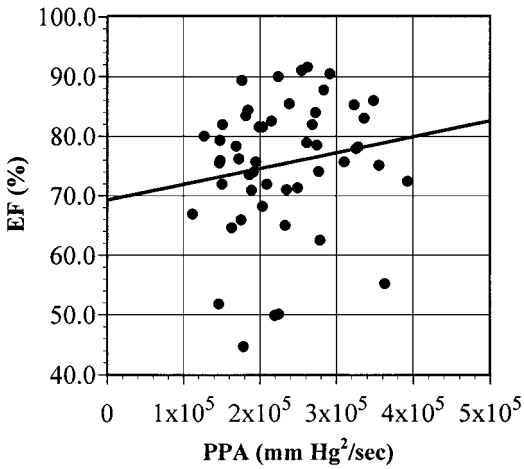

Fig. 3. Analysis of left ventricular (LV) ejection fraction (EF) vs. phase plane area (PPA) showed no apparent linear correlation. A linear fit of the data yielded EF = (2.66 × 10^{-5})PPA + 69.3 (r = 0.17).

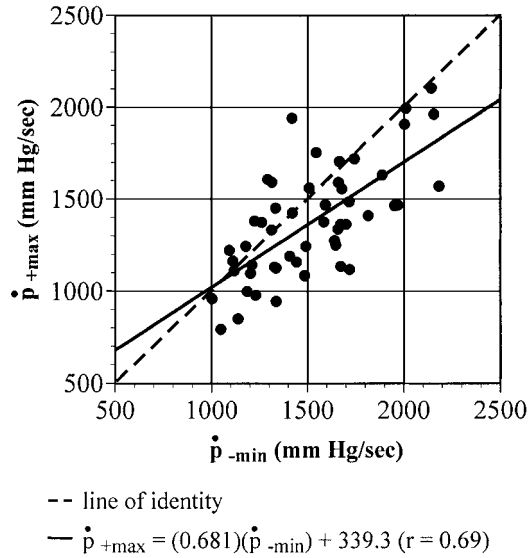

Fig. 4. Correlation between peak +dP/dt ($\dot{p}_{+max}$) during isovolumic contraction and peak −dP/dt ($\dot{p}_{-min}$) during isovolumic relaxation. Thirty-seven of the 51 points (73%) fall below the line of identity, indicating that the relationship is not symmetrical and that $\dot{p}_{-min}$ is generally greater than $\dot{p}_{+max}$. Analysis of the deviation from symmetry using Student's t-test yields $P \ll 0.01$.

Isovolumic relaxation and the assumption of exponential pressure decay. The isovolumic relaxation time (IVRT) is defined as the period commencing at aortic valve closure and concluding at mitral valve opening. Aortic valve closure is known to occur before the time of $\dot{p}_{-min}$ and is indicated by the "incisura" (dicrotic notch) of the aortic root pressure recording. Mitral valve opening is conventionally assumed to occur at a pressure ~5 mmHg above the LVEDP of the previous diastole (8, 11). During this time, the decay of LV pressure from P_R, the pressure at $\dot{p}_{-min}$ (*point 2* in Fig. 1), to a pressure 5 mmHg above LVEDP (*point 3* in Fig. 1) is usually modeled as an exponential decay (8, 11). Two equations have been proposed that differ in the asymptotic value of the pressure at late ($t \rightarrow \infty$) times. In general, we have observed that the asymptote is not zero. For nonzero asymptote, the equation is given by

$$P = P_0 e^{-t/\tau} + P_b \quad (4)$$

where t is time, $P_0 + P_b = P_R$ is the pressure at $\dot{p}_{-min}$ (i.e., at $t = 0$), τ is the time constant of isovolumic relaxation, and P_b is the asymptote (P at $t = \infty$). Therefore, dP/dt is given by

$$\frac{dP}{dt} = -\frac{1}{\tau}P_0 e^{-t/\tau} \quad (5)$$

which, for P_0, yields

$$P_0 = -\tau\frac{dP}{dt}e^{+t/\tau} \quad (6)$$

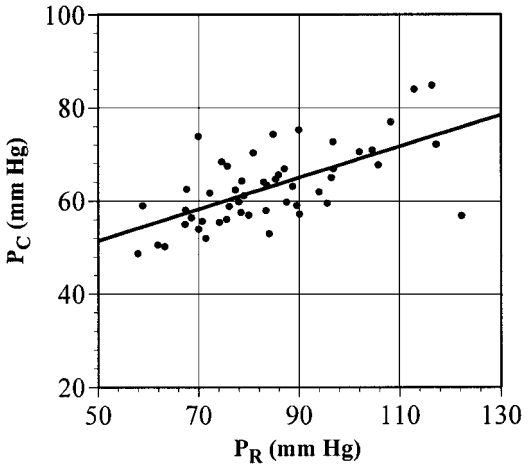

Fig. 5. Correlation between the pressure at which $\dot{p}_{+max}$ occurs (P_C) and the pressure at which $\dot{p}_{-min}$ occurs (P_R). The linear relationship is given by the following equation: $P_C = (0.34)(P_R) + 34.6$ ($r = 0.64$).

Substitution into *Eq. 4* gives

$$P = -\tau \frac{dP}{dt} e^{+t/\tau} e^{-t/\tau} + P_b \qquad (4b)$$

Solving for dP/dt, we obtain

$$\frac{dP}{dt} = -\frac{1}{\tau} P + \frac{P_b}{\tau} \qquad (7)$$

Hence, plotting dP/dt vs. P, with the assumption that P(t) obeys an exponential decay, results in a linear relationship of the form

$$\frac{dP}{dt} = mP + b \qquad (8)$$

where $m = -1/\tau$ is the slope and $b = P_b/\tau$ is the y-intercept. Thus, in the phase plane, τ and the asymptote (P_b) of an assumed exponential pressure decay are easily determined from the slope and the intercept of a straight line.

The ease of determination of τ is important to note. Previously, τ was often computed from hand-digitized data. With the use of the phase plane, however, τ can be determined automatically, using software methods. We observed that for 16 of 44 subjects (36%) without valvulopathy, i.e., having a true isovolumic relaxation period, the relationship between dP/dt and P was *not* linear.

Symmetry of peak +dP/dt vs. peak −dP/dt. Pressure viewed in the phase plane allows for visual comparison between the maximum rate of isovolumic pressure increase, $\dot{p}_{+max}$, and the maximum rate of isovolumic pressure decay, $\dot{p}_{-min}$. Thus phase plane analysis provides direct graphical comparison between an index of contractile function, $\dot{p}_{+max}$, and an index of relaxation, $\dot{p}_{-min}$. Symmetry of $\dot{p}_{+max}$ and $\dot{p}_{-min}$ means that the magnitudes of these two values are equal and that, in the phase plane, the plot of the limit cycle extends equal heights above and below the abscissa.

We found that the maximum rate of pressure decay during isovolumic relaxation, $\dot{p}_{-min}$, and the maximum rate of pressure increase during isovolumic contraction, $\dot{p}_{+max}$, are not symmetrical. For 37 of our 51 total subjects (73%), $\dot{p}_{-min}$ is, on average, greater than $\dot{p}_{+max}$. These peak rates are linearly related, such that $\dot{p}_{+max} = (0.68)(\dot{p}_{-min}) + 339.3$ ($r = 0.69$), as shown in Fig. 4.

Comparison of P_C to P_R. P_C and P_R are also easily visualized in the phase plane. A comparison of these two pressures showed that P_C is lower than P_R for 50 of 51 subjects (98%). Interestingly, we found that P_C and P_R are linearly related, as shown in Fig. 5, where $P_C = (0.34)(P_R) + 34.6$ ($r = 0.64$).

Comparison of average P_C and P_R to average P_{max}. We averaged 35 beats for each subject. When the average values of P_C and P_R for each subject were plotted against the corresponding average P_{max}, we found that P_C and P_R were linearly related to P_{max}, as shown in Fig. 6. Furthermore, when the same plots were made for individual subjects on a beat-by-beat basis, linear relationships were again seen. An example is shown in Fig. 7. The slopes of the relationships for each individual agreed qualitatively with and were similar quantitatively to the results across all the subjects. The results indicate that P_C and P_R are load dependent and that the slope of P_R vs. P_{max} is greater than the slope of

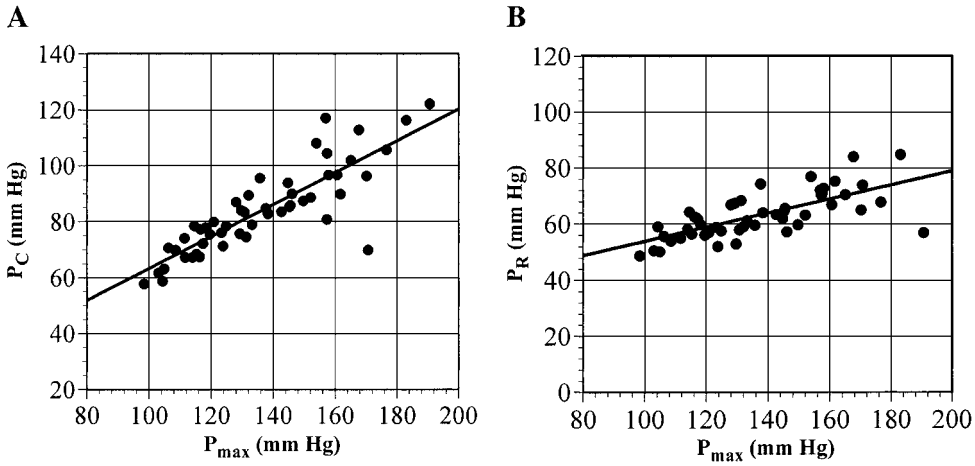

Fig. 6. *A*: correlation between P_C and maximum pressure (P_{max}), where the linear relationship is given by the following equation: $P_C = 0.25 \cdot P_{max} + 28.8$ ($r = 0.71$). *B*: correlation between P_R and P_{max}, where the linear relationship is given by the following equation: $P_R = 0.57 \cdot P_{max} + 6.3$ ($r = 0.85$).

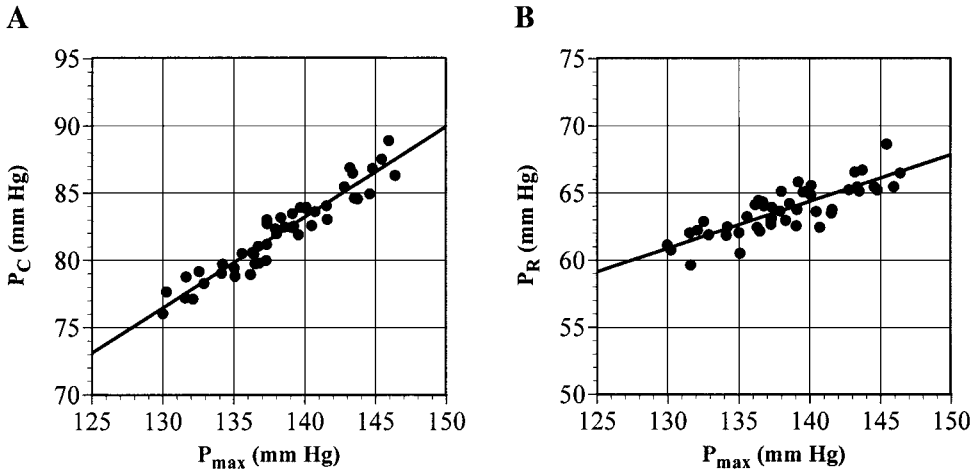

Fig. 7. Correlation between P_C and P_{max} (*A*), where $P_C = 0.35 \cdot P_{max} + 15.5$ ($r = 0.82$), and between P_R and P_{max} (*B*), where $P_R = 0.67 \cdot P_{max} - 11.2$ ($r = 0.95$), for a single representative individual.

P_C vs. P_{max}. We can conclude that relaxation during diastole has a memory of the level of work done in systole and that P_R varies more with load than P_C.

DISCUSSION

It is customary in experimental physiology to characterize relaxation via τ with the use of an exponential fit of pressure decay. Analysis of numerous phase plane plots indicates that the assumption of exponential pressure decay during isovolumic relaxation does not always correspond to the data. To graphically illustrate this, two subjects with widely varying features of the limit cycle corresponding to isovolumic relaxation are shown in Fig. 8. The subject in Fig. 8*A* has "normal" relaxation that is well fit by a linear dP/d*t* vs. P relationship. For the subject in Fig. 8*B*, the limit cycle indicates a curvilinear isovolumic relaxation that is not well fit by a linear dP/d*t* vs. P relationship. Hence, the appropriateness of *Eq. 4* and the applicability of an assumed exponential decline in pressure cannot be viewed as generally valid.

The physiological explanation for linear vs. curvilinear dP/d*t* vs. P relationship during isovolumic relaxation is lacking, although limitations in the use of τ for various clinical scenarios such as mitral regurgitation and aortic stenosis are known (2). For instance, the linearity of dP/d*t* vs. P during pressure decay may not be true in hearts with regurgitant mitral or aortic valves or with extensive coronary artery disease (2). Because ventricles having aortic insufficiency do not have an isovolumic period, the assumption of exponential pressure decay is easily understood not to be applicable.

We found that the portion of the phase plane between $\dot{p}_{-min}$ and P_{min} was nonlinear for three of seven subjects (43%) who had aortic insufficiency or mitral valve regurgitation. However, a similar percentage (36%) of subjects who did not have valvulopathies also showed a nonlinear isovolumic relaxation phase. Furthermore, we observed that 8 of 30 subjects (27%) who had normal LVEF and 1 of 3 subjects (33%) who had low LVEF displayed nonlinear isovolumic relaxation, while 7 of 11 subjects (64%) who had elevated LVEDP showed nonlinear isovolumic relaxation. Thus the phase plane can be used to objectively clarify whether a given LV, in the absence of valvulopathy, is or is not a proper candidate for characterizing its isovolumic relaxation phase using τ. This limitation in the applicability of τ to characterize diastolic function merits recognition.

Furthermore, we note that if an exponential pressure decline during IVRT is assumed, the assumption

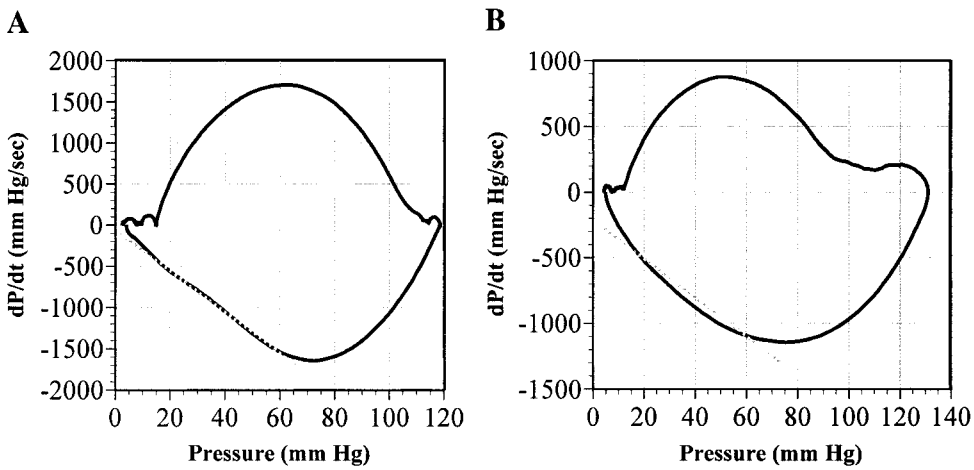

Fig. 8. Comparison of the goodness of fit of an exponential pressure decay, which plots as a straight line in the phase plane, during isovolumic relaxation. The best-fit lines (gray dashed lines) for a linear isovolumic relaxation period (*A*) and a curvilinear isovolumic relaxation period (*B*) are shown superimposed on the phase plane plots.

that the fit should commence at $\dot{p}_{-min}$ (8, 11) does not fit the data. This limitation is particularly clear when the data are viewed in the phase plane. We found that in all our subjects in whom the assumption of linearity of dP/dt vs. P was valid, starting 10 ms after $\dot{p}_{-min}$, rather than at $\dot{p}_{-min}$, gave the best r value for curve fitting. This corresponds to an average relative pressure offset of 10 mmHg below P_R. For the determination of τ, we observed that the assumption of linearity was valid for at least the next 40 ms. This is well within normal range (3).

In our analysis of $\dot{p}_{+max}$ vs. $\dot{p}_{-min}$, we found that when only the subjects with normal hearts were considered, 23 of 30 subjects (77%) had $\dot{p}_{-min}$ greater than $\dot{p}_{+max}$, indicating that the same result is observed for healthy and less healthy subjects. This result provides evidence that the heart relaxes at a faster rate during isovolumic relaxation than it contracts during isovolumic contraction.

Limitations. One limitation of this study was that there was no systematic alteration of beat-to-beat afterload. This is justified by our goal to contrast the relationship between conventional and phase plane-derived indexes of ventricular function and demonstrates the overall utility of phase plane analysis. Moreover, drug manipulation of heart rate, afterload, and other measures of cardiac function has been done by others (5, 6, 9) and, therefore, was not part of the data acquisition protocol in these subjects. However, our examination of average values across all subjects (n = 51) includes data in the (normal) range of heart rates and afterloads and can reveal new physiological relationships using phase plane analysis.

Conclusion. High-fidelity hemodynamic pressure and ventriculographic function (EF) were assessed in 51 subjects. Continuous sets and single-beat LV pressure data were plotted in the phase plane, and the corresponding phase plane loop areas were analyzed. A weak (r = 0.17) correlation between limit cycle area and EF was observed. The standard assumption of exponential pressure decay for a portion of the isovolumic relaxation phase of the LV pressure contour was appropriate for hemodynamic data in 28 of 44 subjects who had an isovolumic relaxation period. In 16 subjects, the pressure decay during the isovolumic phase was curvilinear and not well fit by an exponential relationship. Moreover, the phase plane method graphically shows that the convention of initiating the fit to determine τ at $\dot{p}_{-min}$ introduces a systematic bias. The phase plane method also allowed graphical assessment of the symmetry between peak +dP/dt and peak −dP/dt and showed that $\dot{p}_{-min}$ is greater than $\dot{p}_{+max}$. An explanation for this observation has not yet been offered. P_C and P_R were linearly correlated with each other. Furthermore, P_C and P_R were well correlated with P_{max} and indicated that P_R varies more with load than P_C.

We recognize that the information extracted from the phase plane plot also exists in the original pressure vs. time data. However, the format of the phase plane display facilitates recognition and quantitation of novel physiological/hemodynamic relationships. Therefore, we conclude that phase plane analysis provides a new window for LV hemodynamic analysis and is a source of new physiological insight into LV function and the quantitative relationship between systole, diastole, and load. Additional work, including hemodynamic measurements to fully elucidate and characterize these relationships, is warranted in an effort to improve our understanding of ventricular physiology and to ultimately contribute to improved clinical care.

We thank the staff of the Barnes-Jewish Hospital Cardiac Catheterization Laboratory for assistance in clinical hemodynamic data acquisition and our colleagues in the Cardiovascular Biophysics Laboratory for helpful comments and suggestions.

This study was supported in part by National Heart, Lung, and Blood Institute Grants R01 HL-54179 and K24 HL-04023, the Whitaker Foundation (Rosslyn, VA), and the Alan A. and Edith L. Wolff Charitable Trust (St. Louis, MO).

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