Effect of Phasic Respiration on Left Ventricular Dimension and Performance in a Normal Population
An Echocardiographic Study
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SUMMARY Echocardiographic examination of the left ventricle (LV) in 30 normal subjects, 5 to 47 years of age, was performed in order to analyze the effects of phasic respiration on LV dimensions and derived LV function. Peak expiratory and peak inspiratory LV diastolic and systolic dimensions were measured and extrapolated to volume estimates using a standard formula. Although there was wide variation in the individual measurements, and particularly in systolic dimension, the mean peak inspiratory diastolic dimension, derived diastolic volume, and stroke volume all decreased significantly (P < 0.001); a smaller decrease in ejection fraction was seen (P < 0.02), while the changes in mean end-systolic dimension and end-systolic volume were not significant. While these observed changes may reflect a true physiologic variation, an artifactual component cannot be excluded. Regardless of their physiologic significance, however, these data show that the effect of phasic respiration is a factor to be considered in correlating echocardiographic studies of LV function in both normal and, possibly, pathologic cardiovascular conditions.

SINGLE CRYSTAL ECHOCARDIOGRAPHY is an established technique for evaluating left ventricular (LV) dimensions and function. Correlations between echocardiographic and cineangiographic dimensions have been excellent in both adult and pediatric subjects, provided regional dyskinesis is absent. This noninvasive technique also promises to be useful for serial measurements in the longitudinal follow-up of patients with certain types of cardiovascular disease. Recent attention has been directed to the evaluation of LV volume alterations and, hence, LV function in normal subjects during various acute hemodynamic interventions and the effects of respiration on these same parameters in patients with pericardial effusion. We too have noted marked respiratory variation in the LV internal dimensions in some patients with pericardial effusion, but changes of a similar or even greater magnitude frequently were seen in patients with a normal cardiovascular system. The present study, therefore, was undertaken to quantitate the magnitude of change in echocardiographic LV dimensions and derived function during the respiratory cycle in a group of normal subjects and thereby to provide a frame of reference for the interpretation of these changes in patients with cardiorespiratory pathology.

Methods
A final group of 30 subjects, ranging in age from 5 to 47 years, with a mean age of 20 years, was accepted for evaluation. There was no evidence for cardiopulmonary disease or hypertension in any subject. Echocardiographic evaluation
was performed utilizing a commercially available Ekoline 20 ultrasonoscope and Smith-Kline 2.25 MHz transducers focused at 5 or 10 cm. All recordings for LV function were made with the patient supine or in a slight left lateral decubitus position and with the transducer at an approximate right angle to the chest wall in the third, fourth, or fifth left intercostal space adjacent to the sternum. The transducer was then angulated slightly inferolaterally beneath the mitral valve so that optimum definition of septal and posterior wall endocardial surfaces were obtained. All recordings were made at the conclusion of a standard echocardiographic examination, with the subject as relaxed as possible during quiet respiration, and included an electrocardiogram, respiratory tracing and, often, a high-frequency phonocardiogram. The respirometer was not calibrated to intrathoracic pressure. The signal output was recorded on an Electronics-for-Medicine DR-8 photographic recorder at a paper speed of 50 or 100 mm per second. Left ventricular diastolic dimension was measured on a vertical axis at the peak of the electrocardiographic R wave. Left ventricular systolic dimension was measured on a vertical axis at the maximum anterior excursion of the posterior LV wall endocardial echo. The calipers were placed at the midpoint of the echo created by the blood-endocardial interface and LV dimensions were measured to the nearest millimeter during at least three cycles of quiet phasic respiration and extrapolated to volume, utilizing the formula of Teichholz et al. wherein

$$ \text{volume} = \frac{7}{D + 2.4} \times D^3 $$

where D is the echocardiographic dimension. Occasionally there was respiratory variation in the clarity of the endocardial echo. In such instances the true endocardial echo could be identified by assuming a constant systolic and constant diastolic wall thickness throughout the record (cf., fig. 1).

While the axial resolution (or the ability to distinguish two separate points) of 2.25 MHz ultrasound in body tissue is in the order of 1 mm (reference 12), the accuracy and reproducibility of ultrasound in measuring LV dimensions is undoubtedly diminished by several factors, including the superimposition of phasic respiration. In an attempt to obtain an index of the variability of the measurement, the LV internal dimensions were measured in ten consecutive subjects during held expiration. In each case, the measurement was reproducible to within 1 mm (fig. 2). Therefore, a change in dimension of less than 2 mm (approximately a 5% or less variation in dimension and a 10% or less variation in derived volume) was assumed to be within the error of the method, in agreement with the observations of Redwood et al. To minimize the effect of a significant variation in diastolic filling period, subjects with a marked sinus arrhythmia (greater than a 10% variation in R-R interval) were excluded. In approximately 40% of potential subjects, visualization of the LV during both phases of the respiratory cycle was not adequate (usually with inspiration) and these subjects were also excluded from the final analysis.

The mean and standard error of the absolute change in diastolic and systolic dimension are presented for comparative purposes but, in order to control for the differences in ventricular size from one subject to another, the rest of the measurements for each respiratory cycle were normalized by expressing the changes from peak expiration to peak inspiration as a percentage deviation from the peak expiratory dimensions and derived volumes, e.g.:

$$ \% \text{change in left ventricular diastolic dimension (DD)} = \frac{\text{DD peak expiration} - \text{DD peak inspiration}}{\text{DD peak expiration}} \times (-100) $$

$$ \% \text{change in left ventricular diastolic volume (DV)} = \frac{\text{DV peak expiration} - \text{DV peak inspiration}}{\text{DV peak expiration}} \times (-100) $$

A negative percentage change indicates a decrease in the observed measurement from peak expiration to peak inspiration. Parameters analyzed included percentage change in diastolic dimension and volume, systolic dimension and volume, stroke volume and ejection fraction. For statistical purposes we assumed as the null hypothesis that there is no significant effect of respiration on left ventricular dimensions and derived function (i.e., assumed mean percentage variation = zero). Relative deviates of the measured mean variations were then calculated and levels of significance obtained from standard tables.

**Results**

In absolute terms, the diastolic dimension showed a mean inspiratory decrease of 2.9 ± 0.4 mm (P < 0.001), while the mean systolic dimension decreased 0.7 ± 0.4 mm (not significant). The normalized data show that the mean peak expiratory to peak inspiratory diastolic dimension, derived diastolic volume and stroke volume all decreased significantly [P < 0.001 (see fig. 3)] with a smaller decrease in ejection fraction (P < 0.02). While the mean systolic dimension and derived volume did not change significantly, it is obvious from figure 3 that, in certain individuals, these parameters varied widely. For example, the change in systolic dimension ranged from −14.3% to +13.8%, with a correspondingly larger variation in the derived systolic volume. The significance of this variability is not known (see Discussion) but in the majority (17 subjects) the variation was less than ±5% for systolic dimension and less than ±10% for derived volume. Figures 1 and 4 are examples of subjects with inspiratory decreases in left ventricular dimension. In figure 1, the inspiratory decrease in the minor axis dimension appears to be due in part to both posterior displacement of the septum and anterior displacement of the posterior left ventricular wall at end diastole. In other subjects (fig. 4), posterior displacement of the septum accounted for the majority of the inspiratory decrease in dimension. No subject demonstrated a significant inspiratory increase in LV diastolic dimension and indeed only three subjects showed more than a 5% inspiratory increase in the systolic dimension. Similarly, only two subjects showed an inspiratory increase in stroke volume, and that was 4% and 5%, variations probably within the error of the method.

Discussion

The effect of phasic respiration on LV stroke volume and dimension has been investigated by a variety of techniques in both man and the experimental animal model. Using the pressure gradient technique, Ruskin et al.13 noted a 10% average inspiratory decrease in stroke volume in a group of 11 normal subjects undergoing cardiac catheterization. Guntheroth et al.14 found changes of a similar magnitude (11%) in the instrumented, lightly anesthetized dog. The echocardiographically derived inspiratory decrease in stroke volume of 16% in the present study is in agreement with these earlier reports. Possible explanations for the well-documented inspiratory decrease in LV stroke volume include 1) a decrease in pulmonary venous return due to the more negative intrathoracic pressure; 2) encroachment upon the LV by increased right ventricular (RV) volume; and 3) an inspiratory decrease in LV end-diastolic pressure. These data do not distinguish between the first two possibilities and
Figure 2. Left ventricular dimensions recorded during held expiration in subject W.M., a 24-year-old male, in an attempt to illustrate the reproducibility of the measurements. The dimensions in diastole were measured at the peak of the R wave of the ECG (LVDD$_D$). Note this point differs slightly from the end-diastolic dimension defined by the mitral component of the first heart sound (M$_1$). The systolic dimensions were measured at the peak anterior movement of the posterior endocardial surface, corresponding to the aortic component of the second heart sound (A$_2$). The standard deviation of both the systolic and diastolic dimensions was ±0.4 mm. The respirometer tracing is obscured by the pericardial lung interface echoes and is denoted by the white arrow heads.

Figure 3. ° refers to the mean peak expiratory to peak inspiratory percentage change in the labelled parameters ± the standard error of the mean for the entire group. A negative change corresponds to a decrease from peak expiration to peak inspiration. * refers to a comparison between the mean data for the group and the null hypothesis that there is no significant respiratory variation in the measured parameters. The shaded areas denote a range about the baseline of ±5% for the measured dimensions and ±10% for the derived volumes.
would appear to argue against the third wherein LV diastolic dimension would be expected to remain constant. In figure 4, the RV inspiratory dimension appears to increase as LV minor axis decreases. Given the complexities of right and left ventricular geometry, however, in combination with the limitations of single crystal echocardiography, this investigation cannot resolve this issue and it is therefore preferable not to speculate at any length as to the physiologic significance of the observed dimensional changes.

These data are in contrast to those of Goldblatt et al. who noted no significant change in the end-diastolic distance between LV epicardial clips during quiet respiration. One possible explanation for this discrepancy is that epicardial clips cannot accurately reflect changes in LV internal dimensions. In addition, the study used patients with a variety of cardiovascular diseases, a further contrast to the investigations utilizing normal subjects.

Alternatively, respiratory changes in echocardiographic dimensions may be partially artifactual. The echo beam traverses the LV minor axis at only one location and may not accurately reflect respiration-related changes in LV geometry. For example, there may be a coincident inspiratory increase in the LV major axis that compensates for any decrease in minor axis. Another potential artifact may relate to a respiration-related rotation of the heart relative to a fixed echocardiographic beam.

Figure 4 is from a subject with a striking inspiratory decrease in the diastolic dimension (average 5 mm or 12%) and, to a lesser degree, the systolic dimension (average 4 mm or 14%). These results could be explained if inspiration caused a rotation of the heart so that a slightly different (and smaller) minor axis was traversed by the sound beam. Such a rotational artifact would have two effects: 1) it would magnify any “real” inspiratory decrease in the diastolic dimension, and 2) in systole, because of the method used in normalizing the data, such an artifact would be mathematically amplified to a greater degree than the same artifact in diastole. That is, the artifact would be added to the numerator of a fraction with a small denominator (the expiratory systolic dimension). In this particular subject (and in nine others with decreases of more than 5% in the systolic dimension), a rotational artifact may have been present, but in 17 subjects there was no significant decrease in the systolic dimension.

This potential artifact is analogous to that recognized and corrected for by Goldblatt et al. in their fluoroscopic study, but a similar method of correcting for rotational changes of the heart relative to the single crystal echocardiographic beam does not exist. Such an artifact seems unlikely to be solely responsible for these results, particularly in the 17 subjects with a constant systolic dimension, as it is difficult to conceive of a rotational change that could selectively decrease the inspiratory diastolic dimension while leaving
the systolic dimension unchanged. Furthermore, the absence of an inspiratory change in the systolic dimension is a result that would be anticipated from a consideration of the length-tension characteristics of heart muscle under constant afterload conditions. An inspiratory increase in systolic dimension of greater than 5% is more difficult to explain, particularly since the corresponding diastolic dimensions decreased 5%, 6%, and 9% in these three subjects. A respiration-related rotation of the heart to a larger minor axis is a feasible explanation only if any “real” inspiratory decrease in the LV diastolic dimension exceeded the artifactual increase, resulting in a final net decrease in minor axis. Alternatively, these results could reflect a respiration-related resolution artifact, wherein the true systolic endocardial echo is adequately visualized only during a particular phase of respiration. The latter possibility was excluded by requiring a constant systolic wall thickness during the two phases of respiration in order to aid in the identification of a constant endocardial echo.

In summary, these data indicate that echocardiographic LV diastolic dimensions and the parameters derived from these measurements decrease significantly during quiet phasic inspiration. While the physiologic significance of these changes remains unproven, the changes are similar directionally and in order of magnitude to other studies that utilized invasive techniques to measure respiratory changes in stroke volume. The magnitude of these changes is such that they should be taken into account in any serial echocardiographic measurement of LV function in a given patient, during various hemodynamic interventions, and in any correlation between echocardiographic dimensions and other methods of assessing LV function. Furthermore, given that such variation exists in a normal population, then an even more cogent question relates to the existence, magnitude, and significance of such changes in various pathologic cardiovascular conditions.

References

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