Beat-to-beat Augmentation of Left Ventricular Function by Intraaortic Counterpulsation

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Background: Measuring the effects of intraaortic balloon counterpulsation (IABP) in single cardiac beats may permit an improved understanding of the physiologic mechanisms by which IABP improves the circulation. The objective of the study was to use transesophageal echocardiography in combination with hemodynamic measurements to test the hypothesis that IABP improves global left ventricular systolic function selectively in the IABP-augmented cardiac beats by acutely decreasing left ventricular afterload.

Methods: Twenty-seven studies in which the IABP-to-R wave trigger ratio was serially changed from 1:1, 1:2, 1:4, 0:1 (IABP off) and back to 1:1 were performed in 20 anesthetized cardiac surgical patients during IABP support. Left ventricular short-axis end-diastolic cross-sectional area, end-systolic area, mean end-systolic wall thickness, and ejection time were measured by transesophageal echocardiography at the midpapillary muscle level. Aortic pressure was measured simultaneously from the central lumen of the intraaortic balloon catheter. These measurements were used to calculate the fractional area change, end-systolic meridional wall stress, and heart rate-corrected velocity of circumferential fiber shortening. The echocardiographic and hemodynamic parameters of left ventricular preload, afterload, and systolic function immediately after balloon deflation (IABP augmented cardiac beats) were compared to the parameters measured during nonaugmented cardiac beats to determine the beat-to-beat effects of IABP on left ventricular function.

Results: IABP-augmented cardiac beats had a decreased systolic arterial pressure and end-systolic meridional wall stress and increased diastolic blood pressure, fractional area change, and velocity of circumferential fiber shortening compared to the nonaugmented cardiac beats. IABP did not cause significant beat-to-beat changes in heart rate, pulmonary artery diastolic pressure, or central venous pressure. The improvement in left ventricular systolic function associated with IABP-augmented cardiac beats correlated with the decrease in end-systolic meridional wall stress for that cardiac beat.

Conclusions: Beat-to-beat echocardiographic and hemodynamic measurements performed in anesthetized cardiac surgical patients during IABP support demonstrated improved left ventricular systolic function and decreased left ventricular systolic wall stress in the cardiac beats immediately after balloon deflation. The relationship between left ventricular systolic function and left ventricular systolic wall stress during IABP support suggests that afterload reduction was an important mechanism by which IABP instantaneously improved circulatory function in anesthetized cardiac surgical patients. (Key words: Anesthesia: cardiac. Heart: afterload; coronary artery disease; left ventricular function; ventricular assist devices. Measurement techniques: transesophageal echocardiography. Monitoring: transesophageal echocardiography.)

INTRAORTIC balloon pumping or counterpulsation (IABP) is a common method of providing temporary mechanical circulatory assist to patients undergoing cardiac operations.1-4 It is employed preoperatively or intraoperatively to support patients with coronary insufficiency or left ventricular pump failure that is refractory to medical therapy. The intraaortic balloon is positioned in the descending thoracic aorta and operates by displacing blood within the aortic lumen. Balloon inflation immediately after aortic valve closure augments the diastolic blood pressure, thereby increasing systemic and coronary artery perfusion pressure. Balloon deflation immediately before ventricular ejection decreases systolic blood pressure, thereby reducing the impedance to left ventricular ejection.

The predicted physiologic effects of intraaortic balloon counterpulsation include an increase in diastolic coronary blood flow, a decrease in left ventricular afterload, and an improvement in global left ventricular systolic function as a result of both afterload reduction and increased coronary perfusion pressure.5-8 Although clinical studies have demonstrated improvement in parameters of cardiac performance after the institution of IABP support in critically ill patients,1,2 the measurement of beat-to-beat changes in left ventricular...
preload, afterload, and systolic function to assess the effectiveness of IABP in patients undergoing cardiac operations has not been well studied. Direct real-time imaging of left ventricular contraction using intraoperative transesophageal echocardiography (TEE) provides a method to investigate ventricular function in single cardiac beats in patients requiring IABP support during cardiac operation. Measuring the effects of IABP in single cardiac beats may permit an improved understanding of the physiologic mechanisms by which IABP improves the circulation. The objective of the study was to use TEE in combination with hemodynamic measurements to test the hypothesis that IABP improves global left ventricular systolic function selectively in the IABP-augmented cardiac beats by acutely decreasing left ventricular afterload.

Methods and Materials

In an investigational protocol approved by the University of Pennsylvania Committee on Studies Involving Human Beings, 20 adult patients (10 male and 10 female) undergoing cardiac operations requiring IABP support during their operation were studied prospectively. Patients with intracardiac shunts, with left ventricular aneurysms, or in whom TEE could not be performed were excluded from the study. Seven patients receiving IABP support before surgery were studied before sternotomy and after sternal closure. Studies were performed only in patients who had a sinus or atrially paced cardiac rhythm, who had no significant valvular dysfunction at the time of the study, and who were hemodynamically stable enough to tolerate a brief period of graded weaning of IABP support.

Patients were anesthetized with a fentanyl-based general anesthetic and studied prior to sternotomy before cardiopulmonary bypass and/or after sternal closure following the separation from cardiopulmonary bypass. After intubation of the trachea, a 5-MHz TEE probe (Hewlett Packard, Hanover, MA) was inserted into the distal esophagus or stomach to image the short-axis of the left ventricle at the midpapillary muscle level. Baseline echocardiographic and hemodynamic measurements were obtained at an IABP-to-R wave trigger ratio of 1:1, then repeated serially at IABP-to-R wave trigger ratios of 1:2, 1:4, 0:1 (IABP off), and back at 1:1. The patient's hemodynamic condition was allowed to stabilize for 1–2 min before measurements were made at each IABP-to-R wave trigger ratio. No changes in patient position, intravenous fluid administration, vasoactive drug therapy, anesthetic concentration, or surgical stimulation were made during the periods of study.

All patients had dual-lumen intraaortic balloon catheters with 40-ml balloon volumes (Dataspore, Fairfield, NJ, or Kontron, Everett, MA) placed percutaneously via the femoral artery. The distal tip of the balloon catheter was positioned within the descending thoracic aorta within 2 cm from the left subclavian artery by direct imaging of the proximal descending thoracic aorta using TEE. Balloon inflation was triggered from the R wave of the electrocardiogram obtained from surface electrodes. The balloon was programmed to inflate just before the diastolic notch of the aortic pressure tracing and to deflate just before ventricular systole (fig. 1). At each IABP-to-R wave trigger ratio, an epoch was de-
fined as the minimum number of consecutive cardiac cycles starting from the first R wave used to trigger the IABP that constituted a repetitive unit (fig. 1). Cardiac beats were designated as IABP-augmented or nonaugmented based on the relationship of the cardiac beat to balloon inflation at each IABP-to-R wave trigger ratio (fig. 1).

Hemodynamic and Electrocardiographic Measurements
Pressure measurements were performed using Sorenson 47616-10 transducers (Abbott, Chicago, IL) zeroed at the patient's midaxillary line and interfaced to a Hewlett Packard Merlin model 66 monitoring system with a four-channel chart recorder. The aortic pressure was transduced from the central lumen of the intraaortic balloon catheter. The pulmonary artery and central venous pressures were obtained from an indwelling pulmonary artery catheter (Baxter, Irvine, CA). All pressures were the average obtained from corresponding cardiac beats from three separate epochs at end-expiration. The systolic arterial pressure (SBP) was defined as the peak aortic pressure during ventricular systole. Heart rate was determined from the corresponding R-R intervals. The left ventricular ejection time (LVET), determined using the hemodynamic and electrocardiographic recordings, was defined as the time between the Q wave of the electrocardiogram and the dicrotic notch of the aortic pressure waveform.

Transesophageal Echocardiography
Echocardiographic images were recorded online together with the aortic pressure tracing at a frame rate of 30 Hz on 0.5-inch videotape. The two-dimensional left ventricular short-axis TEE images were recorded simultaneously during procurement of the hemodynamic and electrocardiographic data. Echocardiographic measurements were made offline from videotape recordings using the Hewlett Packard Sonos 1500 by experienced echocardiographers (ATC, JSS, and SFJ) who were blinded to the hemodynamic data. End-diastole was defined as the frame corresponding to the largest left ventricular cross-sectional cavity area before left ventricular ejection. End-systole was defined as the frame corresponding to the smallest left ventricular cross-sectional cavity area during ventricular ejection.

Left ventricular short-axis end-diastolic cross-sectional area, end-systolic cross-sectional area (ESA), end-diastolic endocardial circumference, and end-systolic endocardial circumference for each cardiac beat were measured by manual planimetry of the area circumscribed by the leading edge of the left ventricular endocardial border. The anterolateral and posteromedial papillary muscles were excluded in the area and circumference determinations. The leading edge-to-leading edge technique was used to measure end-diastolic and end-systolic left ventricular anteroposterior cavity diameter, left ventricular anterior wall thickness, and left ventricular inferior wall thickness. All echocardiographic dimensions used for analysis were the mean of measurements performed on three corresponding cardiac beats in three separate epochs. The LVET, determined using TEE, was obtained by dividing the number of video frames between end-diastole and end-systole by the frame rate. The fractional area change (FAC) and fractional circumferential shortening were calculated using previously described formulas. The end-systolic meridional wall stress (SWS) was determined from the ESA, mean left ventricular end-systolic wall thickness (LVWT), and the SBP using the following formula:

\[
SWS \times 10^3 \text{ dynes} \cdot \text{cm}^{-2} = \frac{(0.668)(SBP)(ESA/\pi)^{1/2}}{LVWT[1 + (LVWT)/(2)(ESA/\pi)^{1/2}]}
\]

The heart rate-corrected velocity of circumferential fiber shortening (VCFC) was determined by the following formula:

\[
\text{VCFC} \ (\text{circumference} \cdot \text{s}^{-1}) = \frac{(EDD - ESD)}{[(ESD)(LVWT)(R-R \text{ interval})^{1/2}]},
\]

where

\[
EDD = (2)(\text{end-diastolic circumference}/\pi)
\]

and

\[
ESD = (2)(\text{end-systolic circumference}/\pi).
\]

A baseline description of left ventricular regional systolic wall motion abnormalities was obtained by TEE examination at the start of the study in each patient from the left ventricular short axis image at an IABP-to-R wave trigger ratio of 1:1. Systolic wall motion abnormalities in the inferior, lateral, anterior, and septal left ventricular walls were qualitatively classified as normal, hypokinetic, akinetic, or dyskinetic.

Statistical Analysis
All hemodynamic, electrocardiographic, and echocardiographic data were analyzed as continuous vari-
ables. The 27 studies performed on 20 patients were treated as independent sets of data. Statistical significance was defined as $P < 0.05$. One-way analysis of variance (ANOVA) for repeated measures was used to test whether the measured parameters changed in response to changes in the IABP-to-R wave trigger ratio. For parameters that changed significantly in response to changes in the IABP-to-R wave trigger ratio, individual comparisons of parameters between the IABP-augmented and nonaaugmented cardiac beats were performed using ANOVA contrasts for repeated measures with adjustment for multiple comparisons. A random-effects ANOVA model with subject terms included to control for the correlation induced by having multiple observations per subject was used to test for the significance of the relationship between the measures of left ventricular systolic function and afterload in response to IABP-augmentation.

Results

Seven patients were studied before sternotomy and after sternal closure; 11 were studied only after sternal closure, and 2 were studied only before sternotomy (table 1). Five patients with valvular heart disease were studied only after valve replacement. The patients ranged in age from 39 to 80 yr. All patients had at least one region of left ventricular systolic wall motion abnormality (table 1). None of the patients had left ventricular aneurysms or dyskinetic myocardial segments. IABP was instituted in 11 patients for unstable angina, 3 patients for cardiogenic shock, 1 patient for failed percutaneous balloon coronary angioplasty, and 5 patients for separation from cardiopulmonary bypass (table 2).

IABP caused significant beat-to-beat changes in SBP, SWS, ESA, FAC, and VCFC (figs. 2 and 3). IABP did not cause significant beat-to-beat changes in heart rate, pulmonary artery diastolic pressure, or central venous pressure. The end-diastolic cross-sectional area was not different between IABP-augmented and nonaugmented beats at IABP-to-R wave trigger ratios of 1:2 and 1:4 but decreased when the trigger ratio was changed from 0:1 (IABP off) to 1:1 (fig. 4). The SBP, SWS, and ESA of cardiac beats at an IABP-to-R wave trigger ratio of 1:1 decreased significantly by an average of 16%, 24%, and 20%, respectively, compared to measures obtained from cardiac beats with the IABP off. The FAC, FCS, and VCFC of cardiac beats at an IABP-to-R wave trigger ratio of 1:1 increased by an average of 24%, 17%, and 22%, respectively, compared to measures obtained from cardiac beats with the IABP off. At IABP-to-R wave trigger ratios of 1:2 and 1:4, the SBP, ESA, and SWS of IABP-augmented cardiac beats were significantly less than the measures obtained from subsequent nonaugmented cardiac beats. At IABP-to-R wave trigger ratios of 1:2 and 1:4, the FAC, FCS, and VCFC were significantly greater in the IABP-augmented cardiac beats compared to the measures obtained from subsequent nonaugmented cardiac beats.

There was a significant linear regression between echocardiographic parameters of systolic function (FAC and VCFC) and SWS, the parameter corresponding to left ventricular afterload (fig. 5). The linear regression between the change in echocardiographic parameters of systolic function in adjacent IABP-augmented and nonaugmented cardiac beats and the decrease in SWS was also significant (fig. 6). The mean improvements in the echocardiographic parameters of systolic function (ESA, FAC, and VCFC) when the IABP-to-R wave trigger ratio was changed from a ratio of 0:1 (IABP off) to a ratio of 1:1 were not different after myocardial revascularization in the subgroup of 7 patients who underwent CABG on IABP support for unstable angina (fig. 7).

Discussion

Intraoperative TEE was used in combination with hemodynamic measurements to demonstrate changes in left ventricular systolic function as a consequence of IABP. Varying the IABP-to-R wave trigger ratio during beat-to-beat measurements of ventricular function permitted isolated changes in ventricular function caused by IABP to be identified. IABP acutely improved global left ventricular systolic function and decreased left ventricular SWS. Although the study was not specifically designed to assess the contribution of increased coronary perfusion pressure to left ventricular systolic function, the findings of the study suggested that afterload reduction was an important mechanism by which IABP improved left ventricular systolic function in patients undergoing cardiac operations. The decrease in peak systolic blood pressure and SWS measured in IABP-augmented cardiac beats was consistent with the expectation that intraaortic counterpulsation produced a decrease in left ventricular afterload. Compared to the absence of IABP support, intraaortic counterpul-
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Table 1. Patient Characteristics

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Operation</th>
<th>IABP-to-R Wave Trigger Ratio of 1:1</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>LV Regional Wall Motion Abnormalities</td>
<td>FAC (%)</td>
</tr>
<tr>
<td>1</td>
<td>57</td>
<td>F</td>
<td>Redo CABG</td>
<td>Inferior hypokinesis</td>
</tr>
<tr>
<td>2</td>
<td>39</td>
<td>M</td>
<td>CABG</td>
<td>Anterior, septal, and lateral hypokinesis, inferior akinesis</td>
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<tr>
<td>3</td>
<td>70</td>
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<td>CABG</td>
<td>Inferior and septal hypokinesis</td>
</tr>
<tr>
<td>4</td>
<td>66</td>
<td>M</td>
<td>AVR</td>
<td>Anterior, septal, and inferior hypokinesis</td>
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<tr>
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<td>45</td>
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<td>Anterior and septal hypokinesis</td>
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<td>F</td>
<td>CABG</td>
<td>Inferior akinesis</td>
</tr>
<tr>
<td>7</td>
<td>63</td>
<td>M</td>
<td>MVR, CABG</td>
<td>Inferior and septal hypokinesis</td>
</tr>
<tr>
<td>8*</td>
<td>64</td>
<td>M</td>
<td>Redo CABG</td>
<td>Septal hypokinesis</td>
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<tr>
<td>9</td>
<td>44</td>
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<td>Inferior hypokinesis</td>
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<tr>
<td>10*</td>
<td>62</td>
<td>M</td>
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<td>Anterior, septal, and inferior hypokinesis</td>
</tr>
<tr>
<td>11*</td>
<td>68</td>
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<td>Redo CABG</td>
<td>Inferior hypokinesis</td>
</tr>
<tr>
<td>12*</td>
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<td>CABG</td>
<td>Inferior hypokinesis</td>
</tr>
<tr>
<td>13</td>
<td>80</td>
<td>M</td>
<td>AVR, CABG</td>
<td>Inferior and septal hypokinesis</td>
</tr>
<tr>
<td>14</td>
<td>77</td>
<td>F</td>
<td>Redo CABG</td>
<td>Inferior, septal, and lateral hypokinesis</td>
</tr>
<tr>
<td>15†</td>
<td>65</td>
<td>F</td>
<td>CABG</td>
<td>Anterior and septal hypokinesis</td>
</tr>
<tr>
<td>16†</td>
<td>69</td>
<td>F</td>
<td>CABG</td>
<td>Lateral akinesis, anterior and inferior hypokinesis</td>
</tr>
<tr>
<td>17</td>
<td>56</td>
<td>M</td>
<td>MVR, CABG</td>
<td>Anterior hypokinesis</td>
</tr>
<tr>
<td>18*</td>
<td>53</td>
<td>F</td>
<td>CABG</td>
<td>Inferior and septal hypokinesis</td>
</tr>
<tr>
<td>19*</td>
<td>49</td>
<td>F</td>
<td>CABG</td>
<td>Lateral and anterior akinesis, inferior hypokinesis</td>
</tr>
</tbody>
</table>

F = female; M = male; LV = left ventricle; FAC = short-axis fractional area change at midpapillary muscle level; IABP = intraaortic balloon counterpulsation; LVH = concentric left ventricular hypertrophy; Redo = reoperation; CABG = coronary artery bypass grafting; AVR = aortic valve replacement; MVR = mitral valve replacement.

* Studied before sternotomy and after sternal closure.
† Studied before sternotomy only.

Systolic function at an IABP-to-R wave trigger ratio of 1:1 decreased SWS by an average of 24%. Previous studies demonstrated that measurements of left ventricular wall stress were valid estimates of left ventricular loading conditions. The use of peak systolic arterial pressure and end-systolic left ventricular cross-sectional dimensions to determine SWS had been validated previously in clinical studies of patients without segmental left ventricular contraction abnormalities.

Left ventricular systolic function improved as a consequence of afterload reduction in the IABP-augmented cardiac beats. The beat-to-beat improvement in left ventricular function was detected by echocardiographic parameters of systolic function. IABP-augmented cardiac beats were associated with a decrease in left ventricular ESA, an increase in FAC, and an increase in VCFG (fig. 3). These echocardiographic parameters of left ventricular function have been demonstrated to be load-dependent and to vary indirectly with changes in left ventricular end-systolic wall stress or afterload. The afterload dependence of left ventricular systolic function would explain the regression observed between the echocardiographic measurements of left ventricular systolic function and SWS (fig. 5).

An increase in diastolic coronary artery perfusion pressure and coronary artery blood flow with alleviation of ischemic dysfunction was an alternative explanation for the observed improvement in left ventricular systolic function.

Table 2. Clinical Reason for IABP Support

<table>
<thead>
<tr>
<th>Reason for IABP</th>
<th>Patients (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Separation from CPB</td>
<td>5</td>
</tr>
<tr>
<td>Unstable angina</td>
<td>11</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>3</td>
</tr>
<tr>
<td>Failed PTCA*</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>20</td>
</tr>
</tbody>
</table>

IABP = intraaortic balloon counterpulsation; CPB = cardiopulmonary bypass; PTCA = percutaneous coronary angioplasty.

* Left anterior coronary artery dissection during attempted percutaneous coronary angioplasty.

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complete coronary occlusion.\textsuperscript{20,21} Those experimental observations suggest that only after approximately 10 cardiac beats would myocardial ischemia manifest as regional or global systolic dysfunction, a time course too prolonged to explain the beat-to-beat variations in systolic function during IABP-to-R wave trigger ratios of 1:2 and 1:4. The observation that IABP improved systolic function to the same extent before and after revascularization in the subgroup of patients with un-

tonic function as a consequence of IABP. While some studies have indicated that coronary blood flow increased in response to the augmented diastolic pressure produced by IABP,\textsuperscript{6,7,17,18} the same and other studies have failed to demonstrate an increase in coronary blood flow as a result of IABP in the presence of coronary stenosis.\textsuperscript{17,19} Although changes in coronary blood flow were not measured, it is unlikely that the phasic improvement in left ventricular systolic function observed in every other cardiac beat at the IABP-to-R wave trigger ratio of 1:2 was the consequence of changes in coronary blood flow. Experimental studies have demonstrated that left ventricular wall motion abnormalities and impaired myocardial segment shortening caused by coronary insufficiency were not instantaneous and required 5–15 s to develop, even after com-

Fig. 2. Systolic arterial pressure (SBP) and end-systolic meridional wall stress (SWS), parameters that correspond to left ventricular afterload, are decreased in intraaortic balloon counterpulsation-augmented (A) cardiac beats compared to subsequent nonaugmented (N\textsubscript{0}, N\textsubscript{1}, N\textsubscript{2}, and N\textsubscript{3}) cardiac beats. All values (n = 27) are the mean ± SE. Comparisons were performed using analysis of variance for repeated measures adjusted for multiple comparisons.

Fig. 3. Echocardiographic parameters (end-systolic cross-sectional area, fractional area change, and velocity of circumferential fiber shortening) demonstrate an improvement in left ventricular systolic function for intraaortic balloon counterpulsation-augmented (A) cardiac beats compared to subsequent nonaugmented (N\textsubscript{0}, N\textsubscript{1}, N\textsubscript{2}, and N\textsubscript{3}) cardiac beats. All values (n = 27) are the mean ± SE. Comparisons were performed using analysis of variance for repeated measures adjusted for multiple comparisons.
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Fig. 4. The pulmonary artery diastolic pressure (PAD) and central venous pressure (CVP) did not change in response to intraaortic balloon pump (IABP)-augmentation. The end-diastolic cross-sectional area (EDA) decreased at an IABP-to-R wave trigger ratio of 1:1, suggesting a short-term but not beat-to-beat decrease in left ventricular preload. All values \( n = 27 \) are mean \( \pm \) SE. *Analysis of variance (ANOVA) for repeated measures. †ANOVA for repeated measures adjusted for multiple comparisons.

stable angina suggested also that the beat-to-beat effects of IABP on systolic function could be explained primarily on the basis of afterload reduction. Although the study provided no direct evidence supporting an antiischemic effect of IABP, afterload reduction may provide a mechanism for alleviating myocardial ischemia by decreasing myocardial oxygen demand.

The decrease in left ventricular end-diastolic cross-sectional area observed during the transition from having the IABP off to an IABP-to-R wave trigger ratio of 1:1 suggests that short-term IABP support may decrease left ventricular preload. This finding was consistent with experimental studies of left ventricular failure that showed IABP acutely decreased left ventricular end-diastolic pressure. IABP did not produce beat-to-beat changes in left ventricular preload based on changes in pulmonary artery pressures, central venous pressures, or left ventricular end-diastolic short-axis dimensions at IABP-to-R wave trigger ratios of 1:2 and 1:4. Under those conditions, the increased systolic emptying in the IABP-augmented beats may have been balanced by an increased venous return to the subsequent nonaugmented beat, thereby producing no net change in the measures of left ventricular preload. Measurements of left ventricular end-diastolic pressure may have improved the ability to detect beat-to-beat changes in left ventricular preload during IABP support.

A heterogeneous patient population was chosen for study to provide data representative of the effect of IABP under typical clinical circumstances. Patients with intracardiac shunts, left ventricular aneurysms, and stenotic or regurgitant cardiac valves were specifically excluded from study because those conditions may affect the reliability of the echocardiographic and hemodynamic measures of left ventricular systolic function and afterload. Furthermore, to prevent jeopardizing patient safety, only patients who were stable enough to tolerate the brief period of IABP-to-R wave trigger ratios of less than 1:1 were studied. Studies performed before cardiopulmonary bypass and after sternal closure were treated as independent data sets because the condition of the patient, the function of the heart, the hemodynamic parameters, and pharmacologic support differed enough to be regarded as independent conditions for examining the beat-to-beat effects of IABP. Future studies examining the effects of IABP in specific disease states or patient populations or at specific times in the perioperative period may provide further insights into the mechanisms of action of IABP.

Potential limitations of clinical techniques employed to assess left ventricular afterload by estimating left ventricular SWV include the assumption that left ventricular geometry can be described as an ellipsoid. This assumption may render absolute determinations of left ventricular wall stress in individual patients with left ventricular shapes that deviate from an ellipsoid to be invalid but do not necessarily impair the ability to perform serial comparisons of afterload changes in individual patients during the hemodynamic transients produced by altering the IABP-to-R wave trigger ratio. Left ventricular short-axis measurements have been

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demonstrated to track the serial changes in left ventricular function during hemodynamic transients produced by hypovolemia even in patients with segmental regional wall motion abnormalities. Patients with dyskinetic or aneurysmal left ventricular wall segments were excluded from the study to minimize the confounding effects of severe alterations in left ventricular geometry on cross-sectional measurements of global left ventricular systolic function and meridional wall stress. The measurement of left ventricular wall stress at a single time corresponding to end-systolic represented an additional limitation of the study technique. Comparing changes in end-systolic measurements may underestimate the magnitude of afterload reduction provided by IABP because experimental studies suggest that peak left ventricular wall stress occurred in early systole. Measuring peak left ventricular systolic wall stress would require high-fidelity recordings of left ventricular pressure and the ability to determine the echocardiographic image corresponding to the end of isovolumic contraction.

As with the determination of left ventricular wall stress, determining left ventricular systolic function based on left ventricular short-axis images in patients with regional wall motion abnormalities had inherent limitations. Single cross-sectional imaging planes provide limited information about changes in left ventricular volume and ignore the contribution of longitudinal shortening during ventricular ejection. Nevertheless, experimental studies support the use of ventricular minor axis dimensions to predict left ventricular volume changes in response to alterations in external loading conditions. Myocardial fiber shortening in the plane of the minor axis is the major contributor to changes in left ventricular volume during systole. The presence of regional wall motion abnormalities not visualized in the short-axis imaging plane at the mid-papillary muscle level also may have affected the echo-

![Fig. 5. Relationship between echocardiographic parameters of left ventricular systolic function and end-systolic meridional wall stress (SWS) for individual intraaortic balloon pump-augmented and nonaugmented cardiac beats. The slope (mean ± SE) was significant, indicating that systolic function was inversely proportional to SWS. FAC = fractional area change; VCFC = velocity of circumferential fiber shortening (see text). Random-effects analysis of variance with subject terms included to control for the correlation induced by having multiple observations per subject.](image)

![Fig. 6. The change in echocardiographic parameters of left ventricular systolic function for adjacent intraaortic balloon pump (IABP)-augmented and nonaugmented cardiac beats was inversely proportional to the change in end-systolic meridional wall stress (SWS) produced by IABP. The slope is the mean ± SE. ΔFAC = change in fractional area change in response to IABP augmentation; ΔVCFC = change in velocity of circumferential fiber shortening in response to IABP augmentation; SWS = change in SWS in response to IABP augmentation. Random effects ANOVA with subject terms included to control for the correlation induced by having multiple observations per subject.](image)
cardiographic estimates of global left ventricular systolic function. The imaging plane selected for the study was chosen because it provided simultaneous views of representative myocardial regions perfused by each of the three major coronary arteries. The future development and application of real-time three-dimensional imaging techniques may provide more accurate estimations of left ventricular function and wall stress in patients with ventricular asynergy.

Beat-to-beat echocardiographic and hemodynamic measurements performed in anesthetized cardiac surgical patients during IABP support demonstrated improved left ventricular systolic function and decreased left ventricular systolic wall stress in the cardiac beats immediately after balloon deflation. Although the effects of IABP on coronary blood flow could not be assessed, the relationship between left ventricular systolic function and left ventricular systolic wall stress during IABP support suggested that afterload reduction was an important mechanism by which IABP instantaneously improved circulatory function in anesthetized cardiac surgical patients.

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