Echocardiographic Evaluation of Left Ventricular Function in Critically Ill Patients*
Dynamic Loading Challenge Using Medical Antishock Trousers
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Study objective: We hypothesized that a dynamic left ventricular (LV) evaluation during a loading challenge might enhance diagnostic capabilities of routine transesophageal echocardiography in critically ill patients and selection of therapeutic options against circulatory failure, particularly the choice between volume expansion and vasoactive agent infusion.

Design: Prospective clinical study in a group of 26 patients requiring hemodynamic support by vasoactive infusion because of low systemic arterial pressure (< 90 mm Hg by invasive monitoring) during mechanical ventilation.

Setting: University hospital ICU.

Patients: Patients required respiratory support for an episode of acute respiratory failure of various causes or for an episode of coma. They were studied by transesophageal echocardiography during mechanical ventilation in the controlled mode, before and during a loading challenge made using the legs compartment of medical antishock trousers inflated at 80 mm Hg.

Measurements: A short-axis view of the left ventricle was obtained by a transgastric approach, and end-diastolic and end-systolic areas were measured. LV stroke area (LVSA) and LV fractional area contraction (LVFAC) were calculated.

Results: Changes in LV echocardiographic measurements permitted separation of the patients into two groups. In nine patients (group 1), LVSA, used as an index of stroke output, was significantly increased during the challenge, together with a significant increase in LV end-diastolic area, suggesting preload improvement by the challenge. Conversely, in 17 patients (group 2), LVSA was significantly reduced by the challenge, together with a significant decrease in LVFAC, suggesting a negative effect of increased afterload by the challenge.

Conclusion: Study of the changes in LV dimensions during loading challenge in hemodynamically unstable patients was used to evaluate the balance between the adequacy of preload and the ability of the heart to pump against an increased load, and might thus guide hemodynamic support.

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Key words: circulatory failure; loading challenge; medical antishock trousers; transesophageal echocardiography

Abbreviations: LV = left ventricular; LVD = LV diameter; LVEDA = LV end-diastolic area; LVEDA = LV end-diastolic area; LVESA = LV end-systolic area; LVFAC = LV fractional area concentration; LVr = LV systolic wall stress; LVSA = LV stroke area; MAST = medical antishock trousers; MR = mitral regurgitation; SAP = systolic arterial pressure; TEE = transesophageal echocardiography

Patients hospitalized in ICUs often require permanent or intermittent hemodynamic support, which is achieved by an adequate combination of volume expansion and infusion of vasoactive agents guided by hemodynamic monitoring. As a general rule, hemodynamic monitoring compares an index of left ventricular (LV) preload with an index of LV performance.1,2 The first clinical application was central venous monitoring associated with systemic arterial pressure measurement.3 Later on, a more sophisticated approach involving right heart catheterization was used for simultaneous measurement of pulmonary artery occlusion pressure and cardiac output.4 However, this invasive approach was re-

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recently questioned, and a noninvasive alternative, if available, should be preferred.

During the last 10 years, we have substituted bedside echocardiography for right heart catheterization in our ICU. This noninvasive technique provides a great deal of reliable information about the hemodynamic status of a given patient, thereby simplifying therapeutic decisions. The aim of the present study was to use a load challenge to complete baseline hemodynamic echocardiographic data by means of a dynamic study. During routine examination by transesophageal echocardiography (TEE), this challenge was made in our unit by inflating the legs compartment of medical antishock trousers (MAST).

**Materials and Methods**

We studied 26 patients (15 men, 11 women; mean age, 60 ± 14 years) who required mechanical ventilation during an acute episode of respiratory failure of various causes (23 patients), or because of an episode of coma (3 patients), and who exhibited hemodynamic instability during respiratory support (systolic arterial pressure [SAP] < 90 mm Hg by invasive monitoring). This instability required prolonged hemodynamic monitoring in order to choose and adapt the most appropriate hemodynamic support required to maintain SAP > 90 mm Hg. This goal was initially achieved by vasoactive agent infusion.

**Protocol**

During the study, patients were under controlled ventilation (MA-2; Bennet; Carlsbad, CA), with a tidal volume of 8 mL/kg, a respiratory frequency of 12 to 15 breaths/min, and an end-inspiratory pause of 0.5 s, and zero end-expiratory pressure. At this time, all patients, except those who were comatose, were sedated with midazolam, 10 mg/h, and sufentanil, 30 μg/h. Hemodynamic support by continuous infusion of a vasoactive agent ensured an SAP > 90 mm Hg. This support was not modified during the study, but was eventually modified later, in line with the conclusions of the study.

After completion of a baseline set of measurements, a second set was obtained during application of the legs compartment of MAST inflated at 80 mm Hg to briefly increase LV load. A total MAST inflation time of < 2 min was required to obtain the second set of measurements.

Rapid volume challenge is currently used to test the hemodynamic efficiency of increasing central blood volume in patients with circulatory failure. However, this classical challenge may cause adverse effects in some patients with an already increased central blood volume, and the additional increase produced by volume challenge cannot be quickly removed. Conversely, MAST can be used to increase central blood volume transiently, an effect that is immediately reversed by MAST deflation. Thus this protocol, which was used to assess an acute situation, and which lasted < 2 min, was considered as part of routine clinical practice and no informed consent was obtained from the patients’ next of kin.

**Measurements**

Systemic arterial pressure was measured from an indwelling radial catheter and SAP was used for LV wall stress calculation. Heart rate was obtained from patient’s ECG monitoring. Average central venous pressure for the whole group was 13 ± 2 mm Hg.

Echocardiographic measurements were performed with a Hewlett-Packard machine (model HP 77020 A; Hewlett-Packard; Andover, MA). A standard TEE transducer (5 MHz, transverse plane; Hewlett-Packard) was first used in each patient to check mitral valve competency and the possible presence of regurgitation, using color Doppler echocardiography. The transducer was then positioned to give a short-axis cross-sectional view of the left ventricle at the mid-papillary muscle level. Using a side port of the tracheal tube, airway pressure was displayed on the screen of the echo-Doppler apparatus to accurately locate cardiac events in the respiratory cycle. Echocardiographic images were recorded and reviewed for single-frame, stop-motion analysis. The end-diastolic frame was selected at the peak of the R wave on simultaneous ECG recording, and the end-systolic frame was defined as the smallest ventricular dimension during the last half of the T wave. Using a microcomputer interfaced with the videotape player, stop-motion frames at end-diastole and end-systole, obtained at end-expiration, were displayed on the microcomputer screen to digitize the endocardial outlines of the left ventricle. End-diastolic and end-systolic areas were automatically processed. LV diameter (LVD) and wall thickness at end-systole were measured from M-mode recordings. LV stroke area (LVSA) was calculated as LV end-diastolic area (LVEDA) minus LV end-systolic area (LVESA). LV fractional area contraction (LVFAC) was calculated as LVSA divided by LVEDA. LV systolic wall stress (LVσ) was calculated from the formula of Reichek et al: $\sigma = 0.334 \frac{SAP(LVD)}{h(1 + h/LVD)}$, where h indicates wall thickness at end-systole.

**Statistical Analysis**

Statistical significance was tested using the Statgraphics version 5 package (Manugistics; Rockville, MD). Data are expressed as mean ± SD. Comparisons between baseline and MAST measurements were performed using a Wilcoxon signed ranks test. A test giving a p < 0.05 was considered as statistically significant.

**Results**

Clinical data, including the reasons for respiratory support, the presence of an underlying disease likely to affect cardiovascular status, and the hemodynamic support required to maintain SAP > 90 mm Hg, are summarized in Table 1.

An example of an echocardiographic short-axis view of the left ventricle by the transgastric approach (Figure 1) illustrates the endocardial tracing of the LV cavity areas including papillary muscles.

Hemodynamic changes are illustrated in Figure 2, and average values are summarized in Table 2. MAST inflation produced significant changes in echocardiographic measurements. Nine patients, individualized as group 1, exhibited an increase in LVEDA of > 10%. In this group, LVEDA also increased significantly, whereas LVESA and LVFAC were unchanged. In the 17 remaining patients, individualized as group 2, LVSA was significantly reduced during MAST inflation. In
this subgroup, LVESA was significantly increased and LVFAC was significantly decreased.

**DISCUSSION**

MAST inflation is expected to change both LV preload and afterload.\(^7,8\) By reducing the distensibility of leg vessels, MAST inflation increases mean circulatory pressure and displaces blood from the peripheral compartment to the central compartment. The result is an increase in central blood volume, the filling reserve for the left ventricle, thereby improving preload. By compressing the leg arteries, MAST inflation raises arterial pressure, which in turn increases LVσ and afterloads the left ventricle. Thus, MAST inflation was used to investigate the balance between adequacy of preload and ability of the heart to pump against an increased afterload. Our hypothesis was that in hemodynamically unstable patients, the effect on stroke output of a sudden increase in LV load could differentiate patients with inadequate preload from those with underlying myocardial incompetency, as described in the past using an invasive procedure.\(^1,2\)

In response to an increase in central blood volume, a normally filled left ventricle is not expected to dilate because it operates close to the steep portion of its pressure-volume relation.\(^9\) This is more true for a chronically dilated left ventricle, which actually operates on the steep portion of its pressure-volume relation. Thus, a substantial rise in LV end-diastolic pressure may occur without any discernible increase in LV end-diastolic dimension.\(^10\) However, if LV preload is abnormally reduced, any increase in cen-

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**Table 1—Clinical Data in the Two Groups**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group 1 (n = 9)</th>
<th>Group 2 (n = 17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Need for respiratory support</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute respiratory failure</td>
<td>8</td>
<td>14</td>
</tr>
<tr>
<td>Coma</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Underlying disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>COPD</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Sepsis</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>MR (mild)</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>MR (moderate)</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>Hemodynamic support†</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dobutamine, 5 to 15 μg/kg/min</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Dopamine, 6 to 14 μg/kg/min</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>Epinephrine, 0.1 to 0.7 μg/kg/min</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Norepinephrine, 0.7 to 1.6 μg/kg/min</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

*Data are presented as No.
†Six patients had two drugs simultaneously.

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**Figure 1.** An example of echocardiographic short-axis view of the left ventricle at the papillary muscle level at end-diastole (left) and end-systole (right), at baseline (top) and after MAST inflation (bottom).
tral blood volume may significantly increase LV end-diastolic dimension. MAST has been shown to be an efficient means of restoring adequate LV preload during positive end-expiratory pressure application, and in the present study, it separated our patients into two groups. In group 1, MAST inflation increased LVSA, an index of stroke output, through an increase in LVEDA, an index of preload, suggesting that these patients had a relatively insufficient central blood volume at baseline. In group 2, LVSA decreased in response to MAST, resulting in an increased LVESA. A parallel increase in LVEDA was expected, but was observed in only 7 patients, whereas LVESA was unchanged in 10 patients despite LVESA enlargement, suggesting that LV distensibility was reduced by an increased LV preload.

An increase in LV$_\sigma$ by MAST inflation also resulted in opposite effects in groups 1 and 2. The magnitude of the effect of an increased afterload depends on baseline LV function. Whereas a normal left ventricle is, within a large range,
insensitive to an increase in afterload, a slight increase is enough to worsen the performance of a potentially failing left ventricle. In group 1, LVFAC, an index of LV systolic function, was not affected by MAST inflation, while in group 2, LVFAC significantly decreased. These results suggested that in group 1, patients had normal or near-normal LV systolic function, whereas those in group 2 had underlying depressed LV systolic function.

The practical value of our loading challenge was identification of preload insufficiency or myocardial incompetency in individual patients, whereas baseline echocardiographic data could not characterize the patients with the same accuracy. MAST inflation unmasked preload defect in all group 1 patients, whatever the baseline value of LVEDA. This finding illustrates the fact, in patients who already had a dilated left ventricle at baseline, a preload defect may be present despite increased diastolic dimensions. On the other hand, in group 2, MAST inflation unmasked underlying depressed systolic function, whatever the baseline value of LVFAC. Such a finding was expected because LVFAC alone is not a reliable index of LV systolic function. When the LV is unloaded, as in the case of a septic context that is associated with a fall in systemic arterial resistance, LVFAC may remain within the normal range despite potentially reduced LV pumping ability.

Several possible limitations of our study should be examined. LVSA correlates with invasive determination of stroke volume in experimental and clinical studies. However, use of LVSA as an index of stroke output requires highly accurate echocardiographic measurements to detect a 10% change in LV dimensions, as in the present study. In patients receiving mechanical ventilation, intraobserver and interobserver variability in LVSA measured by TEE in our unit is < 8%, provided it is measured at the same moment of the respiratory cycle. Such accuracy may be unachievable by a transthoracic approach. In the future, we plan to replace LVSA by the Doppler echocardiographic pulmonary or aortic flow velocity-time integral, which is easy to obtain by a transthoracic approach. A second theoretical limitation may arise due to the presence of mitral regurgitation (MR) in four patients. In this case, LVSA reflects the sum of anterograde and retrograde flows, and changes in LVSA during loading challenge are difficult to interpret. We neither observed occurrence of MR caused by MAST nor attempted to quantify MR change by MAST when present at baseline. However, exclusion of patients with MR, most of them being in group 2, would not have influenced our results: in this group 2, LVSA was reduced during loading challenge, whereas a significant effect of MR would paradoxically have increased LVSA. Perhaps more questionable is the fact that the study was performed in patients whose BP was normalized by hemodynamic support. Despite this normalization, loading challenge produced detectable echocardiographic changes. One might hypothesize that vasoactive infusion had not totally normalized hemodynamic status. Also, vasoactive infusion might have markedly altered baseline hemodynamic status, but we did not attempt to stop hemodynamic support during echocardiographic studies for fear of inducing severe hypotension.

In conclusion, we propose a noninvasive dynamic test that investigates, in an individual patient, the balance between adequacy of preload and the ability of the heart to pump against an increased afterload. This test could identify the prevailing factor of hemodynamic instability in critically ill patients and thereby might have therapeutic implications. By assessing its effect on a stroke output index, LV preload defect or depressed systolic function could be separated.

**References**

2 Ross J, Braunwald E. The study of left ventricular function in man by increasing resistance to ventricular ejection with angiotensin. Circulation 1964; 29:739–749
9 Parker J, Case R. Normal left ventricular function. Circulation 1979; 60:4–11
12 Cohn J, Franciosa J. Vasodilator therapy of cardiac failure.