Effect of the mechanical ventilatory cycle on thermodilution right ventricular volumes and cardiac output

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Groeneveld, A. B. Johan, Remco R. Berendsen, Anton J. Schneider, Ioannis A. Pneumatikos, Leo A. Stokkel, and Lambertus G. Thijs. Effect of the mechanical ventilatory cycle on thermodilution right ventricular volumes and cardiac output. J Appl Physiol 89: 89–96, 2000.—The purpose of this study was to evaluate right ventricular (RV) loading and cardiac output changes, by using the thermodilution technique, during the mechanical ventilatory cycle. Fifteen critically ill patients on mechanical ventilation, with 5 cmH₂O of positive end-expiratory pressure, mean respiratory frequency of 18 breaths/min, and mean tidal volume of 708 ml, were studied with help of a rapid-response thermodilution pulmonary artery catheter, allowing 5-ml room-temperature 5% isotonic dextrose thermodilution measurements of cardiac index (CI), stroke volume (SV) index, RV ejection fraction (RVEF), RV end-diastolic volume (RVEDV), and RV end-systolic volume (RVESV) indexes at 10% intervals of the mechanical ventilatory cycle. The ventilatory modulation of CI and RV volumes varied from patient to patient, and the interindividual variability was greater for the latter variables. Within patients also, RV volumes were modulated more by the ventilatory cycle than CI and SV index. Around a mean value of 3.95 ± 1.18 l·min⁻¹·m⁻² (= 100%), CI varied from 87.3 ± 5.2 (minimum) to 114.3 ± 5.1% (maximum), and RVESV index varied between 61.5 ± 17.9 ml/m² during the ventilatory cycle. The variations in the cycle exceeded the measurement error even though the latter was greater for RVEF and volumes than for CI and SV index. For mean values, there was an inspiratory decrease in RVEF and increase in RVESV, whereas a rise in RVEDV largely prevented a fall in SV index. We conclude that cyclic RV afterloading necessitates multiple thermodilution measurements equally spaced in the ventilatory cycle for reliable assessment of RV performance during mechanical ventilation of patients.

right ventricular performance; ejection fraction catheter; critically ill; reliability of thermodilution

IN CRITICALLY ILL PATIENTS, the measurement of right-sided cardiac output by using a pulmonary artery catheter and the thermodilution technique is commonly done (12). The catheter can also be equipped with a rapid-response thermistor, allowing bedside measurement of right ventricular (RV) ejection fraction and volumes (4, 6–10, 17, 25, 29, 35, 36). These latter measurements may also be of mechanistic, diagnostic, therapeutic, and prognostic significance in critically ill patients (4, 7, 12, 17, 29, 35, 36). For instance, RV end-diastolic volume may be a better predictor of preload-recruitable stroke volume by a fluid challenge than filling pressures so that a high volume may preclude a further rise in cardiac output with fluids, independently of filling pressures (3, 4, 7, 35, 36).

Nevertheless, the usefulness of pulmonary artery catheter insertion and thermodilution measurements of cardiac output and, especially, RV volumes has been doubted, partly because of insufficient accuracy, reproducibility, and predictive value for a response to fluid loading, particularly during mechanical ventilation (7, 30, 35, 36). Furthermore, there may be only partial agreement with RV volume measurements by other techniques in patients (9, 10, 17, 27, 36). Measurements are mostly performed at one phase in the mechanical ventilatory cycle, i.e., at the end of expiration, believed to be associated with the greatest reproducibility compared with injections at other phases of the ventilatory cycle, and outliers are usually excluded (1, 3, 4, 9, 10, 17, 20, 25–29, 31, 32, 35, 36). However, the timing of injectates in the ventilatory cycle is known to affect cardiac output measurements, irrespective of measurement errors, possibly via the ventilatory modulation of RV loading associated with cyclic changes in airway pressure and lung volume during mechanical ventilation (1, 10, 12, 14, 15, 17, 18, 24, 30, 32, 33).

Authors have recommended that, for a reliable estimation of mean thermodilution cardiac output, no specific phase in the ventilatory cycle should be selected, and that the best estimation resulted from averaging measurements at three or four equally spaced intervals in the cycle (1, 13–15, 24, 30, 33). Even though there are two (echocardiographic and thermodilution) studies suggesting changing RV volumes during the ventilatory cycle in mechanically ventilated patients, it is unclear how the modulation affects thermodilution RV ejection fraction (RVEF) and volumes (1, 18). Some
authors advised measurement of RV volumes at apnea, even if this is not representative of RV performance during mechanical ventilation (1, 19). Finally, the potential mechanism, i.e., RV pre- or afterload changes, responsible for cardiac output modulations is unclear. In fact, lung inflation could increase RV afterload and volumes, or it could reduce RV preload and volumes and thereby cardiac output, as may occur during incremental positive end-expiratory pressure (PEEP) (10, 13, 16–18, 25, 34).

In consideration of the above data, we hypothesized that changes in RV loading, as assessed from thermodilution volume measurements, are responsible for the cardiac output modulation during the mechanical ventilatory cycle. Moreover, we wanted to quantify the effect and to assess its impact on reliable thermodilution measurements in the mechanical ventilatory cycle.

**METHODS**

**Patients.** Informed consent was obtained from patients’ relatives, and the protocol was approved by the hospital Committee on Ethics. We consecutively studied 15 critically ill patients who were in the surgical intensive care unit and were on continuous volume-controlled positive-pressure ventilation (Siemens Servo 900B, Siemens Elema, Stockholm, Sweden) because of acute respiratory insufficiency. All patients were on 5 cmH₂O of PEEP, at an inspiratory time of 25%, an end-inspiratory hold of 10%, and an expiratory time of 65% of the ventilatory cycle. All patients were sedated with continuous intravenous infusion of fentanyl and midazolam. The patients, without known valvular incompetence, had sinus rhythm and were hemodynamically stable. A radial artery catheter had been inserted for measurement of the mean arterial blood pressure (mmHg). A thermodilution pulmonary artery catheter, equipped with a rapid-response thermistor (model 93A-431H-7.5F, Baxter Edwards, Santa Ana, CA; response time 50 ms) and intracardiac electrodes, was inserted percutaneously via the jugular or subclavian vein until the inflated balloon wedged in a pulmonary artery and the proximal injectate port recorded RV pressure. The catheter was withdrawn thereafter to locate the injectate port just above the tricuspid valve (31). The port was located 21 cm from the tip. Hemodynamically significant tricuspid regurgitation was ruled out in each patient on the basis of absence of V waves in the right atrial pressure recording (27). The injectate temperature was measured by an in-line temperature probe, distally from the injection site (model 93-600 CO-set, Baxter Edwards). The rapid-response thermistor, analog electrocardiograph signal, and the injectate temperature probe were interfaced to the REF-1 computer for signal processing according to in-built algorithms (Baxter Edwards; Refs. 7, 9). The heart rate (HR) was determined from the electrocardiograph signal, and the computer detected the R waves. The first-order exponential downslope of the thermodilution curve was used by the computer to calculate the residual fraction (RF) from the relationship between successive temperature plateaus synchronized to the R wave (Fig. 1). The successive values were averaged, and the RVEF was calculated as 1 – RF. Cardiac output was calculated by integrating the temperature change of the blood. Cardiac index (CI) is cardiac output divided by body surface area calculated from height and weight. Stroke volume (SV) index is derived from CI/HR. The RV end-diastolic volume (RVEDV) index (ml/m²) was calculated as SV index/RVEF.

The RV end-systolic volume (RVESV) index was calculated as RVEDV index – SV index.

**Protocol.** Demographic and clinical features were recorded. The Simplified Acute Physiology Score and Lung Injury Score (LIS) were assessed (11, 22). The latter ranges from 0
Table 1. Patient characteristics

<table>
<thead>
<tr>
<th>Age, yr</th>
<th>62.3 ± 12.6</th>
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<tr>
<td>Men/women</td>
<td>11/4</td>
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<tr>
<td>Simplified acute physiology score</td>
<td>11.8 ± 5.1</td>
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<tr>
<td>Lung injury score</td>
<td>1.65 ± 0.45</td>
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</table>

Conditions requiring intensive care
- Major vascular surgery and complications: 3
- Acute hemorrhagic pancreatitis and complications: 3
- Major large bowel surgery: 2
- Major esophageal/gastric surgery: 3
- Sepsis: 4

Use of vasoactive drugs
- Dobutamine: 3

Temperature, °C: 37.6 ± 1.2

Hospital mortality: 11 (73)

Values are means ± SD or number with percent in parentheses.

**RESULTS**

**General.** Table 1 shows the main features of the patients. All patients had acute lung injury, and one of them had adult respiratory distress syndrome (LIS = 2.5). Table 2 describes baseline global hemodynamic and respiratory variables.

**Modulation of RV performance in the ventilatory cycle.** Figure 2 shows mean and individual CIs as a function of the ventilatory cycle, whereas Fig. 3 shows the absolute mean values of the variables in the ventilatory cycle. The intraindividual variability of RV volumes was greater than that of SV or CI, as judged from higher minimum and maximum values and CVs, indicating that the ventilatory cycle modulated RV volumes to a greater extent than SV and CI.

The interindividual variation (Figs. 2 and 3) did not differ among phases, but it differed among the variables studied (P < 0.001) so that, for instance at 30% of the cycle, the interindividual CV was 8.1 for CI and 24.1% for RVESV index. This indicates less predictability among patients of the modulation of RVESV index than of CI by the ventilatory cycle. Indeed, at 100% of the cycle (end expiration), the CI ranged between 86 and 120%, SV index between 86 and 117, RVEF between 88 and 128, RVEDV index between 84 and 115, and RVESV index between 64 and 124% of mean values in the cycle. Neither the minimum/maximum values (amplitude) nor the mean values at 0 and 100% of the cycle (phase of modulation) for CI, RVEF, and RV volumes related to the absolute level of the hemodynamic variables, including mean pulmonary arterial pressure, or to respiratory variables such as the tidal

**Table 2. Baseline variables**

<table>
<thead>
<tr>
<th>Hemodynamics</th>
<th>97 ± 21</th>
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<tbody>
<tr>
<td>Heart rate, beats/min</td>
<td></td>
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<tr>
<td>Mean arterial pressure, mmHg</td>
<td>78.3 ± 14.5</td>
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<tr>
<td>Mean pulmonary arterial pressure, mmHg</td>
<td>25.0 ± 5.3</td>
</tr>
<tr>
<td>Pulmonary artery occlusion pressure, mmHg</td>
<td>13.7 ± 2.7</td>
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<tr>
<td>Right atrial pressure, mmHg</td>
<td>8.9 ± 4.6</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Respiration</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Tidal volume, ml</td>
<td>708 ± 153</td>
</tr>
<tr>
<td>Respiratory rate, breaths/min</td>
<td>15 ± 3</td>
</tr>
<tr>
<td>Peak inflation pressure, cmH₂O</td>
<td>32 ± 7</td>
</tr>
<tr>
<td>Positive end-expiratory pressure, cmH₂O</td>
<td>5</td>
</tr>
<tr>
<td>Total static respiratory compliance, ml/cmH₂O</td>
<td>28.5 ± 6.9</td>
</tr>
<tr>
<td>Arterial Pco₂, Torr</td>
<td>34 ± 3</td>
</tr>
<tr>
<td>Arterial Po₂, Torr</td>
<td>92 ± 24</td>
</tr>
<tr>
<td>Arterial Pco₂/Po₂, Torr</td>
<td>0.42 ± 0.08</td>
</tr>
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</table>

Values are means ± SD or number. FIO₂, inspiratory O₂ fraction.
volume, frequency, compliance, oxygenation ratio, or LIS.

At 80 and 90% of the cycle, the group mean CI, normalized for the mean in the ventilatory cycle in each patient, was lower than 100% (Fig. 2; \( P < 0.05 \)). The modulation of SV index was not statistically significant, however (Fig. 3). There was a <100% RVEF in the ventilatory cycle at 20 and 40% of the cycle (\( P < \))

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Fig. 2. Modulation of cardiac index (means ± SD) during the mechanical ventilatory cycle (A) and for individual patients (B and C for 7 and 8 patients, respectively) showing interindividual variation in phase and amplitude. Values were normalized for individual means during the cycle. Lines are polynomial fits.

Fig. 3. Right ventricular stroke volume index (A), ejection fraction (B), end-diastolic volume index (C), and end-systolic volume index (D) as a function of the mechanical ventilatory cycle. Values are means ± SD for individual values normalized for individual means during the ventilatory cycle. Solid line, sinusoid that fitted to the data.
Table 4. Right ventricular volumes and cardiac output obtained by multiple room-temperature injections in the mechanical ventilatory cycle

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean Absolute Value in Ventilatory Cycle</th>
<th>% Mean in Cycle</th>
<th>Coefficient of Variation in Cycle,* %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac index, l · min⁻¹ · m²</td>
<td>3.95 ± 1.18</td>
<td>114.3 ± 5.1</td>
<td>8.3 ± 2.7</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>97.0 ± 2.1</td>
<td>107.0 ± 10.1</td>
<td>3.8 ± 3.4</td>
</tr>
<tr>
<td>Stroke volume index, ml/m²</td>
<td>41.7 ± 12.4</td>
<td>113.2 ± 5.3</td>
<td>8.1 ± 2.6</td>
</tr>
<tr>
<td>RV ejection fraction, %</td>
<td>44.9 ± 10.6</td>
<td>126.6 ± 12.7</td>
<td>15.6 ± 7.3</td>
</tr>
<tr>
<td>RV end-diastolic volume index, ml/m²</td>
<td>97.0 ± 21.8</td>
<td>128.4 ± 17.3</td>
<td>16.0 ± 8.4</td>
</tr>
<tr>
<td>RV end-systolic volume index, ml/m²</td>
<td>55.1 ± 17.9</td>
<td>149.8 ± 33.9</td>
<td>29.0 ± 15.8</td>
</tr>
</tbody>
</table>

Values are given in %. n, No. of subjects.

0.05) and a rise in RVEDV and RVESV indexes at 20 and 40% (P < 0.05), followed by a fall, compared with 100%, at 70 and 90% of the cycle (P < 0.05). There were weak correlations (for pooled normalized data) between SV and RVEF changes (r = 0.28, P < 0.005) and between SV and RVEDV index changes (r = 0.20, P < 0.01) in the ventilatory cycle, whereas modulation of CI was largely caused by SV index changes (r = 0.86, P < 0.001), rather than by HR changes (r = 0.28, P < 0.005). Changes in RVEF inversely correlated to RVEDV (r = -0.84, P < 0.001) and RVESV (r = -0.69, P < 0.001) so that the latter two interrelated positively (r = 0.69, P < 0.001). This indicates that the ventilatory modulation of CI was largely caused by changes in SV index, and that changes in both RVEF and RVEDV contributed to changes in SV index. Hence, ventricular dilation, i.e., a rise in RVEDV prevented a fall in SV during a fall in the EF of the RV contracting toward an increased RVESV index.

Error analysis. At a measurement error shown in second column of Table 4, and the mean observed CVs of Table 3, it was calculated (see formula in Calculations and statistical analyses) that the mean CV of ventilatory modulation, independent of measurement error, was 7.2% for CI, 6.4% for SV index, 13.7% for RVEF, 13.5% for RVEDV and 24.9% for RVESV indexes. Otherwise, the measurement error did not depend on the phase in the mechanical ventilatory cycle. For all ventilatory phases together and after correction for measurement error (second column of Table 4), the mean CV for interindividual variation in ventilatory modulation was 6.5% for CI and 24.4% for RVESV index. The above data indicate that the ventilatory cycle modulated RV volumes more than CI and that interindividual differences in the modulation were also greater for the former, irrespective of measurement error.

Prediction of minimum number of at-random measurements for reliable RV volume assessments. In Table 5, the minimum number of at-random measurements for each variable during the ventilatory cycle, necessary for an estimate within certain limits of the mean value, is shown. Five to eight measurements are necessary, at minimum, to reliably assess the RVEF, RVEDV, and RVESV and four for measurements of CI and SV index.

**DISCUSSION**

We show that the modulation of RV volumes and CI by the mechanical ventilatory cycle is greater than the measurement error and that the modulation of RV volumes is greater than that of CI. The difference in modulation between patients is greater for RV volumes than for CI. As judged from group means, RV afterload may rise during lung inflation and this may induce a fall in RVEF and rise in RVESV. A fall in SV is largely prevented by a rise in RVEDV, thereby explaining greater modulation of RV volumes than of CI in the mechanical ventilatory cycle. Because of ventilatory modulation of RV volumes and interindividual differences herein, assessment of RV performance by thermodilution requires multiple determinations at equally spaced intervals, or at least eight at random injections, in the ventilatory cycle.

Our results partly agree with those obtained by other investigators. The relatively large injectate volumes and high respiratory rates, which decrease the CI modulation, may partly explain less ventilatory modu-

Table 5. Number of at-random measurements necessary to yield RV variables with 95% or greater chance to deviate from average within 10, 15, and 30% limits

<table>
<thead>
<tr>
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<th>10% Limits</th>
<th>15% Limits</th>
<th>30% Limits</th>
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</thead>
<tbody>
<tr>
<td>Cardiac index</td>
<td>3.2 ± 2.3</td>
<td>2.2 ± 1.4</td>
<td>1.3 ± 0.8</td>
</tr>
<tr>
<td>Stroke volume index</td>
<td>3.9 ± 3.3</td>
<td>2.5 ± 2.0</td>
<td>1.6 ± 1.1</td>
</tr>
<tr>
<td>RV ejection fraction</td>
<td>5.1 ± 2.7</td>
<td>3.7 ± 2.2</td>
<td>1.7 ± 0.9</td>
</tr>
<tr>
<td>RV end-diastolic volume index</td>
<td>5.3 ± 2.6</td>
<td>3.7 ± 2.1</td>
<td>1.9 ± 1.0</td>
</tr>
<tr>
<td>RV end-systolic volume index</td>
<td>7.6 ± 2.1</td>
<td>6.0 ± 2.8</td>
<td>3.3 ± 2.2</td>
</tr>
</tbody>
</table>

Values are means ± SD.
loration of CI in our study than in the studies by Assmann (1) and Jansen et al. (12–15). Our finding of the tendency for an expiratory fall in CI agrees with the literature (12–15, 19). Even though positive-pressure inflation generally decreases right-sided thermodilution cardiac output, the modulation may be dissimilar among patients in phase and amplitude so that in some patients CI may also increase during lung inflation (1, 12–15, 24, 30). Hence, no specific phase in the ventilatory cycle could be selected for a reliable estimation of mean CI over the ventilatory cycle in all patients. The phase and amplitude of the modulation by the ventilatory cycle may depend on the volume status and absolute blood flow on the one hand and on respiratory variables on the other (1, 12, 13, 15, 30). The fact that modulation may be affected by multiple factors may explain why we could not predict the interindividual differences in hemodynamic modulation by the ventilatory cycle in our patients.

Our data extend those obtained by Assmann et al. (1). They showed that the mechanical ventilatory cycle modulated RVEF and volumes more than CI, when assessed at 0.25 equally spaced fractions of the cycle (1). The assessment of RVEF and volumes by thermodilution was more reproducible during apnea than during mechanical ventilation, and the modulation was lower at higher respiratory rates (1). The intrindividual variability of RVEDV was 11.6% at a respiratory rate of 16 breaths/min (1). The higher variability at a comparable respiratory rate in our study can be explained by the larger number of measurements in the ventilatory cycle and lower injectate volumes (5 ml) than in their study (10 ml). The 5-ml boluses were used to limit fluid overload.

The measurement error for CI in our study may be somewhat lower than that reported before, in which repeated (manual) injections at the same phase in the ventilatory cycle were associated with a CV of 5–10%. Manual injections, however, may be more erroneous than automated ones (1, 10, 23, 29, 33). In vitro, the measurement error of CI and SV may be ~3%, whereas the error of RVEF, RVEDV, and RVESV ranges between 5 and 7% (8). In vivo also, repeated manual (phase-selected) injections in the mechanical ventilatory cycle have revealed greater error for RVEF and volumes than for CI measurements, in agreement with our results (1, 10, 20, 23, 26, 28, 29). In agreement with other investigators (1, 12, 14, 15, 30), we show that, if injections at equal intervals in the ventilatory cycle are impossible, averaging at least four random measurements in the cycle is an adequate strategy to estimate mean CI reliably in patients on mechanical ventilation. Because of greater modulation and measurement error, the minimum number of at-random determinations, necessary to yield an estimate of mean RVEF and volumes over the ventilatory cycle within a certain error, was higher than for CI assessments.

The group means over the ventilatory cycle suggest that lung inflation resulted in a rise in RVEDV and RVESV indexes after a rise in RV afterload, even though the actual thermodilution measurements of RV volumes may have taken place some time after inflation. A delay between lung inflation and actual measurements after injection implies that the rise in volumes could also have been caused by increased filling of the RV, after a reduced intrathoracic pressure and increased venous return, during expiration. This is unlikely, however, because increased filling would not decrease the ejection fraction and would tend to increase the SV (7, 10). This suggests that the volumes measured from injections in the inspiratory phase indeed reflected inspiratory events so that a transient rise in afterload resulted in a fall in RVEF and a rise in RVEDV and RVESV, attempting to maintain SV index. The latter agrees with the transmural pressure measurements and echocardiographic data obtained by Jardin et al. (17, 18) in mechanically ventilated patients. They observed that lung inflation was associated with a rise in RV transmural pressures and volumes after a rise in afterload. Conversely, the patterns of RV volume changes in our study resemble those during preload reductions with PEEP or increases with military antishock trouser inflation (7, 25), resulting in similar decreases and increases, respectively, in RVEDV and RVESV, thereby hardly affecting SV. We cannot exclude RV contractility fluctuations during the cycle, in the absence of end-systolic volume-transmural pressure relationships (7, 17, 20).

The cyclic changes in RV volumes cannot be explained by cyclic tricuspid regurgitation. The disparate rather than parallel fall of RVEF and CI in the inspiratory phase in our study argues against cyclic tricuspid regurgitation. Alternatively, an inspiratory fall in baseline pulmonary blood temperature of ~0.01–0.02°C could lead to a temporary overestimation of ~3% of thermodilution CI (12, 15). We did not observe fluctuations in the baseline temperature, and the CI did not rise during inspiration (Fig. 1). Finally, the distance between the injectate port and thermistor relative to the tricuspid and pulmonary valves, respectively, may affect the absolute values, but not the changes, of RVEF and RV volumes (31, 33).

Although experiments in animals generally show a predominant preload-lowering effect of positive-pressure ventilation, as evidenced by a fall in RV volumes (10), clinical studies, using echocardiography, nuclear angiography, or thermodilution, showed either a fall in RV preload (fall in volumes) or a rise in afterload (rise in volumes) during incremental PEEP ventilation. This seems independent of the measurement technique but dependent, in part, on baseline RV performance and thus underlying disease (4, 6, 17–19, 25). For instance, thermodilution RV volumes may decrease up to 25 cmH$_2$O of incremental PEEP in some studies and may increase in other human studies, particularly when baseline RVEF was decreased and RVEDV increased after coronary artery disease or acute lung injury (1, 6, 17–19, 25). A rise in RV afterload during lung inflation, as suggested in our study, may not exclude a fall in preload during incremental PEEP ventilation, if the former is largely determined by an increased pulmonary air volume and vascular resis-
tance and the latter by an increased intrathoracic pressure and decreased venous return (5). Finally, the effect of positive-pressure inflation may be time dependent, because an inspiratory hold in mechanically ventilated cardiac surgery patients may increase RVEDV and output only in the first 5 s (34).

In previous studies using the thermodilution method, SV, RVEF, and volumes were most often assessed at end expiration (9, 20, 25–29, 31–33, 36) and less often at end inspiration (10, 25, 26, 32). Our study indicates that this practice may have resulted in unpredictable under- and overestimations of RV performance in individual patients. It may also partly explain the reported controversy on effects of PEEP and on the value of RVEDV as a predictor of preoad-recruitable SV (3, 7, 35). In fact, fluid loading and PEEP may alter the phase and amplitude of RV volume and output modulations by the ventilatory cycle (15) so that phase-selected assessments may preclude a reliable judgment of RV volume changes. The modulation by the mechanical ventilatory cycle may also explain why phase-selected thermodilution measurements did not always correlate well to other types of measurements of RV volumes and output (2, 15).

In conclusion, the mechanical ventilation in critically ill patients modulates RV afterloading in a cyclic manner, as assessed by thermodilution, so that the cyclic variation in RV volumes is greater than that of RV output. The cyclic modulation varies among patients. This has considerable impact on the timing of thermodilution measurements in the mechanical ventilatory cycle for the reliable assessment of RV performance.

We thank Dr. Jos Twisk for help with calculations and statistics.

REFERENCES


