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

A. B. Johan Groeneveld, 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 a mean age of 3.95 years, were studied with help of a rapid-response thermistor 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 and 149.3 ± 34.1% of mean 55.1 ± 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 RVESV 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).

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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

Fig. 1. Thermodilution curves as a function of phase of start of injection of room temperature boluses in the mechanical ventilatory cycle in a representative patient. a, b, and c, Three successive temperature plateaus, synchronized to the R wave of the electrocardiogram, allowing the computation of the residual fraction from b/a and c/b. Horizontal line, baseline temperature.
Table 1. Patient characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>62.3 ± 12.6</td>
</tr>
<tr>
<td>Men/women</td>
<td>11/4</td>
</tr>
<tr>
<td>Simplified acute physiology score</td>
<td>11.8 ± 5.1</td>
</tr>
<tr>
<td>Lung injury score</td>
<td>1.65 ± 0.45</td>
</tr>
</tbody>
</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:
- Dopamine: 14
- Dobutamine: 3

Temperature, °C: 37.6 ± 1.2

Hospital mortality: 11 (73)

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

(no acute lung injury) to 4, with values above 2.5 indicating the adult respiratory distress syndrome. The mean arterial pressure, right atrial pressure, mean pulmonary arterial pressure, and pulmonary artery occlusion pressure were measured with patients in the supine position, with the midchest level as reference, and after calibration. The tidal volume, respiratory rate, and peak and plateau airway pressures were recorded. Total respiratory compliance was calculated as tidal volume/(plateau airway pressure – PEEP).

Arterial blood was obtained for determination of blood-gas values. Injection of 5 ml of 5% dextrose at room temperature was automatically performed by a phase controller and a pneumatically driven syringe after manual start of the cardiac output computer (14). The 5-ml bolus was injected at 50 psi within 1 s, by using a power injector, through the pulmonary artery catheter. After 12 s the syringe was automatically refilled. The moment of injection was dependent on a start signal given by the operator and the moment in the ventilatory cycle set on the phase controller. The latter was derived from the Siemens Servo 900B ventilator, which delivers 100 impulses during each ventilatory cycle. The phase zero was at the start of inflation. The injections (n = 11) were performed successively at 10% intervals of the ventilatory cycle, at stable hemodynamics. In 2 patients, 10 successive 5-ml injections were done at 30% of the ventilatory cycle to evaluate measurement error.

Calculations and statistical analyses.

The means of the 11 thermomodulation curve-derived parameters were calculated per patient to normalize results. The Wilcoxon rank-sum test was used to evaluate differences between group means at the phases and 100%. A group coefficient of variation (CV) per patient to normalize results. The Wilcoxon rank-sum test was used to calculate, in each patient, the minimum number of random observations that need to be made from a series of observations to get a reliable estimate of the mean value of that series, with 95% or greater chance to be within 10, 20, and 30% limits of the mean value. Data are expressed as means ± SD, and P < 0.05 was considered statistically significant.

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 means for SV, RVEF, and volumes. Table 3 shows the absolute mean values of the variables in the ventilatory cycle. The interindividual variability of RV volumes was greater than that of SV or CI, as judged from higher minimum and maximum values and CIs, 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>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate, beats/min</td>
<td>97 ± 21</td>
</tr>
<tr>
<td>Mean arterial pressure, mmHg</td>
<td>78.3 ± 14.5</td>
</tr>
<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>
</tr>
<tr>
<td>Right atrial pressure, mmHg</td>
<td>8.9 ± 4.6</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Respiration</th>
<th>Value</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>25.5 ± 6.9</td>
</tr>
<tr>
<td>Arterial Po2, Torr</td>
<td>34 ± 3</td>
</tr>
<tr>
<td>Arterial Po2, Torr</td>
<td>92 ± 24</td>
</tr>
<tr>
<td>FIO2</td>
<td>0.42 ± 0.08</td>
</tr>
<tr>
<td>Arterial Po/FIO2, Torr</td>
<td>222 ± 75</td>
</tr>
</tbody>
</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 ($P <$...
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) be-
tween 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 ventila-
tory 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 Calcula-
tions 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 in-
dexes. Otherwise, the measurement error did not de-
pend 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 mea-
surements for reliable RV volume assessments. In Table
5, the minimum number of at-random measurements
for each variable during the ventilatory cycle, neces-
sary for an estimate within certain limits of the mean
value, is shown. Five to eight measurements are nec-
essary, 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 differ-
ences 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 vol-
umes and high respiratory rates, which decrease the CI
modulation, may partly explain less ventilatory modu-

Table 4. Measurement error

<table>
<thead>
<tr>
<th></th>
<th>11 Measurements</th>
<th>At 30% of Ventilatory Cycle (n = 2)</th>
<th>At 0 and 100% of Ventilatory Cycle (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac index</td>
<td>5.6</td>
<td>4.2</td>
<td>4.2</td>
</tr>
<tr>
<td>Stroke volume index</td>
<td>6.6</td>
<td>4.9</td>
<td>4.9</td>
</tr>
<tr>
<td>RVEF</td>
<td>7.7</td>
<td>7.4</td>
<td>7.4</td>
</tr>
<tr>
<td>RVEDV</td>
<td>9.1</td>
<td>8.5</td>
<td>8.5</td>
</tr>
<tr>
<td>RVESV</td>
<td>12.1</td>
<td>14.8</td>
<td>14.8</td>
</tr>
</tbody>
</table>

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

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>
<th></th>
<th>10%</th>
<th>15%</th>
<th>30%</th>
</tr>
</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>RVEF</td>
<td>5.1 ± 2.7</td>
<td>3.7 ± 2.2</td>
<td>1.7 ± 0.9</td>
</tr>
<tr>
<td>RVEDV</td>
<td>5.3 ± 2.6</td>
<td>3.7 ± 2.1</td>
<td>1.9 ± 1.0</td>
</tr>
<tr>
<td>RVESV</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.
lation 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 intrindivid-ual variability of RVEDV was 11.6% at a respira-tory 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 er-
or, the minimum number of at-random determina-
tions, 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 infla-
ation. 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 in-
crease the SV (7, 10). This suggests that the volumes measured from injections in the inspiratory phase ind-
ed 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 mea-
surements and echocardiographic data obtained by Jardin et al. (17, 18) in mechanically ventilated pa-
tients. They observed that lung inflation was associ-
ated with a rise in RV transmural pressures and vol-
umes 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 RVESV and RVEDV, 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 regurgi-
tation. Alternatively, an inspiratory fall in baseline pulmonary blood temperature of ~0.01–0.02°C could lead to a temporary overestimation of ~3% of thermodi-
lution 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-pres-
Sure 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 cmH2O of incremental PEEP in some studies and may increase in other human studies, particularly when baseline RVEF was decreased and RVEDV in-
creased after coronary artery disease or acute lung inju-
ry (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 venti-
lation, 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 preload-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


