Possitive pressure inspiration differentially affects right and left ventricular outputs in postoperative cardiac surgery patients
van den Berg, P.C.; Grimbergen, C.A.; Spaan, J.A.E.; Pinsky, M.R.

Published in:
Journal of Critical Care

DOI:
10.1016/S0883-9441(97)90002-2

Citation for published version (APA):

General rights
It is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), other than for strictly personal, individual use, unless the work is under an open content license (like Creative Commons).

Disclaimer/Complaints regulations
If you believe that digital publication of certain material infringes any of your rights or (privacy) interests, please let the Library know, stating your reasons. In case of a legitimate complaint, the Library will make the material inaccessible and/or remove it from the website. Please Ask the Library: http://uba.uva.nl/en/contact, or a letter to: Library of the University of Amsterdam, Secretariat, Singel 425, 1012 WP Amsterdam, The Netherlands. You will be contacted as soon as possible.

UvA-DARE is a service provided by the library of the University of Amsterdam (http://dare.uva.nl)
Positive Pressure Inspiration Differentially Affects Right and Left Ventricular Outputs in Postoperative Cardiac Surgery Patients

Paul C.M. Van den Berg, Cornelis A. Grimbergen, Joseph A.E. Spaan, and Michael R. Pinsky

Purpose: The purpose of this study was to determine the dynamic changes in right ventricular (RV) and left ventricular (LV) output during positive airway pressure inspiratory hold maneuvers so as to characterize the interaction of processes in creating steady-state cardiac output during positive pressure ventilation.

Materials and Methods: We examined the disparity of RV and LV outputs at 5 seconds (early) and 20 seconds (late) into a 25-second inspiratory hold maneuver in 14 subjects in the intensive care unit immediately following coronary artery bypass surgery. RV output was measured by the thermodilution technique, whereas LV output was measured by the arterial pulse contour method. RV and LV volumes were also measured by thermal and radionuclide ejection fraction techniques, respectively.

Results: As Paw was progressively increased from 0 to 20 cm H2O in sequential inspiratory hold maneuvers, both RV and LV outputs changed differently both at 5 seconds and 20 seconds into the inspiratory hold maneuvers. When expressed as change in cardiac output (L/min) for every cm H2O Paw increase relative to end-expiratory values, RV output increased at 5 seconds (0.05 ± 0.15 L/min) then decreased at 20 seconds (−0.08 ± 0.21, P < .05). LV output decreased slightly at 5 seconds (−0.14 ± 0.22) and did not change from this minimal depressed level at 20 seconds (P < .05). Changes in RV and LV output were paralleled by changes in RV and LV end-diastolic volumes, respectively.

Conclusion: Positive pressure inspiration induces time-dependent changes in central hemodynamics, which are dissimilar between RV and LV function. Initially, inspiration increases RV output but decreases LV output, such that intrathoracic blood volume increases. However, sustained inspiratory pressures induce proportionally similar decreases in both RV and LV outputs. Thus, the hemodynamic effects of positive pressure ventilation will depend on the degree of lung inflation, the inspiratory time, and when measurements are made within the ventilatory cycle. These data also suggest that positive pressure ventilation with up to 20 cm H2O Paw does not significantly impair ventricular performance in humans.
in lung volume and intrathoracic pressure may initiate intravascular volume shifts, which result in a transient dissimilarity between RV and LV output.

Accordingly, we examined the transient disparity between RV and LV outputs after observing stepwise increases in Psw induced by airway inflation. Taking into account the time constants of the pulmonary and systemic vascular beds, we chose to examine differential RV and LV volumes and outputs at 5 and 20 seconds into an inspiratory hold maneuver. This analysis allowed us to examine both early and late cardiovascular changes during inspiratory hold maneuvers.

**MATERIALS AND METHODS**

Fourteen postoperative cardiac surgery patients were selected for the study. The study was approved by the ethical committee for human experimentation of the hospital, and all patients gave informed consent before surgery. No complications occurred as a result of the study. All patients underwent uncomplicated coronary artery bypass grafting, were functionally and clinically free of congestive heart failure, and had a normal preoperative contraction pattern on left ventriculography.

Patients were excluded from the study if they had an LV end-diastolic pressure >15 mm Hg, LV ejection fraction <30%, atrioventricular conduction defects, arrhythmias, unstable angina pectoris, profoundly impaired myocardial pump function, or a preoperative diagnosis of myocardial infarction. All subjects had no more than mild obstructive pulmonary disease (forced expiratory volume 1 second [FEV1] >75% of expired vital capacity). None of the subjects received β-adrenergic receptor blocking agents at the time of the study. The mean age of the 14 subjects was 59 ± 9 years, and their mean weight was 78 ± 11 kg.

A randomly selected subgroup of six subjects was checked by transesophageal echocardiography at zero and 20 cm H2O of Psw to evaluate whether an increase in Psw to 20 cm H2O resulted in a pressure-dependent tricuspid insufficiency. As a standard measure in all 14 subjects, the Psw wave was also evaluated to detect patterns consistent with free congestive heart failure, and had a normal preoperative contraction pattern on left ventriculography.

Patients were excluded from the study if they had an LV end-diastolic pressure >15 mm Hg, LV ejection fraction <30%, atrioventricular conduction defects, arrhythmias, unstable angina pectoris, profoundly impaired myocardial pump function, or a preoperative diagnosis of myocardial infarction. All subjects had no more than mild obstructive pulmonary disease (forced expiratory volume 1 second [FEV1] >75% of expired vital capacity). None of the subjects received β-adrenergic receptor blocking agents at the time of the study. The mean age of the 14 subjects was 59 ± 9 years, and their mean weight was 78 ± 11 kg.

A randomly selected subgroup of six subjects was checked by transesophageal echocardiography at zero and 20 cm H2O of Psw to evaluate whether an increase in Psw to 20 cm H2O resulted in a pressure-dependent tricuspid insufficiency. In none of these subjects was a pressure-dependent tricuspid insufficiency detected. This analysis allowed us to examine both early and late cardiovascular changes during inspiratory hold maneuvers.

**Arterial Pulse Contour Method**

A pulse contour method was used for the determination of instantaneous LV output (COi). The basic principle of this method is the analysis of the arterial pulse pressure profile. This method of determining cardiac output assumes a constant relationship between arterial pressure, aortic blood flow, and LV stroke volume. The pulse contour method has been compared with other cardiac output measuring methods, both in animals and humans. The correlation coefficient between the thermodilution and the pulse contour method is reported as .90. We used the specific method of Wesseling et al. The advantage of this pulse contour method, compared with other methods, is that it adjusts for changes in mean arterial pressure, heart rate, and age-dependent characteristics of aorta compliance. Using this method, the systolic area of the arterial pressure waveform is integrated and multiplied by the characteristic impedance of the aorta. Because the characteristic impedance of the aorta of each subject was not known, it was cross-referenced and calibrated by thermodilution cardiac output method during periods of prolonged apneic steady states (15 seconds). During such a steady state, this cardiac output value represents both RV and LV output, whereas during a non-steady state, the thermodilution method only represents RV cardiac output (COi). The computation constant of the pulse contour method was then adjusted so that the thermodilution method and pulse contour method gave the same cardiac output results during apneic baseline measurements.

**Nuclear Stethoscope Method**

Relative changes of LV end-diastolic volume were measured using a nuclear probe (Nuclear Stethoscope, Bois Inc., Cleveland, OH). The nuclear probe is a nonimaging scintillation probe with a maximum count rate capacity of 140,000 counts/sec. Recordings of counts over the precordium were made of a 99m-technetium compound in the vascular pool. The high count rate of this marker allows the continuous beat-to-beat analysis of LV volume changes. Positioning of the nonimaging probe over the precordium requires orientation of the detector based on stroke counts (end-diastolic minus end-systolic) and average counts (end-diastolic plus end-systolic divided by two) as recorded by the probe. The LV region of interest was chosen as the position with the maximum ratio of stroke counts to average counts. The background region of interest is chosen as a position immediately inferolateral to the LV region of interest, where
stroke counts are minimal and average counts begin to decrease as previously described. This technique was used to estimate LV ejection fraction, which, when interfaced with CO2-derived stroke volume data during apneic steady-state conditions, allowed for calculation of baseline LV end-diastolic volume as the ratio of LV ejection fraction to stroke volume. Similarly, the ratio of this dynamically changing LV ejection fraction to matched aortic pulse contour-derived stroke volume estimates allowed calculation of beat-to-beat changes in LV end-diastolic volume during inspiratory hold maneuvers.

Protocol

Patients were anesthetized with high-dose fentanyl. This anesthetic technique was used because of its stabilizing effects on the cardiovascular system during both the intraoperative and postoperative intervals. After the coronary bypass operation, all patients were transported to the intensive care unit, and after a stabilization period of at least 2 hours and at maximum 5 hours, a baseline hemodynamic analysis was performed. At that moment all hemodynamic variables (Pmv, Ppa, Pvp, CO2, EF, heart rate, and systemic blood pressure) had to be within 10% of the therapeutic goals determined by the operating team, otherwise the patient was not included in the study. All 14 subjects met these criteria. The experiments were started when the central body temperature was at least 37°C. During the experiments, all subjects were still under the sedative effects of fentanyl given during the operation.

The protocol consisted of repeated inspiratory hold maneuvers of 25 seconds duration. These inspiratory hold maneuvers were separated by 1-minute intervals to regain the same steady-state situation as before the first inspiratory hold. Resumption of a stable baseline was documented by restoration of all measured hemodynamic variables to within 10% of their initial baseline values. Then, the next maneuver was performed, with an inflation volume 250 mL greater than during the previous maneuver. In this way, a series of maneuvers resulted in a stepwise increase in both Pmv and lung volume. During the first two duplicate inspiratory hold maneuvers, no gas was inflated in the lungs and the resultant Pmv equaled ambient pressure. The hemodynamic measurements performed during these maneuvers were used to derive the baseline values of all variables against which changes induced by increased Pmv were compared. Lung inflation was performed with the same FIO2 used to ventilate the subject. Accuracy of the Pmv data during inspiration as not reflecting West zone 1 conditions was validated by the associated increase in Pgas being less than the increase in Pmv.

Each individual inflation challenge was divided into four phases as described in Fig 1. Phase I started at end-expiration and represents the pre-inspiratory period. In this period, the electronic sampling of data was started. Data sampled during this period were used to evaluate the hemodynamic status of the subject, and to evaluate whether hemodynamic deterioration outside the 10% range, as described in the protocol, had taken place. During phase II, an extra volume of gas was inflated in the lungs and created a peak pressure in the trachea. After this peak pressure, phase III started at the moment a plateau in Pmv had been reached. The height of the plateau Pmv was determined by the inflated volume, the functional residual capacity, and the compliance of the lungs and chest wall. All comparisons of hemodynamic and Pmv data were made between baseline values during phase I and 5- or 20-second measured values during phase III. The maximal plateau Pmv we strove for was 30 cm H2O. Phase IV describes the period in which Pmv was allowed to return to its pre-inspiratory pressure by passive exhalation of the extra volume from the lungs.

The effect of an increase in Pmv was evaluated after the pressure peak caused by the inflation with gas was passed and when the Pmv had reached a plateau. In practice, all data used for further analysis were derived either 5 to 6 or 20 seconds after lung inflation was started. The tracheal pressure was measured at the entrance of the endotracheal tube and was used to represent Pmv. Two different thermodilution curves were recorded in phase III, one initiated at 5 to 6 seconds and another initiated at 20 seconds after Pmv had hit a plateau. Each thermal injection was done during a separate inspiratory hold maneuver. All thermal decay profiles were inspected for an exponential thermal decay and return to its baseline value. If this was not the case, the measurements were discarded and the procedure repeated.

A time of 1 minute between consecutive inspiratory hold maneuvers was chosen because the time constant of volume shifts between the thoracic, abdominal, and peripheral compartments of the circulation is well within the order of 1 minute. Between the inspiratory hold maneuvers, the hemodynamic status of the subject was controlled with fluid infusions as shown in Fig 1. Time course of one inspiratory hold maneuver showing some of the measured variables used to evaluate the effects of an increased Pmv on the circulatory system. Data collection starts during a stable apneic phase I. In this example, 1000 mL of oxygen-enriched air was inflated in the lungs at t = 3 seconds to start phase II. After about 1 second, a new inflation state is achieved, which is phase III. The increase in Pmv (cm H2O) is transmitted to the right atrium (Pmv) (mm Hg), the pulmonary artery (Ppa) (mm Hg), and the abdominal cavity (Pabd) (mm Hg). Data are collected during phase III. Sharp peaks in pressure readings are artifacts caused by the automatic thermodilution cardiac output syringe. Cardiac output thermodilution measurement starts approximately 5 seconds after Pmv has been increased. Following removal of the increased airway pressure, lung volumes rapidly decrease during phase IV.
DIFFERENTIAL VENTRICULAR OUTPUT

required by monitoring \( P_{ra} \) (RV filling pressure) and \( P_{rao} \). In this way, subjects were kept in a hemodynamic steady state throughout the protocol. Data from three of the subjects were discarded because of inaccurate pressure measurements. \( SaO_2 \) was continuously measured with a pulse oximeter (Nellcor) and end-tidal \( CO_2 \) levels were checked with a capnograph (Hewlett-Packard, Palo Alto, CA). During all experiments \( SaO_2 \) did not change and the end-expiratory \( CO_2 \) was stable within \( \pm 2\% \).

Data Analysis

All data were digitalized with a sample frequency of 100 Hz, the band width of the used signals being less than 50 Hz. The signals were converted with a 12 bits resolution and were stored on hard disk for later evaluation. Sampling of data started just before the beginning of an inspiratory hold and was stopped 4 seconds after termination of phase III of the maneuver.

The analysis of the data was concentrated on the changes in the hemodynamic variables induced by the increase in \( P_{ra} \). Linear regression analysis was performed, using the method of least squares. The effect of changes in \( P_{ra} \) on the measured hemodynamic variables (\( CO_1, CO_2, Pra, PCWP \)) was made over the entire range of \( P_{ra} \) changes and found to be linear. However, for illustration purposes, changes between 0 and 20 cm H2O are described in the Figs 1 through 5. Analyses of the \( P_{ra} \) effects on the measured hemodynamic variables were performed with repeated measurement analysis of variance using a post hoc...
RESULTS

The baseline hemodynamic data of the 14 subjects before starting the protocol are given in Table 1. An example of the relation between the inflated lung volume and P_{aw} is given in Fig 2. The increase in P_{aw} is transmitted to intravascular structures within the thorax and abdominal cavity, as illustrated during one inspiratory hold maneuver. Per patient, a mean of 7 ± 2 duplicate inflations of the lungs were performed before P_{aw} increased to approximately 20 cm H_{2}O. The P_{aw} range during the experiments ranged from 0 to 19.01 ± 2.7 cm H_{2}O.

Lung inflation induced by increases in P_{aw} increased the intrathoracic vascular pressures and differentially altered the RV and LV outputs in a time-dependent fashion. From a baseline CO_{r} of 5.72 ± 1.5 L/min, right-sided cardiac output, as measured by CO_{r} 5 seconds after the initiation of the plateau phase, increased 0.05 ± 0.15 L/min/cm H_{2}O. This slight increase in CO_{r} was significantly different from the slight decrease in CO_{l} simultaneously measured by the arterial pulse contour method. The change in CO_{l} was −0.14 ± 0.22
Table 1. Baseline Hemodynamic Data Before Lung Inflation

<table>
<thead>
<tr>
<th>Subject</th>
<th>MAP (mm Hg)</th>
<th>Ppv (mm Hg)</th>
<th>Ppa (mm Hg)</th>
<th>Pao (mm Hg)</th>
<th>CO (L/min)</th>
<th>EF (L/min/m²)</th>
<th>CI (L/min/m²)</th>
<th>SVI</th>
<th>EDVI</th>
<th>PCWP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>97</td>
<td>26</td>
<td>9</td>
<td>17</td>
<td>6.51</td>
<td>0.4</td>
<td>3.13</td>
<td>29</td>
<td>73</td>
<td>14</td>
</tr>
<tr>
<td>2</td>
<td>59</td>
<td>16</td>
<td>7</td>
<td>8</td>
<td>5.22</td>
<td>0.59</td>
<td>2.31</td>
<td>36</td>
<td>62</td>
<td>12</td>
</tr>
<tr>
<td>3</td>
<td>70</td>
<td>19</td>
<td>9</td>
<td>15</td>
<td>3.23</td>
<td>0.5</td>
<td>1.75</td>
<td>25</td>
<td>50</td>
<td>11</td>
</tr>
<tr>
<td>4</td>
<td>69</td>
<td>26</td>
<td>12</td>
<td>11</td>
<td>4.88</td>
<td>0.32</td>
<td>2.42</td>
<td>33</td>
<td>102</td>
<td>18</td>
</tr>
<tr>
<td>5</td>
<td>55</td>
<td>23</td>
<td>12</td>
<td>9</td>
<td>6.28</td>
<td>0.46</td>
<td>3.96</td>
<td>41</td>
<td>89</td>
<td>16</td>
</tr>
<tr>
<td>6</td>
<td>91</td>
<td>18</td>
<td>8</td>
<td>8</td>
<td>6.75</td>
<td>0.4</td>
<td>3.29</td>
<td>39</td>
<td>98</td>
<td>12</td>
</tr>
<tr>
<td>7</td>
<td>83</td>
<td>29</td>
<td>9</td>
<td>25</td>
<td>4.31</td>
<td>0.30</td>
<td>2.07</td>
<td>24</td>
<td>80</td>
<td>13</td>
</tr>
<tr>
<td>8</td>
<td>43</td>
<td>21</td>
<td>3</td>
<td>10</td>
<td>8.25</td>
<td>0.45</td>
<td>3.77</td>
<td>36</td>
<td>79</td>
<td>13</td>
</tr>
<tr>
<td>9</td>
<td>39</td>
<td>20</td>
<td>10</td>
<td>10</td>
<td>6.55</td>
<td>0.30</td>
<td>3.27</td>
<td>80</td>
<td>129</td>
<td>17</td>
</tr>
<tr>
<td>10</td>
<td>84</td>
<td>17</td>
<td>6</td>
<td>4</td>
<td>8.24</td>
<td>0.49</td>
<td>3.70</td>
<td>37</td>
<td>76</td>
<td>16</td>
</tr>
<tr>
<td>11</td>
<td>43</td>
<td>24</td>
<td>10</td>
<td>11</td>
<td>7.60</td>
<td>0.40</td>
<td>3.82</td>
<td>38</td>
<td>94</td>
<td>11</td>
</tr>
<tr>
<td>12</td>
<td>47</td>
<td>17</td>
<td>9</td>
<td>10</td>
<td>6.45</td>
<td>0.45</td>
<td>3.26</td>
<td>33</td>
<td>74</td>
<td>15</td>
</tr>
<tr>
<td>13</td>
<td>71</td>
<td>20</td>
<td>9</td>
<td>24</td>
<td>7.00</td>
<td>0.48</td>
<td>3.27</td>
<td>42</td>
<td>87</td>
<td>14</td>
</tr>
<tr>
<td>14</td>
<td>71</td>
<td>19</td>
<td>0</td>
<td>8</td>
<td>5.21</td>
<td>0.38</td>
<td>2.50</td>
<td>33</td>
<td>86</td>
<td>16</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>75 ± 16</td>
<td>21 ± 5</td>
<td>9 ± 4</td>
<td>9 ± 6</td>
<td>5.74 ± 1.52</td>
<td>0.41 ± 0.09</td>
<td>2.93 ± 0.70</td>
<td>36</td>
<td>88 ± 21</td>
<td>14 ± 2</td>
</tr>
</tbody>
</table>

Hemodynamic profile of 14 patients, whose data were used for analysis.
Abbreviations: MAP, mean systemic blood pressure; Ppv, mean pulmonary artery pressure; Ppa, right atrial pressure; Pao, abdominal pressure; CO, right ventricular cardiac output; EF, right ventricular ejection fraction; CI, cardiac index; SVI, stroke volume index; EDVI, end-diastolic volume index; PCWP, pulmonary capillary wedge pressure.

DISCUSSION

Our study demonstrates that positive-pressure inspiration induces time-dependent differential effects on RV and LV output. These data demonstrate that RV filling, CO, and intrathoracic blood volume increase during positive-pressure inspiratory effort of up to a Pp. of 20 cm H2O. Although CO was less than apneic baseline values at both 5 and 20 seconds into an inspiratory hold maneuver, these decreases were most pronounced at 5 seconds returning toward apneic values at 20 seconds. These dynamic changes suggest a complex process by which mean cardiac output is determined by ventilatory phase-dependent changes in both RV and LV function. Although our study could not separate out the selective effects of increases in Pp. from increases in lung volume, these data demonstrate that mean cardiac output is not impaired by up to 20 cm H2O Pp. increases to levels which would be clinically relevant (Table 2). Fessler et al demonstrated in dogs that the application of up to 15 cm H2O PEEP did not alter the gradient for venous return. In support of these findings, they also observed a constant cardiac output. Their data agree with the flow data in our study in postoperative cardiac surgery patients.

Many prior studies in animals and humans have demonstrated that positive pressure inspiration decreases cardiac output. Braunwald et al demonstrated that PEEP decreased cardiac out-
Table 2. Mean Hemodynamic Data During Apneic CPAP

<table>
<thead>
<tr>
<th>P_{aw} range</th>
<th>P_{aw} (cm H2O)</th>
<th>P_{es} (mm Hg)</th>
<th>P_{es} (mm Hg)</th>
<th>P_{aw} (mm Hg)</th>
<th>P_{es} (mm Hg)</th>
<th>P_{total} (mm Hg)</th>
<th>CO_{2} (L/min)</th>
<th>RV_{total} (ml)</th>
<th>CI (mm Hg)</th>
<th>P_{max} (mm Hg)</th>
<th>HR (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-3</td>
<td>0.42 ± 0.05</td>
<td>76.1 ± 11.8</td>
<td>21 ± 3.35</td>
<td>8.67 ± 2.69</td>
<td>9.52 ± 6.89</td>
<td>5.68 ± 2.28</td>
<td>0.4 ± 0.09</td>
<td>2.85 ± 0.96</td>
<td>11.5 ± 3.54</td>
<td>80.4 ± 12.9</td>
<td></td>
</tr>
<tr>
<td>3-6</td>
<td>5.08 ± 0.71</td>
<td>77.7 ± 10.5</td>
<td>22.1 ± 4.46</td>
<td>8.97 ± 2.32</td>
<td>11.5 ± 7.41</td>
<td>5.48 ± 1.06</td>
<td>0.4 ± 0.07</td>
<td>2.79 ± 0.48</td>
<td>11.7 ± 1.87</td>
<td>7.5 ± 3.54</td>
<td></td>
</tr>
<tr>
<td>6-9</td>
<td>7.66 ± 0.66</td>
<td>76.0 ± 14.2</td>
<td>23.7 ± 5.95</td>
<td>8.76 ± 3.61</td>
<td>8.85 ± 6.38</td>
<td>5.73 ± 1.32</td>
<td>0.39 ± 0.09</td>
<td>2.88 ± 0.66</td>
<td>11.2 ± 5.18</td>
<td>3.2 ± 3.54</td>
<td></td>
</tr>
<tr>
<td>9-12</td>
<td>10.6 ± 0.95</td>
<td>71 ± 16.7</td>
<td>25.9 ± 4.52</td>
<td>11.6 ± 3.17</td>
<td>11.9 ± 7.45</td>
<td>5.88 ± 1.89</td>
<td>0.4 ± 0.11</td>
<td>2.95 ± 0.81</td>
<td>12.3 ± 1.71</td>
<td>81.8 ± 14.4</td>
<td></td>
</tr>
<tr>
<td>12-15</td>
<td>13.7 ± 0.83</td>
<td>73.6 ± 18.1</td>
<td>27 ± 5.12</td>
<td>12.6 ± 2.86</td>
<td>11.7 ± 7.88</td>
<td>5.9 ± 1.94</td>
<td>0.4 ± 0.1</td>
<td>2.95 ± 0.86</td>
<td>13.7 ± 3.05</td>
<td>79.9 ± 13.8</td>
<td></td>
</tr>
<tr>
<td>15-18</td>
<td>16.4 ± 0.72</td>
<td>71.6 ± 12.8</td>
<td>28.8 ± 5.15</td>
<td>13.3 ± 2.59</td>
<td>12.8 ± 7.57</td>
<td>6.17 ± 1.81</td>
<td>0.4 ± 0.1</td>
<td>3.12 ± 0.79</td>
<td>14.9 ± 3.07</td>
<td>82.3 ± 13.2</td>
<td></td>
</tr>
<tr>
<td>18-21</td>
<td>19.5 ± 1.11</td>
<td>72.3 ± 20.1</td>
<td>30.5 ± 4.91</td>
<td>14.8 ± 3.53</td>
<td>13.3 ± 8.91</td>
<td>6.29 ± 1.71</td>
<td>0.4 ± 0.1</td>
<td>3.23 ± 0.77</td>
<td>16.7 ± 3.06</td>
<td>82.4 ± 13.4</td>
<td></td>
</tr>
</tbody>
</table>

Note: Pooled mean data for all subjects grouped by date achieving the desired P_{aw} threshold for each patient. Data are presented as mean ± SD. N = 14.
Differential Ventricular Output

pressure inspiration is also a matter of controversy. A decrease in LV volume during sustained increases in \( P_{aw} \) has been described in both animal and human studies. The decrease in LV end-diastolic volume was used to explain the decrease in cardiac output seen in these studies. However, increases in intrathoracic pressure induced by positive pressure ventilation will also decrease LV afterload, and thus this mechanism has been invoked to explain the observed increases in cardiac output in patients with heart failure following the application of PEEP. Additionally, an unchanged cardiac output during a Valsalva maneuver has also been reported and ascribed to squeezing of an overloaded pulmonary circulation. Our data support the hypothesis that if LV afterload was affected by these positive pressure inspiratory hold maneuvers, the effect was small. More likely, the decrease in CO reflects decreasing LV end-diastolic volume owing to ventricular interdependence, since RV end-diastolic volume also increased.

Limitations of the Study

Although CO and CO are measured with different techniques, a high correlation between the two methods has been established. Moreover, if the compliance of the vascular tree changed during the inspiratory hold maneuvers, it would most likely decrease when \( P_{aw} \) increased. This would result in overestimating CO by the pulse contour method. We saw a decrease in CO, thus, the actual decrease in CO may have been even greater than observed. However, Versprille et al demonstrated that during changes in intrathoracic pressure, the aortic pulse contour method accurately reflects CO when the mean arterial pressure does not change, as was the case in our studies. Clearly, CO and CO, although starting at similar points before inspiration, change directionally in opposite directions with inspiration, demonstrate different time-dependent (5 and 20 seconds) phase changes, and demonstrate that markedly different forces interact to define the outputs of the two ventricles. Also, the study of either RV or LV function alone will not accurately describe the dynamic forces determining the equilibrium seen during ventilation.

A discrepancy between radial or brachial artery compared with aortic pressure has been observed in the postoperative period of cardiac surgery subjects. However, this post-bypass difference in central and peripheral systemic blood pressure is transient and resolves with the administration of fluid. Gravlee et al found no difference in radial, brachial, or aortic pressures 20 minutes after cardiopulmonary bypass. Finally, autonomic tone could have changes during the course of these maneuvers, minimizing any hemodynamic deterioration associated with increasing \( P_{aw} \). However, all subjects were under significant narcotic anesthesia during the time of study. Furthermore, both heart rate and calculated peripheral vascular resistance were constant. Accordingly, this human preparation reflects a model with almost no sympathetic responsiveness. In support of the argument that our subjects were markedly suppressed in their endogenous sympathetic responsiveness, six of the subjects had a baseline MAP of 60 mm Hg or less. However, in none of them was there evidence of tissue hypoperfusion, such as lactic acidosis oliguria, ECG changes of ischemia, or subsequent organ dysfunction.

SUMMARY

Our study demonstrates a differential effect of CPAP on RV and LV output, which is phase and time dependent. These changes in outflows are not predicted from assumptions based on selective changes in only \( P_{aw} \), pulmonary vascular resistance, or LV afterload unless one assumes that CPAP differentially alters both RV and LV preload and afterload by affecting these variables, as well as maintains the pressure gradient for venous return.

REFERENCES

6. Brookhart JJ, Boyd TE: Local differences in intrathoracic...
pressure and their relation to cardiac filling pressure in the dog. Am J Physiol 148:434-444, 1947
18. Goldberg HS: Control of cardiac output by the circuit, in Snyder JV, Pinsky MR (eds): Oxygen transport in the critically ill. Chicago, IL, Year Book Medical, 1987, pp 36-45