Measuring central pulmonary pressures during exercise in COPD: how to cope with respiratory effects?

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Abstract

**Introduction:** Respiratory influences are a major confounder when evaluating central hemodynamics during exercise. We studied four different methods to assess mean pulmonary artery pressure (mPAP) and pulmonary capillary wedge pressure (PCWP) in case of respiratory swings.

**Methods:** Central hemodynamics were measured simultaneously with esophageal pressure during exercise in 30 COPD-patients. mPAP and PCWP were assessed at end-expiration, averaged over the respiratory cycle and corrected with a from the right atrial pressure (RAP) waveform estimated intrathoracic pressure and compared with the transmural pressures.

**Results:** Bland-Altman analyses showed the best agreement of mPAP averaged over the respiratory cycle (bias: 2.5mmHg, limits-of-agreement (-6.0 to 11.8) and when corrected with the nadir of RAP (bias: -3.6mmHg, limits: -11.2 to 3.9). Measuring mPAP at end-expiration (bias: 10.3mmHg, limits: 0.5 to 20.3) and mPAP corrected with the RAP-swing (bias: -9.3mmHg, limits: -19.8 to 2.1) resulted in lower levels of agreement. The respiratory swings in mPAP and PCWP were similar ($r^2=0.82$, slope: 0.95±0.1)

**Conclusion:** Central hemodynamics measured at end-expiration leads to an overestimation of intravascular pressures in exercising COPD-patients. Good measures can be acquired even when esophageal pressure is omitted, by averaging pressures over the respiratory cycle or using the RAP waveform to correct for intrathoracic pressure. Assessment of the pulmonary gradient is unaffected by respiratory swings.
**Introduction**

Hemodynamic assessment remains the gold standard in the diagnosis of cardiovascular and pulmonary vascular disease. In an effort to detect early and potentially more treatable pulmonary vascular disease(1, 2), and heart failure with preserved ejection fraction (HfP EF)(3), there has been renewed interest in the direct measurement of central pulmonary hemodynamics during exercise. Both heart and vessel abnormalities are frequently found in chronic obstructive pulmonary disease (COPD) ((4, 5)).

To minimize the effect of increased intrathoracic pressure changes (ITP) to intravascular pressure measurements during a right heart catheterization at rest, it is recommended to analyze pressures at end-expiration (6). During exercise in patients with COPD especially the increase in ITP during expiration, can be a major confounder in the measurement of central pulmonary pressures.

COPD is characterised by a combination of derangements in respiratory mechanics which alter ITP. At rest, the increased lung compliance requires a larger lung volume to balance the chest wall recoil. During exercise, the increased compliance and resistance lead to ineffective and prolonged gas emptying on expiration. This makes that inspiration starts before a full expiration has occurred, so before the lung returned to is relaxation volume. As a consequence, not only the lung volume at end-expiration rises dynamically (dynamic hyperinflation), but also the alveolar pressure at end-expiration becomes higher (intrinsic positive end-expiratory pressure, iPEEP) The consequence of this is that the respiratory muscles have to generate a more negative ITP during inspiration to overcome the presence of iPEEP, but especially generate a large positive ITP during expiration. These pressures are even exaggerated in exercise. When measuring hemodynamics during exercise in COPD, subtracting esophageal pressure (Pes), in order to acquire the true intra-cavitary and thereby deriving actual transmural pressure which is critical in the detection of pulmonary vascular disease, is preferable (7). This is however not practical in daily practice and older studies therefore reported the values of central pressures averaged over one or more respiratory cycles with exercise in COPD(8-10) and healthy subjects (11-13). More recent studies abandoned this and switched to end-expiratory
measurements (14, 15), based on the recommendations for resting measurements under the assumption that they also hold during exercise. An alternative is to use the right atrial pressure (RAP) waveform, which is largely influenced by its surrounding (which is ITP), to estimate ITP (16).

How to measure central pressures with exercise in case of exaggerated ITP swings and the validity of using the RAP-waveform to correct for ITP remain largely unknown. In this study we therefore evaluated the effect of ITP on the different central pressures in patients with COPD in order to come to a method to adequately measure pulmonary vascular pressures without measuring ITP in routine clinical practice. We hypothesized that measuring at end-expiration leads to an overestimation of pulmonary vascular disease and that averaging over the respiratory cycle or correcting the pulmonary artery pressure with an ITP results in better assessment of the intravascular pressure. Secondly, we hypothesize that the respiratory swings with exercise in pulmonary artery pressure and wedge pressure are similar, so that the transpulmonary gradient can be assessed unaffected by respiratory swings.
Methods

Subjects: COPD Patients (n=30) were referred for the analysis of pulmonary hypertension. Patients were selected based on a moderate to very severe airway obstruction (forced expiratory flow in 1 second, FEV₁<80% of predicted), without significant reversibility (<12% or <200 ml change in FEV₁-value). Patients were on optimal medical therapy and had no acute exacerbation for at least 4 weeks. Pulmonary function testing was performed according to published guidelines. (17, 18). All tests were performed in the VU university medical centre and the VU university medical centre ethics committee approved the study. Written informed consent was obtained from all patients.

Preparation of subjects: A balloon tipped, flow directed, triple lumen 7.5F Swan-Ganz catheter (Baxter Healthcare Corp; Irvine, CA), was inserted under local anaesthesia in the jugular vein. The ports of the catheter were positioned in the right atrium, right ventricle and pulmonary artery. A radial artery catheter was inserted at either the right or left wrist under sterile conditions and following local anesthesia. A standard esophageal balloon-catheter (Microtek Medical B.V. Zutphen, The Netherlands) was inserted trans-nasaly with the use of lidocaine gel (2%). The balloon tip was first positioned 45 cm from the nares after and the balloon was emptied by performing a Valsalva maneuver. (19) Five mL of air was injected into the balloon followed by the withdrawal of 4 mL to partially inflate the balloon. The pressure signal was checked and in case of cardiac artefacts the position of the balloon-catheter was slightly adjusted in proximal direction.

Patients were placed on an electromagnetically braked cycle ergometer (Ergoline GmbH, Bitz, Germany), in semi-supine position. The zero reference for the pressure transducers was 5 cm below the middle of the sternum. All catheters were connected to a PowerLab data acquisition system (ADInstruments), in order to record and digitalize pressures from the right atrium, right ventricle, and pulmonary artery simultaneously with $P_{eso}$. 
**Protocol:** The protocol consisted of three minutes of rest after which workload was increased every three minutes until exhaustion. Oxygen consumption (VO2) was measured continuously using a metabolic cart (Vmax 229, Viasys, Yorba Linda, CA, USA). Patients were asked to signal just prior to exhaustion in case the last workload could not be fulfilled for three minutes to complete the data collection at maximal exercise. During the last 30 seconds of every workload pulmonary capillary wedge pressure (PCWP) was acquired and mixed venous, and arterial blood samples were drawn. Cardiac output (CO) was calculated using the direct Fick-method from arterial- and mixed venous oxygen saturation (SaO2, SvO2) and VO2. Pulmonary vascular resistance (PVR) was calculated as (mPAP - PCWP)/CO.

**Post-processing of pressure waveforms:** The recorded pressure tracings were visually checked and 30 seconds of good quality pressure waveforms were selected near the end of each workload as well as the part with the PCPW tracing. At each workload we selected the PAP tracing as close as possible to the PCPW tracing to minimize changes in flow and ITP between the two measurements. Each selected timeframe was analysed by the use of a home-build program for Matlab (The MathWorks, Natick, MA) in two different methods. We automatically acquired systolic (sPAP), mean (mPAP), diastolic (dPAP) together with the transmural values of mPAP (mPAPtm) and PCPW (PCWPtm) by subtracting Pes in a beat-to-beat manner. All values were acquired and averaged over a period of 10-20 inspiratory and 10-20 expiratory heartbeats. This way we acquired robust inspiratory and expiratory values separately as well as the swings (expiratory-inspiratory values).
We then corrected expiratory mPAP and PCWP for the increased Pes in four different manners and compared it with mPAPtm and PCWP, which we considered the gold standard.

1) End-expiratory: mPAP and PCWP measured at the moment of the last heartbeat during expiration without any further correction (mPAP\textsubscript{end-expiratory} and PCWP\textsubscript{end-expiratory})

2) Averaged over the full respiratory cycle: mPAP and PCWP averaged over three full respiratory cycles including inspiratory and expiratory heartbeats. (mPAP\textsubscript{averaged} and PCWP\textsubscript{averaged})

3) Corrected with RAP\_nadir: mPAP and PCWP at expiration corrected for the nadir in the right atrial pressure (RAP) waveform during the same period of expiration. This assumes that during the relaxation period RAP falls to the pressure surrounding the right atrium and therefore is useful in estimating and correction for P\textsubscript{es}. (16) (mPAP\textsubscript{rap-nadir} and PCWP\textsubscript{rap-nadir}). See figure 2 for the determination of the RAP nadir

4) Corrected with RAP swing: mPAP and PCWP at expiration corrected for the swing in RAP from inspiration to expiration during the same breathing cycle. This assumes that the swing in RAP is useful in estimating and correction for P\textsubscript{es} mPAP\textsubscript{rap-swing} and PCWP\textsubscript{rap-swing}. See figure 2 for the determination of the RAP swing

**Statistical methods:** Demographic, pulmonary function, and hemodynamic data are presented as mean ± standard deviation (SD). Differences in the slope and absolute values at rest and exercise before and after correction were tested using a 2-way-ANOVA. The accuracy of the four different methods of measurements of mPAP and PCWP was evaluated by Bland-Altman analyses with mPAP\textsubscript{tm}
and PCWP$_{tm}$ as gold-standard. Relations were analysed by linear regression. Differences between in respiratory swings in different pressures were tested using a two-tailed student t-test. A P-value<0.05 was considered significant. Analyses were performed using GraphPad Prism 5.0.
Results

Study Population.

Fourteen men and sixteen women were included in this study with a mean age of 64 ± 9 years and a body mass index of 27 ± 6. Pulmonary function and hemodynamic measurements are summarized in table 1. All patients were former or current smokers (mean 37 ± 18 pack years) and had been diagnosed with emphysema based on pulmonary function and Computed Tomographic scanning of the chest. The severity of the airflow limitation was moderate in 16, severe in 11 and very severe in 3 patients according to the Global initiative for chronic Obstructive Lung Disease (GOLD) criteria. Nine patients were hypoxemic (PaO₂<60 mmHg) at rest while breathing room air and four patients had a PaCO₂>45 mmHg.

Pressures during exercise with and without correction for ITP. Maximal exercise was at a workload of 48 ± 31 Watt with a VO₂ of 922 ± 379 ml. On average a CO of 10.9± 3.8 L/min was reached with a heart rate of 118 ± 19. The central hemodynamic pressures are summarized in table 2. The large swings in P eso were transduced into all central pressure; on average responsible for a difference between inspiratory values and expiratory values of about 20 mm Hg. The expiratory P eso at maximal exercise ranged from+3 to+25 mmHg (Figure 3). The average slope decreased from 6.4 ± 3.7 to 4.4 ± 3.2 mmHg/L (p<0.001)after correction for ITP. Seven patients had a mPAP/Q slope >3 mmHg before correction which decreased to <3 mmHg after correction for the ITP. Twenty-two patients had aPCWP recording. Nineteen patients of the twenty-two had a PCWP>20 mmHg with exercise without correction for ITP. Seven patients a had PCPW₂<20; in three of them PCPW₂ was between 20 and 25 mmHg.

mPAP measurement and potential correction methods. mPAPtm and PCWPtm at maximal exercise were 47±15 and 17±8 mmHg respectively. The average mPAP values at maximal exercise of the four methods were; mPAP end-exp: 59±14, mPAP averaged: 50±14, mPAP rap-nadir: 44±15 and mPAP rap swing: 38±15 mm Hg. The average PCWP values at maximal exercise of the four methods were; PCWP end-exp: 27±9,
PCWP\textsubscript{averaged}: 20\pm8, \textit{PCWP}\textsubscript{nadir}: 15\pm7 and \textit{PCWP}\textunderscore{swing}: 11\pm8 mmHg. Bland-Altman plots of the four methods of mPAP and PCWP measurements during exercise are shown in figures 4 and 5 and summarized in table 3.

**Effect of ITP swings on PCWP, mPAP and RAP.** See table 2 for a summary of the pressure fluctuations. Figure 6 illustrates the relation between mPAP\textsubscript{swing} and PCWP\textsubscript{swing}. The swing in mPAP and PCWP did not differ (mean difference 0.9 mmHg, \textit{p}=0.35). There was a strict relation between RAP\textsubscript{swing} and PCWP\textsubscript{swing} ($r^2=0.9$, \textit{p}<0.001) with a slope 1.02 and no significant difference between RAP\textsubscript{swing} and PCWP\textsubscript{swing} (mean difference 0.7 mmHg, \textit{p}=0.28).
Discussion

Errors due to respiratory variation are a major concern when interpreting central vascular pressures during exercise, especially in patients with airflow limitation. The aim of this study was to evaluate the potential error introduced in mPAP by measuring at end-expiration in only and to evaluate potential correction methods. We found that: 1) A significant error is introduced when mPAP and PCWP are measured during end-expiration in exercising COPD-patients, due to increases in expiratory ITP and that 2) averaging mPAP and PCWP over the respiratory cycle are better estimates of mPAP_{tm} and PCWP_{tm}, 3) The right atrial waveform can be used to correct for ITP in patients without right heart failure. 4) The transpulmonary pressure gradient (mPAP-PCWP), and thus pulmonary vascular resistance, is unaffected by ITP swings. To better understand individuals pulmonary hemodynamics and mechanics should be reported (averaged over several cycles) at end expiration, end inspiration and averaged over the respiratory cycle raw and corrected for RAP nadir in routine clinical practice.

Our findings support the use of mPAP and PCWP averaged over 2-3 respiratory cycles in order to acquire more accurate assessment of the transmural values of mPAP and PCWP during exercise in COPD-patients. The patients in the present study showed a wide range of expiratory P_{eso} at exercise, reaching to as high as 25 mmHg, which is consistent with previous studies on pulmonary mechanics (20, 21). In these studies, as well as the present study, the positive excursion of P_{eso} during expiration is at least as large as the negative excursion during inspiration. It is therefore not surprising that mPAP averaged over the respiratory cycle is a more realistic measure of intravascular pressure. Albeit more accurate than mPAP_{end-exp}, mPAP_{averaged} was still a slight overestimation, which can be explained by the increased expiratory time. In COPD-patients expiratory time during exercise is longer than inspiratory time(22). As a consequence, mPAP_{averaged} is more influenced by the “high” expiratory mPAP than by the “low” inspiratory mPAP.
The usefulness of the RAP waveform to estimate the pressure surrounding the heart was shown by Tyberg et al. (16). This method assumes that pressure in the very compliant right atrium is predominantly dependent on pressure surrounding the heart (pericardial pressure, or in our case ITP), rather than by right atrial volume. We showed that this method was useful during exercise in COPD-patients, as long as the lowest point of the RAP during expiration was used. This is explained by the fact that during the right atrial contraction dissociation between RAP and ITP is created. Therefore, only the pressure of an empty and relaxing right atrium is useful to estimate ITP. We found a small bias with $mPAP_{tm}$ and $PCWP_{tm}$ when $RAP_{nadir}$ was used to correct expiratory $mPAP$ and $PCWP$, with a very reasonable 95% CI. The small overcorrection in all patients is because it is unlikely that $RAP_{nadir}$ can be lower than ITP at the same moment. This method may not be useful in patients with more pronounced right heart failure, as this causes RAP to rise, even during relaxation. Despite that, the average $mPAP$ at rest in our cohort of COPD-patients is higher than normally reported in COPD (15, 23), only 2 patients showed a $RAP_{nadir}$ higher than ITP during exercise. We previously showed that some patients with COPD have an impaired venous return due to the high expiratory ITP, which makes that $RAP_{nadir}$ is even closer to ITP. We further showed that the $RAP_{swing}$ to estimated expiratory ITP was not useful in our patients. As RAP during inspiration can reach significant negative values, the total swing is larger than the positive excursion of ITP with expiration. Correction of $mPAP$ with $RAP_{swing}$ therefore leads to an underestimation of $mPAP_{tm}$.

Lastly, we showed that the swings in $mPAP$, $PCWP$ and RAP were similar. This has several convenient implications. The consequence of an identical effect of ITP swing on $mPAP$ and $PCWP$ is that the difference between the two, the transpulmonary pressure gradient is unaffected by the swing in ITP. This only holds when both the $mPAP$ and $PCWP$ being recorded at the same time point in the respiratory cycle. So, although individually, $mPAP$ and $PCWP$ are overestimations of intravascular pressure, the $mPAP_{end-exp}$ and $PCWP_{end-exp}$ combined lead to the correct transpulmonary pressure gradient or PVR (transpulmonary gradient/CO). It underscores the importance of PVR as part of the suggested definition of exercise induced pulmonary arterial hypertension (2), as it prevents patients
being diagnosed simply because of an increased ITP. The similar rise in mPAP and PCWP from inspiration to expiration is consistent with the high ITP, per se, not contributing to right ventricular afterload, which is in agreement with previous literature on the effect of positive end expiratory pressure on right ventricular afterload. (24, 25)

The similar effect of ITP swings in RAP and PCWP also has a potential implication in evaluating exercise hemodynamics. The increase in PCWP calculated as a ratio to the increase in RAP (ΔPCWP/ΔRAP), as previously suggested (26), is unaffected by ITP swings. This ratio might therefore be of potential help in the difficult situation of a high PCWP with exercise in the presence of ITP-swings; a situation that is common in COPD-patients. This would be especially helpful in diagnosing exercise-induced HFpEF. (3)

The patients in the present study had at least moderate airflow limitation, likely a worst-case scenario for the influence of ITP on central pressure measurements. We can only speculated to what extent our findings can be extrapolated to patients with less severe airflow limitation or the normal elderly population. The positive expiratory ITP in COPD is mainly due to the use of expiratory muscles, not well related to the airflow limitation and the pattern of expiratory muscle recruitment differs between patients (27). In the healthy population the use of expiratory muscles can lead to substantial positive ITP as well, albeit only at maximal exercise(28). Pulmonary vascular pressure therefore should be averaged over 2 to 3 respiratory cycles not only in COPD (7, 8, 29) but also healthy subjects (11-13). Whether an averaged mPAP is a more accurate estimate of the intravascular pressure at maximal exercise in this population remains unknown and depends on the amplitude and the length of the inspiratory and expiratory excursions in ITP. What is clear is that respiratory variation in central hemodynamic pressures during maximal exercise is not only present in severe COPD.

Limitations of this study warrant discussion. The average mPAP at rest in our cohort of COPD-patients is higher than normally reported in COPD due a referral bias, as our hospital is a pulmonary
hypertension center. Therefore no conclusion should be drawn with regards to the incidence and severity of pulmonary hypertension at rest or during exercise in COPD from this study. We felt it was justified to use mPAP and PCWP after subtracting of \( P_{eso} \) as gold-standard, although small errors might be present in the measurement of \( P_{eso} \). This was the only method in which ITP was taken into account when evaluating mPAP and PCWP and therefore the best way to answer our question.

In conclusion the present study shows that substantial errors are introduced in interpretation of the absolute values of mPAP and PCWP during exercise in COPD when read only at end expiration. In order to acquire more accurate values, averaging over the respiratory cycle or correction with the estimated ITP from the RAP waveform should be performed. The transpulmonary gradient is unaffected by the respiratory swings, when its components are measured at the same time point in the respiratory cycle.
### Table 1. Pulmonary function and hemodynamic characteristics

FEV$_1$ = forced expiratory flow in 1 second, VC = vital capacity, TLC = total lung capacity, FRC = functional residual capacity, Cl = cardiac index, HR = heart rate, PaO$_2$ = arterial oxygen tension, PaCO$_2$ = arterial carbon dioxide tension.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
<th>% of pred</th>
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<tbody>
<tr>
<td>FEV$_1$ (L)</td>
<td>1.58 ± 0.62</td>
<td>56 ± 18</td>
</tr>
<tr>
<td>FEV$_1$/VC (%)</td>
<td>46 ± 18</td>
<td></td>
</tr>
<tr>
<td>VC (L)</td>
<td>3.46 ± 1.09</td>
<td>98 ± 22</td>
</tr>
<tr>
<td>TLC (L)</td>
<td>6.54 ± 1.20</td>
<td>109 ± 17</td>
</tr>
<tr>
<td>FRC (L)</td>
<td>4.19 ± 1.02</td>
<td>136 ± 34</td>
</tr>
<tr>
<td>CI (L/min/m$^2$)</td>
<td>3.3 ± 0.9</td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>80 ± 17</td>
<td></td>
</tr>
<tr>
<td>PaO$_2$ (mmHg)</td>
<td>65 ± 15</td>
<td></td>
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<tr>
<td>PaCO$_2$ (mmHg)</td>
<td>39 ± 9</td>
<td></td>
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</tbody>
</table>
Table 2. Central pressure over the respiratory cycle at rest and during exercise.

Peso = esophageal pressure, sPAP, mPAP, dPAP = systolic, mean and diastolic pulmonary artery pressure, PCWP = pulmonary capillary wedge pressure, mRAP = mean right atrial pressure.

<table>
<thead>
<tr>
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<th></th>
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<th>Exercise</th>
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<td></td>
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<td>Inspiration</td>
<td>Swing</td>
<td>Exp</td>
<td>Inspiration</td>
<td>Swing</td>
<td>Exp</td>
<td>Inspiration</td>
</tr>
<tr>
<td>Peso (mmHg)</td>
<td>3 ± 2</td>
<td>-7 ± 1</td>
<td>10 ± 3</td>
<td>12 ± 6</td>
<td>-11 ± 2</td>
<td>22 ± 6</td>
<td>19 ± 8</td>
<td>12 ± 6</td>
</tr>
<tr>
<td>sPAP (mmHg)</td>
<td>53 ± 20</td>
<td>45 ± 19</td>
<td>8 ± 6</td>
<td>87 ± 24</td>
<td>40 ± 13</td>
<td>19 ± 8</td>
<td>68 ± 21</td>
<td>40 ± 13</td>
</tr>
<tr>
<td>mPAP (mmHg)</td>
<td>33 ± 12</td>
<td>28 ± 11</td>
<td>6 ± 6</td>
<td>59 ± 14</td>
<td>40 ± 13</td>
<td>19 ± 8</td>
<td>19 ± 8</td>
<td>40 ± 13</td>
</tr>
<tr>
<td>dPAP (mmHg)</td>
<td>23 ± 9</td>
<td>15 ± 7</td>
<td>8 ± 5</td>
<td>40 ± 11</td>
<td>23 ± 11</td>
<td>19 ± 8</td>
<td>23 ± 11</td>
<td>23 ± 11</td>
</tr>
<tr>
<td>PCWP (mmHg)</td>
<td>11 ± 4</td>
<td>2 ± 4</td>
<td>9 ± 4</td>
<td>26 ± 8</td>
<td>5 ± 9</td>
<td>21 ± 6</td>
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<td>mRAP (mmHg)</td>
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<td>-2 ± 3</td>
<td>7 ± 4</td>
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<td>20 ± 6</td>
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Table 3. Summary of Bland-Altman Analyses performed. mPAP=mean pulmonary artery pressure, PCWP = pulmonary capillary wedge pressure.
Figure legends

Figure 1

**Pulmonary artery pressure**

- PAP
- Peso

**Transmural pulmonary artery pressure**

- PAP\textsubscript{tm}
- Peso

**Figure 1.** Example of pulmonary artery pressure before (PAP) and after (PAP\textsubscript{tm}) continuous correction for esophageal pressure (P\textsubscript{eso}) at maximal exercise in a patient with severe COPD (FEV1: 30% of predicted).
Figure 2. Example of determinations of right atrial pressures (RAP). Shown are the simultaneous measurement of RAP and esophageal pressure ($P_{eso}$) at maximal exercise in the same patient as shown in figure 1. RAP$_{nadir}$ is the lowest point in RAP during expiration, which represents RAP during relaxation. Note that RAP falls towards $P_{eso}$ during relaxation. RAP$_{swing}$ was determined as the difference between inspiratory RAP and expiratory RAP.

Figure 3. Average pressure flow relations before and after correction for esophageal pressure. mPAP=mean pulmonary artery pressure, mPAP$_{tm}$= transmural mPAP (calculated as mPAP-$P_{eso}$) PCWP= pulmonary capillary wedge pressure. PCWP$_{tm}$= transmural PCWP (calculated as PCWP-$P_{eso}$). ** = p<0.01, ***=p>0.001.
Figure 4. Bland-Altman analyses of the difference between pulmonary artery pressure (mPAP) and transmural mean pulmonary artery pressure (mPAP_{tm}) plotted versus the mPAP_{tm}. A) mPAP measured at end expiration, B) mPAP averaged over the respiratory cycle C) mPAP corrected with the lowest point of RAP during expiration (RAP-nadir) and D) mPAP corrected with the swing in RAP (RAP-swing). Dotted lines represent the 95% coincidence intervals.
Figure 5 Bland-Altman analyses of the difference between pulmonary capillary wedge pressure (PCWP) and transmural pulmonary capillary wedge pressure (PCWP_{tm}) plotted versus the PCWP_{tm}. A) PCWP measured at end expiration, B) PCWP averaged over the respiratory cycle C) PCWP corrected with the lowest point of RAP during expiration (RAP-nadir) and D) PCWP corrected with the swing in RAP (RAP-swing). Dotted lines represent the 95\% coincidence intervals.
Figure 6 The relation between the swing in mean pulmonary artery pressure ($mPAP_{swing}$) and the swing in pulmonary capillary wedge pressure ($PCWP_{swing}$).
Reference List