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# Influence of torso and arm positions on chest examinations by electrical impedance tomography

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#### Abstract

Electrical impedance tomography (EIT) is increasingly used in patients suffering from respiratory disorders during pulmonary function testing (PFT). The EIT chest examinations often take place simultaneously to conventional PFT during which the patients involuntarily move in order to facilitate their breathing. Since the influence of torso and arm movements on EIT chest examinations is unknown, we studied this effect in 13 healthy subjects  $(37 \pm 4 \text{ years}, \text{mean age} \pm \text{SD})$  and 15 patients with obstructive lung diseases  $(72 \pm 8 \text{ years})$  during stable tidal breathing. We carried out the examinations in an upright sitting position with both arms adducted, in a leaning forward position and in an upright sitting position with consecutive right and left arm elevations. We analysed the differences in EIT-derived regional end-expiratory impedance values, tidal impedance variations and their spatial distributions during all successive study phases. Both the torso and the arm movements had a highly significant influence on the end-expiratory impedance values in the healthy subjects (p = 0.0054 and p < 0.0001, respectively) and the patients (p < 0.0001 in both cases). The global tidal impedance variation was affected by the torso, but not the arm movements in both study groups (p = 0.0447 and p = 0.0418, respectively). The spatial heterogeneity of the tidal ventilation distribution was slightly influenced by the alteration of the torso position only in the patients (p = 0.0391). The arm movements did not impact the ventilation distribution in either study group. In summary, the forward torso movement and the arms' abduction exert significant effects on the EIT waveforms during tidal breathing. We recommend strict adherence to the upright sitting position during PFT when EIT is used.

Keywords: pulmonary function testing, regional lung function, EIT, healthy subjects, obstructive lung disease, COPD, posture

(Some figures may appear in colour only in the online journal)

#### 1. Introduction

The ongoing progress in medical science facilitates the development of personalised medicine. Personalised medicine allows tailoring the medical decisions and the management of patients to their individual needs. Electrical impedance tomography (EIT) might contribute to this development.

EIT is a functional imaging modality that makes short-term and long-term continuous chest examinations possible without any radiation exposure in either children or adults (Pillow *et al* 2006, Pulletz *et al* 2010, Miedema *et al* 2011, Vogt *et al* 2014, van der Burg *et al* 2015, Becher *et al* 2015, Nebuya *et al* 2015, Mauri *et al* 2015, Tingay *et al* 2016). It is portable and the costs are relatively low compared with other imaging modalities. Due to its high temporal resolution, EIT is able to detect dynamic changes in the pulmonary air content in the examined chest plane (Hinz *et al* 2007, Pulletz *et al* 2012, Frerichs *et al* 2013). This is the precondition for the characterization of ventilation distribution and the detection of regional ventilation heterogeneities.

An increasing interest in the use of EIT not only in intensive care, but for regional pulmonary function testing (PFT) in spontaneously breathing subjects in pulmonology has recently been observed. The reason is that there exist no comparable non-invasive methods capable of determining the dynamic information about the ventilation distribution with little effort over a short time. In a clinical setting, EIT measurements often take place in parallel with conventional PFT (Vogt *et al* 2012, Zhao *et al* 2012, 2013). During conventional examinations using spirometry or whole-body plethysmography, subjects typically perform a forced full expiration manoeuvre. During this manoeuvre, but even during quiet tidal breathing, subjects move involuntarily in order to facilitate and improve their ventilation and gas exchange. This can be observed particularly in patients suffering from respiratory disorders with airway obstruction (Druz and Sharp 1982, Gosselink 2004). This movement often results in the leaning forward position when the subjects bend their torsos from the waist and may be accompanied by the elevation of arms.

The influence of the torso and the arm movements on EIT chest examinations is unknown. That is why we studied these effects in healthy subjects and in patients with obstructive lung diseases.

#### 2. Methods

This study was approved by the Institutional Ethics Committee and informed written consent was obtained from each study participant. The study took place within the framework of the European Union project WELCOME (Grant No. 611223) aiming to develop an integrated



**Figure 1.** Examination of the effects of the torso (top) and the arm positions (bottom) during quiet tidal breathing in the sitting position. The consecutive study positions are shown.

care approach for continuous monitoring, early diagnosis and detection of worsening events and treatment of patients suffering from chronic obstructive pulmonary disease (COPD) with comorbidities.

We examined 13 healthy subjects  $(37 \pm 4 \text{ years}, 179 \pm 12 \text{ cm} \text{ and } 80 \pm 15 \text{ kg}$ , mean age, mean height, mean weight  $\pm$  SD; female/male: 4/9) with no history of lung disease and 15 patients (72  $\pm$  8 years, 169  $\pm$  10 cm and 89  $\pm$  16 kg; female/male: 3/12) with the main diagnosis of COPD using EIT. Three of the healthy subjects were excluded from the analysis: one subject (male) had a continuing electrode contact failure because of hypertrichosis; two of the healthy subjects (one male, one female) had a cold.

All subjects were examined during quiet tidal breathing. The examinations were split into two sections: the first one dealt with the torso movement, the second one with the arm movement (figure 1). The 'torso part' of the examinations started with the upright sitting position

with both arms adducted to the torso and hands lying on the thighs (called *Upright 1*), followed by the leaning forward position achieved by bending from the waist by about 40° (called *Leaning forward*) and it ended with the return to the upright position (called *Upright 2*). The 'arm part' of the study started with the upright sitting position with arms adducted to the torso and hands lying on the thighs (called *Bilateral adduction 1*), followed by the elevation of the right arm by 90° (called *Right arm elevation*), then the upright sitting position without arm elevation was resumed (called *Bilateral adduction 2*), followed by the elevation of the left arm by 90° (called *Left arm elevation*) and the last return to the upright sitting position with adducted arms (called *Bilateral adduction 3*).

Before the measurements, we attached sixteen self-adhesive electrodes (Blue Sensor L-00-S, Ambu, Ballerup, Denmark) on the chest circumference in the 4th-6th intercostal space and one reference electrode on the abdomen in each studied subject. We recorded three to eight stable consecutive tidal breaths in each study position. A small electrical alternating current (50kHz, 5 mA<sub>rms</sub>) was successively injected through all adjacent pairs of electrodes. During each current injection the remaining passive electrode pairs recorded the resulting potential differences. The EIT data were acquired using the Goe-MF II EIT device (CareFusion, Höchberg, Germany) at 33 images s<sup>-1</sup>. Raw EIT images were reconstructed using the GREIT algorithm (Adler et al 2009). Each EIT image consisted of 912 pixels and each pixel showed the normalised difference between the instantaneous and baseline pixel impedance (i.e. the relative impedance change). The baseline pixel impedance was equivalent to the average pixel impedance determined during a selected tidal breathing phase. In the 'torso part' of the examination, the baseline was selected from the Upright 1 period, in the 'arms part', from the *Bilateral adduction 1* period, when changes in the end-expiratory volumes were analysed. For the analysis of the tidal volume  $(V_T)$  and its distribution, another approach was used: The raw images of each examination phase were generated with the baseline selected from the respective phase (i.e. these data were 'auto-referenced'). Thus, we used three separate baselines in the 'torso part' of the study and five in the 'arm part'.

The pixel EIT waveforms (figure 2) were analysed offline as follows: We determined the minimum (trough) values of relative impedance change at all end-expiration time points in all breaths in all eight measurement periods. The end-expiratory impedance corresponds to the regional end-expiratory lung volume, i.e. the volume that remains in the lungs after a normal tidal exhalation. During quiet tidal breathing, the end-expiratory volume equals the functional residual capacity. Then, we calculated the mean end-expiratory values from each torso and arm position phase for the whole examined chestcross-section, the anterior and posterior thorax regions and the right and left hemithoracic regions, respectively. The pixel waveforms of the auto-referenced data were analysed in a similar way: First, the end-expiratory values were detected as described above but, in addition, the maximum values of the signal corresponding to the end-inspiratory volumes were identified in each phase. The differences between the consecutive end-inspiratory and end-expiratory values corresponded to the tidal impedance variation of each breath. The average tidal variations were calculated for each image pixel for each study phase and summed up to render the global  $V_{\rm T}$  in the chest cross-section in all study participants during all eight study phases. This  $V_{\rm T}$  corresponded to the average gas volume moved into and out of the lungs in the examined chest section during one respiratory cycle. The prerequisite for this calculation was a stable tidal breathing pattern.

In the next step, we normalised the detected differences in the end-expiratory impedance among the phases by the subject's initial average tidal EIT signal variation determined during the first measurement phase. We determined these normalised values in the whole chest crosssection and separately in its anterior and posterior as well as in the right and left regions. For the 'torso part' of the examination, the end-expiratory differences were divided by the tidal



**Figure 2.** Global EIT waveforms of the relative impedance change (rel.  $\Delta Z$ ) acquired in the chest cross-section of one of the examined subjects during the torso (left) and the arm movements (right) during quiet tidal breathing. The successive changes in the torso and the arm positions are highlighted by different shading of the background. The dashed lines correspond to the mean end-expiratory impedance levels in the respective phases.

variations from the *Upright 1* and for the 'arm part' from the *Bilateral arm adduction 1* phase, respectively.

Finally, we determined the coefficients of variation (CV) from all the pixel values of the tidal impedance variation in each study period to characterise the spatial heterogeneity of ventilation distribution and its dependency on the torso and the arm movements. The CV characterised the relative variability in the pixel tidal impedance variations. It was calculated as the standard deviation of all pixel values divided by their mean value. The higher the CV, the more variable the pixel values of the tidal impedance variation and the more heterogeneous the distribution of  $V_{\rm T}$  was.

The statistical analysis was performed using GraphPad Prism version 5.0 (GraphPad Software, San Diego, CA). For testing the differences among the examination periods, we made use of ANOVA for repeated measurements with Bonferroni adjustment. Differences were considered to be statistically significant when p < 0.05.

#### 3. Results

#### 3.1. End-expiratory impedance

We found highly significant differences in the mean end-expiratory impedance level caused by the torso movement from the *Upright 1* to the *Leaning forward* position and from the *Leaning forward* to the *Upright 2* position in both study groups (healthy subjects: p = 0.0054 and patients: p < 0.0001). The end-expiratory impedance values increased in the *Leaning forward* position. There were no differences found between the *Upright 1* and *Upright 2* positions.

This impedance shift (between the *Upright 1* and the *Leaning forward* position) equalled on average 0.77 (95% confidence interval (CI): 0.36-1.19) of the mean tidal impedance variation in the healthy subjects and 0.81 (95% CI: 0.25-1.38) in the patients. The normalised impedance shift between the *Upright 1* and the *Upright 2* positions was not significant. The results are presented in figure 3.



**Figure 3.** Mean end-expiratory relative impedance change (rel.  $\Delta Z$ ) determined by EIT in the healthy subjects (left) and in the patients with COPD (right) during torso movements in the *Leaning forward* and *Upright 2* positions in comparison with the *Upright 1* phase. The values were normalised by the subject's initial average tidal EIT signal variation from the *Upright 1* phase. Individual (filled symbols) and mean values (horizontal lines) are shown. *P* values are indicated. Significant differences between the examined positions are indicated by asterisks. Asterisks with grey background indicate significant differences between the *Upright 1* and the *Leaning forward* phase. \*p < 0.05, \*\*p < 0.01, \*\*\*p < 0.001. Norm. = normalised.

We also found significant differences in the mean end-expiratory impedance induced by the torso movement during the subsequent analysis of smaller image regions. The values were significantly affected by moving from the *Upright 1* to *Leaning forward* position and from the *Leaning forward* to *Upright 2* position in the healthy subjects in both anterior (p = 0.0031) and posterior regions (p = 0.0109). Similar findings were obtained in the COPD patients, however, the changes were more pronounced in the anterior thorax regions (anterior: p < 0.0001; posterior: p = 0.0481).

The corresponding normalised impedance shifts between the *Upright 1* and *Leaning forward* positions equalled in mean 1.51 (95% CI: 0.49–2.53) of the initial tidal variation in the anterior regions in healthy subjects and 2.51 (95% CI: 0.85–4.16) in the patients. In the posterior regions, the *Leaning forward* position caused an impedance increase that was in mean 0.65 (95% CI: 0.13–1.17) of the initial tidal variation in the healthy group. In the COPD patients, the impedance fell as a result of the *Leaning forward* position in mean by -0.86 (95% CI: -1.84 to 0.12) of the initial tidal variation. There were no differences found between the *Upright 1* and the *Upright 2* phases. The results are shown in detail in figure 4.

The torso movement from the Upright 1 to the Leaning forward and the Upright 2 positions significantly affected the mean end-expiratory impedance level in the right hemithorax in both the healthy subjects (p = 0.0019) and the COPD patients (p = 0.0003). Similar effects were observed in the left hemithorax of the healthy subjects (p = 0.0168) and the COPD patients (p = 0.0091).

The normalised mean end-expiratory impedance increased significantly between the *Upright 1* and the *Leaning forward* positions in the right and left hemithoracic regions in both groups (healthy subjects right: 0.68, 95% CI: 0.35–1.01 and left: 0.85, 95% CI: 0.31–1.39; patients right: 0.76, 95% CI: 0.22–1.31 and left: 0.75, 95% CI: –0.05 to 1.55). The movement



**Figure 4.** Mean end-expiratory relative impedance change (rel.  $\Delta Z$ ) determined by EIT in the healthy subjects (left) and in the patients with COPD (right) in anterior (top) and posterior (bottom) thorax regions during the *Leaning forward* and *Upright 2* positions in comparison with the *Upright 1* phase. The values were normalised by the subject's initial average tidal EIT variation from the *Upright 1* phase. Individual (filled symbols) and mean values (horizontal lines) are shown. *P* values are indicated. Significant differences between the examined positions are indicated by asterisks. Asterisks with grey background indicate significant differences between the *Upright 1* and the *Leaning forward* phases. \*p < 0.05, \*\*p < 0.01, \*\*\*p < 0.001. Norm. = normalised.

back to the *Upright 2* position caused significant changes in impedance in the right hemithorax in both groups and in the left hemithorax only in the healthy subjects. There were no differences between the *Upright 1* and *Upright 2* positions in either group. The results are shown in figure 5.

The second study section concerning the arm movement revealed a highly significant effect with differences between the phases *Bilateral adduction 1, 2, 3* and *Right arm elevation* and *Left arm elevation*, respectively, in both healthy subjects and patients (p < 0.0001). In this case, the end-expiratory impedance values were higher in the periods when either the right or the left arm was abducted compared with the periods with the arms adducted. We did not see any differences in global end-expiratory impedance among the three *Bilateral adduction* phases and between the *Right* and *Left arm elevation* periods, neither in the healthy subjects nor in the patients.

In the healthy subjects, the normalised differences in end-expiratory impedance revealed that the impedance shift between the *Bilateral arm adduction 1* and the *Right arm elevation* 



**Figure 5.** Mean end-expiratory relative impedance change (rel.  $\Delta Z$ ) determined by EIT in the healthy subjects (left) and in the patients with COPD (right) in the right (top) and left (bottom) hemithoracic regions during the torso movements to the *Leaning forward* and *Upright 2* positions in comparison with the *Upright 1* phase. The values were normalised by the subject's initial average tidal EIT variation in the *Upright 1* phase. Individual (filled symbols) and mean values (horizontal lines) are shown. *P* values are indicated. Significant differences between the examined positions are indicated by asterisks. Asterisks with grey background indicate significant differences between the *Upright 1* and the *Leaning forward* phases. \*p < 0.05, \*\*p < 0.01, \*\*\*p < 0.001. Norm. = normalised.

amounted on average to 1.01 (95% CI: 0.64–1.38) of the mean tidal impedance variation. The impedance shift elicited by the *Left arm elevation* equalled 1.10 (95% CI: 0.74–1.47) of the mean tidal impedance variation. Similar differences were observed in the patients. For the *Right arm elevation*, the mean impedance shift reached 1.74 (95% CI: 1.11–2.37) and for the *Left arm elevation* 1.88 (95% CI: 0.95–2.82) of the mean tidal impedance variation. The results are presented in figure 6.

These highly significant differences in the mean end-expiratory impedance values induced by arm movements were also observed in the anterior and posterior thoracic regions in both groups (p < 0.0001). They existed between all the phases, only in the healthy subjects. There were no differences between the *Bilateral adduction 2* and *Left arm elevation*, in both anterior and posterior regions.

The corresponding normalised values also exhibited highly significant changes in the healthy subjects, particularly in the anterior region (figure 7). The mean normalised impedance shift from *Bilateral adduction 1* to *Right arm elevation* reached 1.95 (95% CI: 0.97–2.92)



**Figure 6.** Mean end-expiratory relative impedance change (rel.  $\Delta Z$ ) determined by EIT in the healthy subjects (left) and in the patients with COPD (right) during arm movements in the phases *Right arm elevation*, *Bilateral arm adduction 2*, *Left arm elevation* and *Bilateral arm adduction 3* positions in comparison with the *Bilateral arm adduction I* phase. The values were normalised by the subject's initial average tidal EIT variation from *Bilateral arm adduction 1*. Individual (filled symbols) and mean values (horizontal lines) are shown. *P* values are indicated. Significant differences between the examined positions are indicated by asterisks. Asterisks with grey background indicate significant differences between the *Bilateral arm adduction I* and other study phases. \*\*\*p < 0.001. Bilat. add. = bilateral adduction, Norm. = normalised.

and from *Bilateral adduction 1* to *Left arm elevation* 2.05 (95% CI: 1.40–2.71) of the initial mean tidal impedance variation. This effect was also found in the posterior region with similar normalised impedance shifts for the *Right* and *Left arm elevations* (0.51, 95% CI: 0.27–0.75 and 0.51, 95% CI: 0.21–0.82, respectively). We found similar effects in the COPD patients but they were more pronounced in the posterior part of the thorax. Here, the mean normalised impedance increased by 1.40 (95% CI: 0.73–2.07) of the initial tidal variation between *Bilateral adduction 1* and *Right arm elevation* and by 1.78 (95% CI: 1.05–2.51) between *Bilateral adduction 1* and *Left arm elevation*. In the anterior region, we only saw differences between the *Right arm elevation* and *Bilateral adduction 2* phases.

The arm movements influenced the end-expiratory impedance values in the right and left hemithoracic regions as well. In the right hemithorax, we found significant differences between the *Right arm elevation* and the other four study phases: *Bilateral arm adduction 1*, 2, 3 and *Left arm elevation* in both groups (p < 0.0001). In the left hemithorax, significant differences existed between the *Left arm elevation* and the other four study phases (p < 0.0001).

The corresponding normalised impedance values revealed significant increases in the *Right arm elevation* phase in the right and in the *Left arm elevation* phase in the left region in both study groups (figure 8). On average, it amounted to 1.73 (95% CI: 1.07–2.38) of the initial tidal impedance variation in the right hemithorax for the *Right arm elevation* and 2.32 (95% CI: 1.44–3.19) for the *Left arm elevation* in the left hemithorax in the healthy subjects. In the COPD patients, the *Right arm elevation* caused an increase by 2.94 (95% CI: 2.01–3.86) of the initial tidal impedance variation in the right hemithorax, and the *Left arm elevation* by 3.42 (95% CI: 1.84–5.01) in the left hemithorax.



**Figure 7.** Mean end-expiratory relative impedance change (rel.  $\Delta Z$ ) determined by EIT in the healthy subjects (left) and in the patients with COPD (right) in anterior (top) and posterior (bottom) thoracic regions during the examination phases *Right arm elevation*, *Bilateral arm adduction 2*, *Left arm elevation* and *Bilateral arm adduction 3* in comparison with the *Bilateral arm adduction 1* phase. The values were normalised by the subject's initial average tidal impedance variation from the *Bilateral arm adduction 1* phase. Individual (filled symbols) and mean values (horizontal lines) are shown. *P* values are indicated. Significant differences between the examined positions are indicated by asterisks. Asterisks with grey background indicate significant differences between the *Bilateral arm adduction 1* and other study phases. \*p < 0.05, \*\*p < 0.01, \*\*\*p < 0.001. Bilat. add. = bilateral adduction, Norm. = normalised.

#### 3.2. Tidal impedance variation

The global tidal impedance variation was slightly changed by the torso movement. In the healthy subjects, it increased by moving from the *Upright 1* to the *Leaning forward* position (p = 0.0447). In the patients, the global tidal variation was lower during the *Upright 2* than during the *Upright 1* position (p = 0.4180). These findings are shown in figure 9.

The arm movements exhibited a small effect on the global  $V_{\rm T}$  in the healthy subjects (p = 0.0478), however, the post-analysis did not reveal any significant differences between the individual phases (figure 10, left). The arm abduction did not influence the global  $V_{\rm T}$  in the patients (p = 0.2579) (figure 10, right).



## Arm position

**Figure 8.** Mean end-expiratory relative impedance change (rel.  $\Delta Z$ ) determined by EIT in the healthy subjects (left) and in the patients with COPD (right) in the right (top) and left (bottom) hemithoracic regions during the phases *Right arm elevation*, *Bilateral arm adduction 2*, *Left arm elevation* and *Bilateral arm adduction 3* in comparison with the *Bilateral arm adduction 1* phase. The values were normalised by the subject's initial average tidal EIT signal variation in the *Bilateral arm adduction 1* phase. Individual (filled symbols) and mean values (horizontal lines) are shown. *P* values are indicated. Significant differences between the examined positions are indicated by asterisks. Asterisks with grey background indicate significant differences between the *Bilateral arm adduction 1* and the other study phases. \*\*\**p* < 0.001. Bilat. add. = bilateral adduction, Norm. = normalised.

#### 3.3. Homogeneity of tidal impedance variation

The spatial distribution of  $V_{\rm T}$  characterised by the CV of the pixel tidal impedance variations changed to some extent only between the *Leaning forward* and the *Upright 2* positions (p = 0.0391) in the patients. We did not see any changes in the CV values during the torso movement in the healthy subjects (p = 0.9296). These findings are given in figure 11.

We did not find any effect of the arm movements on the CV of the pixel tidal impedance variations, neither in the healthy subjects (p = 0.3080) nor in the patients (p = 0.2899). These results are shown in figure 12.



**Figure 9.** Global tidal volume ( $V_T$ ) in the studied chest cross-section determined by EIT as the sum of the pixel tidal variations of the relative impedance change (rel.  $\Delta Z$ ) in the healthy subjects (left) and in the patients with COPD (right) during the *Upright 1*, *Leaning forward* and *Upright 2* positions. Individual (filled symbols) and mean values (horizontal lines) are shown. *P* values are indicated. Significant differences between the examined positions are indicated by asterisks. \*p < 0.05.  $V_T =$  tidal volume.



**Figure 10.** Global tidal volume ( $V_T$ ) in the studied chest cross-section determined by EIT as the sum of pixel tidal variations of the relative impedance change (rel.  $\Delta Z$ ) in the healthy subjects (left) and in the patients with COPD (right) during the *Bilateral arm adduction 1, Right arm elevation, Bilateral arm adduction 2, Left arm elevation* and *Bilateral arm adduction 3* positions. Individual (filled symbols) and mean values (horizontal lines) are shown. *P* values are indicated. Bilat. add. = bilateral adduction,  $V_T$  = tidal volume.

#### 4. Discussion

The patients examined by conventional PFT often change their baseline upright sitting position by leaning forward their torsos and moving their arms involuntarily. These changes in body position are typical, particularly in patients with chronic obstructive lung diseases where



**Figure 11.** CV of pixel tidal variations of the relative impedance change (rel.  $\Delta Z$ ) determined by EIT in the healthy subjects (left) and in the patients with COPD (right) during the *Upright 1, Leaning forward* and *Upright 2* positions. Individual (filled symbols) and mean values (horizontal lines) are shown. *P* values are indicated. Significant differences between the examined positions are indicated by asterisks. \*p < 0.05.  $V_{\rm T}$  = tidal volume.



**Figure 12.** CV of the pixel tidal variations of the relative impedance change (rel.  $\Delta Z$ ) determined by EIT in the healthy subjects (left) and in the patients with COPD (right) during the *Bilateral arm adduction 1*, *Right arm elevation, Bilateral arm adduction 2*, *Left arm elevation* and *Bilateral arm adduction 3* positions. Individual (filled symbols) and mean values (horizontal lines) are shown. *P* values are indicated. Bilat. add. = bilateral adduction,  $V_{\rm T}$  = tidal volume.

they help the patients to reduce their respiratory effort. Leaning forward improves the diaphragmatic function and the chest wall and the arm movements support the accessory respiratory muscles. Both of these movements optimise the respiratory function (Sharp *et al* 1980, O'Neill and McCarthy 1983, Gosselink 2004).



**Figure 13.** Influence of the examined electrode plane on EIT waveforms acquired in a healthy subject in the course of quiet tidal breathing during different study phases. The changes during the torso movements (top) and during arm movements (bottom) are shown in the caudal electrode plane: 5th–6th intercostal space (left) and in the cranial electrode plane: 3rd–4th intercostal space (right). Changes in global relative impedance change (rel.  $\Delta Z$ ) over the time are presented. The successive changes in the torso and arm positions are highlighted by different shading of the background.

Patients suffering from obstructive lung diseases are the most probable target patient population for EIT-based analysis of regional lung function. Therefore, we examined the influence of the torso and arm movements on EIT chest examinations to determine the extent to which these effects impact EIT waveforms and findings. We examined both the healthy subjects and patients in order to check if these body-movement effects were additionally affected by pulmonary disease or not.

The results of our study underscore that EIT chest examinations are influenced by such body movements and that these effects are present regardless of the pulmonary condition. The most pronounced effects of the studied torso and arm movements were detected in the endexpiratory impedance level. The magnitude of these torso and arm position-related shifts in the end-expiratory impedance was high. The extent of this influence could be best perceived by analysing the end-expiratory differences between the individual study phases normalised to the tidal EIT signal variation. This approach ensured better inter-individual comparability and revealed that the observed differences occasionally amounted to almost twice the value of the tidal impedance variation found during quiet tidal breathing in the baseline upright posture. The effects were comparable between the healthy subjects and patients with COPD. The mean end-expiratory impedance shifts induced by the *Leaning forward* position were lower than the effects of the *Right* and *Left arm elevations*.

The analysis of the end-expiratory impedance values in smaller regions revealed that the effects of torso movement were discernible in both the anterior and posterior regions. The effect of the arm movements was only detected on that side of the chest where the arm was elevated, but not on the contralateral side. The magnitude of ipsilateral changes was very high. In the COPD patients, the increase in the end-expiratory impedance reached about three times the value of the initial tidal impedance variation.

We presume that the reason for this change in the end-expiratory impedance level induced by the studied movements of both torso and arms is the sliding of skin on the torso in relation to the underlying layers like subcutis, muscles and fat. The EIT electrodes attached on the skin surface consequently move with the skin and this may change the examined cross-sectional thorax plane. The studied changes in the torso and arms' positions may also alter the skin resistance because the skin stretches and relaxes at dissimilar locations during the movement. In women, the breast position may affect the findings. All these effects can be expected to change the regional electrical tissue properties not necessarily related to lung aeration when compared with the baseline conditions. Finally, it is known that breathing techniques and alterations in body position improve the length-tension relationship or geometry of the respiratory muscles; in particular of the diaphragm (Gosselink 2004). These changes may modify regional air filling and ventilation distribution as well.

The analysis of the global tidal impedance variation and of the heterogeneity of its spatial distribution revealed that these measures were less markedly affected than the end-expiratory impedance values. Both the healthy subjects and patients did not change their EIT-derived global  $V_{\rm T}$  during the different phases of the second part of the study characterised by the right and left arm movements. In contrast to this, the torso movement slightly affected the global  $V_{\rm T}$ : the healthy subjects increased their global  $V_{\rm T}$  during the *Leaning forward* and the patients decreased it during the *Upright 2* phase. This means that the healthy subjects inhaled and exhaled larger volumes when leaning forward; whereas patients with chronic obstructive lung disease breathed with the same volume as before the torso movement. The healthy subjects adjusted their  $V_{\rm T}$  to the baseline values after having assumed the upright position again. The patients did not exhibit this behaviour; their global  $V_{\rm T}$  decreased.

The heterogeneity of ventilation distribution did not increase significantly due to the studied arm movements in either study group. It was also unaffected by the torso movement in the healthy subjects. Only in the patients, did the CV of the pixel tidal impedance variations increase by moving from the *Leaning forward* to the *Upright 2* posture. This might be related to the movement from the favourable dyspnoea-reducing position to the less favourable erect position that was accompanied by more heterogeneous ventilation distribution. We have to emphasise that we only examined patients with stable COPD, so these effects may be more pronounced during disease exacerbations.

There exist only a few previous studies using conventional PFT that might be of relevance with respect to these findings regarding  $V_{\rm T}$  and its distribution in the lungs: Ogino *et al* found significantly higher lung volumes in conventional lung function in elderly COPD subjects during arm bracing (Ogino *et al* 2015). Studies on asthmatic children examining the effect of

body posture on bronchoconstriction during medication inhalation (Visser *et al* 2015a, 2015b, 2015c) showed that the leaning forward position was beneficial, because it was associated with improved medication delivery to the lower airways (Visser *et al* 2015b). Factors other than the size of the  $V_{\rm T}$  alone contributing to the observed, but rather small impedance changes are possible, but not documented.

The study by Banzett *et al* showed that bracing the arms increased the maximal sustained ventilatory capacity in healthy subjects. The shoulder girdle muscles have generally two functions: they lift the pectoral girdle during inspiration, and support the arms and the pectoral girdle against gravity (Banzett *et al* 1988). However, when the arms are not braced these muscles cannot support the ventilation to the same extent as during bracing. That is why COPD patients tend to use their upper torso and shoulder girdle muscles already during tidal breathing as the airway obstruction becomes more severe during the disease progression (Martinez *et al* 1990, Banzett *et al* 1988). During unsupported arm elevation of a longer duration (2 min) than in our study, the auxiliary respiratory muscles are less available for sufficient respiratory function (Epstein *et al* 1995). These effects are particularly detectable in patients with COPD where the respiratory muscle recruitment changes with a decreased participation of the rib cage inspiratory muscles and an increased participation of the expiratory abdominal muscles. The diaphragm work increases as well (Reid *et al* 1976, Celli *et al* 1988, Celli 1988, Criner and Celli 1988, Martinez *et al* 1990). All these factors may influence regional lung function, but their exact effect on the EIT chest examinations has not been studied until now.

To the best of our knowledge, there exist no previous EIT studies that have followed the effects of the torso and arm movements. This is because the major intended clinical use of EIT identified so far is for pulmonary monitoring of lying, mechanically ventilated patients. Therefore, the postural effects of supine, prone, lateral side and tilted positions have often been examined by EIT, and occasionally compared with the sitting upright position (Bein *et al* 2010, Frerichs *et al* 2004, Reifferscheid *et al* 2011, Lupton-Smith *et al* 2014, van der Burg *et al* 2015), but not with other positions.

Our study has some limitations that we wish to address. The EIT chest examinations were only performed in one chest plane located in the 5–6th intercostal space, thus, different results in other chest planes cannot be excluded. We wondered whether our findings, particularly the large differences in the global end-expiratory rel.  $\Delta Z$ , were only detectable in the examined, rather caudal plane. For this reason, we checked in two healthy subjects whether the influence of the torso and arms' positions on the end-expiratory level of the EIT signal was also detectable when the electrodes were placed in a higher plane in the 3rd–4th intercostal space. However, we detected similar effects in both planes: the end-expiratory impedance levels increased when the torso leaned forward and when the arms were abducted. Example waveforms in one healthy subject are given in figure 13.

We did not use an EIT system that utilises electrode belts in our study, because these belts are typically only used in critically ill lying patients. The electrode contact might be compromised in the most dorsal regions when used in seated subjects. In addition, possible belt displacement could have occurred during body movement, which we wanted to eliminate. We have not measured the overall whole-lung volume changes. This is mainly because we intended to examine the healthy subjects and patients without the necessity to use a mouth-piece that would have potentially led to a different breathing rhythm and modified respiratory airflow resistance. The torso and arm positions were only changed for rather short periods of time (average duration of 4–5 breaths). However, the observed differences in the end-expiratory impedance were almost instantaneous and they immediately stabilised. Therefore, we believe that we were able to manifest reliably the induced changes in the EIT waveforms even during these short examination periods. Finally, the mean age of the study participants

was significantly different between the two groups. However, we do not suspect that our findings are age-specific. The observed changes in the EIT waveforms confirm this, because they were similar in both groups.

#### 5. Conclusion

The forward movement of the torso and the elevation of the arms exert a significant effect on the EIT waveforms registered during otherwise undisturbed tidal breathing. The observed changes in the end-expiratory impedance, regional  $V_{\rm T}$  and ventilation heterogeneity may influence the findings during PFT. In particular, the effects on the end-expiratory impedance level are pronounced. Therefore, we recommend strict adherence to the upright sitting position during PFT when EIT is used. Measurements that might be influenced by baseline impedance shifts should be cautiously interpreted.

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