Can a wearable device detect airway obstruction?

Kofi Odame, Graham Atkins, Maria Nyamukuru, Katherine Fearon

Abstract—Objective: Lung health monitoring may enable early detection and treatment of exacerbations in respiratory diseases such as chronic obstructive pulmonary disease (COPD). Our objective is to explore the feasibility of using a wearable device to continuously monitor lung health. Towards that goal, this work studies the relationship between wearable device measurements of tidal breathing and spirometer lung function parameters.

Methods: Data was collected from 25 single visit adult volunteers with a confirmed or suspected diagnosis of COPD. A respiratory chest belt was used to measure the fractional inspiratory time, respiratory rate and tidal volume of subjects during quiet breathing. The subjects also performed standard spirometry in a pulmonary function testing laboratory under the supervision of trained clinical staff to produce forced expiratory volume in one second ($FEV_1$), forced vital capacity ($FVC$) and the ratio $FEV_1/FVC$. Two classification models were built and trained: one to detect the presence of lung airway obstruction, and another to stratify the severity of airway obstruction.

Results: The classifier detected airway obstruction with a sensitivity of 95% and a specificity of 80%. Severity of airway obstruction was classified as either mild/moderate or severe/very severe with a sensitivity of 91% and a specificity of 78%.

Conclusion: Tidal breathing parameters that are measured with a wearable device can be used to detect airway obstruction with a level of accuracy that is comparable to that of conventional screening tests used in primary care settings. In addition, these parameters can reliably distinguish between severe and mild airway obstruction.

Index Terms—Wearable sensors, biomedical telemetry, chronic obstructive pulmonary disease, COPD

I. INTRODUCTION

The 251 million people worldwide who suffer from chronic obstructive pulmonary disease (COPD) are at risk of experiencing an acute exacerbation of symptoms, which reduces quality of life, increases mortality, and accounts for the majority of COPD-related healthcare costs [20].

Frequent assessment of lung function via spirometry could help detect COPD exacerbations early and mitigate their impact. Patients will experience a drop in forced expiratory volume in 1 second ($FEV_1$) up to 2 weeks before an exacerbation [22], but unfortunately, there is no way to measure lung function reliably in the home environment, as home spirometry is hindered by low patient adherence and poor patient technique in executing the spirometric maneuvers [4].

COPD management is further complicated by improper diagnosis of the disease. The strict diagnosis of COPD requires compatible symptoms plus spirometry that demonstrates a ratio of forced expiratory volume in 1 second to forced vital capacity ($FEV_1/FVC$) that is less than 70%. But COPD is typically first diagnosed in the primary care office, where spirometry can be a challenge because of a lack of equipment and appropriately-skilled personnel. A lack of spirometry leads to widespread underdiagnosis and overdiagnosis of the disease, which result in increased use of healthcare services, without properly treating the patients’ true condition [5, 8].

A solution to these problems of patient non-adherence, improper spirometer technique and access to skilled personnel is automatic lung function monitoring. This would require neither active engagement from the patient nor supervision from skilled clinical staff. To that end, this paper studies the relationship between wearable device measurements of tidal breathing and clinically-measured spirometer parameters.
Fig. 2: A typical respiratory signal. The inspiratory phase lasts $T_i$ seconds, and the full respiratory cycle (inspiratory and expiratory phases) lasts $T_{tot}$ seconds. The amplitude of the signal is measured from trough to peak as $RA$ Newtons.

II. WEARABLE LUNG MONITORING

A respiratory chest belt is an instrumented strap of fabric that is worn just below the breast bone (Fig. 1). It has an embedded sensor (e.g. an accelerometer, or a resistive or inductive stretch sensor) that measures the rising and falling motion of the chest as the wearer breathes in and out.

The chest belt measures the basic tidal breathing pattern at rest, from which we can extract parameters like expiratory phase duration, respiratory rate, and tidal volume. These measurements require no active user engagement, are decoupled from the user’s skill or motivation level and therefore promote user adherence. Despite the passive nature of these measurements, they are still useful for detecting changes in lung health status, as we describe below.

A. Expiratory phase duration

COPD has two contributory pathologies, chronic bronchitis and emphysema, and most patients with COPD have both pathologies. Chronic bronchitis causes inflammation and excess mucus production in the lungs’ airways, resulting in impeded airflow. When breathing in (that is, during the “inspiratory phase” of respiration), COPD patients can overcome this airway obstruction by increasing the work of the diaphragm and intercostal muscles and breathing in more forcefully. Airway diameter also increases slightly in inspiration as pleural pressure becomes more negative than the atmosphere. Breathing out (during the “expiratory phase”) is a more passive process than breathing in, relying on elastic recoil rather than muscular action during normal respiration. So, with no active compensatory mechanism to counter it, airway obstruction will slow down the rate at which patients can breathe out and thus prolong the expiratory phase [14].

Small airways in the emphysematous lung can also collapse in exhalation, a major driver of airflow obstruction in patients with COPD, leading to slow expiratory phase. Some patients with emphysema will utilize pursed lip breathing to facilitate maximal exhalation, which also contributes to a long expiratory phase, but ultimately reduces small airway collapse in expiration [2].

During spirometry, the ratio of forced expiratory volume in 1 second to forced vital capacity (FEV$_1$/FVC) measures how fast one can forcefully empty out the lungs after taking a deep breath. A properly-executed forced exhalation maneuver will make maximal use of the intercostal and abdominal muscles, thus actively working against any airflow resistance due to airway obstruction. However, there is a finite amount of force that a person can exert during exhalation. So, the more obstructed the airways, the slower the lungs can be emptied and the smaller the FEV$_1$/FVC and FEV$_1$; this echoes the prolonged expiratory phase observed in tidal breathing.

B. Tidal volume

At the end of the expiratory phase of tidal breathing, there is an equilibrium between the elastic recoil pressure of the chest wall and that of the lungs. This pressure equilibrium will always leave the lungs partially inflated with some residual amount of air, or functional residual capacity (FRC). But COPD patients experience a loss of lung elastic recoil, and this causes an increase in FRC [6, 7]. As such, the amount of additional air that COPD patients can inhale during tidal breathing (known as “tidal volume”) is decreased [11].

The spirometric analog to tidal volume is forced vital capacity (FVC), which is the volume of air that can be forcefully exhaled from the lungs after taking a deep breath. Just as with tidal breathing, the expiratory phase of forced breathing ends with a pressure equilibrium that leaves some amount of air, the residual volume (RV), remaining in the lungs. The loss of elastic recoil means that RV is typically larger than normal in COPD. So, like tidal volume, FVC is decreased in COPD.

C. Respiratory rate

In addition to diminished elasticity, the air sacs of COPD patients suffer wall damage, which reduces their surface area and decreases diffusion of oxygen into, and carbon dioxide out of, the bloodstream. COPD patients typically overcome this gas exchange inefficiency by breathing faster to increase their lung ventilation [11, 13]. So, worsening COPD is often marked by an increased resting respiratory rate.
Gas exchange efficiency is measured during pulmonary function testing as diffusing capacity of the lung for carbon monoxide (DLCO). But DLCO is not a routine component of COPD management, and spirometric volume parameters like $FEV_1$ and $FVC$ provide no information about the relative concentrations of oxygen or carbon dioxide. Despite the lack of direct correspondence, we can expect some correlation between respiratory rate and the volume parameters, as they all change with increasing COPD severity.

III. METHODS

To answer the question of whether a wearable device can detect airway obstruction, we collected and analyzed data from a number of patients at the pulmonary clinic of the Dartmouth Hitchcock Medical Center (Lebanon, NH). We obtained informed consent from each subject before their inclusion in the study. All aspects of the study protocol were reviewed and approved by the Dartmouth College Institutional Review Board (Committee for the Protection of Human Subjects-Dartmouth; Protocol Number: 00028641; Date of Continuing Review Approval: 1/29/2019).

A. Subjects

Twenty-five adults, 10 men and 15 women, with either suspected or previously-diagnosed COPD, and scheduled to receive a physician-ordered spirometry test, were recruited for this study. A summary of the subjects’ spirometry test results and their anthropomorphic data is listed in Table I.

| Parameter       | Mean ± std |
|-----------------|------------|
| Age (y)         | 67.6 ± 11.6|
| Height (cm)     | 165.3 ± 10.7|
| Weight (kg)     | 73.6 ± 17.1|
| BMI (kg/m²)     | 27.0 ± 6.08|
| $FEV_1/FVC$     | 0.52 ± 0.16|
| $FEV_1$ (L)     | 1.50 ± 0.81|
| $FVC$ (L)       | 2.68 ± 1.09|

TABLE I: Summary of anthropomorphic data and spirometry test results of the subjects recruited for this study (n=25, 10 men).

B. Procedure

Prior to standard-of-care pulmonary function testing, each subject donned a Go Direct Respiration Belt (Vernier Software & Technology, Beaverton, OR) and breathed quietly while seated. The Respiration Belt (see Fig. 1) measured the force produced by the chest as the subject breathed in and out. Figure 2 is a typical example of the resulting tidal breathing signal waveform that we collected from each subject. After producing 15-20 breaths (approximately 1 minute of data collection), the subject removed the Respiration Belt before performing standard spirometry testing.

Each subject performed standard spirometry with a PC-based spirometer under the supervision of trained clinical staff. The subject performed three spirometry maneuvers that were acceptable and reproducible according to the American Thoracic Society criteria. We collected the subject’s best values of $FEV_1/FVC$, $FEV_1$ and $FVC$ for subsequent data analysis.

C. Parameter extraction

For each subject’s tidal breathing signal waveform, we identified a region of at least 6 consecutive respiratory cycles with minimal motion artifact and baseline wander. From this region of clean data, we calculated an average value each for fractional inspiratory time, respiratory rate and amplitude.

Fractional inspiratory time ($FIT$) is the ratio of the inspiratory time to the total respiratory cycle time. Referring to Fig. 2 we calculated this as $FIT = T_i/T_{tot}$. Note that the quantity $1−FIT$ is a measure of the expiratory phase duration, normalized to the respiratory period.

The respiratory rate is the average number of breaths taken per minute, calculated as $RR = 60/(BMI \cdot T_{tot})$. We normalize the respiratory rate with body-mass index (BMI), because they are significantly correlated [3, 12].

The respiratory amplitude, $RA$, is the difference in force between an adjacent trough-peak pair (see Fig 2). This quantity provides an approximation of the tidal volume as follows. The respiratory amplitude is proportional to the change in radius of the chest as it expands during the inspiratory phase. Since BMI is proportional to chest area [9], we can estimate the tidal volume (modulo constants of proportionality) as $TV = RA \cdot BMI$.

D. Data analysis

A correlation matrix was generated for the relation between the tidal breathing parameters (fractional inspiratory time, respiratory rate, and estimated tidal volume) and the spirometric variables ($FEV_1/FVC$, $FEV_1$, $FVC$). We used the results of the correlation analysis to identify which of the tidal breathing measures would be useful as a predictor variable in estimating each spirometric parameter. The tidal breathing measures with the highest correlation coefficients were used to predict $FEV_1/FVC$, $FEV_1$ and $FVC$ via multiple regression.

We built a k-nearest neighbour (KNN) classifier to detect the absence or presence of lung airway obstruction, using $FIT$, $RR$ and $TV$ as the predictor variables, and applying the GOLD criteria on $FEV_1/FVC$ to obtain the
true class labels. We chose a KNN classifier because the ‘normal’ class data points are clustered together in FIT-RR-TV space (see Fig. 3). The classifier was evaluated using leave-one-out cross validation as follows. We held out data from each of the 25 subjects in turn, built a KNN model on the remaining data, and then evaluated the model on the held-out data.

The tidal breathing parameters were also used to classify the severity of airway obstruction. Since this is an ordinal classification problem, we solved it with a classifier that is based on a regression model (independent variables: FIT, RR, TV; dependent variable: percentage predicted FEV\(_1\)). The regression model’s estimated FEV\(_1\) score was used to classify airway obstruction severity, according to the GOLD criteria [19]. We evaluated the classifier using leave-one-out cross validation on two tasks: (1) coarse classification, to distinguish between mild/moderate and severe/very severe obstruction; (2) fine classification, to distinguish between all four severity levels of mild, moderate, severe and very severe obstruction.

### IV. Results

Table II is a matrix of correlations between tidal breathing parameters and spirometric variables. FEV\(_1\) and FEV\(_1\)/FVC are most strongly correlated with FIT (\(R^2 = 0.295, p = 0.005\) and \(R^2 = 0.274, p = 0.007\) respectively), while FVC is most strongly correlated with tidal volume (\(R^2 = 0.329, p = 0.003\)).

|                  | FEV\(_1\)/FVC | FEV\(_1\) | FVC  |
|------------------|---------------|-----------|------|
| **FIT**          | 0.274         | 0.295     | 0.135|
| p                 | 0.007         | 0.005     | 0.070|
| **RR**           | 0.129         | 0.08      | 0.0125|
| p                 | 0.078         | 0.171     | 0.595|
| **TV**           | 0.060         | 0.317     | 0.329|
| p                 | 0.238         | 0.003     | 0.003|

**TABLE II:** Matrix of correlations between tidal breathing parameters and spirometric variables.

\[
\text{FEV}_1/\text{FVC}= 0.094 + 1.57\cdot \text{FIT} - 0.227\cdot \text{RR}
\]

| R\(^2\) | p-value | RMSE |
|---------|---------|------|
| 0.435   | 0.002   | 0.124|

**TABLE III:** FEV\(_1\)/FVC regression model.

\[
\text{FEV}_1 = -1.16 + 5.35\cdot \text{FIT} + 0.005\cdot \text{TV}
\]

| R\(^2\) | p-value | RMSE |
|---------|---------|------|
| 0.427   | 0.002   | 0.640|

**TABLE IV:** FEV\(_1\) regression model.

Tables III, IV and V show summary information for the FEV\(_1\)/FVC, FEV\(_1\) and FVC regression models. Figures 3-5 are scatter plots of true spirometer parameter values versus their corresponding regression model estimates. The coefficients of determination are FEV\(_1\): \(R^2 = 0.427\); FVC: \(R^2 = 0.329\); FEV\(_1\)/FVC: \(R^2 = 0.435\). All three models have a p-value of \(p < 0.005\).

The performance of the airway obstruction detection model is summarized in the confusion matrix of Fig. 8. As Table VI shows, the model performs with a sensitivity of 95% and a specificity of 80%.

The performance of the severity classification model
Fig. 5: FVC regression model (Table V) predictions versus true values. The dashed gray curve is the line of equality.

Fig. 6: Detection of airway obstruction: scatter plot of class labels versus tidal breathing parameter features. Black markers indicate normal airways (FEV₁/FVC > 70%), while red markers indicate airway obstruction. The asterisks are the true class labels, and the circles are the classifier’s inferred output is summarized in Table VII (fine classification task) and Table VIII (coarse classification task). Note that only data from the subjects with airway obstruction (indicated by red asterisk markers in Fig. 6) were included in this experiment.

For the fine classification task, a Cohen’s kappa coefficient of $\kappa = 0.38$ was achieved, indicating fair agreement between the estimated severity and ground truth. The model performed considerably better on the coarse classification task (Table VIII), yielding a sensitivity of 91% and a specificity of 78%. The Cohen’s kappa coefficient for this task was $\kappa = 0.7$, indicating substantial agreement between the estimated severity and ground truth.

**TABLE VI:** Performance of classifier for detecting the presence/absence of airway obstruction.

| Sensitivity | Specificity | Accuracy | F1-Score |
|-------------|-------------|----------|----------|
| 95%         | 80%         | 87.5%    | 95%      |

**TABLE VII:** Precision and recall performance of classifier for 4-class stratification of airway obstruction severity. The classifier attempts to distinguish between mild, moderate, severe and very severe airway obstruction. Cohen’s kappa coefficient is $\kappa = 0.38$.

|                  | Mild | Mod. | Severe | V. severe |
|------------------|------|------|--------|-----------|
| **Precision**    | -    | 75.0%| 60.0%  | 0%        |
| **Recall**       | 0%   | 75.0%| 85.7%  | 0%        |

**Fig. 7:** Detection of airway obstruction: classifier confusion matrix.

**Fig. 8:** Severity of airway obstruction: scatter plot of class labels versus tidal breathing parameter features. Black markers indicate mild or moderate airway obstruction, while red markers indicate severe or very severe obstruction. The asterisks are the true class labels, and the circles are the classifier’s inferred output. Note: only data from the 20 subjects with airway obstruction (red asterisk markers in Fig. 6) were included in this experiment.
V. DISCUSSION

The relatively strong correlation between FEV₁/FVC (and FEV₁) and FIT in Table II is to be expected, given that airway obstruction is a common underlying factor for both parameters. Similarly, FVC showed the strongest correlation with tidal volume, due to the effect of lung elastic recoil on both these measures.

Our FEV₁/FVC model’s accuracy in detecting airway obstruction is comparable to that of conventional screening tests that are used in primary care settings [17, 10]. This suggests that our proposed approach deserves further exploration and refinement as a primary care screening test tool. The benefit of our approach over conventional tools is that it does not require exertion from the patient, and does not depend on the training and technical skill of clinical staff.

Our FEV₁ model can reliably distinguish between severe and mild airway obstruction. However, it falls short in making finer distinctions between, say, severe and very severe airway obstruction. To perform better on the fine classification task, we could use a more sophisticated approach than our current linear regression model. A more powerful model would have more parameters and ultimately require that we collect much more breathing/spirometry data for training. We could also consider a wider range of tidal breathing predictor values, like variability measures and airflow measures, which have been shown to correlate with airflow obstruction [15, 21, 16, 18].

VI. CONCLUSION

In this paper, we studied the relationship between wearable device measurements of tidal breathing and clinically-measured spirometer parameters. Regression analysis revealed a moderate amount of correlation between the two sets of parameters. Further, we built and evaluated simple models that used wearable device measurements to assess lung health. The classifiers accurately detected airway obstruction, and also provided a coarse stratification of the severity of airway obstruction. The results of this study provide a strong premise for further data collection and exploring the concept of wearable devices for lung health monitoring.

VII. ACKNOWLEDGEMENTS

This work was supported in part by the Munck-Pfefferkorn Education and Research Fund. We would like to thank Jonathan Toledo and Orkan Sezer of Dartmouth College and Henry Berube, Jeffrey Vonada and Jennifer Hilton-Hancock of Dartmouth Hitchcock Medical Center for their invaluable support with data collection. The views and conclusions contained in this document are those of the authors and should not be interpreted as necessarily representing the official policies, either expressed or implied, of the sponsors.

REFERENCES

[1] Andrea Aliverti and Antonio Pedotti. Mechanics of breathing: new insights from new technologies. Springer, 2014.
[2] Cintia Laura Pereira de Araujo et al. “Pursed-lips breathing reduces dynamic hyperinflation induced by activities of daily living test in patients with chronic obstructive pulmonary disease: a randomized cross-over study”. In: Journal of rehabilitation medicine 47.10 (2015), pp. 957–962.
[3] Mehdi Chlif et al. “Effects of obesity on breathing pattern, ventilatory neural drive and mechanics”. In: Respiratory physiology & neurobiology 168.3 (2009), pp. 198–202.
[4] Joana Cruz, Dina Brooks, and Alda Marques. “Home telemonitoring in COPD: a systematic review of methodologies and patients’ adherence”. In: International journal of medical informatics 83.4 (2014), pp. 249–263.
[5] Nermin Diab et al. “Underdiagnosis and overdiagnosis of chronic obstructive pulmonary disease”. In: American Journal of Respiratory and Critical Care Medicine 198.9 (2018), pp. 1130–1139.
[6] Bruno-Pierre Dubé et al. “The clinical relevance of the emphysema-hyperinflated phenotype in COPD”. In: COPD Research and Practice 2.1 (2015), p. 1.

[7] Gary T Ferguson. “Why does the lung hyperinflate?” In: Proceedings of the American Thoracic Society 3.2 (2006), pp. 176–179.

[8] Andrea S Gershon et al. “Health services burden of undiagnosed and overdiagnosed COPD”. In: Chest 153.6 (2018), pp. 1336–1346.

[9] Brian B Ghoshhajra et al. “Direct chest area measurement: a potential anthropometric replacement for BMI to inform cardiac CT dose parameters?” In: Journal of cardiovascular computed tomography 5.4 (2011), pp. 240–246.

[10] Shamil Haroon et al. “Diagnostic accuracy of screening tests for COPD: a systematic review and meta-analysis”. In: BMJ open 5.10 (2015).

[11] Kjell Larsson. “Aspects on pathophysiological mechanisms in COPD”. In: Journal of internal medicine 262.3 (2007), pp. 311–340.

[12] Stephen W Littleton. “Impact of obesity on respiratory function”. In: Respirology 17.1 (2012), pp. 43–49.

[13] Stephen H Loring, Mauricio Garcia-Jacques, and Atul Malhotra. “Pulmonary characteristics in COPD and mechanisms of increased work of breathing”. In: Journal of applied physiology 107.1 (2009), pp. 309–314.

[14] Atul Malhotra et al. “Research priorities in pathophysiology for sleep-disordered breathing in patients with chronic obstructive pulmonary disease. An official American Thoracic Society research statement”. In: American journal of respiratory and critical care medicine 197.3 (2018), pp. 289–299.

[15] MJ Morris and DJ Lane. “Tidal expiratory flow patterns in airflow obstruction.” In: Thorax 36.2 (1981), pp. 135–142.

[16] Shayan Motamedi-Fakhr, Rachel C Wilson, and Richard Iles. “Tidal breathing patterns derived from structured light plethysmography in COPD patients compared with healthy subjects”. In: Medical Devices (Auckland, NZ) 10 (2017), p. 1.

[17] Antonius Schneider et al. “Diagnostic accuracy of spirometry in primary care”. In: BMC pulmonary medicine 9.1 (2009), p. 31.

[18] Ville-Pekka Seppä et al. “Expiratory variability index (EVI) is associated with the severity of acute bronchial obstruction in small children: A proof-of-concept study”. In: Pediatric Allergy and Immunology (2020).

[19] Dave Singh et al. “Global strategy for the diagnosis, management, and prevention of chronic obstructive lung disease: the GOLD science committee report 2019”. In: European Respiratory Journal 53.5 (2019).

[20] Sean D Sullivan, Scott D Ramsey, and Todd A Lee. “The economic burden of COPD”. In: Chest 117.2 (2000), 5S–9S.

[21] M Vitacca et al. “Differences in spontaneous breathing pattern and mechanics in patients with severe COPD recovering from acute exacerbation”. In: European Respiratory Journal 13.2 (1999), pp. 365–370.

[22] Henrik Watz et al. “Spirometric changes during exacerbations of COPD: a post hoc analysis of the WISDOM trial”. In: Respiratory research 19.1 (2018), p. 251.