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REVIEW ARTICLE

Continuous remote monitoring of COPD patients—justification and explanation of the requirements and a survey of the available technologies

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Abstract
Remote patient monitoring should reduce mortality rates, improve care, and reduce costs. We present an overview of the available technologies for the remote monitoring of chronic obstructive pulmonary disease (COPD) patients, together with the most important medical information regarding COPD in a language that is adapted for engineers. Our aim is to bridge the gap between the technical and medical worlds and to facilitate and motivate future research in the field. We also present a justification, motivation, and explanation of how to monitor the most important parameters for COPD patients, together with pointers for the challenges that remain. Additionally, we propose and justify the importance of electrocardiograms (ECGs) and the arterial carbon dioxide partial pressure ($PaCO_2$) as two crucial physiological parameters that have not been used so far to any great extent in the monitoring of COPD patients. We cover four possibilities for the remote monitoring of COPD patients: continuous monitoring during normal daily activities for the prediction and early detection of exacerbations and life-threatening events, monitoring during the home treatment of mild exacerbations, monitoring oxygen therapy applications, and monitoring exercise. We also present and discuss the current approaches to decision support at remote locations and list the normal and pathological values/ranges for all the relevant physiological parameters. The paper concludes with our insights into the future developments and remaining challenges for improvements to continuous remote monitoring systems.

Keywords Remote patient monitoring · Telehealthcare · Telemedicine · Telehealth · eHealth · Chronic obstructive pulmonary disease · COPD · Patch ECG · Transcutaneous measurement · Decision support in healthcare

1 Introduction

According to the World Health Organization (WHO) [1], chronic obstructive pulmonary disease (COPD) is currently the fourth, and will soon become the third, most frequent cause of death worldwide. It is also a disabling disease and therefore associated with high costs for treating and managing patients.

At the same time, it is well known that remote patient monitoring can significantly reduce healthcare costs [2]. Such remote monitoring is very applicable to COPD patients, and there is evidence that it reduces costs [3] by at least 14% [4]. However, there are still no widely accepted remote monitoring services involving recently developed sensors that use available technologies efficiently. Currently, COPD patients' physiological parameters are not continuously monitored

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reliably estimate the $PaCO_2$ and even PaO_2 from transcutaneous monitoring [33, 35–46] ($PtcCO_2$ and $PtcO_2$, respectively), which is not affected by the COPD specificities and is therefore reliable for COPD patients. In fact, $PtcCO_2$ has recently become the preferred method for estimating $PaCO_2$ in remote settings [32, 33].

Oxygen is commonly administered to patients during LTOT, but more importantly in acute exacerbations, which saves lives by preventing severe hypoxemia. Oxygen therapy can, however, increase the $PaCO_2$, as explained in the next paragraph. In fact, about a quarter of COPD patients with acute exacerbations are at risk of hypercapnia if they are given a high dose of oxygen [47].

It might seem absurd at first glance that oxygen therapy can cause hypercapnia, but there are several mechanisms responsible for this effect [47]. Here, we mention the most influential:

- Oxygen-induced deterioration of V/Q matching (see Fig. 1). As was already mentioned before, COPD is characterized by V/Q mismatching. There is, however, a physiological mechanism that is correcting the V/Q ratio: for alveoli with reduced ventilation (thus reduced alveolar pO_2—partial pressure of oxygen), pulmonary capillaries supporting these alveoli will constrict, reducing perfusion for the alveoli with reduced ventilation, and consequently improving the V/Q match. In other words, the delivery of blood is reduced to poorly ventilated parts of the lung, causing more blood to go to areas where the gases can be more efficiently exchanged. This mechanism is called hypoxic pulmonary vasoconstriction. The most influential factor for hypoxic pulmonary vasoconstriction is alveolar pO_2. During the oxygen therapy, the pO_2, even in alveoli with low ventilation, will increase, inhibiting hypoxic pulmonary vasoconstriction. As a result, alveoli with relatively impaired ventilation will be well perfused, leading to an increase in V/Q mismatch [48]. Put more intuitively, a perfusion increase in the lung areas that are purely ventilated will decrease the efficiency of the gas exchange and cause an increase in $PaCO_2$.
- Another mechanism for oxygen-induced hypercapnia is the Haldane effect, which refers to the increased capacity of deoxygenated hemoglobin to bind and carry CO_2, compared to the oxygenated form. Thus, when there is an abundance of O_2, CO_2 is released into the blood stream, causing the $PaCO_2$ to rise [48].
- People with healthy lungs have breathing regulation dependent mostly on changes in blood CO_2 levels. Since some patients with COPD have high levels of CO_2 for long periods of time, the brain's regulatory breathing center can, over time, become less sensitive to CO_2 levels, and more dependent on O_2 levels, which causes these patients to rely more on a low arterial O_2 level to stimulate their breathing. This means that oxygen therapy can reduce the stimulus to breathe. As a consequence of this reduced breathing, CO_2 removal from the lungs is decreased. This "hypoxic drive" theorem was traditionally widely accepted, but it was recently challenged at least for the acute situation, as having only a time-limited effect on ventilation that cannot explain the total increase in $PaCO_2$ [48].

Hypercapnia can cause various symptoms, ranging from mild headaches, lethargy, and confusion, to severe ones with a hypnotic effect and acidosis with subsequent organ dysfunction, which can lead to coma and death [47, 49]. It also decreases diaphragm contractility and favors muscle fatigue [50]. Clinical signs of hypercapnia are as follows [47]: vasodilation producing flushing and warm peripheries with dilated blood vessels (including retinal veins), a bounding pulse (a pulse that feels full and spring-like on palpation as a result of an increased thrust of cardiac contraction or an increased volume of circulating blood within the elastic structures of the vascular system), drowsiness, flapping tremor, confusion, and coma.

To decrease the risk of hypercapnia induced by oxygen therapy in acute situations, current clinical guidelines recommend that all patients with COPD receive oxygen therapy targeted at 88–92% SpO_2 until hypercapnia is excluded by an ABG analysis within 1 h of the treatment being started [24, 47]. For some patients, even very small amounts of supplemental oxygen are sufficient to worsen hypercapnia, so they might need an even

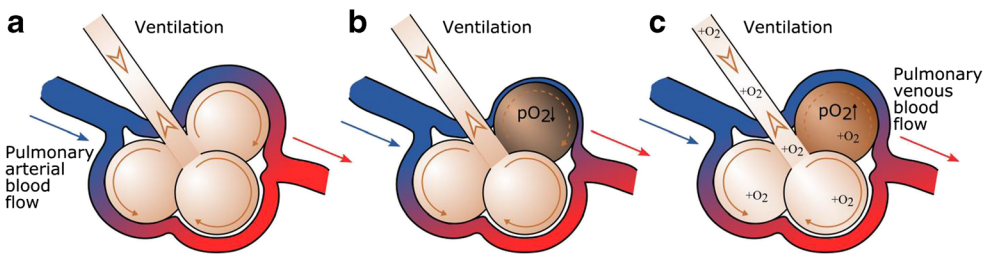

Fig. 1 Hypoxic pulmonary vasoconstriction and its inhibition by oxygen therapy. **a** Normal alveolar ventilation and perfusion. **b** Reduced ventilation in the darker alveolus (thus, the local pO_2 also drops) causes reduced perfusion. **c** Oxygen therapy increases pO_2 in all the alveoli, including the dark one, and inhibits hypoxic pulmonary vasoconstriction

lower target saturation range [47]. On the other hand, if the $PaCO_2$ is normal, oxygen therapy can target the usual saturation range of 94–98% and so be more efficient [47].

For the purpose of keeping the SaO_2 within a desired range, pulse oximeters are used in a closed loop to correct for the administrated oxygen levels. There is clear evidence that this titrated, and therefore closely regulated, acute oxygen therapy reduces the mortality and hypercapnia, compared to non-titrated oxygen therapies [24, 51].

LTOT is also initially titrated for specific needs of each patient but not during the therapy. The goal of the initial titration is to reach $SpO_2 > 90\%$ (resting $PaO_2 > 8$ kPa (60 mmHg)) without a significant rise in CO_2 [52]. This is done by the ABG analysis, but transcutaneous measurements can also be used [52].

Although it is widely accepted that oxygen-induced hypercapnia can develop during COPD exacerbations, few studies have reported on $PaCO_2$ changes during LTOT. Cooper et al. reported a mean PaO_2 increase of 3 kPa, and a mean $PaCO_2$ increase of 0.39 kPa, while patients were breathing oxygen (30% concentration) [53]. Török et al. showed increase in $PaCO_2$ during incremental increase of oxygen flow in the initial adjustment of LTOT but the "rise in CO_2 was not considered to be high enough to lead to a lower prescription of oxygen" [54]. During sleep, however, physiological changes occur that cause the SpO_2 to drop for the patients with COPD (nocturnal oxygen desaturation (NOD)). Since hypoxemia has effects on the cardiovascular system, NOD could possibly also be a reason why COPD patients die more during the night [23]. To counteract NOD, there is a recommendation, inconsistently applied around the world [55], to increase oxygen flow by 1 l/min during sleep and during exercise (to counteract the increased need for oxygen during exercise) [23]. At least one study has reported a significant increase in $PaCO_2$ due to this increase in oxygen flow [56]. Therefore, at least during sleep, patients could benefit from titrated LTOT, much like for acute oxygen therapy, but the patients should also be monitored for hypercapnia [23].

Even though NIV at home is currently not explicitly recommended for COPD patients [30, 57], it is used instead of LTOT for specific patients especially in some geographic areas [58]. In contrast to LTOT, transcutaneous CO_2 measurement was studied for NIV at home as an alternative to ABGs for adjusting NIV settings and was found appropriate [33, 35, 41, 59].

2.2 Relation between COPD and cardiac problems

If low oxygen levels are present for longer periods of time (chronic hypoxemia), they can result in hypoxic pulmonary vasoconstriction (Fig. 1). On the other hand, the destruction of lung tissue leads to a breakdown of the pulmonary capillaries, and hence a reduction of the pulmonary vascular system. These two mechanisms cause an increased resistance of the pulmonary vascular system to the blood stream. The increased resistance causes an increase in the pulmonary artery pressure and makes it harder for the heart to pump blood to the lungs. If this condition continues, it can cause the heart muscle to grow in size (right ventricular hypertrophy) and eventually lead to failure of the right side of the heart [60].

It takes time to develop right-side heart failure, but the heart remodeled with hypertrophy, and receiving hypoxemic blood for its own nutrition, is very prone to arrhythmias and sudden cardiac events. On the other hand, hypercapnia decreases myocardial contraction [61] and also predisposes to arrhythmias [62]. Furthermore, COPD occurs frequently with coronary artery disease [63]. For all these reasons, a substantial proportion of the deaths in patients with COPD is the result of cardiovascular complications [63–65].

In general, cardiovascular diseases are the most frequent comorbidities with COPD and include the following entities: coronary artery disease, heart failure (about 30% of patients with stable COPD show some degree of heart failure), arrhythmias, and hypertension (one of the most frequent comorbidities in COPD) [63, 66].

2.3 Physical activity in COPD

Regular physical activity (PA) is recommended for all patients with stable COPD [66]. Walking is generally accepted, but also stair-climbing, treadmill, or cycling exercises are beneficial. Pulmonary rehabilitation (supervised exercise) or a home exercise program can improve the lung's functional status.

PA in COPD patients decreases the risk of hospitalization [67], reduces the decline in lung function [67], but can also improve the general health status and decrease both disability and mortality [68, 69].

PA is drastically reduced during and after hospitalization caused by exacerbation, but even patients with milder exacerbations, which do not require hospitalization, tend to stay indoors during the exacerbation period, which lowers PA levels [67]. It might take a number of weeks for patients to recover from exacerbation, during which time they lose muscle mass as a result of reduced activity.

Patients whose arterial PaO_2 are borderline at rest may develop worsening hypoxemia during exercise, but even patients without hypoxemia may improve exercise capacity with supplementary oxygen [26]. During exercise, a substantial CO_2 retention ($PaCO_2$ increase of more than 4 mmHg) may also occur frequently in patients with COPD, and can even result in exercise-induced hypercapnia (an elevation of $PaCO_2$ levels greater than 45 mmHg (6.0 kPa). Less frequent is a significant reduction of $PaCO_2$ on exertion which can go even to hypocapnic levels [70].

several days is often taken as an indication of regular use for these drugs [76].

4.7 Detection and classification of exacerbations

Signs are objective, whereas symptoms are subjective, evidence of a health problem.

The symptoms of severe COPD exacerbations that require hospitalization are [76]

1. Change in cough frequency.
2. Change in sputum production and appearance.
3. Increase in dyspnea at rest.

The signs of severe COPD exacerbations that require hospitalization are [76]

4. Inability to speak one full sentence.
5. Temperature > 38.5 °C.
6. Ankle oedema.
7. Respiratory rate > 25/min.
8. HR > 110/min.
9. $PaO_2 < 8$ kPa [75].
10. Worsening cyanosis.
11. Use of accessory muscles.
12. Loss of alertness.
13. PEF < 100 l/min.

All of these parameters are measurable at home. Parameter 12 is significant on its own, whereas parameters 3, 7, 8, 10, and 11 are significant as a group [76]. It is important to note that the exact thresholds in the previous list are not universally accepted; studies in remote monitoring employ diverse exacerbation criteria [21], which might be one of the reasons for the inadequate performance of decision support algorithms (discussed in the next section).

The criteria for severe COPD exacerbations based on parameters that are normally measured in hospitals but can now also be measured in the home environment with portable spirometers, transcutaneous measurements, and portable ECG devices are [76] $FEV_1 < 1$ l, $PaO_2 < 8$ kPa (60 mmHg), $SaO_2 < 90\%$, $PaCO_2 \geq 6.0$ kPa (45 mmHg), and ECG abnormalities. Additional measurements for severe acute exacerbation, which are currently not measurable at home, are chest radiograph, white blood cell count ≥ 12,000, sputum stain/culture, biochemistry (electrolytes, urea, glucose, etc.). The life-threatening events are respiratory or cardiac arrest, confusion or coma, $PaO_2 < 6.7$ kPa (50 mmHg), $PaCO_2 \geq 9.3$ kPa (70 mmHg), pH < 7.3.

COPD exacerbations can often be prevented [66]. It is therefore desirable to predict or at least early detect signs and symptoms of exacerbations. This can be done automatically by using decision support systems featuring the classification of patient states.

4.8 Decision support (exacerbation prediction and detection algorithms)

In most of the existing remote-monitoring systems, the information obtained is analyzed by health caregivers. Only some of them provide automatic decision support systems [15], stand-alone or in combination with human analysis. Figure 6 presents the COPD-related decision support systems data obtained from two existing reviews [20, 21], and reports featuring decision support that came after the reviews [84, 103, 134–137]. The most often used inputs are the self-reporting of symptoms on a PCD, followed by pulse oximetry and spirometry (Fig. 6a). ECG has been used in only two publications: as a source of the features for exacerbation prediction [136], and to detect "clinical alert" (further details not provided) [138], whereas the $PtcCO_2$ was not used at all.

Panel b shows that only about one quarter of the research featuring decision support provides automatic data acquisition. This is related to the frequency of data acquisition and analysis, which was almost exclusively on a daily basis, except in [139] where it was 3 h, in [140] where the interval could be varied depending on each patient's needs, as well as in [136], which is the only report featuring continuous data processing. Patients' compliance is affected by the frequency and method of data acquisition, as discussed in Sect. 4.3, but more importantly, it is not possible to detect immediate life-threatening events without continuous monitoring.

Clinical decisions are traditionally based on a set of predefined universal rules (see previous section). It is for that reason that the approach most often utilized for detecting and predicting exacerbation was by defining universal (population-based) thresholds on obtained symptoms and physiological parameters (panel c). These thresholds were sometimes adjusted to individual patients' needs, but the best results were obtained by using more advanced classification algorithms: linear discriminant classification [141], a Bayesian network [140], a probabilistic neural network classifier [142], multilevel logistic regression [143], classification and regression trees [144], k-means clustering [145], a state machine combined with logistic regression [84], and a hybrid classifier combining a support vector machine, random forest, and a rule-based system [136]. The purpose is to classify the patient's status as being in exacerbation (detection) or as transitional towards exacerbation, i.e., the prodromal period (prediction).

The reported accuracy in detecting exacerbations ranged from 40 to 94%, the sensitivity from 6 to 80%, and the specificity from 61 to 95%. The best accuracy in the early detection of exacerbations is reported for the hybrid classifier with 10 measured parameters and a total of 25 features used as the

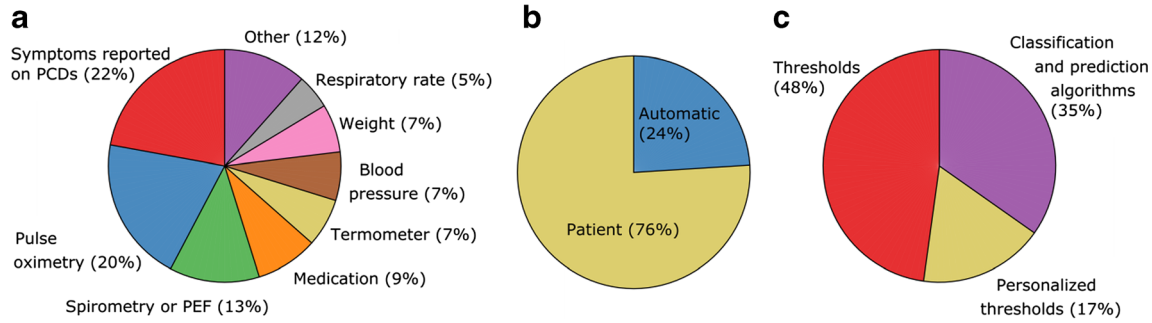

Fig. 6 Overview of existing decision support systems. **a** Sensors used (Other: video/audio (five systems), physical activity (four systems), ECG (two systems), lung and heart sounds (one system), glucometer (one system)). **b** Patient action needed for data acquisition or automatic data acquisition. **c** Algorithms used. Total number of participant used in the studies is 1866, whereas the average duration of the studies was 6.4 months

inputs [136]. Nevertheless, by using only self-reported symptoms as inputs, and k-means clustering, it is possible to obtain a sensitivity of 75% and 90% specificity for early exacerbation detection [145]. Furthermore, a pulse oximeter alone in combination with a classification algorithm can provide a high predictive accuracy [84].

Only two studies [142, 145] reported using patients' electronic health records, but these were developed only for the purpose of the study and did not seem to be integrated into the patients' standard care.

Besides completely outperforming the threshold approach and providing encouraging results, advanced classification algorithms ensure that the classification is adapted to each patient, provide ranking of features based on their predictive power [84], as well as the extraction of new rules [136]. For a concise description of the predictive analytics methods used in healthcare, the reader can refer to [21].

In addition to detecting exacerbations, it is beneficial to assess their severity for the purpose of deciding between home or hospital treatment.

4.9 Assessment of severity and deciding between home or hospital treatment

The severity of an exacerbation is assessed crudely by tachypnoea, tachycardia, the use of accessory respiratory muscles, cyanosis, and evidence of respiratory muscle dysfunction or fatigue (e.g., uncoordinated ribcage motion or paradoxical movement of the abdominal wall during inspiration) [76]. If the severity of an exacerbation is in doubt, it should always be assessed in hospital. Referral to a hospital's emergency department is mandatory in the case of respiratory failure indicated by the increased use of accessory respiratory muscles, paradoxical movement of the abdominal wall during inspiration, and significant deterioration in mental status.

Mild exacerbations of COPD are generally believed to represent an increase in symptoms, especially dyspnea, not necessarily accompanied by increased cough and sputum production, which might be more tenacious than usual. These parameters can be obtained through a self-assessment (see Sect. 4.5). Severe exacerbation, on the other hand, is associated with acute respiratory failure, especially in patients with an impaired lung function, sometimes accompanied by hypercapnia [7], which can be obtained from a $PaCO_2$ measurement. Severe exacerbations cannot be treated at home, so if detected, patients should be transported to a hospital.

There are no definitive clinical guidelines about whether a patient should be cared for at home or in a hospital, and physicians are often uncertain when making this decision. The most important factors are the severity of the exacerbation, acute respiratory failure, the onset of new physical signs (e.g., cyanosis, peripheral edema), and the failure of an exacerbation to respond to initial medical management [30]. Other factors that can be taken into account are cause of the exacerbation (for example, severe pneumonia), a coexisting disorder that requires admission, degree of disability, social factors like the degree of support in the community (e.g., whether the patient lives alone), patient's history, and mental state [7, 75].

4.10 Detection of provoking and predictive factors

It is not clear which factors determine the development and severity of an exacerbation [7]:

- It is commonly thought that viral and bacterial infections of the tracheobronchial tree are the major causes of exacerbations in the later stages of disease [129]. The role of bacterial and viral infections in COPD exacerbations is still considered as controversial by some authors [7].
- Air pollution [66].
- There is some evidence that ozone concentration might be slightly associated with additional hospital admissions [146].
- Poor nutrition, i.e., malnutrition, in combination with respiratory muscle fatigue can aggravate the exacerbation.
- Drugs (especially tranquilizers).
- Stopping regular medication such as diuretics and/or bronchodilators on the patient's own initiative can increase

symptoms, which means that the monitoring of medication compliance is important (Sect. 4.6).
- Inappropriate oxygen administration can aggravate an exacerbation because of a reduction in the hypoxic respiratory drive.
- Conditions that can mimic or aggravate symptoms are pneumonia, pulmonary hypertension, heart failure or arrhythmias, pulmonary embolism, and pneumothorax.
- The cause of about one third of severe exacerbations of COPD cannot be identified [66].

A study involving 64 patients with moderate-to-severe COPD showed evidence that chronic hypercapnic respiratory insufficiency (high $PaCO_2$) and pulmonary hypertension are predictive factors for hospitalization caused by COPD exacerbation [147]. Long-term oxygen therapy and perhaps even long-term noninvasive mechanical ventilation at home are possibly factors that reduce the risk of severe exacerbations, since there is evidence that they reduce hospital admission in COPD with chronic hypercapnia [148].

A study using the SGRQ showed that factors for predicting frequent exacerbations were daily cough, daily wheeze (clinical sign of exhaling difficulties caused by a narrowed tracheobronchial tree), and daily cough and sputum together, and frequent exacerbations in the previous year [149]. Another study showed that SGRQ scores and poor quality of life are associated with re-admission for COPD [150].

4.11 Educational programs

Even though there are reports that educational programs for COPD patients can significantly reduce the utilization of healthcare services and improve health status [151], they have not been as actively promoted as much as programs for asthma patients [76]. Single-topic programs are available (e.g., smoking cessation, long-term oxygen therapy, rehabilitation), but there are insufficient integrated educational materials incorporating all the aspects of disease management [151].

Educational programs should improve people's knowledge about the disease process and its treatment and should also motivate patients to change behavior and lifestyle, with the goal of improving their quality of life [151].

5 Notes about normal and pathological ranges and changes in the measured physiological signals

5.1 SaO_2 and PaO_2

The SaO_2 normal range (two standard deviations (SDs) around mean) for adults aged < 70 years at sea level is 94–98%. For young adults (age 18–24), the 2SDs PaO_2 range is 11.98–14.82 kPa (89.3–110.5 mmHg). The lower limit for this range decreases significantly with increasing age [152], e.g., the range for 64 years old and more is 9.02–14.76 kPa (67.3–110.1 mmHg) [47]. Additionally, the PaO_2 is 0.8 kPa (6 mmHg) lower in the supine position than in the upright position.

Oxygen demand and oxygen delivery increase during exercise and reduce during rest and sleep. Hypoxemia (too low PaO_2) can be defined as PaO_2 below the normal lower limit, but most authors suggest values of less than 8 kPa (60 mmHg), or SaO_2 of 90% [47].

The most common recommendation for patients with COPD is that oxygen is admitted if the resting awake PaO_2 is less than 7.3 kPa (55 mmHg), or if it is 7.3–7.9 kPa (55–59 mmHg) in the presence of an elevated hematocrit (55%) or elevated right ventricular pressure evident from ECG [55].

5.2 $PaCO_2$

The reference range for $PaCO_2$ is 4.6–6.1 kPa (34–46 mmHg) for a healthy adult of 18–38 years [47]. Values above 6.1 kPa are out of the normal range, but values up to 6.7 kPa can be accomplished by holding the breath.

5.3 Body temperature

In humans, the traditional normal value for the oral temperature is around 37 °C [153]. Various parts of the body are at different temperatures, and the magnitude of the temperature difference between the parts varies with the environmental temperature [153]. There are a lot of other variables that can influence temperature measurements, such as the measurement site, time of the day, age, etc. [95].

A body temperature higher than 38.5 °C is one of the factors indicating severe exacerbations [76].

5.4 Electrocardiogram

With the development of pulmonary heart disease due to COPD, the following changes might be seen in a routine ECG [154]:

- The P wave axis is farther right than + 75°.
- Any of the right ventricular hypertrophy criteria.
- Late R wave progression in precordial leads.
- Low voltage.
- Abnormal Q waves in the inferior or anterior leads.
- Supraventricular arrhythmias, especially atrial tachycardia, multifocal atrial tachycardia, and atrial fibrillation.
- Ventricular arrhythmias [155].

On the other hand, some more acutely presenting ECG changes may signal higher risk for the patient. It is known that patients with COPD have higher rates of cardiovascular

diseases and consequently higher incidence of cardiovascular causes of mortality [63–66].

Some ECG changes can be attributable to acute overloading of the right ventricule of the heart which may be seen in acute pulmonary embolism or acute respiratory failure, such as

- S1Q3 pattern in standard leads.
- Acute right bundle branch block.
- Right axis deviation [155].

but some more subtle changes may contribute to the importance of the ECG changes in defining the risk of the patients with otherwise stable COPD:

- Prolongation or shortening of the heart rate corrected QT interval (QTc) suggests higher incidence of sudden cardiac death. It has been shown that these changes may occur more often in patients with COPD [156].
- Reduced heart rate variability is also a marker of sudden cardiac death and has been connected to patients with COPD.
- Dispersion of the QT interval in ECG recordings shows potential to foresee the adverse events and has been appreciated more often in patients with COPD [157].

Supraventricular arrhythmias occur frequently in patients with COPD [63]. Most often, they comprise atrial tachycardias, especially multifocal atrial tachycardia, atrial fibrillation, and atrial undulation which are often chronic. Ventricular tachycardias such as non-sustained ventricular tachycardia may depict patient's higher risk for adverse events. Sustained ventricular tachycardia and ventricular fibrillation are arrhythmias that need to be terminated instantaneously. Arrhythmias are in general life-threatening events and can lead to dangerous complications. For instance, patients with COPD and multifocal atrial tachycardia have higher mortality rates [158].

Cardiac arrhythmia is a group of conditions in which the heartbeat is irregular, too fast, or too slow. If a heartbeat is above 100 beats/min in adults, it is called tachycardia and a heartbeat that is below 60 beats/min is called bradycardia. To distinguish between different arrhythmia types, other diagnostic procedures must be used in addition to the HR analysis.

The ECG can show signs of myocardial ischemia, specifically ST segment and T wave changes, as well as signs of myocardial infarction, specifically changes in the QRS pattern. However, not all patients with acute or previous myocardial infarction exhibit ECG changes [159, 160].

5.5 Arterial blood pressure

"Hypertension is likely to be the most frequently occurring comorbidity in COPD and can have implications for prognosis" [30]. The pressure in the aorta and in the brachial and other large arteries in a young adult human rises to a peak value (systolic pressure) of about 120 mmHg during each heart cycle and falls to a minimum (diastolic pressure) of about 70 mmHg [161]. Conventional notation for the arterial pressure is systolic pressure over diastolic pressure, e.g. 120/70 mmHg. There are, however, a number of variables that can influence normal blood pressure values. For details, the reader is referred to [161] or similar literature.

There are different guidelines that usually define the pressure intervals specifying different hypertension severities. Stage 1 hypertension is defined as "clinical blood pressure of 140/90 mmHg or higher and subsequent ambulatory blood pressure monitoring daytime average or home blood pressure monitoring average blood pressure is 135/85 mmHg or higher" [162]. Therefore, in remote settings, we can consider 135/85 mmHg as a general threshold for hypertension.

5.6 Spirometry output

The presence of a post-bronchodilator FEV_1/FVC of less than 0.70 confirms the presence of COPD [66]. The severity of COPD can be categorized based on FEV_1 (percent of predicted): mild ≥ 70, moderate 50–69, severe < 50 [76], or according to GOLD: mild ≥ 80, moderate 50–79, severe 30–48, very severe < 30. For the complete list of reference values in spirometry, see [91].

5.7 Respiratory rate

One of the signs of COPD exacerbation is a respiratory rate over 25/min [76]. A normal adult human at rest breathes 12–15 times a minute [163].

6 Conclusion and future development

The guideline for home treatment and management of mild COPD exacerbations, as specified by the European Respiratory Society [76], states that patients need to be reassessed every 48 h for worsening of symptoms, signs, and measurements. With the inclusion of continuous monitoring, this interval can be prolonged and the home treatment of exacerbations made more secure.

Two crucial improvements to the current remote-monitoring systems are enabling the continuous monitoring of the most important physiological parameters and enabling real-time decision support based on advanced classification algorithms, which are still to prove their clinical reliability. This will make it possible to detect life-threatening events in real time, consequently reducing mortality and hospitalization. Other expected consequences are an increase in the cost efficiency and an improvement of patients' compliance with the monitoring.