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COPD – Chronic Obstructive Pulmonary Disease

MUDr. Jana Unar Vinklerová, Ph.D., MBA & MUDr. Tomáš Kratochvíl

EVROPSKÁ UNIE
Evropské strukturální a investiční fondy
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1 COPD Definition

COPD is a preventable and treatable disease characterised by persistent respiratory symptoms, bronchial obstruction and abnormalities of the lower airways and alveoli, usually due to exposure to inhaled noxious substances. Other factors, such as genetics, altered prenatal development or frequent respiratory infections in childhood are also involved. The most common symptoms are dyspnoea, cough, and expectoration of sputum.

2 COPD Epidemiology, Pathophysiology and Risk Factors

The prevalence of COPD is steadily increasing, currently estimated at 11.7 % of the world population and 6.7 % in the Czech Republic, which is about 710,000 patients, with only about 230,000 patients registered with pneumologists. COPD is the cause of death in 3,200 to 3,500 patients per year, and therefore COPD diagnosis should be made as early as possible.

The primary risk factor is active and passive smoking and persistent exposure to harmful influences such as environmental or workplace pollution (dust, gases, fumes, etc.). Other factors include asthma, frequent respiratory tract infections, history of pulmonary tuberculosis or HIV infection, use of selected medications, or genetic factors such as alpha-1-antitrypsin (A1AT) deficiency.

COPD is a heterogeneous disease with many extrapulmonary characteristics and comorbidities. It is caused by long-term inflammatory processes due to exposure to various inhaled noxious substances. Processes of oxidative stress, imbalance of proteases and antiproteases, and increased activation of pro-inflammatory cells, predominantly neutrophils and T-lymphocytes, are involved. In some patients, an eosinophilic type of inflammation is also present. Chronic inflammation leads to damage to bronchial walls, damage to alveoli and microvasculature, airway remodelling and chronic mucus hypersecretion. The functional consequences are airway obstruction, hyperinflation, impaired gas exchange and reduced functional capacity. Among the systemic effects, a higher risk of cardiovascular disease, osteoporosis, depression, cachexia, diabetes mellitus or sleep apnoea syndrome. COPD is considered a proven precancerous disease (lung cancer).

These mechanisms vary with the patient and lead to different disease manifestations, allowing us to categorise the clinical phenotypes and features of the disease.

3 COPD Diagnosis

The basic requirements for the diagnosis of COPD are spirometrically confirmed postbronchodilator airway obstruction (FEV1/VCmax < 70%) and evaluation of respiratory symptoms (dyspnoea, limitation of physical performance, cough, sputum expectoration).

The fundamental diagnosis of COPD should encompass:

1. Clinical examination (history taking, assessment of inhalation risks, evaluation of symptom severity, and physical examination),

2. Pulmonary function testing and blood gas analysis (*Appendix 1*),

3. Imaging studies (chest X-ray in two projections (rear and side) to exclude comorbidities; a chest CT scan is not routinely recommended.,

4. Laboratory testing (biochemistry, blood count, A1AT deficiency).

***Figure 1*: Initial diagnostic procedure in new COPD cases**

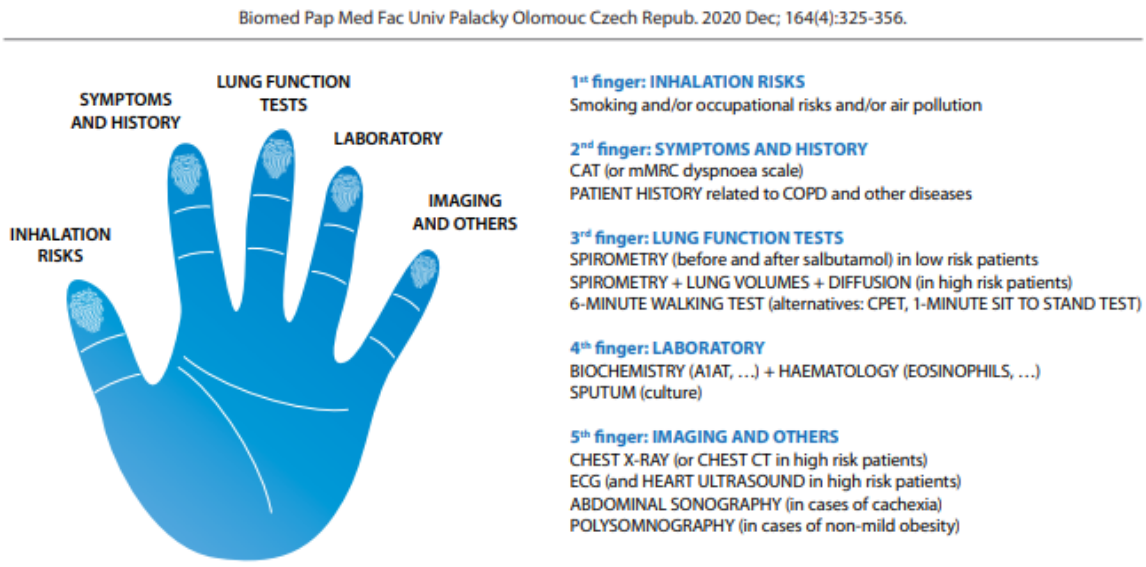


Fig. 3. Initial diagnostic procedures in new COPD cases.

Even an individual without subjective symptoms can have an obstructive ventilation disorder. However, it is often not the case. Most people with COPD suffer from dyspnoea to a certain degree (although they may not be aware of it sometimes due to modification of their behavior and reduction in physical activity). Dyspnoea usually develops very slowly, first with greater physical exertion, which is gradually more and more limited by the feeling of worse breathing. Therefore, it is necessary to focus on "subtle" changes in the patient's behaviour (e.g. extending the journey to the means of transport or using the elevator instead of walking up the stairs as before). Approximately 2/3 of patients suffer from a productive cough with phlegm. About half of the patients experience non-specific fatigue. Some lose weight, others suffer from prolonged autumn and winter colds (these are often acute exacerbations – see below).

A physical examination rarely contributes to the diagnosis of COPD. Physical signs of bronchial obstruction are usually not present in patients with milder degrees of COPD, unless it is an acute exacerbation. Their absence therefore does not exclude COPD. On the other hand, exhalation wheezing and creaking may also be present in other diseases (bronchial asthma, sarcoidosis, left-sided heart failure, etc.). Patients with pulmonary emphysema predominance have diffusely attenuated alveoli breathing with hypersonic tapping and reduction of chest tremors. In extreme cases, people with pulmonary emphysema have a so-called barrel-shaped chest. Central cyanosis, on the other hand, is more frequent in patients with bronchitis COPD.

Other functional examinations of the lungs include bodyplethysmography and transferfactor examination (pulmonary diffusion), which will provide information about possible hyperinflation of the lung parenchyma, restrictive ventilation disorder or diffusion disorder (reduction in emphysema, fibrosis, heart failure). In case of reduced saturation (SpO2 < 94%), a blood gas test should be performed. Stress testing is one of the important functional tests. In practice, the 6-minute walk test (6MWT) is most commonly used.

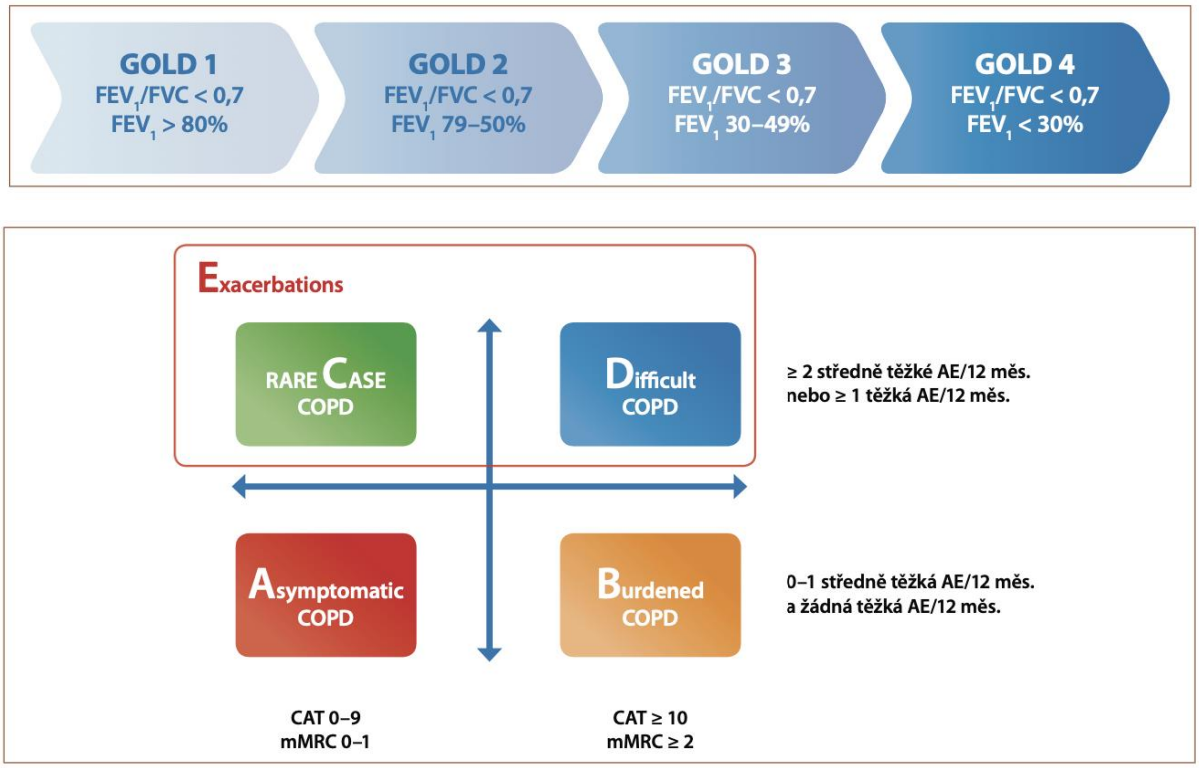
To assess the severity of symptoms, the CAT = COPD Assessment Test questionnaire (*Appendix 2*) or the modified dyspnoea scale – mMRC (*Appendix 3*) is recommended.

For the final diagnosis, differential diagnosis and exclusion of other alternative diagnoses are necessary, in particular: bronchial asthma, gastroesophageal reflux, heart failure, cor pulmonale, pulmonary embolism, bronchiectasis, cystic fibrosis, interstitial lung processes, lung tumor, tracheal stenosis, foreign body aspiration, tracheobronchomalacia, primary ciliary dyskinesia.

4 COPD Classification

For the classification of COPD, we use the GOLD (Global Initiative for Chronic Obstructive Lung Disease) categorization, which divides patients according to spirometric parameters (post-bronchodilator values) into 4 degrees and assigns them categories A, B or E (formerly categories C and D) according to the severity of symptoms (mMRC, CAT) and the incidence of acute exacerbations in the previous 12 months.

Figure 2 *GOLD categorization of COPD*



• According to GOLD, patients of category A have a low risk of exacerbations and few symptoms (these are patients with a stable course and a low incidence of hospitalizations and very low mortality, this group of patients consists of a large proportion of individuals who have not yet been diagnosed). We can call category A **asymptomatic**.

• GOLD category B patients have a low risk of exacerbations, but many symptoms (patients with comorbidities and/or increased subjective perception of dyspnoea, these patients have a higher risk of hospitalization and a higher risk of death). Category B can be called **comorbid**.

• GOLD category E patients have a high risk of exacerbations, regardless of the presence or absence of subjective symptoms. Category E can be called **exacerbation**.

Patients with more frequent exacerbations have a more severe degree of bronchial obstruction. More frequent exacerbations are associated with a faster decline in FEV1 and a faster progression of symptoms and deterioration of quality of life. Patients with GOLD stages 3 and 4 also have a higher risk of hospitalization and death, regardless of the number of exacerbations.

Based on clinical manifestations, imaging examinations, functional changes and other parameters, patients are further divided into 6 phenotypes of COPD disease (*Appendix 4*). This division has practical impact, since it determines the individual treatable features of the disease. Two or more of these phenotypes may be present in individual patients, which gives the possibility of fully individualized phenotype-specific treatment.

Example of notation: COPD GOLD 3E, emphysematic phenotype and frequent exacerbations.

5 COPD Management

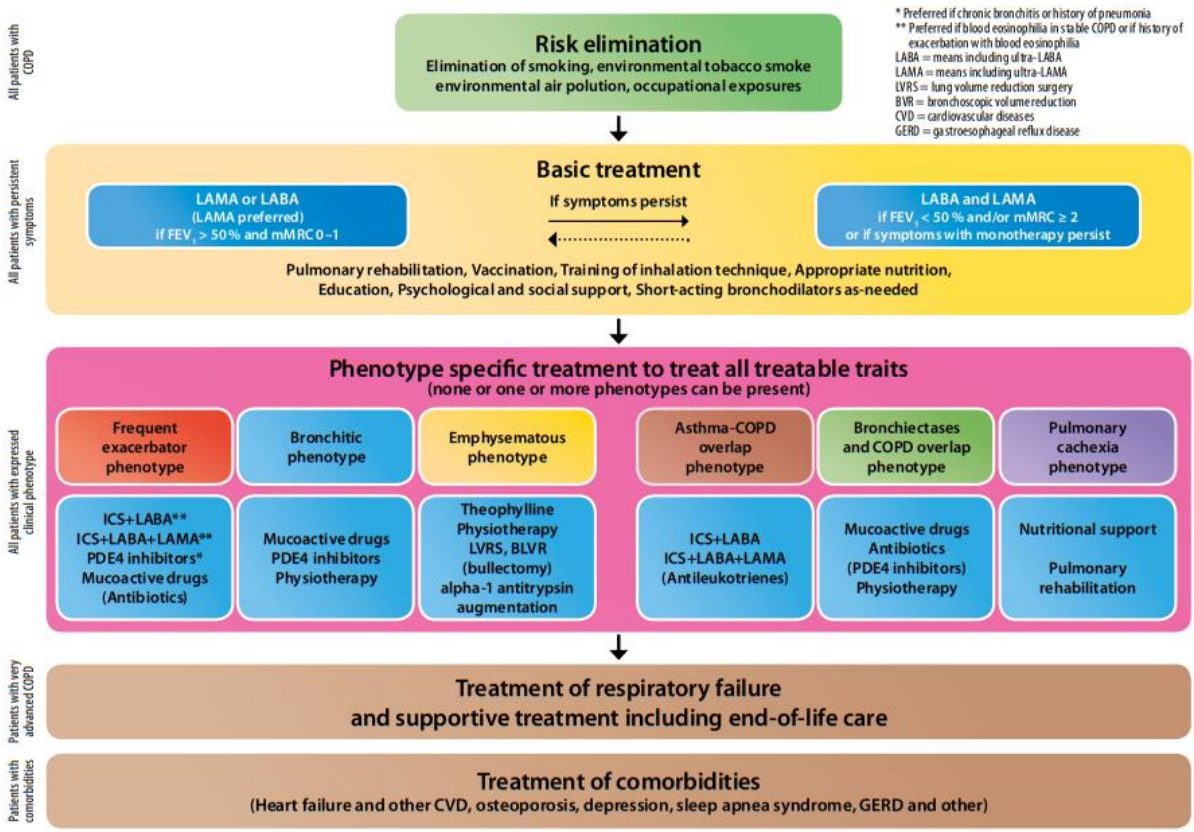
COPD cannot yet be cured or stopped completely. The treatment strategy consists of 5 objectives:

1. elimination of risks, 2. basic treatment, 3. phenotype-specific treatment, 4. treatment of respiratory failure and palliative care, 5. treatment of comorbidities.

Figure 3 *COPD treatment strategy*

Risk elimination includes interventions against tobacco smoking and environmental/occupational exposures. Basic treatment is based on bronchodilator therapy, pulmonary rehabilitation, vaccination, care for appropriate nutrition, inhalation training, education and psychosocial support. Adequate phenotype-specific treatment varies phenotype to phenotype and includes more than ten different pharmacological and non-pharmacological strategies. If more than one clinical phenotype is present, the treatment strategy should be guided by the expression of each phenotypic designation separately.

Figure 4: *Treatment recommendations for stable COPD and phenotype-specific treatment*



The aim is to control symptoms, reduce the number and severity of exacerbations, improve quality of life, and treat complications and comorbidities. Management of COPD is divided into (i) generalised, which is the same for all symptomatic patients regardless of their clinical phenotype, and (ii) personalised, targeting specific features in an individual patient.

Generalised management *(Appendix 8)* includes non-pharmacological approaches such as risk elimination, especially smoking cessation, physical activity (daily aerobic physical exercise, preferably regular walking – the optimal number of steps for COPD patients is approximately 5,000 to 6,000 per day), pulmonary rehabilitation, education on inhalation techniques, and a healthy diet. Pharmacological approaches include influenza and pneumococcal vaccination and bronchodilator therapy *(Annex 9)*. The pharmacological approach to complications and comorbidities, both internal and psychological, is an integral part of generalised management.

The basis of personalised management is treatment specific to individual COPD phenotypes *(Annex 10)*.

For every patient with severe A1AT deficiency, alpha-1-antitrypsin replacement therapy (augmentation therapy) is available to every patient in selected centres.

In the terminal phase of the disease, when patients develop respiratory failure, long-term home oxygen therapy (LTOT), home non-invasive ventilatory support (NIVP), and supportive, nutritional and palliative treatment are indicated. Palliative treatment mainly aims to eliminate severe and otherwise uncontrollable dyspnoea and includes, but is not limited to, the administration of low-dose opioids, hydration, or pain management.

Surgical treatment in case of highly selected patients with COPD includes bullectomy, lung volume reduction surgery (LVRS), bronchoscopic lung volume reduction (BLVR), or lung transplantation (the lung transplant center is at the University Hospital in Motol in Prague).

6 Smoking Cessation

About 40% of patients with COPD continue to smoke. Most smokers would like to quit their addiction, up to 40% of them try to do so every year. However, attempts without professional help are rarely successful (around 3-4%).

The prevalence of smoking in the Czech Republic has gradually decreased, but has been stagnating since 2020. Currently, 24.6% of the population is smokers (population over 15 years of age). E-cigarettes (EC) are used by 6.1% of the population over 15 years of age and heated tobacco by 4.4%. For 21.8% of EC users, it is a means to quit or reduce smoking conventional cigarettes and 24.3% say that they perceive them as less harmful to their health. Passive smoking is also still a major problem, with 18% of the population exposed to cigarette smoke at home and 20% at the workplace.

Tobacco dependence is (i) psycho-socio-behavioural, involving the attachment of smoking to specific situations, society, and activities, and (ii) physical, which is classic drug dependence present in the majority of smokers (80–90 %). It involves the multiplication of acetylcholine-nicotine receptors and, above all, withdrawal symptoms when nicotine is excluded. Withdrawal symptoms can be suppressed by first-line drugs: varenicline, nicotine, bupropion and, more recently, cytisine. However, pharmacotherapy must always be combined with intervention – the smoker must actively change their daily stereotypes and address their psycho-socio-behavioural addiction, which medication cannot help. Many addicts also use nicotine replacement therapy (nicotine patches, gum, lozenges, sachets, etc.), which delivers nicotine to the body without the several thousand harmful substances of tobacco smoke. Smokers who, despite their best efforts, have not been able to quit in the last few years also use nicotine in the form of electronic cigarettes or heated tobacco, an alternative to smoking that has the potential to reduce the health risks of cigarette smoking.

7 Pulmonary Rehabilitation

Pulmonary rehabilitation in patients with COPD is always part of comprehensive interdisciplinary care and is an important part of treatment. It is recommended that all COPD patients who are symptomatic engage in exercise training (3-5× per week, 20-60 minutes, 6-8 weeks) regardless of lung function. This rehabilitation may be indicated during hospitalization, outpatient treatment or spa treatment or treatment in specialized medical institutions.

During pulmonary rehabilitation, newly diagnosed patients with COPD are acquainted with the nature of their disease, lifestyle measures and how to act in times of acute exacerbation.

Regular physical activity (preferably walking) is extremely beneficial for all patients with COPD, so patients should be encouraged to maintain activity and at the same time the level of daily physical activity should be regularly monitored (e.g. with pedometers).

Regular physical activity favourably modifies the course of the disease and eliminates its impacts. The beneficial effect on mortality has already been proven by indirect evidence (more steps = longer life).

8 COPD Exacerbation

The long-term stable course of COPD can occasionally be interrupted in some patients by a permanent deterioration that exceeds the normal daily fluctuations in symptoms. These attacks of worsening symptoms, which last ≥ 2 days and require a change in medication and/or hospitalization, are called acute exacerbations (AEs). COPD exacerbations can be triggered by a variety of factors. The most common cause is respiratory infections.

The treatment of exacerbation aims to minimise the adverse effects and prevent subsequent side effects. In order to initiate treatment, short-acting inhaled beta$_2$ agonists (SABA) alone or with the addition of anticholinergics are recommended. After an exacerbation, long-acting bronchodilators should be started immediately. Systemic corticosteroids (CS) may relieve obstruction (FEV_1), improve oxygenation, accelerate recovery, and reduce hospitalisation time. However, their administration should not be longer than 5–7 days. Antibiotics, if indicated, can speed recovery and reduce the risk of early relapse and treatment failure. They also reduce the length of hospital stays. Treatment should not last longer than 5–7 days. In cases of acute respiratory failure, low-flow oxygen therapy is often necessary. When there's significant hypercapnia or respiratory acidosis, non-invasive mechanical ventilation should be employed. It improves gas exchange, reduces the strain on respiratory muscles, and the need for intubation. Consequently, it shortens the duration of hospitalization and enhances the patient's survival.

Exacerbations are classified according to their severity:

- mild (treated with SABA only)
- moderate (treated with SABA + ATB or oral corticosteroids)
- severe (hospitalisation or emergency department visit is required); usually associated with acute respiratory failure

9 Respiratory failure treatment and palliative care

The inability of the respiratory system to ensure the necessary gas exchange in the lungs is referred to as respiratory insufficiency. This is either type I – hypoxemic (normal PaCO2, hypoxemia ($\bar{}$ PaO2)) or type 2 – hypoxemic-hypercapnic (hypercapnia (PaCO2), hypoxemia ($\bar{}$ PaO2)). It is divided into (i) acute, which is a sudden disorder that is uncompensated, or (ii) chronic, which has developed compensatory and adaptation mechanisms.

Long-term home oxygen therapy (DDOT) in patients with COPD has been shown to alleviate symptoms, exercise capacity, improve cognitive function, quality of life and hospitalization. The criteria for DDOT in the Czech Republic are: resting PaO2 < 7.3 kPa or resting PaO2 7.3–8.0 kPa + one of the following criteria: pulmonary hypertension or polyglobulia or saturation < 90% during at least 30% of sleep time or exercise-induced desaturation.

A 6-month smoking abstinence is required. An oxygen concentrator or portable liquid oxygen is used for a minimum of 16 hours a day.

In stable patients with COPD and chronic hypercapnic respiratory failure, home non-invasive ventilation (dNIV) is indicated. Treatment leads to improved lung function, blood gas levels and quality of life. In the case of concomitant hypoxemia, concomitant DDOT is also indicated.

Palliative and end-of-life care, including pharmacological treatment of COPD symptoms, pain management, rehabilitation, oxygen therapy, non-invasive ventilation, opioid administration, pharmacological sedation, or treatment for depression and anxiety, should be offered to end-stage patients, and the choice of treatment should depend on current clinical challenges.

10 Conclusion

The diagnosis and treatment of COPD have undergone significant changes in recent years. While pharmacotherapy is an essential component of the disease's comprehensive treatment, it is becoming increasingly personalized.

The cornerstone of pharmacotherapy is bronchodilation treatment. Monotherapy with long-acting anticholinergics (LAMA), or dual therapy in a fixed combination with β_2 agonists (LAMA + LABA), is typically employed. For specific phenotypes (ACOS, frequent exacerbations), the inclusion of inhaled corticosteroids (ICS) in the treatment regimen is appropriate. Currently, fixed triple combinations (ICS + LABA + LAMA) are also consolidating their position in the therapeutic scheme for COPD.

Drugs are inhaled and available in different inhalation systems; therefore, continuous and repeated patient education in inhalation techniques is essential.

Non-pharmacological treatment has also significantly developed. The emphasis is mainly on the physical activity of patients and complex pulmonary rehabilitation.

Maximum efforts should be made in prevention, especially not smoking cigarettes or inhaling other fumes.

11 Resources

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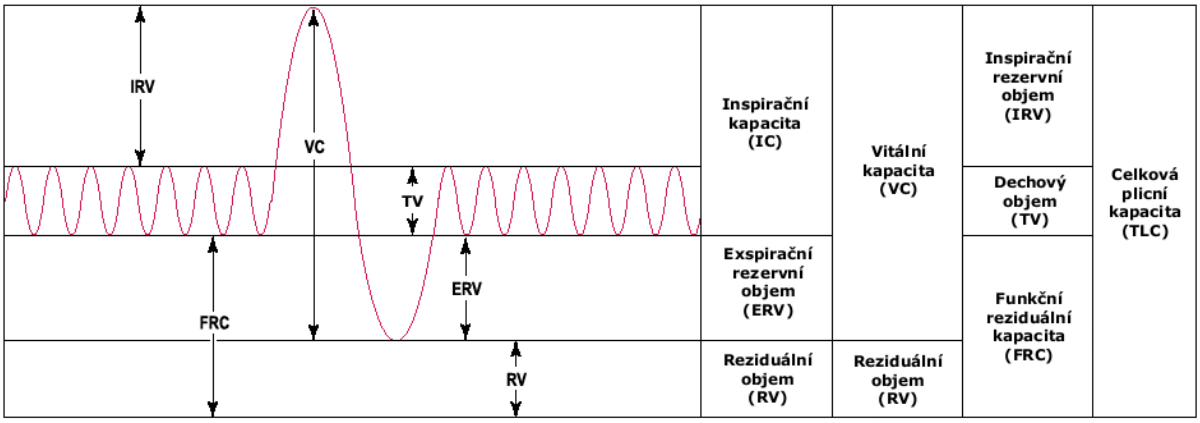
Appendix 1

Lung volumes

Lung volumes are the spaces that are occupied by air in the lungs during breathing. The combination of individual volumes creates the so-called lung capacities. To measure ventilation, a functional examination of the lungs (spirometry) is used, which is an examination of lung volumes, capacities and flows. Measured parameters are divided into static and dynamic. Static parameters are the volumes and capacities of the respiratory system (tidal volume, vital capacity, etc.), which inform about possible restrictive disorders. Dynamic parameters are flow rates or volumes during maximally forced breathing (minute ventilation, maximum minute ventilation, etc.), which inform about obstructive disorders. Lung volumes are affected by the individual's height, weight, age, training, gender, and health status.



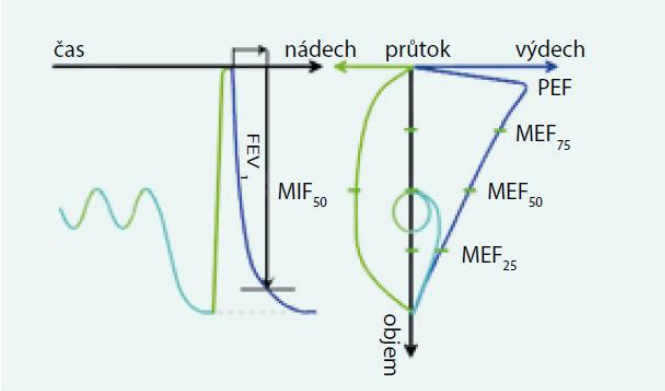
- *Inspirační kapacita (IC)* *Inspiratory Capacity (IC)*
- *Exspirační rezervní objem (ERV)* *Expiratory Reserve Volume (ERV)*
- *Reziduální objem (RV)* *Residual Volume (RV)*
- *Vitální kapacita (VC)* *Vital Capacity (VC)*
- *Inspirační rezervní objem (IRV)* *Inspiratory Reserve Volume (IRV)*
- *Dechový objem (TV)* *Tidal Volume (TV)*
- *Funkční reziduální kapacita (FRC)* *Functional Residual Capacity (FRC)*
- *Celková plicní kapacita (TLC)* *Total Lung Capacity (TLC)*

Pulmonary Function Testing (Spirometry)

Pulmonary function testing is a laboratory method, the results of which have a significant impact on the correct diagnosis and treatment. It is one of the primary internal examination methods, similar to blood pressure measurement or ECG. Concerning the patient, the examination is easy, simple, and infinitely repeatable. It must be performed by trained personnel and standardised procedures.

Spirometry is a physiological test measuring the volume of inhaled or exhaled air as a function of time. The primary examination includes measuring resting (static) and dynamic volume parameters. The basic parameters monitored include: resting expiratory (EVC) or inspiratory (IVC) vital capacity, forced vital capacity (FVC), forced expiratory volume in 1 second (FEV_1), peak expiratory flow (PEF), and maximum expiratory flows at different levels of forced vital capacity (MEF_{25}, MEF_{50}, MEF_{75}). The FEV_1/FVC ratio is called the Tiffeneau index, the physiological value of which is 80 % (0.8). It helps in the diagnosis of obstructive and restrictive lung disease. If the value is 0.7 or less, COPD is suspected.

Either volume or flow is measured. The volume measurement is expressed on a volume/time curve, while the flow measurement is shown on a flow/volume loop:



MEF – maximální výdechové průtoky na různých úrovních FVC
MIF_{50} – střední nádechový průtok v úrovni 50 % nadechnuté FVC

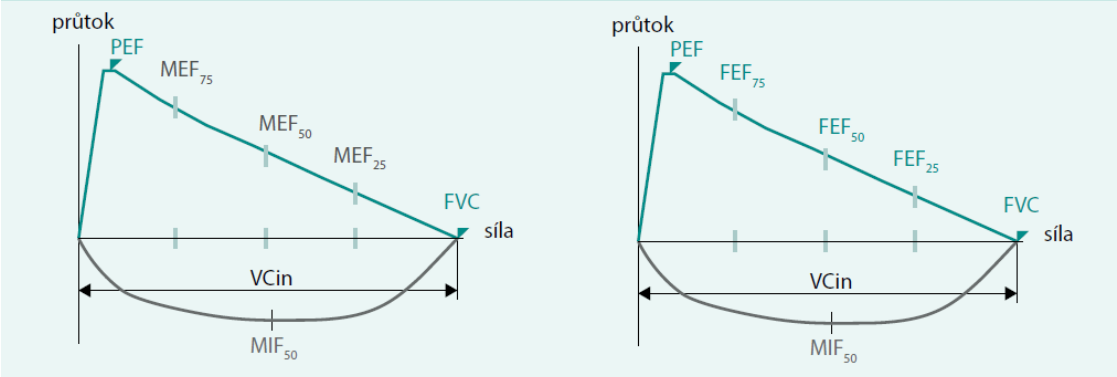
- *čas* — *Time*
- *nádech* — *Inspiration*
- *průtok* — *Flow*
- *výdech* — *Expiration*
- *objem* — *Volume*
- *MEF – maximální výdechové* — *MEF – maximum expiratory flow rates*

průtoky na různých úrovních FVC — *at different FVC levels*

- *MIF_{50} – střední nádechový průtok* — *MIF_{50} – mean inspiratory flow*

v úrovni 50 % nadechnuté FVC — *at 50 % of inspired FVC*

Forced Expiratory Flow Rates:



A_{ex} – plocha pod výdechovou částí křivky průtok-objem **FVC** – usilovná vitální kapacita **FEF_{25-75}** – usilovný výdechový průtok **MEF_{25-75}** – maximální výdechový průtok **MIF_{50}** – střední nádechový průtok v úrovni 50 % nadechnuté FVC **PIF** – maximální průtok dosažený na vrcholu usilovného nádechu **VCin** – nádechová vitální kapacita

- *průtok*	*Flow*
- *síla*	*Power*
- *A_{ex} – plocha pod výdechovou částí*	*A_{ex} – area under the exhalation section*
křivky průtok-objem FVC	*of the flow-volume curve*
- *FVC – usilovná vitální kapacita*	*FVC – forced vital capacity*
- *FEF_{25-75} – usilovný výdechový průtok*	*FEF_{25-75} – forced expiratory flow*
- *MEF_{25-75} – maximální výdechový průtok*	*MEF_{25-75} – maximum expiratory flow*
- *MIF_{50} – střední nádechový průtok*	*MIF_{50} – mean inspiratory flow at 50 % of*
v úrovni 50 % nadechnuté FVC	*inspired FVC*
- *PIF – maximální průtok dosažený*	*PIF – peak inspiratory flow*
na vrcholu usilovného nádechu	
- *VCin – nádechová vitální kapacita*	*VCin – inspiratory vital capacity*

Bronchodilator test (BDT)

It is indicated in the differential diagnosis of dyspnoea, cough, chest pressure, in monitoring the condition of patients and the effect of treatment, to evaluate the effect of individual bronchodilator drugs, in preoperative examination and for assessment purposes. We also perform the test if the values are within the appropriate values, but the examinee reports clinical difficulties.

Ventilation parameters are measured 30 minutes after administration of the bronchodilator. If a bronchodilator beta2 mimetic with a rapid onset of action (salbutamol) is used, it is possible to perform the measurement earlier (but at least in 15 minutes).

A change in FEV1 ≥ 10% indicates test positivity.

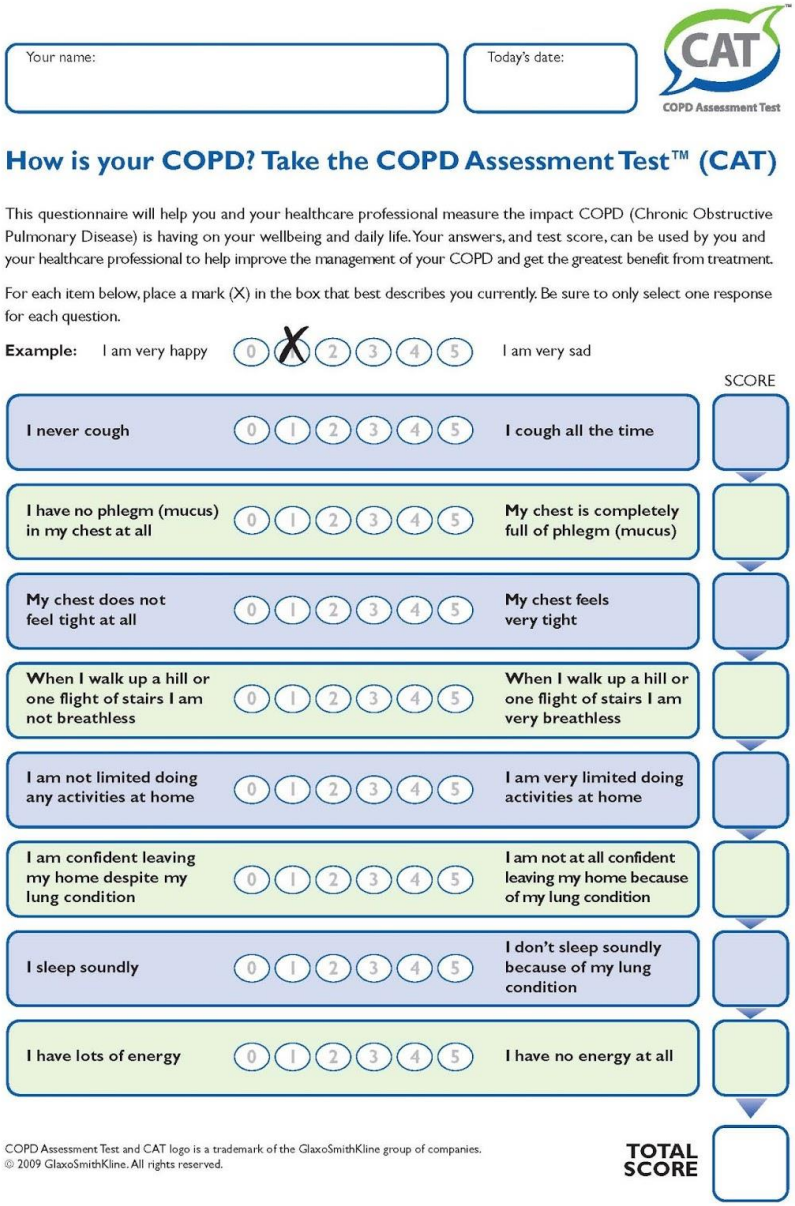
Appendix 2: CAT = COPD Assessment Test

The CAT questionnaire is used to assess the severity of COPD symptoms and includes 8 areas of quality of life.

The score is from 0−40 points (0−5 for each area) and fully depends on the patient's subjective assessment.

It focuses on shortness of breath and other manifestations of COPD – cough, expectoration, chest tightness, limitation of daily activity, sleep disorders and fatigue.

It is advisable to fill in the CAT questionnaire at each check-up of the patient and, based on the results obtained, to monitor the effect of the chosen treatment on the symptoms of COPD.



Your name:

Today's date:

CAT
COPD Assessment Test

How is your COPD? Take the COPD Assessment Test™ (CAT)

This questionnaire will help you and your healthcare professional measure the impact COPD (Chronic Obstructive Pulmonary Disease) is having on your wellbeing and daily life. Your answers, and test score, can be used by you and your healthcare professional to help improve the management of your COPD and get the greatest benefit from treatment.

For each item below, place a mark (X) in the box that best describes you currently. Be sure to only select one response for each question.

Example: I am very happy 0 **X** 2 3 4 5 I am very sad

			SCORE
I never cough	0 1 2 3 4 5	I cough all the time	
I have no phlegm (mucus) in my chest at all	0 1 2 3 4 5	My chest is completely full of phlegm (mucus)	
My chest does not feel tight at all	0 1 2 3 4 5	My chest feels very tight	
When I walk up a hill or one flight of stairs I am not breathless	0 1 2 3 4 5	When I walk up a hill or one flight of stairs I am very breathless	
I am not limited doing any activities at home	0 1 2 3 4 5	I am very limited doing activities at home	
I am confident leaving my home despite my lung condition	0 1 2 3 4 5	I am not at all confident leaving my home because of my lung condition	
I sleep soundly	0 1 2 3 4 5	I don't sleep soundly because of my lung condition	
I have lots of energy	0 1 2 3 4 5	I have no energy at all	
		TOTAL SCORE	

Appendix 3: mMRC – Medical Research Council Modified Dyspnoea Scale

The mMRC scale is used to assess the severity of symptoms, where grades 0–4 are defined.

The physician determines the grades based on the patient's level of exertional dyspnoea, similar to the NYHA classification used in cardiology.

mMRC Grade 0	I only get breathless with strenuous exercise.
mMRC Grade 1	I get short of breath when hurrying on level ground or walking up a slight hill.
mMRC Grade 2	On level ground, I walk slower than people of the same age.
mMRC Grade 3	I stop for breath after walking about 100 yards or after a few minutes on level ground.
mMRC Grade 4	I am too breathless to leave the house, or I am breathless when dressing.

Appendix 4: COPD Phenotypes

There are six clinical phenotypes of COPD, which can be defined based on clinical manifestations, imaging examinations, functional changes, and other parameters.

Some patients may have more than one of these phenotypes.

According to the phenotype, the management of these patients is not only generalised but also personalised.

	BRONCHITIS PHENOTYPE
	• productive cough • (> 3 months/year in the last two years or more)
	EMPHYSEMA PHENOTYPE
	• lifelong absence of productive cough • dry cough may be present • simultaneous signs of pulmonary emphysema • (according to chest HRCT and functional examination)
	COPD BRONCHIECTASIS PHENOTYPE
	• accentuated daily expectoration • younger age • non-smokers or less intense smokers • prolonged or repeated infections of the lungs or lower respiratory tract • haemoptysis, or the presence of blood in the mucus • HRCT signs of bronchiectasis
	COPD OVERLAP WITH BRONCHIAL ASTHMA
	Two major or one major + two minor criteria must be met Major criteria:

(a) significantly positive BDT (rise of $FEV_1 \geq 15$ % a ≥ 400 ml)
(b) positive bronchoconstriction testing
(c) FENO ($\geq$ 45–50 ppb) or eosinophils in sputum ($\geq$ 3 %)
(d) history of bronchial asthma

Minor criteria:
(a) positive BDT (rise of $FEV_1 \geq 12$ % a ≥ 200 ml)
(b) in total IgE
(c) history of atopy

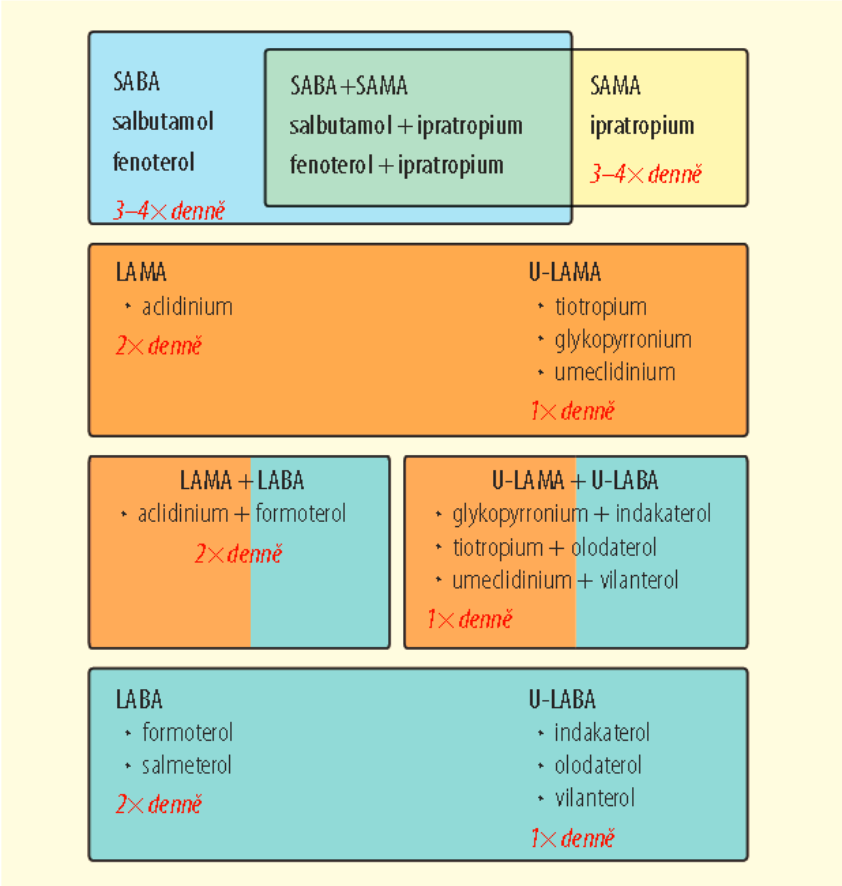
FREQUENT EXACERBATION PHENOTYPE

- frequent acute exacerbations ($\geq$ 2/year) treated with ATB or systemic steroids

PULMONARY CACHEXIA PHENOTYPE

- reduced FFMI (men < 16 kg/m^2, women < 15 kg/m^2), or BMI < 21 kg/m^2 (irrespective of gender) – with no other apparent cause

Appendix 5: Selected Inhaled Bronchodilators



- *3–4xdenně* — *3–4x per day*
- *2xdenně* — *2x per day*
- *1xdenně* — *1x per day*

Principles of bronchodilator treatment:

• **Long-acting bronchodilators** should be used as a first pharmacological step in the treatment of all COPD patients with persistent symptoms who require regular treatment. Long-acting bronchodilators can be divided into two groups: Long-acting beta2 agonists (LABA) and long-acting muscarinic antagonists (LAMA), i.e. drugs with anticholinergic action.

• LAMA only or LABA only monotherapy can be used in patients with milder COPD symptoms, i.e. with a lower degree of dyspnoea with an mMRC of 0–1 and a lower rate of bronchial obstruction. In doing so, we prefer LAMA due to its greater effect on reducing the number of exacerbations compared to LABA. If inhaled corticosteroids (ICS) are indicated, they are usually used in combination with LABA or in triple combination with LABA and LAMA.