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## Usefulness of lung ultrasound in diagnosing causes of exacerbation in patients with chronic dyspnea

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### Abstract

Dyspnea is a non-specific symptom that requires fast diagnostics, accurate diagnosis and proper treatment. The most common causes of dyspnea include exacerbation of chronic obstructive pulmonary disease (COPD) and chronic heart failure (CHF). Distinction between these two medical conditions seems to be critical in diagnostics of emergencies. At the same time, basic diagnostic tools available in emergency room, such as classic radiography (X-ray) of the chest, electrocardiography (ECG) or b-type natriuretic peptide test, are sometimes ambiguous. Therefore looking for additional diagnostic tool seems to be justified and necessary. Transthoracic lung ultrasound assessment is a simple and easily accessible examination, enabling the early and explicit diagnostics of pulmonary oedema and its distinction from other, non-cardiac causes of dyspnea. This review outlines the current knowledge on the subject of transthoracic lung ultrasound (TLUS), particularly in respect of its clinical usefulness in distinction of causes of dyspnea exacerbation.

**Key words:** transthoracic lung ultrasound, dyspnea, cardiogenic pulmonary oedema

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### Introduction

Exacerbation of chronic dyspnea is a non-specific symptom that require fast diagnostics, accurate diagnosis and proper treatment. The most common causes of chronic dyspnea exacerbation include the exacerbation of chronic obstructive pulmonary disease (COPD) or the exacerbation of chronic heart failure (CHF). Other common causes of dyspnea exacerbation include, among others, pulmonary embolism, acute heart failure (AHF) with various etiology, pneumothorax, neoplasms and infectious diseases, including lung cancer and pneumonia or interstitial lung diseases.

According to estimations of WHO, COPD is one of the most common causes of death in the world, and in the immediate years we should expect an increase in the morbidity and mortality

rates caused by COPD. Cardiovascular diseases have long been regarded as diseases of civilization, which have a significant effect on the total morbidity rate of the society and considerably increase the costs of health care in developed countries. At the same time COPD constitutes an independent risk factor of developing cardiovascular diseases [1]. Frequency of hospitalization and mortality connected with exacerbation of COPD and cardiovascular diseases is considerable, and distinction between exactly these two causes of dyspnea seems to be critical in diagnostics of emergencies, and in conditions of the rescue unit it still is a challenge, particularly in the group of elderly patients, with other comorbidities.

Basic diagnostic tools available in emergency room usually include the classic radiography (X-ray) of the chest, electrocardiography (ECG) and laboratory blood tests. This diagnostics is

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sometimes expanded by the echocardiographic examination (ECHO), if it is available on site.

### **Diagnosing the cause of dyspnea in emergency room**

The chest X-ray enables quick confirmation or exclusion of inflammatory lesions in lung parenchyma. Patients suffering from COPD with advanced emphysema present with flattened diaphragm domes, increased lung transparency, and in the radiogram in lateral projection, an increased anteroposterior dimension and retrosternal airspace volume. In the case of pulmonary hypertension, one can expect a decrease or lack of the peripheral vascular pattern, dilation of pulmonary arteries and enlargement of the right heart ventricle. The chest X-ray in the case of non-infectious exacerbation of COPD in a patient without enhanced pulmonary emphysema is usually presented as normal.

The chest X-ray is also the examination of choice in diagnosing dyspneic patients with cardiogenic reasons of exacerbation. It allows revealing increased pulmonary circulation, pulmonary arterial hypertension and pulmonary venous hypertension. The former is found e.g. in congenital heart defects with left-to-right shunt or in hyperkinetic circulation connected with hyperthyroidism, arterio-venous fistulas or anemia. Pulmonary venous hypertension, that is pulmonary oedema, is caused by difficulty in blood outflow from the pulmonary circulation to the left heart atrium and it also causes changes in the radiological lung image. These changes have different images, depending on the pulmonary capillary wedge pressure (PCWP). In the initial period, dilation of higher lobe arteries and veins and narrowing of lower lobe vessels may occur. As PCWP increases, interstitial oedema occurs, visible in the X-ray as increased parenchymal structure, Kerley B lines, peribronchial cuffing and sometimes, the enlargement of the cardiac silhouette. When PCWP exceeds 25 mm Hg, alveolar oedema is developed, visible in the X-ray as symmetric opacities of lung parenchyma surrounding the hila (the so-called butterfly wings), occupying the 2/3 of the lung.

Although the above-mentioned radiological changes are typical for pulmonary oedema, they may not always be visible. Radiological image of oedema and enlargement of the cardiac silhouette is determined by the time of increasing symptoms. During the first 12 hours of acute pulmonary oedema lesions may be invisible or slight [2].

Moreover, in the left ventricular CHF, radiological symptoms of pulmonary oedema depend on the increase in pressure in pulmonary capillaries. In 2/3 of patients with advanced stable CHF, an increase in this pressure is not manifested in the form of significant radiological features of oedema, which is attributed to increased lymphatic drainage. Thus in this group of patients with dyspnea, the chest X-ray may not reveal pictures typical of pulmonary oedema, whereas the pressure in the pulmonary circulation is actually gradually increasing [2, 3].

Also it should be remembered that blood flow in pulmonary vessels depends on gravitation and the patient's position. This is of unquestionable importance in diagnostics of patients in severe conditions, requiring the bedside X-ray examination in AP projection, in lying or half-sitting position. Not only the heart and mediastinum are then subjected to ostensible geometric enlargement, but there are also changes in blood distribution, which often leads to the wrong diagnosis of dilated upper-lobe vessels. Moreover, in this position pleural effusion is located on the posterior wall of the chest and results in shadowing of the most part or the whole chest, which may hinder not only the assessment of the fluid volume but also increased alveolar opacities, typical of pulmonary oedema. Therefore, doubts concerning the causes of cardiac enlargement or the character of opacities on AP radiogram in lying position, even for an experienced radiologist, are by all means understandable. They may result from the technique of examination, actual changes in the pulmonary circulation or both causes at the same time.

One should also bear in mind that in patients with pulmonary emphysema, alveolar oedema may be not visible in the X-ray due to damaged alveolar structure of lung parenchyma in excessively inflated lung areas.

Summing up, the classic chest X-ray can be regarded as an examination of low sensitivity (14–68%) and moderate specificity (53–96%) in diagnosing pulmonary oedema [2–6]. Normal image of the chest X-ray cannot certainly rule out cardiogenic pulmonary oedema as the cause of dyspnea. The more so, because a normal chest X-ray is observed even in clinical situations of an increase in PCWP up to more than 30 mm Hg, particularly in the above-mentioned group of patients with exacerbation of CHF, previously “adopted” to changes in the pulmonary circulation. Moreover, the result of the chest X-ray depends on the experience and skills of the describing

radiologist — X-ray assessment in the position different than vertical makes an additional difficulty in determination of intensity and type of pulmonary densities in radiogram [7].

Determination of natriuretic peptide concentration in the blood is a relatively new laboratory test with high clinical usefulness. It is supposed to serve for identification of patients with heart failure (HF), among others in distinction of cardiac and pulmonary causes of dyspnea. Natriuretic peptides are secreted by cardiomyocytes in response to an increase in their tension at the increase in preload or afterload of the heart. They cause an increase in glomerular filtration and decrease in reabsorption of sodium in kidneys and inhibit secretion of renin and aldosterone. Increasing the concentration of the B-type natriuretic peptide (BNP) or the N-terminal fragment of its prohormone (NT-proBNP) in the blood indicates the activation of compensation mechanism, which occurs before developing symptoms of HF. In some studies, a relationship was proved between BNP/NT-proBNP concentration and the ejection fraction of the left ventricle and PCWP. Concentrations of these peptides correlate with the stage of HF. It is recommended to use BNP/NT-proBNP determinations in order to rule out or confirm HF in patients with ambiguous symptoms.

The normal concentration of natriuretic peptides in a patient without cardiac disease generally rules out a significant heart disease, indicating a “non-cardiac” cause of the symptoms. According to recommendations of the ESC (*European Society of Cardiology*) from 2012, threshold values of BNP and NT-proBNP concentrations are used to rule out HF. Threshold values ruling out HF differ in patients in whom symptoms of exacerbation occurred suddenly in comparison with the patients in whom the symptoms increased gradually. In patients in whom exacerbation of HF symptoms occurred suddenly, the optimal threshold value is a concentration of 100 pg/ml for BNP and 300 pg/ml for NT-proBNP. In patients who are not critically ill, the optimal threshold value ruling out HF is assumed to be 35 pg/ml for BNP and 125 pg/ml for NT-proBNP. Sensitivity and specificity of BNP and NT-proBNP determinations for diagnosing HF are higher in patients with chronically occurring symptoms [8].

Summing up, determination of BNP/NT-proBNP concentration in the blood seems to be an effective and clinically useful method for the fast and reliable identification of HF in people presenting with dyspnea. This is undoubtedly helpful in ruling out cardiac causes of dyspnea due to its high negative predictive value [9–11].

However, this is not a perfect method, due to its limited specificity. Natriuretic peptides are secreted in increased amounts not only in the case of heart failure but in every other situation of increased pre-load of any heart cavities. An increase in these hormones is also observed e.g. in the case of atrial fibrillation, pulmonary embolism, arterial hypertension and some non-cardiac diseases, including renal failure. Natriuretic peptide concentration also increases with age and during corticosteroid therapy. It can decrease, however, in obese patients [8].

Additional limitation of this method is the fact that it is still inaccessible in many hospitals in Poland [5, 12].

Electrocardiogram (ECG) shows the heart rhythm and conduction pathway, allowing the evaluation of arrhythmias as the cause of dyspnea. ECG also can show features of the left ventricular hypertrophy or the presence of Q waves (indicating a scar within myocardium), suggesting a possible cause of HF. AHF occurs very rarely in patients with completely normal ECG (probability < 2%) [8]. In patients with CHF the normal ECG recording is characterized by a slightly lower negative predictive value (probability < 10–14%) [13].

Echocardiography of heart cavities (ECHO) remains the golden standard in diagnosing heart failure as the cause of dyspnea. It enables the assessment of the left ventricular systolic function, the measurement of left ventricular ejection fraction, left ventricular diastolic function and anatomic abnormalities of the heart. Of several accessible methods for heart imaging in patients with suspected HF, ECHO remains the method of choice due to the accuracy, safety and cost of examination [8]. The limitation of this method is still its accessibility [12]. Moreover, ECHO examination should be performed by experienced echocardiographer.

In spite of ability to confirm heart failure as the cause of reported symptoms, ECHO does not allow evaluation of severity of pulmonary oedema or its monitoring.

Therefore, looking for additional diagnostic tool enabling explicit distinction between two most common causes of dyspnea, that is exacerbation of COPD and left ventricular HF, seems to be justified and necessary.

Cardiogenic pulmonary oedema develops at different rate, and accumulation of transudate in lung parenchyma often precedes its clinical manifestation and radiological changes typical for exacerbation of HF. Transthoracic lung ultrasound

(TLUS) is a simple and easily accessible examination, allowing for early and explicit diagnostics of pulmonary oedema and its distinction from other, non-cardiac causes of dyspnea. The early diagnostics of pulmonary oedema should be understood as such diagnostics that enables confirmation of pulmonary oedema in asymptomatic stage of the disease, before it will be visible in the chest X-ray.

Although TLUS can be regarded as a reliable tool to rule out heart failure as the cause of dyspnea, due to high sensitivity and negative predictive value in regard to pulmonary oedema, in everyday practice this examination is currently performed only by a small group of clinicians.

### Transthoracic lung ultrasound

Pulmonary ultrasound diagnostics has been developing relatively low and by stages, remaining in close correlation with technological progress and everyday accessibility of ultrasonographic equipment. The first reports about ultrasound imaging of lung parenchyma, including the Polish author, doctor Janusz Grymiński, date at the turn of the 1970s and 1980s [14–16]. In the 1990s there was a significant progress in lung ultrasound imaging, connected with the use of this method in diagnostics of direct life-threatening conditions. Theoretical and practical aspects of TLUS were systematized then and the ultrasonographic criteria of diagnosing most common pathological changes in the respiratory system were developed. The French researcher Daniel Lichtenstein made the greatest contribution to development and dissemination of lung ultrasound diagnostics. His works focused mostly on the possibility of using ultrasound diagnostics to assess changes in lungs in a group of patients with direct life-threatening condition. In 1992, Lichtenstein was the first to publish the results of study presenting the usefulness of ultrasound in diagnostics of pulmonary oedema. The following years brought much scientific research and meta-analyses which proved the usefulness of this diagnostic tool mainly in emergency [17–37]. Eventually, in 2012 a group of experts appointed to standardize TLUS published evidence-based guideline containing a list of recommendations for clinical application of lung ultrasound [38].

TLUS can be performed with the ultrasound equipment of any class. However, one should bear in mind that in most ultrasound devices technologically advanced imaging options are already available, such as: harmonic imaging (emission of enhanced harmonic echo), XRES or SonoCT

(techniques of artifact elimination). They allow more precise imaging of lung parenchyma consolidation areas, but they considerably change the image of ultrasound artifacts, making their correct interpretation impossible. Therefore, it should always be possible to switch off these options.

The examination uses the *convex* probe (standard probe for assessment of abdominal cavity) with frequency of 3.5–5.0 MHz and a linear probe of high frequency (8.0–10.0–12.0 MHz). TLUS includes standard evaluation of longitudinal scans, for which the recommendations for lung ultrasound were developed with the use of artifact analysis in the study with the *convex* probe. When operating the linear probe, the oblique scans are assessed, which enables obtaining much more detailed images of superficial structures — chest walls, pleura or subpleural focal lesions.

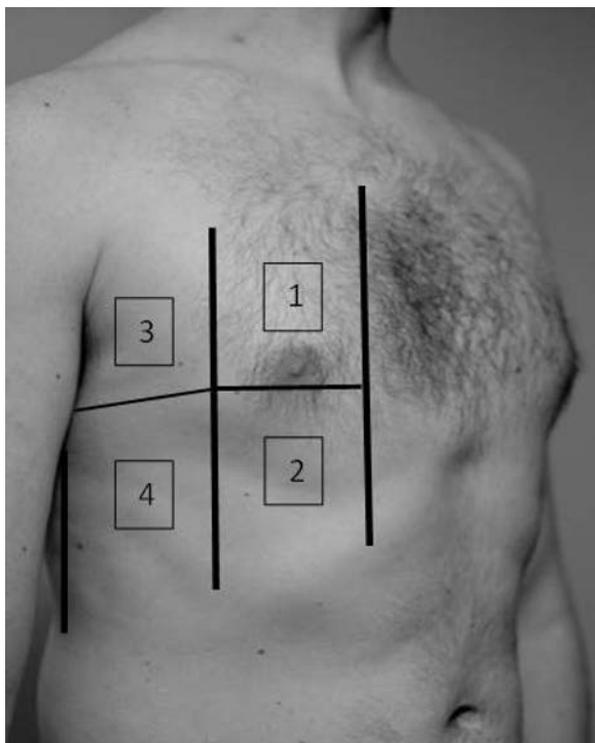
Lung ultrasound examination can be performed in the lying, sitting, half-sitting or standing position (depending on needs and the patient's condition and abilities.) Particular lung areas are assessed moving the probe along the intercostal space in the anterior, lateral and posterior surface of the chest.

There are several TLUS techniques and protocols in dyspnea diagnostics [18, 38–42]. Experts allow three schemes of lung ultrasound examination. In one of them, division of the chest into four areas on each side is proposed, thus the probe application in eight zones on the anterior and lateral surface of the chest is used (Fig. 1).

The other two schemes of the chest ultrasound examination comprise one faster method used in emergencies, with the probe application in two places in front of the chest and the other — more extended, evaluating each intercostal space.

The basic option of lung imaging is the B-mode option. If any abnormalities of lung parenchyma are observed, it is necessary to perform a comparative examination in lung areas regarded as normal. Echogenicity of the observed lesions is compared with the image of reference regions. For solid focal lesions (regions of lung parenchyma consolidations) the reference region is the echogenicity of normal liver parenchyma. For fluid lesions, including pleural effusion, the echogenicity of the gall bladder. Echogenicity of a lesion is referred to as isoechogenic if it is equal to the echogenicity of the normal liver parenchyma and respectively, hypoechogenic (lower) or hyperechogenic (higher) than the echogenicity of liver parenchyma [43].

In healthy individuals revealing the organs situated inside the chest is not possible due to the

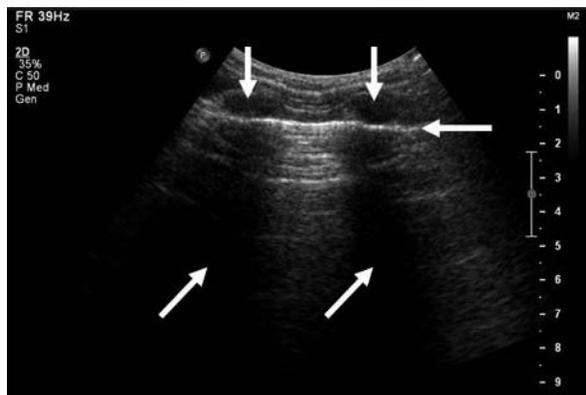


**Figure 1.** The four chest areas per side considered for complete lung ultrasound examination, modified from Volpicelli et al. Areas 1 and 2 denote the upper anterior and lower anterior chest areas, respectively. Areas 3 and 4 denote the upper lateral and basal lateral chest areas. The horizontal lines are from median line: parasternal line, anterior axillary line and posterior axillary line, respectively [38]

presence of air in lungs, which according to the laws of physics prevents ultrasound transmission into deeper structures, causing almost complete reflection and dispersing of emitted ultrasonic waves. Thus according to one of the researchers, Giovanni Volpicelli, the ultrasound image of a normally aerated lung can be compared to the mirror effect [44].

In lung parenchyma air stays in close contact with water in a relatively large area, and mutual interaction of those two media leads to formation of artifacts, enabling lung ultrasound imaging. These artifacts can be divided into static, dynamic and those used in assessment of inflammatory lesions in lungs and in pulmonary embolism.

The normal lung ultrasound image is composed of the following structures: skin and subcutaneous tissue, a layer of muscles, intrathoracic fascia, the pleural line and homogenous, slightly hyperechogenic pattern of reflections corresponding with a normally aerated lung. In the longitudinal scan, acoustic shadows of two neighboring ribs are visible (due to lack of ultrasound penetration through bone structures) and a hyperechogenic line between them (visible about



**Figure 2.** Ultrasound scan of a normal lung, using convex probe. Two adjacent ribs (vertical arrows) with acoustic shadows below (oblique arrows) and hyperechogenic pleural line (horizontal arrow) creating ‘the bat sign’

5 mm below the rib line), giving the image of the so-called “bat sign” (Fig. 2). This hyperechogenic line is the line of pleura, which is composed of the parietal pleura, the visceral pleura and a small amount of fluid between them. In normal conditions it is smooth, its thickness should not exceed 2 mm and it moves synchronously with respiration, giving the characteristic, dynamic sign of lung sliding [43].

A static artifact occurring in a healthy lung is A-lines. These are horizontal, slightly hyperechogenic lines arising. In normal conditions A-lines are formed on the pleural line and are artifacts of reverberation that is the effect of ultrasound wave reflection on the boundary between the air in the alveoli below the visceral pleura and the chest wall. Hence the distance between successive A lines is a repetition of the distance between the pleural line and the chest wall. The positive sign of lung sliding combined with the presence of A lines make the image of a healthy, normally aerated lung.

Apart from the above-mentioned air filling the alveoli, the presence of osteo-chondrous structure of the chest (ribs, scapulae, spine) may seem to be additional difficulty in imaging in TLUS. However, intercostal spaces make sufficient physiological acoustic windows, enabling the examination of lung parenchyma.

In about 98.5% of cases clinically significant pathological changes occurring in adult patients in emergencies have direct contact with the visceral pleura [19]. Such location fully justifies the use of lung ultrasound imaging in emergency room.

In lung parenchyma partial loss of airflow results in disturbances of balance between the

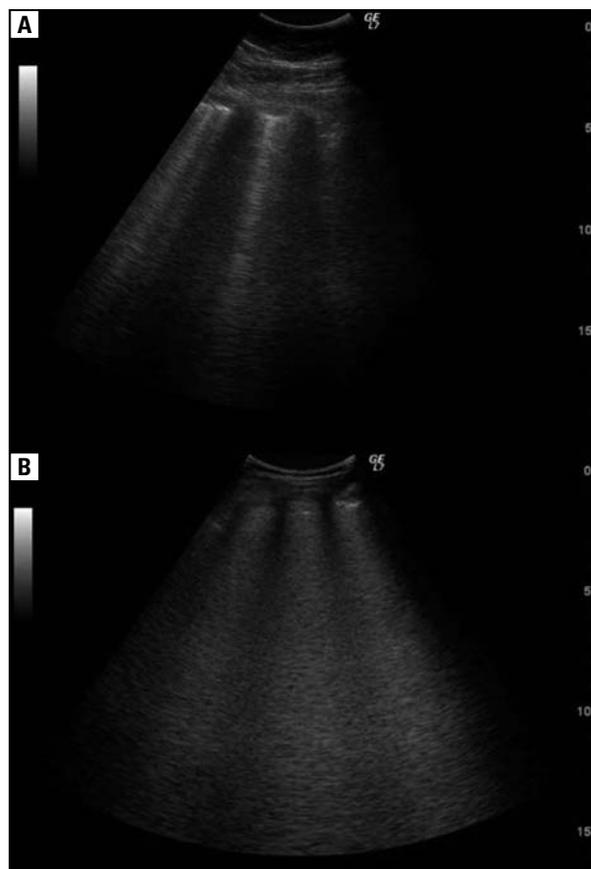
content of water and air, and ultrasound images are highly sensitive to changes in this balance. Pulmonary oedema is the perfect example of this.

In pulmonary oedema there is an increase in pressure in pulmonary capillaries, which results in transudate to parenchyma and pulmonary alveoli. Thus there is the above-mentioned disturbance of balance between water and air volume in the lung parenchyma. These disturbances result in appearance of B lines in ultrasound lung imaging. Phenomena which result in formation of B lines are still the subject of discussion [40, 45, 46]. These artifacts are most likely connected with the presence of a small amount of water under the visceral pleura, most often in interalveolar septa and a difference in sound transmission velocity for water and air. In ultrasound examination B-lines, also addressed as comet-tail artifacts, are presented as vertical, hyperechogenic acoustic shadows arising from the pleural line, extending to the bottom of the screen without fading and moving synchronously with lung sliding.

The presence of one or two B-line artifacts in one longitudinal scan of the chest is regarded as normal (Fig. 3A). These artifacts are more hyperechogenic during inhalation, and their number is closely connected with the degree of lung aeration. Hence single B lines are observed in a healthy, normally aerated lung, usually in parbasal parts of the lower lobes, which should be regarded as physiological [38, 43].

In conditions of pulmonary oedema of different etiology, B-lines are more numerous creating the so-called interstitial syndrome, occurring bilaterally and symmetrically. Observation of 3 and more B lines bilaterally, in at least two areas described above, justifies diagnosing the interstitial syndrome (Fig. 3B).

Summing up, observing numerous artifacts of B lines indicates explicitly an increase in extravascular water volume in lung parenchyma. It was documented that the presence of B lines  $7 \text{ mm} \pm 1$  apart in imaging with the convex (or sector) probe results from interlobular septal thickening and it is characteristic of interstitial oedema (described as B7-lines profile). B lines located  $3 \text{ mm} \pm 1 \text{ mm}$  apart correspond to both increased oedema of interlobular septa and the presence of fluid in the lumen of pulmonary alveoli and correlate with the image of the so-called ground glass observed in the chest CT of high resolution. Thus the number of observed B lines correlates with PCWP and with radiological signs of pulmonary oedema [17]. Ultrasound image confirming the presence of the interstitial syndrome was regarded as explicit



**Figure 3.** Ultrasound scan of a single B-line in a normal lung (A) and interstitial syndrome in pulmonary oedema called 'white lung' (B)

and justifying distinction of cardiac pulmonary oedema from exacerbation of COPD [38].

Ultrasound examination in pulmonary oedema appears to have an advantage over the chest X-ray, both in respect of sensitivity and specificity [38]. Also a strict correlation was proved between the presence of B lines in TLUS and the concentration of BNP in the blood of patients with cardiogenic pulmonary oedema [47].

An additional advantage of TLUS as a repeatable, dynamic and safe examination is the possibility of monitoring patients with pulmonary oedema. It allows an objective assessment of treatment effectiveness or possible verification of the initial diagnosis [38, 48–50].

Moreover, according to the experts, the qualitative assessment of B lines is the prognostic index in the group of patients with decompensated heart failure [38]. Correlation between the number of B lines and mortality in a 3-month observation in a group of patients with exacerbation of chronic heart failure was described [38]. According to the previous reports, the number of B lines allows even forecasting the duration of a patient's stay in hospital [51].

**Table 1. Summary of studies included by Al. Deeb et al. in meta-analysis evaluating the relevance of point-of-care ultrasonography for the diagnosis of acute cardiogenic pulmonary oedema in patients presenting acute dyspnea [5]**

Author	Lichtenstein	Lichtenstein	Gargani	Liteplo	Vitturi	Prosen	Cibinel
Year	1998	2008	2008	2009	2011	2011	2012
Journal	Intensive Care Medicine	Chest	European Journal of Heart Failure	Academic Emergency Medicine	Ultrasound	Critical Care	International Emergency Medicine
Number of patients (number in final analysis)	146 (146)	301 (260)	149 (149)	100 (94)	152 (152)	248 (218)	5 (56)
TLUS protocol	Lichtenstein	Lichtenstein	Comet score	Volpicelli	Comet score	Volpicelli	Volpicelli
Time to complete TLUS	< 1 min	< 3 min	< 5 min	< 5 min	< 3 min	< 1 min	< 5 min
Sensitivity (%)	100	97	76.2	58	97	100	93.6
Specificity (%)	97	95	88	85	79	95	84

It is notable that the time of TLUS in a patient with dyspnea, depending on the used examination protocol, lasts from 1 to 5 min [20, 23, 41]. The image of B line artifacts is very characteristic and it does not require large experience of the investigator [5, 29].

Ultrasound imaging in diagnostics of pulmonary oedema is documented in many studies that were subjected to meta-analysis in 2014 by AlDeeb et al. Eventually, 7 articles were included in the systematic review (Table 1). The authors regarded TLUS revealing B lines as an examination with a high positive predictive value among patients with moderate and high risk of cardiogenic pulmonary oedema. In the group of patients with low risk of such oedema, the lack of B lines in TLUS allows with high probability ruling out a cardiac cause of dyspnea.

Apart from oedema, B lines are also found in other diseases. Focally located B lines are present in pneumonia, atelectasis, lung injury and pulmonary infarction, proliferative diseases or pleural diseases. Numerous, dispersed B lines, located bilaterally, occur in interstitial lung diseases, fibrosis of different etiology or ARDS. However, the assessment of other ultrasonographic artifacts, e.g. the symptom of lung sliding, pleural irregularities small subpleural consolidations, combined with the clinical picture and results of other additional examinations, allow their reliable distinction.

TLUS is also useful in diagnostics of less common causes of dyspnea, for instance in pneumothorax. In 2004, the concept of EFAST (*Extended Focused Assessment with Sonography for Trauma*) was introduced. Thus the classic diagnostic algorithm, referred to as FAST (*Focused*

*Assessment with Sonography for Trauma*), was extended to include lung ultrasound examination in patients with life-threatening conditions, in order to rule out or confirm the presence of pneumothorax and pleural effusion. In 2008, Lichtenstein et al. presented the so-called *BLUE protocol (BLUE, Bedside Lung Ultrasound in Emergency)* — the first ultrasound algorithm directed towards identification of life-threatening conditions leading to acute respiratory distress [23]. This algorithm has already found its place in emergency.

Ultrasound lesions suggesting pulmonary embolism and those typical of pneumonia have already been well described as well. They are equally characteristic and clinically useful.

## Conclusion

The usefulness of TLUS in distinction of the most common causes of dyspnea, that is exacerbation of COPD and cardiogenic pulmonary oedema, is unquestionable. The need of an additional diagnostic tool in distinction of those two groups of patients seems to be obvious and ultrasonography can effectively satisfy this need in conditions of the hospital emergency unit. TLUS allows for fast verification of pulmonary changes, especially in cases when the result of the chest X-ray is ambiguous. In the group of patients with acute respiratory failure, when time is short, the accuracy of initial diagnosis is critical and it determines the further conduct and treatment.

There are reliable arguments for benefits resulting from the practical usefulness of TLUS, in the form of convincing studies and meta-analyses. Therefore it seems that presently the biggest

drawback of TLUS is the lack of its acceptance among clinicians, which makes it impossible to add this useful tool to the panel of basic diagnostic examinations that facilitate distinction between causes of dyspnea.

### Conflict of interest

The authors declare no conflict of interest.

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