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Review article

Pleural manometry–historical background, rationale for use and methods of measurement

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ABSTRACT

Subatmospheric pleural pressure (Ppl), which is approximately −3 to −5 cmH_2O at functional residual capacity (FRC) makes pleura a unique organ in the human body. The negative Ppl is critical for maintaining the lungs in a properly inflated state and for proper blood circulation within the thorax. Significant and sudden pleural pressure changes associated with major pleural pathologies, as well as therapeutic interventions may be associated with life-threatening complications.

The pleural pressure may show two different values depending on the measurement method applied. These are called pleural liquid pressure and pleural surface pressure. It should also be realized that there are significant differences in pleural pressure distribution in pneumothorax and pleural effusion. In pneumothorax, the pressure is the same throughout the pleural space, while in pleural effusion there is a vertical gradient of approximately 1 cm H_2O/cm in the pleural pressure associated with the hydrostatic pressure of the fluid column.

Currently, two main methods of pleural pressure measurement are used: simple water manometers and electronic systems. The water manometers are conceptually simple, cheap and user-friendly but they only allow the estimation of the mean values of pleural pressure. The electronic systems for pleural pressure measurement are based on pressure transducers. Their major advantages include precise measurements of instantaneous pleural pressure and the ability to display and to store a large amount of data. The paper presents principles and details of pleural pressure measurement as well as the rationale for its use.

1. Introduction

Although the pleura is only a potential space between the lung and chest wall it plays an important role in respiratory physiology. The function of respiratory muscles results in cyclic changes in pleural pressure which are directly responsible for both inspiration and expiration. Additionally, the narrow space between the visceral and parietal pleura, that typically contains a thin layer of fluid (0.5–2 ml) reduces frictional forces and allows the lung to slide along the internal surface of the chest cavity during respiratory movements [1]. The normal pleural pressure, which is approximately −3 to −5 cmH_2O at functional residual capacity (FRC), results from the counteracting elastic recoil forces of the lung and the chest wall [1,2]. As pleural pressure is lower than the atmospheric pressure during the whole respiratory cycle, it is often referred to as “a negative pleural pressure”. In this regard the pleura is a unique organ in the human body.

The subatmospheric pleural pressure is an important factor involved in pathophysiology of pleural diseases. This refers to both major pleural pathologies, i.e. pneumothorax and pleural effusion. When alveolar wall ruptures or penetrating thoracic trauma creates a communication between the pleural cavity and ambient air, the air flows into the pleural space in order to equalize the pressures. Air accumulation in the pleural space that is termed pneumothorax is invariably associated with an increase in pleural pressure which results in partial or complete lung collapse and increase in size of the hemithorax. The negative pleural pressure can also promote accumulation of pleural fluid. The disruption of the delicate balance between the production and reabsorption of pleural fluid in favor of its production may result in pleural fluid accumulation. Similarly to pneumothorax, accumulation of pleural effusion is usually associated with an increase in pleural pressure with a secondary decrease in the volume of the affected lung and increase in size of the hemithorax.

Both pneumothorax and pleural effusion are common conditions in clinical practice. It is estimated that pleural effusion affects approximately 1.5 million patients per year in the USA [3]. The number of diagnostic and therapeutic thoracenteses in the USA is quite high and was reported to be between 127,000 and 173,000 procedures per year [4,5]. Withdrawal of a large volume of pleural fluid (therapeutic

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thoracentesis) or pneumothorax aspiration may be associated with a significant decline in pleural pressure. It is thought that an uncontrolled decrease in pleural pressure is one of the mechanisms involved in development of reexpansion pulmonary edema, a rare but severe complication of pleural air or pleural fluid removal. To reduce the risk of reexpansion pulmonary edema, the measurement of pleural pressure changes during pleural fluid withdrawal seems to be a reasonable solution. More than 30 years ago Light et al. [6] showed that in individual patients therapeutic thoracentesis was associated with enormous variability in pleural pressure changes. These changes could not be predicted by clinical or radiological data [6]. Hence, the use of pleural pressure measurement during the procedure is the only way to follow pleural pressure changes. Thus, with regard to safety issues, the use of pleural manometry may be in some way similar and complementary to bedside ultrasonography which is known to reduce the number of complications associated with thoracentesis [7,8]. Application of pleural manometry may not only allow an increase the safety of large-volume thoracentesis, reducing the risk of pressure-related complications, but may also add some useful clinical information that impact the diagnosis and/or management of patients with pleural effusion and pneumothorax. For example, a diagnosis of transudative pleural effusion associated with trapped lung is important, as these patients usually do not require specific therapy. It should be emphasized that pleural manometry is the only method to calculate pleural elastance and evaluate lung expandability. These data are crucial in predicting the risk of unsuccessful pleurodesis [2,6,9–14]. More data on the usefulness of pleural manometry in clinical practice were presented in a comprehensive review published recently [15].

2. Physiology and pathophysiology of the pleural pressure

The pleura is inactive in terms of pressure generation. Therefore, the pressure inside the pleural cavity is a result of external forces affecting the parietal and visceral pleura. The parietal pleura appoints the external borders of the pleural space and is tightly attached to the diaphragm and to the internal surface of the thoracic walls. Due to these direct anatomical relationships, the respiratory movements of the parietal pleura follow the movements of diaphragm and the thoracic walls. These respiratory shifts are generated by the respiratory muscle function. The contraction of the diaphragm and the external intercostal muscle enlarges the volume of thoracic cavity downward and anterolaterally, respectively. This leads to an expansion of the external border of the pleural space and a decrease in the pleural pressure. The decline in pleural pressure helps to transfer inspiratory muscle power to the lungs increasing their volume, decreasing the intraalveolar pressure and ultimately resulting in air inflow to pulmonary alveoli [3,16]. The work performed during inspiration is spent on distending the lung's resilient elements and overcoming airflow resistance. During the inspiratory phase of quiet breathing, the intrapleural pressure fluctuates between -6 and -10 cmH_2O (Fig. 1A) [17,18]. However, under some circumstances (e.g. forced inspiration against increased airway resistance) it might be as low as -100 cm H_2O [19]. During normal expiration, the respiratory muscles relax and the major forces responsible for pleural pressure changes is that of elastic recoil of the chest wall and the lung. These forces result in a slight increase in pleural pressure compared to inspiratory phase. However, the pleural pressure remains negative also during expiratory phase reaching the value of -3 to -5 cmH_2O at FRC (Fig. 1B). This is due to opposite elastic recoil properties of the lung and the chest wall. As unstressed volume of the chest is relatively high, it shows a tendency to recoil outward. This is in contrast to the lungs which, under normal volumes, display strong inward recoiling forces. Hence, the opposing recoils of the lungs and chest wall create a subatmospheric (negative) intrapleural pressure during expiratory phase and at the resting end-tidal position (FRC). The negative pleural pressure is critical for maintaining the lungs in a properly inflated state. If the negative pleural pressure is eliminated, the lungs will collapse under their own elastic recoil. This may happen in patients with pneumothorax or large volume pleural effusion. The negative pleural pressure is also important for proper blood circulation within the thorax. As both heart and lungs are distensible organs enclosed in the thoracic cavity, the volume of these organs depends on the pressure gradient across their walls. Cyclic pressure changes inside the thoracic cage affect cardiac pre- and afterload. Decreased inspiratory pressure in the thorax leads to an increase in right ventricle filling [20–22] but excessively negative pressure in the thoracic cavity yields in large vein collapse [21,22]. Transthoracic echocardiography showed that increased pleural pressure may result in ventricular collapse and decreased cardiac output [23,24].

It should be realized that the pleural pressure is not uniform throughout the entire pleural cavity. A vertical gradient in pleural pressure which is probably mainly related to gravity is present. Thus, the upper part of the pleural space is characterized by the lower pressure, while higher pressure prevails in the basal, peridiaphragmatic regions. This gradient results in the alveoli in the upper parts of the lung being larger than those in the lower parts. It may also be responsible for the formation of subpleural blebs in the apex of the lungs [25–27].

There are significant differences in pleural pressure distribution in pneumothorax and pleural effusion. In pneumothorax, the pressure is the same throughout the pleural space, while in pleural effusion there is a vertical gradient of 1 cm H_2O/cm in the pleural pressure associated with the hydrostatic pressure of the fluid column (Fig. 2). Thus, in patients with pneumothorax the upper lobes are relatively more affected, whereas in patients with pleural effusion the lower lobes are put in a more disadvantageous position [3]. In both clinical entities the function of the respiratory and cardiovascular system can be compromised to a varying degree. The extreme examples are tension pneumothorax or tension hydrothorax that may lead to acute respiratory failure and significant reduction in cardiac output (probably due to decreased venous return as a consequence of increased pleural pressure). This may eventually result in profound deterioration in blood oxygenation, hypotension, shock and even death [3]. Hence, adequate pleural interventions including pneumothorax aspiration, thoracentesis and chest tube drainage are critical to restore normal or near normal anatomical conditions and normal pleural pressure.

Measurement of pleural pressure may show two different values depending on the measurement method applied. This observation became a basis for the concept of two different pleural pressures. If the pressure is measured with application of fluid-filled catheter, the vertical gradient is approximately 1 cmH_2O/cm vertical height [3]. This is called pleural liquid pressure. This pressure probably plays a role in the absorption of pleural fluid. However, when the pressure is measured with the use of surface balloon or suction cup, the vertical gradient is significantly smaller and approximates to 0.3 cmH_2O/cm vertical height. This measurement reflects the so-called pleural surface pressure [3]. It is thought that it represents the balance between pressures generated by the thoracic cavity and the lungs [3]. The difference between pleural surface pressure and pleural liquid pressure probably results from deformation forces generated by surface of parietal and visceral pleural contact [11]. However, it is still not clear whether there is only one pleural pressure or two different pressures.

Assuming there are two different pleural pressures and applying Agostini's animal model of hydrothorax, the pressure of the liquid remains in hydrostatic equilibrium maintained by vertical gradient (1 cmH_2O/cm height) and three zones in the pleural space can be distinguished. When pleural fluid accumulates in the pleural cavity, the thickness of the fluid layer in the upper zone of the pleural space is normal as the pleural liquid pressure is lower than the pleural surface pressure (because over it the lung still contacts the chest wall). In the middle zone, where the pleural fluid thickness is increased, the pleural liquid pressure equalizes with the pleural surface pressure. And finally, the lower zone of the pleural cavity is characterized by a liquid pressure higher than pleural surface pressure (that pushes the lung and chest

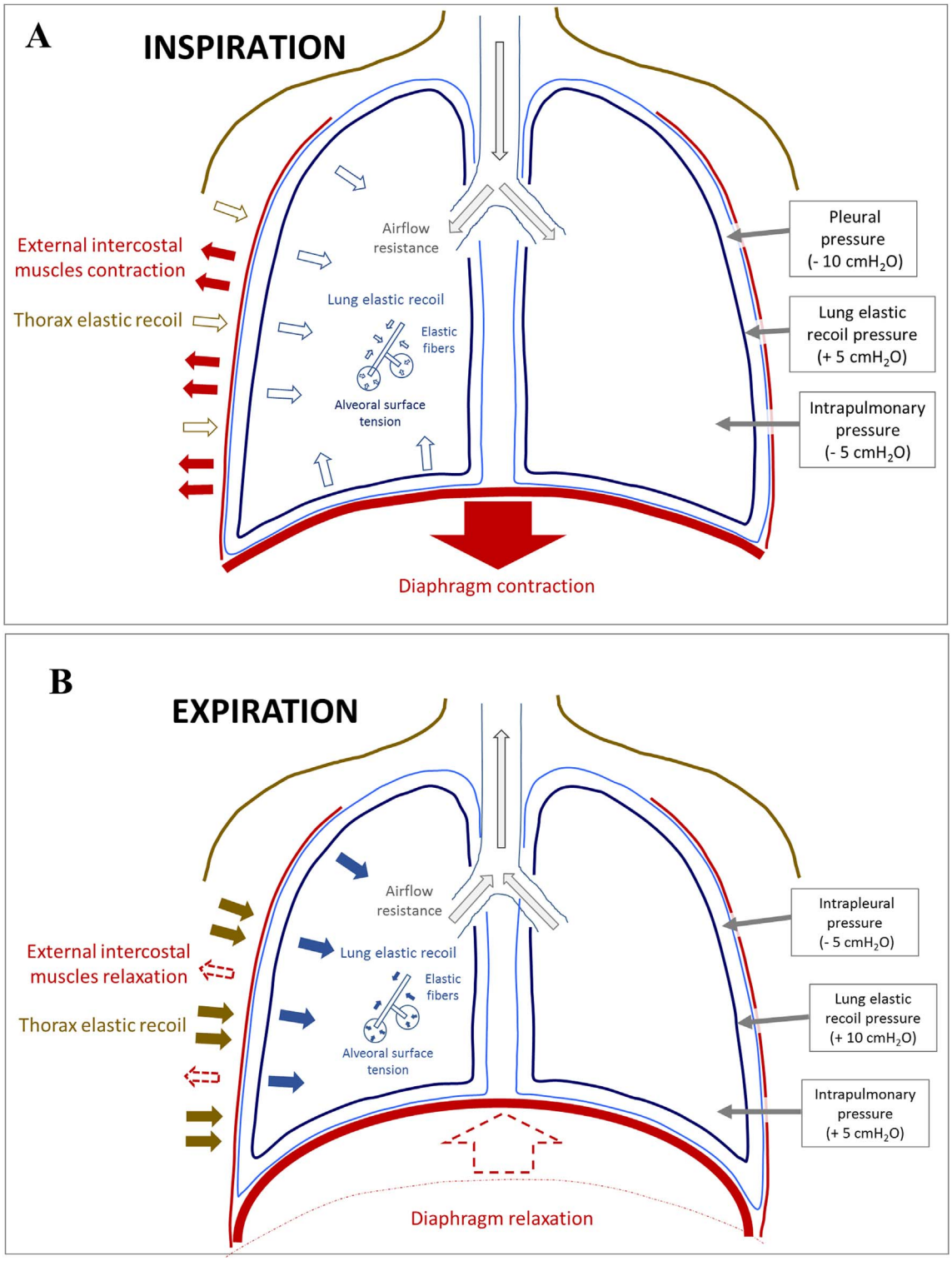

Fig. 1. Schematic representation of pleural pressure (Ppl) changes during respiratory phases (the right sides of the figure) and the major mechanisms responsible for Ppl swings (the left side of the figure). **A.** Inspiration – a contraction of the inspiratory muscles (mainly the intercostals and the diaphragm) overcomes the elastic recoils of the lung and the chest wall and expands the volume of the thorax which create a more negative pleural pressure. As the intrapulmonary pressure is the sum of pleural pressure and lung elastic recoil pressure, a relatively low pleural pressure is necessary to create a negative intrapulmonary pressure that drives inspiratory airflow. **B.** Expiration – due to the relaxation of the inspiratory muscles and the increased recoil pressure of the lung and the chest wall (due to the extension of the elastic fibers during inspiration) the balance of forces shifts in favor of elastic recoils. The red lines represent the inspiratory muscles (intercostals – thin red lines and the diaphragm - thick red line; a light and dark blue line represents parietal pleura and the lung covered by visceral pleura, respectively; the solid arrows show the vectors of the predominant forces while the outlined arrows show the overcome forces during inspiration and expiration. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

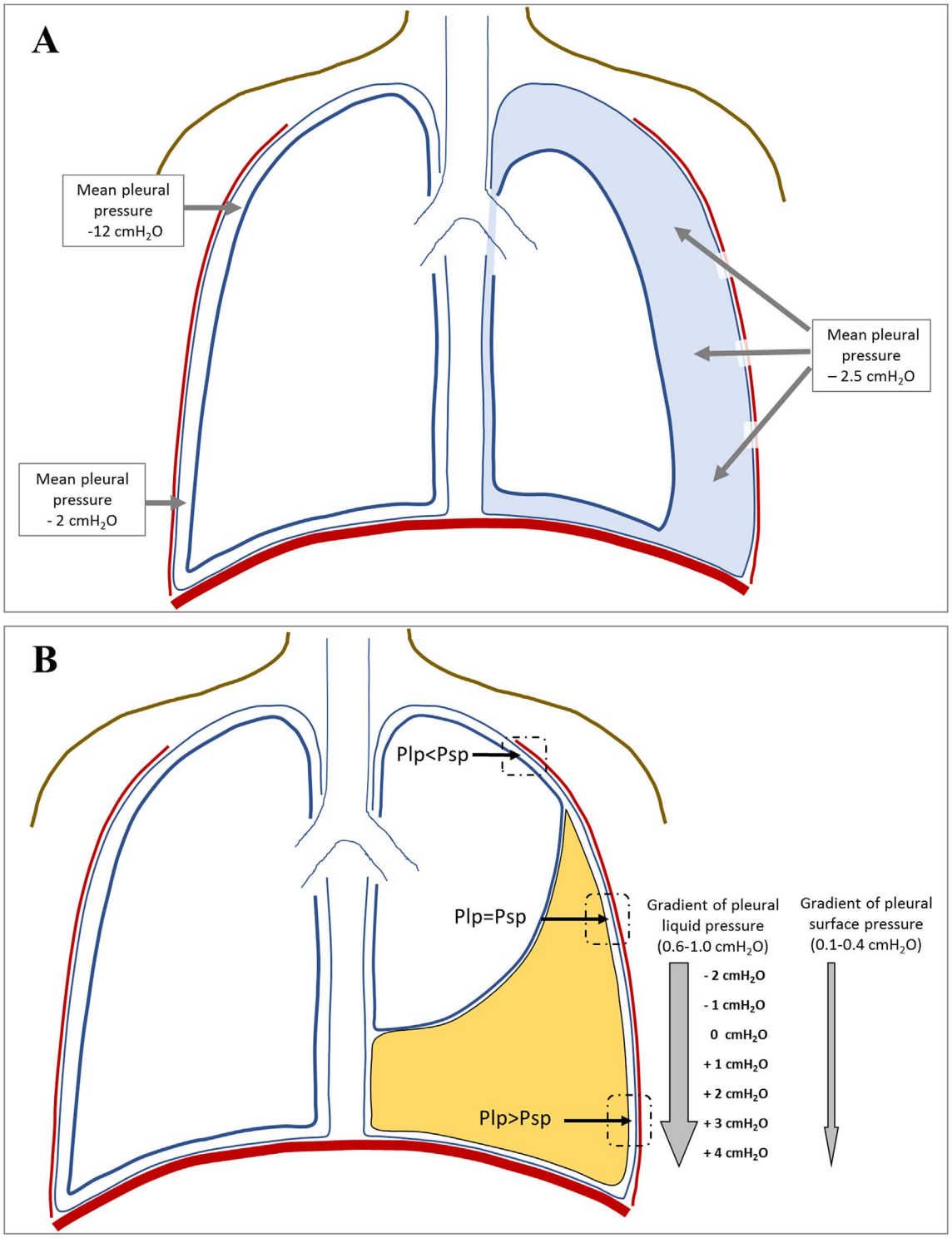

Fig. 2. Schematic presentation of the differences between pleural pressure in pneumothorax **(A)** and pleural effusion **(B)**. Pale blue and yellow filled areas represents pneumothorax and pleural effusion, respectively. Ppl – pleural liquid pressure, Psp – pleural surface pressure. See text for details. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

wall apart) (Fig. 2B) [1,6].

The value of pleural pressure measurement depends on the level at which the system is opened to atmosphere and calibrated as zero point. By establishing this point near the upper level of pleural fluid, more precise measurements of pleural pressure can be performed. If the pleural catheter is inserted in the more dependent part of the pleural space, the initial influence of the hydrostatic fluid column will be more pronounced. When the height of the pleural fluid column above the catheter decreases during thoracentesis, the influence of the hydrostatic pressure gradient on pleural pressure measurement becomes significantly reduced. Therefore, some authors recommend inserting the pleural catheter/needle at the level of moderate amount of fluid and push it to the most dependent part of the effusion. This approach enables maximizing the amount of fluid that can be withdrawn and, at the same time, to minimize deformation forces between catheter and lung [1]. It should, however, be stressed that if the hypothesis on the

equality of pleural liquid pressure and pleural surface pressure is true (and only one pleural pressure exists), the pressure measured at the level of the catheter accurately indicates the hydrostatic pressure in the fluid [3].

3. Development of pleural manometry–historical perspective

Pleural manometry has gained much interest over the last four decades. However, the history of the pleural pressure measurements is much longer, and the studies performed in the last 40 years reflect rediscovery of pleural manometry rather, than pioneering works. Nonetheless, the new studies that used more technically advanced tools and measurement methods provide new interesting data that have widened the knowledge on pleural pathophysiology.

German internist Heinrich Irenaeus Quincke (1842–1922) is credited as performing the first pleural pressure measurement during pleural fluid withdrawal in 1878 [11,28]. His name is better known from the first description of angioedema (1882) which is commonly referred to as “Quincke's edema”. An introduction of needle lumbar puncture technique (1891) was yet another important Quincke's contribution to world medicine.

In the first decades of the twentieth century, pleural manometry became an important component of collapse therapy in pulmonary tuberculosis (TB). It was commonly used during creation and management of artificial pneumothorax that was a primary therapeutic procedure applied in the pre-antibiotic era [17]. Manometry played a dual role in the creation of artificial pneumothorax. First, the measurement of pleural pressure helped to assess the position of the needle which had to be introduced to the pleural cavity. Negative pressure with cyclic fluctuations concordant with respiratory movements confirmed the proper placement of the needle tip. Then nitrogen introduction into the pleural cavity could be started. Second, the pleural pressure was carefully monitored during gas infusion. The procedure was usually terminated when the pleural pressure of 3 cmH_2O was reached [29,30]. Pleural pressure measurement was also applied during subsequent inductions of pneumothorax which were necessary to maintain lobar collapse. An interesting observation directly associated with collapse therapy was the development of unexpandable lung in some patients treated with repeated artificial pneumothoraces. Approximately 5% of patients demonstrated features of trapped lung with failure to expand between the therapeutic refills [30]. These patients were characterized by lower pleural pressure than usually found during the refilling procedure. Trapped lung and the persistence of pleural space were the prerequisites for development of *ex vacuo* pleural effusion [30,31]. Thus, pleural manometry played a key role in the early concept and the diagnosis of trapped lung.

After the introduction of effective anti-tuberculous agents, collapse therapy was abandoned, so was pleural manometry for the most part. The measurement of pleural pressure was still applied to create an artificial pneumothorax only in some centers performing medical thoracoscopy. Its role remained the same as during collapse therapy – to safely create an artificial pneumothorax before the thoracoscopy procedure [32,33].

There was a renewed interest in pleural pressure measurement in the last two decades of the twentieth century and was associated with impressive results of the study by Light and colleagues published in 1980. The authors followed pleural pressure changes during therapeutic thoracentesis and demonstrated three different patterns of pleural elastance. These patterns which corresponded to normal pleural physiology, lung entrapment and trapped lung [6,12,28] are briefly characterized in Table 1. Since then, pleural manometry has been used for studying various pathophysiological aspects of thoracentesis and pleural fluid removal including the safety of high volume thoracentesis, immediate detection of an unexpandable lung, as well as differentiation between trapped lung and lung entrapment [9,34–36]. There have been two leading groups which are particularly active in research on pleural manometry, namely those in Baltimore, MD, USA, and Charleston, SC, USA. Other centers which have a significant contribution in this field include Rochester MN, USA, Madrid, Spain, Herston Brisbane, Australia, Amsterdam, the Netherlands, Taipei, Taiwan and Warsaw, Poland.

4. Methods of pleural pressure measurement

There are two main methods of pleural pressure measurement: simple water manometers and electronic systems. Both methods have their advantages and disadvantages. The water manometers are conceptually simple, cheap and user-friendly but their disadvantage is inability to measure and register a true instantaneous pleural pressure. This is associated with continuous oscillations of water column during respiration as well as relatively high inertia and flow resistance of the system when water is used as an indicator [37,38]. Thus, simple water manometers allow only the estimation of the mean values of pleural pressure. Additionally, the measurement can be significantly falsified in case of sudden pressure changes, for example those occurring during cough [37].

The electronic systems for pleural pressure measurement are based on pressure transducers that generate an electric signal dependent on the pressure exerted on a sensor/membrane. The major components of the electronic system are pressure transducer and physiologic measurement system consisting of a signal conditioner and a display or data acquisition system [37,39]. The main advantages of these systems are precise measurements of instantaneous pleural pressure as well as the ability to display and to store a large amount of data which can be analyzed during the procedure and also after its completion.

Simple U-tube water manometer was used in the first contemporary study by Light et al. This was connected via a plastic tube and a three-way stopcock to Abrams needle joined (via another three-way stopcock) with a 60-ml syringe and a big bottle used to pull out and collect pleural fluid, respectively [6]. As discussed above, only mean pleural pressure could be measured and the results of measurements had to be manually recorded. Also, it was important not to let fluid enter the plastic catheter between pleural fluid removal circuit and the manometer. Although the measurements were certainly demanding, the authors elegantly showed that the change in pleural pressure during pleural fluid withdrawal differs significantly from patient to patient. These variability reflected different patterns of pleural elastance [6].

The use of similar equipment was later reported by various groups, including Lan et al. Villena et al. and recently by Galal et al. [9,34,40]. Lan et al. adopted virtually the same technique as described by Light et al. with the exception of using a 16-gauge pleural catheter instead of an Abrams needle and a commercial set of plastic tubes for central venous pressure measurement using the U-tube manometer. The zero point on the scale of central venous pressure monitor was set at the level of the puncture site. The authors measured mean of the pleural fluid pressures observed during inspiration and expiration [9]. In the study by Galal et al. pleural pressure was measured by a simple water column manometer that consisted of two lengths of intravenous tubing connected through a three-way stopcock to a 16-gauge catheter inserted to the pleural space. The system of IV tubing was purged of air with normal saline. The authors stated that the end-expiratory pleural pressure values were recorded during therapeutic thoracentesis [40]. However, due to water column oscillations and internal resistance of this set, the reliability of instantaneous end-respiratory pressure seems to be questionable. Villena et al. made some other minor modifications compared to the original set of Light and coworkers. These included the use of thoracentesis kit needle (Mill-Rose), a pleural biopsy needle (Cope or Castelain), or an angiocatheter instead of Abrams biopsy needle. The measurement of pleural pressure was performed with a water column manometer designed for monitoring central venous pressure with its scale reshaped to a range between −25 and +10 cmH_2O [34]. The set allowed them to follow the mean of the inspiratory

Table 1
Brief characteristics of three major patterns of pleural pressure changes during therapeutic thoracentesis [6,12,14,15,28,30].

Pattern of lung expandability	Characteristics of pleural pressure changes and their causes	Clinical significance
Normal lung	• usually positive initial pleural pressure, • small decrease in pressure during pleural fluid withdrawal, • monophasic, slightly downward slope of pressure/volume curve, • typical underlying diseases: congestive heart failure, hepatic hydrothorax.	• usually well-tolerated and safe procedure even when a large volume of pleural fluid is withdrawn, • low risk of serious complications (e.g. pneumothorax), • possible full lung reexpansion and successful pleurodesis
Lung entrapment	• usually positive initial pleural pressure, • biphasic pressure/volume curve: initially slightly sloping downwards, then abruptly turning down and becoming much steeper, • this elastance curve is related to active inflammatory or malignant processes (intrapulmonary or pleural), • typical underlying diseases: lymphangitic carcinomatosis, pleural malignancies, complicated parapneumonic effusion, rheumatoid pleurisy.	• initially well-tolerated procedure but tolerance may worsen with increasing pleural elastance, • different pleurodesis success rates depending on pleural elastance. • may lead to development of trapped lung
Trapped lung	• negative initial pleural pressure • monophasic steep slope of the pressure volume/curve. • this elastance curve is a late sequela of pleural inflammation, formation of an irreversible fibrous peel over the visceral pleura • typical underlying diseases: post coronary artery bypass grafting, uremic pleurisy, pleural empyema.	• often associated with small-volume asymptomatic pleural effusions, • transudative effusion, • rapid recurrence of fluid after thoracentesis • increased risk of pneumothorax after thoracentesis • unsuccessful pleurodesis due to unexpandable lung

and expiratory pressure after removal of a predetermined pleural fluid volume [34].

The above studies show that albeit pleural pressure measurement with a simple U-tube water manometer has some obvious limitations, it may be successfully applied to follow pleural elastance during pleural fluid withdrawal.

A modified version of water manometer was proposed in 2004 by Doelken et al. In contrast to previously used underdamped water manometers, the authors referred to their modified device as overdamped manometer. The idea of overdamped manometer was to significantly damp water column oscillations to allow a direct measurement of mean pleural pressures. To accomplish this a resistor was interposed to the system. The resistor was a simple 22-gauge needle inserted into an injection terminal and imposed between two lengths of IV tubing that formed the U-tube [37]. The manometer usually stabilized in 30 s and the water column oscillated with an amplitude of 2–4 mmH_2O around the mean value of pleural pressure. Thus, true pressure oscillations were not measured by such a system but an overdamped water manometer was found to be efficient enough to directly measure the mean pleural pressure. In the above study, Doelken et al. compared the results of pleural pressure measurements with the overdamped water manometer and an electronic manometer. Both devices were routinely used in the authors' institution. They reported that a properly calibrated electronic system was a reference standard but this system was not readily available to most clinicians [37]. At the Medical University of South Carolina, the authors used a typical hemodynamic transducer with carrier demodulator and a personal computer data acquisition system. The pressure signal was sampled at a rate of 50 times per second. This system was sufficient to precisely measure and display pressure oscillations and instantaneous pleural pressure. A water manometer was, however, necessary to calibrate the electronic system. The reference zero point was arbitrarily defined at the level of pleural catheter insertion into the chest cavity. The authors found a very strong correlation between mean pleural pressure obtained by the overdamped water manometer and that measured by the electronic transducer ($r = 0.97$).

Several relevant papers on pleural space physiology published by Feller-Kopman and colleagues from John Hopkins University Medical School, Baltimore, MD, USA also pointed out the use of both water manometer and electronic transducer system [2,10,35]. In one of these papers, the authors discussed the advantages and disadvantages of these two techniques concluding that at the time of publication it was impossible to recommend one method of pleural pressure measurement over the other. Although they underlined several convincing advantages of electronic transducer systems, they also listed some problems which may be encountered during the measurement of pleural pressure with commercially available vascular transducers. These include complexity and the necessity of having an additional equipment and special calibration. If the transducer is to be used with a standard ICU monitor, the system requires adequate re-adjustment. This is because standard ICU monitors are calibrated to measure only positive pressure. Therefore, an adjustment of the actual height relative to the zero reference level or calibration of the offset is necessary. Furthermore, a conversion factor should be applied to display the results in cmH_2O instead of mmHg [2]. Bearing in mind all these requirements, Feller-Kopman et al. reasonably suggested to use a system that can be adopted, easily available and replicated on each patient at one's local institution [2].

The above presented problems with proper data display and storage and the lack of commercially available pleural manometers had encouraged other groups to develop their own electronic systems dedicated for pleural pressure measurements. In 2011 Krenke et al. described a process of construction and validation of an electronic pleural manometer. The authors used only widely accessible elements, i.e. vascular pressure transducer (which changes pressure into an electric signal), signal conditioner (which detects, filters and amplifies the electric signal) and personal computer and demonstrated that the set can be built at a reasonable cost of approximately 2000 € [39]. They also demonstrated that the new device reliably measured positive as well as negative instantaneous pressure in the laboratory and clinical setting compared to water manometer. In Bland-Altman analysis a very high precision of the measurements with narrow 95% confidence interval for agreement ranging from -0.3 to $+0.2$ cmH_2O was found. The authors reported a very high correlation coefficient between measurements with electronic manometer and water manometer ($r = 0.999$; $p < 0.001$) [39]. The electronic manometer had very low inertia and high frequency of measurements (50 Hz) allowing reliable measurements of pleural pressure even during cough.

A modified and improved method of electronic pleural pressure was presented in 2014 by Salamonsen et al. The authors used pressure transducer which was connected to a standard urodynamics machine that computed the data and displayed them on the monitor. However, to enable continuous measurement of pleural pressure, i.e. without the need to temporarily cease the flow through the catheter, they separated the pressure measuring circuit from the chest tube used to remove pleural fluid. This was done by the insertion of a small-bore epidural catheter through a self-sealing intravenous bung into the chest tube [41]. Consequently, the set consisting of a small-bore catheter, a pressure transducer and a urodynamics computer was used for continuous measurements of pleural pressure during fluid drainage. Interestingly,

the volume of drained pleural effusion was also continuously measured by a weighted cell, which transduced the change in weight (a surrogate measure of drain volume) into an electric signal. Information was further wired to the urodynamics machine. Having the above data, pleural elastance could have been calculated and displayed in real time. Thus, the technique allows not only for automated, continuous recording of pleural pressures, but also for continuous monitoring of pleural elastance [41]. The need for the use of two different transducers and two separate catheters might be considered as a disadvantage but the technique may become more straightforward if double lumen chest tubes and thoracentesis kits become commercially available.

In recent years, the first digital, handheld electronic pleural manometer became commercially available in United States. The device is compact and can be easily attached in-line to a pleural needle or catheter and used during routine thoracentesis. The reliability of measurements with this digital manometer was recently compared by Lee et al. to the U-tube manometer and the electronic transducer system. The authors found a strong linear correlation between elastance calculated for the digital manometer and the electronic transducer ($r = 0.9582$, $p < 0.001$). Similarly, analysis with linear regression on the association between digital manometer and electronic transducer measurements in patients who developed chest discomfort during pleural fluid removal showed highly significant correlation ($R^2 = 0.824$, $P < 0.001$) [38]. In contrast, the measurements made with the U-tube manometer correlated neither with the digital manometer nor the electronic transducer measurements. The authors concluded that the handheld digital manometer provided a valid and easy-to-use method to measure pleural pressure during thoracentesis [38]. The device is mainly available in the United States and the cost of one single use pressure transducer is approximately $70.

4.1. The procedure of pleural pressure measurement with an electronic system

The procedure of pleural manometry largely depends on the method of pleural pressure measurement and the type of thoracentesis kit or pleural catheter that are used to drain pleural fluid. Below we present a brief description of the procedure which is based on our own experience and the literature data. After discussing the procedure, written informed consent should be obtained from the patient. Then, the patient is comfortably positioned in the upright sitting position as for the therapeutic thoracentesis (Fig. 3A). Ultrasound is used to choose and mark a safe point of entry for a blunt pleural needle or pleural catheter. The skin is disinfected and the whole procedure is performed under sterile conditions. After local anaesthesia, the pleural needle/catheter is inserted into the pleural cavity and a few milliliters of pleural fluid is aspirated to confirm its proper position. A vascular pressure transducer is placed in the base which is connected with a signal conditioner and personal computer (Fig. 4 B and 3B). A length of intravenous tubing is attached to the transducer and carefully purged of air with sterile saline. The second end of the tubing is positioned at the level of the puncture site and opened for atmospheric air. The reference zero point is set simply by pressing “0 level” key on the PC keyboard. The proper position of “0 level” is confirmed on LCD display. Then, the tubing is attached to a side port of the three-way stopcock, while the outlet port of the stopcock is connected to a pleural catheter. The second three-way stopcock is attached via an inlet port with the 50–60 ml syringe and pleural fluid bag attached to its port (Fig. 3A). Repositioning of the stopcock handles allows efficient pleural fluid aspiration, ejection to the pleural fluid bag and pleural pressure measurement. The first measurement is performed after connection of the pleural catheter with pressure transducer. The next measurements are repeated after removal of every 200 ml of pleural fluid, up to 1000 ml. When the volume of pleural fluid withdrawn exceeds 1000 ml, the measurements are performed with every 100 ml of pleural fluid withdrawn. The procedure is usually terminated when there is no more fluid or pleural pressure drops below -20 cmH_2O or the patient develops cough or chest discomfort that is difficult to control. Pleural manometry prolongs the procedure of therapeutic thoracentesis only for approximately 5–15 min, depending on the amount of pleural fluid removed, frequency of measurements and the duration of single measurement [5,39]. It should be emphasized, however, that continuous pleural pressure measurement as described by Salamonsen et al. was not associated with increased duration of the procedure [41].

5. Summary

Proper understanding of pleural pressure changes and their consequences is of particular importance in patients with pleural diseases. The role of pleural manometry is not limited to its historical significance or providing data for basic physiological science. Contrary, it has a direct impact on efficacy and safety of pleural procedures. Both

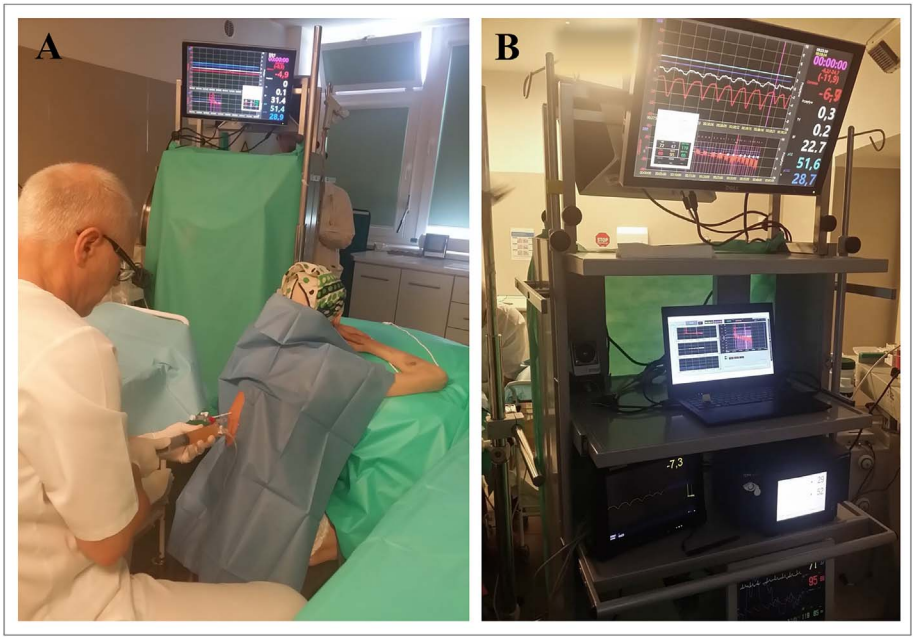

Fig. 3. The procedure of therapeutic thoracentesis combined with pleural manometry. **A.** A position of the patient and the operator during the procedure. **B.** Endoscopy trolley with an electronic signal conditioner, personal computer and two external monitors.

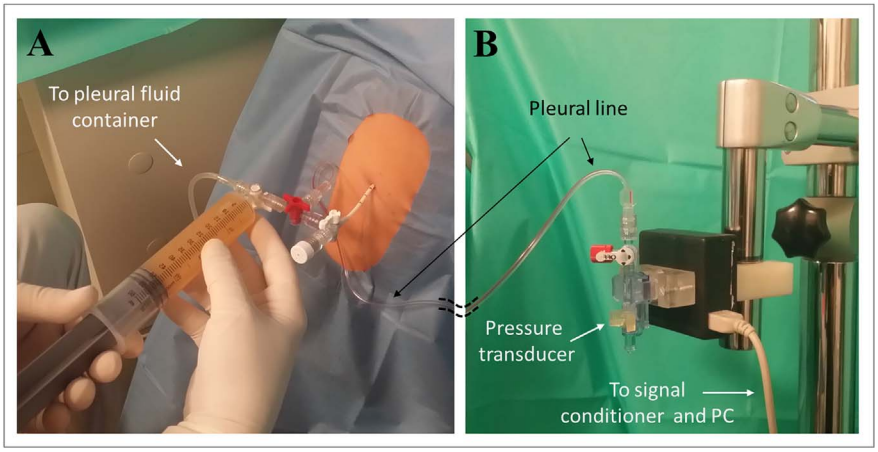

Fig. 4. The main single-use elements used for pleural pressure monitoring during a therapeutic thoracentesis. **A.** Fluid withdrawal kit containing a small bore pleural catheter, a red-handle three-way-stopcock connected with vascular pressure transducer; and a white-handle tree-way-stopcock (in upside down position) connected with a syringe and a pleural fluid container. **B.** Vascular pressure transducer connected to a pleural catheter (pleural line) and the signal conditioner (seen in Fig. 3B). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

dissemination of knowledge on pleural manometry and wider access to homemade or commercial pleural manometers will allow the optimization of the medical approach to patients with pleural diseases. The clinical applications of pleural manometry will be presented in detail in a second review that will be published in one of the coming issues of this journal. Future will show whether all major applications have already been explored or if there are still new fields for the use of pleural manometry.

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Conflicts of interest

None declared.

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