

PLEURAL PATHOPHYSIOLOGY

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SUMMARY – According to recent concepts, pleura is defined as an organ with its own biological and functional properties. Pleura is located between two anatomically and functionally different parts of the integral thoracopulmonary system. One is the thorax, which has an active and driving role in the process of ventilation, and the other are the lungs, which passively follow thoracic movements. The balance of the thoracopulmonary system can be compromised by a number of primary and even more commonly secondary pathologic processes of the pleura that can manifest in the form of pleural effusions, air in the pleural cavity, pleural cavity obliteration, or a combination of these phenomena. Variation in the histologic structure of the visceral and parietal pleura, intracavitary fluid exchange, and pathogenesis of pleural effusions are presented. The presence of air in the pleural cavity indicates total disintegration of the thoracopulmonary system and substantially impedes the respiratory function of the lungs. Pleural obliteration or adhesions result in partial or complete pleural space reduction and, depending on the extension and localization of adhesions, cause restriction or restriction-obstruction ventilation disturbances, modify lung hemodynamics, and with time lead to pulmonary hypertension and chronic pulmonary heart. The development of permanent anatomical and functional lesions of the thoracopulmonary system can be timely prevented by an early diagnosis and appropriate treatment of pleural changes.

Key words: *Pleura, pathophysiology; Pleural diseases; Pleural effusion; Pneumothorax*

Introduction

The following systems are involved in the process of external breathing, i.e. exchange of gases on the alveo-capillary membrane: 1) nervous (central, peripheral, and vegetative); 2) musculoskeletal (ribs, diaphragm, intercostal musculature); 3) respiratory (upper and lower airways); 4) cardiovascular; and 5) hematopoietic (red blood cells). Obviously, pleura as one of the important structures in the thoracopulmonary system function is not mentioned at all. We have been taught that pleura is a thin serous membrane lining the lungs and inner wall of the thoracic cavity, thus creating a space that is not visualized in physi-

ologic conditions¹. However, many authors disagree with such a definition of the pleura, stating that pleura is not an accompanying reactive pulmonary tissue but an organ with its own biological and functional properties². Whatever definition be accepted, we have to admit that pleura is situated like a 'buffer' in-between two anatomically and functionally different parts of the integral thoracopulmonary system. One of these two is the thorax, which has an active and driving role in the process of ventilation, and the other are the lungs that passively increase in volume while following the anterior-posterior and cranio-caudal expansion of the thorax³. With its negative pressure and mechanism of maintaining minimal fluid volume and low protein concentration within the intrapleural cavity, pleura compensates for the shear forces of the thorax and the lungs. This balance of the thoracopulmonary system can be deranged by various primary and even more commonly secondary pathologic processes of the pleura, which can manifest as: 1) pleural effusion; 2) air in the pleural space;

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Received January 27, 2002, accepted February 21, 2002

3) obliteration of pleural cavity (adhesions); or 4) a combination of these phenomena.

Histologic Structure of the Pleura

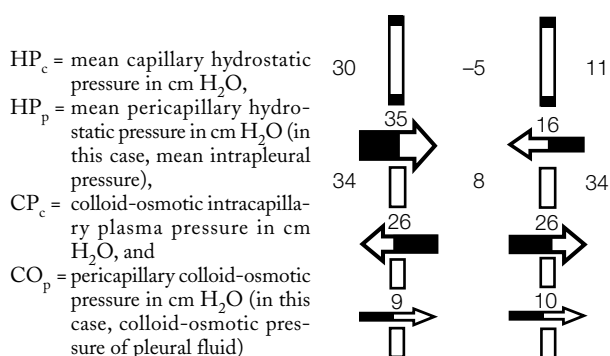
The parietal and visceral pleurae consist of two layers. The first layer consists of squamous epithelial cells, so-called mesothelium with basal membrane and a thin layer of connective tissue, and overlies the other, main layer consisting of a network of elastic and collagenous fiber⁴. As there are no junctions (hemidesmosomes) between the two layers, any irritation can readily induce scaling, i.e. stratification and mesothelial lesion. In the parietal pleura, a loose connective tissue sheath, endothoracic fascia, containing a network of blood and lymph vessels and nerve fiber, is found beneath the main layer. Parietal pleura is supplied *via* branches of the systemic circulation originating from the thoracic aorta (intercostal, pericardiophrenic, phrenic and musculophrenic arteries). Venous blood is drained to the azygos, hemiazygos and mammary veins. Lymph drainage of the parietal pleura runs *via* initial lymph capillaries and lymph orifices (stoma) located between mesothelial cells, mostly in the region of costal, mediastinal and diaphragmatic pleura. Visceral pleura has no endothoracic fascia but the pleural elastic fiber continue to the elastic fiber of pulmonary interstitium, where blood and lymph capillaries are located. Visceral pleura is mostly irrigated by branches of the pulmonary artery, and only partly (mediastinal, interlobar and in part diaphragmatic surface) by branches of bronchial arteries. Venous blood is mostly drained to pulmonary veins. As differentiated from parietal pleura, visceral pleura has no lymph orifices. Initial lymph capillaries are subpleurally located, draining the fluid that is resorbed *via* visceral pleura. Microvilli are found on the free surface of mesothelial cells. Microvilli are minute, cilium-like processes facilitating sliding and increasing the absorptive area. They are more abundant on visceral than on parietal pleura^{5,6}.

Intrapleural Fluid Flow

About 20 ml of low-protein (~1 g/dl) fluid are found in the pleural space. The fluid is distributed in a thin layer over the pleural filaments allowing for their smooth sliding motion. The fluid flow in the pleural space is well understood and regulated by Starling's law (Fig. 1a). The fluid is filtered on the parietal pleura under the pressure

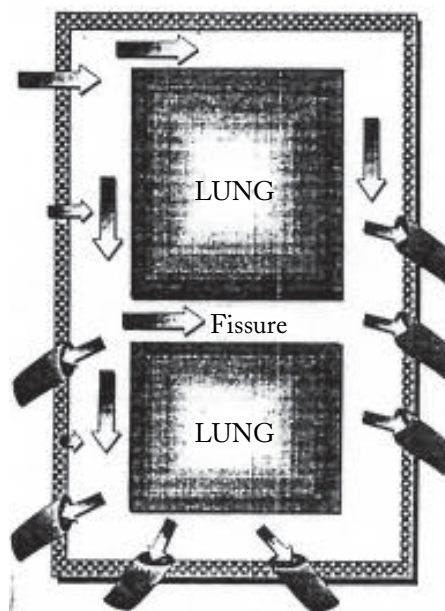
of +9 cm H₂O, and reabsorbed on visceral pleura under the pressure of -10 cm H₂O. The rate of filtration on the parietal pleura and reabsorption on the visceral pleura depends on the hydrostatic and colloid-osmotic pressure in the capillaries of parietal and visceral pleura, and on the intrapleural pressure. The flow of fluid through the pleural cavity can be presented by the following equation:

Fig. 1a. Intrapleural fluid flow⁷



$$\text{Fluid flow} = C \times (HP_c - HP_p) - (CP_c - CO_p), \text{ where } C = \text{coefficient of filtration,}$$

Fig. 1b.



In physiologic conditions, water and small particles are eliminated by this route. Major effusions and large particles, especially protein, are cleared from the pleural space

via lymph orifices (stoma) of the parietal pleura, whose capacity is 20-fold absorption capacity of the visceral pleura, and which makes about 80% of total intrapleural fluid volume (Fig. 1b). When both of these regulatory mechanisms of intrapleural fluid flow fail due to some pathologic process, the fluid accumulates in the pleural cavity, with an amount of fluid exceeding 20 ml considered a pathologic event⁷⁻⁹.

Pathogenesis of Pleural Effusions

Pleural effusion as a primary pleural pathology, or as a secondary manifestation of pathologic alterations of the adjacent organs or organ systems can develop by any of the following mechanisms: 1) hydrostatic pressure increase in the parietal and visceral pleura capillaries; 2) colloid-osmotic plasma pressure decrease; 3) increased capillary permeability; 4) lymphostasis; 5) rupture of a blood vessel, thoracic duct, or esophagus; and 6) combination of these mechanisms.

In patients with cardiac insufficiency or constrictive pericarditis, pleural effusions occur due to increase in intracapillary hydrostatic pressure, whereas in those with liver cirrhosis, hypoalbuminemia, hydronephrosis or nephrotic syndrome, pleural effusions are due to decrease in plasma colloid-osmotic pressure. In these patients, pleura is not pathologically altered. Effusions are usually bilateral, moderate in extension, and of a transudate nature. If left in place and treated with diuretics, they may produce a picture of pseudoexudate.

Inflammatory effusions mostly develop in association with specific or nonspecific inflammation of the lungs and other adjacent organs, whereas primary pleurisy is rare. Capillary permeability is increased in the pleural inflammatory area, which leads to exudation and elevated concentration of water binding protein, thus increasing the effusion volume. Tuberculous pleurisy develops due to the pleural space invasion with caseous masses and tuberculosis bacilli. According to the character of effusion, serofibrinous and caseous pleurisy are differentiated. Serofibrinous tuberculous effusion is clear, amber-colored, has the characteristics of an exudate, and shows a tendency of spontaneous regression. The presence of tuberculosis bacilli is difficult to demonstrate, as they represent pleural hyperergic reaction to the small amount of *Mycobacterium (M.) tuberculosis* in the pleural space. Caseous effusion is turbid, with a high fibrin content, and the presence of *M. tuberculosis* is easily detected. This type of effusion usually involves a large

part of the pleural and subpleural area, is associated with a more severe clinical picture, and entails extensive posttuberculous intrapleural sequelae. Nonspecific pleural effusions generally accompany lobar bacterial pneumonia, pulmonary abscesses, inflamed bronchiectasis and cysts, and are less frequently found in focal pneumonia, legionellosis, rickettsial, mycoplasmal and viral inflammation. According to the time of development, parapneumonial effusions occurring during pneumonia, and metapneumonial or postpneumonial effusions that develop secondary to the clinical picture of pneumonia are distinguished. The latter are more extensive, more difficult to treat, and pathogenic bacteria can rarely be detected.

Malignant pleural effusions develop in primary and even more commonly in secondary (metastatic) pleural tumors. These effusions are very extensive, recur soon after puncture, and can occur due to increased capillary permeability, blood vessel rupture, lymphostasis, or a combination of these mechanisms.

The effusions that occur due to increased capillary permeability and/or blood vessel rupture generally are hemorrhagic, have characteristics of an exudate, and malignant cells are easily detectable. The effusions occurring due to lymphostasis have a transudate nature, and the presence of malignant cells is more difficult to demonstrate.

In autoimmune diseases such as rheumatoid arthritis, systemic lupus erythematosus or Sjögren's syndrome, minor effusions, usually bilateral and with characteristics of an exudate, can occur. These effusions develop because of increased capillary permeability and are caused by an immune reaction on small and medium blood vessels (vasculitis).

Hemothorax and chylothorax are conditions that occur secondary to rupture of a blood vessel or thoracic duct. Esophageal rupture may occasionally be associated with effusion.

In addition to these effusions that are most frequently encountered in clinical practice, effusions may also be found in various other pathologic conditions such as uremia, myxedema, postinfarction syndrome, pulmonary infarction, Meigs' syndrome, endometriosis, yellow nail syndrome, then effusions after radiotherapy, some medication, or exposure to asbestos inhalation. In the general internist population, pleural effusions are found in some 10% of cases and may pose a major diagnostic problem. Statistical data show that cardiac effusions are most common, accounting for 30% – 40% of all effusions. Statistical data on noncardiac effusions differ between America and Europe, as illustrated in Tables 1 and 2. In Croatia, exact data on the prevalence of effusions are still lacking¹⁰⁻¹³.

Table 1. Prevalence of noncardiac pleural effusions¹²

Etiology	N	%
Malignant		42
Tuberculous		17
Pneumonia		12
Various		14
Idiopathic		15
Total	1,500	

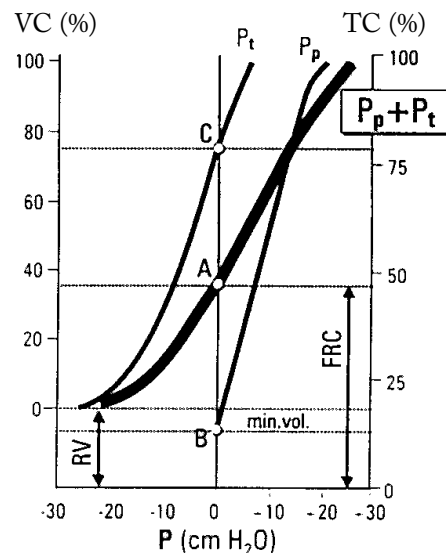
Table 2. Prevalence of pleural effusions in the USA¹⁴

Etiology	n	% of total	% of noncardiac
Cardiac	500,000	37.5	
Pneumonia	400,000	30.0	48.0
Neoplasms	200,000	15.0	24.0
Pulmonary embolism	150,000	11.0	18.0
Liver cirrhosis	50,000	4.0	6.0
Abdominal pathology	25,000	2.0	3.0
Autoimmune diseases	6,000	0.5	0.7
Tuberculosis	2,500	0.2	0.3
Asbestos effusion	2,000	0.15	0.25

Air in the Pleural Space (Pneumothorax)

In physiologic conditions, there is no air in the pleural cavity, and the mean intrapleural pressure at the level of functional residual capacity (FRC) is -5 cm H₂O. If the air occurs in the pleural cavity for any reason, the intrapleural pressure rises, and the function of the thoracopulmonary system fails. This is best illustrated by the curves of relaxation of the thoracopulmonary system as a whole and of each of its structures individually (Fig. 2). These curves show that the thorax and lungs differ both in their shape and elastic properties. Point A on $P_p + P_t$ curve is the site of relaxation of the entire thoracopulmonary system, representing the FRC volume, i.e. the volume at balance and rest, when the force drawing air into the lungs equals the thoracic force drawing air out of the lungs. Within the lungs, the pressure is zero, there is no air flow, and the intrapleural pressure is -5 cm H₂O. Point B on P_p curve is the site of full pulmonary relaxation. In these conditions, the lungs have lowest volume, which is even below the residual volume. Point C on P_t curve is the site of complete thorax relaxation. At this point, thorax

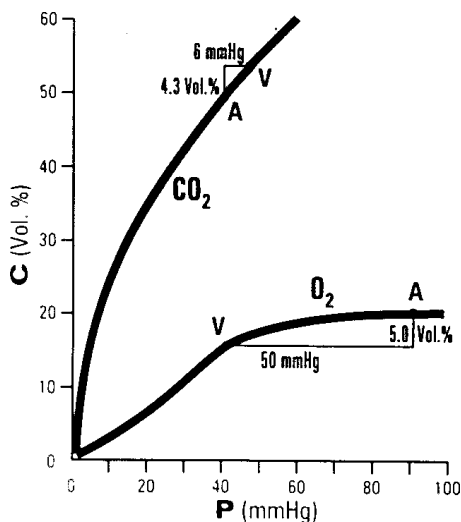
Fig. 2. Thoracopulmonary system relaxation curves



does not exert any outward force anymore and has reached complete relaxation, which is achieved at high volumes at the level of 75% of vital capacity. This impairment of the thoracopulmonary system function modifies the level of ventilation, perfusion and ventilation/perfusion ratio, and influences the hemodynamics; any of these segments in the process of external respiration can be objectively recorded in pneumothorax. Depending on the pneumothorax extension, decreased values of the vital capacity (VC), inspiration reserve volume (IRV), expiration reserve volume (ERV), residual volume (RV), functional residual capacity (FRC) and total capacity (TC) can be recorded by the method of helium irrigation. If the method of plethysmography is used, however, RV, intrathoracic gaseous volume (IGV) and TC may be elevated. In a collapsed lung, hypoventilation and shunt effect develop, thus hypoxemia may be observed in arterial blood.

Air is being slowly resorbed from the pleural space. If the intrapleural air be left to spontaneous resorption, then, according to Magyar and Miskovitsh¹⁷, 37 days would be required for resorption of the air of completely collapsed lungs, or 29 days for 8-cm, 18 days for 5-cm and 14 days for 2- to 3-cm air mantle. Air resorption from the pleural space occurs due to the gradient of intrapleural air pressure and total gas pressure in the venous segment of the capillary. In other words, total venous gas pressure is lower than total gas pressure in the alveolus, artery and pleural space. This difference occurs at the moment of arterial blood venozation, i.e. when PO_2 decrease exceeds by many millimeters of mercury (from 90 to 40 mm Hg)

Fig. 3. Air resorption from pleural space¹⁸



Air resorption from the pleural space (Gauer)

	Alv.	art.	ven	Pleural space
P _{H₂O}	47	47	47	47
P _{CO₂}	38	40	46	46.1
P _{O₂}	100	90	40	40.9
P _{N₂}	562	564	564	613
ΣP	747	741	697	747

the PCO₂ increase (from 40 to 46 mm Hg). The reason for such a pattern of respiratory gases lies in the different shape of dissociation curves for oxygen and carbon dioxide (Fig. 3).

Air resorption from the pleural space is limited by nitrogen, because it acts as an inert gas, i.e. is characterized by poor water solubility and poor chemical binding. In contrast, air resorption from the pleural space is accelerated by the use of oxygen to increase PaO₂, which is frequently low in patients with pneumothorax¹⁴⁻¹⁹.

Intrapleural Obliteration (Adhesions)

Adhesions are permanent pleural lesions that occur secondary to specific or nonspecific pleurisy, empyema, trauma, treated pneumothorax, asbestos exposure, or in association with some systemic diseases. Histologically,

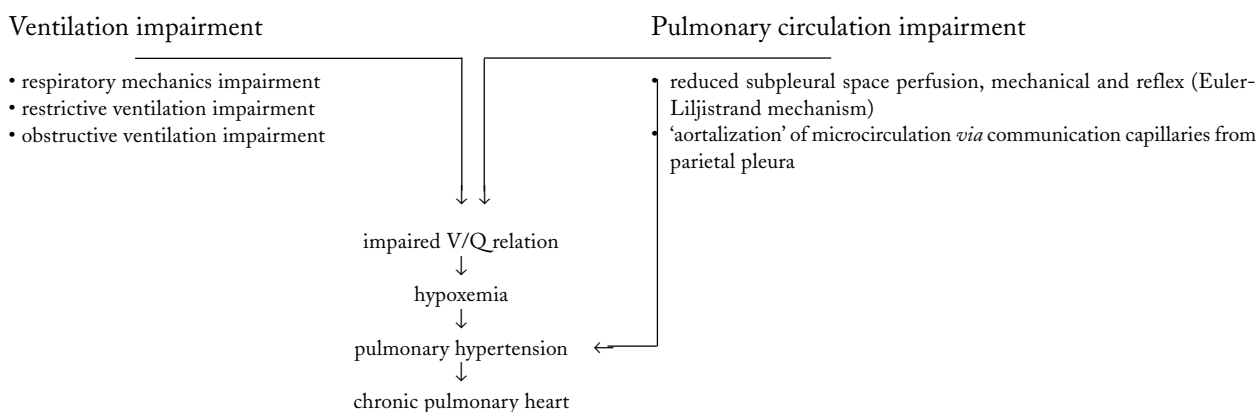
adhesions consist of connective tissue with some collagenous fiber, and are well vascularized *via* branches of systemic circulation. This should be borne in mind, as these vessels may sustain rupture and cause abundant hemorrhage in pneumothorax. Depending on the size and extension of intrapleural adhesions, they lead to anatomic modifications of the thorax and lungs, whereas in the late stage of disease the respiratory function of the lungs may be impeded. The process of parietal pleura fibroization may involve endothoracic fascia and intercostal muscle, leading to the narrowing of intercostal spaces and weakening of the chest movements. Visceral pleura fibroization involves subpleural region and leads to morphologic changes of the interstitium, peripheral vasculature and bronchi. Intrapleural pressure disappears with the formation of adhesions. The elastic forces of the lungs are directly connected to the elastic shear forces of the thorax, thus being mostly abolished, which results in small, low-volume thorax excursions.

Impairment of the respiratory function of the lungs depends on the extension and localization of adhesions. Apical adhesions do not induce any major impairment of the pulmonary function. The adhesions localized in the region of diaphragm and laterobasal parts of the thorax cause more severe events in the respiratory function of the lungs, because the main inspiration muscle, the diaphragm, has thus been rendered nonfunctional. Such unilateral total diaphragmatic dysfunction causes more severe functional impairments than partial pneumothorax, lobectomy or thoracoplasty. On spirometry, it is manifested by a decrease in IRV, then in VC and TC, and these values are read-off as a restriction impairment of pulmonary ventilation. A combination of apicomediastinal and costodiaphragmatic adhesions with cranial hilum retraction, and with restrictive disturbances also leads to obstruction impairments of ventilation. In this case, the uniform traction force relations in the lungs are changed, with a prevalence of vertical stretching of pulmonary parenchyma, which in turn leads to morphological changes of the bronchi and vasculature. Obliteration of the entire pleural space is described by the clinical and radiologic term of fibrothorax. It is a condition where pleural space does not exist anymore but has been replaced by connective tissue, and the process of fibroization has involved the entire thorax, leading to its malformation and reducing the driving force of the thorax to the minimum. On the other hand, the process of fibroization is transferred from visceral pleura to pulmonary parenchyma, where rearrangement of the pulmonary structures occurs, including an-

gulation, dilatation and deformities of the bronchi with stasis of discharge, destruction of alveolar septa, and development of paraseptal emphysema. The changes described compromise ventilation as well as pulmonary cir-

pleural mesothelioma or bronchial carcinoma may develop after a long latency period of 30-40 years²⁴⁻²⁷.

Fig. 4. Pathogenesis of the lung respiratory function impairment in fibrothorax



ulation, which leads to the development of pulmonary hypertension and chronic pulmonary heart (Fig. 4).

Although adhesions generally present a definite condition, clinically the possibility of tuberculosis reactivation, secondary infection with pleurocutaneous fistula, cholesterol pleurisy, and malignant diseases should be considered²⁰⁻²³.

Pleural Plaques

The individuals inhaling air contaminated with asbestos dust can sustain pleural and pulmonary lesions, which manifest as pleural plaques, asbestos pleurisy, costodiaphragmatic adhesions, pulmonary fibrosis (asbestosis), and malignant mesothelioma.

Pleural plaques are most common of these morphologic changes, and can serve as a marker of the level of atmosphere contamination with asbestos. Large-scale radiologic studies have shown the presence of pleural plaques in 0.02% to 3% of the general population, and in up to 30% of individuals at occupational asbestos exposure. Pleural plaques appear as small islets of smooth surface, sharply demarcated, consisting mostly of hyalin, and are usually located on parietal pleura (diaphragmatic, costal and mediastinal). Pleural plaques do not cause any major impairments in the respiratory function of the lungs. The persons found to have pleural plaques belong to a risk group who require systematic follow-up, because

Conclusion

The concepts presented lead to the following conclusions: pleura has a very important role in the function of thoracopulmonary system; pathologic processes in the lungs and other organ systems are very early recorded and signalled by the pleura; unless appropriate diagnosis and treatment of pleural processes are performed on time, permanent anatomical and functional lesions of the thoracopulmonary system can occur, mostly manifesting as impaired respiratory function of the lungs.

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Sažetak

PATOFIZIOLOGIJA PLEURALNOG PROSTORA

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Pleura se prema novijim shvaćanjima definira kao organ koji ima svoja biološka i funkcijska svojstva. Pleura je smještena između dvaju anatomski i funkcijski različitih dijelova jedinstvenog torakopulmonalnog sustava. Jedan je toraks koji ima djelatnu i pokretačku ulogu u procesu ventilacije, a drugi su pluća koja pasivno prate pokrete toraksa. Ravnotežu torakopulmonalnog sustava mogu narušiti različiti primarni, ali češće sekundarni patološki procesi pleure, koji se mogu očitovati u obliku izljeva u pleuralnom prostoru, zraka u pleuralnom prostoru, obliteracijom pleuralnog prostora ili kombinacijom spomenutih pojava. Prikazane su raznolikosti u histološkoj građi visceralne i parijetalne pleure, zatim izmjena tekućine u pleuralnom prostoru, te patogeneza pleuralnih izljeva. Zrak u pleuralnom prostoru označava potpun raspad torakopulmonalnog sustava i bitno narušava respiracijsku funkciju pluća. Obliteracije ili priraslice djelomice ili potpuno smanjuju pleuralni prostor, a ovisno o proširenosti i lokalizaciji priraslica uzrokuju restriksijske ili restriksijsko-opstruksijske smetnje ventilacije, mijenjaju plućnu hemodinamiku i s vremenom dovode do plućne hipertenzije i kroničnog plućnog srca. Ranom dijagnostikom, a potom i liječenjem pleuralnih promjena može se na vrijeme zaustaviti nastanak trajnih anatomskih i funkcijskih promjena torakopulmonalnog sustava.

Ključne riječi: Pleura, patofiziologija; Pleuralne bolesti; Pleuralni izljevi; Pneumotoraks