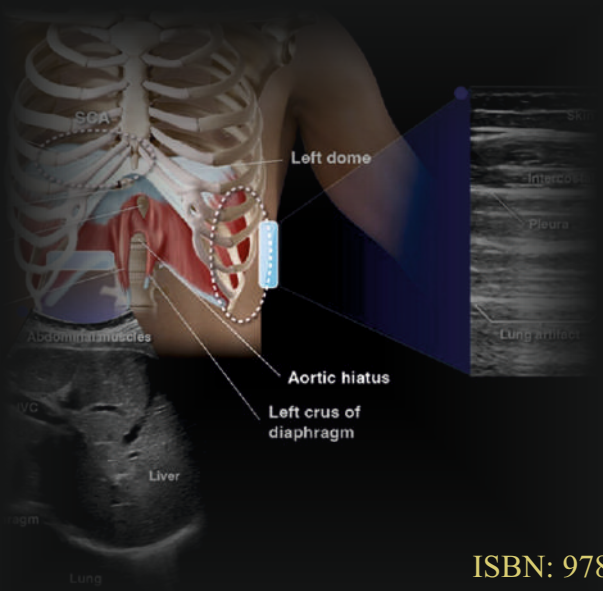
DIAPHRAGMATIC SURGERY: FROM ANATOMY TO ADVANCED PRACTICE

EDITOR
Assoc. Prof. Dr. Turkan DUBUS, MD



ISBN: 978-625-5923-70-7
Ankara -2025

Assoc. Prof. Dr. Guntug BATIHAN, MD
Asst. Prof. Dr. Mahmut OZBEY, MD
Asst. Prof. Dr. Suleyman Emre AKIN, MD
Dr. Eren ERDOGDU, MD, Thoracic Surgeon
Dr. Leyla Nesrin ACAR, MD, Thoracic Surgeon
Dr. Murat HANOZU, MD, Thoracic Surgeon
Dr. Mehmet Ali UNAL, MD, Thoracic Surgery Resident
Dr. Muhammet KERTMEN, MD, Thoracic Surgery Resident

DIAPHRAGMATIC SURGERY: FROM ANATOMY TO ADVANCED PRACTICE

EDITOR

Assoc. Prof. Dr. Turkan DUBUS
ORCID ID: 0000-0002-7222-7998

AUTHORS

Assoc. Prof. Dr. Guntug BATIHAN, MD [1]

Asst. Prof. Dr. Mahmut OZBEY, MD[2]

Asst. Prof. Dr. Suleyman Emre AKIN, MD[3]

Dr. Eren ERDOGDU, MD, Thoracic Surgeon[4]

Dr. Leyla Nesrin ACAR, MD, Thoracic Surgeon[5]

Dr. Murat HANOZU, MD, Thoracic Surgeon[6]

Dr. Mehmet Ali UNAL, MD, Thoracic Surgery Resident[7]

Dr. Muhammet KERTMEN, MD, Thoracic Surgery Resident[8]

[1]Department of Thoracic Surgery, Faculty of Medicine, Canakkale Onsekiz Mart University, Canakkale, Turkey, **gbatihan@hotmail.com**
ORCID ID: 0000-0001-5258-2856

[2]Department of Thoracic Surgery, Faculty of Medicine, Sivas Cumhuriyet University, Sivas, Turkey, **mahmutozbey@cumhuriyet.edu.tr**
ORCID ID: 0000-0001-8641-1772

[3]Department of Thoracic Surgery, Faculty of Medicine, Süleyman Demirel University, Isparta, Turkey, **suleymanemreakin@yahoo.com**
ORCID ID: 0000-0002-0641-5229

[4]Department of Thoracic Surgery, Basaksehir Cam and Sakura City Hospital, Istanbul, Turkey, **eren.erdogdu@istanbul.edu.tr**
ORCID ID: 0000-0001-8153-0107

[5]Department of Thoracic Surgery, Ankara Atatürk Sanatorium Training and Research Hospital, Ankara, Turkey, **acar8106@gmail.com**
ORCID ID: 0009-0001-8920-7416

[6]Department of Thoracic Surgery, Istanbul Mehmet Akif Ersoy Chest Cardiovascular Surgery Training and Research Hospital, Istanbul, Turkey, **mhanozu@gmail.com**
ORCID ID: 0009-0003-3979-4442

[7]Department of Thoracic Surgery, Basaksehir Cam and Sakura City Hospital, Istanbul, Turkey, **mehmetaliunal44@gmail.com**
ORCID ID: 0000-0003-0876-4964

[8]Department of Thoracic Surgery, Basaksehir Cam and Sakura City Hospital, Istanbul, Turkey, **muhammedkertmen@gmail.com**
ORCID NO: 0009-0001-6636-3109

DOI: https://doi.org/10.5281/zenodo.15873689

E mail: ubakyayinevi@gmail.com
www.ubakyayinevi.org
It is responsibility of the author to abide by the publishing ethics rules.
UBAK Publishing House – 2025©

ISBN: 978-625-5923-70-7
July / 2025
Ankara / Turkey

PREFACE

Due to its unique anatomical position and complex physiological functions, the diaphragm is an extremely important structure within the thoracoabdominal system. It serves not only as a dynamic barrier between the thoracic and abdominal cavities, but also as the primary respiratory muscle essential for effective ventilation. The complexity of its functions necessitates surgical interventions that require specialized expertise, extensive knowledge and high precision.

Recent advances in diaphragmatic surgery, particularly the widespread adoption of minimally invasive and robotic techniques, have enabled surgeons to perform procedures with greater efficiency and less morbidity. Accordingly, there is a growing need for a comprehensive reference covering the entire spectrum of diaphragmatic pathology, from embryologic abnormalities to congenital and acquired disorders, functional disorders, tumors, and cystic lesions.

This book takes a multidisciplinary approach to the diagnosis and treatment of diaphragmatic disorders. It begins with a detailed discussion of the embryological development and anatomical structure of the diaphragm and then comprehensively covers topics such as congenital diaphragmatic hernias, acquired disorders, functional disorders, eventration and plication procedures, tumors and cysts. In addition, special attention is given to the role of minimally invasive and robotic surgical techniques in the light of current scientific literature.

Each chapter has been carefully written by recognized specialists in thoracic surgery who combine basic theory with real clinical cases,

detailed illustrative material and modern surgical approaches. The book is designed to provide basic and advanced knowledge to both practicing thoracic surgeons and residents in training.

I firmly believe that this work will serve as a valuable guide for the diagnosis and treatment of complex diaphragmatic disorders commonly encountered in thoracic surgical practice. In addition, it should make an important contribution to the training of young thoracic surgeons and support their academic endeavors.

I would like to express my sincere gratitude to all the esteemed authors who have generously shared their expertise in the preparation of this volume. I hope this book proves to be a practical tool for clinical application and an inspiring reference for ongoing scientific research in the field of thoracic surgery.

14.07.2025

Assoc. Prof. Dr. Turkan DUBUS

Editor

TABLE OF CONTENTS

CHAPTER 1

EMBRYOLOGICAL DEVELOPMENT, ANATOMY, AND FUNCTION OF THE DIAPHRAGM

Dr. Eren ERDOGDU, MD, Thoracic Surgeon
Dr. Muhammet KERTMEN, MD, Thoracic Surgery Resident

INTRODUCTION

Although the diaphragm has been known as an independent anatomical unit since antiquity, its full developmental and functional complexity was only recognized in the course of the last century. First described by Hippocrates and Galen as a membranous partition between the thoracic and abdominal cavities, in the past it was only recognized as a structural boundary. However, its most important physiological functions were only discovered with the advances of modern biomedical science (Netter, 2018; Standring, 2021).

The diaphragm is the primary muscular apparatus of respiration and serves as the main drive for the inhalation mechanism. Its rhythmic contraction generates intrathoracic pressure and thus allows the lungs to inflate, while its relaxation enables passive exhalation. However, the functions of the diaphragm go far beyond ventilation dynamics. By regulating intra-abdominal pressure, it plays a central role in a number of physiological processes such as defecation, micturition, vomiting and childbirth. Maintaining the integrity of the diaphragm is therefore

essential not only for respiratory efficiency, but also for the maintenance of homeostatic and visceral functions that are critical for quality of life and survival (Negrini, 2022).

Anatomically, the diaphragm is a structurally complex, asymmetrical, bilaterally curved muscle-tendon segment. Its integrity is essential for maintaining the separation between the thoracic and abdominal cavities and thus serves as a dynamic barrier against herniation of abdominal organs (Clugston et al., 2010). From an embryological perspective, its formation is the result of a highly coordinated and temporally regulated morphogenetic sequence involving the integration of multiple tissues of different embryonic origin (Sadler et al., 2024; Sefton et al., 2018) This multifaceted developmental trajectory reflects the critical physiological functions of the diaphragm and its susceptibility to a wide range of congenital abnormalities (Donahoe et al., 2016; Zani et al., 2022).

1. Embryological development of the diaphragm

The diaphragm is the most important respiratory muscle in mammals and serves as an anatomical partition between the thoracic and abdominal cavities. Its embryological development is a highly coordinated and temporally regulated process that involves the migration, proliferation, differentiation and integration of cell populations derived from different embryonic sources (Persaud & Torchia, 2025; Sadler et al., 2024). Successful formation of a structurally and functionally competent diaphragm requires precise orchestration of mesenchymal, myogenic, neurogenic and vasculogenic elements (Dannenberg & Seaver, 2018). Disruptions at any stage of this

complex developmental cascade can lead to severe congenital anomalies, many of which are associated with significant morbidity and high perinatal mortality (Dannenberg & Seaver, 2018; Donahoe et al., 2016).

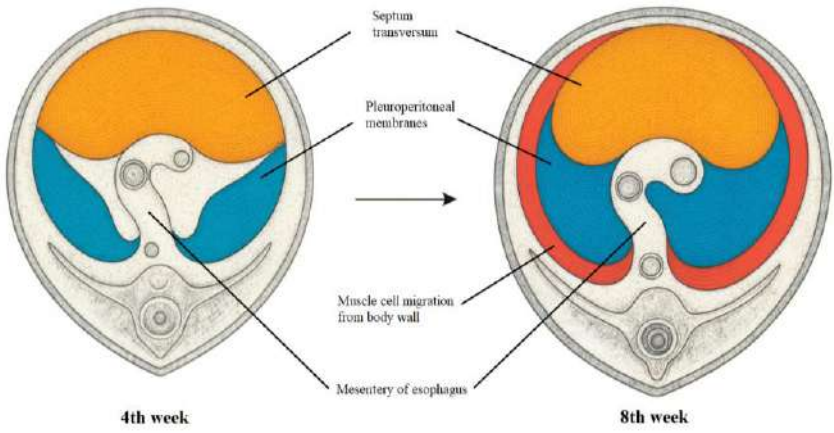


Figure 1. Development of the diaphragm from the 4th to the 8th week of pregnancy.

In the human embryo, development of the diaphragm begins around the fourth week of gestation and is nearly complete by the end of the eighth week (Persaud & Torchia, 2025; Sadler et al., 2024) (Figure 1). This complicated morphogenetic process is composed of four main components of the embryo, each contributing different structural elements to the mature diaphragm.

1. Transverse septum
2. Pleuroperitoneal membranes

3. Dorsal mesentery of the esophagus

4. Parietal mesoderm

Each embryologic progenitor contributes different anatomic and histologic elements to the developing diaphragm. The coordinated morphogenetic interplay of these components is crucial for the structural integrity and physiological functionality of the diaphragm (Sefton et al., 2018).

1.1. Transverse septum

The transverse septum is the earliest embryologic structure that appears during the development of the diaphragm and is initially located at the level of the cervical region (Sadler et al., 2024). During embryonic folding, it is displaced ventrocaudally and is eventually located cranial to the developing liver (Persaud & Torchia, 2025). Although this mesenchymal structure does not contain myogenic cells, it gives rise to the central tendon of the diaphragm. It also serves as a physical barrier between the liver and the pericardial cavity (Sadler et al., 2024; Sefton et al., 2018).

1.2 Pleuroperitoneal membranes

The pleuroperitoneal folds (PPFs) are bilaterally symmetrical mesenchymal structures that transiently delineate the thoracic and abdominal cavities during early embryogenesis. These folds become morphologically recognizable at the end of the fifth week of pregnancy and increasingly expand laterally-medially in the sixth and seventh week. This growth process culminates in their fusion with the transverse

septum and the dorsal mesentery of the esophagus, allowing the formation of the pericardioperitoneal canals. The PPFs form the majority of the posterolateral and lateral components of the diaphragm. Disruption of the formation or fusion of these structures is an important pathogenic mechanism underlying posterolateral congenital diaphragmatic hernias, particularly the Bochdalek type (Clugston et al., 2010; Donahoe et al., 2016; Sefton et al., 2018).

1.3 Dorsal mesentery of the esophagus

The dorsal mesentery of the esophagus forms the medial muscle columns of the diaphragm, the so-called crura, and contributes to the morphogenesis of the esophageal hiatus. This structure plays a central role in maintaining the axial stability of the diaphragm. Disruptions in its development have been associated with a number of median diaphragmatic defects, most notably congenital hiatal hernias (Persaud & Torchia, 2025; Sadler et al., 2024).

1.4. Parietal mesoderm

The somatic mesoderm of the body wall plays a central role in the development of the peripheral muscle components of the diaphragm. In particular, the ventrolateral muscle fibers arise from mesenchymal progenitor cells that originate from this region and undergo spatially regulated migration with subsequent myogenic differentiation. In addition, the connective tissue elements also originate from this mesodermal source alongside the parietal pleura and peritoneum (Sadler et al., 2024; Sefton et al., 2018).

1.5. Migration of the myogenic precursors and innervation of the diaphragm

The muscles of the diaphragm arise from myogenic progenitor cells that originate from the dermatomyotomes of the cervical somites, particularly from segments C3 to C5. These progenitor cells migrate towards the pleuroperitoneal folds, where they undergo proliferative expansion and subsequently differentiate into myoblasts and later into myotubes. The formation of muscle tissue is closely synchronized with the projection of axons of the phrenic nerve into the same region. The phrenic nerve arises from motor neurons in the C3–C5 segments of the spinal cord and supplies the diaphragm with both motor and sensory nerve fibers. This coordinated neuro-muscular interaction is crucial for the terminal differentiation of muscle fibers and the acquisition of their contractile function (Allan & Greer, 1997; Sefton et al., 2018).

1.6. Vascularization and tendonogenesis of the diaphragm

The arterial supply to the diaphragm is primarily via the inferior phrenic artery, with additional contributions from various thoracoabdominal vessels. The organization of the diaphragmatic vascular network follows the migration and localization of the myogenic cells and ensures an efficient blood supply to the developing musculature. The connective tissue elements of the diaphragm, in particular the central tendon, develop from mesenchymal precursor cells from the transverse septum. Tendonogenesis precedes myogenic differentiation both temporally and morphogenetically and plays a crucial role in conferring biomechanical stability to the developing diaphragm through early

deposition of extracellular matrix and tensile reinforcement (Clugston et al., 2010; Persaud & Torchia, 2025; Sadler et al., 2024).

1.7 Timeline for development

The development of a fully functional and anatomically intact diaphragm depends on the temporal and spatial coordination of the complex embryological events described above (Sefton et al., 2018). Disruptions in the early stages of diaphragmatic development play a central role in the pathogenesis of various congenital anomalies, particularly congenital diaphragmatic hernia (Clugston et al., 2010; Donahoe et al., 2016) (Table 1).

Table 1. Developmental timeline of the diaphragm

Week of gestation	**Events**
Week 4	Septum transversum appears.
Week 5-6	The PPFs emerge and begin to enlarge.
Week 6	The migration of the muscle precursor cells begins.
Week 7	Projections of the phrenic nerve and interaction of the nerve muscles.
Week 8	The formation of the structural diaphragm is largely complete.

PPFs: pleuroperitoneal folds.

1.8 Microscopic structure and histology

The diaphragm consists of typical striated skeletal muscle tissue and fulfills an autonomic function that is important for breathing. Its muscle fibers consist predominantly of type I (slow twitch, oxidative) and type IIa fibers, which are well suited to maintaining the continuous, fatigue-free contractions required for respiratory activity. These fibers are arranged radially and attach to the tendo centrale (central tendon), which enables coordinated and efficient contraction. Both the muscular and tendinous components of the diaphragm can be characterized immunohistochemically by the expression of specific muscle-associated proteins, including desmin, myogenin and titin (Allan & Greer, 1997; Clugston, Zhang, & Greer, 2010; Donahoe, Longoni, & High, 2016; Persaud & Torchia, 2025; Sadler et al, 2024; Sefton et al, 2018).

2. Developmental anomalies of the diaphragm

Congenital anomalies of the diaphragm are caused by incomplete or defective development of the diaphragmatic structure during embryogenesis. These developmental anomalies cover a broad clinical spectrum, ranging from mild respiratory distress to severe, life-threatening respiratory failure in newborns. Among these anomalies, congenital diaphragmatic hernia (CDH) is the most common and best-studied entity; however, other, less common diaphragmatic malformations have also been documented (Clugston et al., 2010; Sadler et al., 2024).

2.1. Congenital diaphragmatic hernia (CDH)

In CDH, defective development of the diaphragm during embryonic development leads to a hernia of the abdominal organs into the thoracic cavity. This anomaly is often accompanied by pulmonary hypoplasia and pulmonary vascular dysplasia, which together contribute to significant respiratory impairment. CDH occurs in approximately 1 in 2,500 to 4,000 live births and is one of the most common causes of neonatal respiratory failure (Ackerman & Pober, 2007; Keijzer & Puri, 2010; Zani et al., 2022).

2.1.1. Posterolateral hernia (Bochdalek type)

This is the most common form of congenital diaphragmatic hernia, occurring in about 80–85% of cases and is predominantly localized in the left hemithorax. It results from the pleuroperitoneal folds not closing completely around the sixth week of pregnancy, a critical step in the development of the diaphragm. As a result, abdominal viscera, including the stomach, small intestine, spleen and occasionally liver, can herniate into the thoracic cavity and interfere with ipsilateral lung development (Merrell & Kardon, 2013; Torfs et al., 1992).

2.1.2. Anterior hernia (Morgagni type)

This rarer subtype of congenital diaphragmatic hernia, which accounts for about 2–5% of cases, is typically located in the right hemithorax, behind the pericardium. It is caused by a defect in the anterior part of the central tendon. Morgagni hernias are frequently asymptomatic and

are often discovered incidentally in late childhood or adulthood (Katsaros et al., 2021; Nasr & Fecteau, 2009).

2.1.3. Central diaphragmatic hernias

This rare form is associated with defects in the development of the central tendon. Clinically, it can be associated with both respiratory and gastrointestinal symptoms (Merrell & Kardon, 2013).

2.2. Diaphragmatic agenesis

Diaphragmatic agenesis is a rare but usually fatal congenital anomaly characterized by the complete absence of diaphragmatic tissue. Bilateral agenesis is generally incompatible with life and often leads to intrauterine death. In unilateral agenesis, the prognosis depends largely on the herniating viscera and the degree of associated pulmonary hypoplasia. Embryologically, this defect reflects a severe failure in the development of the transverse septum, pleuroperitoneal folds and parietal mesoderm (Fukuoka et al., 2022).

2.3. Diaphragmatic hypoplasia

In diaphragmatic hypoplasia, the diaphragm is structurally present but has reduced thickness, muscle mass or functional capacity. It often occurs in conjunction with CDH and causes a disturbance in the motor activity of the diaphragm. Radiologic imaging plays an important role in the differential diagnosis (Zani et al., 2022).

2.4. Diaphragmatic eventration

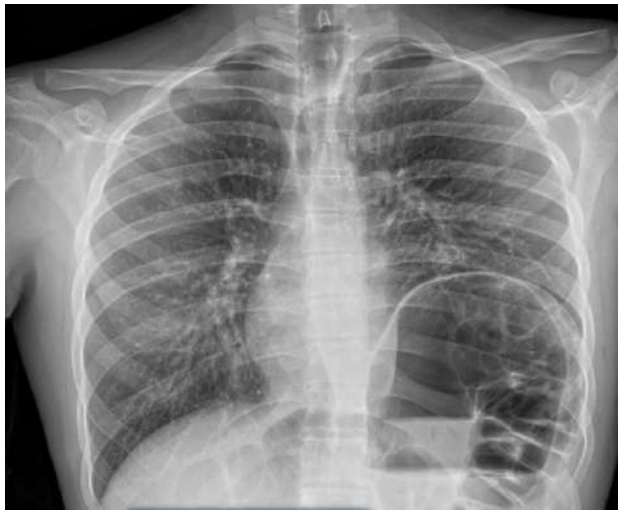


Figure 2. Posteroanterior chest radiograph showing a left-sided diaphragmatic ventration.

In diaphragmatic eventration, the diaphragm remains anatomically intact and in its normal position, but the muscle tissue is partially or completely replaced by fibrous-elastic connective tissue. This condition typically results from a primary myogenic developmental defect or insufficient innervation by the phrenic nerve. Clinically, it can lead to paradoxical diaphragmatic movements and unilateral respiratory insufficiency (Konstantinidi et al., 2023; Shibuya & De Coppi, 2022) (Figure 2).

2.5. Diaphragmatic duplication

Diaphragmatic duplication is an extremely rare developmental anomaly resulting from the formation of two different diaphragmatic layers

during embryogenesis. Although its pathophysiological basis is not fully understood, it is most commonly observed as an incidental anatomical change without clinical significance (Becmeur et al., 1995).

2.6. Anomalies of the phrenic nerve

The phrenic nerve, which originates from motor neurons in the spinal cord segments C3-C5, provides the motor innervation of the diaphragm. Disturbances in the axonal projection of the phrenic nerve during embryogenesis can impair the correct development of the diaphragmatic musculature. Primary abnormalities in the development of the phrenic nerve can lead to congenital diaphragmatic paralysis (Fogarty et al., 2018; Kokatnur & Rudrappa, 2018; McCool & Tzelepis, 2012).

3. Anatomy and physiology of the diaphragm

3.1. Anatomical features

The diaphragm is a highly complex structure, both in terms of its embryological origin and its functional properties, and represents the most important musculoskeletal barrier between the thoracic and abdominal cavities. This dome-shaped, thin muscle serves as the main driver of the respiratory mechanism and at the same time plays a crucial role in maintaining the anatomical position of the mediastinal organs. Due to the volumetric influence of the liver, the right hemidiaphragm is positioned higher than the left and often extends to the level of the fourth intercostal space (Netter, 2018; Standring, 2021).

3.1.1. Substructures and components

Anatomically, the diaphragm is made up of four main components:

- *Pars costalis:* Arises from the inner surfaces of the lower six ribs and their costal cartilage.

- *Pars lumbalis:* The thickest part of the diaphragm, which originates from the upper lumbar vertebrae (L1–L3) and contains the right and left crural fibers (crus dextrum and crus sinistrum).

- *Pars sternalis:* Runs from the posterior part of the xiphoid process of the sternum to the central tendon.

- *Central tendon:* A non-muscular, fibrous structure in which the muscle fibers converge. The fibrous pericardium is firmly anchored to this central tendon.

3.1.2. Anatomical openings

The diaphragm contains several physiological openings that allow the passage of vital thoracoabdominal structures:

- *Hiatus aorticus (T12):* The descending aorta, the thoracic duct and the azygos vein run through it.

- *Esophageal hiatus (T10):* The esophagus and the anterior and posterior trunks of the vagus nerve run through it.

- *Foramen venae cavae (T8):* A fibrous foramen that allows passage of the inferior vena cava and dilates with contraction of the diaphragmatic to facilitate venous return.

In addition to these main foramina, smaller vascular and neural structures traverse smaller openings or areas of lower resistance, such as Bochdalek's triangle and Morgagni's foramen.

3.1.3. Fascial relationships and adjacent structures

- *Upper (thoracic) surface:* Lies in close relationship to the heart and lungs. The fibrous pericardium is firmly attached to the central tendon.

- *Lower (abdominal) surface:* In contact with the liver, stomach, spleen and left kidney. The adrenal glands also have a close anatomical relationship with the diaphragm.

3.2. Physiological functions

3.2.1. Role in breathing

The diaphragm is the main muscle of inspiration and contributes to about 70 % of tidal breathing at rest. When it contracts, it descends, increasing the volume of the thoracic cavity and decreasing intrathoracic pressure, creating a negative pressure gradient that facilitates passive airflow into the alveoli (Schepens, Fard, & Goligher, 2020).

- *Inhalation:* Contraction of the diaphragm increases thoracic volume.

- *Exhalation:* The diaphragm relaxes and lifts passively, reducing the thoracic volume.

3.2.2 Intra-abdominal pressure and visceral functions

The diaphragm plays an important role in increasing intra-abdominal pressure, which facilitates vital functions such as defecation, micturition, vomiting and childbirth. These actions are mediated in conjunction with the abdominal musculature and form a functional synergy that is critical to visceral biomechanics (Hodges & Gandevia, 2000).

3.2.3. Effects on the circulatory and lymphatic system

Diaphragmatic excursions modulate the thoracoabdominal pressure gradient and thus improve venous return. During inspiration, the negative pressure exerted on the inferior vena cava promotes increased blood flow into the right atrium. In addition, the diaphragmatic movements facilitate lymphatic drainage by exerting a suction effect on the thoracic duct (Hruby & Martinez, 2021; Negrini, 2022).

3.2.4. Role in phonation and postural stability

By modulating intrathoracic pressure, the diaphragm plays an essential role in voice production during phonation. It is also a key component of trunk stability and postural control. In maintaining the integrity of the spine, the diaphragm works synergistically with the transversus abdominis and pelvic floor muscles (Badaró et al., 2023; Elhofy et al., 2025).

4. Innervation and vascularization of the diaphragm

4.1. Innervation

The primary innervation of the diaphragm is provided by the phrenic nerve, which contains both motor and sensory fibers. Understanding its embryological origin and anatomic course is of particular importance for clinical practice (Moore & Dalley, 2018; Sadler et al., 2024; Standring, 2021).

Motor innervation

The motor supply of the entire diaphragm is provided bilaterally by the phrenic nerves.

- *Origin:* Arises from the anterior branches of the cervical spinal nerves C3–C5, with the largest contribution coming from C4.

- *Course:* In the cervical spine, the nerve runs anterior to the scalene anterior muscle and enters the thoracic cavity. It runs laterally to the pericardium and continues in the anterior mediastinum to reach the tip of the diaphragm.

The right phrenic nerve runs in front of the hilum of the right lung in the immediate vicinity of the superior and inferior vena cava and enters the diaphragm via the cavity opening.

The left phrenic nerve runs close to the aortic arch and the left ventricle and continues to the left pulmonary hilum before ending at the left diaphragmatic dome.

Sensory innervation

- *Central tendon and thoracic surface:* Innervated by the sensory fibers of the phrenic nerve.

- *Peripheral regions:* Receive sensory input via the anterior branches of the 6th to 12th thoracic spinal nerves, particularly the intercostal and subcostal nerves.

- *Abdominal surface:* Partial sensory transmission via visceral branches of the lower thoracic nerves.

4.2. Arterial supply

The diaphragm has a rich vascular network supplied by both the thoracic and abdominal arterial systems, with extensive anastomotic connections ensuring good perfusion.

Pericardiacophrenic artery:

- *Origin:* Internal thoracic artery.

- *Course:* Runs along the phrenic nerve and supplies the pericardium and the adjacent diaphragmatic regions.

Arteriae musculophrenicae:

- *Origin:* Terminal branch of the internal thoracic artery.

- *Distribution:* Supplies the anterior and lateral sides of the diaphragm with blood.

Lower posterior intercostal arteries (T7–T12):

- *Supplies small segmental branches that support the ribbed portion of the diaphragm.*

Inferior phrenic artery:

- *Origin:* abdominal aorta.

- *This is the most important arterial supply to the diaphragm and also leads to the upper adrenal branches.*

The presence of numerous anastomoses between these vessels supports the functional integrity of the diaphragm and should be taken into account during surgical procedures, especially with regard to hemostasis.

4.3. Venous drainage

The venous drainage of the diaphragm runs parallel to its arterial supply and includes both the thoracic and abdominal venous systems.

Thoracic surface

- *Pericardiocephalic veins* → Brachiocephalic veins.

- *Musculophrenic veins* → Internal thoracic veins → Subclavian vein.

- *Inferior intercostal veins* → Azygos and hemiazygos veins.

Surface of the abdomen

- *The inferior diaphragmatic veins open directly into the inferior vena cava.*

- *On the left side, drainage can take place via the left renal vein, although there are anatomical differences here.*

4.4. Lymphatic drainage

The diaphragm has a bidirectional lymphatic drainage and provides a connection between the thoracic and abdominal lymphatic areas.

Upper area:

- *Anterior region:* Parasternal lymph nodes (Nodi lymphoidei parasternales).

- *Posterior regions:* Posterior mediastinal lymph nodes (Nodi lymphoidei mediastinales posteriores).

Inferior surface:

- *Anterior and lateral regions:* Preaortic and celiac lymph nodes (nodi lymphoidei praeaortici et coeliaci).

- *Posterior regions:* Lumbar lymph nodes (nodi lymphoidei lumbales).

This extensive lymphatic plexus plays a decisive role in the transdiaphragmatic spread of intra-abdominal and intrathoracic malignant tumors, e.g. in the metastasis of hepatocellular carcinomas to thoracic lymph nodes (Locatelli et al., 2020).

5. Evaluation of the function of the diaphragm

The diaphragm is a key component of the respiratory mechanism, and its functional integrity depends on both its anatomical structure and neuromuscular control systems. In the diagnosis of diaphragmatic

dysfunction, it is important to examine not only structural abnormalities but also movement patterns. Various imaging techniques are used for this purpose, each with specific advantages and limitations (Gridelli et al., 2015; Houston et al., 1994; Nason et al., 2012; Shanmuganathan et al., 2000).

5.1. Radiography of the chest

Chest radiography is a common and easily accessible imaging modality for the initial evaluation of diaphragmatic pathology. Posteroanterior and lateral radiographs of the chest in the upright position can reveal height asymmetries of the diaphragmatic domes, contour irregularities, silhouette abnormalities and diaphragmatic elevation (Houston et al., 1994; Nason et al., 2012).

Advantages:

- *Inexpensive, rapid and widely available.*

- *Provides diagnostic clues for conditions such as diaphragmatic elevation, eventration or herniation.*

Limitations:

- *Does not provide functional assessment.*

- *Dynamic movement analysis is not possible and postural effects may contribute to diagnostic uncertainty.*

5.2. Diaphragmatic ultrasound examination

Ultrasonography allows assessment of diaphragmatic parameters such as excursion, muscle thickness and degree of thickening. Imaging can be performed via subcostal or intercostal approaches, with the right hemidiaphragm being more accessible (Gridelli et al., 2015; Shanmuganathan et al., 2000).

Advantages:

- *Enables real-time and repeatable functional analysis.*

- *Radiation free; suitable for bedside use, especially in critically ill patients.*

Limitations:

- *Operator-dependent and more technically demanding for the left hemidiaphragm.*

- *Image quality may be affected by obesity or intestinal gas.*

5.3. Dynamic fluoroscopy

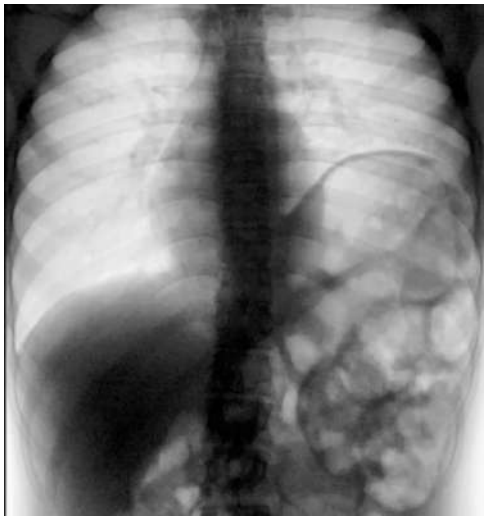


Figure 3. Diaphragmatic eventration in dynamic fluoroscopy.

Fluoroscopy enables dynamic assessment of diaphragmatic movement in real time. In particular, imaging during a special maneuver, the so-called “sniff test”, allows the direction and amplitude of diaphragmatic movement to be assessed. In this test, the patient is asked to perform rapid and forceful nasal breathing.

Under normal conditions, both hemidiaphragms move synchronously in a caudal direction during this maneuver (Chow & Hatem, 2022) (Figure 3).

The importance of the sniff test:

In individuals with diaphragmatic paralysis, the affected hemidiaphragm shows a paradoxical movement, i.e. it moves cranially

rather than caudally. This characteristic finding makes the fluoroscopic sniff test the gold standard for the diagnosis of unilateral diaphragmatic paralysis (Houston et al., 1994; Nason et al., 2012).

Technical details:

- Imaging is preferably performed in an upright position, although a sitting or supine position can be used if necessary.

- Bilateral projection views are obtained to allow comparative assessment of both hemidiaphragms.

- Diaphragmatic excursions, symmetry and timing are carefully observed.

Advantages:

- Enables real-time visualization that provides direct information about the motor function of the diaphragm.

- High specificity and sensitivity in detecting unilateral diaphragmatic dysfunction.

Limitations:

- Use of ionizing radiation, which limits repeated use.

- Requires patient cooperation, which makes it difficult to use in pediatrics or in patients with severe respiratory distress syndrome.

5.4. Computed tomography (CT)

Thoracoabdominal CT offers better resolution of the soft tissues for morphologic assessment of the diaphragm. It is indispensable for the diagnosis of diseases such as diaphragmatic hernias, traumatic ruptures and mass infiltrations. With the advent of dynamic CT techniques in recent years, limited functional data can also be obtained (Shanmuganathan et al., 2000).

Advantages:

- *Provides a high level of anatomical detail.*

- *Allows simultaneous assessment of adjacent structures.*

Limitations:

- *Requires high radiation exposure.*

- *Functional analysis remains limited.*

5.5. Magnetic resonance imaging (MRI)

MRI can accurately visualize structural features of the diaphragmatic muscle, such as atrophy and fibrosis. In particular, cine MRI techniques can be used to assess the movement of the diaphragm during the respiratory cycle non-invasively and without radiation exposure (Nason et al., 2012).

Advantages:

- *Excellent soft tissue resolution.*

- *Radiation-free and considered safe.*

Limitations:

- *Relatively limited accessibility and higher cost.*

- *Long acquisition times and motion artifacts can affect diagnostic quality.*

5.6. Spirometry and volumetric assessment

Spirometry and volumetric breath tests are often used to assess diaphragmatic function in an indirect but practical way. These tests provide important clues, especially when diaphragmatic paralysis or weakness is suspected (McCool & Tzelepis, 2012).

Comparison of forced volume in the seated resting position

In normal individuals, there is a 5-10% decrease between the forced vital capacity (FVC) values measured in the sitting and supine positions.

However, in the case of bilateral diaphragmatic paralysis, the vital capacity value can decrease by up to 50% in the supine position. With unilateral paralysis, a decrease of 15-25% is usually observed.

This difference is related to the pressure of the abdominal organs on the diaphragm under the influence of gravity and the inability of the weak side of the muscle to compensate for this (McCool & Tzelepis, 2012).

REFERENCES

Ackerman, K. G., & Pober, B. R. (2007). Congenital diaphragmatic hernia and pulmonary hypoplasia: New insights from developmental biology and genetics. *American Journal of Medical Genetics Part C: Seminars in Medical Genetics*, *145C*(2), 105–118. https://doi.org/10.1002/ajmg.c.30128

Allan, D. W., & Greer, J. J. (1997). Embryogenesis of the phrenic nerve and diaphragm in the fetal rat. *Journal of Comparative Neurology*, *382*(4), 459–468.

Badaró, F., Caleffi-Pereira, M., Macedo, J. P., Behlau, M., & de Albuquerque, A. L. P. (2023). Vocal impact in patients with unilateral diaphragmatic paralysis. *Journal of Voice.*

Becmeur, F., Horta, P., Donato, L., Christmann, D., & Sauvage, P. (1995). Accessory diaphragm Review of 31 cases in the literature. *European Journal of Pediatric Surgery*, *5*(1), 43–47. https://doi.org/10.1055/s-2008-1066162

Chow, J., & Hatem, M. (2022). Quantitative analysis of diaphragm motion during fluoroscopic sniff test to assist in diagnosis of hemidiaphragm paralysis. *Radiology Case Reports*, *17*(5), 1750–1754.

Clugston, R. D., Zhang, W., & Greer, J. J. (2010). Early development of the primordial mammalian diaphragm and cellular mechanisms of nitrofen-induced congenital diaphragmatic hernia. *Birth Defects Research Part A: Clinical and Molecular Teratology*, *88*(1), 15–24.

Dannenberg, L. C., & Seaver, E. C. (2018). Regeneration of the germline in the annelid Capitella teleta. *Developmental Biology, 440*(2), 74–87.

Donahoe, P. K., Longoni, M., & High, F. A. (2016). Polygenic causes of congenital diaphragmatic hernia produce common lung pathologies. *The American Journal of Pathology, 186*(10), 2532–2543.

Elhofy, N. A., Youssef, A. M., Osman, D. A., & Abdellatife, A. N. (2025). The relationship between dynamics of pelvic floor muscles and phonation patterns: A narrative review. *Deraya International Journal for Medical Sciences and Rehabilitation, 1*(1). https://doi.org/10.21608/dijms.2025.354333.1001

Fogarty, M. J., Mantilla, C. B., & Sieck, G. C. (2018). Breathing: Motor control of diaphragm muscle. *Physiology (Bethesda), 33*(2), 113–126. https://doi.org/10.1152/physiol.00002.2018

Fukuoka, S., Yamamura, K., Nagata, H., Toyomura, D., Nagatomo, Y., Eguchi, Y., Ochiai, M. (2022). Clinical outcomes of pulmonary agenesis: A systematic review of the literature. *Pediatric Pulmonology, 57*(12), 3060–3068.

Gridelli, C., Rossi, A., Carbone, D. P., Guarize, J., Karachaliou, N., Mok, T., & Rosell, R. (2015). Non-small-cell lung cancer. *Nature Reviews Disease Primers, 1*, 15009. https://doi.org/10.1038/nrdp.2015.9

Hodges, P. W., & Gandevia, S. C. (2000). Activation of the human diaphragm during a repetitive postural task. *Journal of Physiology, 522*(1), 165–175.

Houston, J., Angus, R., Cowan, M., McMillan, N., & Thomson, N. (1994). Ultrasound assessment of normal hemidiaphragmatic movement: Relation to inspiratory volume. *Thorax, 49*(5), 500–503.

Hruby, R. J., & Martinez, E. S. (2021). The lymphatic system: An osteopathic review. *Cureus, 13*(7), e16448.

Katsaros, I., Katelani, S., Giannopoulos, S., Machairas, N., Kykalos, S., Koliakos, N., Schizas, D. (2021). Management of Morgagni's hernia in the adult population: A systematic review of the literature. *World Journal of Surgery, 45*(10), 3065–3072.

Keijzer, R., & Puri, P. (2010). Congenital diaphragmatic hernia. *Seminars in Pediatric Surgery, 19*(3), 180–185.

Kokatnur, L., & Rudrappa, M. (2018). Diaphragmatic palsy. *Diseases, 6*(1), 16. https://doi.org/10.3390/diseases6010016

Konstantinidi, A., Liakou, P., Kopanou Taliaka, P., Lampridou, M., Kalatzi, N., Loukas, I., Sokou, R. (2023). Congenital diaphragmatic eventration in the neonatal period: Systematic review of the literature and report of a rare case presenting with gastrointestinal disorders. *Pediatric Reports, 15*(3), 442–451.

Locatelli, F., Ambrosi, F., & Rossi, G. (2020). Mechanisms of tumor dissemination in thoracic neoplasms. *The Thorax*, 1–33.

McCool, F. D., & Tzelepis, G. E. (2012). Dysfunction of the diaphragm. *The New England Journal of Medicine, 366*(10), 932–942. https://doi.org/10.1056/NEJMra1007236

Merrell, A. J., & Kardon, G. (2013). Development of the diaphragm – A skeletal muscle essential for mammalian respiration. *The FEBS Journal, 280*(17), 4026–4035.

Moore, K. L., & Dalley, A. F. (2018). *Clinically oriented anatomy* (8th ed.). Wolters Kluwer India Pvt Ltd.

Nason, L. K., Walker, C. M., McNeeley, M. F., Burivong, W., Fligner, C. L., & Godwin, J. D. (2012). Imaging of the diaphragm: Anatomy and function. *Radiographics, 32*(2), E51–E70.

Nasr, A., & Fecteau, A. (2009). Foramen of Morgagni hernia: Presentation and treatment. *Thoracic Surgery Clinics, 19*(4), 463–468.

Negrini, D. (2022). Morphological, mechanical and hydrodynamic aspects of diaphragmatic lymphatics. *Biology, 11*(12), 1803.

Netter, F. H. (2018). *Atlas of human anatomy: Latin terminology E-book* (7th ed.). Elsevier Health Sciences.

Persaud, T. V. N., & Torchia, M. G. (2025). *The developing human: Clinically oriented embryology* (12th ed.). Retrieved from https://www.clinicalkey.com/dura/browse/bookChapter/3-s2.0-C20220018183

Sadler, T. W., Sadler-Redmond, S. L., Tosney, K., Byrne, J., Imseis, H., Langman, J., & Wolters, K. (2024). *Langman's medical embryology* (15th ed., International edition). Wolters Kluwer.

Schepens, T., Fard, S., & Goligher, E. C. (2020). Assessing diaphragmatic function. *Respiratory Care, 65*(6), 807–819. https://doi.org/10.4187/respcare.07410

Sefton, E. M., Gallardo, M., & Kardon, G. (2018). Developmental origin and morphogenesis of the diaphragm, an essential mammalian muscle. *Developmental Biology*, *440*(2), 64–73.

Shanmuganathan, K., Killeen, K., Mirvis, S. E., & White, C. S. (2000). Imaging of diaphragmatic injuries. *Journal of Thoracic Imaging*, *15*(2), 104–111.

Shibuya, S., & De Coppi, P. (2022). Eventration of diaphragm. In *Fundamentals of Pediatric Surgery* (pp. 765–771). Springer.

Standring, S. (2021). *Gray's anatomy* (42nd ed.). Elsevier Health Sciences.

Torfs, C. P., Curry, C. J., Bateson, T. F., & Honoré, L. H. (1992). A population-based study of congenital diaphragmatic hernia. *Teratology*, *46*(6), 555–565.

Zani, A., Chung, W. K., Deprest, J., Harting, M. T., Jancelewicz, T., Kunisaki, S. M., Keijzer, R. (2022). Congenital diaphragmatic hernia. *Nature Reviews Disease Primers*, *8*(1), 37. https://doi.org/10.1038/s41572-022-00353-3

CHAPTER 2

CONGENITAL DIAPHRAGMATIC HERNIAS IN ADULTS

Dr. Leyla Nesrin ACAR, Thoracic Surgeon

INTRODUCTION

Congenital diaphragmatic hernias (CDH) are defined as the herniation of abdominal organs into the thoracic cavity through a defect in the diaphragm. The etiology of CDH remains unclear; however, several mechanisms have been proposed, including insufficient innervation of the diaphragm by the phrenic nerve, abnormal development of lung tissue, underdevelopment or weakness of the diaphragmatic muscle fibers in the herniation area (Gomes-Da Silva de Rosenzweig et al., 2024).

CDH was first identified incidentally during an autopsy by Lazarus Riverius in 1697 and subsequently described (Al-Emadi et al., 1999).

1. Anatomy

The diaphragm is a dome-shaped musculoaponeurotic structure that separates the thoracic and abdominal cavities. It attaches to the sternum, ribs, and lumbar vertebrae. Its intrathoracic surface is covered by pleura and the abdominal surface by peritoneum. The central part consists of the aponeurotic centrum tendineum, while the peripheral muscular portion attaches to the apertura thoracica inferior.

The muscular region is divided into three parts: pars sternalis, pars costalis, and pars lumbalis (Sanford et al., 2018).

CDH is rare and epidemiological data require population-based studies. The prevalence in Europe is approximately 2.3 per 100,000 live births with a male-to-female ratio of 1:0.69 (McGivern et al., 2015). Globally, the incidence ranges from 1 in 2,000 to 1 in 12,500 live births (Goh et al., 2007).

2. Classification

CDH is classified into two major types:

-Congenital posterolateral diaphragmatic hernia (Bochdalek hernia)

-Parasternal hernia (Morgagni hernia)

2.1. Bochdalek hernia

Bochdalek hernia (BH) is characterized by the herniation of abdominal organs through a defect between the pars lumbalis and pars costalis, resulting from the failure of closure of the pleuroperitoneal canal by the 8th week of gestation. It was first described by Victor Alexander Bochdalek in 1867 (Mullins et al., 2001).

BH occurs in approximately 1 in 2,000–5,000 live births. While typically diagnosed antenatally or shortly after birth due to respiratory distress, about 10% of cases remain asymptomatic and are diagnosed

incidentally in adulthood (Taylor et al., 2009; Pober et al., 2007). BH accounts for 95% of adult CDH cases. The estimated prevalence in the general population ranges between 0.17–12.7% (Kinoshita et al., 2009; Mullins et al., 2001)

Due to the early development of the right hemidiaphragm and the liver's barrier effect, BH occurs predominantly on the left side (70–90%) **(**Eren et al., 2005; Gale, 1985; Kinoshita et al., 2009; Taylor et al., 2009). However, Mullins et al. reported rates of 18% left-sided, 68% right-sided, and 14% bilateral (Mullins et al., 2001).

Adults are often asymptomatic; when present, symptoms relate to respiratory or gastrointestinal systems, such as dyspnea, chest pain, recurrent pulmonary infections, abdominal pain, anorexia, nausea, vomiting, and diarrhea. Complications may include intestinal obstruction, strangulation, perforation, gastric volvulus, and splenic rupture (Mullins et al., 2001; Robb et al., 2006; Sakorafas et al., 2001).

2.2. Morgagni hernia

Morgagni hernia (MH) involves herniation of abdominal contents through an anterior defect between the pars costalis and pars sternalis, typically at the cardiophrenic angle (Larrey's space). Giovanni Battista Morgagni first identified this condition in 1769, while Larrey described a surgical route to the pericardium via this space in 1828,

giving rise to the terms Larrey's space and Morgagni hernia (Döner, 2013; Nasr et al., 2009).

MH comprises 1–5% of adult CDH cases. Risk factors include pregnancy, trauma, obesity, chronic constipation, and chronic cough (Contreras et al., 2024; Khandelwal et al., 2011; Nasr et al., 2009; Schumacher et al., 2009). Due to the presence of the pericardial sac on the left side, MH is seen predominantly on the right side (90%) (Nasr et al., 2009).

Unlike BH, a peritoneal sac (true hernia) is always present in MH, commonly containing omentum, but may also involve the colon, stomach, or small intestines (Contreras et al., 2024). Patients are typically asymptomatic until adulthood. When symptomatic, complaints include mild to severe substernal pain, dyspnea, gastroesophageal reflux, dysphagia, and recurrent constipation. Diagnosis often occurs during routine imaging or emergently due to strangulation or volvulus (Bragg et al., 1996; Swain et al., 2001).

3. Diagnosis

On posteroanterior chest radiographs, suspicion arises with the presence of air-fluid levels suggesting stomach or intestinal loops in the thorax. Lateral chest X-rays aid in identifying BH. Other imaging modalities include barium studies, fluoroscopy, computed tomography (CT), and magnetic resonance imaging (MRI). Differential diagnosis should include anterior mediastinal masses, lipoma, right middle lobe

collapse, pericardial fat pad, and pulmonary sequestration (Habib et al., 2002; Köse et al., 2024).

In MH, air-fluid levels adjacent to the heart on PA view and retrosternal positioning on lateral view are characteristic (Kilic et al., 2001). Thorax CT imaging showing discontinuity of the diaphragm helps differentiate CDH from diaphragmatic eventration (Panicek et al., 1988).

4. Treatment

Surgery is recommended for patients diagnosed with CDH because of the risk of strangulation, perforation and death. The aim of surgery is to reduce the organs herniated into the thorax to the abdomen, treat strangulation/perforation if present, and close the defect in the diaphragm (Gomes-Da Silva de Rosenzweig et al., 2024; Kilic et al., 2001).

There is no definitive method reported for the surgical approach. There are approaches using thoracic, abdominal or both. Thoracotomy, video-assisted thoracic surgery (VATS), laparotomy or laparoscopy, robotic surgery are the methods used. The surgical approach method can be determined according to the condition of the patient and hernia and the experience of the surgeon. (Gomes-Da Silva de Rosenzweig et al., 2024; Horton et al., 2008; Kilic et al., 2001).

In right-sided hernias, the thoracic or abdominal approach can be used according to the surgeon's preference. However, since advanced adhesions may develop on the left side, the thoracic approach is more appropriate. Since the hernia sac may be adherent to the mediastinum, pleura and lung, the hernia sac can be seen, dissected and the abdominal organs can be reduced to the abdomen more easily with the transthoracic approach (de Oliveira et al., 1984; Schumacher et al., 2009; Yamaguchi et al., 2002). Some authors advocate the abdominal approach because it allows the herniated organs to be pulled into the abdomen, a good position can be given in the abdomen and repair can be performed in the presence of any strangulation (Ohtsuka et al., 2017; Ulas et al., 2020)

Nowadays, with the development of minimally invasive surgical techniques, the preference for endoscopic surgery is increasing. Wide exposure, reduced pain, shorter hospital stay and better cosmetic appearance are stated as advantages of endoscopic methods. Thoracoscopic approach also provides better visualization of the hernia sac and allows safe dissection of possible pericardial and pleural adhesions (Aljuhani et al., 2025; Aydin et al., 2014; Sanford et al., 2018; Schumacher et al., 2009; Young et al., 2019). With the widespread use of robotic surgery, its use in CDH repair has been reported and its advantages such as less morbidity and wide field of vision have been stated (Jambhekar et al., 2018; Huang et al.,2024).

Whether to excise the hernia sac remains controversial; to avoid phrenic nerve injury, some advocate leaving the sac in place (Kilic et al., 2001).

Repair of diaphragmatic defects varies according to the size of the defect. Primary closure method is used for small defects up to 10 cm2. In larger defects, the use of grafts is recommended to reduce tension (Gomes-Da Silva de Rosenzweig et al., 2024; Mardani et al., 2024; Pfannschmidt et al., 2004). Polytetrafluoroethylene graft is the most commonly used graft type because it does not erode the gastrointestinal organs and has less adhesion formation (Toydemir et al., 2012).

Outcomes are excellent across all approaches. (Schumacher et al., 2009; Aydin et al., 2014). Recurrence was reported in only 1 case in a large case series (Horton et al., 2008).

5. Conclusion

Most patients with CDH are asymptomatic or present with respiratory or gastrointestinal symptoms. Due to the potential for strangulation or incarceration, surgical treatment yields excellent results and is recommended.

REFERENCES

Al-Emadi, M., Helmy, I., Nada, M. A., & Al-Jaber, H. (1999). Laparoscopic repair of Bochdalek hernia in an adult. *Surgical Laparoscopy, Endoscopy & Percutaneous Techniques, 9*(6), 423–425.

Aljuhani, A., Alsumaili, A. A., Alyaseen, E. M., Daak, L. I., Esmail, A., Alzohari, J. E., Alqahtani, A., Junaidallah, E. A., Alghamdi, H. A., & Saeedi, F. (2025). Minimally invasive approach versus traditional approach for treating congenital diaphragmatic hernia: A systematic review and meta-analysis. *Cureus, 17*(1), e77596. https://doi.org/10.7759/cureus.77596

Aydin, Y., Altuntas, B., Ulas, A. B., Daharli, C., & Eroglu, A. (2014). Morgagni hernia: Transabdominal or transthoracic approach? *Acta Chirurgica Belgica, 114*(2), 131–135.

Bragg, W. D., Bumpers, H., & Flynn, W. (1996). Morgagni hernias: A common cause of chest masses in adults. *American Family Physician, 54*(6), 2021–2024.

Contreras, N., Varghese, T. K. Jr, & Mitzman, B. (2024). Management of congenital hernias in adults: Foramen of Morgagni hernia. *Thoracic Surgery Clinics, 34*(2), 147–154.

De Oliveira, F., & Oliveira, F. J. (1984). Congenital posterolateral diaphragmatic hernia in the adult. *Canadian Journal of Surgery, 27*(6), 610–611.

Döner, E. (2013). Morgagni hernisi. In İ. Ökten & H. Ş. Kavukçu (Eds.), *Göğüs cerrahisi* (2. baskı, ss. 1481–1485). İstanbul Tıp Kitabevi.

Eren, S., & Ciriş, F. (2005). Diaphragmatic hernia: Diagnostic approaches with review of the literature. *European Journal of Radiology, 54*(3), 448–459.

Gale, M. E. (1985). Bochdalek hernia: Prevalence and CT characteristics. *Radiology, 156*(2), 449–452.

Goh, B. K., Teo, M. C., Chng, S. P., & Soo, K. C. (2007). Right-sided Bochdalek's hernia in an adult. *American Journal of Surgery, 194*(3), 390–391.

Gomes-da Silva de Rosenzweig, P., Vázquez-Minero, J. C., Delgado-Casillas, O. M., et al. (2024). Diaphragmatic hernia repair in adult patients: A retrospective institutional experience. *Cureus, 16*(11), e74601. https://doi.org/10.7759/cureus.74601

Habib, E., Bellaiche, G., & Elhadad, A. (2002). Complications of misdiagnosed Bochdalek hernia in adults: Literature review. *Annales de Chirurgie, 127*(3), 208–214.

Horton, J. D., Hofmann, L. J., & Hetz, S. P. (2008). Presentation and management of Morgagni hernias in adults: A review of 298 cases. *Surgical Endoscopy, 22*(6), 1413–1420.

Huang, Y.-J., & Fang, Y.-L. (2024). Robot-assisted congenital diaphragmatic hernia repair in adults: A case series. *Medicine, 103*(43), e39918.

Jambhekar, A., Robinson, S., Housman, B., et al. (2018). Robotic repair of a right-sided Bochdalek hernia: A case report and literature review. *Journal of Robotic Surgery, 12*(2), 351–355.

Khandelwal, S., & Oelschlager, B. K. (2011). Video laparoscopic repair of congenital bilateral Morgagni hernia. *Surgical Endoscopy, 25*(6), 2010. https://doi.org/10.1007/s00464-010-1337-y

Kilic, D., Nadir, A., & Döner, E. (2001). Transthoracic approach in surgical management of Morgagni hernia. *European Journal of Cardio-Thoracic Surgery, 20*(5), 1016–1019.

Kinoshita, F., Ishiyama, M., Honda, S., et al. (2009). Late-presenting posterior transdiaphragmatic (Bochdalek) hernia in adults: Prevalence and MDCT characteristics. *Journal of Thoracic Imaging, 24*(1), 17–22.

Köse, E. E., & Yalçınkaya, İ. (2024). Congenital diaphragmatic hernia. *Turkish Journal of Thoracic and Cardiovascular Surgery, 32*(1), 89–97. https://doi.org/10.5606/tgkdc.dergisi.2024.25705

Mardani, P., Dalfardi, F., Bahmani, S., Rahmati, R., & Zarimeidani, F. (2024). Clinical presentations and surgical features of Morgagni hernia in adults: A retrospective study. *Health Science Reports, 7*, e70248.

McGivern, M., Best, K. E., Rankin, J., et al. (2015). Epidemiology of congenital diaphragmatic hernia in Europe: A register-based study. *Archives of Disease in Childhood - Fetal and Neonatal Edition, 100*(2), 137–144.

Mullins, M. E., Stein, J., Saini, S. S., & Mueller, P. R. (2001). Prevalence of incidental Bochdalek's hernia in a large adult population. *AJR American Journal of Roentgenology, 177*(2), 363–366.

Nasr, A., & Fecteau, A. (2009). Foramen of Morgagni hernia: Presentation and treatment. *Thoracic Surgery Clinics, 19*(4), 463–468.

Ohtsuka, Y., & Suzuki, T. H. (2017). Right-sided Bochdalek hernia in an elderly patient: A case review of adult Bochdalek hernias from 1982 to 2015 in Japan. *Acute Medicine & Surgery, 4*(2), 209–212. https://doi.org/10.1002/ams2.249

Panicek, D. M., Benson, C. B., Gottlieb, R. H., & Heitzman, E. R. (1988). The diaphragm: Anatomic, pathologic, and radiologic considerations. *Radiographics, 8*(3), 385–425.

Pfannschmidt, J., Hoffmann, H., & Dienemann, H. (2004). Morgagni hernia in adults: Results in 7 patients. *Scandinavian Journal of Surgery, 93*(1), 77–81.

Pober, B. R. (2007). Overview of epidemiology, genetics, birth defects, and chromosome abnormalities associated with CDH. *American Journal of Medical Genetics Part C: Seminars in Medical Genetics, 145C*(2), 158–171.

Robb, B. W., & Reed, M. F. (2006). Congenital diaphragmatic hernia presenting as splenic rupture in an adult. *Annals of Thoracic Surgery, 81*(3), e9–e10.

Sakorafas, G. H., & Delibasis, G. (2001). Large right congenital diaphragmatic (Bochdalek) hernia: An incidental finding in an

aged patient with acute intestinal obstruction. *Digestive Surgery, 18*(6), 431. https://doi.org/10.1159/000050188

Sanford, Z., Weltz, A. S., Brown, J., Shockcor, N., Wu, N., & Park, A. E. (2018). Morgagni hernia repair: A review. *Surgical Innovation, 25*(4), 389–399.

Schumacher, L., & Gilbert, S. (2009). Congenital diaphragmatic hernia in the adult. *Thoracic Surgery Clinics, 19*(4), 469–472. https://doi.org/10.1016/j.thorsurg.2009.08.004

Swain, J. M., Klaus, A., & Achen, S. R. (2001). Congenital diaphragmatic hernia in adults. *Seminars in Laparoscopic Surgery, 8*(4), 246–255.

Taylor, G. A., Atalabi, O. M., & Estroff, J. A. (2009). Imaging of congenital diaphragmatic hernias. *Pediatric Radiology, 39*(1), 1–16.

Toydemir, T., Akıncı, H., Tekinel, M., et al. (2012). Laparoscopic repair of an incarcerated Bochdalek hernia in an elderly man. *Clinics (São Paulo), 67*(2), 199–201.

Ulaş, A. B., Aydın, Y., & Eroğlu, A. (2020). Laparoscopic approach in the treatment of Morgagni hernia. *Turkish Journal of Thoracic and Cardiovascular Surgery, 28*(3), 514–520. https://doi.org/10.5606/tgkdc.dergisi.2020.19209

Yamaguchi, M., Kuwano, H., Hashizume, M., et al. (2002). Thoracoscopic treatment of Bochdalek hernia in the adult: Report of a case. *Annals of Thoracic and Cardiovascular Surgery, 8*(2), 106–108.

Young, M. C., Saddoughi, S. A., Aho, J. M., et al. (2019). Comparison of laparoscopic versus open surgical management of Morgagni hernia. *Annals of Thoracic Surgery, 107*(1), 257–261. https://doi.org/10.1016/j.athoracsur.2018.08.021

CHAPTER 3

ACQUIRED DIAPHRAGMATIC DISEASES

Assoc. Prof. Dr. Guntug BATIHAN

INTRODUCTION

Acquired diaphragmatic diseases encompass a diverse spectrum of pathological conditions that affect the diaphragm after birth, in contrast to congenital anomalies (Hanna et al., 2009). These disorders may result from trauma, surgery, neoplasms, infections, or neurologic and inflammatory processes, and they often present with nonspecific respiratory or gastrointestinal symptoms (Anraku et al., 2009). The diaphragm, as the primary muscle of respiration, plays a pivotal role in pulmonary mechanics and intra-abdominal pressure regulation. Consequently, any acquired dysfunction can lead to significant clinical consequences, ranging from asymptomatic incidental findings to life-threatening respiratory compromise or visceral herniation. Understanding the etiology, pathophysiology, and diagnostic strategies related to acquired diaphragmatic diseases is crucial for timely intervention and optimal patient outcomes.

1. Traumatic diaphragmatic injuries

Traumatic diaphragmatic injuries (TDI) are often overlooked yet critically important conditions in thoracoabdominal trauma. The diaphragm, serving as both the primary muscle of respiration and an anatomical barrier between the thoracic and abdominal cavities, is susceptible to significant functional compromise following trauma (Abdellatif et al., 2020). Both penetrating and blunt mechanisms can cause diaphragmatic rupture, leading to herniation of abdominal organs into the thoracic cavity, respiratory dysfunction, and hemodynamic instability. Prompt diagnosis and effective surgical intervention are essential and often lifesaving, making TDI a vital concern in the practice of thoracic surgeons (Grimes et al., 1974).

1.1. Epidemiology and injury pattern

TDI occur in 0.8% to 5% of all thoracoabdominal traumas. The incidence may reach up to 15% in penetrating injuries, while blunt trauma accounts for approximately 0.5–1.8% of cases (Lim et al., 2017). Injuries most commonly involve the left hemidiaphragm at the level of the 3rd to 5th intercostal space. Right-sided injuries are less frequent due to the protective effect of the liver and are more prone to delayed diagnosis (Shackleton et al., 1998).

1.2. Mechanisms of injury: blunt and penetrating diaphragmatic trauma

Blunt and penetrating traumas lead to diaphragmatic injuries through distinct pathophysiological mechanisms, both of which require different diagnostic considerations and surgical approaches.

1.2.1. Blunt trauma

In blunt thoracoabdominal trauma, typically caused by high-energy mechanisms such as motor vehicle collisions, falls from significant heights, or crush injuries, a sudden and severe increase in intra-abdominal pressure occurs. This rapid pressurization acts against a fixed and relatively thin muscular diaphragm, particularly its posterolateral aspect, which is structurally more vulnerable due to its embryological fusion lines (the pleuroperitoneal canals). The resultant force surpasses the tensile strength of the diaphragm, causing it to rupture.

These ruptures are often large ranging from 5 to 10 centimeters and usually occur on the left hemidiaphragm due to the cushioning effect of the liver on the right side. The injury may initially go unnoticed, especially when overshadowed by more apparent injuries such as long bone fractures, pelvic trauma, or head injuries. Moreover, in supine trauma patients, herniated abdominal contents may spontaneously reduce, further delaying the diagnosis.

Diaphragmatic tears in blunt trauma are frequently associated with multiple injuries, including splenic rupture, rib fractures, pulmonary contusions, or spinal trauma. The presence of such polytrauma necessitates a high index of suspicion and a systematic diagnostic approach. Importantly, delayed diagnosis may lead to progressive herniation of abdominal organs into the thoracic cavity, resulting in respiratory compromise or strangulation.

1.2.2. Penetrating trauma

Penetrating injuries to the diaphragm are typically caused by stabbing, gunshot wounds, or impalement. Unlike blunt trauma, the injury is the result of direct mechanical disruption of the diaphragmatic musculature by a foreign object. These injuries may appear deceptively minor externally, but they often involve both thoracic and abdominal structures due to the diaphragm's location as a transitional zone.

Penetrating trauma creates a defect whose size depends on the object’s trajectory, velocity, and angle of entry. The injury path may violate pleural, pericardial, hepatic, gastric, or colonic structures, often leading to complex clinical presentations. Additionally, diaphragmatic wounds from penetrating trauma can function as one-way valves, facilitating herniation during inspiration while trapping abdominal contents in the thorax, increasing the risk of ischemia.

The side of injury also influences clinical presentation and management. Left-sided injuries more readily result in herniation due

to the absence of a solid organ buffer, whereas right-sided injuries may be masked by the presence of the liver, leading to delayed complications like biliothorax or subphrenic abscesses.

Given the potential for occult injury and delayed herniation, even small penetrating wounds in the thoracoabdominal transition zone should prompt diagnostic laparoscopy or thoracoscopy when imaging is inconclusive, especially if the trajectory suggests diaphragmatic violation.

1.3. Clinical presentation

TDI present with a wide spectrum of symptoms, ranging from asymptomatic to life-threatening respiratory or circulatory compromise. Auscultation of bowel sounds in the chest is considered pathognomonic, while dyspnea, hypoxemia, and abdominal pain may be attributed to herniated abdominal organs compressing thoracic structures. Delayed presentations are common, particularly for right-sided injuries, which are often masked by the liver (Rashid et al., 2017).

Early diagnosis remains challenging due to nonspecific signs and concurrent injuries. Initial chest radiographs may show indirect clues but have limited sensitivity. Contrast-enhanced computed tomography (CT) with multiplanar reconstructions is the imaging modality of choice, with findings such as the “collar sign” and “dependent viscera sign” aiding diagnosis (Tiberio et al., 2005). Focused Assessment with

Sonography for Trauma (FAST) can assist in detecting associated thoracic injuries but has limited utility for direct diaphragm evaluation.

In hemodynamically stable patients with penetrating trauma or equivocal imaging, diagnostic laparoscopy or thoracoscopy allows for both confirmation and immediate repair, enhancing both detection and management outcomes.

1.4. Surgical management

Once a traumatic diaphragmatic injury is confirmed, surgical repair becomes imperative, as conservative management carries a high risk of herniation, strangulation, and long-term complications. The choice of surgical approach depends on several factors, including the mechanism and timing of injury, the patient's hemodynamic stability, and the presence of other associated injuries.

In the acute setting, patients who are hemodynamically stable may undergo laparoscopy, particularly when the injury is suspected to be confined to the abdominal side. Laparoscopy allows for adequate inspection of the diaphragm and can be therapeutic in selected cases. However, in patients with multiple injuries or those who are unstable, an open exploratory laparotomy is the preferred approach, as it allows full access to evaluate and address concurrent intra-abdominal injuries.

The diaphragmatic tear is typically repaired using interrupted or running non-absorbable sutures such as polypropylene or polyester.

Care must be taken to achieve a tension-free closure, and in large or posterior defects where tissue integrity is compromised, reinforcement with mesh may be necessary. Double-layer closure may be applied in high-tension repairs or when delayed healing is anticipated.

In cases where the diagnosis is delayed, often presenting with chronic herniation of abdominal organs into the thoracic cavity, a thoracic approach may be favored. Thoracotomy or video-assisted thoracoscopic surgery (VATS) provides better access for adhesiolysis and safe reduction of the herniated viscera. Adhesions between herniated organs and the pleura or lung parenchyma may be dense and fibrotic, increasing the risk of inadvertent injury. The diaphragmatic defect is then closed primarily if feasible, or with the aid of mesh in cases of large tissue loss. In contaminated surgical fields, such as those involving strangulated bowel or purulent effusion, the use of biological mesh is preferred to reduce the risk of infection and fistula formation.

Ultimately, meticulous surgical technique, appropriate approach selection, and intraoperative vigilance are paramount in reducing the risk of recurrence, postoperative complications, and mortality associated with TDI. In Figures 1 and 2, intraoperative images of diaphragmatic repair performed via open and minimally invasive surgery are presented.

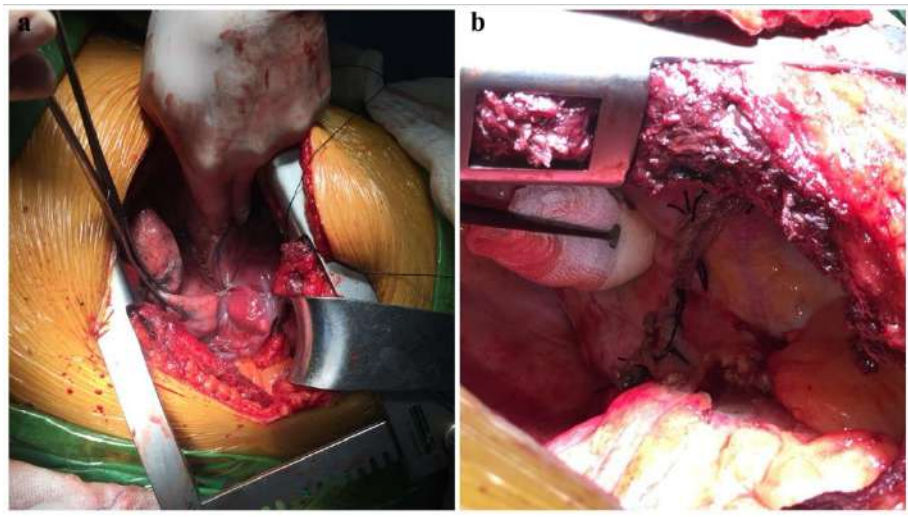


Figure 1. Diaphragmatic laceration repair via thoracotomy approach (a) is shown. The lacerated area of the diaphragm was sutured using separate non-absorbable mattress sutures (b).

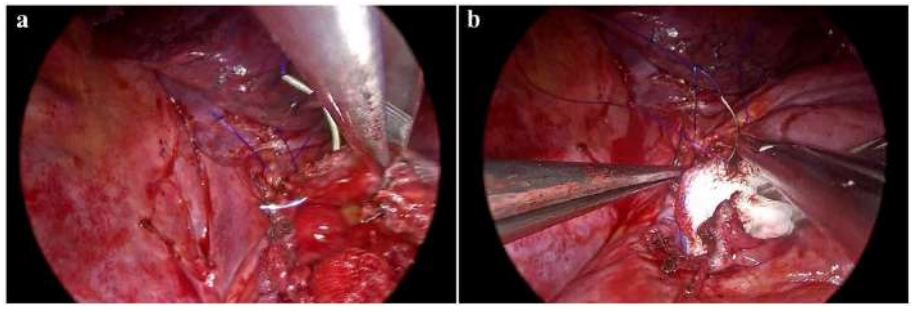


Figure 2. In Figures 2a and 2b, videothoracoscopic repair of a diaphragmatic laceration is demonstrated. As shown in Figure 2b, intra-abdominal organs were retracted during suturing to prevent injury.

Intraoperative images of diaphragmatic repairs performed with open and minimally invasive surgery are shown in Figure 1 and Figure 2.

1.5. Complications and long-term follow-up

Traumatic diaphragmatic injuries, if not promptly and adequately treated, can lead to significant early and late complications, many of which are potentially life-threatening. One of the most serious and time-sensitive complications is the strangulation and subsequent necrosis of herniated abdominal organs. When loops of bowel, stomach, or even solid organs herniate into the thoracic cavity through a diaphragmatic defect, they may become compressed at the diaphragmatic hiatus, compromising their vascular supply. This can rapidly progress to ischemia, necrosis, and even perforation, resulting in peritonitis or empyema if contamination extends into the pleural space.

Respiratory complications are also common, particularly in the early postoperative period. These may include respiratory insufficiency due to lung compression or failure of re-expansion, as well as atelectasis and pneumonia. These risks are particularly elevated in cases with delayed presentation or large herniations, where prolonged lung collapse has occurred.

Infectious complications such as empyema, infected pleural effusions, or subphrenic abscesses may arise either from intraoperative contamination or secondary to bowel perforation. The presence of necrotic tissue, retained hemothorax, or mesh prostheses further

increases the risk of such infections and may necessitate re-intervention, prolonged antibiotic therapy, or drainage procedures.

Recurrence of diaphragmatic herniation is another long-term concern and is typically attributed to technical failure during initial repair or to insufficient suture strength in the setting of increased intra-abdominal pressure. Proper surgical technique including the use of non-absorbable sutures, adequate tissue overlap, and reinforcement with mesh in appropriate cases can significantly reduce recurrence rates.

Lastly, wound complications such as surgical site infection or dehiscence can compromise healing and predispose to secondary herniation. These are more common in contaminated fields, obese patients, or in those with poor nutritional or immunologic status.

Close follow-up with imaging and clinical evaluation is essential in the postoperative period, especially for patients who underwent delayed repair or had complicated intraoperative findings. Preventive measures, including pulmonary rehabilitation, infection control, and nutritional optimization, play an important role in improving long-term outcomes.

1.6. Differential diagnosis

Several clinical conditions may mimic the radiological or clinical presentation of traumatic diaphragmatic injuries, particularly in the acute trauma setting. Differentiating between these entities is essential to avoid misdiagnosis and to guide appropriate management strategies.

One important differential is diaphragmatic eventration**,** a congenital or acquired condition characterized by an abnormal elevation of the intact hemidiaphragm due to muscular weakness or paralysis. Unlike traumatic rupture, eventration maintains continuity of the diaphragmatic contour and lacks associated herniation of abdominal organs, although imaging may sometimes raise suspicion for TDI in cases of marked elevation.

Hiatal hernia is another consideration, particularly when stomach or bowel loops are visualized within the thoracic cavity. This condition typically involves herniation through the esophageal hiatus and is more common on the left side. However, unlike post-traumatic herniation, hiatal hernias are usually chronic, often associated with gastroesophageal reflux symptoms, and have characteristic radiographic findings.

Pneumothorax or pneumoperitoneum may also confound the diagnosis, especially when free air is detected on imaging. In pneumothorax, the lung margin is displaced centrally and the diaphragm remains intact, whereas pneumoperitoneum may cause diaphragmatic elevation but does not involve disruption of the diaphragmatic musculature. Both conditions can coexist with TDI, further complicating the diagnostic picture.

Lastly, pleural effusion may obscure diaphragmatic contours on imaging and mimic or mask signs of herniation. In trauma patients, the presence of hemothorax or effusion should prompt further

investigation, particularly if diaphragmatic injury is clinically suspected.

Recognition of these differential diagnoses, supported by high-resolution imaging and, when necessary, direct visualization via laparoscopy or thoracoscopy, is crucial for timely and accurate identification of true diaphragmatic injury.

1.7. Conclusion

Traumatic diaphragmatic injuries are diagnostically elusive but surgically correctable conditions. Thoracic surgeons must maintain a high index of suspicion, interpret advanced imaging appropriately, and select optimal surgical approaches tailored to the individual patient. Early intervention remains key to reducing morbidity and mortality.

2. Acquired diaphragmatic hernias

Acquired diaphragmatic hernias (ADH) represent a spectrum of diaphragmatic defects that develop secondary to non-congenital causes, including trauma, surgical interventions, or idiopathic processes (Johnson et al., 1988). Unlike congenital diaphragmatic hernias, which are detected early in life, acquired forms may present in any age group and often pose diagnostic and therapeutic challenges due to their insidious onset and variable clinical manifestations. Thoracic surgeons must be adept at recognizing the etiology, presentation, and appropriate management strategies for these entities.

2.1. Classification and etiology

ADH can be classified into three major etiological categories: traumatic, iatrogenic, and spontaneous. Each type has a distinct pathophysiological mechanism, clinical trajectory, and surgical implication. Understanding these distinctions is essential for accurate diagnosis and appropriate management.

2.1.1. Traumatic diaphragmatic hernias (TDH)

TDH result from physical disruption of the diaphragm due to blunt or penetrating trauma, as discussed extensively in the preceding sections. While many injuries are diagnosed acutely during initial trauma care, a significant proportion remain clinically silent and are only detected months or even years later, typically when herniation of abdominal contents becomes symptomatic. The posterolateral region of the left hemidiaphragm is most commonly affected due to its relative lack of protection compared to the right, which is shielded by the liver. Over time, small unrecognized diaphragmatic defects can enlarge under the influence of negative intrathoracic pressure and repeated increases in intra-abdominal pressure, eventually allowing visceral herniation. These delayed presentations often mimic spontaneous hernias and may be complicated by dense adhesions and organ entrapment, necessitating more complex surgical repair.

2.1.2. Iatrogenic diaphragmatic hernias (IDH)

IDH are increasingly recognized in the era of complex thoracoabdominal and oncologic surgeries. These hernias typically arise from inadvertent injury to the diaphragm or from intentional diaphragmatic incisions that were inadequately closed or subjected to increased tension postoperatively. Common precipitating procedures include liver resections (particularly right hepatectomy), esophagectomy with diaphragmatic hiatus mobilization, splenectomy, gastrectomy, adrenalectomy, and diaphragmatic stripping during peritonectomy. Laparoscopic and robotic approaches, while minimally invasive, may contribute to under-visualization and missed injuries. IDH may present as early postoperative complications with respiratory distress or bowel obstruction, or as delayed findings with chronic chest discomfort, dyspnea, or gastrointestinal symptoms. Prompt recognition is critical, as delayed diagnosis may lead to strangulation of intrathoracic bowel. Surgical repair should be tension-free, and mesh reinforcement is often required, particularly in reoperative fields or in the presence of large defects.

2.1.3. Spontaneous diaphragmatic hernias (SDH)

SDH are rare and defined by the absence of prior trauma or surgical insult. The pathogenesis remains incompletely understood but is thought to involve a combination of inherent diaphragmatic weakness and sudden elevation in intra-abdominal pressure (Figure 3. and 4). Risk factors include episodes of violent coughing, parturition,

defecation, vomiting, or lifting heavy objects. Structural conditions such as collagen vascular diseases (e.g., Ehlers-Danlos syndrome, Marfan syndrome) may predispose to muscular dehiscence. SDH frequently present acutely with chest pain, dyspnea, or signs of bowel obstruction and may be mistaken for other intrathoracic emergencies such as pneumothorax or pulmonary embolism. In many cases, diagnosis is established intraoperatively during exploratory laparotomy or thoracotomy (Tapias et al., 2009). The hernia defect often involves the central tendon or posteromedial muscle bundles and may require mesh repair depending on size and tissue quality.

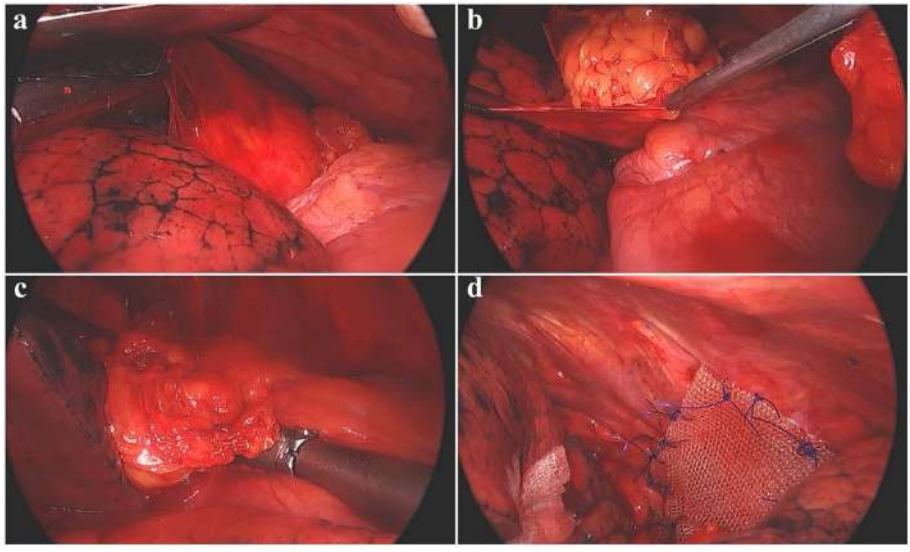


Figure 3. A herniation originating from the foramen of Morgagni was observed. Review of the patient's previous imaging revealed a minimal herniation that had spontaneously progressed over time. Through a right videothoracoscopic approach, the hernia sac located in the anteromedial

area was opened (a), and the omentum along with intra-abdominal organs was mobilized (b). The portion of the omentum that could not be reduced into the abdominal cavity was carefully partially resected (c). Subsequently, the diaphragmatic defect was sutured and reinforced with a polypropylene mesh (d).

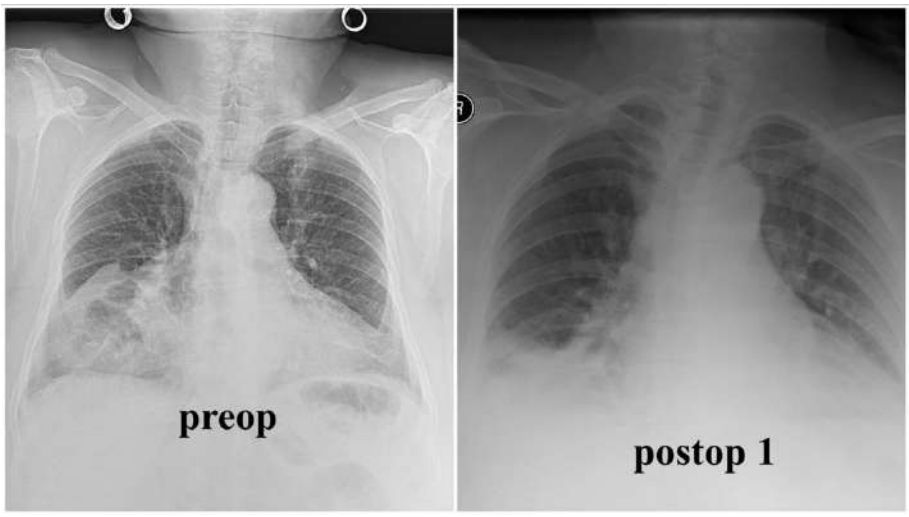


Figure 4. Preoperative and postoperative chest X-rays of the patient whose operative images are presented above are shown.

The pathogenesis is not yet fully understood, but it is thought to be a combination of congenital diaphragmatic weakness and a sudden increase in intra-abdominal pressure. Representative surgical and radiologic images of a spontaneous diaphragmatic hernia are shown in Figure 3 and Figure 4.

2.2. Clinical presentation

ADHs present with variable symptoms depending on defect size, herniated organ type, and presence of complications. Many patients remain asymptomatic, while others may experience nonspecific symptoms such as chest discomfort, exertional dyspnea, or postprandial bloating. Larger or fixed hernias can cause abdominal pain, vomiting, or respiratory symptoms like cough, orthopnea, and reduced breath sounds.

Acute presentations due to strangulation or incarceration may lead to severe pain, bowel obstruction, or systemic signs like sepsis. In such cases, emergent surgery is often required. Delayed diagnosis is common, especially in iatrogenic and spontaneous cases. Therefore, any new respiratory or gastrointestinal symptom following thoracoabdominal surgery should prompt evaluation for ADH.

2.3. Diagnosis

Accurate diagnosis of ADHs requires a high index of suspicion and a multimodal imaging approach, especially due to their often subtle and variable presentations. Imaging plays a critical role not only in detecting the presence of a hernia but also in determining the contents, assessing for complications (e.g., strangulation or perforation), and guiding surgical planning (Testini et al., 2017; Lamture et al., 2024).

Chest Radiography is frequently the first-line imaging modality, especially in emergency settings. Although its sensitivity is limited, it can provide early diagnostic clues. Typical findings include elevation or blurring of the hemidiaphragm, the presence of gas-filled bowel loops in the thoracic cavity, mediastinal shift, or air-fluid levels within the hemithorax. In patients with a nasogastric tube in place, upward migration of the tube into the thorax can be a helpful sign of gastric herniation. Right-sided hernias may be more difficult to detect due to the radiodensity of the liver masking herniated structures.

Contrast-enhanced CT with multiplanar reconstruction (MPR) is considered the gold standard for diagnosis in stable patients. CT provides direct visualization of the diaphragmatic defect, herniated contents, and associated thoracic or abdominal pathology.

Upper gastrointestinal (GI) series or fluoroscopy may be beneficial in patients with suspected intermittent or functional hernias, particularly those involving the stomach or small bowel. This modality allows real-time observation of organ movement across the diaphragm during respiratory cycles or positional changes and can help in cases where CT findings are inconclusive.

MRI is not routinely used in the emergency setting but may be helpful in select cases, especially for distinguishing soft tissue structures or evaluating complex postoperative anatomy. It is particularly advantageous in patients for whom radiation exposure is a concern, such as pregnant women or children.

Ultrasound, including diaphragm-specific ultrasonography or Extended -FAST in trauma settings, has limited sensitivity for chronic hernias but may occasionally detect abnormal diaphragmatic motion, discontinuity, or thoracoabdominal organ displacement. It remains more valuable in the acute trauma setting or when used by experienced operators.

Finally, diagnostic laparoscopy or thoracoscopy may be necessary in ambiguous or borderline cases. These minimally invasive approaches provide direct visualization of the diaphragmatic surface and permit immediate repair if a defect is identified. They are especially useful in post-surgical patients with atypical symptoms and inconclusive imaging.

In summary, while chest X-rays may provide initial suspicion, contrast-enhanced CT with multiplanar reconstructions remains the cornerstone for definitive diagnosis, with adjunctive modalities applied based on clinical scenario and anatomical complexity.

2.4. Surgical management

Surgical intervention remains the definitive treatment for ADHs and is generally indicated in all patients, regardless of symptom severity, due to the unpredictable risk of visceral incarceration, strangulation, and subsequent ischemic complications. Even in incidentally discovered asymptomatic hernias, elective repair is recommended to avoid emergency presentations with higher morbidity (Petrone et al., 2007).

The choice of surgical approach is influenced by multiple factors including the timing of diagnosis (acute vs. chronic), hernia size, location, contents, presence of adhesions, and whether the setting is elective or emergent. The surgeon's expertise and institutional resources (e.g., availability of advanced minimally invasive platforms) also play a significant role (Reber et al., 1998).

2.4.1. Laparoscopic approach

In cases where the hernia is diagnosed early or when the defect is accessible from the abdominal side such as in iatrogenic or anterior hernias laparoscopy offers an excellent minimally invasive option. It allows complete reduction of herniated contents, assessment of visceral viability, and primary repair of the diaphragmatic defect. Laparoscopy also permits simultaneous inspection and repair of intra-abdominal injuries if present. The technique is particularly advantageous in patients with minimal adhesions and no prior upper abdominal surgery.

2.4.2. VATS approach

VATS has emerged as a preferred modality in select cases, especially in chronic diaphragmatic hernias with dense intrathoracic adhesions or in post-traumatic defects that present late. VATS allows excellent visualization of the thoracic cavity, facilitates safe and controlled reduction of herniated viscera, and enables meticulous dissection of fibrous adhesions to lung, pleura, or pericardium. It also provides better

access to posterior and lateral defects, which may be difficult to approach transabdominally (Figure 5-7).

In chronic TDH and long-standing SDH, where herniated contents may be fixed within the thoracic cavity, VATS offers the advantage of reduced surgical trauma and faster recovery compared to thoracotomy. However, dense adhesions, loss of domain, or large incarcerated organs may necessitate conversion to open thoracotomy, and the surgical team should be prepared for such a contingency.

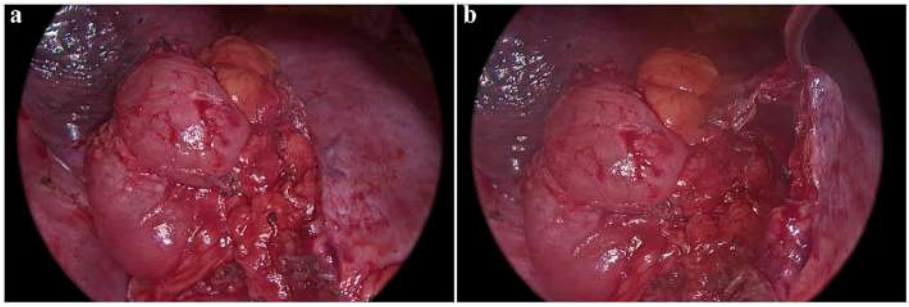


Figure 5. In Figure 5a, a diaphragmatic hernia that developed in the late postoperative period following prior intra-abdominal surgery is observed. In Figure 5b, the herniated abdominal organs have been mobilized and are prepared for reduction into the abdominal cavity.

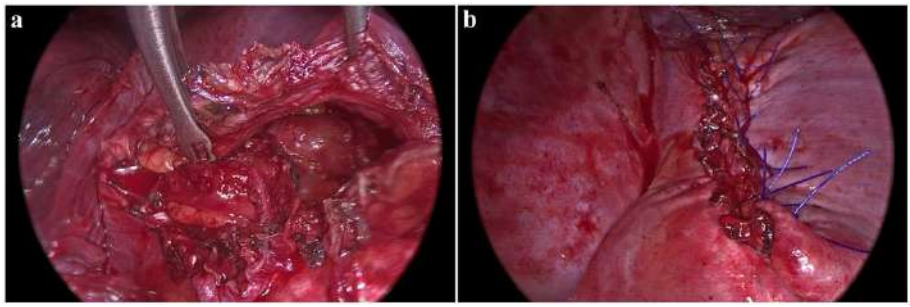


Figure 6. In Figure 6a, the herniated abdominal organs have been reduced below the diaphragm, and in Figure 6b, the diaphragmatic defect is shown after being sutured.

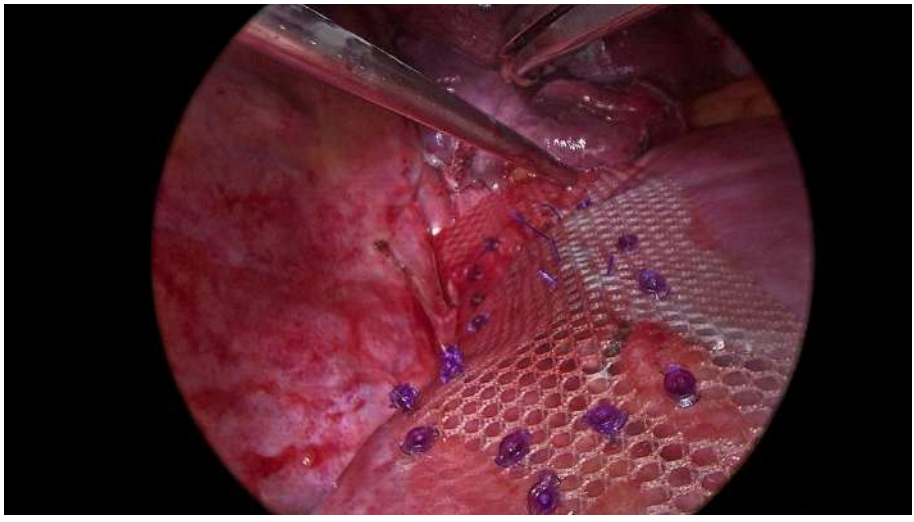


Figure 7. To reinforce the suture line and reduce the risk of recurrent herniation, the diaphragmatic suture line can be supported with a mesh.

Figures 5 to 7 show a representative case of a chronic post-traumatic diaphragmatic hernia treated with the VATS procedure. The images show the successive steps of hernia identification, reduction of the abdominal contents, closure of the defect and mesh reinforcement.

2.4.3. Open repair

Laparotomy or thoracotomy remains necessary in certain settings, particularly in hemodynamically unstable patients, complex reoperative cases, or when massive herniation and organ ischemia are suspected. The thoracic approach (via posterolateral thoracotomy) is typically preferred in chronic presentations with dense adhesions or when VATS is not feasible. For massive bilateral hernias or where both abdominal and thoracic access is required, combined thoracoabdominal incisions may be considered.

2.4.4. Repair technique

The fundamental principle is closure of the diaphragmatic defect without tension. Small defects can be repaired primarily with interrupted or running non-absorbable sutures such as polypropylene or polyester. For larger defects, or when tissue integrity is compromised, prosthetic reinforcement is recommended (Figure 7). Synthetic meshes (e.g. polytetrafluoroethylene [PTFE], polypropylene) are often used for diaphragm or abdominal wall repairs due to their strength and durability. In contrast, biological meshes (e.g. acellular dermal matrices) are often preferred in contaminated surgical areas or in

patients at high risk of infection, as they have better biocompatibility and lower infection rates.

The mesh should be anchored circumferentially with non-absorbable sutures or tacks, ensuring adequate overlap with healthy diaphragmatic tissue. Care should be taken to avoid injury to adjacent structures such as the pericardium, lung parenchyma, liver, or stomach.

2.5. Postoperative considerations

Chest tube placement is typically required when the thoracic cavity is entered, and lung re-expansion should be monitored closely. Pain control, respiratory physiotherapy, and gradual return to upright activity are key to preventing postoperative complications such as atelectasis or pneumonia. Patients with large preoperative herniations may require gradual abdominal wall accommodation due to loss of domain.

In summary, surgical management of ADHs should be individualized, with a growing preference for minimally invasive techniques such as laparoscopy and VATS when feasible. Thoracic surgeons must be familiar with both approaches and prepared to escalate to open surgery in complex cases to ensure complete, durable, and safe hernia repair.

2.6. Prognosis and follow-up

With timely diagnosis and proper repair, outcomes are generally favorable. However, recurrence may occur if the repair is under tension or in the setting of ongoing elevated intra-abdominal pressure. Patients

with iatrogenic or spontaneous hernias require long-term surveillance, particularly if underlying risk factors persist.

3. Diaphragmatic ruptures and diagnostic pitfalls

Diaphragmatic rupture is a complex clinical entity that often poses diagnostic challenges due to its varied presentations, nonspecific findings, and the limitations of conventional imaging modalities. Ruptures may result from blunt trauma, penetrating injury, or iatrogenic causes, and can range from small, clinically silent defects to massive disruptions associated with visceral herniation and respiratory compromise. Failure to recognize diaphragmatic rupture in a timely manner may result in delayed treatment, increased morbidity, and even mortality. Understanding the diagnostic nuances and potential pitfalls is therefore essential for thoracic and trauma surgeons.

3.1. Mechanisms and clinical context

Diaphragmatic rupture occurs when the structural integrity of the diaphragm is compromised, most commonly due to a sudden increase in intra-abdominal pressure surpassing the tensile capacity of the muscle. Blunt trauma from motor vehicle collisions, falls, or crush injuries accounts for the majority of cases. Penetrating injuries may cause smaller, well-localized tears, while iatrogenic ruptures often result from inadvertent injury during upper abdominal or thoracic surgeries. Left-sided ruptures are more common due to the protective buffering effect of the liver on the right.

3.2. Diagnostic challenges

Diagnosing diaphragmatic rupture is often difficult due to its subtle and nonspecific presentation, especially in the acute trauma setting where more obvious injuries such as long bone fractures or organ lacerations dominate attention. Symptoms like dyspnea, abdominal pain, or shoulder discomfort may be misattributed to more common thoracic injuries or overlooked entirely in sedated or intubated patients.

In delayed cases, overlooked injuries may later present as herniation with vague respiratory or gastrointestinal symptoms. These may mimic chronic obstructive pulmonary disease (COPD), gastroesophageal reflux disease (GERD) or functional disorders, leading to misdiagnosis. The lack of a clear history of trauma, particularly in older or co-morbid patients, further complicates recognition. Maintaining a high level of clinical suspicion is essential, especially in patients with a history of thoracoabdominal trauma, regardless of its initial severity.

3.3. Imaging pitfalls

While imaging plays a critical role in the diagnosis of diaphragmatic rupture, each modality comes with specific limitations, and misinterpretation or underrecognition of subtle signs remains a key contributor to delayed or missed diagnoses.

Chest radiography is often the initial imaging modality obtained in trauma patients due to its rapid availability and ease of use. However,

its sensitivity for detecting diaphragmatic injury is notoriously low, ranging between 17–50% in various studies. Typical findings, when present, are indirect and include elevation or blurring of the hemidiaphragm, obliteration of the diaphragmatic contour, mediastinal shift, and the presence of gas-filled bowel loops within the thoracic cavity. However, such signs are frequently subtle or masked by hemothorax, pulmonary contusion, or subcutaneous emphysema. Moreover, in supine trauma patients, air-fluid levels may not be readily apparent. Right-sided injuries are particularly elusive due to the presence of the liver, which can obscure herniated structures and create false reassurance.

CT has significantly enhanced the ability to detect diaphragmatic defects, particularly with modern multidetector scanners and the use of multiplanar reformatted images. Nonetheless, CT is not infallible. Small or partial-thickness tears, intermittent herniations, and injuries masked by adjacent hemorrhage or soft tissue distortion may evade detection. A common pitfall is overreliance on axial slices, which may miss defects oriented in the sagittal or coronal planes. Therefore, complete review of reconstructed images is essential.

Radiologists and clinicians must be familiar with classic CT signs associated with diaphragmatic rupture:

- The *"collar sign"***,** which indicates a waist-like constriction of herniated organs at the defect site.

- The *"dangling diaphragm"*, where torn diaphragmatic muscle edges appear to hang freely.
- The *"dependent viscera sign"*, which refers to abnormal positioning of abdominal organs against the posterior ribs without diaphragmatic support.

These signs are more readily seen in the left hemithorax, again due to the masking effect of the liver on the right. Interpretation errors are particularly common in the setting of polytrauma, where attention is often focused on more dramatic injuries such as intracranial bleeding or visceral organ lacerations.

MRI, though not routinely used in acute settings, offers excellent soft tissue resolution and can be helpful in selected cases particularly in differentiating between diaphragmatic eventration, tumors, or chronic herniation. MRI may also be beneficial in pediatric or pregnant patients where radiation exposure is a concern.

Fluoroscopic studies, such as dynamic sniff tests or contrast-enhanced gastrointestinal series, can be valuable tools in evaluating diaphragmatic motion and intermittent herniation. These studies are particularly useful in functional defects or in chronic presentations, where standard imaging fails to capture transient visceral displacement. However, their utility is limited in the emergency context, and they require patient cooperation and radiologist expertise.

In summary, the diagnosis of diaphragmatic rupture remains radiologically challenging, particularly in the absence of overt herniation or when attention is diverted by concurrent injuries. Multiplanar CT evaluation with targeted suspicion, awareness of specific imaging signs, and adjunctive modalities where indicated, are all essential to reduce the risk of missed or delayed diagnoses.

3.4. Clinical pitfalls

Diagnostic errors are commonly due to a lack of clinical suspicion or misinterpretation of radiological findings. In some cases, diaphragmatic injury is incorrectly diagnosed as eventration, pleural effusion, or pneumothorax, leading to mismanagement. Surgical findings during unrelated procedures may unexpectedly reveal diaphragmatic defects, highlighting the importance of intraoperative vigilance.

In particular, spontaneous reduction of herniated viscera in supine trauma patients may mask the severity of injury, resulting in false-negative imaging studies. Similarly, ventilation-induced lung expansion during mechanical support may temporarily obscure diaphragmatic contour irregularities.

3.5. Avoiding missed diagnoses

A high index of suspicion should be maintained in any patient with thoracoabdominal trauma, especially when imaging reveals unexplained hemithorax opacification, diaphragmatic contour

asymmetry, or abnormal position of abdominal organs. Multi-modality imaging should be pursued when initial results are inconclusive. Diagnostic laparoscopy or thoracoscopy should be considered in equivocal cases, especially in penetrating trauma.

Routine checklist-based review of trauma CTs by experienced radiologists, ideally in collaboration with surgeons, may further reduce the rate of missed injuries. Training programs should emphasize recognition of key radiographic signs and maintain low thresholds for surgical exploration in high-risk scenarios.

3.6. Conclusion

Diaphragmatic rupture remains a frequently underdiagnosed entity due to its elusive clinical and radiological characteristics. Meticulous attention to mechanism of injury, imaging interpretation, and surgical awareness is essential. Early recognition and timely repair are paramount to prevent life-threatening complications such as visceral strangulation, respiratory failure, and recurrent herniation. A structured diagnostic strategy, combined with a multidisciplinary trauma approach, is the most effective way to minimize diagnostic oversight.

4. Diaphragmatic paralysis and paresis

Diaphragmatic paralysis and paresis refer to a spectrum of conditions characterized by impaired motor function of the diaphragm due to disruption of its neural control. These dysfunctions may be unilateral or

bilateral and result from either central or peripheral etiologies. While paresis implies partial weakness with preserved motion, paralysis denotes complete loss of diaphragmatic function (Laroche et al., 1990). Clinically, these conditions can range from asymptomatic incidental findings to profound respiratory insufficiency, depending on the extent of involvement and underlying cause (Cordeiro et al., 2016).

4.1. Anatomy and innervation

The diaphragm is a dome-shaped musculotendinous structure that serves as the principal muscle of inspiration. It separates the thoracic and abdominal cavities and is essential for generating negative intrathoracic pressure during respiration. Understanding its neuroanatomy is crucial for recognizing the diverse causes and clinical implications of diaphragmatic dysfunction (Gibson et al., 1989).

Motor innervation to the diaphragm is provided by the phrenic nerves**,** which originate predominantly from the anterior rami of the C3, C4, and C5 cervical spinal nerve roots, with C4 providing the main contribution. The mnemonic "C3–C5 keeps the diaphragm alive" underscores the importance of these segments in diaphragmatic function.

After arising from the cervical plexus, the phrenic nerve descends vertically through the neck, running anterior to the anterior scalene muscle and deep to the sternocleidomastoid. It then enters the thoracic cavity by passing posterior to the subclavian vein and anterior to the

root of the lung. The course of the right phrenic nerve continues along the lateral aspect of the superior vena cava and pericardium, ultimately penetrating the diaphragm near the caval hiatus. The left phrenic nerve passes over the pericardium adjacent to the left ventricle and pierces the diaphragm near the cardiac apex (Merrell et al., 2013).

Each phrenic nerve provides motor innervation to the corresponding hemidiaphragm and sensory fibers to the central diaphragm, pericardium, mediastinal pleura, and diaphragmatic peritoneum. This dual motor-sensory role explains referred pain patterns (e.g., shoulder pain) associated with diaphragmatic irritation.

Central control of diaphragmatic movement originates from the respiratory centers in the medulla oblongata, particularly the dorsal respiratory group (DRG) and ventral respiratory group (VRG), which generate rhythmic respiratory impulses. These impulses descend via the corticospinal and bulbospinal pathways to the anterior horn cells at the C3–C5 spinal cord levels, where they synapse with lower motor neurons of the phrenic nerves.

Due to its long and exposed course, the phrenic nerve is vulnerable to multiple sites of injury**:**

- Centrally, lesions affecting the medullary respiratory centers (e.g. stroke, tumors, demyelinating diseases) or the spinal cord (e.g. cervical trauma, syringomyelia, amyotrophic lateral

sclerosis [ALS]) can affect the unilateral or bilateral innervation of the diaphragm.

- Peripherally**,** the phrenic nerve is at risk during procedures involving the neck (e.g., cervical spine surgery), thorax (e.g., cardiac surgery, thoracic tumor resection), or mediastinum (e.g., central line placement, lymphadenectomy).
- The nerve's proximity to major vascular and cardiac structures also places it at risk for compression by aneurysms, tumors, or enlarged lymph nodes**.**

An appreciation of this detailed anatomy is vital for clinicians when interpreting symptoms, evaluating imaging, or planning surgical approaches involving the cervical spine, thorax, or diaphragm (Nason et al., 2012).

Etiology Diaphragmatic dysfunction can be categorized into central and peripheral causes (Ko et al., 2009; Kokatnur et al., 2018):

- Central Causes**:**
    - Brainstem infarcts or hemorrhages affecting the medullary respiratory centers
    - High cervical spinal cord trauma (especially above C5)
    - Neurodegenerative diseases such as ALS, multiple sclerosis
    - Central demyelinating conditions
    - Intracranial tumors or surgical complications
- Peripheral Causes**:**

- Phrenic nerve injury due to trauma, thoracic surgery (e.g., cardiac or esophageal procedures), or cervical spine surgery
- Compression by mediastinal tumors, lymphadenopathy, or aortic aneurysms
- Neuralgic amyotrophy (Parsonage-Turner syndrome)
- Diabetic neuropathy or other systemic neuropathies
- Iatrogenic injury from central line placement or nerve blocks
- Viral infections such as herpes zoster affecting cervical roots or the phrenic nerve

4.2. Clinical presentation

The clinical manifestations of diaphragmatic paralysis and paresis are highly variable and depend on several factors, including whether the involvement is unilateral or bilateral, the rapidity of onset, the patient's baseline cardiopulmonary reserve, and the presence of comorbid conditions such as COPD, obesity, or neuromuscular disorders (Dubé et al., 2016).

Unilateral diaphragmatic paresis or paralysis is often clinically silent and may be discovered incidentally during evaluation for unrelated pulmonary symptoms or radiographic imaging showing an elevated hemidiaphragm. However, when symptoms do occur, they usually reflect mild to moderate reductions in ventilatory capacity, especially

in physically active individuals or those with pre-existing lung disease. Common complaints include:

- *Exertional dyspnea,* particularly during uphill walking or climbing stairs
- *Orthopnea,* defined as breathlessness in the supine position, due to loss of diaphragmatic contribution to inspiratory volume
- *Platypnea,* a less common positional symptom characterized by shortness of breath that worsens when upright
- *Reduced exercise tolerance* or early fatigue

Clinically, paradoxical abdominal movement may be observed during inspiration especially in thin individuals where the abdominal wall is drawn inward instead of outward, indicating paradoxical motion of the paralyzed hemidiaphragm.

Bilateral diaphragmatic paralysis**,** in contrast, is almost always symptomatic and often severely disabling. Because the accessory muscles of respiration (e.g., intercostals, scalene, sternocleidomastoids) must compensate for the absent diaphragmatic function, even minimal exertion can lead to respiratory distress. These patients commonly present with:

- *Marked dyspnea on minimal exertion* or even at rest
- *Severe orthopnea,* often requiring patients to sleep in an upright position or propped with multiple pillows

- *Sleep-disordered breathing*, including nocturnal hypoventilation and central hypopneas
- *Morning headaches and daytime somnolence*, suggestive of chronic hypercapnia and nocturnal CO_2 retention
- *Fatigue and cognitive fog*, due to poor sleep quality and chronic hypoxia

Physical examination may reveal shallow, rapid breathing with minimal abdominal excursion and use of accessory muscles, especially in supine positioning. Auscultation may show reduced breath sounds at the lung bases due to reduced expansion. Paradoxical breathing is more pronounced and can be visually dramatic in bilateral cases.

In children or infants, bilateral diaphragmatic dysfunction may manifest as episodic apnea, feeding difficulty, poor weight gain, or cyanotic spells, often misdiagnosed as central apnea or cardiac pathology.

In both unilateral and bilateral cases, symptoms may worsen with increased abdominal pressure (e.g., post-prandially, pregnancy, ascites), due to further reduction in diaphragmatic excursion. Importantly, the degree of functional impairment may not correlate with radiographic findings, necessitating objective evaluation through imaging and pulmonary function testing (Celli et al., 2002).

4.3. Diagnosis

Diagnosis involves a combination of clinical, radiological, and functional assessments (Jesus F et al., 2025):

- *Chest X-ray:* Elevated hemidiaphragm, reduced lung volume
- *Fluoroscopy (Sniff test):* Paradoxical upward motion of the affected hemidiaphragm during sniffing indicates paralysis
- *Ultrasound:* Assesses diaphragmatic excursion and thickening ratio during respiration (Fayssoil et al., 2017), (Haji et al., 2016)
- *Pulmonary Function Tests (PFTs):* Show restrictive patterns; decreased forced vital capacity (FVC) more pronounced in supine position
- *Electromyography (EMG) and Nerve Conduction Studies (NCS):* Help distinguish neurogenic from myopathic causes
- *MRI or CT:* May identify compressive or structural lesions along the phrenic nerve pathway

4.4. Management

The management of diaphragmatic paralysis and paresis is multifactorial and depends on several variables, including the laterality (unilateral vs. bilateral), the underlying etiology, the severity of respiratory compromise, and the patient's overall functional status. A tailored, patient-specific approach is essential, often involving a multidisciplinary team that includes thoracic surgeons, pulmonologists, neurologists, and physiotherapists.

4.4.1. Observation and conservative management

Asymptomatic unilateral diaphragmatic paresis, especially when incidentally discovered in patients with preserved pulmonary reserve, typically requires no immediate intervention. These cases can be managed conservatively with periodic clinical monitoring and repeat imaging or pulmonary function testing. Most idiopathic or viral-induced cases may resolve spontaneously within 6–12 months, particularly in younger or otherwise healthy individuals.

4.4.2. Non-invasive ventilatory support (NIV)

In patients with bilateral diaphragmatic dysfunction or significant symptomatic hypoventilation, non-invasive positive pressure ventilation (NIPPV) is often the cornerstone of management. Bilevel positive airway pressure (BiPAP) or volume-assured pressure support modes can alleviate nocturnal hypoventilation, improve gas exchange, reduce morning symptoms (e.g., headaches, fatigue), and enhance quality of life. NIV is especially indicated in:

- Documented nocturnal desaturation or hypercapnia
- Sleep-disordered breathing secondary to diaphragm weakness
- Preoperative respiratory optimization
- Patients not suitable for surgical intervention

4.4.3. Diaphragmatic plication

For patients with unilateral diaphragmatic paralysis who experience exertional dyspnea, orthopnea, or poor pulmonary function despite

conservative measures, surgical diaphragmatic plication is a well-established option (Groth et al., 2009).

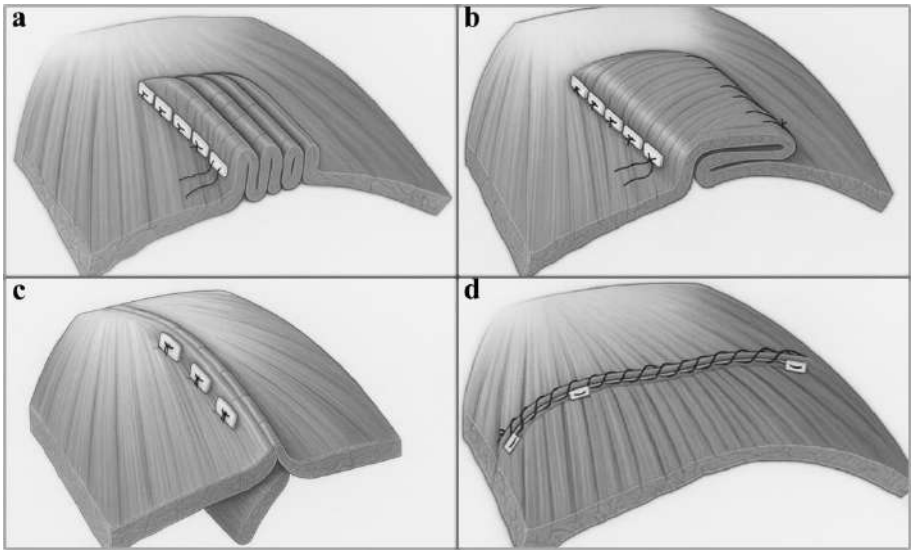


Figure 8. Illustration of four different diaphragmatic plication techniques.

This schematic representation demonstrates commonly used techniques for diaphragmatic plication aimed at flattening the redundant, elevated diaphragm in cases such as diaphragmatic eventration or paralysis.

- **(a)** Multiple *horizontal folding technique with interrupted sutures*: Multiple parallel horizontal folds are created and secured individually using interrupted non-absorbable pledged sutures.
- **(b)** *Continuous running suture over horizontal folds*: Similar horizontal folding is performed but secured using a single continuous running suture.

- **(c)** Horizontal fold technique with interrupted sutures; the plication is inverted toward the abdominal cavity, embedding the fold on the peritoneal side.
- **(d)** *Single-layer plication with continuous suture*: A single longitudinal fold is reinforced using a continuous running suture placed along the plication line.

These techniques are selected based on surgeon preference, diaphragm condition, and procedural access (open vs. minimally invasive approach) (Groth et al., 2010).

In this procedure, the paralyzed hemidiaphragm is folded and sutured to flatten its dome and prevent paradoxical movement, thereby improving lung expansion and respiratory mechanics (Figure 8). It can be performed via thoracotomy, VATS nefits typically include:

- Reduced orthopnea.
- Improved FVC and exercise tolerance.
- Enhanced lung perfusion to the affected side.
- Shortened intensive care unit (ICU) or ventilator dependency in selected cases.

4.4.4. Phrenic nerve reconstruction or stimulation

In select patients, particularly those with recent traumatic or iatrogenic phrenic nerve injury, surgical phrenic nerve repair**,** neurolysis, or nerve

grafting (e.g., using intercostal or sural nerve autografts) may be attempted. More experimental techniques include:

- *Phrenic nerve stimulation* via implanted diaphragmatic pacemakers in cases of high cervical spinal cord injury or central hypoventilation syndrome
- *Diaphragm pacing***,** which may help reduce dependence on mechanical ventilation, though it requires intact lower motor neurons and functional neuromuscular junctions

4.4.5. Respiratory rehabilitation and physiotherapy

Regardless of the underlying treatment path, structured pulmonary rehabilitation is essential to optimize respiratory efficiency. This includes:

- Diaphragmatic breathing exercises
- Incentive spirometry
- Positional training (e.g., sleeping semi-upright)
- Chest physiotherapy for secretion clearance
- Avoidance of sedatives or opioids that may depress respiratory drive

Additionally, nutritional optimization, management of comorbidities (e.g., COPD, heart failure), and vaccination against respiratory pathogens (influenza, pneumococcus) are crucial adjuncts in reducing the risk of complications such as pneumonia.

4.4.6. Long-term monitoring

Patients should undergo regular follow-up with spirometry (including supine vs upright FVC), sleep studies when indicated, and imaging to assess for resolution, progression, or compensatory changes. Reassessment for surgical options may be warranted if functional decline occurs over time.

4.5. Prognosis

The prognosis varies depending on the cause and extent of injury. Idiopathic or viral paresis may resolve spontaneously over months. Traumatic or iatrogenic injuries have variable recovery, while neurodegenerative and central causes typically follow a progressive course (Qureshi et al., 2009).

5. Special considerations in diaphragmatic dysfunction

Diaphragmatic dysfunction encompasses a diverse range of clinical scenarios, each requiring a tailored approach based on the underlying etiology, patient population, and care setting. Among these, three distinct situations warrant particular attention: tracheostomized and mechanically ventilated patients, pregnancy-associated diaphragmatic issues, and diaphragm impairment in intensive care settings, especially ventilator-induced diaphragmatic dysfunction (VIDD) (Greising et al., 2018).

5.1. Diaphragm management in tracheostomized and mechanically ventilated patients

In patients undergoing prolonged mechanical ventilation, particularly those with tracheostomy, diaphragmatic atrophy and weakness are frequent complications. Disuse atrophy due to prolonged diaphragmatic inactivity, coupled with critical illness polyneuromyopathy, can lead to ventilator dependency. Diaphragmatic ultrasonography is a valuable bedside tool to assess diaphragm thickness and excursion, guiding weaning strategies. Interventions such as early mobilization, diaphragmatic pacing, and inspiratory muscle training have shown potential in preserving or restoring diaphragmatic function in this population.

5.2. Diaphragmatic challenges in pregnancy

Pregnancy induces significant anatomical and physiological adaptations that affect diaphragmatic mechanics. The upward displacement of the diaphragm by the enlarging uterus reduces functional residual capacity and may exacerbate any pre-existing diaphragmatic weakness. While these changes are typically well tolerated, they can complicate the clinical picture in women with neuromuscular diseases or prior diaphragmatic injury. Careful respiratory monitoring and a multidisciplinary approach are essential, particularly during the peripartum period, to prevent respiratory compromise.

5.3. Ventilator-induced diaphragmatic dysfunction (VIDD) in the ICU

VIDD is a well-recognized phenomenon resulting from controlled mechanical ventilation, where the absence of diaphragmatic contractions leads to oxidative stress, proteolysis, and myofibrillar damage. Even short durations of controlled ventilation can lead to significant diaphragm weakness. Strategies to prevent or mitigate VIDD include the use of assisted or spontaneous ventilation modes, early weaning protocols, and regular diaphragm monitoring using imaging modalities such as ultrasound or electromyography. Understanding and addressing VIDD is crucial to improve outcomes in critically ill patients and to reduce the duration of mechanical ventilation.

In conclusion, while diaphragmatic dysfunction has broad implications, its management must be individualized for specific clinical contexts. Greater awareness of these special considerations can help clinicians implement proactive strategies that preserve diaphragmatic function and improve patient outcomes.

REFERENCES

Abdellatif, W., Chow, B., Hamid, S., Khorshed, D., Khosa, F., Nicolaou, S., & Murray, N. (2020). Unravelling the mysteries of traumatic diaphragmatic injury: An up-to-date review. *Canadian Association of Radiologists Journal, 71*(4), 482–490. https://doi.org/10.1177/0846537120905133

Anraku, M., & Shargall, Y. (2009). Surgical conditions of the diaphragm: Anatomy and physiology. *Thoracic Surgery Clinics, 19*(4), 419–429. https://doi.org/10.1016/j.thorsurg.2009.08.002

Celli, B. (2002). Respiratory management of diaphragm paralysis. *Seminars in Respiratory and Critical Care Medicine, 23*(3), 275–281. https://doi.org/10.1055/s-2002-33036

Cordeiro, J., Almeida, A. K., Senival, S., & Filho, I. A. (2016). Diaphragmatic eventration: Review of current knowledge, diagnostic, and management options. *Journal of Thoracic Disease, 8*(11), E1551–E1559.

Dubé, B.-P., & Dres, M. (2016). Diaphragm dysfunction: Diagnostic approaches and management strategies. *Journal of Clinical Medicine, 5*(12), 113. https://doi.org/10.3390/jcm5120113

Fayssoil, A., Franques, J.-B., & Brun, C. (2017). Diaphragm: Pathophysiology and ultrasound imaging in neuromuscular disorders. *Journal of Neuromuscular Diseases, 4*(2), 95–105.

Gibson, G. (1989). Diaphragmatic paresis: Pathophysiology, clinical features, and investigation. *Thorax, 44*(12), 990–996.

Greising, S. M., Mantilla, C. B., Sieck, G. C., & Sieck, E. L. (2018). Diaphragm plasticity in aging and disease: Therapies for muscle weakness go from strength to strength. *Journal of Applied Physiology, 125*(5), 1440–1452.

Gritsiuta, A. I., Ivanov, S. P., & Kuznetsova, O. V. (2022). Minimally invasive diaphragm plication for acquired unilateral diaphragm paralysis: A systematic review. *Innovations, 17*(4), 300–310. https://doi.org/10.1177/15569845221097761

Grimes, O. (1974). Traumatic injuries of the diaphragm: Diaphragmatic hernia. *American Journal of Surgery, 128*(2), 175–181.

Groth, S. S., & Andrade, R. S. (2009). Diaphragmatic eventration. *Thoracic Surgery Clinics, 19*(4), 511–519.

Groth, S. S., & Andrade, R. S. (2010). Diaphragm plication for eventration or paralysis: A review of the literature. *Annals of Thoracic Surgery, 89*(6 Suppl), S2146–S2150.

Haji, K., Kim, Y.-S., & Nguyen, T. V. (2016). Interpreting diaphragmatic movement with bedside imaging: Review article. *Journal of Critical Care, 34*, 56–65.

Hanna, W., & Deslauriers, J. (2009). Acute traumatic diaphragmatic injury. *Thoracic Surgery Clinics, 19*(4), 485–489.

Jesus, F., Martin, L., & Okafor, C. (2025). Diaphragm dysfunction: How to diagnose and how to treat? *Breathe, 21*(2), 145–154.

Johnson, C., & Butler, J. (1988). Acquired hernias of the diaphragm. *Postgraduate Medical Journal, 64*(753), 12–15.

Ko, M., & Saito, H. (2009). Acquired paralysis of the diaphragm. *Thoracic Surgery Clinics, 19*(4), 501–510.

Kokatnur, L., & Rudrappa, M. (2018). Diaphragmatic palsy. *Diseases, 6*(1), 16.

Lamture, Y., Singh, P., Kumar, A., et al. (2024). Spontaneous acquired diaphragmatic hernia: Case series and review of literature. *Journal of Emergencies, Trauma, and Shock, 17*(1), 35–39.

Laroche, C. M., & Mier, A. (1990). Diaphragmatic paresis: Pathophysiology, clinical features, and investigation. *Thorax, 45*(4), 294–298.

Lim, B., Tan, C., Wong, J., et al. (2017). Traumatic diaphragmatic injuries: A retrospective review of a 12-year experience at a tertiary trauma centre. *Singapore Medical Journal, 58*(6), 325–329.

Merrell, A. J., & Kardon, G. (2013). Development of the diaphragm – A skeletal muscle essential for mammalian respiration. *FEBS Journal, 280*(17), 4026–4036. https://doi.org/10.1111/febs.12274

Nason, L. K., Walker, C. M., McNeeley, M. F., Burivong, W., Fligner, C. L., & Godwin, J. D. (2012). Imaging of the diaphragm: Anatomy and function. *Radiographics, 32*(2), E51–E70.

Petrone, P., et al. (2007). Diaphragmatic injuries: Challenges in the diagnosis and management. *Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine, 15*, 9.

Qureshi, A. (2009). Diaphragm paralysis. *Seminars in Respiratory and Critical Care Medicine, 30*(3), 315–320.

Reber, P. U., Weber, C., & Schmid, R. A. (1998). Missed diaphragmatic injuries and their long-term sequelae. *Journal of Trauma, 44*(1), 183–188.

Rashid, F., Chakrabarty, M., Boulton, R. F., & Phillips, A. R. (2017). Delayed presentation of diaphragmatic rupture: Review. *World Journal of Emergency Surgery, 12*(1), 45.

Shackleton, K. L., Singh, J., Jessop, F., et al. (1998). Traumatic diaphragmatic injuries: Spectrum of radiographic findings. *Radiographics, 18*(1), 49–59. https://doi.org/10.1148/radiographics.18.1.9460108

Tapias, L., et al. (2009). Hernias diafragmáticas: Desafío clínico y quirúrgico. *Revista Chilena de Cirugía, 61*(2), 145–149.

Testini, M., et al. (2017). Emergency surgery due to diaphragmatic hernia: Case series and review. *World Journal of Emergency Surgery, 12*(1), 23.

Tiberio, G. A., et al. (2005). Traumatic lesions of the diaphragm: Experience in 33 cases and review of the literature. *Acta Chirurgica Belgica, 105*(6), 653–657.

CHAPTER 4

DIAPHRAGMATIC TUMORS AND CYSTS

Asst. Prof. Dr. Mahmut OZBEY

INTRODUCTION

The diaphragm, a dome-shaped musculotendinous structure, serves as a critical anatomic and functional boundary between the thoracic and abdominal cavities. It plays an essential role in respiration and helps maintain intra-abdominal pressure, with its unique anatomical position predisposing it to involvement from both thoracic and abdominal pathologies (Tasnim, et al., 2024).

Despite its physiological importance, primary lesions of the diaphragm are rare and often underestimated. Diaphragmatic tumors and cysts represent a heterogeneous group of benign and malignant diseases, including primary neoplasms, metastatic involvement and congenital or acquired cystic formations. Advances in imaging techniques such as multidetector computed tomography (CT) and magnetic resonance imaging (MRI) have improved the detection of these lesions, even in asymptomatic individuals (Nakamura et al., 2024; Dres et al., 2025).

Malignant involvement of the diaphragm is more commonly secondary, due to direct invasion from adjacent organs such as the liver or lungs, or peritoneal carcinomatosis. Primary malignancies, although

exceedingly rare, include sarcomas and mesothelioma (Engelking, et al., 2024).

Given the diaphragm's proximity to vital structures, accurate diagnosis and appropriate surgical or oncological management of diaphragmatic lesions require a multidisciplinary approach. Recognition of these conditions is vital to avoid misdiagnosis and ensure optimal outcomes (Luna Russo et al., 2020).

1. Benign diaphragmatic lesions

Benign diaphragmatic lesions are rare entities, often discovered incidentally during thoracic or abdominal imaging. Although frequently asymptomatic, they can occasionally cause mass effect or clinical symptoms depending on their size and location (Guo et al., 2021).

1.1. Lipoma

Diaphragmatic lipomas are benign tumors composed of mature adipose tissue. They are usually slow-growing and well-encapsulated, often discovered incidentally on imaging studies. On CT scans, they typically appear as well-circumscribed, non-enhancing hypodense masses. Most lipomas are asymptomatic; however, large lesions may cause chest discomfort or dyspnea due to compression (Alawneh et al,. 2020).

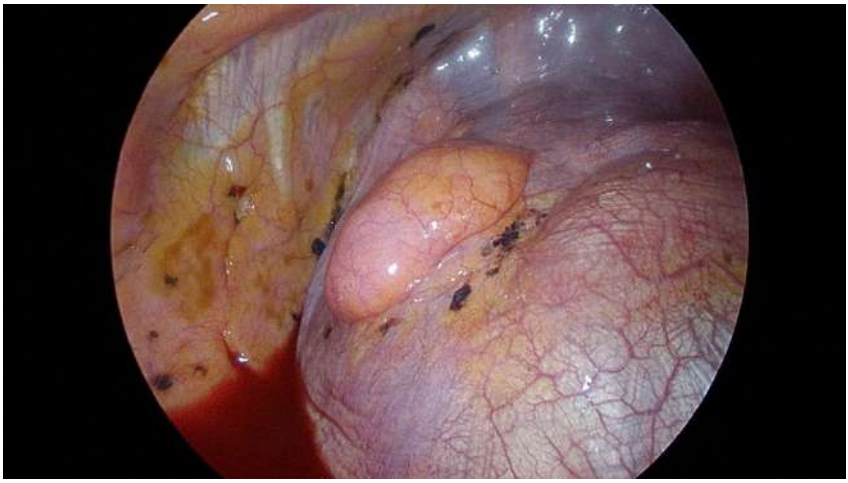


Figure 1. Intraoperative view of a diaphragmatic lipoma located on the left posterolateral diaphragm.

Surgical excision is indicated in symptomatic cases or when diagnosis remains uncertain (Figure 1).

1.2. Cysts

Cystic lesions of the diaphragm can include mesothelial, bronchogenic, or pericardial cysts. Mesothelial cysts, the most common congenital variant, are typically located on the right hemidiaphragm and are lined by a single layer of mesothelial cells. They are unilocular, fluid-filled, and usually benign. Diagnosis is often incidental, and intervention is generally not required unless the cyst is symptomatic or shows signs of infection or hemorrhage (Ben Ismail et al., 2025).

1.3. Endometriosis

Diaphragmatic endometriosis is an uncommon form of extrapelvic endometriosis, more frequently observed in women of reproductive age. It is often associated with catamenial pneumothorax or cyclic thoracic pain. Lesions predominantly affect the right hemidiaphragm, potentially due to clockwise peritoneal fluid circulation (Matsuki et al., 2021).

MRI is considered a valuable tool for diagnosis, particularly when performed during menstruation. Sensitivity ranges from 78% to 83% in detecting diaphragmatic implants (Rousset et al., 2016). Despite these advancements, MRI may underestimate the extent of thoracic involvement (Naem et al., 2024). Minimally invasive approaches such as video-assisted thoracoscopic surgery (VATS) combined with laparoscopy are often employed for diagnosis and treatment (Luna Russo et al., 2021). Hormonal therapy may be used adjunctively to reduce recurrence.

2. Malignant diaphragmatic lesions

Malignant diaphragmatic lesions may be either primary in origin or secondary due to contiguous invasion or metastatic spread. Secondary involvement is significantly more prevalent than primary tumors, as reported in multiple series (Baldes, N., et al,. 2016).

2.1. Primary malignant tumors

Primary diaphragmatic malignancies are exceedingly rare. These include sarcomas (such as liposarcoma, fibrosarcoma) and mesothelioma involving the diaphragmatic pleura or peritoneum (Guo et al., 2021) . Primary pulmonary sarcomas and diaphragmatic mesothelioma are particularly uncommon, with recent imaging-based case studies emphasizing the role of advanced ultrasound and contrast-enhanced imaging in their early detection (Möller et al., 2024).

2.2. Secondary malignant involvement

2.2.1. Lung cancer

Non-small cell lung carcinoma (NSCLC), especially tumors located in lower lobes, may invade the diaphragm directly. Though rare, such cases are associated with poorer prognosis; however, aggressive surgical management including en bloc resection of lung, diaphragm, and even liver, has shown encouraging outcomes with disease-free survival extending beyond 24 months in select patients (Batıhan et al., 2021).

2.2.2. Hepatic tumors

Hepatocellular Carcinoma (HCC) and hepatic metastases particularly from colorectal primaries can infiltrate the diaphragm. Imaging may reveal characteristic diaphragmatic thickening with enhancement, and prompt surgical resection can yield favorable outcomes. For recurrent

HCC lesions invading the diaphragm, early detection and resection correspond to improved control (Kim et al,. 2022). Recent reviews confirm that combining hepatectomy with diaphragmatic resection is becoming increasingly standard in appropriate cases (L et al., 2012) .

2.2.3. Peritoneal and pleural mesothelioma

Malignant mesotheliomas, whether pleural or peritoneal, may invade the diaphragm diffusely or surround it from above or below. Diagnosis is based on multimodality imaging (CT, MRI, positron emission tomography [PET]), thoracoscopic or laparoscopic biopsy and analysis of biomarkers such as mesothelin-related peptides (Porcel, 2022). Desmoplastic malignant peritoneal mesothelioma is a rare, aggressive subtype that primarily affects the subdiaphragmatic peritoneum, with case reports emphasizing its rapid invasion and poor prognosis (Wang et al., 2024).

3. Diaphragm Involvement in Hepatic and Pulmonary Tumors

Given its anatomic location at the interface of the thoracic and abdominal cavities, the diaphragm is uniquely vulnerable to direct invasion by tumors arising in adjacent organs most notably the liver and lungs.

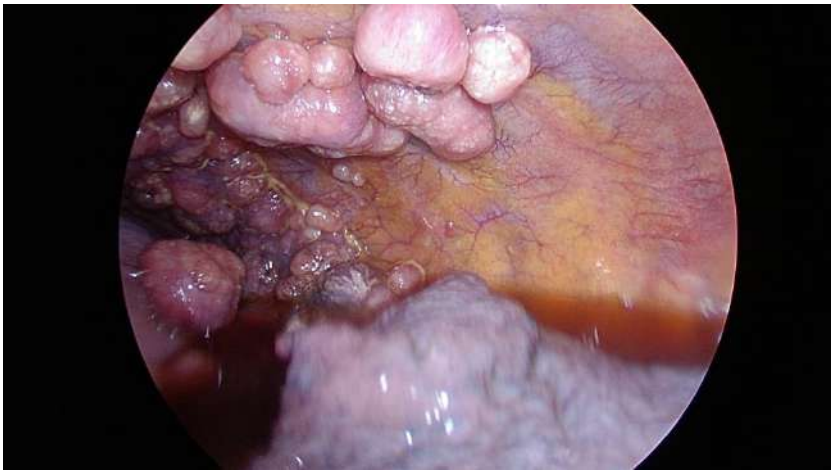


Figure 2. Intraoperative view showing diaphragmatic metastasis of invasive breast carcinoma.

Diaphragmatic invasion by such tumors has critical implications for staging, treatment planning, and prognosis (Shroff, et al., 2023) (Figure 2).

3.1. Liver Tumors Involving the Diaphragm

HCC and metastatic colorectal cancer (CRC) are the most common hepatic tumors that can invade the diaphragm. Invasion usually occurs via direct extension, particularly when tumors are located in the superior right hepatic lobe. Radiologically, this may be observed as obliteration of the fat plane between the liver and diaphragm, diaphragmatic thickening, or enhancement on contrast-enhanced CT and MRI (Kocjan et al., 2024).

Surgical resection involving en bloc hepatectomy with partial diaphragm excision has been demonstrated to be both feasible and effective in selected patients. Several large series report no significant increase in morbidity or mortality with diaphragm resection compared to standard hepatectomy, provided meticulous surgical technique is applied (Kazaryan et al., 2020; Liu et al., 2018). Furthermore, laparoscopic and robotic-assisted techniques for combined liver and diaphragm resections are being increasingly reported with favorable short-term outcomes (Wasson et al., 2015).

3.2. Lung Tumors Involving the Diaphragm

Lower lobe lung cancers, especially those located posteriorly or near the costodiaphragmatic recess, may directly invade the diaphragm. In such cases, T4 staging is warranted according to the 8th edition of the TNM classification. Multimodal imaging, particularly PET-CT, is useful for evaluating true diaphragmatic invasion versus mere adherence due to inflammation.

Extended surgical resections including lobectomy or pneumonectomy with en bloc diaphragm excision have been performed successfully in highly selected patients. In a retrospective review, diaphragmatic resection was not independently associated with worse survival when complete tumor resection (R0) was achieved (Acar et al., 2023). Reconstruction of the diaphragm can be performed with direct closure or prosthetic mesh, depending on the extent of resection.

4. Highlights

- The diaphragm is a rare but important site for both benign and malignant lesions, often diagnosed incidentally due to advances in imaging.

- Benign diaphragmatic lesions include lipomas, cysts, and endometriosis, which generally have favorable outcomes with appropriate management.

- Primary malignant tumors of the diaphragm are extremely rare; secondary involvement from adjacent organs like the liver and lungs is more common and clinically significant.

- Hepatic and pulmonary tumors invading the diaphragm require careful imaging evaluation and may necessitate complex surgical resections to achieve complete tumor removal.

- Minimally invasive techniques, including laparoscopic and robotic-assisted surgeries, are emerging as effective options for diaphragmatic tumor management.

- Multidisciplinary collaboration is essential for optimal diagnosis, treatment planning, and improving patient prognosis in diaphragmatic lesions.

REFERENCES

Acar, A., Ceylan, K. C., Mestan, H., & Yücel, N. (2023). Complete videothoracoscopic en-bloc resection of lung cancer invading the diaphragm. *Turk Gogus Kalp Damar Cerrahisi Dergisi*, *31*(3), 416–419. https://doi.org/10.5606/tgkdc.dergisi.2023.22052

Alawneh, K. Z., Raffee, L. A., Alawneh, K. M., Alawneh, H., & Aleshawi, A. (2020). Diaphragmatic lipoma in a woman with rheumatoid arthritis: A case report and literature review. *The American Journal of Case Reports*, *21*, e925174. https://doi.org/10.12659/AJCR.925174

Baldes, N., & Schirren, J. (2016). Primary and secondary tumors of the diaphragm. *The Thoracic and Cardiovascular Surgeon, 64*(8), 641–646. https://doi.org/10.1055/s-0036-1582256

Batıhan, G., & Kaya, Ş. Ö. (2021). Extended resection for unexpected invasion of the left sided lung cancer into the liver: Combined lung, diaphragm, and liver resection. *Indian Journal of Thoracic and Cardiovascular Surgery, 37*(3), 348–350. https://doi.org/10.1007/s12055-020-01093-x

Ben Ismail, I., Mlika, M., Sghaier, M., Boujemaa, M., Zenaidi, H., Rebii, S., & Zoghlami, A. (2025). Symptomatic mesothelial cyst of the diaphragm misdiagnosed as a hepatic hydatid cyst. *Radiology Case Reports, 20*(4), 2218–2221. https://doi.org/10.1016/j.radcr.2025.01.049

Dres, M., Doorduin, J., Boussouar, S., & Bachasson, D. (2025). Advancements in imaging techniques for monitoring the respiratory muscles. *Critical Care, 29*(1), 110. https://doi.org/10.1186/s13054-025-05339-1

Engelking, M., & Rao, M. (2024). Management of diaphragm tumors. *Thoracic Surgery Clinics, 34*(2), 189–195. https://doi.org/10.1016/j.thorsurg.2024.01.009

Guo, C., Liu, L., Zhang, J., Wang, G., Chen, Y., Huang, C., Liu, H., & Li, S. (2021). Analysis of clinicopathological features of primary diaphragm tumors: A single-center study. *Thoracic Cancer, 12*(12), 1810–1813. https://doi.org/10.1111/1759-7714.13988

Kazaryan, A. M., Aghayan, D. L., Fretland, Å. A., Semikov, V. I., Shulutko, A. M., & Edwin, B. (2020). Laparoscopic liver resection with simultaneous diaphragm resection. *Annals of Translational Medicine, 8*(5), 214. https://doi.org/10.21037/atm.2020.01.62

Kim, C. H., Song, K. D., & Woo, J. H. (2022). Infiltrative invasion of the diaphragm: An uncommon manifestation of recurrent hepatocellular carcinoma. *Precision and Future Medicine, 6*(1), 78–84. https://doi.org/10.23838/pfm.2021.00086

Kocjan, J., Rydel, M., & Adamek, M. (2024). Hepatocellular carcinoma (HCC) metastasis to the diaphragm muscle: A systematic review and meta-analysis of case reports. *Cancers, 16*(17), 3076. https://doi.org/10.3390/cancers16173076

Li, G. Z., Turley, R. S., Lidsky, M. E., Barbas, A. S., Reddy, S. K., & Clary, B. M. (2012). Impact of simultaneous diaphragm resection during hepatectomy for treatment of metastatic colorectal cancer. *Journal of Gastrointestinal Surgery, 16*(8), 1508–1515. https://doi.org/10.1007/s11605-012-1896-4

Liu, Y. C., Mao, Y. Z., Wang, J. C., Wang, J., Lao, X. M., Chen, M. S., & Li, S. P. (2018). Hepatocellular carcinoma with en bloc diaphragmatic resection: A single-center experience over 14 years. *International Journal of Surgery, 53*, 93–97. https://doi.org/10.1016/j.ijsu.2018.03.033

Luna Russo, M., Dassel, M., Raymond, D., Richards, E., Falcone, T., & King, C. (2021). Considerations for the surgical management of diaphragmatic endometriosis. *Journal of Minimally Invasive Gynecology, 28*(7), 1282. https://doi.org/10.1016/j.jmig.2020.09.011

Matsuki, M., Numoto, I., Hamakawa, T., Ishii, K., & Chikugo, T. (2021). Primary diaphragmatic clear cell carcinoma associated with endometriosis: A case report and literature review. *Gynecologic Oncology Reports, 36*, 100733. https://doi.org/10.1016/j.gore.2021.100733

Möller, K., Dietz, F., Ludwig, M., Eisenmann, S., Görg, C., Safai Zadeh, E., Blank, W., Jenssen, C., Vetchy, V., Möller, B., & Dietrich, C. F. (2024). Comments and illustrations of the European Federation of Societies for Ultrasound in Medicine (EFSUMB) guidelines: Rare malignant pulmonal and pleural tumors: Primary pulmonary sarcoma and mesothelioma,

imaging features on transthoracic ultrasound. *Diagnostics, 14*(20), 2339. https://doi.org/10.3390/diagnostics14202339

Naem, A., Roman, H., Martin, D. C., & Krentel, H. (2024). A bird-eye view of diaphragmatic endometriosis: Current practices and future perspectives. *Frontiers in Medicine, 11*, 1505399. https://doi.org/10.3389/fmed.2024.1505399

Nakamura, H., Hirai, T., Kurosawa, H., Hamada, K., Matsunaga, K., Shimizu, K., Konno, S., Muro, S., Fukunaga, K., Nakano, Y., Kuwahira, I., & Hanaoka, M. (2024). Current advances in pulmonary functional imaging. *Respiratory Investigation, 62*(1), 49–65. https://doi.org/10.1016/j.resinv.2023.09.004

Porcel, J. M. (2022). Pleural mesothelioma. *Medicina Clinica, 159*(5), 240–247. https://doi.org/10.1016/j.medcli.2022.03.007

Rousset, P., Gregory, J., Rousset-Jablonski, C., Hugon-Rodin, J., Regnard, J. F., Chapron, C., & Coste, J. (2016). MR diagnosis of diaphragmatic endometriosis. *European Radiology, 26*(11), 3968–3977. https://doi.org/10.1007/s00330-016-4226-5

Shroff, N., Choi, W., Elshikh, M., Wong, B., & Bhargava, P. (2023). Multimodality imaging approach in identifying invasive hepatocellular carcinoma. *Clinical Imaging, 97*, 34–43. https://doi.org/10.1016/j.clinimag.2023.03.002

Tasnim, S., Bribriesco, A. C., & Sudarshan, M. (2024). Surgical diaphragm: Anatomy and physiology. *Thoracic Surgery Clinics, 34*(2), 111–118. https://doi.org/10.1016/j.thorsurg.2024.01.002

Wang, J., & Deng, H. (2024). Case report: Radiological features of a case of desmoplastic malignant mesothelioma of peritoneum. *Frontiers in Oncology, 14*, 1502105. https://doi.org/10.3389/fonc.2024.1502105

Wasson, M. N., Magtibay, P., & Magrina, J. (2015). Robotic excision of diaphragm metastases with liver involvement. *Journal of Minimally Invasive Gynecology, 22*(6S), S119. https://doi.org/10.1016/j.jmig.2015.08.335

CHATER 5

DIAPHRAGMATIC FUNCTION DISORDERS

Asst. Prof. Dr. Suleyman Emre AKIN
Orcıd ID:0000-0002-0641-5229

INTRODUCTION

The diaphragm plays a critical role in respiration as the principal muscle of inspiration, contributing significantly to effective ventilation and maintaining intrathoracic pressure gradients. Diaphragmatic function disorders encompass a wide range of conditions characterized by partial or complete loss of diaphragmatic motion, which can be unilateral or bilateral, acute or chronic, and structural or functional.

These disorders may result from various etiologies, including neurological, traumatic, infectious, iatrogenic, or idiopathic causes.

Accurate diagnosis and timely intervention are essential, as diaphragmatic dysfunction can lead to significant respiratory compromise, particularly in patients with underlying pulmonary or neuromuscular diseases.

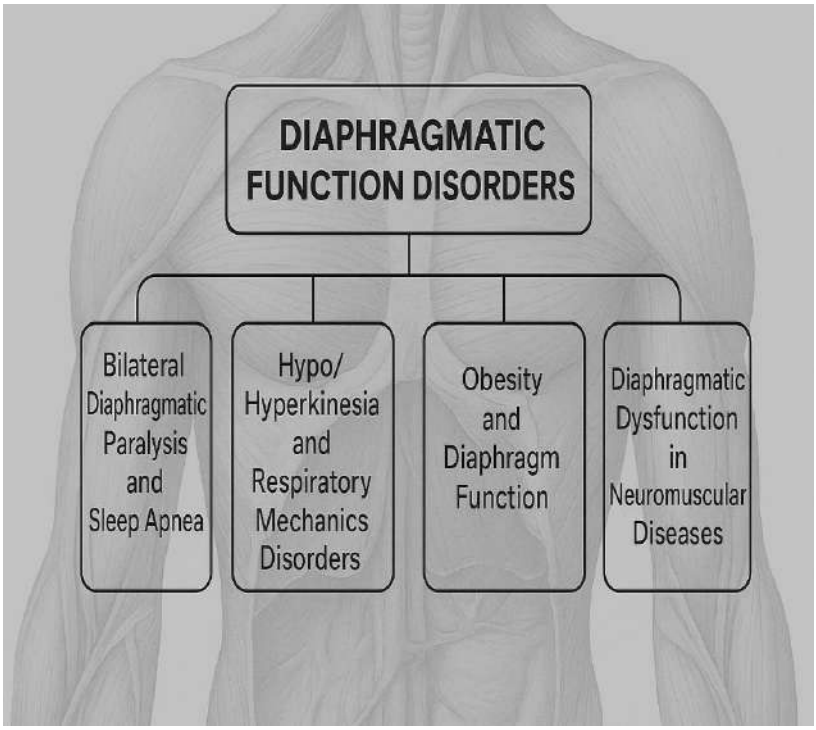


Figure 1. Diaphragmatic function disorders.

Understanding the pathophysiology, clinical presentation, and available diagnostic and therapeutic approaches is key to optimizing outcomes in affected individuals. Diaphragmatic function disorders are divided into the following subheadings and will be explained in detail (Figure 1).

1. Bilateral diaphragmatic paralysis and sleep apnea

Bilateral diaphragmatic paralysis (BDP) is a critical respiratory condition characterized by complete loss of function in both

hemidiaphragms. The diaphragm is essential for ventilatory mechanics any compromise in its integrity leads to severe reductions in tidal volume, lung compliance, and overall breathing efficiency. These effects are especially pronounced during sleep, a period when muscle tone naturally diminishes, resulting in an increased risk of nocturnal hypoventilation and sleep apnea. Recent research highlights the substantial clinical burden imposed by BDP, especially in populations with concomitant comorbidities (Piper & Yee, 2014; Shah et al., 2024).

The loss of diaphragmatic function triggers compensatory reliance on accessory respiratory muscles. The inability to generate appropriate negative intrathoracic pressure leads to reduced inspiratory capacity and an increase in work of breathing (Withana, 2024). Clinically, patients with BDP typically report profound dyspnea, orthopnea, and frequent awakening at night due to breathlessness. During rapid eye movement (REM) sleep, the loss of compensatory muscle tone unmasks the severity of diaphragmatic dysfunction, exacerbating episodes of central and obstructive sleep apnea (BaHammam & Hunasikatti, 2024). Other common findings include morning headaches (due to nocturnal hypercapnia), cognitive impairment, and, with prolonged hypoxemia, signs of right-sided heart failure (Le Pimpec-Barthes et al., 2016).

Diagnostic work-up starts with bedside observation paradoxical abdominal wall movement and reduced chest expansion suggest diaphragmatic dysfunction (Locke et al, 2022). Pulmonary function tests reveal a restrictive pattern with a significant drop in vital capacity, especially in the supine position. Dynamic imaging, particularly

ultrasonography and fluoroscopy, helps visualize absent or paradoxical diaphragmatic movement (Piper & Yee, 2014). For the assessment of sleep-disordered breathing, overnight polysomnography is the gold standard, quantifying apnea–hypopnea indexes and characterizing the type of apnea (BaHammam & Hunasikatti, 2024). Phrenic nerve conduction studies may elucidate neurogenic causes.

The mainstay of BDP management is ventilatory assistance. Non-invasive ventilation (NIV), used primarily nocturnally, has demonstrated benefit in improving sleep architecture, daytime alertness, and gas exchange (Shah et al., 2024). For carefully selected patients, diaphragmatic pacing or surgical plication may be considered (Le Pimpec-Barthes et al., 2016). Respiratory physiotherapy and regular pulmonary function monitoring form the cornerstone of long-term care. In some cases, urgent management of acute hypercapnic respiratory failure with invasive ventilation becomes necessary.

1.1. The Role of diaphragmatic weakness in sleep apnea

Diaphragmatic paralysis drastically alters upper airway dynamics during inspiration, increasing pharyngeal collapsibility and significantly raising the risk for obstructive and central sleep apnea syndromes (Withana, 2024). REM sleep further compounds this vulnerability by physiologically reducing accessory muscle compensation. Therefore, targeted diaphragmatic and sleep pathology treatment is crucial and improves patient-centered outcomes (BaHammam & Hunasikatti, 2024).

Highlights

-BDP leads to profound nocturnal hypoventilation and is strongly associated with sleep-disordered breathing.

-Polysomnography and dynamic imaging are essential diagnostic modalities.

-Nocturnal NIV is the mainstay of therapy; surgical and pacing interventions remain adjunctive.

-Sleep apnea in BDP requires a comprehensive, multidisciplinary management approach.

2. Hypo/hyperkinesia and respiratory mechanics disorders

Alterations in diaphragmatic movement hypokinesia (decreased movement) or hyperkinesia (exaggerated movement) have profound implications for pulmonary mechanics and gas exchange. Hypokinesia is frequently observed in neuromuscular diseases, chest trauma, and metabolic imbalances, whereas hyperkinesia is rarer and often associated with compensatory mechanisms or specific movement disorders. Both phenomena disrupt the delicate balance necessary for effective inhalation and contribute to respiratory insufficiency and morbidity (Cesanelli et al., 2024; Dua et al., 2019).

Hypokinesia describes a diminished amplitude of diaphragmatic contraction, leading to shallow and ineffective breaths. Etiologies

include phrenic nerve dysfunction, muscle weakness, metabolic disturbances, and prolonged intensive care unit (ICU) stays (Walters, 2022). Hyperkinesia reflects excessive or dyskinetic movement, often seen in cases of partial neuromuscular recovery or as an adaptive response in contralateral healthy hemidiaphragms (Xu et al., 2022).

Decreased diaphragmatic motion is associated with a reduction in tidal volume, functional residual capacity, and vital capacity (Cesanelli et al., 2024). This leads to a restrictive ventilatory defect and an increased risk for atelectasis and chronic hypoxemia. Hyperkinesia, while rarely the primary pathology, can create abnormal transdiaphragmatic pressure gradients that impair the synchrony of chest wall and lung expansion. Both states can provoke compensation from accessory muscles, increased respiratory rate, and ventilatory inefficiency (Doherty & Chaudhry, 2021; Walters, 2022).

Bedside examination remains valuable, especially with paradoxical or asymmetric breathing patterns. Ultrasound is the preferred imaging modality to quantify diaphragmatic excursion and thickness during quiet and deep breathing (Xu et al., 2022). Fluoroscopy and magnetic resonance imaging (MRI) provide additional structural details but are less commonly used in routine practice. Pulmonary function tests characterize the resulting restrictive or mixed ventilatory defects. Electromyography can differentiate between neural and muscular causes (Dua et al., 2019).

Management centers on correction of the underlying etiology addressing neuromuscular dysfunction, electrolyte disturbances, or trauma (Doherty & Chaudhry, 2021). Respiratory muscle training may mitigate weakness in selected patients. In the context of persistent hypoventilation, non-invasive ventilation or mechanical support may be required. Close clinical monitoring (including serial ultrasound assessments) guides therapy and determines prognosis (Cesanelli et al., 2024).

Highlights

-Hypo- and hyperkinesia disrupt normal lung volumes and cause restrictive or mixed ventilatory defects.

-Comprehensive assessment requires bedside observation, imaging, and pulmonary function testing.

-Treatment must be individualized, often combining supportive care with targeted intervention.

3. Obesity and diaphragm function

Obesity is a global epidemic that significantly impairs respiratory function. The accumulation of adipose tissue within the thoracoabdominal compartment restricts diaphragmatic excursions, leading to mechanical and functional limitations. These changes predispose patients to restrictive ventilatory deficits, hypoventilation, and complications such as obstructive sleep apnea and obesity

hypoventilation syndrome. Recent studies reveal a complex interplay between increased body mass index (BMI), diaphragm muscle mechanics, and metabolic derangement, with clear implications for both clinical outcomes and therapeutic strategies (Cerundolo et al., 2025; Dixon & Peters, 2018).

Obesity elevates intra-abdominal and intrathoracic pressures due to increased fat deposition, raising the resting position of the diaphragm and reducing its functional reserve (Wang & Hu, 2022). The result is decreased tidal volume and lung compliance, requiring the diaphragm to generate greater force for adequate ventilation (Parameswaran et al., 2006). Studies using imaging and ultrasound report decreased diaphragmatic excursion and thickening among obese individuals, independent of age and comorbidities (Cerundolo et al., 2025; Dixon & Peters, 2018). Over time, chronic processes contribute to muscle fatigue and diminished inspiratory reserve (Rodrigues et al., 2020).

The severity of restrictive ventilatory patterns in obese patients is proportional to the degree of excess weight, vital capacity, and total lung capacity decline with rising BMI. This mechanical constraint is augmented during recumbency and sleep, aggravating nocturnal hypoventilation and gas exchange abnormalities (Parameswaran et al., 2006). Additionally, respiratory muscle inefficiency increases susceptibility to atelectasis and postoperative pulmonary complications (Wang & Hu, 2022).

Obese individuals often present with dyspnea on exertion, orthopnea, and unexplained fatigue (Dixon & Peters, 2018). Pulmonary function testing reveals restrictive deficits and reduced maximal inspiratory/expiratory pressures. Imaging (ultrasound, MRI) quantifies the degree of diaphragmatic impairment, while arterial blood gas analysis may demonstrate hypercapnia in severe cases. Sleep studies are frequently employed to diagnose concurrent sleep disorders (Cerundolo et al., 2025; Chokroverty, 2010).

Weight loss via lifestyle interventions, pharmacotherapy, or bariatric surgery can dramatically improve diaphragmatic function and overall respiratory mechanics. Recent meta-analyses demonstrate improved spirometric indices and increased diaphragmatic excursion following significant weight loss (Parameswaran et al., 2006; Cerundolo et al., 2025). Early intervention is recommended to prevent irreversible changes and optimize long-term quality of life (Chokroverty, 2010).

Highlights

-Obesity impairs diaphragmatic function, reducing tidal volume and increasing respiratory effort.

-Imaging and pulmonary function testing are essential in evaluating dysfunction severity.

-Weight reduction improves respiratory performance and prevents complications.

4. Diaphragmatic dysfunction in neuromuscular diseases

Neuromuscular diseases (NMDs) such as amyotrophic lateral sclerosis (ALS), muscular dystrophies, and myasthenia gravis, frequently involve the diaphragm due to direct muscular degeneration or impaired neuromuscular transmission. This results in progressive respiratory insufficiency, often culminating in chronic hypoventilation, sleep-disordered breathing, and ultimately respiratory failure the leading cause of mortality in advanced NMDs. Modern diagnostic tools and therapeutic strategies have improved early identification and management, providing substantial gains in quality of life and survival (Casey et al., 2023; Charalampopoulou & Maragakis, 2024).

Disruption of the neuromuscular pathway ranging from motor neuron degeneration (as in ALS), transmission defects (as in myasthenia gravis), or primary myopathies results in either weakness or paralysis of the diaphragm (Charalampopoulou & Maragakis, 2024). This impairs the generation of negative intrathoracic pressure, reduces lung volumes, and leads to ineffective cough, predisposing to recurrent infections and secretions retention (Graustein et al., 2023). The progression and pattern of diaphragmatic involvement may vary with disease type, but eventually, all compromise respiratory pump efficiency.

Symptoms often begin subtly with exertional dyspnea, orthopnea, sleep disturbances, and frequent nocturnal awakenings (Graustein et al., 2023). Reduced voice volume, altered cough strength, and recurrent

respiratory tract infections are common. Diagnosis is established by spirometry (showing reduced forced vital capacity, particularly supine), arterial blood gas analysis, and diaphragm ultrasound or fluoroscopy to directly visualize the loss of motion (Casey et al., 2023). Serial sleep studies are recommended due to the high prevalence of nocturnal hypoventilation in this population (Aboussouan, 2015).

Non-invasive positive pressure ventilation (NIPPV) is a lifesaving measure, improving both survival and quality of life (Aboussouan, 2015). Secretion management using cough-assist devices, chest physiotherapy, and suctioning is an essential adjunct. Early education and periodic reassessment are critical, as escalation to invasive ventilation may be warranted as disease progresses (Graustein et al., 2023). Multidisciplinary care, involving neurologists, pulmonologists, and physiotherapists, is strongly associated with improved outcomes and patient satisfaction (Charalampopoulou & Maragakis, 2024).

Diaphragm pacing, gene therapy, and advanced pharmacotherapies (e.g., antisense oligonucleotides for spinal muscular atrophy) represent the frontier of care (Casey et al., 2023). Tailored pulmonary rehabilitation, including inspiratory muscle training, has been shown to slow decline in respiratory function (Torres-Castro et al., 2021). Access to clinical trials and specialist centers can offer eligible patients potentially disease-modifying interventions.

Highlights

-Diaphragmatic dysfunction is central to morbidity and mortality in neuromuscular disease.

-Early diagnosis and regular reassessment are critical components of care.

-NIPPV and secretion management dramatically improve survival and quality of life.

CONCLUSION

Disorders of diaphragmatic function represent a clinically diverse group of conditions with potentially significant impacts on respiratory performance and overall quality of life. Advances in diagnostic imaging, pulmonary function testing, and neuromuscular assessment have improved early detection and characterization of diaphragmatic dysfunction. Therapeutic strategies vary depending on the underlying cause and severity, ranging from observation and supportive care to surgical intervention or phrenic nerve stimulation. A multidisciplinary approach involving pulmonologists, neurologists, radiologists, and thoracic surgeons is often necessary for comprehensive management. Continued research is needed to refine diagnostic criteria and develop targeted therapies for this underrecognized but clinically important group of disorders.

REFERENCES

Aboussouan, L. S. (2015). Sleep-disordered breathing in neuromuscular disease. American Journal of Respiratory and Critical Care Medicine, 191(9), 979–989. https://doi.org/10.1164/rccm.201412-2224CI

BaHammam, A. S., & Hunasikatti, M. (Eds.). (2024). *Sleep apnea frontiers: Pathophysiology, diagnosis, and treatment strategies* [Eds.]. Springer Nature.

Casey, K., Sharma, G., Selim, B., Zielinski, D., Aboussouan, L. S., McKim, D., & Gay, P. (2023). Respiratory management of patients with neuromuscular weakness. Chest: Critical Care Guidelines and Consensus Statements.

Cerundolo, N., Siniscalchi, C., Okoye, C., Scarlata, S., Parise, A., Rendo, M., ... & Ticinesi, A. (2025). Integrated lung, diaphragm, and lower limb muscular ultrasound: Clinical correlations in geriatric patients with acute respiratory illness. Diagnostics, 15(1), 87. https://doi.org/10.3390/diagnostics15010087

Charalampopoulou, A., & Maragakis, N. J. (2024). Overview of the pathophysiology and epidemiology of neurologic disorders affecting the respiratory system. In Pulmonary complications of neuromuscular disease (pp. 1–36). Springer Nature Switzerland. https://doi.org/10.1007/978-3-031-65335-3_1

Chokroverty, S. (2010). Overview of sleep & sleep disorders. Indian Journal of Medical Research, 131(2), 126–140.

Cesanelli, L., Cesanelli, F., Degens, H., & Satkunskiene, D. (2024). Obesity-related reduced spirometry and altered breathing pattern are associated with mechanical disadvantage of the diaphragm. Respiratory Physiology & Neurobiology, 325, 104267. https://doi.org/10.1016/j.resp.2024.104267

Dixon, A. E., & Peters, U. (2018). The effect of obesity on lung function. Expert Review of Respiratory Medicine, 12(9), 755–767. https://doi.org/10.1080/17476348.2018.1506331

Doherty, L., & Chaudhry, V. (2021). Inpatient diagnosis and management of neuromuscular disorders. Seminars in Neurology, 41(5), 493–510. https://doi.org/10.1055/s-0041-1733794

Dua, K., Malyla, V., Singhvi, G., Wadhwa, R., Krishna, R. V., Shukla, S. D., ... & Hansbro, P. M. (2019). Increasing complexity and interactions of oxidative stress in chronic respiratory diseases: An emerging need for novel drug delivery systems. Chemico-Biological Interactions, 299, 168–178. https://doi.org/10.1016/j.cbi.2018.12.009

Graustein, A., Carmona, H., & Benditt, J. O. (2023). Noninvasive respiratory assistance as aid for respiratory care in neuromuscular disorders. Frontiers in Rehabilitation Sciences, 4, 1152043. https://doi.org/10.3389/fresc.2023.1152043

Le Pimpec-Barthes, F., Legras, A., Arame, A., Pricopi, C., Boucherie, J. C., Badia, A., & Panzini, C. M. (2016). Diaphragm pacing: The state of the art. Journal of Thoracic Disease, 8(Suppl 4), S376–S386. https://doi.org/10.21037/jtd.2016.03.97

Locke, B. W., Lee, J. J., & Sundar, K. M. (2022). OSA and chronic respiratory disease: Mechanisms and epidemiology. International Journal of Environmental Research and Public Health, 19(9), 5473. https://doi.org/10.3390/ijerph19095473

Parameswaran, K., Todd, D. C., & Soth, M. (2006). Altered respiratory physiology in obesity. Canadian Respiratory Journal, 13(4), 203–210. https://doi.org/10.1155/2006/834786

Piper, A. J., & Yee, B. J. (2014). Hypoventilation syndromes. Comprehensive Physiology, 4(4), 1639–1676. https://doi.org/10.1002/j.2040-4603.2014.tb00591.x

Rodrigues, G. C., Rocha, N. N., Maia, L. D. A., Melo, I., Simões, A. C., Antunes, M. A., & Silva, P. L. (2020). Impact of experimental obesity on diaphragm structure, function, and bioenergetics. Journal of Applied Physiology, 129(5), 1062–1074. https://doi.org/10.1152/japplphysiol.00262.2020

Shah, N. M., Steier, J., Hart, N., & Kaltsakas, G. (2024). Effects of non-invasive ventilation on sleep in chronic hypercapnic respiratory failure. *Thorax*, *79*(3), 281–288. https://doi.org/10.1136/thorax-2023-220036

Torres-Castro, R., Lima, Í., Resqueti, V. R., & Fregonezi, G. A. (2021). Weaning from mechanical ventilation in people with neuromuscular disease: A systematic review. BMJ Open, 11(9), e047449. https://doi.org/10.1136/bmjopen-2020-047449

Walters, J. (2022). Weakness in the intensive care unit. Practical Neurology, 22(5), 358–375. https://doi.org/10.1136/pn-2022-003422

Wang, Y., & Hu, C. (2022). Leptin and asthma: What are the interactive correlations? Biomolecules, 12(12), 1780. https://doi.org/10.3390/biom12121780

Withana, S. (2024). Multidisciplinary approach to sleep disordered breathing. Asian Journal of Lung Health, 1, 5.

Xu, Q., Yang, X., Qian, Y., Hu, C., Lu, W., Cai, S., & Li, J. (2022). Comparison of assessment of diaphragm function using speckle tracking between patients with successful and failed weaning: A multicentre, observational, pilot study. BMC Pulmonary Medicine, 22, 459. https://doi.org/10.1186/s12890-022-02260-z

CHAPTER 6

DIAPHRAGMATIC EVENTRATION AND PLICATION

Dr. Murat HANOZU, MD, Thoracic Surgeon

Dr. Mehmet Ali UNAL, MD, Thoracic Surgery Resident

INTRODUCTION

The diaphragm is a very important muscle. A dysfunction of the diaphragm manifests itself in various symptoms. In some cases, surgery is required to improve these symptoms. Diaphragmatic surgery can be performed using robotic, thoracoscopic, laparoscopic, thoracotomy, sternotomy and laparotomy methods. All methods have advantages and disadvantages compared to the others (Ricoy et al., 2019).

1. Diaphragmatic eventration

Diaphragmatic eventration occurs more frequently in the left diaphragm than in the right diaphragm and is more common in men. (Krishnani et al., 2024).

This disease usually manifests as shortness of breath and gastrointestinal symptoms. Diaphragmatic eventration is a disease that can be cured by surgery (Forter-Chee-A-Tow & Smith, 2023).

Plication surgery is an effective treatment for diaphragmatic eventration. After diaphragm plication intrathoracic volume increases and the lung subsequently expands better (Agarwal & Lone, 2024).

There may be congenital and acquired causes for the etiology of diaphragmatic ventration. The causes of diafragmatic eventration include some infections, tumors, granulomatous diseases, intrathoracic surgery, muscle-nerve diseases, and trauma. (Özkan et al., 2016).

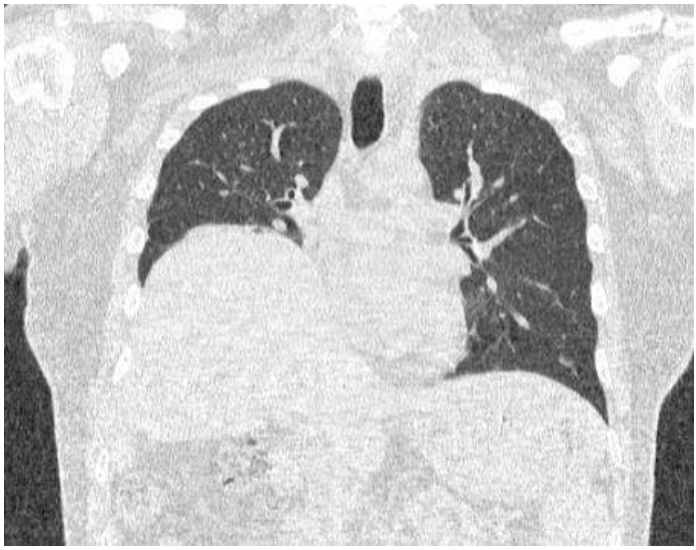


Figure 1. Example of a computed tomography image of a case of right diaphragmatic eventration.

The most commonly used imaging modalities for diagnosis are computed tomography of the thorax (Figure 1.), ultrasound, magnetic resonance imaging, fluoroscopy and chest radiographs (Laghi et al., 2021).

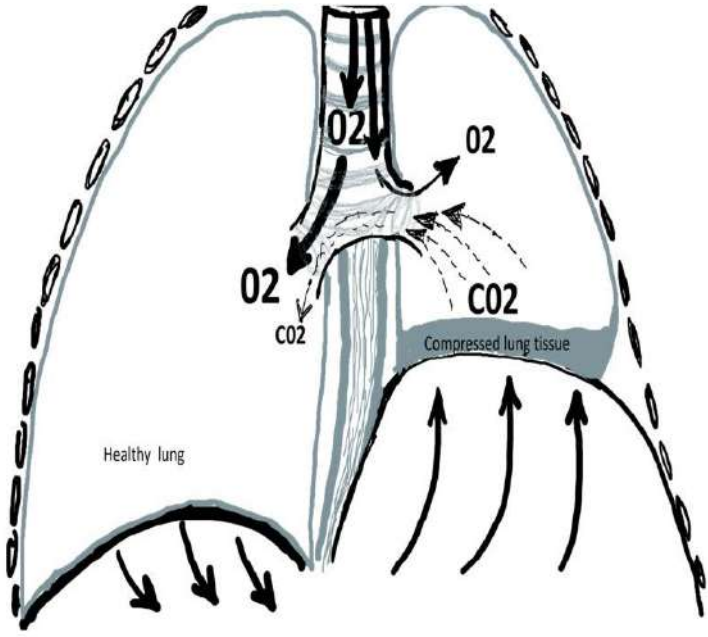


Figure 2. A paralyzed high diaphragm causes gas intolerance. This process is described in the drawing (Beshay et al ., 2023).

In patients with a high diaphragm, paralysis can also occur outside the eventration. This can also be examined with the aid of fluoroscopy. A paralyzed elevated diaphragm causes gas mismatch (Figure 2). Diaphragmatic paralysis occurs as a result of damage to the phrenic nerve or pressure on this nerve. In these cases, diaphragm plication is performed. (Tiryaki et al., 2006).

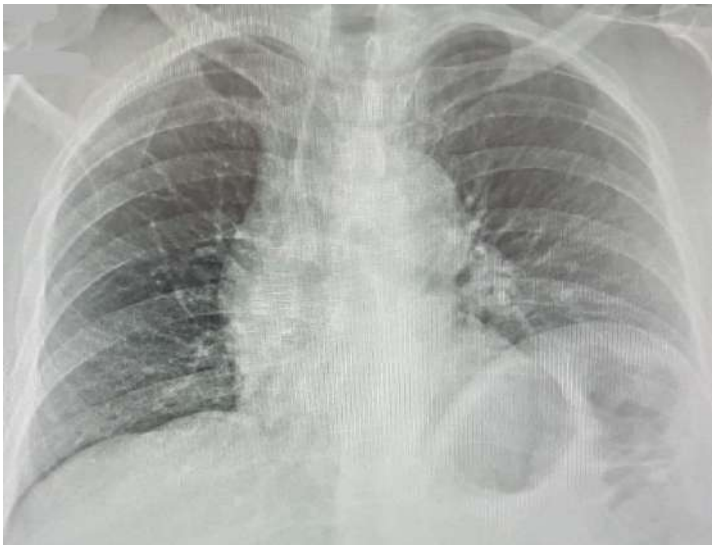


Figure 3. X-ray image before left diaphragm plication.

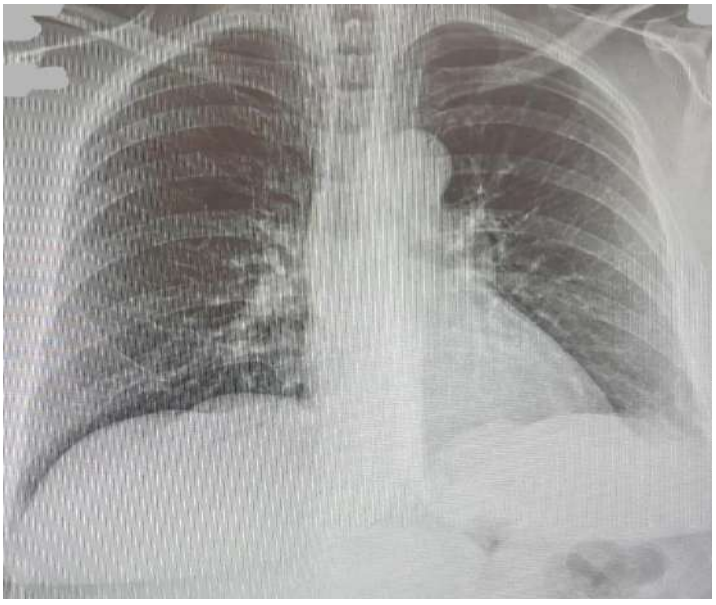


Figure 4. X-ray image after diaphragm plication on the left.

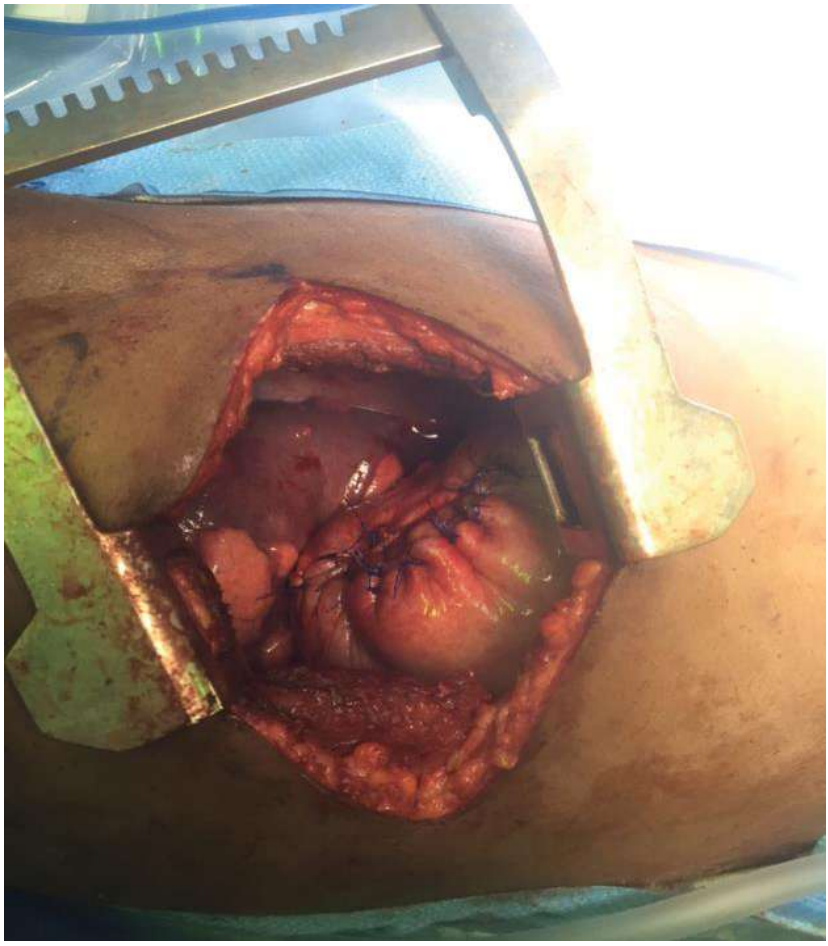


Figure 5. Method of left thoracotomy for diaphragm plication (Shwaartz et al., 2017).

The aim of diaphragm plication is to tighten and compress the diaphragm. Surgeons often prefer to compare chest x-rays before (Figure 3) and after (Figure 4) the plication operation. If diaphragmatic plication is planned via a thoracotomy (Figure 5) or thoracoscopic

approach, the surgeon prefers one-lung ventilation, preferably with CO_2 insufflation, and inserts a chest tube for drainage (Stamenovic, 2017).

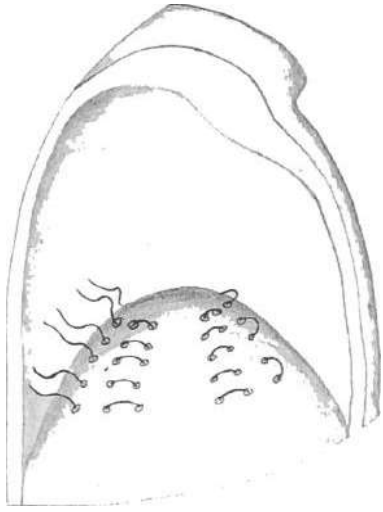


Figure 6. Mattress stitch technique (Freeman et al., 2009).

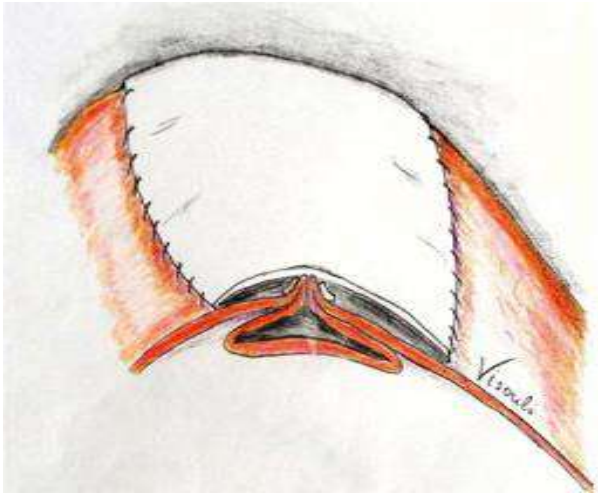


Figure 7. Drawing of the diaphragm, supported by plication and patch. (Visouli et al., 2012).

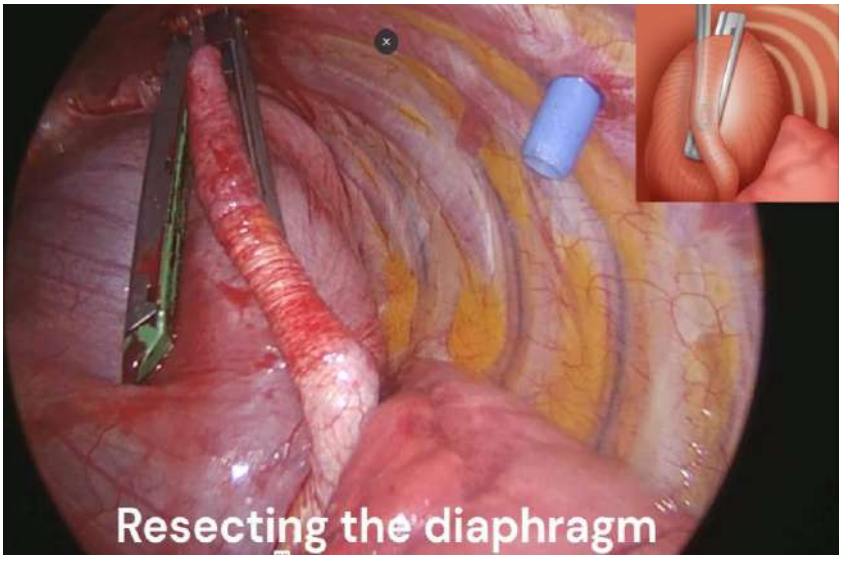


Figure 8. Stapling technique with stapling device (Yun &Yoon 2023).

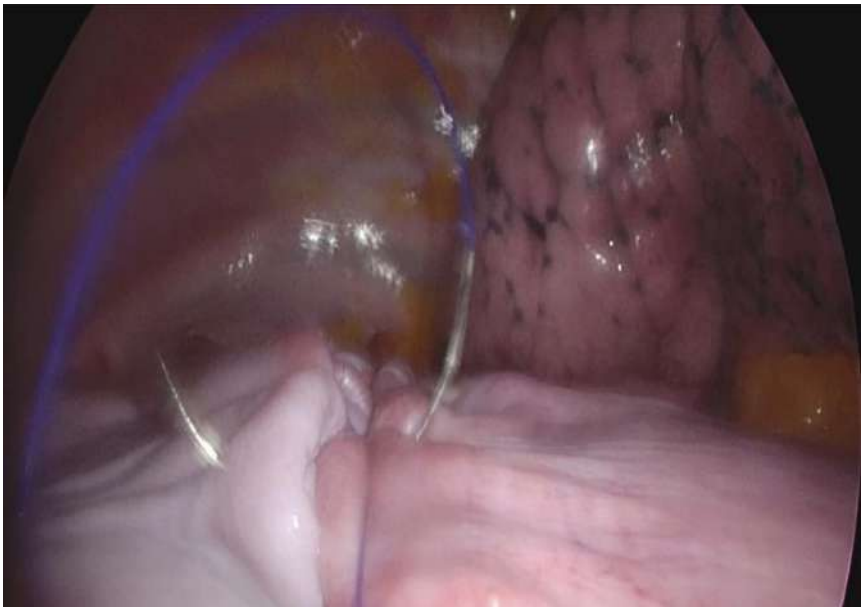


Figure 9. Continuous suturing tecnique.

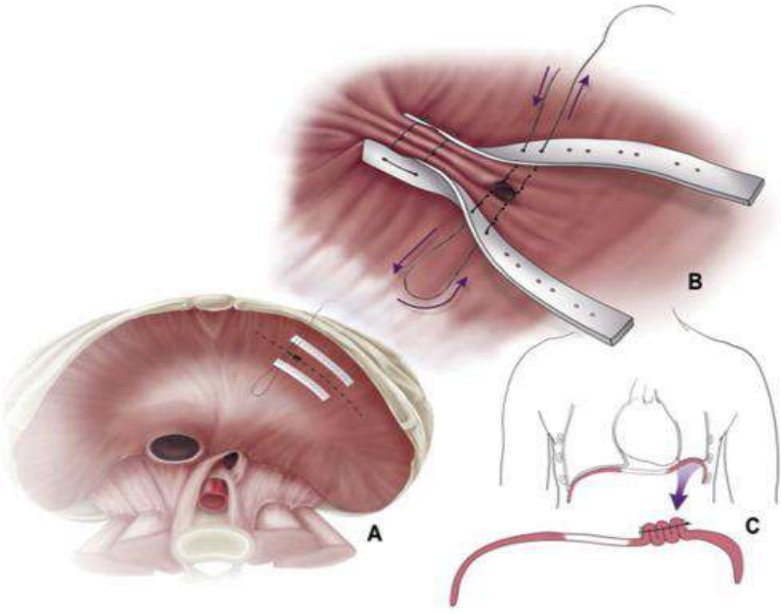


Figure 10. (A) Schema of diaphragm, and (B) Accordion technique. Pledgets and mattress stitches shown (C) Diaphragm after surgical technique. The occurrence of a tense diaphragm (Zwischenberger et al., 2016)**.**

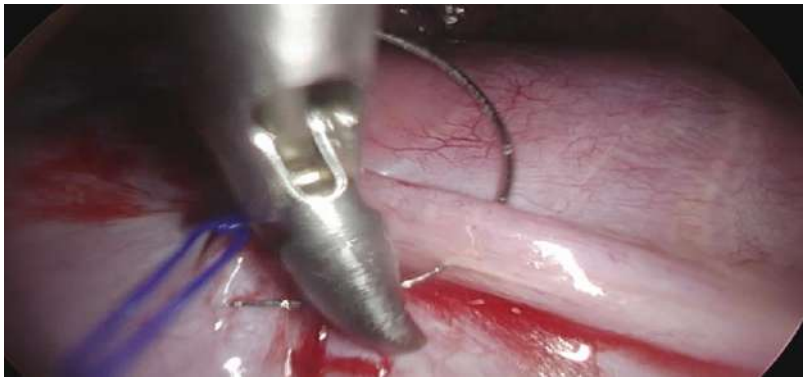


Figure 11. VATS (Video-assisted thoracoscopic surgery) diaphragmatic plication (with non-absorbable suture).

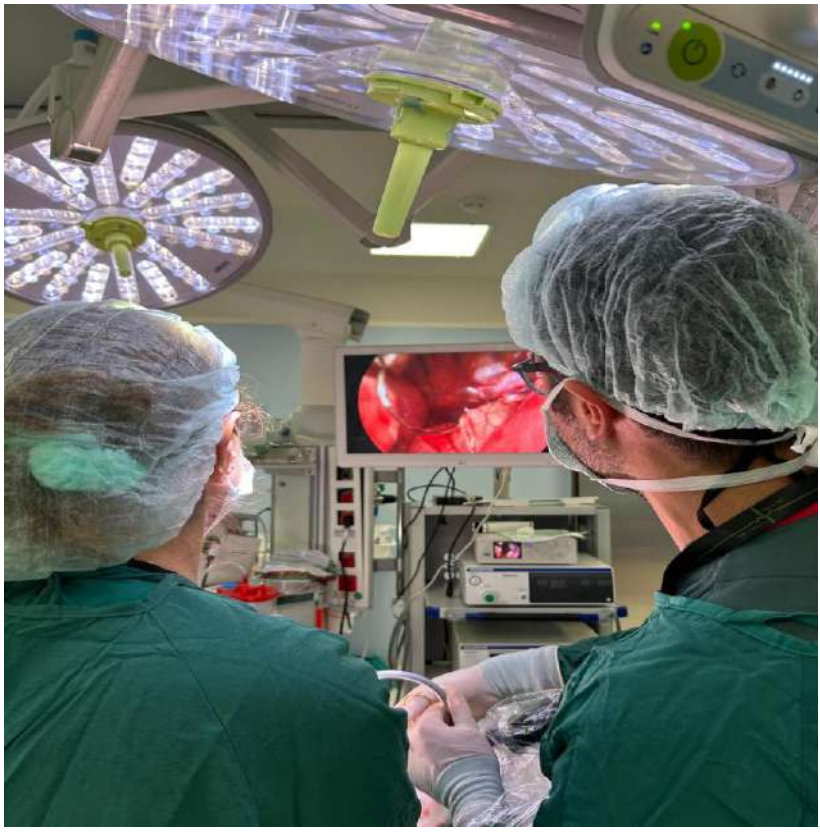


Figure 12. VATS left diaphragmatic plication with non-absorbable suture.

Commonly used techniques for plication include ‘U-mat stitches (Figure 6), applying mash (Figure 7), stapling with staplers (Figure 8), continuous suturing (Figure 9), putting pledget (Figure 10), and accordioning. Non-dissolvable sutures are preferred (Figure 11). The operation is complete when adequate plication is achieved. Thoracic surgeons may opt for single-port or multi-port thoracoscopic surgery (Figure 12). Complications of the procedure include abdominal organ

injury and bleeding, pneumonia, pleural effusion and subsequent re-elevation of the diaphragm (Evman et al., 2016).

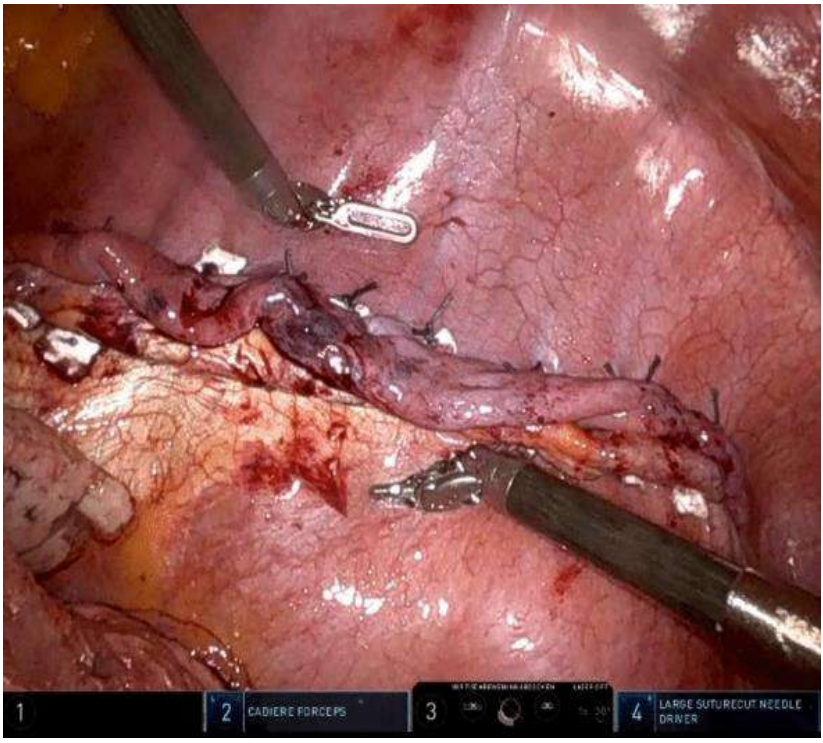


Figure 13. Robotic diaphragm plication (Le et al ., 2023).

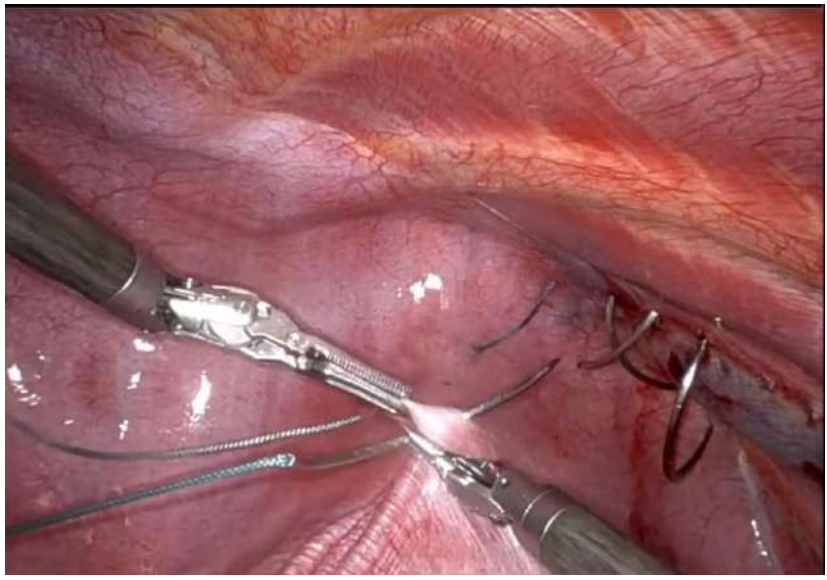


Figure 14. The method in which the diaphragm is sutured to the chest wall. The diaphragm attached to the ribs is placed under tension (Mughal & Habib, 2023).

Robot-assisted surgery for diaphragm plication (Figure 13) is being used more and more frequently today. (Figure 14). The high costs can be seen as a disadvantage. However, it offers the surgeon a great advantage in terms of appearance and comfort (Şengül et al ., 2023).

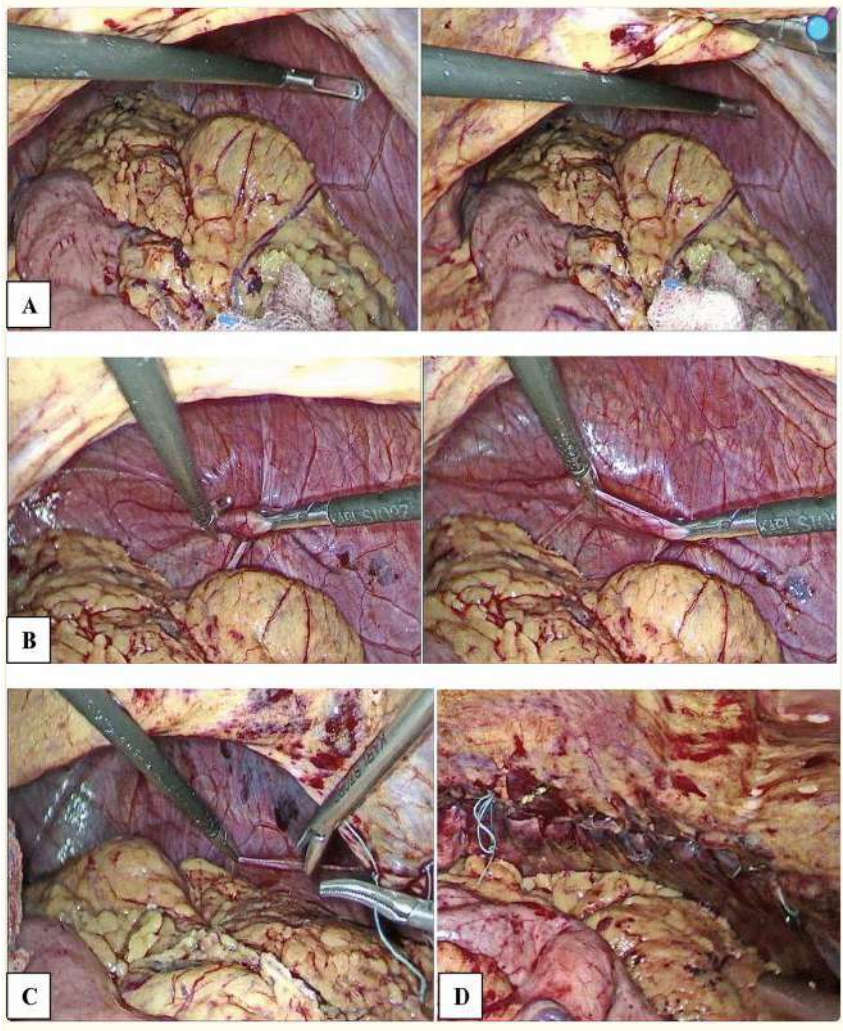


Figure 15. A, Diaphragmatic eventration. **B,** Holding of the diaphragm. **C,** Beginning of the operation. **D,** After completion of diaphragm plication (El-Magd et al., 2024).

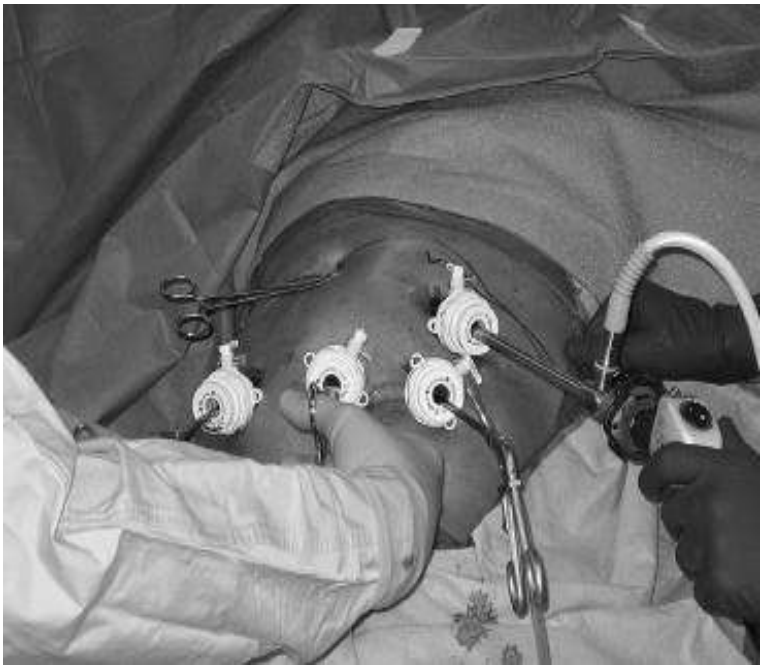


Figure 16. Location of ports in laparoscopic diaphragm plication (Groth et al., 2010).

The laparoscopic (Figure 15) or laparotomy approach may also be preferred as it does not require single lung ventilation and allows access to both diaphragms. With the laparoscopic approach, as with thoracoscopy, placement of the port (Figure 16) is important. (Zeng et al., 2024).

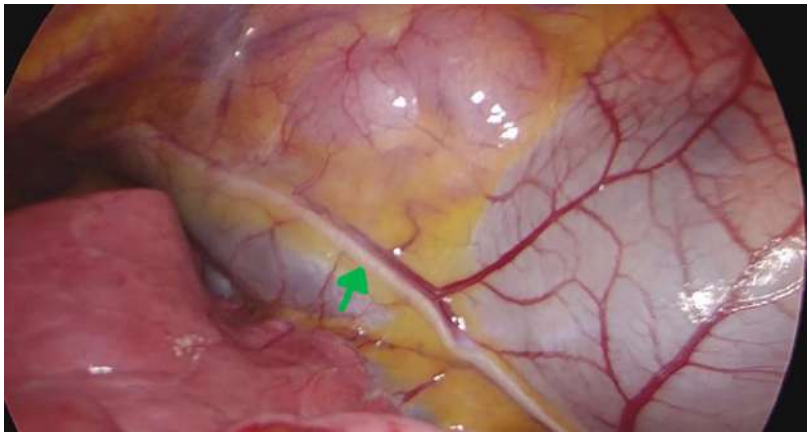


Figure 17. The green arrow indicates the phrenic nerve.

The diaphragm is innervated by the phrenic nerve (Figure 17). Diaphragmatic ventrication may result from damage to the phrenic nerve due to surgery such as cardiac surgery, resection of the anterior mediastinum, and other tumors (e.g., thymic carcinoma, lung cancer) that invade the phrenic nerve, and plication may be required. Some tumors directly invade the phrenic nerve and the surgeon must cut the phrenic nerve. In these patients, a diaphragmatic plication is also performed during the same procedure (Espana et al., 2021).

HIGHLIGHTS

Minimally invasive techniques are increasingly preferred today. Less pain and a more aesthetic appearance after the operation are very effective. Non-minimally invasive techniques are used for recurrences or adhesions of organs.

REFERENCES

Agarwal, A. K., & Lone, N. A. (2024). Diaphragm Eventration. In *StatPearls*. StatPearls Publishing.

Beshay, M., Abdel Bary, M., Kösek, V., Vordemvenne, T., Mertzlufft, F., & Schulte Am Esch, J. (2023). Minimally-Invasive Diaphragmatic Plication in Patients with Unilateral Dia8Fphragmatic Paralysis. *Journal of clinical medicine*, *12*(16), 5301. https://doi.org/10.3390/jcm12165301)

El-Magd, E. A., Elgeidie, A., Abbas, A., Elmahdy, Y., LotfyAbulazm, I., & Hamed, H. (2024). Laparoscopic approach in the management of diaphragmatic eventration in adults: gastrointestinal surgical perspective. *Updates in surgery*, *76*(2), 555–563. https://doi.org/10.1007/s13304-023-01665-7)

España, M. I., Sastre, I., Franco, C. A., Emilia, Q., Ceballos, R. J., & Bustos, M. E. F. (2021). Video-assisted thoracoscopic diaphragmatic plication. *Multimedia manual of cardiothoracic surgery: MMCTS*, *2021*, 10.1510/mmcts.2021.043. https://doi.org/10.1510/mmcts.2021.043)

Evman, S., Tezel, C., Vayvada, M., Kanbur, S., Urek, S., Baysungur, V., & Yalçınkaya, I. (2016). Comparison of Mid-Term Clinical Outcomes of Different Surgical Approaches in Symptomatic Diaphragmatic Eventration. *Annals of thoracic and cardiovascular surgery: official journal of the Association of Thoracic and Cardiovascular Surgeons of Asia*, *22*(4), 224–229. https://doi.org/10.5761/atcs.oa.16-00018)

Forter-Chee-A-Tow, N., & Smith, A. (2023). Case report: repair of eventration of the diaphragm in an octogenarian. *Journal of surgical case reports*, *2023*(10), rjad581. https://doi.org/10.1093/jscr/rjad581)

Freeman, R. K., Van Woerkom, J., Vyverberg, A., & Ascioti, A. J. (2009). Long-term follow-up of the functional and physiologic results of diaphragm plication in adults with unilateral diaphragm paralysis. *The Annals of thoracic surgery*, *88*(4), 1112–1117. https://doi.org/10.1016/j.athoracsur.2009.05.027)

Groth, S. S., Rueth, N. M., Kast, T., D'Cunha, J., Kelly, R. F., Maddaus, M. A., & Andrade, R. S. (2010). Laparoscopic diaphragmatic plication for diaphragmatic paralysis and eventration: an objective evaluation of short-term and midterm results. *The Journal of thoracic and cardiovascular surgery*, *139*(6), 1452–1456. https://doi.org/10.1016/j.jtcvs.2009.10.020)

Krishnani, N., Gupta, P., & Patankar, R. (2024). Ruptured Diaphragmatic Eventration: Lessons Learned. *Cureus*, *16*(10), e71929. https://doi.org/10.7759/cureus.71929) It is seen at a higher rate in men.)

Laghi, F. A., Jr, Saad, M., & Shaikh, H. (2021). Ultrasound and non-ultrasound imaging techniques in the assessment of diaphragmatic dysfunction. *BMC pulmonary medicine*, *21*(1), 85. https://doi.org/10.1186/s12890-021-01441-6)

Le, U. T., Titze, L., Hundeshagen, P., Passlick, B., & Schmid, S. (2023). Robotic diaphragm plication: functional and surgical outcomes

of a single-center experience. *Surgical endoscopy*, *37*(6), 4795–4802. https://doi.org/10.1007/s00464-023-09942-7)

Mughal, A. Z., & Habib, A. M. (2023). Right robotic-assisted hybrid diaphragm plication - central tendon fixation. *Multimedia manual of cardiothoracic surgery: MMCTS*, *2023*, 10.1510/mmcts.2023.041. https://doi.org/10.1510/mmcts.2023.041)

Ozkan, S., Yazici, Ü., Aydin, E., & Karaoğlanoğlu, N. (2016). Is surgical plication necessary in diaphragm eventration?. *Asian journal of surgery*, *39*(2), 59–65. https://doi.org/10.1016/j.asjsur.2015.05.003)

Ricoy, J., Rodríguez-Núñez, N., Álvarez-Dobaño, J. M., Toubes, M. E., Riveiro, V., & Valdés, L. (2019). Diaphragmatic dysfunction. *Pulmonology*, *25*(4), 223–235. https://doi.org/10.1016/j.pulmoe.2018.10.008)

Shwaartz, C., Duggan, E., Lee, D. S., Divino, C. M., & Chin, E. H. (2017). Diaphragmatic eventration presenting as a recurrent diaphragmatic hernia. *Annals of the Royal College of Surgeons of England*, *99*(7), e196–e199. https://doi.org/10.1308/rcsann.2016.0342)

Stamenovic D. (2017). New technique of diaphragmatic plication by means of uniportal video-assisted thoracoscopic surgery. *Interactive cardiovascular and thoracic surgery*, *25*(1), 162–163. https://doi.org/10.1093/icvts/ivx022)

Şengül İnan, M., Kavaklı, K., Işık, H., Sapmaz, E., Gürkök, S., İlhan Sezer, E., Çaylak, H., & Genç, O. (2023). Transthoracic robotic

plication for diaphragmatic elevation. *Turk gogus kalp damar cerrahisi dergisi*, *31*(2), 215–221. https://doi.org/10.5606/tgkdc.dergisi.2023.23283)

Tiryaki, T., Livanelioğlu, Z., & Atayurt, H. (2006). Eventration of the diaphragm. *Asian journal of surgery*, *29*(1), 8–10. https://doi.org/10.1016/S1015-9584(09)60285-2)

Visouli, A. N., Mpakas, A., Zarogoulidis, P., Machairiotis, N., Stylianaki, A., Katsikogiannis, N., Tsakiridis, K., Courcoutsakis, N., & Zarogoulidis, K. (2012). Video assisted thoracoscopic plication of the left hemidiaphragm in symptomatic eventration in adulthood. *Journal of thoracic disease*, *4 Suppl 1*(Suppl 1), 6–16. https://doi.org/10.3978/j.issn.2072-1439.2012.s001)

Yun, J., & Yoon, C. (2023). Video-assisted thoracoscopic diaphragm oblique resection simplified with an endostapler for diaphragm eventration. *Multimedia manual of cardiothoracic surgery : MMCTS*, *2023*, 10.1510/mmcts.2023.037. https://doi.org/10.1510/mmcts.2023.037)

Zeng, B., Gan, W., Zhou, T., Chen, S., Huang, E., Yuan, Z., Liu, F., Liang, Z., & Li, Y. (2024). Laparoscopic management of diaphragmatic eventration: a three-step procedure of diaphragm reconstruction. *Gastroenterology report*, *12*, goae043. https://doi.org/10.1093/gastro/goae043)

Zwischenberger, B. A., Kister, N., Zwischenberger, J. B., & Martin, J. T. (2016). Laparoscopic Robot-Assisted Diaphragm

Plication. *The Annals of thoracic surgery*, *101*(1), 369–371. https://doi.org/10.1016/j.athoracsur.2015.05.118)

ISBN: 978-6-25592-370-7