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UNIVERSITE MOHAMMED V DE RABAT
FACULTE DE MEDECINE
ET DE PHARMACIE
RABAT

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THE IMPACT OF SURGICAL PLASTY IN DIAPHRAGMATIC EVENTRATION

THESIS

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BY

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Dedications

To the greatest parents in the World

Boughdadi Hamid and Toumi Lalla Latifa

I kept postponing dedications, I knew I would tear up as soon as I tried to put words on my feelings. Saying I'm grateful would be an understatement. Every single accomplishment in my life, the person I have become, and everything the future still withholds for me, I owe to you.

Saying you're the best parents in the world might sound cliché or inauthentic, it isn't. This is as close as I can get to an honest phrasing of my sentiments. Miryam once said these wise words: "If I was born to different parents, I would leave them, to go look for you."

I know that no matter how hard I try, my whole life wouldn't suffice to give back the tiniest part of all you did for me. I hence won't try to do that. Instead, I'll do my best every day to make you proud. I'll strive to never let you down. I'll always aim to be the good person you brought me up as. Do not worry though, I'll still take good care of myself and never forget about my own happiness. I know you wouldn't want it any other way.

Thank you for your unconditional Love, immutable to the point I often take it for granted.

May Miryam, Yasmine and I only bring you joy, happiness and delight.

I'll start by dedicating this work to you.

I love you Babati, I love you Mamati

And forever will

To the most wonderful sisters in the World

Boughdadi Miryam and Boughdadi Yasmine

What would my life be like without you? Calmer probably, boring unquestionably, a nightmare undoubtedly.

What kind of person would I have become if you weren't there? The worst possible version of myself.

I could write an essay, thanking you for always being there for me, trying to verbalize the intensity of my love for you, and sharing our most epic stories. But that would only serve to make anyone who doesn't have you for sisters jealous. It also would be overly sentimental.

I will say this though, thank you for putting me back in my place every time I need it. Thank you for standing up for me and invariably being on my side. Thank you for choosing to be my best friends, when you were already born with the heavy assignment of being the best baby sisters.

I'll finally seize this occasion to officially apologize for all the times I took advantage of you, on account of being older. To redeem myself, and as the greatest older sister in the Universe and soon-to-be doctor, I'll do my best to never let anything or anyone hurt you.

I'm proud of the incredible women you have become and know you'll both achieve great things in the future. You'll always have my unconditional Love and absolute support.

I dedicate this work to you.

I love you Miryam I love you Yasmine

To my adoring Mi and Henna

To the memory of Ba and Jeddi

May you rest in piece

Knowing that none of you will ever be able to read this work, either because Death took you away too soon or because education was refused to your gender seventy years ago, is my one source of heartache. Yet I have no doubt about how proud you all are of me. Thank you for loving and indulging me in a way only grandparents would. Thank you for all the happy memories that I'll cherish forever.

I love you all

To all my uncles, aunts and cousins

I'm grateful for being able to call you my family. Thank you for your love, support and encouragements. Thank you for always believing in me. Each of you has contributed in their own way to forge the person I've become.

I love you all and hope this work makes you proud.

To Kenza, Alia and Malak

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Our bond is special, I dedicate this work to you. I love you girls and hope the six of us stay close forever.

To all my friends

To the kindest of them Bourotte Basma

and the most caring of them Sif Nasr Karima

When we first met, I didn't think we could become friends, look at us now, you're two of the most important people in my life. No matter how hard I tried, I couldn't write about one of you without bringing up the other. We went through some of the happiest and hardest moments together. Such friendships are a privilege, not everyone gets to have complete strangers choose them, and decide to love them and be there for them no matter what.

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You're two of the best people I know. You're strong, you're incredibly brilliant, you're gorgeous. Never forget it, or let anyone tell you otherwise!

Let's be friends forever, I love you girls.

To the one who's been there since the beginning

Elhabti Sanae

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We shared our dreams and hopes for an always brighter future. I hope we both get to fulfill every single one of them.

I'm grateful for all the years and memories, thank you for invariably being there for me. With all my love.

To the funniest of them Bouhajla Achraf

Sometimes life puts people on your path for a reason, med school put you on mine since our first osteology class. We sat next to each other through innumerable classes and exams because of our alphabetically close last names, but that wouldn't have been enough to make us the friends we are today. Your jokes helped me deal with many stressful moments, I still wish you would take some things more seriously though.

I know you'll always be there for me, and you know you can infallibly count on me.

To the one I saved for last Addou Ashraf

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To Salma, Ikram, Nabil, Oussama, Amine, Anas

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To our President and thesis advisor

Colonel Doctor Kabiri El Hassane

Professor of thoracic surgery

Hmimv - Rabat

I wish to sincerely thank my honorable thesis president and advisor, who trusted me with this work.

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Your kind acceptance and approval, gave me the freedom to imprint this work and make it my own.

I am profoundly grateful for the honor I was granted working under your valued supervision.

To our Professor and jury member

Colonel Doctor Bounaim Ahmed

Professor of general surgery

Hmimv - Rabat

I would like to offer you my endless gratitude for accepting to be part of my thesis committee.

My first acquaintance with surgery as a third-year medical student, happened under your kind supervision. My internship in your unit marked the beginning of my partiality towards the operating theater.

I am thereby deeply honored to submit this work to your valued judgement. Please find in it a testament of my respect to you as a reputed surgeon and highly esteemed professor.

To our Professor and jury member

Doctor Professor Mssrouri Rahal

Professor of general surgery

Chu Ibn Sina – Rabat

I am profoundly grateful that you accepted my invitation as a member of my thesis committee.

I am proud to have the privilege to count you, an esteemed surgeon and renowned professor as part of this jury.

It is an honor to present this work to your respected judgement. Please find in it a tribute to your contribution in both the academic and medical fields.

To our Professor and jury member

Lieutenant Colonel Doctor El Kaoui Hakim

Professor of general surgery

Hmimv - Rabat

I am greatly honored that you accepted to be part of the judging committee for this thesis. I am sincerely thankful to count you, a renowned doctor and admired professor as a jury member.

It is a marked privilege to submit this work to your reputable judgement.

Please find in this work an acclamation of my high regard to your academic work and medical intake.

To our mentor and associated member

Doctor Commander El Hammoumi Mohammed Massine

Assistant Professor of thoracic surgery

Hmimv – Rabat

I finally would like to express my utmost wholehearted gratitude to Pr. M.M El Hammoumi, whose invaluable guidance and permanent availability provided the means for this work.

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Your dedication as a mentor and your immutable devotion for thoracic surgery inspired me and encouraged me to strive in pursuit of excellence.

I am proud to have had the honor to work under your direction. Please find in this work the statement of my eternal respect and thankfulness.

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Open Access Full Text Article

ORIGINAL RESEARCH

Impact of surgical treatment in acquired dipaphragmatic eventration: experience of a Moroccan center

Impact du traitement chirurgical dans l'évènementration dipaphragmatique acquise: expérience d'un centre Marocain

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List of abbreviations

Abbreviations

aPTT : Activated partial thromboplastin time

BMI : Body Mass Index

COPD : Chronic Obstructive Pulmonary Disease

CPAP : Continuous Positive Airway Pressure

CT scan : Computed Tomography scan

DE : Diaphragmatic Eventration

DLCO : Diffusing Capacity for Carbon Monoxide

DP : Diaphragmatic Paralysis

EMGdi : Diaphragmatic electromyography

FEV1 : Forced Expiratory Volume in the first second

Fig. : Figure

FVC : Forced Vital Capacity

GER : Gastroesophageal Reflux

IMT : Inspiratory Muscle Training

INR : International normalized ratio

MRC : Medical Research Council

MRI : Magneti Resonance Imaging

NPPV : Noninvasive positive-pressure ventilation

Pdi : Transdiaphragmatic pressure

Pes : Esophageal pressure

PFT : Pulmonary Functional Tests

Pga **:** Gastric pressure

PLT **:** Posterolateral Thoracotomy

ppoDLCO **:** Percentage of predicted postoperative diffusing capacity for carbon monoxide

ppoFEV1 **:** Percentage of predicted postoperative forced expiratory volume in one second

REM **:** Rapid Eye Movement

RFT **:** Respiratory Functional Tests

SD **:** Standard Deviation

SPSS **:** Statistical Package for Social Sciences

VATS **:** Video-assisted thoracoscopic surgery

VC **:** Vital Capacity

WHO **:** World health organization

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Summary

Introduction

Diaphragmatic eventration is defined as the abnormal elevation of a portion of, or the entire hemidiaphragm. All the normal anatomical attachments to the dorso-lumbar spine, ribs and sternum are maintained, as opposed to diaphragmatic hernia and rupture.

It can be classified using its origin into congenital or acquired diaphragmatic eventration, the etiology and pathology of each being different, but with similar results: a thin weakened portion of the diaphragm, leading to reduced functions and various clinical symptoms.[1]

The congenital form also called “true diaphragmatic eventration”, is the result of a defect in the muscularization of the diaphragm.[2] The acquired form also called diaphragmatic paralysis is more common in the adult population. Its numerous etiologies can be divided into central and peripheral. In general, all affections of the neuromuscular axis between the cervical spinal cord and the diaphragm can lead to a diaphragmatic eventration.[3] Anatomical criteria can also be used to classify eventrations as unilateral or bilateral, and complete or partial.

Our work will focus on acquired forms of eventration, from peripheral etiologies. The therapeutic management of this pathology isn’t standardized, but plication, a surgical technique based on folding the weak elevated diaphragm has been used as a symptomatic treatment for decades.[4] Many studies proving the efficiency of this method have been published, but, to our knowledge, none yet in our country.

In the treatment of symptomatic eventration, can plication show better results than a medical treatment based on physical therapy?

Can we suggest a diagnostic and therapeutic algorithm that would simplify the management of diaphragmatic eventration?

It is in this perspective, with answering these questions as a goal, that this work was undertaken.

Anatomical and physiological reminder

1. Anatomy of the diaphragm

A knowledge of the anatomy of a structure is fundamental to understanding any of its pathologies. The term diaphragm derives from the Greek words *dia* (in between) and *phragma* (fence). It is a musculo-fibrous dome-shaped membrane separating the thoracic cavity from the abdominal one, and represents the most important inspiratory muscle. It has three major openings, through which vital structures pass: the caval, esophageal and aortic hiatus.

a. Muscular and tendinous portions of the diaphragm

The diaphragm is composed of a central noncontractile aponeurosis surrounded by two major muscular portions: lumbar and costal, and a minor sternal one. These muscular portions form a continuous structure with the transversus abdominis i.e., the inner layer of the abdominal wall.

The crural or lumbar part of the diaphragm is the most powerful one and is located on either side of the vertebral column. This portion has a complex "crisscross" fibers arrangement. The right crura is larger and originates from the vertebrae L1-4, whereas the left crura arises from L1-2. Both right and left crus contribute to the formation of the esophageal hiatus.

The costal portion of the diaphragm originates from the 7^{th} to 12^{th} ribs. A triangular area covered only by fascia can often be found between the costal and lumbar diaphragm, called Bochdalek's gap.

The sternal portion is the smallest one, extending from the back of the xiphoid process and the posterior layer of the rectus sheath. The triangular areas lying between the sternal and costal diaphragm are only covered in connective tissue, and are called triangle of Morgani on the right and of Larrey on the left.

All the muscular striated fibers composing the diaphragm have an insertion on the central tendinous part, which is a clover-leaf shaped fascial aponeurosis with three leaves (right, left and anterior). This tendon's location is more anterior, and the right leaf is the most prominent of all three. The central part lies under the pericardium, to which the superior surface of the tendinous part is firmly attached.

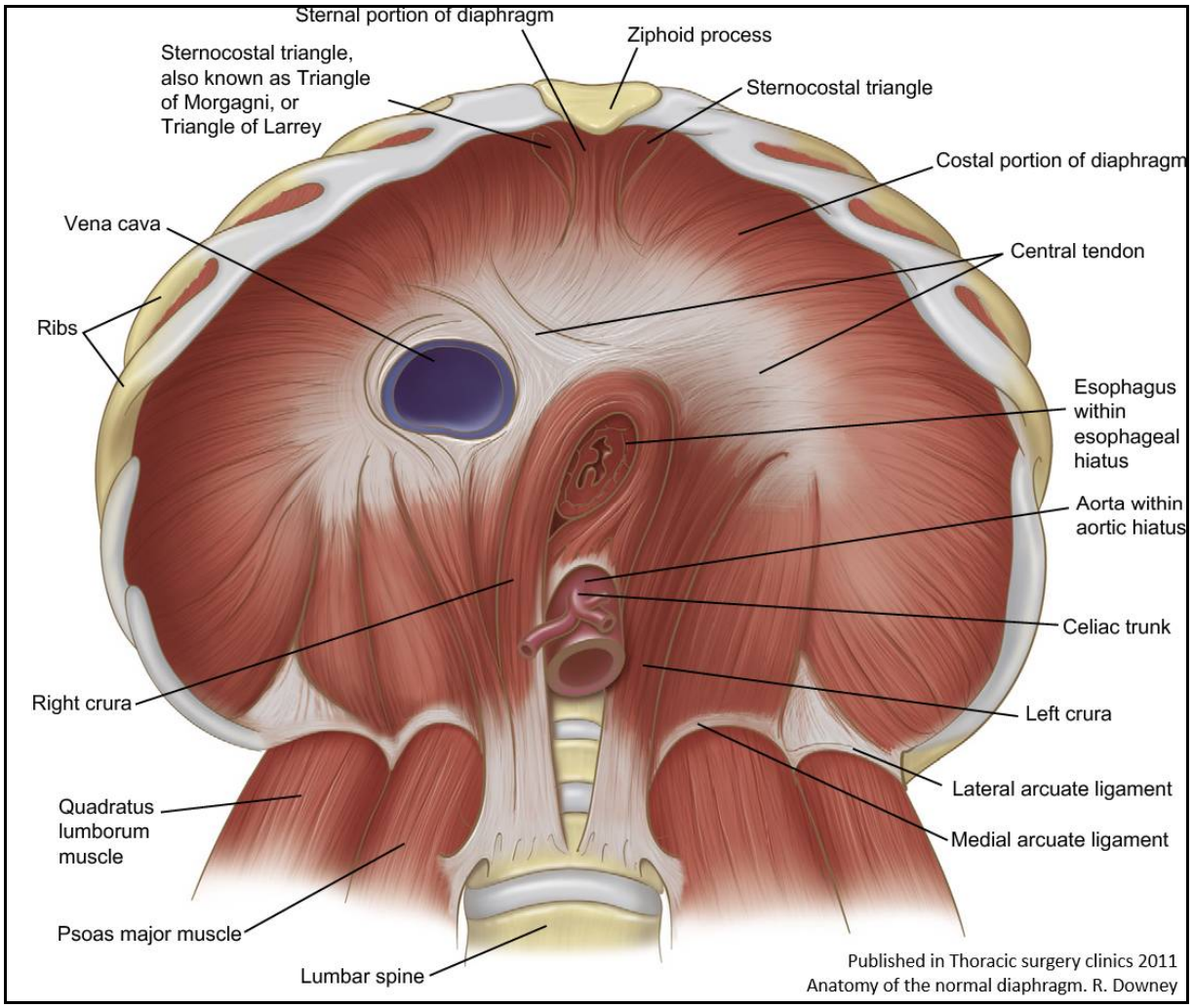


Figure 1: Abdominal view of the diaphragm showing its muscular portions

The right and left diaphragmatic domes are lateral to the heart and mobile depending on the ventilation's extent. At rest, the right dome is at the 4th intercostal space's level and the left one at the 5th's. During deep inspiration, both dome levels descend by approximately two intercostal spaces. [5, 6]

b. Blood supply of the diaphragm

The diaphragmatic arterial blood supply is rich, ensured by the pericardiophrenic arteries, the musculophrenic arteries, the superior and inferior phrenic and the 5 lowest intercostal arteries. The superior phrenic arteries originate from the thoracic aorta. The musculophrenic arteries and the pericardiophrenic ones both derive from the internal thoracic artery. All of these supply to the thoracic side of the diaphragm. The right and left inferior phrenic arteries on the abdominal side of the diaphragm, can be direct branches of the abdominal aorta or sometimes arise from the celiac trunk. Being considerably larger than all the other arteries, they are the main source of oxygenated blood supply to the diaphragm. [5, 7]

The venous drainage follows the arterial supply. It is insured by the azygos and hemiazygos veins on the thoracic side of the diaphragm, and by the inferior phrenic veins to the inferior vena cava on the abdominal side. The intercostal and internal thoracic veins are responsible for the venous drainage of the peripheral costal and sternal segments.

The diaphragmatic blood supply has the particularity of being respiratory-stage dependent: increasing during expiration which is the muscle's relaxation phase, then decreasing during inspiration, all the more when this one is forced.[8]

c. Lymphatic drainage of the diaphragm

The diaphragmatic lymphatic system consists of 3 main lymph node groups. Next to the xyphoid process is the anterior group, which drains to the parasternal nodes. The right and left lateral groups run along the phrenic nerve and drain into the posterior mediastinum nodes. The dorsal or posterior lymphatic system is located around the diaphragmatic crura, draining to the posterior mediastinal and lateral aortic nodes. [5, 6]

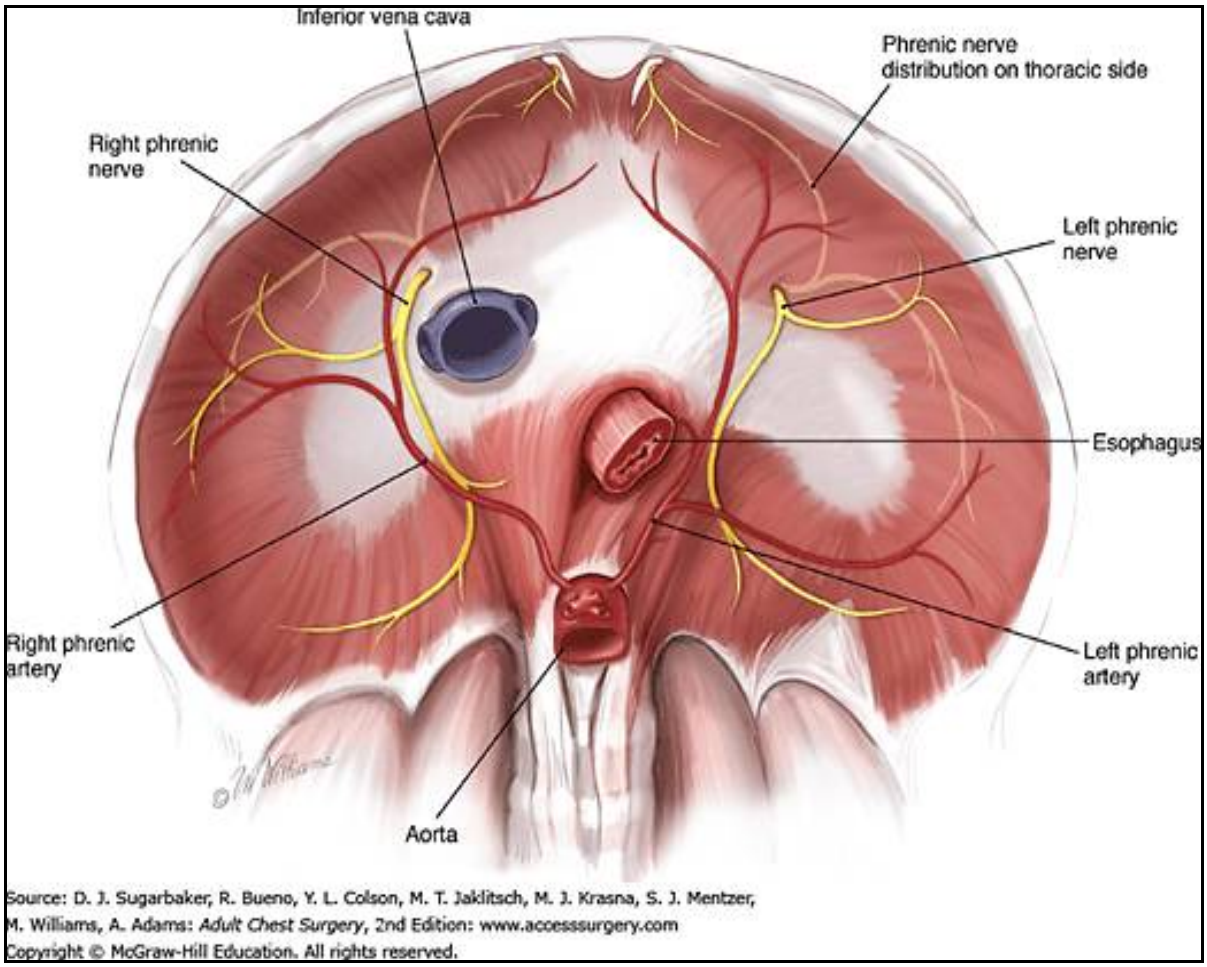


Figure 2: Blood supply and innervation on the diaphragm's abdominal surface

d. Innervation of the diaphragm

The phrenic nerve is the chief supplier of the diaphragm's innervation, although branches of the intercostal nerves can also contribute to it.

The right and left phrenic nerves are composed of motor, sensory and sympathetic fibers originating from the anterior rami of the cervical plexus nerve roots C3, C4 and C5. The nerves descend vertically, next to the internal jugular veins. In the thorax, both nerves are anterior to the lungs' hilum. They then attach to the lateral surface of the pericardium, following the same trajectory as the pericardiophrenic vessels, and providing pericardial branches. The right phrenic nerve then follows the vena cava, and enters the central tendon of the diaphragm anterolateral to the foramen vena cava. The left phrenic nerve enters the diaphragm on the left border of the heart.

Each nerve gives rise to 4 branches: sternal, anterolateral, posterolateral, and crural. The left nerve then passes through the hiatus esophagus innerving the peritoneum and upper abdominal structures. It is important to note that these branches are commonly not visible, since deeply implanted in the muscle. The phrenic nerve and its branches provide total motor innervation to the diaphragmatic domes, leading to the muscle's contraction during inspiration and its relaxation during expiration. The phrenic nerves also provide sensitive innervation to the central tendon. The sensory innervation is completed by the intercostal nerves. [5, 6, 9]

An accessory phrenic nerve can be found in more than 60% cases, arising most often from the subclavian nerve.[10]

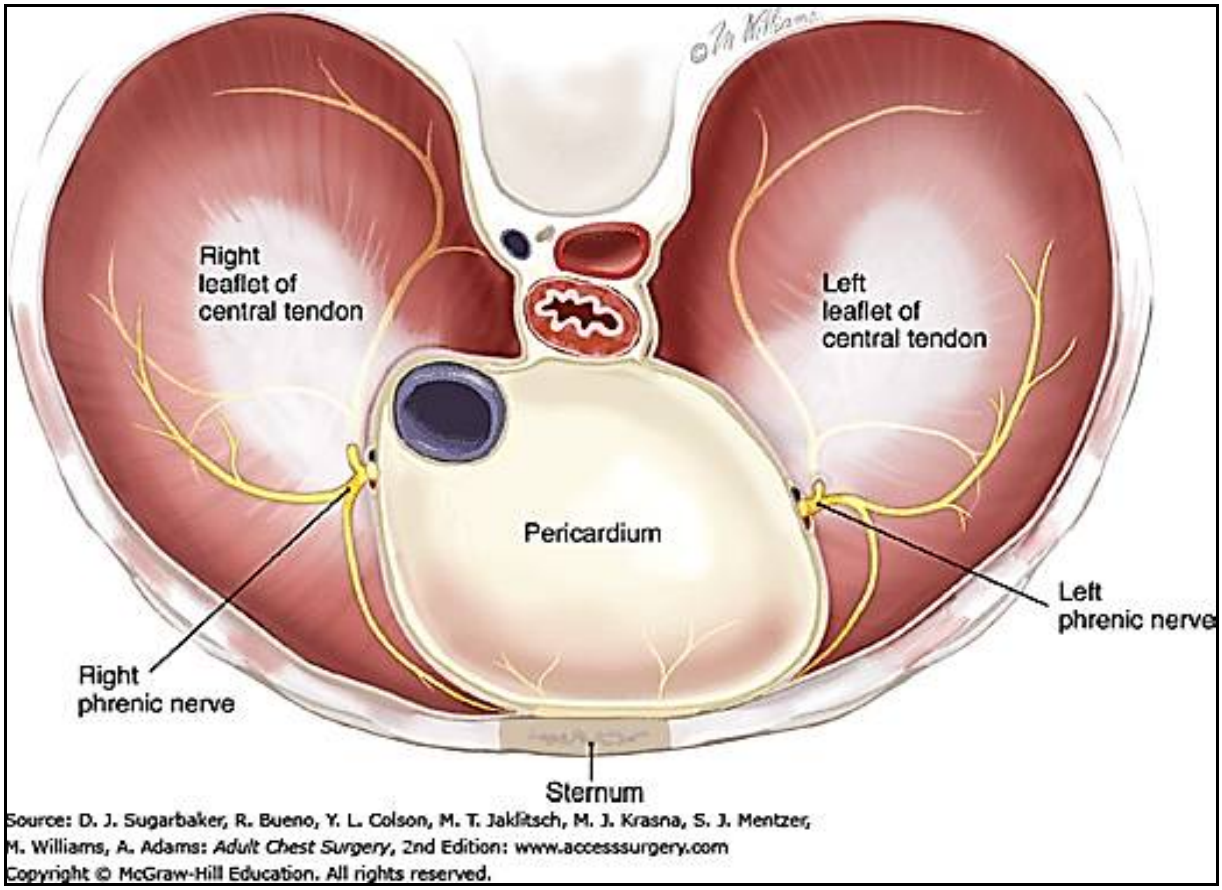


Figure 3: Thoracic view of the diaphragm showing its innervation

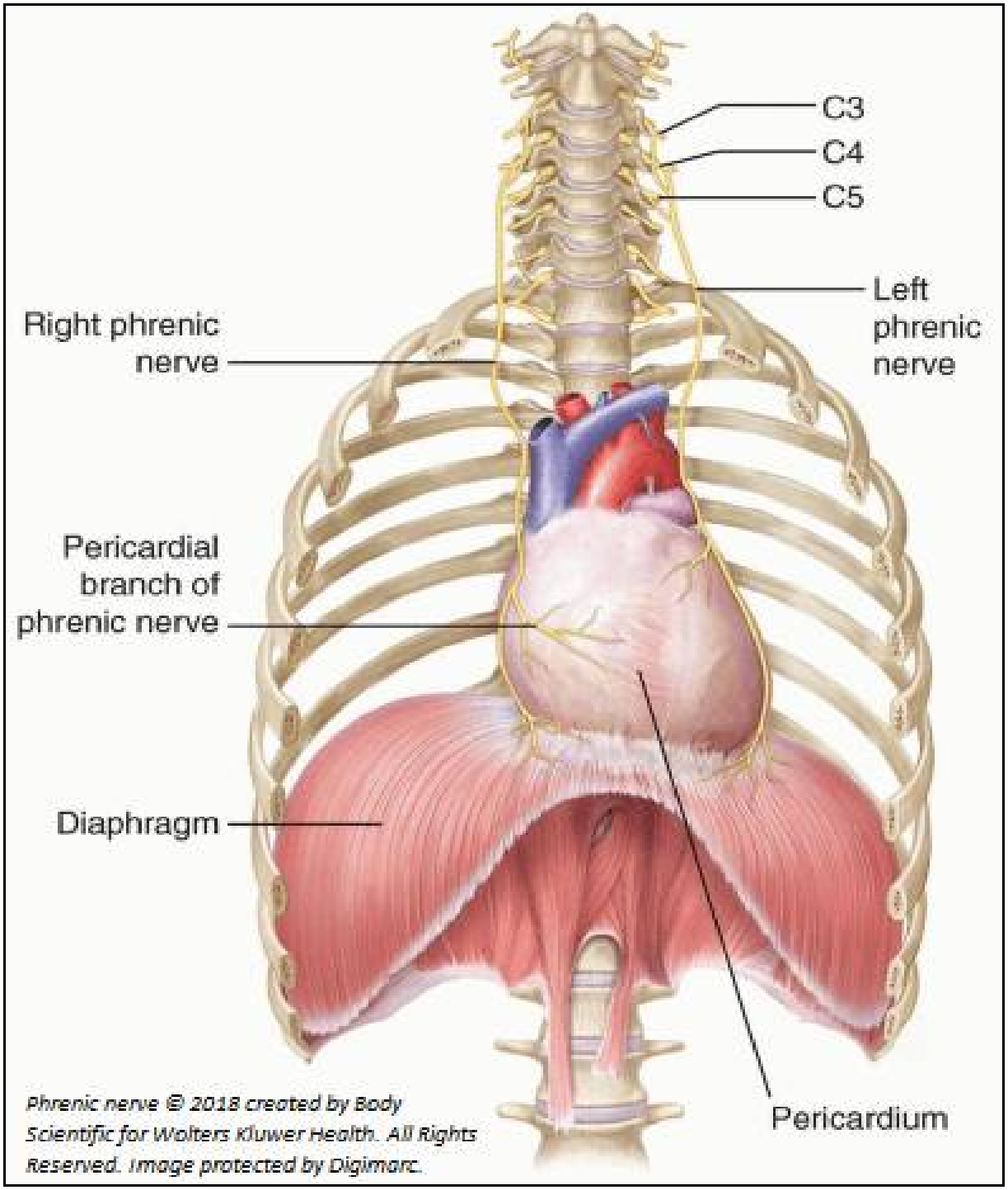


Figure 4: The anatomical course of the phrenic nerves

e. Openings in the diaphragm

The diaphragm is characterized by the passage of major structures between the thoracic and abdominal cavities, through three main openings: aortic, esophageal and vena cava. In addition to these, the triangular gaps between the sternal and costal portions of the diaphragm, and the ones between the lateral arcuate ligaments and costal diaphragm act as minor orifices.

The inferior vena cava foramen lies on the right leaf of the central tendon at vertebrae's T8-9 level. It's only bounded by tendinous tissue. In addition to the inferior vena cava, this orifice also allows passage to the right phrenic nerve and lymphatics.

The esophageal hiatus is located behind the central tendon, slightly left to the midline and anterior to the aortic hiatus, at T10's level. It has the particularity of being formed by the muscular fibers of the right crus anterolaterally, and those from the median arcuate ligament posteriorly. This helps closing the inferior esophagus during diaphragmatic contraction. The esophageal orifice transmits the esophagus, both the anterior and posterior vagal trunks, the phrenicoabdominal branch of the left phrenic nerve and branches of the left gastric artery and vein.

The aortic hiatus is anterior to the body of T12-L1 which constitute its dorsal border, while the crural diaphragm acts as its lateral ones and the median arcuate ligaments as the anterior one. It transmits the aorta, the aortic plexus, the thoracic duct, lymphatics and sometimes the azygos vein.

The Larrey and Morgani gaps are the crossing point of the internal thoracic arteries and veins, which then become the superior epigastric artery and vein, accompanied by a few lymphatics.

The medial and lateral clefts respectively allow passage of the greater splanchnic nerve, azygos and hemiazygos veins for the first, and the truncus sympathicus for the second. [5, 6]

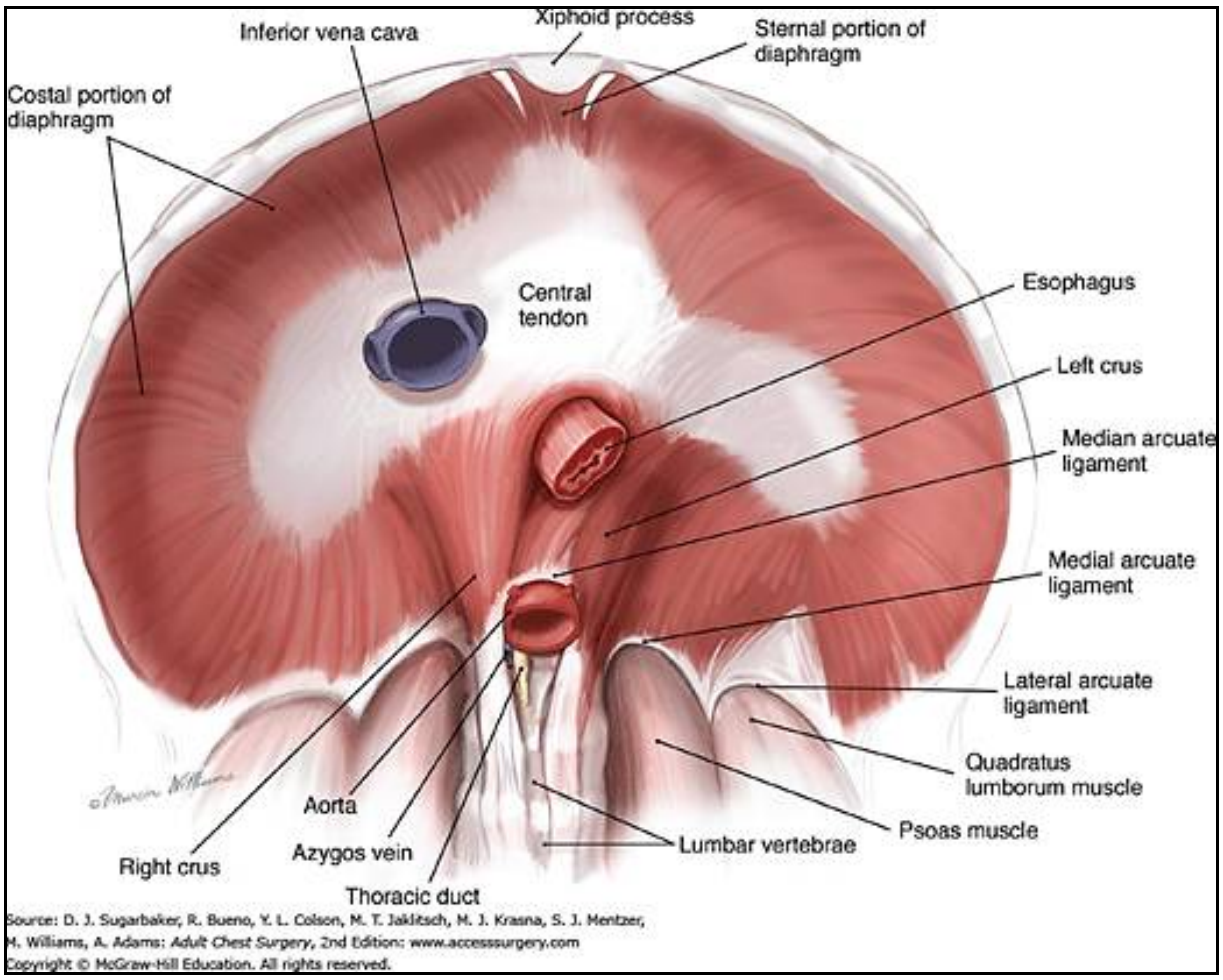


Figure 5: Abdominal view of the diaphragm showing its openings

2. Physiology of the diaphragm

The diaphragm plays a major role both in respiration, being the primary ventilatory muscle, and gastroesophageal functions such as emesis, esophageal emptying and anti-reflux. It also contributes to expulsive acts, since a deep inspiration is often taken before coughing, sneezing, laughing or defecating. Moreover, the diaphragm provides thoracic and abdominal organs with anatomic stability.[5]

The diaphragm should be seen as two main distinct muscles: crural and costal, synchronous throughout respiration but with different gastroesophageal functions.[11]

a. Contractile properties

As seen in the anatomical reminder, the diaphragm is a composite structure with a central collagenous tendon, relatively inelastic, and a contractile elastic muscular part.[12]

The diaphragm's peripheral portion is made of striated muscles, and thus presents characteristic responses to stimulation. The stimulation being, in this case, controlled by autonomic and voluntary neural pathways provided by the phrenic nerve. In vivo, measuring the speed of a contraction and relaxation of the human diaphragm, secondary to phrenic nerve stimulation, shows the following results: a TPT of 70ms and ½ RT of 67ms, where TPT: the time to peak tension is the speed of one twitch contraction, and ½ RT: one half relaxation time is the time required for peak tension to fall by half.[5, 13]

b. Role in ventilation

The diaphragm is mainly an inspiratory muscle, this function is realized through many mechanisms, influenced by the cranio-caudal position of the diaphragm and the zone of apposition between the diaphragm and the rib cage. The first mechanism results from the muscle fibers' contraction and shortening, which pulls the central tendon down resulting in the expansion of the chest volumes. Simultaneously, the domes push the abdominal organs down while descending, thus increasing the intra-abdominal pressure.

The apposition zone transmits this elevated abdominal pressure, which pushes the lower ribs outward and expands the thorax. The costal portion of the diaphragm provides the major musculature for this inspiratory phase.[5] (Fig.6)

Breathing is a continuous process, the diaphragm's work, like the heart's, is an endurance one, based on lifelong repetitive contractions. [14] The composition of the diaphragm fits this task perfectly. In adult humans, 55% of the fibers are type I oxidative, slow twitch ones, highly resistant to fatigue. The remaining fibers are type II fast twitch ones, with approximately 25% of the oxidative, glycolytic intermediate type, which is susceptible to fatigue though still resistant, and around 20% of the rapid glycolytic one, which tires easily. These type II fibers are only recruited when the breathing rate increases. [13]

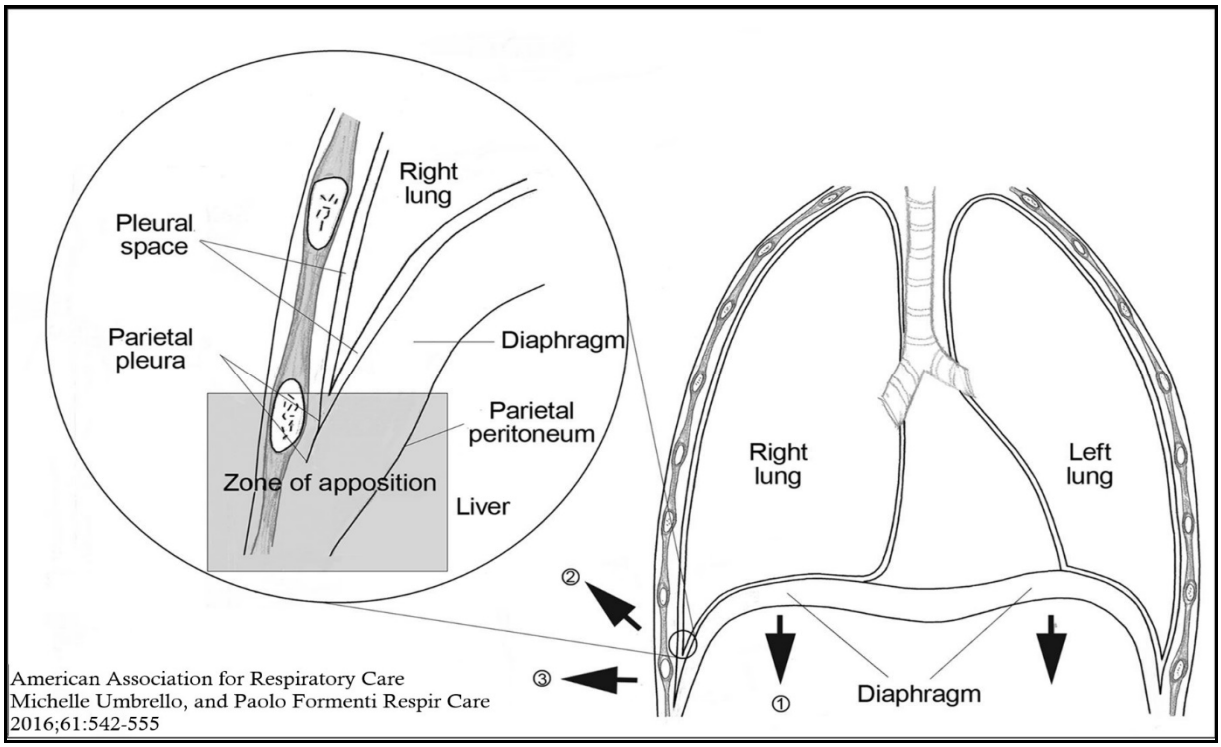
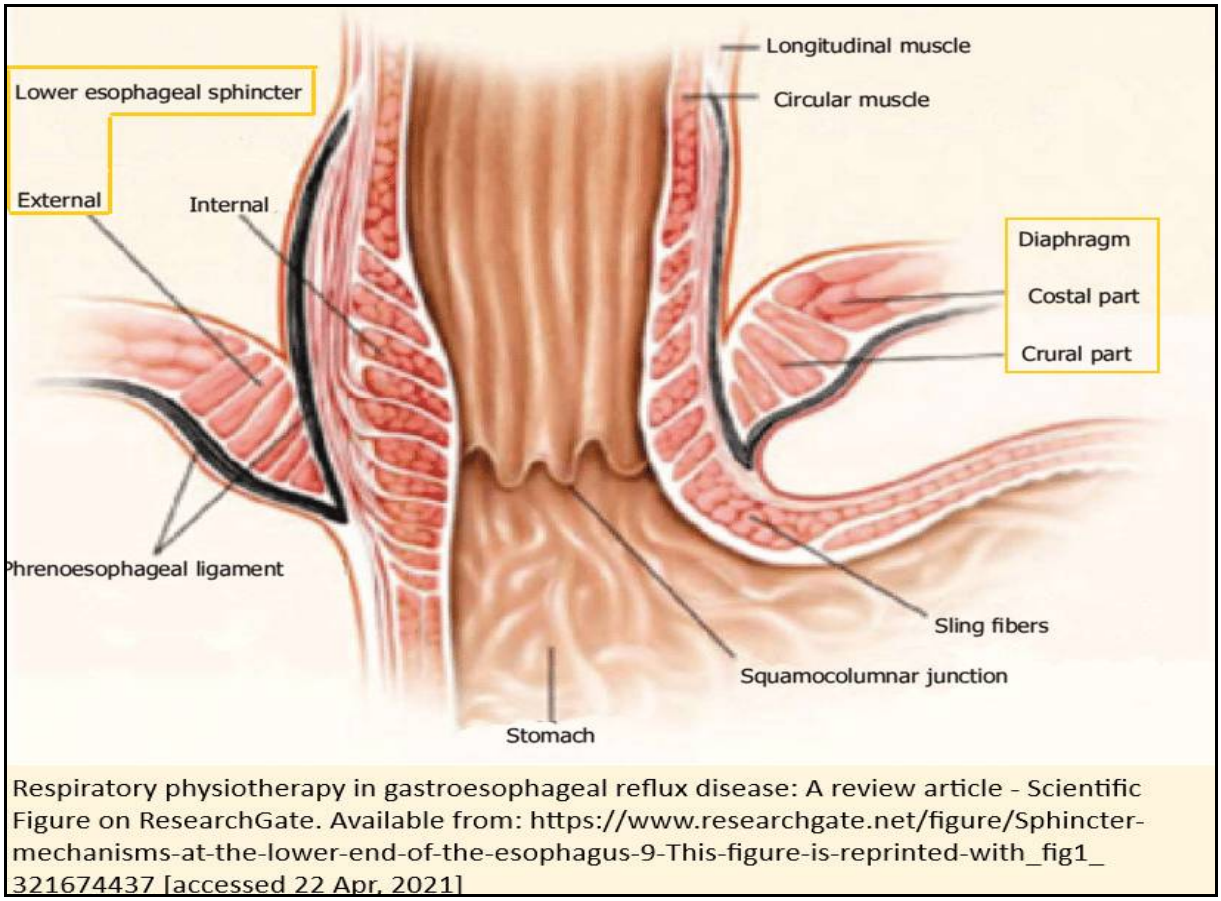


Figure 6: Schematic view of the diaphragm and the zone of apposition

The arrows in the schema represent the forces acting on the structures. (1) Shortening of diaphragmatic fibers and caudal movement of the diaphragm during inspiration. The diaphragmatic contraction lowers the pleural pressure and increases the abdominal one, leading to (2) an inflationary effect on the lungs produced by the reduction in pleural pressures, and (3) the expansion of the rib cage secondary to the increased abdominal pressure.

c. Role in gastroesophageal functions

The crural diaphragm's function in respiration is relatively small. In the gastroesophageal area though, it acts as an external sphincter, which prevents gastroesophageal reflux and relaxes to allow the smooth passage of food bolus from the esophagus to the stomach during the swallowing process. It also plays an important part in emesis, which involves the contraction of the diaphragm along with the abdominal muscles, gastrointestinal tract, and other respiratory muscles.[5, 11]



Respiratory physiotherapy in gastroesophageal reflux disease: A review article - Scientific Figure on ResearchGate. Available from: https://www.researchgate.net/figure/Sphincter-mechanisms-at-the-lower-end-of-the-esophagus-9-This-figure-is-reprinted-with_fig1_321674437 [accessed 22 Apr, 2021]

Figure 7: Schematic view of the lower esophageal sphincter

Materials and methods

1. The study type

This work is a retrospective descriptive case series study, based on the comparison of therapeutic results between two groups of patients, A and B. The study uses statistical analysis to show the significance of all results obtained.

2. The study period

The study concerned patients seen and treated between January 2010 and December 2018.

3. The study location

The study took place at the Thoracic surgery Department of the Mohammed V Military Hospital, Rabat, Morocco.

4. Inclusion criteria

Were concerned all adult patients diagnosed with a symptomatic diaphragmatic eventration. Clinically, this included patients presenting chronic progressive dyspnea, palpitations, or chronic digestive symptoms. All the therapeutic measures and follow-ups were performed at our structure during the study's period of time.

5. Exclusion criteria

Reasons for exclusion from the study included poor general state, and eventual associated malignant tumor. Were also excluded, patients with a highly limited respiratory function.

6. Data collection

a. Patients' characteristics

i. Demographic characteristics

- Sex: Male or Female
- Age

ii. Clinical data

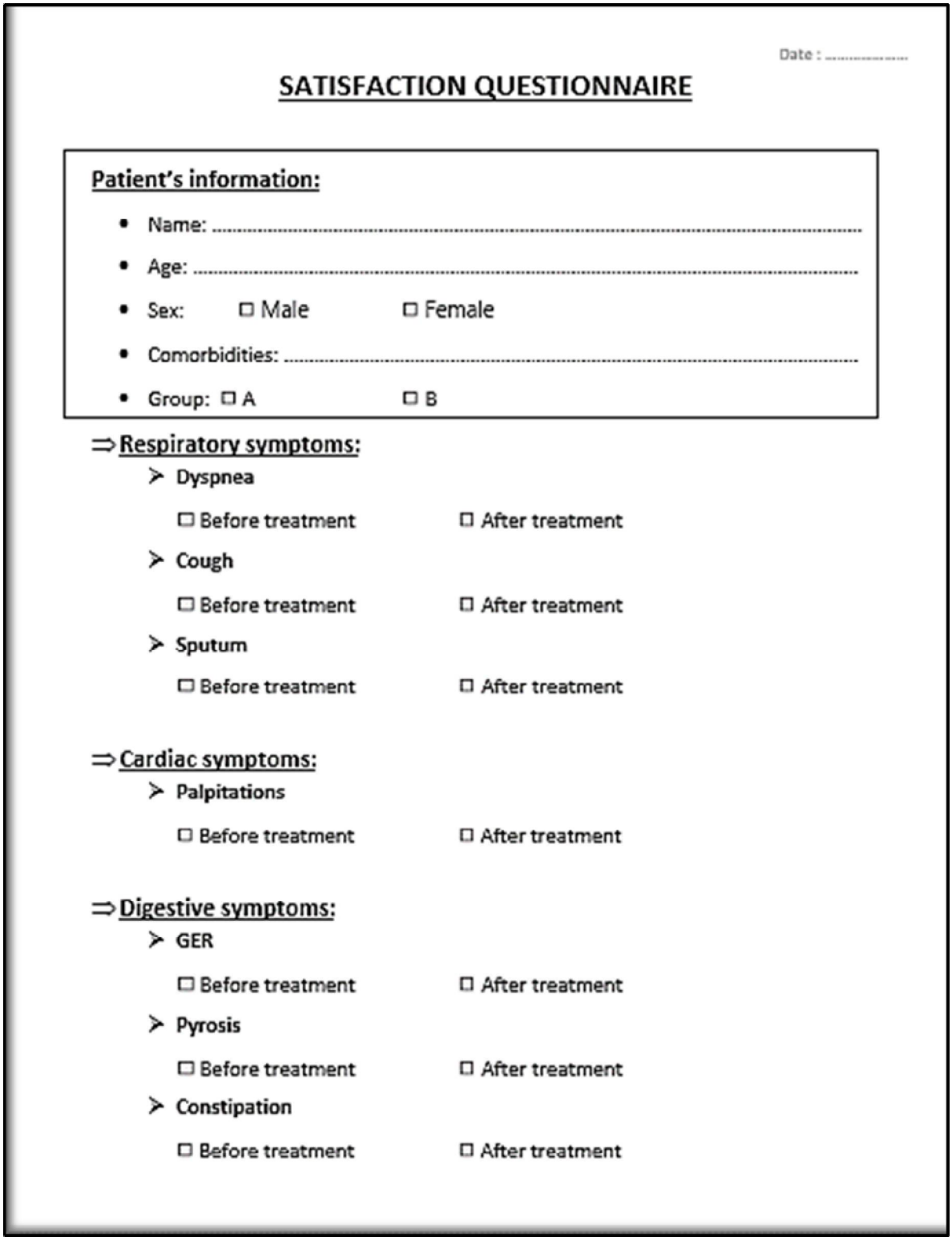
- Body Mass Index
- History of smoking
- Medical history of chest surgery or traumatism
- Comorbidities: COPD, asthma, diabetes
- Eventration side: right, left or bilateral
- Clinical symptoms:
  - Respiratory
  - Digestive
  - Cardiac

b. Instruments

All patients included in the study underwent a chest X-ray and a thoracic CT scan. In some cases, an additional MRI was indicated because of the suspicion of a diaphragmatic rupture. Respiratory functional tests (FEV1 and FVC) were performed for all cases, before treatment and 03 months after the chosen therapeutic procedure. Were also compared, the radiological levels of DE between the pre- and post-treatment phases.

The patients were divided in two groups, depending on the received treatment. Group A included symptomatic DE patients whose treatment was exclusively medical using physical therapy. While Group B's patients all received surgical plication of the diaphragm in addition to physical therapy.

The clinical improvement and regression of respiratory and digestive symptoms were measured using a simplified satisfaction questionnaire.



Date : .....

SATISFACTION QUESTIONNAIRE

Patient's information:

- Name: .....
- Age: .....
- Sex: ☐ Male ☐ Female
- Comorbidities: .....
- Group: ☐ A ☐ B

⇒ **Respiratory symptoms:**

➢ Dyspnea

☐ Before treatment ☐ After treatment

➢ Cough

☐ Before treatment ☐ After treatment

➢ Sputum

☐ Before treatment ☐ After treatment

⇒ **Cardiac symptoms:**

➢ Palpitations

☐ Before treatment ☐ After treatment

⇒ **Digestive symptoms:**

➢ GER

☐ Before treatment ☐ After treatment

➢ Pyrosis

☐ Before treatment ☐ After treatment

➢ Constipation

☐ Before treatment ☐ After treatment

Figure 8: Copy of the satisfaction questionnaire used in our study

7. Surgery

a. Preanesthetic assessment

An initial preoperative assessment taking into account the particularities of a thoracic surgery (lateral decubitus position, one-lung ventilation, an open thorax) was performed for all patients. It included:

- History, comorbidities and physical examination
- Known allergies
- Biological assessment, oriented by the clinical examination and history: complete blood count, electrolytes and creatinine, INR and aPTT, fasting glucose…
- Electrocardiograph
- Chest radiograph
- An assessment of the patient's physical status using the ASA (American Society of Anesthesiologists) classification system.
- Pulmonary function tests: FVC, FEV1 with the calculation of ppoFEV1, and DLCO with the estimation of ppoDLCO.
- Discussing postoperative pain management.

b. Operative indications

The patients that were candidates for surgery, had to present a surgical indication, principally:

- Chronic disabling dyspnea
- Visible lung destruction

- Chronic digestive symptoms
- Chronic cardiac symptoms

At least one of these indications was present in all of group B's patients.

c. Surgical approach

All operated patients underwent diaphragmatic plication. They were put under general anesthesia, with a double-lumen endotracheal tube allowing selective lung ventilation. The patients were then put in a lateral decubitus position.

The approach was a diaphragmatic plication through classical posterolateral thoracotomy in most cases. The choice of this more invasive surgical approach was justified by the presence of important pleural adhesions in the majority of patients, which developed due to the delay in diagnosis. Even though a minimally invasive thoracoscopic approach was possible in some cases.

Traditional open transthoracic plication as performed in our unit, began with an incision through the 7^{th} or 8^{th} intercostal space, allowing an examination of the lung and mediastinum, thus confirming the DE and excluding unexpected etiologies. Then the procedure started, based on creating pleats in the weakened diaphragm using U-stitches. Beginning on the central tendinous portion of the diaphragm, seven to nine lines of silk sutures were put, going from the posteromedial to the anterolateral parts, while crossing the phrenic nerve's fibers. Repositioning the diaphragm in a central position was achieved using hanged sutures and parallel plication.

Additional reinforcement was necessary in some cases and achieved by using a prosthetic plate.

The surgery ended with the insertion of a 28-32Fr intercostal chest tube that was left in place.

d. Postoperative course

i. Clinical monitoring

Standard surveillance of vitals, temperature and the surgical wounds were daily assessed.

Pain management was a major aspect of the post-operative course. Systemic analgesics using the pain ladders set by the WHO were used to find the most effective symptomatic treatment. Opioids were avoided as much as possible, and used in association with other analgesics when needed, to prevent side-effects, mainly respiratory depression.

ii. Chest tube management

The chest tube was only kept for a short period of time and usually removed 2 to 3 days after the operation. A negative suction was applied and maintained as long as the tube was kept.

iii. Respiratory physiotherapy

Physiotherapy was started as soon as the state of the patient allowed it.

iv. Biological and radiological assessments

Oriented by the clinical surveillance, biological and radiological exams were prescribed when needed, and if a complication was suspected.

e. Follow up

The first follow-up examination was programmed 10 days after the surgery, then the rhythm followed was: after 1 month, then 6 months, then a year after surgery. The clinical state of the patient was assessed at every appointment, the reappearance of symptoms possibly indicating a recurrence, and orienting towards further explorations.

8. Statistical analysis

Our study aims to show the results of surgical plication as a treatment for symptomatic DE. The question being whether it can constitute a more effective therapeutic approach than the medical one, which is based on physical therapy.

All values were expressed in the text and tables as “mean±SD”.

Unpaired t-test, Fisher exact test, and $\chi 2$ test were used for group differences.

A p value inferior to 0.05 was considered statistically significant. The data analysis was carried out using the software Statistical Package for the Social Sciences 21st version for Windows (SPSS, Inc., Chicago, IL, USA).

Results

1. Patients' flow chart

During the study period, 125 patients were diagnosed with symptomatic DE, and treated in our unit. 39 of them were excluded for various reasons (Fig.9).

Out of the 86 patients included in the study, 48 received exclusive medical treatment and 38 underwent surgical plication of the diaphragm. 7 of the group B patients were lost during follow-up.

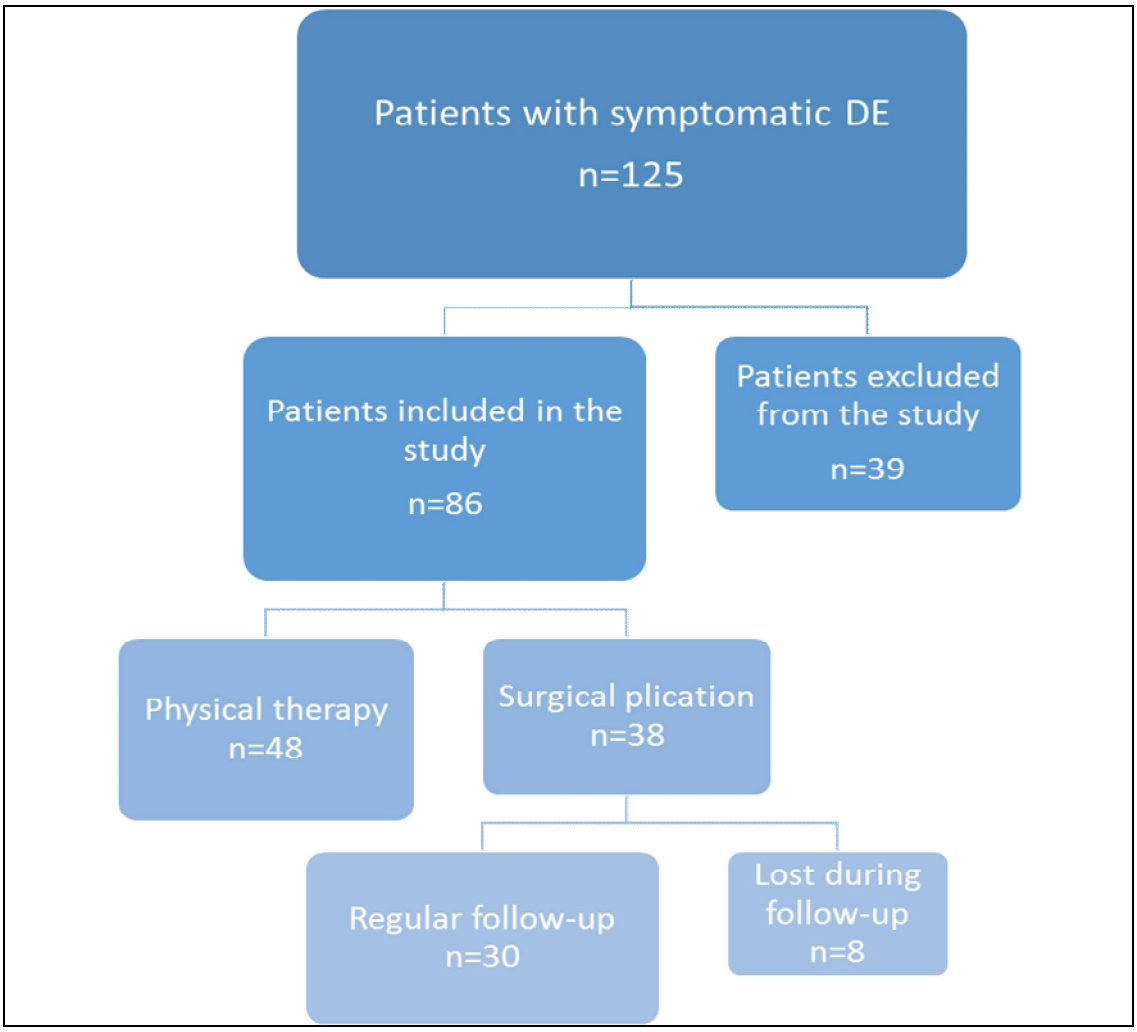


Figure 9: Flow chart of study participants

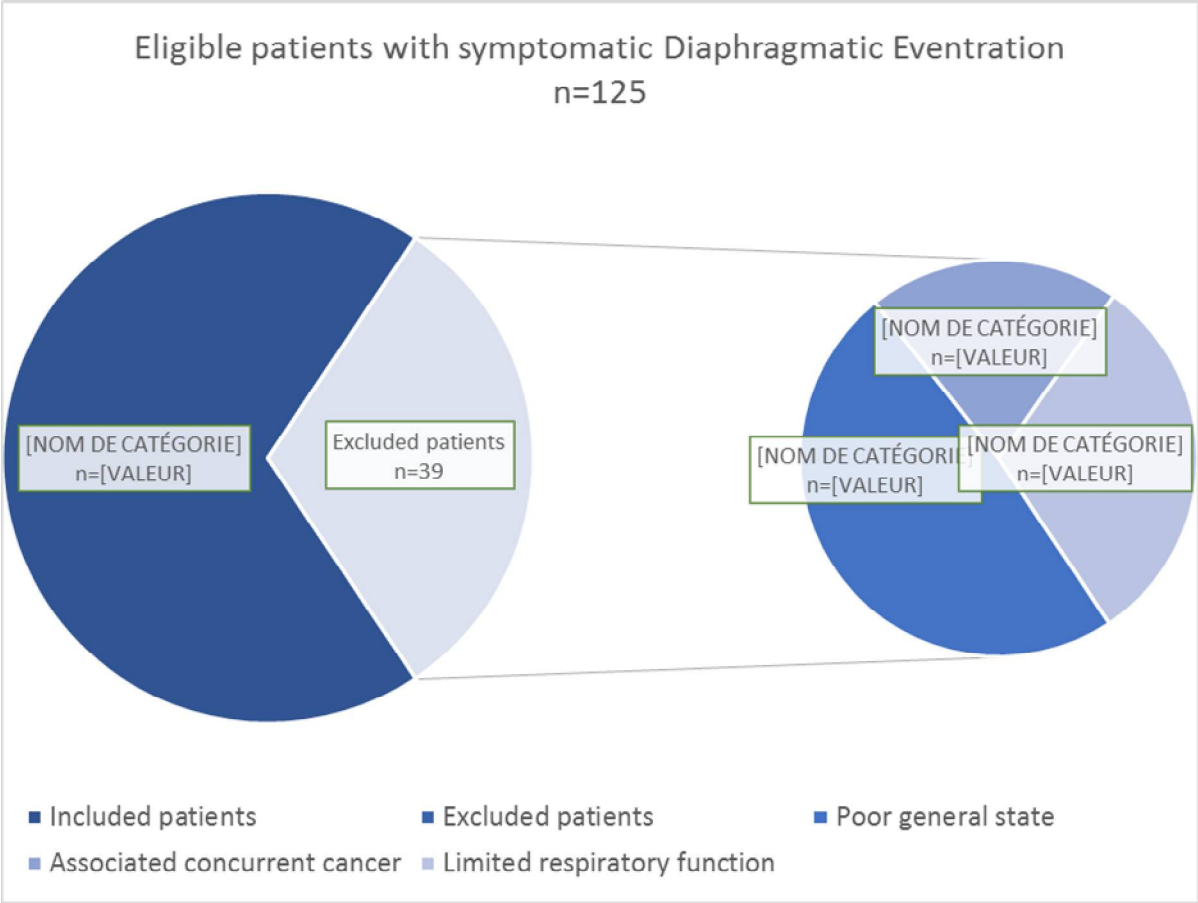


Figure 10: Diagram of included and excluded patients showing exclusion criteria

2. Studied population's characteristics

a. Sex

The gender distribution of affected patients was different in both groups. A slight male predominance was observed in group A with a gender ratio of 1.3 male per female. While a female predominance was noted in group B, with a ratio of 0.6 male per female.

The gender ratio in the whole studied group was of 0.95 male per female.

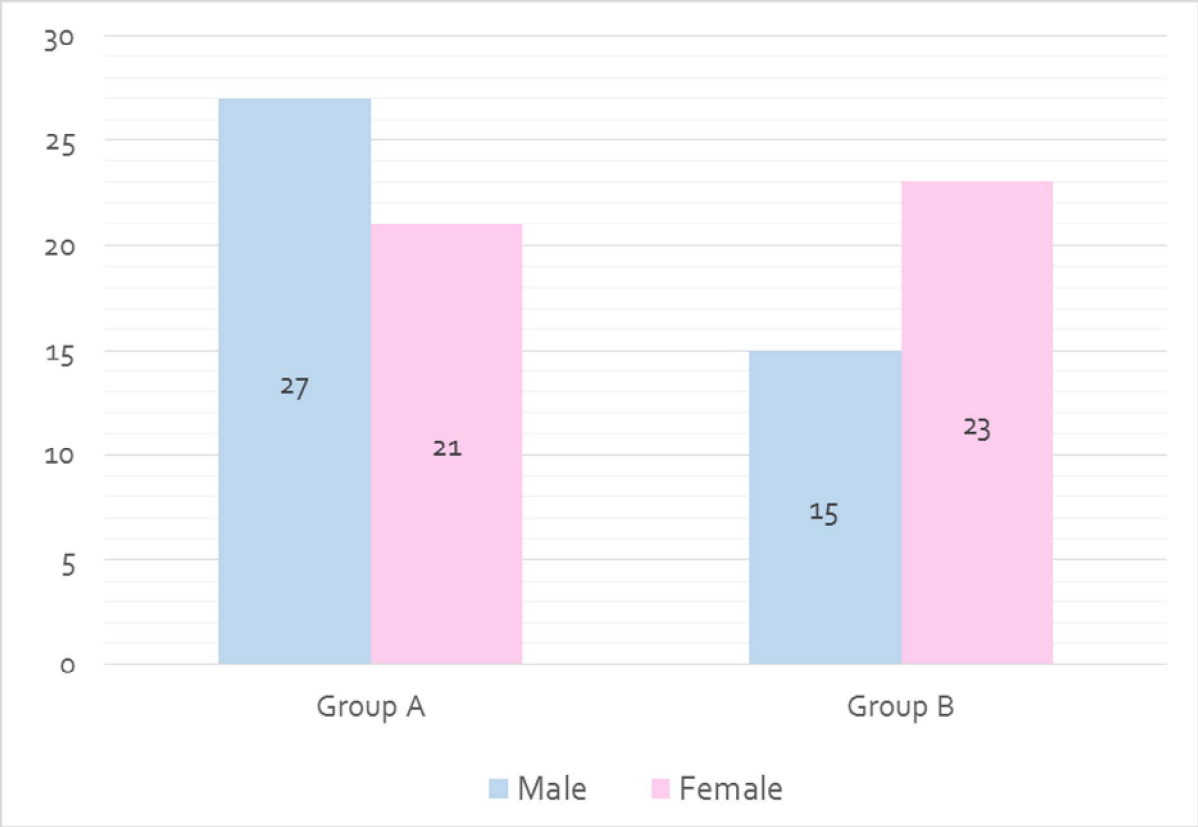


Figure 11: Diagram of the gender distribution in studied groups

b. Age

The median of age in group A patients was 42.5±5.2 years, and 46.8±3.8 years in group B patients.

c. BMI

The mean BMI of group A patients was 21.8±2.9, while it was 25.8±2.6 for group B patients.

d. Smoking history

We were able to classify patients based on their smoking history in three categories: those who never smoked (n=11 in group A, and n=21 in group B), those who ceased smoking (n=19 in group A and n=11 in group B), and the ones whose smoking history remained unknown (n=18 in group A and n=6 in group B).

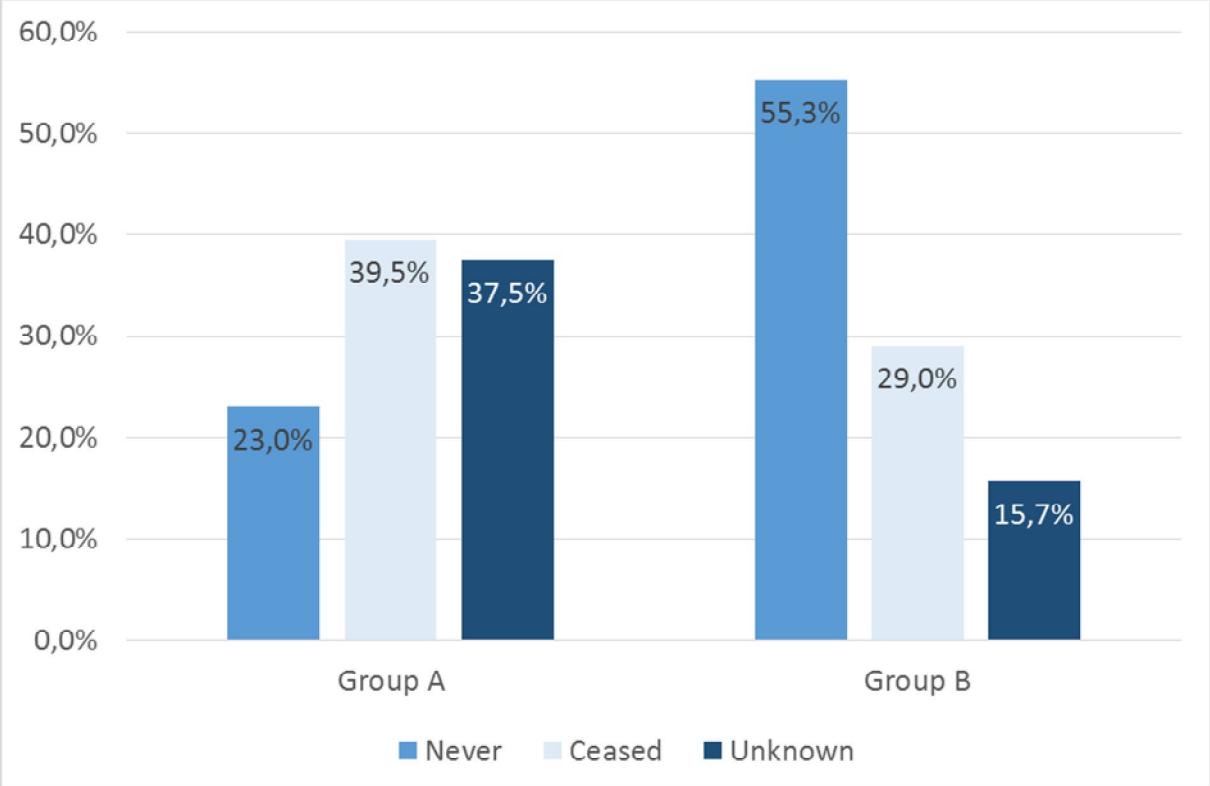


Figure 12: Diagram of the smoking history in studied groups by percentage

e. Medical history

Most patients in our study had no major medical or surgical history. Only a few (n=7) of group A's patients and none of group B's 38 patients, had a recorded medical history.

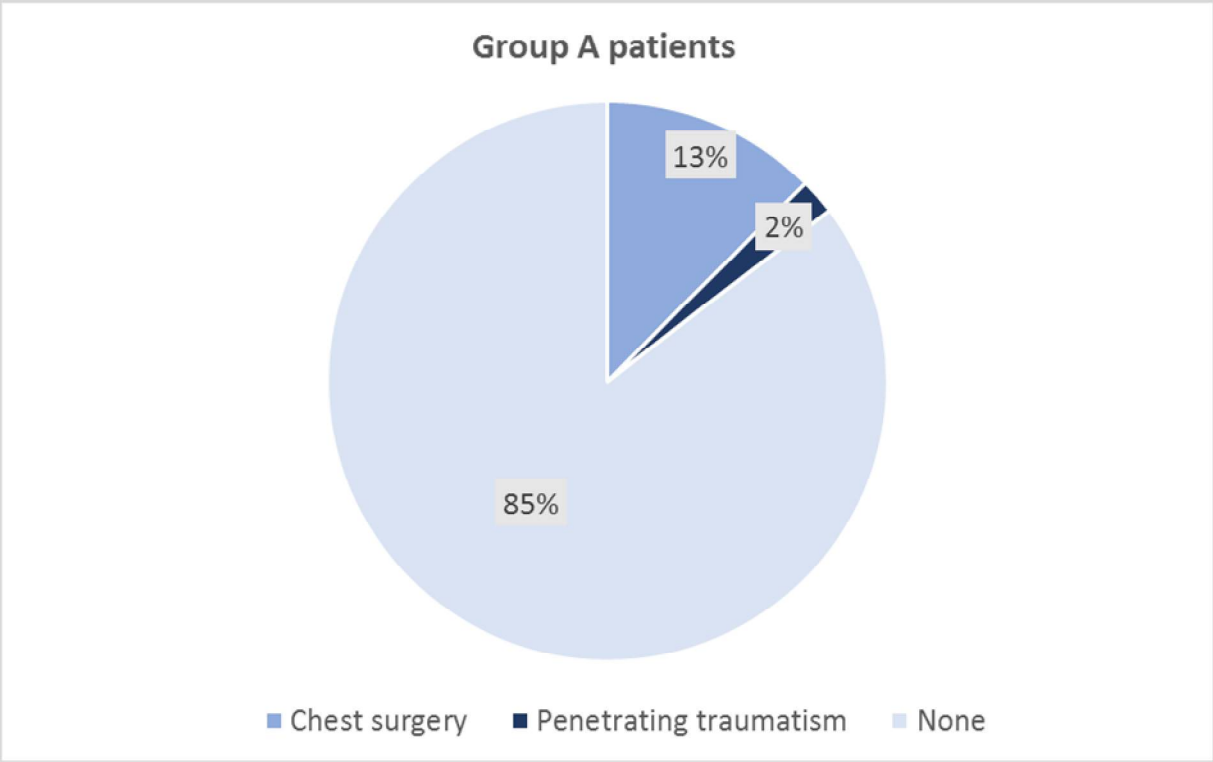


Figure 13: Diagram of group A's patients' medical history

f. Comorbidities

An associated comorbidity was found in 48% (n=23) of group A's patients, and 24% (n=9) of group B's patients.

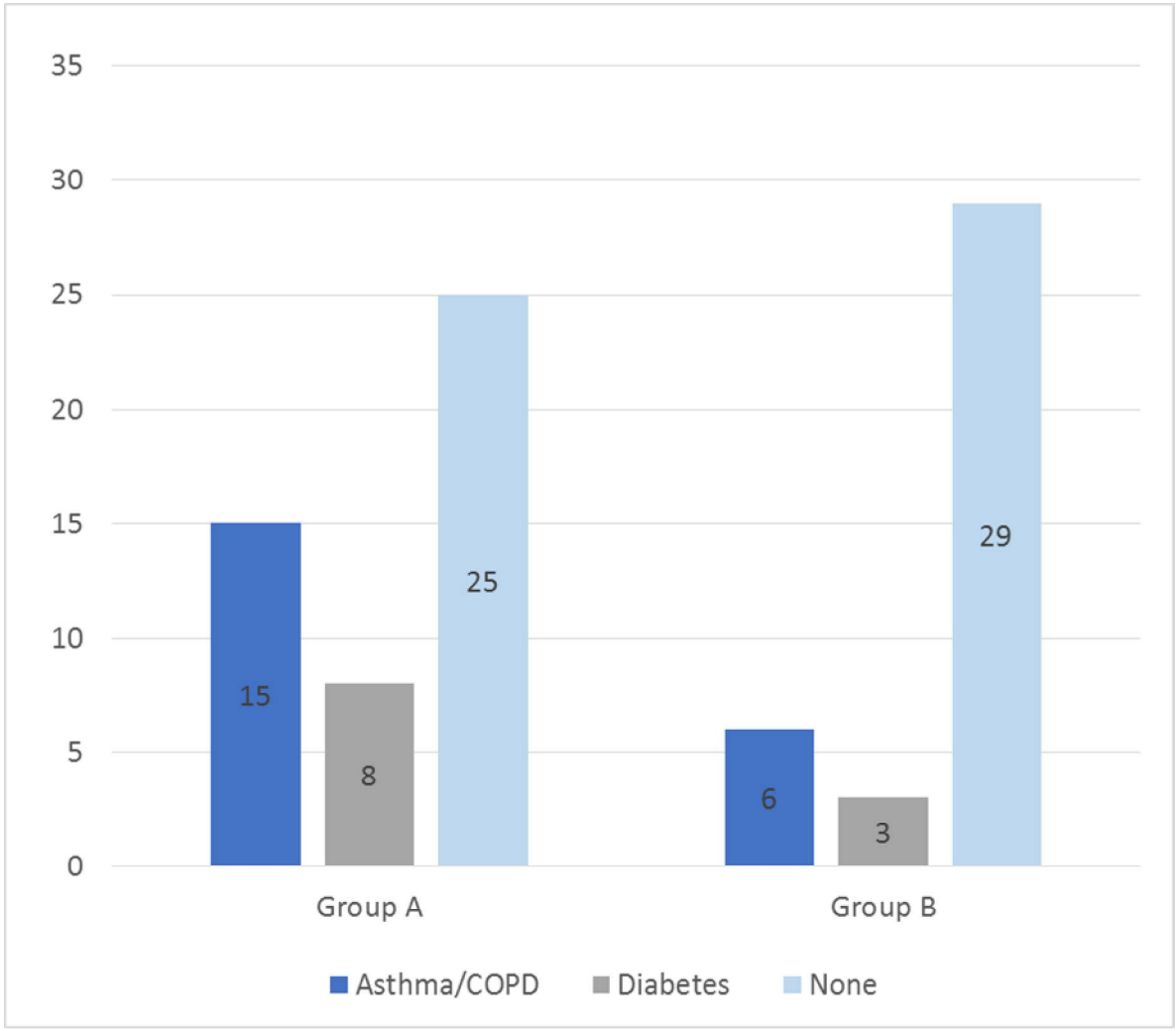


Figure 14: Diagram of the comorbidities found in the two groups' patients

g. Clinical symptoms

i. Respiratory symptoms

In both groups, respiratory symptoms were the most recurring ones:

- In group A, 46 out of the 48 patients had a respiratory clinical presentation.
- In group B, recurrent dyspnea, and orthopnea were the most common pulmonary symptoms, reported by 36 patients.

Other respiratory presentations, such as recurrent pneumonia with purulent sputum, chronic cough and thoracic pain were noted in 3 cases in group B.

ii. Digestive symptoms

Gastroesophageal reflux, pyrosis and constipation were the most commonly reported digestive symptoms:

- 30 patients from group A presented digestive manifestations.
- 31 patients from group B suffered from dyspeptic symptoms or a transit disorder.

iii. Cardiac symptoms

Patients with cardiac symptoms suffered mainly from palpitations, tachycardia, and in some cases arrythmia. These presentations were reported by:

- 16 patients from group A.
- 32 patients from group B.

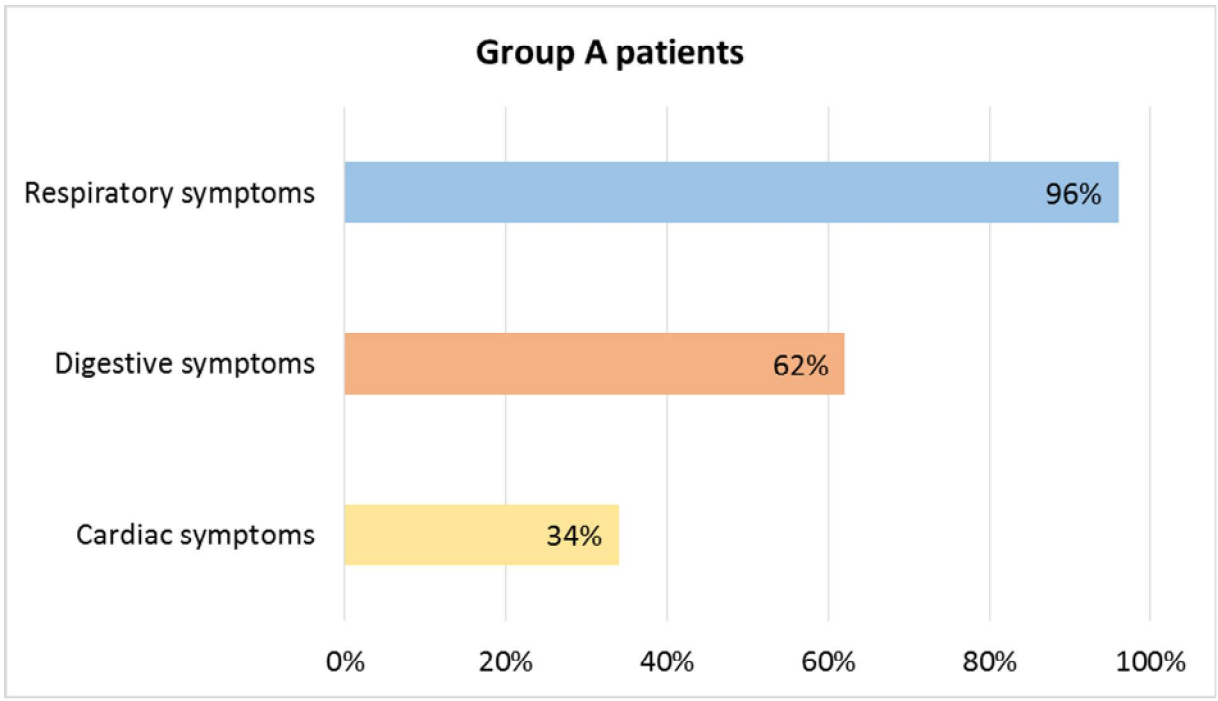


Figure 15: Diagram of clinical symptoms' distribution in group A

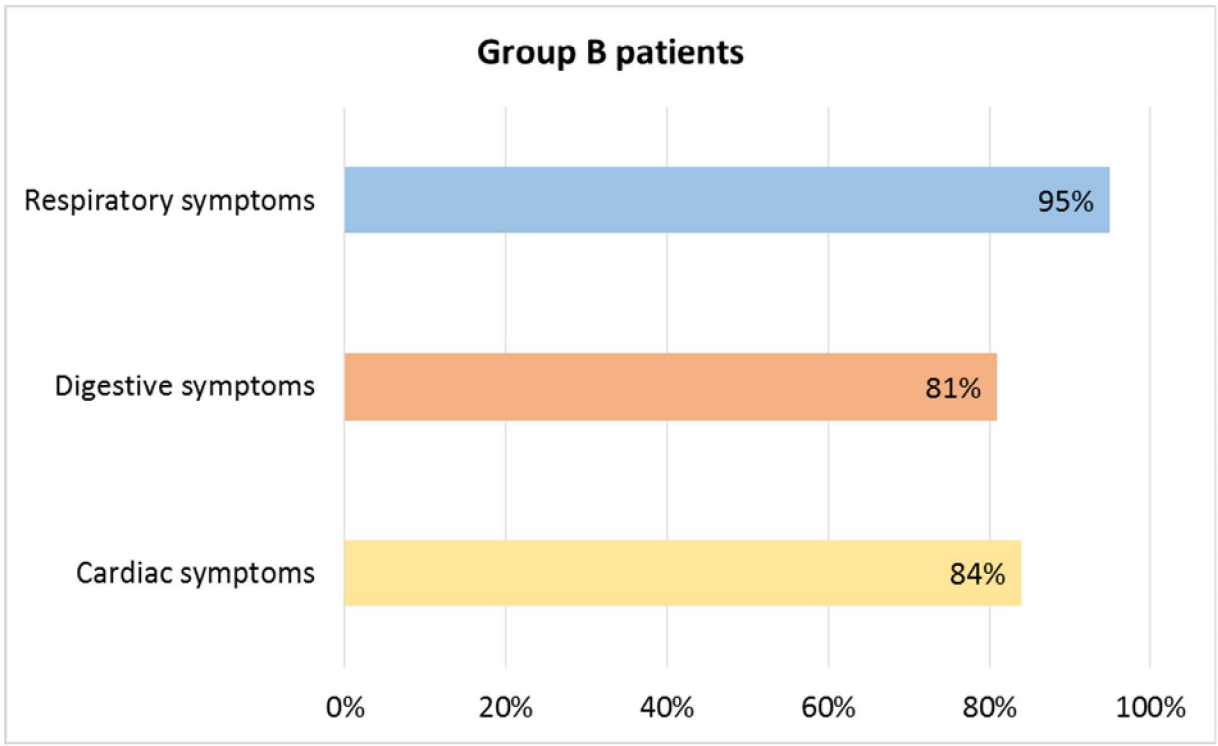


Figure 16: Diagram of clinical symptoms' distribution in group B

h. Eventration side

A significant predominance of left sided eventrations was observed in both groups: with 37 cases in group A, and 31 cases in group B. While the right side was only affected in 9 and 7 cases, respectively in groups A and B. For bilateral eventration, 2 cases were noted in group A and none in group B.

Out of a total number of 86 patients, 68 had a left sided eventration which constitutes a percentage of 79%. The right side was affected in 16 (18.6%) cases, and only 2 (2.3%) cases of bilateral eventration were observed.

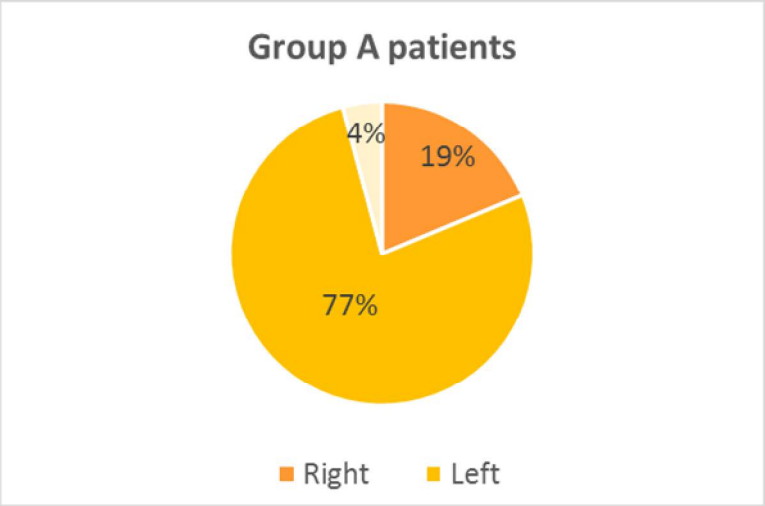


Figure 17: Diagram showing the distribution of eventration sides in group A

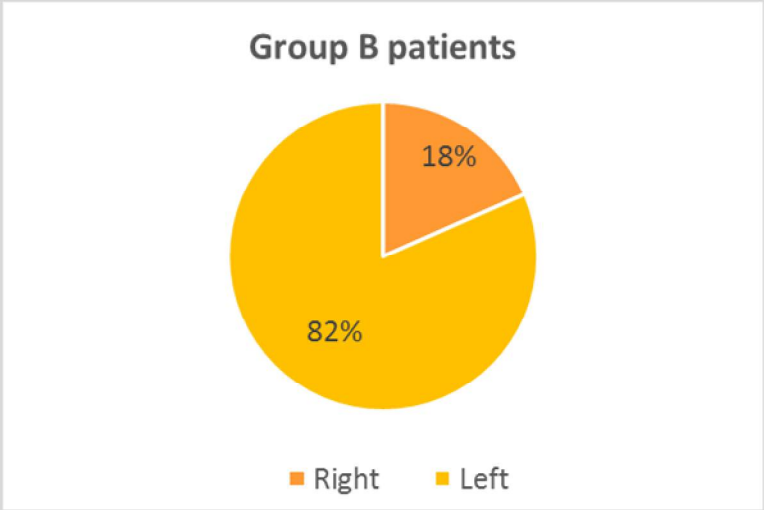


Figure 18: Diagram showing the distribution of eventration sides in group B

Table I: Recapitulation of the clinical and demographic characteristics presented by the patients with DE selected for the study

Variables		Group A (n=48)	Group B (n=38)	p value
Sex	Male	27	15	0.082
	Female	21	23	
Age (in years)		42.5±5.2	46.8±3.8	0.025
BMI (in Kg/m²)		21.8±2.9	25.8±2.6	0.035
History of smoking	Never	11	21	0.625
	Ceased	19	11	0.269
	Unknown	18	05	0.063
Medical history	Chest surgery	06	00	-
	Penetrating traumatism	01	00	
Comorbidities	Asthma, COPD	15	06	0.025
	Diabetes	08	03	0.086
Eventration side	Right	09	07	0.456
	Left	37	31	0.247
	Bilateral	02	00	0.350

3. Instruments

a. Radiology

i. Chest X-ray

Chest radiographs were the first step in the paraclinical assessment of DE and showed the side and extent of it.

Chest imaging showed the elevated hemidiaphragm, and allowed to later compare the degree of eventration between the pre- and post-therapeutic phases.

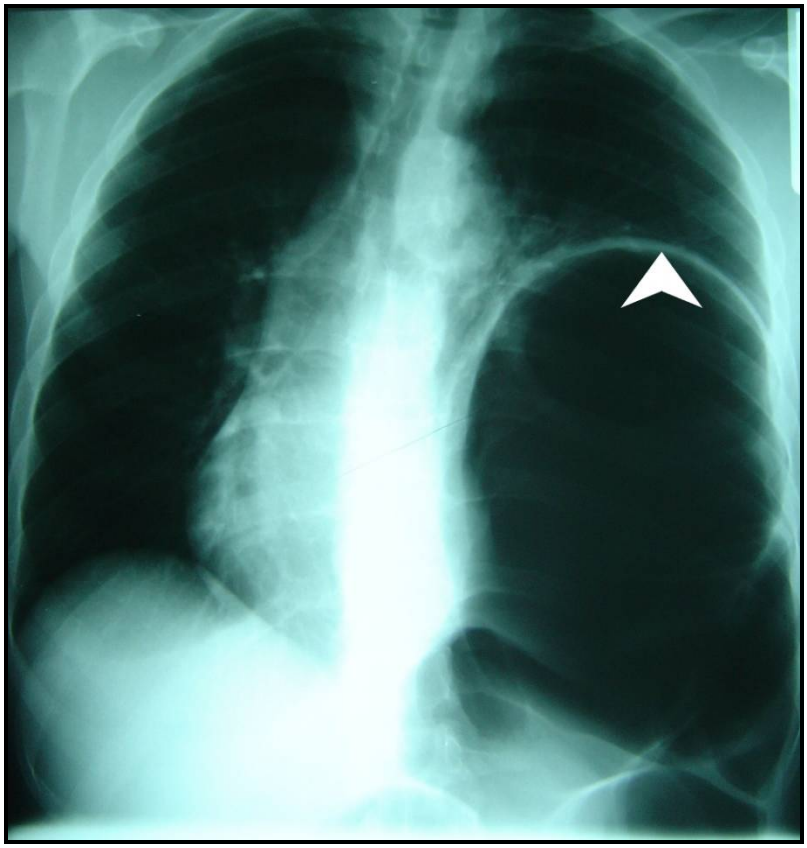


Figure 19: Antero-posterior chest radiograph showing left elevated hemidiaphragm (arrow) with ascension of the stomach and colon

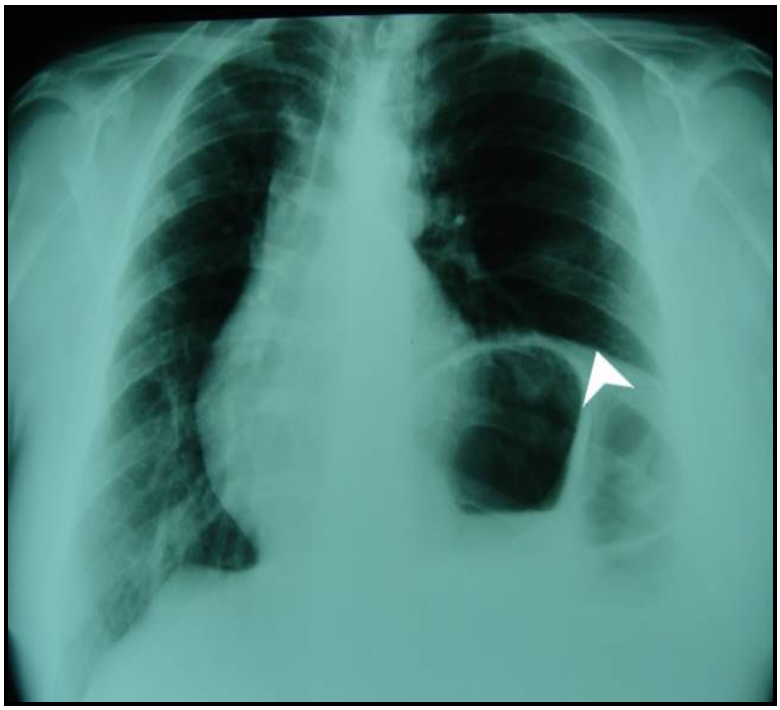


Figure 20: Antero-posterior chest radiograph showing a left elevated hemidiaphragm (arrow) with an ascension of the stomach

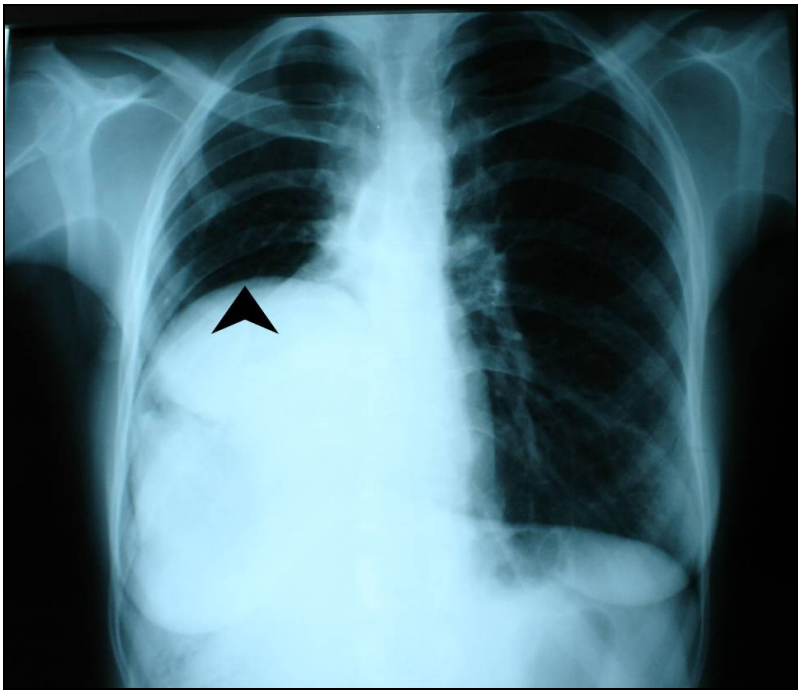


Figure 21: Antero-posterior chest radiograph showing a right elevated hemidiaphragm (arrow) with an ascension of the liver

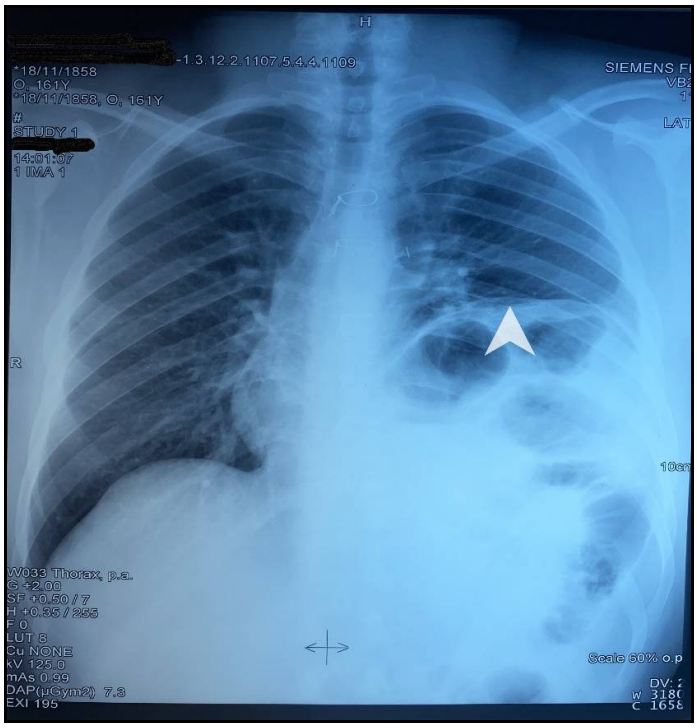


Figure 22: Preoperative anteroposterior chest X-ray showing an elevated left hemidiaphragm (arrow) with an ascension of the stomach and colon

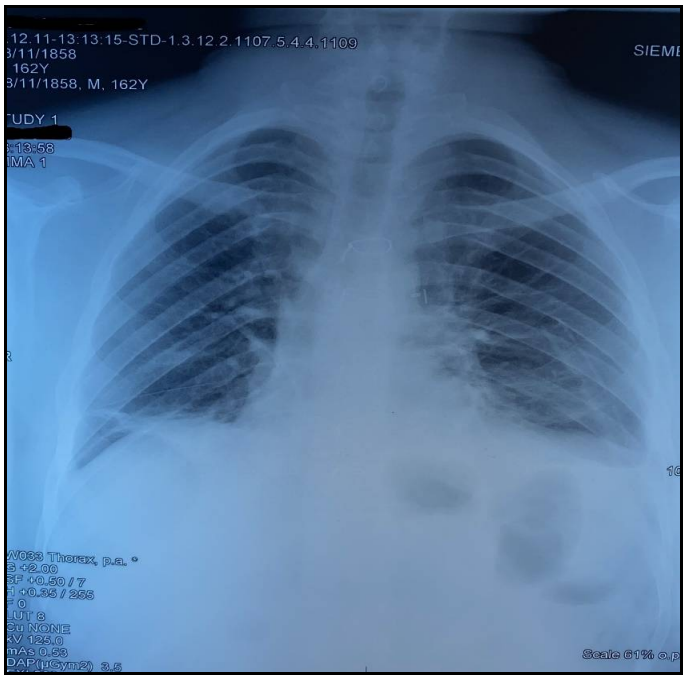


Figure 23: Postoperative anteroposterior chest X-ray of the same patient from Fig.22 after a thoracoscopic diaphragm plication

Fig.22 shows an elevation of the left hemidiaphragm following a thymectomy in a patient who was suffering from a thymic tumor. During the surgical treatment of the neoplastic pathology, the left phrenic nerve was sectioned. At follow-up, the patient started presenting respiratory symptoms, mainly dyspnea, and the radiological presentation in Fig.22 was found. After a surgical plication of the diaphragm through a thoracoscopic approach, the result showed in Fig.23 was obtained.

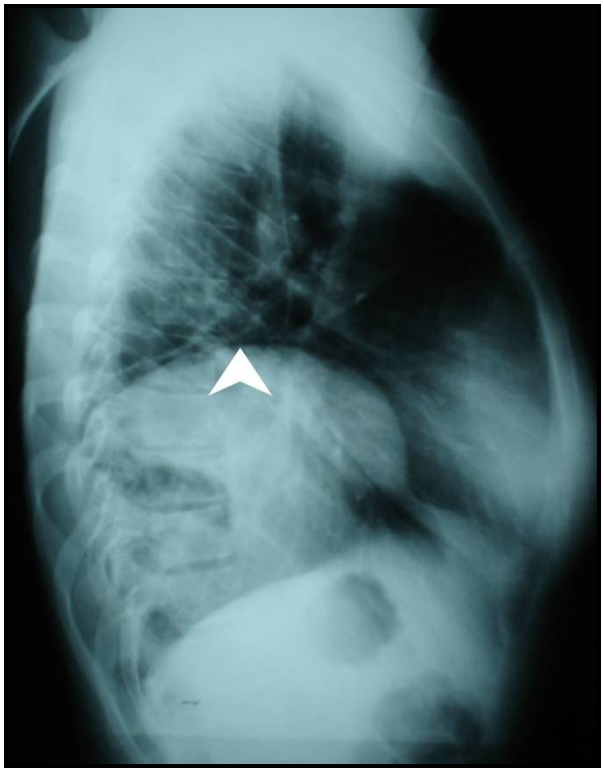


Figure 24: Lateral chest radiograph showing an elevated right hemidiaphragm (arrow)

ii. CT scan

A chest CT-scan was performed for all patients, it gave a better view of the dysfunctional diaphragm and of the abdominal organs that migrated cephalically. It also allowed the elimination of possible differential diagnosis.

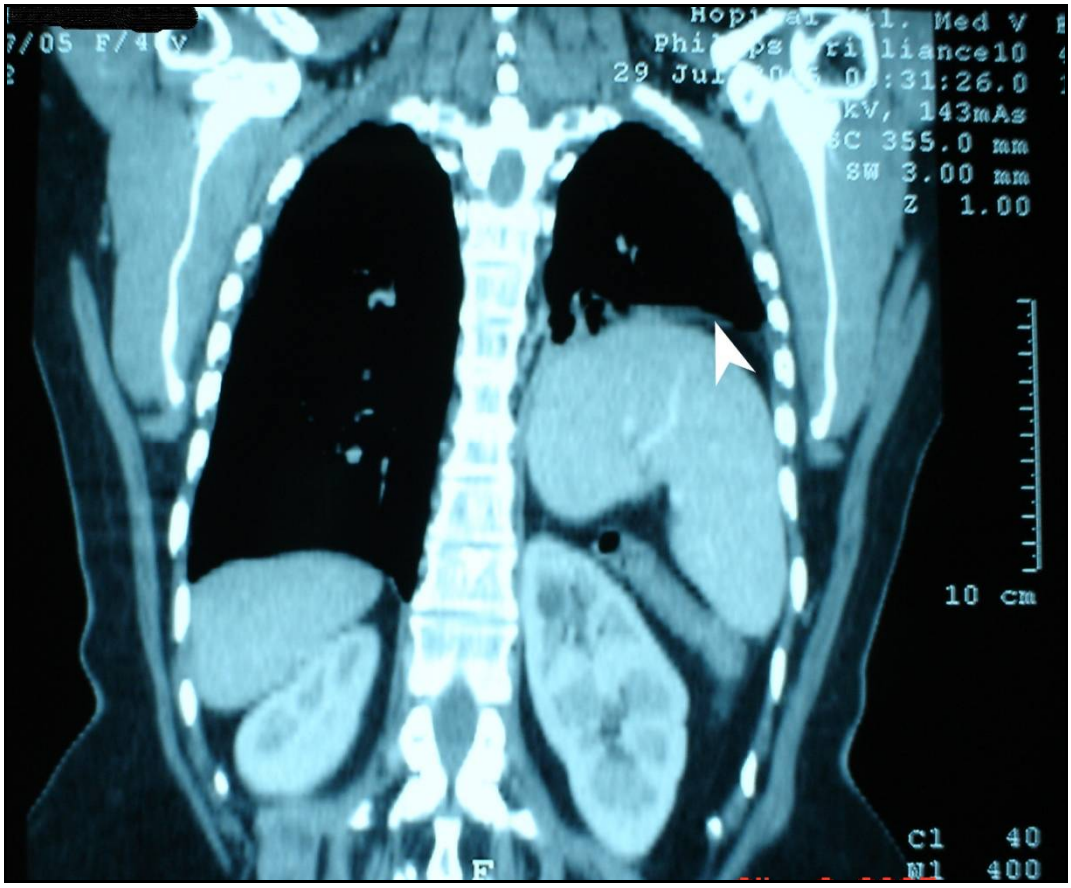


Figure 25: Chest CT-scan in the coronal plane showing a left DE with an ascension of the spleen, stomach and colon (arrow)

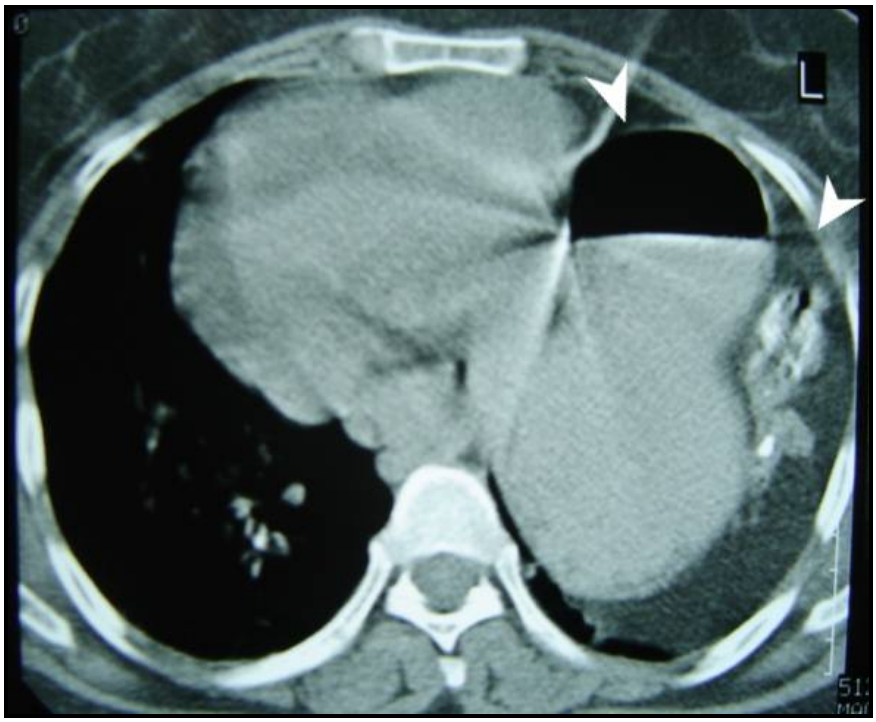


Figure 26: Chest CT-scan in the axial plane showing a left DE with an ascension of the spleen, stomach and colon (arrows)

Figure 27: Chest CT-scan in the axial plane showing a right DE with an ascension of the liver and epiploon (arrow)

iii. MRI

In 4 cases from our study, a chest MRI was realized. The principal indication for it was the suspicion of a diaphragmatic rupture. In these cases, the MRI was the most performant imaging tool to eliminate the differential diagnosis with an eventual rupture of the diaphragm.

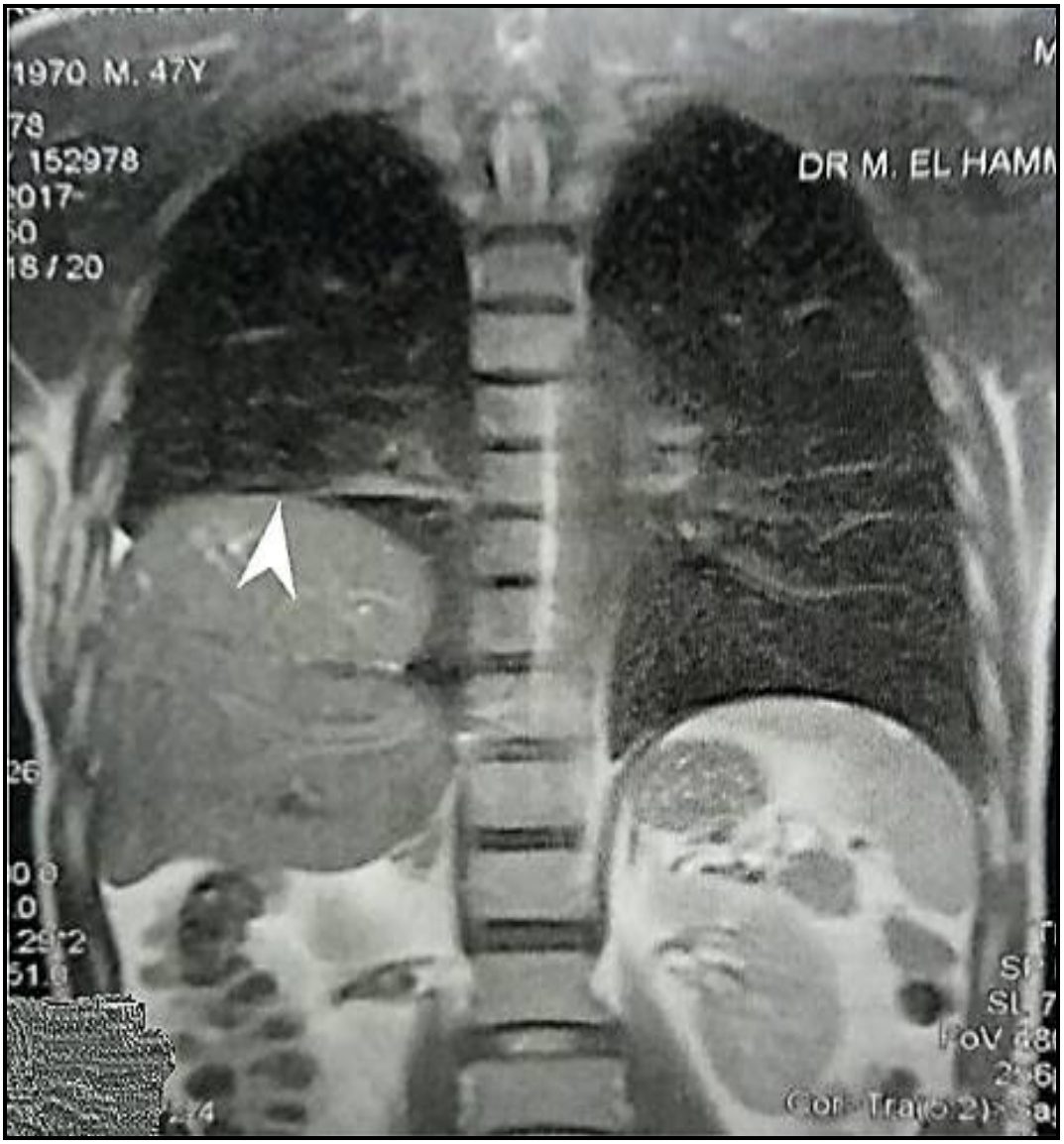


Figure 28: Chest MRI in the coronal plane showing a right DE with an ascension of the liver (arrow)

b. Pulmonary functional tests

The respiratory function was measured before and 3 months after treatment in both groups, using pulmonary functional tests (PFT), mainly the FEV1 and FVC. The obtained results are compared in Table II.

Table II: Pre- and post-therapeutic PFT results in both groups

Measured pulmonary functions	Group A			Group B		
	Before physio-therapy	After physio-therapy	p value	Before surgery	After surgery	p value
Forced expiratory volume in one second (in Liter and %predicted)	2.10±0.56L 78%±25%	2.40±0.25L 82%±28%	p=0.056	1.50±0.79L 60%±10%	1.90±0.81L 75%±5%	**p=0.028**
Forced vital capacity (in Liter)	2.99±0.68L	3.20±0.53L	p=0.063	3.35±0.89L	3.87±1.23L	**p=0.015**

After treatment, a difference in spirometry results was noted in both groups. But the improvement was only statistically significant in group B, with a p value inferior to 0.05. The FEV1 registered an improvement of 15% and the FVC improved by a mean 0.52L.

Surgery improved PFT results significantly, while medical treatment didn't show any conclusive spirometry results with an FEV1 rising by 4% and an FVC by 0.21L (p value>0.05).

4. Surgery

a. Operative indications

All patients that were put in group B, presented a symptomatic DE with at least one indication for surgical management:

- Chronic progressive dyspnea or orthopnea was present in 36 of the 38 group's patients.
- Damage to the pulmonary parenchyma was be observed in 3 of the patients.
- Abdominal digestive chronic symptoms such as gastroesophageal reflux (GER), pyrosis and chronic transit disorders were present in 31 patients.
- Cardiac symptoms, mainly presenting as palpitations, were a surgical indication for 32 patients.
- Finally, surgical cure was indicated for 20 patients who desired a rapid improvement of their symptoms.

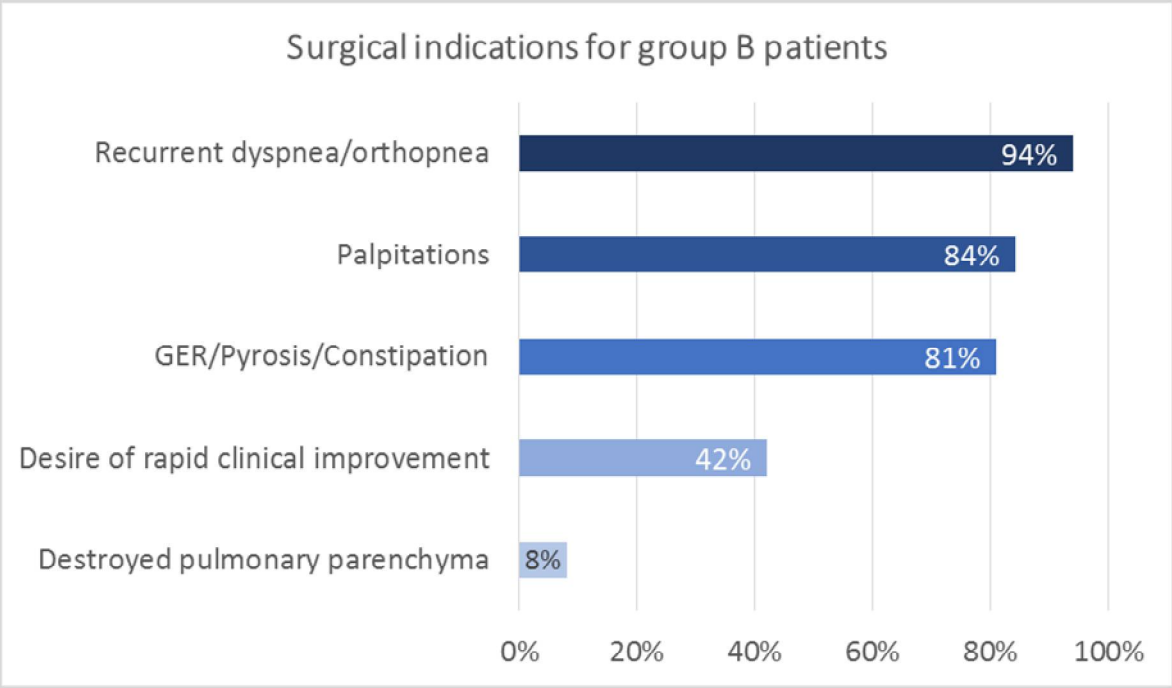


Figure 29: Diagram showing the distribution of the main surgical indications found in our patients

b. Surgical procedures

The type of procedure chosen for each patient depended on his comorbidities, the degree of eventration and the eventual lung parenchyma destruction. In most cases, the approach was through standard open thoracotomy.

95% (n=32) of the patients underwent diaphragmatic plication through PLT, out of which 58% (n=22) didn't require any prosthetic reinforcement. In 26.3% (n=10) cases, the use of a diaphragmatic patch was found necessary, due to the distension and weakness of the diaphragm. This approach was adopted in our unit to prevent recurrences, in cases with a very thin diaphragm.

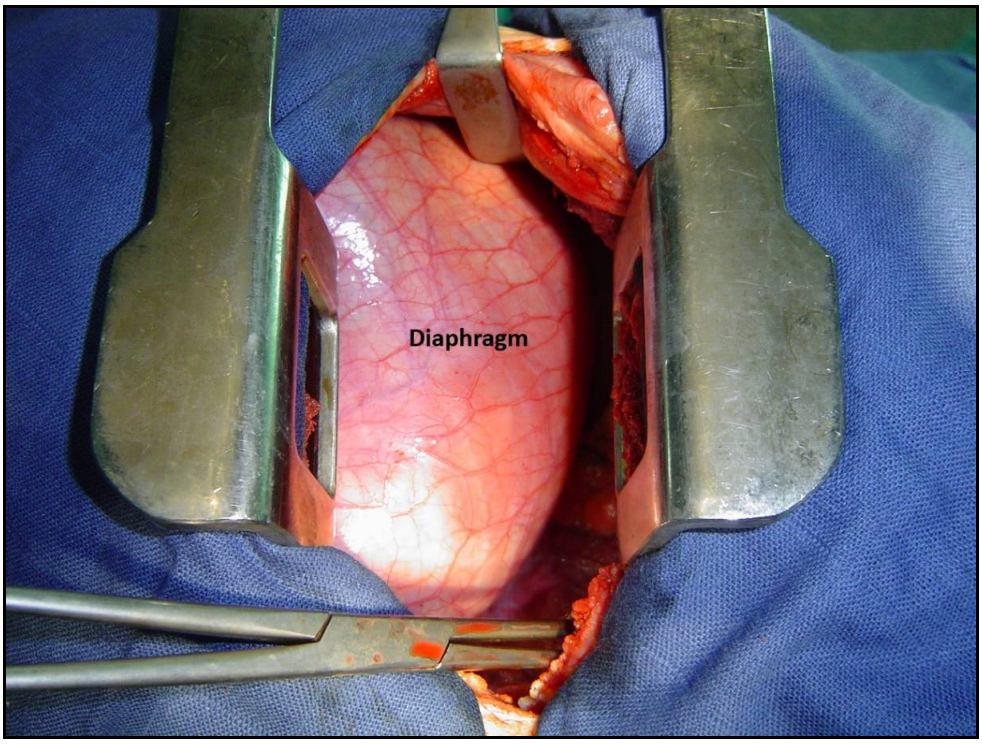


Figure 30: Initial per-operative view of DE through thoracotomy

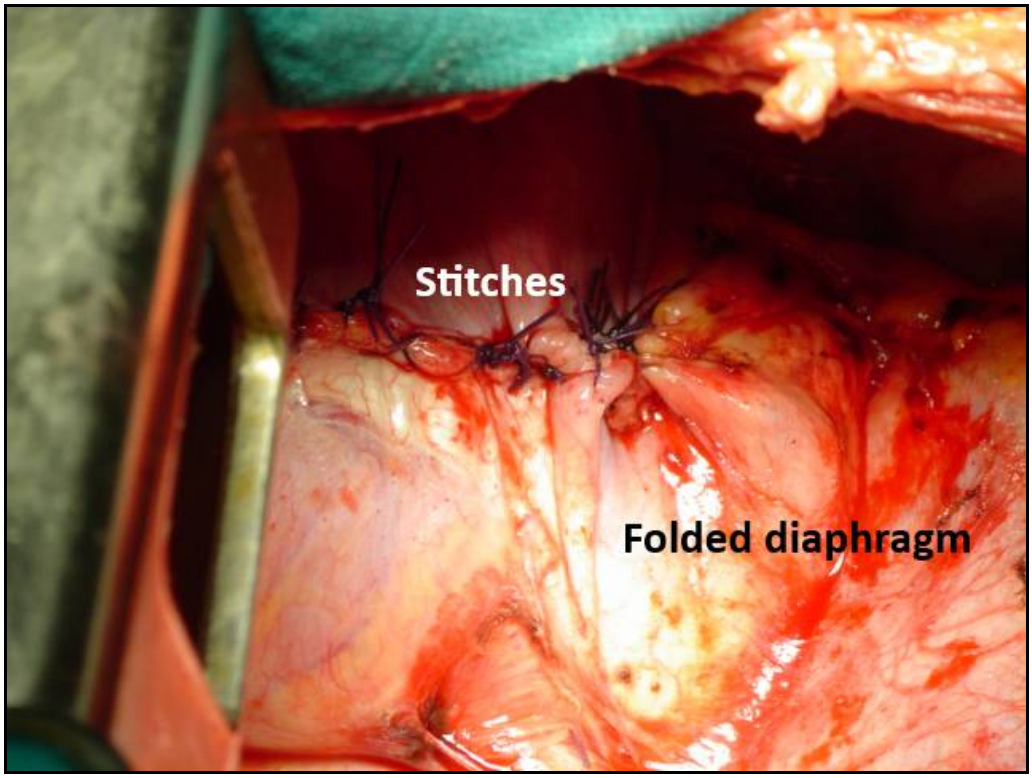


Figure 31: Per-operative view of diaphragmatic plication

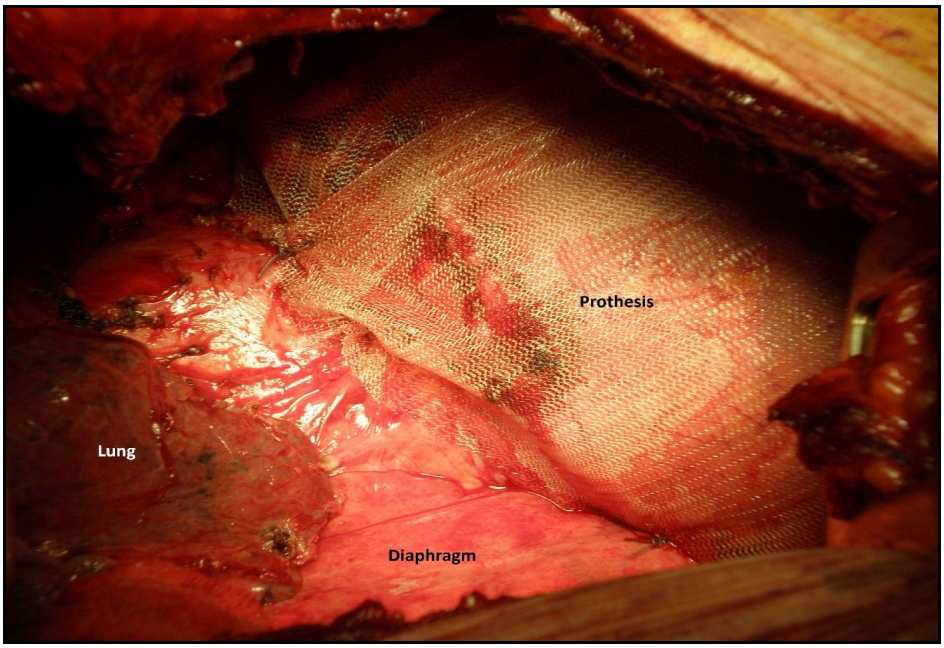


Figure 32: Per-operative view of prosthetic plate reinforcement of diaphragmatic plication

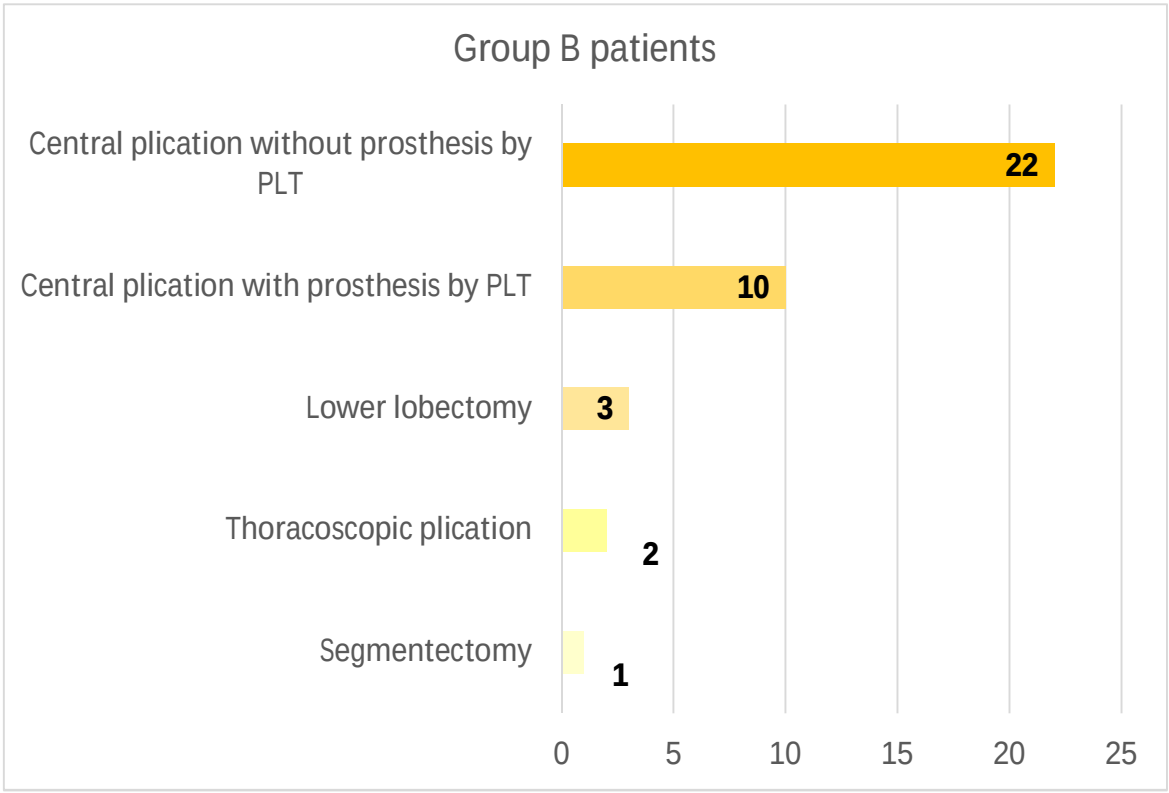


Figure 33: Diagram of the surgical procedures in studied patients

A minimally invasive approach, i.e., thoracoscopic plication, was possible in 5.3% (n=2) of cases, and successfully completed.

Finally, a destroyed pulmonary parenchyma required an associated systematized lung resection for 10.5% (n=4) of patients, three of which were lower lobectomies, and in one case a segmentectomy.

c. Postoperative period

The morbidity rate in group B patients was of 15.8%, while the post-operative mortality was null. The details of complications secondary to surgical treatment in our study's patients are reported in Table III.

Table III: Post-operative mortality and morbidity rates in group B patients

Post-operative complications		Number of group B patients	Management of complications
Mortality		0	-
Morbidity	**Recurrence**	3 (7.9%)	Reoperation with plate reinforcement
	Pyothorax	2 (5.3%)	Medical treatment with thoracic drainage
	Hemothorax	1 (2.6%)	Reoperation
Total		6 (15.8%)	-

The cases that were complicated by a hemothorax or a pyothorax had benefitted from a central plication through thoracotomy, without plate reinforcement. The patient presenting a hemothorax was reoperated, without any further postoperative complications. The 2 pyothorax patients weren't reoperated, and medical treatment with thoracic drainage was sufficient to obtain a satisfactory evolution. None of the 3 patients who underwent plication surgery with an associated lobectomy showed complications in the form of a bronchopleural fistula.

The chest tube was kept in place for a median duration of 2.5 days after the surgery. In the cases with a normal non-complicated postoperative course, the hospitalization period was of 4 to 5 days.

5. Clinical satisfaction questionnaire

Realized during the follow-up, this questionnaire demonstrates the functional subjective improvement felt by the patient after the chosen method of treatment.

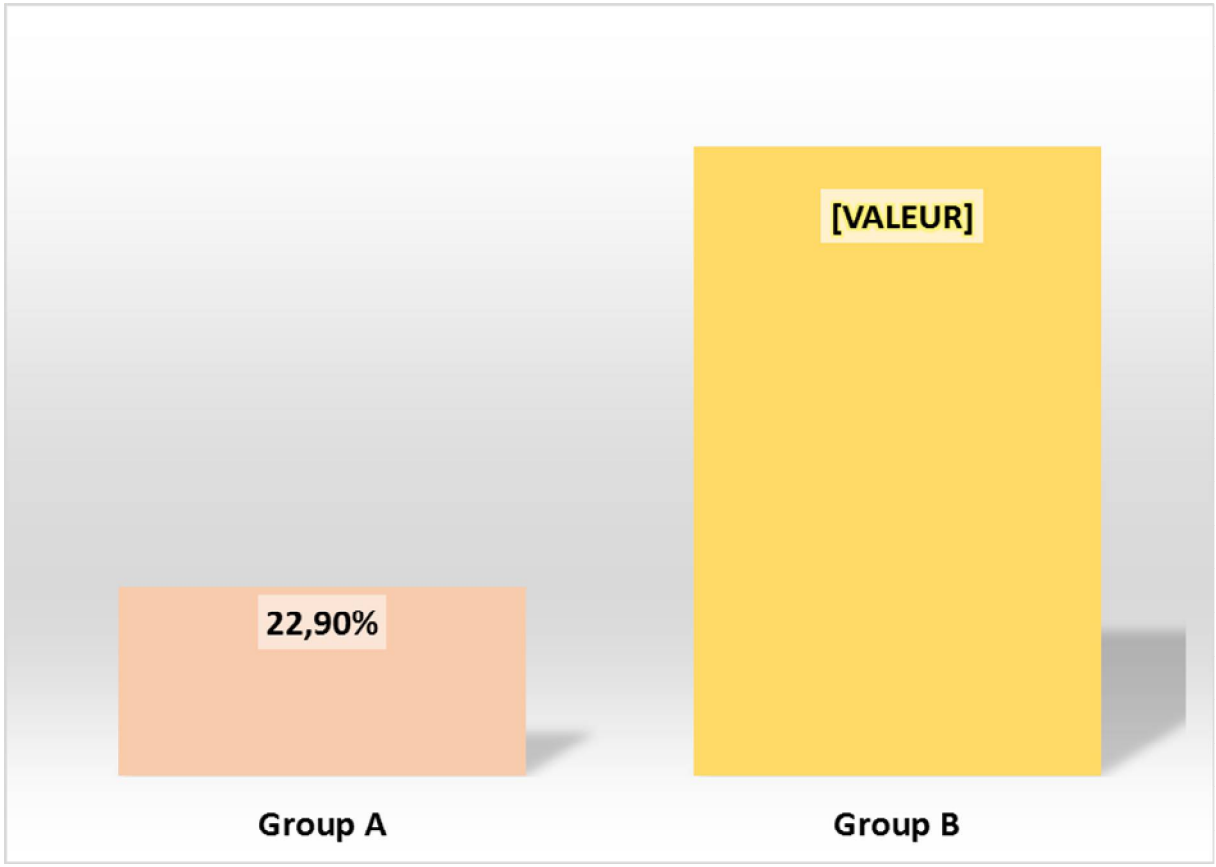


Figure 34: Diagram showing the percentage of patients with a clinical improvement in both groups

A clinical improvement of the respiratory and digestive symptoms was observed in 29 of the 38 cases in group B. In group A, functional rehabilitation had clinically significant results in 11 cases, out of the 48 medically treated patients. The difference between both groups is statistically significant, with a p value=0.025.

6. Follow-up

a. Group A

In this group, 3 patients died: 2 of pulmonary embolism and 1 of acute heart failure. The 45 patients left were regularly followed-up.

Their post-therapeutic results were quantified using spirometry for the respiratory function. (Table II)

The subjective clinical improvement felt by the patient was evaluated using the satisfaction questionnaire. (Fig.34)

b. Group B

Out of the 38 operated patients, 8 (21%) were lost to follow-up. The rest of them (n=30) had regular follow-ups with a median period of 11.5 months.

The eventual clinical improvement at this stage was quantified using a satisfactory questionnaire, and compared to the results obtained for group A's patients. (Fig.34)

The pulmonary function improvement was quantified using spirometry, and results were compared with those from the preoperative exam. (Table II)

A recurrence of the DE was observed in 3 of group B's patients, 15 months at least after the procedure. These patients didn't benefit from a prosthetic reinforcement during the initial plication procedure. A reoperation, this time with the use of a prolene plate, was indicated and successfully achieved.

Discussion

1. Definition

The diaphragm is known as the primary muscle of inspiration, although external intercostal muscles aid in it. This means that any diaphragmatic dysfunction or weakness can vitally impede respiratory functions.

Diaphragmatic eventration can be defined as the absence (when congenital), or loss (when acquired) of the diaphragm's muscular power, while maintaining all the normal anatomical attachments. Macroscopically, the eventrated diaphragm has an attenuated appearance. Microscopically, a congenital form will show diffuse fibroelastic alterations and a rarity of muscular fibers.[15] While a diaphragm with an acquired dysfunction will appear to have a normal, although atrophic, amount of muscle fibers.[16] The thin stretched diaphragm, is unable to contract, leading to the typical aspect of elevated (hemi)diaphragm.

The credit for the first description of this condition goes to Jean Louis Petit (1774), who discovered it during a postmortem examination.[17] The first successful surgical repair of a DE was reported by Morrison in 1923.[18]

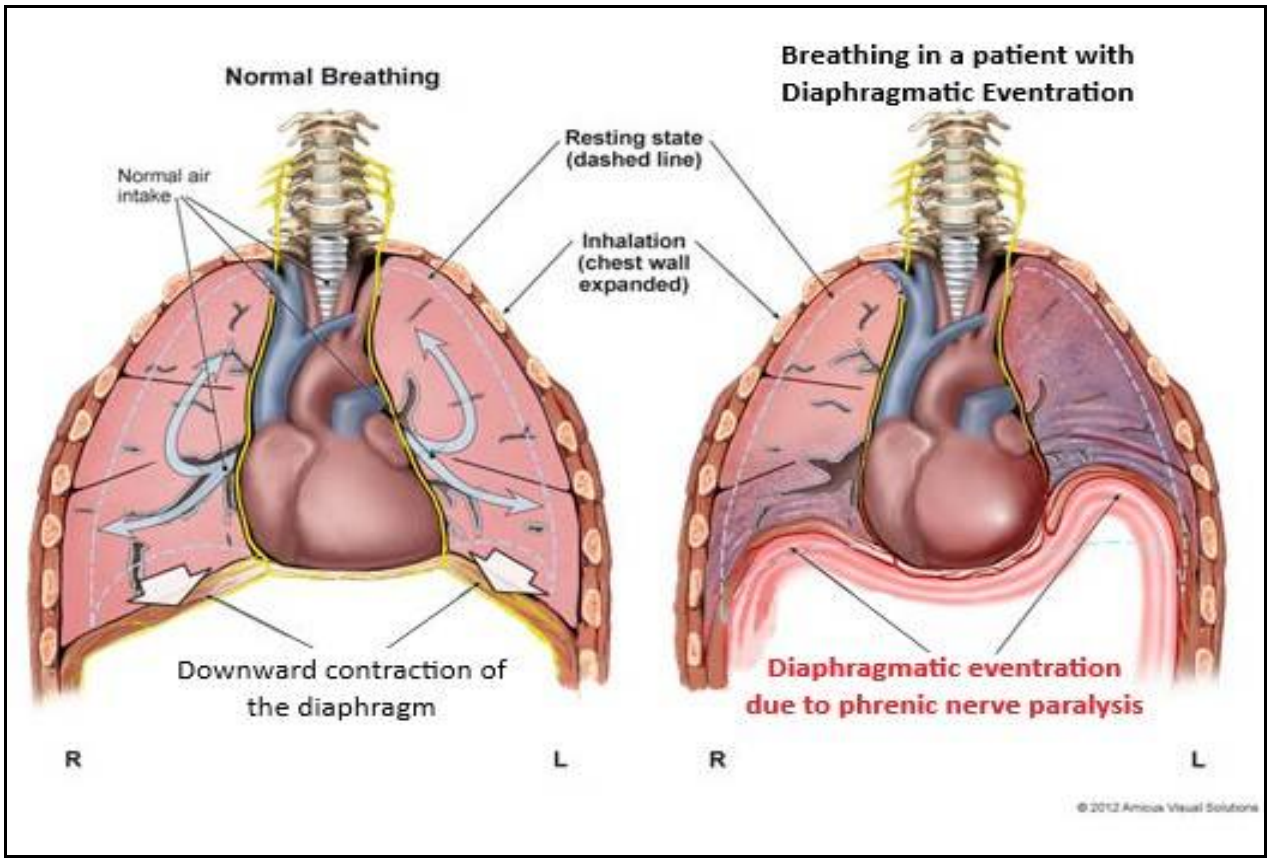


Figure 35: Comparison between a normal and a paralyzed diaphragm

2. Epidemiology

a. General incidence

The frequency of most diaphragmatic disorders can't be exactly estimated. The incidence and prevalence of DE being most likely underestimated, due to subtle non-specific clinical presentations.

The data presented in most studies still defines DE as a rarity, with an estimated incidence of 0.07% (1 in 1400 subjects) in an English mass X-ray unit.[19] The available case reports have suggested an incidence inferior to 0.05%.[1] Even though the frequency of acquired DE, i.e. diaphragmatic paralysis or paresis, is probably higher, and the pathology most likely under-diagnosed.[16]

b. Gender distribution

Diaphragmatic eventration has been reported as more frequent in the male population. A male predominance between 65% and 94% can be observed in reviewed studies.[18, 20, 21]

In our present study however, we didn't note a pronounced gender predominance, with a sex ratio of 0.95 male per female.

c. Affected side

The unilateral form of eventration, presenting as an elevated hemidiaphragm is more common than the bilateral one. The left side of the diaphragm is more often affected than the right one, as reported in reviewed literature sources, with rates between 55% and 81%.[18, 20, 21]

Our study confirms these facts, with a pronounced left side eventration predominance, the rate of which was 79%.

3. Clinical diagnosis

DE is an uncommon disease, the non-specificity of clinical symptoms or their complete absence, are the principal causes of the under-diagnosis of this pathology. A study from Laxdal and colleagues estimated that at least half of the patients presenting a DE were asymptomatic. As a general rule, DE should be looked into as a differential diagnosis in cases of unexplained dyspnea.[22, 23]

a. Functional symptoms

i. Unilateral DE

Adult patients with an elevated hemidiaphragm are usually asymptomatic. In that case, the diagnosis might be accidental, made during a chest radiograph.[24]

When symptoms are present, they're mainly respiratory. They consist of dyspnea -mainly on exertion-, orthopnea, a decrease in exercise performance and chest wall pain. Piehler and colleagues, in their study of diaphragmatic paralysis in 142 patients, found dyspnea, chest wall pain and coughing as the most common functional signs.[25-28]

Gastrointestinal disorders secondary to the migration of abdominal organs in the thoracic cavity can also reveal a unilateral DE, especially if it's on the left side. The symptoms observed are nonspecific and mainly dyspeptic, such as regurgitation, nausea, epigastric pain or pyrosis.[29, 30]

Cardiac symptoms, principally palpitations, are probably secondary to the displacement of the mediastinum.[18, 31]

Since none of these symptoms is specific, a meticulous review of the patient's history is crucial. The investigation of any other cause for dyspnea (heart failure, obesity, pulmonary disease…) must not be overlooked. If found, such factors should be corrected before imputing the symptoms to a unilateral eventration or paralysis, which remains an exclusion diagnosis.[29]

The main characteristic common to these symptoms is that they often intensify with the change of posture. This can be explained by the extrinsic compression applied by the abdominal organs on the thoracic cavity in supine position, when the dysfunctional diaphragm is no longer capable of preventing it. Another factor is the aggravation of the diaphragmatic excursion loss when lying down flat.[32, 33]

ii. Bilateral DE

Symptoms are more frequent and intense for patients with a bilateral disorder. Dyspnea is still the main symptom, it can be on exertion, though more often present at rest or supine position.[34] Possible dyspnea during immersion in water to the abdomen's level has also been reported.[35]

Orthopnea is more evocative of diaphragmatic weakness, and can be severe enough to completely prevent the patient from lying in supine position.[24] Orthopnea is often the main complaint of the patient, and the reason for medical consultation.[36]

Sleep hypoventilation or even apnea have also been reported as signs of DE. In a case presented by Stradling and colleagues, concerning a patient suffering from bilateral DP, these disorders were present during the REM sleep phase before diurnal respiratory failure.[37]

Hypersomnia, fatigue and depression due to sleep disturbance are often reported by patients.[33]

Digestive and cardiac symptoms are similar to the ones found in unilateral disorders.

As with the unilateral presentation, these symptoms are all nonspecific, and a history of cardiac surgery, cervical traumatism or neuromuscular disease will help comfort the acquired DE diagnosis.

b. Physical signs

Physical signs are usually nonspecific and don't add much to the diagnosis. But a thorough clinical examination is still required, to assess the physical consequences and progression of dyspnea and orthopnea and eliminate any other possible cause of dyspnea.[24, 29]

The most frequently found physical signs include tachypnea, a decrease in breath sounds at the affected lung base(s), and sometimes with unilateral DE an asymmetrical motion of the wall in supine position. Tachycardia can also be noted. [24, 32, 34]

The only characteristic sign of diaphragmatic weakness is the abdominal paradox, more visible in supine position. During inspiration, the abdominal wall moves inward while the rib cage expands outward. This is consequent to the decrease of pleural pressure caused by the contraction of accessory inspiratory and intercostal muscles. Leading to an upward motion of the dysfunctional diaphragm while the abdomen is drawn inward. The use of accessory inspiratory muscles can be confirmed during inspiration, by the contraction of the sternocleidomastoid muscle felt by palpating the neck.[25, 34, 38]

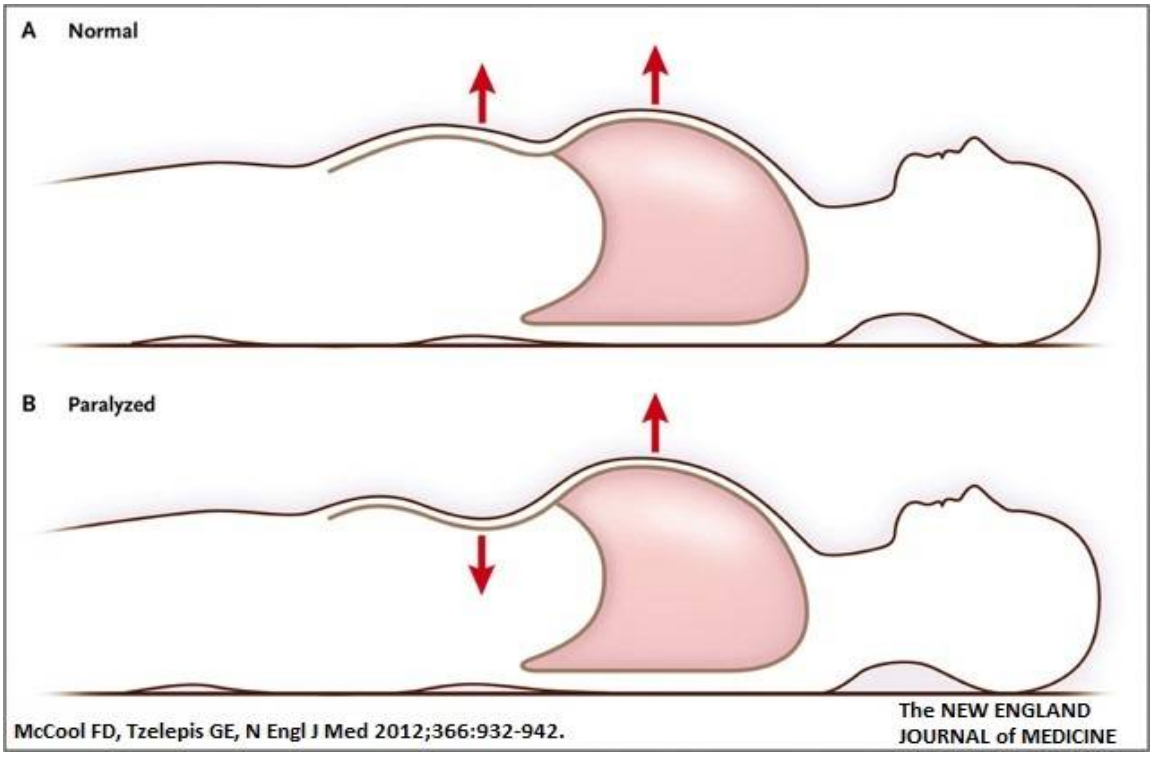


Figure 36: Rib cage and abdominal wall motion in normal (A) and paralyzed (B) diaphragms

A study conducted by Mier-Jedrzejowicz and colleagues (1988), suggested that the presence of abdominal paradox meant that the transdiaphragmatic pressure (Pdi) was lower than 30cmH_2O.[39] This sign is rarely observed in unilateral DE, unless an impairment in inspiratory muscle strength or cardiopulmonary disease is associated.[40]

c. Complications

Diaphragmatic eventration is sometimes revealed by a complication:

i. Respiratory

The limited thoracic excursion that accompanies DE is a risk factor for lung infections. This explains the frequency of recurrent infections of the lower respiratory tract in patients with eventration, particularly in chronic or bilateral forms. Another common complication is atelectasis due to the compression of the basal part of the lung.[30, 34]

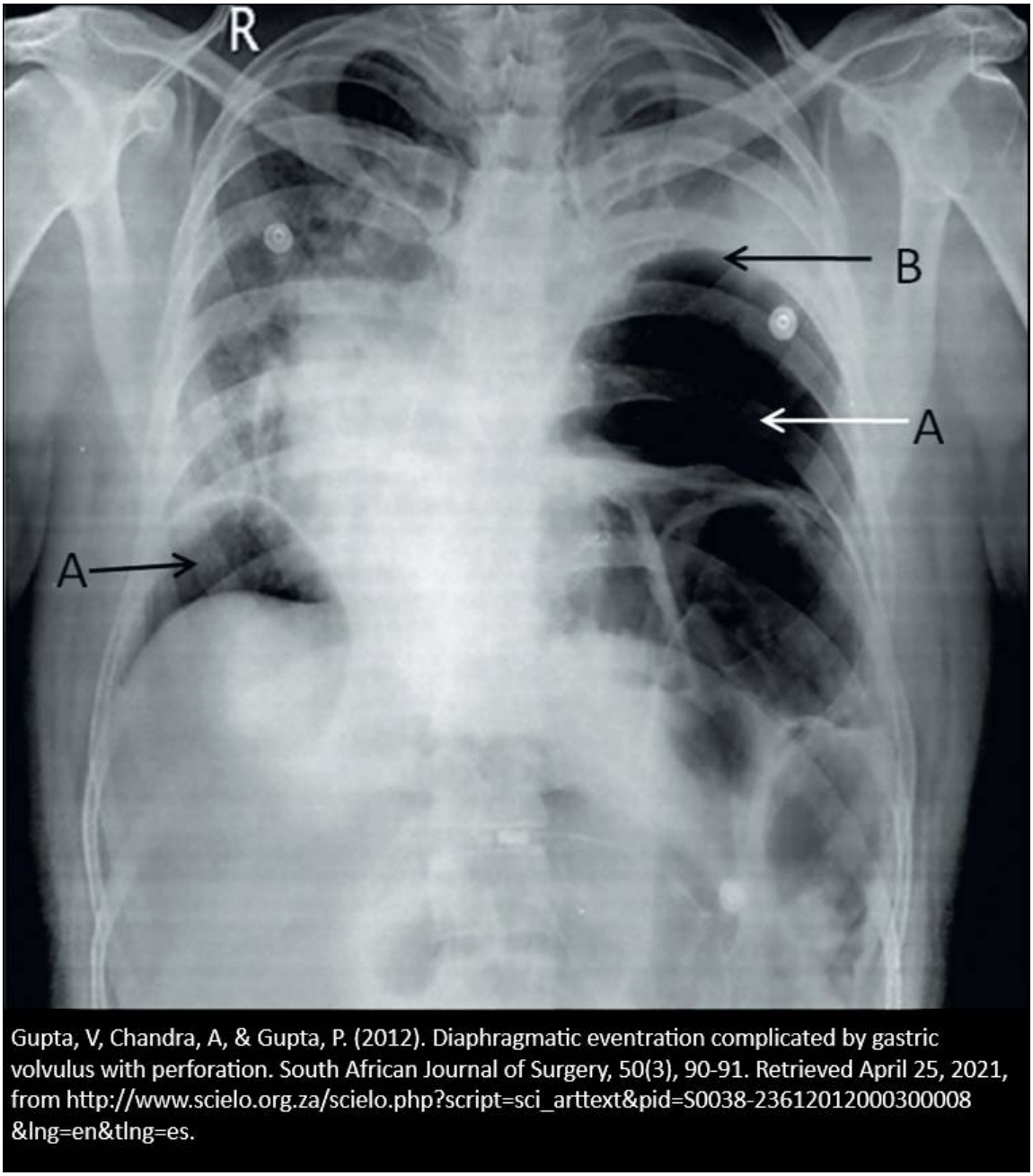
The multifactorial changes in lung anatomy (destruction) and physiology can in turn cause respiratory distress.[32] Watanabe and colleagues reported a case of right total DE complicated by acute progressive respiratory distress requiring an emergency operation.[41]

ii. Cardiac

Chronic hypoventilation, more common in bilateral forms, can cause hypoxia and hypercapnia. On the long term, this can lead to a right heart failure clinical presentation.[33]

iii. Digestive

Beside the complications secondary to dyspeptic symptoms, principally the GER. DE can also be associated with gastric volvulus, which is considered a surgical emergency, because of the risk of ischemia, necrosis and perforation. This condition though uncommon, has been repeatedly reported.[42-44]



Gupta, V, Chandra, A, & Gupta, P. (2012). Diaphragmatic eventration complicated by gastric volvulus with perforation. South African Journal of Surgery, 50(3), 90-91. Retrieved April 25, 2021, from http://www.scielo.org.za/scielo.php?script=sci_arttext&pid=S0038-23612012000300008 &lng=en&tlng=es.

Figure 37: Antero-posterior chest X-ray showing pneumoperitoneum (A) and left elevated hemidiaphragm (B)

4. Paraclinical diagnosis

a. Morphological assessment

i. Chest X-ray

The diagnosis of DE in asymptomatic patients can be incidental, made during a chest radiograph. If the diagnosis is suspected, because of the clinical presentation, antero-posterior and lateral conventional radiographies in a standing position and full inspiration are the first-line imaging tool. It classically shows unilateral or bilateral elevation of the diaphragm as a continuous unbroken curved line on an antero-posterior incidence. This elevation isn't specific and could be due to a decrease in pulmonary or abdominal compliance, or pleural adhesions if bilateral, or other diaphragm abnormalities if one-sided. A lateral incidence allows a comparison of level between the elevated and normal side. Radiographs of the stomach erect and in Trendelenburg's position can reveal the degree of stomach dislocation.[24, 30]

An elevation on chest X-ray is quite sensitive when the disorder is unilateral. A study conducted by Chetta and colleagues (2004) concluded to a 90% sensitivity but only a 44% specificity for unilateral diaphragm dysfunction.[45] With bilateral DE however, chest X-ray is limited by a low sensibility and specificity.

To conclude, in both unilateral and bilateral forms, further studies must be conducted if an elevated hemidiaphragm is found on the standard chest radiograph of a dyspneic patient.

ii. Fluoroscopy

This technique is based on a “sniff test”, it allows an evaluation of the diaphragm’s motion during a short, sharp inspiration. Normally, the diaphragm moves caudally during inspiration. In case of unilateral DE, the opposite happens and the dysfunctional hemidiaphragm will paradoxically stay in a cephalic position. Unfortunately, this test has many limitations. First, it hasn’t proven reliable in the diagnosis of bilateral DE.[16, 34] Second, it lacks specificity, with frequent false positives. This was proven in a study conducted by C Alexander, which concluded to rates as high as 6% of unilateral paradoxical movement in normal subjects.[46] Finally, the paradoxical motion may be absent in a dysfunctional diaphragm, leading to false negatives.

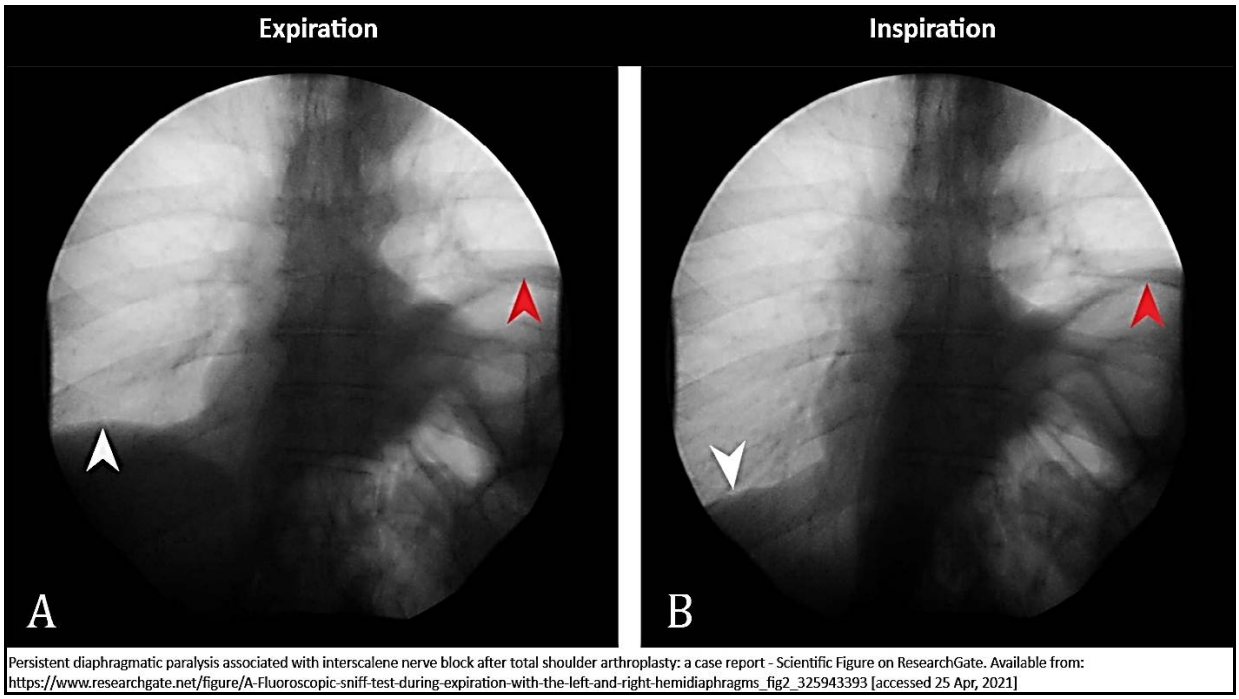


Figure 38: Fluoroscopic sniff test during expiration (A) with left and right hemidiaphragms’ elevation, and during inspiration (B) with physiologic depression of the right hemidiaphragm but persistent elevation of the left hemidiaphragm (arrows)

The limitations of this technique consequently make its results difficult to interpret. A paradoxical motion of at least 2cm has been recommended as the minimum to consider the test result significant.[24]

iii. Ultrasound

Ultrasonography of the diaphragm, especially at the zone of apposition with the rib cage, allows to evaluate the diaphragm's thickness and how it changes during respiration. It doesn't involve any radiation as opposed to the imaging methods previously cited. A comparative study of ultrasound with fluoroscopy was conducted by Houston and colleagues. It showed a concordance rate of 81% in the diagnosis of abnormal hemidiaphragm movements. It also noted technical failures using fluoroscopy and the possibility to detect milder dysfunctions with ultrasound.[47] This noninvasive imaging technique also has the advantage of being effective in the diagnosis of both unilateral and bilateral DP, and even evaluating the post-treatment recovery.[48] The ultrasound diagnostic criteria for a diaphragm dysfunction are: a diaphragm thickening fraction (DTF) below 20% in B-mode, and paradoxical breathing (a curve below the baseline) in M-mode.[48-50]

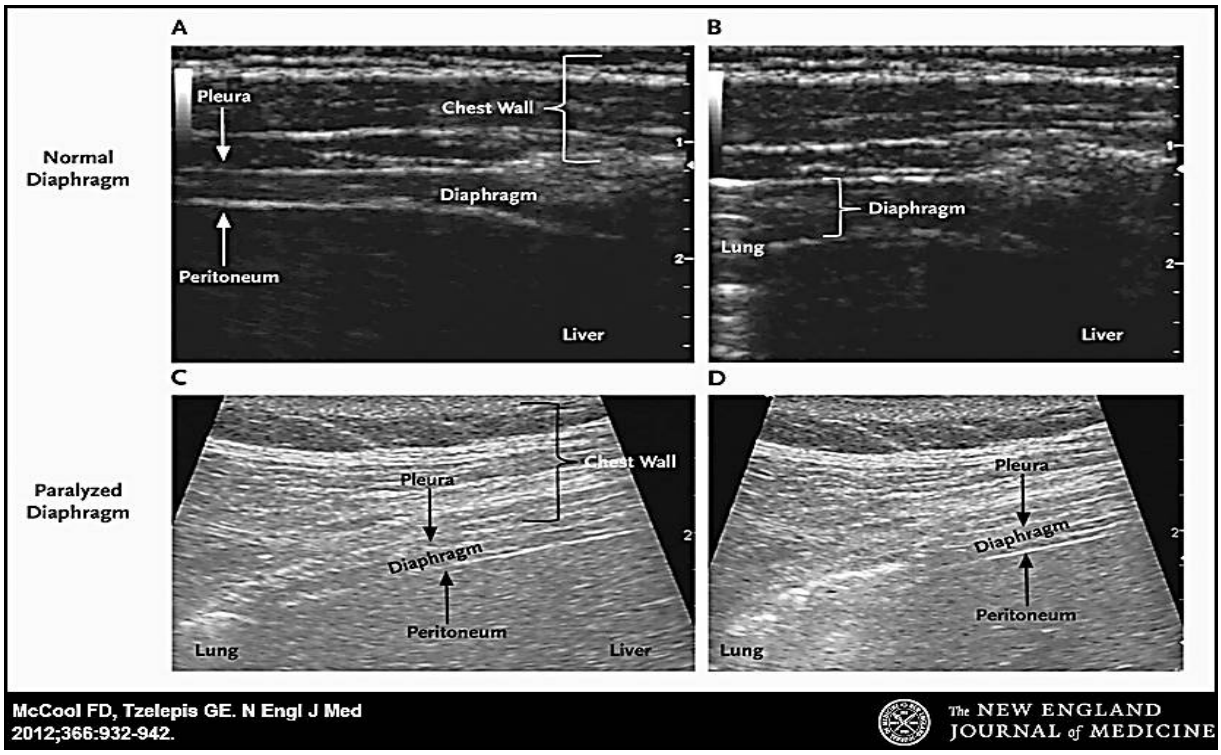


Figure 39: B-mode ultrasound pictures of normal (A, B) and paralyzed (C, D) diaphragm

The normal diaphragm is thin during expiration (A) and thickens during inspiration (B). The dysfunctional diaphragm, thin during expiration (C), does not thicken during inspiration (D).

Ultrasound offers both a static and dynamic evaluation of the diaphragm, a study by Lloyd and colleagues (2006) showed the interest of M mode ultrasonography. While B mode is widely used in the diagnosis of DP, it is limited by the patient's ability to take deep breaths and his cooperation. These limitations aren't present with motion sonography, it can be done at the patient's bedside and only requires a few minutes with an experienced operator.[50]

Although of rising interest in the diagnosis of diaphragmatic dysfunctions in adults, the technique needs further study, especially the M mode. It still calls for an experimented operator and visualizes the right hemidiaphragm better than the left.[24]

iv. Computed tomography

A chest CT scan can estimate the thickness of the diaphragm and the height of each dome. The comparison between standard standing radiographs and supine CT scan images allows an appreciation of the diaphragmatic amyotrophy. It also shows eventual organs' compressions or deviations. But the main indication for it is the exclusion of eventual cervical or intrathoracic tumoral etiologies, or a subphrenic process responsible for a hemi-diaphragmatic elevation. A CT scan is most commonly required if one of these etiologies is suspected.[16, 24, 29]

v. Magnetic resonance imaging

MRI is the most effective imaging modality in the comprehension and evaluation of diaphragmatic dysfunctions. Like ultrasound, it has the advantages of being radiation-free and enabling both morphological and functional evaluations. Furthermore, it can detect paradoxical movement of the paralyzed diaphragm.[51] The imaging technique can also show early signs of diaphragmatic weakness, when spirometry results are still normal. This was shown in a study about Pompe disease by Harlaar and colleagues (2021).[52]

The use of MRI is unfortunately limited by its high cost, long duration and the patient's compliance. It is consequently not recommended as a routine tool in the evaluation of DE.[29]

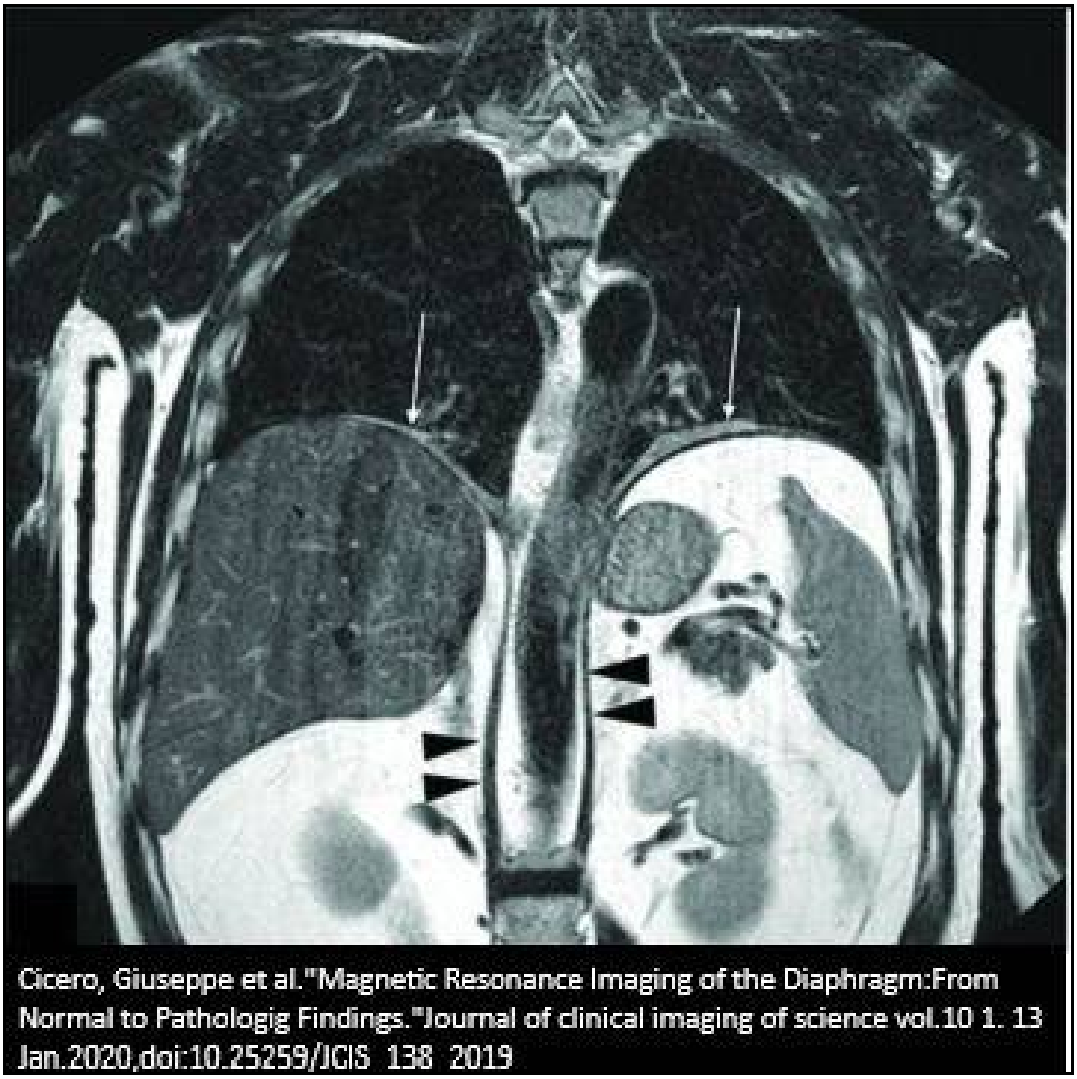


Figure 40: MRI image showing an elevation of both hemidiaphragms, bilateral basal atelectasis (arrows) and normal diaphragmatic crura (arrowheads)

b. Neuromuscular functional study

The study of a dysfunctional diaphragm, requires the full assessment of the conduction pathway from its central origin to the diaphragmatic muscle.

i. Transdiaphragmatic pressure measurement

Transdiaphragmatic pressure (Pdi) can be measured using two balloon manometers, one in the esophagus to measure the esophageal pressure (Pes) which reflects the pleural one, and the other in the stomach (Pga) to measure the gastric (intra-abdominal) pressure. Knowing that the Pdi is calculated as:

Pdi = Pga - Pes

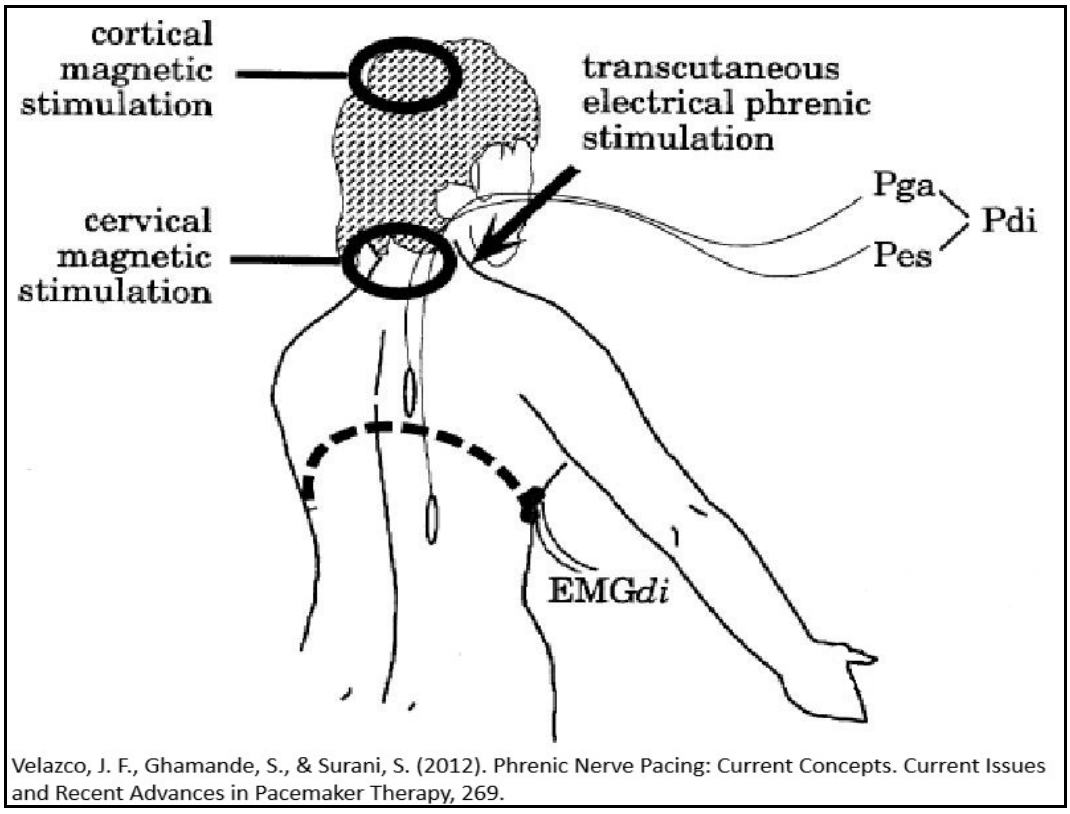
During inspiration the Pga will normally rise and Pes decrease. The numbers obtained are compared to static and sniff-test reference values, which are: >90cmH2O for men and >70cmH2O for women during static inspiration, and >100cmH2O for men and >80 cmH2O for women during dynamic (sniff-test) inspiration.[53]

ii. Electromyography and phrenic stimulation

Electromyographic activity can be recorded using surface electrodes during spontaneous breathing. But this technique is limited by the impossibility of obtaining purely diaphragmatic measures, and is consequently not commonly used.[53]

Recording the electromyographic response to a phrenic stimulation is the recommended technique for the neuromuscular functional study of the diaphragm. It’s based on registering the result of cervical phrenic nerve electrical or magnetic stimulation, using surface electrodes. The electrical stimulation has a high specificity and is the reference method for the study of the

phrenic nerve. But it's limited by its discomfort for the patient due mainly to the painful electric shocks. Magnetic stimulation on the other side is painless and technically easier, but has no specificity for the diaphragm.[53]



Velazco, J. F., Ghamande, S., & Surani, S. (2012). Phrenic Nerve Pacing: Current Concepts. Current Issues and Recent Advances in Pacemaker Therapy, 269.

Figure 41: Schema of the techniques used in the functional study of the diaphragm

The response to electrical or magnetic stimulation is measured through:

***EMGdi**: diaphragmatic electromyography*

***Pdi**: trasdiaphragmatic pressure, result of the difference between measured Pga (gastric pressure) and Pes (esophageal pressure)*

The electromyographic response obtained is then studied: its aspect, latency and the amplitude of motor response are thoroughly analyzed. The normal response is a monophasic expansion. A biphasic expansion is the sign of a force asymmetry between both hemidiaphragms. In traumatic situations, diaphragmatic EMG has a prognostic value: a complete abolition of the electromyographical response meaning that a spontaneous recovery is unlikely. A measurement of the Pdi is commonly associated with phrenic stimulation.

In conclusion, despite its ability to analyze the phrenic nerve's functionality and diaphragm contractility, electromyographic response to phrenic stimulation doesn't play a major role in the positive diagnosis of DP, because of its limited availability.[53, 54]

c. Evaluation of impact

i. Pulmonary functional tests

The diaphragm is the main inspiratory muscle, which is why when it's dysfunctional, pulmonary function tests will often show suggestive abnormalities. A restrictive pattern (i.e., low FVC and low FEV1), can often be observed with diaphragmatic dysfunctions, secondary to a reduction in the thoracic wall's compliance. Measurements of the vital capacity, in both upright and supine positions, are essential.[16, 34] A decreased FVC is frequently present in the erect position, but most importantly while supine due to the elevation of the diaphragm in this posture.[36] Since a posture-related fall in vital capacity can also be seen in normal subjects, a study conducted by Allen and colleagues, concluded that FVC differences higher than 25% should indicate further tests.[55]

In unilateral DP, vital capacity could drop to 75% of the predicted value.[39] With a further decrease when supine that stays within the normal range (<25%) in most cases.[27] With bilateral DP on the other hand, the vital capacity is around 50% of predicted value. In supine position the decrease is this time significant, since it is between 30 and 50%.[38] Total lung capacity can also be reduced.[39]

PFT are noninvasive, easily available and contribute to both positive and gravity diagnosis of DP. The therapeutic choice is also oriented by the results obtained during this exam. It is, in conclusion an essential tool in the assessment of DE.

ii. Gasometry

An arterial gasometry is imperative in the evaluation of a diaphragmatic dysfunction's impact. Although both its sensitivity and specificity are low.[53]

iii. Evaluation of sleep

Nocturnal anomalies must be researched, and if suspected clinically or because of the gasometry results, further tests are indicated. Nocturnal oximetry can be quite sensitive, although the absence of desaturation episodes can't exclude the diagnosis. In second intention, polysomnography is the gold standard.[38, 53]

5. Differential diagnosis:

DE in both its unilateral and bilateral forms, doesn't have any specific clinical presentation. When the diagnosis is suspected, all possible etiologies of dyspnea and orthopnea must be excluded. With unilateral DE, alternative causes of elevated hemidiaphragm must also be considered.

a. Diaphragmatic rupture

Rupture of the diaphragm is an acquired disruption of the diaphragm. It is most commonly post-traumatic, with a reported incidence up to 5.8% in blunt thoracoabdominal trauma.[56] High velocity trauma secondary to motor vehicle accidents seems to be the most frequent cause, with associated thoracic or abdominal injuries.[57]

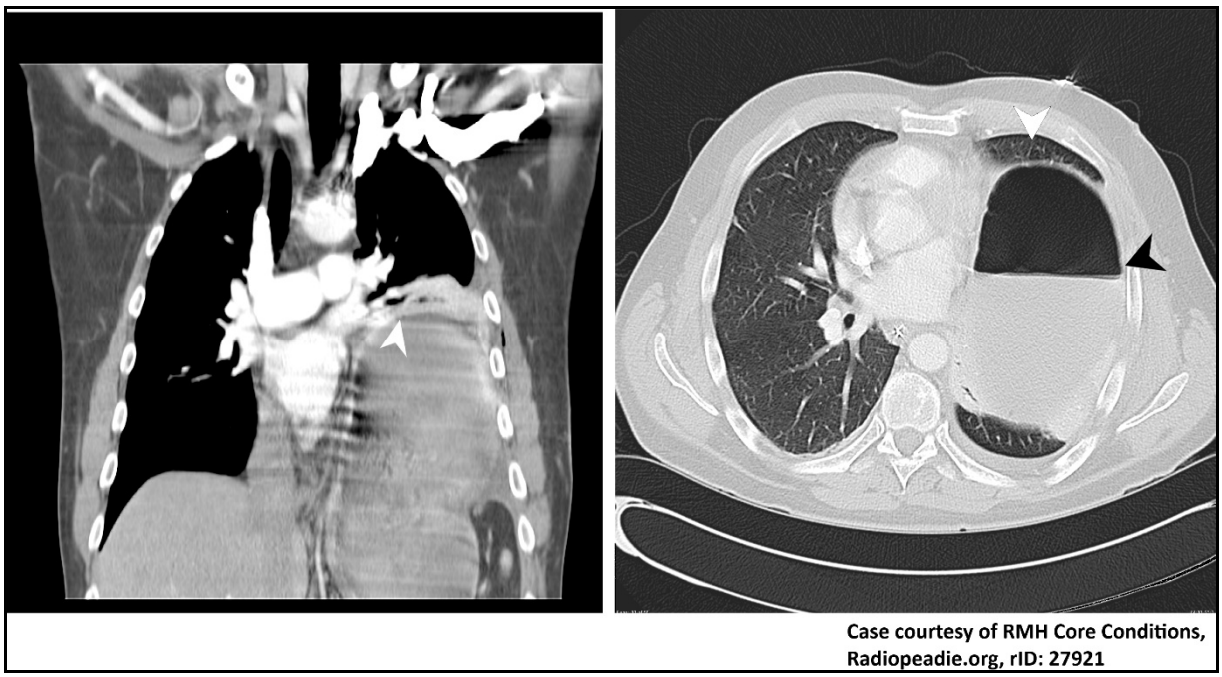


Figure 42: CT scan showing left diaphragmatic rupture with herniation of the stomach into the left thoracic cavity

A thoraco-abdominal CT scan, or when available an MRI, are the methods of choice in the diagnosis of traumatic diaphragmatic rupture. With computed tomography, the maximum sensitivity is 63% and maximum specificity is 100% for the pathognomonic collar sign.[58]

Despite the high-performance imaging tools, around half cases are still diagnosed during laparoscopy, laparotomy or thoracotomy for concomitant injuries. An undiagnosed traumatic diaphragmatic rupture will lead to visceral herniation and strangulation, or pulmonary complications and respiratory distress. These complications may explain the high morbidity and mortality rates that can rise to 50%.[59] It is recommended to repair the defect through a laparotomy using non-absorbable suturing.[56]

History consistent with diaphragm eventration or paralysis doesn't rule out an associated rupture. A case report of traumatic rupture in a patient with DE history shows the possible association of both situations.[59] Consequently, the notion of acute blunt trauma must lead to a careful imaging assessment looking for any signs of disruption in the diaphragm, and sometimes even exploratory laparoscopy. The risks associated with an uncorrected defect being too great to allow a missed diagnosis.

b. Diaphragmatic hernia

Congenital undiagnosed diaphragmatic hernia may appear as an elevated hemidiaphragm on a chest radiograph. These defects are the consequence of an incomplete closure of the muscular portion of the diaphragm. Bochladek hernia, Morgagni hernia and hiatal hernia can all be mistaken for a DE on standard imaging. The main difference with DE is the disruption of the diaphragm's continuity, causing a direct contact between the abdominal and thoracic organs.[60, 61] A surgical correction of the defect after the replacement of the abdominal viscera into the abdominal cavity is the treatment of choice.

c. Diaphragmatic tumors

A tumor extending along the surface of the diaphragm may resemble an elevated hemidiaphragm. Primary tumors of the diaphragm are rare, the most frequent are cystic benign formations, but malignant sarcomas are also common.[62] Secondary tumors with a thoracic or abdominal origin are more frequent. A complete surgical resection is recommended in most cases.[63]

d. Extra-diaphragmatic disease

- Subphrenic mass or abscess
- Subpulmonic effusion
- Basal atelectasis
- Pleural tumor
- Pulmonary or mediastinal mass
- Lung resection
- Ascites
- Hepatomegaly or splenomegaly

6. Etiological diagnosis

The etiologies responsible for an eventration of the diaphragm are numerous and can be classified using different criteria. The pathology may be congenital or acquired. When acquired, the etiologies are categorized based on the level of impairment (Fig.43), starting from central neurological diseases, to peripheral neurological, then phrenic nerve damage and myopathies. Though simplified to a certain extent, this classification allows to structure the extensive list of acquired DE etiologies.

This work mainly focuses on acquired DE, and only peripheral etiologies starting from the phrenic nerve's level will be detailed. One must keep in mind that, although neurological diseases are only cited here, they're frequent causes of DE, particularly the bilateral forms.[60]

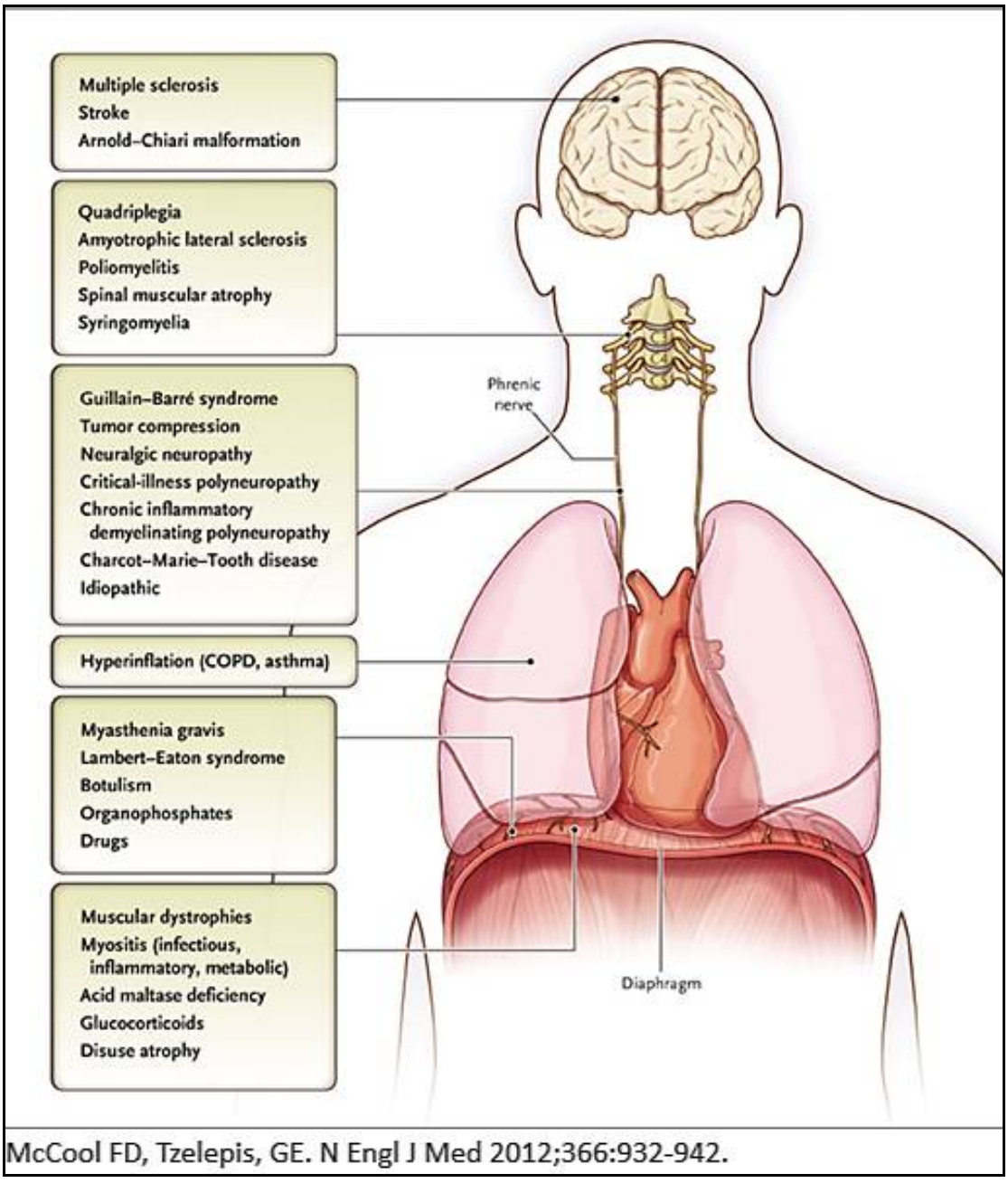


Figure 43: DE etiologies according to the level of impairment

a. Neurological diseases

- Spinal cord injuries can lead to DP, the risk and gravity decreasing with lower levels of medullary transection. When the injury occurs at C1 or C2 level, DP is complete, whereas the diaphragmatic function can be partially preserved from C3 to C5.[64]

- Multiple sclerosis often leads to respiratory complications, but diaphragmatic weakness only seems to be involved in around a third of the cases.[65]

- Amyotrophic lateral sclerosis.[66]

- Poliomyelitis was a major cause of permanent respiratory failure before vaccination became available.[33]

- Parsonage Turner syndrome (brachial neuritis)[67]

- Spinal surgery

- Chiropractic manipulation of the cervical spine has been reported as a possible cause of diaphragmatic paralysis.[68]

b. Phrenic nerve injury

i. Traumatic lesions

Traumatic damage to the phrenic nerve is most commonly iatrogenic, with the phrenic nerve being injured during cardiothoracic or neck surgery. The cause may be mechanical, hypothermic or ischemic.[34, 69]

Cardiac surgery is a well-recognized etiology of phrenic nerve injuries, though the incidence of DP varies in the literature due to various reasons. In a study conducted by Wheeler and colleagues the use of a cold slush solution for

topical cardiac hypothermia was proven to lead to an elevation of the left hemidiaphragm in 60% of the cases. Cardiac insulation pads were proven a less invasive method to protect the heart.[70]

The high right internal mammary artery harvesting was shown to lead to right phrenic nerve injury in 4% of cases in a prospective study by Deng and colleagues (2003).[71]

Overall, surgical procedures located on the path of the phrenic nerve, can lead to its injury. This includes otorhinolaryngological surgery and brachial plexus surgery. Traumatic injuries of the upper limb can also be responsible of phrenic injuries.[67]

ii. Infections

Cervical and more rarely thoracic herpes zoster should be considered as a possible cause of diaphragmatic paralysis. Case reports of the etiologies in the literature are numerous.[72-74]

Lyme neuroborreliosis has been reported as the cause of unilateral or bilateral diaphragmatic paralysis in many cases.[75, 76] The disease should always be considered for patients with unexplained DP in endemic areas.

iii. Compression

All thoracic tumors close to the phrenic nerve may damage it, by direct infiltration or by external compression. Bronchogenic carcinoma and metastatic hepatocellular carcinoma were both reported as etiologies of unexplained DP.[28]

The phrenic nerve may be compressed by a large goiter, leading to the nerve's paralysis.[77]

Neurofibromatosis in the context of Von Recklinghausen disease has also been reported as a possible cause of diaphragmatic paralysis.[78]

Cervical spondylosis can also lead to phrenic nerve compression and should be considered and evaluated in patients with DP.[79]

iv. Polyneuropathy

Guillain-Barré syndrome is the most common cause of neuromuscular respiratory failure, occurring in a third of all Guillain-Barré patients.[80]

Other causes of neuropathy (Critical illness polyneuropathy, Charcot-Marie-Tooth disease, Acute porphyria) are less frequent but should still be ruled out.[81]

v. Inflammatory diseases

DP can be secondary to various systemic conditions. Systemic lupus erythematosus can be complicated by shrinking lung syndrome, characterized by a weakened elevated diaphragm associated with progressive dyspnea.[82]

Dermatomyositis, collagen vascular disease and sarcoidosis may also cause DP, secondary to either a phrenic nerve injury or a myopathy.[33, 83]

c. Diaphragm myopathies

Myasthenia Gravis can cause respiratory failure secondary to bilateral diaphragmatic paralysis.[84] The symptomatology often occurs during a myasthenic crisis.[34]

Metabolic disorders such as hypocalcemia, hypokalemia, hypomagnesemia, or hypophosphatemia are known reversible causes of myopathies.[34]

Botulinum toxins may interfere with acetylcholine release which impairs the diaphragmatic function.[85]

Hypothyroidism is associated with a peripheral myopathy which can present as a diaphragmatic weakness, reversible after hormonal replacement.[86]

d. Pulmonary causes

Pneumonia has been reported as a possible etiology of diaphragmatic paralysis. Cases of unilateral DP following a pulmonary inflammation on the same side have been observed.[87, 88]

Lung hyperinflation as seen in COPD has shown detrimental effects on the diaphragm. It induces mechanical disadvantages and may flatten the diaphragm, and reduce its zone of apposition.[89]

e. Idiopathic

Finally, when no apparent etiology can be found despite all investigations, diaphragmatic dysfunction is said idiopathic.[60, 67]

7. Treatment

a. Objectives

Whichever method of treatment is chosen, the purpose is ultimately to:

- Improve the dyspnea and treat other symptoms.
- Prevent and/or treat the complications.
- Prevent the recurrences.

b. Medical treatment

i. Management of comorbidities

Obesity and COPD are both conditions associated with dyspnea and lower exercise tolerance. Which is why the therapeutic management of DE should start with an optimal medical treatment of these conditions, if present.

•Obesity

Obesity is a proven risk factor for dyspnea,[90] and the improvement of respiratory function after weight loss has also been demonstrated[91]. The most appropriate therapy depends on the individual. Traditional behavioral programs based on diet and physical activity are important. When insufficient, pharmacotherapy can be associated. In the most extreme cases, bariatric surgery is the final option.[92]

•COPD

COPD can lead to a diaphragmatic dysfunction through lung hyperinflation, or be associated to a DE caused by a different etiology, in which case it may aggravate the dyspnea. Treating COPD is thus an essential part of

the management of diaphragmatic dysfunction. This treatment must be carried out according to the GOLD guidelines, and adapted to each patient's situation.

ii. Inspiratory muscle training

A 2013 prospective study from Kodric and colleagues used an adjustable pressure device designed for IMT to train the diaphragm after cardiac surgery. Results showed increased muscle mobility and improved inspiratory diaphragm strength in patients with post-surgery diaphragm dysfunction. This allowed to avoid a surgical management of DP.[93] A 2009 study from Petrovic and colleagues, has shown an improvement that enabled the cessation of nocturnal use of non-invasive ventilation therapy in patients previously dependent on it.[94]

In our study, the group benefiting from physical therapy as sole treatment for their DE, didn't show significant improvement both clinically and in their spirometry results.

iii. Non-invasive ventilation techniques

When nocturnal symptoms are present in the form of sleep-disordered breathing, continuous positive airway pressure is the treatment of choice. In more severe cases, particularly bilateral DP, non-invasive positive pressure ventilation is the treatment of choice.[60]

These non-invasive ventilation techniques are described in the symptomatic management of DP and prevention of ventilatory failure.[95] A 2007 study from Versteegh and colleagues, reports the beneficial use of CPAP masks for several patients of their study, though the negative impact on their daily life is not to be disregarded.[96]

c. Surgical treatment

The first published surgical repair of DE dates back to 1923.[4] Still today, most surgical procedures are based on plication techniques, which is the immobilization of the weak diaphragm, using folds that reduce the dysfunctional muscle's paradoxical movement during breathing.

i. Indications of plication

Diaphragmatic plication is a symptomatic treatment of DE. It only aims to improve symptoms, and is thus exclusively indicated for symptomatic patients.

"We don't operate a radiological image": The presence of a paradoxical motion or an elevated hemidiaphragm do not, by themselves warrant surgery.

The severity and duration of symptoms must be considered, and other possible causes of said symptoms eliminated, before diaphragmatic plication is performed. Some studies recommend to consider surgical plication for patients with quality-of-life impairing dyspnea caused by DE, present for at least 6 months, since a spontaneous diaphragmatic recovery is possible.[16, 21, 97, 98]

For adult patients presenting a DE following cardiac surgery, an observation period of 1 to 2 years is often recommended. Multiple studies having proven an improvement of the phrenic nerve function with time.[48, 99-101]

ii. Contraindications of plication

Relative contraindications to surgical treatment of DE include:

- Morbid obesity: Patients with a BMI >35 should only be considered for surgery after a significant weight loss. First because dyspnea may

improve after medically or surgically treating the obesity, thus canceling the indication for a plication. But also, since in overweight patients, the procedure is more technically challenging, and the efficacy of the plication limited.[16, 98, 102]

- Neuromuscular disorders: the benefits of plication are too low compared to the risk of complications.[16, 98]
- Pleural carcinosis or any pathology with a short survival period.[54]
- Any general anesthesia contraindication.

Patients from all these categories require an individualized multidisciplinary approach to determine if they're possible candidates for diaphragmatic plication.

iii. Thoracic approaches

Open transthoracic plication

Open transthoracic plication is the most traditional approach, in the treatment of symptomatic DE. Though techniques may vary, the plication is performed under general anesthesia and with single-lumen intubation, through a posterolateral thoracotomy in either the 6^{th}, 7^{th} or 8^{th} intercostal space. After examining the lung, mediastinum, and phrenic nerve, the diaphragm is plicated in multiple rows, until taut. Using U-stitches, mattress sutures or running ones with pledgets sometimes to prevent tearing out. An intercostal drain is left in place and the thoracotomy closed in layers. [20, 103-105]

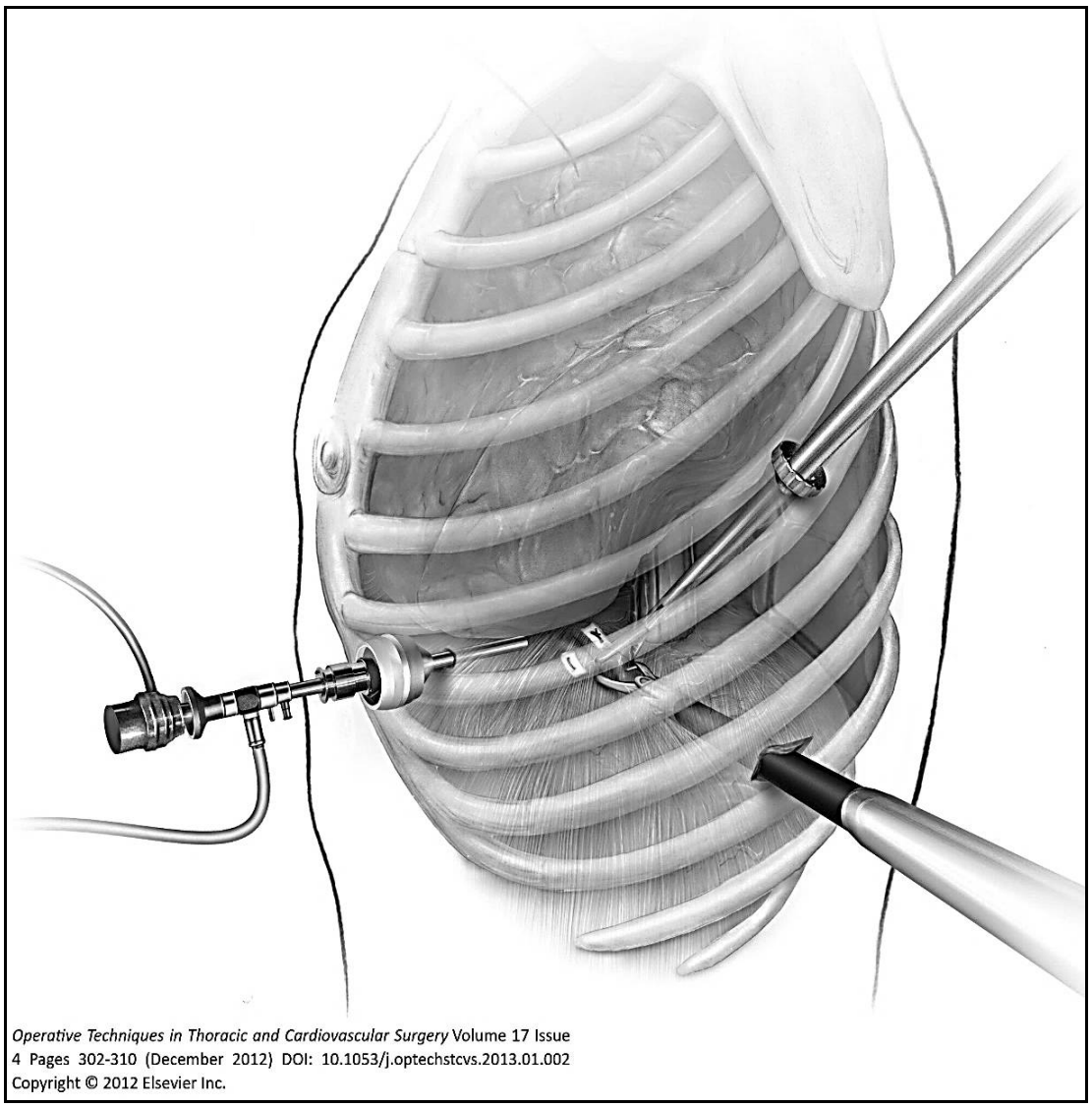
A 2010 study from Balci and colleagues of plication through a limited thoracotomy incision, compared the use of plication alone and that of diaphragmatic patches over the plicated diaphragm. It concluded to the possible decrease of recurrences with the patch reinforcement.[106]

The results that can be achieved by this approach have been reported by multiple studies. Verseteegh and colleagues studied the long-term effects of plication on dyspnea levels and pulmonary function on 22 patients between 1996 and 2006. They were the first to describe bilateral diaphragm plication and show its results. A transition dyspnea index was used [-9 - +9] and showed a postoperative functional improvement of +6.38 in patients with unilateral DP, and +2.67 in patients with bilateral DP. PFT registered an improvement of both VC (13%) and FEV1 (10%) at the 4-year follow-up.[96]

Between 2002 and 2013, Shah and colleagues followed 38 patients that underwent plication through thoracotomy. Dyspnea was graded according to MRC scale [Grade 1 to 4], and values went from 2.6 before surgery to 0.56 after it. Spirometry values improved by a mean of 11.5% for FVC, and 11.7% for FEV1. Most patients whose daily activities were compromised because of the dyspnea, were able to return to work.[18]

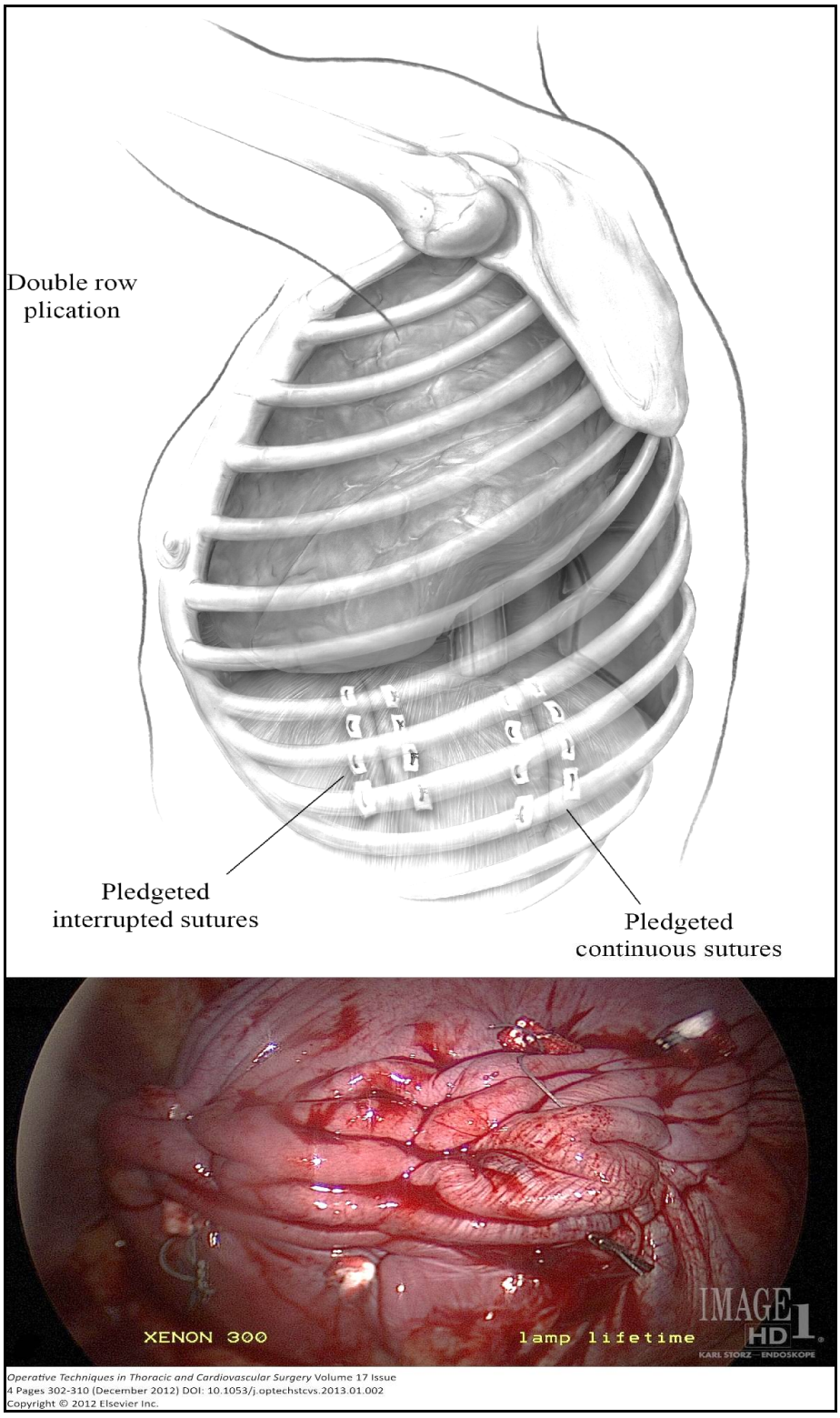
Thoracoscopic plication

Following the development of video-assisted thoracoscopic surgery, minimally invasive plication has become an alternative to the standard transthoracic open one. Using 2 (mini thoracotomy) to 3 ports, and with a contralateral single lung ventilation, the weak hemidiaphragm is plicated. As with open thoracotomy, a series of U-stitches or interrupted horizontal mattress sutures and pledgets can be used. Some teams opt for a partial resection of the stretched diaphragm, additionally simplified by the use of an endostapling device for the suturing, but we don’t agree with this method which could increase the risk of rupture. A chest tube is left in place before closing the incisions.[21, 107-110]



Operative Techniques in Thoracic and Cardiovascular Surgery Volume 17 Issue 4 Pages 302-310 (December 2012) DOI: 10.1053/j.optechstcvs.2013.01.002

Figure 44: Schema of VATS port arrangement for endo-suturing assist device during diaphragm plication



Operative Techniques in Thoracic and Cardiovascular Surgery Volume 17 Issue
4 Pages 302-310 (December 2012) DOI: 10.1053/j.optechstcvs.2013.01.002

Figure 45: Schema and photograph of thoracoscopic double row plication technique with pledgeted continuous and interrupted sutures

Since its first introduction by Moroux in 1996, thoracoscopic plication has advanced a lot. The approach can now be uniportal, or robot-assisted as reported using the Da Vinci Xi Robot (Intuitive Surgical, Sunnyvale, CA), avoiding many technical difficulties.[111-113]

Between 1992 and 2003, Mouroux and colleagues evaluated the results of diaphragm plication through VATS in 12 patients. All patients reported a complete disappearance of symptoms after the surgery. The 1-year spirometry, done for 10 of the patients, showed an improvement of 30% in FVC and 22% in FEV1. A 5-year follow-up was possible for 6 patients, offering long-term results of a technique that was still, at the time uncommon. The improvement compared to the preoperative values was of 18% for the FVC and 25% for the FEV1. The study was able to recommend the minimally invasive approach as an alternative to standard thoracotomy.[31]

Freeman and colleagues followed 41 patients who underwent plication through either thoracoscopy (30) or thoracotomy (11), from 2001 to 2007. Dyspnea improved significantly after the plication in 37 patients, with MRC dyspnea scores [1–5] going down by 2 compared to preoperative value, this improvement was still present at the 48-month follow-up. 31 patients, whose dyspnea prevented from being professionally active, were able to return to work after the plication. Mean spirometry values showed significant and constant improvement in all 41 patients: +19% in FVC 6-month after surgery, and +17% 48-month after it. FEV1 values registered a 23% improvement at the 6-month follow-up, and a 21% rise after 48-month.[102]

The experience in our unit

In our unit, diaphragmatic plication through thoracotomy was, for decades, the standard approach. Reinforced, when patients were at risk of recurrences, by a diaphragmatic patch. As said above, in our practice no diaphragm resection is used because of the high risk of suture rupture that could be associated with it.

With the development of minimally invasive techniques in our unit, thoracotomy is now reserved for cases that don't qualify for a VATS, which is associated with lower morbidity and consequently shorter hospitalization periods. Although during the period of this study it wasn't of common practice yet, it is now the method of reference with good results.

The results of our study showed an FEV1 improvement of 15%, and an FVC improvement of 0.52L for surgically treated patients. Furthermore, 76% of these group B patients reported a clinical improvement of their symptoms and were satisfied with the treatment's results. The comparison with the exclusively medically treated patients supports the significance of these results. Since in this group A, spirometry's mean values didn't show any marked improvement with an FEV1 rise of 4% and an FVC improvement of 0.21L. Additionally, patients were only satisfied with the clinical result in 22.9% of the cases.

Surgical plication in our practice, was associated with relatively low morbidity rates and no mortality.

We were limited by the absence of a standardized score for dyspnea, and the relatively short-term follow-up. Further studies of thoracoscopic techniques now that they're commonly used could prove helpful.

Our study confirms, however the conclusion from previous ones: the use of surgical plication through a thoracic approach leads to significant results, and is thus an efficient method in the treatment of symptomatic DE.

iv.Abdominal approaches

Laparoscopic plication

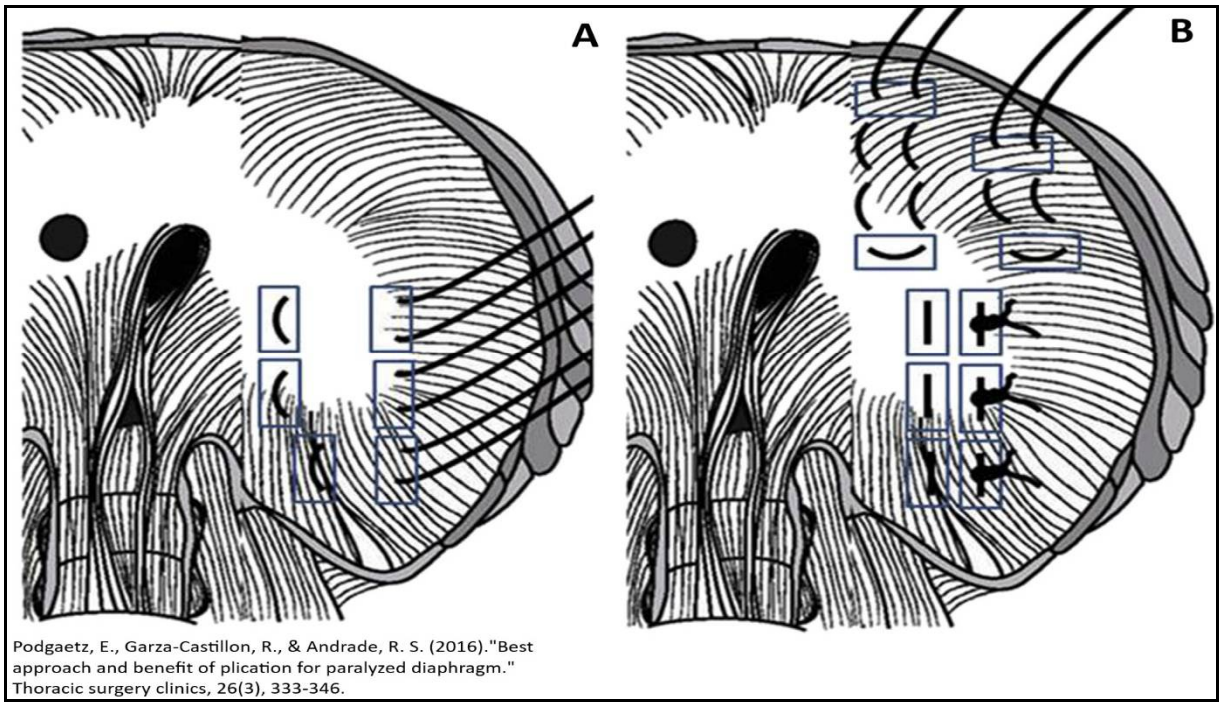
The use of laparoscopy is another minimally invasive approach that has been successfully used in the management of DE. Most teams use 4 ports, after an abdominal insufflation of CO2, a controlled pneumothorax is created through a 5mm defect in the diaphragmatic dome, facilitating the access to the diaphragm. The plication is then constructed using pledgeted U-stitches.[98, 114] The reinforcement of the repair using a patch on the abdominal side of the hemidiaphragm has been reported.[115]

Similar to the evolution seen with the thoracic approach, abdominal robot-assisted diaphragmatic plication is benefitting from rising interest. The laparoscopic use of the Da Vinci Surgical System (Intuitive Surgical, Sunnyvale, CA) has been reported as a viable option with good clinical outcomes.[116, 117]

Hüttl and colleagues introduced laparoscopic plication in a study between 1994 and 1998. Their study had many limitations, but concluded to the attractiveness of this alternative minimally invasive method.[118]

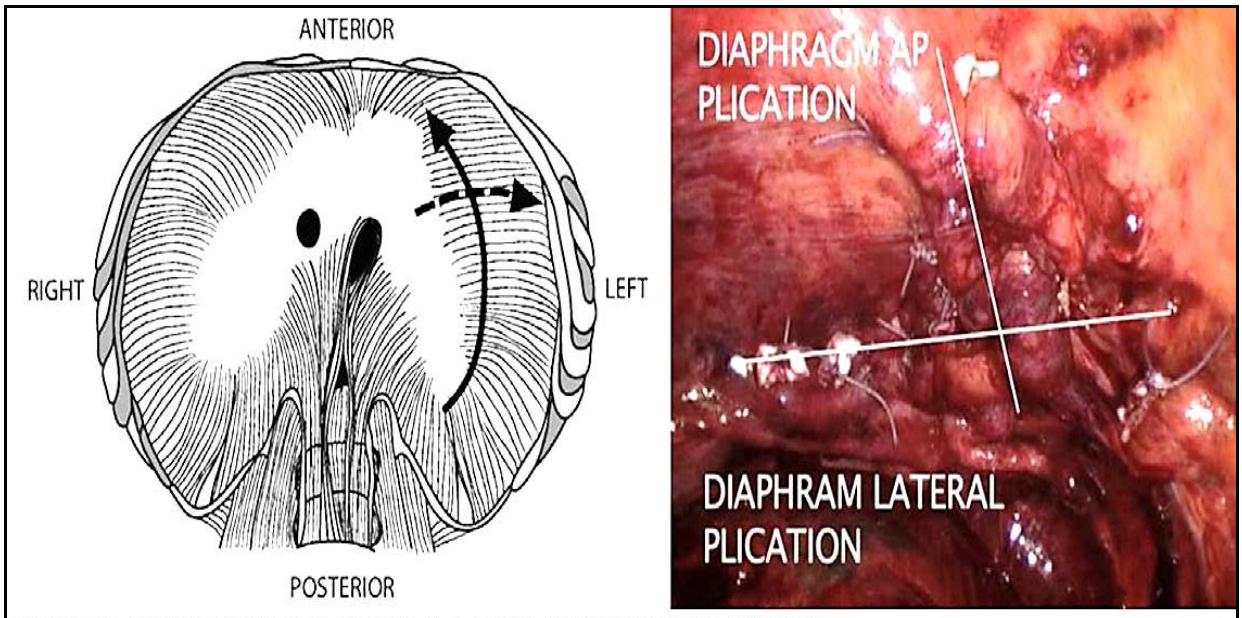
A retrospective study conducted by Groth and colleagues between 2005 and 2008, was able to offer a better review of laparoscopic plication. It followed 25 patients for a year, using a respiratory quality-of-life questionnaire score, that showed a reduction of at least 20 points at both the 1-month and 1-year follow-ups. Spirometry values went up, with an improvement of both FVC (+10.3% after 1 month, +3% after 1 year) and FEV1 (+12.3% after 1 month, +7.4% after 1 year). The study concluded to the possibility of obtaining the same positive results with laparoscopic plication while avoiding the limitations of thoracic approaches.[114]

A more recent study, from Roy and colleagues, retrospectively followed 22 patients who underwent transabdominal robot-assisted diaphragm plication. Conducted between 2012 and 2016, the study aimed to introduce the novel technique, prove its efficiency and benefits as a minimally invasive approach that provides the surgical dexterity of an open one. The mean MRC dyspnea score [1–5] went down by 2 points at postoperative follow-up (around 2 years after surgery). The study was limited by the absence of postoperative PFT data making objective functional evaluation impossible.[117]



Podgaetz, E., Garza-Castillon, R., & Andrade, R. S. (2016)."Best approach and benefit of plication for paralyzed diaphragm." Thoracic surgery clinics, 26(3), 333-346.

Figure 46: Schema of laparoscopic plication stitches: medio-lateral (A) and postero-anterior (B)



Podgaetz, E., Garza-Castillon, R., & Andrade, R. S. (2016). Best approach and benefit of plication for paralyzed diaphragm. Thoracic surgery clinics, 26(3), 333-346.

Figure 47: Schema and photograph of final "T-shaped" result of laparoscopic plication

Gastropexy

When digestive symptoms are present, particularly an important gastroesophageal reflux, an anti-reflux procedure might be attempted, alone or associated to the plication. Gastropexy is a simple and fast intervention that can instantly improve digestive symptoms, based on the repositioning and fixation of the stomach to avoid its migration under the elevated hemidiaphragm. It doesn't correct the eventration but can be associated to a plication through either a laparotomy or laparoscopy.[119]

Performed through an abdominal subcostal incision, allowing access to both the diaphragm and stomach, the most common procedure associates a plication of the weak hemidiaphragm, with an anterior gastric fixation to the abdominal wall.[120, 121]

Laparoscopic anterior gastropexy has also been successfully attempted, in cases that didn't require an emergency treatment.[42, 122]

The use of gastropexy has also been reported as an emergency procedure in cases of gastric volvulus complicating a DE. Symptomatic gastric volvulus associated with DE requires emergency surgical repair, because of the risk of ischemia and perforation. In this case, the procedure is commonly performed through an open diaphragm, and a reduction of the volvulus with a control of the ischemic stomach precedes the gastropexy.[43, 121]

v. Complications of plication

In most studies, a feeling of tightness in the lower chest or upper abdomen is commonly reported after the surgery.[96, 104]

Complications of plication, though cited in literature, aren't frequent in published series.[20, 123] The most commonly reported complications are shown in the following table:

Table IV: The most frequent complications of surgical diaphragm plication

	Complications
Respiratory	- Pneumonia [31, 102-104] - Prolonged chest tube drainage [114] - Pleural effusion [32, 114] - Surgical emphysema [18] - Recurrence [104]
Digestive	- Paralytic ileus [96, 102] - Abdominal compartment syndrome [123] - Upper gastrointestinal hemorrhage [114]
Cardio-vascular	- Deep vein thrombosis [21, 102] - Arrythmia (most often atrial fibrillation) [18, 102, 114] - Pulmonary embolism [96]
Cutaneous	- Superficial site infection [18, 21]
Neuropathic	- Post-thoracotomy pain syndrome [124]

vi. Comparison between the surgical plication approaches

To our knowledge, no study comparing the efficacy and morbidity of different surgical techniques has been published yet. In our practice, thoracoscopic plication has proven less invasive than the open one, with fewer post-operative complications and a shorter hospitalization. Although our current study can't make any comparison because of the small proportion of thoracoscopically treated patients. Previous studies have shown the benefits of minimally invasive techniques over open ones.[125] An additional advantage of the thoracoscopic approach that must be cited is the lower risk of post-thoracotomy pain syndrome.

Laparoscopy also presents all the advantages of a minimally invasive approach. It allows the dissection of any abdominal adhesions to the diaphragm and a gastropexy can be associated in case of digestive symptoms. It also avoids selective lung ventilation and chronic neuropathic pain. But it doesn't offer any exploration of the thoracic structures.

In the end, the choice depends on the surgeon's experience and preference, and the available equipment. Regardless of the chosen approach, all plication techniques are based on the same principle: restoring a strong barrier between the thoracic cavity and the abdominal one in patients with symptomatic DE.

vii. Phrenic nerve approaches

Phrenic nerve reconstruction

Recent studies have shown that phrenic nerve reconstruction may be an effective alternative to diaphragm plication in the management of selected cases of unilateral DP.[126] Based on peripheral nerve surgery techniques, this surgical procedure starts with nerve decompression, followed by nerve grafting for segmental injuries of the phrenic nerve, or nerve transfer in case of cervical root or proximal phrenic nerve lesions. The efficacy of this technique has been demonstrated, and new algorithms including it in the therapeutic management of symptomatic DP proposed.[126, 127]

Kaufman and colleagues evaluated phrenic nerve reconstruction as an alternative treatment possibility in selected cases. Between 2008 and 2012, they reviewed 68 patients with DP who underwent phrenic nerve surgery, and compared the results with those obtained from a plication group and a nonsurgical group. Spirometry follow-up values registered a mean improvement of 13% in FEV1 and 14 % in FVC with phrenic nerve surgery. Plication surgery improved PFT values by 17% for FEV1 and 17% for FVC. The nonsurgical treatment group showed an average improvement of 1.7% in FEV1, but FVC dropped by an average 0.4%. Despite the slightly better results obtained with plication, the study concludes to the possibility of further gradual improvement in the phrenic nerve surgery group, which could be assessed by a longer follow-up (>12 months).[127]

A study led by the same team, Kaufman and colleagues, was able to prove this point in 2015, thanks to a long-term follow-up of 180 phrenic nerve reconstruction patients. They demonstrated the incrementality of the

improvement in diaphragm motor amplitude with longer follow-ups. Which increased from 37% 1 year after the surgery, to 125% after 2 years. Supporting the notion that peripheral nerve regeneration's effect on muscle strength may take several years to reach optimal levels.[126]

Phrenic pacing

Phrenic pacing is indicated for ventilator-dependent patients presenting a bilateral DP with intact phrenic nerves. Those are patients with eventration due to central causes, mainly high cervical cord quadriplegia and central hypoventilation.

This technique is based on an artificial phrenic stimulation provided by a surgically implanted device. The surgical approach can be either thoracic (mini-thoracotomy or VATS) or abdominal (laparoscopic). In both cases, surgery allows the restoration of the diaphragm's contractile activity and shows good results in selected patients.[34, 128]

8. Management of a peripheral diaphragmatic paralysis

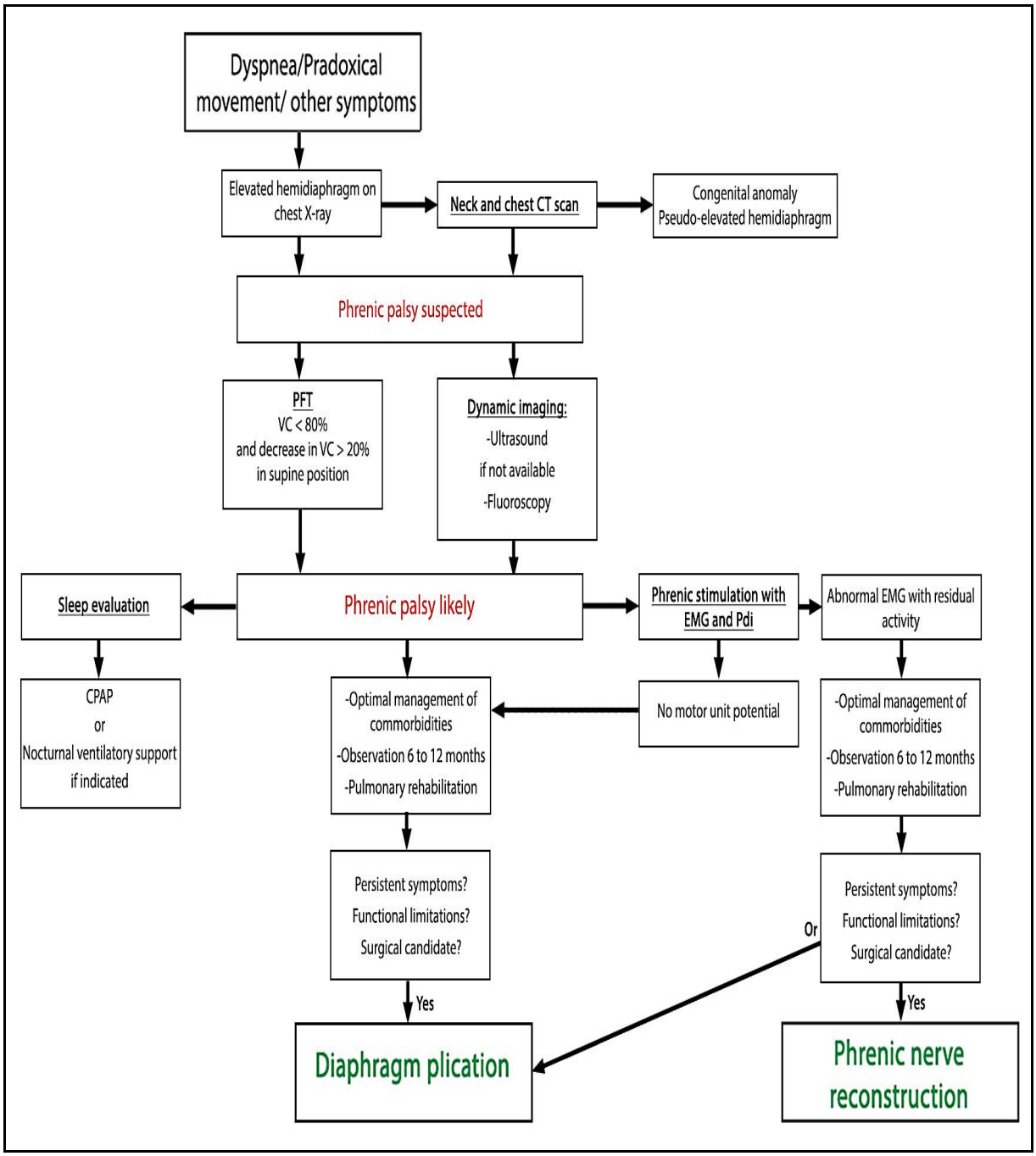


Figure 48: Suggested diagnostic and treatment algorithm for symptomatic DE

Conclusion

DE is a dysfunction of the diaphragm. It can be defined as a congenital or acquired inability to contract, leading to a loss of the diaphragm's power as a barrier between the thoracic and abdominal cavities. It can affect one or both hemidiaphragms, and be partial or total. Although the dysfunction commonly occurs on the left hemidiaphragm.

The etiologies responsible for this rare condition can be central or peripheral, but phrenic nerve injuries causing a DP are most often to blame.

The clinical presentation of DE is diverse and nonspecific, it is typically associated with an elevated hemidiaphragm on chest radiograph. The positive diagnosis is based on static and dynamic imaging techniques. An evaluation of pulmonary impact, mainly using PFT is crucial. Functional assessment of the diaphragm and phrenic nerve complete the paraclinical diagnosis.

The therapeutic management of this pathology can be medical. Based on the management of comorbidities, inspiratory muscle training, and non-invasive ventilation techniques.

Surgical treatment was proven to be the gold standard for symptomatic patients' management. Plication through open or minimally invasive thoracic approaches has shown great results. It can considerably improve the patients' quality of life. Furthermore, the continuous development of surgical techniques simplifies the procedure and decreases its morbidity. The recent addition of phrenic nerve reconstruction to the surgical treatment methods of DE, and its promising results, is also of interest for selected patients.

Our study was able to objectively prove the therapeutical benefit of surgical plication through a thoracic approach for patients with symptomatic DE. Based on our results and those of reviewed literature, we were able to suggest an algorithm, which could guide the diagnostic and therapeutic management of DE in symptomatic patients.

Abstract

Abstract

Title: The impact of surgical plasty in diaphragmatic eventration

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Director: Pr KABIRI El Hassane

Keywords: Diaphragm; Eventration; Paralysis; Surgery; Plication

Introduction: Diaphragmatic eventration is a rare condition, defined by an abnormal unilateral or bilateral elevation of the diaphragm while maintaining all its anatomical attachments. It can be congenital or acquired, due to central or peripheral causes. Our study aims to determine the therapeutic benefit of surgical diaphragm plication for symptomatic patients presenting this condition.

Material and methods: A retrospective descriptive case series study was conducted from January 2010 to December 2018. It followed 86 patients divided in two groups. Group A included 48 patients who received exclusively medical treatment by physical therapy. Group B included 38 patients who underwent plication surgery through a thoracic approach. Spirometry results (FEV1, FVC) and clinical improvement (using a satisfactory questionnaire) were evaluated pre- and post-therapeutically, and compared between both groups.

Results: In group B, 36 patients underwent plication through thoracotomy and 2 through thoracoscopy. The median of chest tube drainage was 2.5 days, and that of hospitalization was 4.5 days. 3 patients presented complications: pyothorax (n=2) and hemothorax (n=1). During follow up, recurrence was observed in 3 cases, all successfully reoperated. There was an average improvement in FEV1 of 15% and in FVC of 0.52L. These results were statistically significant (p value<0.05). A clinical improvement was reported in 29 (76%) cases. In group A, FEV1 only increased by 4% and FVC by 0.21L, these results weren't statistically significant (p value>0.05). A clinical improvement was reported in only 11 (22.9%) patients.

Conclusion: Our evaluation of diaphragmatic plication proved its objective benefit in the treatment of symptomatic eventration of the diaphragm, by demonstrating significant clinical and functional improvement.

Résumé

Titre: Impact de la chirurgie sur l'éventration diaphragmatique

Auteur: BOUGHDADI Soraya

Directeur: Pr KABIRI El Hassane

Mots clés: Diaphragme; Éventration; Paralysie; Chirurgie; Plicature

Introduction : L'éventration du diaphragme est une pathologie rare, définie par l'élévation anormale du diaphragme, sans rupture de sa continuité. Elle peut être congénitale ou acquise, de cause centrale ou périphérique. Notre étude a pour but de déterminer l'intérêt thérapeutique de la plicature chirurgicale chez les patients symptomatiques.

Matériel et méthodes : Entre janvier 2010 et décembre 2018, 86 patients divisés en 2 groupes étaient inclus dans une étude rétrospective, descriptive d'une série de cas. Le groupe A (n=48) a reçu un traitement médical uniquement, par kinésithérapie. Les patients du groupe B (n=38) ont été opérés par voie thoracique pour plicature diaphragmatique. Les résultats de la spirométrie (FEV1, FVC) et l'amélioration clinique (par un questionnaire de satisfaction), évalués avant et après traitement, ont été comparés chez les deux groupes.

Résultats : Chez le groupe B, 36 patients ont été traités par thoracotomie, et 2 par thoracoscopie. La médiane de drainage thoracique était 2,5 jours, celle d'hospitalisation 4,5 jours. Chez 3 patients, des complications postopératoires ont été observées et traitées : pyothorax (n=2) et hémothorax (n=1). Pendant le suivi, 3 cas de récidive ont été observés et réopérés avec succès. Une amélioration moyenne de 15% du FEV1 et de 0,52L du FVC a été notée. Ces résultats étaient statistiquement signifiants ($p<0.05$). Cliniquement, 29 patients (76%) ont rapporté une amélioration. Chez le groupe A, l'élévation de 4% du FEV1 et de 0,21L du FVC, n'étaient pas statistiquement signifiantes ($p>0.05$). Seuls 11 patients (22,9%) étaient cliniquement satisfaits.

Conclusion : Notre étude a objectivement prouvé l'intérêt de la plicature diaphragmatique dans la prise en charge thérapeutique des patients présentant une éventration symptomatique du diaphragme.

ملخص

العنوان: تأثير الجراحة في اندحاق الحجاب الحاجز

الكاتبة: ريــاصـو بغــدادي

المشرف: الحســن كبـــيري الأســـتاذ

الكلمـــات ةىالأســـاس: الحجاب الحاجز - اندحاق الحجاب – الشلل – الجراحة - الثني

مقدمة: اندحاق الحجاب الحاجز حالة مرضـــية، يتم تحديدها من خلال ارتفاع غير طبيعي من جانب واحد أو ثنائي للحجاب الحاجز مع الحفاظ على جميع ملحقاته التشريحية. يمكن أن يكون خلقيًا أو مكتسبًا لأسباب مركزية أو محيطية. تهدف هذه الدراسة إلى تحديد الفائدة العلاجية لجراحة ثني الحجاب الحاجزالمرضــى لـدى الذين يعانون من أعراض.

الطرق و التدابر: من يناير2010 إلى غاية دجنبر2018 تم إجراء دراسة سلسلة حالات وصفية تراجعية و التي تتبعت 86 مريضًا منقسمين إلى مجموعتين. تضم المجموعة "أ" 48 مريضًا تلقوا علاجًا طبيًا عن طريق العلاج الطبيعي فقط. بينما ضمت المجموعة "ب" 38 مريضا خضعوا لجراحة الثني عن الصــدر طـريق. تم تقييم نتائج قياس التنفس (FEV1، FVC) والتحسن السريري (باستخدام استبيان مرضي) قبل وبعد العلاج ، ومقارنتها بين المجموعتين.

النتائج: في المجموعة "ب"، خضع 36 مريضا للثني من خلال شق الصدر و 2 من خلال تنظير الصدر. كان متوسط صرف الأنبوب الصدري 2.5 يوما، والمكوث في المستشفى 4.5 يوما.

عانى 3 مرضى من مضاعفات: تقيح الصدر (n = 2) وتدمي الصدر (n = 1). لوحظ عودة الأعراض في 3 حالات أعيـدت جراحتها جميعًا بنجاح. كان هناك تحسن متوسط في FEV1 بنسبة 15٪ وفي FVC قدره 0.52 لتر. كانت هذه النتائج ذات دلالة إحصائية (قيمة p <0.05). تم الإبلاغ عن تحسن سريري في 29 حالة (76٪).

في المجموعة "أ"، سـجلت زيادة FEV1 بنسبة 4٪ فقط و FVC بمقدار 0.21 لتر، ولم تكن هذه النتائج ذات دلالة إحصائية (القيمة p> 0.05). تم الإبلاغ عن تحسن سريري عنـد 11 مريض فقط (22.9٪).

الخلاصة: أثبت تقييمنا لثني الحجاب الحاجز فائدته في علاج أعراض اندحاق الحجاب، من خلال إظهار تحسن سريري و وظيفي بـــارزين.

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Serment d' H ippocrate

Au moment d'être admis à devenir membre de la profession médicale, je m'engage solennellement à consacrer ma vie au service de l'humanité.

- *Je traiterai mes maîtres avec le respect et la reconnaissance qui leur sont dus.*
- *Je pratiquerai ma profession avec conscience et dignité. La santé de mes malades sera mon premier but.*
- *Je ne trahirai pas les secrets qui me seront confiés.*
- *Je maintiendrai par tous les moyens en mon pouvoir l'honneur et les nobles traditions de la profession médicale.*
- *Les médecins seront mes frères.*
- *Aucune considération de religion, de nationalité, de race, aucune considération politique et sociale ne s'interposera entre mon devoir et mon patient.*
- *Je maintiendrai le respect de la vie humaine dès la conception.*
- *Même sous la menace, je n'userai pas de mes connaissances médicales d'une façon contraire aux lois de l'humanité.*
- *Je m'y engage librement et sur mon honneur.*

قسم أبقراط

بسم الله الرحمان الرحيم

أقسم بالله العظيم

في هذه اللحظة التي يتم فيها قبولي عضوا في المهنة الطبية أتعهد علانية:

- بأن أكرس حياتي لخدمة الإنسانية.
- وأن أحترم أساتذتي وأعترف لهم بالجميل الذي يستحقونه.
- وأن أمارس مهنتي بوازع من ضميري وشرفي جاعلا صحة مريضي هدفي الأول.
- وأن لا أفشي الأسرار المعهودة إلي.
- وأن أحافظ بكل ما لدي من وسائل على الشرف والتقاليد النبيلة لمهنة الطب.
- وأن أعتبر سائر الأطباء إخوة لي.
- وأن أقوم بواجبي نحو مرضاي بدون أي اعتبار ديني أو وطني أو عرقي أو سياسي أو اجتماعي.
- وأن أحافظ بكل حزم على احترام الحياة الإنسانية منذ نشأتها.
- وأن لا أستعمل معلوماتي الطبية بطريق يضر بحقوق الإنسان مهما لاقيت من تهديد.
- بكل هذا أتعهد عن كامل اختيار ومقسما بالله.

والله على ما أقول شهيد.

المملكة المغربية
جامعة محمد الخامس بالرباط
كلية الطب والصيدلة
الرباط

سنة : 2021

أطروحة رقم: 199

تأثير الجراحة في اندحاق الحجاب الحاجز

أطروحة

قدمت ونوقشت علانية يوم : / /2021

من طرف

السيدة صوريا بغدادي

المزدادة في **01** يناير **1996** بالحاجب

لنيل شهادة

دكتور في الطب

الكلمات الأساسية : الحجاب الحاجز؛ اندحاق الحجاب؛ الشلل؛ الجراحة؛ الثني

أعضاء لجنة التحكيم:

السيد الحسن كبيري أستاذ في جراحة الصدر	**رئيس ومشرف**
السيد أحمد بونعيم أستاذ في الجراحة العامة	**عضو**
السيد رحال مسروري أستاذ في الجراحة العامة	**عضو**
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السيد محمد مسين الحمومي أستاذ مساعد في جراحة الصدر	**عضو مشارك**