

Janusz Kocjan<sup>1</sup>, Mariusz Adamek<sup>1</sup>, Bożena Gzik-Zroska<sup>2</sup>, Damian Czyżewski<sup>1</sup>, Mateusz Rydel<sup>1</sup>

<sup>1</sup>Chair and Department of Thoracic Surgery, Faculty of Medicine and Dentistry, Medical University of Silesia, Katowice, Poland

<sup>2</sup>Department of Biomaterials and Medical Devices Engineering, Silesian University of Technology, Zabrze, Poland

## Network of breathing. Multifunctional role of the diaphragm: a review

The authors declare no financial disclosure

### Abstract

The diaphragm is the primary muscle involved in active inspiration and serves also as an important anatomical landmark that separates the thoracic and abdominal cavity. However, the diaphragm muscle like other structures and organs in the human body has more than one function, and displays many anatomic links throughout the body, thereby forming a 'network of breathing'. Besides respiratory function, it is important for postural control as it stabilises the lumbar spine during loading tasks. It also plays a vital role in the vascular and lymphatic systems, as well as, is greatly involved in gastroesophageal functions such as swallowing, vomiting, and contributing to the gastroesophageal reflux barrier. In this paper we set out in detail the anatomy and embryology of the diaphragm and attempt to show it serves as both: an important exchange point of information, originating in different areas of the body, and a source of information in itself. The study also discusses all of its functions related to breathing.

**Key words:** diaphragm, breathing, diaphragm function

**Adv. Respir. Med. 2017; 85: 224–232**

### Introduction

The scientific interest in diaphragm dates back to the end of the 19<sup>th</sup> c., when Sewall and Pollard investigated the relationship of movement between the thoracic cavity and the diaphragm. They assessed the diaphragmatic component of respiration by observing changes of circumference of the abdomen and concluded that the diaphragm contracts and descends into abdominal cavity [1]. At the beginning of the nineteenth century, Bell demonstrated that life could be maintained by diaphragmatic breathing alone after injuries to the cervical spinal cord in a man [2]. In the following decades researchers have investigated physiological mechanisms, biomechanical functions, and anatomical attachments of the diaphragm. Currently, it is well documented that

the diaphragm acts not only as a breathing muscle, but also plays multiple distinct physiological roles. Anatomically, the diaphragm only separates thoracic and abdominal cavities. However, from a functional perspective, this muscle extends from the trigeminal system to the pelvic floor, as well as has many links throughout the body and is an important crossroads of information involving the entire body. The phrenic nerve innervates the diaphragm, and runs from the roots of C3 to C5. The pathway of the phrenic nerve involves the entire brachial plexus and the entire cervical plexus. Along its pathway, the phrenic nerve anastomoses with the vagus nerve, which is joined to the medial longitudinal fasciculus and is in contact with the spinal trigeminal nucleus *via* afferent connections. Furthermore, the vagal nerve is firmly connected with the hypoglossal

**Address for correspondence:** Janusz Kocjan, Chair and Department of Thoracic Surgery, Faculty of Medicine and Dentistry, Medical University of Silesia, Katowice, Poland, e-mail: j\_kocjan@wp.pl

DOI: 10.5603/ARM.2017.0037

Received: 20.03.2017

Copyright © 2017 PTChP

ISSN 2451–4934

nerve, which is closely related to the trigeminal system and receives a multitude of presynaptic impulses from the phrenic nerve. With reference to neurological connections, dysfunction of the diaphragm can cause symptoms observed in the region of the cervical base and eyes, as well as in the floor of the mouth (poor swallowing or sleep apnoea). From lower side, the connection between the diaphragm and pelvic floor is less complicated. The respiration process has to be supported by the pelvic floor in order to properly control intra-abdominal pressure. During physiological diaphragmatic action (e.g. respiration, coughing), when the diaphragm descends into the abdominal cavity, the corresponding action of lowering of the pelvic floor is also observed. In this point it is also important to mention that electrical activity of the pelvic floor muscle is noticed before inhalation [3]. To summarise, Caroline Stone states: 'the diaphragm is one of the most remarkable areas of the body in that it has so much influence and the consequences of its dysfunction can manifest anywhere from the head to the toes' [4]. While Andrew Still, the founder of osteopathy and osteopathic medicine wrote: 'by (diaphragm) action we live, and by its failure we shrink, or swell, and die' [5].

### **Anatomy and anatomic connections of the diaphragm**

The diaphragm is a thin (2–4 mm), movable dome-shaped sheet of internal skeletal muscle that separates the thoracic and abdominal cavities. It also plays a vital role in managing the information related to both cavities. In anatomic position, the diaphragm curves into right and left domes (also known as cupolae). The right dome is slightly higher than the left one and reaches as high as the upper border of the fifth rib. The left dome may reach up to the lower border of the fifth rib. The reason behind the right dome being higher than the left one is probably the larger size of the right lobe of the liver. The central tendon, which lies between the two domes, remains at the level of the xiphisternal joint. The domes of the diaphragm support the right and left lung while the central tendon provides support to the heart. The position and shape of the diaphragm is not absolute, but varies with the phase of respiration. With full exhalation, the dome of the diaphragm can rise to the level of the fourth intercostalspace anteriorly (nipple level). With full inhalation, the diaphragm flattens, bringing the thoracic cavity down to the level of costal margin anteriorly

and the 12<sup>th</sup> rib posteriorly. The position also depends upon the posture of the body (lower when a person is sitting or standing and higher in supine position) and degree of distention of the abdominal viscera [6, 7].

Structurally, the diaphragm comprises two parts: a centrally placed, non-contractile tendon — a flat aponeurosis made of dense collagen fibers, and the peripheral muscle — that converge on the central tendon. The muscular portion is divided into three parts based on fibers origin. Fibers of the sternal parts are short and arise as small slips from the posterior surface of the xiphoid process. The costal region (laterally on either side of the xiphoid) is made up of several wide muscle segments originating from the internal surface of the caudal six ribs at the costal cartilages (costal margin), while the lumbar part of the diaphragm has its origins on the lumbar vertebra by two crura and three arcuate ligaments. The right crus attaches distally to the anterior portions of the first through third lumbar vertebrae (L1-3) and the left crus attaches distally on the first and second lumbar vertebrae (L1-2). Anatomists name the crura left or right by their origin from the left or right side of the vertebral bodies, but surgeons name the crura left or right by their relation to the esophagus. Lateral to the crura on both sides, the diaphragm arises from the medial and lateral arcuate ligaments. Arcuate ligaments are thickened upper margin of fascia covering the psoas muscle on its anterior surface (medial arcuate ligament) and the quadratus lumborum muscle (lateral arcuate ligament). The paired lateral arcuate ligaments extend from the tip and lower margin of the 12<sup>th</sup> rib and arch over quadratus lumborum to the transverse processes of L1 [6–8]. Although the lateral arcuate ligament is commonly described in anatomy books as attaching to the first lumbar vertebra (L1), other instances have been found in cadaver studies with attachments to either the second (L2) or third (L3) lumbar vertebra [9]. Approximately in 5% of people inferolateral extensions of the lateral arcuate ligaments is present as thickened nodular areas adjacent to the lateral diaphragmatic surface [10]. The paired medial arcuate ligaments, in contrast to the lateral ones, complete the journey arching over the psoas major from the tip of the transverse processes of L1 (sometimes also, of L2) to the tendinous portion of each diaphragmatic crus. The medial margins of the right and left crura unites to form a poorly defined arch — the median arcuate ligament, through which pass the aorta, the azygos vein, and the thoracic duct [8].

The diaphragmatic ligaments are structures that connect the diaphragm to the viscera. The inferior pulmonary ligament is a pleural thickening connecting the diaphragm to the base of the lungs. The phrenopericardial ligament connects the diaphragm to the heart and is the fulcrum around which the diaphragm is supported when it comes to distribute its contractile tension laterally. The hepatic ligaments (the falciform ligament and the right and left triangular ligaments) represent a subdiaphragmatic peritoneal thickening. The phrenicoesophageal ligament joins the esophagus and the diaphragm and is composed of loose connective tissue. And finally, the phrenicocolic ligament connects the diaphragm to the angle of the right ascending colon, while the ligament of Treitz is made up of a series of muscular tracts that start in the main left crura and go to the duodenojejunal angle [3].

### Embryology of the diaphragm

The diaphragm develops from four embryonic structures derived from the mesoderm, each of which either becomes totally incorporated into the diaphragm or merely contributes the part of itself to the diaphragm. The septum transversum is the first and the most important component present in the developing diaphragm. In the third week of embryonic life, the transverse septum lies at the level of the third cervical vertebra. By the end of diaphragm development in week 8, the early diaphragm descends to its ultimate position at the first lumbar segment, secondary to the rapid growth of the vertebral column. The septum serves as the initial barrier between the thoracic and abdominal cavities, however, it does not separate the thoracic and abdominal cavities entirely, but after the headfold forms, it becomes a thick incomplete partition between the cavities with an opening on each side of the gut, the pleural canals. The septum forms the central tendon of the diaphragm. The pleuroperitoneal membranes (folds), which originates from the caudal end of the pericardioperitoneal canals are the second important component of the developing diaphragm. These two transient, pyramidal-shaped structures lying on either side of the esophagus protrude from the body wall between the pleural and peritoneal cavities. The pleuroperitoneal membranes fuse with the dorsal mesentery of the esophagus and with the dorsal portion of the septum transversum to complete the partition between the pleural and peritoneal cavities, form the primitive diaphragm. It is cur-

rently unclear whether the pleuroperitoneal folds are simply transient embryonic structures with no adult derivatives or if they give rise to cells or tissues of the adult diaphragm. After the early diaphragm is formed, the dorsal mesentery of the esophagus (third embryonic structure) fuses with the two previously mentioned structures (septum transversum and the pleuroperitoneal membranes) to form the median portion of the diaphragm, while the body wall tissue (fourth major component) splits into two layers as a result of the enlargement of the lungs and the pleural cavities. The parts of the inner layer form the peripheral sections of the diaphragm. Extensions of the pleural cavities into the body walls form the costodiaphragmatic recesses, which forms the dome-shaped adult diaphragm [11, 12]. In terms of the embryonic origins, the diaphragm consists of two distinct muscles each of which has different actions on the rib cage. The crura of the diaphragm develop from muscle fibers, which grow into the esophageal mesentery, while myoblasts from the lateral body walls become the costal parts [13]. DeTroyer *et al.* [14] reported that when the costal muscle of dogs was electrically stimulated, lung volume and abdominal pressure increased, and there was an outward displacement of the abdomen and the lower rib cage. Electrical stimulation of the crural muscle had similar effects except there was no effect on the rib cage dimension. The crural muscle, which is attached to the lumbar vertebrae but not attached to the rib cage, functions mainly to exert abdominal pressure of the fully developed diaphragm. Crurae are pairs of diverging bundles of muscles.

The human diaphragm is supplied by the phrenic nerve, which contains nerve fibers emerging from the same segmental levels from which the diaphragm skeletal muscles arise (mnemonic, 'C3, 4, 5 keep the diaphragm alive'). Since the phrenic nerve is a bilateral structure running on either side of the midline, each nerve supplies motor innervation to one-half (ipsilateral) of the diaphragm on the same side. Sensory fibres from the phrenic nerve supply the central part of the diaphragm (including the surrounding pleura and peritoneum) and convey sensation from the diaphragm to the central nervous system (at C3-C5). Neurons at these spinal cord levels also receive sensation from the shoulders *via* the supraclavicular nerves (medial, intermediate and lateral) [15]. Therefore, irritation of the diaphragm or stimulation of sensory fibers in the phrenic nerve can be experienced as referred pain in the shoulder area, due to the embryological origins of the

diaphragm. After gynaecological laparoscopy, shoulder tip pain is induced by stretching of the diaphragm on insufflation of carbon dioxide to maintain the pneumo-peritoneum [16]. Women with a ruptured ectopic pregnancy may develop shoulder pain on depressing the head of the bed, presumably due to irritation of the diaphragm by blood and fluid [17]. Another examples of the same phenomenon is irritation of the under-surface of the diaphragm by blood leaking from a ruptured spleen [18]. Sutton *et al.* [19] in a single case study reported that shoulder pain was caused by phrenic artery rupture. Furthermore, diseases of nearby organs may also irritate the diaphragm. Peritonitis or gallbladder inflammation can irritate the phrenic endings in the central part of diaphragmatic peritoneum [20]. Söyüncü *et al.* [21] observed the left shoulder pain signaled by the phrenic nerve in case of splenic abscess.

### Mechanical action of the diaphragm

Although breathing process appears involuntary, the diaphragm's proper function and mechanical action efficiency largely depends on its anatomic arrangement with the lower rib cage. The area of attachment (apposition) between the diaphragm and the rib cage is referred as the zone of apposition (ZOA), and extends from the diaphragm's caudal insertion near the costal margin, cephalad to the costophrenic angle, where the fibers break away from the rib cage to form the free diaphragmatic dome. ZOA is a crucial and one of the most important aspects of breathing, responsible for: efficient length-tension relationships of the diaphragm, *i.e.* maintaining vertical alignment of diaphragm muscle fibers, postero-lateral (bucket-handle) movement of the lower rib cage. In upright position during quiet breathing, the zone of apposition represents about one third of the total surface area of the inner rib cage. During quiet inspiration, the diaphragm contracts, its axial length diminishes and the dome of the diaphragm descends relative to its costal insertions. The height of ZOA decreases by about 15mm, while the dome of the diaphragm remains relatively constant in size and shape. At maximum inspiratory capacity of the lungs, ZOA is almost zero [22, 23].

The area of apposition is controlled by the abdominal muscles and directs diaphragmatic tension. During the inhalation phase of ventilation, when the diaphragm contracts and descends downwards into the abdominal cavity, intra-abdominal pressure increases and distends

the abdominal wall in a three dimensions with accompanied rotation of the ribs outward. The abdominal wall opposes the action of the diaphragm with an eccentric contraction of all abdominal muscles, controlling the length-tension relationship of the diaphragm muscle. This eccentric contraction ensures the maintainance of the dome shape of the diaphragm and sustains the zone of apposition long enough to produce postero-lateral expansion of the lower rib cage and thereby facilitates the increased force of the diaphragm. Additionally, less activity of the abdominal muscles allows visceral displacement due to the dome of the diaphragm dropping. During expiration this action is reversed. The abdominal muscles contract concentrically, compresses the viscera in the abdominal cavity, whereby the diaphragm is forced in cephalad direction and the ribs internally rotate [24].

When ZOA is decreased (in a suboptimal position), the diaphragm has lesser ability to draw air into the thoracic cavity due to less caudal movement upon contraction and less effective tension of the diaphragm on the ribs and therefore lower transdiaphragmatic pressure [25]. This situation is accompanied by decreased expansion of the rib cage, postural alterations, and a compensatory increase of abdominal expansion [24]. As a result, the adaptive breathing strategies can develop, such as relaxation of the abdominal musculature more than necessary on inspiration what allows for thoraco-abdominal expansion or increased use of accessory muscle of respiration [25]. This situation leads to several potential negative consequences: shortness of breath (dyspnoea), decreased respiratory efficiency, decreased exercise tolerance, decreased intra-abdominal pressure, increased lumbar lordosis, increased hamstring length, increased abdominal length, sternum elevation, increased lumbo-pelvic instability, increased activity of paraspinals, low back pain, thoracic outlet syndrome, sacroiliac joint pain, headaches, asthma [26].

### (Multi)function of the diaphragm

The diaphragm is mainly recognised as the primary respiratory muscle of the body responsible for about 80% of all of the respiratory work in normal tidal breathing. However, like other structures in the human body, the diaphragm muscle has more than one function. By modulation of intra-abdominal pressure, it is related with postural stability, and assists in-micturition, defecation and parturition. It is also important for

cardiac function and lymphatic flow, furthermore, it plays a role in emesis, swallowing and as an anti-reflux barrier.

### Postural function

The postural function of the diaphragm, understood as trunk stabilisation and postural trunk control during repetitive movements, is inextricably linked with its breathing function. Trunk bracing maintains all spinal segments in a biomechanically neutral position during the course of any movement and is dependent on the dynamic coordination of numerous synergist and antagonist muscles for precise control of excessive joint motion. There is also a general consensus that an increase of intra-abdominal pressure (IAP) stabilises the spine. The diaphragm cannot move the trunk voluntarily, but its contraction contributes to trunk (spinal) stability *via* an increase of pressure in the abdominal cavity. This dual function of the diaphragm (ventilation and posture) is performed simultaneously [27–29].

During early postural development, the diaphragm functions primarily as a respiratory muscle. With continued central nervous system (CNS) maturation and development to about 4½ months of age, sagittal stabilisation of the spine, pelvis, and chest is fully established for subsequent movements that occur in the transverse plane, (*e.g.* rolling, turning, creeping, crawling) and eventually the transition to upright posture. The diaphragm begins to fulfill its dual function as both a respiratory and postural muscle when abdominal breathing is coordinated with chest breathing at about 6 months of age [30].

Several findings provide support for dual function theory of the diaphragm. Skladal *et al.* [31] already in 1969 were the first to provide indirect evidence of a contribution of the diaphragm to postural control. These studies documented contraction of the diaphragm prior to contraction of the rectus abdominis in preparation for rising onto the toes. Further studies reported a close relationship between transdiaphragmatic pressure and intra-abdominal pressure. Hemborg *et al.* [32] demonstrated that the diaphragm is tonically activated during lifting objects. The first direct evidence that the diaphragm may contribute to the postural control of the human trunk in addition to its role in respiration was demonstrated by Hodges and colleagues. They showed that electromyographic (EMG) activity of the diaphragm increased prior to the onset of activity of the muscle responsible for movement of

the contralateral upper limb. With rapid flexion of the shoulder in response to a visual stimulus, EMG activity in the costal and crural diaphragm occurred about 20 ms prior to the onset of deltoid muscle EMG. This feedforward feedback occurred irrespective of the phase of respiration. The diaphragm EMG activity was associated with an increase of transdiaphragmatic pressure. Ultrasonographic measurements also revealed that the costal diaphragm shortened and then lengthened progressively during the increase in transdiaphragmatic pressure [28]. Next studies conducted by Hodges have provided further confirmation of this function. Most of all, the postural activation of the diaphragm was unchanged when subjects performed an identical task in sitting position with the trunk unsupported. However, diaphragm activity was not present with single movements of small distal segments of the upper limb. Additionally, the amplitude of diaphragm EMG was linearly related to the peak acceleration of the limb and thus to the forces transmitted to the spine. And finally, rapid repetitive movement of the upper limb during apnoea (breath holding at end-expiration) also activated the diaphragm [33]. Recent two interesting MR imaging studies of the diaphragm conducted by Kolar *et al.* [34] support the thesis of dual function of respiration and spinal stabilisation. The first study demonstrated that the diaphragm has a postural function that can be voluntarily controlled and is independent of breathing. The results showed that the diaphragm's postural position is lower or similar to that of tidal breathing in 81% of the subjects, but the diaphragm range of movement (ROM) during both respiration and postural activities differ among individuals. There is also a significant correlation between the respiration volume and the diaphragm ROM during tidal breathing [34]. The second study demonstrated that the diaphragm's dual function can be performed simultaneously. The diaphragm can achieve its respiration function from a lowered position to ensure sufficient intra-abdominal pressure that is produced when required for a postural task [35]. At the base of results of the both studies mentioned above, the authors concluded that there is an individual ability to control the postural function of the diaphragm. Individuals with limited capability to contract their diaphragm for stabilisation of the body may have higher likelihood of development of back pain. Insufficient and uncoordinated diaphragm activation in people with weak body stabilising function of the diaphragm may leads to overloading of spi-

nal segments [36, 37]. It was already previously observed by Hodges, namely that if the demand for breathing increases, the role of the diaphragm in low back stability declines [36], and again it was confirmed later by other authors. Janssens *et al.* [37] showed that an increased demand for one of diaphragm functions (an inspiratory loading task) will inevitably abolish the other function, in terms of impaired balance control. In another study, Vostatek *et al.* [38] demonstrated that individuals with low back pain move their diaphragms about half less, compared to healthy subjects. And once again the next study performed by Janssens *et al.* [39], in 2013. They found that individuals with low back pain exhibited significant diaphragm fatigue after inspiratory muscle loading, what was not observed among healthy controls. The authors suggest that fatigability of the diaphragm may be a potential underlying mechanism in the aetiology of recurrent non-specific low back pain [39].

### Cardiac function

Breathing not only involves gas exchange to and from the lungs and blood stream, but also is a modulator of cardiovascular control reducing negative intrathoracic pressure through inhalation, thereby decreasing left ventricular afterload and generating a transdiaphragmatic gradient that promotes venous return. Stone suggested that diaphragm movement may influence the movement of the heart as the pericardial sac is connected to the diaphragm by phrenicopericardial ligaments, and a lack of diaphragm movement may reduce heart contractility and blood circulation throughout the body [3]. Respiratory sinus arrhythmia (RSA), one of the physiologic interactions between respiration and circulation, is heart rate variability in synchrony with respiration, by which the R-R interval on an ECG is shortened during inspiration and prolonged in expiration [40]. Kulur *et al.* [41] investigated the effect of diaphragmatic breathing on heart rate variability in ischaemic heart disease. They noted that regular practice of diaphragmatic breathing significantly improves heart rate variability (HRV) with a favourable prognostic picture in ischaemic heart disease patients. There was also a significant increase in HRV in normal control subjects who practiced diaphragmatic breathing for one year. Lee *et al.* [42] investigated the effects of normal breathing and diaphragmatic breathing on blood pressure (BP) and heart rate (HR) in a single case study, in which BP and HR were being re-

corded during three weeks. The authors reported that diaphragmatic breathing was associated with a statistically significant reduction in systolic and diastolic blood pressure. Furthermore, the most of others parameters of the cardiac function such as ejection fraction, aortic pressure, and pulmonary arterial pressure, preload and afterload and even tissue oxygenation have been shown to be mode-locked to breathing [43, 44]. Diaphragmatic contractions also increase cardiac output under conditions of pre-load dependency, which has actually been observed in healthy humans [45] and in patients with cardiac pacemakers, in whom diaphragmatic contractions were induced *via* the pacemaker [46]. Interestingly, D'Alonzo and Krachman have also shown that poor diaphragm biomechanics may lead to decreased cardiac output [47].

Beneficial haemodynamic effects of diaphragm contractions induced by phrenic pacing during quiet breathing have been described by Roos *et al.* [46]. A recent study conducted by Aliverti *et al.* [45, 48] demonstrated that the modulation of the splanchnic vascular bed as a result of an increase in intra-abdominal pressure *via* diaphragmatic contraction contribute to inferior vena cava blood return, that is, during inspiration, splanchnic venous return is favoured, whereas, during expiration, a venous return of femoral blood flow below the entry of the hepatic vein is preferred. The consequence is that abdominal breathing promotes an extra blood volume mobilisation (from splanchnic circulation) increasing net venous return to inferior vena cava, that is, diaphragmatic inspiration implies that inferior vena cava venous return is facilitated primarily by the central translocation of blood from the vessels of the abdomen and is not the result of facilitation of venous return from the lower limbs [49]. Thus, a greater diaphragm contraction contributed to abdominal circulatory pump results in haemodynamic benefits secondary to net increase in blood venous return and, through Frank-Starling mechanism, greater cardiac stroke volume [45, 48]. It should be also mentioned that during inhalation, the inferior vena cava diameter decreases [50] and the efficiency of venous drainage reaches its climax in slow and deep respiration [51].

The breathing cycle also reflects the balance between the parasympathetic and sympathetic divisions of the autonomic nervous systems with fluctuations in heart rate variability (HRV) being associated with improved oxygen uptake. Each time during inhalation and exhalation, au-

tonomic status swings from parasympathetic to sympathetic (inhalation) and from sympathetic to parasympathetic (exhalation). The increase in heart rate is indicative of a net increase in sympathetic emphasis during inhalation and the decrease in heart rate is suggestive of a net increase in parasympathetic emphasis during exhalation [52]. Diaphragmatic breathing reduce sympathetic activity by enhancing central inhibitory rhythm [53]. Due to increased tidal volume during deep diaphragmatic breathing, there is the activation of the Hering-Breuer reflex, which reduces the chemoreflex sensitivity and might enhance the baroreflex and reduce the sympathetic activity [54].

### Lymphatic function

The diaphragm has a lymphatic drainage system, which is particularly effective for rapid absorption from the peritoneal cavity and returning it to the vascular system. Several studies aimed at elucidating the pathways of peritoneal fluid reabsorption indicate that the peritoneal surface of the diaphragm is an important site of lymphatic drainage. It is clearly shown by morphological studies that the mesothelial cells covering the peritoneal surface of the diaphragm rest on a connective tissue matrix layer, within which lies a rich plexus of lymphatic vessels [55]. These well-developed structures of the diaphragmatic lymphatics comprise of two layers, *i.e.* the submesothelial network and the deeper network of lymphatics which connect with each other by the side branches. They both are found in the muscular portion, while there is only a single layer lymphatic network in the tendinous portion of the diaphragm. The lymphatic network, however, is denser in the tendinous portion than that in the muscular portion. Further, *via* the peritoneal stomata, the lymph of the peritoneal cavity flows into the subperitoneal channels, and then, by the regulation of lymphatic drainage units, into the lymphatic lacunae. The lymphatic lacunae occur only in the muscular portion of the human diaphragm. They are broad, enlarged and blind-ended terminal lymphatic vessels. The right half of the diaphragm has more lacunae than the left one. Finally, lymph passes through the lymphatic plexus under the diaphragmatic pleura to the thoracic duct and right lymphatic duct. In the lymph drainage pathway, the back flow of the lymph fluid from the lymphatic lacunae into the peritoneal cavity is prevented by a few valve-like cytoplasmic processes from the mesothelial cells

and endothelial cells, and numerous filamentous processes from the connective tissue, as well as by the overlapping of endothelial cells in the lymphatic lacunae [56].

The diaphragm is a lymphatic pump, since about 60% of all lymph nodes in the human body are located just under the diaphragm. Shields, in his study '*Lymph, lymph glands, and homeostasis*' reported that diaphragmatic breathing stimulates the cleansing of the lymph nodes by creating a negative pressure pulling the lymph through the lymphatic system [57]. Lymphatic absorption firstly depends on the rhythmicity and stretching of the diaphragm, then on intraperitoneal pressure and the posture of the individual [55]. These concepts are important because they exemplify how incorrect functionality of the diaphragm can negatively affect the lymphatic system. Further, it is important to remember the cisterna chyli, which is located under the diaphragmatic crural region and is the main destination point for the lymph.

### The role of diaphragm in emesis, anti-reflux barrier and swallowing

De Troyer *et al.* [14] showed that whilst the costal diaphragm expands the lower rib cage, the crural diaphragm does not change the dimensions of the rib cage appreciably. According to this, the crural diaphragm has probably an unimportant respiratory role, but is greatly involved in gastroesophageal functions, such as swallowing, vomiting, and contributing to the gastroesophageal reflux barrier.

The physiological process of emesis is complex. In the retching phase, the diaphragm contracts strongly as a single muscle along with the abdominal muscles, increasing the gastric pressure. However, the gastric contents cannot readily traverse the diaphragm because of the simultaneous increase in pressure of the oesophagogastric junction due to the crural contraction. At the next stage of the vomiting cycle, the crural and costal diaphragm dissociate their activities, with the crural diaphragm relaxing to allow the ejection of the gastric contents and the costal diaphragm contracting to increase the abdominal pressure and thus force the gastric contents outwards [58].

Divergence of the activity of the crural and costal diaphragm is also seen during swallowing and esophageal distension. The precise mechanism that mediates the reflex inhibition of the crural diaphragm during oesophageal distension is still poorly understood and unclear. Oyer *et al.* [59]

found that oesophageal distension produced a complete inhibition of the crural diaphragm electromyogram, while at the same time there was only a partial inhibition of the efferent discharge in the crural branch of the phrenic nerve.

Descent of the costal diaphragm creates a thoraco-abdominal pressure gradient, which favours acid reflux. Crural muscle respiratory rhythm gripping the oesophagus and opposing the action of the costal diaphragm can potentially rhythmically squeeze acid from the stomach to oesophagus. It was confirmed by Mittal *et al.* [60] who reported that selective crural myotomy created a state of frequent gastroesophageal reflux.

### Conclusions

The mammalian diaphragm has traditionally been studied as a respiratory muscle. In scientific world there is a general agreement that diaphragm muscle is an essential for breathing and its respiratory function is undeniable. However, the diaphragm has a multiple, non-ventilatory functions affecting the whole body. First of all, the respiratory and cardiovascular systems share similar control mechanisms, and alterations in one system will modify the functioning of the other. Diaphragm function is associated with regulation of many of cardiovascular parameters, *i.e.* cardiac output, stroke volume or venous drainage. The previous study showed that regular practice of diaphragmatic breathing significantly improves heart rate variability (HRV) with a favourable prognostic picture in ischaemic heart disease patients. The diaphragmatic lymphatic system plays also a major role in draining fluids from the peritoneal cavity. Many authors reported that the diaphragm is involved in the control of postural stability during sudden voluntary movement of the limbs. From the point of view of embryology, the diaphragm is a source and exchange point of information. This lead to conclusion that the diaphragm muscle should not be seen as a segment or barrier between two cavities, but as a part of a whole body system. Therefore its uncompromised functionality constitutes broad and relevant issue for a number of fields of medicine, including pulmonology, cardiology, chest surgery, orthopaedics as well as rehabilitation.

### Conflict of interest

The authors declare no conflict of interest.

### References:

- Sewall H, Pollard ME. On the Relations of Diaphragmatic and Costal Respiration, with particular reference to Phonation. *J Physiol.* 1890; 11(3): 159–178, indexed in Pubmed: [16991922](#).
- Bell C. *The Anatomy and Physiology of the Human Body.* 6th ed. Longman, Rees, London 1826.
- Bordoni B, Zanier E. Anatomic connections of the diaphragm: influence of respiration on the body system. *J Multidiscip Healthc.* 2013; 6: 281–291, doi: [10.2147/JMDH.S45443](#), indexed in Pubmed: [23940419](#).
- Stone C. *Science in the art of osteopathy. Osteopathic principles and practice.* Stanley Thornes Ltd, Cheltenham 1999.
- Still AT. *Philosophy of osteopathy.* Mo: A.T. Still, Kirksville 1899.
- Downey R. Anatomy of the normal diaphragm. *Thorac Surg Clin.* 2011; 21(2): 273–279, doi: [10.1016/j.ihorsurg.2011.01.001](#), indexed in Pubmed: [21477776](#).
- Snell RS. *Clinical anatomy by regions.* PA: Lippincott Williams & Wilkins, Philadelphia 2008.
- Moore KL, Dalley AF. *Clinically oriented anatomy.* 5th ed. PA: Lippincott Williams and Wilkins, Philadelphia, 2006.
- Deviri E, Nathan H, Luchansky E. Medial and lateral arcuate ligaments of the diaphragm: attachment to the transverse process. *Anat Anz.* 1988; 166(1-5): 63–67, indexed in Pubmed: [3189849](#).
- Silverman PM, Cooper C, Zeman RK. Lateral arcuate ligaments of the diaphragm: anatomic variations at abdominal CT. *Radiology.* 1992; 185(1): 105–108, doi: [10.1148/radiology.185.1.1523290](#), indexed in Pubmed: [1523290](#).
- Perry SF, Similowski T, Klein W, et al. The evolutionary origin of the mammalian diaphragm. *Respir Physiol Neurobiol.* 2010; 171(1): 1–16, doi: [10.1016/j.resp.2010.01.004](#), indexed in Pubmed: [20080210](#).
- Merrell AJ, Kardon G. Development of the diaphragm -- a skeletal muscle essential for mammalian respiration. *FEBS J.* 2013; 280(17): 4026–4035, doi: [10.1111/febs.12274](#), indexed in Pubmed: [23586979](#).
- Pickering M, Jones JFX. The diaphragm: two physiological muscles in one. *J Anat.* 2002; 201(4): 305–312, indexed in Pubmed: [12430954](#).
- De Troyer A, Sampson M, Sigrist S, et al. Action of costal and crural parts of the diaphragm on the rib cage in dog. *J Appl Physiol Respir Environ Exerc Physiol.* 1982; 53(1): 30–39, indexed in Pubmed: [7118646](#).
- Fell SC. Surgical anatomy of the diaphragm and the phrenic nerve. *Chest Surg Clin N Am.* 1998; 8(2): 281–294, indexed in Pubmed: [9619305](#).
- Ingelmo PM, Bucciero M, Somaini M, et al. Intraperitoneal nebulization of ropivacaine for pain control after laparoscopic cholecystectomy: a double-blind, randomized, placebo-controlled trial. *Br J Anaesth.* 2013; 110(5): 800–806, doi: [10.1093/bja/aes495](#), indexed in Pubmed: [23293276](#).
- King M, Bewes PC, Cairns J, Thornton J. *Primary surgery: non-trauma.* Vol 1. Oxford University Press, Oxford 1990.
- Russell RC. Spleen. In: Mann CV. ed. *Bailey and Loves' short practice of surgery.* Chapman & Hall, London 1992.
- Sutton CD, Marshall LJ, White SA, et al. Kehr's sign - a rare cause: spontaneous phrenic artery rupture. *ANZ J Surg.* 2002; 72(12): 913–914, indexed in Pubmed: [12523356](#).
- Goodman CG, Snyder TK. *Differential Diagnosis for Physical Therapists,* 5th Ed. Elsevier. 2013.
- Söyüncü S, Bektaş F, Cete Y. Traditional Kehr's sign: Left shoulder pain related to splenic abscess. *Ulus Travma Acil Cerrahi Derg.* 2012; 18(1): 87–88, indexed in Pubmed: [22290058](#).
- Goldman MD, Mead J. Mechanical interaction between the diaphragm and rib cage. *J Appl Physiol.* 1973; 35(2): 197–204, indexed in Pubmed: [4723027](#).
- Mead J. Functional significance of the area of apposition of diaphragm to rib cage [proceedings]. *Am Rev Respir Dis.* 1979; 119(2 Pt 2): 31–32, doi: [10.1164/arrd.1979.119.2P2.31](#), indexed in Pubmed: [426349](#).
- De Troyer A, Estenne M. Functional anatomy of the respiratory muscles. *Clin Chest Med.* 1988; 9(2): 175–193, indexed in Pubmed: [3292122](#).



25. Lando Y, Boiselle PM, Shade D, et al. Effect of lung volume reduction surgery on diaphragm length in severe chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. 1999; 159(3): 796–805, doi: [10.1164/ajrccm.159.3.9804055](https://doi.org/10.1164/ajrccm.159.3.9804055), indexed in Pubmed: [10051253](https://pubmed.ncbi.nlm.nih.gov/10051253/).
26. Boyle KL, Olinick J, Lewis C. The value of blowing up a balloon. *N Am J Sports Phys Ther*. 2010; 5(3): 179–188, indexed in Pubmed: [21589673](https://pubmed.ncbi.nlm.nih.gov/21589673/).
27. Hodges PW, Eriksson AE, Shirley D, et al. Intra-abdominal pressure increases stiffness of the lumbar spine. *J Biomech*. 2005; 38(9): 1873–1880, doi: [10.1016/j.jbiomech.2004.08.016](https://doi.org/10.1016/j.jbiomech.2004.08.016), indexed in Pubmed: [16023475](https://pubmed.ncbi.nlm.nih.gov/16023475/).
28. Hodges PW, Butler JE, McKenzie DK, et al. Contraction of the human diaphragm during rapid postural adjustments. *J Physiol*. 1997; 505 (Pt 2): 539–548, indexed in Pubmed: [9423192](https://pubmed.ncbi.nlm.nih.gov/9423192/).
29. Hodges PW, Gandevia SC. Changes in intra-abdominal pressure during postural and respiratory activation of the human diaphragm. *J Appl Physiol* (1985). 2000; 89(3): 967–976, indexed in Pubmed: [10956340](https://pubmed.ncbi.nlm.nih.gov/10956340/).
30. Frank C, Kobesova A, Kolar P. Dynamic neuromuscular stabilization & sports rehabilitation. *Int J Sports Phys Ther*. 2013; 8(1): 62–73, indexed in Pubmed: [23439921](https://pubmed.ncbi.nlm.nih.gov/23439921/).
31. Skladal J, Skarvan K, Ruth C, et al. propos de l'activite posturale du diaphragme chez l'Homme. *Journale de Physiologie*. 1969; 2: 405–406.
32. Hemborg B, Moritz U, Löwing H. Intra-abdominal pressure and trunk muscle activity during lifting. IV. The causal factors of the intra-abdominal pressure rise. *Scand J Rehabil Med*. 1985; 17(1): 25–38, indexed in Pubmed: [3159082](https://pubmed.ncbi.nlm.nih.gov/3159082/).
33. Hodges PW, Gandevia SC. Activation of the human diaphragm during a repetitive postural task. *J Physiol*. 2000; 522 Pt 1: 165–175, indexed in Pubmed: [10618161](https://pubmed.ncbi.nlm.nih.gov/10618161/).
34. Kolar P, Neuwirth J, Sanda J, et al. Analysis of diaphragm movement during tidal breathing and during its activation while breath holding using MRI synchronized with spirometry. *Physiol Res*. 2009; 58(3): 383–392, indexed in Pubmed: [18637703](https://pubmed.ncbi.nlm.nih.gov/18637703/).
35. Kolar P, Sulc J, Kyncl M, et al. Stabilizing function of the diaphragm: dynamic MRI and synchronized spirometric assessment. *J Appl Physiol* (1985). 2010; 109(4): 1064–1071, doi: [10.1152/jappphysiol.01216.2009](https://doi.org/10.1152/jappphysiol.01216.2009), indexed in Pubmed: [20705944](https://pubmed.ncbi.nlm.nih.gov/20705944/).
36. Hodges PW, Heijnen I, Gandevia SC. Postural activity of the diaphragm is reduced in humans when respiratory demand increases. *J Physiol*. 2001; 537(Pt 3): 999–1008, indexed in Pubmed: [11744772](https://pubmed.ncbi.nlm.nih.gov/11744772/).
37. Janssens L, Brumagne S, Polspoel K, et al. The effect of inspiratory muscles fatigue on postural control in people with and without recurrent low back pain. *Spine (Phila Pa 1976)*. 2010; 35(10): 1088–1094, doi: [10.1097/BRS.0b013e3181bee5c3](https://doi.org/10.1097/BRS.0b013e3181bee5c3), indexed in Pubmed: [20393397](https://pubmed.ncbi.nlm.nih.gov/20393397/).
38. Vostatek P, Novák D, Rychnovský T, et al. Diaphragm postural function analysis using magnetic resonance imaging. *PLoS One*. 2013; 8(3): e56724, doi: [10.1371/journal.pone.0056724](https://doi.org/10.1371/journal.pone.0056724), indexed in Pubmed: [23516397](https://pubmed.ncbi.nlm.nih.gov/23516397/).
39. Janssens L, Brumagne S, McConnell AK, et al. Greater diaphragm fatigability in individuals with recurrent low back pain. *Respir Physiol Neurobiol*. 2013; 188(2): 119–123, doi: [10.1016/j.resp.2013.05.028](https://doi.org/10.1016/j.resp.2013.05.028), indexed in Pubmed: [23727158](https://pubmed.ncbi.nlm.nih.gov/23727158/).
40. DeBurgh DM. Interactions between respiration and circulation. In: Cherniack NS, Widdicombe JG. ed. *Handbook of Physiology, Section 3: The Respiratory System, Vol II: Control of Breathing, Part 2*. American Physiological Society, Bethesda 1986: 529–594.
41. Kulur AB, Haleagrahara N, Adhikary P, et al. Effect of diaphragmatic breathing on heart rate variability in ischemic heart disease with diabetes. *Arq Bras Cardiol*. 2009; 92(6): 423–463, indexed in Pubmed: [19629309](https://pubmed.ncbi.nlm.nih.gov/19629309/).
42. Lee JS, Lee MS, Lee JY, et al. Effects of diaphragmatic breathing on ambulatory blood pressure and heart rate. *Biomed Pharmacother*. 2003; 57 Suppl 1: 87s–91s, indexed in Pubmed: [14572682](https://pubmed.ncbi.nlm.nih.gov/14572682/).
43. Bernardi L, Spadacini G, Bellwon J, et al. Effect of breathing rate on oxygen saturation and exercise performance in chronic heart failure. *Lancet*. 1998; 351(9112): 1308–1311, doi: [10.1016/S0140-6736\(97\)10341-5](https://doi.org/10.1016/S0140-6736(97)10341-5), indexed in Pubmed: [9643792](https://pubmed.ncbi.nlm.nih.gov/9643792/).
44. van Dixhoorn J. [Favorable effects of breathing and relaxation instructions in heart rehabilitation: a randomized 5-year follow-up study]. *Ned Tijdschr Geneesk*. 1997; 141(11): 530–534, indexed in Pubmed: [9190510](https://pubmed.ncbi.nlm.nih.gov/9190510/).
45. Aliverti A, Uva B, Laviola M, et al. Concomitant ventilatory and circulatory functions of the diaphragm and abdominal muscles. *J Appl Physiol* (1985). 2010; 109(5): 1432–1440, doi: [10.1152/jappphysiol.00576.2010](https://doi.org/10.1152/jappphysiol.00576.2010), indexed in Pubmed: [20813981](https://pubmed.ncbi.nlm.nih.gov/20813981/).
46. Roos M, Kobza R, Jamshidi P, et al. Improved cardiac performance through pacing-induced diaphragmatic stimulation: a novel electrophysiological approach in heart failure management? *Europace*. 2009; 11(2): 191–199, doi: [10.1093/europace/eun377](https://doi.org/10.1093/europace/eun377), indexed in Pubmed: [19168496](https://pubmed.ncbi.nlm.nih.gov/19168496/).
47. D'Alonzo GE, Krachman SL. *Pulmonology*. In: Ward RC. ed. *Foundations for osteopathic medicine* (2nd ed.). Lippincott Williams & Wilkins, Philadelphia 2003.
48. Aliverti A, Bovio D, Fullin I, et al. The abdominal circulatory pump. *PLoS One*. 2009; 4(5): e5550, doi: [10.1371/journal.pone.0005550](https://doi.org/10.1371/journal.pone.0005550), indexed in Pubmed: [19440240](https://pubmed.ncbi.nlm.nih.gov/19440240/).
49. Miller JD, Pegelow DF, Jacques AJ, et al. Skeletal muscle pump versus respiratory muscle pump: modulation of venous return from the locomotor limb in humans. *J Physiol*. 2005; 563(Pt 3): 925–943, doi: [10.1113/jphysiol.2004.076422](https://doi.org/10.1113/jphysiol.2004.076422), indexed in Pubmed: [15649978](https://pubmed.ncbi.nlm.nih.gov/15649978/).
50. Kimura BJ, Dalugdugan R, Gilcrease GW, et al. The effect of breathing manner on inferior vena caval diameter. *Eur J Echocardiogr*. 2011; 12(2): 120–123, doi: [10.1093/ejechoard/jeq157](https://doi.org/10.1093/ejechoard/jeq157), indexed in Pubmed: [20980326](https://pubmed.ncbi.nlm.nih.gov/20980326/).
51. Byeon K, Choi JO, Yang JH, et al. The response of the vena cava to abdominal breathing. *J Altern Complement Med*. 2012; 18(2): 153–157, doi: [10.1089/acm.2010.0656](https://doi.org/10.1089/acm.2010.0656), indexed in Pubmed: [22339104](https://pubmed.ncbi.nlm.nih.gov/22339104/).
52. Stauss H. Heart rate variability. *American Journal of Physiology - Regulatory, Integrative and Comparative Physiology*. 2003; 285(5): R927–R931, doi: [10.1152/ajpregu.00452.2003](https://doi.org/10.1152/ajpregu.00452.2003).
53. Montano N, Cogliati C, Porta A, et al. Central vagotonic effects of atropine modulate spectral oscillations of sympathetic nerve activity. *Circulation*. 1998; 98(14): 1394–1399, indexed in Pubmed: [9760293](https://pubmed.ncbi.nlm.nih.gov/9760293/).
54. Bernardi L, Gabutti A, Porta C, et al. Slow breathing reduces chemoreflex response to hypoxia and hypercapnia, and increases baroreflex sensitivity. *J Hypertens*. 2001; 19(12): 2221–2229, indexed in Pubmed: [11725167](https://pubmed.ncbi.nlm.nih.gov/11725167/).
55. Abu-Hijleh MF, Habbal OA, Moqattash ST. The role of the diaphragm in lymphatic absorption from the peritoneal cavity. *J Anat*. 1995; 186 ( Pt 3): 453–467, indexed in Pubmed: [7559120](https://pubmed.ncbi.nlm.nih.gov/7559120/).
56. Li J, Zhao Z, Zhou J, et al. A study of the three-dimensional organization of the human diaphragmatic lymphatic lacunae and lymphatic drainage units. *Ann Anat*. 1996; 178(6): 537–544, doi: [10.1016/S0940-9602\(96\)80113-0](https://doi.org/10.1016/S0940-9602(96)80113-0), indexed in Pubmed: [9010570](https://pubmed.ncbi.nlm.nih.gov/9010570/).
57. Shields JW. Lymph, lymph glands, and homeostasis. *Lymphology*. 1992; 25(4): 147–153, indexed in Pubmed: [1293429](https://pubmed.ncbi.nlm.nih.gov/1293429/).
58. Miller AD. Respiratory muscle control during vomiting. *Can J Physiol Pharmacol*. 1990; 68: 237–241.
59. Oyer LM, Knuth SL, Ward DK, et al. Reflex inhibition of crural diaphragmatic activity by esophageal distention in cats. *Respir Physiol*. 1989; 77(2): 195–202, indexed in Pubmed: [2781162](https://pubmed.ncbi.nlm.nih.gov/2781162/).
60. Mittal RK, Sivri B, Schirmer BD, et al. Effect of crural myotomy on the incidence and mechanism of gastroesophageal reflux in cats. *Gastroenterology*. 1993; 105(3): 740–747, indexed in Pubmed: [8359645](https://pubmed.ncbi.nlm.nih.gov/8359645/).