

*LITERATURE REVIEW*

## **The Effect of Diaphragm Breathing Exercises on Physiological and Psychological Stress**

**Edwin Goutama<sup>1</sup>, Siti Chandra Widjanantie<sup>2</sup>**

<sup>1</sup> Department of Physical Medicine and Rehabilitation, Universitas Indonesia, Cipto Mangunkusumo General Hospital, Jakarta, Indonesia

<sup>2</sup> Department of Physical Medicine and Rehabilitation, Universitas Indonesia, Persahabatan General Hospital, Jakarta, Indonesia

### **ABSTRACT**

Physiological and psychological stress causes changes in homeostasis characterized by an imbalance of activity between the autonomic nervous systems. Overactivity of the sympathetic nervous system leads to physical, psychological, and behavioral disorders.

Diaphragmatic breathing exercises routinely have been shown to suppress sympathetic nerve activity through the activation of the parasympathetic nervous system and an increase in tidal volume that creates a change in the cell membrane potential to hyperpolarization, with the result being an improvement in physiological and psychological stress conditions, which appear in various parameters. This paper reviews the anatomy, physiology, and biomechanics of the diaphragm muscle, as well as the mechanisms of diaphragmatic breathing exercises in improving physiological and psychological stress conditions from a Physical Medicine and Rehabilitation point of view.

**Keywords:** breathing, diaphragm, exercise, physiological stress, psychological stress

## ABSTRAK

Stres fisiologis dan psikologis menyebabkan perubahan homeostasis yang ditandai kondisi ketidakseimbangan aktivitas sistem saraf otonom. Overaktivitas sistem saraf simpatis memunculkan gangguan fisik, psikis, dan perilaku.

Latihan pernafasan diafragma secara rutin terbukti menekan aktivitas saraf simpatis melalui aktivasi sistem saraf parasimpatis dan peningkatan volume tidal yang menciptakan perubahan membran potensial sel menjadi hiperpolarisasi, dengan hasil akhir berupa perbaikan kondisi stres fisiologis maupun psikologis, yang nampak pada berbagai parameter. Makalah ini meninjau anatomi, fisiologi, dan biomekanika otot diafragma, serta mekanisme latihan pernafasan diafragma dalam memperbaiki kondisi stres fisiologis dan psikologis dari sudut pandang Kedokteran Fisik dan Rehabilitasi.

**Kata kunci:** pernafasan, diafragma, latihan, stres, fisiologis, psikologis

---

Correspondent Detail:

**Edwin Goutama**

Email: edwinikfrfkui@gmail.com Department of Physical Medicine and Rehabilitation, Faculty of Medicine, Universitas Indonesia, Dr. Cipto Mangunkusumo Hospital, Jakarta, Indonesia

## INTRODUCTION

Stress is a global epidemic occurring in all parts of the world. According to the WHO global stress assessment in 2016, more than 350 million people worldwide are exposed to stress.<sup>1</sup> In Indonesia, a welfare score survey by Cigna found that the percentage of Indonesian people's stress levels is around 75%.<sup>2</sup>

Stress is a process in which the demands of the environment are not met, resulting in adverse effects on physical and psychological health.<sup>1</sup> The physiological stress response is triggered

by activation of the sympathetic nervous system through the release of cortisol, causing an increase in respiratory rate, heart rate, and systolic blood pressure. Activation of the sympathetic nervous system also triggers depolarization of cellular membranes, which then becomes a significant risk factor for psychological disorders such as anxiety and depression.<sup>1</sup> One exercise that is known to have a potential effect on stress is diaphragmatic breathing.<sup>3</sup>

Diaphragmatic breathing is a slow, deep breathing technique that optimizes the force of contraction of the diaphragm at a rate of 6 breaths per minute. This technique allows activation of the parasympathetic dominant over the sympathetic, providing a physiological and psychological homeostatic effect.<sup>1</sup> Therefore, in this paper, we will discuss the mechanism of diaphragmatic breathing in improving homeostasis, especially in the cardiorespiratory and limbic systems, to reduce physiological and psychological stress levels.

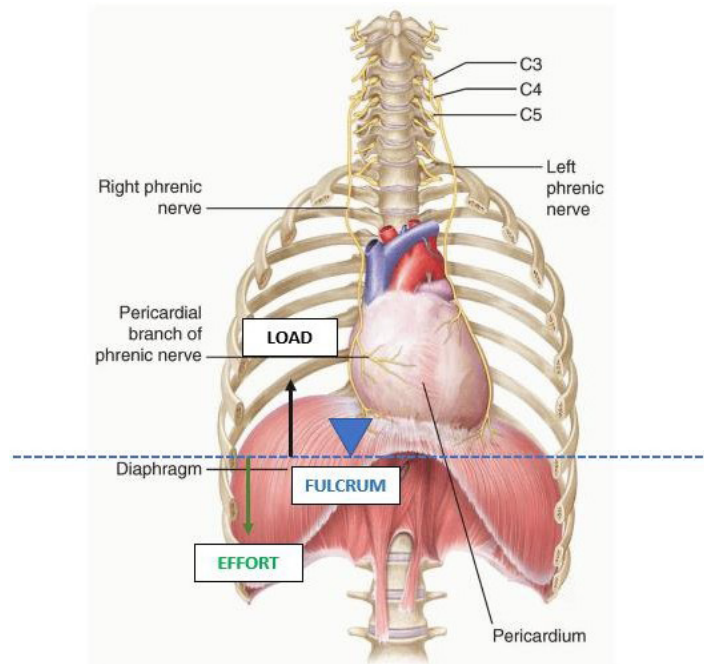
**Anatomy**

The diaphragm is composed of a mixture of type I and type II muscle fibers. The percentage of type I fibers is the highest compared to type II, namely 55% compared to 25% of type IIa and 20% of kind IIb.<sup>4</sup> This composition is very beneficial, considering the continuous work of the diaphragm muscle.<sup>4,5</sup> From a functional perspective, there are two essential areas of the diaphragm: the crural and costal.<sup>6</sup> The area that plays a direct role in the inspiration process is the costal area. In contrast, the costal area is more involved in the contraction-relaxation mechanism of the distal esophagus.<sup>7</sup>

**Biomechanics**

Biomechanically, the diaphragm muscle belongs to the lever arm class II (mechanical

advantage), with the right and left phreno-pericardial ligaments acting as the fulcrum points in each dome.<sup>8,9</sup> In the diaphragmatic contraction stimulation experiment, it was found that the motor unit action potentials (MUAPs) of the medial costal area increased significantly compared to the crural site.<sup>7</sup> The force load is exerted by intra-abdominal pressure when the diaphragm contracts, which causes the roof of the diaphragm to take the shape of a dome.<sup>10</sup> When referring to torque, the rotation of this force load is counterclockwise. Therefore, the force generated by the diaphragm when it contracts will be clockwise.<sup>7</sup> The type II lever arms provide the advantage that the power generated is greater than the other two types of the lever arms.



**Figure 1. The biomechanic lever arm of the diaphragm.**<sup>7,8,9,10</sup>

**The voluntary breathing control mechanism**

Voluntary breathing is an individual’s conscious, modified breathing for a specific purpose, such as when regulating speech, blowing out

candles, or breathing exercises. The voluntary respiratory control center is located in the higher brain, namely the cerebral cortex. Electrical stimulation of the motor cortex in humans shows

that the respiratory muscles receive efferent impulses from the corticospinal pathways. Vice versa, as feedback, the somatosensory cortex receives impulses from afferents of the respiratory muscles. The premotor cortex is said to influence voluntary control of breathing, thereby proving that efferent impulses from the cortical can bypass involuntary respiratory centers in the brainstem to affect the respiratory muscles directly.<sup>11</sup>

**The role of the autonomic nervous system in respiration**

The sympathetic and parasympathetic autonomic nervous systems have two essential roles in the respiratory process: directly to the respiratory organs and indirectly through changes in blood PaO<sub>2</sub> and H<sup>+</sup> homeostasis through peripheral chemoreceptors.<sup>12,13</sup> In homeostasis, the cell membrane is in polarization condition, which means there is a balance between the positive charge inside the cell and the negative charge outside the cell. The decrease in PaO<sub>2</sub> and increased intra-cellular H<sup>+</sup> inhibits the opening of the potassium channel pump and induces an

influx of Ca<sup>2+</sup>. The accumulation of potassium and intracellular Ca<sup>2+</sup>, both positively charged, causes the intracellular membrane to be too positive, which is called depolarization. Depolarization of the aortic cell membrane and carotid bodies causes vagus nerve afferents (aortic bodies) to release the neurotransmitter nor-epinephrine and glossopharyngeal nerves (carotid bodies) to the respiratory center in the medulla, to activate the DRG. Activation of these inspiratory neurons further causes increased pulmonary ventilation to restore homeostasis. In the case where PaO<sub>2</sub> increases with low intracellular H<sup>+</sup>, the opposite is an efflux of K ions followed by an influx of Cl<sup>-</sup>, causing the intracellular charge to be more negative than the resting membrane potential. This state is called hyperpolarization, which then stimulates the release of the neuro-transmitter acetylcholine. Acetylcholine input will be carried by vagus afferents to the Pre-Botzinger complex in the medulla, resulting in a slow and deep breathing pattern, decreasing amygdala neuronal activity through the mechanism of cell membrane hyperpolarization.<sup>12</sup>

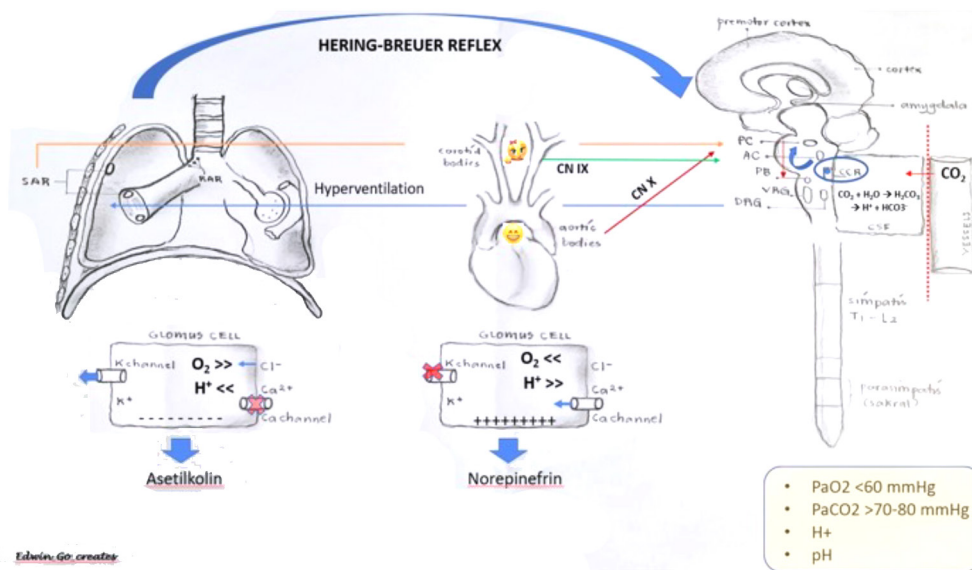


Figure 2. Schematic of the respiratory response to changes in homeostasis.<sup>11,12,13</sup>

## Stress

Psychological stress is a relationship between the individual and his environment that the individual judges as burdening or exceeding his resources and endangering his well-being. The structure in the brain that plays a dominant role in the stress response is the amygdala.<sup>14</sup> Negative moods or moods such as stress, anxiety, and depression cause sympathetic activation due to the depolarization process that extends to all parts of the brain and body.<sup>15</sup> Excitatory activity increases neuronal activity in the amygdala and the hypothalamic-pituitary-adrenal axis (HPA axis), causing the secretion of excitatory neurotransmitters, such as norepinephrine, serotonin, dopamine, and GABA, which increase heart rate, respiratory rate, blood pressure, and muscle tension. In addition, membrane depolarization is essential for skeletal muscle contraction, suggesting that increased muscle tension during stressful conditions is resulted from an extensive membrane depolarization potential.<sup>16</sup>

According to Hans and Seyle, physiological stress is a response to changes to maintain conditions of stability and homeostasis. Hans and Seyle define stress as a response to changes to maintain conditions of equilibrium and homeostasis.<sup>17</sup> Kenneth and Hambly describe a maladaptive condition in which an overactivity of the sympathetic nervous system causes physical, psychological, and behavioral disorders. Hans and Seyle also divide the body's response into three stages of response. The first stage is the "alarm reaction stage," where the body reacts to the stressor with a "fight-or-flight response" and activates the sympathetic nervous system. The second stage is the "resistance stage," where the body begins to adapt to the stressor. During

this phase, the parasympathetic nervous system returns physiological functions to normal, and the body focuses on its resources to fight the stressor. At this stage, an individual's physical appearance appears normal, but blood glucose, cortisol, and adrenaline levels remain elevated. If the stressor continues beyond the body's ability to cope, the individual will run out of resources and become vulnerable to illness and death. This state is called the "exhaustion stage." There are not many biological markers used to define stress. The most common biological marker for the definition of stress-related physiological disorders is cortisol.<sup>17</sup>

Under stress, the HPA axis is activated. The paraventricular nucleus of the hypo-thalamus secretes corticotropin-releasing hormone (CRH), which then stimulates the anterior lobe of the pituitary gland to release adrenocorticotropin hormone (ACTH). ACTH further stimulates the adrenal glands to secrete cortisol, a stress hormone found to be dominant in humans. High cortisol levels activate the glucocorticoid receptor (GR) on cell membranes at the cellular level. Cortisol binding to GR induces cytochrome C in the mitochondria to activate caspase 3, an enzyme that plays a role in cellular apoptosis.<sup>18</sup> It is known that mitochondria are the dominant intracellular organelles in the respiration process. Consumption of oxygen for the production of ATP occurs in the process of oxidative phosphorylation or electron transport chain that takes place in the mitochondria of cells. Therefore, stress-induced mitochondrial dysfunction will cause a significant disruption of homeostasis. The high cortisol level in the mitochondrial cell membrane, in addition to triggering cell apoptosis, also stimulates the opening of calcium channels, allowing  $Ca^{2+}$

ions to enter intracellularly (influx). Closure of potassium channels, causing K<sup>+</sup> ions to remain intracellular. This condition then upsets the equilibrium of the membrane potential, wherein the intracellular charge is too positive for depolarization. In addition, cortisol also causes oxidative stress by modifying endogenous antioxidant activity. High cortisol levels decrease blood glutathione (GSH) and superoxide dismutase (SOD) erythrocyte cells. Other enzymes involved in glucocorticoid-induced oxidative stress include NADPH oxidase, xanthine oxidase, and nitric oxide.<sup>19</sup>

### Diaphragmatic breathing exercises

Diaphragmatic breathing is slow, deep breathing through the nose using the force of contraction of the diaphragm with minimal chest movement in the supine position. Diaphragmatic breathing is typically performed with inspiration and an expiration of 5 seconds each. During normal breathing, the diaphragm contracts and drops 1 cm on inspiration. When breathing deeply, the

diaphragm can drop up to 10 cm. Seventy-five percent of the enlargement of the chest cavity during inspiration is caused by contraction of the diaphragm.<sup>20</sup> Diaphragmatic breathing at a rate of 6 breaths per minute decreases the chemoreflex response to hypoxia and hypercapnia compared to normal breathing of 15 breaths per minute. Slow breathing increases lung capacity and facilitates better oxygen exchange in the alveoli.<sup>20</sup> In addition, contraction of the diaphragm provides a compressive effect on the visceral organs, increasing the volume and flow of the portal venous system back to the right heart. Diaphragmatic breathing exercises increase tidal volume, followed by an increase in intravascular PaO<sub>2</sub>. Under sufficient O<sub>2</sub>, peripheral chemoreceptor glomus cells tend to hyperpolarize the cell membrane. The parasympathetic nervous system becomes dominant over the sympathetic, inhibiting activation of the HPA axis, resulting in a decrease in cortisol levels, blood pressure, heart rate, and respiratory rate.<sup>20</sup>

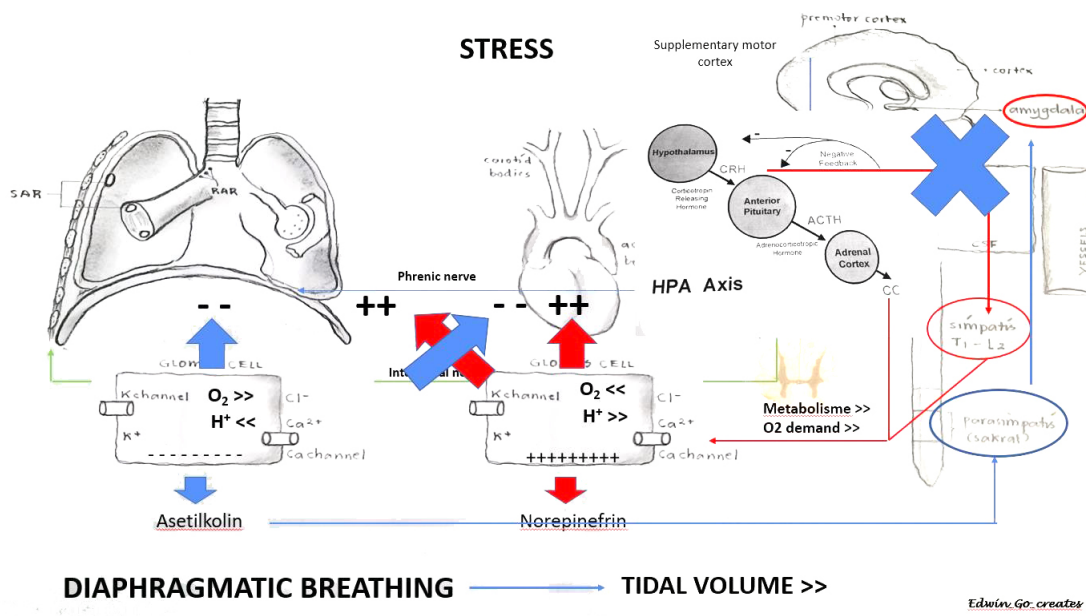


Figure 3. Physiology of diaphragmatic breathing exercises in reducing stress.<sup>17,20</sup>

**Diaphragm breathing technique**

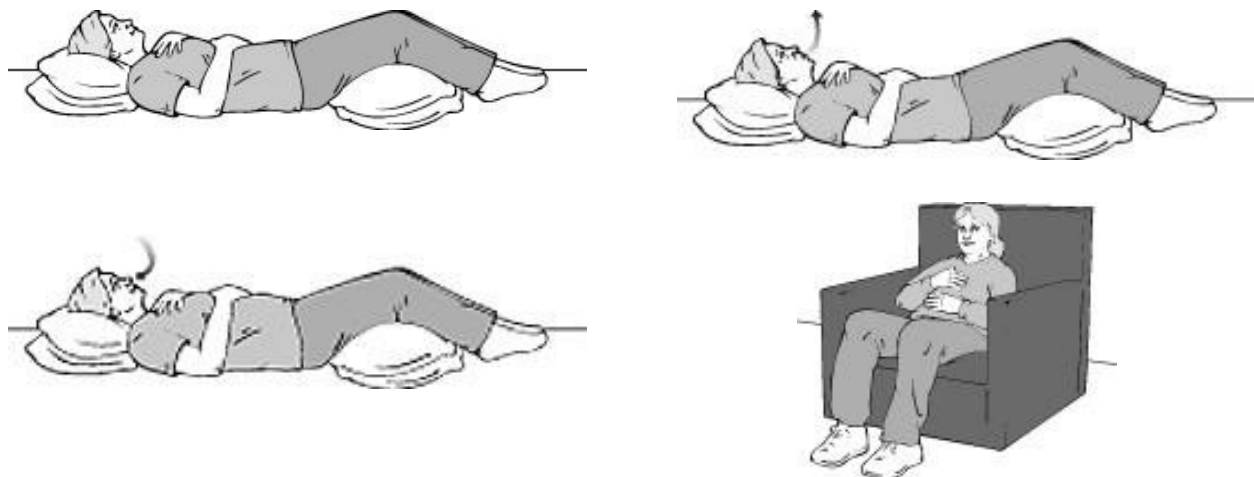
Diaphragmatic breathing is intended to help someone use the diaphragm muscle correctly and adequately when breathing with the aim of:<sup>21</sup>

- a. Strengthening the diaphragm muscle itself
- b. Reducing work of breathing by slowing the rate of breathing
- c. Reducing oxygen demand
- d. Using less energy and effort in breathing

The diaphragmatic breathing technique is as follows:<sup>21</sup>

- 1. Supine position with a flat base on the mat or mattress. The knees are bent with a pillow underneath, and the head is propped with a pillow. Place one hand on the chest and the other hand on the abdomen just below the rib cage. This maneuver is intended to be able to feel the movement of the diaphragm when breathing.
- 2. Breathe slowly through the nose until the stomach expands so that the hands on the abdomen are raised. Hands placed on the chest as much as possible do not come up.

- 3. Then tighten the abdominal muscles, and let the stomach back down along with the exhalation through pursed lips. The hand on the abdominal side will come back down, and the hand on the chest will remain still.
- 4. In the early stages, to facilitate the exercise process, it can be done in a supine position, as in the instructions above. But along with intense routines and exercises, diaphragmatic breathing can also be done in a sitting position in a chair. Diaphragmatic breathing technique while sitting on a chair is not much different from lying on your back. Both knees are bent by sitting in a comfortable chair and position the head, neck, and shoulders in a relaxed state. After that, the breathing technique is the same as described above.
- 5. Diaphragmatic breathing exercises are carried out in the early stages for 5-10 minutes per session, with a frequency of 3-4 times daily. The intensity of the exercise can be increased by adding weights such as books to the abdomen.



**Figure 4. Diaphragmatic breathing exercise positions**

A study by Vasuki and Sweety on individuals aged 30-50 years with pre-hypertension and hypertension found that diaphragmatic breathing exercises six times per minute twice a day with a duration of 10 minutes for 12 weeks resulted in a decrease in systolic, diastolic, and blood pressure. Significant mean arterial pressure (MAP) compared to the control group without intervention.<sup>22</sup> Yu-Fen Chen et al. involved 30 people with chronic obstructive pulmonary disease (COPD) who had periods of anxiety in the last month, with a Beck Anxiety Inventory (BAI) score above 8. The intervention group performed diaphragmatic breathing exercises under the supervision of a senior psychiatrist with a frequency of 2 times a day, every ten times, as much as 12 times in 8 weeks. BAI assessment at the end of week 8 (end of session) showed a significant decrease in BAI score ( $p < 0.05$ ) in the intervention group when compared to the control group without intervention. This study indicates that diaphragmatic breathing exercises in COPD patients with high anxiety levels (BAI score  $> 8$ ) significantly reduce anxiety levels or psychological stress.<sup>23</sup> Research by Perciavalle et al. in 2016 on 38 healthy subjects aged 18-28 years provided an intervention for diaphragmatic breathing exercises. It assessed mood disorders, salivary cortisol levels, and heart rate. The exercise protocol was administered once a week for ten sessions. Outcome measurements were carried out at the beginning of the study (phase I), the 5th session (phase II), and the last session (phase III). There was a significant improvement in the intervention group between phase I and phase III, as well as in the outcomes of heart rate. On the output of salivary cortisol levels, better results were obtained, namely a decrease in cortisol levels between phases I and III and between phases II and III.<sup>24</sup>

## CONCLUSION

Diaphragmatic breathing exercises six times per minute have been shown to reduce psychological and physiological stress through direct changes in membrane potential charge on the cardiorespiratory and limbic systems. The increase in  $\text{PaO}_2$  is accompanied by a decrease in intracellular  $\text{H}^+$ , causing hyperpolarization, which then triggers the release of acetylcholine and activation of the parasympathetic nervous system, resulting in improvements in various parameters such as systolic blood pressure, MAP, cortisol, DASS and BAI scores.

## REFERENCES

1. Hopper SI, Murray SL, Ferrara LR, Singleton JK, Orga-TIL. Effectiveness of diaphragmatic breathing on quantitative systematic review protocol. 2018;1367–72.
2. Andriani D. Tingkat stres orang Indonesia terendah selama pandemi. *Bisnis.com*. 2020;
3. Russo M, Santarelli D. The physiological effects of slow breathing in the healthy human. 13(4):298–309.
4. Polla B, D'Antona G, Bottinelli R, Reggiani C. Respiratory muscle fibres: Specialisation and plasticity. *Thorax*. 2004;59(9):808–17.
5. Fogarty MJ, Mantilla CB, Sieck GC. Breathing: Motor control of diaphragm muscle. *Physiology*. 2018;33(2):113–26.
6. Finucane KE, Singh B. Human diaphragm efficiency estimated as power output relative to activation increases with hypercapnic hyperpnea. *J Appl Physiol*. 2009;107(5):1397–405.



7. Shafik A, Shafik I, El-Sibai O, Mostafa RM. Does the crural diaphragm share in the contractile activity of the costal diaphragm? The concept of an “autonomous esophageal crus” and its role in esophageal competence. *Med Sci Monit.* 2004;10(8).
8. Bordoni B, Zanier E. Anatomic connections of the diaphragm: Influence of respiration on the body system. *J Multidiscip Healthc.* 2013;6:281–91.
9. Paoletti S. *The Fasciae: Anatomy, Dysfunction and Treatment.* In: 1st ed. Seattle: Eastland Press; 2006.
10. Massery M. Multisystem consequences of impaired breathing mechanics and/or postural control. *Cardiovasc Pulm Phys Ther Evid Pract.* 2006;695–717.
11. Macefield BYG, Gandeviat SC. 5 . During quiet breathing , in which subjects were relaxed and distracted from. 1991;545–58.
12. Widdicombe JG. *The Autonomic Nervous.* 2015;
13. Cahyono ID, Sasongko H, Primatika AD. Neurotransmitter Dalam Fisiologi Saraf Otonom. 2009;I:42–55.
14. Best B. The Amygdala and The Emotions. In: *Anatomy of the mind.* 2009. p. 223–7.
15. Murik S. Polarization theory of motivations, emotions, and attention. *Bull Eastern-Siberian Sci Cent SB RAMS.* 2005;167–74.
16. Ma X, Yue Z, Gong Z, Zhang H, Duan N, Shi Y. The Effect of Diaphragmatic Breathing on Attention , Negative Affect and Stress in Healthy Adults. 2017;8(June):1–12.
17. Kim H, Cheon E, Bai D, Lee YH, Koo B. Stress and Heart Rate Variability : A Meta-Analysis and Review of the Literature. 2018;
18. Chirichigno JW, Manfredi G, Beal MF, Albers DS. Stress-induced mitochondrial depolarization and oxidative damage in PSP cybrids. 2002;951:31–5.
19. Martarelli D, Cocchioni M, Scuri S, Pompei P. Diaphragmatic Breathing Reduces Exercise-Induced Oxidative Stress. 2011;2011.
20. Bernardi L, Gabutti A, Porta C, Spicuzza L. Slow breathing reduces chemore<sup>-</sup> ex response to hypoxia and hypercapnia , and increases barore<sup>-</sup> ex sensitivity. 2:2221–9.
21. Goldfried, Davison. Diaphragmatic Breathing. In: *Clinical Behavior Therapy*
22. Vasuki G, Sweet LM. The study of usefulness of deep breathing exercise on blood pressure in pre- hypertensive and hypertensive patients. 2017;4(3):400–3.
23. Chen Y, Huang X, Chien C. The Effectiveness of Diaphragmatic Breathing Relaxation. 2016;0:1–8.
24. Perciavalle V, Blandini M, Fecarotta P, Buscemi A, Di D, Luana C, et al. The role of deep breathing on stress. 2016;