v. 4, n. 93, 2024

International Journal of Health Science

Acceptance date: 24/10/2024

ASSESSMENT OF DIAPHRAGMATIC THICKNESS USING ULTRASOUND AND IMPLEMENTATION OF A DIAPHRAGM REHABILITATION STRATEGY IN A MECHANICALLY VENTILATED PATIENT: CASE REPORT[1]

Natan Macedo dos Santos

Marcelo Rocha S. Silva

Caroline da Silva Lima

Wesley Luís da Silva Cunha

Jusley da Silva Gil

Jimmy Lennon S. Brigda

George Santos Simões

Jacyara de Oliveira Vanini

Bruno Pereira Bandeira

Eric Lennon S. Silva

Amanda Voigt dos Santos

Jullyane dos Santos Skruk

1. Cardiopulmonary and Metabolic Rehabilitation Research Group/ Intensive Care Unit, Hospital Arquidiocesano Cônsul carlos Renaux, Brusque- SC, Brazil

Amanda Lourin M. Pereira

Lucas Couto Santos

INTRODUCTION

The diaphragm muscle is considered to be the main muscle involved in breathing. It is characterized by its dome shape, a thin muscular lamina, and its regions of origin are the sternal portion, the posterior face of the xiphoid process, the costal portion, which is the internal region of the costal arches, forming the zone of apposition to the rib cage, and the lumbar portion, which comes from the lumbar vertebrae (L2-L4). It is innervated by the phrenic nerves originating from the C3, C4 and C5 segments. The muscle is made up of approximately 55% type 1 fibres and 45% type 2 fibres, providing greater resistance to fatigue, with type 1 fibres being characterized as slow twitch. When the diaphragm contracts during breathing, it moves in a cephalo-caudal direction by approximately 1 cm to 2 cm, increasing the size of the chest cavity and favoring the movement of air into the lungs. [1,2]

Diaphragmatic dysfunction in intensive care units is correlated with prolonged periods of mechanical ventilation, reducing the muscle's ability to generate motor force and reducing its resistance to fatigue[16] . In the vast majority of cases, this weakness is associated with prolonged ventilator weaning, increasing the length of stay in the ICU and consequently exposing the patient to a greater risk of infections. .[3]

The use of bedside ultrasound to diagnose this dysfunction has proven to be a viable, fast, non-invasive method without exposure to ionizing radiation, as is the case with other methods. Among the techniques used to assess the diaphragm, we have the measurement of the thickness of the muscle, in the apposition zone, between the eighth and ninth intercostal space, being a hypoechoic structure (muscle mass) between two hyperechoic lines, the more superficial pleural line and the deeper peritoneal line .[3,4,5]

In order to strengthen the diaphragm muscle and reverse this dysfunction, it is necessary to implement a muscle training protocol based on three principles: overload, where loads will be imposed on the muscle beyond the limit to which it is accustomed, gradually seeking an increase and adaptation of this imposed load; the principle of specificity, where the effects of training will be directed at a certain type of muscle fiber, adjusting the training to the goal to be achieved, and finally reversibility, which aims to maintain and continue the process, so that the changes remain, if there is no stimulus the gains are reduced and can return to the initial stage [15,17].

This study aims to present a case report of a patient on mechanical ventilation and the change in diaphragmatic thickness during hospitalization. To highlight the variation in thickness and respiratory effort with different ventilation modes and settings and the variation in Tdi after a training protocol.

CASE REPORT

Female patient, 60 years old, 165cm, 65kg, BMI 23.8, eutrophic, admitted to the intensive care unit due to traumatic brain injury following a fall from a height. Hemodynamically stable, with an endotracheal tube, on mechanical ventilation, with no signs of ventilatory drive, under analgesia (Fentanyl and Midazolan, dose 2mcg/kg), RASS scale - 4, without the use of vasoactive drugs. The imaging exams described: "areas of subarachnoid hemorrhage in the left and right frontoparietotemporal regions, extensive subdural hemorrhage in the left and right frontoparietotemporal regions and areas of parenchymal contusions in the right temporal regions", reports issued by a neurologist and a radiologist.

To assess expiratory diaphragmatic thickness (Tdi-exp), the patient was placed in the supine position, head at 30-45°. The device used was the Mindray M7, a high-frequency linear transducer (7-13MHz), measured between the eighth and ninth intercostal spaces, between the anterior axillary line and the mid-axillary line, over the Apposition Zone[5] . The protocol consisted of an initial assessment on admission (Day 1) and an assessment every 48 hours until ventilatory support was withdrawn (Days 3,5,7,8).

At the initial assessment, the patient was on mechanical ventilation, WL3 ventilator, PCV ventilation mode, parameters: PC above peep: 17cmH2O; Vt: 410ml; PEEP: 7 cmH2O; RR: 16ipm; FIO2: 0.28. No ventilatory drive during the period. Tdi-exp values 0.30cm. RASS - 4 , under sedation (Fentanyl and Midazolan, dose 2mcg/kg), without the use of vasoactive drugs, with diet infusion via nasoenteral tube at 30% of the expected calorie target.

On D3 of mechanical ventilation, under fixed sedation, RASS - 4, without the use of vasoactive drugs, nutritional intake at 100% of the expected calorie target. New Tdi values of 0.22cm were collected, a reduction of 26.6%.

When data was collected on D5, sedoanalgesia was weaned, RASS -2, with no signs of hemodynamic instability. The ventilatory mode was changed from pressure-controlled to pressure-support. The ventilatory parameters during collection were: PS above peep 12cmH20; PEEP: 7cmH2O; Vt: 540ml; RR: 17ipm; FIO2: 0.30; P0.1: -1.2cmH2O; Pmus: 3.0cmH20; the Tdi value was 0.14cm, a reduction of 53.3% on the value measured on admission. To calculate Pmus, we used the formula proposed by Natsumi, based on P0.1, Pmus = -2.99 x P0.1 + 0.53[18] . Normal values for Pmus are between 5 and 10cmH2O, but there is disagreement in the literature regarding exact values .[19]

In order to strengthen the respiratory muscles, a training protocol was instituted, consisting of a gradual reduction in support pressure, reaching Pmus values of between 5 and 6cmH20, during 3 minutes of training

with a 1 minute break, being applied on two consecutive days, the values collected on day 1 of training are shown in Table 1.

Training	Support pressure	P0.1	Pmus
Pre-training	12cmH2O	1.2 cmH2O	3.0 cmH2O
Training	9cmH2O	2.0 cmH2O	5.4 cmH2O
Training	0cmH20	2.0 cmH2O	5.4 cmH2O
After training	12cmH2O	1.6 cmH2O	4.2 cmH2O

Table 1 - First day of respiratory muscle training.

On the second day of the protocol, it was started with a lower support value, but following the same method as before, of gradual reduction and measurement of P0.1 and Pmus. The data collected on day 2 of the protocol is shown in Table 2.

Training	Support pressure	P0.1	Pmus
Pre-training	9cmH2O	0.6 cmH2O	1.2 cmH2O
Training	6cmH2O	1.8 cmH2O	4.8 cmH2O
Training	0cmH20	2.1 cmH2O	5.7 cmH2O
After training	9cmH2O	0.6 cmH2O	1.2 cmH2O

Table 2 - Second day of respiratory muscle training.

On day 7, the patient was reassessed, with ventilatory parameters: PS above peep: 8cmH2O; PEEP: 7cmH2O; Vt: 540ml; RR: 27; FIO2: 0.21; P0.1: -1.4cmH2O; Pmus: 3.6 cmH2O. Tdi-exp of 0.22cm, an increase of 57.1% compared to the measurement on day 5 of ventilation.

On day 8, before extubation, the last measurement was taken, with ventilatory parameters: PS above peep: 5cmH2O; PEEP: 5cmH2O; Vt: 490ml; RR: 26; FIO2: 0.21; P0.1: 1.2 cmH2O; Pmus: 3 cmH2O. Tdi value obtained 0.22cm.

The values measured for Tdi throughout the ventilation period, ventilation mode, blood pressure levels, P0.1 and Pmus are shown in Table 3.

DISCUSSION

Assessment of the diaphragm muscle using ultrasound has recently become an increasingly practical and easily reproducible method. By visualizing the diaphragm muscle, the classification of patients with dysfunction becomes clearer, and allows the team to implement an early and targeted strategy for the dysfunction in question[9,10] .

The prolonged duration of mechanical ventilation in critically ill patients is directly related to diaphragmatic atrophy. The fact that the muscle is partially or totally inactive has an influence on the speed of the dysfunction process, loss of contractile capacity and reduction in muscle mass. Factors that can predispose to these changes are closely linked to the patient's clinical condition, such as sepsis, trauma, polyneuropathy, use of vasoactive drugs and malnutrition, resulting in reduced protein synthesis and increased proteolysis.[7,8]

Studies indicate that the loss of diaphragm muscle mass begins in the first 12 hours of mechanical ventilation. This is because the peripheral muscles are subjected to more frequent overload and are less susceptible to the atrophy imposed by immobility. The diaphragm, on the other hand, being a muscle that is active 24 hours a day, ends up suffering greater atrophy in situations of inactivity .[12,13]

Over the course of the hospital stay, we observed a loss of diaphragm thickness in controlled modes, without the presence of a ventilatory drive. The total thickness loss was 53.3%, reaching 0.14cm in 5 days. After the transition to PSV mode and the application of the training protocol, an increase of 57.1% was seen, reaching a value of 0.22cm, maintaining this parameter until the moment of extubation, a value considered adequate in the literature. The studies show a divergence regarding the daily reduction in muscle thickness, due to the heterogeneity of the protocols adopted and the variation in the number of days on ventilation .[5]

Day	VM mode	PC/PS (above PEEP)	Tdi	P0.1	Pmus
1	PCV	17 cmH20	0.30cm		
3	PCV	17 cmH20	0,22cm		
5	PSV	12 cmH20	0,14cm	-1.2 cmH20	3.0 cmH2O
7	PSV	8 cmH20	0,22cm	-1.4 cmH20	3.6 cmH2O
8	PSV	5 cmH20	0,22cm	-1.2 cmH2O	3 cmH2O

Table 3

The transition from controlled to spontaneous ventilation proved effective in preventing atrophy. As already described in the literature, reducing diaphragm loss and maintaining diaphragm thickness is closely linked to muscle activation. It is important to highlight the pressure settings adopted, which can directly influence the process of protecting the diaphragm .[9,14]

For inspiratory muscle training, a preset pressure support reduction protocol was applied. With this reduction in ventilatory support, there is an increase in muscle pressure (Pmus) to maintain adequate ventilation. This increase in the load imposed on the muscles is one of the main concepts of training, where in order to gain strength, it is necessary to subject the muscles to periods of overload, as previously described [2,15].

Analyzing table 1 and 2, we can see an increase in Pmus compared to pre-training and during training, an increase of 70% over the initial resting value. By reducing the level of support and subjecting these muscles to an effort for a certain period of time, increasing the load and the work imposed, we can observe a double conditioning response, possibly improving strength and consequently endurance. This concept is described by McConnell, where the author mentions that stronger muscles end up performing certain tasks at lower percentages of their maximum capacity, compared to weaker muscles, so they are better able to sustain overload activities for longer periods of time. Therefore, a muscle with a certain fatigue threshold, which has its maximum strength increased, this previously imposed threshold ends up becoming a lower percentage load, imposing a considerably lower level of overload .[17]

Adjustments to ventilatory support levels, guided by P0.1, Pmus and Tdi, have proved effective, providing the therapist with parameters that reflect the patient's respiratory effort, thus guiding them towards adequate and synchronous ventilation, preventing under- or over-assistance, a strategy described as protective ventilation of the diaphragm, with the main objective of preventing myotrauma and maintaining an effective effort .[10,11]

The evaluation method used in the study is widely described in the literature and has proven to be efficient for data collection, providing practicality and easy data interpretation, but requires prior training and expertise of the evaluators .[5]

Evaluating the thickness of the diaphragm using ultrasound during the period of mechanical ventilation is a valuable tool for measuring the process of muscle atrophy and implementing measures early on to protect the diaphragm. Studies have shown that the combination of a muscle training protocol and the adoption of spontaneous ventilation modes has proved effective in reversing diaphragmatic dysfunction. There is a lack of consensus among the authors as to the percentage of daily loss, but this result is to be expected due to the heterogeneity of the samples.

CONCLUSION

The main objective of this study was to highlight the variation in thickness and respiratory effort with different ventilatory modes and settings and the variation in Tdi after a training protocol, correlating the mean time of mechanical ventilation. Therefore, in the final analysis of this report, it is inferred that the use of this respiratory muscle training protocol in this patient was relevant, given the significant increase in diaphragmatic thickness, which made it possible to reduce ventilatory parameters, consequently ventilatory weaning, resulting in shorter mechanical ventilation time. In view of this, we suggest and recognize the need for further studies to be carried out with a larger sample, in order to better guarantee the effectiveness of this protocol.

REFERENCES

1. WEST, John B. **Fisiologia respiratória-: Princípios básicos**. Artmed Editora, 2013.

2. Fisioterapia Respiratória/ Uma nova visão/ 3ª ed. Bruno Lombaerde Varella Presto, Luciana Damázio de Noronha Presto/ Bruno Lobaerde Varella Presto Editor Produtor Editor, Rio de Janeiro, 2007.

3. Dias AS, Sachetti A. Disfunção diafragmática induzida pelo ventilador: uma visão fisioterapêutica. In: Associação Brasileira de Fisioterapia Cardiorrespiratória e Fisioterapia em Terapia Intensiva; Martins JA, Reis LFF, Andrade FMD, organizadores. PROFISIO Programa de Atualização em Fisioterapia em Terapia Intensiva Adulto: Ciclo 10. Porto Alegre: Artmed Panamericana; 2019. p. 123–56.

4. Gibson GJ. Diaphragmatic paresis: pathophysiology, clinical features, and investigation. Thorax. 1989;44(11):960-970. https://doi.org/10.1136/thx.44.11.960

5. Santana PV, Cardenas LZ, Albuquerque ALP, Carvalho CRR, Caruso P. Diaphragmatic ultrasound: a review of its methodological aspects and clinical uses. J Bras Pneumol. 2020;46(6):e20200064.

6. López MF. Ultrassonografia cinesiológica na covid-19. In: Associação Brasileira de Fisioterapia Cardiorrespiratória e Fisioterapia em Terapia Intensiva; Martins JA, Reis LFF, Andrade FMD, organizadores. PROFISIO Programa de Atualização em Fisioterapia em Terapia Intensiva Adulto: Ciclo 12. Porto Alegre: Artmed Panamericana; 2021. p. 59–89. (Sistema de Educação Continuada a Distância, v. 1). https://doi.org/10.5935/978-65-5848-361-8.C0002.

7. Willhelm, Fernanda Franz, Escobar, Mariana e Perry, Ingrid D. SchweigertAlterações na composição corporal e em parâmetros antropométricos de dependentes de crack internados em unidade de adição. Jornal Brasileiro de Psiquiatria [online]. 2013, v. 62, n. 3 [Acessado 2 Outubro 2022] , pp. 183-190. Disponível em: <https://doi.org/10.1590/S0047-20852013000300002>. Epub 30 Out 2013. ISSN 1982-0208. https://doi.org/10.1590/S0047-20852013000300002.

8. Ferreira, Iára Kallyanna CavalcanteTerapia nutricional em Unidade de Terapia Intensiva. Revista Brasileira de Terapia Intensiva [online]. 2007, v. 19, n. 1 [Acessado 2 Outubro 2022] , pp. 90-97. Disponível em: <https://doi.org/10.1590/S0103-507X2007000100012>. Epub 23 Out 2007. ISSN 1982-4335. https://doi.org/10.1590/S0103-507X2007000100012.

9. Goligher EC, Laghi F, Detsky ME, Farias P, Murray A, Brace D, Brochard LJ, Bolz SS, Rubenfeld GD, Kavanagh BP, Ferguson ND. Measuring diaphragm thickness with ultrasound in mechanically ventilated patients: feasibility, reproducibility and validity. Intensive Care Med. 2015 Apr;41(4):642-9. doi: 10.1007/s00134-015-3687-3. Epub 2015 Feb 19. Erratum in: Intensive Care Med. 2015 Apr;41(4):734. Sebastien-Bolz, Steffen [corrected to Bolz, Steffen-Sebastien]. PMID: 25693448.

10. Shi Z, Zhou JX, Heunks L. Diaphragm-Protective Mechanical Ventilation: Saving the Diaphragm from the Detrimental Effects of Mechanical Ventilation. J Transl Crit Care Med 2022;4:5

11. Schepens, Toma; Dres, Martinb,c; Heunks, Leod; Goligher, Ewan C.e,f,g. Diaphragm-protective mechanical ventilation. Current Opinion in Critical Care: February 2019 - Volume 25 - Issue 1 - p 77-85 doi: 10.1097/MCC.0000000000000578.

12. Powers SK, Wiggs MP, Sollanek KJ, Smuder AJ. Ventilator-induced diaphragm dysfunction: cause and effect. Am J Physiol Regul Integr Comp Physiol. 2013 Sep;305(5):R464-77.

13. Hudson NJ, Franklin CE. Maintaining muscle mass during extended disuse: aestivating frogs as a model species. J Exp Biol. 2002 Aug;205(Pt 15):2297-303. doi: 10.1242/jeb.205.15.2297. PMID: 12110663.

14. Goligher EC, Dres M, Patel BK, Sahetya SK, Beitler JR, Telias I, Yoshida T, Vaporidi K, Grieco DL, Schepens T, Grasselli G, Spadaro S, Dianti J, Amato M, Bellani G, Demoule A, Fan E, Ferguson ND, Georgopoulos D, Guérin C, Khemani RG, Laghi F, Mercat A, Mojoli F, Ottenheijm CAC, Jaber S, Heunks L, Mancebo J, Mauri T, Pesenti A, Brochard L. Lung- and Diaphragm-Protective Ventilation. Am J Respir Crit Care Med. 2020 Oct 1;202(7):950-961. doi: 10.1164/rccm.202003-0655CP. PMID: 32516052; PMCID: PMC7710325.

15. Caruso P, Denari SD, Ruiz SA, Bernal KG, Manfrin GM, Friedrich C, et al. Inspiratory muscle training is ineffective in mechanically ventilated critically ill patients. Clinics (Sao Paulo). 2005 Dec;60(6):479-84.

16 Hudson NJ, Franklin CE. Maintaining muscle mass during extended disuse: aestivating frogs as a model species. J Exp Biol. 2002 Aug;205(Pt 15):2297-303. doi: 10.1242/jeb.205.15.2297. PMID: 12110663.

17. McConnell A. Breathe Strong, Perform Better. Champaign: Human Kinetics; 2011.

18. Hamahata NT, Sato R, Yamasaki K, et al. Estimating actual inspiratory muscle pressure from airway occlusion pressure at 100 msec. J Mech Vent 2020; 1(1)8-13.

19.Goligher EC, Jonkman AH, Dianti J, Vaporidi K, Beitler JR, Patel BK, Yoshida T, Jaber S, Dres M, Mauri T, Bellani G, Demoule A, Brochard L, Heunks L. Clinical strategies for implementing lung and diaphragm-protective ventilation: avoiding insufficient and excessive effort. Intensive Care Med. 2020 Dec;46(12):2314-2326. doi: 10.1007/s00134-020-06288-9. Epub 2020 Nov 2. PMID: 33140181; PMCID: PMC7605467.