detect the changes. 2) In the PEEP change group although an overdistention trend was observed, a possible explanation of the observed low hemodynamic impact could be precisely the low PEEP that was used, probably starting at low transpulmonary pressure and low end expiratory volumes. The increment in these two last variables, as a consequence of PEEP increment, could have not resulted in an excessively unfavourable situation for the relation between the pulmonary volume and pulmonary vascular resistance.

Our study is concordant with the hemodynamic findings mentioned by Evrard et al.,[4] the size of the cohort being similar. However, in our sample, as mentioned, no patient showed ventricular dysfunction.

An increase in PEEP tended to worsen pulmonary mechanics but without an enormous impact in hemodynamics.

The main limitation of the study is the small sample size which does not allow to draw more generalized conclusions regarding the objective of our research. Another limitation of our study is that the hemodynamic monitoring did not include advanced techniques that are able to detect cardiovascular change with high sensitivity.

Our result regarding the impact of MV on hemodynamics may be relevant at the time of choosing the best management strategy for this kind of patients.

In memoriam

In memoriam of our dear friend, Juan Martinez-Milla MD, PhD, who will always belong to the heart of our team.

References

1. Gattinoni L, Coppola S, Cressoni M, Busana M, Rossi S, Chiumello D. Covid-19 does not lead to a ''typical'' Acute Respiratory Distress Syndrome. Am J Respir Crit Care Med. 2020, http://dx.doi.org/10.1164/rccm.202003-0817LE. Published online March 30.
2. Gattinoni L, Chiumello D, Rossi S. COVID-19 pneumonia: ARDS or not? Crit Care. 2020;24:154.
3. Capotosto L, Nguyen BL, Ciardi MR, Mastroianni C, Vitarelli A. Heart, COVID-19 and echocardiography. Echocardiography. 2020;37:1454–64.
4. Evrard B, Goudelin M, Montmagnon N, Fedou AL, Lafon T, Vignon P. Cardiovascular phenotypes in ventilated patients with COVID-19 acute respiratory distress syndrome. Crit Care. 2020;24:236.

Ana-Maria Ioan[a], Oscar Gonzalez-Lorenzo[b], Andrea Kallmeyer Mayor[b], César Pérez-Calvo[a], Arnoldo Santos[a,*]

[a] *Department of Intensive Care Medicine, Hospital Universitario Fundación Jiménez Díaz, Avda. Reyes Católicos, 2, 28040 Madrid, Spain*
[b] *Department of Cardiology, Hospital Universitario Fundación Jiménez Díaz, Avda. Reyes Católicos, 2, 28040 Madrid, Spain*

* Corresponding author.
E-mail address: asantos@fjd.es (A. Santos).

Development of pectoral muscle atrophy in critically ill patients

Desarrollo de atrofia de los músculos pectorales en pacientes críticos

Dear Editor:

Skeletal muscle wasting and weakness have proven to be important determinants of critically ill patients' outcome. Reduced skeletal muscle mass on intensive care unit (ICU) admission has been associated with increased mortality and disability after discharge.[1–3] Moreover, muscle wasting occurring during ICU stay has also been associated with adverse outcomes.[4] However, the development of muscle atrophy in critically ill patients is highly heterogeneous among different muscle types.[5,6] Pectoral muscle area (PMA) on ICU admission, determined by computed tomography (CT) scan, has been associated with mortality.[1,7] On the contrary, tomographic evolution of PMA and its impact on patients' outcomes has not been reported. Therefore, we aimed to determine if the evolutionary pattern of PMA after ICU admission was associated with patient survival. We hypothesized that PMA wasting would be greater in non-survivors.

Thirty mechanically ventilated patients admitted to the ICU of a University Hospital (Hospital de Clínicas, Montevideo) from February 2016 to April 2020 and requiring two chest CT scans were retrospectively included in the study. Median time from ICU admission to the first CT scan was 0 (0–1) days and 12 (9–15) days for the second one. PMA measurement was performed as previously described from a single axial slice of the CT scan.[1] Muscles were manually shaded in the first axial slice above the superior aspect of the aortic branch using specific software (Weasis Medical Viewer) and PMA was computed in square centimeters as the aggregated area of right and left major and minor pectoral muscles (Supplementary Fig. 1). The study was approved by the institution's Research Ethics Committee.

Categorical variables are reported as absolute numbers (percentage) and were compared using Chi-square test or Fisher exact test. Continuous variables are expressed as mean ± standard deviation if normally distributed, or median (25th–75th percentile) if not. PMA evolution was analyzed through Wilcoxon signed-rank test. Student *t*-test or Mann–Whitney *U* test were performed to compare variables between groups. The Spearman correlation test was used to analyze bivariate correlations. Kaplan-Meier curves and the log-rank test were applied to compare ICU mortality in patient groups stratified by PMA. A P value < 0.05 was considered statistically significant.

DOI of refers to article: https://doi.org/10.1016/j.medin.2022.10.003

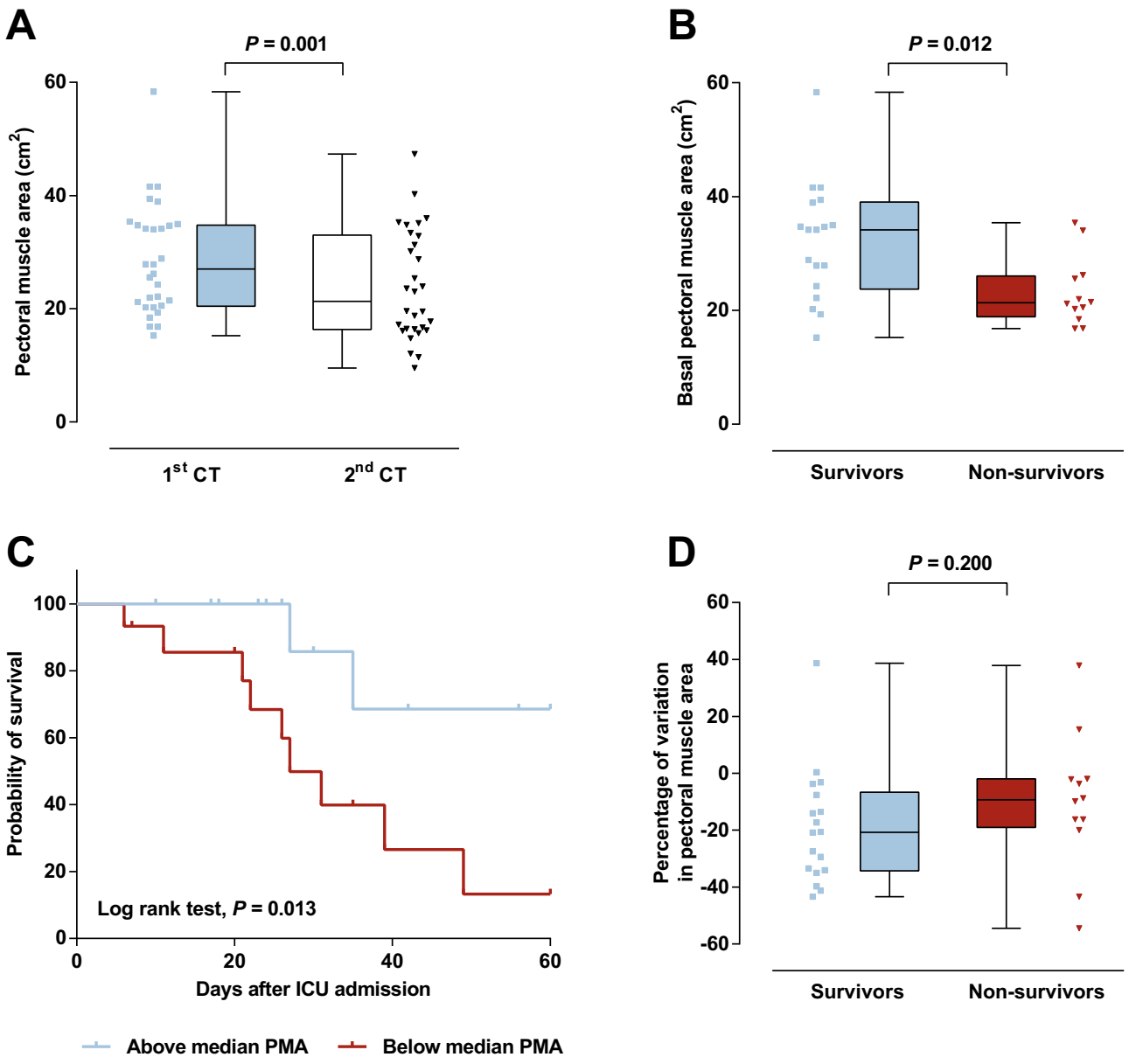

Figure 1 **(A)** A significant decrease in pectoral muscle area (PMA) was observed between first (1st) and second (2nd) CT scans. **(B)** Survivors presented a higher PMA on admission than non-survivors. **(C)** Survival curves for patients with admission PMA below or above the median value of 27.0 cm^2. **(D)** No significant difference was observed in the variation of PMA between 1st and 2nd CT scans.

Patient's characteristics are summarized in Table 1. Most patients were male (77%), median time on mechanical ventilation was 21 (13–32) days and ICU mortality was 40%. The vast majority (87%) presented a low nutritional risk as assessed by the modified NUTRIC score (mNUTRIC).[8] Median PMA on admission CT scan (PMA_1) was 27.0 (20.5–34.8) cm^2, and was inversely correlated with patients' age ($r_s = -0.506$, $P = 0.004$), mNUTRIC ($r_s = -0.717$, $P < 0.001$) and SAPS III ($r_s = -0.657$, $P < 0.001$; Supplementary Figure 2). Males had a higher PMA on admission than females (34.1 (20.5–35.4) cm^2 *versus* 21.5 (19.3–24.3) cm^2, $P = 0.033$). PMA measured on the second CT scan (PMA_2) was significantly reduced from baseline to 21.3 (16.3–33.0) cm^2 ($P = 0.001$; Fig. 1A), with a median variation of −16.2 ([−33.6]–[−3.5])%. Neither PMA measurements (PMA_1 and PMA_2) nor its percentage of variation were correlated with the duration of mechanical ventilation or ICU stay. Baseline PMA was significantly higher in patients discharged alive from ICU than in non-survivors (34.1 (23.8–39.1) cm^2 *versus* 21.3 (18.9–26.0) cm^2, $P = 0.012$; Fig. 1B). A significant difference in ICU mortality was observed between patients whose admission muscle area was above or below the median PMA (13% *versus* 67%, respectively, $P = 0.013$; Fig. 1C). The percentage of variation from PMA_1 to PMA_2 was not significantly different between survivors and non-survivors ($P = 0.200$; Fig. 1D). However, a trend towards a more pronounced reduction in PMA was observed in survivors, which could be related to the higher muscle mass

Table 1 Baseline characteristics of the cohort.

Variable	All patients (n = 30)	Survivors (n = 18)	Non-survivors (n = 12)	*P* value
Gender, male/female	23/7	16/2	7/5	0.053
Age, years	53 (29–68)	45 (29–64)	59 (29–75)	0.200
SAPS III	66 ± 14	60 ± 10	75 ± 15	0.003
SOFA on admission	6 ± 3	5 ± 2	7 ± 3	0.018
mNUTRIC	3 (2–5)	2 (2–4)	5 (3–6)	0.010
Body mass index (kg/m^2)	28.5 ± 8.1	27.8 ± 7.2	29.9 ± 10.8	0.687
Reason for ICU admission, n (%)				0.183
Sepsis	15 (50)	8 (44)	7 (58)	
Trauma	11 (37)	9 (50)	2 (17)	
Neurologic dysfunction	3 (10)	1 (6)	2 (17)	
Respiratory failure	1 (3)	0 (0)	1 (8)	
Medication, n (%)				
Midazolam	30 (100)	18 (100)	12 (100)	–
Opioids	30 (100)	18 (100)	12 (100)	–
Propofol	9 (30)	6 (33)	3 (25)	0.704
Atracurium	11 (37)	7 (39)	4 (33)	0.999
Vasopresors	14 (47)	9 (50)	5 (42)	0.722
Steroids	10 (33)	6 (33)	4 (33)	0.999
Aminoglycosides	5 (17)	4 (22)	1 (8)	0.622
Nutritional support				
Caloric intake day 3 (kcal)	1131 ± 409	1113 ± 391	1169 ± 504	0.834
Caloric intake day 7 (kcal)	1559 ± 388	1525 ± 438	1650 ± 260	0.659
Renal replacement therapy	8 (27)	2 (11)	6 (50)	0.018
Invasive mechanical ventilation	30 (100)	18 (100)	12 (100)	–
Days on mechanical ventilation	21 (13–32)	20 (13–24)	26 (13–35)	0.249
ICU length of stay, days	26 (18–35)	24 (17–35)	27 (21–38)	0.439

Categorical variables are presented as n (%) and continuous variables as median (25th–75th percentile) or mean ± standard deviation. *P* value represents significance between survivors and non-survivors.

on admission in these patients. Of note, caloric intake on the third and seventh days was similar between survivors and non-survivors ($P = 0.834$ and 0.659, respectively), and was not correlated with the percentage of variation from PMA_1 to PMA_2 ($P = 0.505$ and 0.849, respectively).

CT-determined cross-sectional area of different skeletal muscles at the time of admission to the ICU has been associated with prognosis. In concordance with our results, Jaitovich et al. reported higher survival in patients with larger admission PMA.[1] Furthermore, reduced baseline erector spinae,[2] psoas,[9] and total abdominal muscles' cross-sectional area (evaluated at the third lumbar vertebra)[3] have been associated with negative outcomes in critically ill patients. While the relevance of different muscles' mass on admission has been consistently demonstrated, their evolution during critical illness and its impact on patients' prognosis seems more heterogeneous. Upper and lower limb muscle atrophy after ICU admission has been described by numerous authors,[5,10] and its development is associated with increased mortality.[4] On the contrary, abdominal muscle area was not reduced in patients with acute pancreatitis requiring ICU admission.[6] Previous work by Vivier et al. studied pectoral muscle thickness using ultrasound in 34 critically ill patients.[11] Overall, no significant difference was found in pectoral muscle thickness between day 1 and day 5 of ICU stay. Moreover, when the authors analyzed the subgroup of patients that developed pectoral muscle atrophy (*i.e.*, thickness reduction $\geq$ 10% between days 1 and 5) found no difference in mortality compared to the other patients. In contrast to Vivier et al., we found a significant decrease in PMA after ICU admission. This might be explained by a longer period between measurements (9 *versus* 5 days) and different methods used to evaluate pectoral muscle mass (CT scan *versus* ultrasound). However, neither of the studies found an association between the reduction in pectoral muscle mass and mortality. The different relationship between limb or pectoral muscle atrophy and patients' outcome could be related to the distinct functional domains involved (*i.e.*, locomotor and non-locomotor).

Our study has certain limitations. First, it was a retrospective study conducted in a single center, including a small number of patients with heterogeneous characteristics. Therefore, the independent impact of PMA on patients' outcomes after adjusting for other variables (*e.g.*, age, SAPS III, *etc.*) could not be determined. Second, data regarding the catabolic state and fluid balance of the patients could not be obtained. Finally, muscle strength and functional status after ICU discharge was evaluated.

In conclusion, development of pectoral muscle atrophy after ICU admission was demonstrated by CT scan for the first time. Admission PMA was associated with patients' survival, although further studies are required in order to confirm these results and evaluate the effect of pectoral muscle atrophy developed during ICU stay.

Funding

None.

Conflict of interests

None.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.medine.2022.11.001.

References

1. Jaitovich A, Khan M, Itty R, Chieng H, Dumas C, Nadendla P, et al. ICU admission muscle and fat mass, survival, and disability at discharge: a prospective cohort study. Chest. 2019;155(2):322–30.
2. Jaitovich A, Dumas CL, Itty R, Chieng H, Khan M, Naqvi A, et al. ICU admission body composition: skeletal muscle, bone, and fat effects on mortality and disability at hospital discharge—a prospective, cohort study. Crit Care. 2020;24(1):566.
3. Weijs PJ, Looijaard WG, Dekker IM, Stapel S, Girbes A, Oudemans-van Straaten H, et al. Low skeletal muscle area is a risk factor for mortality in mechanically ventilated critically ill patients. Crit Care. 2014;18(2):R12.
4. Nakanishi N, Oto J, Tsutsumi R, Akimoto Y, Nakano Y, Nishimura M. Upper limb muscle atrophy associated with in-hospital mortality and physical function impairments in mechanically ventilated critically ill adults: a two-center prospective observational study. J Intensive Care. 2020;8(1):87.
5. Carambula A, Pereyra S, Barbato M, Angulo M. Combined diaphragm and limb muscle atrophy is associated with increased mortality in mechanically ventilated patients: a pilot study. Arch Bronconeumol. 2021;57(5):377–9.
6. Brewster DJ, Strauss BJ, Crozier TM. Measuring visceral fat, subcutaneous fat and skeletal muscle area changes by computed tomography in acute pancreatitis: a retrospective, single-centre study. Crit Care Resusc. 2014;16(1):42–7.
7. Moon SW, Kim SY, Choi JS, Leem A, Lee S, Park M, et al. Thoracic skeletal muscle quantification using computed tomography and prognosis of elderly ICU patients. Sci Rep. 2021;11(1):23461.
8. Heyland DK, Dhaliwal R, Jiang X, Day AG. Identifying critically ill patients who benefit the most from nutrition therapy: the development and initial validation of a novel risk assessment tool. Crit Care. 2011;15(6):R268.
9. Rangel EL, Rios-Diaz AJ, Uyeda JW, Castillo-Angeles M, Cooper Z, Olufajo O, et al. Sarcopenia increases risk of long-term mortality in elderly patients undergoing emergency abdominal surgery. J Trauma Acute Care Surg. 2017;83(6):1179–86.
10. Puthucheary ZA, Rawal J, McPhail M, Connolly B, Ratnayake G, Chan P, et al. Acute skeletal muscle wasting in critical illness. JAMA. 2013;310(15):1591–600.
11. Vivier E, Roussey A, Doroszewski F, Rosselli S, Pommier C, Carteaux G, et al. Atrophy of diaphragm and pectoral muscles in critically ill patients. Anesthesiology. 2019;131(3):569–79.

O. Nosiglia[a], V. Cambón[a], P. Filippi[a], A. Ríos[a], Y. Romero[a], A. Vacca[a], C. Gaiero[c], M. Angulo[a,b,*]

[a] *Departamento de Fisiopatología, Hospital de Clínicas, Universidad de la República, Montevideo, Uruguay*
[b] *Laboratorio de Exploración Funcional Respiratoria, Centro de Tratamiento Intensivo, Hospital de Clínicas, Universidad de la República, Montevideo, Uruguay*
[c] *Centro de Tratamiento Intensivo, Hospital de Clínicas, Universidad de la República, Montevideo, Uruguay*

* Corresponding author.
E-mail address: martin.angulo@hc.edu.uy (M. Angulo).

Effect of a specific training program on patient-ventilator asynchrony detection and management

Efecto de un programa específico de entrenamiento en la identificación y manejo de asincronía paciente-ventilador

In patients undergoing mechanical ventilation (MV), patient-ventilator asynchrony (PVA) is a common phenomenon associated with poor clinical outcomes including increased mortality and longer duration of MV.[1,2,3] Our group have previously shown that proper identification and management of PVA at the bedside, using waveform analysis, is very challenging for health care professionals (HCPs) regardless of profession and clinical experience.[4,5] Although, some studies have shown the effects of a specific training program on the ability of HCPs to identify PVA,[4,5,6,7] no studies have currently explore the effect of a training program on the ability of HCPs to determine potential causes and management of PVA.

The aim of the scientific letter is to communicate the effect of a specific training program on the ability of HCPs, with less than one year of clinical experience, to identify and determine potential causes and management of PVA. This pilot study was approved by the Ethics committee of the Hospital Clínico de la Universidad de Chile (File N° 1097/20).

We conducted a quasi-experimental study in which an invitation was sent to HCPs from 20 hospitals in Latin America to participate in a specific online training program designed to assess the effect on proper recognition, management, and determine the potential causes of PVA. The inclusion criteria for HCPs were: 1) less than one year of experience proven by a certificate validated by their hospitals; 2) HCPs without formal training on PVA; 3) Currently experience working in the ICU and mechanically ventilated patients.

The program included 6 h of live streaming using the ZOOM® platform in which HCPs could interact with the instructor. Subsequently, HCPs had access to interact with the instructor for a period of one month after they completed the program using an online direct contact. An assessment tool was designed and validated by three experts with an inter-observer agreement of 100%. The assessment