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CARDIAC OUTPUT IN CRITICALLY ILL PATIENTS CAN BE ESTIMATED EASILY AND ACCURATELY USING THE MINUTE DISTANCE OBTAINED BY PULSED-WAVE DOPPLER

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ABSTRACT—**Background:** Cardiac output (CO) assessment is essential for management of patients with circulatory failure. Among the different techniques used for their assessment, pulsed-wave Doppler cardiac output (PWD-CO) has proven to be an accurate and useful tool. Despite this, assessment of PWD-CO could have some technical difficulties, especially in the measurement of left ventricular outflow tract diameter (LVOTd). The use of a parameter such as minute distance (MD) which avoids LVOTd in the PWD-CO formula could be a simple and useful way to assess the CO in critically ill patients. Therefore, the aim of this study was to evaluate the correlation and agreement between PWD-CO and MD. **Methods:** A prospective and observational study was conducted over 2 years in a 30-bed intensive care unit (ICU). Adult patients who required CO monitoring were included. Clinical echocardiographic data were collected within the first 24 h and at least once more during the first week of ICU stay. PWD-CO was calculated using the average value of three LVOTd and left ventricular outflow tract velocity-time integral (LVOT-VTI) measurements, and heart rate. Minute distance was obtained from the product of LVOT-VTI × heart rate. Pulsed-wave Doppler cardiac output was correlated with MD using linear regression. Cardiac output was quantified from the MD using the equation defined by linear regression. Bland-Altman analysis was also used to evaluate the level of agreement between CO calculated from MD (MD-CO) and PWD-CO. The percentage error was calculated. **Results:** A total of 98 patients and 167 CO measurements were analyzed. Sixty-seven (68%) were male, the median age was 66 years (interquartile range [IQR], 53–75 years), and the median Acute Physiology and Chronic Health Evaluation II score was 22 (IQR, 16–26). The most common cause of admission was shock in 81 patients (82.7%). Sixty-nine patients (70.4%) were mechanically ventilated, and 68 (70%) required vasoactive drugs. The median CO was 5.5 L/min (IQR, 4.8–6.6 L/min), and the median MD was 1,850 cm/min (IQR, 1,520–2,160 cm/min). There was a significant correlation between PWD-CO and MD-CO in the general population (R^2 = 0.7; P < 0.05). This correlation improved when left ventricular ejection fraction (LVEF) was less than 60% (R^2 = 0.85, P < 0.05). Bland-Altman analysis showed good agreement between PWD-CO and MD-CO in the general population, the median bias was 0.02 L/min, the limits of agreement were −1.92 to +1.92 L/min. The agreement was better in patients with LVEF less than 60% with a median bias of 0.005 L/min and limits of agreement of −1.56 to 1.55 L/min. The percentage error was 17% in both cases. **Conclusion:** Measurement of MD in critically ill patients provides a simple and accurate estimate of CO, especially in patients with reduced or preserved LVEF. This would allow earlier cardiovascular assessment in patients with circulatory failure, which is of particular interest in difficult clinical or technical conditions.

KEYWORDS—Cardiac output; pulsed-wave doppler; minute distance; shock; intensive care; ejection fraction

INTRODUCTION

The main objective in the management of patients with circulatory failure is to achieve adequate organ perfusion and oxygen delivery to meet tissue requirements. To achieve this goal, the assessment of cardiac output (CO) is essential because it allows the identification of the type of shock, selection of the most appropriate therapeutic intervention, and evaluation of the response to therapy (1). Various technics are currently used to measure CO in critically ill patients. Among them, pulsed-wave Doppler cardiac output (PWD-CO) has shown reasonable agreement with standard thermodilution techniques such as pulmonary artery catheter (PAC) and transpulmonary thermodilution (2). It is therefore proposed as a useful tool for the early assessment of patients in shock (3–5). This is mainly because of its ability to determine the type of shock (hypovolemia, cardiac dysfunction, cardiac tamponade, acute cor pulmonale, and/or a distributive circulation) and to infer immediate therapeutic decisions (6–8) but also because of some specific characteristics such as noninvasiveness, low cost, bedside application, and widespread availability.

The measurement of PWD-CO combines the recording of the left ventricular outflow tract velocity-time integral (LVOT-VTI) and calculation of the left ventricular outflow tract diameter (LVOTd). However, although the measurement of LVOT-VTI can be a feasible and reliable parameter for assessment in the intensive care unit (ICU) or emergency department (9), LVOTd can be difficult to measure because of characteristics such as variations associated with the cardiac cycle during systole and the use of mechanical ventilation in critically ill patients. In addition, it has been reported that measurement error can be higher than 20% in inexperienced hands (10–13). Considering these technical difficulties and the fact that any change in the CO is essentially due to variations in the LVOT-VTI or heart rate (HR), it could be argued that the use of a parameter that avoids LVOTd in the PWD-CO formula could be a simple, reliable, and useful way to assess CO in critically ill patients. This parameter could be the minute distance (MD), defined as the product of LVOT-VTI and HR.

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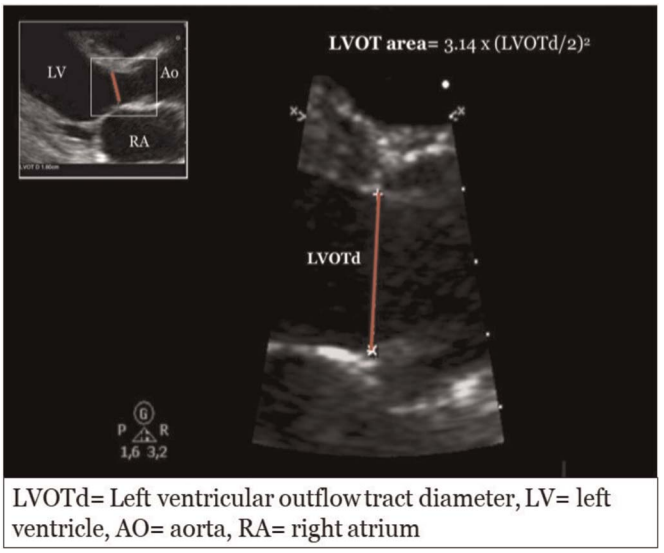

FIG. 1. **Measurement of left ventricular outflow tract cross-sectional area.** LVOTd indicates left venticular outflow tract diameter; LV, left ventricle; AO, aorta; RA, right atrium.

Therefore, the aim of the present study was to investigate the correlation and agreement between PWD-CO and MD in critically ill patients and to evaluate factors that influence this relationship, such as left ventricular ejection fraction (LVEF).

MATERIALS AND METHODS

Study design

This prospective and observational study was conducted for 2 years from November 2019 to October 2021 in a 30-bed ICU in a university hospital (Tarragona, Spain).

Approval was obtained from the Ethics Committee of the Joan XXIII University Hospital (Ref. CEIM:130/2019), and the study was considered to have minimal risk to the participants. Informed consent was obtained from each participant or their next kin.

Patients

The inclusion criteria were as follows: patients older than 18 years with a diagnosis of shock that required transthoracic echocardiography. Monitoring was decided by the treating physician in accordance with the protocols of our ICU and the European Society of Intensive Care Medicine Task Force (14). The investigators did not change the medical management based on the results of this study.

The exclusion criteria included a medical history of congenital heart disease, severe tricuspid regurgitation, severe aortic regurgitation, aortic stenosis, pregnancy, atrial fibrillation, and low-quality images.

Data collection

Anthropometric data were obtained from medical records or relatives. In some cases, weight and height were measured in the ICU.

We defined shock as follows: systolic arterial pressure <90 mm Hg, MAP <70 mm Hg, or decrease ≥40 mm Hg from baseline; associated with some evidence of tissue hypoperfusion: poor peripheral perfusion (cold skin, lividity, prolonged capillary refill >2 s); oliguria: diuresis <0.5 mL/kg/h; altered mental status (disorientation, clouding, or confusion); lactate >2 mEq/L; or the need for vasoactive drugs (14).

Study protocol and data measurements

Subjects were enrolled during the first 24 h after admission to the ICU. The following demographic, clinical, and physiological data were collected: age, sex, weight, height, HR, comorbidities, MAP, diuresis, Acute Physiology and Chronic Health Evaluation II score (15), the Sequential Organ Failure Assessment score (16), use of mechanical ventilation, use of renal replacement therapy, and need for vasoactive drugs. All echocardiographic measurements were performed using a Philips Sparq ultrasound system (Philips Ultrasound; Royal Philips Electronics, Bothell, WA) equipped with a 3.5 MHz phased-array transducer.

Echocardiographic measurements

Measurements were performed by senior intensivists during routine echocardiographic studies in critically ill patients. All ultrasound images obtained were stored in a digital format.

Once a subject was enrolled, we performed echocardiographic measurements within the first 24 h and at least once more during the first week of their ICU stay.

To measure the LVOTd, we first acquire a high-quality parasternal long-axis view focusing on the left ventricular outflow tract (LVOT) and the aortic valve. Second, we zoom and freeze the image to see the aortic valve open at maximum midsystole and finally measure the distance from the inner edge to the inner edge of the LVOT in a line parallel to the aortic annulus from the base of coronary cusp of the right aortic valve to the base of the noncoronary cusp (Fig. 1). The average of three LVOTd measurements was used to calculate the LVOT cross-sectional area (17). It was calculated as follows:

$$\text{LVOT area} = 3.14 \times (\text{LVOTd}/2)^2.$$

Left ventricular outflow tract velocity-time integral was measured by obtaining an apical five-chamber view and then placing the pulsed-wave Doppler sample volume in the center of the LVOT, below the aortic valve annulus. The resulting tracing was of the LVOT systolic velocity. We traced the systolic velocity flow curve, and the cardiac software calculated the area under the curve to obtain the LVOT-VTI. It was measured simultaneously during the respiratory cycle, ideally at the end of expiration, and the average of the three values was used. The LVOT-VTI was measured in centimeters and represented the distance that blood propelled forward from the LVOT to the aorta with each cardiac contraction (Fig. 2).

Pulsed-wave Doppler cardiac output was calculated using the concept that a volume of blood in the form of a cylinder is pushed forward by the volume of the cylinder whose base is the LVOT cross-sectional area and whose height is the LVOT-VTI. The LVOT-VTI represents the distance that this cylinder of blood travels with each left ventricular contraction (Fig. 3). We calculated the PWD-CO as follows:

$$\text{PWD-CO}\,(\text{L/min}) = \text{LVOT area}\,(\text{cm}^2) \times \text{LVOT-VTI}\,(\text{cm}) \times \text{HR}\,(\text{min}^{-1}).$$

Heart rate was calculated by cardiac ultrasound cardiac and not by physical examination or telemetry.

We calculated MD by excluding LVOTd from the PWD-CO formula described previously:

$$\text{MD}\,(\text{cm/min}) = \text{LVOT-VTI}\,(\text{cm}) \times \text{HR}\,(\text{min}^{-1}).$$

Left ventricular ejection fraction was calculated using the monoplane or biplane method of disks from apical two- and four-chamber views.

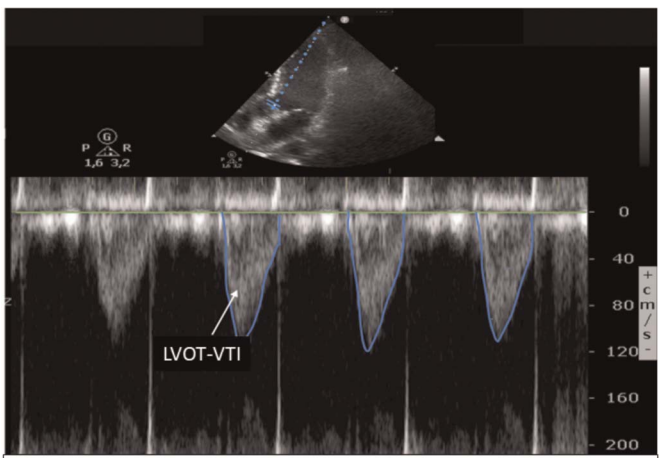

FIG. 2. **Measurement of left ventricular outflow tract velocity-integral time.** LVOT-VTI indicates left ventricular outflow tract velocity-integral time.

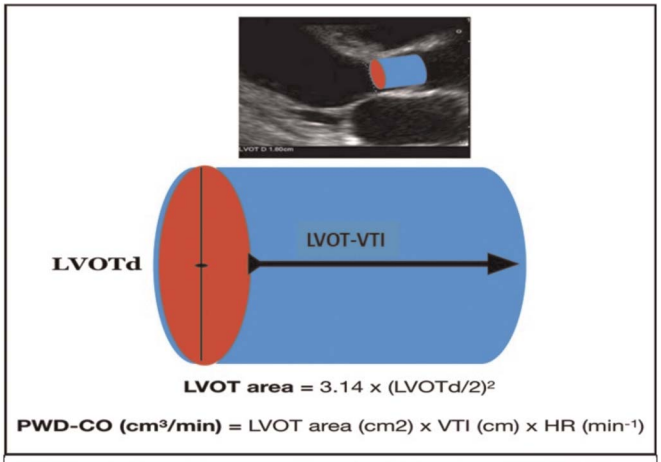

FIG. 3. **Pulsed-wave Doppler cardiac calculation.** LVOTd indicates left ventricular outflow tract diameter; LVOT-VTI, left venticular outflaw tract velocity-integral time; PWD-CO, pulsed-wave Doppler cardiac output; HR, heart rate.

Endpoints

The primary endpoint of our study was to assess the correlation and agreement between PWD-CO and CO calculated from MD (MD-CO). Secondary endpoints included the correlation of these measurements with high LVEF.

Statistical analysis

The normality of the data distribution was checked using the Kolmogorov-Smirnov test. Data are reported as number (percentage) for categorical variables, median (interquartile range [IQR]) for nonparametric variables, and medium and SD for parametric variables. Pulsed-wave Doppler cardiac output was correlated with MD using linear regression and MD-CO using the equation defined by linear regression. Bland-Altman analysis was also used to evaluate the level of agreement between MD-CO and PWD-CO. The percentage error (PE) of agreement between the two techniques was calculated using the following equation:

$$\text{PE PAC}-\text{PWD} = \sqrt{\left[(\text{precision PAC})^2 + (\text{precision PWD})^2\right]}.$$

All statistical analyses were performed using IBM SPSS Statistics for Windows, version 24.0 (IBM Corp., Armonk, NY). The threshold for statistical significance was set at $P < 0.05$

RESULTS

Patient characteristics at admission

A total of 98 patients requiring PWD-CO monitoring at ICU admission were included in the study. Sixty-seven (68%) were male, the median age was 66 years (IQR, 53–75 years), and the median Acute Physiology and Chronic Health Evaluation II score was 22 (IQR, 16–26). The most common cause of admission was shock in 81 patients (82.7%). Sixty-nine patients (70.4%) were mechanically ventilated, and 68 (70%) required vasoactive drugs. We assessed LVEF in 45% of patients, with a mean ± SD of 51.1 ± 14.1. The baseline characteristics of the general population are shown in Table 1.

CO measurements

From 241 echocardiographic studies, CO was measured in 167 patients (Fig. 4). The median CO was 5.5 L/min (IQR, 4.8–6.6 L/min), and the median MD was 1,850 cm/min (IQR, 1,563–2,160 cm/min) (Table 2). Patients with an LVEF >60% had significantly higher LVOT-VTI (24.15 m/s vs. 20 m/s, $P < 0.001$), MD (2,075 cm/min vs. 1,822 cm/min, $P = 0.003$), and left ventricular end-systolic volume index (9.6 cm^3 vs. 15.5 cm^3, $P < 0.001$) than patients with lower LVEF (Table 3).

There was a significant correlation between PWD-CO and MD-CO in the general population ($R^2 = 0.7$, $P < 0.05$: Figure 5). When we evaluated this correlation according to the LVEF, we found that it improved when LVEF was less than 60% ($R^2 = 0.85$, $P < 0.05$; Figure 6).

TABLE 1. **Baseline characteristics of general population**

Variables	Population (N = 98)
Demographics	
Age (years, median, IQR)	66 (53–75)
Male (n, %)	67 (68.4)
BMI (kg/m^2, median, IQR)	26.1 (23.7–30.4)
BSA (mean ± SD) (m_2)	1.87 ± 0.19
Overweight (n, %)	31 (31.6)
Obesity (n, %)	25 (25.5)
Hypertension (n, %)	52 (53.1)
Diabetes mellitus (n, %)	31 (31.6)
Chronic renal disease (n, %)	8 (8.2)
COPD (n, %)	13 (13.3)
Cardiovascular disease (n, %)	7 (7.1)
Immunosuppression (n, %)	27 (27.6)
Severity score	
APACHE II score (median, IQR)	22 (16–26)
At admission	
Sepsis (n, %)	75 (76.5)
Lactate at admission mmol/L (median, IQR)	2.7 (1.8–4)
Shock (n, %)	81 (82.7)
>Septic shock (n, %)	51 (62.9)
LVEF at admission (median ± SD) [n]	51.1 ± 14.1 [44]
ICU mortality (n, %)	19 (19.4)
Mechanical ventilation (n, %)	69 (70.4)

BMI, body mass index; BSA, body surface area; COPD, chronic obstructive pulmonary disease; APACHE II, Acute Physiology and Chronic Evaluation II; LVEF, left ventricular ejection fraction; ICU, intensive care unit.

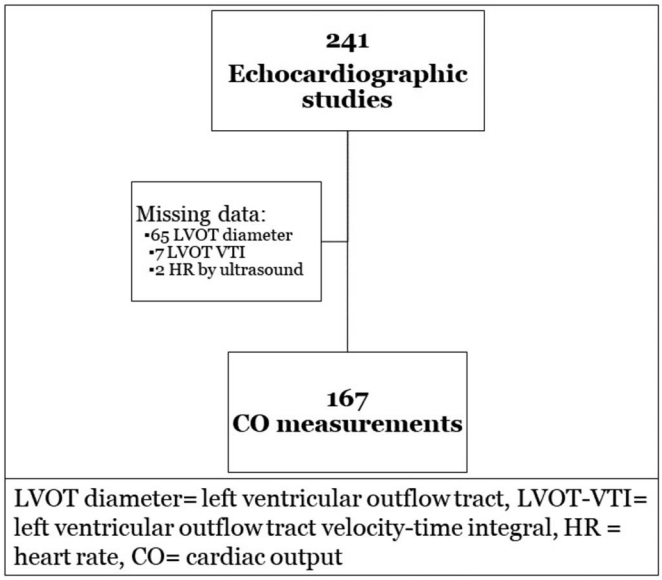

FIG. 4. **Flowchart of CO measurements.** LVOT indicates left ventricular outflow tract; LVOT-VTI, left ventricular outflow tract velocity-time integral; HR, heart rate; CO, cardiac output.

TABLE 2. **Hemodynamic parameters of the measurements**

Hemodynamic parameters	Measurements (n = 167)
HR (bpm, mean ± SD) [n]	90 ± 16.9
MAP (mm Hg, median, IQR) [n]	80.8 (75–90)
Norepinephrine (μg/kg/min, median, IQR) [n]	0.4 (0.14–0.6) [105]
LVOT-VTI (m/s, median, IQR) [n]	21 (18.3–24)
Aortic peak velocity (m/s, mean ± SD) [n]	125 ± 22.4 [62]
Aortic acceleration (mean ± SD) [n]	2,327 ± 865 [61]
SVI (mL/m^2, mean ± SD) [n]	19.9 ± 5.17
S′ LV (cm/s, median, IQR) [n]	11 (9.4–13.6) [57]
LVOTd (cm, median, IQR) [n]	1.96 (1.86–2.1)
LV-EDVI (mL/m^2, median, IQR) [n]	31.8 (25.9–41.7) [63]
LV-ESVI (mL/m^2, median, IQR) [n]	13.8 (9.6–17.7) [63]
LVEF (median, IQR) [n]	56 (47–63) [87]
PWD-CO (L/min, median, IQR) [n]	5.5 (4.8–6.6)
MD (cm/min, median, IQR) [n]	1,850 (1,563–2,160)

HR, heart rate; LVOT-VTI, left ventricular outflow tract velocity-time integral; SVI, stroke volume index; S′ LV, S′ left ventricle; LVOTd, left ventricular outflow tract diameter; LV-EDVI, left ventricular end-diastolic volume index; LV-ESVI, left ventricular end-systolic volume index; LVEF, left ventricular ejection fraction; PWD-CO, pulsed-wave Doppler cardiac output; MD, minute distance.

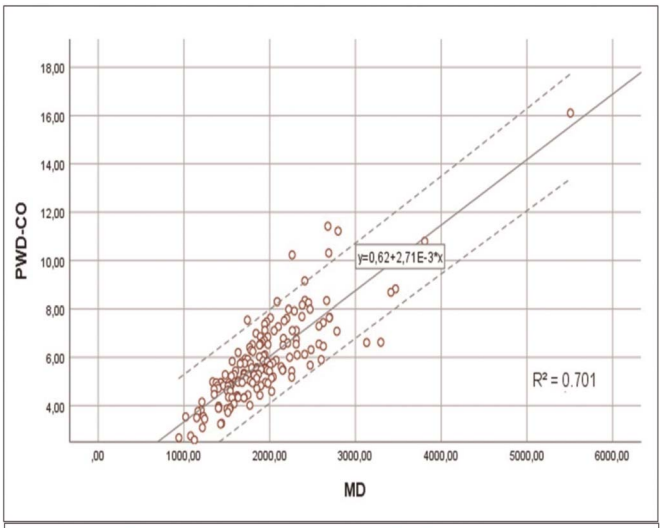

FIG. 5. **Correlation between PWD-CO and MD.** Equation defined by lineal regression. PWD-CO indicates pulsed-wave Doppler cardiac output; MD, minute distance.

Bland-Altman analysis showed good agreement between PWD-CO and MD-CO in the general population, the median bias was 0.02 L/min, and the limits of agreement were −1.92 to +1.92 L/min (Fig. 7). This agreement was better in patients with LVEF less than 60% with a median bias of 0.005 L/min and limits of agreement ranging from −1.56 to 1.55 L/min (Fig. 8). The PE was 17% in both cases.

DISCUSSION

In this study, we found that, in critically ill patients requiring CO monitoring, MD provides a simple and accurate estimation of CO, despite the exclusion of LVOTd in the measurement. Furthermore, this estimation appears to be more accurate in patients with reduced or preserved LVEF. This may allow its use in difficult clinical or technical conditions.

Traditionally, CO estimation has been assessed using invasive thermodilution methods and limited to the ICU or operating room; however, with the increasing availability of echocardiography since the 1980s, several studies have demonstrated that PWD signals provide a simple, practical, and accurate measurement of CO in stable patients (18–20). Similarly, subsequent studies have demonstrated the accuracy of PWD-CO assessment and monitoring in critically ill patients compared with PAC, even by intensivists with basic training (7,8,21,22).

Nevertheless, the acquisition of high-quality images to measure the LVOTd is the main limitation in the estimation of PWD-CO. First, because it is squared, any error in the measurement of LVOTd leads to a multiplicative effect on the accuracy of CO measurement, and second, transthoracic echocardiography tends to underestimate the measurement of LVOTd (23). In this regard, some studies have described that, in critically ill patients,

TABLE 3. **Hemodynamic parameters according to LVEF**

Hemodynamic parameters	LVEF >60% n = 29	LVEF <60% n = 58	*P*
HR (bpm, mean ± SD) [n]	90.3 ± 17.8	92 ± 18.7	0.664
MAP (mm Hg, median, IQR) [n]	80 (74–91)	81 (75–90)	0.708
Norepinephrine (μg/kg/min, median, IQR) [n]	0.6 (0.3–1) [14]	0.4 (0.13–0.42) [38]	0.021
LVOT-VTI (m/s, median, IQR) [n]	24.15 (21.9–27.5)	20 (17–22)	0.000
Aortic peak velocity (m/s, mean ± SD) [n]	137.5 ± 19.3 [20]	120 ± 21.3 [38]	0.003
Aortic acceleration (mean ± SD) [n]	2,740 ± 831.2 [21]	2,129 ± 824.2 [36]	0.009
SVI (mL/m^2, mean ± SD) [n]	22.8 ± 4.8 [29]	19 ± 5.7 [58]	0.003
S′ LV (cm/s, median, IQR) [n]	12.7 (10.9–15.15) [18]	10.3 (9.1–12) [35]	0.014
LVOT diameter (cm, median, IQR) [n]	1.9 (1.8–2.05)	1.9 (1.8–2.1)	0.298
LV-EDVI (mL/m^2, median, IQR) [n]	28.2 (23.1–38.6) [21]	32.2 (27.2–47.4) [40]	0.122
LV-ESVI (mL/m^2, median, IQR) [n]	9.6 (7.5–14.5) [21]	15.5 (12.5–21) [40]	0.000
PWD-CO (L/min, median, IQR) [n]	5.9 (5–7.2)	5.5 (4.3–6.7)	0.068
MD (cm/min, median, IQR) [n]	2,075 (1,862–2,365)	1,822 (1,486–2,151)	0.003

LVEF, left ventricular ejection fraction; HR, heart rate; LVOT-VTI, left ventricular outflow tract velocity-time integral; SVI, stroke volume index; S′ LV, S′ left ventricle; LVOT, left ventricular outflow tract; LV-EDVI, left ventricular end-diastolic volume index; LV-ESVI left ventricular end-systolic volume index; PWD-CO, pulsed-wave Doppler cardiac output; MD, minute distance.

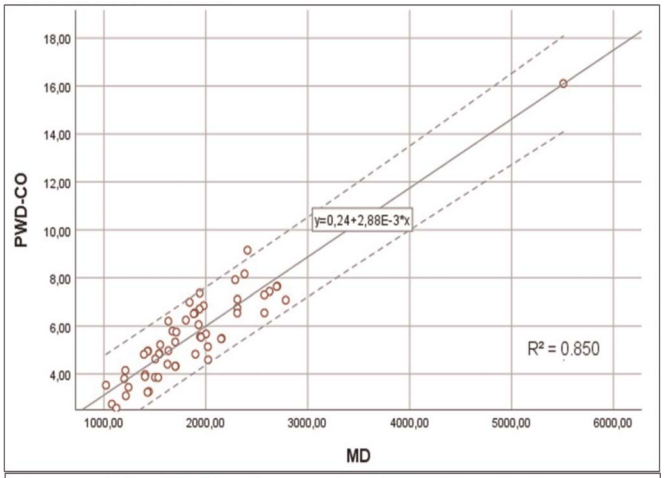

Fig. 6. **Correlation between PWD-CO and MD, with LVEF <60%.** Equation defined by lineal regression. PWD-CO indicates pulsed-wave Doppler cardiac output; MD, minute distance; LVEF, left ventricular ejection fraction.

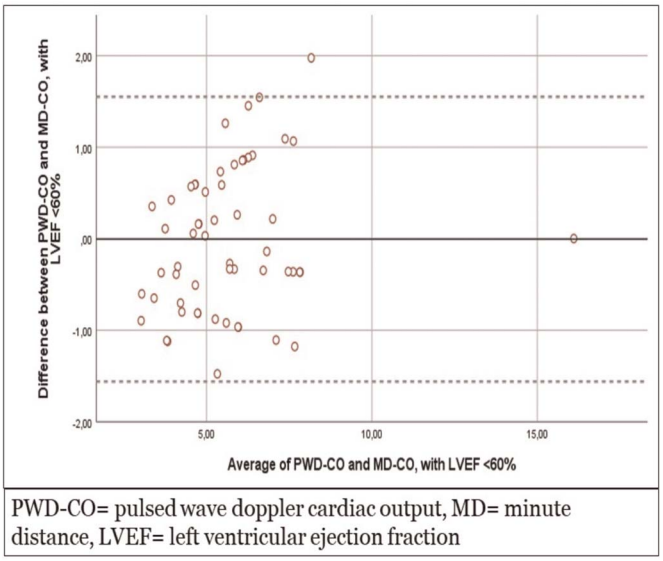

Fig. 8. **Bland-Altman between PWD-CO and MD, with LVEF <60%.** PWD-CO indicates pulsed-wave Doppler cardiac output; MD, minute distance; LVEF, left ventricular ejection fraction.

the inability to obtain high-quality images can reach more than 20% of cases; most of them attributed to factors such as mechanical ventilation, high positive end-expiratory pressure levels, weight gain, or the use of chest tubes and abdominal wall dressings (12,19). To address this problem, some studies have evaluated the use of transesophageal echocardiography as an alternative, but this technique could be limited at the point of care because of limited portability and perceived risk in nonventilated patients and, more importantly, because many intensive care teams currently do not have a probe for their own unrestricted use (24).

Considering that LVOTd is a static anatomical measurement related to body habitus, we could assume that any change in systolic volume would depend on LVOT-VTI and that any change in CO would depend on LVOT-VTI and HR. (25) Therefore, MD could be considered as an attractive, simple, and reliable method to assess CO. Accordingly, in our study, we confirmed a strong correlation between PWD-CO and MD. We also found that it was possible to extrapolate PWD-CO from MD with good agreement.

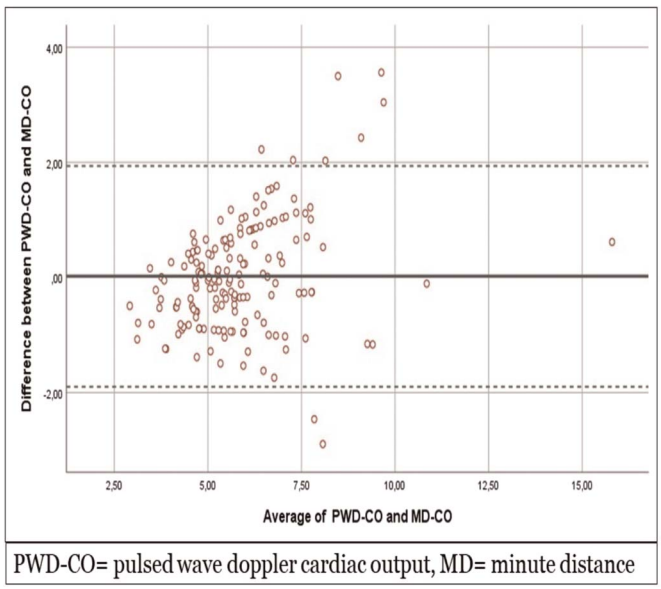

Fig. 7. **Correlation between PWD-CO and MD.** PWD-CO indicates pulsed-wave Doppler cardiac output; MD, minute distance.

Although, currently, there is not enough literature to indicate that MD calculated from aortic flow velocity is related to CO in critically ill patients, some studies have explored this tool with similar results to our study. Inatsugi et al. (26) compared MD with CO measured by thermodilution during cardiovascular surgery in 32 patients and found that MD calculated from pulmonary venous flow velocity tracings was highly related to CO. They also showed that the trend of changes in MD was very similar to that of CO. This suggests that MD is strongly related to CO and that changes in CO can be estimated from MD (26). In another study, Goldman et al. (27) found that MD could be accurately measured in patients with cardiac pathology rather than CO because it was independent of body size, in contrast to the dependence of PWD-CO on body surface area because of the measurement of LVOTd. Thus, MD could be proportional to CO, as long as the LVOT area and aortic flow remained constant (27). Conversely, in a report, Blancas et al. (28), found a weak correlation between LVOT-VTI and systolic volume index determined by thermodilution, and although these findings could be extrapolated to the correlation with MD, there were some factors that could affect their results, as they included patients with atrial fibrillation in 25% of cases. They also compared PWD-CO with two different thermodilution techniques, PAC and transpulmonary thermodilution, and observed significant differences in patient characteristics in terms of body surface area and sex. These findings may have had a significant impact on the results, as LVOTd and systolic volume index, unlike LVOT-VTI, are related to these factors, supporting the idea that MD is proportional to CO in the same conditions as LVOT-VTI (29,30).

Another finding of our study was that the correlation between PWD-CO and MD-CO improved in patients with LVEF <60%. We also found that those patients with LVEF <60% had significantly higher cardiac volumes and lower changes in these volumes

compared with patients with LVEF >60%. This may be explained by the large changes in ventricular and aortic diameters during the cardiac cycle in patients with hypovolemia, where there are relatively small ventricles compared with patients with normal or dilated ventricles. In fact, previous data showed that PWD-CO was more accurate in patients with ventricular enlargement in the setting of extensive myocardial infarction with reduced LVEF (20). In this regard, Maeder et al. (31) assessed patients with nondilated left ventricles and preserved LVEF and found that PWD-CO did not accurately reflect cardiac index when compared with Fick or thermodilution methods. Similar results were found in a study carried out in heart transplant recipients who had relatively normal sized left ventricles, showing worse agreement between PWD-CO and thermodilution-CO. These authors evaluated the LVOTd using three-dimensional echocardiography and found that the previous assumptions related to cylindrical geometry and laminar flow were inaccurate, the peak velocity was not laminar, and the LVOT area was elliptical rather than circular. In addition, the shape of the LVOTd was dynamic over the cardiac cycle and under different hemodynamic conditions (32). Consistent with these findings, some previous studies have shown that changes in arterial pressure can produce dynamic changes in the circumferential distensibility of the aortic diameter, which decreases as pressure increases (33). This could be an argument in favor of using MD as a parameter that avoids the errors of LVOTd measurement.

Our study has several limitations

The first and most important limitation is that we did not compare MD-CO with the standard thermodilution method for CO measurement as the PAC. However, the aim of our study was to evaluate a simple and sufficiently accurate method of assessing CO using PWD without measuring LVOTd compared with the usual method. Furthermore, previous studies have compared PWD-CO with standard thermodilution methods and demonstrated its accuracy in measuring CO in both stable and critically ill patients (7,18).

Second, we did not assess the number of patients who were excluded or the reason for exclusion. This was probably due to, among other things, the logistical limitations imposed by the urgency of containing the coronavirus disease 2019 pandemic. Therefore, we could not explain which cases were excluded because of an inability to obtain high-quality echocardiographic views or valvular regurgitation or other significant structural or functional abnormalities. However, we believe that previous studies provided a good indication of the proportion of patients that might affect the assessment of MD-CO. (10,11)

Third, we were unable to assess the LVEF in 50% of measurements, mainly because of difficulties in obtaining high quality images. This prevented better assessment of ventricular sizes or volemia states, which might have helped us better understand the reasons for the lower correlation observed in patients with higher LVEF.

Fourth, our study did not evaluate hemodynamic changes following a therapeutic setting, which could have affected our results. However, the CO measurements were performed simultaneously to avoid discrepancies between the values obtained.

CONCLUSIONS

In critically ill patients, MD measurement provides a simple and accurate estimate of CO, especially in patients with reduced or preserved LVEF.

Minute distance would allow earlier cardiovascular assessment in patients with circulatory failure, which is of particular interest in difficult clinical or technical conditions. It could also have a major impact on daily management by allowing serial noninvasive monitoring. However, further research is required to assess its impact on daily practice.

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