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REVIEW

Assessing aortic flow with doppler echocardiography in cardiogenic shock: A crucial diagnostic tool

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Abstract

Purpose: Cardiogenic shock still has a high mortality. In order to correctly manage these patients, it is useful to have available haemodynamic parameters, invasive and non-invasive. The aim of this review is to show the current evidence on the use of echocardiographic aortic flow assessment by left ventricular outflow tract - velocity time integral.

Methods: Publications relevant to the discussion of echocardiographic aortic flow assessment by left ventricular outflow tract - velocity time integral and cardiogenic shock, were retrieved from PubMed®.

Results: Left ventricular outflow tract - velocity time integral is an easily sampled and reproducible parameter that has already been shown to have prognostic value in various cardiovascular pathologies, including myocardial infarction and heart failure. Although there are still few data available in the literature, the LVOT-VTI also seems to have an important role in CS from prognosis to guidance in the escalation/de-escalation of vasoactive therapy and to support devices by allowing an estimate of patient's probability of response to fluid administration.

Conclusion: Aortic flow assessment can become a very useful invasive parameter in the management of cardiogenic shock.

KEYWORDS
cardiogenic shock, escalation/de-escalation, fluid responsiveness, LVOT-VTI, prognostic role

1 | INTRODUCTION

Cardiogenic shock (CS) is a syndrome characterized by tissue hypoperfusion due to cardiac causes, leading to multiple organ failure and, eventually, death. Mortality in a patient with CS is still very high, ranging from 30% to 60%, despite the better familiarity with the use of vasoactive drugs and the advent of new device to support the circulation. Its management is based on the immediate stabilization of hemodynamic parameters with the use of inotropes/vasopressors and possible escalation to mechanical circulatory supports (MCSs) in addition to the management of the cause responsible for the clinical setting. In order to correctly manage these patients, it is therefore useful to have available invasive and non-invasive hemodynamic parameters, which allow for an early identification of patients at greater risk of an adverse prognosis and for a serial monitoring in order to be able to carry out a correct escalation and de-escalation of pharmacological therapy/MCS.[1]

The left ventricular (LV) outflow tract (LVOT)-velocity time integral (VTI) (LVOT-VTI) is a readily available hemodynamic parameter obtained through Doppler echocardiography that can aid in risk stratification and management of patients with CS. It is easily sampled and can be used as a cardiac output (CO)/stroke volume (SV) surrogate for serious monitoring, although its accuracy may be reduced in some conditions that may be frequent in CS patients such as arrhythmias,

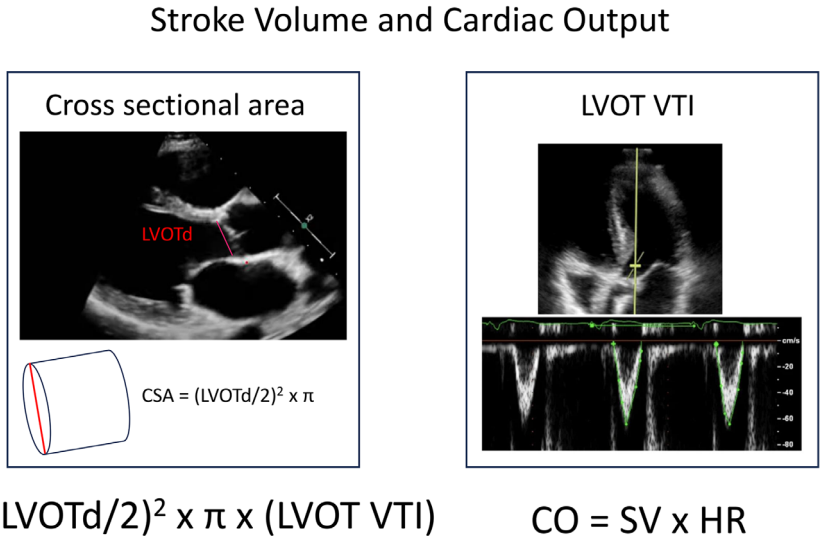

FIGURE 1 Echocardiographic quantification of SV and CO. CO, cardiac output; CSA, cross sectional area; HR, heart rate; LVOTd, diameter of the left ventricular outflow tract; LVOT-VTI, left ventricular outflow tract-velocity time integral; SV, stroke volume.

mechanical ventilation or severe aortic regurgitation (AR)/mitral regurgitation (MR).

The aim of this review is to show the current evidence on the use of LVOT-VTI in patients with CS.

2 | METHODS

We searched for clinical studies, reviews and meta-analysis in MEDLINE/Pubmed. We used the keywords and headings of the medical topic "cardiogenic shock", "LVOT-VTI", "Doppler-Echocardiography", "vasoactive drugs", "LVOT diameter", "mechanical ventilation", and additional text words (such as abbreviations). We also searched through the bibliography of identified studies and meta-analysis.

3 | ROLE OF LVOT-VTI IN CO AND SV MEASUREMENT

Transthoracic echocardiography (TTE) has emerged a valuable tool to identify the etiology of CS and its management. It allows non-invasive, bedside, low-cost, repeatable, and widely available hemodynamic monitoring of flow parameters such as SV and CO, essential to assess organ perfusion and oxygen distribution.[2]

3.1 | Method of sampling

Doppler derived CO is typically obtained by measuring flow across the LVOT which is determined by the VTI of the Doppler signal directed across the aortic valve, multiplied by the cross sectional area (CSA) of the LVOT and heart rate (HR) (Figure 1).

In more detail, to calculate the CSA it is important to first obtain an accurate measurement of the diameter of LVOT (LVOTd). Current guidelines recommend measuring the LVOTd in the zooming long parasternal axis view in the mid-systole from inner edge to inner edge.[3] The exact measurement location is debated: some experts recommend that LVOTd should be calculated 3–10 mm below the annulus level, while others support annulus-level measurement.[3] The rationale for measuring 3–10 mm below the annulus is derived from the fact that, for the continuity equation to be accurate, the volume of the pulsed wave Doppler (PWD) sample must be in the identical anatomical plane in which the CSA is calculated. Once the LVOTd has been calculated, the CSA is obtained from the formula $(LVOTd/2)^2 \times \pi$. As regards LVOT-VTI sampling, in an apical five-chamber view, the PWD should be placed just proximal to the aortic valve with the PWD interrogation line that should be parallel to the blood flow within the LVOT (if the angle of insonation is within 20°, the error from the true Doppler shift is about 6%). PWD should then be carefully moved into the LVOT (approximately at 1 cm distance to the aortic valve) so that LVOT-VTI is represented by an envelope, which is "dark" inside with a "light" outline. It's important to be as precise as possible and approximate the outer edge as close as possible when drawing the curve.

3.2 | Correlation between invasive and non-invasive CO sampling

Multiple studies have demonstrated a close correlation between CO calculated by Doppler echocardiography and invasive thermodilution and Fick methods.[4,5] Gola et al. showed that Doppler echocardiography underestimated CO measured with Fick's method only of 0.22 L/min in patients with chronic heart failure (HF) and the correlation between Fick and Doppler echocardiography measurements of CO was satisfactory also in subgroups with severe tricuspid regurgitation, and with low CO.[4] Similarly, Davies et al. demonstrated that Doppler-derived CO compares well with traditional methods of measurement (Fick and thermodilution). Indeed, linear regression analysis

for Doppler against thermodilution provided better correlation (coefficient (*r*) of 0.81, $p < 0.002$, for Doppler and *r* 0.76, $p < 0.02$, for Fick method).[6] These data suggest that the Doppler echocardiographic method for measuring CO may represent a valid alternative to invasive methods.

3.3 | Advantage of using only LVOT-VTI in CO sampling

According to the formula used to calculate the LVOT-CSA, any measurement error in the LVOTd will be squared; therefore, there may be a large error in the calculation of the SV even for minimal errors in the calculation of the LVOTd.[7,8] This can occur more easily in some anatomic conditions such as in the presence of a calcification of the annulus in which the inner edge is not always well recognizable or in the case when the echocardiographic acoustic window is not optimal. In addition, the LVOT-CSA has a non-circular elliptical shape. Consequently, the LVOT area is underestimated with the 2D method compared to the 3D planimetric area. This results in a CO of about 10% lower with the 2D method.[9] In order to avoid this limitation, since LVOTd is constant, it is possible not to measure it repeatedly but only at baseline, thus using the same LVOT-CSA for the serial estimates of SV and CO. Another solution to the possible error in the calculation of LVOTd is to use only LVOT-VTI, a reliable surrogate for CO in the absence of abnormalities in the LV outflow.[10] In a study that evaluated CO measured using only LVOT-VTI and HR, rather than the classic method, there was a significant correlation between the two methods, in particular when LV ejection fraction (LVEF) was less than 60% ($R^2 = .85, p < .05$).[11] Moreover, a study demonstrated that Doppler-TTE variables, including VTI, derived from the LV assist device (LVAD) outflow cannula can reliably predict CO in patients supported with LVADs and may mitigate the need for invasive testing also in these patients.[12]

However, in these cases, it is recommended to average at least five LVOT-VTI values in order to obtain a more accurate VTI value.[2] As reference values, Goldman et al. showed that when HR is within the normal range, the average LVOT-VTI values are about 20 ± 3 cm. When HR is below 55 bpm, the LVOT-VTI values should be above 18 cm; otherwise, the presence of a low SV and CO must be considered; finally, when the HR is above 95 bpm, the LVOT-VTI values must be less than 22 cm; otherwise, a high SV and CO are suggested.[13] Analyzing baseline LVOT-VTI values by gender, women tend to have a higher baseline value than men.[14] Intra-observer and inter-observer analysis showed very good reproducibility of its assessment.[14]

4 | ROLE OF LVOT-VTI IN CARDIOVASCULAR DISEASES

Studies have evaluated LVOT-VTI in different diseases, especially from a prognostic perspective in acute setting (Table 1).

Jentzer et al. analyzed 6957 patients admitted to the cardiac intensive care unit (CICU) for acute coronary syndrome in 54.3% and HF in 50.0%. The strongest TTE predictor of hospital mortality was LVOT-VTI and a value <16 cm showed the best accuracy.[15]

When analyzing only the acute myocardial infarction setting, mortality rate at 1 month and 5 years were 18% and 43% respectively when PWD assessment was less than 65% predicted.[16]

In the acute HF setting, LVOT-VTI was associated with long-term mortality, independently of other known TTE prognostic factors in a cohort of 350 elderly patients.[17] It is very important to point out that in this study LVEF was associated with mortality only when LVOT-VTI was excluded from the analysis, highlighting a superiority of this value in prognostic stratification as compared to LVEF. Further evidence has been shown in a cohort of 100 patients with advanced HF, where the occurrence of death and LVAD implantation was associated with a lower LVOT-VTI, defined below 8.1 cm, but not LVEF or CO and remained statistically significant also in multivariate analysis.[18] In fact, in an acute HF setting, LVEF may have important limitations, including its dependence on volemic load and overestimation in patients with MR, a common finding in these patients. In contrast, LVOT-VTI can provide more accurate information about LV systolic function by reflecting the LV ejection into the aorta and, thus, representing a more reliable parameter than the global LVEF.[19] As proof of this, a trial demonstrated that LV anterior output improves outcome prediction in patients with HF and low LVEF in presence of secondary MR, and its variation pre- and post- percutaneous mitral valve repair could further help in the prognostic stratification of such patients.[20]

Not only, LVOT-VTI upon admission could be useful for better risk stratification in hospitalized patients with HF with preserved ejection fraction by identifying patients with low flow.[21]

In patients with acute pulmonary embolism (PE), this Doppler echocardiographic parameter could be a signal of right ventricular (RV) dysfunction, and therefore a predictor of poor outcomes. In fact, low LVOT-VTI (≤15 cm) was associated with in-hospital death or cardiac arrest and shock or need for reperfusion in a multivariable model.[22] Its sampling could be important in patients with PE at intermediate-high risk, an area that is still grey as fibrinolysis is currently not indicated in all patients but in whom there may be clinical deterioration associated with death.[23] The presence of a parameter that allows immediate monitoring and at the same time an indicator of negative prognosis can in fact identify patients who could for example benefit from percutaneous thrombolysis strategies by means of devices.

Finally, LVOT-VTI is part of the evaluation of the severity of aortic stenosis and its pre-operative value can also be used for prognostic purposes.[24] In this work, a prognostic model including height, chronic lung disease, STS score, preoperative LVEF, age, and precisely preoperative LVOT-VTI are the variables related to 30-day mortality. To evaluate its possible role in identifying patients with a worse prognosis who could benefit from a transcatheter or surgical aortic valve replacement procedure although still asymptomatic.

TABLE 1 evidence on the prognostic role of LVOT-VTI in cardiovascular diseases.

Trial	Type of patients	Number of patients	LVOT-VTI cut-off	Results
Jentzer et al.[15]	CICU	6957	<16 cm	LVOT-VTI was the strongest TTE predictors of hospital mortality (adjusted OR .912 per 1 cm higher, 95% CI .883–.942, $p < .0001$)
Trent et al.[16]	AMI	378	/	LVOT-VTI was independently related to mortality within 1 month ($p < .0001$) and 7 years ($p < .0001$) in multivariate logistic regression analysis
Zhong et al.[17]	Acute HF	350	≤15.7 cm	LVOT-VTI > 15.7 cm was associated with 45% lower risk for death during 5-years of follow-up (age-adjusted HR .55, 95% CI .42–.72, $p < .0001$)
Tan et al.[18]	Advanced HF	100	<8.1 cm	A lower LVOT-VTI was associated with death and LVAD implantation in multivariate analysis ($p = .003$). Survival analysis by LVOT-VTI tertile demonstrated an unadjusted HR of 4.755 (95% CI 1.576–14.348, $p = .006$) for combined LVAD and mortality at 1-year compared to patients with LVOT-VTI between 8.1 and 10 cm
Omote et al.[21]	Acute HF with preserved LVEF	214	<15.8 cm	Lower LVOT-VTI was significantly associated with all-cause death and readmission due to HF compared with higher LVOT-VTI ($p = .005$). At multivariable Cox regression analyses lower LVOT-VTI was an independent determinant all-cause death and readmission due to HF (HR .94, 95% CI .91–.98).
Gentile et al.[20]	HF and secondary MR	287	≤17 cm	Patients with an LVOT-VTI ≤17 cm showed the greatest risk of cardiac death (log rank 44.3, $p < .001$) and all-cause mortality (log rank 8.6, $p = .003$)
Yuriditsky et al.[22]	Pulmonary embolism	188	≤15 cm	In a multivariable model, LVOT-VTI ≤ 15 remained significant for death or cardiac arrest (OR 3.48, 95% CI 1.02, 11.9; $p = .047$) and for shock or need for reperfusion (OR 8.12, 95% CI 1.62, 40.66; $p = .011$). Among intermediate-high-risk patients, low LVOT-VTI was the only variable associated with the composite outcome of death, cardiac arrest, shock, or need for reperfusion (OR 14, 95% CI 1.7, 118.4; $p = .015$).
Prosperi-Porta et al.[23]	Pulmonary embolism	665	/	LVOT-VTI was lower in intermediate-high risk patients with related cardiopulmonary death or cardiopulmonary decompensation ($p < .001$). At Univariate Logistic Regression analysis OR per 1 cm decrease was 1.50 (1.29–1.74, $p < .001$)
Lertsanguansinchai et al.[24]	Severe aortic stenosis	186	/	A prediction model that includes height, chronic lung disease, STS score, preoperative LVEF, age, and preoperative LVOT-VTI is correlated to 30-day mortality

Abbreviations: AMI, acute myocardial infarction; CI, confidence interval; CICU, cardiac intensive care unit; HF, heart failure; HR, hazard ratio; LVAD, left ventricular assist device; LVEF, left ventricular ejection fraction, LVOT-VTI, left ventricular outflow tract-velocity time; MR, mitral regurgitation; OR, odds ratio; STS, society of thoracic surgeons; TTE, transthoracic echocardiography.

5 | ROLE OF LVOT-VTI IN CARDIOGENIC SHOCK

There is very little data available from clinical studies concerning the role of LVOT-VTI in CS. Below is summarized its role in three scenarios (Figure 2).

5.1 | Fluid responsiveness

An important goal in the treatment of patients in a critical condition is the administration of intravenous fluids for initial resuscitation avoiding volume overload that has been associated with higher mortality.[25] Previous studies in non-CS patients showed that a variation of this Doppler echocardiographic parameter >12%–15% after volemic load or passive leg raise is able to predict with a good diagnostic performance the fluid responsiveness of those patients.[26–28]

Unlike in some settings, for example septic shock or in post-cardiac surgery patients, in which fluid replacement has become a routine procedure, this strategy for resuscitation in CS patients remains a point of debate in clinical practice. In fact, in this setting, the application of fluid therapy needs a balance between achieving sufficient volume for peripheral perfusion and avoiding fluid overload because excessive fluid administration can lead microcirculatory dysfunction due to edema formation. Moreover, in CS, many patients may be mechanically ventilated. Changes in right atrial pressure are the main factor determining cyclic changes in venous return during mechanical ventilation. In fact, the application of a positive pressure increases the right atrial pressure reducing venous return. However, the increase in pressure even at the intra-abdominal level can partially or totally counterbalance the increase in right atrial pressure by mobilizing an amount of venous blood in the capacitance vessels.[29] This will result in a LVOT-VTI variation that may limit the assessment of fluid responsiveness. In a systematic review of 11 studies including 406 CS patients,[30] fluid therapy played a crucial role in CS management but necessitates integration into an appropriate treatment strategy, accounting for individual circumstances, comorbidities, and etiology.

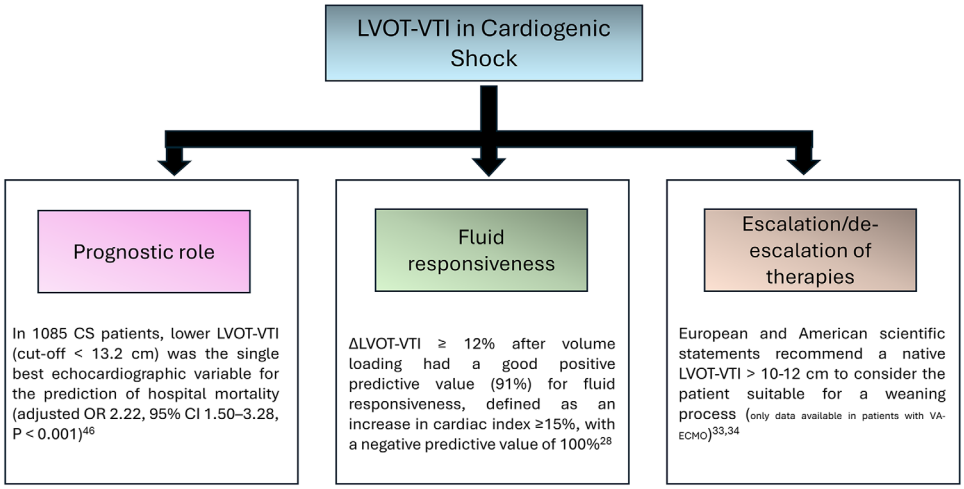

FIGURE 2 Three scenarios in which LVOT-VTI can be used in patients with CS. CI, confidence interval; CS, cardiogenic shock; LVOT-VTI, left ventricular outflow tract-velocity time integral; OR, odds ratio; VA-ECMO, venoarterial extracorporeal membrane oxygenation.

Currently, the European Society of Cardiology (ESC) and the American Heart Association (AHA) recommend considering a fluid challenge as a first-line treatment for CS, provided there are no signs of fluid overload.[31,32] Therefore, the monitoring of such hemodynamic parameter by echocardiography emerges as an important component of patient management, allowing for the optimization of volume status, myocardial contractility, and tissue perfusion while helping to avoid fluid overload.

5.2 | Role in escalation/de-escalation of inotropes/vasopressor and MCSs

The ideal treatment of CS should be a multistep approach based on hemodynamics and metabolic characteristics of patients going through a first phase of "rescue", in which the goal is to maintain adequate tissue perfusion, a subsequent phase of treatment "optimization", possibly combining vasoactive drugs and MCS, and a final phase of "weaning" with the goal of removing drugs and MCS once recovery has taken place.[1]

At the moment, the evidence on the role of LVOT-VTI concerns the weaning phase. In fact, in a clinical consensus statement of the Association for Acute Cardio Vascular Care of the ESC, the European Society of Intensive Care Medicine, the European branch of the Extracorporeal Life Support Organization, and the European Association for Cardio-Thoracic Surgery about step by step daily management of short-term MCS for CS[33] and in a scientific statement from the AHA about escalating and de-escalating temporary MCS in CS,[34] the use of LVOT-VTI is only mentioned in the weaning process by MCSs reporting a LVOT-VTI cut-off of 10−12 cm to consider patients suitable for a weaning process. It is important to highlight that this recommendation is based only on data available in patients with venoarterial extracorporeal membrane oxygenation (VA-ECMO).[35–37] In fact, among patients on VA-ECMO successfully weaned during follow-up, the most widely used parameters to track LV recovery were a higher Doppler echocardiographic measures at the time of weaning, reflecting a better SV. The most commonly reported threshold to predict successful weaning was >9.5 cm. To underline the importance of ventricular recovery, an important role in predicting successful weaning was also played by the VTI ratio from cannulation to weaning attempt.[35]

Since the purpose of using LVOT-VTI in this context is to assess the presence of myocardial recovery, it can be assumed that these data can be extrapolated and also applied to the de-escalation of other MCS such as Impella and intra-aortic balloon pump (IABP) but also to the weaning phase from vasoactive therapy. Future clinical studies will need to evaluate this topic. It will also be important to consider the possible role of VTI in evaluating cardiac recovery in the correct timing of weaning from mechanical ventilation or non-invasive ventilation to avoid weaning-induced pulmonary edema by increased RV preload and LV afterload during the transition from positive pressure to negative pressure.

Use in the escalation phase is based on a different concept, evaluate its increase in response to titration of vasoactive therapy or MCS as an indicator of increased perfusion. Indeed, in the literature its correlation with tissue perfusion is well known. In fact, it showed the stronger association with hypotension/hypoperfusion in 5375 patients admitted to CICU.[38] Similarly, Chinen et al. analyzed 212 patients admitted for acute decompensated HF. Low LVOT-VTI (≤15 cm) patients showed a higher prevalence and low-output syndrome signs, such as pulsus alternans (17.7% vs. 8.1%, $p = .0006$), cold ends (26.6% vs. 9.1%, $p = .0012$) and proportional pulse pressure <25% (17.7% vs. 8.1%, $p = .0436$), suggesting the importance of PWD measurements to assess low CO. Of note, these patients presented high inotropic administration (37.2% vs. 14.1%, $p = .0002$).[39] Lastly, a LVOT-VTI < 15 cm was strongly correlated with a higher stroke index, a well-known hemodynamic parameter in critical care medicine, calculated by dividing the HR by the systolic blood pressure (SBP), that can be used to identify CICU patients with unfavorable hemodynamics (in case of value above 1) who may require aggressive treatment.[40]

There is no direct evidence at the moment in patients with CS. However, Zarragoikoetxea et al. found that in patients with low CO syndrome, LVOT-VTI showed a marked improvement after dobutamine administration (~37% increase).[41] Similarly, although in patients with septic shock, LVOT-VTI increased from 18 to 20 cm, during norepinephrine infusion, result that could also be valid for patients in CS with arterial hypotension and with mixed cardiogenic and septic shock.[42] Other data to support the LVOT-VTI also in the escalation process comes from a study of Xu et al.[43] This study assessed the regional hemodynamic effect of IABP on patients undergoing VA-ECMO by ultrasonography and indicated that IABP was associated with increased LVOT-VTI.

It will therefore be important, in the years to come, to have available data also in the setting of the CS, not only on the use of the MCS but also of the vasoactive drugs. As in evaluating the patient's fluid responsiveness, it could in fact be an immediate parameter of the patient's response with CS to treatment both in the rescue phase and in that of hemodynamic optimization. It will also be important to decide whether to consider a patient responsive to treatment for any variation in absolute value or to use a variation cut-off as for the fluid challenge.

5.3 | Prognostic role

Previous studies have shown a consistent association between invasively measured hemodynamic variables such as cardiac index or cardiac power output and mortality in patients with CS. As mentioned above, these systemic hemodynamic variables are calculated from the LVOT-VTI, which itself could be the best predictor of mortality, without the need for complex calculations. The Society for Cardiovascular Angiography and Interventions (SCAI) shock stages classification stratifies mortality risk in CICU patients.[44] Higher SCAI shock stages were associated with lower LVEF and worse non-invasive hemodynamic parameters reflecting forward flow, including LVOT-VTI values ($p < .001$).[45] Jentzer et al. retrospective analyzed 1085 patients with an admission diagnosis of CS and a TTE within 1 day of admission.[46] Lower LVOT-VTI (cut-off < 13.2 cm) was the single best echocardiographic variable for the prediction of hospital mortality (adjusted OR 2.22, 95% CI 1.50–3.28, $p < .001$). In fact, patients with an LVOT-VTI < 13.2 cm had higher hospital mortality in each LVEF group, although this was not significant in patients with severe LV systolic disfunction ($p = .07$). Another study analyzed the role of LVTO-VTI values according to the characteristics of ventricular dysfunction. Patients with RV dysfunction had the highest mean values (18.9 ± 5.1 cm), patients with isolated LVEF < 40% had intermediate values (16.8 ± 4.1 cm), while lower LVOT-VTI values were found in patients with biventricular dysfunction (14.1 ± 4.4 cm). Of note, only biventricular dysfunction, associated with lower LVOT-VTI values, was associated with in-hospital mortality (adjusted HR 1.815; 95% CI, 1.237–2.663; $p = .0023$).[47]

6 | LIMITS OF LVOT-VTI AS SURROGATE FOR SV

Although we have seen that LVOT-VTI is a simple parameter to sample, reproducible and which can be used as an excellent surrogate for SV and CO in various clinical settings, this condition fails in some contexts (Table 2). A correct sampling of LVOT-VTI might not always be immediate in case of difficulties to a correct alignment or in patients without optimal echocardiographic acoustic window, for example in cases of obesity or lung disease. In addition, patients are often in supine decubitus obligated, which further limits the accuracy of echocardiographic examination. A common issue for an inaccurate LVOT-VTI determination is the presence of arrhythmias, especially atrial fibrillation and frequent extrasystoles, since different filling times result in beat-to-beat VTI variability. Sampling of at least five consecutive beats is recommended in these cases.[48] Moreover, the LVOT-VTI is not reliable for estimating SV/CO when there is moderate to severe AR because the increased LV end-diastolic volume leads to a supranormal, overestimated LVOT-VTI value. Possibility of overestimation of LVOT-VTI value could be also in states lead to a high CO at baseline such as cirrhosis, thyrotoxicosis, anemia, pregnancy, and arterio-venous fistulae. The opposite condition is instead the presence of more than moderate MR, associated with a reduction in forward SV and therefore with a underestimation of LVOT-VTI. Another condition where LVOT-VTI is not reliable for estimating SV/CO is subaortic obstruction (LVOTO), typical in extreme cases hypovolemia, asymmetric hypertrophy of the LV septum, anterior myocardial infarctions with compensatory hyperdynamic basal segments of the interventricular septum and in Takotsubo syndrome.[2] Instead, in the setting of a LVOTO, the velocity of blood flow leads to a phenomenon known as aliasing. In this condition, the ultrasound machine is unable to determine the true direction and velocity of blood flow across the area being sampled, and it is represented by a spectral envelope that cannot be reliably traced.[48]

TABLE 2 Conditions under which LVOT-VTI may be less accurate.

Settings
Acquisition errors (poor image quality or incorrect alignment)
Moderate to severe aortic regurgitation
Moderate to severe mitral regurgitation
Fixed sub-aortic obstruction (ex. sub-valvular stenosis)
Dynamic sub-aortic obstruction (ex. Takotsubo syndrome)
Hyperdynamic circle (ex. cirrhosis, thyrotoxicosis, anemia, pregnancy and arterio-venous fistula)
Arrhythmias (ex. Atrial fibrillation or frequent extrasystoles)
Mechanical ventilation

Abbreviation: LVOT-VTI, left ventricular outflow tract-velocity time integral.

Finally, as previously mentioned, a patient with CS in CICU could need of mechanical ventilation. Wang et al. performed a study to evaluate which TTE parameters could be more affect with mechanical ventilation. There were significantly fewer patients in the study group who had optimal images acquisitions for parameter assessments with M-mode method, PWD method and endocardium-tracing method, including LVOT VTI (37.8% vs. 85.6%, $p < .001$).[49] In addition, we have seen how in this situation it is difficult to assess the reactivity of the fluid for cyclic changes in venous return induced by positive pressure. More importantly, in a study that compared the correlation between

LVOT-VTI and SV index calculated by pulmonary artery catheter or a Pulse index Contour Cardiac Output (PiCCO®), there was poor correlation, especially with higher LVOT-VTI values.[50] Further testing will be necessary, but if this data is confirmed, it may be an important limitation for LVOT-VTI use in CICU. Therefore, currently, the advice in the ventilated patient is to use it carefully and not as the only decision parameter.

7 | CONCLUSION

LVOT-VTI is an easily sampled and reproducible parameter on TTE that has already been shown to have prognostic value in various CV pathologies, including myocardial infarction and HF. Although there are still few data available in the literature, the LVOT-VTI also seems to have an important role in CS from prognosis to guidance in the escalation/de-escalation of vasoactive therapy and to support devices by allowing an estimate of patient's probability of response to fluid administration. Further data will be necessary to confirm these initial findings and to improve patients management by using non-invasive echocardiographic parameters.

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The authors have nothing to report.

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