Comparison of continous-wave Doppler ultrasound monitor and echocardiography to assess cardiac output in intensive care patients

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ABSTRACT

Background: Continuous-wave Doppler (CWD) ultrasound through the left ventricular outflow tract is one modality used for non-invasive cardiac output measurement. The ultrasonic cardiac output monitor (USCOM) is a relatively new monitor which uses a small, transcutaneous ultrasound probe to measure cardiac output with CWD via the suprasternal window. It is faster and less complex to train new users than conventional echocardiography. In addition to stroke volume (SV), the USCOM can calculate stroke volume variation (SVV) and the Smith–Madigan inotropy index (SMII), which is an estimate of the pre-load independent contractility of the myocardium.

Objective: To assess the level of agreement between cardiac output measured with conventional echocardiography and with USCOM.

Methods: A prospective, observational, multicentre trial of patients admitted to the intensive care units of two hospitals. After excluding patients with aortic stenosis, any patient undergoing a clinically indicated echocardiogram also underwent a subsequent USCOM study for comparison.

Results: We enrolled 121 patients in the study, with aortic stenosis the main reason for patient exclusion. Of the study patients, 63% were mechanically ventilated, 84% were in sinus rhythm, and the mean age of the study cohort was 66 years (SD, 17 years). There was a very strong correlation between SV as measured by the USCOM and by echocardiography. The mean difference in SV was 0.33 mL (SD, 5.62 mL), $r^2 = 0.956$, and Bland–Altman analysis confirmed no significant bias with acceptable limits of agreement between the methods. Patients who were fluid responsive had an SVV cut point on the receiver operating characteristic curve of 21%, and sensitivity and specificity of 95%. A low SMII (< 1.1 watts/m^2) calculated with the USCOM did not correlate well with low cardiac output status, with a sensitivity of only 69%.

Conclusions: SV (and thus cardiac output) measured using the USCOM correlated well with echocardiographic cardiac output measurement, which suggests that the USCOM could be a valuable haemodynamic tool for assessment of cardiac output and fluid responsiveness in critically ill patients if patients with aortic stenosis are excluded. Inotropy, as a parameter of low cardiac output, was not useful in this cohort of patients.

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Echocardiography is commonly used in critically ill patients for haemodynamic assessment, and there are multiple emerging invasive and non-invasive bedside haemodynamic monitors available for the intensive care unit. In the 21st century, formal echocardiography is accepted as a gold standard for assessment of cardiac output and cardiac function,[1] but is time consuming and relies on the availability of appropriately trained personnel. Echocardiography provides reliable non-invasive information in real time for making critical decisions, such as about fluid management, vasoactive or inotropic drugs, and the need for pericardiocentesis.[2,3]

Stroke volume (SV) is assessed echocardiographically by first measuring the diameter (and thus the radius) of the left ventricular outflow tract (LVOT) in the parasternal long axis window. Then flow is measured with pulse-wave (PW) Doppler ultrasound of LVOT in the apical five-chamber view, and the velocity–time integral (Vti) is calculated. SV is calculated from the equation:

$$SV = \pi r^2 \times Vti\ (LVOT)$$

in which r = radius of the LVOT (in cm), calculating the SV in millilitres.[4] Cardiac output is calculated by multiplying SV by heart rate.

Echocardiography can be used to assess volume status by measuring the inferior vena cava (IVC) diameters and looking for respiratory variation in patients who are predicted to be fluid responsive. An IVC collapsibility index [(maximum diameter – minimum diameter) ÷ maximum diameter] × 100 that exceeds 40% in spontaneously breathing patients, and an IVC distensibility index [(maximum diameter – minimum diameter) ÷ minimum diameter] × 100 that exceeds 15%, predicts fluid responsiveness in mechanically ventilated patients with a sensitivity and specificity of 90%.[5,6,7] PW Doppler scan of LVOT can be used in mechanically ventilated patients in sinus rhythm to assess respiratory changes in SV and predict fluid responsiveness. A variation of more than 12%–17% predicts a positive response to fluid.[8]

The ultrasonic cardiac output monitor (USCOM) (Uscom Ltd) is a relatively new monitor based on the same suprasternal continuous-wave (CW) Doppler principles, and therefore measures the highest velocity in its directed path, which is across the valve (see uscom.com.au). The CW Doppler transducer is positioned suprasternally and the operator directs the beam progressively in three dimensions, with the aim of achieving the highest velocity aligned to aortic valve flow. Aortic valve annulus diameter is assumed from a validated nomogram based on height and weight.[9] The Doppler ejection waveform is automatically traced to calculate VTi, and thus SV, after multiplication by the assumed annulus area. Beat-to-beat stroke volume variation (SVV) is also measured. The main difference between echocardiography and the USCOM is that the USCOM uses CW (without 2-D) and echocardiography uses PW (with 2-D assist). Variability of the PW sample volume positioning is eliminated in the USCOM method by using CW Doppler scan and the 25% variability of the VTi is reduced to 5%–10%.

The operator uses the three-dimensional process to search for the smaller Vti of the aorta and then manipulates the transducer further to fulfil the higher and wider Vti of the aortic valve flow. The only way the USCOM differentiates between the LVOT, aortic valve and ascending aorta is through the operator getting the highest velocity with the best Doppler waveform.

The Smith–Madigan inotropy index (SMII), labelled inotropy in the USCOM method, is derived from an algorithm to estimate the length-independent myocardial contractility, such that an increase in SMII leads to an increase in cardiac output, irrespective of pre-load. The USCOM measures SMII through complex equations which summate potential energy and kinetic energy produced by the heart, then index it to body surface area (measured in watts/m^2).[10]

An USCOM examination takes 2 minutes.[11] Small studies have shown that USCOM data in adult and paediatric populations are consistent with echocardiography and correlate with direct catheter measurements in animals.[12-15] Studies of training in the use of the USCOM suggest that proficiency can be achieved in 2–4 days.[16] The USCOM has been used as a haemodynamic monitor to aid management in small trials,[17,18] but its measurements have not been validated in larger trials against echocardiography performed by trained, accredited sonographers.[19]

Methods

Our aim was to evaluate the agreement between the USCOM and echocardiographically derived haemodynamic measurements. Our objectives were to assess:

- agreement between SV as measured by the USCOM and by echocardiography
- agreement between Vti and peak velocity (Vpk) as measured by the USCOM and by echocardiography
- the relationship and cut point between the USCOM SVV and clinical fluid responsiveness
- the relationship and cut points between the SMII and cardiac output in a subset of patients with low cardiac output (defined as a left ventricular ejection fraction of < 35%) and low cardiac output on echocardiography.

This was a prospective, observational, multicentre study in two tertiary ICUs. All patients for whom a conventional echocardiogram was requested for clinical reasons were eligible. The patient or person responsible consented to a USCOM examination by a different operator, and the USCOM operator was blinded to the echocardiogram result. Full diagnostic echocardiograms were performed by an accredited sonographer according to the American Society of Echocardiography guidelines. Patients were excluded if they refused consent for the additional examination, if they had a history of congenital heart disease or any degree of aortic stenosis (AS), or if the echocardiogram identified previously undiagnosed AS (of at least mild severity, based on valve area or median velocity). The study was approved by the human research ethics committee of Nepean Blue Mountains Local Health District.

For our study, the LVOT diameter was measured three times and the largest reading stored, then LVOT Vti was measured by PW Doppler scan in the apical five-chamber view (or the three-chamber view if the five-chamber view was not possible). The IVC maximum and minimum diameters were measured for spontaneously breathing patients and for deep-sedated mechanically ventilated patients, in the subcostal view with tidal volume set to 8mL/kg.

Immediately after the echocardiogram, an USCOM study was done by the principal investigator. Using the transducer placed in the suprasternal notch, the best Doppler flow profile was recorded through the aortic valve. Three measurements were performed and the average of the three readings was used. The SMII on the USCOM was also recorded, and was analysed in the subset of patients for whom the echocardiographer recorded a low cardiac output state.

Patients who, for clinical reasons, received a fluid bolus within 1 hour of the USCOM study had another USCOM study after receiving the fluid bolus to assess fluid responsiveness. Additional USCOM measurements were obtained before and after the fluid bolus was administered, up to 48 hours after enrolment, and SVV was calculated. Fluid responsiveness was defined as an increase in SV of > 15% after the patient had received 250–500 mL of crystalloid fluid over 10–15 minutes.[20]

Sample size and statistics

Assuming an alpha error of < 5% and a beta error of < 20% for 80% power, the minimum number of subjects required is 50 + 8*m*, where *m* is the number of independent variables. With six USCOM variables and a margin for possible technical difficulties, we planned to recruit 120 patients into the study.

We performed statistical analysis using correlation analysis of the Pearson product–moment correlation coefficient for parametric data, and Bland–Altman analysis to rule out bias.[21] We used multiple regression analysis for continuous dependent variables, and logistic regression for categorical or binary (dichotomous) dependent variables.

Results

We screened 135 patients and included 121 in the study. The reasons for exclusion were aortic stenosis (six patients), refused consent (two patients), unable to get accurate LVOT measurements on echocardiography (four patients) and unable to acquire adequate USCOM tracing (two patients).

Demographic data are shown in Table 1. A total of 63% of our patients were mechanically ventilated, and 84% were in sinus rhythm. The mean age of the study cohort was 66.5 years (SD, 17.2 years) and the most common admitting diagnoses were septic and cardiogenic shock.

There was no significant difference between SV as measured with the USCOM and with echocardiography (P = 0.52), with the mean difference in SV between the USCOM and echocardiography measurements being only 0.33 mL (SD, 5.62 mL). There was minimal bias on Bland–Altman analysis and acceptable limits of agreement (Figure 1 and Table 2).

LVOT diameter measured with echocardiography correlated well with the aortic valve diameter estimated from the nomogram in the USCOM machine, with the mean difference measured at only 0.014 mm (SD, 0.13 mm) (Figure 2 and Table 3).

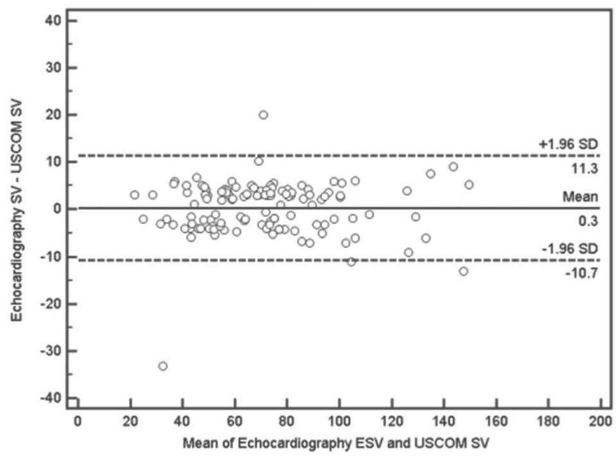
Aortic valve Vpk measured by the USCOM and measured by echocardiography of the LVOT were similar (P = 0.056; r^2 = 0.874). Neither USCOM Vpk nor echocardiography LVOT Vpk correlated with echocardiographic aortic valve Vpk (Tables 4–6 and Figures 3–5). Vti also showed good correlation between USCOM and echocardiographic measurements, with unbiased Bland–Altman curve. (Figure 6 and Table 7)

There was no statistical agreement between SVV > 10% (cut point used in pulse index continuous cardiac output [PiCCO] and FloTrac cardiac output monitors) or SVV > 15% and fluid responsiveness. Assessment of the receiver operating characteristic (ROC) curve for SVV and fluid responsiveness showed that an SVV of > 21% showed very good correlation with fluid responsiveness, with specificity and sensitivity of 95% (Figure 7 [online at cicm.org.au/Resources/Publications/Journal] and Tables 8 and 9).

Table 1. Demographic data of the study cohort

Variable	Frequency (n = 121)	Proportion (%)
Sex		
Male	73	60.3%
Female	48	39.7%
Mechanical ventilation		
Yes	77	63.6%
No	44	36.4%
Rhythm		
Sinus rhythm	101	84.2%
Atrial fibrillation	18	15.0%
Third degree atrioventricular block	2	0.8%
Admitting diagnosis		
Cardiac	56	46.2%
Respiratory	30	24.8%
Septic shock	26	21.5%
Renal	24	19.8%
Surgical	13	10.8%
Other	10	8.3%

Figure 1. Bland–Altman graph for SV with USCOM and echocardiography



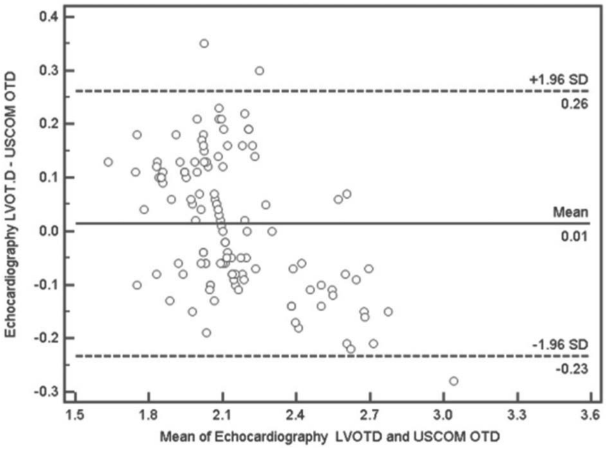
SV = stroke volume. USCOM = ultrasonic cardiac output monitor.

Table 2. Comparison of USCOM and echocardiographic SV measurement

Measure	SV measured by echocardiography, mL (n = 121)	SV measured by USCOM, mL (n = 121)
Minimum, maximum	15.90, 152.20	20.0, 154.0
Mean (SD)	71.71 (26.65)	71.38 (26.53)
Median	71.50	68.0
Mean difference (SD)	0.33 (5.62)	
P_1*	0.519	

USCOM = ultrasound cardiac output monitor. SV = stroke volume. SD = standard deviation. * P for one sample t test between the difference and 0 (if significant, there is fixed bias).

Figure 2. Bland–Altman graph for LVOT diameter with USCOM and echocardiography



LVOT = left ventricular outflow tract. USCOM = ultrasonic cardiac output monitor

Table 3. Comparison of USCOM and echocardiographic OTD measurement

Measure	LVOTD measured by echocardiography, mL (*n* = 121)	OTD measured by USCOM, mL (*n* = 121)
Minimum, maximum	1.70, 2.90	1.57, 3.18
Mean (SD)	2.15 (0.22)	2.13 (0.29)
Median	2.10	2.09
Mean difference (SD)	0.014 (0.13)	
P_1*	0.238	

USCOM = ultrasound cardiac output monitor. OTD = outflow tract diameter. LVOTD = left ventricular outflow tract diameter. SD = standard deviation. * *P* for one sample *t* test between the difference and 0 (if significant, there is fixed bias).

Table 4. Comparison of USCOM and echocardiographic LVOT Vpk measurement

Measure	LVOT Vpk measured by echocardiography, cm/s (*n* = 121)	Vpk measured by USCOM, cm/s (*n* = 121)
Minimum, maximum	51.0, 173.0	44.0, 180.0
Mean (SD)	100.65 (24.24)	98.88 (28.02)
Median	99.0	95.0
Mean difference (SD)	1.78 (10.14)	
P_1*	0.055	

USCOM = ultrasound cardiac output monitor. LVOT = left ventricular outflow tract. Vpk = peak velocity. SD = standard deviation. * *P* for one sample *t* test between the difference and 0 (if significant, there is fixed bias).

In the subgroup of 31 patients diagnosed with a low cardiac output state, confirmed with echocardiography showing an ejection fraction of < 35%, we examined the sensitivity and specificity of SMII of < 1.1 watts/m^2 (proposed in some research as a cut point to diagnose low cardiac output status). Results showed a scuffed ROC curve (Figure 8 [online at cicm.org.au/Resources/Publications/Journal]) with poor sensitivity (< 70%) to diagnose a low cardiac output status for SMII of < 1.1 watts/m^2 (Table 10).

Table 5. Comparison of LVOT Vpk and AV Vpk measured by echocardiography

Measure	LVOT Vpk, cm/s (*n* = 121)	AV Vpk, cm/s (*n* = 121)
Minimum, maximum	51.0, 173.0	42.0, 241.6
Mean (SD)	100.65 (24.24)	123.99 (33.12)
Median	99.0	121.50
Mean difference (SD)	23.16 (22.38)	
P_1*	< 0.001†	

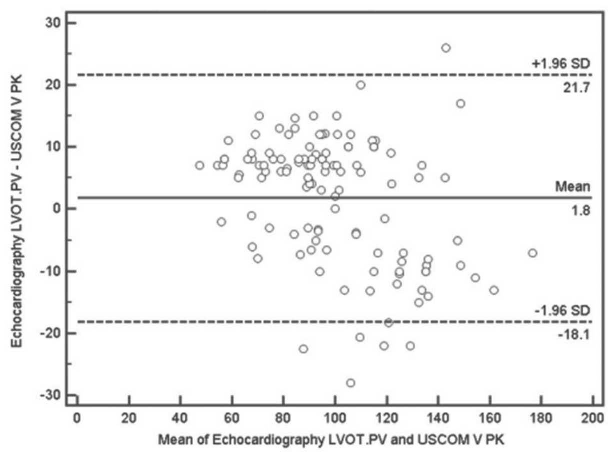
LVOT = left ventricular outflow tract. PV = peak velocity. AV = aortic valve. SD = standard deviation. * *P* for one sample *t* test between the difference and 0 (if significant, there is fixed bias). † Significant.

Table 6. Comparison of AV Vpk measured by echocardiography and Vpk measured by USCOM

Measure	AV Vpk measured by echocardiography, cm/s (*n* = 121)	Vpk measured by USCOM, cm/s (*n* = 121)
Minimum, maximum	42.0, 241.6	44.0, 180.0
Mean (SD)	123.99 (33.12)	98.88 (28.02)
Median	121.50	95.0
Mean difference (SD)	24.94 (22.98)	
P_1*	< 0.001†	

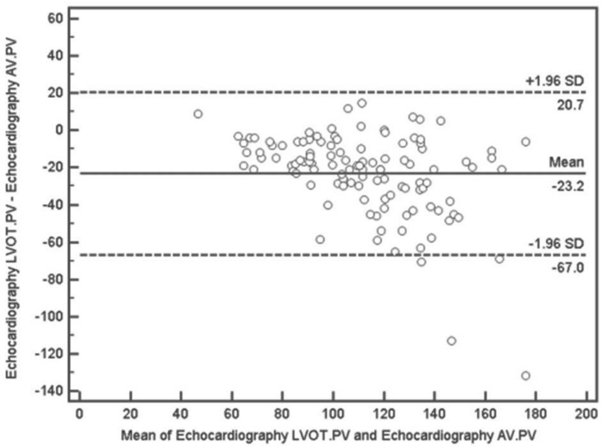
AV = aortic valve. Vpk = peak velocity. USCOM = ultrasound cardiac output monitor. SD = standard deviation. * *P* for one sample *t* test between the difference and 0 (if significant, there is fixed bias). † Significant

Figure 3. Bland–Altman graph for LVOT PV with USCOM and echocardiography



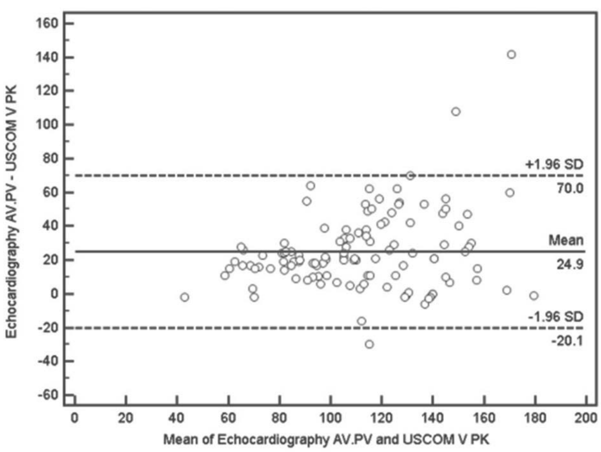
LVOT = left ventricular outflow tract. PV = peak velocity. Vpk = peak velocity. USCOM = ultrasonic cardiac output monitor.

Figure 4. Bland–Altman graph for LVOT PV and AV PV echocardiography



LVOT = left ventricular outflow tract. PV = peak velocity. AV = aortic valve.

Figure 5. Bland–Altman graph for AV Vpk echocardiography and Vpk USCOM

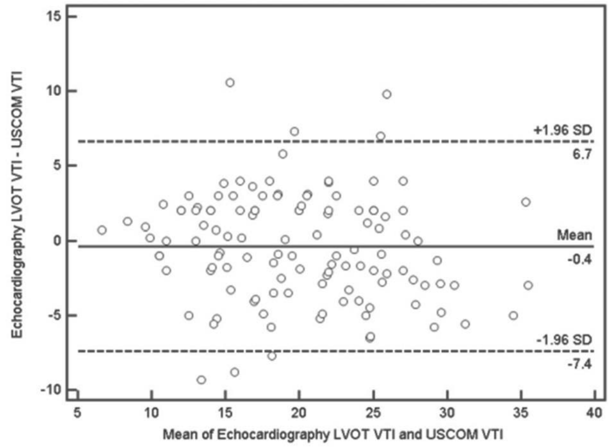


AV = aortic valve. PV = peak velocity. Vpk = peak velocity. USCOM = ultrasonic cardiac output monitor.

Discussion

In our analysis, there was a clear and unbiased correlation between SV measured by the USCOM and by formal echocardiography. This confirms that a simple device such as the USCOM can accurately estimate SV (and therefore cardiac output) in intensive care patients, because there is similar Doppler signal acquisition between the two modalities. The small differences in flow in the blinded USCOM technique (when the operator might have scanned flow anywhere from the ascending aorta to the aortic valve), despite our efforts to get the best aortic valve signal, did not affect the overall area under the curve and reliable stroke volume measurement by the USCOM.

Figure 6. Bland–Altman graph for Vti with USCOM and echocardiography



Vti = velocity–time integral. USCOM = ultrasonic cardiac output monitor. LVOT = left ventricular outflow tract.

Table 7. Comparison of Vti measured by echocardiography and by the USCOM

Measure	LVOT Vti measured by echocardiography, cm (n = 121)	Vti measured by USCOM, cm (n = 121)
Minimum, maximum	7.0, 36.60	6.30, 37.0
Mean (SD)	19.80 (6.06)	20.19 (6.56)
Median	20.10	20.0
Mean difference (SD)	0.38 (3.59)	
P_1*	0.246	

Vti = velocity–time integral. USCOM = ultrasound cardiac output monitor. LVOT = left ventricular outflow tract. SD = standard deviation. * P for one sample t test between the difference and 0 (if significant, there is fixed bias).

We compared LVOT and aortic valve diameters measured by echocardiography with aortic valve diameter estimated by the USCOM. Our study showed that the USCOM nomogram is accurate in this population, with the mean difference ≤ 0.1 mm. This is similar to previous studies comparing nomograms to echocardiography.[22] We excluded all patients with any degree of aortic stenosis (4.5% of our screened population). However, we did examine two patients with the USCOM (using echocardiographically measured valve size) who were found to have moderate AS on echocardiography (based on valve area) and were therefore excluded from our study. In these two patients, the measured stroke volumes were 63 mL and 75 mL, respectively (measured with echocardiography), and 64 mL and 76 mL, respectively (measured with USCOM). Research is needed to understand the utility of the USCOM in patients with mild and moderate AS.

Table 8. Agreement (sensitivity, specificity and accuracy) for USCOM stroke volume variation

Criterion	Sensitivity (%)	95% CI	Specificity (%)	95% CI	+ PV	– PV
> 10	100.00%	94.0–100.0	0	0.0–5.9	49.6	–
> 17	100.00%	94.0–100.0	67.21%	54.0–78.7	75.0	100.0
> 18	98.33%	91.1–100.0	75.41%	62.7–85.5	79.7	97.9
> 19	98.33%	91.1–100.0	88.52%	77.8–95.3	89.4	98.2
> 20	95.00%	86.1–99.0	93.44%	84.1–98.2	93.4	95.0
> 21	95.00%	86.1–99.0	95.08%	86.3–99.0	95.0	95.1
> 23	91.67%	81.6–97.2	95.08%	86.3–99.0	94.8	92.1
> 24	88.33%	77.4–95.2	96.72%	88.7–99.6	96.4	89.4
> 36	31.67%	20.3–45.0	96.72%	88.7–99.6	90.5	59.0
> 37	28.33%	17.5–41.4	98.36%	91.2–100.0	94.4	58.3
> 41	20.00%	10.8–32.3	98.36%	91.2–100.0	92.3	55.6
> 42	20.00%	10.8–32.3	100.00%	94.1–100.0	100.0	56.0
> 66	0	0–6.0	100.00%	94.1–100.0	–	50.4

Table 9. Relation between *F* responsiveness and USCOM stroke volume variation

	F responsiveness				
USCOM SVV	No, *n* (%) (*n* = 61)	Yes, *n* (%) (*n* = 60)	χ^2	*P*	κ
≤ 21	58 (95.1%)	3 (5.0%)	98.18	< 0.001	0.901
> 21	3 (4.9%)	57 (95.0%)			

USCOM = ultrasound cardiac output monitor. SVV = stroke volume var-iation.

Table 10. Agreement (sensitivity, specificity and accuracy) for SMII to predict patients with low cardiac output

	Low cardiac output				
SMII	No, *n* (%) (*n* = 90)	Yes, *n* (%) (*n* = 31)	χ^2	*P*	κ
> 1.16	76 (84.4%)	9 (29.0%)	33.875	< 0.001	0.526
≤ 1.16	14 (15.6%)	22 (71.0%)			

SMII = Smith–Madigan inotropy index.

We found that although the PV measured by USCOM and LVOT echocardiography correlated well, neither correlated with PV at the aortic valve by echocardiography. We attribute this to two outlier measurements in the aortic valve PV by echocardiography (seen in Figures 4 and 5) that led to the significant difference between the measurements, and hence the poor correlation. Despite this small difference between the PVs, there was no effect on the Vti curve, which had good agreement between the two machines.

We could not show a relationship between fluid re-sponsiveness and SVV of > 10%. Our ROC curve of fluid responsiveness with SVV suggested that an SVV of 21% correlated well with fluid responsiveness, with sensitivity and specificity of > 95%. As expected, this was better for patients who were mechanically ventilated than those who were not ventilated (κ = 0.901 v 0.821), but as 63% of the total cohort were ventilated, there was a good overall correlation. Our findings are similar to the findings of others who examined echocardiographic SVV measurements in fluid responsive patients, which ranged from 15% to 17%.[23,24] In one study using oesophageal Doppler-derived SVV in ventilated patients, the cut-off point for fluid responsiveness was 14%, compared with 21% using the USCOM.[25]

Finally, an SMII of < 1.1 watts/m^2 has been taken by some researchers to be the cut point for a low cardiac output state. We only examined SMII in a subgroup of 31 patients with low cardiac output, but there was only a fair correlation (κ = 0.526) and a sensitivity of < 70%. We suggest that derived indices such as the SMII have limited value in a critically ill patient cohort such as ours.

Strengths and limitations

We studied a large number of patients in two centres. We were dependent on the quality of the echocardiograms which we used as the standard for comparison because, with any ultrasound investigation, there is a skill dependence and inter-interpreter variation which can affect the accuracy and reliability the results. Our four sonographers all had more than 6 years echocardiographic experience and all our echocardiographic studies were reported by consultant cardiologists or intensivists specifically trained in echocardiography.

For our subgroup of patients with low cardiac output, for whom we recorded SMII, we lacked a precise definition for cardiogenic shock and low cardiac output. This, with the small sample size, makes any conclusions speculative only, but they warrant further investigation.

We excluded all patients with even mild AS because this was a validation study. The utility of the USCOM in patients with mild-to-moderate AS remains an important question. Specific testing is needed to assess how different degrees of AS can affect the USCOM readings and whether, if measured aortic valve size is used rather than estimated size, the measurements would be reliable.

Despite the fact that none of our patients had LVOT obstruction diagnosed on echocardiography, we should have excluded it in the study protocol.

As with any blinded CW Doppler scan, the USCOM machine accuracy can be affected by using aortic valve nomograms, because variation from actual valve size may exist.

Lastly, our USCOM operator had more than 6 months training with the USCOM previously, and may have therefore acquired better Doppler tracings than operators might after the recommended 2–4 days' training.

Conclusion

SV (and thus cardiac output) measured using the USCOM, a simple non-invasive monitor, correlates well with echocardiographic cardiac output measurement. This suggests that the USCOM could be a valuable haemodynamic tool for assessment of cardiac output and fluid responsiveness in critically ill patients if AS is excluded. SMII as a parameter of low cardiac output was not useful in this cohort of patients.

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Competing interests

We declare no financial interest or relation of any type with the company Uscom Ltd. The USCOM machine used in this study was loaned to us unconditionally by the manufacturer, who had no access to our data. We take full responsibility for the veracity and analysis of these data.

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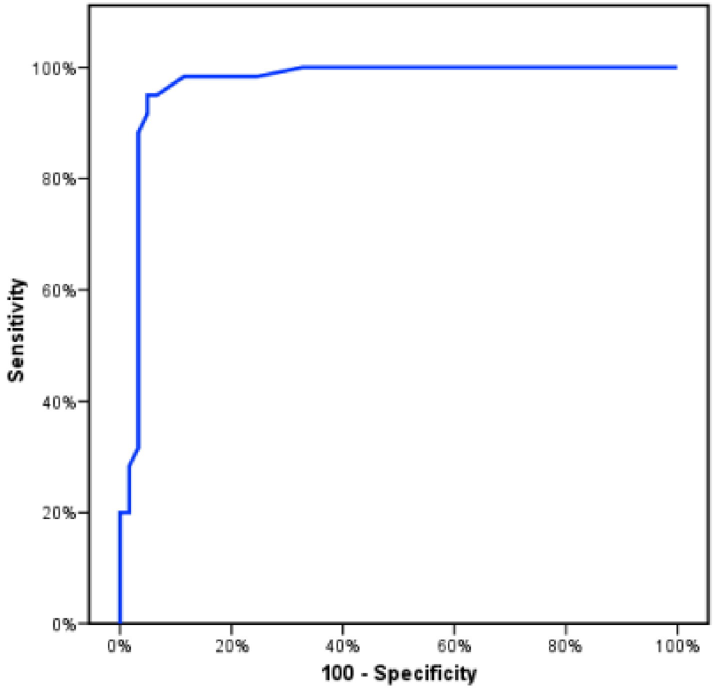
Appendix
This appendix was part of the submitted manuscript and has been peer reviewed. It is posted as supplied by the authors.

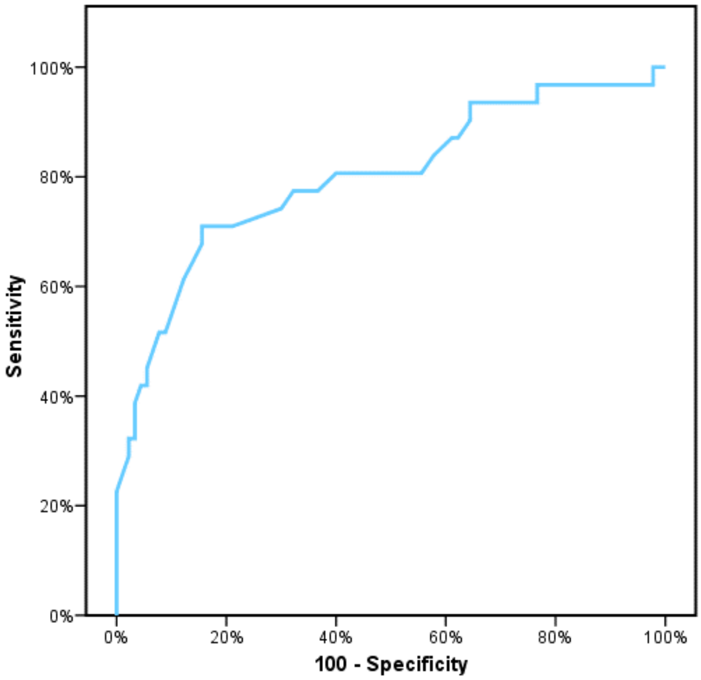
Figure7: ROC curve for USCOM SVV for predicting F responsiveness (compares SVV in SV>15% and not SV>15%)



	AUC	P	Cut off point	Sensitivity	Specificity	PPV	NPV	Accuracy
USCOM SVV	0.968	<0.001*	>21	95.0	95.1	95.0	95.1	95.1

Value of κ	**Strength of agreement**
< 0.20	Poor
0.21 - 0.40	Fair
0.41 - 0.60	Moderate
0.61 - 0.80	Good
0.81 - 1.00	Very good

Figure 8: ROC curve for SMII ability to predict cases with low cardiac output state (LVEF <35%).



	AUC	P	Cut off point	Sensitivity	Specificity	PPV	NPV	Accuracy
SMII	0.799*	<0.001*	≤1.16	69.97	84.44	61.1	89.4	80.99