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REVIEW

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Clinical examination for diagnosing circulatory shock

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Purpose of review
In the acute setting of circulatory shock, physicians largely depend on clinical examination and basic laboratory values. The daily use of clinical examination for diagnostic purposes contrasts sharp with the limited number of studies. We aim to provide an overview of the diagnostic accuracy of clinical examination in estimating circulatory shock reflected by an inadequate cardiac output (*CO*).

Recent findings
Recent studies showed poor correlations between *CO* and mottling, capillary refill time or central-to-peripheral temperature gradients in univariable analyses. The accuracy of physicians to perform an educated guess of *CO* based on clinical examination lies around 50% and the accuracy for recognizing a low *CO* is similar. Studies that used predefined clinical profiles composed of several clinical examination signs show more reliable estimations of *CO* with accuracies ranging from 81 up to 100%.

Summary
Single variables obtained by clinical examination should not be used when estimating *CO*. Physician's educated guesses of *CO* based on unstructured clinical examination are like the 'flip of a coin'. Structured clinical examination based on combined clinical signs shows the best accuracy. Future studies should focus on using a combination of signs in an unselected population, eventually to educate physicians in estimating *CO* by using predefined clinical profiles.

Keywords
cardiac output, circulatory shock, clinical examination, critical illness, diagnostic accuracy, physical examination, shock

INTRODUCTION

Many critically ill patients suffer from circulatory shock, which places them at increased risks of multi-organ failure, long-term morbidity and mortality [1,2]. Combinations of clinical, hemodynamic and biochemical variables are recommended for diagnosing shock [3,4].

Daily use of clinical examination (in any patient) for diagnostic purposes contrasts with the limited number of studies, so that the level of evidence in the critically ill is considered best practice [4]. Much remains unknown about the value of clinical examination in diagnosing shock, reflected by an inadequate cardiac output (*CO*) or maldistribution of blood flow. More knowledge on this topic could assist physicians in the diagnostic process and guide interventions. Previous overviews have evaluated the value of physical examination in sepsis patients [5], cardiovascular patients [6▪▪] and in hemodynamically unstable patients for predicting fluid responsiveness [7▪]. We aim to provide an overview of the diagnostic test accuracy of clinical examination findings for estimating *CO* in critically ill patients.

BACKGROUND

'Clinical examination' of the cardiovascular system has been performed for a long time. The first evaluations of heart rate by palpation of the arterial pulse rate date back as far as approximately 335–280 B.C.

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KEY POINTS

- Clinical examination findings are poorly associated with *CO* in single-variable and multivariable analyses.
- The physician's accuracy to subjectively estimate *CO* based on clinical examination equals the flip of a coin.
- Physicians are likely insufficiently capable to recognize a low *CO* by using clinical examination.
- Estimating *CO* by using a predefined combination of clinical signs seems the most accurate method to diagnose shock.
- Future studies on estimating *CO* should be conducted in a representative population, use standardized clinical examination and use appropriate statistical indices of diagnostic accuracy.

[8]. Around the second century A.D., physicians recognized the value of pulse rate in diagnosing diseases. Pulse quality and quantity were extensively evaluated and distinctions were made in pulse fullness, rate, rhythm and size [9]. However, it would still take hundreds of years before the clinical assessment of circulatory shock 'had evolved' into the way as it is conducted today. In 1941, Ebert *et al.* [10] elaborately described the complexity of symptoms seen in systemic and peripheral circulatory failure in septic shock patients. He encountered the same clinical picture that we still face today:

> (..) All the patients studied presented a similar clinical picture. They were stuporous or comatose. The rectal temperatures ranged from 36.1 to 41.3 degrees Celsius. The skin was pale and often covered with perspiration. The extremities were cold, and this finding usually preceded the fall in arterial pressure. The skin of the body was usually warm, although in terminal stages it too became cool. The radial pulse was feeble or impalpable. The pulse rate was rapid. (..)

For years, clinical examination was considered the cornerstone for diagnosing shock. Reliance on examination declined when Swan *et al.* [11] introduced pulmonary artery catheterization (PAC) in 1970. PAC allowed a wide range of pressure and flow-based hemodynamic measurements, including variables such as pulmonary capillary wedge pressure, systemic vascular resistance and *CO* [12]. Several studies concluded that the use of PAC frequently resulted in change of therapy compared with clinical examination [13–18]. However, PAC remained controversial because of its invasiveness in the absence of any clinical benefit [19–22]. Today, PAC has largely been replaced by less-invasive methods for assessment of *CO*, ranging from echo to pulse pressure analysis devices [23–26].

Despite these technological improvements, clinical examination still holds a prominent position in diagnosing circulatory shock [4,27]. We aimed to provide an overview of the diagnostic accuracy of clinical examination for the assessment of circulatory shock measured by *CO* or cardiac index (*CI*). We only included studies that estimated *CO* using clinical examination based on a one-time snapshot. Physicians mostly use changes in clinical examination findings as proxy for changes in *CO* to guide their interventions. To evaluate the diagnostic accuracy of changes in clinical examination in relation to changes in *CO* was beyond the scope of this review. In this review, we were mainly interested which clinical examination findings may accommodate clinical needs, because in daily practice these snapshot measurements guide treatment decisions as triggers for interventions.

METHODS

A sensitive search strategy was used to identify eligible studies (Appendix 1, http://links.lww.com/COCC/A17). In addition, we used the snowball and citation search methods on the selected articles. We attempted to include all studies that provided results on clinical examination findings in relation to *CO*. We excluded prognostic studies. We separated studies that evaluated univariable associations from studies that used multivariable analyses. Varying statistical indices for describing diagnostic test accuracy as well as a varying prevalence of low *CO* were encountered, limiting interstudy comparison. Whenever available, we used likelihood ratios as the preferred modality to describe diagnostic accuracy. Likelihood ratios may provide valuable information on disease probability in an individual and do not change with pretest probability (i.e. the prevalence of disease) [28–30]. We calculated sensitivity, specificity, predictive values and likelihood ratios of clinical examination for the detection of low *CO* whenever possible.

RESULTS

Our search resulted in 8128 hits of which 28 publications were selected. An additional six publications were identified through snowballing. After selection, we included 34 publications in this overview.

UNIVARIABLE STUDIES

Thirteen studies evaluated univariable associations of clinical examination variables with *CO*, including

skin temperature or temperature gradients ($n=8$) [31–38], capillary refill time (CRT; $n=1$) [39], temperature gradient and CRT ($n=1$) [40], mottling ($n=1$) [41], heart rate and mean arterial pressure ($n=1$) [42] and central venous pressure ($n=1$; Table 1) [31–43]. The method used for measuring *CO* varied, including, for example thermodilution with the PAC or Doppler wave with transesophageal or transthoracic echocardiography.

Circulatory shock may lead to compensatory vasoconstriction of nonvital, peripheral tissues such as the skin. Peripheral perfusion can easily be evaluated by measurement of skin temperature, CRT and degree of skin mottling. Two studies demonstrated that a subjectively cool skin temperature was associated with a lower *CO* [31,32]. Studies evaluating the correlation between objective temperature measurements and *CO* showed conflicting results; some observed moderate correlations [33,35,40], whereas most observed no correlation [34–38]. Skin temperature measurement methods differ widely and are likely influenced by several factors: age, ambient temperature, hypothermia, peripheral vascular disease, vasopressors, pain and anxiety have all been proposed as influencing circumstances [44,45]. This may explain the conflicting results and may limit its usefulness for estimating *CO* in clinical practice. Several studies have emphasized the prognostic value of prolonged CRT and mottling of the skin [39,41,46–49], but only three studies have evaluated their associations with *CO* and found no relevant correlations [39–41].

Prospective studies on systemic hemodynamic variables showed that heart rate, mean arterial pressure and central venous pressure were not directly correlated to *CO* [42,43,50]. Only during episodes of deep hypotension, one study observed a moderate correlation between mean arterial pressure and *CO* [42]. These systemic hemodynamic variables seem to be poor indicators of *CO*, which supports the common conception that low blood pressure is a late sign of circulatory shock and should not be relied on for early diagnosis [4,51].

MULTIVARIABLE STUDIES

Twenty-one studies evaluated multivariable associations of clinical variables with *CO*. Because of the differing methods of estimating *CO*, we subdivided our results into studies that evaluated the capacity of physicians to estimate *CO* ($n=17$; Table 2) [13–18, 52–61,62▪▪] and studies that constructed clinical profiles based on multiple variables ($n=3$) or a multivariable model ($n=1$) to correlate clinical examination findings with *CO* (Table 3) [63–66]. Furthermore, we could calculate the diagnostic test accuracy for physician's estimation of low *CO* in nine studies (Table 2).

PHYSICIAN'S CAPACITY TO ESTIMATE *CO* BASED ON CLINICAL EXAMINATION

Seventeen studies evaluated the accuracy of physician's estimates or 'educated guesses' of *CO* as compared to objectively measured *CO*. Estimates were based on clinical examination, with or without knowledge of medical history, biochemical values and/or radiological imaging (Table 2). Some studies used a categorical variable for *CO* estimates (e.g. 'low', 'normal' or 'high'), whereas others used a continuous scale (e.g. 1–12 l per min) [15,17, 62▪▪]. Physician's estimates were correct in 42–62% of the time [13–18,52–61]. Moderate-to-reasonable correlations and a high percentage error were found when physician's estimates of continuous *CO* were compared to objectively measured *CO* [15,16,62▪▪]. Moderate-to-very poor agreements were found in studies that used weighted κ statistics to address agreement occurring by chance [55,59,60,67]. In addition, two studies reported that 21 and 26% of the *CO* estimations were completely disparate (an estimated high *CO* when the objective *CO* was low or vice versa) [55,59].

Nine studies provided enough data for calculation of the diagnostic accuracy of physician's estimates for detecting low *CO*. The overall results appeared disappointing [13,14,16,17,53,54,56,58, 60] (Table 2). Furthermore, two studies concluded that physicians more frequently overestimated (31–33%) rather than underestimated (18–23%) *CO* [14,57], implicating that physicians were more prone to miss an insufficient *CO*. Perel *et al.* [62▪▪] found the opposite when physicians were asked to estimate *CO* on a continuous scale.

These results suggest that physicians are not very capable to subjectively estimate *CO* based on clinical examination. The widely varying diagnostic accuracies are probably the result of different populations or cutoffs for a low *CO*, but overall it seems that physician's estimates are 'an inaccurate diagnostic test'. This is in accordance with two studies of Saugel *et al.* [67,68], which both demonstrate the incapability of physicians to reliably assess volume status using simple clinical signs. Furthermore, five out of six studies concluded that predictions of senior staff members were equally bad as those of residents or fellows [13,18,54,61,62▪▪,69]. Finally, one study found that the accuracy of estimates was unrelated to the level of confidence physicians had in their assessment [69].

Several important limitations apply. Many studies did not elaborate their methods of clinical

Table 1. Prediction of cardiac output using a single variable

Author, year	Patients	Population	Variables of interest	Measurement method	Results	
					Nonsignificant	Significant
Peripheral temperature						
Kaplan *et al.* 2001 [31]	264[a]	Surgical ICU patients	Temp, subjective: foot ('cool' or 'warm')	PAC, technique not mentioned	–	'Cool' : $CI = 2.9 \pm 1.2$ 'Warm': $CI = 4.3 \pm 1.2$
Schey *et al.* 2009 [32]	10[a]	Post cardiac surgery	Temp, subjective: foot: ('cool' or 'cool-warm' or 'warm') Temp, objective of foot	PAC, thermodilution	T_{skin}, objective: $r = 0.11$	'Cool' : $CO = 3.71$ 'Cool-warm': $CO = 4.83$ 'Warm' : $CO = 5.12$
Joly *et al.* 1969 [33]	100	Circulatory shock	Temp, objective: toe ΔT: toe – ambient (ΔTp-a)	Indicator dilution technique	–	T_{skin} objective: $r = 0.71$ ΔTp-a: $r = 0.73$
Woods *et al.* 1987 [34]	26[a]	Circulatory shock	ΔT: central – toe (ΔTc-p)	PAC, thermodilution	ΔTc-p: no correlation	
Vincent *et al.* 1988 [35]	15[a]	Cardiogenic and septic shock	ΔT: toe – ambient (ΔTp-a)	PAC, thermodilution	ΔTp-a in septic shock: no correlation	ΔTp-a in cardiogenic shock: $r = 0.63$
Bailey *et al.* 1990[b] [40]	40[a]	Post cardiac surgery	ΔT: central – toe (ΔTc-p)	PAC, thermodilution	ΔTc-p day of operation: no correlation	ΔTc-p postoperative day 1: $r = -0.60$
Sommers *et al.* 1995 [36]	21[a]	Post cardiac surgery	T_{skin}, objective: axillary, groin, knee, ankle, toe	PAC, thermodilution	T_{skin}, objective: no correlation in any site	–
Boerma *et al.* 2008 [37]	35	Sepsis and septic shock	ΔT: central – foot (ΔTc-p)	TEE, Doppler wave	ΔTc-p: $r = -0.15$	–
Bourcier *et al.* 2016 [38]	103[a]	Sepsis and septic shock	ΔT: toe – ambient (ΔTp-a)	TTE, technique not mentioned	ΔTp-a: no correlation	–
Capillary refill time						
Bailey *et al.* 1990[b] [40]	40[a]	Post cardiac surgery	CRT: site not mentioned	PAC, thermodilution	CRT: no correlation	–
Ait-Oufella *et al.* 2014 [39]	59	Septic shock	CRT: index finger	FloTrac, arterial pressure waveform analysis	CRT: no correlation	–
Skin mottling						
Ait-Oufella *et al.* 2011 [41]	60	Septic shock	Mottling score: knee	TTE, Doppler wave	Mottling score: no correlation	–
Systemic hemodynamic variables						
Wo *et al.* 1993 [42]	256[a]	Severe injury and critically ill postoperative	HR, MAP	PAC, thermodilution	HR: $r = 0.27$, $r^2 = 0.07$, MAP: $r = -0.01$, $r^2 = 0.0001$,	MAP during severe hypotension: $r = 0.50$, $r^2 = 0.25$
Kuntscher *et al.* 2006 [43]	16[a]	Major burns	Central venous pressure	Thermal dye double indicator dilution	–	Central venous pressure: $r = 0.40$

[a]=repeated measurements in each patient.
[b]=same study population.
ΔTc-p, central-to-peripheral temperature gradient (°C); ΔTp-a, peripheral-to-ambient temperature gradient (°C); *CI*, cardiac index (l/min/m^2); *CO*, cardiac output (l/min); CRT, capillary refill time (s); HR, heart rate (beats/min); MAP, mean arterial pressure (mmHg); PAC, pulmonary artery catheter; TEE, transoesophageal echocardiography; Temp, temperature (°C); TTE, transthoracic echocardiography.

Table 2. Physician's capacity to estimate cardiac output based on clinical examination

Author, year	Patients	Setting	Variables of interest		Measurement method	Results	
			Classification	Estimation based on		Estimation	Diagnostic accuracy for low *CO* (95% CI)
Connors *et al.* 1983 [13]	62[a]	ICU	*CI* categorical: <2.5; 2.5–3.5; >3.5	Clinical assessment, laboratory and X-ray	PAC, thermodilution	44% correct estimation	Sens 58% (45–68%); Spec 60% (48–71%) PPV 58% (49–65%); NPV 60% (52–67%) LR+ 1.43 (1.02–2.00); LR– 0.71 (0.51–0.98)
Eisenberg *et al.* 1984 [14]	97	ICU	*CO* categorical: <4.5; 4.5–7.5; >7.5	Not described	PAC, thermodilution	51% correct estimation	Sens 71% (54–85%); Spec 56% (43–69%) PPV 48% (39–57%); NPV 78% (66–86%) LR+ 1.64 (1.15–2.33); LR– 0.51 (0.29–0.89)
Tuchschmidt *et al.* 1987 [15]	35	ICU	*CO* continuous	Clinical assessment and X-ray	PAC, thermodilution	$r=0.72$	–
Connors *et al.* 1990 [17]	461	ICU	*CI* dichotomous: <2.2; ≥2.2 *CI* continuous	Clinical assessment, laboratory, X-ray and ECG	PAC, thermodilution	64% correct estimation Mean CI-difference in $CI=1.0\pm0.9$	Sens 49% (40–57%); Spec 70% (65–75%) PPV 43% (38–49%); NPV 74% (71–77%) LR+ 1.62 (1.28–2.05); LR– 0.73 (0.62–0.87)
Celoria *et al.* 1990[b] [16]	114	Surgical ICU	*CO* categorical: <4; 4–8; >8	Clinical assessment, laboratory and X-ray	PAC, thermodilution	51% correct estimation $r=0.47$	Sens 67% (30–93%); Spec 80% (71–87%) PPV 22% (14–34%); NPV 97% (92–99%) LR+ 3.33 (1.83–6.07); LR– 0.42 (0.16–1.05)
Steingrub *et al.* 1991[b] [53]	152	Surgical and medical ICU	*CO* categorical: <4; 4–8; >8	Clinical assessment, laboratory and X-ray	PAC, thermodilution	51% correct estimation	Sens 54% (37–70%); Spec 73% (63–81%) PPV 40% (31–51%); NPV 82% (76–87%) LR+ 1.96 (1.29–2.98); LR– 0.64 (0.44–0.91)
Mimoz *et al.* 1994 [18]	112	ICU	Combinations of *CI*, PAOP and SVRI	Clinical assessment, laboratory, X-ray and echocardiography	PAC, thermodilution	56% correct estimation	–
Staudinger *et al.* 1998 [54]	149	ICU	*CI* categorical: <2.0; 2.0–4.0; >4.0	Clinical assessment, medical history, laboratory and X-ray	PAC, thermodilution	62% correct estimation	–
Rodriguez *et al.* 2000 [55]	33	ED + respiratory distress or hypotension	*CI* categorical: <2.6; 2.6–4.0; >4.0.	Clinical assessment, medical history, laboratory, X-ray and ECG	TEE, Doppler wave	$\kappa1=-0.04$ (95% CI–0.31–0.24) $\kappa2=0.07$ (95% CI –0.17–0.31)	–
Linton *et al.* 2002 [56]	50	Post cardiac surgery	*CI* categorical: <1.9; 1.9–3.5; >3.5	Not described	LiDCO, indicator-dilution	54% correct estimation	Sens 42% (15–72%); Spec 74% (57–87%) PPV 33% (18–54%); NPV 80% (71–87%) LR+ 1.58 (0.67–3.72); LR– 0.79 (0.47–1.32)
Iregui *et al.* 2003 [57]	105	ICU	*CI* categorical: <2.5; 2.5–4.5; >4.5	Clinical assessment, laboratory and X-ray	TEE, Doppler wave	44% correct estimation	–
Veale *et al.* 2005 [58]	68	ICU	*CI* categorical: <2.5; 2.5–4.2; >4.5	Not described	BioZ *CO* monitor, Impedance cardiography	42% correct estimation	Sens 22% (6–48%); Spec 66% (51–79%) PPV 19% (8–38%); NPV 70% (63–76%) LR+ 0.65 (0.25–1.68); LR– 1.18 (0.86–1.62)
Rodriguez *et al.* 2006 [59]	31	ED + endotracheal intubation	*CI* categorical: ranges not specified	Clinical assessment, medical history, laboratory and X-ray	TEE, Doppler wave	$\kappa=0.57$ (95% CI 0.36–0.77)	–
Nowak *et al.* 2011 [60]	38	ED + respiratory distress	*CO* categorical <4.0; 4.0–8.0; >8.0	Clinical assessment and medical history	Nexfin, ABP waveform analysis	50% correct estimation $\kappa=-0.02$ (95% CI –0.25–0.20)	Sens 33% (4–78%); Spec 63% (44–79%) PPV 14% (5–36%); NPV 83% (73–90%) LR+ 0.89 (0.26–3.00); LR– 1.07 (0.57–2.00)
Duan *et al.* 2014 [61]	132	ICU	*CI* categorical: <3; 3–5; >5	Not described	PiCCO, thermodilution	50% correct estimation	–
Perel *et al.* 2016 [62■■]	206[a]	ICU	*CO* continuous	Clinical assessment	PiCCO, thermodilution	Percentage error=66% Absolute mean difference in $CO=-1.5\pm2.2$	–

[a]=repeated measurements in each patient.
[b]=overlapping study populations.
95% CIs, 95% confidence intervals; *CI*, cardiac index (l/minute/m^2); *CO*, cardiac output (l/min); ECG, electrocardiography; ICU, intensive care unit; LiDCO, lithium dilution cardiac output; LR–, negative likelihood ratio; LR+, positive likelihood ratio; NPV, negative predictive value; PAC, pulmonary artery catheter; PAOP, pulmonary artery occlusion pressure (mmHg); PiCCO, pulse contour cardiac output; PPV, positive predictive value; Sens, sensitivity; Spec, specificity; SVRI, systemic vascular resistance index (dynes · s/cm^5 · min^2); TEE, transesophageal echocardiography.

Table 3. Combined signs of clinical examination for estimation of *CO*

Author, year	Patients	Population	Variables of interest: Clinical profile	Variables of interest: Clinical profile based on	*CO*-measurement	Results
Combined clinical profiles						
Ramo *et al.* 1970 [63]	98	AMI	I (normal *CI*): no signs of HF II (normal *CI*): mild-to-moderate HF III (low *CI*): overt pulmonary edema IV (low *CI*): cardiogenic shock	Mean arterial pressure, cool extremities, urine output, mental status, third heart sound gallop rhythm and rales	PAC, indicator-dilution technique	I (normal *CI*): 23 of 45 (51%) II (normal *CI*): 19 of 30 (63%) III (low *CI*): 10 of 10 (100%) IV (low *CI*): 13 of 13 (100%)
Forrester *et al.* 1977 [64]	200	AMI	I (normal *CI*): no pulmonary congestion or peripheral hypoperfusion II (normal *CI*): pulmonary congestion only III (low *CI*): hypoperfusion only IV (low *CI*): both	Heart rate, blood pressure, cool extremities, urine output and mental status	PAC, thermodilution	Overall: 81% correct estimations of *CI* I & II (normal *CI*): 84 of 95 (88%) III & IV (low *CI*): 76 of 105 (72%)
Grissom *et al.* 2009 [65]	405	ALI	I: All three clinical signs aberrant II: Any one clinical sign aberrant	Capillary refill time, knee mottling and cool extremities	PAC, thermodilution	92% correct estimations of *CI* in class I: Sens 12% (3–28%); Spec 98% (97–99%) PPV 40% (17–69%); NPV 93% (92–93%) LR+ 7.52 (2.23–25.3); LR– 0.89 (0.79–1.01) 75% correct estimations of *CI* in class II: Sens 52% (34–69%); Spec 78% (73–82%) PPV 17% (12–23%); NPV 95% (93–96%) LR+ 2.31 (1.58–3.38); LR– 0.62 (0.44–0.89)
Multivariable analysis						
Sasse *et al.* 1996 [66]	23[a]	ICU patients	*CO* continuous	Heart rate, respiratory rate, mean arterial pressure and temperature	PAC, thermodilution	Heart rate: $R^2 = 0.05$ Respiratory rate: $R^2 = 0.14$ Mean arterial pressure: $R^2 = 0.03$

[a]=repeated measurements in each patient.

ALI, acute lung injury; AMI, acute myocardial infarction; *CI*, cardiac index (l/min/m^2); *CO*, cardiac output (l/min); HF, heart failure; LR–, negative likelihood ratio; LR+, positive likelihood ratio; NPV, negative predictive value; PAC, pulmonary artery catheter; PPV, positive predictive value; Sens, sensitivity; Spec, specificity.

examination in terms of variables used and definitions employed, leaving variability at the physician's discretion so that these studies cannot be reproduced. PAC was used in most studies, but only in selected patients who failed to respond to initial therapy or in whom clinical examination alone was deemed insufficient, so that evaluation of the accuracy of clinically estimated *CO* will be biased by definition. Likewise, many other studies also used convenience samples, which hampers generalizability of their results. Clinical examination should be performed in a standardized fashion, according to a protocol, to maximize interobserver agreement and generalizability.

COMBINED SIGNS OF CLINICAL EXAMINATION FOR ESTIMATION OF *CO*

Three studies have compared predefined clinical profiles based upon clinical examination with objectively measured *CI* (Table 3). Forrester *et al.* [64] found a good agreement in patients with acute myocardial infarction (AMI). In their study, 75% of patients with low *CI* and 96% of patients with very low *CI* had clinical signs of peripheral hypoperfusion, such as decreased skin temperature, confusion or oliguria in conjunction with either arterial hypotension or tachycardia. Ramo *et al.* [63] observed 100% correct estimation of low *CI* when patients with AMI had overt signs of pulmonary edema or signs of cardiogenic shock. In their study, clinical signs of overt pulmonary edema were defined by rales or a third heart sound gallop rhythm and cardiogenic shock was diagnosed by the presence of a systolic blood pressure below 90 mmHg, oliguria, cold extremities and disorientation. These findings suggest that physicians can diagnose cardiogenic shock in patients with AMI using clinical examination. Accurate estimation of *CO* for diagnosing shock in all critically ill patients based on clinical examination might appear much more difficult because of large interindividual differences. Grissom *et al.* [65] combined CRT, mottling and skin temperature to predict *CI* in an unselected cohort of patients with acute lung injury. The presence of all three physical signs had a high specificity (98%) but a low sensitivity (12%) for diagnosing shock, suggesting that these three signs accurately rule in, but inaccurately rule out circulatory shock. Varying types of shock are probably associated with varying clinical signs [70], so that a 'one size fits all' approach seems inappropriate. Roughly, one-third of all patients with circulatory shock suffer from a low *CO*, whereas two-thirds have distributive shock with associated high *CO* [1,71]. Especially in the latter, clinical examination may indicate inadequate circulation regardless of the height of *CO* and it is difficult to establish how much *CO* is sufficient for each individual patient.

PREDICTING *CO* USING A MULTIVARIABLE MODEL

One study used multivariable regression analyses to estimate *CO* based on heart rate, respiratory rate, mean arterial pressure and central temperature (Table 3) [66]. These multivariable results confirm that systemic hemodynamic variables do not correspond well with *CO*. Future diagnostic studies of *CO* should therefore incorporate all clinical and hemodynamic variables in a multivariable model.

CONCLUSION

Clinical examination findings are poorly associated with *CO* in single-variable and multivariable analyses. Physicians seem to be insufficiently capable to estimate *CO* or recognize a low *CO* using their clinical examination. The most promising results were found when *CO* was estimated by using predefined profiles composed of combined clinical examination signs. However, most studies were conducted in highly selected populations and the details of estimations were not specified. On the basis of current evidence, using clinical examination to diagnose *CO* can, to our opinion, not be considered best practice. Future studies on this topic should be conducted in a representative population, use standardized clinical examination and use appropriate statistical indices of diagnostic accuracy. Ultimately, these results should guide education of physicians to estimate *CO* using predefined clinical profiles.

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Conflicts of interest

There are no conflicts of interest.

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- ■ of special interest
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