

Research Article

Association of Early Peripheral Perfusion Parameters with Organ Dysfunction and Mortality in Critically Ill Patients with Circulatory Shock: A Prospective Observational Study

Dr. Nityanand Rao Patil^{1*}, Dr. Patil Archana², Dr. Shubhangi S. Chavan³

^{1*}Senior Consultant, Department of Critical Care Medicine, Medcover Hospitals, Hyderabad, Telangana, India.

²Associate Professor, Department of Pathology, TRR Institute of Medical Sciences (TRR IMS), Inole, Hyderabad, Telangana, India.

³Consultant, Critical Care Medicine, Medcover Hospitals, Hyderabad, Telangana, India.

Email: ^{1*}nityanandraopatil@gmail.com, ²patil18@yahoo.com

Corresponding Author: Dr. Nityanand Rao Patil

Senior Consultant, Department of Critical Care Medicine, Medcover Hospitals, Hyderabad, Telangana, India.

Email: nityanandraopatil@gmail.com

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ABSTRACT

Background: Peripheral perfusion abnormalities may provide early information about tissue hypoperfusion in circulatory shock. This study evaluated whether capillary refill time (CRT), mottling, and peripheral perfusion index (PI) were associated with organ dysfunction and mortality.

Methods: In this prospective observational study, 200 adults with circulatory shock underwent baseline and six-hour peripheral perfusion assessment. Associations with 48-hour SOFA score and 28-day mortality were examined using adjusted regression models.

Results: Twenty-eight-day mortality was 24.0%. Baseline CRT was associated with mortality in unadjusted analysis but not after adjustment (OR 1.40 per second, 95% CI 0.92-2.14). Baseline mottling and PI were also not independently associated with mortality. In contrast, six-hour worsening in CRT and mottling was associated with higher 48-hour SOFA scores ($B=1.47$ and 0.82 , respectively), while improvement in PI was associated with lower SOFA ($B=-0.65$; all $p \leq 0.001$).

Conclusion: Early changes in peripheral perfusion were more strongly associated with evolving organ dysfunction than baseline values alone.

Keywords: Circulatory Shock, Capillary Refill Time, Mottling, Peripheral Perfusion Index, Sofa.

INTRODUCTION

Circulatory shock is an acute circulatory failure that is life threatening in which tissue oxygen delivery and utilization are inadequate to meet cellular needs. While hypotension is often seen, restoration of mean arterial pressure does not necessarily mean the restoration of tissue perfusion. As a result, clinical, biochemical and microcirculatory parameters have been added to traditional systemic haemodynamic parameters in the assessment of shock [1]. Peripheral circulation is an easy place to look at tissue perfusion as compensatory vasoconstriction during circulatory failure occurs first in the skin and extremities. Therefore, several bedside observations such as delayed capillary refill, cool peripheral temperature and mottling of the skin can be signs of ongoing hypoperfusion despite apparently good systemic haemodynamics. An

objective measure obtained via pulse oximetry is peripheral perfusion index (PI), which quantifies the ratio of the pulsatile and non-pulsatile parts of the peripheral signal [2]. Persistently abnormal peripheral perfusion after initial stabilization has also been associated with greater organ dysfunction and adverse clinical outcome in critically ill patients [3]. Capillary refill time (CRT) is especially appealing because it is fast, inexpensive, repeatable, and does not need any special equipment. Ait-Oufella et al. showed that in septic shock, prolonged CRT after initial resuscitation was associated with tissue hypoperfusion and mortality, suggesting that CRT should be considered as a dynamic marker of circulatory recovery, and not just a sign at admission [4]. If effective resuscitation is achieved, peripheral perfusion may improve relatively quickly and serial assessment of the CRT may be helpful in

determining whether tissue perfusion is adequate or not in patients who remain inadequately perfused despite correction of arterial pressure.

Another clinically recognisable form of impaired cutaneous microcirculation is skin mottling. Ait-Oufella et al. created an ordinal mottling score based on the extent of discoloration around the knee and found a strong relationship between increasing score and mortality in septic shock [5]. The prognostic value of mottling has since been demonstrated in more extensive populations of critically ill patients, and mottling and its persistence have been linked to increased ICU mortality [6]. These observations help to assess peripheral perfusion as a multi-dimensional process and not just on a single bedside sign.

The clinically significant result of prolonged circulatory failure is organ dysfunction. The Sequential Organ Failure Assessment (SOFA) score is a standardised way of measuring dysfunction in the respiratory, coagulation, hepatic, cardiovascular, neurological and renal systems and is now widely used to assess the severity and progression of critical illness [7]. This may help elucidate the temporal relationship between changes in peripheral perfusion and subsequent changes in SOFA.

The clinical usefulness of peripheral perfusion monitoring has also been investigated as a resuscitation goal. In the ANDROMEDA-SHOCK randomized trial, a CRT normalization strategy in early septic shock led to a reduction in organ-dysfunction scores at 72 hours compared to a lactate-targeted strategy, but the reduction in 28-day mortality did not meet conventional statistical significance [8]. The results indicate that peripheral perfusion may be a valuable additional source of data to lactate and systemic haemodynamics in early resuscitation. Most of the previous studies have focused on septic shock or have studied a single peripheral perfusion parameter at a time. There is less information available on comparative and longitudinal behaviour of CRT, mottling and PI in a heterogeneous population of septic, cardiogenic, hypovolaemic, obstructive and mixed circulatory shock. Moreover, the degree of association between early changes in these bedside parameters and subsequent organ dysfunction and mortality is still unclear. The present study thus examined peripheral perfusion parameters at baseline and at 6 hours in critically ill adults with circulatory shock and their relationship with organ dysfunction at 48 hours and 28-day mortality.

PATIENTS AND METHODS

Study design and participants

This is an observational prospective study in the adult intensive care unit (ICU) of a tertiary care centre from January 2025 to June 2026. Recruitment was carried out between January 2025 and May 2026 to allow for follow-up of 28 days. Adults ≥ 18 years of age with new recognition of circulatory shock were enrolled if the baseline study assessment could be achieved within 6 hours of shock recognition. Circulatory shock was defined as sustained mean arterial pressure (MAP) < 65 mmHg, systolic blood pressure (SBP) < 90 mmHg, or requiring vasopressor support to maintain arterial pressure, plus at least one of the following: lactate > 2 mmol/L, urine output < 0.5 mL/kg/hour for at least 2 hours, or acute altered mentation not due to sedation or a primary neurological disorder. These included septic/distributive, cardiogenic, hypovolaemic, obstructive, and mixed shock, and the predominant aetiology and underlying cause were noted.

Exclusion criteria included shock diagnosed > 6 hours before baseline, severe peripheral vascular or local limb disease that would not allow for a reliable assessment of capillary refill, core temperature $< 32^{\circ}\text{C}$ or therapeutic temperature management, mechanical circulatory support at baseline, post-cardiac-arrest syndrome, comfort-focused care before enrolment, and repeat enrolment during the same admission. Patients were kept in the cohort if they went on to die, develop complications, be placed on mechanical support, or if follow-up measurements were not available.

Clinical and peripheral perfusion assessment

Demographic data, comorbidities, time to admission and shock recognition, vasoactive treatment, mechanical ventilation, fluid and blood-product administration, lactate, urine output, haemodynamics and temperature were recorded. BMI was defined as $\text{weight}/\text{height}^2$. Peripheral perfusion was measured at baseline and at 6 hours in a window of ± 1 hour. Capillary refill time (CRT) was measured at a finger site after 10 seconds of blanching pressure and recorded continuously in seconds. Primary exposure was baseline CRT and secondary descriptive classification was CRT > 3 seconds. Mottling was scored on an ordinal scale from 0 to 5. Unassessable mottling was recorded as unavailable rather than as score 0. Peripheral

perfusion index (PI) was derived from the pulse-oximeter signal with the use of the pulsatile to non-pulsatile signal ratio provided by the device. No universal abnormal PI threshold was applied, and measurements with poor signal quality were treated as missing. Changes in scores were determined as the 6-hour value minus the baseline value. Thus, a negative change in CRT and mottling was considered to be an improvement, while a positive change in PI was considered to be an improvement. When both lactate levels were available, and baseline lactate was positive, the percentage change in lactate was calculated as $100 \times (\text{six-hour lactate} - \text{baseline lactate}) / \text{baseline lactate}$. No six-hour measurements were taken post mortem.

Organ dysfunction and outcomes

The conventional six-component Sequential Organ Failure Assessment (SOFA) score was used to assess organ dysfunction. Respiratory scoring included $\text{PaO}_2/\text{FiO}_2$ and status on respiratory support; coagulation included platelet count; hepatic dysfunction included bilirubin; cardiovascular dysfunction included MAP and status on vasoactive support; neurological dysfunction included Glasgow Coma Scale (GCS); and renal dysfunction included creatinine or 24-hour urine output, whichever gave the more severe score.

Baseline SOFA only included data collected at or before baseline assessment. The most recent reliable pre-sedation GCS in the specified time frame was used for sedated patients; if this was unavailable, the neurological component and total SOFA were considered missing. Follow-up SOFA was calculated from the worst component values within the 24-hour period from 24 to 48 hours after baseline. Deaths that occurred prior to the end of this period were reported separately and were not given an arbitrary follow-up score. The SOFA change was defined as the 48-hour minus the baseline SOFA.

The main clinical outcome was all-cause mortality within 28 days of baseline, including deaths after hospital discharge. Secondary outcomes were 48-hour SOFA, SOFA change, ICU and hospital mortality, mechanical

ventilation, new renal replacement therapy, post-baseline mechanical circulatory support, and the length of stay in the ICU and hospital.

Statistical analysis

Data for continuous variables were presented as mean $\pm$ SD or median [IQR], as appropriate, and those for categorical variables as n (%). Missing observations were analyzed using available-case analysis.

Wilcoxon signed-rank tests were used to compare baseline to 6-hour changes in CRT, mottling, PI, and lactate, and a paired t test was used to compare changes in MAP. The Wilcoxon signed-rank test was used to compare baseline and 48-hour SOFA scores in patients who had both scores.

Logistic regression was used to assess the relationship between baseline CRT and 28-day mortality, adjusting for age, baseline SOFA and shock aetiology; baseline lactate was included in a sensitivity model. Baseline mottling and PI were evaluated in separate models with the same covariates. Mottling was analysed per 1-point increase and PI was $\log_2$ transformed. Linear regression with HC3 robust standard errors was used to assess associations with 48-hour SOFA, adjusting for baseline SOFA, age and shock aetiology. The 6-hour change models also adjusted for the corresponding baseline perfusion value.

Mortality analyses of 6-hour perfusion changes were performed using a six-hour landmark approach, excluding patients who died prior to 6 hours. No outcomes were imputed. All tests were two-tailed and $p < 0.05$ was taken as statistically significant.

RESULTS

Cohort characteristics

The cohort included 200 adults with circulatory shock. Mean age was 59.0 ± 16.6 years and 121 (60.5%) were male. Septic/distributive shock was the most frequent aetiology, occurring in 101 (50.5%), followed by cardiogenic shock in 40 (20.0%). Baseline clinical, perfusion, and organ-dysfunction characteristics are summarized in Table 1.

Table 1. Baseline characteristics of the study cohort

Characteristic	Overall (N=200)
Age, years	59.0 ± 16.6
Male sex	121 (60.5%)
BMI, kg/m^2	24.3 ± 4.3
Diabetes mellitus	64 (32.0%)

Hypertension	77 (38.5%)
Chronic kidney disease	43 (21.5%)
Chronic liver disease	15 (7.5%)
Chronic heart failure	38 (19.0%)
Pre-existing renal replacement therapy	4 (2.0%)
Septic/distributive shock	101 (50.5%)
Cardiogenic shock	40 (20.0%)
Hypovolaemic shock	24 (12.0%)
Obstructive shock	7 (3.5%)
Mixed shock	28 (14.0%)
Recognition-to-baseline interval, h	3.09 [1.91–4.34]
Baseline lactate, mmol/L	3.38 [2.58–4.37]
Baseline MAP, mmHg	76.1 ± 6.1
Norepinephrine at baseline	170 (85.0%)
Norepinephrine dose among users, µg/kg/min	0.122 [0.090–0.187]
Mechanical ventilation at baseline	118 (59.0%)
Baseline SOFA score	7.0 [5.0–9.0]
Baseline CRT, s	3.1 [2.3–4.0]
Baseline CRT >3 s	105 (53.0%)
Baseline mottling score	0.0 [0.0–1.8]
Baseline peripheral perfusion index	1.88 [1.14–2.82]

Values are mean ± SD, median [IQR], or n (%), as appropriate. Denominators for variables with missing measurements are based on available observations.

Early peripheral perfusion and haemodynamic changes

Baseline CRT was available in 198 (99.0%) patients and exceeded 3 seconds in 105 (53.0%) of those assessed. Among paired

observations, CRT and lactate decreased over six hours while peripheral perfusion index increased; the overall mottling-score distribution did not materially change. Detailed paired estimates are shown in Table 2.

Table 2. Early changes in peripheral perfusion, haemodynamics, lactate, and SOFA

Variable	Paired n	Baseline	Follow-up assessment	Test statistic	p-value
Capillary refill time, s	191	3.1 [2.3–3.9]	2.8 [1.9–3.6]	W=3192.0	<0.001
Mottling score	179	0.0 [0.0–1.5]	0.0 [0.0–2.0]	W=976.0	0.805
Peripheral perfusion index	171	1.91 [1.19–2.91]	2.43 [1.44–3.75]	W=2261.5	<0.001
Lactate, mmol/L	187	3.42 [2.59–4.37]	2.72 [1.91–3.49]	W=499.0	<0.001
MAP, mmHg	195	76.2 ± 6.1	77.4 ± 7.6	t=4.15	<0.001
SOFA score*	185	7.0 [5.0–9.0]	6.0 [4.0–9.0]	W=3374.5	<0.001

*SOFA comparison is restricted to patients with both baseline and 48-hour SOFA totals. Wilcoxon signed-rank tests were used for baseline-to-six-hour changes in CRT, mottling, PI, and lactate, and for baseline-to-48-hour change in SOFA; a paired t test was used for baseline-to-six-hour change in MAP. Six-hour measurements were unavailable for patients who died before the six-hour assessment.

Baseline perfusion parameters and 28-day mortality

Day-28 vital status was known for 196 (98.0%) patients, of whom 47 (24.0%) died. Baseline CRT was associated with mortality in the unadjusted analysis, but the association attenuated after adjustment for age, baseline SOFA, and shock aetiology (OR 1.40 per 1-second increase, 95% CI 0.92–2.14; z=1.56, p=0.118). Baseline mottling score and peripheral perfusion index were not independently associated with mortality in their corresponding adjusted models. The six-hour landmark analyses likewise did not show clear

independent associations between changes in CRT, mottling, or PI and subsequent day-28 mortality (Table 3; Figure 1).

Table 3. Association of baseline and six-hour peripheral perfusion measures with 28-day mortality

Model/exposure	n / deaths	Adjusted OR	95% CI	z	p-value
Crude baseline CRT, per 1 s	194 / 46	1.92	1.35–2.72	3.66	<0.001
Baseline CRT, per 1 s	190 / 45	1.40	0.92–2.14	1.56	0.118
Age, per 10 years	190 / 45	0.95	0.76–1.20	-0.42	0.673
Baseline SOFA, per 1 point	190 / 45	1.27	1.08–1.49	2.97	0.003
Cardiogenic vs septic/distributive shock	190 / 45	2.38	0.93–6.07	1.81	0.070
Other/mixed vs septic/distributive shock	190 / 45	1.02	0.41–2.53	0.05	0.960
CRT + baseline lactate sensitivity model, CRT per 1 s	190 / 45	1.32	0.84–2.07	1.21	0.226
Baseline mottling score, per 1 point	186 / 44	1.25	0.89–1.75	1.30	0.193
Baseline PI, per doubling	180 / 41	0.96	0.61–1.52	-0.16	0.873
Six-hour landmark: CRT change, per +1 s	183 / 43	1.76	0.87–3.55	1.57	0.117
Six-hour landmark: Mottling change, per +1 point	171 / 40	1.44	0.80–2.58	1.22	0.222
Six-hour landmark: PI change, per +1 unit	164 / 37	0.63	0.36–1.12	-1.58	0.115

The primary adjusted model included baseline CRT, age, baseline SOFA, and shock aetiology (septic/distributive reference; cardiogenic; other/mixed). Secondary baseline-marker models used the same core adjustment set. The lactate sensitivity model additionally included

baseline lactate. Six-hour landmark models included the change variable, its baseline value, age, baseline SOFA, and shock aetiology; patients dead before six hours were excluded. PI was analyzed per doubling at baseline and per +1-unit absolute change longitudinally.

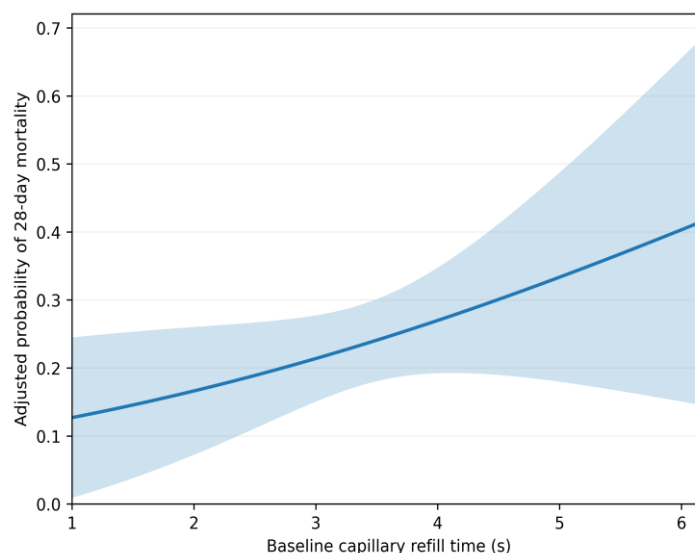


Figure 1. Adjusted marginal probability of 28-day mortality across baseline capillary refill time.

The line represents predictions from the primary logistic model adjusted for age, baseline SOFA, and shock aetiology; the shaded band denotes the 95% confidence interval.

Peripheral perfusion and 48-hour organ dysfunction

Ten (5.0%) patients died before 48 hours and were not assigned a follow-up SOFA score. A 48-hour SOFA total was available in 189 (94.5%) patients. After baseline SOFA

adjustment, baseline CRT showed a modest association with higher 48-hour SOFA that did not reach statistical significance (B=0.27 per second, 95% CI -0.05 to 0.58; t=1.67, p=0.095). In contrast, worsening CRT and

mottling over six hours and failure of PI to improve were independently associated with greater 48-hour organ dysfunction (Table 4; Figure 2).

Table 4. Associations of peripheral perfusion measures with 48-hour SOFA score

Exposure	n	B	Robust SE	95% CI	t	P-value	R ²
Baseline CRT, per 1 s	184	0.27	0.16	-0.05–0.58	1.67	0.095	0.655
Baseline mottling score, per 1 point	180	0.18	0.14	-0.10–0.45	1.28	0.202	0.650
Baseline PI, per doubling	174	-0.33	0.17	-0.66–0.00	-1.94	0.052	0.664
CRT change, per +1 s	179	1.47	0.21	1.06–1.89	6.96	<0.001	0.761
Mottling change, per +1 point	168	0.82	0.25	0.32–1.32	3.22	0.001	0.717
PI change, per +1 unit	160	-0.65	0.16	-0.97–-0.33	-3.96	<0.001	0.741

Linear regression estimates use HC3 heteroskedasticity-robust standard errors. Baseline-marker models adjust for baseline SOFA, age, and shock aetiology. Change models additionally adjust for the

corresponding baseline perfusion marker. Positive CRT and mottling changes indicate worsening; positive PI change generally indicates improvement.

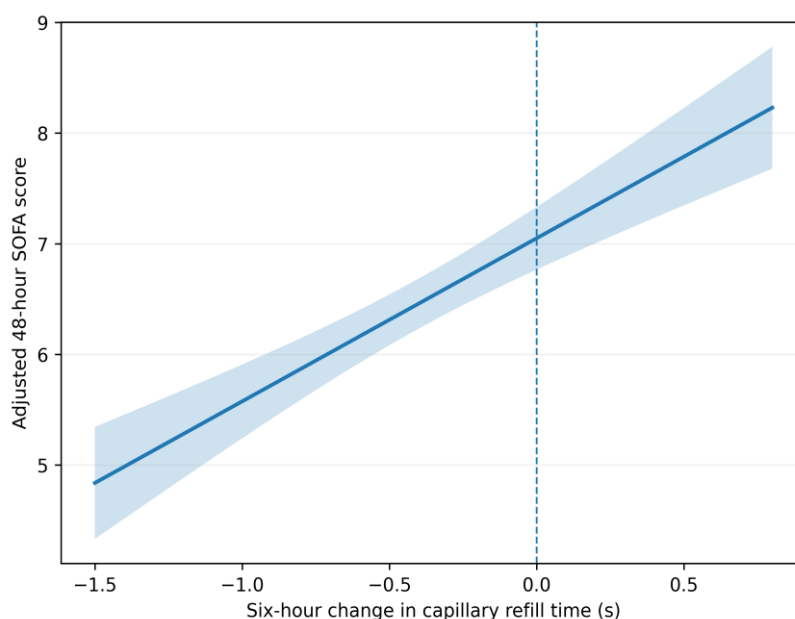


Figure 2. Adjusted 48-hour SOFA score according to six-hour change in capillary refill time.

Positive change indicates prolongation of CRT (worsening). The line represents model-adjusted mean SOFA and the shaded band its 95% confidence interval; the vertical dashed line denotes no change.

Clinical outcomes and follow-up

ICU mortality occurred in 38 (19.0%) patients and hospital mortality in 46 (23.0%). Mechanical ventilation was used at some point in 135 (67.5%), and 29 (14.5%) received new renal replacement therapy. Four (2.0%) patients had incomplete day-28 vital-status follow-up. Additional outcome and follow-up details are provided in Table 5.

Table 5. Clinical outcomes and follow-up completeness

Outcome / follow-up item	Result
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Day-28 follow-up complete	196 (98.0%)
Day-28 all-cause mortality	47 (24.0%)
Day-28 vital status unknown	4 (2.0%)
Deaths before six hours	2 (1.0%)
Deaths before 48 hours	10 (5.0%)
48-hour SOFA available	189 (94.5%)
ICU mortality	38 (19.0%)
Hospital mortality	46 (23.0%)
Mechanical ventilation at any time	135 (67.5%)
New renal replacement therapy	29 (14.5%)
Mechanical circulatory support after baseline	1 (0.5%)
ICU length of stay, days	4.13 [2.84–6.41]
Hospital length of stay, days	9.64 [6.90–13.16]

Day-28 mortality is reported among participants with known day-28 vital status. Length-of-stay values are median [IQR].

DISCUSSION

In the present study, peripheral perfusion abnormalities were found in patients with circulatory shock, with their prognostic value varying between baseline and serial evaluation. Baseline CRT was found to be associated with 28-day mortality in unadjusted analysis, but this association was dampened when SOFA, age, and shock aetiology were adjusted for. Baseline mottling and PI were also not independently related to mortality. Conversely, deterioration of CRT and mottling, and failure of PI to improve after 6 h were independently associated with increased 48-hour organ dysfunction. The results demonstrate that early perfusion trends could be more useful than single baseline values.

This interpretation is supported by the study of Hernández et al., which showed in 41 patients with sepsis-related circulatory dysfunction that normalization of the CRT and peripheral temperature gradients at 6 hours was associated with successful resuscitation at 24 hours, with CRT normalizing earlier than several metabolic variables [9]. Lara et al. also demonstrated that the initial CRT value had less prognostic significance than persistent abnormal CRT, with adverse outcomes being seen in 88% of patients with persistently abnormal CRT versus 20% with normalized CRT, and hospital mortality of 63% versus 9%, respectively [10]. These data are very similar to our data, in which changes in CRT were more strongly associated with subsequent SOFA than was the baseline value.

There is less consistent evidence on mottling. Dumas et al. found that a one-point rise in mottling score was independently associated with 14-day mortality in septic patients (OR 2.26), with improvement in mottling score

during resuscitation being associated with a better outcome [11]. On the other hand, Ferraris et al. demonstrated that mottling was associated with a decrease in skin temperature in septic shock, but that neither mottling score nor skin temperature was independently associated with 28-day mortality [12]. In our heterogeneous shock cohort, mottling was not independently associated with mortality; however, the increase in mottling over six hours was associated with a 0.82-point increase in the adjusted 48-hour SOFA score per score point. Variation among studies may be due to differences in shock etiology, vasopressor use, and assessment time.

The results of the PI also supported serial measurement over a single measurement. He et al. reported that septic nonsurvivors had lower PI and that a PI <0.2 was predictive of mortality at a similar level as lactate [13]. Recently, Chi et al. showed that both six- and 12-hour PI values were independent predictors of 28-day mortality, with AUCs of 0.802 and 0.945, respectively [14]. Although baseline PI was not independently associated with mortality in our study, a one-unit increase in PI over six hours was associated with a 0.65-point reduction in adjusted 48-hour SOFA. This is consistent with the notion that better peripheral pulsatile flow is associated with early good physiological recovery.

The direct relationship between CRT and microcirculatory function has been demonstrated. Contreras et al. reported very low skin blood flow in septic-shock patients with abnormal CRT and a strong association between measured skin blood flow and CRT ($R^2=0.76$) [15]. These findings reinforce the hypothesis that CRT offers more than just information on systemic arterial pressure and

can reflect clinically relevant microvascular dysfunction.

Peripheral perfusion also seems relevant outside septic shock. Merdji et al. analyzed 61 patients with cardiogenic shock and found that a CRT >3 seconds was strongly associated with the composite of 90-day mortality or extracorporeal circulatory support (HR 12.38). The inclusion of CRT in the CardShock score improved the prognostic discrimination to an AUC of 0.93 [16]. The inclusion of cardiogenic and other non-septic shock states in the present study therefore increases clinical generalizability but may also have diminished associations seen in more homogeneous groups.

The association with mortality is weaker than with subsequent SOFA, which is clinically plausible. Mortality is related to perfusion as well as age, baseline organ dysfunction, shock mechanism, comorbidities, treatment response, and downstream interventions. Conversely, 48-hour SOFA is closer in time to the initial perfusion insult. In a meta-analysis of 1667 patients with sepsis or septic shock, peripheral perfusion assessment had a pooled sensitivity of ~70% and pooled specificity of 75.9% for mortality, suggesting that it was a useful but incomplete prognostic discriminator [17]. Therefore, peripheral perfusion measures should be used in conjunction with, and not in place of, existing severity measures.

The present study has a number of strengths: it is a prospective study, it includes multiple shock phenotypes, it evaluates multiple parameters (CRT, mottling, and PI) simultaneously, and it has repeated measurements within a defined 6-hour window. The landmark approach also minimized bias due to patients who died prior to reassessment. The limitations are observational design, single-centre setting, the possibility of measurement variability of CRT and mottling, the dependence of PI on the device, and the lack of repeat measurements in patients who deteriorated early. The complexity of adjusted mortality models was also restricted by the number of deaths.

Overall the results show that dynamic changes in peripheral perfusion in the first hours of shock management are more closely associated with evolving organ dysfunction than are single baseline measurements. Therefore, serial CRT, mottling and PI assessment may be useful adjunctive data to consider when assessing early response to resuscitation.

CONCLUSION

Baseline peripheral perfusion measurements were less strongly associated with subsequent organ dysfunction than were dynamic changes in peripheral perfusion during the early phase of circulatory shock. Worsening of capillary refill time and mottling, in addition to failure of peripheral perfusion index to improve, were each independently associated with increased 48-hour SOFA scores. Therefore, serial bedside perfusion assessment may be helpful in providing complementary information in early shock resuscitation.

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