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Stroke Volume and Stroke Volume Variation, but not Cardiac Index Is Associated With Survival of Majorly Burned Patients in Early Burn Shock

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Abstract

Adequate fluid therapy is crucial to maintain organ function after burn trauma. Major burns lead to a systemic response with fluid loss and cardiac dysfunction. To guide fluid therapy, measurement of cardiac pre- and afterload is helpful. Whereas cardiac function is usually measured after admission to intensive care unit (ICU), in this study, hemodynamic monitoring was performed directly after arrival at hospital. We conducted a prospective cohort study with inclusion of 19 patients (male/female 13/6, 55 ± 18 years, mean total body surface area 36 ± 19%). Arterial waveform analysis (PulsioFlexProAqt®, Getinge) was implemented immediately after admission to hospital to measure cardiac pre- and afterload and to guide resuscitation therapy. Cardiac parameters 3.75 (2.67-6.0) h after trauma were normal regarding cardiac index (3.45 ± 0.82) L/min/m², systemic vascular resistance index (1749 ± 533) dyn sec/cm^5 m^2, and stroke volume (SV; 80 ± 20) mL. Stroke volume variation (SVV) was increased (21 ± 7) % and associated with mortality (mean SVV survivors vs nonsurvivors 18.92 (±6.37) % vs 27.6 (±5.68) %, $P = .017$). Stroke volume was associated with mortality at the time of ICU-admission (mean SV survivors vs nonsurvivors 90 (±20) mL vs 50 (±0) mL, $P = .004$). Changes after volume challenge were significant for SVV (24 ± 9 vs19 ± 8%, $P = .01$) and SV (68 ± 24 vs 76 ± 26 mL, $P = .03$). We described association of SVV and SV with survival of severely burned patients in an observational study. This indicates high valence of those parameters in the early postburn period. The use of an autocalibrated device enables a very early monitoring of parameters relevant to burn shock survival.

Keywords

intensive care unit, cardiac index, hemodynamic monitoring, resuscitation, volume status, severe burn injury, arterial waveform analysis, burn shock

Introduction

Severe burn injury leads to burn shock, a systemic inflammatory response with immense fluid and protein loss into surrounding tissues due to capillary leak.[1] It also causes relevant myocardial depression and influences cardiac output (CO) as well as pulmonary- and systemic vascular resistance.[2] The known changes in cardiac function during burn shock appear as a decrease in CO, stroke volume (SV), venous return, mean arterial pressure (MAP), and stroke work.[3] In addition, the occurring severe intravascular hypovolemia is followed by hypoperfusion and microcirculatory impairment.[4,5] Without adequate fluid resuscitation, this leads to organ dysfunction, but fluid overload is also associated with deleterious outcomes.[6,7] To guide this therapy, comprehensive monitoring addressing disturbance of both macro- and microcirculation is elementary.

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For individual, patient-adjusted fluid resuscitation, early insights into volume capacity and fluid responsiveness are highly relevant. A common way to measure cardiac pre- and afterload in burn care medicine is to initiate enhanced hemodynamic monitoring after patient admission to the intensive care unit (ICU) to guide fluid resuscitation. According to literature, this measurement typically starts 6 to 12 h after trauma.[8–10]

To improve resuscitation, it is important to consider the relationships between fluid responsiveness and cardiac dysfunction. There are several studies describing hemodynamic alterations and myocardial performance during burn shock period.[3,8,11] Parameters of cardiac pre- and afterload seem to be relevant for outcome after burn trauma. Initial low SV and low CO, 6 h after trauma, are shown to be associated with mortality.[12,13] Despite this knowledge, we mainly must rely on experimental studies in animals to describe hemodynamic changes in the very early period of burn shock.[14–16] Increased vascular permeability, caused by interendothelial gaps, lipid peroxidation, and loss of intercellular adhesion,[17] is described to appear rapidly after trauma with an abrupt increase of tissue water content.[18–20] The decrease in CO and reduced myocardial contractility is registered immediately after burn trauma.[14,21] As cardiac depression starts immediately and capillary leak peaks 2 to 4 h after burn trauma, information about patient myocardial function and fluid responsiveness should be achieved as soon as possible to improve clinical care.[1,2,14,15,17]

In this study, we used an autocalibrated enhanced hemodynamic monitoring system (PulsioFlexProACT®) to determine fluid responsiveness and cardiac function directly after burnt patients were admitted to hospital. Parameters of cardiac pre- and afterload were measured during initial wound debridement even before ICU admission. In a secondary analysis of data published previously,[22] we describe the very early course of hemodynamic after burn trauma in a prospective cohort study and its changes during fluid resuscitation. Furthermore, we investigated the prognostic value of cardiac pre- and afterload parameters to patient outcome.

Materials/Methods

Patients

We conducted an observational, prospective cohort study of patients admitted between August 2020 and June 2021 with major burn injuries to the burn center of our hospital (BG Klinikum Unfallkrankenhaus Berlin, a supraregional trauma center of the German Federal Statutory Accident Insurance). Approval from the local ethics committee (Aerztekammer Berlin, 20/05/07, Eth-04/20) was obtained, and the study was registered (20/05/26, DRKS00020826, UTN U1111-1248-8066). We included patients with burn injury affecting ≥20% total body surface area (TBSA) II-IV°, age ≥18 years and first debridement and hydrotherapy in general anesthesia with mechanical ventilation. Exclusion criteria were age <18 years, pregnancy, atrial fibrillation, severe heart insufficiency, or other reasons preventing sufficient arterial waveform analysis. Patients with toxic epidermal necrolysis were also excluded. Informed consent was obtained from the patients, their nearest relative or legal guardian.

After hospital admission, patients were transferred directly to the burn operating theatre. All patients received general anesthesia, mechanical ventilation, and a jugular central venous catheter, as well as an invasive arterial blood pressure measurement. After patient's weight determination, PulsioFlexProAQT®-sensor and the PiCCO®-Monitor were connected. We documented the first acquired cardiac parameters following hospital admission. Fluid therapy was guided by the Parkland formula and was adjusted to standard parameters such as heart rate (HR), hourly urinary output, base excess, and lactate levels, as well as parameters of enhanced hemodynamic monitoring (cardiac index [CI], SV variation [SVV], and systemic vascular resistance index [SVRI]). Stroke volume was calculated, using captured CI and HR. Goal parameters for resuscitation were defined ($CI \geq 2.5$ L/min/m², $SVV \leq 20\%$, $SVRI \geq 1250$ dyn sec/cm^5 m^2, hourly urine output 0.5 to 1 mL/kgKG/h, $BE > -2$, $Lactate < 2$ mmol/L, and $HF < 120$/min) according to the German burn resuscitation guidelines.[23] After the first surgical wound treatment, patients were transferred to the burn ICU. Enhanced hemodynamic monitoring was continued for 24 h or until the patient reached spontaneous breathing. In addition to maintaining fluid infusion at fixed rates, at clinician's discretion volume challenges were possible. We documented hemodynamic parameters before and after fluid bolus which was administered over 60 min. We analyzed extra bolus applications over 1000 mL in 1 h. Study parameters were documented at the time of hospital admission (T0), after reaching the burn operating theatre (T1), before first wound cleaning and scrub (T2), at admission to ICU (T3) and after 6, 12, 18, and 24 h (T3.1-T3.4).

Statistics

Patient charts from the hospital and the ICU data management system Medico (Cerner Health Services®) and ICM (Dräger®) were reviewed. Data extraction was performed in a pseudonymous way. After digitalization in Microsoft Excel, rigorous plausibility checks were conducted before the data were imported to SPSS® (Version 27, IBM).

If not indicated differently, metric study parameters were presented as an average with standard deviation. For group comparison, Student *t* test was performed when normal distribution and equality of variance was present (Shapiro-Wilk test and Levene-test). If either requirement was not met, we used the nonparametric Mann-Whitney *U* test. For connected variables with intraindividual measurement at 3 or more time points, we used 2-factor analysis of variance for ranks according to Friedman, for analysis of 2 connected samples Wilcoxon signed-rank test was used. To indicate the discriminatory ability of parameters we used receiver operating curves. The area under the curve is given with 95% confidence intervals and significance testing was performed with $AUROC = 0.5$ as null hypothesis.

Categorical variables were shown as counts and percentages, a test of significance was then performed with the χ^2 test.

Two-sided *P* values of <.05 were considered statistically significant.

Results

Twenty patients were included in the study. Due to missing informed consent in one case, 19 patients were analyzed (male/female 13/6, mean age 55 ± 18 years, mean TBSA 36 ± 19%, mean body mass index 28 ± 5 kg/m^2). Sixteen patients suffered from flame injury, in 3 cases from scald-type burns. Demographic data and data at admission are shown in Tables 1 and 2. In 12 patients, enhanced hemodynamic monitoring was implemented immediately after admission to burn center, in 7 patients first documented measurement was slightly later due to technical difficulties.

We started measurement of hemodynamic parameters, cardiac pre- and afterload, as well as fluid responsibility in median 3.75 (2.67-6.0) h after burn trauma.

Cardiac index at admission was 3.45 ± 0.82 L/min/m^2. Initial SVV was 21 (±7) %, SV (80 ± 20) mL, SVRI 1749 ± 533 dyn sec/cm^5 m^2, and HR 94 ± 27 (Table 3). Heart rate significantly decreased from time of admission to hospital until ICU admission (94 ± 27 vs 86 ± 22 bpm, *P* = .001) and significantly increased from time to admission ICU to 18 h (86 ± 22 bpm vs 89 ± 23, *P* = .03) and 24 h (86 ± 22 vs 92 ± 22 bpm, *P* = .01) after trauma (Table 4).

Table 1. Demographic Data.

Number (%) or Mean ± SD	Study group (*n* = 19)
Sex (%)	
Female	6 (31.6)
Male	13 (68.4)
Age (years)	55 ± 18
TBSA (%KOF)	36 ± 19
Inhalation Injury	7 (36.8%)
Third degree (%)	10 (52.6)
Fourth degree (%)	1(5.3)
BMI (kg/m^2)	28.1 ± 5.3
rBaux-Index	99 ± 30
ABSI-Score	9 ± 3

Abbreviations: TBSA, total body surface area; BMI, body mass index; rBaux-Index, revised Baux-Index.

Table 2. Data at Time of Admission to Hospital.

Number (%) or Mean ± SD	Study group (*n* = 19)
pH	7.31 ± 0.12
BE (mmol/L)	3.15 ± 4.86
Lactate (mmol/L)	2.2 ± 1.7
HR (bpm)	94 ± 27
MAP (mm Hg)	95 ± 32
Prehospital applied fluid (mL)	1607 ± 882

Abbreviations: MAP, mean arterial pressure; HR, heart rate.

Patients who survived until at least 28d had initially a significant lower SVV (mean SVV survivors vs nonsurvivors 19 (±6) % vs 29 (±6) %, *P* = .007; Figure 1). Also, lower SV at the time of ICU-admission, 6.04 ± 1.77 h after trauma, was associated with mortality after 28d (mean SV survivors vs nonsurvivors 90 (±20) mL vs 50 (±0) mL, *P* = .004; Figure 2). There was no difference in body surface area (BSA) between the 2 groups (mean BSA survivors vs nonsurvivors 2.08 (±0.3) m^2 vs 2.10 (±0.16) m^2, *P* = .86).

At this time point, HR showed a significant difference (mean HR survivors vs nonsurvivors 78 (±18) bpm vs 108 (±27) bpm, *P* = .04). Initially measured CI with 3.41 (±0.88) versus 3.57 (±0.77) L/min/m^2 survivors versus nonsurvivors, *P* = .521, Figure 3 and SVRI with 1838 (±573) versus 1618 (±393) dyn sec/cm^5 m^2, survivors versus nonsurvivors, *P* = .521, Figure 4, were no predictors of survival in our cohort.

ROC curves comparing the ability of initial SVV, ABSI, and revised Baux-Index (rBaux-Index) to discriminate 28-day mortality gave areas under curves of 0.88 (CI 0.71-1.04) for SVV, *P* < .01; 0.91 (CI 0.77-1.05) for ABSI, *P* < .01 and 0.92 (CI 0.80-1.05) for rBaux-Index, *P* < .01 (Figures 5 and 6).

Table 3. Earliest Measured Cardiac Pre- and Afterload Parameters, 3.75 h After Trauma.

Mean ± SD	
CI (L/min/m^2)	3.45 ± 0.82
SVV (%)	21 ± 7
SVRI (dyn sec/cm^5 m^2)	1749 ± 533
SV (mL)	80 ± 20

Abbreviations: SVRI, systemic vascular resistance index; SV, stroke volume; SVV, stroke volume variation.

Table 4. Course of Hemodynamic Parameters During Points of Treatment.[a]

Mean ± SD	CI	SVV	SVRI	SV	HR
T0					94 ± 27[b]
T1	3.48 ± 0.89	21 ± 7	1640 ± 413	80 ± 20	92 ± 16
T2	3.64 ± 0.86	21 ± 8	1618 ± 454	80 ± 20	97 ± 18
T3	3.06 ± 0.89	18 ± 8	2148 ± 739	80 ± 20	86 ± 22
T3.1	3.12 ± 0.79	21 ± 10	1818 ± 738	70 ± 20	95 ± 24
T3.2	2.68 ± 0.74	18 ± 10	1468 ± 794	60 ± 20	86 ± 21
T3.3	2.65 ± 0.81	20 ± 10	1340 ± 757	60 ± 20	89 ± 23[b]
T3.4	2.9 ± 0.92	17 ± 8	1325 ± 930	70 ± 20	92 ± 25[b]
P	0.123	0.187	0.470	0.921	*<0.05

Abbreviations: CI, cardiac index in L/min/m^2; SVV, stroke volume variation in %; SVRI, systemic vascular resistance index in (dyn sec/cm^5 m^2); SV, stroke volume in mL; HR, heart rate in bpm; ICU, intensive care unit.

[a] T0: admission to hospital T1: admission to burn center T2: during first wound scrub T3: admission to ICU T3.1: 6 h after trauma T3.2: 12 h after trauma T3.3: 18 h after trauma T3.4: 24 h after trauma.

[b] *P* < .05 for HR: admission to hospital versus ICU admission, 18 h post trauma versus ICU admission, 24 h post trauma versus ICU admission.

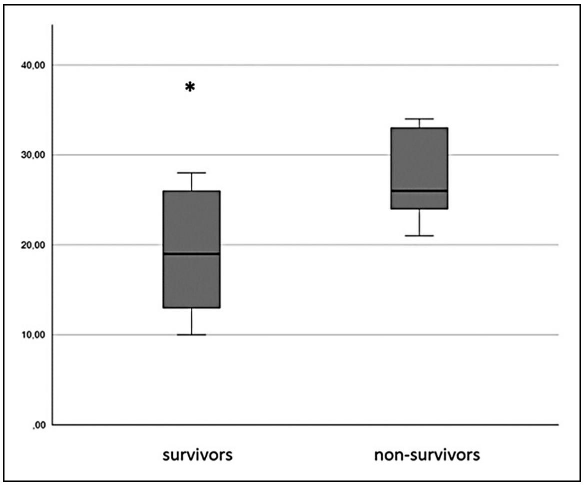

Figure 1. Stroke volume variation as predictor for mortality, x-axis: mortality after 28 d y-axis: stroke volume variation in %, $^{*}P = .007$.

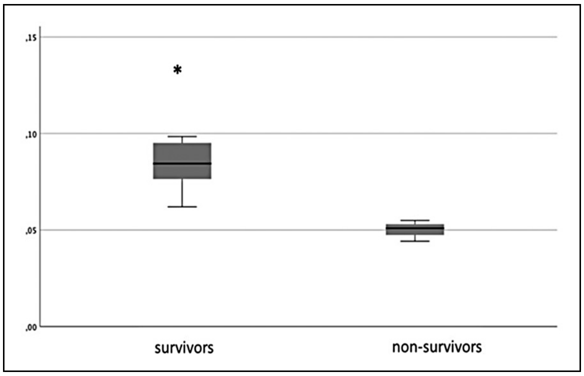

Figure 2. Stroke volume as predictor for mortality, x-axis: mortality after 28 d y-axis: stroke volume variation in mL, $^{*}P = .004$.

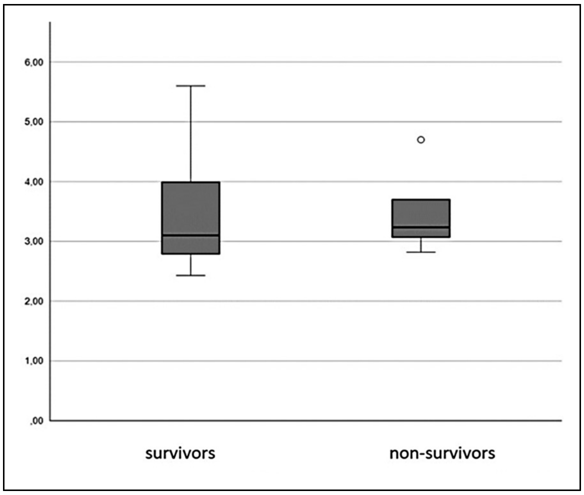

Figure 3. Cardiac index as no predictor for mortality, x-axis: mortality after 28 d y-axis: cardiac index in L/min/m^2, $P = .521$.

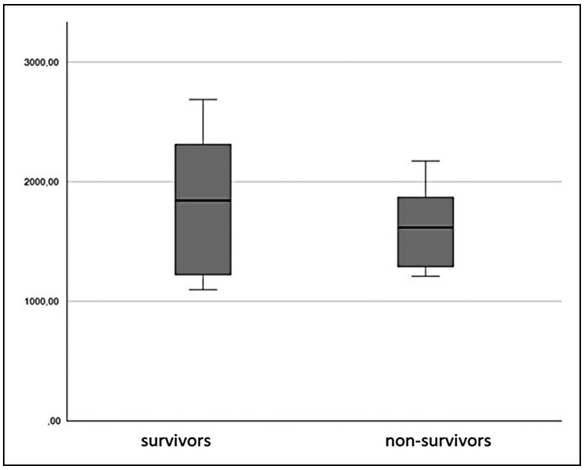

Figure 4. Systemic vascular resistance index as no predictor for mortality, x-axis: mortality after 28 d y-axis: systemic vascular resistance index in dyn sec/cm^5 m^2, $P = .521$.

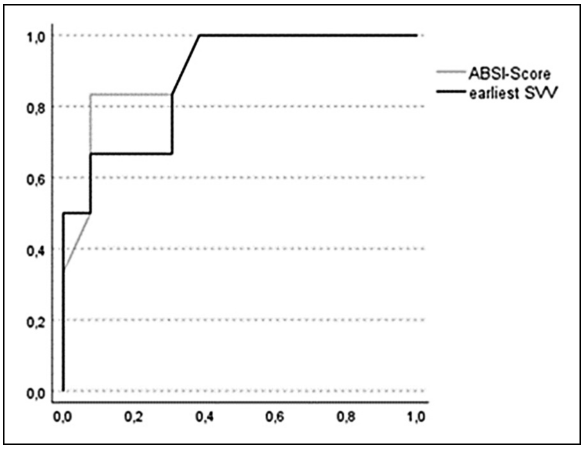

Figure 5. ROC-analysis, describing ability of initial stroke volume variation (SVV) and ABSI-score to discriminate 28-day mortality y-axis: sensitivity, x-axis: specificity.

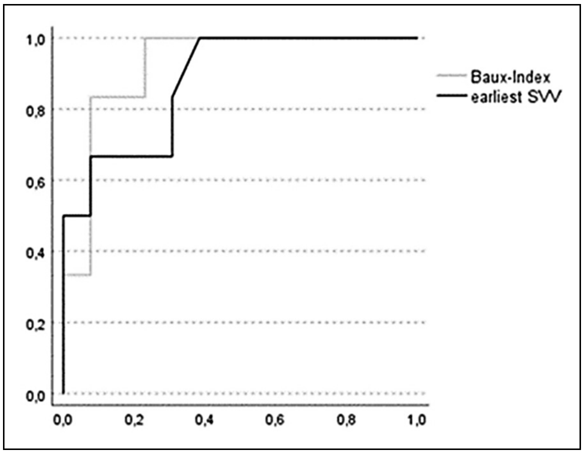

Figure 6. ROC-analysis, describing ability of initial stroke volume variation (SVV) and rBaux-Index to discriminate 28-day mortality y-axis: sensitivity, x-axis: specificity.

Table 5. Change of Hemodynamic Parameters During Volume Challenge.[a]

Mean ± SD	T_{before}	T_{after}	P
HR (bpm)	96 ± 24	87 ± 24	.03
MAP (mm Hg)	71 ± 19	75 ± 14	.25
CI (L/min/m²)	3.00 ± 0.53	3.05 ± 0.77	.43
SVV (%)	24 ± 9	19 ± 8	.01
SV (mL)	68 ± 24	76 ± 26	.03
SVRI (dyn sec/cm^5 m^2)	1877 ± 631	2036 ± 674	.02
Norepinephrine (µg/kg/min)	0.15 ± 0.15	0.14 ± 0.16	.41

Abbreviations: CI, cardiac index; SVRI, systemic vascular resistance index; SV, stroke volume; SVV, stroke volume variation; MAP, mean arterial pressure; HR, heart rate.

[a] T_{before}: data before volume challenge; T_{after}: data directly after administration of 1000 mL crystalloid solution (RAC) in 60 min.

Volume challenge with 1000 mL crystalloid-Bolus in 60 min, in median administered 6.75 (3.62-9.25) h after trauma, showed significant changes of hemodynamic parameters regarding SVV (24 ± 9 vs19 ± 8%, $P = .01$), SV (68 ± 24 vs 76 ± 26 mL, $P = .03$), SVRI (1877 ± 631 vs 2036 ± 674 dyn sec/cm^5 m^2, $P = .02$), and HR (96 ± 24 vs 87 ± 24 bpm, $P = .03$) but not for CI (3.00 ± 0.53 vs 3.05 ± 0.77 L/min/m², $P = .43$) and MAP (71 ± 19 vs 75 ± 14 mm Hg, $P = .25$; Table 5).

There was also no significant association between the initial cardiac performance or fluid responsibility and further outcome parameters like organ failures or organ replacement therapy. Also, all other time points of measurement (T1-T3.4) showed no significant association with outcome parameters. A total of 6 patients (31.6%) died during 28d in the hospital.

Discussion

The measurement of the early hemodynamic changes after severe burn trauma is important for improvement of ongoing fluid resuscitation. To address cardiac depression and fluid deficit adequately, timely information about cardiac pre- and afterload, myocardial function, and fluid responsibility is crucial.

We describe cardiac performance and volume capacity in 19 severely burned patients in median 3.75 h after burn trauma, which is 2 to 8 h earlier than recent clinical publications document cardiac parameters.[12,24–27] Literature researched only revealed one article with similarly quick measurement, Sanchez et al published data in retrospective evaluation with a mean time of 4.2 h after trauma.[28]

In our cohort, we observed CI within normal ranges, normal SVRI and SV, and upper normal values of SVV at the time of admission to burn center. Experimental studies in animals lead to the theory, that cardiac depression starts immediately after burn trauma and lasts at least for 24 h.[2,14,15] Temporal analysis of these events in human burn patients, especially early after burn trauma, has not been largely available. Gong et al described low CI 12 h after trauma with slow postburn recovery until hour 36.[29] Pruitt et al observed low CO already 6 h after trauma but were able to detect earlier recovery after 12 h.[30] In our cohort, we found only a nonsignificant trend toward lower CO starting 12 h after burn trauma, so we are not able to support the results published so far.

Systemic vascular resistance is supposed to rise within 2 h after burn trauma, caused by elevated sympathetic activity and high-level released catecholamines and comes back to preburn level 18 to 24 h postburn.[2,11,31] In our cohort, we saw upper normal values of SVRI at time of admission to ICU with a decreasing almost to reference value levels 24 h after trauma, this result suggests coherence with published experimental and clinical studies.

Like previously published clinical data, small sample sizes and changes in hemodynamic parameters also limited statistical analysis in our study. We could only find trends in CI, SVRI, and SVV which did not reach statistical significance. The demonstrated significant change in HR should also be interpreted cautiously. In summary, there is still a lack of knowledge concerning development of hemodynamic parameters immediately after burn shock outside animal experiments, whereby our data add important information.

To improve our understanding of the relationship between fluid resuscitation and cardiac performance during burn shock, we investigated the changes of hemodynamic parameters after bolus administration of crystalloids. Volume challenge is considered potentially harmful as worsening of tissue edema is possible.[32] Nevertheless, if circulatory instability persists, additional fluid administration may be necessary. It was only used in our study when the attending clinicians considered it not to be avoidable. Lavrentieva et al investigated hemodynamic changes after fluid challenge in a prospective cohort, using thermodilution. A significant change in CI, SVV, and SVRI was described.[27] Our data confirm those findings for SVV and SVRI using autocalibrated monitoring. A significant change in the CI during the volume challenge could not be confirmed in our cohort. This can be explained, supported by animal experimental data, by a very slow change in CO after fluid application and a described independence of cardiac depression from fluid resuscitation.[2,19,33]

The results for systemic resistance need to be interpreted cautiously as a marked variation in noradrenaline dosing under volume challenge was observed. PulsioFlex® is described as a reliable device for tracking fluid-induced changes in nonburned patients.[34] We could describe usefulness of autocalibrated monitoring in a cohort of severely burned patients regarding fluid responsiveness, even though bolus-administration rate was lower than in similar investigations.

Some authors were able to describe associations of lower CI with worse outcomes. Bernard and Holm used cumulated hemodynamic parameters during the first 72 h of treatment, whereas Soussi focussed first measurement of CI 6 h after thermal injury.[12,13,35]

We cannot confirm those results in our prospective cohort with early detected CI 3.75 h after trauma, as it showed no significant association with mortality at this point of measurement or at later time points. This discrepancy can be explained by the timely implementation of enhanced cardiac monitoring in our cohort. In contrast to the studies we did not use thermodilution, but patients were already receiving closely monitored volume and catecholamine therapy at the later time points which might influence hemodynamic parameters. Additional prospective investigations with standardized measurement methods and treatment protocols as well as larger cohort sizes are needed to give insights on the conflicting data regarding CO in early burn shock.

Stroke volume is also supposed to be associated with burn shock survival, with survivors showing higher SV at time of admission than nonsurvivors.[12] We could not find any differences between survivors and nonsurvivors in early measured SV after admission to burn center 3.75 h after trauma, but at time of ICU admission, SV was higher in the group of survivors than in the patients who died. Soussi et al demonstrated this association 6 h after trauma, which is coherent with our data.

Stroke volume variation is previously shown to be a reliable value for fluid responsiveness in ventilated patients with circulatory failure.[36,37] As adequate fluid resuscitation remains the main tool to prevent burn shock deaths, early detection of fluid responsiveness given additional myocardial depression appears to be more important than ever during burn shock. Chiao et al as well as Tokarik et al showed that addressing SVV for goal-directed therapy during fluid resuscitation leads to less unnecessary fluid administration and optimizes resuscitation.[24,38] In a prospective cohort study, Lavrentieva et al showed SVV as valuable to predicting fluid responsiveness in severely burned patients with a significant decrease under fluid administration.[27]

In our cohort, in the group of burn trauma survivors initially measured SVV was lower than in nonsurvivors. This could allow the interpretation of increased SVV as an early marker of burn shock severity with pronounced vascular permeability and high fluid loss and reveal the crucial importance of this parameter in the early treatment period. Early measurement of SVV as a predictor of fluid responsiveness and mortality can give important additional information about individual compensation. It enables the attending physicians to guide resuscitation therapy and react to the individual fluid capacity even before ICU admission.

To describe the discriminative capability of SVV regarding mortality, we performed ROC analysis with SVV as a new marker and established scores predicting burn-caused mortality such as ABSI and rBaux-Index.[39,40] Stroke volume variation exhibited a good discriminatory ability but it should only be used additionally in predicting patient survival. Generally, one should only use SVV prognostically when it is combined with clinical context and well-established predictive scores such as ABSI and rBaux.

Limitations of this study are the small group and the heterogeneity of patients. For a robust statistical analysis, a larger study group is necessary. Whether the clinician's early reaction to the acquired additional hemodynamic information translates to patient-centered outcome improvement should be investigated in randomized controlled fashion. Another limiting factor is the use of autocalibrated monitoring for CO measurement. There are reported deviations, regarding absolute values of CO between devices with autocalibration and methods using calibration via thermodilution, which restrict the interpretation of absolute values. However, as the ability to implement a complex device and perform calibration with thermodilution during admission, initial care and wound cleansing is limited and the usefulness of autocalibrated devices in fluid resuscitation has been demonstrated,[34,41] the use of an arterial waveform analysis is a viable alternative. A further limitation of the study is the exclusion of patients with atrial fibrillation or severe valvular abnormalities, as it is precisely these patients who might benefit from precise hemodynamic monitoring. Since the dynamic parameters of volume responsiveness such as SVV in patients with atrial fibrillation do not allow reliable conclusions to be drawn from the pulse contour analysis, a different procedure must be recommended for future studies. Possibly the use of transthoracic echocardiography to determine CO in response to fluid administration, combined with the course of metabolic parameters such as lactate and base excess is of additional value in the challenging cohort of patients with underlying cardiac disease.

Conclusion

Our data provide supplementary information on hemodynamic parameters and fluid capacity in burn shock. It was already known from animal and clinical studies, that cardiac dysfunction and the individualized fluid administration following burn trauma is of high relevance. We can confirm the finding of altered hemodynamic parameters and specifically add information on the hemodynamic state of adult burn patients in their very early postburn period. In our cohort, cardiac dysfunction measured before ICU admission was not severely altered and not associated with mortality, while SVV and SV helped predict patient survival. To prove the hypothesis, that earlier detection of alterations in CO and fluid responsibility and quick adjustment of therapy can improve survival, further randomized controlled trials are necessary.

Declaration of Conflicting Interests

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