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Biochemical Profile and Outcomes in Trauma Patients Subjected to Open Cardiopulmonary Resuscitation: A Prospective Observational Pilot Study

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Abstract

Background The predictive factors to regain a heartbeat following emergency department resuscitative thoracotomy (EDT) for trauma are poorly understood. The objective of the present study was to prospectively assess the electrolyte profile, coagulation parameters, and acid-base status from intracardiac blood samples in trauma patients subjected to open cardiopulmonary resuscitation (CPR) in the presence of established cardiac arrest.

Methods All patients who underwent EDT following trauma were considered for inclusion. Prior to the injection of any resuscitative medications, a sample of intracardiac blood from the right ventricle was obtained for analysis.

Results During the study period, a total of 22 patients had intracardiac blood samples obtained and were eligible for analysis. Twelve patients never regained cardiac activity, and 10 patients transiently regained a heartbeat for a mean of 51 ± 69 min, but ultimately died. Some 91 % (20/22) of patients presented with severe acidosis (pH < 7.20). The pCO_2 was <45 mmHg in 68 % (15/22) of patients, and the pO_2 level was >75 mmHg in 77 % (17/22) of patients. Patients who never regained cardiac activity had a significantly higher lactate level than those with a return of cardiac rhythm (17.1 ± 2.6 vs. 10.6 ± 4.9 mmol/L, $p = 0.018$). The sodium and potassium levels were higher for those who never regained a rhythm than for those who did regain a pulse (sodium: 155 ± 14 vs. 147 ± 9 mmol/L, $p = 0.094$; potassium: 6.0 ± 1.1 vs. 4.6 ± 1.0 mmol/L, $p = 0.014$). Severe hyperkalemia (potassium > 5.5 mmol/L) occurred significantly more often in patients who did not regain a heart beat ($p = 0.030$). Coagulopathy (INR > 1.2 and/or prothrombin time >15 s and/or platelet count <100,000/μL) was noted in 96 % of patients.

Conclusions Most patients undergoing open CPR have normal blood gas levels. Severe lactic acidosis, hyperkalemia, and hypernatremia are associated with decreased probability for return of cardiac function. Calcium and magnesium levels were not significantly different between the two groups, making the therapeutic role of these electrolytes very questionable.

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Introduction

Emergency Department resuscitative thoracotomy (EDT) and open cardiopulmonary resuscitation (CPR) can be a life-saving procedure for patients suffering traumatic cardiac arrest [1–4]. We have previously shown that return of cardiac activity can be achieved in approximately one third of patients by open CPR [5]. However, the vast majority of patients subsequently die during the post-resuscitation phase. After successful initial open CPR, many of these patients re-enter into ventricular fibrillation or go into asystole. The reasons causing these delayed cardiac lethal arrhythmias or asystole are unknown.

Knowledge of the biochemical pathophysiology of trauma patients suffering cardiac arrest could affect the management of these patients. Only scant literature exists concerning non-traumatic cardiac arrest victims with severe lactic acidosis, hyperkalemia, hypochloremia, and hyperphosphatemia [6, 7]. In victims of traumatic cardiac arrest, hypocalcemic states were described by one investigator; however, its impact on survival remains uncertain [8]. Although various drugs and interventions have been studied to improve survival and neurological outcome after CPR, the survival remains poor, and data on traumatic cardiac arrest victims are still lacking [9, 10].

The objective of this pilot study was to prospectively assess the serum electrolyte levels and coagulation parameters, along with the acid-base status from intracardiac blood samples taken from trauma patients subjected to open CPR with established cardiac arrest, and to correlate any abnormalities with the outcome of the initial resuscitation.

Materials and Methods

Study Design and Setting

After Institutional Review Board (IRB) approval, a prospective observational study was conducted at the Los Angeles County + University of Southern California Medical Center (LAC + USC). The study period extended from 9/1/2008 to 4/30/2010. At the LAC + USC Medical Center, an aggressive policy of EDT is in place. Injured patients who arrive in the resuscitation area with lost cardiac activity undergo immediate resuscitative thoracotomy, irrespective of injury mechanism, time of cardiac arrest, and injured body area. A left-sided thoracotomy through the 4th or 5th intercostal space is performed, followed by opening of the pericardial sac, cardiac massage, and clamping of the thoracic aorta. In case the heart does not start after the first few manual compressions, epinephrine, vasopressin, atropine, bicarbonate, and/or calcium is injected directly into the heart or through the central venous line, at the discretion of the attending trauma surgeon. The endpoint of open CPR is a visible beating heart.

Selection of Patients

All patients who underwent resuscitative thoracotomy from 9/1/2008 to 4/30/2010 were considered for inclusion in the present study. Before injecting any resuscitative medications into the heart, blood was aspirated to ensure the intracardiac location of the tip of the needle. For study purposes, approximately 20 mL of blood was obtained from the right ventricle (two EDTA tubes, one citrate tube, and one blood gas tube). For the purposes of the present study, only patients who required intracardiac application of any resuscitative medications and with intracardiac blood samples were included for further analysis. Patients who did not have intracardiac blood drawn or in whom cardiac activity was restored without intracardiac drug administration were excluded from further analysis. This group of patients regained spontaneous cardiac activity, and an intracardiac blood sample solely for study purposes was not drawn due to ethical concerns.

Data Collection and Processing

The following variables were collected for all patients who underwent resuscitative thoracotomy: age, gender, mechanism of injury, vital signs on admission (blood pressure, heart rate), glasgow coma scale (GCS) score, injury characteristics, including source of bleeding, location where the patient experienced cardiac arrest (on scene, en route, emergency department), duration of closed CPR prior to admission, restoration, and duration of cardiac activity after resuscitative thoracotomy.

The intracardiac blood samples were analyzed for chemistry, coagulation, and basic hematological parameters. In addition, a blood gas analysis was performed.

To detect any selection bias, a sensitivity analysis was performed by comparing the demographics, injury characteristics, and clinical outcomes of the excluded patients and the included patients. Subsequently, the study population was divided into those patients who regained cardiac rhythm and those who did not. Again, demographics and injury characteristics were compared between these two groups. In addition, the results of the analyses of the intracardiac blood samples were compared between these two groups.

Primary Data Analysis

Data were entered into a computerized spreadsheet (Microsoft Excel 2003, Microsoft corporation, Redmond, WA), and all statistical analysis was performed with the Statistical Package for Social Sciences (SPSS Windows), version 16.0 (SPSS Inc., Chicago, IL).

Continuous and categorical variables are reported as means ± standard deviation (SD), and percentages, respectively. The p values were obtained from the chi-square test or Fisher's exact test for proportions and the Mann–Whitney U test for continuous variables.

Results

During the 20 month study period, a total of 108 patients underwent resuscitative thoracotomy. Return of heart rhythm was achieved in 47 patients (43.5 %) (Fig. 1). A comparison of the characteristics of patients who regained a heartbeat and those who never showed any cardiac activity is shown in Table 1.

Overall, nine patients (8.3 %) survived to the intensive care unit, and three of them (2.8 %) eventually survived to

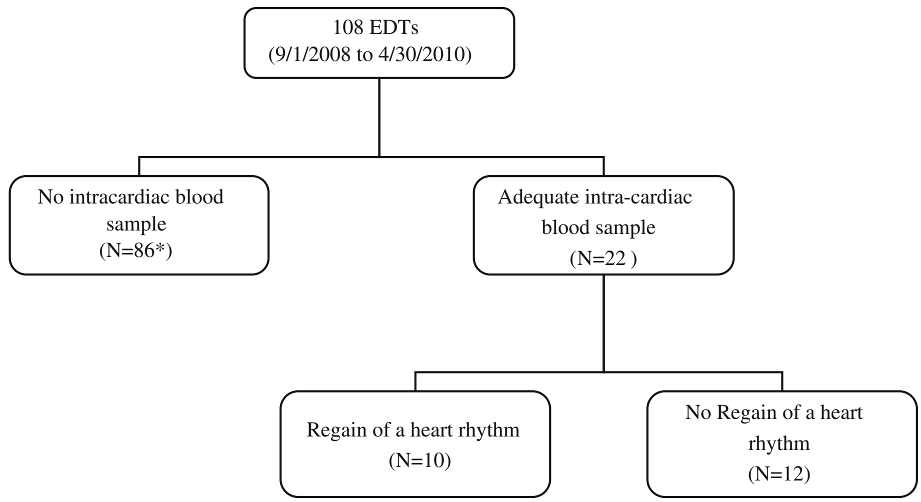

Fig. 1 Study outline. * No intracardiac blood sample was available because a peripheral blood sample was obtainable ($n = 45$), restoration of a heartbeat occurred without intracardiac drug administration ($n = 13$), there was not enough intracardiac blood or the samples were clotted or contaminated with air ($n = 6$), other reasons ($n = 22$)

Table 1 Comparison of the patients who regained cardiac activity to those who never regained any cardiac activity

	Return of cardiac activity ($n = 47$)	No return of cardiac activity ($n = 61$)	p value[b]
GSW	31.9 % (15/47)	44.3 % (27/61)	0.192
SW	19.1 % (9/47)	11.5 % (7/61)	0.266
Traffic accidents	36.2 % (17/47)	26.2 % (16/61)	0.266
Blunt severe TBI	12.8 % (6/47)	3.3 % (2/61)	0.062
Penetrating severe TBI	0.0 % (0/47)	4.3 % (2/61)	0.104
Major source of bleeding			
Heart	21.3 % (10/47)	19.7 % (12/61)	0.837
Major thoracic vessels	23.4 % (11/47)	26.2 % (16/61)	0.737
Major abdominal vessels	21.3 % (10/47)	14.8 % (9/61)	0.377
Abdominal solid organ injury	23.4 % (11/47)	8.2 % (5/61)	0.027
Arrest in the field	14.9 % (7/47)	34.4 % (21/61)	0.022
Arrest en route	19.1 % (9/47)	21.3 % (13/61)	0.782
Arrest in the ED	53.2 % (25/47)	26.2 % (16/61)	0.004
Duration of closed CPR prior to EDT, min[a]	3.7 ± 8.0	9.8 ± 13.3	0.003

GSW gunshot wound; *SW* stab wound; *TBI* traumatic brain injury; *ED* emergency department; *CPR* cardiopulmonary resuscitation; *EDT* emergency department thoracotomy

[a] Mean ± standard deviation

[b] chi-square test, Fisher's exact test, or Mann–Whitney *U* test

discharge. All three survivors experienced cardiac arrest in the emergency department and immediately underwent EDT. Two of the three survivors sustained a stab wound (SW) to the heart that was immediately repaired. Both were discharged home with a good neurological outcome. The other survivor sustained multiple SW to the chest and abdomen and was down for several hours. After EDT he underwent laparotomy with repair of a mesenteric bleed and an injury to the small bowel. He was discharged to a special facility because he remained in a vegetative state.

In 28 patients (25.9 %) an intracardiac blood sample was drawn during open CPR. Of these, six were excluded because not enough blood could be drawn or the samples were clotted or contaminated with air. Resulting in a total of 22 adequate intracardiac blood samples (Fig. 1). The remaining 80 patients had no intracardiac blood sample attempted because a peripheral blood sample was available ($n = 45$), because restoration of a heartbeat occurred without intracardiac drug administration ($n = 13$), or for other reasons ($n = 22$).

Characteristics of the Study Population

The demographics, injury characteristics, as well as the proportion of patients who regained a heart rhythm did not differ significantly between the 22 patients with intracardiac blood collected and the remaining 86 patients without a valid intracardiac blood sample (Table 2). However, significantly more patients with an intracardiac blood sample lost their vital signs en route to the hospital, leading to a significantly longer duration of closed CPR prior to EDT (11.8 ± 13.3 vs. 4.3 ± 9.3 min, $p = 0.009$; Table 2).

Cross-clamping of the thoracic aorta was performed in all patients with intracardiac blood samples. Intracardiac

Table 2 Demographics, injury characteristics, and outcome of EDT: comparison between patients with intracardiac blood samples collected ($n = 22$) and patients without intracardiac blood samples collected ($n = 86$)

	Intracardiac blood sample ($n = 22$)	No intracardiac blood sample ($n = 86$)	p Value[b]
GSW	40.9 % (9/22)	38.4 (33/86)	0.828
SW	18.2 % (4/22)	14.0 % (12/86)	0.737
Traffic accidents	31.8 % (7/22)	30.2 % (26/86)	0.885
Fall	9.1 % (2/22)	3.5 % (3/86)	0.268
Other mechanism of injury	0.0 % (0/22)	14.0 % (12/86)	0.121
Blunt severe TBI	9.1 % (2/22)	7.0 % (6/86)	0.664
Penetrating severe TBI	0.0 % (0/22)	2.3 % (2/86)	1.000
GCS on admission[a]	4.7 ± 4.2	5.3 ± 4.0	0.251
Major source of bleeding			
Heart	27.3 % (6/22)	18.6 % (16/86)	0.368
Major thoracic vessels	40.9 % (9/22)	20.9 % (18/86)	0.053
Major abdominal vessels	13.6 % (3/22)	18.6 % (16/86)	0.758
Abdominal solid organ injury	13.6 % (3/22)	15.1 % (13/86)	1.000
Pelvis	0.0 % (0/22)	3.5 % (3/86)	1.000
Neck	0.0 % (0/22)	2.3 % (2/86)	1.000
Other/unknown	22.7 % (5/22)	24.4 % (21/86)	0.868
Arrest in the field	22.7 % (5/22)	26.7 % (23/86)	0.701
Arrest en route	40.9 % (9/22)	15.1 % (13/86)	0.007
Arrest in the ED	31.8 % (7/22)	39.5 % (34/86)	0.506
Arrest in the OR	4.5 % (1/22)	14.0 % (12/86)	0.461
Arrest in the ICU	0.0 % (0/22)	4.7 % (4/86)	0.580
Duration of closed CPR prior to EDT, min[a]	11.8 ± 13.3	4.3 ± 9.3	0.006
Return of cardiac activity	45.5 % (10/22)	43.0 % (37/86)	0.830
Duration of cardiac activity after EDT, min[a]	50.7 ± 69.3	73.9 ± 101.0	0.381

GCS Glasgow coma score; *OR* operating room; *ICU* intensive care unit

[a] Mean ± standard deviation

[b] chi-square test, Fisher's exact test, or Mann–Whitney *U* test

drugs administered included epinephrine ($n = 20$, 91 %), sodium-bicarbonate ($n = 18$, 82 %), atropine ($n = 8$, 36 %), and vasopressin ($n = 2$, 9 %). Of the included 22 patients, 12 (55 %) never regained cardiac activity, and 10 (46 %) patients transiently regained a heartbeat for a mean of 50.7 ± 69.3 min. The same variables shown in Table 2 were compared between these two groups. No statistical significant differences were found for the demographics and injury characteristics. None of these 22 patients ultimately survived to hospital discharge.

A comparison of the intracardiac blood samples between the 12 patients who never regained a heartbeat and the 10 patients with transient return to cardiac rhythm are summarized in Tables 3 and 4.

Blood Gases

No statistically significant differences of the partial pressure of carbon dioxide (pCO_2) and oxygen levels (pO_2) were found between the two groups (Table 3). The pCO_2 was normal (35–45 mmHg) in 36 % (8 of 22) of patients and low (<35 mmHg) in 32 % of patients (7 of 22) (Table 4). All 15 patients with normal or low pCO_2 levels had normal pO_2 levels (>75 mmHg). The remaining 7 patients (32 %) had elevated pCO_2 levels (>45 mmHg) and of those, 5 also had low pO_2 levels (≤75 mmHg).

The mean pH of the 22 patients was measured at 7.08 ± 0.16 pH units (Table 3). A total of 91 % (20/22) had a pH < 7.20, and 86 % (19/22) had a lactate level of >10 mmol/L (Table 4). Patients who never regained a heartbeat had a significantly higher lactate level than those who regained a cardiac rhythm (17.1 ± 2.6 versus 10.6 ± 4.9 mmol/L, $p = 0.018$). In parallel, the base deficit was higher for those patients who never regained cardiac activity compared to those who did; however, this trend did not reach statistical significance (19.8 ± 4.0 vs. 13.7 ± 6.0 mmol/L, $p = 0.066$) (Table 3).

Hematological and Coagulation Parameters

The range of measured hemoglobin (Hb) and hematocrit (Hct) levels showed a wide range (Hb 5.6–15.2 g/dL, Hct 16.0–45.3 %). Overall, 55 % (12/22) of patients had an Hb level ≥8 g/dL.

Regarding coagulation parameters, all but one patient (96 %) were in a coagulopathic state (INR > 1.2 and/or

Table 3 Analysis of intracardiac blood samples

Laboratory parameter	Total ($n = 22$)	Return of cardiac activity ($n = 10$)	No return of cardiac activity ($n = 12$)	p value[a]
Arterial blood gas analysis				
pH, units	7.08 ± 0.16	7.10 ± 0.17	7.02 ± 0.14	0.625
Lactate, mmol/L	13.5 ± 4.8	10.6 ± 4.9	17.1 ± 2.6	0.018
pCO_2, mmHg	42 ± 27	50 ± 29	27 ± 15	0.178
pO_2, mmHg	194 ± 126	214 ± 145	161 ± 88	0.386
HCO_3^-, mmol/L	17 ± 15	15 ± 9	20 ± 22	0.664
Base deficit, mmol/L	15.8 ± 5.9	13.7 ± 6.0	19.8 ± 4.0	0.066
O_2sat, %	84 ± 25	83 ± 28	86 ± 21	0.691
Blood cell count and coagulation parameters				
Hemoglobin, g/dL	9.5 ± 3.3	8.5 ± 3.4	10.2 ± 3.2	0.224
Hematocrit, %	27.2 ± 9.5	24.5 ± 9.2	29.5 ± 9.6	0.183
Platelets ($\times 10^3$/μL)	167 ± 80	147 ± 54	186 ± 101	0.337
Prothrombin time, s	21.5 ± 5.3	22.3 ± 5.1	20.4 ± 6.2	0.522
INR	1.81 ± 0.56	1.89 ± 0.52	1.69 ± 0.68	0.454
aPTT, s	48.6 ± 21.5	53.9 ± 19.6	40.7 ± 24.6	0.286
Electrolytes				
Sodium, mmol/L	151 ± 12	147 ± 9	155 ± 14	0.094
Potassium, mmol/L	5.3 ± 1.0	4.6 ± 1.0	6.0 ± 1.1	0.014
Calcium ionized, mg/dL	4.0 ± 0.9	4.0 ± 0.8	4.1 ± 1.2	0.540
Chloride, mmol/L	117 ± 7	118 ± 6	116 ± 9	0.353
Phosphate, mg/dL	7.1 ± 2.0	6.5 ± 1.8	7.5 ± 2.3	0.480
Magnesium, mg/dL	2.3 ± 0.5	2.2 ± 0.3	2.4 ± 0.6	0.724

[a] Mann–Whitney *U* test

prothrombin time (PT) > 15 s, and/or platelet count <100,000/μL). Their PT ranged from 18.3–30.2 s (normal range: 12.3–14.7 s), and the INR ranged from 1.30–2.69 (normal range: 0.89–1.11). Thirteen of 22 patients (59 %) had an INR >1.5. No difference was found for this cutoff between the patients who did or did not regain a heartbeat. Five patients (23 %) additionally had a platelet count <100,000/μL (Table 4).

Electrolytes

Fourteen of 22 patients (64 %) had a sodium level >145 mmol/L (normal range: 135–145 mmol/L), and 12 patients (55 %) had a potassium level >4.9 mmol/L (normal range: 3.3–4.9 mmol/L). Those who never regained cardiac activity showed higher sodium and potassium levels than those who did regain heart rhythm. This difference, however, was statistically significant only for the potassium levels (sodium: 155 ± 14 vs. 147 ± 9 mmol/L, $p = 0.094$; potassium: 6.0 ± 1.1 versus 4.6 ± 1.0 mmol/L, $p = 0.014$). Additionally, severe hyperkalemia (potassium >5.5 mmol/L) occurred significantly more often in patients who did not regain a heartbeat (Table 4).

In 20 of 22 patients (91 %), the blood phosphorus level was clearly elevated, ranging from 4.9 to 9.9 mg/dL (normal range: 3.0–4.5 mg/dL); however, no statistical difference was detected between those patients who never regained a heartbeat and those who did (Tables 3 and 4). In addition, 17 of 22 patients (77 %) experienced elevated blood chloride levels (range: 111–132 mmol/L; normal range: 100–110 mmol/L). Again, no statistical difference was detected between those patients who never regained cardiac activity and those who did (Tables 3 and 4).

Hypocalcemic states (ionized calcium: <4.5 mg/dL) occurred more often in patients who regained heart rhythm than in those who never regained cardiac activity; however, this difference did not reach statistical significance (70 vs. 42 %, $p = 0.231$) (Table 4).

Hypermagnesemia (magnesium: >2.3 mg/dL) occurred in a total of 41 % (9/22) of patients. No statistical difference was found between those who regained cardiac activity and those who never regained cardiac activity (Table 4). The remaining 59 % of patients had blood magnesium levels within normal limits (1.7–2.3 mg/dL).

Discussion

In the present pilot study, the biochemical profiles of 22 trauma patients undergoing resuscitative thoracotomy were assessed by analyzing their intracardiac blood samples. It is unknown why some patients with traumatic cardiac arrest

Table 4 Incidences of selected abnormal laboratory results

Laboratory parameter	Total ($n = 22$) (%)	Return of cardiac activity ($n = 10$) (%)	No return of cardiac activity ($n = 12$) (%)	p value[a]
Arterial blood gas analysis				
Low pO_2 ($\leq$ 75 mmHg)	32 (7)	30 (3)	33 (4)	1.000
High pCO_2 (> 45 mmHg)	32 (7)	40 (4)	25 (3)	0.625
Low pCO_2 (< 35 mmHg)	46 (10)	30 (3)	58 (7)	0.231
Severe acidosis (pH < 7.20)	91 (20)	80 (8)	100 (12)	0.195
Severe lactic acidosis (lactate > 10 mmol/L)	86 (19)	70 (7)	100 (12)	0.078
Electrolytes				
Hyperkalemia (K^+ > 4.9 mmol/L)	55 (12)	30 (3)	75 (9)	0.084
Severe hyperkalemia (K^+ > 5.5 mmol/L)	50 (11)	20 (2)	75 (9)	0.030
Hypernatremia (Na^+ > 145 mmol/L)	64 (14)	50 (5)	75 (9)	0.378
Severe hypernatremia (Na^+ > 155 mmol/L)	32 (7)	20 (2)	42 (5)	0.381
Hyperphosphatemia (P^+ > 4.5 mg/dL)	91 (20)	80 (8)	100 (12)	0.195
Hyperchloremia (Cl^- > 110 mmol/L)	77 (17)	90 (9)	67 (8)	0.323
Hypocalcemia (Ca^{2+} < 4.5 mmol/L)	55 (12)	70 (7)	42 (5)	0.231
Severe Hypocalcemia (Ca^{2+} < 4.0 mmol/L)	46 (10)	50 (5)	42 (5)	1.000
Hypermagnesemia (Mg^+ > 2.3 mg/dL)	41 (9)	30 (3)	50 (6)	0.415
Blood cell count and coagulation parameters				
INR > 1.5	59 (13)	70 (7)	50 (6)	0.415
Platelets < 100,000	23 (5)	30 (3)	17 (2)	0.624
Platelets < 150,000	27 (6)	40 (4)	17 (2)	0.348

[a] Fisher's exact test

regain a heart rhythm and some never show any cardiac activity. We opted to assess the differences in electrolyte profiles and blood gases from intracardiac blood samples of patients who did or did not regain a cardiac rhythm. To the best of our knowledge, such a study has not been previously conducted.

The analyzed patients were a total 22 of 108 patients suffering traumatic cardiac arrest undergoing open CPR within the study period. To observe any selection bias due to the relatively small sample size, a comparison of the demographics, injury characteristics, and outcome of the included and excluded patients was performed (Table 2). The compared groups were statistically similar in almost all assessed variables. However, the rate of patients who experienced cardiac arrest en route to the hospital was significantly higher for the patients with intracardiac blood samples accrued, which resulted in an increased duration of closed CPR prior to EDT for this group of patients. Interestingly, this difference did not affect outcome.

Unexpectedly, most of the patients showed adequate O_2-saturations and pO_2, along with normal or even low pCO_2 levels. The cardiopulmonary arrest in combination with a right shift of the standard oxygen–hemoglobin dissociation curve due to the lactic acidosis are expected to alter blood gas levels for the worse. It seems that by open CPR, the gas exchange was adequate and blood circulation to the mechanically ventilated lungs could be achieved.

As expected, the current study population was in a severe acidotic state, with decreased base excess levels indicating severe tissue hypoperfusion. This severe acidosis was significantly accentuated for those patients who never regained cardiac rhythm. Prolonged hypoperfusion of tissues results in irreversible cellular injury. Accordingly, the alterations in the blood electrolyte levels noted in the current study group can be explained by disseminated hypoxic cell death. In particular, the increased potassium levels in parallel with the profound acidosis likely were caused by severe tissue ischemia and subsequent reperfusion injury. Massive blood transfusion or the use of paralyzing agents (i.e., succinylcholine) might be additional reasons. Similarly, the sodium and phosphate levels were increased. However, statistical significance was only found for severe hyperkalemic states (K^+ > 5.5 mmol/L) among patients who never regained a heartbeat. This indicates a potential irreversible moribund physiologic state due to a deep hemorrhagic shock, possibly predicting a poorer outcome.

Other electrolytes that are highly debated as being important components of the overall electrolyte disturbance in patients with traumatic cardiac arrest are calcium and magnesium. Hypocalcemic states frequently occurred in the current series. Severe hemorrhagic shock and/or ischemia-reperfusion injury appear to be important causative factors. However, similar to previous investigations, no statistical impact on outcomes could be observed, making a recommendation difficult [8]. Magnesium also has important electrophysiological effects, and normal concentrations are required to maintain regular cardiac conduction, rhythm, and vascular tone [11]. In the current series, the magnesium levels were elevated or within normal limits, and no trend was found when comparing the study groups. This is in concordance with other trials showing no effect of magnesium administration on outcomes [11–13]. However, these published studies included only victims of non-traumatic cardiac arrest.

What was common in almost all patients was a severe coagulopathic state on admission. Hemodilution and consumption of coagulation factors might be potential reasons for this severe coagulopathic state. However, hemoglobin and hematocrit levels as surrogate markers for the degree of (iatrogenic) hemodilution were only moderately decreased on admission. The prehospital "scoop and run" approach with short transportation times and a policy of "hypotensive resuscitation" are expected to limit prehospital volume substitution. In addition, hypoperfusion of tissues resulting in severe acidosis and the initiation of the activated protein C pathway may contribute to the early coagulopathy observed in the present series [14].

Limitations

Because of the overall low number of study patients, interpretation of the results is made with caution. In addition, the inclusion criteria of patients may have led to a potential selection bias. A sensitivity analysis was therefore performed by comparing the demographics, injury characteristics, and outcomes between the study group and the excluded patients (Table 2). That comparison showed that the two groups were statistically similar in almost all assessed variables.

Different times of intracardiac blood sampling potentially affected the results. The time of the intracardiac blood draw was at the attending trauma surgeon's discretion and, because of the challenging nature of a resuscitative EDT, was difficult to schedule in advance. In addition, a defined point in time for the intracardiac blood draw could not be set due to ethical concerns.

Conclusions

Most patients undergoing open CPR showed normal blood gas levels, with the exception of severe lactic acidosis. It seems that by open CPR, the gas exchange is adequate and blood circulation to the mechanically ventilated lungs could be achieved. Severe hyperkalemia and hypernatremia in combination with lactic acidosis were significantly more common in patients who never regained cardiac function. Calcium and magnesium levels were not significantly different between the two groups, making the therapeutic role of these electrolytes very questionable. Further investigation is warranted to delineate therapeutic implications in the management of patients undergoing resuscitative thoracotomy.

References

1. Rhee PM, Acosta J, Bridgeman A et al (2000) Survival after emergency department thoracotomy: review of published data from the past 25 years. J Am Coll Surg 190:288–298
2. Demetriades D, Rabinowitz B, Sofianos C (1987) Emergency room thoracotomy for stab wounds to the chest and neck. J Trauma 27:483–485
3. Fialka C, Sebok C, Kemetzhofer P et al (2004) Open-chest cardiopulmonary resuscitation after cardiac arrest in cases of blunt chest or abdominal trauma: a consecutive series of 38 cases. J Trauma 57:809–814
4. Powell DW, Moore EE, Cothren CC et al (2004) Is emergency department resuscitative thoracotomy futile care for the critically injured patient requiring prehospital cardiopulmonary resuscitation? J Am Coll Surg 199:211–215
5. Schnuriger B, Inaba K, Branco BC et al (2010) Organ donation: an important outcome after resuscitative thoracotomy. J Am Coll Surg 211:450–455
6. Makino J, Uchino S, Morimatsu H et al (2005) A quantitative analysis of the acidosis of cardiac arrest: a prospective observational study. Crit Care 9:R357–R362
7. Cannon LA, Heiselman DE, Dougherty JM et al (1987) Magnesium levels in cardiac arrest victims: relationship between magnesium levels and successful resuscitation. Ann Emerg Med 16:1195–1199
8. Vivien B, Langeron O, Morell E et al (2005) Early hypocalcemia in severe trauma. Crit Care Med 33:1946–1952
9. Reis AG, Ferreira de Paiva E et al (2008) Magnesium in cardiopulmonary resuscitation: critical review. Resuscitation 77:21–25
10. Stiell IG, Wells GA et al (1995) Association of drug therapy with survival in cardiac arrest: limited role of advanced cardiac life support drugs. Acad Emerg Med 2:264–273
11. Allegra J, Lavery R, Cody R et al (2001) Magnesium sulfate in the treatment of refractory ventricular fibrillation in the prehospital setting. Resuscitation 49:245–249
12. Fatovich DM, Prentice DA, Dobb GJ (1997) Magnesium in cardiac arrest (the magic trial). Resuscitation 35:237–241
13. Hassan TB, Jagger C, Barnett DB (2002) A randomised trial to investigate the efficacy of magnesium sulphate for refractory ventricular fibrillation. Emerg Med J 19:57–62
14. Brohi K, Cohen MJ, Ganter MT et al (2007) Acute traumatic coagulopathy: initiated by hypoperfusion: modulated through the protein C pathway? Ann Surg 245:812–818