https://doi.org/10.21320/1818-474X-2024-3-142-151

Changes in clinical and laboratory parameters depending on the severity of traumatic shock: a retrospective cohort study

R.E. Lakhin[1,2,*], A.S. Kusai[1], E.A. Usoltsev[3], R.R. Kasimov[1,3], K.A. Tsygankov[1], D.O. Starostin[4,5], V.V. Shustrov[1], A.V. Shchegolev[1]

[1] *Military Medical Academy, St. Petersburg, Russia*
[2] *Saratov State Medical University named after V.I. Razumovsky, Saratov, Russia*
[3] *442 military clinical hospital named after Z.P. Solovyov, St. Petersburg, Russia*
[4] *1586 Military Clinical Hospital, branch No. 8, Moscow region, Russia*
[5] *Federal Research and Clinical Center of Intensive Care Medicine and Rehabilitology, Moscow, Russia, Moscow, Russia*

Изменение клинических и лабораторных показателей в зависимости от тяжести травматического шока: ретроспективное когортное исследование

Р.Е. Лахин[1,2,*], А.С. Кусай[1], Е.А. Усольцев[3], Р.Р. Касимов[1,3], К.А. Цыганков[1], Д.О. Старостин[4,5], В.В. Шустров[1], А.В. Щеголев[1]

[1] *ФГБВОУ ВО «Военно-медицинская академия имени С.М. Кирова» МО РФ, Санкт-Петербург, Россия*
[2] *ФГБОУ ВО «Саратовский государственный медицинский университет им. В.И. Разумовского» Минздрава России, Саратов, Россия*
[3] *ФГКУ «442 военный клинический госпиталь им. З.П. Соловьева» МО РФ, Санкт-Петербург, Россия*
[4] *ФГКУ «1586 Военный клинический госпиталь» МО РФ, Московская область, Россия*
[5] *ФГБНУ «Федеральный научно-клинический центр реаниматологии и реабилитологии (ФНКЦ РР)», Москва, Россия*

Abstract

INTRODUCTION: Traumatic shock is a complex pathophysiological process that includes impaired tissue perfusion, the development of circulatory and tissue hypoxia, metabolic disorders, and homeostasis disorders. **MATERIALS AND METHODS:** A retrospective cohort study that included 329 people with combat injury. The level of systolic blood pressure (SBP), heart rate (HR), acid-base state (CBS), general and gas blood tests were examined upon admission. **RESULTS:** There were 55 patients without shock (30 %), 83 (25 %) with I degree shock, 80 (25 %) of II degree, 51 (15 %) of III degree, 16 (5 %) of IV degree. The analysis of the values of the SBP, HR and shock index (SI), base deficiency (BE) showed the presence of statistical differences depending on the degree of shock. SI without shock was 0.7 (0.7; 0.8); with shock of the I degree — 0.8 (0.7; 0.9); II degree — 1.3 (1.1; 1.5), III degree — 1.8 (1.5; 2.1), IV degree — 2.5 (2.1; 3.8). The value without shock was –3.1 (3.9; –2.2); with degree I shock –3.7 (–2.1; –6.2); II degree: –4.6 (–3.1; –6.8); III degree: –8.5 (–6.5; –11.8); IV degree: –22.1 (–15.6; –26.1). Regression analysis of the relationship with the degree of shock showed

Реферат

АКТУАЛЬНОСТЬ: Травматический шок является сложным патофизиологическим процессом, который включает в себя нарушения перфузии тканей, развитие циркуляторной и тканевой гипоксии, метаболических расстройств, нарушение гомеостаза. **МАТЕРИАЛЫ И МЕТОДЫ:** Ретроспективное когортное исследование, в которое было включено 329 раненых. Исследовали при поступлении уровень систолического артериального давления (АДсист), частоту сердечных сокращений (ЧСС), кислотно-основное состояние (КОС), общий и газовый анализ крови. **РЕЗУЛЬТАТЫ:** Без шока было 99 пациентов (30 %), с шоком I степени — 83 (25 %), II степени — 80 (25 %), III степени — 51 (15 %), IV степени — 16 (5 %). Анализ величины АДсист, ЧСС и шокового индекса (ШИ), дефицита оснований (BE) показал наличие статистических различий в зависимости от степени шока. АДсист (мм рт. ст.) у пациентов без шока было 120,0 (120,0; 135,0), при шоке I степени — 130,03 (100,5; 135,0), II степени — 89,5 (80,0; 95,0), III степени — 70,8 (65,0; 85,0), IV степени — 55,0 (50,00; 69,55). ШИ без шока был 0,7 (0,7; 0,8), при шоке

two models that described a 45.9 % and 50.2 % probability. The first model includes a constant and VE ($p < 0.01$). The second model includes the constant, VE and the number of platelets ($p < 0.01$). **CONCLUSION:** A retrospective analysis of clinical and laboratory parameters for traumatic shock demonstrated differences in SBP, HR, SI, red blood counts, CBS, lactate levels depending on the degree of shock. The severity of acidosis and lactate levels turned out to be higher than expected for the degree of shock, due to the nature and severity of the injuries received. Base deficiency and platelet count were associated with the severity of shock.

KEYWORDS: shock, traumatic, blood gas analysis, acid-base equilibrium, arterial pressure, wounds and injuries, lactate

* *For correspondence*: Roman E. Lakhin — Doctor of Medical Science, Professor, Department of Anesthesiology and Intensive Care, Military Medical Academy, Saint Petersburg, Russia; e-mail: doctorlahin@yandex.ru

For citation: Lakhin R.E., Kusai A.S., Usoltsev E.A., Kasimov R.R., Tsygankov K.A., Starostin D.O., Shustrov V.V., Shchegolev A.V. Changes in clinical and laboratory parameters depending on the severity of traumatic shock: a retrospective cohort study. Annals of Critical Care. 2024;3:142–151. https://doi.org/10.21320/1818-474X-2024-3-142-151

Received: 06.03.2024
Accepted: 06.06.2024
Published online: 31.07.2024

I степени — 0,8 (0,7; 0,9), II степени — 1,3 (1,1; 1,5), III степени — 1,8 (1,5; 2,1), IV степени — 2,5 (2,1; 3,8). BE без шока было –3,1 (3,9; –2,2), при шоке I степени: –3,7 (–2,1; –6,2), II степени: –4,6 (–3,1; –6,8), III степени: –8,5 (–6,5; –11,8), IV степени: –22,1 (–15,6; –26,1). Регрессионный анализ связи со степенью шока показал две модели, которые описывали 45,9 и 50,2 % вероятности. В первую модель включены константа и BE ($p < 0,01$), во вторую модель — константа, BE и количество тромбоцитов ($p < 0,01$). **ВЫВОДЫ:** Ретроспективный анализ клинических и лабораторных показателей при травматическом шоке в летний период продемонстрировал различия АДсист, ЧСС, ШИ, показателей красной крови, КОС, уровня лактата в зависимости от степени шока. Выраженность ацидоза, уровень лактата оказались выше ожидаемых для степени шока значений, что обусловлено характером и тяжестью получаемых ранений. Дефицит оснований и количество тромбоцитов были связаны с тяжестью шока.

КЛЮЧЕВЫЕ СЛОВА: травматический шок, анализ газов крови, кислотно-щелочное равновесие, артериальное давление, раны и ушибы, лактат

* *Для корреспонденции*: Лахин Роман Евгеньевич — д-р мед. наук, профессор кафедры военной анестезиологии и реаниматологии ФГБВОУ ВО «Военно-медицинская академия им. С.М. Кирова» МО РФ, Санкт-Петербург, Россия; e-mail: doctor-lahin@yandex.ru

Для цитирования: Лахин Р.Е., Кусай А.С., Усольцев Е.А., Касимов Р.Р., Цыганков К.А., Старостин Д.О., Шустров В.В., Щеголев А.В. Изменение клинических и лабораторных показателей в зависимости от тяжести травматического шока: ретроспективное когортное исследование. Вестник интенсивной терапии им. А.И. Салтанова. 2024;3:142–151. https://doi.org/10.21320/1818-474X-2024-3-142-151

Поступила: 06.03.2024
Принята к печати: 06.06.2024
Дата онлайн-публикации: 31.07.2024

DOI: 10.21320/1818-474X-2024-3-142-151

Introduction

The main cause of lethal outcome in victims with trauma received during the military conflict in the first hours is acute massive blood loss [1–3]. Under conditions of blood loss and haemorrhagic shock, a rapid decrease in circulating blood volume (BV) leads to a decrease in cardiac output and oxygen delivery. Increasing changes in tissues with the participation of the sympathetic system lead to hypoxic redistribution of blood, due to the organ-specific microcirculatory reaction with a reduction in blood flow in the skin, muscles, intestines, kidneys and other organs except for the heart, brain and adrenal glands. This mechanism of adaptation has been called "centralization of circulation". Compensation for further reduction of blood volume and lowering of blood pressure (BP) is carried out due to tachycardia, tachypnoea, slowing of blood flow in the lungs as a result of spasm of postcapillary sphincters (which increases the time of oxygen saturation of erythrocytes in pulmonary capillaries). In this case, systolic BP (SBP) may remain within normal lim-

its for a long time, although circulatory and tissue hypoxia progresses, thereby exacerbating the severity of metabolic acidosis. Correction with infusion therapy, particularly with unbalanced solutions aggravates metabolic complications at the prehospital stage [4, 5]. Moreover, high ambient temperature in summer leads to additional fluid loss, forming initial latent hypovolemia with haemoconcentration, which leads to tissue hypoxia and metabolic acidosis in wounded and injured people, that masking the severity of the condition by normal haemoglobin level [5].

According to a number of studies and clinical guidelines, the severity of shock can only be objectified by laboratory tests [4, 5, 7, 8]. Decrease in pH, increase in base deficit/base excess (BE) [9, 10], increased lactate [9, 11, 12]; decreased fibrinogen [13], increased international normalised ratio [14], low haemoglobin [15, 16], low platelet count [17] are independent predictors of mortality.

Objective

To analyse clinical and laboratory parameters characterizing traumatic shock in patients with combat surgical trauma.

Materials and methods

A retrospective cohort study of medical data of the wounded at the stage of qualified surgical medical care (level 2) in a separate medical battalion was performed.

Inclusion criteria: wounded and injured patients aged 18 to 60 years who were admitted to the shock room or operating theatre with traumatic shock. Inclusion criteria were: wounded and injured with penetrating skull wounds, in a state of clinical death and ineffective resuscitation measures. After applying the inclusion and non-inclusion criteria, 329 injured and wounded were selected for analysis.

The localisation of wounds and the extent of surgical interventions are presented in table 1 and table 2, respectively. In 253 (77%) the wounds were combined, therefore, surgical interventions were performed on several segments.

The grade of shock was determined on the basis of a comprehensive assessment of the injured person presented in table 3 [5, 18].

All victims with shock were catheterized in 2 veins (2 peripheral or central + peripheral) on admission to the antishock unit, followed by blood sampling for determination of blood group, Rh factor, and total blood count, which was performed on an automatic haematological blood analyser Mindray BC-20. Arterial blood was collected from the radial artery for the study of acid-base and gas composition of the blood using a portable device EDAN i15 (China). Then rapid intravenous infusion of warm balanced crystalloid solution of 1000 ml was started with subsequent evaluation of hemodynamic parameters. If against the background of crystalloid infusion, the SBP was less than 70 mmHg, microjet infusion of norepinephrine was started at a starting rate of 0.1 mcg/kg/min, which was increased until reaching the target SBP 90 mmHg. Indications for haemotransfusion and the necessary volume of blood components were assigned taking into account the severity of shock and areas of anatomical injury with calculation of the approximate volume of blood loss according to the guidelines for the treatment of combat surgical trauma [18]. In patients with acute massive blood loss requiring surgical intervention, tranexamic acid 1 g with a repeated dose of 1 g after 8 hours was administered.

Table 1. Localization of wounds requiring surgery ($n = 329$)

Localization	*n*	%
Chest wounds	158	48
Abdominal wounds	145	44
Wounds to the extremities	217	66

Table 2. The volume of surgical interventions ($n = 329$)

Name	*n*	%
Drainage of the pleural cavity	158	48
Laparocentesis	42	13
Laparotomy	171	37
Fragment repositioning, fixation in a rod apparatus of external fixation (MFRK, CTK)	204	62
Amputations by PST	17	5
Temporary prosthesis of brachial, femoral, and hamstring arteries	30	9
Suture of the brachial, femoral, and hamstring arteries	10	3
Primary surgical treatment	79	24

The initial information was accumulated and systematized in Microsoft Office Excel spreadsheets. Statistical analysis was performed by the SPSS-26 program for Windows (Statistical Package for Social Science, SPSS Inc. Chicago IL, USA). When data were normally distributed, they were represented by mean and standard deviation M (± SD); descriptive quantitative data that did not obey the law of normal distribution were presented as median and 25th and 75th percentiles, Me (Q1; Q3), or mean with 95% confidence interval, M (95% CI). Analyses were performed to describe and summarize the distributions of the variables. The description of frequencies in the study sample was presented with the obligatory indication of the sample characteristic given (*n* (%)), or *n* (95% CI). The presence of group differences was determined using the Kruskal-Wallace test. Then, in order to analyse the differences between groups in a detailed sequential manner, after group differences were identified, posterior pairwise comparisons were performed; the statistical significance of posterior comparisons was cal-

Table 3. Criteria for assessing the degree of traumatic shock

Parameters	Grade of shock			
	I	II	III	IV (terminal condition)
Blood loss, % of BV	< 15	15–30	30–40	> 40
Blood loss, ml	< 750	750–1500	1500–2000	> 2000
HR, min^{-1}	< 100	100–120	120–140	> 140
SBP, mmHg	norma	may be lower	low	very low
Respiratory rate, min^{-1}	norma	norm	could be tachypnoea	tachypnoea
Diuresis, ml/h	norma	norma	reduced	anuria
Consciousness level	norma	norma / agitation	oppressed	significantly oppressed
BE, mmol/l	0 … –2	–2 … –6	–6 … –10	< –10
The need for haemotransfusion	not	possibly	yes	massive haemotransfusion

Notes: BE — deficiency or excess of bases; BV — volume of circulating blood; HR — heart rate; SBP — systolic blood pressure.

culated using the Bonferroni correction (see Appendix 1). The relationship between the indicators was assessed by constructing a linear regression model using the stepwise exclusion method. Tests were considered statistically significant at $p < 0.05$.

Results

A total of 329 injured patients were included in the study. In the study cohort, there were 99 patients (30 %) without shock, 83 (25 %) with grade I shock, 80 (25 %) with grade II, 51 (15 %) with grade III, and 16 (5 %) with grade IV. The time from wounding to admission to the stage of skilled care for shock was 3.8 (2.1; 6.4) hours.

The analysis of SBP, heart rate (HR) and shock index (SI) showed statistical differences depending on the grade of shock (Table 4). Thus, SBP decreased, HR and SI increased. Pairwise analysis of a posteriori comparisons of SBP, HR, and SI showed statistical differences between shocks of different grade (Appendix 1). It should be noted that there were no statistical differences between hemodynamic parameters in shock of III and IV grades.

Clinical blood counts demonstrated that there were no differences in patients with no shock and first-grade shock, between I and II grades, and between III and IV grade of shock (Appendix 1). All differences were between I, II and III, IV grades. Traumatic shock led to the metabolic acidosis. The pH decreased and base deficiency increased (Table 4). There were statistical differences in BE values when comparing all groups except for patients without shock and shock of the first grade.

Regression analysis was performed to assess the relationship between the grade of shock and the most significant parameters (Table 5).

Stepwise regression identified two models that described 45.9 % and 50.2 % probabilities. Haemoglobin, erythrocytes, SBP and lactate showed no association with the grade of shock. The first model includes constant and base deficit (grade of shock =1.75 + (–0.09 × BE)). In the second model, in addition to the constant and base deficit, platelet count was included (grade of shock = 2.41 + (–0.09 × BE – 0.003 × platelets). The analysis revealed that base deficit and platelet count were the indicators demonstrating the severity of shock manifestation.

Massive destruction of soft tissues during wounds, arterial blood flow disturbance, tourniquets, necrotic areas, wound contamination, rapid development of traumatic oedema disturb local perfusion of tissues, which worsens with systemic haemodynamic disturbances in shock (Figure 1). All this leads to a rapid increase in lactate level even in I and II grade of shock (Table 4). On admission in patients with shock and massive soft tissue destruction the lactate level was extremely high. In III, IV grades of shock it is due to systemic severe tissue hypoperfusion, requiring adequate replenishment of blood volume by blood products to ensure proper oxygen delivery to tissues and avoid excess crystalloid solutions.

Discussion

At the stage of qualified surgical care in a separate medical battalion, a protocol of massive haemotransfusion therapy was used: the wounded with signs of traumatic shock of grade II were given haemotransfusion in the volume of at least 3 unites of homologous red blood cells and 3 unites of fresh frozen plasma (FFP), and of grade III — at least 6 unites. In case of traumatic shock of the grade IV (terminal condition) the volume of haemotransfusion therapy is selected individually under the control of blood gas and acid-base state. Haemotransfusion was performed in 109 wounded with shock during the study period, who received 273 unites of red blood cells and 280 units of FFP.

Table 4. Distribution of the studied indicators by degree of shock severity

Parameters	No shock (n = 99)	Grade of shock, Me (Q1; Q3)				H Kruskal-Wallis test; p
		I (n = 83)	II (n = 80)	III (n = 51)	IV (n = 16)	
Age, years	33 (27; 40)	34 (29; 41)	35 (29; 41)	38 (30; 44)	40 (31; 43)	H = 4.97; p = 0.29
SBP, mmHg	120.0 (120.0; 135.0)	130.03 (100.5; 135.0)	89.5 (80.0; 95.0)	70.8 (65.0; 85.0)	55.0 (50.0; 69.5)	H = 227.54; p < 0.01
HR, min^{-1}	90.0 (80.0; 95.0)	100.0 (90.0; 110.0)	117.5 (110.0; 124.0)	132.0 (120.4; 138.3)	144.0 (133.5; 151.8)	H = 183.21; p < 0.01
SI	0.7 (0.7; 0.8)	0.8 (0.7–0.9)	1.3 (1.1; 1.5)	1.8 (1.5; 2.1)	2.5 (2.1; 3.8)	H = 238.26; p < 0.01
SpO_2, %	98.0 (97.0; 98.0)	97.0 (96.0; 98.0)	95.0 (92.0; 97.0)	90.0 (88.0; 94.0)	84.0 (78.8; 86.8)	H = 88.89; p < 0.01
Body T, °C	36.4 (36.3; 36.6)	36.3 (36.2; 36.5)	35.9 (35.3; 36.3)	35.1 (33.9; 36.1)	32.5 (31.9; 33.8)	H = 85.95; p < 0.01
Erythrocytes, 10^{12}/L	3.62 (3.20; 4.45)	3.78 (3.50; 4.05)	3.56 (3.39; 3.74)	2.97 (2.73; 3.22)	2.77 (2.16; 3.39)	H = 25.79; p < 0.01
Haemoglobin, g/L	121.0 (114.8; 123.8)	102.73 (98.3; 114.0)	99.20 (97.0; 129.0)	87.9 (81.8; 110.7)	68.0 (63.5; 97.0)	H = 53.28; p < 0.01
Haematocrit, %	36.5 (32.3; 38.8)	32.0 (31.0; 34.0)	31.0 (29.0; 34.04	28.6 (24.4; 32.6)	24.2 (19.0; 28.0)	H = 54.89; p < 0.01
Platelets, $\times 10^9$/L	320.87 (207.4; 385.5)	261.87 (219.8; 303.9)	256.8 (237.6; 275.9)	223.5 (203.8; 243.2)	188.8 (132.5; 245.0)	H = 14.13; p = 0.01
pH	7.3 (7.3; 7.4)	7.3 (7.3; 7.48)	7.2 (7.1; 7.3)	7.2 (7.1; 7.3)	7.0 (6.7; 7.1)	H = 58.18; p < 0.01
BE, mmol/l	–3.1 (3.9; –2.2)	–3.7 (–2.1; –6.2)	–4.6 (–3.1; –6.8)	–8.5 (–6.5; –11.8)	–22.1 (–15.6; –26.1)	H = 83.42; p < 0.01
Lactate, mmol/l	2.3 (2.1; 3.1)	4.1 (2.4; 6.2)	4.2 (2.9; 6.1)	5.7 (3.9; 9.2)	16.7 (10.7; 19.3)	H = 55.85; p < 0.01

Notes: BE — deficiency or excess of bases; HR — heart rate; SBP — systolic blood pressure; SI — shock index; SpO_2 — blood oxygenation; pH — negative decimal logarithm of hydrogen ion concentration.

Table 5. Regression analysis indicators of the relationship between the degree of shock and the studied indicators

Model	Summary for the model			ANOVA		Coefficients		
	r	r^2	критерий Дарбина—Уотсона	F	p	Coefficient	B	p
1 (constant, BE)	0.677	0.459	1.783	84.817	< 0.01	Constant	1.75	< 0.001
						BE	–0.09	< 0.001
2 (constant, BE, platelets)	0.709	0.502	1.784	49.907	< 0.01	Constant	2.41	< 0.001
						BE	–0.09	< 0.001
						Platelets	–0.003	0.004

An important result of this study is the fact that the severity of acid-base state and tissue perfusion disorders even in summer time was higher than traditionally accepted [5, 18]. The study shows that BE is the leading indicator characterising the severity of shock. The high value of BE was shown in other studies [2, 9, 19, 20].

Acute massive blood loss is one of the main causes of lethal outcome in victims with combat trauma, the clinical manifestation of which is hypotension. However, in our work we obtained the results that SBP in shock of I and II grades were quite high (above 90 mmHg), and in the grade III we met individual cases with a sufficiently high SBP, but accompanied by high tachycardia. This compensation, maintaining high SBP relative to the grade of shock, was caused also by sympathetic stimulation due to pain syndrome and stress. Anaesthesia was performed due to general anaesthesia or regional anaesthesia, the SBP decreased, demonstrating the true picture of haemorrhagic shock. The decrease in SBP was corrected by infusion and transfusion therapy. Vasopressor support was initially required in 10 wounded. Initiation or increase of vasopressor support after regional anaesthesia was in 4 wounded, and after induction of general anaesthesia (ketamine and\or propofol + fentanyl + rocuronium bromide) in 19 wound-

ed. The inclusion of propofol in the scheme of induction anaesthesia, as a rule, always led to a decrease in SBP, which does not allow us to recommend the use of this drug in victims in a state of uncompensated shock due to a pronounced depression of the circulatory system, suppression of the protective reflex in response to hypotension, tendency to bradycardia.

As a rule, acute massive blood loss is accompanied by a decrease in haemoglobin. In this study, the haemoglobin level in all grades of shock was quite high. In confirmation of our results, table 6 presents the results of arterial blood gas analysis of a patient with III grade shock. In this analysis the haemoglobin level was 97 g/l (high index, at which even blood preparations are not transfused in usual "civil" practice), but on admission the patient had SBP 60 mm Hg, stunned, expressed disturbances of acid-base state (Table 6). At the prehospital stage, 1000 ml of 0.9 % sodium chloride was transfused, which increased acidosis due to hyperchloremia.

Despite the warm weather (during the study period, the daytime temperature exceeded 30 °C), extremely severe patients with IV grade shock were admitted in a state of hypothermia, exacerbating the severity of the wounded patient's condition. A patient with a gunshot fracture of the pelvis, left femur was admitted in extremely severe condition with high doses of vasopressor and inotropic support with norepinephrine and epinephrine. The temperature on admission was 30.2 °C. Indices of gas exchange, acid-base state and haemoglobin level on admission are presented in Table 7. The severity of acid-base state disturbances was significant, which was beyond the capability of the gas analyser, so the device gave pH less than 6.5. The base deficit was –37.2 mmol/l. Warming of the patient with a thermal blanket and administration of warm solutions was started. Stabilisation of the pelvic ring and gunshot fracture of the femur with external fixation devices was performed. After infusion of 800 ml of sodium hydrogen carbonate, pH increased to 6.9, base deficit –25.4 mmol/l. After infusion of a total of 1200 ml of 4 % sodium hydrogen carbonate solution, warming the patient to 36.2 °C, pH in arterial blood was 7.32, BE — 5.6 mmol/l. Further stabilisation of acid-base state was carried out with balanced solutions. Against the background of blood loss replenishment (a total of 10 units of erythrocyte transfusions and 10 units of FFP), it was firstly possible to withdraw from epinephrine support, to reduce vasopressor support with norepinephrine to 0.05 μg/kg/min. At the same time, SBP was between 110 and 130 mmHg. Lactate levels decreased from 17.8 to 10.1 mmol/l. Diuresis appeared, the rate of which was up to 100 ml per hour. On the background of stabilisation of the condition, the patient was sent for evacuation. This example emphasises the need for intensive therapy of severe blood loss under control of acid-base state, the importance of compensation of severe acidosis.

A limitation of this work is its single-centre nature with a relatively small sample of victims in the summer time pe-

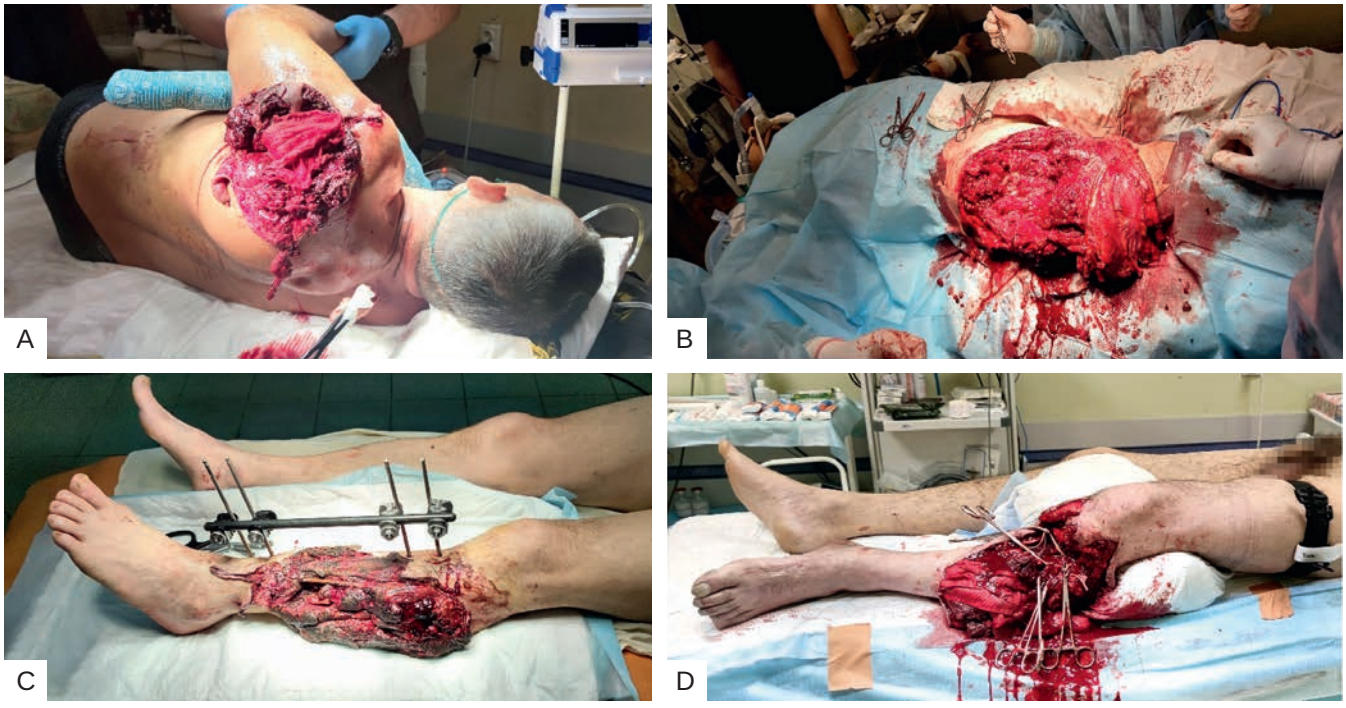

Fig. 1. Soft tissue destruction in gunshot wounds

A — wound of the shoulder, shoulder joint with destruction of the scapula; *B* — fragmentation wound of the thigh with fracture of the femur; *C* — fragmentation wound with fracture of the tibia bones and wound contamination; *D* — fragmentation wound of the knee joint with fracture of the tibia bones and damage to the hamstring artery.

Table 6. Indicators of gas exchange, CBS and hemoglobin level in a wounded person with shock of the 3rd degree

Parameters	Result
SBP, mmHg	60
HR, min^{-1}	132
SI	2.2
Haemoglobin, g/L	97
Haematocrit, %	28
PaO_2, mmHg	184
$PaCO_2$, mmHg	34.1
pH	7.028
BE, mmol/l	–22.1
HCO_3^-, mmol/l	8.8
Лактат, mmol/l	16.6
Na^+, mmol/l	143
K^+, mmol/l	3.6
Cl^-, mmol/l	113

Notes: BE — deficiency or excess of bases; HR — heart rate; $PaCO_2$ — partial pressure of carbon dioxide in arterial blood; PaO_2 — partial pressure of oxygen in arterial blood; pH — negative decimal logarithm of hydrogen ion concentration; SBP — systolic blood pressure; SI — shock index.

Table 7. Gas exchange parameters, CBS and hemoglobin levels in a wounded patient with grade IV shock and hypothermia

Parameters	Results		
	A	B	C
SBP, mmHg	50	95	115
HR, min^{-1}	153	110	95
SI	3.06	1.16	0.82
Haemoglobin, g/L	83	68	99
Haematocrit, %	24	20	29
PaO_2, mmHg	205	130	241
$PaCO_2$, mmHg	42.3	41.4	40.9
pH	< 6.5	6.887	7.318
BE, mmol/l	–37.2	–25.4	–5.6
HCO_3^-act, mmol/l	2.9	7.7	20.5
Lactate, mmol/l	17.8	14.4	10.1
Na^+, mmol/l	153	152	149
K^+, mmol/l	4.6	2.2	3.3
Cl^-, mmol/l	122	112	105

Notes: BE — deficiency or excess of bases; HR — heart rate; SBP — systolic blood pressure; SI — shock index; PaO_2 — partial pressure of oxygen in arterial blood; $PaCO_2$ — partial pressure of carbon dioxide in arterial blood; pH — negative decimal logarithm of hydrogen ion concentration.

riod, which requires validation and comparison with other techniques for correction of metabolic disorders, which will be a task for future work.

Conclusion

The retrospective analysis of clinical and laboratory parameters characterising traumatic shock in patients with combat surgical trauma in the summer period of time has demonstrated differences in SBP, HR, SI, clinical blood test results, acid-base state, lactate level depending on the grade of shock. The severity of acidosis and lactate level were higher than the values traditionally accepted for the grade of shock, which is due to the nature and severity of the wounds received and the duration of evacuation to the stage of receiving specialised care. The deficit of bases and platelet count are the indicators demonstrating the severity of shock manifestation.

Disclosure. The authors declare no competing interests.

Author contribution. All authors according to the ICMJE criteria participated in the development of the concept of the article, obtaining and analyzing factual data, writing and editing the text of the article, checking and approving the text of the article.

Ethics approval. This study was approved by the local Ethical Committee of the Military Medical Academy (reference number: 273 of 06/27/2023).

Information about financing. The authors declare that there is no external funding for the study.

Data Availability Statement. Data that support the findings of this study are available from the corresponding author, upon reasonable request.

Consent for publication. Not required since patient photos are presented in unidentifiable manner.

Acknowledgements. The authors would like to thank Ekaterina A. Zhirnova, an anaesthesiologist of the Department of anaesthesiology and intensive care of the Saint Petersburg State University Hospital, for her assistance in translating the article into English.

Author's ORCID:

Lakhin R.E. — 0000-0001-6819-9691
Kusai A.S. — 0000-0002-0668-9676
Usoltsev E.A. — 0000-0001-6497-1429
Kasimov R.R. — 0000-0001-5866-8378
Tsygankov K.A. — 0000-0002-2357-0685
Starostin D.O. — 0000-0002-5069-6080
Shustrov V.V. — 0000-0001-5144-3360
Shchegolev A.V. — 0000-0001-6431-439X

References

[1] *Kronstedt S., Lee J., Millner D. et al*. The Role of Whole Blood Transfusions in Civilian Trauma: A Review of Literature in Military and Civilian Trauma. Cureus. 2022; 14(4): e24263. DOI: 10.7759/cureus.24263
[2] *Eastridge B.J., Malone D., Holcomb J.B*. Early predictors of transfusion and mortality after injury: a review of the data-based literature. J Trauma. 2006; 60(6 Suppl): S20–S25. DOI: 10.1097/01.ta.0000199544.63879.5d
[3] *Kauvar D.S., Lefering R., Wade C.E*. Impact of hemorrhage on trauma outcome: an overview of epidemiology, clinical presentations, and therapeutic considerations. J Trauma. 2006; 60(6 Suppl): S3–S11. DOI: 10.1097/01.ta.0000199961.02677.19
[4] *Григорьев Е.В., Лебединский К.М., Щеголев А.В. и др*. Реанимация и интенсивная терапия при острой массивной кровопотере у взрослых пациентов. Анестезиология и реаниматология. 2020; 1: 5–24. DOI:10.17116/anaesthesiology20200115 [*Grigor'ev E.V., Lebedinskii K.M., Schegolev A.V., et al.* Resuscitation and intensive care in acute massive blood loss in adults (clinical guidelines). Russian Journal of Anesthesiology and Reanimatology. 2020; 1: 5–24. DOI:10.17116/anaesthesiology20200115 (In Russ)]
[5] Военно-полевая хирургия. Национальное руководство. Под ред. И.М. Самохвалова. 2-е изд., перераб. и доп. М.: ГЭОТАР-Медиа, 2023. DOI: 10.33029/9704-8036-6-VPX-2023-1-960 [Military field surgery. National guideline. Ed. I.M. Samohvalov. 2nd ed. Moscow: GEOTAR-Media, 2023. DOI: 10.33029/9704-8036-6-VPX-2023-1-960 (In Russ)]
[6] *Остапченко Д.А., Гутников А.И., Давыдова Л.А.* Современные подходы к терапии травматического шока (обзор). Общая реаниматология. 2021; 17(4): 65–76. DOI: 10.15360/1813-9779-2021-4-65-76 [*Ostapchenko D.A., Gutnikov A.I., Davydova L.A*. Current Approaches to the Treatment of Traumatic Shock (Review). General Reanimatology. 2021; 17(4): 65–76. DOI: 10.15360/1813-9779-2021-4-65-76 (In Russ)]
[7] *Chang R., Holcomb J.B.* Optimal Fluid Therapy for Traumatic Hemorrhagic Shock. Crit Care Clin. 2017; 33(1): 15–36. DOI: 10.1016/j.ccc.2016.08.007
[8] *Rossaint R., Afshari A., Bouillon B., et al*. The European guideline on management of major bleeding and coagulopathy following trauma: sixth edition. Crit Care. 2023; 27(1): 80. Published 2023 Mar 1. DOI: 10.1186/s13054-023-04327-7
[9] *Qi J., Bao L., Yang P., Chen D*. Comparison of base excess, lactate and pH predicting 72-h mortality of multiple trauma. BMC Emerg Med. 2021; 21(1): 80. DOI: 10.1186/s12873-021-00465-9
[10] *Mutschler M., Nienaber U., Brockamp T., et al*. Renaissance of base deficit for the initial assessment of trauma patients: a base deficit-based classification for hypovolemic shock developed on data from 16,305 patients derived from the TraumaRegister DGU®. Crit Care. 2013; 17(2): R42. DOI: 10.1186/cc12555
[11] *Odom S.R., Howell M.D., Silva G.S., et al*. Lactate clearance as a predictor of mortality in trauma patients [published correction appears in J Trauma Acute Care Surg. 2014 Mar; 76(3): 902]. J Trauma Acute Care Surg. 2013; 74(4): 999–1004. DOI: 10.1097/TA.0b013e3182858a3e
[12] *Martín-Rodríguez F., López-Izquierdo R., Castro Villamor M.A., et al*. Prognostic value of lactate in prehospital care as a predictor of early mortality. Am J Emerg Med. 2019; 37(9): 1627–32. DOI: 10.1016/j.ajem.2018.11.028
[13] *McQuilten Z.K., Wood E.M., Bailey M., et al*. Fibrinogen is an independent predictor of mortality in major trauma patients: A five-year statewide cohort study. Injury. 2017; 48(5): 1074–81. DOI: 10.1016/j.injury.2016.11.021
[14] *Verma A., Kole T*. International normalized ratio as a predictor of mortality in trauma patients in India. World J Emerg Med. 2014; 5(3): 192–5. DOI: 10.5847/wjem.j.issn.1920-8642.2014.03.006
[15] *Lalwani S., Gera S., Sawhney C., et al.* Mortality Profile of Geriatric Trauma at a Level 1 Trauma Center. J Emerg Trauma Shock. 2020; 13(4): 269–73. DOI: 10.4103/JETS.JETS_102_18
[16] *Lichtveld R.A., Panhuizen I.F., Smit R.B., et al*. Predictors of Death in Trauma Patients who are Alive on Arrival at Hospital. Eur J Trauma Emerg Surg. 2007; 33(1): 46–51. DOI: 10.1007/s00068-007-6097-6
[17] *Brown L.M., Call M.S., Margaret Knudson M., et al*. A normal platelet count may not be enough: the impact of admission platelet count on mortality and transfusion in severely injured trauma patients. J Trauma. 2011; 71(2 Suppl 3): S337–S342. DOI: 10.1097/TA.0b013e318227f67c
[18] *Тришкин Д.В., Крюков Е.В., Чуприна А.П. и др*. Методические рекомендации по лечению боевой хирургической травмы. Санкт-Петербург: Военно-медицинская академия имени С.М. Кирова, 2022. 373 с. [*Trishkin D.V., Kryukov E.V., Chuprina A.P., et al*. Guidelines for the treatment of combat surgical trauma. Saint Petersburg: S.M. Kirov Military Medical Academy. 2022. 373 p. (In Russ)]
[19] *Schork A., Moll K., Haap M., et al*. Course of lactate, pH and base excess for prediction of mortality in medical intensive care patients. PLoS One. 2021; 16(12): e0261564. Published 2021 Dec 20. DOI: 10.1371/journal.pone.0261564
[20] *Ward C.L., Olafson S.N., Cohen R.B., et al*. Combination of Lactate and Base Deficit Levels at Admission to Predict Mortality in Blunt Trauma Patients. Cureus. 2023; 15(6): e40097. Published 2023 Jun 7. DOI: 10.7759/cureus.40097

Appendix 1. Significance level (*p*) of pairwise comparisons with Bonferroni correction for 4 groups

Parameters	Comparison of groups by grades of shock, *p*									
	0–1	0–2	0–3	0–4	1–2	1–3	1–4	2–3	2–4	3–4
SBP, mmHg	0.37	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	0.03	0.006	0.154
HR, min^{-1}	0.02	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	0.012	< 0.01	0.485
SI	0.23	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	0.04	< 0.01	0.99
Erythrocytes, 10^{12}/L	0.063	0.167	< 0.01	< 0.01	0.784	< 0.01	0.133	0.001	0.258	0.461
Haemoglobin, g/L	0.397	< 0.01	< 0.01	< 0.01	0.768	0.001	< 0.01	< 0.01	< 0.01	0.182
Haematocrit, %	0.014	< 0.01	< 0.01	< 0.01	0.665	0.112	0.29	0.035	0.437	0.187
Platelets $\times 10^9$/L	0.088	0.059	< 0.01	< 0.01	0.376	0.059	0.011	0.013	0.039	0.844
Body T, °C	0.63	0.64	0.076	< 0.01	0.784	< 0.01	0.133	0.046	0.258	0.212
pH	0.34	0.084	0.05	< 0.01	0.05	0.029	0.002	0.182	0.039	0.162
BE, mmol/l	0.127	0.05	< 0.01	< 0.01	0.02	< 0.01	0.031	0.002	0.05	0.05
Lactat, mmol/l	0.02	< 0.01	< 0.01	< 0.01	0.19	0.018	0.012	0.035	< 0.01	0.008