DOI: 10.23934/2223-9022-2019-8-2-124-131

Oxygen Transport System and Its Compensatory Capabilities for Victims During Operations for Abdominal Trauma, Complicated by Blood Loss

V.H. Timerbaev[1]*, V.V. Valetova[2], A.V. Dragunov[3], OV Smirnova[4], T.F. Tukibaeva[3]

Department of Anesthesiology
[1] N.V. Sklifosovsky Research Institute for Emergency Medicine of the Moscow Healthcare Department
3 Bolshaya Sukharevskaya Square, Moscow 129090, Russian Federation
[2] Russian Medical Academy of Continuing Professional Education
2/1 Barrikadnaya St., b. 1, Moscow 125993, Russian Federation
[3] A.K. Yeramishantsev Moscow Clinical Hospital
15 Lenskaya St., Moscow 129327, Russian Federation
[4] Moscow Clinical Hospital No. 52
3 Pekhotnaya St., Moscow 123182, Russian Federation

*** Contacts:** Vladimir H. Timerbaev, Dr. Med. Sci., Chief of the Department of Anesthesiology, N.V. Sklifosovsky Research Institute For Emergency Medicine. E-mail: timerbaev56@inbox.ru

BACKGROUND Acute hemorrhage remains the leading cause of death on the operating room in emergency surgery. However, the correlation of the central hemodynamics, oxygen balance and homeostasis in such victims during emergency surgical treatment with the outcomes of surgical treatment has not yet been evaluated.

MATERIAL AND METHODS We examined 100 patients with acute massive blood loss, who had emergency surgery. We determined heart rate, arterial pressure by direct and indirect methods, central venous pressure, oxygen saturation of blood, cardiac index, systemic vascular resistance, gas and acid-base contents of arterial and venous blood, oxygen consumption, oxygen delivery, oxygen extraction ratio according to generally accepted formulas. Two groups of patients were formed of 50 people, depending on the oxygen balance (Group 1 — subcompensation, Group 2 — decompensation).

RESULTS At the time of admission to the operating room and after the surgery, the indicators of systemic hemodynamics in patients of both groups did not differ statistically significantly. In the Group 2, at the time of admission to the operating room, there were statistically significantly higher VO_2 (195 (158, 256) ml/(min·m^2) and 112.5 (86; 145.3) ml/(min·m^2)), ERO_2 (50 (45.1, 60) % and 25.1 (19.6, 33.2) %) and low SvO_2 (54.4 (48.5, 67.5) % and 75.1 (67,8; 83) %) ($p<0.001$ for all indicators). In the Group 2, there were increased values of ERO_2 and VO_2 ($p=0.001$) at the end of the operation compared to the Group 1, although the glucose and lactate levels did not differ statistically significantly between the groups. The course of the postoperative period was complicated in 9 (18%) patients of the Group 1 and 2 (4%) patients died. The course of the postoperative period of patients in the Group 2 was complicated in 9 (18%) patients and 7 (14%) patients died.

CONCLUSION Circulatory insufficiency persisted in patients who had severe oxygen deficiency disorders, despite management of bleeding, replenishment of blood loss, intraoperative intensive therapy, indicating the depletion of the compensatory mechanisms of the oxygen transport system. This was confirmed by a higher mortality rate among the victims of this group.

Keywords: oxygen balance, acute massive hemorrhage, oxygen consumption, oxygen delivery, oxygen extraction ratio

For citation Timerbaev V.H., Valetova V.V., Dragunov A.V., et al. Oxygen transport system and its compensatory capabilities for victims during operations for abdominal trauma, complicated by blood loss. *Russian Sklifosovsky Journal of Emergency Medical Care.* 2019; 8(2): 124–131. DOI: 10.23934/2223-9022-2019-8-2-124-131 (In Russian)

Conflict of interest Authors declare lack of the conflicts of interests

Acknowledgments The study had no sponsorship

Affiliations

Vladimir H. Timerbayev	Dr. Med. Sci., Chief of the Department of Anesthesiology, N.V. Sklifosovsky Research Institute For Emergency Medicine, ORCID: 0000-0002-1604-9947.
Valeria V. Valetova	Dr. Med. Sci., Professor of the Department of Anesthesiology and Emergency Medicine, Russian Medical Academy of Continuous Professional Education, ORCID: 0000-0001-6132-463x.
Anton V. Dragunov	Anesthesiologist of the A.K. Yeramishantsev Moscow Clinical Hospital, ORCID: 0000-0001-7455-8726.
Olga V. Smirnova	Anesthesiologist of the Moscow Clinical Hospital No. 52, ORCID: 0000-0002-8729-0754.
Tamila F. Tukibayeva	Anesthesiologist of the A.K. Yeramishantsev Moscow Clinical Hospital, ORCID: 0000-0002-3075-6090.

BE — base excess
BP_d — diastolic arterial blood pressure
BP_{mean} — mean arterial blood pressure
BP_s — systolic arterial blood pressure
CBV — circulating blood volume
CI — cardiac index
ctO_2 — oxygen content in blood
CVP — central venous pressure
$DavO_2$ — arterio-venous difference in oxygen content
DO_2 — oxygen delivery
ERO_2 — oxygen extraction ratio
Hb — hemoglobin
HCO_3 — bicarbonates
HR — heart rate
Ht — hematocrit
ITT — infusion-transfusion therapy
pCO_2 — partial pressure of carbon dioxide
pO_2 —oxygen partial pressure
SvO_2 — oxygenation of venous blood
SVRI — systemic vascular resistance index
VO_2 — oxygen consumption

BACKGROUND

In emergency surgery, acute blood loss remains the main cause of deaths on the operating table and in the immediate postoperative period [1–8]. The effectiveness of blood loss compensation depends on the chosen tactics of treatment, the means of therapy used and monitoring methods [3, 9-25].

The concept of optimizing intraoperative hemodynamic and oxygen transport disorders was formulated by *W.C. Shoemaker* in 1988 [26]. The trigger for the development of therapeutic algorithms of hemodynamic disorders in blood loss was the fact that among patients who had lower rates of oxygen delivery and consumption, a higher mortality rate was observed. The obvious reason is insufficient physiological reserves of the body with increasing metabolic needs against the background of blood loss [27].

The concept of modern intensive therapy of any critical conditions was formed in the 1980–1990s, when extensive studies of central hemodynamics and oxygen metabolism became possible [2, 26–31]. However, in the available domestic and foreign literature, real data on changes in central hemodynamics, oxygen balance and homeostasis during emergency surgical treatment of victims with massive blood loss are not sufficiently described today.

The aim of the study: to analyze the state of the oxygen transport system and its compensatory abilities in victims with injuries of the abdominal cavity and acute blood loss in the intraoperative period of bleeding management.

MATERIAL AND METHODS

The study included 100 patients with injuries of the abdominal organs, complicated by acute massive blood loss. Among them, there were 98 male patients (98%) and 2 female patients (2%). The average age of patients was 33 (25; 45) years. The severity of damage according to *ISS* was 35 (26; 44) score. The diagnosed injuries: spleen —32 (32%); intestines — 21 (21%); liver — 18 (18%); kidney — 8 (8%); vessels — 6 (6%); bladder — 5 (5%); stomach — 3 (3%); gallbladder — 3 (3%); tissue damage in the retroperitoneal space — 2 (2%); pancreas — 1 (1%); adrenal glands — 1 (1%).

In order to stop intra-abdominal bleeding in patients, depending on the type of damage, the following types of surgery were performed, in addition to laparotomy: splenectomy — 32; suturing of ruptured diaphragm — 27; closure of liver ruptures — 14; hemihepatectomy — 4; cholecystectomy — 3; suturing of defects or resection of the small and large intestine — 21; closure of stomach ruptures — 3; nephrectomy, closure of kidney ruptures, suturing of bladder rupture and epicistostoma —13; stiching, coagulation of damaged vessels — 6; laparotomy, suturing of mesentery — 34. Nasogastrointestinal intubation was performed in 70 patients. Revision, rehabilitation and drainage of the abdominal cavity (and/or retroperitoneal space) were carried out in 100% of patients. Most patients underwent surgery for combined injuries of the abdominal organs.

The average duration of operations was 140 (100; 190) min, the volume of blood loss was 2,000 (1,900; 2,775) ml, 24 patients (24%) were in a state of intoxication.

ANESTHESIA METHODS

All patients and victims were operated on an emergency basis according to vital indications for stopping bleeding. Anesthesia was standard. Induction was performed with benzodiazepines, Ketamine and Fentanyl in conventional dosages. Tracheal intubation was performed after administration of Cisatracurium or Rocuronium. Anesthesia was maintained by fractional administration of Ketamine and Fentanyl, myoplegia was maintained with Cisatracurium or Rocuronium. Artificial ventilation of the lungs was performed in the normal ventilation mode with an air-oxygen mixture (FiO_2 0.5–0.8).

STAGES AND METHODS OF RESEARCH

All patients underwent standard intraoperative monitoring of vital functions (*Agilent* monitor *Anesthesia V*24*C*, France). We monitored heart rate (HR), blood pressure (BP_s, BP_{mean}, BP_d) with direct and indirect methods, central venous pressure (CVP), oxygen saturation (SpO_2). The electrocardiogram was recorded in the 2nd standard lead.

We conducted continuous mini-invasive determination of central hemodynamic parameters (cardiac index – CI, systemic indicators of oxygen balance: gases of arterial and venous blood, and also oxygen consumption (VO_2) [1], oxygen delivery (DO_2) [2], oxygen extraction ratio (ERO_2) [3] by the generally accepted formulas. The gas composition of the arterial and venous blood acid-base status, hemoglobin, hematocrit, electrolytes, glucose, lactate were measured using the analyzer *Radiometer Copenhagen ABL 700/800* (*"Radiometer"*, Denmark). These indicators were studied in the express laboratory. Blood sampling of patients, as well as recording of hemodynamic parameters was performed when the patient was admitted to the operating room, immediately after introductory anesthesia or at the beginning of

surgery (stage I) and at the end of the operation, when the bleeding was arrested and surgical intervention was performed (stage II). Arterial blood samples were taken through the catheter in the radial artery, the venous samples were taken with the help of a catheter advanced through the right internal jugular vein into the cavity of the right atrium.

Statistical processing of the material was performed using the *SPSS* program *(SPSS Inc., USA)*. When analyzing quantitative traits, the median (*Me*), the standard deviation (*s*), the lower quartile (*LQ*) and upper quartile (*UQ*) were determined. Given the small number of observations, and distribution characteristics different from the normal, parameters descriptive statistics of quantitative attributes were represented as *Me (LQ, UQ).* The distribution of qualitative signs were presented in percent of the total number of patients in the subgroup. When comparing the signs between groups and evaluating the results of treatment, nonparametric criteria were used: Mann-Whitney test, Kruskal-Wallis test by ranks, Wilcoxon signed-rank test, Friedman two-way test. To determine the relationship of signs, the criterion χ^2 (chi-square) and the Spearman's rank correlation coefficient *R* were used. The results were considered statistically significant with a probability value of $p < 0.05$.

RESULTS AND DISCUSSION

During the period of retrospective analysis of the dynamics of the intraoperative oxygen balance data of the victims for the classification of signs characterizing the oxygen balance, and the selection of homogeneous groups for the purpose of further study, all the obtained data were classified using two-stage cluster analysis. As characteristics to be separated, SvO_2 (oxygen saturation of venous blood), ERO_2 and VO_2 were used. Using the procedure of two-stage cluster analysis (average silhouette 0.6), 2 clusters that were homogeneous in terms of the indicators studied were obtained, each of which had 50 victims. The basic principle of dividing victims into groups was the degree of compensation of their oxygen balance upon admission to the operating room, which affected the statistically significant differences of SvO_2, ERO_2 and VO_2 (p <0.001 for all indicators). In addition to SvO_2, ERO_2 and VO_2, there were significant intergroup differences in ctO_2 (v) and pvO_2 (p <0.001) upon admission, which were lower in the 2nd group of victims. Upon admission, $DavO_2$ was also statistically significantly higher in the 2nd group, and together with low SvO_2 and high ERO_2 it confirmed the significant stress of the oxygen balance compensation mechanisms, despite unaltered average DO_2.

When admitted to hospital, patients in both groups did not statistically significantly differ in age — 32.5 (22.6; 45) and 33 (25.7; 44.5) years; severity of damage on the *ISS* scale — 33.5 (26.3; 42.2) and 38.1 (27.8; 48.4) score; hemoglobin levels — 101 (80; 116) and 100 (86; 118) g/l; and hematocrit — 31.4 (25.2; 35.3)% and 30.9 (26.6; 36.3)%.

Upon admission to the operating room, moderate tachycardia, arterial hypotension and low CVP values were noted in patients of the 1st group; CI and SVRI were normal (Table 1). Consequently, upon admission to the operating room, hemodynamic indicators of the victims confirmed hypovolemia and its compensation with adequate performance of the heart and vascular tone.

In patients of this group (Table 1), despite continued internal bleeding, the average hemoglobin and hematocrit showed no critical reduction, although 20% of the victims at that time already had indications for blood transfusion (hematocrit less than 25%).

Table 1

Hemodynamic and oxygen balance of the affected in the Group 1 [n=50; Me (25%; 75%)]

Parameters	Values at stages of study		P value
	I stages	II stage	
HR beats per min	100 (90; 112)	89 (78; 96)	<0.001
BP_{mean}, mm Hg	75 (66; 88)	88 (83; 98)	0.001
CVP, mm Hg	0 (0;1.4)	4.1 (3.6; 5.1)	<0.001
CI, l/(min*m^2)	2.96 (2.47; 3.64)	2.7 (2.34; 3.16)	0.019
SVRI, dyn*sec*cm^5/m^2	1,872 (1,324; 2,336)	2,323 (1,874; 3,029)	<0.001
pH (a)	7.33 (7.29; 7.39)	7.35 (7.31; 7.39)	0.181
pO_2 (a), mm Hg	186 (119; 227)	186 (127; 271)	0.225
HCO_3 (a), mmol/l	20.3 (18.1; 22.6)	21.5 (20.2; 23.6)	0.057
pCO_2 (a), mm Hg	37.4 (34.6; 41.2)	38.9 (35.5; 45.2)	0.036
BE (a), mmol/l	-4.8 (-7.7; -2.4)	-3.7 (-4.8; -1.5)	0.04
pCO_2 (v), mm Hg	45.9 (41.9; 50.5)	43.3(38.9; 48.6)	0.135
pO_2 (v), m Hg	48.7 (39.8; 53.2)	54.9 (46.5; 62.1)	0.004
SvO_2, %	75.1 (67.8; 83)	80.9 (75.5; 86.3)	0.003
$DavO_2$, ml/dl	3.0 (2.03; 4.18)	3.4 (2.2; 4.1)	0.692
Hb, g/l	101 (80.8; 116)	100 (89.3; 110.3)	0.89
Ht, %	31 (25.2; 35.2)	30.6 (27; 33.5)	0.854
Glucose (a), mmol/l	9.05 (7.38; 13.53)	8.3 (7.05; 9.5)	0.004
Lactate (a), mmol/l	2.85 (2.12; 3.83)	2.75 (2.28; 3.53)	0.911
DO_2, ml/(min*m^2)	400 (314; 526)	357 (309; 433)	0.217
VO_2, ml/(min*m^2)	112 (86; 145)	97 (78;138)	0.465
ERO_2, %	25.1 (19.6; 33.2)	24.5 (19.2; 30.2)	0.969
ctO_2 (a), ml/dl	13.2 (11.1; 16.0)	13.9 (12.6; 14.8)	0.257
ctO_2 (v), mg/dl	10.5 (8.6; 12.8)	11.2 (9.2; 12.8)	0.176

Notes: BE — base excess; BP_{mean} — mean arterial blood pressure; CI — cardiac index; ctO_2 — oxygen content in the blood; CVP — central venous pressure; $DavO_2$ — arterio-venous difference in oxygen content; DO_2 — oxygen delivery; ERO_2 — oxygen extraction ratio; Hb — hemoglobin; HCO_3 — bicarbonates; HR — heart rate; Ht — hematocrit; pCO_2 — carbon dioxide partial pressure; pO_2 — oxygen partial pressure; SvO_2 — oxygenation of venous blood; SVRI — systemic vascular resistanse index; VO_2 — oxygen consumption

PO_2 and pCO_2 values were within normal limits, although the total oxygen content in arterial blood was reduced due to anemia. The decrease in DO_2 was associated with continued bleeding, hypovolemia and moderate anemia. However, VO_2 was in the range of normal values with normal ERO_2.

Upon admission to the operating room, the patients of the 1st group showed signs of subcompensated metabolic

acidosis with a decrease in pH and base excess *BE*) (Table 1). Despite the correspondence to the proper values of the mean values of pvO_2 and SvO_2, in 30% of patients of the 1st group, these indicators exceeded the reference values upon admission, which indicated possible arteriovenous bypass of blood. The content of lactate in the blood were moderately elevated.

By the end of surgical intervention, blood loss in victims was 2,000 (1,800; 2,725) ml. In total, during the operation, we administered 4,582 (3,239; 5,923) ml of infusion and transfusion fluids, including: colloids 800 (500; 1,400) ml; crystalloids 2,300 (1,800; 3,050) ml; fresh frozen plasma 635 (548; 1016) ml; donor erythrocytes 518 (303; 729) ml; autoerythrocytes 650 (418; 968) ml. Diuresis during the operation averaged 500 (300; 1,200) ml.

By the end of surgery, almost all studied hemodynamic parameters have changed significantly: heart rate decreased, mean arterial blood pressure, CVP and SVRI increased (for all indicators $p < 0.05$ when compared with the previous stage); CI has not changed much. That is, when bleeding was managed intraoperatively and blood loss was replenished, positive hemodynamic changes have been noted. There has been an increase in SVRI as a compensatory-adaptive response to injury and blood loss.

Red blood counts: hemoglobin and hematocrit changed statistically insignificantly after arresting the bleeding and initiating infusion-transfusion therapy (Table 1). Upon admission to the operating room, 20% of the victims had indications for blood transfusion (hematocrit less than 25%), at the end of the operation 10% of patients still had indications for continuing blood transfusion.

By the end of the surgical intervention, the indices of pO_2 and pCO_2 remained normal. When calculating the oxygen content in the blood, attention was drawn to the fact that ctO_2 values less than 15 mg/ml were observed at both stages of the study, being associated with a reduced amount of hemoglobin.

The decrease in DO_2 upon admission was associated with continued bleeding, hypovolemia and moderate anemia. Despite the cessation of bleeding, the compensation of the CBV and the lost globular volume, by the end of the operation DO_2 even slightly decreased. It is important that in the absence of obvious signs of an imbalance of oxygen metabolism in 30% of victims, DO_2 was below the critical level of 320 ml/(min*m^2) both before the operation and after the surgical intervention. This is a situation when oxygen consumption becomes directly dependent on oxygen delivery, as well as metabolic disorders characteristic of tissue ischemia and shock, appear and progress.

At both stages of the study, VO_2 was slightly lower than the reference values. Despite intraoperative compensation of CBV and non-critical decrease in hemoglobin after surgery, lower values of the indicator were noted in 68% of the 1st group patients. The decrease in VO_2 might possibly be associated with anesthesia, myoplegia, and induced hypothermia. However, one cannot completely exclude the fact that a decrease in DO_2 was also associated with inhibition of aerobic metabolism and switch to anaerobic metabolism.

Despite the low values of indicators of transport and oxygen consumption, the average values of ERO_2 corresponded to the lower limit of the norm, which does not exclude the tension of compensatory mechanisms and the formation of oxygen debt. Thus, upon admission to the operating room, in 36% of the injured patients of the 1st group ERO_2 was lower than 22%, and in 10% it exceeded 35%. By the end of the operation, ERO_2 remained normal in 36% of the affected, and in 18% of cases it exceeded the reference values. Therefore, we cannot exclude the fact that the normal values of ERO_2 at the 2nd stage of the study reflect the effect of anesthesia drugs and/or arteriovenous bypass mechanisms on the oxygen consumption. At first glance, the dynamics of SvO_2 during the operation seems to be positive. However, with a detailed assessment, it could be noted that already in 56% of the affected SvO_2 exceeded the norm. Despite the absence of statistically significant differences in the oxygen content in arterial and venous blood before and after surgery, we found that the arteriovenous difference in oxygen ($DavO_2$) at both stages of the study did not reach 5 mg/dl , which might indicate oxygen imbalance and tension of compensatory mechanisms of metabolism regulation, an increase in arteriovenous bypass and centralization of blood circulation. It is possible that this process did not depend on the promptness of the performed hemostasis and the adequacy of CBV replenishment, but was associated with the terms of the injury and the general reaction to trauma and blood loss.

The imbalance of oxygen metabolism and the stress of compensatory mechanisms were also reflected in an increased level of glycemia and a moderate increase in the blood lactate content at the stages of the study (Table 1). Despite a statistically significant decrease in glycemia at the end of the operation, we did not reveal any significant changes in lactate levels.

The postoperative period was complicated in 9 patients (18%) of the 1st group, including 8 (16%) with pneumonia and purulent tracheobronchitis, and one (2%) with acute cardiovascular insufficiency. All victims survived surgery. But in the postoperative period, 2 patients (4%) of the 1st group died.

Upon admission to the operating room, patients of the 2nd group also had moderate tachycardia, hypotension and low CVP values. Circulatory reaction was normal or even closer to the hypokinetic: CI and SVRI approached the reference values (Table 2). It should be noted that upon admission to the operating room, hemodynamic parameters of did not statistically significantly differ in both groups. In patients of the 2nd group, like in the 1st one, despite continued internal bleeding, the average hemoglobin and hematocrit did not reflect a critical reduction, although 18% of the victims already had indications for blood transfusion (hematocrit less than 25%).

On admission to the operating room, pO_2 and pCO_2 were within normal limits, but the total oxygen content in arterial blood was reduced due to anemia. A decrease in DO_2 upon admission (Table 2) was associated with continued bleeding, hypovolemia, moderate anemia, and a decrease in cardiac performance. However, VO_2 was in the range of normal values, which was provided by a significantly increased ERO_2 .

Table 2

Hemodynamic and oxygen balance of the affected in the Group 1 [n=50; Me (25%; 75%)]

Parameters	Values at stages of study		P value
	I stage	II stage	
HR, beats per min	100 (96; 113)	90 (85; 99)	p<0.001
$BP_{mean,}$ mm Hg	71 (57; 97)	88 (80; 94)	p=0.004
CVP, mm Hg	0 (0;0.5)	4.2 (3.5; 5.8)	p<0.001
CI, l/(min*m^2)	2.7 (2.3; 3.7)	2.9 (2.7; 3.2)	p=0.297
SVRI, dyn*sec*cm^5/m^2	1,761 (1,246; 2,265)	2,150 (1,902; 2,698)	p=0.031
pH (a)	7.31 (7.28; 7.37)	7.36 (7.32; 7.41)	0.001
pO_2 (a), mm Hg	163 (118; 212)	162 (127; 189)	0.919
HCO_3 (a), mmol/l	19.9 (18.1; 21.9)	22.5 (21.3; 24.0)	<0.001
pCO_2 (a), mm Hg	39.3 (35.2; 46.3)	40.2 (36.3; 43.7)	0.721
BE (a), mmol/l	-5.25 (-7.03; -2.5)	-3 (-4.18; 0.2)	0.001
pCO_2 (v), mm Hg	43.7 (40.8; 50.9)	44.6 (41.8; 49.7)	0.824
pO_2 (v), m Hg	38.5 (29.0; 45.9)	46.9 (37.4; 56.3)	<0.001
SvO_2, %	54.4 (48.5; 67.5)	74.6 (62.5; 80.7)	<0.001
$DavO_{2,}$ ml/dl	5.75 (4.0; 7.95)	3.75 (3.2; 5.2)	<0.001
Hb, g/l	100 (86; 118.3)	92 (81.3; 105)	0.015
Ht, %	30.9 (26.6; 36.3)	28.4 (23.9; 32.4)	0.008
Glucose (a), mmol/l	9.5 (6.9; 12.3)	8.9 (7.4; 12.2)	0.585
Lactate (a), mmol/l	2.7 (1.72; 3.68)	2.8 (1.0; 3.85)	0.242
$DO_{2,}$ ml/(min*m^2)	409 (304; 495)	384 (321; 452)	0.377
$VO_{2,}$ ml/(min*m^2)	195 (158; 256)	131 (102; 201)	<0.001
ERO_2,%	50 (45.1; 60)	38 (25.3; 52)	<0.001
ctO_2 (a), ml/dl	14.1 (11.9; 16.4)	13.2 (11.6; 14.9)	0.099
ctO_2 (v), mg/dl	8.0 (5.9; 9.4)	9.4 (7.8; 11.2)	0.001

Notes: BE — base excess; BP_{mean} — mean arterial blood pressure; CI — cardiac index; ctO_2 — oxygen content in the blood; CVP — central venous pressure; $DavO_2$ — arterio-venous difference in oxygen content; DO_2 — oxygen delivery; ERO_2 — oxygen extraction ratio; Hb — hemoglobin; HCO_3 — bicarbonates; HR — heart rate; Ht — hematocrit; pCO_2 — carbon dioxide partial pressure; pO_2 — oxygen partial pressure; SvO_2 — oxygenation of venous blood; SVRI — systemic vascular resistanse index; VO_2 — oxygen consumption

Also, blood tests showed signs of moderate decompensated metabolic acidosis with decreased pH and base excess (Table 2). The mean SvO_2 values were lower than the reference values in 68% of patients, and in 8% this indicator exceeded 80%. However, the arteriovenous oxygen difference was within normal limits and the blood lactate did not indicate the predominance of anaerobic metabolism in body tissues (Table 2).

By the end of surgery, total blood loss was 2,000 (1,975; 2,825) ml. The volume of intraoperative infusion therapy in patients of this group was 7,605 (5,563; 10,395) ml and included: 1,550 (900; 2,500) ml of colloids; 3,100 (2,675; 4,250) ml of crystalloids; 1,280 (699; 1,704) ml of fresh frozen plasma; 625 (375; 1,109) ml of fresh frozen plasma; 860 (349; 1,426) ml of autocellular component. Diuresis during the operation averaged 700 (500; 1,200) ml.

By the end of surgery the majority of hemodynamic parameters significantly improved (heart rate, mean arterial blood pressure, CVP, CI, SRVI (p<0.05)). It should be noted that all hemodynamic parameters at the end of the operation differed slightly in both groups. Like in the 1st group of victims, SRVI increased by the end of the operation in the 2nd group. Consequently, despite the differences in baseline oxygen balance, the response of the cardiovascular system to injury, surgery, anesthesia, and therapy was similar in both groups.

Despite hemostasis and transfusion of hemoglobin-containing fluids, hemoglobin and hematocrit levels were statistically insignificant at the end of the operation, which may have been associated with hemodilution after infusion-transfusion therapy (ITT). After the end of the surgical procedure, 34% of patients still had indications for continued blood transfusion.

Oxygenating and ventilation function of the lungs of patients of this group during the whole observation period remained normal, and the oxygen content in arterial blood was almost unchanged.

The statistically significant dynamics of the medians of pvO_2 and SvO_2 reflected the stabilization of the oxygen balance, despite the low values of ctO_2 (a). However, at the end of the operation in 42% of patients SvO_2 was below 60%, and in 26% of victims it exceeded 80%. Such a difference in the indices indicated that the tension of the mechanisms of oxygen metabolism and the presence of a significant proportion of victims with arteriovenous bypass of blood persisted. The decrease in $DavO_2$ by the end of the operation reflected an imbalance between oxygen delivery and the need for it. At the same time, the value of $DavO_2$ was significantly below the normal value — 3.75 (3.2; 5.2) ml/dl (Table 2).

The tension of the compensatory mechanisms in the imbalance of oxygen metabolism in the 2nd group was also manifested by moderate hyperglycemia and lactate acidosis. We have not identified significant dynamics of these indicators after surgery. Medium DO_2 was below the reference values, but exceeded the critical level. A detailed analysis showed that upon admission to the operating room DO_2 was below 320 ml/(min*m^2) in 34% of patients, and after completion of the operation low DO_2 values were revealed in 26% of the patients.

Upon admission to the operating VO_2 exceeded the normal values in 76% cases of the 2nd group. After surgery and

ITT, the DO_2 indicators corresponded to reference values (Table 2), but in 36% it exceeded the norm. Probably, higher oxygen consumption was due to prior oxygen debt or a stress response to trauma and surgery. The discrepancy between the need for oxygen and its delivery on admission is reflected in elevated values of ERO_2 in almost all the victims of the 2nd group. Normalization of VO_2 by the end of the operation in 50% of patients of the 2nd group was accompanied by a decrease in ERO_2 to the reference values. DO_2 decreased by the end of the operation, however, there was no significant decompensation of the oxygen balance, since VO_2 decreased statistically significantly at the same time.

The postoperative period was complicated in 9 patients (18%) of the 2nd group: one patient (2%) had pneumonia, 5 (10%) had purulent-septic complications, 2 (4%) had acute cardiovascular failure, and one patient (2%) had posthypoxic encephalopathy.

In the postoperative period, 7 victims (14%) of the 2nd group died.

CONCLUSION

In both groups of victims with injuries of the abdominal cavity organs, complicated by massive blood loss, at the time of admission to the operating room, the average systemic hemodynamics did not demonstrate the severity of their condition and were not significantly different. There were signs of hypovolemia such as decreased CVP, moderate tachycardia, arterial hypotension with normal vascular tone and cardiac output. After surgery to manage bleeding and intraoperative blood loss replenishment hemodynamic parameters improved — heart rate approached to normal, blood pressure and CVP increased, CI remained stable. At the same time, an increase in SVRI was observed, indicating an increase in vascular tone.

Upon admission to the operating room, patients of the 2nd group were already in a more serious condition than patients of the 1st group, the severity of their condition was only manifested by oxygen balance indicators. Thus, the injured of the 2nd group statistically significantly had higher VO_2 (195 (158; 256) ml/(min*m^2) and 112.5 (86; 145.3) ml/(min*m^2)), ERO_2 (50 (45.1; 60)% and 25.1 (19.6; 33.2)%) and low SvO_2 (54.4 (48.5; 67.5)% and 75.1 (67.8; 83)%) (p <0.001 for all indicators). These data indicate that victims experienced severe respiratory or circulatory hypoxia during the injury, which was partially managed during the period of primary care upon admission to the operating room or transportation, but the victims still had “laboratory signs of oxygen debt”.

By the end of the operation, the average oxygen balance was statistically significantly different between the groups. The Group 2 still had the tension of the compensatory and adaptive mechanisms to maintain aerobic metabolism such as ERO_2 and VO_2 (p=0.001). Accordingly, at the end of the operation, the Group 2 had lower values of ctO_2 (v) (p=0.002), pvO_2 (p=0.017), SvO_2 (p=0.001), although the level of glucose and lactate between the groups differed statistically insignificantly. Consequently, despite the control of bleeding in patients of the 2nd group, replenishment of blood loss and the conducted intraoperative intensive therapy, signs of circulatory insufficiency persisted, indicating that the compensatory mechanisms of the oxygen transport system were exhausted. This was confirmed by a higher mortality rate among the victims of this group.

The modern practice of intensive care requires the addition of new criteria and its individualization for assessing the severity of the condition and the quality of intensive care based on a set of indicators — parameters of central and systemic hemodynamics. Particular attention should be paid to the dynamics of transport and oxygen consumption, as well as metabolic processes in tissues.

REFERENCES

1. Akaraborworn O. Damage control resuscitation for massive hemorrhage. *Chin J Traumatol*. 2014; 17(2): 108–111. PMID: 24698581.
2. Shoemaker W.C., Appel P.L., Waxman K., et al. Clinical trial of survivors’ cardiorespiratory patterns as therapeutic goals in critically ill postoperative patients. *Crit Care Med*. 1982; 10(6): 398–403. PMID: 7042206.
3. Cohen M.J. Towards hemostatic resuscitation: the changing understanding of acute traumatic biology, massive bleeding, and damage-control resuscitation. *Surg Clin North Am*. 2012; 92(4): 877–891, viii. PMID: 22850152. DOI: 10.1016/j.suc.2012.06.001.
4. Perrotta S., Stiehl D.P., Punzo F., et al. Congenital erythrocytosis associated with gain-of-function HIF2A gene mutations and erythropoietin levels in the normal range. *Haematologica*. 2013; 98(10): 1624–1632. PMID: 23716564. PMCID: PMC3789469. DOI: 10.3324/haematol.2013.088369.
5. Spoerke N., Michalek J., Schreiber M., et al. Crystalloid resuscitation improves survival in trauma patients receiving low ratios of fresh frozen plasma to packed red blood cells. *J Trauma*. 2011; 71 (2, Suppl 3.): S380–383. PMID: 21814108. DOI: 10.1097/TA.0b013e318227f1c5.
6. Bansal V., Fortlage D., Lee J.G., et al. Hemorrhage is more prevalent than brain injury in early trauma deaths: the golden six hours. Eur *J Trauma Emerg Surg.* 2009; 35(1): 26–30. PMID: 26814527. DOI: 10.1007/s00068-008-8080-2.
7. Curry N., Hopewell S., Dorée C., et al. The acute management of trauma hemorrhage: a systematic review of randomized controlled trials. *Crit Care.* 2011; 15(2): R92. PMID: 21392371. PMCID: PMC3219356. DOI: 10.1186/cc10096.
8. Zehtabchi, S., Nishijima D.K. Impact of transfusion of fresh-frozen plasma and packed red blood cells in a 1:1 ratio on survival of emergency department patients with severe trauma. *Acad Emerg Med*. 2009; 16(5): 371–378. PMID: 19302364. DOI: 10.1111/j.1553-2712.2009.00386.x.
9. Valetova V.V., Ermolov A.S., Timerbaev V.H., Dragunov A.V. The influence of intraoperative infusion-transfusion therapy on massive blood loss patients lethality. *Anesteziologiya i reanimatologiya*. 2012; (2): 23–27. (In Russian).
10. Gorodetskiy V.M. Modern philosophy of transfusion therapy for traumatic massive blood loss. *Gematologiya i transfuziologiya*. 2012; (3): 3–5. (In Russian).
11. Bouglé, A., Harrois A., Duranteau J. Resuscitative strategies in traumatic hemorrhagic shock. *Ann Int Care*. 2013; 3(1): 1. PMID: 23311726. PMCID: PMC3626904. DOI: 10.1186/2110-5820-3-1.
12. Bunn F., Trivedi D., Ashraf S. Colloid solutions for fluid resuscitation. *Cochrane Database Syst Rev*. 2011; 3: CD001319.
13. Duchesne J.C., Kimonis K., Marr A.B., et al. Damage control resuscitation in combination with damage control laparotomy: a survival advantage. *J Trauma*. 2010; 69(1): 46–52. PMID: 20622577. DOI: 10.1097/TA.0b013e3181df91fa.
14. Cotton B.A., Reddy N., Hatch Q.M., et al. Damage control resuscitation is associated with a reduction in resuscitation volumes and improvement survival in 390 damage control laparotomy patients. *Ann Surg.* 2011; 254: 598–605. PMID: 21918426. PMCID: PMC3816774. DOI: 10.1097/SLA.0b013e318230089e.
15. Duchesne J.C., McSwain N.E. Jr., Cotton B.A., et al. Damage control resuscitation: the new face of damage control. *J Trauma*. 2010; 69(4): 976–990. PMID: 20938283. DOI: 10.1097/TA.0b013e3181f2abc9.
16. Annane D., Siami S., Jaber S., et al. Effects of fluid resuscitation with colloids vs crystalloids on mortality in critically ill patients presenting with hypovolemic shock: the CRISTAL randomized trial. JAMA. 2013; 310(17): 1809–1817. PMID: 24108515. DOI: 10.1001/jama.2013.280502.
17. Fröhlich M., Lefering R., Probst C., et al. Epidemiology and risk factors of multiple-organ failure after multiple trauma: An analysis of 31,154 patients from the TraumaRegister DGU. J Trauma Acute Care Surg. 2014; 76(4): 921–928. PMID: 24662853. DOI: 10.1097/TA.0000000000000199.
18. Geeraedts L.M. Jr., Kaasjager H.A., van Vugt A.B., Frölke J.P. Exsanguination in trauma: A review of diagnostics and treatment options. Injury. 2009; 40(1): 11–20. PMID: 19135193. DOI: 10.1016/j.injury.2008.10.007.
19. Bacter C.R., Canizaro P.C., Carrico C.J., Shires G.T. Fluid resuscitation of hemorrhagic shock. Postgrad Med. 1970; 48(3): 95–99. PMID: 5460379.

20. Kaafarani H.M., Velmahos G.C. Damage Control Resuscitation In Trauma. Scand J Surg. 2014; 103(2): 81–88. PMID: 24777616. DOI: 10.1177/1457496914524388.
21. Kesinger, M.R., Puyana J.C., Rubiano A.M. Improving Trauma Care in Low- and Middle-Income Countries by Implementing a Standardized Trauma Protocol. World J Surg. 2014; 38(8): 1869–1874. PMID: 24682314. DOI: 10.1007/s00268-014-2534-y.
22. Kobayashi L., Costantini T.W., Coimbra R. Hypovolemic shock resuscitation. Surg Clin North Am. 2012; 92(6): 1403–1423. PMID: 23153876. DOI: 10.1016/j.suc.2012.08.006.
23. Page C., Retter A., Wyncoll D. Blood conservation devices in critical care: a narrative review. Ann Intensive Care. 2013; 3(1): 14. PMID: 23714376. PMCID: PMC3673809. DOI: 10.1186/2110-5820-3-14.
24. Palmer L., Martin L. Traumatic coagulopathy (part 1). Resuscitative strategies. J Vet Emerg Crit Care (San Antonio). 2014; 24(1): 63–74. PMID: 24382014. DOI: 10.1111/vec.12130.
25. Cotton B.A., Au B.K., Nunez T.C., et al. Predefined massive transfusion protocols are associated with a reduction in organ failure and postinjury complications. J Trauma. 2009; 66(1): 41–48. PMID: 19131804. DOI: 10.1097/TA.0b013e31819313bb.
26. Shoemaker W.C., Appel P.L., Kram H.B. Tissue oxygen debt as a determinant of lethal and nonlethal postoperative organ failure. Crit Care Med. 1988; 16(11): 1117–1120. PMID: 3168504.
27. Shere-Wolfe R.F., Galvagno S.M. Jr., Grissom T.E. Critical care considerations in the management of the trauma patient following initial resuscitation. Scand J Trauma Resusc Emerg Med. 2012; 20: 68. PMID: 22989116. PMCID: PMC3566961. DOI: 10.1186/1757-7241-20-68.
28. Shoemaker W.C., Appel P.L., Kram H.B., et al. Prospective trial of supranormal values of survivors as therapeutic goals in highrisk surgical patients. Chest. 1988; 94(6): 1176–1186. PMID: 3191758. DOI: 10.1378/chest.94.6.1176.
29. Shoemaker W.C., Appel P.L., Kram H.B. Hemodynamic and oxygen transport responses in survivors and nonsurvivors of high-risk surgery. Crit Care Med. 1993; 21(7): 977–990. PMID: 8319478.
30. Shoemaker W.C., Appel P.L., Kram H.B. Measurement of tissue perfusion by oxygen transport patterns in experimental shock and in high-risk surgical patients. Int Care Med. 1990; 16(Suppl 2): S135–144. PMID: 2289979.
31. Shoemaker W.C., Appel P.L., Kram H.B. Role of oxygen debt in the development of organ failure sepsis, and death in highrisk surgical patients. Chest. 1992; 102(1): 208–215. PMID: 1623755. DOI: 10.1378/chest.102.1.208.

Received on 14.09.2018

Accepted on 11.10.2018