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Experimental Medicine

Arterial and Venous Blood Gas Monitoring in Hemorrhagic Shock

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ABSTRACT. We aimed at evaluating the informativeness of arterial and venous blood indices at hemorrhagic shock and gravity of hemorrhagic shock in animal models with the use of the in vitro system AVL (England).

The model of severe hemorrhagic shock (HS) in 2-4 kg anesthetized (nembutal narcosis - 40 mg/kg) male cats (n=8) was induced by the Wiggers-fine' method through bloodletting. Intraarterial blood effusion summary 40mg/kg, till arterial blood pressure dropped to 40mm/hg for 1 hr. Arterial and venous blood gas monitoring was conducted in dynamics: at 30th min (HS 30), at 60th min (HS 60) and at 80th min. Blood was taken from femoral vein and artery (v.et.a.femoralis) using microcapillary TYPPE 551T 1/18 5/100 and studied on AVL (England). A total of 174 blood gas samples: arterial and venous pH, partial pressure of $PC0_2$, mmHg, partial pressure of PO_2, mmHg, bases excess BE mmol/l, buffer bases BB mmol/l, bicarbonate HCO_3, mmol/l, saturated O_2 sat mmHg, alveolo-arterial oxygen deficiency ($AaDO_2$) and cardio circulatory oxygen deficiency $CCDO_2$ - % were measured. The obtained results were analyzed statistically with the use of STX program. Reduction of arterial blood effusion to 40 ml/kg/ led to the following alterations: decreases in mean arterial blood pressure (from 124 mmHg to 60 mmHg – at HS30 and 40 mmHg - at HS60), In venous blood control $CCDO_2$ increases from 17.10±1.20 % to 21.43±1.15 % . At HS60 and 80th min $CCDO_2$ increases from 43.3±3.60% to 47.80±1.03% ($p< 0.05$, $p< 0.01$, $p<0.01$). In arterial blood control $CCDO_2$ was 0%, while at HS30, HS60 and at 80th min $CCDO_2$ increases and is equal to 9.0±1.3%, 43.3±3.63% and 50.1±1.3%, respectively. ($p<0.01$, $p<0.01$, $p<0.01$). Arterial $PC0_2$ varies and at HS30, HS60 and 80th min it is 40.55±1.09 mmHg, 32.40±2.40 mmHg ($p< 0.05$), 38.66±1.20 mmHg and 45.60±1.3 mmHg ($p< 0.05$). After 80th min irreversible changes develop. In the experiments carried out PCO_2 differs from control data. Arterial and venous blood gas monitoring revealed close correlation of altered PCO_2 and $CCDO_2$ indices with acidosis, which could be used for assessment of acidosis at hemorrhagic shock. Changes in venous PCO_2 and $CCDO_2$ have recently been shown to correlate with changes in global tissue perfection (cardio-vascular system). Such data, available immediately via continuous venous blood gas monitoring, may be useful for monitoring shock and the response to resuscitation. The results of blood gas monitoring agrees closely with the results obtained with the use of fiberoptic three-dimensional sensors.

This method could be used in different unstable situations. In vitro AVL system provides additional possibilities to investigate other vitally important parameters (buffers, $CCDO_2$, $AaDO_2$) etc. *© 2011 Bull. Georg. Natl. Acad. Sci.*

***Key words**: AVL, hemorrhagic shock, buffer systems, BB, BE, $CCDO_2$, PCO_2, PO_2, $AaDO_2$, $CCDO_2$, O_2sat, cats.*

The adaptive and compensatory reactions developed at hemorrhagic shock fail against the background of total hypoxia and subsequent accumulation of metabolic waste products as a result of dramatically disordered hemocirculation. Alterations in arterial and venous blood gas indices at HS and their continuous monitoring at various stages of HS provides valuable information about the disorders of metabolic processes in the organism. Reaction of the organism at cellular, subcellular and organ levels, developed in response to progressive hypovolemia, is a whole system of metabolic processes, which is under influence of various factors and has been revealed by disordered microhemocirculation, altered neuro-humoral regulation, abnormal membrane permeability and disordered transmission of vitally important elements [1-3]. A good indicator for evaluation of the above-mentioned disorders is the monitoring of arterial and venous blood gases and investigation of buffer systems in organism. The current standard for blood gas analysis is intermittent blood gas sampling with measurements performed in vitro, in a blood gas analyzer. Recent advances in optical sensor technology have allowed the development of miniaturized fiberoptic devices that can be placed intravascularly to continuously measure changes in PO_2, PCO_2 and pH. Continuous monitoring and investigation of arterial and venous blood gases is possible with the use of the Puritan-Bennetti 3300 system (PB3300) and fiberoptic three-dimensional sensors. This method provides information in vivo in unstable conditions [4].

Material and methods: The model of severe hemorrhagic shock (HS) in 2-4 kg anesthetized (nembutal narcosis - 40 mg/kg interarterial blood exfusion 40 ml/kg) male cats (n=8) was induced by the Wiggers-Fine method [1] through bloodletting. Animals were bled to a femoral arterial blood pressure 40 ml/Kg. Catheter was inserted in left femoral artery (a.femoralis sinistra) for measurement and monitoring of arterial blood pressure. The blood was withdrawn from the same place (left femoral artery-a.femoralis sinistra). Blood was taken from right femoral vein and artery (v.et a.femoralis dexter) using microcapillary TYPPE 551T 1/18 5/100 and studied on AVL (England). Blood test was conducted at 37 ^{0}C on the AVL.

Arterial and venous blood gas monitoring were conducted in dynamics: in norm (control), at 30th min (HS30) at arterial pressure 60 mmHg, on 60th min (HS60) at arterial pressure 40 mmHg and on 80th min, when arterial pressure was 20 mmHg. Fluid with equivalent amount of withdrawn blood and diuresis was retransfused in left femoral artery (a.femoralis sinistra). Body core temperature was maintained at 37 ^{0}C. A total of 174 blood gas samples: arterial and venous pH, partial pressure of PCO_2, mmHg, partial pressure of PO_2, mmHg, Bases Excess BE mmol/l, Bufer Bases BB mmol/l, bicarbonate HCO_3, mmol/l, saturated O_2sat %, alveolo-arterial oxygen deficiency ($AaDO_2$) and cardio circulatory oxygen deficiency $CCDO_2$ - % were measured. Obtained results were analyzed statistically with the use of STX program.

Results and discussion. The experimental results showed (see tables 1, 2; Figs. 1- a), b), 2- a), b)) that some parameters of the arterial and venous gas structure and buffer system had a tendency to increase, some to decrease. In the arterial blood PCO_2, BE, $AaDO_2$, $CCDO_2$ and $AaDO_2$ increase, but - PH, BB, HCO_3, PO_2, O_2 sat, $AaDO_2$ and $CCDO_2$ – decrease. In the venous blood PCO_2 and $CCDO_2$ increase, but PH, BE, BB, HCO_3, PO_2 and O_2 sat decrease. The activity of biological macromolecule structure and finally, function of cell defines PH. The majority of ferments have determined PH, whose increase or decrease regress their activity. In arterial blood the control group pH was 7.276+0.034 and it decreased to 7.112+0.012 at 80th min ($P<0.01$). In venous blood pH decreased from 7.245+0.048 to 7.049+0.032 ($P<0.01$). The reduction ten-

Table 1.

Arterial blood gases change of hemorrhagic shock in male cats (M±m, n=8)

Index	Control group	HS30	HS60	80th min
pH	7.276±0.34	7.271±0.086	7.217±0.021 p<0.001	7.112±0.012 p<0.01
PCO_2 (mmHg)	40.55±1.09	32.4±2.4	38.66±1.2	45.6±1.3 p<0.05
BE (mmol/l)	-7.87±1.14	-7.71±1.95	-13.22±1.41 p<0.05	-14.3±1.98 p<0.05
BB (mmol/l)	40.02±1.14	39.17±3.39	35.27±1.06 p<0.05	33.601±1.11 p<0.01
HCO_3(mmol/l)	18.3±1.04	17.22±1.62	13.21±1.14	13.6±1.11 p<0.05
PO_2 (mmHg)	112.53±13.75	101.9±7.72	76.6±5.5 p<0.05	55.2±9.8 p<0.001
O_2 sat %	97.04±1.4	96.23±1.1	89.98±1.1 p<0.01	81.1±2.15 p<0.001
$AaDO_2$ %	0	0	21.9±1.1 p<0.01	39.21±14.44 p<0.001
$CCDO_2$%	0	9±1.3	43.3±3.63 p<0.001	50.1±1.3 p<0.001

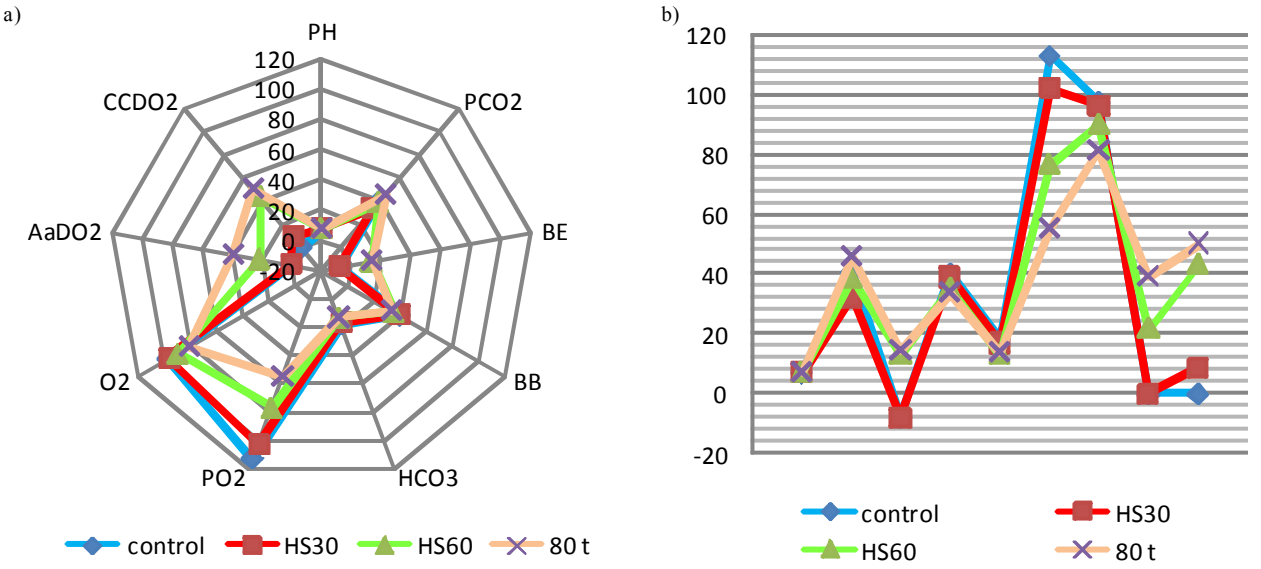

Fig. 1. Arterial blood gases structure at hemorrhagic shock in male cats (M±m, n=8). a) Arterial blood gases structure priority parameter change in femoral artery; b) The dynamic change of hypothetic model in arterial blood gases structure in femoral artery.

dency in pH points to disordered metabolic processes in organism, which has been revealed in internal organs (heart, liver) by increased level of lactic acid [5-7]. These data reflect initial hypoxic processes developed in internal organs.

In control group animals the venous PCO_2 was increased from 44.03±3.16 to 64.23±3.18 mmHg ($p< 0.001$), pointing to early stage of shock during which organism reacts by hyperventilation and adequate perfusion. In arterial blood the same parameter PCO_2 was 40.55±1.09 mmHg, at HS30 it decreased to 32.4±2.4 mmHg ($p< 0.05$), at HS60 started elevation and it was 38.66±1.2 mmHg ($p<0.05$) and at 80th min PCO_2 increased to 45.60±1.30 mmHg ($P<0.05$).

This decrease in PCO_2 at HS30 could be explained by consumption of CO_2 for synthesis of bicarbonates in renal tubules, but the latter increase in PCO_2 indicates malfunctioning of the kidney in producing bicarbonates.

At HS, BE and BB increase in both femoral artery and vein (a.et v.femoralis). In arterial blood BE increases from -7.87±1.14 to -14.3±1.98 mmol/l ($p<0.05$) and in venous blood – from 8.18±1.62 to -14.8±1.13 mml/l ($P<0.001$). In arterial and venous blood BE at 80th min decreases almost equally. Depletion of BE develops against the background of progressive acidosis, indicating a malfunctioning of the liver in restoring pH level at a late stage of the shock. At 80th min utilization of lactate also decreases, which is initially revealed in venous- and later in arterial blood (see Tables 1 and 2). BB sharply decreases in arterial and venous blood. Arterial BB decreases from 40.02±1.14 to 33.60±1.11 mmol/l ($P<0.01$), venous BB decreases from 39.78±2.26 to 33.61±1.31 mmol/l ($P<0.05$). This decrease in BB correlates with the severity of shock. HCO_3 in arterial blood decreases from 18.30±1.04 to 13.60±1.11 mmol/l ($P<0.05$), in venous blood HCO_3 decreases from 18.45±1.57 to 13.80±1.13 mmol/l ($P<0.05$). This decrease in HCO_3 indicates renal malfunctioning, leading to reduced excretion of bicarbonates and impaired functioning of the liver to compensate acidosis. In arterial blood PO_2 and O_2 sat decrease also from 112.53±13.75 to 55.20±9.8 mmHg ($P<0.01$) and from 97.04±1.4 % to 81.10±2.15%, respectively ($P<0.001$). PO_2 and O_2 sat in venous blood decrease from 56.86±7.68 to 31.10±2.5mmHg ($P<0.01$) and from 61.17±7.06 % to 20.70±3.05 %, respectively ($P<0.001$). Decrease in PO_2 and O_2 sat is the result of total hypoxia and points to anaerobic processes. The reduction of this index indicates profound impairment of oxidative processes. Reuptake of O_2 from alveoli tissue to blood and saturation of hemoglobin indicates significant destructive process of hypoxia, leading to conversion of the aerobic process to anaerobic.

$AaDO_2$ in arterial blood increases from 0 to 39.21±14.44% ($P<0.01$) and $CCDO_2$ from 0 to 50.1±1.3% ($P<0.001$), in venous blood $CCDO_2$ increases from 17.10±1.20 % to 47.80±1.03% ($P<0.001$). It is an important fact that oxygen deficit in the venous blood during HS3O is significantly reduced, $p<0.05$. Massive exfusion, which was 40 ml/kg per body mass, a cardio-circulatory oxygen deficit and lack of arterio-alveolar oxygen were seen. Composition of the blood samples to a great extent depends on the exact site of the blood withdrawal. So, the blood gas structure depends on the depth of the catheter insertion. Initially, at HS30 when arterial blood pressure was 60 mmHg (erectile phase) only venous $CCDO_2$ $P<0.05$ and

Table 2.

Venous blood gases change at hemorrhagic shock in male cats (M±m, n=8)

Index	Control group	HS30	HS60	80th min
pH	7.245±0.048	7.22±0.001	7.156±0.08	7.049±0.031 p<0.01
PCO_2 (mmHg)	44.03±3.16	49.52±4.97	54.69±1.08 p<0.05	64.23±3.18 p<0.001
BE (mmol/l)	-8.18±1.62	-7.12±2.55	-9.76±2.98	-14.8±1.13 p<0.001
BB (mmol/l)	39.78±2.26	40.7±2.57	38.11±2.9	33.61±1.31 p<0.05
HCO_3(mmol/l)	18.45±1.57	19.62±1.64	18.52±2.05	13.8±1.13 p<0.05
PO_2(mmHg)	56.86±7.68	40.08±8.28	38.05±11.01	31.1±2.15 p<0.01
O_2 sat %	61.17±7.06	55.52±13.32	55.1±15.25	20.7±3.05 p<0.001
$CCDO_2$ %	17.1±1.2	21.43±1.15 p<0.05	43.3±3.6 p<0.001	47.8±1.03 p<0.001

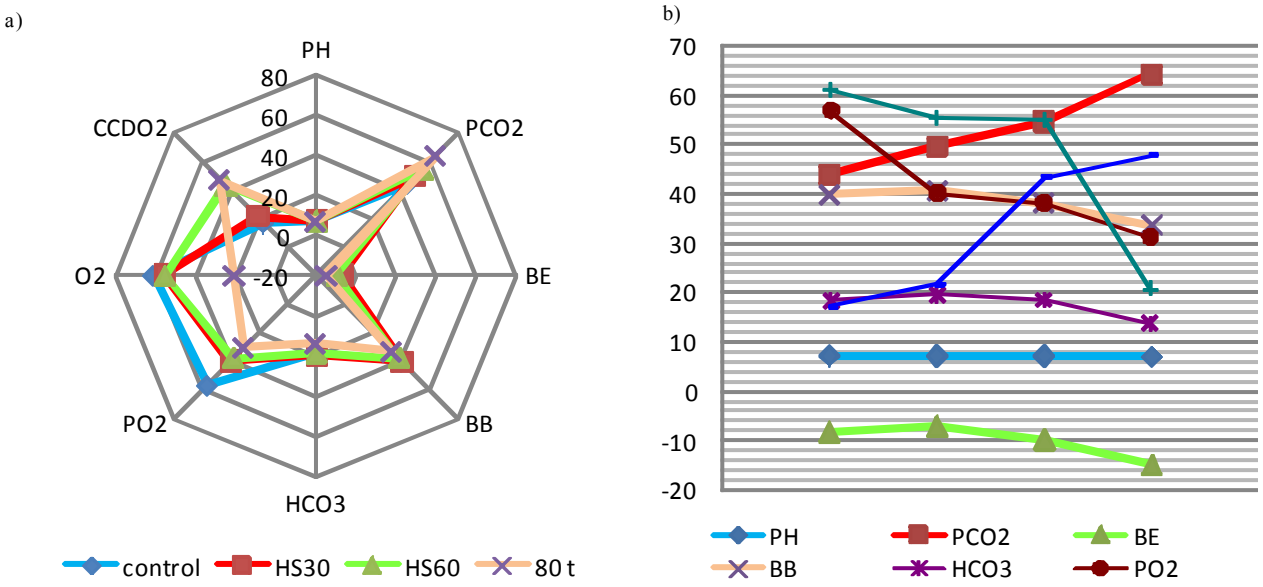

Fig. 2. Venous blood gases structure at hemorrhagic shock in male cats (M±m, n=8) a) Venous blood gases structure priority parameter change in femoral artery; b) The hypothetic model of dynamic change of venous blood gases structure in femoral artery.

arterial PCO_2 (P<0.05) and $CCDO_2$ (P<0.001) were decreased. Reduced PCO_2 in arterial and venous blood caused well expressed $CCDO_2$ via exaggerated processes of erectile phase.

The second phase is presented by inhibitory phase and corresponds to torpid phase (HS60). At this stage the initial arterial blood pressure is 40 mmHg. The pumping function of the heart is significantly inhibited. Systolic ejection and cardiac output decrease due to altered PCO_2 gradient in venous-arterial blood (Tables 1 and 2). The third – preagony leading to disorders of symbiotic connections of all vitally important organs, corresponds to 80th min. In these processes the leading role is played by cerebral cortex, permeability of blood-brain barrier and subcortical structures. Stressful situations as protective at HS are characterized by increased functioning of the adrenal cortex, but later, collaptoid state develops. If we take into consideration the fact that at HS hypovolemia and electrolytes' disbalance develop, the venous blood monitoring will be more informative than arterial in the case of HS and other pathologies (respiratory) [8-10]. Alterations in PH are more expressed in arterial rather than in venous blood causing metabolism deviations, acidosis and homeostatic disorders. Hypercapnia develops, metabolic acidosis worsens, PO_2 in arterial blood dramatically decreases at HS60 P<0.05 and at 80th min P<0.01. The same tendency is observed in the case of O_2 reduction [11-13]. Thus, if we discuss the physiologic properties of each parameter, it could be concluded that drastically decreases the detoxicative function of the liver in maintenance of the excess bases. The activity of the lungs, its compensative-adaptive ability changes via hyperpnoea and alveolar opening when $AaDO_2$ deficiency develops. Renal disorders are reflected in both arterial and venous

blood. Disorders of buffer systems are also well expressed especially in venous blood at the 80th min. Increased arterio-venous O_2 difference, reduced O_2 in venous blood indicates that circulatory hypoxia results in "burning" of the cardio-vascular system, its overload and finally failure and later development of collaptoid situation [14].

Thus, according to the results of our investigations it can be concluded that the most susceptible indicator in femoral artery and venous blood gas structure and buffer parameter is PCO_2, revealed in arterial blood at HS30. The dynamic gravity of this parameter is more expressed in venous blood. PCO_2, $P>0.05$, $P>0.05$ and $P<0.05$ in arterial blood, while in venous blood $P>0.05$, $P<0.05$ and $P<0.001$. These data correlate with hemodynamic parameters. However, increase in PCO_2 may result in reversible reduction of the contractility of the heart. Also $CCDO_2$ has sensitive parameter both in arterial and venous blood [15, 16]. Severity of shock according to the arterial and venous blood gases is well expressed in venous blood, pointing to irreversible changes at 80th min.

ექსპერიმენტული მედიცინა

არტერიული და ვენური სისხლის გაზოვანი სტრუქტურის მონიტორინგი ჰემორაგიული შოკის დროს

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თბილისის სახელმწიფო სამედიცინო უნივერსიტეტი

(წარმოდგენილია აკადემიკოს ნ. ჯავახიშვილის მიერ)

ჰემორაგიული შოკის (HS) დროს ბარძაყის ვენასა და არტერიაში ვითარდება სისხლის გაზოვანი სტრუქტურის უხეში დარღვევა. არტერიულ სისხლში ექსფუზიური აგრესიის შემდეგ: PCO_2 ჰშ30-ზე მეტად რეაგირებს, თუმცა ის HS60-ზე ვენურთან შედარებით მცირედით იცვლება. ის კორელაციაშია ქსოვილების მინიმალურ პერფუზიასთან და ჰემოდინამიკურ მაჩვენებლებთან, ხოლო $CCDO_2$-ის ზრდა არტერიულ სისხლში მკვეთრადაა წარმოდგენილი. მე-80 წუთი განისაზღვრება შეუქცევად პროცესში გადასვლით, რომელზეც ვენური სისხლის გაზოვანი სტრუქტურის ცვლილების მონიტორინგი მიგვანიშნებს. არტერიული და ვენური სისხლის გაზოვანი სტრუქტურის მონიტორინგით გამოვლინდა PCO_2 და $CCDO_2$, რომლებიც ყველაზე მეტად რეაგირებენ მზარდი აციდოზის ფონზე და მიუთითებენ მის დამძიმებაზე.

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