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# INFLUENCE OF SUBARACHNOID ANESTHESIA IN THE PRONE POSITION ON THE ENERGY OF CIRCULATION

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## ABSTRACT

The energy supply of the body has an energy price, which is paid, in particular, by the myocardium. This price depends on the circulatory conditions, which can also be affected by iatrogenic factors, such as subarachnoid anesthesia (SA) in the prone position. Decreasing of a vascular tone under the influence of SA requires the myocardium to increase energy consumption to maintain an adequate cardiac index (CI). With a resistive type of circulation, autoregulation of blood circulation may become untenable, that requires artificial maintenance of vascular tone with the  $\alpha$ 1-adrenomimetics, that also contribute to an increase in energy expenditure by the myocardium. All this makes the assessment of blood flow energy relevant.

The aim of the study was to study the effect of SA in the prone position on the energy of circulation. 65 patients underwent transpedicular stabilization of 1-2 segments under SA in the prone position have been examined. On the eve of the operation, the prognostic index of hemodynamic instability (PIHI) has been determined by the N. Lysohub's method. At three stages (the position on the back after performing SA, 5 and 20 minutes after turning on the prone position), kinetic (systolic index, ejection fraction, heart rate and SI), dynamic (effective, or average, AP — APe, central venous pressure, the difference between them — systemic perfusion pressure and specific peripheral vascular resistance) and hemic (blood oxygen content) indicators have been determined. Based on these indicators and the energy potential of oxygen (the energy obtained by glucose oxidation by 1 mol of oxygen), the following energy parameters have been calculated: flow power index (FPI), tissue power consumption index (TPCI), oxygen reserve (OR) and circulatory reserve index (CRI). APe was maintained at least 60 mm Hg during anesthesia by infusion and, if necessary, by administration of the  $\alpha$ 1-adrenomimetic phenylephrine. Patients have been retrospectively divided into 2 groups: patients of the VF1 group (n = 25) needed phenylephrine administration, and patients of the VF2 group (n = 40) did not need it. The following results have been obtained. The minimum PIHI in the VF1 group was 0.1, which made it possible to expect a high probability of hemodynamic instability, while in the VF2 group it was no higher than 0.05. The integral kinetic parameter CI in the groups did not differ significantly, but the energy consumption and their efficiency between the groups significantly differed. FPI in the VF1 group was significantly lower than in the VF2 group, although TPCI did not differ significantly between the groups. TPCI compliance with tissue needs was determined by OR, which was significantly lower in the VF1 group. The final parameter CRI was significantly lower in the VF1 group, although CI did not differ significantly from the VF2 group level. This indicates energy insufficiency of circulation in patients with a resistive type of circulation against the background of SA in the prone position, so with this type, the presence of which is confirmed by a high PIHI, preference should be given to another type of anesthesia.

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**Introduction.** Today, it does not require additional evidence that for the vital activity and preservation of the integrity of a living organism, it needs a constant flow of energy from the outside at a sufficient speed. It had long been established that at rest, an organism weighing about 70 kg requires about 1800 kcal of energy per day, which is about 90 W [1, 2]. This power must be developed, for example, when lifting a weight of 9 kg to a height of 1 m in 1 second. This example provides a visual representation of the minimum rate of continuous energy consumption by the body [3].

The necessary speed of energy production is provided by aerobic oxidation of substances obtained from food, so in the process of evolution, a perfect system of oxygen transport (SOT) was formed, one of the links of which is the circulatory system (CS). However, the CS, which must bring an adequate supply of energy to the tissues by means of transport of the carrier of potential chemical energy (PCE) — oxygen, itself needs energy for its functioning, and the ratio between the consumed and delivered energy ("energy price of energy supply") must be optimal. In other words, the target parameter of CS regulation is the volume velocity of blood flow (cardiac output — CO), and the means of regulation is the blood flow power (BFP). If there is a violation of the regulation of CS, maintaining an adequate CO and, consequently, satisfying the energy needs of tissues become impossible [3].

Iatrogenic drop in vascular tone during subarachnoid anesthesia (SA) is one of the reasons for the violation of CS regulation. This situation can be aggravated by changing the position of the body. All this leads to violation of conformity of volume of circulating blood (VCB) to volume of vessels that can be supported by the body during SA only with increase in force and rate of heart contractions. This increases power consumption by the myocardium, while his ability to do this can be in varying degrees limited. It is known that SA, performed using the same technique and reaching the same level, does not lead to circulatory disorders in some patients, while in others it causes severe destabilization [4]. The only reasonable explanation for this fact, from our point of view, is that the autoregulation of CS in different individuals is carried out with the predominance of different mechanisms, which, however, provide an adequate supply of energy to tissues under average daily loads. It is only when conditions go beyond the average limits that these differences are revealed. This view is confirmed by published research results, according to which there are three types of circulatory regime: resistive, capacitive and balanced [5]. Apparently, it is in the resistive type of blood circulation that SA leads to the destabilization of hemodynamics. Recently, a method was found for predicting a high probability of developing circulatory instability in SA in the prone position by calculating the predictive index of hemodynamic instability (PIHI). If its level exceeds 0.5, the probability of hemodynamic destabilization during SA in the prone position is very high [6, 7]. Iatrogenic correction in this case should consist in artificial filling of the vascular bed (infusion), if this measure is ineffective — in an artificial increase in vascular tone with  $\alpha_1$ -adrenomimetics. In any case, all regulatory influences, both spontaneous and artificial, will affect the energy of blood circulation and its effectiveness.

**The aim of the study** the effect of subarachnoid anesthesia in the prone position on the energy of blood circulation.

**Materials and methods.** The course of SA in the prone position has been studied in 65 patients operated for degenerative diseases of the spine. On the eve of the operation, in all patients PIHI has been calculated using the method developed By N. Lizogub and co-authors [6, 7]. All patients have been undergone the same surgical intervention (transpedicular stabilization of 1-2 segments) under SA, performed using the same method, which consisted of the following. V. subclavia has been catheterized in the operating room, then volemic preparation has been performed with a balanced crystalloid solution in the volume of  $6.38 \pm 1.14$  ml/kg. SA was performed with 0.5 % hyperbaric bupivacaine solution administered in the L2-L3 interval in a sitting position through a G25 needle for 20 seconds, after which

the patient was laid on his back for 5-7 minutes, and then turned on prone position. The study was performed up to 20 minutes after turning on the prone position. In the case of a decrease in effective (average) blood pressure (APe) to 60 mm Hg, the administration of phenylephrine was started using a syringe pump (10 mg in 50 ml of saline solution) at the speed necessary to maintain APe.

Depending on the presence or absence of the need to use  $\alpha_1$ -adrenomimetics, patients have been retrospectively divided into 2 groups. The group VF1 (n = 25) included patients whose hemodynamic stabilization was possible only with the help of the  $\alpha_1$ -adrenomimetic phenylephrine, and the group VF2 (n = 40) — patients who did not require the use of vasopressors. The characteristics of patients are presented in table 1, which shows that the destabilization of blood circulation occurred more often in young men with excessive body weight. Probably, the type of blood circulation in patients of the VF1 group is unbalanced (more often — resistive), and in patients of the VF2 group — balanced (a combination of resistive and capacitive types).

Table 1. Distribution of patients by groups

Group	Age, years, M $\pm$ $\sigma$	Body mass, kg, M $\pm$ $\sigma$	Height, sm, M $\pm$ $\sigma$	Body surface area, m <sup>2</sup> , M $\pm$ $\sigma$	Body mass index, kg/m <sup>2</sup> , M $\pm$ $\sigma$	Men n (%)	Women n (%)
VF1 (n = 25)	42,0 $\pm$ 5,4	95,0 $\pm$ 9,9	173,3 $\pm$ 7,0	2,19 $\pm$ 0,15	31,6 $\pm$ 1,9	16 (64,0 $\pm$ 9,6)	9 (36,0 $\pm$ 9,6)
VF2 (n = 40)	45,9 $\pm$ 8,7	72,0 $\pm$ 8,7	174,9 $\pm$ 6,2	1,96 $\pm$ 0,14	23,5 $\pm$ 2,5	17 (42,5 $\pm$ 7,8)	23 (57,5 $\pm$ 7,8)
p	0,046	< 0,001	> 0,3	< 0,001	< 0,001		

Since the energy parameters of blood circulation are determined by mechanical (kinetic and dynamic) and hemic parameters [3], they have been determined during SA at three stages: 1) in the back position after performing SA; 2) 5 minutes after turning on the prone position; 3) 20 minutes after turning on the prone position (table 2).

Table 2. Blood circulation indicators

Kinetic	Dynamic	Hemic	Energy
SI, EF, HR, CI	SPP, SPVR	Hb, Ht, $S_{O_2}$ , $p_{O_2}$ , $C_{O_2}$ , $T_{O_2}$ , $V_{O_2}$	IBFP, ITPC, OTP, OCP, OR, ICR

Kinetic parameters (determined by echocardiography): stroke index (SI), ejection fraction (EF), heart rate (HR), and cardiac index (CI). Dynamic parameters: systemic perfusion pressure (SPP), i.e. the difference between APe and central venous pressure, and specific peripheral vascular resistance (CPVR), calculated according to the Poiseuille-Franck law. Blood content ( $C_{O_2}$ ), transport ( $T_{O_2}$ ) and consumption ( $V_{O_2}$ ) of oxygen have been calculated based on the measured concentrations of hemoglobin, hematocrit, hemoglobin saturation with oxygen ( $S_{O_2}$ ) and partial pressure of dissolved oxygen ( $p_{O_2}$ ) in the conventional way, the amount of oxygen was measured not in units of volume, but in units of mass (mol). Hemic parameters of arterial blood have been determined by taking capillary blood with its preliminary arterialization by placing the fingers of the hand in water at a temperature of 39 °C. The energy potential of oxygen (EPO) is determined from the final equation of complete aerobic glucose oxidation and is equal to 480 kJ per 1 mol of oxygen.

Using mechanical and hemic parameters, oxygen and energy transport parameters have been calculated: blood flow power index (IBFP,  $W/m^2$ ), tissue power consumption index (ITPC,  $W/m^2$ ), oxygen transport and consumption pressure (OTP and OCP,  $kJ/mol$ ), oxygen reserve (OR, dimensionless value) and circulatory reserve index (ICR,  $W/m^2$ ). IBFP reflects the useful work of the myocardium, OTP and OCP — the energy of the myocardium, which is accounted for by transport and consumption of 1 mole of oxygen, respectively. OR shows the correspondence of oxygen consumption by tissues to their needs, its calculation is based on the final equation of anaerobic glucose oxidation with the production of lactate. The integrating energy parameter is the ICR, which is equal to the product of IBFP and OR and takes into account all multidirectional undefined fluctuations in the mechanical and hemic parameters of oxygen transport. A decrease in the IBFP corresponds to a certain degree of circulatory failure. The reference values of energy parameters have been previously determined in the study of CS of healthy volunteers.

**Results of the research.** The PIHI of patients in the VF1 group was  $0.53 \pm 0.42$ , that is, the spread of its values was significant, but only in 8 patients it was below 0.1, in the rest it exceeded 0.3. In the VF2 group, the PIHI was less than 0.05, that is, this parameter has a reliable prognostic value for the course of SA in the prone position.

There were no significant changes in such kinetic parameters as SI and EF during the study, while these parameters significantly differed in the groups of patients when compared with each other and when compared with reference values (Fig. 1). CI did not show significant differences when comparing groups of patients, although in the VF2 group in stage 2 the CI decrease was significant, but not to critical values. In both groups of patients, CI was significantly lower than the reference values: according to the study stages in the VF1 group, it was  $2.8 \pm 0.4$ ,  $2.7 \pm 0.4$  and  $2.8 \pm 0.4$   $l/min \cdot m^2$ , in the VF2 group —  $3.0 \pm 0.4$ ,  $2.8 \pm 0.4$  and  $2.9 \pm 0.4$   $l/min \cdot m^2$ . The absence of significant differences between groups of patients in CI in the presence of significant differences in SI and EF reflects a compensatory reaction in the form of tachycardia, which was not significantly inhibited by SA and vasopressors (in the VF1 group).

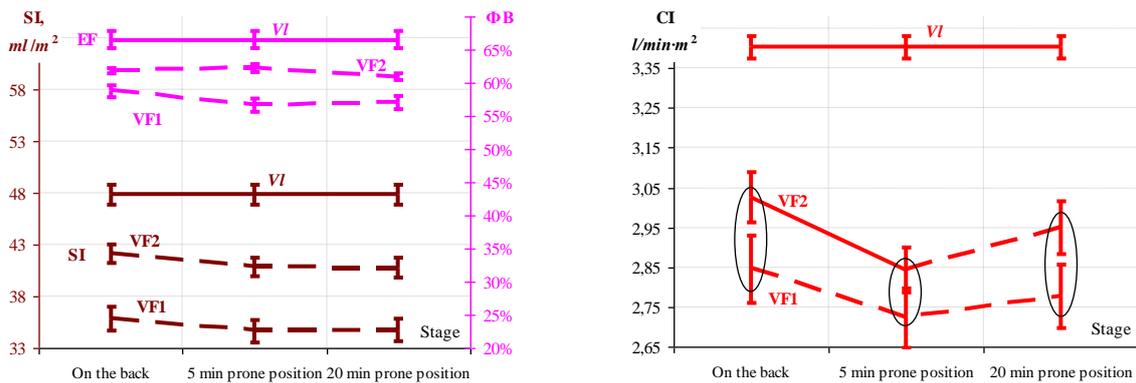


Fig. 1. Kinetic parameters during SA,  $M \pm m$

(here and further: —  $p < 0,05$  compared to the previous stage, - - -  $p > 0,05$  compared to the previous stage, 0—  $p > 0,05$  between groups, VI — group of healthy volunteers).

In contrast to kinetic, dynamic indicators were subjected to significant changes throughout the study, which had a certain effect on the level of energy parameters (Fig. 2). At the beginning of the study, SPP in the VF1 group was significantly higher ( $95.9 \pm 12.5$  mm Hg) than in healthy individuals ( $89.5 \pm 8.2$  mm Hg) and in the VF2 group ( $85.4 \pm 10.3$  mm Hg), between the latter there were no significant differences in SPP. At stage 2, SPP significantly decreased in both groups, becoming significantly lower than in healthy individuals, and in the VF1 group it decreased more despite the administration of vasopressors (to  $71.5 \pm 8.1$  mm Hg versus  $75.5 \pm 7.8$  mm Hg in the VF2 group). This dynamic was stable and continued until the end of the study: in the VF1 group, the SPP decreased to  $65.5 \pm 5.3$ , and in the VF2 group — to  $69.9 \pm 5.4$  mm Hg.

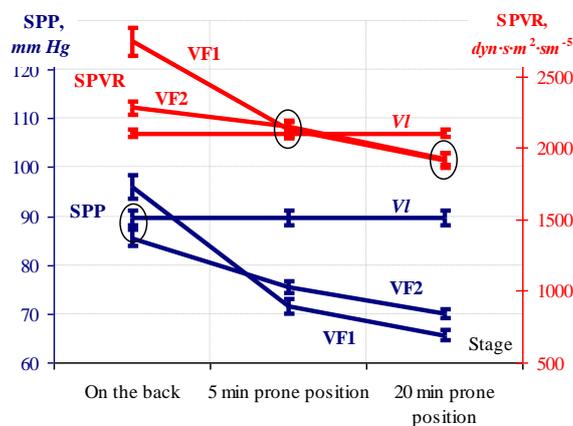


Fig. 2. Dynamic parameters during SA,  $M \pm m$ .

The SPVR at stage 1 was significantly different when compared both between groups of patients and when comparing groups of patients and healthy individuals ( $2743 \pm 490$ ,  $2279 \pm 291$  and  $2100 \pm 127$   $dyn \cdot s \cdot m^2 \cdot cm^{-5}$ , respectively, in groups VF1, VF2 and in healthy volunteers). At stage 2,

SPVR decreased significantly in both groups, and in the VF1 group more significantly despite the administration of vasopressors, and thus this parameter has become the same in both patients and healthy (respectively  $2125 \pm 295$ ,  $2144 \pm 265$  and  $2100 \pm 127 \text{ dyn}\cdot\text{s}\cdot\text{m}^2\cdot\text{cm}^{-5}$ ). Further the dynamics of SPVR in both groups of patients were the same (in the group VF1 —  $1914 \pm 265$ , in the group VF2 —  $1923 \pm 253 \text{ dyn}\cdot\text{s}\cdot\text{m}^2\cdot\text{cm}^{-5}$ ), but patients in the group VF1 received phenylephrine.

The described dynamics of mechanical parameters of blood circulation can be explained in such way. Patients of the VF1 group are characterized by an unbalanced circulatory regime with a predominance of resistive type of regulation, whereas patients of the VF2 group had a balanced type of blood circulation. In patients of the VF1 group, there seems to be some tension in blood circulation compensation, manifested by a tendency to arterial hypertension, a decrease in SI and CI, and an increase in SPVR. Primary may be a slight decrease in the contractile capacity of the myocardium (CCM) with a compensatory increase in the SPVR, or an increase in the SPVR for various reasons (hypertension, psychoemotional stress, etc.) with insufficient compensation from the CCM. A greater risk of this state, according to the results of our study, occurs in younger people with an increased body mass index. In any case, we can say that these patients have formed a certain blood circulation regime that meets the needs of tissues for oxygen in everyday life with average physical activity. Under the influence of SA, there is a decrease in SPVR, the increased level of which is compensatory without anesthesia. With insufficient myocardial reserves, arterial hypotension and a decrease in CI develop, which forces to compensate for the decrease in SPVR with the help of  $\alpha_1$ -adrenomimetics [7].

Support of blood circulation in patients of the VF1 group using vasopressors prevented a significant decrease in the integral kinetic index — CI (there were no significant differences in the CI level between the groups of patients), but other mechanical parameters of blood circulation in the VF1 and VF2 groups differed significantly (except for SPVR at the 2<sup>nd</sup> stage of the study), which could not but affect the energy efficiency of oxygen and energy transport.

IBFP at the 1<sup>st</sup> stage of the study in both groups did not differ and was significantly lower than in healthy individuals: in the group VF1, it was at the level of  $606 \pm 126 \text{ mW}/\text{m}^2$ , VF2 —  $578 \pm 124 \text{ mW}/\text{m}^2$ , in healthy individuals —  $678 \pm 90 \text{ mW}/\text{m}^2$  (Fig. 3). This did cause a lower CI against the background of increased SPVR, and the increase in SPP in patients of the VF1 group was not sufficient to maintain CI. In other words, the myocardium of patients in this group did not have sufficient reserves to overcome the increased SPVR. As the vasoplegic effect of SA developed, the TPVR decreased to the reference level, but the strength of myocardial contractions remained at the same level (SI and EF, as well as heart rate, did not significantly change), which has led to a significant decrease in SPP. The described changes were reflected in a significant decrease in IBFP at the 2<sup>nd</sup> stage of the study, especially in the group VF1 — up to  $434 \pm 91 \text{ mW}/\text{m}^2$ , (VF2 — up to  $479 \pm 92 \text{ mW}/\text{m}^2$ ). It is noteworthy that the dynamics of IBFP is due to changes in dynamic parameters, the kinetic parameters changed slightly, this illustrates the insufficiency of assessing the state of the CS only using CI. At stage 3, the IBFP in the VF1 group was already significantly lower ( $404 \pm 75 \text{ mW}/\text{m}^2$ ) than in the VF2 group ( $459 \pm 86 \text{ mW}/\text{m}^2$ ).

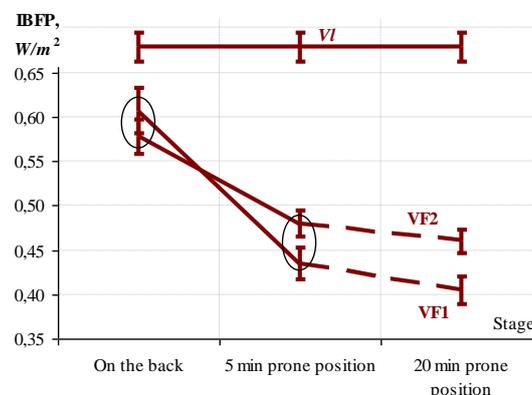


Fig. 3. Blood flow power during SA,  $M \pm m$ .

A low IBFP may mean that most of the myocardial energy is spent directly on oxygen transport and less on other transport needs, i.e. the oxygen (energy) cost of tissue energy supply increases. This price is reflected by indicators such as OTP and OCP, but for their determination, hemic indicators of oxygen transport are necessary (Fig. 4).

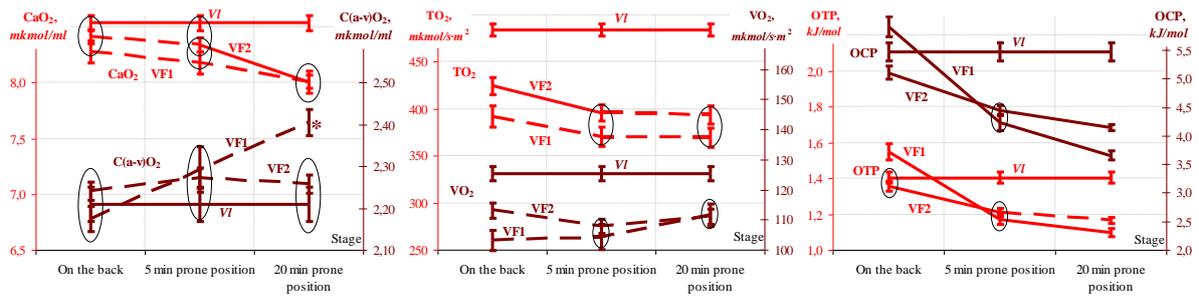


Fig. 4. Hemic parameters during SA,  $M \pm m$ .

At the beginning of the study, the blood oxygen content was the same in both groups, not differing from the level of healthy volunteers ( $C_{aO_2} — 8.53 \pm 0.54 \text{ mmol/ml}$ ,  $C_{(a-v)O_2} — 2.21 \pm 0.23 \text{ mmol/ml}$ ). At stage 2, there was a tendency to decrease  $C_{aO_2}$  (to  $8.17 \pm 0.49$  in the VF1 group and to  $8.24 \pm 0.40 \text{ mmol/ml}$  in the VF2 group) and increase  $C_{(a-v)O_2}$  (to  $2.29 \pm 0.27$  in the VF1 group and to  $2.27 \pm 0.16 \text{ mmol/ml}$  in the VF2 group). At stage 3,  $C_{aO_2}$  in both groups has become almost the same ( $2.40 \pm 0.16$  and  $2.26 \pm 0.14 \text{ mmol/ml}$ , respectively), what is significantly lower than the level of healthy individuals, but the dynamics of  $C_{(a-v)O_2}$  in the patient groups was different. In the VF2 group, this indicator has not been changed, and in the VF1 group it continued to increase (to  $2.40 \pm 0.16 \text{ mmol/ml}$ ), becoming significantly higher than the initial level. Against this background, tissue oxygen consumption has not been changed significantly throughout the study, although it was lower than in healthy individuals, as was oxygen transport. Thus, we can conclude that when the oxygen transport fluctuates within certain limits, the tissues extract all the oxygen they need from the blood, regardless of the level of its delivery. This is illustrated by the dynamics of OTP and OCP.

OTP, equal to the ratio of SPP to  $C_{aO_2}$ , shows the work of the myocardium, accounting for the movement of 1 mole of oxygen, OCP is equal to the ratio of SPP to  $C_{(a-v)O_2}$  and shows the work of moving the consumed part of the oxygen. If at the beginning of the study these parameters differed from the level of healthy (OTP —  $1.40 \pm 0.17 \text{ kJ/mol}$ , OCP —  $5.46 \pm 0.84 \text{ kJ/mol}$ ) insignificantly, then they significantly decrease (OTP to  $1.09 \pm 0.11 \text{ kJ/mol}$  in the VF1 group and to  $1.17 \pm 0.11 \text{ kJ/mol}$  in the VF2 group; OCP — to  $3.65 \pm 0.40$  and  $4.13 \pm 0.35 \text{ kJ/mol}$ , respectively). A decrease in these parameters means that more and more of the myocardial energy is spent directly on the movement of oxygen, that is, the energy price of tissue energy supply is increasing.

Against this background, the ITPC that repeats the  $V_{O_2}$  dynamics, changed little for the same reason that  $V_{O_2}$  did not change much, but the ITPC compliance with the energy needs of tissues in the VF1 group was violated, as evidenced by changes in the OR (Fig. 5).

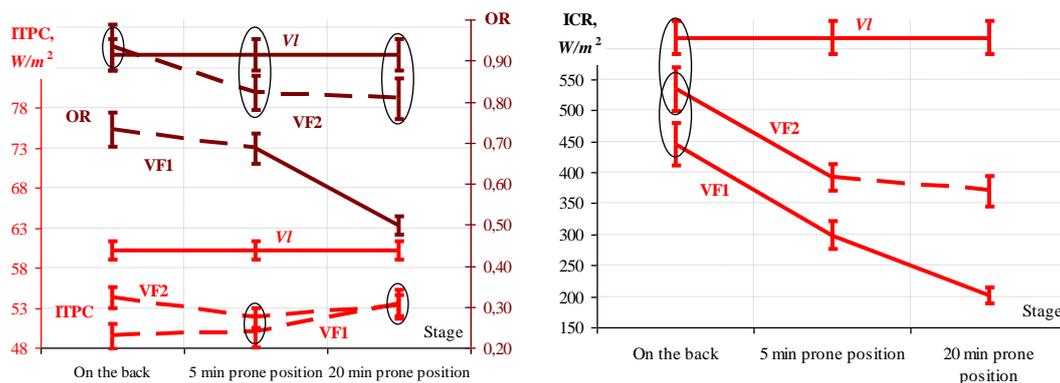


Fig. 5. Energy parameters during SA,  $M \pm m$ .

In the VF2 group, OR did not significantly differ from the level of healthy volunteers during the entire study, although the kinetic and dynamic parameters of patients in this group differed from the reference values. This can be regarded as the transition of an organism with a balanced type of blood circulation to a more economical mode of life, although the reasons and mechanisms for this transition are not clear. It can be assumed that the use of adrenomimetics in patients of the VF1 group increases the need for tissue energy, which can not be met at the same level of ITPC.

Changes in all parameters of blood circulation are summed up in the ICR. Already at the first stage of the study, in patients with an unbalanced type of blood circulation (group VF1), this parameter was significantly lower ( $445 \pm 175 \text{ mW/m}^2$ ) than in healthy individuals ( $617 \pm 145 \text{ mW/m}^2$ ). This means that the useful work of the myocardium is not sufficient to fully meet the energy needs of tissues, which is confirmed by a reduced OR. In the future, the ICR decreases in both groups, but to a greater extent — in the VF1 group. In this group, at the end of the study, the ICR decreased to  $202 \pm 65 \text{ mW/m}^2$ , and in the VF2 group — to  $370 \pm 158 \text{ mW/m}^2$ .

The total dose of phenylephrine required to maintain APe at least the target level (60 mm Hg) in patients of the VF1 group was  $5.86 \pm 2.85 \text{ mcg/kg}$ .

Naturally, the question arose about the predictive value of parameters of the initial state of the kinetics, dynamics and energy of blood circulation. To solve this issue, we studied the relationship of the initial ICR with PIHI and the dose of phenylephrine required to maintain blood circulation. The results were such as the next. The correlation coefficient of the initial ICR with the dose of phenylephrine was  $-0.80 \pm 0.07$ , while the association of PIHI with the initial ICR was absent ( $-0.23 \pm 0.19$ ). From this, as well as from other results obtained, it follows that PIHI, the calculation of which is based on kinetic and dynamic indicators of blood circulation, with great accuracy allows to predict the instability of hemodynamics when performing SA in the abdominal position. The severity of hemodynamic instability can be predicted with high probability when taking into account the initial energy indicators of blood flow.

Thus, for evaluating the state of CS traditional mechanical indices (SI, CI, SPP) is not enough, it is necessary to consider the energy circulation and energy supply of tissues. The energy approach to the study of CS during SA in the prone position allows to identify differences in the efficiency of energy supply to tissues depending on the initial blood circulation regime, which determines the response of CS to SA.

**Conclusions.** 1. One of the main compensatory reactions of the circulatory system when the supply of oxygen to tissues deteriorates is an increase in vascular tone. The increased severity of this reaction is characteristic of an unbalanced type of blood circulation with a predominance of the resistive component. The blockade of this reaction against the background of SA violates the compensation of blood circulation, especially in its resistive type.

2. In order to fully assess the effectiveness of the CS, it is necessary to take into account its energy characteristics.

3. Energy analysis of blood circulation during SA in the prone position has been shown that with the initial unbalanced blood circulation mode with a predominance of the resistive component, artificial increase in vascular tone with  $\alpha_1$ -adrenomimetics does not allow to maintain the correspondence of the energy supply of tissues to their needs, so to choose the optimal anesthetic aid, it is necessary to determine the type of blood circulation before surgery by calculating the predictive index of hemodynamic instability (PIHI) by Lyzohub.

**Conflict of interest.** The authors do not declare a conflict of interest.

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