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INFLUENCE OF ACUTE BLOOD LOSS ON THE IONIC BALANCE OF THE BRAIN IN THE PERIOPERATIVE PERIOD

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ВПЛИВ ГОСТРОЇ КРОВОТРАТИ НА ІОННИЙ БАЛАНС ГОЛОВНОГО МОЗКУ В ПЕРІОПЕРАЦІЙНОМУ ПЕРІОДІ

Тkachuk A.Ю.

В клінічних умовах (63 пацієнта з СЖК) вивчали вплив гострої крововтрати на іонний гомеостаз мозку. Встановлено можливі причини церебральної недостатності та порушення свідомості у пацієнтів даної категорії.

Ключові слова: гостра крововтрата, церебральна недостатність, метаболізм мозку, артеріо-яремна різниця.

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In a clinical setting (63 patients with FFA), the effect of acute blood loss on ionic homeostasis of the brain was studied. Possible causes of cerebral insufficiency and impaired consciousness in patients of this category have been established.

Key words: acute blood loss, cerebral insufficiency, brain metabolism, arterio-jugular difference.

Introduction. Today, acute blood loss (ABL) is one of the main causes that cause serious disorders of the internal environment of the body and lead to the death of people of all ages [1, 2]. Under the action of circulatory-hemic hypoxia, which develops in ABL, metabolic processes in all organs and systems of the body are disrupted [3]. Due to compensatory-adaptive reactions, the function of the heart and brain in ABL is disrupted last [4], and cerebral insufficiency develops after a violation of the mechanisms of autoregulation of cerebral circulation [5-6]. In recent years, the number of experimental studies that indicate the role of ionic imbalance in brain cell damage has increased. The fact of ionized hypocalcemia and hypomagnesemia in critically ill patients has been established, but the connection between these ionic disorders and acute blood loss has not been fully disclosed [7-8]. Intensive care for acute blood loss today remains among the relevant areas in emergency medicine [9-10].

All the above determined the need to study the ionic balance of the brain in patients with cerebral insufficiency and impaired consciousness in acute blood loss.

Material and methods. The study included 63 patients with acute gastrointestinal bleeding (AGIB). Selection was performed at the stage of admission to the ICU, the criteria for inclusion in the study were the level of hemoglobin below 90 g / l and the level of systolic blood pressure below 90 mm Hg. The only criterion for dividing patients into groups was the level of consciousness. The main group (32 patients) was defined as patients who had a disturbance of consciousness on admission. If the patient was conscious on admission to the ICU, he was assigned to the comparison group (31 patients). Patients with a history of diabetes mellitus, liver pathology, and renal failure were excluded from the study. The Glasgow Com Scale was used to determine the level of consciousness. The volume of blood loss was calculated by the Moore formula [12], the degree of stress of the body's compensatory mechanisms in ABL was estimated using the compensation coefficient (CC) [13].

The source of acute blood loss was a complicated duodenal and / or gastric ulcer. All subjects underwent surgery for vital signs in the first days of their stay at the institute, the operations were performed under general anesthesia.

Clinical and laboratory parameters were determined on admission and on the third day after surgery. The method of Ludwig and London was used to study metabolic processes in the brain. The essence of the method is to assess the metabolism of the brain by changing the composition of the blood flowing to it and flowing from it. Blood from the carotid artery and internal jugular vein was examined. Statistical processing of the obtained data was performed using a computer program for Excel Windows XP and BIOSTAT.

Research results and their discussion. Patients in the comparison group were admitted conscious, the number of GCS scores was 14.86 ± 0.35 . The degree of depression of consciousness in patients of the main group ranged from moderate stunning to stupor, the average number of GCS points in this group was 11.13 ± 0.83 points, $p \leq 0.05$. Most of the examined (53.33%) were admitted in a state of deep stunning, 16.66% of patients were admitted in stupor. Moderate stunning was noted in 30% of patients. The volume of blood loss in patients with impaired consciousness was significantly higher in relation to the comparison group ($38.17 \pm 9.19\%$ and $33.21 \pm 6.16\%$ BCC, respectively, $p \leq 0.05$, Table 1).

The initial values of systolic blood pressure and hemoglobin in the compared groups did not differ significantly. In the main group, AP syst. was determined at the level of

Table 1

Laboratory and calculated parameters of patients with acute blood loss

Indicators	Admission		3 days	
	Main group	Comparison group	Main group	Comparison group
% blood loss	33,21±6,16	38,17±9,19*	-	-
HB, g / l	73,33±12,09	69,7±9,68	79,65±7,33	85,04±8,4
Erythrocytes, · 10 ¹²	2,52±0,8	2,21±0,73	2,7±0,52	3,01±0,64
pH art	7,35±0,05	7,33±0,06	7,37±0,05	7,39±0,05
CC, units	3,18±1,45	2,43±1,49*	5,76±1,59	5,66±1,36**
GCS, points	14,87±0,34	11,47±1,37*	14,53±1,01	14,26±1,25**

* - reliably relative to the comparison group at $p \leq 0.05$; ** - reliably relative to admission at $p \leq 0.05$

73.58 ± 19.7 mm Hg, $p \geq 0.05$. Upon admission, the hemoglobin level in the main group was 69.7 ± 9.68 g / l, in the comparison group 73.33 ± 12.09 g / l, $p \geq 0.05$. Clinical and laboratory (hemoglobin level) indicators at admission indicated a high degree of stress in compensatory-adaptive reactions. This was confirmed by the low value of the compensation coefficient (CC).

When studying the ionic balance of the brain in patients with acute blood loss, severe hypocalcemia was observed (Table 2).

There was a positive arterio-jugular difference (AJD) in total calcium (0.04 ± 0.02 mmol / l), which was almost 2.5 times higher than that in the comparison group (0.016 ± 0.02 mmol / l, $p \leq 0.05$).

On the third day, in relation to the data on admission, there was an increase in total calcium, in the carotid artery 1.86 ± 0.25 mmol / l, in the jugular vein 1.87 ± 0.26 mmol / l. In the dynamics, AJR changed its direction, became negative and significantly differed from AJR in total calcium upon admission (-0.012 ± 0.07 mmol / l, $p \leq 0.05$).

The level of ionized calcium in the carotid artery and jugular vein on admission contributed to a positive AJD (0.03 ± 0.02 mmol / l, $p \leq 0.05$). In dynamics on the third day, ionized calcium increased, positive AJD upon admission changed its direction and significantly differed from AJD on the third day (-0.01 ± 0.09 mmol / l), $p \leq 0.05$.

The magnesium level in patients with OK complicated by impaired consciousness was low. In the jugular vein, Mg significantly differed from the comparison group (0.82

Table 2

Ionic balance of the brain in patients with acute blood loss

Indicators	Admission		3 days	
	Main group	Comparison group	Main group	Comparison group
Ca total, art. mmol / l	1,82±0,26	1,84±0,27	1,89±0,36	1,86±0,25
Ca total, jug. mmol / l	1,81±0,32	1,8±0,25	1,89±0,32	1,87±0,26**
AJD for Ca total	0,016±0,02	0,04±0,02*	-0,011±0,09	-0,012±0,07**
Ca ion. art. mmol / l	0,88±0,29	0,89±0,3	0,97±0,19	0,93±0,24
Ca ion. jugul mmol / l	0,87±0,24	0,86±0,26	0,97±0,18	0,93±0,19**
AJD for Ca ion.	0,015±0,03	0,03±0,02*	-0,01±0,085	-0,01±0,09**
Mg art mmol / l	0,93±0,25	0,84±0,2	0,97±0,26	0,96±0,15**
Mg yugul mmol / l	0,95±0,27	0,82±0,21*	0,99±0,34	0,97±0,18**
AJD for Mg	-0,03±0,1	0,025±0,1*	-0,02±0,11	-0,03±0,09**
Phosphorus art mmol / l	1,13±0,3	1,09±0,23	1,14±0,23	1,11±0,29
Phosphorus jug. mmol / l	1,12±0,29	1,08±0,21	1,14±0,31	1,1±0,32
AJD for Ph	0,05±0,17	0,026±0,15	0,02±0,21	0,02±0,19
Potassium art., mmol / l	3,89±0,4	4,16±0,32	4,1±0,35	4,12±0,47
Potassium jug., mmol / l	3,85±0,44	4,11±0,42	4,2±0,49	4,08±0,4
AJD for potassium	0,07±0,1	0,06±0,13	0,09±0,18	0,06±0,12
Sodium art, mmol / l	143,4±4,37	142,1±3,13	149,2±4,47	150,67±5,4**
Sodium jugul, mmol / l	142,85±4,7	143,65±4,1	148,39±3,46	149,8±4,8**
AJD for sodium	0,66±1,35	-0,19±1,96	0,13±1,58	0,27±1,68

± 0.21 mmol / l, $p \leq 0.05$). Acute blood loss in patients with impaired consciousness was accompanied by a positive AJD for magnesium (0.025 ± 0.1 mmol / l, $p \leq 0.05$). This made it possible to make an assumption about the possible “compensatory absorption” of magnesium ions by the cells of the brain. Based on the results obtained, the treatment regimen for patients with ABL in the postoperative period included magnesium sulfate in the form of drip infusion in physiological saline at a daily dosage of 0.15-0.2 g / kg for at least three days after the operation.

The Mg content on the third day in the carotid artery (0.96 ± 0.15 mmol / l) and jugular vein (0.97 ± 0.18 mmol / l) significantly increased over time. AJD in dynamics changed its direction and became, as in the comparison group, negative (-0.03 ± 0.09 mmol / l, $p \leq 0.05$ in relation to AJD on admission).

The level of inorganic phosphorus did not have statistically significant differences between the groups upon admission, as well as in dynamics. Inorganic phosphorus values were within normal limits. The level of potassium and sodium in the blood flowing to and from the brain in patients with acute blood loss complicated by impaired consciousness did not go beyond normal values.

Conclusion. Cerebral insufficiency and impaired consciousness in patients with acute blood loss are accompanied by “compensatory absorption” of magnesium ions by the cells of the brain, as well as the transition of calcium ions from the extracellular sector to the intracellular one, with possible participation in the disturbance of the central nervous system.

Further research is planned to study the ionic imbalance of the brain in acute blood loss.

ЛІТЕРАТУРА

1. Williams M., Lee J.K. Intraoperative blood pressure and perfusion of the brain: strategies for clarifying hemodynamic goals // Paediatr. Anaesth. — 2014. — Vol. 24. — P. 657-67.
2. Bickler P.E., Feiner J.R., Rollins M.D. Factors affecting the performance of 5 brain oximeters during hypoxia in healthy volunteers // Anesth. Analg. — 2013. — Vol. 117. — P. 813-23.
3. Weiss M., Vutskits L., Hansen T.G., Engelhardt T. Safe anesthesia for each purpose: the SAFE-TOTS initiative // Curr. Opin. Anaesthesiol. — 2015. — Vol. 28. — P. 302-7.
4. Erdoğan S., Oto A., Boşnak M. Reliability of cerebral oximeter in non-invasive diagnosis and follow-up of hypercapnia // Turk. J. Pediatr. — 2016. — Vol. 58, № 4. — P. 389-394.
5. Ancora G., Maranella E., Locatelli C., Pierantoni L., Faldella G. Changes in cerebral hemodynamics and amplitude integrated EEG in an asphyxiated newborn during and after cool cap treatment // Brain Dev. — 2009. — Vol. 31. — P. 442-4.
6. Zulueta J.L., Vida V.L., Perisinotto E., Pittarello D., Stellan G. Role of intraoperative regional oxygen saturation using near infrared spectroscopy in the prediction of low output syndrome after pediatric heart surgery // J. Card. Surg. — 2013. — Vol. 28, № 4. — P. 446-52.
7. Michelet D., Arslan O., Hilly J., Mangalsuren N., Brasher C., Grace R., Bonnard A., Malbezis S., Nivoche Y., Dahmani S. Intraoperative changes in blood pressure associated with cerebral desaturation in infants // Paediatr. Anaesth. — 2015. — Vol. 5, № 7. — P. 681-8.
8. De Graaff J.C., Pasma W., van Buuren S., Duijghuisen J.J., Nafiu O.O., Kheterpal S., van Klei W.A. Initial values for noninvasive blood pressure in children during anesthesia: a multicentre retrospective observational cohort study // Nesthesiology. — 2016. — Vol. 12. — P. 904-13.
9. Verhagen E.A., van Braeckel K.N., van der Veere C.N., Groen H., Dijk P.H., Hulzebos C.V., Bos A.F. Cerebral oxygenation is associated with a neurological outcome of premature infants aged 2 to 3 years // Dev. Med. Child Neurol. — 2015. — Vol. 57. — P. 449-55.

10. Alderliesten T, Lemmers P.M., van Haastert I.C., de Vries L.S., Bonestroo H.J., Baerts W., van Bel F. Hypotension in preterm neonates: only low blood pressure does not affect the result of the development of the nervous system // *J. Pediatr.* — 2014. — Vol. 164. — P. 986.
11. Padmanabhan P, Oragwu C., Das B., Myers J.A., Raj A. Utility of Non-Invasive Monitoring of Cardiac Output and Cerebral Oximetry during Pain Management of Children with Sickle Cell Disease in the Pediatric Emergency Department // *Children (Basel)*. — 2018. — Vol. 29. — P. 5. 4
12. Lai N., Saidel G.M., Iorio M., Cabrera M.E. Non-invasive estimation of metabolic flux and blood flow in working muscle: effect of blood-tissue distribution // *Adv. Exp. Med. Biol.* — 2009. — Vol. 645. — P. 155-60.
13. Van den Brand J.G., Verleisdonk E.J., van der Werken C. Near infrared spectroscopy in the diagnosis of chronic exertional compartment syndrome // *Am. J. Sports Med.* — 2004. — Vol. 32. — P. 452-6.

REFERENCES

1. Williams, M., Lee, J.K. Intraoperative blood pressure and perfusion of the brain: strategies for clarifying hemodynamic goals. *Paediatr. Anaesth.*, 2014, vol. 24, pp. 657-67.
2. Bickler, P.E., Feiner, J.R., Rollins, M.D. Factors affecting the performance of 5 brain oximeters during hypoxia in healthy volunteers. *Anesth. Analg.*, 2013, vol. 117, pp. 813-23.
3. Weiss, M. et al. Safe anesthesia for each purpose: the SAFETOTS initiative. *Curr. Opin. Anaesthesiol.*, 2015, vol. 28, pp. 302-7.
4. Erdoğan, S., Oto, A., Boşnak, M. Reliability of cerebral oximeter in non-invasive diagnosis and follow-up of hypercapnia. *Turk. J. Pediatr.*, 2016, vol. 58, № 4, pp. 389-394.
5. Ancora, G. et al. Changes in cerebral hemodynamics and amplitude integrated EEG in an asphyxiated newborn during and after cool cap treatment. *Brain Dev.*, 2009, vol. 31, pp. 442-4.
6. Zulueta, J.L. et al. Role of intraoperative regional oxygen saturation using near infrared spectroscopy in the prediction of low output syndrome after pediatric heart surgery. *J. Card. Surg.*, 2013, vol. 28, № 4, pp. 446-52.
7. Michelet, D. et al. Intraoperative changes in blood pressure associated with cerebral desaturation in infants. *Paediatr. Anaesth.*, 2015, vol. 5, № 7, pp. 681-8.
8. De Graaff, J.C. et al. Initial values for noninvasive blood pressure in children during anesthesia: a multicentre retrospective observational cohort study. *Nesthesiology*, 2016, vol. 12, pp. 904-13.
9. Verhagen, E.A. et al. Cerebral oxygenation is associated with a neurological outcome of premature infants aged 2 to 3 years. *Dev. Med. Child Neurol.*, 2015, vol. 57, pp. 449-55.
10. Alderliesten, T. et al. Hypotension in preterm neonates: only low blood pressure does not affect the result of the development of the nervous system. *J. Pediatr.*, 2014, vol. 164, pp. 986.
11. Padmanabhan, P. et al. Utility of Non-Invasive Monitoring of Cardiac Output and Cerebral Oximetry during Pain Management of Children with Sickle Cell Disease in the Pediatric Emergency Department. *Children (Basel)*, 2018, vol. 29, pp. 5. 4
12. Lai, N. et al. Non-invasive estimation of metabolic flux and blood flow in working muscle: effect of blood-tissue distribution. *Adv. Exp. Med. Biol.*, 2009, vol. 645, pp. 155-60.
13. Van den Brand, J.G., Verleisdonk, E.J., van der Werken, C. Near infrared spectroscopy in the diagnosis of chronic exertional compartment syndrome. *Am. J. Sports Med.*, 2004, vol. 32, pp. 452-6.

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