КОНЦЕНТРАЦИЯ α-ТУМОРНЕКРОТИЧЕСКОГО ФАКТОРА В СЫВОРОТКЕ КРОВИ БЕРЕМЕННЫХ, ИНФИЦИРОВАННЫХ ВИРУСОМ ЛИМФОЦИТАРНОГО ХОРИОМЕНИНГИТА

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Резюме. Статья посвящена анализу концентрации α-туморнекротического фактора в сыворотке крови беременных, инфицированных вирусом лимфоцитарного хориоменингита. Проведено определение концентрации α-туморнекротического фактора в сыворотке крови 62 беременных, инфицированных вирусом лимфоцитарного хориоменингита. Установлено, что существует взаимосвязь между концентрацией α-туморнекротического фактора в сыворотке крови и осложненным течением беременности у женщин, инфицированных вирусом лимфоцитарного хориоменингита. В группе женщин с осложненным течением беременности достоверно ($p<0,05$) более высокой была концентрация α-туморнекротического фактора в сыворотке крови, чем у женщин с неосложненным течением беременности.

Ключевые слова: α-туморнекротический фактор, беременность, вирус лимфоцитарного хориоменингита.

THE CONCENTRATION OF TUMOR NECROSIS FACTOR-A IN GRAVIDAS BLOOD SERUM INFECTED WITH LYMPHOCYTIC CHORIOMENINGITIS VIRUS

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Abstract. The paper deals with an analysis of the concentration of tumor necrosis factor-α in gravidas blood serum, infected with lymphocytic choriomeningitis virus. An assessment of the concentration of tumor necrosis factor-α in the blood serum of 62 gravidas infected with the lymphocytic choriomeningitis virus has been performed. It has been established that there exists a correlation between the tumor necrosis factor-α concentration in the blood serum and a complicated course of pregnancy in women infected with the virus of lymphocytic choriomeningitis. The concentration of tumor necrosis factor-α in the blood serum was considerably higher ($p<0,05$) in the group of women with a complicated course of pregnancy than in the women with an uncomplicated course of pregnancy.

Key words: tumor necrosis factor-α, pregnancy, lymphocytic choriomeningitis virus.

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EFFECTS OF VOLUME THERAPY ON SPLANCHNIC PERFUSION AND MYOCARDIAL OXYGENATION AFTER CARDIAC SURGERY

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Abstract. The aim of the present study was to analyse the effects of postoperative fluid volume on splanchnic perfusion and myocardial oxygenation after cardiac surgery. 20 patients were studied in two stratified by the median of the postoperative volume replacement groups. In the low-volume group the stroke volume index and cardiac index increased continuously throughout the study. In the high-volume group an increase was only observed during the first 5 hours and myocardial oxygenation was significantly impaired and ventilation prolonged. This suggests that fluid loading without a beneficial effect on hemodynamic may not only be ineffective in improving splanchnic perfusion but may have detrimental effects on myocardial oxygenation and pulmonary function. Fluid therapy should be titrated according to individual physiological responses.

Key words: hypovolaemia, volume replacement therapy, splanchnic perfusion, myocardial oxygenation, gastric tonometry.

Introduction. Extracorporal circulation causes serious disturbances in human hormonal, metabolic and fluid homeostasis. Surgical procedures with cardiopulmonary bypass (CPB) lead to a form of circulatory, hypovolaemic shock which is followed by angiotensin II-induced splanchnic vasoconstriction [1]. Thus, during the initial postoperative period patients after CPB may have in splanchnic hypoperfusion to protect the essential organs such as the brain, heart and kidneys [2]. It is known that severe and prolonged splanchnic hypoperfusion is associated with higher morbidity, e.g. multiple organ failure or even mortality [3, 4]. Recently it has been shown that perioperative plasma volume expansion reduces the

the incidence of splanchnic hypoperfusion and improves the outcome [5].

In 1996 the Third European Consensus Conference on tissue oxygenation pointed out the importance of volume replacement in hypovolaemic patients to improve their oxygen delivery [6]. In case of persisting tissue oxygenation deficiency after adequate volume replacement, inotropic agents should be added [7]. But it is still under discussion how to define adequacy of fluid replacement therapy. As splanchnic perfusion is early reduced in hypovolaemia according to physiologic redistribution [8] it would be worthwhile to monitor splanchnic oxygenation or perfusion. Gastric-mucosal tonometry is the only clinically available method for an accurate diagnosis of compromised splanchnic blood [9].

Since volume replacement therapy may also have negative effects on the lungs or the myocardium, a restrictive volume therapy in cardiac surgery is widely adopted [6]. This "traditional" approach has never been evaluated thoroughly in terms of perfusion or oxygenation of the splanchnic area or other organ systems. Splanchnic perfusion can be assessed by means of gastric tonometry [9]. During open heart surgery it is possible to insert microprobes directly into the myocardium to measure tissue partial oxygen pressure. In this way the effects of therapeutic strategies on different organ systems on the regional level can be measured.

The object. Our object was to study retrospectively the impact of volume replacement therapy on splanchnic perfusion and myocardial oxygenation in cardiac surgery patients.

Material and methods. Upon the approval by the local ethics committee and a written informed consent 20 patients, undergoing elective coronary artery bypass surgery, were studied. The patients were divided in two groups by the median (1850 ml) of the total amount of volume replacement during the first 10 hours after operation: the group of low volume (LV) and the group of high volume (HV). Premedication, induction and maintenance of anaesthesia were standardized in both groups. Mechanical ventilation in a volume controlled mode was adapted to achieve arterial normocapnia. All patients underwent standard open heart surgery with non-pulsatile CPB in moderate hypothermia. During CPB the mean peripheral arterial pressure (MPAP) was maintained at 60-80 mmHg. Global hemodynamic parameters and regional oxygenation were taken at intervals of 15 minutes (blood sampling every 30 minutes) intra- and postoperatively. The study ended up 10 hours after the admittance to the intensive care unit. All the patients were mechanically ventilated as long as clinically appropriate and were sedated with continuous infusion. Fluid management was adjusted to achieve and maintain a central venous pressure (CVP) between 8 to 12 mmHg. Volume replacement was performed with Ringer's solution and gelatine polysuccinate, as appropriate.

Additionally to standard monitoring all patients were equipped with a pulmonary artery catheter, a nasogastric tonometry catheter and special polarographic microprobes for a myocardial partial oxygen pressure examination. A pulmonary artery catheter was used to determine mean pulmonary artery pressures (MPAP), automated semicontinuous measurement of cardiac output (CO) / cardiac index (CI), and a continuous measurement of mixed venous oxygen saturation (SvO_2). Arterial blood samples were drawn for the determination of pH, oxygen (pO_2) and carbon dioxide (pCO_2) tension, base excess (BE), the bicarbonate, haemoglobin and lactate concentration. A nasogastric tonometry catheter was measured gastric luminal pCO_2 ($pgCO_2$) which is in equilibrium with gastric mucosal pCO_2 by automatic gas capnometry [10] and calculated the difference between gastric and arterial pCO_2, (CO_2-gap), which has been shown to reflect gastro-intestinal stagnant hypoxia secondary to hypovolaemia [9, 11]. To rule out intragastric CO_2 generation following the buffering of acid with bicarbonate, patients received 300 mg of ranitidine [12]. Using special polarographic microprobes (GMS Inc., Germany), myocardial partial oxygen pressure (pO_2myo) was determined. The probe was placed in the ischemic left ventricular area between a diagonal branch and the left anterior descending artery.

In a statistical analysis Fischer´s probability test and Wilcoxon´s signed rank test were used. Differences between groups were tested with the Mann-Whitney U test. A p value less than 0,05 was regarded significant.

Results and discussion. All the patients had an uneventful postoperative course without signs of myocardial infarction as determined by an electrocardiogram and cardiac enzyme levels taken every 6 hours postoperatively. Demographic and perioperative data are presented in Table 1.

The total urine output was 4153 ± 1288 ml in the LV group and 4472 ± 1294 ml in the HV group ($p>0,05$). Both groups received approximately two third of the total amount of volume during the first five hours after admittance to the ICU. There were no differences of the CO_2-gap between the groups at any time point, nor any change over time. The pO_2myo steadily increased over the whole observation period in the LV group ($p<0,001$), but showed no improvement in the HV group ($p>0,05$) (Fig.1).

Hemodynamic data are summarized in Table 2. The LV group showed a reduction of MAP in the second half of the observation period. CVP and MPAP did not show differences between groups. The CI increased and SVR decreased in both groups over time. SvO_2 in the LV group was higher, showing significance at five and six hours after admittance to the ICU. SVI increased in parallel to the CI (LV group, $p <0,001$, HV group, $p<0,01$).

Table 1

Demographic and perioperative patients' data in volume replacement groups (mean ± SD)

Patients data	Group LV	Group HV	p
Age (years)	69,6±8,0	69,4±8,7	0,97
Gender (f / m)	4 / 6	2 / 8	0,62
CPB time (minutes)	94,7 ± 15,7	89,0 ± 18,3	0,49
X-clamp time (minutes)	47,4 ± 17,1	45,0 ± 26,1	0,54
Ventilation time (hours)	12,6 ± 5,5	16,0 ± 1,7	0,02
Volume of fluid (ml)	1070 ± 521	2865 ± 567	0,001

Table 2

Analysis of volume replacement: Haemodynamics (mean ± SD)

Data	Group	Time of infusion				
		01 h	02 h	05 h	07 h	10 h
MAP	LV	81,0±9,0	76,0±7,0[a,b]	75,0±12,0	71,0±4,0[b]	75,0±6,0[b]
	HV	79,0±10,0	69,0±6,0	73,0±9,0	75,0±10,0	73,0±14,0
HR	LV	99,0±8,0	100,0±8,0	98,0±8,0	96,0±6,0	96,0±10,0
	HV	96,0±9,0	98,0±13,0	96,0±18,0	93,0±14,0	93,0±12,0
CVP	LV	7,0±3,0	9,0±3,0	10,0±2,0	10,0±3,0	11,0±3,0
	HV	7,0±3,0	9,0±3,0	10,0±4,0	9,0±4,0	9,0±3,0
MPAP	LV	17,0±4,0	19,0±5,0	18,0±3,0	20,0±2,0	18,0±3,0
	HV	16,0±4,0	17,0±4,0	21,0±4,0[b]	20,0±2,0[b]	18,0±4,0
SVR	LV	1299±360	1079±364	942±306[b]	850±169[a,b]	861±233[b]
	HV	1342±316	994±222[b]	996±256[b]	1101±261[b]	1046±363[b]
CI	LV	2,6±0,6	3,0±1,0	3,0±0,6[b]	3,2±0,8	3,4±0,9
	HV	2,3±0,4	2,7±0,6[b]	2,8±0,7[b]	2,7±0,5[b]	2,9±0,6[b]
SvO_2	LV	66,0±7,0	64,0±7,0	71,0±9,0 [a]	70,0±8,0	69,0±11,0
	HV	63,0±6,0	65,0±5,0	61,0±6,0	64,0±8,0	64,0±8,0

MAP: Mean arterial pressure (mmHg); HR: Heart rate (bpm); CVP: Central venous pressure (mmHg); MPAP: Mean pulmonary artery pressure (mmHg); SVR: Systemic vascular resistance (dyn · sec · cm^{-5}); CI: Cardiac index ($l \cdot min^{-1} \cdot m^{-2}$); SvO_2: Mixed venous oxygen saturation (%). [a] $p<0.05$ between groups (Mann-Whitney-U-test); [b] $p<0.05$ different to baseline data (Wilcoxon test)

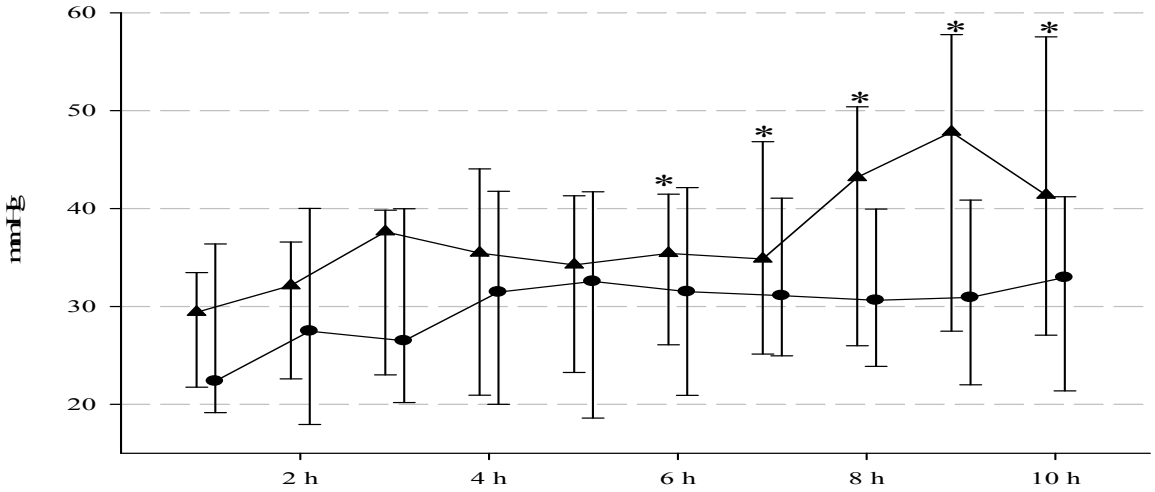

Fig. 1. Myocardial partial oxygen tension (pO_2myo). (Values are expressed as median ± quartiles. Group LV = solid triangles (▲), group HV = solid circles (●). * significant differences $p<0,05$ from baseline data)

Looking independently at the first and second half of the observation period the course of hemodynamic and oxygenation values seem to be divided in two different parts. SVI only increased from 01 h to 05 h, but remained stable in the HV group from 06 h until 10 h (p=0,85), whereas tendency continued to increase in the LV group (p=0,08). During the second half of the observation period the CO_2-gap decreased in the LV group (p<0,05) and remained stable in the HV group (p>0,05).

In this retrospective study we could show the known positive effects of volume replacement therapy on global hemodynamic in patients after cardiac surgery. In both groups CI and SVI increased significantly, but during the second half of the observation period the HV group could not benefit as much as the LV group, maybe because of a fluid overload. By more improved global hemodynamic during the second half of the observation period the LV group could increase splanchnic perfusion, shown in decreasing CO_2-gaps. Despite of receiving a higher amount of fluids the HV group could not benefit. Consistent with the idea of a fluid overload and a slight congestive cardiac insufficiency, myocardial oxygenation showed an impairment in the HV group. It is important to note that the two groups did not differ in conventional global parameters of volume status as HR, CVP, or MPAP nor showed high filling pressures as a sign of myocardial decompensation.

Volume and fluid deficits are extremely common in postoperative cardiac surgery patients. Preoperative depletion, intraoperative fluid losses and bleeding can produce absolute, while vasodilatation mediated by vasodilating substances may cause relative volume deficits. In addition hypovolaemia may develop secondary to an impairment of the endothelial barrier during inflammation after extracorporal circulation, resulting in diffuse capillary leakage. Untreated hypovolaemia causes prolonged splanchnic hypoperfusion and ischemia by increased levels of angiotensin II and reduced levels of bradykinin [1, 13]. Later on, as a sign of persisting tissue ischemia, elastase of polymorphnuclear granulocytes rises [14]. Finally, mucosal permeability increases and systemic inflammation derives from endotoxinaemia [15]. A deadly vicious circle could cause a mortality and morbidity increase [4].

It has been shown, that a low stroke volume in the initial postoperative period is a good marker for predicting a postoperative complication [16] and Mythen and Webb demonstrated that perioperative plasma volume expansion reduces not only the incidence of gut mucosal hypoperfusion, but also improves the outcome [5]. Consistent with these results we showed a positive effect of volume therapy on global hemodynamics. In both groups SVI and CI increased, but in the HV group only during the first half of the observation period. Looking at splanchnic perfusion, our patients developed only slightly higher CO_2-gap values compared to older studies. Jakob and colleagues reported a maximum CO_2-gap of 14,5±7,1 mmHg within 12 hours after cardiac surgery [17]. Our patients had maximum CO_2-gaps of 17,0±13,0 mmHg (03 h, LV group) and 16,0±5,0 mmHg (06 h, HV group). But patients in the HV group showed no decrease in CO_2-gaps at that moment, when no further increase in SVI could be achieved by volume therapy, whereas patients in the LV group could decrease their CO_2-gaps by increasing SVI.

Our data suggest that a fluid overload leads to a deterioration or at least no improvement of myocardial partial oxygen pressure. Five hours after admission to the ICU patients in the HV group were not able to increase their SVI with fluid loading anymore. Myocardial tissue pO_2 did not rise in this group, in contrast, patients in the LV group showed a steady increase. PO_2myo in patients with high volume replacement remained low with values around 33 mmHg ten hours after admission to the ICU. The LV group showed higher values than the "normal" course of pO_2myo after cardiac surgery. The mean pO_2myo in patients with uneventful recovery after cardiac surgery has been reported to be around 36 mmHg twelve hours after CPB [18]. The mean value of the LV group was 43,0±18,0 mmHg at that time point, compared to 33,0±16,0 mmHg in the HV group.

These changes occurred without significant changes of CVP and only minor changes of MPAP in the HV group. This supports the view that routinely measured standard cardiovascular variables (CVP, PAP, Wedge-Pressure, MAP, and HR) are of a limited value in assessing the actual volume status. Flow-derived values may probably be more helpful than measuring filling pressures [5]. However, the results give more evidence to the fact, that too much volume replacement at the wrong time could be detrimental or at least not supportive to the myocardium. This supports the view that no monitoring method should be looked separately, but into clinical context with all other available information.

This study was neither designed nor powered enough to serve as an outcome study, so the significant difference in ventilation time between the two groups should be interpreted very cautiously.

Conclusion

Volume therapy guided by conventional markers of volume status, i.e. CVP, MAP, HR, may lead to a fluid overload and different effects on regional perfusion in different organs, i.e. the splanchnic region and the myocardium. Flow derived markers of volume status, e.g. SVI may work better. If regional parameters, e.g. the CO_2-gap, more sensitively reflect the adequacy of intravascular volume status, they should be studied in further prospective investigations.

References

1. Reilly P.M., Bulkley G.B. Vasoactive mediators and splanchnic perfusion // Crit. Care Med. – 1993. – V. 21, №2 (Suppl.). – P. 55-68.
2. Groeneveld A.B., Kolkman J.J. Splanchnic tonometry: a review of physiology, methodology, and

clinical applications // J. Crit. Care. – 1994. – V. 9, № 3. – P. 198-210.
3. Hamilton-Davies C., Barclay G.R., Cardigan R.A. et al. Relationship between preoperative endotoxin immune status, gut perfusion, and outcome from cardiac valve replacement surgery // Chest. – 1997. – № 5. – P. 1189-1196.
4. Kavarana M.N., Frumento R.J., Hirsch A.L. et al. Gastric hypercarbia and adverse outcome after cardiac surgery // Intensive Care Med. – 2003. – № 5. – P. 742-748.
5. Mythen M.G., Webb A.R. Perioperative plasma volume expansion reduces the incidence of gut mucosal hypoperfusion during cardiac surgery // Arch Surg. – 1995. – № 4. – P. 423-429.
6. Richard C. Tissue hypoxia. How to detect, how to correct, how to prevent? // Intensive Care Med. – 1996. – № 11. – P. 1250-1257.
7. Dellinger R.P., Carlet J.M., Masur H. et al. Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock // Crit. Care Med. – 2004. – № 3. – P. 858-873.
8. Hamilton-Davies C., Mythen M.G., Salmon J.B. et al. Comparison of commonly used clinical indicators of hypovolaemia with gastrointestinal tonometry // Intensive Care Med. – 1997. – № 3. – P. 276-281.
9. Chapman M.V., Mythen M.G., Webb A.R., Vincent J.L. Report from the meeting: Gastrointestinal Tonometry: State of the Art // Intensive Care Med. – 2000. – № 26. – P. 613-622.
10. Creteur J., De Backer D., Vincent J.L. Monitoring gastric mucosal carbon dioxide pressure using gas tonometry: in vitro and in vivo validation studies // Anesthesiology. – 1997. – № 3. – P. 504-510.
11. Schlichtig R., Mehta N., Gayowski T.J. Tissue-arterial pCO_2 difference is a better marker of ischemia than intramural pH (pHi) or arterial pH-pHi difference // J. Crit Care. – 1996. – № 2. – P. 51-56.
12. Bams J.L., Kolkman J.J., Roukens M.P. et al. Reliable gastric tonometry after coronary artery surgery: need for acid secretion suppression despite transient failure of acid secretion // Intensive Care Med. – 1998. – № 11. – P. 1139-1143.
13. Uhlig T., Kuss O., Kuppe H. et al. [Gastric mucosal tonometry as a monitoring method in cardiac anesthesia. Empirical findings on the postoperative outcome under various volume controls] // Anasthesiol Intensivmed Notfallmed Schmerzther. – 1998. – V. 33, № 2 Suppl. – P. 99-105.
14. Mythen M.G., Purdy G., Mackie I.J. et al. Postoperative multiple organ dysfunction syndrome associated with gut mucosal hypoperfusion, increased neutrophil degranulation and C1- esterase inhibitor depletion // Br. J. Anaesth. – 1993. – № 6. – P. 858-863.
15. Andersen L.W., Landow L., Baek L. et al. Association between gastric intramucosal pH and splanchnic endotoxin, antibody to endotoxin, and tumor necrosis factor-alpha concentrations in patients undergoing cardiopulmonary bypass // Crit. Care Med. – 1993. – № 2. – P. 210-217.
16. Poeze M., Ramsay G., Greve J.W., Singer M. Prediction of postoperative cardiac surgical morbidity and organ failure within 4 hours of intensive care unit admission using esophageal Doppler ultrasonography // Crit. Care Med. – 1999. – № 7. – P. 1288-1294.
17. Jakob S.M., Ruokonen E., Takala J. Assessment of the adequacy of systemic and regional perfusion after cardiac surgery // Br. J. Anaesth. – 2000. –№ 5. – P. 571-577.
18. Misfeld M., Heinze H., Sievers H.H., Kraatz E.G. Intramyocardial oxygen monitoring in coronary artery bypass surgery // J. Clin. Monit. Comput. – 2004. – № 2. – P. 93-101.

ВПЛИВ ОБ'ЄМУ ПІСЛЯОПЕРАЦІЙНОЇ ІНФУЗІЙНОЇ ТЕРАПІЇ НА ЧЕРЕВНИЙ КРОВОТІК ТА ОКСИГЕНАЦІЮ МІОКАРДА В КАРДІОХІРУРГІЧНИХ ХВОРИХ

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Резюме. Метою роботи було проаналізувати вплив об'єму рідини, що уводили кардіохірургічним хворим у післяопераційному періоді, на черевний кровотік та оксигенацію міокарда. Обстежено 20 хворих, розподілених на дві групи за медіаною об'єму післяопераційної замісної інфузії. У групі з меншим об'ємом рідини ударний об'єм серцевого викиду та серцевий індекс постійно зростали впродовж спостереження. У групі з більшим об'ємом рідини таке підвищення спостерігали тільки в перші 5 год, у той же час оксигенація міокарда суттєво порушувалась, а штучна вентиляція була більш тривалою. Можна вважати, що надлишкове навантаження рідиною є несприятливим для гемодинаміки, неефективним у покращанні черевного кровотоку і також має шкідливий вплив на оксигенацію міокарда та легеневу функцію. Замісна терапія рідиною повинна дозуватися відповідно до індивідуальних фізіологічних реакцій.

Ключові слова: гіповолемія, замісна терапія, черевний кровотік, оксигенація міокарда, шлункова тонометрія.

ВЛИЯНИЕ ОБЪЕМА ПОСЛЕОПЕРАЦИОННОЙ ИНФУЗИОННОЙ ТЕРАПИИ НА БРЮШНОЙ КРОВОТОК И ОКСИГЕНАЦИЮ МИОКАРДА У КАРДИОХИРУРГИЧЕСКИХ БОЛЬНЫХ

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Резюме. Целью работы было проанализировать влияние объема жидкости, вводимого кардиохирургическим больным в послеоперационном периоде, на брюшной кровоток и оксигенацию миокарда. Обследовано 20 больных, распределенных на две группы по медиане объема послеоперационной заместительной инфузии. В группе с мень-

шим объемом жидкости ударный сердечный объем и сердечный индекс постоянно возрастали в течение наблюдения. В группе с большим объемом жидкости такой рост отмечался только в течение первых 5 часов, в то же время оксигенация миокарда была существенно нарушена, а искусственная вентиляция была более длительной. Можно полагать, что избыточная нагрузка жидкостью является не только неблагоприятной для гемодинамики и брюшного кровотока, но и имеет отрицательное влияние на оксигенацию миокарда и легочную функцию. Заместительная терапия жидкостью должна дозироваться в соответствии с индивидуальными физиологическими реакциями.

Ключевые слова: гиповолемия, заместительная терапия, брюшной кровоток, оксигенация миокарда, желудочная тонометрия.

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ОСОБЛИВОСТІ АНТИСЕКРЕТОРНОЇ ДІЇ ВНУТРІШНЬОВЕННОГО ОМЕПРАЗОЛУ В ОСІБ ІЗ КРОВОТОЧИВИМИ ВИРАЗКАМИ ДВАНАДЦЯТИПАЛОЇ КИШКИ

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Резюме. Досліджено особливості змін pH шлунка в осіб з активними кровотечами та стигматами недавніх кровотеч із виразок дванадцятипалої кишки під впливом внутрішньовенного уведення омепразолу. Проведено порівняння ефективності двох схем уведення омепразолу.

Ключові слова: омепразол, кровоточиві виразки дванадцятипалої кишки.

Вступ. В етіопатогенезі виникнення та прогресування пептичних виразок дванадцятипалої кишки (ДПК), а також розвитку таких грізних ускладнень, як кровотечі, пенетрація та інші залишається ціла низка нез'ясованих питань. Однак незаперечним є факт порушень рівноваги між механізмами захисту слизової оболонки гастродуоденальної зони та чинниками "агресії" з переважанням останніх [1-4]. Особливо зростає роль кислотно-пептичного чинника при виразкових кровотечах шлунка та ДПК, оскільки дослідження in vitro показали, що кислота несприятливо впливає на коагуляцію крові та агрегацію тромбоцитів і відіграє важливу роль у лізисі кров'яного згустку [4, 7, 8].

Отже, визначаючи та контролюючи pH у шлунку, за рахунок впливу на рівень секреції соляної кислоти, можна значною мірою, запобігти лізису тромбу та виникнення рецидиву виразкових кровотеч [6].

Мета дослідження. Обгрунтувати особливості впливу інгібітору протонної помпи (ІПП) омепразолу на кислотоутворювальну функцію шлунка та порівняння на цій основі ефективності двох режимів його внутрішньовенного призначення в осіб із кровоточивими виразками ДПК.

Матеріал і методи. Обстежено 28 пацієнтів віком від 22 до 71 року, які поступали із шлунково-кишковими кровотечами в Київську міську клінічну лікарню швидкої допомоги в 2005-2006 роках. Обстеженню підлягали тільки особи з кровоточивими виразками ДПК, а саме з активною кровотечею (FIA, FIB) та стигматами недавньої кровотечі: наявністю «тромбованої судини» діаметром менше 2мм (FIIA) та наявністю фіксованого згустку діаметром більше 2мм (FIIB).

При поступленні всім пацієнтам проводилася екстрена езофагогастродуоденоскопія (ЕГДС) за стандартною методикою з використанням апаратів фірми "Olympus" та "Fujinon". При виявленні джерела активної кровотечі або її стигмат відразу проводили мініінвазивні ендоскопічні втручання (МЕВ). Вони включали електрокоагуляцію, ендоскопічну ін'єкційну терапію, поєднану з електрокоагуляцією, і направлені на зупинку кровотечі та профілактику її рецидиву. МЕВ здійснювалися за допомогою розробленого нами ендоскопічного коагулюючого інжектора (патент України на корисну модель №10669) та монополярного коагуляційного зонда. Під час ендоскопічного обстеження також проводилося вимірювання пристінкового та внутрішньошлункового pH за допомогою приладу ИКЖ – 2 за загальноприйнятою методикою [5]. У подальшому пацієнтам протягом доби проводився добовий гастро-pH-моніторинг із вимірюванням внутрішньошлункового pH щогодини.

Після ендоскопічного гемостазу пацієнтам проводилася загальноприйнята гемостатична та протишокова інфузійна терапія, за необхідності призначалося переливання крові, плазми, еритроцитарної маси, кровозамінників тощо. Додатково до загальноприйнятої терапії особам із кровоточивими виразками ДПК проводилося внутріш-