2. Використання транексамової кислоти дозволяє знизити періопераційну крововтрату на 38,65 % у хворих, які перенесли гістеректомію. Транексамова кислота є ефективним препаратом для обмеження періопераційної крововтрати при виконанні гістеректомій.

3. Транексамова кислота пригнічує фібринолітичну активність (уповільнює швидкість лізису тромбу), але ніяк не впливає на коагуляційну ланку гемостазу. З цієї причини тромбоутворення в глибоких венах нижніх кінцівок ніяк не пов'язане з використанням транексамової кислоти. Застосування транексамової кислоти є безпечним методом обмеження періопераційної крововтрати.

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THORACIC EPIDURAL ANESTHESIA/ANALGESIA PREVENTS BNP LEVEL INCREASING AFTER MAJOR ABDOMINAL SURGERY

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ТОРАКАЛЬНАЯ ЭПИДУРАЛЬНАЯ АНЕСТЕЗИЯ/АНАЛГЕЗИЯ ПРЕДУПРЕЖДАЕТ ПОВЫШЕНИЕ УРОВНЯ BNP ПОСЛЕ АБДОМИНАЛЬНЫХ ХИРУРГИЧЕСКИХ ВМЕШАТЕЛЬСТВ

Изучались факторы, влияющие на высвобождение натрийуретического пептида типа В (BNP) в периоперационном периоде при проведении открытых абдоминальных хирургических вмешательств у пациентов с повышенным риском кардиальных осложнений. Пациенты, у которых наблюдалось повышение уровня данного пептида во время операции, отличались большими (средними за время операции) показателями частоты сердечных сокращений, наличием эпизодов тахикардии свыше 90 ударов в минуту и более высоким уровнем гликемии в конце операции. В раннем послеоперационном

периоде с повышением уровня гормона были связаны более высокие значения среднего артериального давления и интенсивности болевого синдрома как в покое, так и при кашле. Использование торакальной эпидуральной анестезии с последующей продленной аналгезией позволило предотвратить повышение уровня BNP как во время операции, так и в раннем послеоперационном периоде.

Ключевые слова: кардиальный риск, натрийуретический пептид типа В, торакальная эпидуральная анестезия и аналгезия.

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THORACIC EPIDURAL ANESTHESIA/ANALGESIA PREVENTS BNP LEVEL INCREASING AFTER MAJOR ABDOMINAL SURGERY

Background. B-type natriuretic peptide (BNP) was shown to be a marker of myocardial ischemia and elevated BNP and NT-proBNP levels identify patients undergoing major noncardiac surgery at high risk of cardiac complications. With this study, we aimed to determine the factors associated with increase in BNP level during surgery.

Methods. Thirty two consecutive patients, American Society of Anesthesiologists status II or III, scored as 2 or 3 on the Revised Cardiac Risk Index, scheduled to undergo major abdominal surgery were included in the study. Hemodynamic responses and pain scores were recorded throughout the procedure and on the first postoperative day. Blood samples were drawn before the procedure, 1 hr after incision, at the end of surgery and in the morning of the first postoperative day and analysed for BNP, troponin I, glucose, fibrinogen and C-reactive protein level.

Results. Comparing the patients with increase in BNP level at the end of the surgery and those without significant differences were found in type of anesthesia (general anesthesia, 100% vs. 41.6%; combined (epidural + general) anesthesia, 0% vs. 58.3%; p=0,004), in mean heart rate (HR) during surgery (86.97 [79.71–90.18] vs. 65.35 [60.28–71.18]; p=0.002), in the percentage of time during surgery HR remained at more than 90 beats/min (34.7% [20.4–47.9] vs. 0% [0–0]; p=0.0001), in glucose level 1 hr after incision (6.4 [5.7–7.6] vs. 4.5 [4.0–5.4]; p=0.005). Significant differences between patients with increase in BNP level in the morning of the first postoperative day and those without were found in type of analgesia (intravenous patient-controlled analgesia with morphine, 100% vs. 33.3%; epidural analgesia, 0% vs. 66.6%; p<0.001), in mean postoperative MAP (118.33 [112.72–119.33] vs. 93,33 [88,38–110.0]; p<0.001), mean postoperative pain scores at rest (33.5 [29.0–38.0] vs. 16.66 [14.0–24.0]; p<0.001) and during coughing (57.87 [50.75–65.0] vs. 29.33 [26.33–43.00]; p<0.001).

Conclusions. Increase in BNP level during abdominal surgery was associated with tachycardia and hyperglycaemia. In the postoperative period hypertension and high pain scores predicted BNP level elevation. Thoracic epidural anaesthesia and analgesia abolish increase in BNP level during abdominal surgical procedures and on the first postoperative day.

Key words: cardiac risk, B-type natriuretic peptide, thoracic epidural anaesthesia and analgesia.

B-type natriuretic peptide (BNP) is released from ventricular cardiac myocytes in response to increased ventricular wall stress and ischemia [1–3]. BNP measurement is an established diagnostic and prognostic tool for heart failure, stable coronary artery disease and acute coronary syndrome [4–9]. Elevations in preoperative BNP and NT-proBNP identify patients at high risk of cardiac complications after both cardiac and major noncardiac surgery [10–16]. The elevated peak postoperative BNP has recently emerged as being a predictor of hospital length of stay as well as worse longterm function 6 months to 2 yr after surgery [17; 18]. In addition, median perioperative NT-proBNP increase has been shown to be greater in patients sustaining cardiovascular

events as compared with those event free [19]. Postoperative BNP reflects not only the preoperative condition of the heart but also the dynamic consequences of surgery and anesthesia; therefore its increase indicates an impaired cardiac performance induced by intraoperative and early postoperative myocardial stress. However, the association between perioperative management and postoperative BNP release remains uncertain.

Thoracic epidural analgesia (TEA) has many potential benefits with regard to myocardial protection. Several meta-analyses have demonstrated that TEA continued for 24 hrs after surgery reduced the incidence of postoperative myocardial infarction [20; 21]. It is recommended to consider performing TEA in high-risk surgery for patients with cardiac disease [22].

The objective of the current study conducted in adult patients undergoing elective major abdominal surgery was to determine the factors associated with increase in BNP level during surgery. The hypothesis tested was that anesthesia techniques influence hemodynamic profile and have different abilities to attenuate stress and inflammatory response, differ in capacity to myocardial stress amelioration according to BNP plasma level.

Materials and Methods

Study Population

The Local Ethics Committee of a hospital center approved this study and written informed consent was obtained from all patients. Preliminary results of the study are described in this manuscript. Thirty two patients scheduled for elective major abdominal surgery were enrolled prospectively into the study from September 2011 to April 2012 at the Mechnikov Dnepropetrovsk Regional Hospital (Ukraine).

Patients were included if they were scored as 2 or 3 on Revised cardiac risk index, had sinus rhythm, had a left ventricular ejection fraction of at least 40% and were scheduled to undergo open abdominal surgery. All patients were ASA physical status II or III. Patients with unstable coronary syndromes (acute or recent myocardial infarction, unstable or severe angina pectoris), decompensated heart failure and severe renal dysfunction (requiring hemodialysis or having preoperative serum creatinine > 3 mg/dl) were excluded. Patients who had contraindications to epidural anesthesia were also excluded from analysis.

Perioperative Management

Patients were randomly allocated (by opening of an envelope) to receive either combined anesthesia with continuous thoracic epidural analgesia (n=14; CA group) or general anesthesia followed by intravenous patient-controlled analgesia (n=18; GA group). The study was conducted in an open manner because the performance of a sham epidural insertion was considered unethical due to the risk of epidural hematoma formation.

Before surgery, patients underwent a routine clinical evaluation including detailed medical history, physical evaluation, routine laboratory tests, 12-lead electrocardiography and chest radiography. Preoperative cardiac medications were continued until the day of surgery.

All patients were premedicated with intramuscular diazepam 10 mg and atropine 0.01 ml kg^{-1} 40 min before surgery. In the operating room, routine monitoring including five-lead electrocardiogram with continuous ST-segment monitoring (leads II and V5), heart rate (HR), respiratory rate, noninvasive arterial pressure (systolic, diastolic and mean), pulse oximetry and end-tidal CO_2 was established. Bispectral Index (BIS) was measured to monitor the depth of anesthesia (Aspect Medical Systems, Norwood, Massachusetts, USA). All patients received a preoperative loading dose of 7 ml/kg^{-1} of crystalloid over 15 min, continued at a baseline rate of 7 ml/kg^{-1} h^{-1}. In the CA group,

an epidural catheter was inserted then between the T_7 and T_{10} using a median approach and loss-of-resistance technique. Five minutes after administration of a 3 ml test dose of 0.75% ropivacaine a 3–5 ml main dose was administered, followed by continuous perfusion of 0.2% ropivacaine at a rate of 5–7 ml/h, which was continued up to 48 hrs after surgery. Anesthesia was then induced in all patients with thiopental (3–5 mg/kg), fentanyl (3 μg/kg), and pipecuroniun (0.06 mg/kg). Anesthesia was maintained with thiopental infusion and additional bolus injections of pipecuroniun. Intraoperative analgesia in both groups was achieved with additional bolus injections of fentanyl (0.1 mg) at the discretion of the attending anesthesiologist.

Both groups were managed to keep BIS between 40 and 60 and the mean arterial pressure (MAP) within 30% of baseline values. If the BIS was above the predefined range, thiopental infusion was increased, and if the BIS was below the predefined range, thiopental infusion was decreased. If the MAP was more than 30% from baseline value or tachycardia occurred while the BIS was in the predefined range, additional fentanyl bolus (0.1 mg) was administered. If hypertension persisted, nitroglycerine infusion was started. If the MAP decreased by more than 30% from the baseline value or was below 60 mm Hg, the crystalloid fluid infusion was accelerated. If hypotension persisted for more than 10 min, colloid was administered. If despite the volume loading the MAP remained persistently below 70% of baseline or less than 60 mm Hg, a continuous infusion of phenylephrine was started. Bradycardia, defined as HR less than 50 beats min^{-1}, was treated with atropine. Throughout the procedure noninvasive arterial pressure and HR were measured every 5 min and mean values and the percentages of time HR remained at more than 90 beats/min^{-1} and less than 60 beats/min^{-1} were calculated. Also the percentages of time while MAP were more or less than 20% from baseline value were calculated.

After surgery, all patients were transferred to the ICU and stayed for at least 24 h. All patients had access to i.v. morphine via a patient-controlled analgesia (PCA) device (Injectomat CP PACOM; Fresenius, Bad Homburg, Germany) with 0.5 mg bolus doses, 6 min lockout time, a maximal dose of 20 mg in a 4-hour period and without continuous background infusion. Ketoprofen, 100 mg every 8 hrs, was administered as a supplemental analgesic in both groups. Post-operative pain was assessed at rest and on coughing using a visual analogue scale (VAS) (0 mm=no pain; 100 mm=the worst pain imaginable) every 2 hrs if the patient was awake. The arterial pressure and heart rate were recorded every hour after the operation.

Blood samples

For BNP measurement venous blood samples were taken after 20 minutes of supine rest into a chilled syringe. Samples in chilled EDTA vacutainers, containing aprotinin (500 IU/ml), were placed on ice and centrifuged within 20 min at 4°C, and the plasma was stored at -70°C until analysis. For cardiac troponin I and C-reactive protein measurement venous blood samples were collected and centrifuged within 15 min. Serum was frozen and stored at -20°C.

BNP (Peninsula Laboratories, San Carlos, CA, USA) was determined before the procedure, at the end of surgery and in the morning of the first postoperative day. C-reactive protein (Roche Diagnostics GmbH, Mannheim, Germany) was determined before procedure and on the first postoperative day. Troponin I (Diagnostic automation, Calabasas, CA, USA) was measured before surgery, 4–6 hrs after the end of surgery and in the morning of the first postoperative day. The measurement fibrinogen level was performed for the same time points, and glucose was measured for all above listed time points and also 1 hr after incision.

Statistical Analysis

All the data were tested for normal distribution by Shapiro–Wilk test. The data are shown as mean ±SD, medians and interquartile range [25–75th percentile], or absolute frequencies as appropriate. The Wilcoxon signed-rank test was used to compare differences in BNP and troponin I concentrations between the different study time points. Univariate comparisons between patients with and without BNP increase as well as between group comparisons were performed using the Fisher exact test, Mann–Whitney U test, or unpaired Student *t* test as appropriate. The level of significance was set at a two-tailed *P* value less than 0.05. The analyses were performed using STATISTICA 6.1 software (StatSoft, Tulsa, OK, USA).

Results

The patients in both groups were similar with respect to the biometric data, comorbidities, preoperative medication and the type of surgery (Table 1).

The perioperative data are listed in Table 2. Both groups were comparable with respect to duration of surgery, thiopental consumption, blood loss and urine output. Patients in the CA group required smaller doses of fentanyl during surgery. There were more incidences of nitroglycerine use in GA group. The infusion rate was lower, however not significantly (p=0.06), in GA group during the procedure and differed between groups in the postoperative period.

None of the patients experienced any adverse cardiac events as assessed by routine clinical examination. No patient had detectable levels of troponin I at any time point.

Baseline BNP concentrations were similar in both groups (CA group 237 [175–533] pg/mL; GA group 249 [165–327] pg/mL). There was no significant difference in plasma BNP concentrations between groups and in comparison whit baseline at any time point.

Table 1

Baseline Patient Characteristics

Parameter	CA group, n=14	GA group, n=18	Parameter	CA group, n=14	GA group, n=18
Age, yr	66±6	67±7	*Preoperative medications (n)*		
Sex, male/female	8/6	8/10	ACE inhibitors	2	3
Body mass index, kg/m^2	23±3	26±3	β-Blockers	1	2
Comorbidities (n)			Nitrates	0	0
Angina pectoris	5	7	Statins	0	1
Chronic heart failure			Diuretics	0	0
NYHA I	1	1	Calcium blockers	0	0
NYHA II	8	13	*Type of surgery (n)*		
NYHA III	1	1	Gastrectomy/distal	3	3
NYHA IV	0	0	gastrectomy		
Diabetes mellitus	4	5	Gastric bypass surgery	2	1
Previous stroke/TIA	4	5	Biliary bypass surgery	6	10
Hypertension	9	14	Hemicolectomy	3	4

Values are expressed as mean ±SD or number of patients *(n)*.
CA group — combined anesthesia group, GA group — general anesthesia group, ACE inhibitors — angiotensin-converting enzyme inhibitors.

Table 2

Perioperative Data

Parameter	CA group, n=14	GA group, n=18
Duration of surgery, min	158±80	152±71
Consumption of anesthetics, mg		
Thiopental	1300 [1100–1500]	1430 [950–2200]
Fentanyl	0.4 [0.4–0.5]*	1.1 [0.9–1.4]
Intraoperatively administered medication *(n)*		
Phenylephrine	1	0
Atropine	1	0
Nitroglycerine	0*	7
Intraoperative blood loss, ml	225 [175–300]	200 [175–225]
Urine output, ml		
Intraoperative	175 [150–200]	175 [125–200]
First postoperative day	1000 [650–1275]	900 [700–1075]
Volume infusion, ml		
Intraoperative	2630±867	2098±646
First postoperative day	2190±548*	1611±296

Data are mean ± standard deviation, number of patients *(n)*, or median (interquartile range).

CA group — combined anesthesia group, GA group — general anesthesia group.

* $P<0.01$, compared with other group.

Increase in BNP level at the end of the surgery was defined as elevation of BNP serum concentration by more than 10% from the pre-operative baseline level. We compared patients with increase in BNP level (n=8) and those without (n=24) (Table 3). There were significant differences in type of anesthesia, in mean HR during surgery, in the percentage of time during surgery HR remained at more than 90 beats/min and in glucose level 1 hr after incision. The patients in both groups were comparable with respect to duration of surgery, volume of intraoperative infusion, preoperative BNP level, mean MAP and episodes of hypertension.

Also the patients with increase in BNP level in the morning of the first postoperative day (n=11) and those without (n=21) were compared. Significant differences were found in type of analgesia, in mean postoperative MAP, mean postoperative pain scores at rest and on coughing. The patients in both groups were similar with respect to infusion volume, preoperative BNP level, mean HR, glucose, fibrinogen and C-reactive protein level.

Discussion

TEA has been shown to reduce cardiac sympathetic drive that subsequently leads to reduction in myocardial contractility and heart rate, as well as change in cardiac loading conditions. Also TEA attenuates neurohormonal stress response and provides better postoperative pain relief compared with parenteral (including PCA) opioid administration.

Suttner et al. [23] demonstrated that BNP elevations after major abdominal surgery were attenuated by continuous intraoperative and postoperative thoracic epidural analgesia. Our results are in accordance with these findings; however, there are some differences in the obtained results. In the current study we did not observe BNP increase

Table 3

Characteristics of Patients Separated by Occurrence of Increase in BNP Level in Comparison with Baseline

Parameter	Patients with	Patients without	P value
Increase in BNP level in the end of the surgery			
Type of anesthesia *(n)*			
general anesthesia	8	10	0.004
combined anesthesia	0	14	
Duration of surgery, min	135 [95–162]	160 [112–172]	0.356
Intraoperative volume infusion, ml	2000 [1500–2500]	2500 [1600–3200]	0.160
Preoperative BNP level, pg/ml	170 [147–301]	258 [194–438]	0.124
Intraoperative hemodynamic responses			
Mean HR, beats/min	87 [80–90]	65 [60–71]	0.002
HR < 60 beats/min, %*	0 [0–5]	1 [0–29]	0.379
HR > 90 beats/min, %*	35 [20–48]	0 [0–0]	0.0001
Mean MAP, mm Hg	93 [88–109]	91 [80–100]	0.593
MAP > 120% of baseline value, %*	7 [5–14]	3 [0–12]	0.219
MAP < 80% of baseline value, %*	3 [0–8.5]	21 [5–45]	0.029
Glucose level, mmol/l			
1 hr after incision	6.4 [5.7–7.6]	4.5 [4.0–5.4]	0.005
end of surgery	5.5 [4.9–6.4]	4.8 [4.2–6.1]	0.363
Increase in BNP level in the morning of POD1			
Type of analgesia *(n)*			
i.v. PCA with morphine	11	7	0.001
epidural analgesia	0	14	
Volume infusion on POD1, ml	1600 [1300–2000]	1900 [1800–2100]	0.096
Preoperative BNP level, pg/ml	182 [165–258]	289 [214–599]	0.051
HR, beats/min**	74 [66.5–78]	71 [66–79]	0.896
MAP, mm Hg**	118 [113–120]	93 [88–110]	0.001
Postoperative pain scores			
at rest**	33.5 [29–38]	16,6 [14–24]	<0.001
on coughing**	58 [51–65]	29 [26–43]	<0.001
Glucose level, mmol/l			
4–6 hrs after the end of surgery	5.2 [4.8–8.2]	5 [3.5–6.8]	0.311
morning of POD1	5.7 [5.2–6.1]	6 [4.9–6.5]	0.860
CRP POD1, mg/l	80 [77.8–91]	127 [81–160]	0.061
Fibrinogen POD1, g/l	4.4 [4–5.8]	5.6 [3.6–6.5]	0.718

Data are number of patients *(n)* or median [interquartile range].

BNP — B-type natriuretic peptide, HR — heart rate, MAP — mean arterial pressure, POD1 — first postoperative day, i.v. PCA — intravenous patient-controlled analgesia.

* — Percent of total anesthesia time, ** — mean values on first postoperative day.

in CA group at the end of surgery as it was reported by Suttner et al. A likely explanation is that perioperative management was quite different especially concerning volume infusion.

Preoperative BNP levels were much higher than those previously reported by Suttner et al. This fact can be explained by inadequate chronic cardiac medication.

Another finding of our study is that increase in BNP level during abdominal surgery associated with tachycardia and hyperglycaemia, although this finding can be secondary to the difference in type of anesthesia. Glucose blood level reflects neurohormonal stress response to surgery. Catecholamine surge results in an increased heart rate, which is the major determinant of myocardial oxygen demand. Our results are in accordance with findings reported by Beattie et al. [24] in their meta-analysis, where a decreased incidence of postoperative MI associated with a larger effect of β-blockers on HR was demonstrated. Cardioprotection was shown only for trails where the maximal HR was less than 100 beats per minute. These findings of the current study lay stress on importance of tight heart rate control for adverse cardiac events prevention.

Postoperative pain scores and MAP were found to be higher in patients with increase in BNP level. Inadequate pain relief after surgery deteriorates the balance between myocardial oxygen supply and demand due to proinflammatory cytokines release, hypercoagulability and neurohormonal response and may lead to perioperative cardiac complications.

Some remarks must be included to indicate the limitations of the current study. BNP was not analyzed after the first postoperative day, although a greater number of adverse cardiac events take place after this period. Also, the patients included in the study received inadequate chronic cardiac medication. Therefore, a certain caution should be taken in extrapolating these results to other patient populations. Finally, only the univariate analysis was used to identify variables associated with BNP increase. Multivariate approaches are required to assess the independent value of each variable for predicting increase in BNP level. Better understanding of the factors influencing BNP release from ventricular cardiac myocytes in the perioperative period may help interpret predictive value of BNP measurement in surgical setting and develop cardioprotection.

In conclusion, thoracic epidural anaesthesia and analgesia abolish increase in BNP level during abdominal surgical procedures and on the first postoperative day. Perioperative BNP increase associated with tachycardia, hyperglycaemia, hypertension and inadequate pain relief.

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ГОСТРЕ ПІСЛЯОПЕРАЦІЙНЕ ПОШКОДЖЕННЯ НИРОК

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ОСТРОЕ ПОСЛЕОПЕРАЦИОННОЕ ПОВРЕЖДЕНИЕ ПОЧЕК

Цель — установить факторы риска развития острого послеоперационного повреждения почек.

Материалы и методы. Выполнено ретроспективное, обсервационное, когортное исследование острого послеоперационного повреждения почек (ОППП) с целью определения частоты развития и тяжести повреждения у больных после операции резекции абдоминального отдела аорты (n=165); грыжесечения, грыжепластики (n=74), перитонита (n=68), панкреонекроза (n=107). Острое послеоперационное повреждение почек диагностировали на основании шкалы RIFLE.

Результаты и их обсуждение. Из 414 пациентов в дооперационном периоде риск "R" развития почечного повреждения определялся у 18,5 % больных, а в послеоперационном периоде оказалось, что ОППП в стадии "I" (повреждение) наблюдалось почти в 2 раза чаще, чем предполагали. Кроме того, почти у 50 % больных в стадии "I" формировалась более тяжелая степень "F".

Установлена связь объема кровопотери и уровня мочевины и креатинина плазмы, аллотрансфузии и уровня мочевины, креатинина плазмы, а также между внутрисосудистым гемолизом и уровнем мочевины и креатинина, объемом гемодилюции и уровнем мочевины и креатинина плазмы крови.

Полученные данные стали основанием для изменения тактики внутриоперационной интенсивной терапии и сроков проведения заместительной почечной терапии. Важнейшим фактором как развития, так и исхода ОППП являются особенности оперативного вмешательства и его осложнений.

Выводы. Определен риск развития ОППП до операции на основании шкалы RIFLE, SAPS, ASA. Установлены «операционные» факторы риска развития острого послеоперационного повреждения почек. Для исхода острого послеоперационного почечного повреждения важным фактором является развитие послеоперационных осложнений.

Ключевые слова: острое повреждение почек, кровопотеря, заместительная почечная терапия.