ORIGINAL PAPERS

Adv Clin Exp Med 2007, **16**, 3, 383–388
ISSN 1230-025X

Rafał Kleczyk[1], Andrzej Mysiak[1], Piotr Brzostowicz[1],
Małgorzata Kobusiak-Prokopowicz[1], Marek Pelczar[2]

The Dynamics of Perioperative Changes in Serum BNP and Troponin I Concentrations in Patients Undergoing Heart-Valve Correction with Extracorporeal Circulation*

Dynamika zmian stężeń BNP i troponiny I w surowicy w okresie okołooperacyjnym u chorych poddanych zabiegowi korekty wady serca w krążeniu pozaustrojowym

[1] Department of Cardiology, Silesian Piasts University of Medicine in Wrocław, Poland
[2] Department of Cardiac Surgery, Silesian Piasts University of Medicine in Wrocław, Poland

Abstract

Background. Measurement of BNP (brain natriuretic peptide) and troponin I in the diagnostics of heart-vessel disease allows assessment of heart dysfunction (BNP) and necrosis of the heart muscle (troponin). The effectiveness of these markers in the diagnostics of myocardium function in cardiac surgery patients has not been clearly established. Heart surgery with extracorporeal circulation can lead to varied extents of damage and impairment of heart function which influence troponin and BNP concentrations.
Objectives. The aim of this study was to describe the dynamics of concentration changes in BNP and troponin I in the perioperative period in patients undergoing cardiac surgery with extracorporeal circulation to correct a valve defect.
Material and Methods. BNP and troponin I concentrations were measured in 17 patients (10 women and 7 men) aged 27–79 years who had aortic, mitral, or tricuspidal heart valve defects and therefore underwent cardiac surgery. The measurements were made before and 24 hours after surgery. The control group consisted of 18 people without heart-vessel diseases aged 54–75 years.
Results. Statistically significant higher concentrations of BNP in the patient group than in the control group were observed before (636.63 ± 472.13 pg/ml vs. 45.79 ± 21.92 pg/ml) and after cardiac surgery (628.22 ± 482.9 vs. 45.79 ± 21.92 pg/ml). BNP concentration did not change significantly before and after surgery. Patients in NYHA class III/IV had higher BNP concentrations before surgery than those in NYHA II (837.35 ± 580.96 pg/ml vs. 410.81 ± 116.96 pg/ml). The preoperative concentration of troponin I did not vary significantly from that of the control group. The concentration of troponin I was much higher 24 hours after the operation than the control group value. A difference in troponin I concentration in NYHA class II and class III/IV patients was not observed.
Conclusions. The increase in troponin I 24 hours after valve surgery is probably a result of damage to cardiomyocytes during the operation. Moreover, the chronic pathophysiology connected with the valve defect caused continuation of myocardium function disturbance after cardiac surgery. The positive correlation between BNP concentration 24 hours after surgery and aortic cross-clamping time indicates a relationship between ischemia and BNP release (**Adv Clin Exp Med 2007, 16, 3, 383–388**).

Key words: troponin I, BNP, extracorporeal circulation, cardiac surgery.

Streszczenie

Wprowadzenie. Jednoczesne oznaczenie troponiny oraz BNP w diagnostyce chorób układu sercowo-naczyniowego pozwala na ocenę dysfunkcji czynnościowej (BNP) i ewentualnej martwicy mięśnia sercowego (troponiny). Dotychczas nie została jednoznacznie określona skuteczność wymienionych markerów w diagnostyce czynności mio-

* This work has been supported by grant no. 1060.

kardium u pacjentów po zabiegu kardiochirurgicznym. Procedury związane z zabiegiem kardiochirurgicznym w krążeniu pozaustrojowym mogą prowadzić do uszkodzenia mięśnia sercowego i upośledzenia jego funkcji, czego wyrazem jest zwiększone stężenie troponin i mózgowego peptydu natriuretycznego (BNP).
Cel pracy. Określenie okołooperacyjnej dynamiki zmian stężeń mózgowego peptydu natriuretycznego (BNP) oraz troponiny I (Tn I) w surowicy pacjentów poddanych zabiegowi korekty wady zastawkowej.
Materiał i metody. Badaniem objęto 17 pacjentów (10 kobiet i 7 mężczyzn) w wieku 27–79 lat, u których przeprowadzono zabieg korekty wady lub wymiany zastawki serca. Oznaczanie BNP oraz troponiny I wykonano przed zabiegiem kardiochirurgicznym oraz 24 godziny po operacji. Grupę kontrolną stanowiło 18 osób bez współistniejących chorób układu sercowo-naczyniowego.
Wyniki. Wykazano znamiennie większe stężenia BNP w grupie badanych w porównaniu z grupą kontrolną zarówno przed (636,63 ± 472,13 pg/ml *vs* 45,79 ± 21,92 pg/ml), jak i po zabiegu (628,22 ± 482,9 pg/ml *vs* 45,79 ± ± 21,92 pg/ml). U badanych chorych po zabiegu operacyjnym nie wykazano znamiennych zmian stężeń BNP w porównaniu ze stężeniem przed zabiegiem operacyjnym. U pacjentów w klasie NYHA III/IV stwierdzono większe stężenia BNP przed zabiegiem operacyjnym niż u pacjentów w klasie NYHA II (837,35 ± 580,96 pg/ml vs 410,81 ± ± 116,96 pg/ml). Stężenie troponiny I w surowicy przed zabiegiem u badanych nie różniło się istotnie statystycznie w porównaniu ze stężeniem w grupie kontrolnej. 24 godziny po operacji stężenie troponiny I było istotnie większe niż w grupie kontrolnej. Nie wykazano różnicy w stężeniu troponiny I w grupie pacjentów w klasie NYHA II w porównaniu z pacjentami w klasie NYHA III/IV.
Wnioski. Zwiększenie stężenia troponiny I we wczesnym okresie po zabiegu chirurgicznej korekty wady zastawkowej wskazuje na uszkodzenie kardiomiocytów podczas tej procedury. Przewlekłe zjawiska patofizjologiczne związane z wadą zastawkową sprawiają, że pomimo przeprowadzenia zabiegu chirurgicznego, czynność miokardium nadal jest zaburzona. Dodatnia korelacja między stężeniem BNP po zabiegu operacyjnym a czasem zakleszczenia aorty wskazuje na związek niedokrwienia z uwalnianiem tego peptydu (**Adv Clin Exp Med 2007, 16, 3, 383–388**).

Słowa kluczowe: troponina I, BNP, krążenie pozaustrojowe, operacja kardiochirurgiczna.

The procedures connected with the performance of cardiac surgery with extracorporeal circulation can lead to a varied range of damage and impairment of heart function that influence troponin and BNP (brain natriuretic peptide) concentrations. BNP and troponin I measurements in the diagnostics of heart-vessel diseases allows assessment of heart dysfunction (BNP) and necrosis of heart muscle (troponin). The effectiveness of these markers in the diagnostics of myocardium action in patients after cardiac surgery has not yet been established. An incorrect BNP concentration in serum can also result from an increase in pressure in the heart's interior due to diastolic and systolic dysfunction and hypertrophy of the heart muscle. BNP is also an indicator of myocardial function [1]. Additionally, an incorrect Tn I concentration in serum is a result of cell necrosis or an increase in the permeability of cell membrane and the release of the cytoplasmic compartment of the protein [2].

Higher troponin I and T concentrations are associated with worse prognosis in patients with acute coronary syndrome, coronary vasospasm, severe aortal stenosis, and percutaneous coronary angioplasty. It was also shown that the troponin concentration increases in patients with tachycardia, myocarditis, bleeding from the digestive tract, septic shock, hypertrophic muscle of the heart, sever heart failure, stroke, mechanical or electrical trauma (including electrical cardioverion), pulmonary embolism, escalated COPD, and ketone acidosis in diabetic patients. In these cases the higher troponin concentration in serum, caused by a different mechanism from ischemia, makes the prognosis worse [3–6].

The hemodynamic dysfunction associated with valve pathology significantly influences the synthesis and release of BNP. A higher serum BNP concentration in these patients can indicate advancement of the defect and hemodynamic and structural disturbances and can also help in choosing the optimal moment to perform surgical correction. Moreover, a higher BNP concentration is related to the advancement of heart failure, which is defined by NYHA class but is not related to the ejection fraction of the left ventricular [7–9].

The aim of this study was to describe the dynamics of concentration changes of brain natriuretic peptide and troponin I in the perioperative period in patients undergoing an operation to correct a valve defect in extracorporeal circulation.

Material and Methods

Seventeen patients (10 women and 7 men) aged 27–79 years undergoing surgical valve correction or replacement of an aortal, mitral, or tricuspidal valve were examined. Five had a mitral valve replacement (MVR), six had aortal valve replacement (AVR, one patient of whom had a patent foramen ovale closure), and one patient had both mitral and aortal valve replacements. Three other patients underwent a reconstruction of the tricuspid valve modo De Vega. One of the above had aortal valve replacement with recon-

struction of the mitral valve. Each patient underwent coronarography before the operation, which did not show any significant hemodynamically important narrowing in the coronary artery. Nine patients were classified as NYHA class III/IV and eight as NYHA class II. The control group consisted of 18 people without heart-vessel diseases aged 54–75 years.

Blood was taken twice from the patients' cephalic vein: one day before and 24 hours after surgery. Blood for the BNP test was put into polypropylene tubes containing 1 mg EDTA and 500 KIU aprotynin per ml of blood. The collected samples were centrifuged at 1600 × *g* for 15 minutes at 4°C. The serum was stored at –70° C. The BNP concentration was determined using the radioimmunological method (RIA type, Peninsula Laboratories Inc., cat. no. RIK 9086). Blood for the troponin test was put into polypropylene tubes and centrifuged at 1600 × *g* for 15 minutes at 4°C. The serum was stored at –70°C. Troponin I was tested using an immunochemical method (Abbott Axsym System).

The patients were operated on using extracorporeal circulation with antegrade cold crystalloid cardioplegia (BOSTON): 12.6 ml 15% potassium chloride, 12 ml 8.4% sodium bicarbonate, 56 ml 10% sodium chloride, 5.4 ml 40% glucose, and 914 ml 0.9% sodium chloride. Before each operation, echocardiography was performed using a Hewlett Packard Sonos 5500 in accordance with the American Society of Echocardiography rules.

The statistical analysis was performed using STATISTICA 6.0 PL software. The results are given as the average ± the standard deviation. The variability of the data distribution was analyzed using the Shapiro-Wilk test. The Mann-Whitney *U* and Wilcoxon tests were also used because of the nonparametric data distribution for the analysis of the results. Correlations between the parameters were analyzed using Spearman's correlation coefficient. Differences were considered statistically significant with $p < 0.05$.

Table 1. Patients chracteristics before cardiac surgery (n = 17)

Tabela 1. Charakterystyka pacjentów przed operacją (n = 17)

n = 17	Average (Średnia)	Minimum (Wartość minimalna)	Maximum (Wartość maksymalna)
Age – years (Wiek – lata)	60.6	27	79
Weight (Masa ciała) kg	75.7	58	100
Height (Wzrost) cm	167.52	150	186
EF%	60.8	30	81
ECC (min)	94.4	50	175
Aorta stop (min)	60.8	35	105

EF – left ventricle ejection fraction.
ECC – time of extracorporeal circulation.
Aorta stop – aortic cross-clamp time.

EF – frakcja wyrzutowa lewej komory.
ECC – czas trwania krążenia pozaustrojowego.
Aorta stop – czas zakleszczenia aorty.

Results

The profile of the patients is presented in Table 1 and the results are shown in Tables 2 and 3. Statistically significant higher concentrations of

Table 2. Comparison of BNP and troponin I serum concentrations before surgery (BNP 0, Tn 0) and 24 hours after cardiac surgery (BNP 24, Tn I 24) and control group values

Tabela 2. Porównanie stężenia BNP i troponiny I w surowicy krwi przed operacją (BNP 0, Tn 0) i 24 godziny po operacji (BNP 24, Tn I 24) z pacjentami w grupie kontrolnej

	Examined patients (Grupa badana) n = 17		Control group (Grupa kontrolna) n = 38	*p*
Tn I (0) ng/ml	0.09 ± 0.23	vs.	0.026 ± 0.01	0.503
Tn I (24) ng/ml	9.03 ± 6.72	vs.	0.026 ± 0.01	0.05
p	< 0.05			
BNP (0) pg/ml	636.63 ± 472.13	vs.	45.79 ± 21.92	0.05
BNP (24) pg/ml	628.22 ± 482.9	vs.	45.79 ± 21.92	0.05
p	0.831			

Table 3. Comparison of BNP and troponin I serum concentrations before cardiac surgery (BNP 0, Tn I 0) and 24 hours from cardiac surgery (BNP 24, Tn I 24) depending on NYHA class

Tabela 3. Porównanie stężenia BNP i troponiny I w surowicy przed (BNP 0, Tn I 0) i 24 godziny po operacji (BNP 24, Tn I 24) w zależności od klasy czynnościowej NYHA

	Patients in NYHA II (Pacjenci w NYHA II) (n = 8)		Patients in NYHA III/IV (Pacjenci w NYHA III/IV) (n = 9)	*p*
Tn I (0) ng/ml	0.17 ± 0.33	vs.	0.02 ± 0.02	0.42
vs.	vs.			
Tn I (24) ng/ml	9.72 ± 6.66	vs.	8.4 ± 7.11	0.6
p	< 0.05		< 0.05	
BNP (0) pg/ml	410.81 ± 116.96	vs.	837.35 ± 580.96	< 0.05
vs.	vs.			
BNP (24) pg/ml	431.96 ± 196.44	vs.	802.67 ± 599.91	0.074
p	0.88		0.76	

$p < 0.05$ indicates statistical significance.

p < 0,05 oznacza znamienność statystyczną.

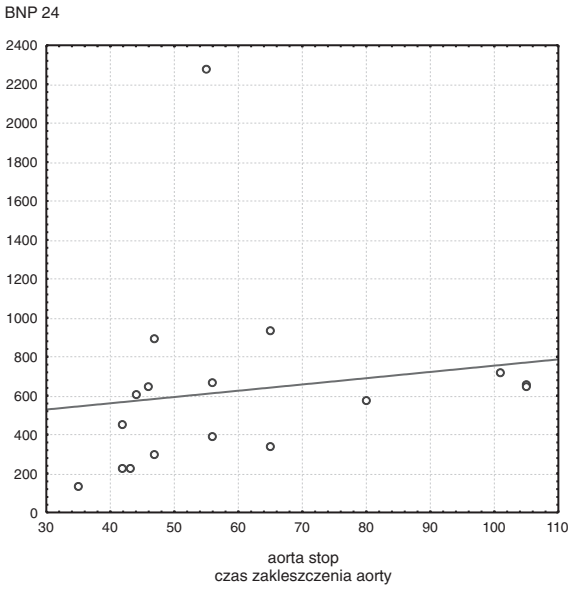

Fig. 1. Relation between BNP concentration 24 hours after cardiac surgery and aortic cross-clamp time

Ryc. 1. Korelacja między stężeniem BNP po 24 godzinach (BNP 24) od zakończenia operacji a czasem zakleszczenia aorty

BNP in the patient group before and after cardiac surgery than in the control group were observed ($p < 0.05$). The concentration of troponin I before operation did not differ significantly from that measured in the control group. The concentration of troponin I 24 hours after surgery was much higher than the control group value.

The patients with NYHA class III/IV had much higher serum BNP concentrations before surgery than those in the NYHA II group. After surgery the difference persisted, although the data did not reach statistical significance ($p = 0.074$). The troponin I concentration in serum increased after surgery regardless of the cardiac failure level. However, the BNP concentration did not change statistically significantly in either group (Table 3).

A significant correlation between BNP concentration before and after cardiac surgery and ejection fraction was not observed. A statistically significant correlation between aortic cross-clamp time and post-surgical serum BNP concentration was found (Fig. 1). However, a statistically significant relationships between extracorporeal circulation time, BNP, and troponin I concentration in the short period of time after the operation was not shown. Similarly, a statistically significant correlation between aortic cross-clamp time and troponin I concentration in serum during the tested time after the operation was not shown.

Discussion

Many studies have shown that measuring the dynamic changes in both BNP and troponin serum concentrations can be used to specify the risk of surgical heart revascularisation and the correction of valve defects. Hutflessa et al. studied patients undergoing surgical revascularization and valve correction. They showed that intraortal contrapulsation was more often necessary in patients with significantly higher BNP serum concentrations before cardiac surgery than those with lower concentrations. It was also shown that patients with higher serum BNP concentrations before surgery had to stay longer than 10 days in hospital after surgery and often died within one year after surgery [10]. It was also shown that an increase in serum troponin I concentration to over 23.8 ng/ml

after cardiac revascularization involves a higher risk of death caused by heart vessel disease within two years after operation [11]. It was also demonstrated that in patients with significant disturbance of heart valve function in whom surgical correction of the heart valve was necessary because of structural and hemodynamic disorders, the BNP concentration before operation did not correlate with lower ejection fraction, as observed in patients with heart failure [12, 13].

In the present study the initial BNP concentration corresponded with NYHA class and did not correspond with the ejection fraction of the left ventricle. BNP concentrations were much higher in the patients than in the controls. In most cases the higher troponin concentration shows necrosis of cardiomyocytes. However, the higher troponin concentration in the small group of the patients can be, as mentioned above, a consequence of higher cell membrane permeability with secondary release of the cytoplasmic compartment into the serum [14]. The data concerning the role of the changes in BNP concentration during a heart valve operation have not been thoroughly studied so far.

Berendea and coworkers showed that changes in serum BNP concentration during the perioperative period depended on the type of cardiac surgery. In the case of heart revascularization, the initially higher BNP concentration before cardiac surgery increased significantly 24 hours after the operation. It was also shown that the higher BNP concentration was dependent on the time of extracorporeal circulation, aorta cross-clamp time, and the postoperative troponin concentration. It seems that the main cause of the higher release of BNP was ischemia of the myocardium, which led to stunning and systolic dysfunction of the heart. In patients with a defect of the mitral or aortal valve, the preoperative BNP concentration increased 3- and 14-times, respectively. The preoperative BNP concentration did not change after the operation and also did not correlate with the time of extracorporeal circulation, the postoperative troponin concentration, or aorta cross-clamp time. The increase in the synthesis and release of BNP is connected with the consequences of the valve defect, such as overload of the left ventricle and higher tension of heart's wall as a result of hypertrophy myocardium and the increase in end diastolic pressure [15].

The present study showed that the BNP concentration in serum in the patients after cardiac surgery did not change significantly from that before surgery. The BNP concentration correlated neither with the time of extracorporeal circulation nor with the postoperative troponin I concentration, but only with the aorta cross-clamp time. It can also be responsible for the influence of ischemia on higher BNP release.

On the other hand, the study did not show significant differences in BNP concentration before operation in patients with coronary artery diseases, mitral valve insufficiency, and aortal valve stenosis who were qualified for operation. In patients with ischemic heart disease and mitral valve insufficiency, significantly higher BNP concentrations 8 and 12 hours after cardiac surgery were observed [16]. However, stenosis of the aortal valve did not cause the BNP concentration to increase. The high diversity of the obtained BNP concentrations in the groups of patients in that study, as in the present study, decrease the credibility of the obtained results. It was also shown that the troponin I serum concentration after cardiac surgery increased significantly from the concentration before operation. However, aorta cross-clamp time, extracorporeal circulation time, NYHA class, BNP concentration, and the type of cardiac surgery did not show any statistically significant correlations. These studies just proved the result obtained by Opfermann and coworkers, who did not observe any relationship between the increase in troponin concentration, time of operation, extracorporeal circulation time, aorta cross-clamp time, and the dose of cardioplegin [17]. It was shown that OPCAB (off-pump coronary artery bypass graft) operation is connected with lower cardiac damage described by lower troponin I and T concentrations after cardiac surgery compared with patients operated with extracorporeal circulation [18].

According to some other researchers, an increase in serum BNP concentration over 450 pg/ml and an increase in serum troponin I concentration over 5.4 ng/ml result in a 12-fold increase in the risk of heart failure. However, the determination of BNP and troponin concentrations in serum in the early perioperative period indicated heart failure more correctly than each of these markers separately. A BNP concentration over 352 pg/ml influenced higher mortality within the first year of observation [19]. It also seems that determination of the BNP and troponin serum concentrations before operation and a short time after operation can be useful in defining the overload of the left ventricle, its dysfunction, ischemia, and also necrosis of the heart muscle.

The authors conclude that an increase in the troponin I concentration in serum 24 hours after surgical correction of a valve defect suggests cardiomyocyte damage during the procedure. A long-term high BNP concentration in serum 24 hours after surgical correction of a valve defect is responsible for the existence of the chronic patho-

physiological phenomenon during disease, the consequences of which did not recede directly after surgery. The BNP concentration in serum, regardless of initial the BNP concentration, did not change within the short time after cardiac surgery. The positive correlation between aortic cross-clamp time and serum BNP concentration 24 hours after surgical correction of a valve defect showed that perioperative ischemia of the heart muscle influenced BNP release.

References

[1] Bursi F, Weston SA, Redfield MM, Jacobsen SJ, Pakhomov S, Nkomo VT, Meverden RA, Roger VL: Systolic and diastolic heart failure in the community. JAMA 2006, 8, 2209–2216.

[2] Bleier J, Vorderwinkler KP, Falkensammer J, Mair P, Dapunt O, Puschendorf B, Mair J: Different intracellular compartmentations of cardiac troponins and myosin heavy chains: a causal connection to their different early release after myocardial damage. Clin Chem 1998, 44, 1912–1918.

[3] Mahajan N, Mehta Y, Rose M, Shani J, Lichstein E: Elevated troponin level is not synonymous with myocardial infarction. Int J Cardiol 2006, 111, 442–449.

[4] Nienhuis MB, Ottervanger JP, Dikkeschei B, Suryapranata H, de Boer MJ, Dambrink JH, Hoorntje JC, van 't Hof AW, Gosselink M, Zijlstra F: Prognostic importance of troponin T and creatine kinase after elective angioplasty. Int J Cardiol 2006, 18, Abstract.

[5] Mehta NJ, Khan IA, Gupta V, Jani K, Gowda RM, Smith PR: Cardiac troponin I predicts myocardial dysfunction and adverse outcome in septic shock. Int J Cardiol 2004, 95, 13–17.

[6] Janata K, Holzer M, Laggner AN, Mullner M: Cardiac troponin T in the severity assessment of patients with pulmonary embolism: cohort study. BMJ 2003, 326, 312–313.

[7] Iltumur K, Karabulut A, Yokus B, Yavuzkir M, Taskesen T, Toprak N: N-terminal proBNP plasma levels correlate with severity of mitral stenosis. J Heart Valve Dis 2005, 14, 735–741.

[8] Arat-Ozkan A, Kaya A, Yigit Z, Balci H, Okcun B, Yazicioglu N, Kucukoglu S: Serum N-terminal pro-BNP levels correlate with symptoms and echocardiographic findings in patients with mitral stenosis. Echocardiography 2005, 22, 473–478.

[9] Weber M, Arnold R, Rau M, Brandt R, Berkovitsch A, Mitrovic V, Hamm C: Relation of N-terminal pro-B-type natriuretic peptide to severity of valvular aortic stenosis. Am J Cardiol 2004, 15, 740–745.

[10] Hutfless R, Kazanegra R, Madani M, Bhalla MA, Tulua-Tata A, Chen A, Clopton P, James C, Chiu A, Maisel AS: Utility of B-type natriuretic peptide in predicting postoperative complications and outcomes in patients undergoing heart surgery. J Am Coll Cardiol 2004, 19, 1873–1879.

[11] Fellahi JL, Gue X, Richomme X, Monier E, Guillou L, Riou B: Short- and long-term prognostic value of postoperative cardiac troponin I concentration in patients undergoing coronary artery bypass grafting. Anesthesiology 2003, 99, 270–274.

[12] Bursi F, Weston SA, Redfield MM, Jacobsen SJ, Pakhomov S, Nkomo VT, Meverden RA, Roger VL: Systolic and diastolic heart failure in the community. JAMA 2006, 8, 2209–2216.

[13] Sutton TM, Stewart RA, Gerber IL, West TM, Richards AM, Yandle TG, Kerr AJ: Plasma natriuretic peptide levels increase with symptoms and severity of mitral regurgitation. J Am Coll Cardiol 2003, 41(12), 2280–2287.

[14] Bleier J, Vorderwinkler KP, Falkensammer J, Mair P, Dapunt O, Puschendorf B, Mair J: Different intracellular compartmentations of cardiac troponins and myosin heavy chains: a causal connection to their different early release after myocardial damage. Clin Chem 1998, 44, 1912–1918.

[15] Berendes E, Schmidt C, Van Aken H, Hartlage MG, Rothenburger M, Wirtz S, Scheld HH, Brodner G, Walter M: A-type and B-type natriuretic peptides in cardiac surgical procedures. Anesth Analg 2004, 98, 11–19.

[16] Georges A, Forestier F, Valli N, Plogin A, Janvier G, Bordenave L: Changes in type B natriuretic peptide (BNP) concentrations during cardiac valve replacement. Eur J Cardiothorac Surg 2004, 25, 941–945.

[17] Opfermann UT, Peivandi AA, Dahm M, Hilgenstock H, Hafner G, Loos A, Oelert H: Postoperative patterns and kinetics of cTnI, cTnT, CK-MB-activity and CK-activity after elective aortic valve replacement. Swiss Med Wkly 2001, 22, 550–555.

[18] Guerin V, Ayed SB, Varnous S, Golmard JL, Leprince P, Beaudeux JL, Gandjbakhch I, Bernard M: Release of brain natriuretic-related peptides (BNP, NT-proBNP) and cardiac troponins (cTnT, cTnI) in on-pump and off-pump coronary artery bypass surgery. Surg Today 2006, 36, 783–789.

[19] Provenchere S, Berroeta C, Reynaud C, Baron G, Poirier I, Desmonts JM, Iung B, Dehoux M, Philip I, Benessiano J: Plasma brain natriuretic peptide and cardiac troponin I concentrations after adult cardiac surgery: association with postoperative cardiac dysfunction and 1-year mortality. Crit Care Med 2006, 34, 995–1000.

Address for correspondence:

Rafał Kleczyk
Szybka 3/50
50-421 Wrocław
Poland
Tel.: +48 600 92 88 59
e-mail: rkleczyk@dilnet.wroc.pl

Conflict of interest: None declared

Received: 7.02.2007
Revised: 8.05.2007
Accepted: 10.05.2007