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Cardiac Troponin I Is an Independent Predictor of In-hospital Death after Adult Cardiac Surgery

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Background: **Although myocardial injury during cardiac surgery is associated with impaired clinical outcome, little is known about the prognostic value of cardiac troponin I (cTnI), a cardiac-specific biologic marker. The purpose of this prospective study was to evaluate the prognostic value of cTnI concentrations measured 20 h after the end of surgery in adult patients undergoing coronary artery bypass grafting or conventional valve surgery.**

Methods: **Baseline and perioperative characteristics of 502 consecutive patients undergoing conventional heart surgery during a 1-yr period were collected. In-hospital death (n = 28) and major clinical outcomes, *e.g.*, low cardiac output, ventricular arrhythmia, and renal failure, were recorded.**

Results: **Multivariate analysis, using a stepwise logistic regression, showed that cTnI concentration was an independent predictor of in-hospital mortality (for cTnI concentration > 13 ng/ml, odds ratio = 6.7 [95% confidence interval, 2.3–19.3]), as were diabetes, altered preoperative cardiac function, emergent surgery, cardiopulmonary bypass duration, postoperative Pao_2 level and total chest drainage volume. Further, elevated cTnI concentrations were associated with a cardiac cause of death and with major clinical outcomes.**

Conclusions: **Our results demonstrated that cTnI concentration measured 20 h after the end of surgery is an independent predictor of in-hospital death after cardiac surgery. In addition, elevated concentrations of cTnI are associated with a cardiac cause of death and with major postoperative complications.**

CARDIAC surgery with cardiopulmonary bypass is often complicated by some degree of perioperative myocardial injury, despite improvements in cardioprotective strategies and surgical techniques. Whatever the pathophysiology of such perioperative myocardial injury (coronary artery embolism, graft occlusion, inadequate myocardial protection, and so on), the issue is the lack of markers that can easily and precisely identify and quantify the extent of cardiac damage.[1] Electrocardiographic (ECG) changes are of limited value after cardiac surgery.[1,2] Transesophageal echocardiography is used to noninvasively examine left ventricular (LV) function and regional wall motion but may lack the sensitivity to detect more subtle degrees of myocardial injury. Finally, the specificity of biochemical markers such as total creatine kinase (CK) and CK-MB is limited by the presence of skeletal muscle damage.[3] It does, however, appear to be crucial to recognize perioperative myocardial injury because it is associated with a decreased LV function and impaired outcomes.[4] Further, the assessment of such injury enables different techniques of myocardial protection to be compared and should help in the perioperative treatment of patients.[5,6]

The development of commercially available assays for the determination of cardiac troponin has been the most important innovation in the field of cardiovascular diagnostics in the past decade. Cardiac troponin isoforms, either I or T (cTnI, cTnT), are proteins belonging to the thin filament regulatory system of the contractile complex. Cardiac troponins are specific for the heart and are never expressed in skeletal muscle.[3,7] Because of their high sensitivity and specificity for the heart, troponins are appropriate markers for the diagnosis of perioperative myocardial infarction (PMI).[8]

In cardiac surgery patients, several studies have described the kinetics of cardiac troponin elevation in the plasma. It has been shown that cardiac surgery *per se* induces an increase in plasma level of cardiac troponins, even in the absence of ischemic myocardial damage. This increase depends on the type of surgery and the subsequent degree of direct surgical trauma. In cases of significant PMI, such as Q-wave PMI, peak levels of cTnI occur 20 h after the end of cardiac surgery and decrease slowly thereafter.[9–11] Conversely, in the absence of documented myocardial infarction, cTnI peaks earlier, and plasma levels remain lower. Nevertheless, it is difficult to define a cut-off value to diagnose PMI because there is a systematic release of cTnI resulting from cardiac surgical trauma and because ischemic myocardial damage after cardiac surgery is a continuous phenomenon. Finally, the prognostic value of isolated cardiac troponin elevation after cardiac surgery, independent of significant ECG changes, is not well defined.

We hypothesized that, whatever the mechanisms of perioperative myocardial damage, postoperative cardiac troponin concentrations might reflect the extent of myocardial injury and therefore be an independent predictor of in-hospital outcome after cardiac surgery. Our prospective study was thus designed to determine the prognostic value of cTnI concentrations 20 h after the end of

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surgery in adult patients undergoing coronary artery bypass grafting (CABG) or conventional valve surgery.

Methods

Patient Population

Consecutive patients who underwent either CABG or valve surgery from February 1997 to January 1998 at the Bichat Hospital were eligible for the study. The protocol was approved by the local ethic committee, which waived the need for informed consent because cTnI measurements were performed from a blood sample withdrawn for other routine blood tests. Reasons for exclusion were acute myocardial infarction (MI, assessed by ECG changes and an elevation of cTnI $\geq$ 1.5 ng/ml) within 7 days before surgery (n = 7), active bacterial endocarditis (n = 15), and mitral valve replacement with a human mitral valve homograft (n = 22). Two patients who died within 24 h postoperatively were also not included. Finally, 502 patients were studied.

Preoperative LV function was assessed by echocardiography. All examinations were performed by a cardiologist, who classified LV systolic function as altered, mildly altered, or normal (LV ejection fraction $<$ 30%, 30–50%, or $>$ 50%, respectively). Surgical priority (elective, urgent within 24 h, or emergent) and types of surgery were also recorded.

Anesthetic and Surgical Management

Premedication was standardized for all patients; β-blocker agents were given until the day of surgery. Standardized anesthesia (midazolam, high doses fentanyl, pancuronium bromide) and monitoring techniques (ECG, arterial and pulmonary pressure monitoring) were used in all patients. Cardiopulmonary bypass (CPB) was conducted during normothermia. Myocardial protection was achieved by intermittent anterograde cold blood cardioplegia and a warm reperfusion just before the removal of aortic cross clamp. After aortic unclamping, use of internal cardioversion (and the number of shocks) was recorded in all patients. Shocks for ventricular defibrillation were given at 20 J, increased to 30 J after the second.

Postoperative Management

To detect PMI, ECGs were performed at arrival in the intensive care unit (ICU) and daily thereafter for 4 days. Postoperative echocardiography was performed in the ICU in case of hemodynamic instability or ECG changes, or later in ward by the cardiologists. ECGs and echocardiography were analyzed by two experienced clinicians unaware of clinical and biochemical information. Q-wave PMI was defined as new Q waves on ECG ($>$ 0.04 ms in at least two contiguous leads) and occurrence of postoperative severe wall motion abnormalities in the same area.

Routine biologic variables and chest radiographs were obtained at arrival in the ICU, daily, and when necessary thereafter. Arterial blood gases on arrival in the ICU were measured during mechanical ventilation at an inspired oxygen concentration 1. Postoperative bleeding was assessed by the measurement of total chest drainage volume.

Clinical Outcomes

In-hospital mortality was defined as death occurring at any time from day one after surgery to discharge from hospital. Causes of death were recorded and classified as cardiac (heart failure, ventricular arrhythmia, in the absence of signs of sepsis) or not cardiac (hemorrhage, respiratory failure, sepsis, or other causes). Dead patients were separated in two groups, according to their cTnI concentrations ($<$ or $\geq$ 13 ng/ml) to assess the linkage between causes of death and cTnI concentration.

Other postoperative complications, potentially related to myocardial dysfunction, were recorded. The occurrence of low cardiac output was defined as a cardiac index of 2 $l \cdot min^{-1} \cdot m^{-2}$ or less for more than 4 h, assessed by the pulmonary artery catheter, during the ICU stay. Postoperative ventricular arrhythmia was defined as any sustained ventricular arrhythmia (assessed by a five-lead ECG monitoring) requiring treatment. Postoperative acute renal failure was defined by the requirement of dialysis.

Measurements of Cardiac Troponin I Concentration

Blood samples were collected 20 h after the end of surgery. Samples were immediately centrifuged at 3000 rpm for 10 min, and serum samples stored at -20°C. cTnI was measured on a STRATUS II analyser (Dade Behring, Paris, France) according to the manufacturer's instructions. The upper reference limit in a control population was 0.4 ng/ml. The within-run coefficient of variation was 6%, and the between-run coefficient of variation was 13%.

Statistical Analysis

Results are expressed as mean $\pm$ SD or median [25th, 75th percentiles], as appropriate. The link between cTnI concentrations and perioperative variables or major postoperative complications was studied by nonparametric tests (Mann–Whitney or Kruskall–Wallis with the Bonferroni correction for multiple comparisons) for categorical variables and with the Spearman rank order test for continuous variables.

Potential association between perioperative variables and in-hospital death were first tested by univariate analysis using Student *t* test or the chi-square test (or Fisher exact test) for categorical data.

To assess the best cut-off limit of cTnI to predict mortality (threshold associated with the maximum of

sensitivity + specificity), receiver–operator characteristic (ROC) curves were performed.

The variables identified by univariate analysis were then analyzed with multivariate procedure, a stepwise logistic regression (Biomedical Data Processing Package, University of California, Los Angeles, CA). To avoid overestimation of the number of predictive variables, we selected these variables with conservative criteria as follows: limits to enter or remove variables in regression equation must have had a 5% probability value; the ratio between the corresponding regression coefficient and its standard error must have been greater than 2; and results were verified by two numerical procedures: an asymptotic covariance estimate and the maximum likelihood method. cTnI was initially entered in the model as a continuous variable.

At last, to better describe the relation between cTnI concentrations and in-hospital death, a second stepwise regression logistic was performed using cTnI as a categorical variable, according to the cut-off limit found with the ROC curve. Multivariate odds ratios and their 95% confidence intervals (CI) were calculated from the model (adjusted odds ratios were thus calculated from this model with two different concentrations of cTnI, low [< 13 ng/ml] and high [≥ 13 ng/ml]).

In all tests, a two-tailed *P* value less than 0.05 was considered as significant.

Results

Patients Characteristics

During the study period, 502 consecutive patients (mean age, 63 ± 14 yr) were prospectively included. Surgical procedures were CABG (n = 187, 37%), mitral valvuloplasty (n = 97, 19%), valve replacement (n = 162, 32%), or combined surgery (valve + CABG; n = 56, 11%). Among all patients, 7% were operated for urgent or emergent procedures. Mean durations of CPB and aortic clamping were respectively 94 ± 33 and 69 ± 24 min.

Cardiac Troponin I Concentrations and Surgical Procedures

As shown in table 1, surgical procedures were associated with different cTnI concentrations ($P < 0.001$). Combined surgery and mitral valvuloplasty were associated with the highest postoperative cTnI concentrations. By contrast, no difference in cTnI concentrations was found between patients scheduled for CABG surgery or valve replacement. Patients scheduled for elective surgery had lower cTnI concentrations than those operated for urgent or emergent procedures ($P = 0.01$). cTnI concentrations and aortic clamping time were slightly correlated ($r^2 = 0.1$, $P < 0.05$). No relationship was found between preoperative LV function and postoperative cTnI concentrations.

Table 1. Surgical Procedures and Cardiac Troponin I Concentrations Measured 20 h after the End of Surgery

	Number of Patients (%)	cTnI Values (ng/ml)
Surgical Procedures		
CABG	187 (37)	3 [2.0;7.0]
Combined surgery	56 (11)	8.1 [4.9;12.9]*
Valve replacement	162 (32)	4.9 [2.4;8.4]
Mitral valvuloplasty	97 (19)	5.6 [3.7;9.1]†

Data of cTnI are shown as median [25th; 75th percentiles] $P < 0.0001$, Kruskall–Wallis test; * $P < 0.0001$ *versus* the three other groups; † $P < 0.001$ *versus* CABG.

CABG = coronary artery bypass grafting.

Cardiac troponin I concentrations in patients who received an internal cardioversion at the end of CPB (n = 254, 51%) were slightly higher than cTnI concentrations in patients without internal cardioversion (5.4 [2.8; 9.8] *vs.* 4.1 [2.3; 7.6] ng/ml, respectively, $P < 0.05$). Nevertheless, when the type of surgery was considered, there was no significant association between cTnI concentration and cardioversion, suggesting that internal cardioversion *per se* did not increase cTnI concentration 20 h after surgery.

Cardiac Troponin I Concentrations and Clinical Outcomes

The association between main postoperative complications and postoperative cTnI concentrations is reported in table 2.

Twenty-eight patients (5.6%, [95% CI, 3.6–7.6]) died during the postoperative course. Mortality rates of patients undergoing CABG and valve surgery were similar (respectively, 5.3% [95% CI, 3.3, 7.3] and 4.2% [95% CI, 2.4, 5.9]), whereas the mortality rate in patients with combined surgery was higher (12.5% [95% CI, 9.6–15.4], $P = 0.04$). Patients who died were older than survivors (68 ± 13 *vs.* 63 ± 14 yr, $P = 0.05$). In patients undergoing elective CABG, the mortality rate was 2.6% in young patients (< 60 yr) and 8.9% in elderly patients

Table 2. Cardiac Troponin I Concentrations and Clinical Outcome

	With Complication	Without Complication
Q wave PMI	56.5 [43;175.5]* n = 7	4.2 [2.4;7.6] n = 495
Ventricular Arrhythmias	8.4 [4.2;28.2]* n = 17	4.3 [2.4;8.0] n = 485
Low cardiac output	9.5 [4.4;26.7]* n = 81	4.3 [2.4;7.7] n = 421
Prolonged MV (48h)	8.1 [5.2;22.3]* n = 46	4.5 [2.5;8.1] n = 456
ARF requiring dialysis	18.2 [9.1;33.5]* n = 18	4.6 [2.5;8.2] n = 484

cTnI values (ng/ml) are expressed as median (25th; 75th percentiles) * $P <$ 0.0001.

PMI = perioperative myocardial infarction; MV = mechanical ventilation; ARF = acute renal failure.

Table 3. Univariate Analysis of In-hospital Death

	Survivors (n = 474)	Nonsurvivors (n = 28)	*P* Value	Odds Ratio (95% CI)
Age (y)	63 ± 14	68 ± 13	0.05	
Female (%)	37	29	0.49	0.7 (0.3–1.7)
BMI	25.4 ± 4.3	26.1 ± 5.9	0.39	
Creatinine clearance (ml/min)	68 ± 25	58 ± 25	0.06	
Diabetes (%)	16	43	<0.0001	4.8 (2.2–10.6)
Previous MI (%)	23	25	0.32	1.5 (0.6–3.6)
Altered LV function (%)	14	46	<0.0001	9.3 (3.4–25.4)
NYHA IV (%)	4	21	<0.0001	13 (2.8–56.8)
Combined surgery (%)	4	25	<0.0001	2.9 (1.2–7.1)
Emergent surgery (%)	1	11	<0.0001	18.0 (3.7–84.1)
CPB duration (min)	93 ± 30	123 ± 56	<0.0001	
Pa_{O_2} ICU (mmHg)	340 ± 101	247 ± 138	<0.0001	
Total chest drainage (ml)	750 [545; 1030]	1300 [615; 2545]	<0.0001	
cTnI (ng/ml)	4.6 [2.5; 8.2]	14.9 [5.5; 40]	<0.0001	

Data are mean ± SD (median and 25th–75th percentiles for cTnI and total chest drainage) for continuous variables and as percentages for the categorical ones. BMI = body mass index; MI = myocardial infarction; LV = left ventricular; NYHA = New York Heart Association; CPB = cardiopulmonary bypass; ICU = intensive care unit.

(> 70 yr). We found no significant association between in-hospital death and the different surgeons (n = 3) or anesthesiologists (n = 6).

Pre- and perioperative variables significantly associated with in-hospital death in the univariate analysis are shown in table 3. In-hospital death was associated with higher cTnI concentrations (14.9 [5.5; 40] *vs.* 4.6 [2.5; 8.2] ng/ml, $P < 0.0001$). Figure 1 shows that mortality rate increases with elevated cTnI concentrations. When cTnI was above 40 ng/ml (n = 16), in-hospital death reached 44%.

Receiver–operator characteristic curve analysis showed that the best cut-off value to predict in-hospital death was 13.4 ng/ml, rounded to 13 ng/ml (fig. 2). As shown in table 4, dead patients having cTnI values above 13 ng/ml (n = 16) had a higher risk of cardiac death than dead patients with lower concentrations of cTnI (n = 12; 75% *vs.* 8%, $P < 0.0006$, Fisher exact test). We found a trend for earlier death in patients with high concentrations of cTnI (median of postoperative day of death 3.5 [3; 19] *vs.* 13 [7.5; 24], $P = 0.12$).

Multivariate analysis showed that preoperative independent predictive variables of in-hospital death were diabetes, altered LV function, and emergent surgery. The peri- and postoperative variables independently associated with in-hospital death were total chest drainage volume, Pa_{O_2}, and cTnI concentrations (table 5).

Discussion

The main result of this study is that cTnI concentration 20 h after the end of cardiac surgery is an independent predictor of in-hospital mortality after CABG or conventional valve surgery. Further, high cTnI concentrations were associated with a cardiac cause of death.

The in-hospital mortality rate reported in the present study is in agreement with other works.[12–15] In a recent multicenter European study, Roques *et al.*[15] reported an overall mortality of 4.8%. This substantial mortality could

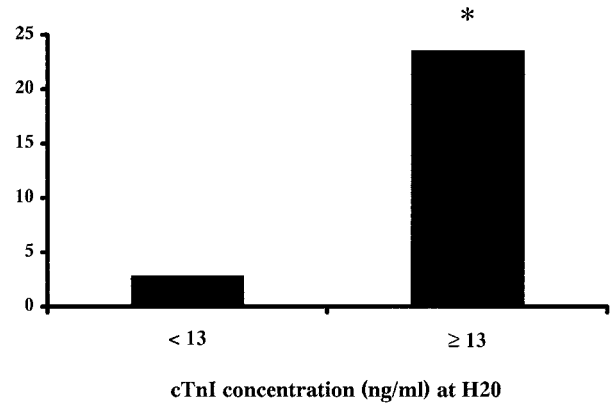

Fig. 1. Mortality rate for different concentrations of postoperative cardiac troponin I: low (< 13 ng/ml, n = 445) and high (≥ 13 ng/ml, n = 57). * $P > 0.0001$.

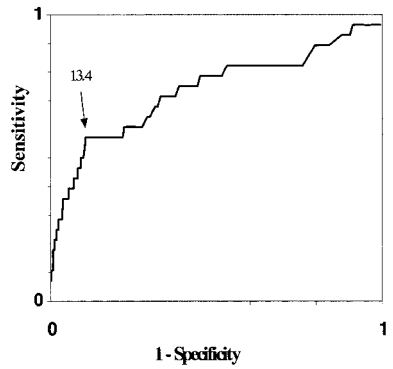

Fig. 2. Receiver–operator characteristic (ROC) curve showing that 13.4 ng/ml was the serum cardiac troponin I (cTnI) concentration (measured at hour 20) giving the most accurate prediction of in-hospital death.

Table 4. Causes of Death According to Cardiac Troponin I Concentrations

	cTnI < 13 ng/ml *n (%)*	cTnI ≥ 13 ng/ml *n (%)*
Cardiac death	*1 (8)*	*12 (75)**
Cardiogenic shock	1 (8)	11 (69)
Ventricular fibrillation	0	1 (6)
Noncardiac death	*11 (92)**	*3 (19)*
Sepsis	5 (42)	2 (12)
Gastric bleeding	2 (17)	1 (6)
Postoperative hemorrhage	2 (17)	
Respiratory distress	2 (17)	
Unknown†	0	*1 (6)*

Numbers in parentheses are percentages of patients. Cardiogenic shock was defined as a cardiac index $< 2l \cdot min^{-1} \cdot m^{-2}$ with a pulmonary wedge pressure > 18 mmHg.

* Cardiac death was significantly associated with cTnI concentrations higher than 13 ng/ml ($P = 0.001$); † One patient died at day 12 without any previous complication. Although the probability of a sudden death (severe arrhythmia?) was high, we classified it as " unknown" cause.

be explained by an increased number of high-risk patients proceeding to surgery, especially elderly patients.[13,14] In their large cohort of consecutive CABG patients, O'Connor *et al.*[12] reported a mortality rate of 4.5%, and interestingly, the major cause of death was postoperative heart failure. The first cause of in-hospital death was also heart failure in our study.

In the context of cardiac surgery, several factors, mainly direct surgical cardiac trauma and ischemic myocardial damage (resulting from inadequate myocardial protection, coronary artery embolism, graft occlusion or embolism, or other complications of the procedure), can trigger the release of cTnI. In our study, we only included patients scheduled either for CABG or conventional valve surgery because both surgical procedures, in the absence of significant ischemic myocardial damage, do not induce a large release of cTnI.[16,17] Data from patients undergoing CABG on beating heart without CPB and aortic clamping suggest that coronary artery anastomosis *per se* does not lead to an important release of cTnI,[18] in contrast with more complex procedures (*e.g.*, mitral valve replacement with human mitral valve homograft, Ross procedures, or some types of pediatric cardiac surgery).[19–21] Another issue concerning the interpretation of postoperative cTnI concentrations in terms of ischemic myocardial damage could be the frequent use of internal defibrillation before the end of CPB. It has been previously demonstrated that external defibrillation does not increase cTnI concentrations, in contrast to biochemical markers originating from skeletal muscles.[22] As far as we know, no data are available for internal defibrillation during cardiac surgery. We did not find any link between internal defibrillation and cTnI concentration at day one, when the type of surgery was considered, which does not exclude an early or a slight release of cTnI, as shown after repeated internal atrial shocks.[23] Thus, in our cohort of patients, cTnI concentration measured 20 h after the end of surgery mainly reflected ischemic perioperative myocardial damage.

Table 5. Multivariate Analysis of In-hospital Death

	P Value	Odds Ratio (95% CI)
Diabetes	0.02	4.7 (1.5–14.6)
Altered LV function	0.01	5.2 (1.7–16.4)
Emergent surgery	0.02	15.8 (2.0–123)
P_{AO_2} (mmHg)	0.03	1.1 (1.0–1.1)*
Total chest drainage	0.001	1.1 (1.0–1.2)†
cTnI (≥13ng/ml)	0.002	6.7 (2.3–19.3)

P_{AO_2} was measured upon arrival in the intensive care unit. * Odds ratio per 10 mmHg increase; †Odds ratio for 100 ml bleeding.

LV = left ventricular; CI = confidence interval.

The main finding of our study is that cTnI concentration 20 h after the end of surgery is an independent predictor of in-hospital mortality after cardiac surgery. Interestingly, the increased risk of in-hospital mortality appeared to be marked above a value of 13 ng/ml, which is close to the cut-off value that we previously found for the diagnosis of severe perioperative myocardial damage.[9] Further, the causes of death also depend on cTnI concentrations because values above 13 ng/ml were associated with the highest risk of cardiac death. All these absolute values have to be considered cautiously because there is no standardization for the cTnI determination.[24] Nevertheless, whatever the immunoassay used, the correlation between cardiac troponin concentrations and the degree of ventricular dysfunction or short-term prognosis has been already reported in other experimental or clinical settings, such as acute coronary syndrome, acute MI, pulmonary embolism, and septic shock.[25–32] The prognostic implication of the occurrence of PMI after cardiac surgery, whatever its mechanism, has been previously reported.[33] The crucial issue remains the criteria used for the diagnosis of PMI. Indeed, Q-wave PMI are far less frequent than non–Q-wave PMI after cardiac surgery.[4,34] In agreement with this, our results showed that among 502 patients, only 7 developed a Q-wave PMI. Thus, perioperative myocardial damage would not have been recognized in the majority of patients in the absence of cTnI measurement or of more sophisticated and expensive tools such as myocardial scintigraphy.[4,34] The possibility of assessing global myocardial damage with an early, inexpensive, and easy to use diagnostic tool, which is an independent predictor of in-hospital mortality, is therefore attractive.

In our cohort of patients, we found that cTnI values are also associated with major postoperative complications, even in the absence of Q-wave PMI. Our data show an association between troponin concentrations and low cardiac output, severe ventricular arrhythmia, and severe renal dysfunction. In a small cohort of children, Immer *et al.* showed that cTnI serum concentrations after pediatric cardiac surgery were correlated with the

incidence of postoperative complications.[35] Greenson *et al.* also found, in a recent study, that peak cTnI concentrations after open heart surgery were associated with adverse clinical outcomes.[36]

In addition to the cTnI predictive value, our multivariate analysis individualized other risk factors of mortality. Most of them have already been reported, such as diabetes, altered LV function, emergency, and postoperative bleeding. In our study, Pa_{O_2} level was also an independent predictor. Interpretation should however be cautious because changes in Pa_{O_2} can be linked to many clinical situations not specifically investigated in the study.

In the present study, we investigated the association between clinical outcome and one determination (at 20 h) of cTnI value. This time point was chosen because cTnI values peaked 20–24 h after surgery in case of significant intraoperative ischemic myocardial damage.[9–11,36] Further, serial measurements for a kinetic study would not have been easy to perform in daily clinical practice in our large cohort of patients. One can argue that we might have found similar results with an earlier determination of cTnI (10–12 h after surgery). Nevertheless, using ROC curves with different time points, two previous studies have clearly demonstrated that the better time point (associated with MI or cardiac events) was hour 20 or hour 24.[11,36] On the basis of its delayed kinetics in cases of MI and its early release as a result of direct surgical trauma, cTnI cannot be used in the early postoperative period after cardiac surgery as a useful tool to assess the quality of myocardial revascularization or graft patency, allowing an emergent surgical reexploration to decide.

As one measurement of cTnI concentration is strongly associated with in-hospital mortality, especially with cardiac death, it may help in the treatment of patients. However, our study did not identify the causes of cTnI elevation, and it remains to be determined if a particular care (*e.g.*, as prolonged ICU stay) on the basis of postoperative cTnI values would be beneficial. Moreover, further studies are needed to show that postoperative cTnI concentrations may be associated with the long-term prognosis and, therefore, if long-term treatments (*e.g.*, β-adrenoceptor blockers or angiotensin-converting enzyme inhibitors) would be necessary in patients with high postoperative cTnI concentrations.

In conclusion, our study demonstrates that high values of cTnI concentration at one time point, 20 h after the end of surgery, are associated with major postoperative complications after adult cardiac surgery. cTnI concentration 20 h after the end of surgery is an independent predictor of in-hospital mortality, and elevated concentrations of cTnI are associated with a cardiac cause of death.

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