Early and Delayed Myocardial Infarction after Abdominal Aortic Surgery

Yannick Le Manach, M.D., Azriel Perel, M.D., Pierre Coriat, M.D., Gilles Godet, M.D., Michèle Bertrand, M.D., Bruno Riou, M.D., Ph.D.

Background: Although postoperative myocardial infarction (PMI) after vascular surgery has been described to be associated with prolonged ischemia, its exact pathophysiology remains unclear.

Methods: The authors used intense cardiac troponin I (cTnI) surveillance after abdominal aortic surgery in 1,136 consecutive patients to better evaluate the incidence and timing of PMI (cTnI ≥ 1.5 ng/ml) or myocardial damage (abnormal cTnI < 1.5 ng/ml).

Results: Abnormal cTnI concentrations was noted in 163 patients (14%), of which 106 (9%) had myocardial damage and 57 (5%) had PMI. In 34 patients (3%), PMI was preceded by a prolonged (> 24 h) period of increased cTnI (delayed PMI), and in 21 patients (2%), the increase in cTnI lasted less than 24 h (early PMI). The mean times from end of surgery to PMI were 37 ± 22 and 74 ± 39 h in the early PMI and delayed PMI groups, respectively (P < 0.001). The mean time between the first abnormal cTnI and PMI in the delayed PMI group was 54 ± 35 h, during which the cTnI profiles of the myocardial damage and delayed PMI groups were identical. In-hospital mortality rates were 24, 21, 7, and 3% for the early PMI, delayed PMI, myocardial damage, and normal groups, respectively.

Conclusions: Intense postoperative cTnI surveillance revealed two types of PMI according to time of appearance and rate of increase in cTnI. The identification of early and delayed PMI may be suggestive of different pathophysiologic mechanisms. Abnormal but low postoperative cTnI is associated with increased mortality and may lead to delayed PMI.

THE understanding of the pathophysiology, definition, and identification of acute cardiac events is constantly improving. This is mainly due to development of assays of biomarkers that have led to a better characterization of these processes and to a better prediction of prognosis. Specifically, biomarkers such as cardiac troponin I (cTnI) have greatly enhanced our ability to identify and quantify myocardial damage (MD) in the perioperative period.1–6 However, in spite of our improved ability to detect postoperative myocardial infarction (PMI) and to assess its impact on the outcome, the exact pathophysiology of PMI has yet to be elucidated. We still do not know whether PMI is due mostly to sudden development of a thrombotic process associated with vulnerable plaque rupture, as is the case in most nonsurgical myocardial infarction, or whether it is due to the cumulative deleterious effects on myocardial oxygenation of several factors that are present after major surgery, such as increased thrombogenicity, high concentrations of circulating tissue factor, and sympathetic overactivity.2 Does PMI occur more often as a result of sudden coronary artery occlusion followed by massive necrosis, or is it mostly due to the cumulative effects of repeated or prolonged ischemic episodes, which by themselves may not cause extensive MD, as has been suggested previously?8,9 The answers to these questions may have both pathophysiologic and therapeutic implications that may potentially lead to the reduction of the still considerable cardiac morbidity and mortality after major vascular surgery.10–12 Therefore, we have attempted to better evaluate the true incidence and timing of PMI and, in particular, how often it is preceded by prolonged low cTnI concentrations, by using intense surveillance of cTnI concentrations in a large cohort of consecutive patients undergoing abdominal aortic surgery.

Materials and Methods

Since September 1995, we have used an intensive cTnI surveillance policy in all patients undergoing infrarenal aortic surgery, to better evaluate the incidence and better understand the pathophysiology of postoperative MD. All postoperative cTnI data are included in the Pitié-Salpêtrière Vascular Surgery Registry, which is a comprehensive, prospectively recorded database describing clinical and surgical characteristics of all patients undergoing aortic surgery at our institution. A systematic audit by one of the authors (M.B.) constantly verifies the accuracy of the coded data. All patients undergoing aortic surgery have been preoperatively screened in accordance with the recommendations proposed by the American Heart Association/American Col-
lege of Cardiology Task Force. This study was approved by our institutional review board (Comité Consultatif de Protection des Personnes se Prêtant à la Recherche Médicale Pitié-Salpêtrière, Paris, France). Since the study data were collected, although care of patients conformed to standard procedures currently used in our institution, authorization was granted to waive informed consent for the study.

Postoperatively, cTnI was measured on arrival to the intensive care unit (time 0); 6, 12, and 24 h later; and every 24 h thereafter for 3 days. When an abnormal concentration of cTnI was observed, the measurement was repeated 12 h later. cTnI was measured by an immunoenzymofluorometric assay on a Stratus autoanalyser (Dade-Behring, Paris La Défense, France), with values above 0.5 ng/ml being considered abnormal in our institution from September 1995 to November 1999, and values above 0.2 ng/ml being considered abnormal from November 1999 until today. PMI was defined as a cTnI concentration of at least 1.5 ng/ml, as recommended by a recent consensus conference for epidemiologic and clinical research studies. Two physicians classified each patient according to the pattern of cTnI measurements. In the case of a disagreement, a third physician was consulted.

Patients who underwent repeated surgery or had a major septic event postoperatively were excluded from the study. The overall amount of released cTnI (ng · ml⁻¹ · h⁻¹) was calculated as the area under the cTnI concentration curve by the use of a curve-fitting application that generated a series of rectangles between consecutive points on the curve, with the overall area under the curve being the sum of the areas of these rectangles. To enable us to compare the duration of hospitalization in different groups while taking into account the mortality rates, we calculated the number of hospital-free days within 1 month after surgery, with all dead patients being given a score of 0 hospital-free days. In-hospital mortality was defined as all-cause mortality before hospital discharge.

Statistical Analysis

Data are expressed as mean ± SD, median, and interquartile for nongaussian variables (e.g., duration, number of hospital-free days), or number and percentage with 95% confidence interval (CI). In the analysis of baseline characteristics, comparison of two proportions was performed with use of the chi-square test or Fischer exact test where appropriate, comparison of means was performed with use of the Student t test, and comparison of medians was performed with use of the Mann–Whitney and Kruskal–Wallis tests. Comparison of several means was performed by one-way analysis of variance. Significance was set at the 0.05 level, with all values being two-tailed. The relation between cTnI concentration and short-term mortality was assessed by logistic stepwise regression. We applied the clinical risk model of Lee et al. and collapsed the clinical risk assessment into one index variable. To compose the Lee risk index, 1 point is assigned to each of the following characteristics: high-risk type of surgery, known ischemic heart disease, a history of congestive heart failure, a history of cerebrovascular disease, diabetes mellitus, and renal failure. Subsequently, the prognostic value of this risk index and the additional and additive prognostic value of age and postoperative cTnI concentration were analyzed by logistic regression analyses. Calibration of the model was assessed using Hosmer-Lemeshow statistics (P > 0.05 for no significant difference between predictive model and observed data). All analyses were performed using SPSS 11.5 (Chicago, IL).

Results

The data of 1,152 consecutive patients, who underwent abdominal infrarenal aortic surgery from September 1995 to November 2002, were included in the study. Sixteen patients who had an abnormal postoperative increase of cTnI were not included in the final analysis because of a need for repeated surgery (n = 7) or a major septic event (n = 9). All of these patients had increased cTnI concentrations, and 9 of them had at least one cTnI measurement above 1.5 ng/ml.

Four different patterns of postoperative cTnI measurements led us to define four groups of patients (table 1). Out of the study population of 1,136 patients, 204 patients (18%) had one missing cTnI value, and 57 patients (5%) had two missing values. Patients who had cTnI values within the normal range were allocated to the no-MD group. In the case of at least one abnormal cTnI value, patients were classified as having MD or one of the two types of PMI, according to the level of the abnormal cTnI value and its pattern. Despite the occurrence of some missing values, as mentioned above, all patients except two (see below) could be allocated into one of the four groups using the other cTnI values.

Of the final study population that included 1,136 patients, 163 patients (14%) had at least one abnormal cTnI concentration. Of these patients, cTnI concentrations were always less than 1.5 ng/ml in 106 patients (10%) (MD group) and 1.5 ng/ml or greater in 57 patients (5%) who were considered to have PMI. Patients with PMI included 34 patients (3%) in the delayed PMI (DPMI) group, 21 patients (2%) in the early PMI (EPMI) group, and 2 patients in whom the cTnI profile was incomplete and precluded the definition of either EPMI or DPMI. The main preoperative clinical characteristics of the four groups of patients are summarized in table 2. History of previous myocardial infarction was significantly more frequent in the EPMI group only (table 2). Intraoperative blood loss for the whole patient population was 1,220 ±
TABLE 1. Definition of Groups According to Cardiac Troponin I Profile

<table>
<thead>
<tr>
<th>Group</th>
<th>cTnI Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>No myocardial damage</td>
<td>No abnormal cTnI values during the first 3 postoperative days</td>
</tr>
<tr>
<td>Myocardial damage</td>
<td>Patients with abnormal values of cTnI that have not exceeded at any time</td>
</tr>
<tr>
<td>Early postoperative myocardial infarction</td>
<td>a postoperative myocardial infarction threshold value of 1.5 ng/ml</td>
</tr>
<tr>
<td>Delayed postoperative myocardial infarction</td>
<td>Patients in whom the cTnI concentration exceeded the 1.5-ng/ml threshold</td>
</tr>
</tbody>
</table>

cTnI = cardiac troponin I.

320 ml, and aortic cross clamp lasted between 20 and 50 min.

The mean time from end of surgery to the detection of both PMI threshold and peak cTnI concentrations was significantly longer in the DPMI group compared with the EPMI group (fig. 1). The mean times to detection of threshold PMI cTnI concentrations were 37 ± 22 h in the EPMI group and 74 ± 39 h in the DPMI group (P < 0.001). The mean times to detection of peak cTnI concentration were 51 ± 30 and 92 ± 34 h in the EPMI and DPMI groups, respectively (P < 0.001). However, the mean times to the detection of the first abnormal cTnI concentration were similar in the two groups (29 ± 23 vs. 20 ± 20 h in the EPMI and DPMI groups, respectively) and in the MD group, as well (29 ± 18 h) (fig. 1).

In the EPMI group, PMI occurred in 10 patients (48%) by 24 h and in 18 patients (86%) by 48 h postoperatively (fig. 1). In the DPMI group, only 17 patients (50%) experienced PMI within the first 48 h, and only 10% experienced PMI on the fifth and sixth postoperative days (fig. 1). Abnormal cTnI concentrations were present in only 27% of all patients with PMI at 6 h postoperatively and in 93% by 48 h. The positive predictive value of an abnormal cTnI value for the development of PMI was 47% at 6 h and 36% at 48 h (table 3), implying that the earlier one has abnormal cTnI values, the higher the chance is that one already has or will have PMI. The negative predictive value of abnormal cTnI concentrations for PMI was 99.6% at 48 h, meaning that if by that time cTnI was normal, the chances of development of PMI were approximately 0.4% (95% CI, 0.2-0.6%; table 3).

Although the times to PMI in the DPMI and EPMI groups were significantly different, the areas under the cTnI concentration curves of both groups were similar (502 ± 600 vs. 574 ± 673 ng · ml⁻¹ · h⁻¹) and significantly higher than that of the MD group (32 ± 78 ng · ml⁻¹ · h⁻¹; P < 0.001).

TABLE 2. Main Clinical Characteristics of the Four Groups of Patients

<table>
<thead>
<tr>
<th></th>
<th>No-MD Group (n = 973)</th>
<th>MD Group (n = 106)</th>
<th>DPMI Group (n = 34)</th>
<th>EPMI Group (n = 21)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>67 ± 11</td>
<td>68 ± 11</td>
<td>70 ± 11</td>
<td>70 ± 10</td>
</tr>
<tr>
<td>Male</td>
<td>875 (90)</td>
<td>90 (85)</td>
<td>33 (97)</td>
<td>19 (90)</td>
</tr>
<tr>
<td>Female</td>
<td>98 (10)</td>
<td>16 (15)</td>
<td>1 (3)</td>
<td>2 (10)</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>339 (35)</td>
<td>45 (42)</td>
<td>19 (56)*</td>
<td>12 (57)*</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>164 (17)</td>
<td>26 (24)</td>
<td>7 (21)</td>
<td>9 (43)*</td>
</tr>
<tr>
<td>Previous coronary revascularisation</td>
<td>160 (18)</td>
<td>19 (18)</td>
<td>6 (18)</td>
<td>7 (33)*</td>
</tr>
<tr>
<td>Previous CABG</td>
<td>50 (5)</td>
<td>3 (3)</td>
<td>0 (0)</td>
<td>1 (5)</td>
</tr>
<tr>
<td>Previous PCI</td>
<td>110 (11)</td>
<td>16 (15)</td>
<td>6 (18)</td>
<td>6 (29)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>523 (54)</td>
<td>63 (59)</td>
<td>24 (71)</td>
<td>17 (81)</td>
</tr>
<tr>
<td>β Blockers</td>
<td>263 (27)</td>
<td>30 (28)</td>
<td>13 (38)</td>
<td>9 (43)</td>
</tr>
<tr>
<td>Angiotensin-converting enzyme inhibitors</td>
<td>250 (26)</td>
<td>26 (24)</td>
<td>11 (32)</td>
<td>9 (43)</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>391 (40)</td>
<td>45 (42)</td>
<td>13 (38)</td>
<td>7 (33)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>83 (8)</td>
<td>6 (6)</td>
<td>2 (8)</td>
<td>1 (5)</td>
</tr>
<tr>
<td>Chronic renal failure</td>
<td>156 (16)</td>
<td>16 (15)</td>
<td>6 (18)</td>
<td>5 (24)</td>
</tr>
<tr>
<td>Abdominal aortic aneurysm</td>
<td>675 (69)</td>
<td>71 (67)</td>
<td>22 (65)</td>
<td>16 (76)</td>
</tr>
<tr>
<td>Lee Risk Score</td>
<td>1.6 ± 0.8</td>
<td>1.7 ± 0.7</td>
<td>1.9 ± 0.8</td>
<td>2.0 ± 0.7</td>
</tr>
<tr>
<td>1</td>
<td>543 (56)</td>
<td>50 (47)</td>
<td>9 (27)</td>
<td>5 (25)</td>
</tr>
<tr>
<td>2</td>
<td>306 (31)</td>
<td>40 (39)</td>
<td>21 (61)</td>
<td>13 (60)</td>
</tr>
<tr>
<td>≥ 3</td>
<td>124 (13)</td>
<td>15 (14)</td>
<td>4 (12)</td>
<td>3 (15)</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SD or number of patients (%).

* P < 0.05 vs. no-MD group.

CABG = coronary artery bypass graft; DPMI = delayed postoperative myocardial infarction; EPMI = early postoperative myocardial infarction (see table 1 for definitions); MD = myocardial damage; no MD = no abnormal troponin Ic values; PCI = percutaneous coronary intervention.
In the DPMI group, the mean time between the first abnormal cTnI value and the development of PMI was 54 ± 35 h. During this period, the cTnI profiles of the MD and DPMI groups were comparable, as can be seen from the representative cTnI concentration curves of two patients from the MD and DPMI groups (fig. 2).

Global in-hospital mortality for the whole patient population was 4%. Mortality rates in the EPMI (24%; 95% CI, 8–47%) and DPMI (21%; 95% CI, 9–38%) groups were not different. Mortality in the PMI group (22%; 95% CI, 12–35%) was significantly higher than that of the MD group (7%; 95% CI, 2–12%), which in turn was significantly higher than the mortality of patients with normal cTnI concentrations (3%; 95% CI, 2–4%) (fig. 3). Increased postoperative cTnI, even at a low level, was an independent predictor of increased in-hospital mortality (table 4) and was associated with a significant decrease in the number of postoperative hospital-free days within 1 month after surgery (fig. 3).

**Discussion**

The postoperative cTnI profiles of our large group of consecutive patients undergoing aortic surgery suggest that there are two types of PMI that are different in their timing and biomarker pattern. EPMI occurs in the early...
postoperative period and is not preceded by subinfarc-
tion MD, whereas DPMI occurs later and is preceded by
a long period (> 24 h) of MD in which cTnI values are
increased. Because these two distinct PMI profiles were
identified in a large population with an overall inciden-
ty of PMI similar to that previously reported, we be-
lieve that our findings may have a significant clinical
relevance because they allow a better understanding of
the pathophysiology of PMI. In addition, these findings
highlight the significance of the presence of low cTnI
values after major vascular surgery.

Approximately 60% of the patients who had PMI had
their infarction preceded by a prolonged period of MD
(DPMI group). This delayed pattern of PMI is consist-
ent with previous observations by Mangano et al.,16 in
which a clear association has been demonstrated be-
tween postoperative ischemia and PMI. Landesberg
et al.9 have later shown that there is a close correlation
between the duration of postoperative ischemia, as re-
flexed by the electrocardiogram, and MD reflected by
cTnI concentrations. These authors have postulated that
prolonged ischemia, in the presence of severe but stable
coronary artery disease, may be a causative factor for
PMI, rather than acute coronary occlusion.9 The caus-
ative role of prolonged ischemia in the development of
PMI has been previously postulated as well.8

However, our findings do suggest that, in addition to
the well-recognized entity of DPMI, there is another PMI
entity, namely EPPI, which may have been hitherto
unrecognized in the postoperative setting. EPMI occurs
quite early in the postoperative period, is not preceded
by prolonged MD, and is associated with a sudden in-
crease in cTnI. Therefore, the cTnI profile of EPMI re-
sembles that of acute nonsurgical myocardial infarc-
tion and is most probably due to acute coronary occlusion
resulting from plaque hemorrhage, rupture, and throm-
bus formation, as has been observed in autopsies of fatal
PMI.17

We may therefore postulate that the mechanism un-
derlying EPMI is that of the “vulnerable plaque,” which
includes all thrombosis-prone plaques and plaques prone
to undergo rapid progression.18,19 The postopera-
tive inflammatory syndrome may greatly contribute to
this process because increased C-reactive protein plasma
concentrations have been shown to provoke plaque rup-
ture.20 Although the vulnerability of the plaque is de-

defined by its structural and functional properties, patient-
related factors may play a role in the outcome that will
result from its disruption.18,19 In this context, it is inter-
esting to note that the EPMI population in our study had
a significantly higher incidence of previous myocardial
infarction compared with the patients who experienced
DPMI, reflecting a possible higher patient-vulnerabili-
ty.21 The cTnI pattern of DPMI is more similar to that
observed during prolonged unstable angina culminating
in myocardial infarction. This pattern has been recently
defined as unstable angina class IIIa, which develops in
the presence of extracardiac conditions that intensify
myocardial ischemia (secondary unstable angina).22

Although the pathophysiology of EPMI and DPMI may
be different, we have found the extent of MD and the
associated mortality to be the same for the two types of
PMI, although PMI-related mortality was significantly
higher than that of the MD group (MD not reaching the
cTnI threshold of PMI). In this regard, postoperative
abnormal sub-PMI threshold cTnI concentrations may be
considered as a marker of unstable coronary artery dis-

case, explaining their association with lower survival

compared with patients who had normal cTnI values
(no-MD group). These findings, which are in accord with
previous reports in which mortality after vascular sur-
gery was found to correlate with cTnI concentrations,1–5
demonstrate that even postoperative abnormal although
sub-PMI threshold cTnI concentrations may worsen
outcome.

Although EPMI and DPMI seem to have a similar extent

| Table 4. Multivariate Model for the Prediction of In-hospital Mortality by Lee Risk Index, Age, and Postoperative Peak Cardiac Troponin I Concentration |

<table>
<thead>
<tr>
<th>Variables</th>
<th>OR</th>
<th>95% CI for OR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Lower</td>
</tr>
<tr>
<td>Postoperative peak cardiac troponin I</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abnormal and ≥ 1.5 ng/ml</td>
<td>8.1</td>
<td>2.9</td>
</tr>
<tr>
<td>Abnormal and &lt; 1.5 ng/ml</td>
<td>3.9</td>
<td>1.8</td>
</tr>
<tr>
<td>Normal</td>
<td>1.0</td>
<td>—</td>
</tr>
<tr>
<td>Age (per 1-yr increase)</td>
<td>1.05</td>
<td>1.03</td>
</tr>
<tr>
<td>Lee Risk Index* (per 1-point increase)</td>
<td>2.7</td>
<td>1.6</td>
</tr>
</tbody>
</table>

* To compose the Lee Risk Index, 1 point was assigned to each of the following characteristics: high-risk type of surgery, known ischemic heart disease, history of congestive heart failure, history of cerebrovascular disease, diabetes mellitus, and renal failure.

Cl — confidence interval; OR — odds ratio.

Fig. 3. (A) Number of postoperative hospital free days within 1 month after surgery in normal cardiac troponin I, myocardial damage (MD), and postoperative myocardial infarction (MI) groups. (B) In-hospital mortality of the postoperative MI, MD, and normal cardiac troponin I (control) groups.
of MD and to carry similar mortalities, the identification of these two different types of PMI may have some therapeutic implications. In patients who are prone to development of PMI, and especially in those who are prone to development of EPMI, prevention might be achieved by better preoperative identification of the vulnerable plaque and by better plaque stabilization, either metabolically (e.g., statins) or by actual coronary stenting. However, the patients who have DPMI do present an additional opportunity for the prevention of PMI during the period of increased biomarkers of myocardial injury. Because this period of subinfarction MD lasts for approximately 60 h, it should be regarded as a "golden period" in which various interventions (e.g., intensive β-blocker therapy, adequate analgesia, correction of anemia) may potentially prevent PMI. Our finding that close to 50% of the patients with abnormal cTnI concentrations at 6 h either have or will have PMI indicates that these patients might get the benefit of optimal monitoring and care until cTnI concentrations normalize. These data complement previous studies in which the majority of ischemic events, including those culminating in PMI, started immediately at the end of surgery or within 12–24 h after surgery. The very strong negative predictive value of cTnI suggests that, in the presence of normal cTnI values, the chance of developing of PMI is 3.6% at 6 h and only 0.4% after 48 h.

Our study has a few potential shortcomings, of which our definition of PMI is a major one. The introduction of cTnI as a biomarker of MD has dramatically increased our ability to detect even small amounts of damage and is far superior to the use of sophisticated but cumbersome electrocardiogram interpretation. However, the use of such a sensitive biomarker has made MD to be a "golden period" in which various interventions (e.g., statins) or by actual coronary stenting. However, the patients who have DPMI do present an additional opportunity for the prevention of PMI during the period of increased biomarkers of myocardial injury. Because this period of subincharction MD lasts for approximately 60 h, it should be regarded as a "golden period" in which various interventions (e.g., intensive β-blocker therapy, adequate analgesia, correction of anemia) may potentially prevent PMI. Our finding that close to 50% of the patients with abnormal cTnI concentrations at 6 h either have or will have PMI indicates that these patients might get the benefit of optimal monitoring and care until cTnI concentrations normalize. These data complement previous studies in which the majority of ischemic events, including those culminating in PMI, started immediately at the end of surgery or within 12–24 h after surgery. The very strong negative predictive value of cTnI suggests that, in the presence of normal cTnI values, the chance of developing of PMI is 3.6% at 6 h and only 0.4% after 48 h.

Our study has a few potential shortcomings, of which our definition of PMI is a major one. The introduction of cTnI as a biomarker of MD has dramatically increased our ability to detect even small amounts of damage and is far superior to the use of sophisticated but cumbersome electrocardiogram interpretation. However, the use of such a sensitive biomarker has made MD to be a "golden period" in which various interventions (e.g., intensive β-blocker therapy, adequate analgesia, correction of anemia) may potentially prevent PMI. Our finding that close to 50% of the patients with abnormal cTnI concentrations at 6 h either have or will have PMI indicates that these patients might get the benefit of optimal monitoring and care until cTnI concentrations normalize. These data complement previous studies in which the majority of ischemic events, including those culminating in PMI, started immediately at the end of surgery or within 12–24 h after surgery. The very strong negative predictive value of cTnI suggests that, in the presence of normal cTnI values, the chance of developing of PMI is 3.6% at 6 h and only 0.4% after 48 h.

Our study has a few potential shortcomings, of which our definition of PMI is a major one. The introduction of cTnI as a biomarker of MD has dramatically increased our ability to detect even small amounts of damage and is far superior to the use of sophisticated but cumbersome electrocardiogram interpretation. However, the use of such a sensitive biomarker has made MD to be a "golden period" in which various interventions (e.g., intensive β-blocker therapy, adequate analgesia, correction of anemia) may potentially prevent PMI. Our finding that close to 50% of the patients with abnormal cTnI concentrations at 6 h either have or will have PMI indicates that these patients might get the benefit of optimal monitoring and care until cTnI concentrations normalize. These data complement previous studies in which the majority of ischemic events, including those culminating in PMI, started immediately at the end of surgery or within 12–24 h after surgery. The very strong negative predictive value of cTnI suggests that, in the presence of normal cTnI values, the chance of developing of PMI is 3.6% at 6 h and only 0.4% after 48 h.

Our study has a few potential shortcomings, of which our definition of PMI is a major one. The introduction of cTnI as a biomarker of MD has dramatically increased our ability to detect even small amounts of damage and is far superior to the use of sophisticated but cumbersome electrocardiogram interpretation. However, the use of such a sensitive biomarker has made MD to be a "golden period" in which various interventions (e.g., intensive β-blocker therapy, adequate analgesia, correction of anemia) may potentially prevent PMI. Our finding that close to 50% of the patients with abnormal cTnI concentrations at 6 h either have or will have PMI indicates that these patients might get the benefit of optimal monitoring and care until cTnI concentrations normalize. These data complement previous studies in which the majority of ischemic events, including those culminating in PMI, started immediately at the end of surgery or within 12–24 h after surgery. The very strong negative predictive value of cTnI suggests that, in the presence of normal cTnI values, the chance of developing of PMI is 3.6% at 6 h and only 0.4% after 48 h.

Our study has a few potential shortcomings, of which our definition of PMI is a major one. The introduction of cTnI as a biomarker of MD has dramatically increased our ability to detect even small amounts of damage and is far superior to the use of sophisticated but cumbersome electrocardiogram interpretation. However, the use of such a sensitive biomarker has made MD to be a "golden period" in which various interventions (e.g., intensive β-blocker therapy, adequate analgesia, correction of anemia) may potentially prevent PMI. Our finding that close to 50% of the patients with abnormal cTnI concentrations at 6 h either have or will have PMI indicates that these patients might get the benefit of optimal monitoring and care until cTnI concentrations normalize. These data complement previous studies in which the majority of ischemic events, including those culminating in PMI, started immediately at the end of surgery or within 12–24 h after surgery. The very strong negative predictive value of cTnI suggests that, in the presence of normal cTnI values, the chance of developing of PMI is 3.6% at 6 h and only 0.4% after 48 h.

Our study has a few potential shortcomings, of which our definition of PMI is a major one. The introduction of cTnI as a biomarker of MD has dramatically increased our ability to detect even small amounts of damage and is far superior to the use of sophisticated but cumbersome electrocardiogram interpretation. However, the use of such a sensitive biomarker has made MD to be a "golden period" in which various interventions (e.g., intensive β-blocker therapy, adequate analgesia, correction of anemia) may potentially prevent PMI. Our finding that close to 50% of the patients with abnormal cTnI concentrations at 6 h either have or will have PMI indicates that these patients might get the benefit of optimal monitoring and care until cTnI concentrations normalize. These data complement previous studies in which the majority of ischemic events, including those culminating in PMI, started immediately at the end of surgery or within 12–24 h after surgery. The very strong negative predictive value of cTnI suggests that, in the presence of normal cTnI values, the chance of developing of PMI is 3.6% at 6 h and only 0.4% after 48 h.

In conclusion, based on intense cTnI surveillance, we have identified, in patients undergoing vascular surgery, two cTnI patterns that correspond to an early and a delayed PMI. Monitoring cTnI in the postoperative period after aortic surgery enables the identification of patients with abnormal but low postoperative cTnI concentrations. This may have an important clinical implication because these low cTnI concentrations are associated with increased mortality and may lead to delayed PMI. In addition, monitoring cTnI concentrations postoperatively may allow the institution of early aggressive intervention to prevent the evolution of PMI during the “golden period” of approximately 2 days before the development of delayed PMI. Further understanding of the mechanisms underlying PMI, as well as its early identification, may contribute to the reduction of the incidence of PMI and its associated mortality in the future.

References
EARLY AND DELAYED POSTOPERATIVE MYOCARDIAL INFARCTION


Copyright © by the American Society of Anesthesiologists. Unauthorized reproduction of this article is prohibited.

Anesthesiology, V 102, No 5, May 2005

Downloaded from anesthesiology.pubs.asahq.org by guest on 07/24/2019