Anesthesiology 2004; 101:1262–8

Incidence and Significance of Cardiac Troponin I Release in Severe Trauma Patients

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Background: **The incidence and significance of troponin I release and its mechanism are unknown in severe trauma patients. The characteristics of this release were prospectively studied in such patients and correlated with presence of shock, existence of myocardial contusion, and outcome.**

Methods: **During a 24-month period, serial electrocardiogram recordings and troponin I measurements were performed in all trauma patients admitted at a surgical intensive care unit. The diagnosis of a significant myocardial contusion was made on electrocardiographic criteria. According to the time course of troponin I, three groups of patients were defined *a priori*: very transient (≤ 12 h) and limited release (troponin I < 2 μg/l), transient (≤ 36 h) and significant release (troponin I ≥ 2 μg/l), and sustained (> 36 h) and significant release (troponin I > 2 μg/l). In the last group, coronary artery angiography was performed.**

Results: **The incidence of troponin I release was 12% (95% confidence interval [CI], 9.6–14.4%) in 728 patients. A significant myocardial contusion was found in 35 patients (5%; 95% CI, 3.4–6.6%) and may occur in the absence of chest trauma and without troponin I release. Sensitivity, specificity, and positive and negative predictive values of troponin I for the diagnosis of myocardial contusion were 63, 98, 40, and 98%, respectively. Troponin I release was observed in 54 early (> 48 h) survivors (7%; 95% CI, 5.6–9.6%) without preexisting coronary artery disease. A sustained and significant release of troponin I (17 patients) was frequently associated with chest trauma (82%) and constantly with electrocardiographic abnormalities. A coronary artery injury was found in 7 patients (2 major and 5 minor vascular injuries) (1% of the whole group; 95% CI, 0.4–2.0%). Mortality was similar in early survivors with (15%; 95% CI, 7–27%) or without (12%; 95% CI, 9–14%) troponin I release. The odds ratio for late mortality was 1.32 (95% CI, 0.61–2.85) in patients with troponin I release.**

Conclusions: **Serial electrocardiogram recordings and troponin I assessments may be proposed for initial screening in high-risk trauma patients to detect anatomical cardiac injuries through the time course of circulating protein. Troponin I release does not have a prognosis value in trauma patients.**

This article is accompanied by an Editorial View. Please see: Riou B: Troponin: Important in severe trauma and a first step in the biological marker revolution. ANESTHESIOLOGY 2004; 101:1259–60.

THE routine repeated assessment of cardiac enzymes for detecting a blunt cardiac injury or myocardial injury is a controversial practice during the treatment of trauma patients.[1] The lack of cardiac specificity of creatine kinase MB isoform greatly contributes to explain the low diagnosis value of this marker for such an elusive pathology in patients who generally have peripheral muscle injuries. Conversely, immunoassays provide an accurate assessment of troponin I cardiac release, which gains popularity for the diagnosis of myocardial contusion even if the diagnosis time window is narrower than that reported for "medical" myocardial ischemia.[2,3] However, circulating troponin I has been observed in a large range of clinical settings, especially circulatory failure of septic or hemorrhagic origin in the absence of direct cardiac trauma.[4–7] Because studies about troponin I have generally been concerned with limited series of selected chest trauma patients, the aims of the current prospective observational study were therefore (1) to determine the incidence and the profile of troponin I release in consecutive severe blunt trauma patients admitted at a level I surgical and trauma intensive care unit and (2) to correlate the biologic results with the presence of shock, the existence of myocardial contusion, and the outcome of the patients.

Materials and Methods

The investigation was approved by the hospital review board (Le Kremlin Bicêtre, France). Because data were recorded without any specific intervention and according to a protocol used in routine in the unit, authorization was given to waive informed consent in most trauma patients. Nevertheless, informed consent was obtained from all subjects or from their relatives when coronary angiography was performed because this investigation was not previously performed on a routine basis. During a period of 24 months (January 2001 to December 2002), all patients consecutively admitted in the unit with significant blunt trauma were prospectively included in the study. All these patients were cared for by a mobile intensive care unit (French Service d'Aide Médicale Urgente system).[8] The severity of the trauma was considered high enough by the prehospital team to war-

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Received from the Service d'Anesthésie-Réanimation, Hôpital de Bicêtre, Le Kremlin Bicêtre, France. Submitted for publication April 16, 2004. Accepted for publication August 30, 2004. Supported by grants from the French Ministère de la Recherche, Paris, France, and the Conseil Scientifique, Faculté de Médecine Paris Sud, Le Kremlin Bicêtre, France. Dr. Felten was a recipient of funding from the Fondation pour la Recherche Médicale, Paris, France.

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rant direct admission of these patients to the emergency room of a level 1 trauma center. Revised Trauma Score,[9] Injury Severity Score[10] and Simplified Acute Physiology Score[11] were calculated at the end of the resuscitative phase to assess the magnitude of the trauma and its general consequences. An Abbreviated Injury Scale score for the head or the thorax of 3 or greater defined a severe brain or thoracic injury, respectively. A Shock Score of 3 or greater defined a significant hemorrhagic shock during the early phase.[12] The care of the patients was directed by the same existing protocols and was not modified by the study. Volume loading was made to obtain a mean arterial pressure of 70 mmHg or greater, a hemoglobin concentration of 8 g/dl or greater, a prothrombin time of 16 s or greater, and a platelet count of 75×10^9/l or greater. Norepinephrine was given for persistent hypotension when the volume status was considered adequate to compensate the estimated blood loss[13] and/or to maintain a cerebral perfusion pressure of 70 mmHg or greater.[14] Plasma lactate concentration was measured using routine assays. Plasma lactate concentration was used as a biologic marker of ischemia.[15] An early death was defined as a death occurring within the 48 h after trauma.

All patients had a serum troponin I concentration drawn and 12-lead electrocardiography performed at admission. The plasma troponin I concentration was again tested and an electrocardiogram was again recorded between 6 and 12 h after admission. Thereafter, additional troponin I concentrations and electrocardiographic recordings were repeated as long as the troponin I concentration remained increased, the electrocardiogram remained abnormal, or both. Troponin I concentration was measured using an immunologic method of chemiluminescence detection and was expressed in micrograms per liter (ACS:180SE; Bayer, Paris, France). This assay showed no cross-reactivity with skeletal muscle troponin I or other cardiac proteins. The limit of detection was 0.04 μg/l. The coefficient of intraindividual variation was between 2.8 and 3.9%, and the coefficient of interindividual variation between 3.1 and 4.1%.[16] A plasma concentration above 0.4 μg/l corresponded to a significant myocardial injury, and the threshold for a diagnosis of myocardial necrosis was 1.5 μg/l.[17,18] Considering this peak value and the time course of cardiac contractile protein release after acute myocardial infarction, three troponin I release profiles were defined *a priori*[7]: very transient (≤ 12 h) and limited release (peak troponin I < 2 μg/l), transient (≤ 36 h) and significant release (peak troponin I ≥ 2 μg/l), and sustained (> 36 h) and significant release (peak troponin I ≥ 2 μg/l). In this last group of patients, the area under the curve of troponin I concentrations as a function of time corresponded to that observed during severe acute coronary syndromes.[19] Whatever the pathologic electrocardiographic signs, this sustained troponin I cardiac release associated with a recent blunt trauma, without any evidence of preexisting coronary artery disease, was considered an indication for coronary angiography.[20] The angiography was therefore performed as soon as possible. Routine left heart catheterization was made using the Seldinger technique with a 6-French sheath introduced into the femoral artery. A 6-French straight pigtail catheter was used for left ventricular angiography. Coronary angiography was performed by using a 6-French multipurpose catheter. The contrast medium used was ioxaglate sodium and meglumide (Hexabrix®, 320 mg; Guerbet, Villepinte, France). Two successive orthogonal monoplane x-ray digitized left ventricular cineangiographies (Integris V3000; Philips, Eindhoven, The Netherlands) were performed. Cineangiograms of left ventricle and coronary arteries were obtained at 25 images/s in stable sinus rhythm. Digitized images (512^2 matrix) were stored on a CD-ROM and analyzed by two independent observers.[21]

The adopted criteria for making the diagnosis of myocardial contusion were significant electrocardiographic signs detected after admission and during the period of observation, regardless of the values of troponin I concentrations.[22] These electrocardiogram criteria were defined as a cardiac rhythm other than normal sinus or transient sinus tachycardia (ventricular dysrhythmias, supraventricular tachycardia [atrial fibrillation or flutter], junctional tachycardia, sinus bradycardia), a conduction abnormality, a right or left bundle branch block pattern, a prolonged QT interval, a Q-wave formation, an ST-segment depression or elevation greater than 1 mm, or flat or inverted T waves or both in two or more leads.

Transthoracic echocardiography was selectively performed in patients with shock, electrocardiographic abnormalities, or significant troponin I release, within 24–48 h after injury. The echocardiographic criteria adopted to confirm a suspected myocardial contusion were pericardial effusion, regional wall motion abnormality (defined as a reduction in wall motion and in systolic thickening), and acute valvular dysfunction.[22] Two-dimensional echocardiographic images were obtained as recommended by the American Society of Echocardiography, and all echocardiographic data were reviewed by two observers unaware of the patients' clinical and biochemical histories.[23]

Statistical Analysis

The study was performed considering an α error of 5% and a power of 90% and assuming an expected increase in mortality of at least 100% in patients with troponin I release as compared with patients without release of the marker.[24] Three hundred fifty blunt trauma patients are usually admitted to the unit each year, with an overall mortality of approximately 20%. Assuming that the incidence of troponin I release may be of the same magnitude for shocked trauma patients as for patients with septic shock (approximately 60%)[25] and knowing that

approximately 20% of trauma victims are admitted to the unit with hemorrhagic shock, a total of 560 patients were requested, and a 2-yr study was therefore conducted.

Data are expressed either as mean ± SD or as frequency with 95% confidence interval (CI). Comparisons between groups were performed using the chi-square test for proportions and the Student *t* test (two sided) for normally distributed variables after a check for normality by a Shapiro-Wilk test. The two tests were corrected for multiple comparisons by the Bonferroni correction if needed.

The probability of survival was calculated using the Trauma and Injury Severity Score methodology.[8] To compare the current population with that of the Major Trauma Outcome Study (MTOS), the observed survival was compared to the expected survival by calculating the M, W, and Z scores. A value of $M < 0.88$ indicated a disparity in the severity match between the study group and the MTOS group. The W score was the percentage of survivors more (or less) than would be expected from the MTOS prediction. A Z score between −1.96 and +1.96 indicated no significant difference between the actual and expected number of survivors. Moreover, a logistic regression was performed to determine the precise factors associated with mortality in this group of blunt trauma patients. The following factors were entered in the model and successively deleted in a stepwise manner: age, Revised Trauma Score, Injury Severity Score, Simplified Acute Physiology Score, troponin I release (yes/no), and presence of shock (yes/no). The level of significance chosen was $P < 0.05$.

Results

During the study period, 728 trauma patients (male/female, 532/196; 73%/27%) were admitted at the unit. These young patients (aged 37 ± 19 yr) suffered a significant trauma (Injury Severity Score, 28 ± 18) with physiopathologic consequences (Revised Trauma Score, 8.3 ± 3.3; Simplified Acute Physiology Score, 28 ± 17). The value of the Injury Severity Score was equal or lower than 10 in only 56 patients (8%; 95% CI, 6–10%). Brain and chest injuries were noted in 240 (33%; 95% CI, 30–37%) and 169 patients (23%; 95% CI, 20–27%), respectively. An abnormal electrocardiogram leading to the diagnosis of significant myocardial contusion was found in 35 patients (4.8%; 95% CI, 3.4–6.6%). The observed electrocardiographic abnormalities were as follows: ST segment depression or elevation greater than 1 mm, 19; right bundle branch block, 10; ventricular dysrhythmia, 8; inverted T wave in more than two leads, 8; Q-wave formation, 2; anterior left bundle branch block, 2; auriculoventricular block, 1. Twenty-three of the 35 patients with myocardial contusion underwent emergency surgery. Catecholamine infusion was more frequently needed in these 23 patients during the early phase as compared with the remaining 12 patients (91% *vs.* 58%; $P < 0.05$). For the whole group, shock was observed in 149 (20%; 95% CI, 18–24%) patients. Ventricular dysrhythmias or conductive abnormalities never justified a specific treatment. For the whole group, mortality was 17% (95% CI; 15–21%). The number of late survivors (n = 598; 88%; 95% CI, 85–90%) was similar to that expected (n = 590; 87%). The W score was +1.1% ($Z = -1.3$). The value of $M = 0.86$ indicated a slight disparity in the severity match between the study group and the MTOS group. The disparity was due to a higher proportion of patients with a rather low probability of survival (Trauma and Injury Severity Score < 0.75, 18% *vs.* 8%). These latter patients experienced a slightly higher number of survivors than that expected by MTOS ($W = +4.1\%$, $Z = -2.1$; $P < 0.05$).

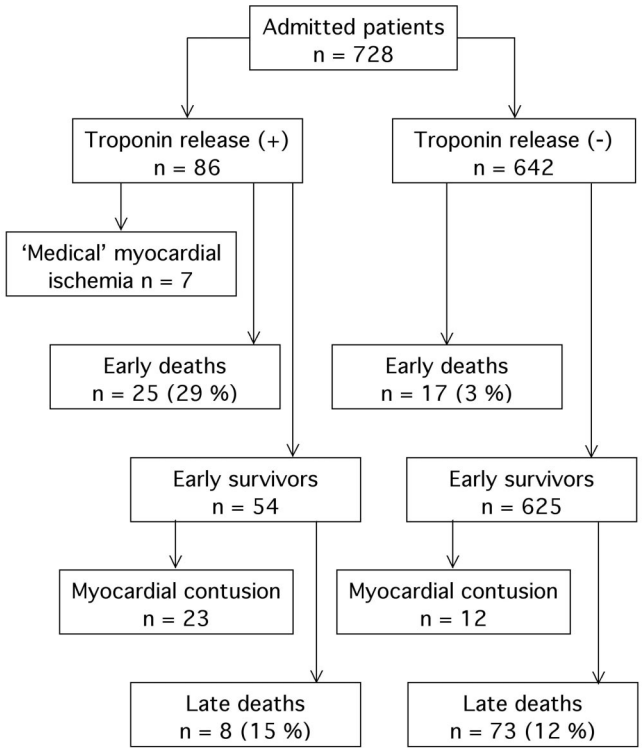

Fig. 1. Trial profile of the 728 patients according to the existence of troponin I release, electrocardiographic diagnosis of blunt cardiac injury (or myocardial contusion), and outcome (early death, during the first 48 h after trauma).

Troponin I release was observed in 86 patients (fig. 1). This release was attributed to "medical" myocardial ischemia in 7 patients (chronic coronary artery disease, 4; cardiac arrest, 3). Twenty-five of these 86 patients were early nonsurvivors (brain death, 14; exsanguination, 9; early multiple organ failure, 2). Seventeen early nonsurvivors were noted among the 642 patients without troponin I release; therefore, early death occurred in 42 admitted patients (5.8%; 95% CI, 4.2–7.7%; fig. 1). Finally, troponin I release was observed in 54 early survi-

Table 1. Characteristics of the 54 Early Survivors with Posttraumatic Release of Troponin I in the Absence of "Medical" Myocardial Ischemia

	Very Transient and Limited Troponin I Release	Transient and Significant Troponin I Release	Prolonged and Significant Troponin I Release
n	22	15	17
ISS	32 ± 11	35 ± 14	31 ± 11
SAPS II	35 ± 15	47 ± 23	30 ± 17
Brain injury	8 (36%)	12 (80%)*	9 (53%)
Chest injury	13 (59%)	8 (53%)	14 (82%)*
Myocardial contusion	3 (13%)	2 (13%)	17 (100%)*
Shock	17 (77%)	12 (80%)	11 (65%)
Peak troponin, μg/l	1.0 ± 0.3	5.7 ± 6.7	14.2 ± 19.1*
Peak lactate, mM	4.2 ± 4.6	6.8 ± 4.5*	3.8 ± 2.6
Mortality	4 (18%)	4 (27%)	0

* $P < 0.05$ *vs.* the two other groups of patients.

ISS = Injury Severity Score; SAPS II = Simplified Acute Physiological Score.

vors without preexisting coronary artery disease (7%; 95% CI, 5.6–9.6%). Brain injury (56% *vs.* 29%; $P < 0.05$), chest injury (67% *vs.* 19%; $P < 0.05$), blunt cardiac trauma (40% *vs.* 2%; $P < 0.05$), and shock (78% *vs.* 17%; $P < 0.05$) were more frequent in these patients than in the 625 early survivors without troponin I release. The logistic regression demonstrated a significant effect of high age ($P < 0.0001$), low Revised Trauma Score ($P < 0.0001$), and presence of shock at admission ($P = 0.012$) on mortality and a trend toward an effect of high Simplified Acute Physiology Score ($P = 0.0677$). Neither Injury Severity Score nor release of troponin I showed any significant effect on the survival rate. Mortality was similar in early survivors with (15%; 95% CI, 7–27%) and without (12%; 95% CI, 9–14%) troponin I release (fig. 1). The odds ratio for late mortality was 1.32 (95% CI, 0.61–2.85%) in patients with troponin I release.

The breakdown of the 54 patients by profile of troponin I release is depicted in table 1. A very transient and limited release of troponin I was observed in 22 hemodynamically unstable patients (3% of the whole group; 95% CI, 1.9–4.5%) (shock, 77%; blunt cardiac trauma, 13%). The high incidence of brain injury and shock was the main feature of the 15 patients with significant but transient troponin I release (2.0% of the whole group; 95% CI, 1.2–3.4%). Finally, sustained and significant troponin I release occurred in 17 patients (2.3% of the whole group; 95% CI, 1.4–3.7%) frequently associated with chest trauma and constantly associated with electrocardiographic abnormalities (table 1). Echocardiographic abnormalities were found in 9 (53%) out of these 17 patients (regional wall motion abnormality, 8; pericardial effusion, 2). Sensitivity, specificity, positive predictive value, and negative predictive value of troponin I release over time for the diagnosis of a significant myocardial contusion were 63, 98, 40, and 98%, respectively, on the basis of electrocardiographic abnormalities.

Coronary angiography was not undertaken in 2 of the 17 patients with prolonged release of the marker (table 2) because of renal failure (patient 10) or ongoing sepsis (patient 12). Coronary angiography was usually performed at the end of the first posttraumatic week (range, 2–18 days). A coronary artery injury was found in 7 patients (1% of the whole group; 95% CI, 0.4–2.0%; 47% of angiographies; 95% CI, 21.2–73.4%). Two patients had a major vascular injury: rupture of a distal branch of a dominant right coronary in a pseudoaneurysm of the left ventricular diaphragmatic wall (patient 1; fig. 2) or prox-

Table 2. Characteristics of the 17 Patients with Significant and Sustained Troponin Release

	Sex	Age, yr	Mechanism of Trauma	ISS	SAPS	Shock	Peak Troponin I, μg/l	Norepinephrine Infusion	Emergency Surgery	Coronary Angiography Results (Delay, days)	ICU LOS, days
1	M	24	Motorcycle	29	83	Yes	74.8	Yes	No	Major (6)	23
2	M	44	Pedestrian	16	35	Yes	41.4	Yes	Yes (L + O)	Major (3)	19
3	M	35	Motor vehicle	25	25	No	2.2	Yes	No	Normal (15)	18
4	M	37	Fall	27	15	No	10.4	No	Yes (L)	Minor (4)	7
5	F	23	Motor vehicle	43	41	Yes	3.7	Yes	Yes (L)	Normal (4)	58
6	F	21	Motorcycle	50	37	Yes	11.4	Yes	Yes (L)	Normal (17)	22
7	F	24	Fall	43	21	No	2.2	No	No	Normal (18)	17
8	F	20	Pedestrian	9	8	No	2.4	No	No	Normal (2)	8
9	M	66	Motor vehicle	16	22	No	5.0	No	No	Normal (3)	3
10	M	47	Motorcycle	41	42	Yes	8.2	Yes	Yes (L + O)	NA	40
11	M	27	Motorcycle	34	24	Yes	34.5	Yes	Yes (L)	Minor (15)	10
12	M	18	Motor vehicle	48	26	Yes	9.7	Yes	Yes (O)	NA	8
13	F	19	Motor vehicle	24	20	No	3.8	Yes	Yes (O)	Normal (4)	24
14	M	22	Motor vehicle	34	28	Yes	13.2	Yes	Yes (O)	Minor (8)	13
15	M	23	Fall	34	20	Yes	4.6	Yes	Yes (O)	Normal (16)	27
16	M	26	Motor vehicle	29	20	Yes	7.3	Yes	Yes (L)	Minor (8)	16
17	M	38	Motor vehicle	29	35	Yes	8.7	No	No	Minor (8)	9

Major and *minor* refer to the vascular injury.

ICU = intensive care unit; ISS = Injury Severity Score; L = laparotomy; LOS = length of stay; NA = not available; O = orthopedic surgery; SAPS II = Simplified Acute Physiological Score.

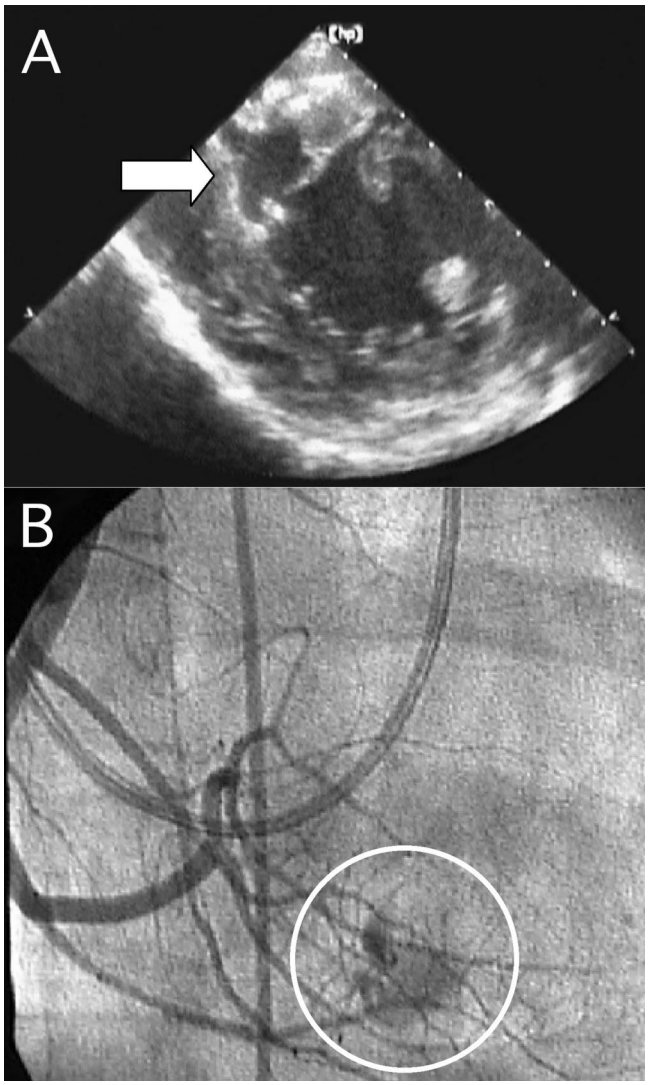

Fig. 2. Patient 1. (*A*) Echographic view of the pseudoaneurysm of the left ventricular diaphragmatic wall. (*B*) Rupture of a distal branch of a dominant right coronary in the pseudoaneurysm.

imal dissection of the left anterior descending coronary artery with spontaneous reperfusion and anteroapical left ventricular aneurysm (patient 2; fig. 3). Five patients had only minor vascular injuries: distal dissection of the left anterior descending coronary artery (patient 14) or occlusion of distal left obtuse marginal branches arising from the circumflex artery (patients 4, 11, 16, and 17). Regional left ventricular wall motion abnormalities were found in the 2 patients with major vascular injury (patients 1 and 2), in 4 of the 5 patients with minor vascular injury (patients 4, 11, 14, and 16), in 1 patient with normal coronary angiography (patient 8), and in 1 of the 2 patients in whom angiography could not be performed (patient 12). The 7 patients with abnormal coronary angiography were given β-adrenergic blocker and salicylate as soon as possible. Surgical treatment of ventricular aneurysm was needed at the 47th posttraumatic day

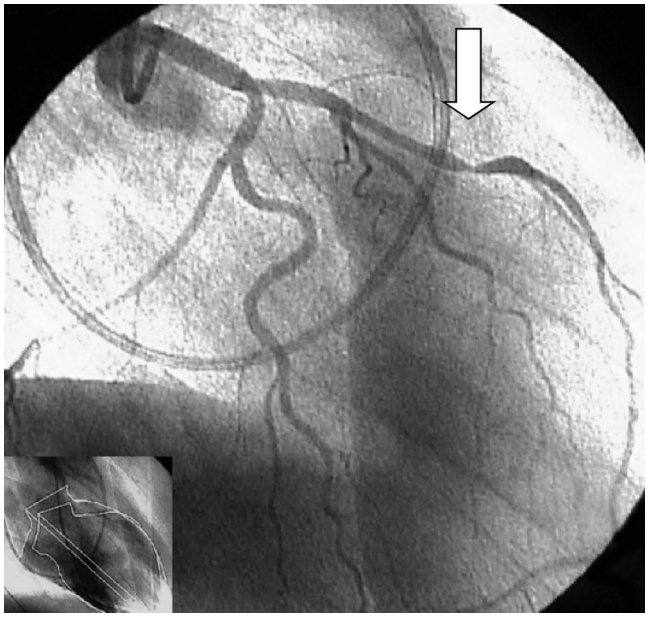

Fig. 3. Patient 2. Proximal dissection of the left anterior descending coronary artery with spontaneous reperfusion and anteroapical left ventricular aneurysm (ventricular cineangiogram in the *left lower part* of the figure showing the anterior akinesia).

(patient 1). All of the patients with significant and sustained release of troponin I left the unit alive.

Discussion

The current prospective study demonstrates that troponin I release is frequent in severe trauma patients and may occur in the absence of a significant direct myocardial contusion. The time course of the marker may predict cardiac anatomical injuries. In contrast with cardiac, noncardiac critically ill, and septic patients, troponin I release does not have a prognosis value in severe trauma patients.

Release of troponin I frequently occurred during the hyperadrenergic states that induced myocardial contraction bands,[26] such as nontraumatic subarachnoid hemorrhage,[27] brain death,[28] septic shock,[4–6] or hemorrhagic shock in the absence of direct cardiac trauma.[7] This mechanism probably explains the results of large prospective studies and the current results reporting a questionable value of troponin I in depicting myocardial contusion with a positive predictive value between 21 and 75%, unlike earlier reports.[29,30] A 12% overall incidence of troponin I release was observed in our series. Posttraumatic electrocardiographic abnormalities in the absence of previous coronary artery disease remain the reference for the diagnosis of myocardial contusion and the best predictor of subsequent cardiovascular complications.[31] In the emergency room, electrocardiographic recording is easy to perform, in contrast with an appropriate echocardiography. Furthermore, segmental wall

motion abnormalities and valvular dysfunction may be observed in the absence of myocardial contusion in severe trauma patients with hypovolemia,[32,33] increase in intrathoracic pressure,[34] or subarachnoid hemorrhage.[35,36] The current study suggests a 5% incidence of a significant myocardial contusion revealed by the electrocardiogram in severe trauma patients and underlines the fact that cardiac injury may be observed with or without chest trauma[37,38] and with or without troponin I release.[22,30] The absence of circulating marker may be related to the limited amount of tissue necrosis, *e.g.*, prominent involvement of the right ventricular wall,[39] timing of blood sampling, hemodilutional phenomenon. Because myocardial contusion may induce significant cardiovascular complications as suggested by the current need for catecholamines during emergency surgery,[40,41] this diagnosis must be clearly established and must justify the association of repeated electrocardiogram recordings and circulating troponin I detection.[3,30,42–44]

Serial blood sampling for troponin I measurement provides an estimate of the amount of necrosed myocardial tissue during experimental and clinical ischemia[45,46] and during experimental traumatic cardiac injury.[47] Increased cardiac troponin I also predicts the severity of the coronary artery lesions during unstable angina.[48] Using an adequate threshold (5 times the upper limit of normal value)[18] and a reasonable duration of troponin I release measurement,[19] the current results demonstrate that the time course of circulating troponin I may predict coronary vascular injuries whose incidence seems to be at least 1% in severe trauma patients. Coronary artery injuries have been involved in posttraumatic cardiac dysfunction[49,50] and could be implicated in some rare delayed cardiac complications of myocardial contusion.[51] As suggested by the current observations, both left and right coronary arteries may be concerned with various lesions (dissection, stenosis, thrombosis, compression, aneurysm, and others) associated with major cardiac muscle injuries (dyskinesia, pseudoaneurysm, rupture).[39] Interestingly, posttraumatic release of troponin I does not have the same prognostic value as that after acute coronary syndrome[52,53] or septic shock,[54] because mortality was similar in early survivors with or without circulating marker. A previously normal coronary bed probably explains the favorable short- and long-term outcomes after blunt cardiac injury.[19]

The results of this study concern severe trauma patients exclusively. Because coronary artery injury may be more frequent than suggested by numerous anecdotical reports, serial electrocardiographic recordings and troponin I assessments may be recommended for initial screening in these high-risk trauma patients.[30,44] The second limit of this study was the absence of long-term surveying of the patients, especially those with significant and transient troponin I release. Long-term cardiac complications cannot be excluded, thus justifying additional early investigations and prolonged follow-up. Because coronary angiography does not seem appropriate, noninvasive cardiac imaging, such as specific use of multislice spiral computerized tomography,[55] must be developed to compensate for the limits of the current study.

The authors thank Jean-Xavier Mazoit, M.D., Ph.D. (Service d'Anesthésie-Réanimation, Hôpital de Bicêtre, Le Kremlin Bicêtre, France), for his friendly and efficient statistical advice.

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