# METHODS

# Experimental Model of Heart Contusion

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The proposed experimental model reproduces the mechanism of isolated contusion of the heart resultant from the chest stroke against the steering wheel prop in a car accident. The conditions of experiment rule out the additional pathogenetic factors occurring in clinical practice (extracardiac injuries, alcohol intoxication, blood loss, pain, *etc.*) and hence, it is possible to study the mechanisms of developing myocardial dysfunction proper and its contribution to the course and outcome of the posttraumatic period. Clinical, hemodynamic, and pathomorphological equivalents of this kind of blunt injury of the heart were obtained.

**Key Words:** *myocardial contusion; experimental simulation*

Contusion of the heart (myocardial contusion) can be a component of multiple trauma and an isolated injury. The contribution of myocardial contusion to mortality, diagnostic and prognostic criteria of this type of heart injuries remains an object of discussions [4]. The most likely explanation of this fact is that heart contusion in the patients is rarely an isolated injury, while in multiple trauma it is paralleled by other thoracic and extrathoracic injuries and hence, pain and blood loss. In addition, the underlying diseases and alcoholic intoxication are also essential circumstances. All these factors, no doubt, impede the definition of precise diagnostic criteria and evaluation of the contribution of myocardial contusion to the score of the injury severity.

The conditions of experiment allow minimization of the additional factors, largely determining the clinical course, severity of clinical status, and mortality in multiple injury. The pain afferentation is ruled out under conditions of an experiment; there is no blood loss and alcoholic intoxication; the underlying diseases (including cardiac) can also be ruled out with a high probability. Histological study, the "golden" diagnostic standard of myocardial contusion, is possible under experimental conditions, which is particularly important, because due to this study other myocardial injuries occurring in blunt cardiac injury (concussion, rupture of the myocardium, traumatic myocardial infarction, traumatic myocardiodystrophy) can be ruled out and possible injuries to the pericardium, valvular system, and papillary muscles can be detected.

## MATERIALS AND METHODS

A chest stroke against the wheel prop in car accidents is the most frequent mechanism of heart contusion [4]. The model proposed in this paper reproduces the mechanism of contusion resultant from the chest stroke against the steering wheel prop in a car accident. A mobile mechanism with a vertically fixed rat is set to motion by a free-falling load. The trajectory of the mobile mechanism's movement is blocked with a barrier with a fragment protruding at the level of heart projection on the chest. The experimental animal hits the barrier with its heart projection area. As a result of

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the collision, the falling of the load ceases, the chest is compressed in the anteroposterior direction, and when the system is equilibrated, the chest compression process is over. The anterior chest remains in a state of compression of permanent force during several fractures of a second or several seconds, depending on the elastic properties of the chest. The thread is broken. After hundredth fractures of a second the chest resistance starts to surpass the stroke force, and due to elastic characteristics of the chest, the mobile mechanism slides back at a certain velocity. The chest acquires the initial sagittal size, the thoracic organs return to the initial position. This model allows coordination between the level of the protruding fragment of the barrier and the heart projection area, precise dosing of the force of stroke and hence, the severity of trauma. The priority is protected by a useful model patent [1].

Experiments were carried out on 60 outbred male albino rats (250-300 g) narcotized with sodium thiopental (Sintez Company; 60 mg/kg intraperitoneally). Respiration rate (RR), heart rate, blood pressure in the left carotid artery (AP; by the direct method), ECG in three standard leads, integral rheogram, and first derivative of differential rheogram were recorded before simulation of myocardial contusion and during the first hour of the posttraumatic period. The following parameters were calculated: stroke volume (SV), cardiac output (CO), and total peripheral vascular resistance (TPVR).

For morphologic studies under a light microscope, the hearts were fixed in neutral buffered formalin, dehydrated in ethanol, and embedded in paraffin by routine methods. Histotopographic sections of the ventricles were stained with hematoxylin and eosin for examination in transmitting and polarized light. In order to detect early ischemic lesions in cardiomyocytes, the preparations were stained with hematoxylin, basic fuchsin, and picric acid (HBPF) as described previously [5].

## RESULTS

Blunt trauma of the chest caused changes in the respiration rate, rhythmic and contractile functions of the heart, and in the central hemodynamics in general.

A significant but transient deceleration of heart rhythm (to 65.55% of initial value) was recorded in 100% cases directly after the injury. It was paralleled by deceleration of respiration rate and even its complete arrest in some cases. Sinus bradycardia was the only ECG deviation of the early posttraumatic period in 6.66% cases, in other cases (93.3%) it was paralleled by automatism, excitation, and conduction disorders (Table 1).

The central hemodynamic parameters also changed significantly during the early posttraumatic period (Table 2). The leading hemodynamic shift was arterial hypotension, persisting throughout the entire period of observation. Cardiac output also decreased significantly, first at the expense of pronounced bradycardia and stroke volume reduction, and then, after partial recovery of heart rate, at the expense of the stroke volume value.

Myocardial structure in controls corresponded to the known morphology of this condition (according to optic microscopy) [2]. The majority of cardiomyocytes had regular transverse striation in polarized light.

**TABLE 1.** Incidence of ECG Deviations (% of Animals) Recorded during the First Hour of Posttraumatic Period Following Experimental Contusion of the Heart

| ECG deviation | Sinus bradycardia | Sinus arrhythmia | Voltage reduction | SES | CES | PVT |
|---|---|---|---|---|---|---|
| Incidence | 100 | 10 | 13.33 | 38.3 | 18.3 | 1.66 |
| ECG deviations | Heterotopic rhythms | | | | | |
| AR | MNR | INR | SPMM | IVR | AVD | |
| Incidence | 41.66 | 11.66 | 1.66 | 16.66 | 13.33 | 1.66 |
| ECG deviations | IACD and IVCD | AV block | | | *ST* rise | *ST* rise |
| | | first degree | second degree | third degree | | |
| *ST* depression | | | | | | |
| Incidence | by 8.33 | 25 | 1.66 | 8.33 | 38.3 | 3.3 |

**Note.** SES: supraventricular extrasystoles; VES: ventricular extrasystoles; PVT: paroxysmal ventricular tachicardia; AR: atrial rhythms; MNR: mean nodular rhythm; INR: inferior nodular rhythm; SPMM: supraventricular pacemaker migration; IVR: idioventricular rhythm; AVD: atrioventricular dissociation; IACD: intra-atrial conduction disorders; IVCD: intraventricular conduction disorders.

Staining (HBPF) showed solitary cardiomyocytes with diffusely fuchsinophilic cytoplasm.

One hour after the trauma, numerous ruptures, stratification of structural elements of cardiac membranes, focal and punctate hemorrhages (mainly in the epicardial and subepicardial zones) were macroscopically seen.

Examination of panoramic and semithin preparations showed erythrocyte aggregations in the form of "coin bars" and clots in the microvessels, mainly in venules. The arteriolar lumen was as a rule empty and its profile was oval or slightly pleated. The layers of arteriolar walls were well discernible. The interstitium was edematous (Fig. 1). Myofibrils in the subendocardial portion of the myocardium were located far from each other and the space between the fibrils looked empty. Foci of myocardial muscle fibers dissociation and relaxation (Fig. 2, *a*), accumulations of erythrocytes and lymphocytes (Fig. 2, *b*) were detected.

Examination in polarized light showed cardiomyocytes with more intense A-disc anisotropy and shortening of I-discs. Sometimes cells with signs of second degree segmentary contracture formed complexes from several neighboring cardiomyocytes. First

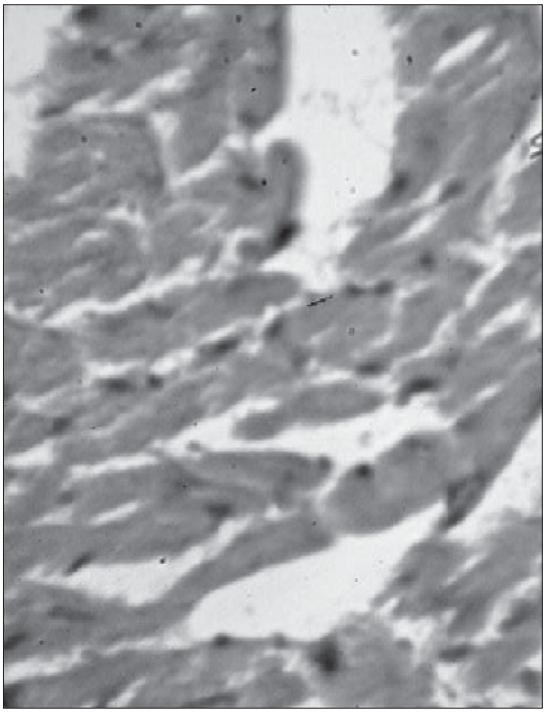

**Fig. 1.** Myocardial edema in the myocardial contusion zone. Hematoxylin and eosin staining, ×220.

**TABLE 2.** Parameters of Central Hemodynamic during Posttraumatic Period after Experimental Myocardial Contusion (*M*±*m*)

| Posttraumatic period, min | | Heart rate, $min^{-1}$ | SV, μl | CO, ml/min | AP, mm Hg | TPVR, $10^3$din×sec×$cm^{-5}$ |
|---|---|---|---|---|---|---|
| Initially | group I | 377±3.7 | 115±0.8 | 43±0.4 | 125±1.7 | 230±3.9 |
| | group II | 374±2.3 | 115±1.11 | 43±0.95 | 125±1.22 | 232±1.61 |
| 3 | group I | 375±2.4 | 115±0.8 | 43±0.4 | 125±1.7 | 230±3.9 |
| | subgroup IIa | 228±2.5*+ | 89±1.72*+ | 20±0.43*+ | 67±1.38*+ | 263±2.21*+ |
| | subgroup IIb | 246±3.1*+ | 92±1.69*+ | 23±0.65*+ | 84±1.33*+ | 99±2.62*+ |
| 10 | group I | 376±2.9 | 115±0.6 | 43±0.4 | 124±1.6 | 230±4.3 |
| | group II | 307±10.1*+ | 94±0.62*+ | 29±1.39*+ | 78±1.41*+ | 214±2.56*+ |
| 20 | group I | 377±2.6 | 115±0.2 | 43±0.3 | 123±1.6 | 227±3.6 |
| | group II | 333±3.8*+ | 96±0.5*+ | 31±1.37*+ | 79±1.41*+ | 198±5.0*+ |
| 30 | group I | 379±3.1 | 116±0.5 | 44±0.4 | 122±0.9 | 223±2.4 |
| | group II | 343±6.6*+ | 96±0.45*+ | 33±1.02*+ | 81±1.46*+ | 196±4.65*+ |
| 40 | group I | 375±3.1 | 115±0.5 | 43±0.4 | 124±1.3 | 231±2.8 |
| | group II | 335±4.5*+ | 98±0.82*+ | 33±1.05*+ | 85±1.25*+ | 206±3.57*+ |
| 50 | group I | 371±2.5 | 116±0.5 | 43±0.3 | 123±1.4 | 230±2.7 |
| | group II | 334±3.5*+ | 98±0.7*+ | 33±1.1*+ | 86±1.18*+ | 210±3.11*+ |
| 60 | group I | 376±5.2 | 116±0.5 | 44±0.2 | 124±1.5 | 227±2.9 |
| | group II | 335±2.6*+ | 98±2.4*+ | 33±0.92*+ | 86±2.02*+ | 210±3.26*+ |

**Note.** Group I: control (intact); group II: animals with trauma; IIa and IIb: subgroups with different degree of AP reduction after injury. $p<0.001$ *compared to initial values in the same group (subgroup), +between groups I and II during the same period of observation.

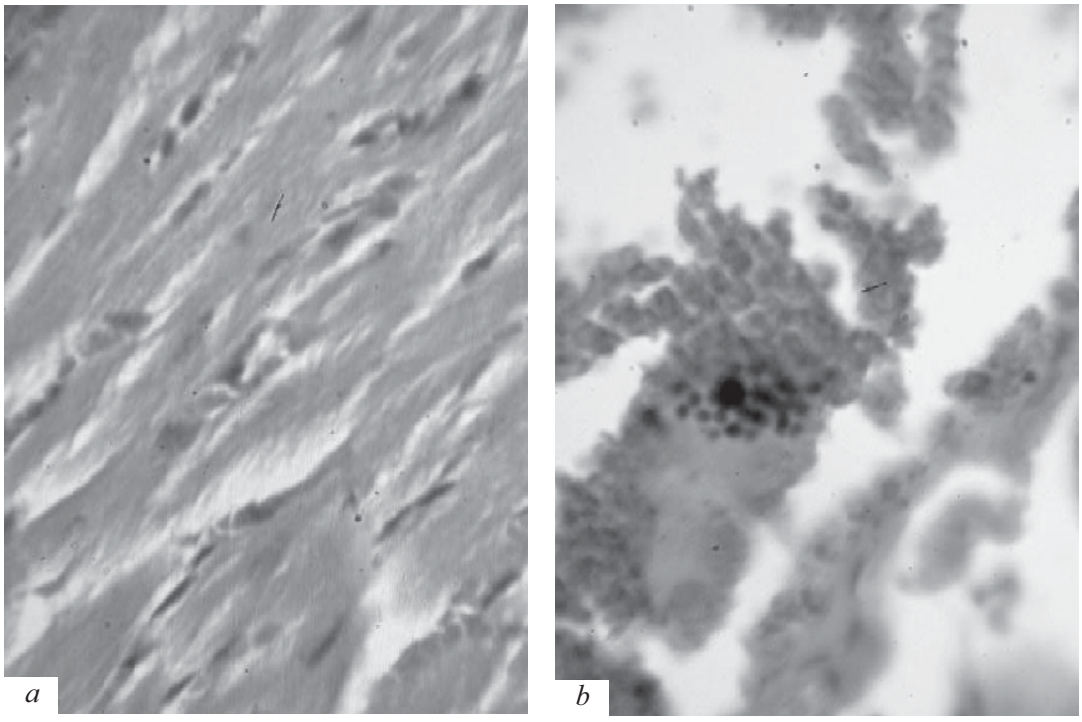


**Fig. 2.** Pathomorphology of heart contusion zone. Hematoxylin and eosin staining, ×220. *a*) erythrocyte diapedesis and destruction in the heart contusion zone; *b*) accumulation of erythrocytes and lymphocytes in surface layers of the myocardium in the heart contusion zone.

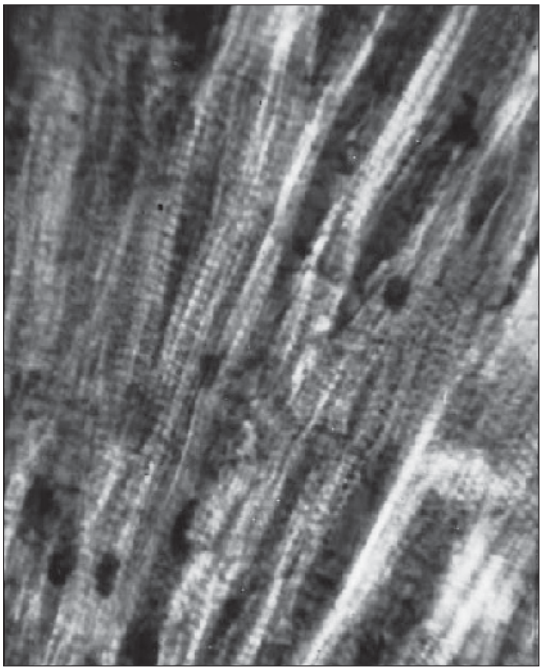

**Fig. 3.** Segmentary contracture, first degree. Polarized light, ×336.

degree segmentary contractures acquired a focal pattern (Fig. 3). Myocytolysis, vacuolation, and lumpy degradation of cardiomyocytes were observed.

Hence, degenerative changes in capillary endothelium and disorders in the blood aggregation developed by the end of the first hour of the posttraumatic period in the myocardium of experimental animals. Augmenting interstitial edema was paralleled by deformation of the microvessels. Myofibril lesions (segmentary contractures, lumpy degradation, and myocytolysis) developed in cardiomyocytes. This pathomorphological picture corresponded to that described for heart contusion [3], and hence, our experimental model can be considered adequate.

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