

## PULMONARY CONTUSION – HISTOLOGICAL FINDINGS: SELF-EXPERIENCE EXPERIMENTAL STUDY

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### ABSTRACT.

**Background.** Pulmonary contusion is a common finding after blunt chest trauma.

**The aim** of this experimental study is to present the histological findings in pulmonary contusion as a result of blunt chest trauma.

**Material and Methods.** Twenty male Wistar albino rats were used in the study. The animals were divided into four groups, according to the kinetic energy of blunt chest trauma: 2,0 J, 2,2 J, 2,4 J and 2,6 J. The animals were autopsied at 24<sup>th</sup> hour after blunt chest trauma. Histopathological findings were examined and scored. Correlation between histopathological findings and energy of chest trauma was performed

**Results.** At the 24<sup>th</sup> hour post-contusion pulmonary tissue showed diffuse areas of parenchymal hemorrhage, alveolar disruption and alveolar atelectasis. Severity of histological lung contusional changes was in significant correlation with the energy of blunt chest trauma.

**Conclusion.** This experimental study presents the histological findings in pulmonary contusion as a result of blunt chest trauma.

**Key words:** pulmonary contusion, blunt chest trauma, histological findings.

### Introduction.

Pulmonary contusion is the most common intrathoracic injury in blunt chest trauma. Despite the development of respiratory therapy for the last two decades the mortality of pulmonary contusion still remains in a very high level (1,2,3). This fact can explain the interest in experimental study of this serious problem in thoracic surgery clinical practice.

**The aim** of this experimental study is to present the histological findings in pulmonary contusion as a result of blunt chest trauma.

### Materials and Methods.

Twenty male Wistar albino rats (body weight – 310,0 ±20,0 ) were used in the study. The animals were divided into four groups, according to the kinetic energy of blunt chest trauma: 2,0 J, 2,2 J, 2,4 J and 2,6 J. A special platform unique for this experimental study was designed and produced in the mechanical engineering laboratory of our university. Pulmonary contusion in blunt chest trauma was induced by dropping a weight of 500 g the left hemithorax of the animals. The mechanical impact on the chest wall of the animals with energy of 2,0 J, 2,2 J, 2,4 J and 2,6 J was induced by dropping the falling body of the platform from a height of 41, 45, 49 and 53 cm.

After general anesthesia, the animals were autopsied at 24<sup>th</sup> hour after blunt chest trauma. The lung was dissected and lung tissues were fixed in 10 % formaldehyde solution. Tissue samples were embedded in paraffin blocks and cut into five micron slices. Samples were stained with hematoxylin-eosin and evaluated by a light microscopy. In every sample, microscopical fields were examined and lung pathological findings were determined.

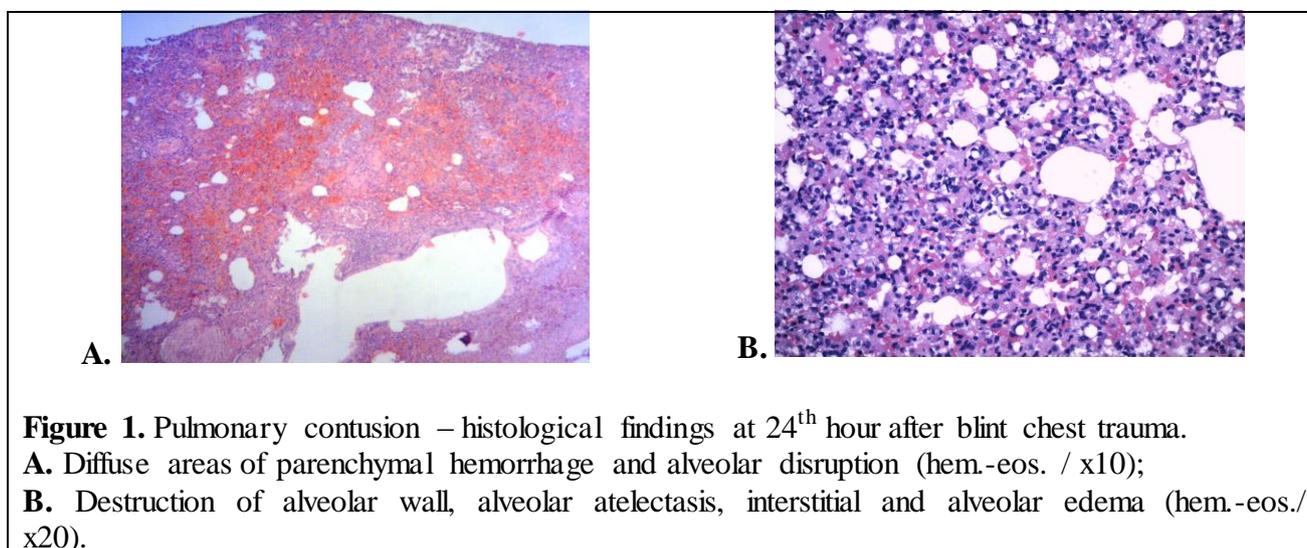
Histopathological findings were examined and scored by two experienced laboratory pathologists. According to our scoring system, histological findings were scored as: 0 – no changes,

1- mild, 2 – moderate; 3 – severe. Data are expressed as mean and standard deviation. Correlation between histopathological findings and energy of chest trauma was performed (Pearson correlation).

This study was performed with the permission of our university local ethic committee and Bulgarian food safety agency.

### Results.

Histopathological sings of pulmonary contusion were observed in all trauma groups of animals. At the 24<sup>th</sup> hour post-contusion pulmonary tissue showed diffuse areas of parenchymal hemorrhage (fig. 1A). The alveoli in affected lung areas were filled with blood. There was also hemorrhage into the interstitial space. Destruction of alveolar wall and alveolar atelectasis in contusional areas of the lung was established (fig. 1B). Because of parenchymal hemorrhage, alveolar disruption and alveolar atelectasis the architecture of the lung in affected areas was entirely obliterated at the 24<sup>th</sup> hour after blunt chest trauma (fig. 1A).

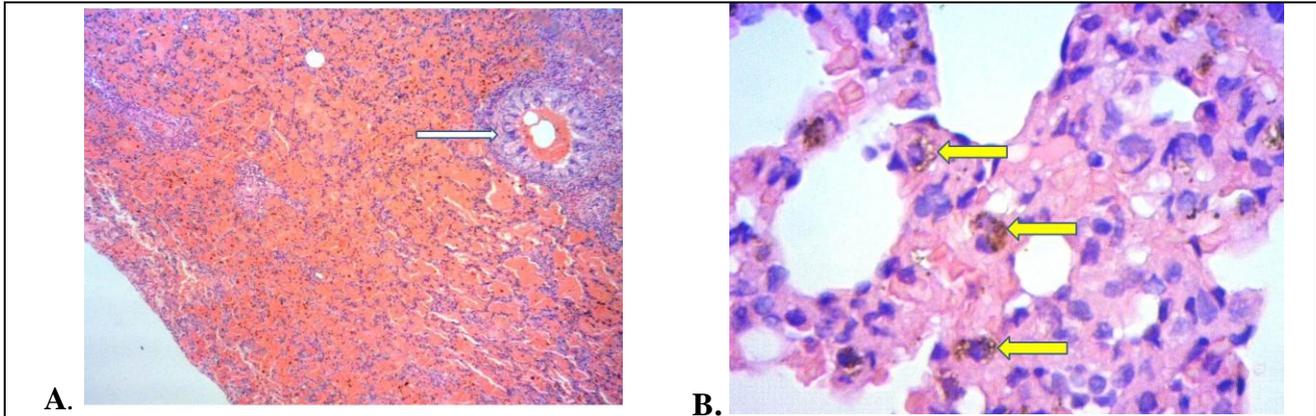


**Figure 1.** Pulmonary contusion – histological findings at 24<sup>th</sup> hour after blint chest trauma.  
**A.** Diffuse areas of parenchymal hemorrhage and alveolar disruption (hem.-eos. / x10);  
**B.** Destruction of alveolar wall, alveolar atelectasis, interstitial and alveolar edema (hem.-eos./ x20).

Damage of the terminal bronchioles in affected lung areas was established. Disruptions of the bronchial walls and bronchial hemorrhages (into the lumen of bronchioles and in their walls) were seen (fig. 2A).

At the 24<sup>th</sup> hour after blunt chest trauma infiltration of polymorphonuclear and mononuclear cells in the injured lung areas was established. Hemosiderin was accumulated in areas of pulmonary contusion. Granules of hemosiderin were found in cytoplasm of lung macrophages - hemosiderophages (2B).

Mean severity scores of the histopathological sings of pulmonary contusion were compared. The results are shown in table 1. It was established that severity of histological lung contusional changes was in significant correlation with the energy of blunt chest trauma ( $p < 0,05$ ).



**Figure 2.** Pulmonary contusion – histological findings at 24<sup>th</sup> hour after blunt chest trauma. **A.** Diffuse areas of parenchymal hemorrhage. Arrow points hemorrhage in the wall of terminal bronchiolus (hem.-eos. / x10); **B.** Infiltration of polymorphonuclear and mononuclear cells in the injured lung tissue. Yellow arrows point macrophages with granules of hemosiderin into their cytoplasm (hem.-eos./ x40).

Severity of pulmonary contusion according to the energy of blunt chest trauma – histological evaluation [data are presented as mean and Standard Deviation- (SD)]. **Table 1.**

Lung histological findings	Energy of blunt chest trauma			
	2,0 J	2,2 J	2,4 J	2,6 J
pulmonary hemorrhage	1,33 (0,52)	1,83 (0,41)	2,50 (0,55)	2,83 (0,41)
alveolar disruption	1,50 (0,54)	2,16 (0,41)	2,50 (0,54)	3,00 (0)
bronchiolar damage	1,16 (0,41)	1,50 (0,54)	2,16 (0,41)	2,83 (0,41)

**Discussion.**

Pulmonary contusion is a common finding after blunt chest trauma. Despite the development of respiratory therapy for the last two decades the mortality of pulmonary contusion still remains in a very high level (1,2). This fact can explain our interest as clinicians and scientific workers in experimental study of this serious problem in thoracic surgery clinical practice.

Pulmonary contusion is an acute parenchymal injury resulting in hemorrhage (alveolar and interstitial), alveolar disruption, alveolar atelectasis, interstitial and alveolar edema (3,4,5, 8-10). We had established all of these parenchymal lung histological findings at the 24<sup>th</sup> hour after blunt chest trauma in our experimental study. These parenchymal lung injuries, demonstrated in our study, may lead to pathophysiologic changes, the severity of which depends on the extension of pulmonary contusion. At this point, pulmonary contusion may lead to acute respiratory failure because of ventilation/perfusion mismatch, elevations in intrapulmonary shunt, increases in lung fluid and loss of lung compliance (6,7).

We had determined the correlation between severity of pulmonary contusion and kinetic energy of blunt chest trauma on the base of expression of histological lung injuries. As we had expected, it was significant correlation between severity of pulmonary contusion and energy of mechanical impact on the chest of the animals. This result will help us to choose the energy of blunt chest trauma in other future experimental studies dedicated to pulmonary contusion.

The lung of the laboratory animals (rats – in our study) is too vulnerable at the time of autopsy and lung dissection. At this point, iatrogenic damage of the lung may be caused at the time of its dissection (3,9,10). This iatrogenic damage may result in parenchymal hemorrhage and alveolar disruption – histological findings of pulmonary contusion. Hemosiderin is an iron-storage complex that is found in macrophages and is especially abundant in situations following hemorrhages (5). Producing and accumulation of hemosiderin in macrophages occurs at 18<sup>th</sup> to 24<sup>th</sup> after tissue hemorrhages. This fact can explain why we had chosen 24<sup>th</sup> hour after blunt chest trauma as a term of autopsy of the animals. We had demonstrated granules of hemosiderin in cytoplasm of lung macrophages. This histological finding demonstrates that parenchymal lung hemorrhage was caused by blunt chest trauma and wasn't result of iatrogenic damage. At this point, this fact confirms that using our platform of blunt chest trauma we had created a reproducible model of pulmonary contusion in small laboratory animals. This model will give us opportunities for future studying the different aspects of this common thoracic injury in blunt chest trauma.

### Conclusion.

This experimental study presents the histological findings in pulmonary contusion as a result of blunt chest trauma. Lung histological findings have confirmed that we had created a reproducible model of pulmonary contusion in small laboratory animals. Using this model of pulmonary contusion our next step in experimental work will be the progression of postcontusional lung changes.

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