The Macklin Effect*

A Frequent Etiology for Pneumomediastinum in Severe Blunt Chest Trauma

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Study objectives: To review the etiology and pathophysiology of pneumomediastinum in severe blunt trauma, with a special interest in one of its possible origins, the Macklin effect. The Macklin effect relates to a three-step pathophysiologic process: blunt traumatic alveolar ruptures, air dissection along bronchovascular sheaths, and spreading of this blunt pulmonary interstitial emphysema into the mediastinum. The clinical relevance of the Macklin effect was also evaluated.

Setting: A university hospital serving as a reference trauma center.


Inclusion criteria: Severe trauma or high-speed deceleration justifying chest CT; if chest CT demonstrated a pneumomediastinum, bronchoscopy and esophagoscopy were performed to rule out tracheobronchial or esophageal injury.

Design: Retrospective analysis of patients’ clinical files, chest CT, and bronchoscopy and esophagoscopy reports. The Macklin effect was diagnosed when an air collection adjacent to a bronchus and a pulmonary vessel could be clearly identified on the chest CT. Clinical relevance of the Macklin effect was statistically evaluated regarding its repercussions on the pulmonary gas exchange function, the respective durations of intensive care and total hospital stay, and the associated injuries.

Results: Twenty (39%) Macklin effects and 5 tracheobronchial injuries (10%) were identified. One tracheobronchial injury occurred simultaneously with the Macklin effect. The presence of the Macklin effect affected neither the clinical profile nor the result of pulmonary gas analysis on hospital admission, but was associated with a significant (p < 0.001) lengthening of the intensive care stay.

Conclusions: The Macklin effect is present in 39% of severe blunt traumatic pneumomediastinum detected by CT. Its identification does not rule out a tracheobronchial injury. The Macklin effect reflects severe trauma, since it is associated with significantly prolonged intensive care stay.

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Key words: chest radiograph; CT; diagnostic imaging; lung; mediastinal emphysema; nonpenetrating wounds; pulmonary interstitial emphysema

Pneumomediastinum, also termed mediastinal emphysema, relates to air collections surrounding mediastinal structures. Pneumomediastinum is reported in up to 10% of patients with severe blunt chest trauma.1,2 In < 2% of patients, pneumomediastinum results from blunt tracheobronchial lesions. It rarely originates from esophageal injuries.3–6 However, in most instances, the origin of the pneumomediastinum remains obscure. It may result from the extension of cervical or thoracic subcutaneous emphysema, or of a retropneumoperitoneum consecutive to a hollow viscus rupture.1,2 In other cases, it has been attributed to the Macklin effect. This pathophysiologic process, first described by Macklin7 in 1939 and further studied by others,8–10 is summed up in three steps: alveolar ruptures, air dissection along bronchovascular sheaths, and spreading of this pulmonary interstitial emphysema into the mediastinum. The Macklin effect is involved in blunt traumatic pneumomediastinum but also in pneumomediastinum arising in various conditions, such as neonatal respiratory distress syndromes, asthma crises, positive-pressure mechanical ventilation, and Valsalva maneuvers (parturition, Boerhaave’s syndrome, and epileptic seizures).10–13 The Macklin effect has recently been identified on CT examinations.13–16

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Our purpose was to review our series of blunt trauma patients whose hospital admission chest CT survey revealed a pneumomediastinum, in order to identify its origin and thus determine the epidemiologic importance of the Macklin effect. Moreover, particular interest was devoted to the clinical relevance of the Macklin effect identified in patients with blunt trauma.

**Materials and Methods**

A total of 1,269 patients were admitted to our institution with a trauma diagnosis between March 1995 and March 2000. In cases of severe trauma and suggestive biomechanics (high-speed deceleration), trauma patients admitted to our institution undergo, as part of the initial survey, a spiral chest CT (CTi or CT Lightspeed unit; General Electric Medical Systems; Milwaukee, WI) with the following acquisition parameters: 120 kV, 200 mA, 3-mm or 2.5-mm thickness, and during IV injection of iopamidol (Iopamiro 300; Bracco; Milano, Italia) or iohexol (Accupaque 300; Nycomed; München, Germany). The purpose of this CT survey is to detect blunt thoracic lesions, notably to identify occult pneumothoraces, pulmonary contusions, or a possible blunt aortic injury. Chest radiographic or CT diagnosis of a pneumomediastinum systematically leads us to proceed to bronchoscopy and esophagoscopy in order to rule out a possible tracheobronchial or esophageal injury.

Among the 297 trauma patients who underwent chest CT, 51 patients with severe blunt trauma whose hospital admission chest CT revealed a pneumomediastinum and who had undergone complete bronchoscopy and esophagoscopy were retrospectively identified. The corresponding chest CT surveys were reviewed with pulmonary window settings, so as to track subcutaneous emphysema, pneumothorax, and/or the Macklin effect. The latter features tiny air lucencies contiguous to small pulmonary vessels and relating bronchi on lung fields. However, these air lucencies cannot be clearly distinguished from blunt traumatic lung lacerations, so that the Macklin effect is diagnosed convincingly only when the pulmonary vessel next to the bronchus could be identified without doubt with an adjacent air collection on the chest CT (Fig 1). Patients’ chest CT surveys were reviewed by two independent readers who were blinded to the clinical data at the time of review, and a Macklin effect was diagnosed only with two reviewers’ agreement. When the two readers’ opinion differed, diagnosis was made by consensus. Patients with a diagnosed Macklin effect formed patient group 1, whereas those without a Macklin effect constituted patient group 2.

Since the Macklin effect involves alveolar ruptures, possible repercussions on the pulmonary gas exchange function of the alveolar lining could be suspected. Hospital admission blood gas analyses were thus reviewed in both patient groups for pH and Pco₂. Pco₂ could not be used because of the frequent administration of oxygen in blunt trauma patients. Finally, associated injuries and durations of the intensive care and total hospital stays were chosen as severity hallmarks of the patients’ clinical condition.

Statistical analysis was obtained for the 51 patients included in the study, regarding age, sex ratio, hospital admission blood gas analyses, duration of intensive care and total hospital stays, and associated injuries. The small number of patients in both groups justified the use of two-sample Wilcoxon rank-sum (Mann-Whitney) tests for continuous variables and of Fisher’s Exact Tests for discrete ones.

**Results**

In our series of 51 patients with blunt traumatic pneumomediastinum, 20 patients (39%) were involved in a motor vehicle accident, 8 patients (16%) were involved in a motorcycle accident, 4 patients (8%) were hit as pedestrians, 18 patients (35%) were injured in a fall, and 1 patient (2%) received injury in a domestic accident.

Pneumomediastinum in patients with severe blunt trauma may have various origins: the Macklin effect, a tracheobronchial lesion, a pneumothorax, or a subcutaneous emphysema, some of these causes being sometimes concomitant (Table 1). The Macklin effect was identified in 20 of the 51 patients. Only one case led to different diagnosis on the part of the two reviewers; after review, the Macklin effect was finally ruled out.

The patient with concomitant tracheobronchial injury and Macklin effect suffered from a posterior, transmural tracheal tear, 1 cm above the carina. Identification of the Macklin effect as origin of a blunt traumatic pneumomediastinum did not allow ruling out a tracheobronchial injury (p = 0.340; Table 2).

**Table 1—Origins of Pneumomediastinum in 51 Patients With Severe Blunt Trauma**

<table>
<thead>
<tr>
<th>Patients, No. (%</th>
<th>Macklin Effect</th>
<th>Tracheobronchial Injury</th>
<th>Pneumothorax</th>
<th>Subcutaneous Emphysema</th>
<th>Unknown</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 (19)</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>1 (2)</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>6 (12)</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>3 (6)</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>3 (6)</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>1 (2)</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>16 (31)</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>1 (2)</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>5 (10)</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>5 (10)</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>
The average age (mean ± SD) of the 51 patients involved in the study was 43 ± 17 years. Age (p = 0.072) and sex ratio (p = 0.304) did not significantly differ in both groups of patients. Ages ranged from 13 to 60 years (mean, 38.5; SD, 15) in patient group 1, and from 18 to 80 years (mean, 49; SD, 18) in patient group 2. The sex ratios (predominantly male) were 1.7 and 1.8 in patient group 1 and patient group 2, respectively.

pH and Pco2 levels at hospital admission were 7.31 ± 0.10 and 46 ± 11 mm Hg (6.1 ± 1.5 kPa) in patient group 1, and 7.32 ± 0.13 and 43 ± 10 mm Hg (5.8 ± 1.4 kPa) in patient group 2, respectively. No significant difference could be observed regarding either the pH (p = 0.470) or the Pco2 (p = 0.463), relating to the presence or absence of a Macklin effect.

The duration of the intensive care stay was 14.4 ± 5.5 days in patient group 1 and 5.8 ± 5.6 days in patient group 2. It thus was significantly higher (p < 0.001) in patients with a Macklin effect. However, the duration of the complete hospital stay was 30.9 ± 12.8 days in patient group 1 and 20.4 ± 9.5 days in patient group 2, which was not significantly shorter (p = 0.220).

No significant association could be identified between the presence of a Macklin effect and other blunt injuries. Craniocerebral trauma affected 40% of patients in group 1 and 29% of patients in group 2 (p = 0.767), cardiovascular lesions were found in 30% of patients in group 1 and 19% of patients in group 2 (p = 0.704), flail chest or sternal fracture was found in 20% of patients in

\[ \text{Table 2—Macklin Effect and Tracheobronchial Injuries in Patients With Blunt Trauma and Pneumomediastinum}^* \]

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group 1, Macklin Effect</th>
<th>Group 2, No Macklin Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>With tracheobronchial injury</td>
<td>1 (2)</td>
<td>4 (8)</td>
</tr>
<tr>
<td>Without tracheobronchial injury</td>
<td>19 (37)</td>
<td>27 (53)</td>
</tr>
</tbody>
</table>

*Data are presented as No. (%).
group 1 and 9.5% of patients in group 2 (p = 0.514), while diaphragmatic ruptures were observed in 0% of patients in group 1 and in 9.5% of patients in group 2 (p = 0.668). The mortality rate was similar in patient group 1 (n = 2, 10%) and in patient group 2 (n = 4, 13%).

**Discussion**

We reviewed a group of 51 patients with blunt chest trauma whose severe condition or accident biomechanics justified chest CT survey. The latter demonstrated a pneumomediastinum, which in turn led to the performance of bronchoscopy and esophagoscopy. Fifty-nine percent of these patients with blunt chest trauma were involved in traffic accidents. Fifty percent of patients were between 27 years and 55 years of age, with a predominantly male sex ratio of 1.8:1.

Pneumomediastinum relates to air collections within the mediastinum. These air collections are thought to arise from various origins. Tracheobronchial or esophageal ruptures create an air leak into the mediastinum. The Macklin effect involves alveolar ruptures with air dissection along bronchovascular sheaths (Fig 1) to the mediastinum; these alveolar ruptures are either isolated or confluent and then result in pulmonary lacerations. In case of a pneumothorax, a concomitant tear of the parietal pleura may allow the free pleural air to enter the mediastinal compartment. Finally, subcutaneous emphysema created by rib fractures (in association with a pneumothorax or not) may progress along fascial sheaths and extend into the mediastinum. Conversely, a pneumomediastinum may dissect along fascial sheaths to create a cervical or thoracic subcutaneous emphysema.

Our data are consistent with these pathophysiological hypotheses (Table 1). We identified 20 patients with a Macklin effect, as well as 5 patients with tracheobronchial lesions, representing 39% and 10% of our patients, respectively. No esophageal injury was identified. The origin of the blunt traumatic pneumomediastinum remained undefined in five patients (10%).

Blunt traumatic pneumomediastinum may find its origin in several concomitant events. Notably, one patient was admitted to the hospital with simultaneous tracheal injury and Macklin effect. Thus, identification of a Macklin effect should not prevent the patient from undergoing a bronchoscopy.

There was no significant difference in age or sex ratio between groups of patients. However, no patient with the Macklin effect could be observed beyond the age of 60 years, whereas six patients were aged from 60 to 80 years in group 2. This may result from an increased stiffness of the pulmonary interstitium in the elderly, preventing air leak and dissection along peribronchovascular sheaths.

In our patients, the presence of the Macklin effect does not alter the result of blood gas analysis performed on hospital admission. Ruptures of a few pulmonary alveoli, with subsequent air dissection along bronchovascular sheaths, do not affect pulmonary gas exchanges. However, the Macklin effect reflects a severe blunt chest trauma, since it is associated with a significantly prolonged intensive care stay. Moreover, there was a trend to a longer duration of the complete hospital stay (about 10 days) in the 20 patients with the Macklin effect. However, this was not statistically significant, probably because there were not enough patients in each group. A specific origin for the prolonged intensive care stay could not be statistically inferred from our series of 20 patients with the Macklin effect: this prolonged stay related to sepsis from various origins (pulmonary, urinary, etc) in 4 patients, ARDS in 3 patients, and cardiovascular complications in 2 patients, whereas there were no complications in the remaining 11 patients.

**Conclusion**

In conclusion, the Macklin effect was identified in 39% of our patients with severe blunt traumatic pneumomediastinum. Its identification, as possible origin of pneumomediastinum, should not prevent the performance of bronchoscopy and esophagoscopy in order to detect a possible concomitant tracheobronchial or esophageal injury. Identification of the Macklin effect is associated with significantly longer intensive care stay.

**References**

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