Crushed Chest Injury and Artificial Ventilation*

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Crushing injuries of the chest have steadily increased in the past decade with the increase in number of cars and speed of automobile traffic. In a series of 1678 persons injured, 1.5 per cent had a crushing injury of the chest, and approximately 25 per cent of all traffic deaths were due to chest injuries. Unfortunately, many injuries of the thorax occur at sites distant from medical centers. It is important, therefore, for the physician to familiarize himself with the emergency treatment of such patients. The purpose of this paper is to emphasize the use of internal pneumatic stabilization in the emergency treatment of patients with severe crushing injuries of the chest, and to question the necessity of open reduction in patients with a severely dislocated flail thoracic segment.

Material

Seventy-five patients with closed chest trauma were seen and treated at the Surgical Hospital of the University of Würzburg, Würzburg, Germany, from January, 1962 to February, 1964 (Table 1). Fifty-three of these patients had only mild trauma to the thorax and will not be discussed further in this paper. The remaining 22 patients had severe injuries of the chest. Thoracic injury alone was seen in eight patients and was associated with other severe injuries in the remaining 14. Four sustained an injury of the thorax and head; two of the thorax and abdomen; and eight had injuries of the chest associated with injuries of the head, abdomen, pelvis and extremities.

The 15 patients who required artificial ventilation with intermittent positive pressure breathing (IPPB) were divided into two groups. Group 1 contained the seven survivors, and group 2 the eight who died. The severity of chest injury was similar in both groups. Three had fractured sternum and three had ruptured diaphragm. Five had pneumothorax and eight had hemothorax. A traumatic wet lung was diagnosed in ten. Five showed no sign of traumatic wet lung, although they suffered from crushed chest injury. Eight exhibited paradoxical respiration. Abdominal injuries were more frequently encountered in group 2, and six of the eight in this group had such an injury. Head injuries occurred in both groups in four instances. However, the severity of head injury in group 2 was more extensive as evidenced by the sustained unconsciousness of the four with head injury in this group.

Treatment

The major pathologic abnormalities of our patients with a crushed chest injury were lung trauma, paradoxical motion of the chest wall, and traumatic wet lung. These pathologic abnormalities led to inadequate pulmonary ventilation which in turn produced hypoxia, hypercapnia, atelectasis, pneumonitis. In these severely injured patients, artificial ventilation with IPPB markedly improved pulmonary function. This was accomplished by connecting a cuffed tracheostomy tube to a positive pressure breathing machine.

Restoration of pulmonary function, decompression of hemopneumothoraces, and the treatment of shock were the important features of the therapy. Often all three were accomplished simultaneously in the emergency room. In our patients, artificial ventilation with IPPB was begun soon after the patient's admission to the hospital or whenever it was deemed advisable. The
my became very important to avoid secondary complications. Sterile precautions were taken to prevent unnecessary contamination of the airway. The nursing staff was instructed to use sterile gloves and sterile catheters during each aspiration. Since aspiration was done and as frequently as nearly 10 to 20 minutes, unnecessary trauma to the tracheal mucosa was avoided. Cultures with sensitivity tests of tracheal secretions were obtained at regular intervals and appropriate systemic antibiotics were given. Frequent changes of the patient's posture enhanced the drainage of bronchial secretions into the trachea where they were removed. In some cases, bronchial toilet under direct vision with the bronchoscope was necessary due to retained secretions.

Those who needed prolonged artificial ventilation required special humidification of the inspired gases. We used a heated nebulizer†† to maintain high humidity, which prevented crusting of bronchial secretions. Bronchodilators were used when indicated.

RESULTS

One patient in group 1 was dismissed from the hospital with a tracheal cannula in place. He is now a pulmonary cripple; however, he had had severe pulmonary emphysema prior to the accident. Three are restricted in their physical activities. One 12-year-old boy has no physical restrictions, and the hospital stay of two others was prolonged due to other reasons.

The cause of death of three patients in group 2 was cardiac arrest in two and severe head injury in three. One with an

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| TABLE 1—75 Patients with Closed Injuries of the Chest (Jan. 1962—Feb. 1964) |
|-------------------|-----------------|----------------|
| Mild to moderate injuries without complications | Number of Patients |IPPB|Survived|Died |
| Thorax | 53 | |53| |
| Thorax and head | 4 | 2 | 2 | | |
| Thorax and abdomen | 2 | |2| |
| Thorax and head pelvis abdomen extremities | 8 | 7 | 1 | 6 |
| TOTAL | 22* | 15 | 7 | 8 |

*Seven of the 22 patients were not ventilated and of these patients, five survived and two died.

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| TABLE 2—Test of Pulmonary Function on Four Patients After Crushed Chest Injury |
|-------------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
|---------|-----|-----|-------------|-------------|-------------|-------------|-------------|-------------|
| 1. | 60 | F | 14-16 | 20 | 7.10 | 12.60 | 210 | 390 | 59.00 | 27.00 | 3450 | 2100 |
| 2. | 59 | M | 14-16 | 21 | 6.00 | 7.25 | 180 | 270 | 53.20 | 17.25 | 2950 | 1650 |
| 3. | 70 | M | 14-16 | 18 | 6.09 | 9.18 | 190 | 270 | 44.43 | 20.52 | 2962 | 1200 |
| 4. | M50 | 14-16 | 26 | 8.05 | 11.70 | 240 | 180 | 74.20 | 16.55 | 3905 | 1110 |

††Puritan heated nebulizer, Puritan International S. A. Baltimore, Maryland.
extensive head injury developed severe bronchopneumonia, which contributed to his death. Two died of pulmonary embolism, and one had an associated undiagnosed adrenal medullary insufficiency. One died from uncontrolled hemorrhage from both internal mammary arteries.

Studies of pulmonary function were done in four patients after injury and are summarized in Tables 2 and 3. Although three had only minor complaints, the results of these tests indicated severe restrictive abnormalities in all cases. Unfortunately, the studies which were done did not clearly elucidate the degree of restrictive disease.

<table>
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<th>Sex</th>
<th>FEV 1.0″*</th>
<th>FEV 1.0″%</th>
<th>FVC 1.0″*</th>
<th>FVC 1.0″%</th>
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<td>60</td>
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<tr>
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<td>1</td>
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<td>M</td>
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<td>65</td>
<td>18</td>
<td>1</td>
<td>1.58</td>
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</tbody>
</table>

*FEV 1.0″—Timed Vital Capacity in 1 second. **FVC—Predicted vital capacity.

**DISCUSSION**

Crushing chest injuries have always carried a high mortality rate due to ventilatory insufficiency. Proper treatment of these patients depends upon the correction of the ventilatory insufficiency and accompanying shock. One must immediately establish a clear airway, carry out a careful tracheobronchial toilet, and institute adequate alveolar ventilation once the diagnosis of ventilatory insufficiency is made.

Artificial ventilation with IPPB appeared to correct ventilatory insufficiency and produced internal pneumatic stabilization. This form of treatment for patients with a crushed chest injury was first introduced by Avery, Mörch, and Benson.

Intermittent positive pressure breathing impeded paradoxic movements of a flail segment, and was thus lifesaving. However, IPPB was not able to re-establish correct anatomic position of multiple doubly fractured ribs with severe dislocation (Fig. 1).

Test of pulmonary function from this study indicated the presence of restrictive disease in all patients following recovery from the accident. Because of this finding, we now believe that open reduction of the dislocated flail segment may reduce the post-traumatic thoracoplasty-like effect in those patients. However, open reduction is certainly not necessary as an initial
treatment since internal pneumatic stabilization is effective in alleviating ventilatory insufficiency.

The seven patients who survived may have died had not artificial respiration been used. However, prolonged artificial respiration with IPPB certainly has its own risk. The most serious complications are tension pneumothorax, reduced cardiac output, alveolar hypoventilation with CO₂ retention, and contamination of tracheobronchial tree and lungs. Tension pneumothorax must be avoided and its presence should be ruled out in the initial phase of emergency treatment, especially when thoracentesis must be done. Reduced cardiac output can be further accentuated in the hypovolemic patient by IPPB. Because CO₂-tension is difficult to detect clinically, regular determinations of arterial CO₂ plus O₂ tensions are necessary.

Sometimes it is difficult to decide when a patient can benefit from artificial ventilation. One cannot rely on the determination of arterial blood gases alone. We believe that either significant elevation of the Pco₂ above the level of 60 mm.Hg or steadily rising values of the Pco₂ are definite indications for artificial respiration. One with a normal Pco₂ value may need artificial ventilation, since it does not indicate how the normal ventilation was achieved. One at the border of compensation can be tipped into respiratory failure by the slightest additional complication. One of the two patients who died without artificial respiration had a Pco₂ which was not elevated (32 to 44 mm.Hg), and it was interesting to note that he had fractured nearly all the ribs of his left hemithorax. Retrospectively we now believe that this patient was a good candidate for prolonged artificial respiration with IPPB. This thought was confirmed by the necropsy examination which showed evidence of central respiratory failure.

The clinical impression of the status of the patient is the single most important factor which can determine whether artificial ventilation is required. However, the impression of the effectiveness of ventilation by inspection can be difficult. A normal respiratory pattern does not necessarily mean the patient has an adequate alveolar ventilation. This is specially true in the presence of hemorrhagic shock, as shown in the studies of Gerst and co-workers, and Freeman and Nunn. The work of these authors, as well as our own, shows that during hemorrhagic hypotension the ratio of physiologic dead space to tidal volume may increase to 75 per cent. This means the decision to ventilate a patient artificially must be made from the impression of the patient’s clinical status and from the determination of the arterial blood gases.

We would like to emphasize that oxygen therapy, intercostal nerve block and tracheostomy, as the initial or only treatment of patients with minor crushed chest injuries, are important. Artificial respiration should be employed either when ventilation is insufficient or when the work of respiration is too great for the patient. Through the use of artificial ventilation we believe that no patient today with intact lungs who has sustained a crushed chest

![Figure 1B: X-ray film taken one year after injury. Note the continued dislocation and thoracoplastry-like effect of the upper left rib cage.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21440/ on 06/27/2017)
injury alone, should die from respiratory insufficiency.

**Summary**

Fifteen patients with severe crushing injury of the chest were artificially ventilated with IPPB. They were divided into two groups; group 1 contained seven survivors and group 2 contained the eight who died. It was concluded that the associated injuries were frequently responsible for the final outcome. The indications for the use of artificial ventilation with IPPB were discussed, and the initial emergency treatment of patients with a crushed chest injury was reviewed.

**Resumen**

Quince pacientes con traumatismos aplastantes del torax fueron ventilados artificialmente con presión positiva intermitente, RPPI. De ellos siete sobrevivieron y ocho fallecieron; el desenlace fatal parece frecuentemente relacionado con las lesiones asociadas. Las indicaciones de la ventilación artificial por RPPI son analizadas y el tratamiento inicial de emergencia en estos casos revisado.

**Résumé**

Chez 15 malades atteints de graves écrasements thoraciques, on pratiqua la ventilation artificielle. Ces malades se divisaient en 2 groupes: 7 survivants dans le 1er et 8 morts dans le 2nd. Cette étude amène à conclure que les lésions associées ont été souvent responsables de l'évolution fatale. L'auteur discute les indications de la ventilation artificielle et fait l'exposé des traitements immédiats d'urgence chez les traumatisés du thorax par écrasement.

**Zusammenfassung**


**References**


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**AMERICAN COLLEGE OF CHEST PHYSICIANS 1967 ESSAY CONTEST**

Medical students wishing to enter the 1967 Alfred A. Richman Essay Contest of the American College of Chest Physicians must observe the following rules:

1. Complete application form in duplicate, have original copy signed by the dean of the medical school, and return original copy to the offices of the American College of Chest Physicians.
2. Five copies of the manuscript, typewritten in English (double spaced) must be submitted to the American College of Chest Physicians in Chicago not later than March 15, 1967.
3. The length for manuscripts is optional: 2500-4500 words suggested.
4. The only means of identification of the author shall be a motto or other device on the title page. A sealed envelope bearing the same motto on the outside and enclosing the name and address of the author must accompany the essay. (Motto may be a word or brief phrase which has a significant meaning to the author.)

The first prize will be $500; second prize will be $300; third prize will be $200. Each winner will also receive a certificate of merit. A trophy, inscribed with the name of the first prize winner and the name of his school will be awarded to the winner's school.

The winning contributions will be selected by a committee of chest specialists and will be announced at the 33rd Annual Meeting of the American College of Chest Physicians to be held in Atlantic City, New Jersey, June 15-19, 1967. All manuscripts become the property of the American College of Chest Physicians and may be considered for publication in the College journal.

It is suggested that applicants study the format of the College journal, DISEASES OF THE CHEST, to guide them in preparing the essay. 