Immediate Operative Treatment for Massive Hemoptysis*

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A series of 15 patients with life threatening, massive hemoptysis is reported. Thirteen of these patients underwent immediate operative treatment with only three deaths. One died after bronchoscopic identification of the bleeding site while awaiting elective thoracotomy. The other patient left the hospital against medical advice.

Operative intervention often must be considered in the initial management of patients who present with life threatening hemoptysis. In the past an arbitrary definition of massive hemoptysis (600 ml) which requires pulmonary resection has been proposed.† However, in the severely obtunded or debilitated patient, small amounts of blood may not be cleared adequately from the tracheobronchial system and suffocation can ensue. Therefore, in those patients whose life appears to be threatened by further bleeding, identification of the bleeding area by bronchoscopy and definitive excision of the bleeding part should be done unless specific contraindications exist. This report summarizes experience with 15 such patients in Baylor College of Medicine Affiliated Hospitals.

**Clinical Material.

Fifteen patients with life threatening hemoptysis have been seen by the Surgical Services at the Ben Taub General Hospital and the Houston Veterans Administration Hospital during the past five years. Six of these have been reported previously.³ There were twelve men and three women. Eight were Negro and seven were Caucasian. Ages ranged from 23 to 63 years. Blood replacement before operation varied from none to ten units.

Operation was not performed in two patients, while pulmonary resection was carried out in the other 13. Among these 15 patients, causes of massive hemoptysis (Table 1) were cavitary tuberculosis (5 patients), bronchiectasis (3 patients), pyogenic abscess with aspergillum (2 patients), pneumonia (2 patients), metastatic carcinoma (1 patient), and bronchial carcinoma (1 patient). In these patients, causes of massive hemoptysis (Table 1) were cavitary tuberculosis (5 patients), bronchiectasis (3 patients), pyogenic abscess with aspergillum (2 patients), pneumonia (2 patients), metastatic carcinoma (1 patient), and bronchial carcinoma (1 patient).

Table 1—Diagnosis and Location of Lesion

<table>
<thead>
<tr>
<th>Case No</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Volume (ml/24 hrs)</th>
<th>Location by Bronchoscopy</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>48</td>
<td>F</td>
<td>Tuberculosis</td>
<td>1000</td>
<td>Right upper lobe</td>
</tr>
<tr>
<td>2</td>
<td>45</td>
<td>F</td>
<td>Tuberculosis</td>
<td>400</td>
<td>Right upper lobe</td>
</tr>
<tr>
<td>3</td>
<td>30</td>
<td>F</td>
<td>Tuberculosis</td>
<td>600</td>
<td>Right upper lobe</td>
</tr>
<tr>
<td>4</td>
<td>48</td>
<td>M</td>
<td>Tuberculosis</td>
<td>700</td>
<td>Right upper lobe</td>
</tr>
<tr>
<td>5</td>
<td>43</td>
<td>M</td>
<td>Tuberculosis</td>
<td>800</td>
<td>Right upper lobe</td>
</tr>
<tr>
<td>6</td>
<td>35</td>
<td>M</td>
<td>Bronchiectasis</td>
<td>250</td>
<td>Right upper lobe</td>
</tr>
<tr>
<td>7</td>
<td>61</td>
<td>M</td>
<td>Bronchiectasis</td>
<td>150</td>
<td>Right upper lobe</td>
</tr>
<tr>
<td>8</td>
<td>55</td>
<td>M</td>
<td>Bronchiectasis</td>
<td>600</td>
<td>Right middle lobe</td>
</tr>
<tr>
<td>9</td>
<td>48</td>
<td>M</td>
<td>Aspergillum</td>
<td>300</td>
<td>Left upper lobe</td>
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<tr>
<td>10</td>
<td>53</td>
<td>M</td>
<td>Aspergillum</td>
<td>400</td>
<td>Left upper lobe</td>
</tr>
<tr>
<td>11</td>
<td>45</td>
<td>M</td>
<td>Recurrent Pneumonia</td>
<td>300</td>
<td>Right upper lobe</td>
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<tr>
<td>12</td>
<td>23</td>
<td>M</td>
<td>Staphylococcus pneumonia (heroin addict)</td>
<td>500</td>
<td>Left upper lobe</td>
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<tr>
<td>13</td>
<td>46</td>
<td>M</td>
<td>Metastatic sarcoma</td>
<td>800</td>
<td>Right upper lobe</td>
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<tr>
<td>14</td>
<td>63</td>
<td>M</td>
<td>Normal lung</td>
<td>700</td>
<td>Right upper lobe</td>
</tr>
<tr>
<td>15</td>
<td>49</td>
<td>M</td>
<td>Not determined</td>
<td>500</td>
<td>Left lung</td>
</tr>
</tbody>
</table>

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152 McCollum et al

CHEST, 67: 2, FEBRUARY, 1975
recurrent pneumonia (1 patient) and necrotic metastatic sarcoma which had responded to chemotherapy (1 patient). The other patient with unrelenting hemoptysis requiring operation, had a normal chest roentgenogram and the resected specimen did not reveal the cause of bleeding.

Bronchoscopy was performed in all 15 patients immediately after hospital admission. The site of bleeding was identified on initial examination in all except two patients. Repeat bronchoscopy during an episode of active hemoptysis identified the bleeding site in one of these.

The double-lumen Carlens tube was used for endotracheal intubation in 11 of the 13 patients undergoing resection for massive hemoptysis. By this means hemorrhage was limited to the involved lung and the dependent lung was utilized for ventilation. After excision of the involved lobe, the open-ended bronchus was aspirated of retained blood and secretion prior to closure.

**Results**

Ten of the 13 patients undergoing emergency resection for massive hemoptysis were long term survivors (Table 2). One patient in whom a Carlens tube was not used sustained a cardiac arrest secondary to hypoxia from blood in the dependent mainstem bronchus during operation. Another patient died after operation from progressive hypoxia secondary to inadequate pulmonary reserve. One patient bled from the chest wall, required reoperation for evacuation of a clotted hemothorax, had inadequate pulmonary expansion resulting in empyema and requiring thoracoplasty, and eventually died in sepsis.

Complications occurred in two patients surviving operation. One developed hemorrhage from the pulmonary artery, which required reoperation, and subsequently developed a bronchopleural fistula, which was managed satisfactorily by long term tube drainage. Another patient in whom a Carlens tube was not used developed respiratory insufficiency secondary to extensive spread of blood throughout the tracheobronchial tree and required prolonged ventilatory support prior to recovery.

Two patients did not undergo resection. One with a history of intermittent hemoptysis for two years, but without roentgenographic evidence of pulmonary disease, left the hospital against advice after spontaneous cessation of hemorrhage for 48 hours. He was seen last four months later, at which time there had been no further episodes of bleeding. The other patient, who was found at bronchoscopy to be bleeding from the left upper lobe, stopped bleeding spontaneously. Elective resection was planned, but massive hemoptysis recurred 36 hours after bronchoscopy, and he suffocated before operation could be performed.

**Discussion**

The presence of large amounts of free and clotted blood in the tracheobronchial system must be treated as a life threatening emergency. The site of bleeding must be identified rapidly and resection carried out expeditiously. Although history and physical examination, as well as roentgenographic examination of the chest, may suggest the site of bleeding, precise identification of this area must be done by direct visualization with a bronchoscope. Massive pulmonary hemorrhage may occur from an area which appears normal by routine roentgenographic examination, and bronchoscopy is the most reliable means of identifying the bleeding site.4,5

Recent interest in use of the fiberoptic bronchoscope relates primarily to the ability of the endoscopist more adequately to evaluate the tracheobronchial tree, particularly in its more peripheral aspects, with this instrument than with the rigid bronchoscope. In the patient with massive hemoptysis, however, only the rigid bronchoscope provides the means for adequate clearing of blood from the tracheobronchial tree and maintenance of a satisfactory airway. Occasionally, passage of the fiberoptic scope through the rigid scope aids in evaluating
segmented bronchi and arriving at a definitive etiologic diagnosis, but the fiberoptic scope should not be used as the primary means of bronchoscopy in the patient with massive hemoptysis.

When bronchoscopy is performed, hemorrhage occasionally is so rapid that the site of origin cannot be identified immediately. Under these circumstances, the bronchoscope should not be removed but should be used to clear blood from the tracheobronchial tree and to maintain an airway. Light sedation without depressing the cough reflex and topical oropharyngeal anesthesia are used, and the Trendelenburg position may be helpful in the adequate evacuation of blood. Once the bleeding site is identified, topical application of epinephrine and pressure may aid in temporary control of hemorrhage. Under extenuating circumstances passage of a gauze pack through the bronchoscope into the involved bronchus may be necessary for temporary control of hemorrhage, or the bronchoscope may be placed into the uninvolved mainstem bronchus to maintain an airway while general anesthesia is initiated. Alternately, a Fogarty catheter may be passed through the bronchoscope and the balloon inflated to obstruct the bleeding from a secondary bronchus. Utilization of cardiopulmonary bypass for oxygenation of a suffocating patient in whom an airway cannot be maintained by either technique, also has been proposed.

After bronchoscopic examination of the tracheobronchial tree and the induction of general anesthesia, a Carlens tube should be placed and the patient prepared for definitive thoracotomy. If the Carlens tube is not available or the anesthesiologist is not familiar with its use, a large endotracheal tube can be passed into the mainstem bronchus of the uninvolved lung with complete isolation of the bronchus from the bleeding site. The disadvantage of the conventionally placed endotracheal tube is that blood may continue to spread throughout the tracheobronchial tree. Close cooperation between anesthesiologist and surgeon is particularly essential while the bleeding site is being identified and intubation performed.

When a patient presents with life threatening hemoptysis, but a bleeding site cannot be identified by initial bronchoscopy, it should be presumed that the patient will bleed again. Therefore, the patient should be placed in an intensive care unit so that immediate therapy can be instituted in the event hemorrhage recurs. Suffocation from recurrent hemoptysis may occur if thoracotomy is delayed after the bleeding site is identified by bronchoscopy. One death in the present series resulted from such an untoward occurrence. Once the location of hemorrhage is identified, resection therapy should be performed immediately.

In the patient in whom the bleeding site cannot be identified on initial bronchoscopy, additional investigations should be carried out in the intensive care unit. In addition to causes of massive hemoptysis seen in this series (Table 1), other possible causes, such as broncholithiasis, should be considered. Sputum studies should be performed, and the use of bronchography, tomodensitigraphy may be helpful in identifying the source of bleeding. Pulmonary function studies for evaluation of pulmonary reserve also may be helpful. However, when the patient is actively bleeding, exploratory thoracotomy should not be delayed for such studies since continued bleeding will result in reduction of functioning lung tissue and further decrease the patient's chances of survival.

Crocco et al reported an 81 percent survival among 32 patients with massive hemoptysis undergoing operative therapy, as compared to a 22 percent survival for nine patients who were treated nonsurgically. Other authors also have found resective therapy superior to nonsurgical management. In the present series, 10 of 13 patients undergoing resection survived. Among patients with hemoptysis, massive bleeding is rare, but when it occurs, it is a life threatening emergency and resection offers the patient the best chance for survival.

Although the preferred treatment for massive hemoptysis is operative resection, there are certain patients in whom a nonsurgical approach is preferable. Disseminated terminal pulmonary cancer is a contraindication to resection. Pulmonary hemorrhage secondary to mitral stenosis is best controlled by emergency cardiac surgery rather than pulmonary resection. Coagulopathies secondary to anticoagulant medication must be corrected prior to considering resection as definitive treatment for massive hemoptysis. In addition, von Willebrand's disease and Goodpasture's syndrome may present with abnormal bleeding tendencies and massive hemoptysis which is not amenable to localized resection. There will also be an occasional patient whose pulmonary reserve prohibits thoracotomy and resection. Finally, inability to localize the bleeding site is a contraindication to attempts at resectional therapy until such localization has been accomplished.

References
The title refers to the current geriatric population of the United States. The increased incidence of acute and chronic illnesses, with possible protracted invalidism and/or potentially fatal outcome, constitute a challenge to the medical profession which should be more than of passing interest. It is a pathetic commentary to admit that the fundamental substrate of aging, its causes and dubious amenability by medical intervention are matters of speculation rather than irrefutable scientific facts. It is hoped that by reviewing some of the salient facets of pertinent problems renewed interest may be added to proficient explorations conducive to progress in this field. It is the consensus that "natural" structural and functional changes condition the aging human body to disease. This conditioning may be attributable to weakening of bodily resistance, defense and repair. Impairment of this sort may be due to unfavorable alterations in the "milieu interieur" of Claude Bernard brought about by faulty, deficient nutrition. Too, it may be influenced unfavorably by adverse emotional stress and strain resulting from social and/or economic circumstances. In persons of advanced age, decrease in the number of neurons and retrogressive changes in the brain, with the latter's substantially reduced weight, may cause weakened senile existence. In the aged one finds frequently degenerative changes in the locomotor system, particularly muscle weakness, and osteoarthritis; the latter being due to repetitive wear and tear of mechanical trauma, and aggravated by obesity. Ecologic influences may accelerate aging. Some of the harmful agents in this regard are ozone, oxides of nitrogen, some of the industrial waste products, motor vehicle exhaust fumes, and radioactive air contaminants. The term arteriosclerosis was coined by Lobstein in 1833. The condition has been identified as of common occurrence since the earliest days of human history. It is associated with vascular changes, such as fragmentation of the elastic components, and the replacement of the muscle of media by fibrous tissue. This results not only in various degrees of vascular deformity but also in interference with normal circulation by increased peripheral resistance. According to Tomanek, R et al (J Gerontol 27:33, 1972) aging is associated with increase in the heart weight, pronounced increase in the collagen concentration in the ventricular walls and within the atrioventricular bundle. The cardiac output (L/min/M²) is diminishing progressively with advancing age until it becomes about 54 percent of that observed at the age of 20 years. In the text edited by Shock, NW (Biologic Aspects of Aging, New York, Columbia University Press, 1962) it is stated that "the amount of oxygen the blood takes up from the lungs and transports to the tissues during exercise falls substantially with age. The blood of a twenty year old man takes up, on the average, almost 4 L of oxygen per minute, whereas at the age of 75 the rate is only 1.5 L per minute. In order to double the level of oxygen uptake during exercise the older individual must move 50 percent more air in and out of his lungs." With aging, respiratory excursions of the chest wall may be restricted by frequent costovertebral arthritises. Also, one is likely to find dorsal kyphosis, barrel-deformity of the chest together with hyperinflation of the lung. The amount and functional competence of the pulmonary elastic tissue are reduced, consequently the inspiratory recoil of the lung is lessened. There is dehydration of the lung tissue. Pulmonary blood vessels show sclerotic changes. The alveolar component of the lung volume and the vital capacity of the lung are decreased. There are increase in functional residual capacity and decrease in diffusion capacity for oxygen. Correction of acid-base imbalance is slower in older subjects; the same holds true of return to resting levels of blood pressure and pulse rate after exercise. Impairment of mucosal sensitivity of the upper air passages with advanced age facilitates inadvertent aspiration of food particles, mineral oil, regurgitated gastric contents, infectious exudate from the throat and paranasal sinuses, with serious consequences, such as pneumonitis, postpneumonic pulmonary fibrosis, atelectasis, and bronchiectasis. Awareness of the impaired resistance, defense and recuperative power of the body in old persons calls for urgently needed specific geriatric standards preventively, diagnostically and therapeutically. The precepts of reconditioning exercise programs advocated by Kraus, H and Raab, W (Hypokinetic Disease, Springfield, C C Thomas, 1961) merit a great deal of attention. 

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