CHAPTER 116

Interventional Bronchoscopy and Massive Hemoptysis

HIREN J. MEHTA and MICHAEL A. JANTZ

INTRODUCTION

Hemoptysis is defined as the expectoration of blood that originates from the lower respiratory tract. Pseudohemoptysis is the expectoration of blood from a source other than the lower respiratory tract such as the nares, oropharynx, larynx, or the gastrointestinal tract. Massive hemoptysis is defined as expectoration of blood exceeding 200 to 1,000 mL over a 24-hour period, with expectoration of more than 600 mL in 24 hours being the most commonly used definition (1).

In practice, the rapidity of bleeding and ability to maintain a patent airway are critical factors; life-threatening hemoptysis can alternatively be defined as the amount of bleeding that compromises ventilation (2). Only 3% to 5% of patients with hemoptysis have a massive bleed, with the mortality rate ranging from 20% to as high as 80% in some case series (3–5). Most patients who die from massive hemoptysis do so from asphyxiation secondary to airway occlusion by clot and blood—not from exsanguination. Prognostic factors associated with an increased risk of death from massive hemoptysis include bleeding in excess of 1,000 mL/24 hr, hemoptysis due to neoplasms, radiographic evidence of aspiration, and hemodynamic instability (3,6).

ETIOLOGY OF MASSIVE HEMOPTYSIS

Hemoptysis has multiple causes usually categorized under parenchymal diseases, airway diseases, and vascular diseases. Bleeding may originate from small or large lung vessels. The causes of massive hemoptysis are listed in Table 116.1. Virtually all causes of hemoptysis may result in massive hemoptysis. Infections associated with bronchiectasis, tuberculosis, lung abscess, and necrotizing pneumonia are commonly responsible for the massive bleeding. Other common causes include bronchogenic carcinoma, mycetoma, invasive fungal diseases, chest trauma, cystic fibrosis, pulmonary infarction, and coagulopathy. Although massive hemoptysis is usually due to bleeding from the bronchial circulation, alveolar hemorrhage due to conditions such as granulomatosis with polyangiitis (Wegener’s) granulomatosis and Goodpasture syndrome may occasionally cause massive hemoptysis (Table 116.2).

ANATOMIC SOURCES OF HEMOPTYSIS

The sources of lower respiratory tract bleeding include the pulmonary and bronchial circulations. Two arterial vascular systems supply blood to the lungs: the pulmonary and the bronchial arteries. The pulmonary arteries provide 99% of the arterial blood to the lungs and are involved in gas exchange. The pulmonary circulation is a low pressure circuit under normal circumstances. The bronchial arteries serving the intrapulmonary airways and lung parenchyma arise from the thoracic aorta at the level of the third through the eighth thoracic vertebrae, originating most commonly at the level of the fifth and sixth vertebrae and drain through the bronchopulmonary anastomoses into the pulmonary veins, which empty into the left side of the heart. Bronchial circulation supplies nourishment to the extra- and intrapulmonary airways and to the pulmonary arteries (vasa vasorum), without being involved in the gas exchange (7). Complex capillary anastomoses exist between the pulmonary arteries and the systemic bronchial arteries. When the pulmonary circulation is compromised (e.g., in thromboembolic disease, vasculitic disorders, or in hypoxic vasoconstriction), the bronchial supply gradually increases, causing a hyperflow in the anastomotic vessels, which become hypertrophic with thin walls and tend to break into the alveoli and bronchi, giving rise to hemoptysis. Likewise, in chronic inflammatory disorders, such as bronchiectasis, chronic bronchitis, tuberculosis, mycotic lung diseases, and lung abscess, as well as in neoplastic diseases, the release of angiogenic growth factors promote neovascularization and pulmonary vessel remodeling, with engagement of collateral systemic vessels (8). These new and collateral vessels are fragile and prone to rupture into the airways.

Angiographic studies of patients with active hemoptysis have demonstrated that bleeding originates from bronchial and pulmonary arteries in 90% and 5% of cases, respectively (1,9). In the remaining 5% of cases, hemoptysis may derive from nonbronchial systemic arteries (9). Very rarely, hemoptysis has been reported originating from pulmonary and bronchial veins (10) and capillaries (11). A recent study by Noe et al. (12) shows that bleeding from bronchial arteries can coexist with bleeding from nonbronchial and pulmonary arteries in the same patient.

INITIAL EVALUATION

A detailed history and physical examination should be performed. Patients with a history of tuberculosis may have bleeding from rupture of a pulmonary artery aneurysm in the cavity lumen, known as a Rasmussen aneurysm, or by breakdown of bronchopulmonary anastomoses within the wall of old cavities. Bronchogenic carcinoma should be suspected in smokers older than 40 years of age. Repeated episodes of hemoptysis over months to years suggest bronchiectasis or a carcinoid tumor. Chronic sputum production predating the
diffuse pulmonary infiltrates. The triad of upper airway disease, lower airway disease, and renal disease suggests granulomatosis with polyangiitis (Wegener granulomatosis) (17). Goodpasture syndrome should be suspected in young men with alveolar hemorrhage and microscopic or macroscopic hematuria (18). Patients with a history of systemic lupus erythematosus (SLE) may develop alveolar hemorrhage at any time during the course of their disease, and alveolar hemorrhage may be the initial manifestation (19). Alveolar hemorrhage should be considered in patients with diffuse pulmonary infiltrates who have recently undergone hematopoietic stem cell or bone marrow transplantation (20). Although uncommon, tracheoinnominate artery fistula is an important consideration in patients with tracheostomy presenting with massive hemoptysis (21,22). The peak incidence is between the first and second week, although hemorrhage can occur as early as 48 hours and as late as 18 months after the procedure. A sentinel self-limited bleed is observed in 35% to 50% of patients. Trauma from suctioning, particularly in the setting of abnormal coagulation, may also cause hemoptysis in patients with a tracheostomy tube or in those who have an endotracheal tube (ET) in place. The possibility of traumatic rupture of a pulmonary artery should be considered in patients with a pulmonary artery catheter in place (23,24).

**Physical Examination**

The physical examination may provide clues to the diagnosis of massive hemoptysis. A saddle nose deformity and/or septal perforation suggest Wegener granulomatosis. Stridor or unilateral wheezing indicates a possible laryngeal tumor, tracheobronchial tumor, or airway foreign body. Pulmonary embolism should be considered in patients with tachypnea,
a pleural friction rub, and lower extremity phlebitis. Diffuse
rales on examination raises the possibility of diffuse alveolar
hemorrhage, diffuse parenchymal lung disease, or cardiac dis-
ease as the cause of the hemoptysis. The presence of telangi-
cestasias of the skin or mucous membranes suggests hereditary
hemorrhagic telangiectasia or a connective tissue disease as the
cause. Ecchymoses or petechiae suggest a hematologic abnor-
mality or coagulopathy. Clubbing of the fingers may be a sign
of a lung carcinoma, bronchiectasis, and cystic fibrosis. The
finding of pulsation of the tracheostomy tube is of concern for
the development of a tracheoinnominate fistula.

Laboratory Studies

Laboratory studies, including a complete blood count (CBC),
coagulation studies, urinalysis, and chest radiograph, should
be obtained in all patients. The CBC may suggest an infectious
process or hematologic disorder as the cause of hemoptysis
and indicate the need for blood transfusion. Coagulation stud-
ies may provide evidence for a hematologic disorder as the
cause for the hemoptysis, or may identify a coagulopathy that
is causing or contributing to the bleeding from another dis-
 ease. Hematuria may be noted on urinalysis, which suggests
the diagnosis of Goodpasture syndrome, Wegener granuloma-
tosis, or another systemic vasculitis.

Chest Radiograph

The chest radiograph is an important study to identify the
cause and side of bleeding. The chest radiograph may dem-
onstrate abnormalities such as lung masses, cavitary lesions,
atelectasis, focal infiltrates, and diffuse infiltrate. Single or
multiple pulmonary cavities suggest neoplasm, tuberculosis,
fungal disease, lung abscess, septic pulmonary emboli, para-
sitic infection, or Wegener granulomatosis as the cause for
hemoptysis. The presence of a mass within a cavitary lesion
indicates a possible mycetoma (aspergilloma). The appearance
of a new air-fluid level in a cavity or infiltrate around a cavity
is suggestive of the site of bleeding. A solitary pulmonary
nodule that has vessels going toward the nodule may be an
arteriovenous malformation. Diffuse pulmonary infiltrates
suggest diffuse alveolar hemorrhage (Table 116.2), bleeding
from coagulopathy, lung contusions from blunt chest trauma,
hemorrhage with multiple areas of aspiration, or pulmonary
edema with a cardiac cause for hemoptysis. Chest radiographs
may be normal or nonlocalizing in 20% to 45% of patients
(25,26). Therefore, in patients presenting with hemoptysis, a
negative CXR warrants other diagnostic studies.

Computed Tomography

Computed tomography (CT) represents a noninvasive and
highly useful imaging tool in the clinical context of hemoptysis,
allowing a comprehensive evaluation of the lung parenchyma,
airways, and thoracic vessels by using contrast material. Mul-
tidetector CT (MDCT) may identify the bleeding site in 63%
to 100% of patients with hemoptysis (27); the role of CT in
the management of massive hemoptysis is however somewhat
controversial. CT may demonstrate abnormalities that are not
visible on the chest radiograph. It is helpful in the diagnosis of
bronchiectasis (28), although abnormalities from bronchiect-
sis can usually be appreciated on the chest radiograph. CT
with contrast may detect pulmonary emboli, thoracic aneu-
rysms, or arteriovenous malformations. CT scans may also
demonstrate cavitation with a surrounding infiltrate, the halo
sign, which suggests a necrotizing infection such as aspergil-
lossis or mucormycosis (29,30). Some studies have noted that
CT scanning before bronchoscopy may increase the yield of
bronchoscopy (31). In one retrospective study of 80 patients
with large or massive hemoptysis, chest CT was superior to
chest radiograph or bronchoscopy in determining the cause
of bleeding, and was similar to bronchoscopy in successfully
localizing the site of bleeding (32). CT is useful to create a
detailed and accurate map of the thoracic vasculature that may
guide further treatment, depicting the number and origin of
bronchial arteries and the coexistence of an additional non-
bronchial arterial supply. CT can thus assist in choosing ecto-
pic vessels amenable to embolization, preventing recurrence
after initial successful embolization, reducing angiography
procedure time, fluoroscopy radiation dose, contrast load, and
decreasing iatrogenic complications (12). Some authors have
argued that transport of the potentially unstable patient with
massive hemoptysis may not be justifiable, however; thus, the
patient should be adequately stabilized prior to obtaining a
chest CT.

Angiography

Angiography can determine the site of bleeding in 90% to 95%
of cases. However, in one case series, routine use of diagnostic
angiography provided a diagnosis not identified on bronchos-
copy in only 4% of patients (33). Angiography can be helpful
in detecting a pseudoaneurysm that has formed after healing
of a pulmonary artery tear from pulmonary artery catheter-
ization (34). As previously noted, the bronchial arteries and
other collateral systemic arteries account for the source of
bleeding in most cases with massive hemoptysis. Pulmonary
angiography is usually performed only when there is suspi-
cion for pulmonary aneurysms, arteriovenous malformations,
and pulmonary embolism. Technetium labeled red blood cell
or colloid studies rarely provided any information that is not
obtained by bronchoscopy and chest CT. The use and timing
of bronchoscopy will be discussed in a subsequent section.

Bronchoscopy

Bronchoscopy, performed with either a rigid or flexible endo-
scope, is helpful for identifying active bleeding and for checking
the airways in patients with massive hemoptysis. The capability
and success of bronchoscopy in localizing the bleeding site
may vary according to the rate and severity of the hemorrhage.
Hirschberg et al. (4) found that bronchoscopy was more effec-
tive in finding the bleeding site in patients with moderate to
severe hemoptysis (64% and 67%) than in those with mild
hemoptysis (49%). Bronchoscopy has an overall lower sensi-
tivity than MDCT in detecting the underlying causes of bleed-
ing (25,27,31). Nevertheless, bronchoscopy yields additional
information on endobronchial lesions and allows samples for
tissue diagnosis and microbial cultures.

Other Studies

Depending on the suspected causes of massive hemoptysis,
additional studies may be indicated. Echocardiography

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may be performed if a cardiac cause is considered. If diffuse alveolar hemorrhage syndromes are suspected, laboratory testing, including antiglomerular basement membrane antibody, antineutrophilic cytoplasmic antibody, antinuclear antibody, rheumatoid factor, complement levels, cryoglobulins, and antiphospholipid antibodies, should be performed, depending on the causes that are being considered. Transbronchial lung biopsy, open lung biopsy, or kidney biopsy may be indicated in some cases of alveolar hemorrhage to establish a diagnosis.

MANAGEMENT OF MASSIVE HEMOPTYSIS

Airway Protection and Stabilization

Once the diagnosis of massive hemoptysis is established, the initial priorities are to protect the airway and stabilize the patient. In general, the patient with massive hemoptysis should be monitored in the ICU setting, even if intubation and mechanical ventilation are not required. Large bore IV access should be established and supplemental oxygen provided. Blood should be drawn for a CBC, arterial blood gas analysis, coagulation studies, electrolytes, renal function tests, and liver function tests. The patient should be type and cross-matched for blood, with 4 to 6 units of packed red blood cells always available. Correction of thrombocytopenia and coagulopathy, if present, with appropriate blood products should be considered. Attempts to lateralize the site of bleeding should be made in anticipation of steps to prevent aspiration into the nonbleeding lung; the patient may be positioned in a lateral decubitus position with the bleeding lung down.

Airway patency must be ensured in patients with massive hemoptysis, as deaths from this process are predominantly due to asphyxiation. Most patients with ongoing massive hemoptysis will require intubation and mechanical ventilation, although select patients who are not hypoxemic and are able to keep the airway clear on their own may not require intubation. Although intubation generally preserves oxygenation and facilitates blood removal from the lower respiratory tract, the ET can become obstructed by blood clots, leading to the inability to oxygenate and ventilate the patient. The largest possible ET should be inserted to allow the use of bronchoscopes with a 2.8- to 3.0-mm working channel for more effective suctioning and to allow for better ventilation with the bronchoscope in the airway for prolonged periods of time. In severe cases, the mainstem bronchus of the nonbleeding lung can be selectively intubated under bronchoscopic guidance to preserve oxygenation and ventilation from the normal lung.

Some authors have recommended the use of a double-lumen ET to isolate the normal lung and permit selective intubation. Although double-lumen ETs have been used successfully in the airway management of massive hemoptysis, there are several potential pitfalls. First, placement of a double-lumen ET is difficult for less experienced operators, particularly with a large amount of blood in the larynx and oropharynx. Second, the individual lumens of the ET are significantly smaller than a standard ET and are at significant risk of being occluded by blood and blood clots. Last, positioning of the double-lumen ET and subsequent bronchoscopic suctioning of the distal airways require a small pediatric bronchoscope with working channels of 1.2 to 1.4 mm. Adequate suctioning of large amounts of blood and blood clots through such bronchoscopes is extremely problematic. In one series of 62 patients with massive hemoptysis, death occurred in four of seven patients managed with a double-lumen ET due to loss of tube positioning and aspiration (35). In general, we do not recommend the use of double-lumen ETs for airway management in massive hemoptysis. As an alternative to selective mainstem bronchial intubation or intubation with a double-lumen ET, an ET that incorporates a bronchial blocker, such as the Univent tube, may be used.

Localization of Source and Cause of Hemoptysis

Once the patient is stabilized and airway patency is achieved, the source of bleeding should be localized as precisely as possible, and the cause of bleeding determined. Identification of the cause and location of the bleeding potentially allows for more specific therapy. Methods of localization include patient history, physical examination, chest radiograph, chest CT, bronchoscopy, and angiography. In one study of 105 patients with hemoptysis, patients themselves were able to localize the side of bleeding in 10% of cases, but with an accuracy of 70% when able to do so (36); localization by a physical examination performed by a physician was possible in 43% of patients. Chest radiographs were able to localize bleeding in 60% of cases. Bronchoscopy was accurate in localizing the source of bleeding in 86% of patients. In another study, 9 of 24 patients were able to accurately localize the side of their bleeding (37). Chest radiographs should be routinely obtained to help localize the source of bleeding and determine the cause. As discussed earlier, chest CT may provide additional information beyond the chest radiograph, and may be more accurate in localizing the bleeding and determining the cause, although concerns about transporting a potentially unstable patient out of the ICU exist (7,38). Bronchoscopy and angiography remain the modalities for localizing the source of hemoptysis and offer potential therapeutic intervention.

Early—rather than delayed—bronchoscopy should be performed to increase the likelihood of localizing the source of bleeding. Bronchoscopy performed within 48 hours of bleeding onset successfully localized bleeding in 34% to 91% of patients, depending on the case series, as compared to successful localization in 11% to 52% of patients if delayed bronchoscopy was performed (39). Bronchoscopy performed within 12 to 24 hours may provide an even higher yield. Bedside flexible bronchoscopy should not be performed to establish a diagnosis of a tracheoarterial fistula such as a tracheoinnominate fistula (21,40).

Bronchoscopic Therapies to Control Hemoptysis

Rigid bronchoscopy is the most efficient means of clearing the airways from blood clots and secretions, ensuring effective tamponade of the bleeding airway and safe isolation of the nonaffected lung, thereby preventing asphyxia and preserving ventilation. However, it requires a trained bronchoscopist, who is not always readily available. A variety of maneuvers can be performed with the flexible bronchoscope to control bleeding.
Balloon Tamponade

Endobronchial tamponade via flexible bronchoscopy can prevent aspiration of blood into the contralateral lung and preserve gas exchange in patients with massive hemoptysis. Endobronchial tamponade can be achieved with a 4-Fr Fogarty balloon-tipped catheter. The catheter may be passed directly through the working channel of the bronchoscope, or the catheter can be grasped with biopsy forceps placed though the working channel of the bronchoscope prior to introduction into the airway of the bronchoscope and catheter. The catheter is held in place adjacent to the bronchoscope by the biopsy forceps, and both are then inserted as a unit into the airway. The catheter tip is inserted into the bleeding segmental orifice, and the balloon is inflated. If passed through the suction channel, the proximal end of the catheter is clamped with a hemostat, the hub cut off, and a straight pin inserted into the catheter channel proximal to the hemostat to maintain inflation of the balloon catheter. The clamp is removed, and the bronchoscope is carefully withdrawn (41–43). The catheter can safely remain in position between 15 minutes and 1 week, until hemostasis is ensured by surgical resection of the bleeding segment or bronchial artery embolization. It should be deflated for a few minutes three times a day, in order to preserve mucosal viability and to check for bleeding recurrence. Right heart balloon catheters have been used in a similar fashion (44). A modified technique for placement of a balloon catheter has been described using a guidewire for insertion. A 0.035-in soft-tipped guidewire is inserted through the working channel of the bronchoscope into the bleeding segment. The bronchoscope is withdrawn, leaving the guidewire in place. A balloon catheter is then inserted over the guidewire and placed under direct visualization after reintroduction of the bronchoscope (45). The use of endobronchial blockers developed for unilateral lung ventilation during surgery may hold promise for management of massive hemoptysis in tamponading bleeding and preventing contralateral aspiration of blood (46). The Arndt endobronchial blocker is placed through a standard ET and directly positioned with a pediatric bronchoscope. Suctioning and injection of medications can be performed through the lumen of the catheter after placement. The Cohen tip deflecting endobronchial blocker is also placed through a standard ET and directed into place with a self-contained steering mechanism under bronchoscopic visualization. At this time, there is limited published experience with these blockers in the setting of massive hemoptysis, although the author has successfully used them for this application.

Other Bronchoscopic Techniques

Additional bronchoscopic techniques may be useful as temporizing measures in patients with massive hemoptysis. Bronchoscopically administered topical therapies, such as iced sterile saline lavage or topical 1:10,000 or 1:20,000 epinephrine solution, may be helpful (47). Direct application of a solution of thrombin or a fibrinogen–thrombin combination solution has been used (48). The use of bronchoscopy-guided topical hemostatic tamponade therapy using oxidized regenerated cellulose mesh has recently been described (49). Endobronchial placement of a silicone spigot can prove adequate for temporary control of bleeding, allowing patients to stabilize before endovascular embolization (50). Successful tamponade and isolation of the bleeding site in patients with massive hemoptysis can be achieved by the placement of covered self-expanding stent orifice of the bleeding airway (51). Although anecdotal, the author has had success with topical application of a sodium bicarbonate solution.

For patients who have hemoptysis due to endobronchial lesions, particularly endobronchial tumors, hemostasis may be achieved with the use of neodymium:yttrium aluminum garnet (Nd:YAG) laser phototherapy, electrocautery, argon plasma coagulation (APC), or cryotherapy via the bronchoscope.

Angiography and Embolization

Angiography can identify the bleeding site in more than 90% of cases. As noted, the bronchial arteries are the most frequent source of bleeding in massive hemoptysis. In some cases, systemic vessels other than the bronchial arteries can be the source of bleeding (52). The pulmonary arteries may be the source for massive hemoptysis in 8% to 10% of cases (53). Visualization of extravasated dye from a vessel is relatively uncommon. Signs suggesting a particular vessel is the source of bleeding include vessel tortuosity, increased vessel diameter, and aneurysmal dilatation.

Bronchial artery embolization has been widely used to control massive hemoptysis; as a temporary measure to stabilize patients before surgical resection or medical treatment (antibiotics/antituberculous drugs) or as a definitive therapeutic approach in patients who refuse surgery, who are not considered as candidates for surgery (poor lung function, bilateral pulmonary disease, comorbidities), or patients in whom surgery is contraindicated. Bronchial artery embolization is considered the most effective nonsurgical modality for treatment of massive hemoptysis. The immediate success rates from bronchial artery embolization range from 51% to 100% (3,9,37,54–66). This wide range of success rates across multiple studies can be partially attributed to heterogeneity with regard to analysis of results with some series, including patients whom bronchial artery cannot be canialized or that spinal artery was seen coming off the bronchial vessel preventing embolization in the final analysis (61). Embolization has been performed with Gelfoam, polyurethane particles, polyvinyl alcohol particles, and vascular coils. Sclerosing agents may cause subsequent lung necrosis and should be avoided. Recurrence of bleeding, although usually nonmassive, has been noted in 16% to 46% of patients (9,54). Recurrence of hemoptysis may be due to incomplete embolization of the bronchial vessels, recanalization of the embolized arteries, presence of nonbronchial systemic arteries, or development of collateral circulation in response to continuing pulmonary inflammation (60,67). Repeat embolization may be required in some patients (37,59,63,68). Complications include chest pain, fever, vessel perforation and intimal tears, and embolization of material to mesenteric and extremity arteries. The most serious complication is embolization of the anterior spinal artery, which may arise from the bronchial artery, with subsequent spinal artery infarction and paraparesis; the risk of this occurrence is less than 1%.

Rupture of the Pulmonary Artery

The pulmonary artery may potentially be ruptured from right heart catheterization. This complication should be suspected...
in patients who develop hemoptysis with a pulmonary artery catheter in place. Balloon tamponade and contralateral selective intubation should be performed (69). The catheter should be withdrawn 5 cm with the balloon deflated, and the balloon then reinflated with 2 mL of air and allowed to float back into the ruptured vessel to occlude it. Patients who stop bleeding should undergo angiographic evaluation to localize the tear and identify the formation of a pseudoaneurysm (34,70). If a pseudoaneurysm is identified, embolization of the affected vessel should be considered to prevent subsequent hemorrhage.

Surgery

Emergency surgery for control of massive hemoptysis is performed less often due to the advent of bronchial artery embolization. Mortality rates for surgical management of massive hemoptysis range from 1% to 50% (3,71–76). Surgical resection of the source of bleeding offers definitive treatment as long as the lesion can be completely resected and the patient is able to tolerate resectional surgery. It is often difficult to accurately determine if these patients will be able to tolerate surgery; as they are often too ill to undergo pulmonary function tests, or are intubated and thus unable to perform pulmonary function tests. Surgical resection may be considered in patients when bronchial artery embolization is unavailable, if bleeding continues despite embolization, or if the cause of the hemoptysis is unlikely to be controlled with embolization. Surgery also remains the strategy of choice for the management of massive hemoptysis caused by diffuse and complex arteriovenous malformations, iatrogenic PA rupture, chest trauma, and mycetoma not responding to other therapeutic strategies, or associated with recurrent life-threatening hemoptysis as outlined above. Bronchovascular fistulae—with ensuing massive bleeding, is most often encountered following surgery, local infection, associated with vascular aneurysms and, less frequently, following lung transplantation surgery—are also managed by surgical repair once the patient is stabilized (1).

Diffuse Alveolar Hemorrhage

Patients with diffuse alveolar hemorrhage syndromes are not candidates for bronchial artery embolization or surgery; treatment for these groups of patients is pharmacologic. Corticosteroids are typically used and are effective for a wide range of the alveolar hemorrhage syndromes (77). Doses of 1 to 2 mg/kg/day of methylprednisolone have been most commonly used. For life-threatening alveolar hemorrhage, initial doses of 500 to 1,000 mg/day of methylprednisolone have been recommended. For Goodpasture disease, granulomatosis polyangiitis, and other vasculitides, adjunctive cytotoxic therapy or plasmapheresis may be considered.

PROGNOSIS

Factors associated with high mortality in patients with massive hemoptysis include a bleeding rate of at least 1,000 mL within a 24-hour period, aspiration of blood in the contralateral lung, massive bleeding requiring single lung ventilation, and broncho(genic carcinoma as an underlying etiology (3,6). Patients seem to fare better when tuberculosis, bronchitis, or bronchiectasis are responsible for the massive hemoptysis. Patients who experience recurrent bleeding following embolization for massive hemoptysis have significantly higher mortality (78). Overall mortality rates for massive hemoptysis range from 9% to 38% (58,61,79), with significant reduction in mortality in recent years since bronchial artery embolization is considered as first-line therapy.

INTERVENTIONAL BRONCHOSCOPY

Interventional bronchoscopy is a field that utilizes minimally invasive techniques for the management of a variety of tracheobronchial disorders. There are a number of circumstances for which interventional bronchoscopy has application in the ICU. The most common need for such procedures arises from central airway obstruction (CAO), both malignant and benign in etiology. Other potential situations that interventional bronchoscopy may be of benefit include management of hemoptysis and management of persistent airleaks.

Malignant Airway Obstruction

CAO from malignancy may arise from endoluminal tumor, submucosal tumor infiltration, extrinsic compression by a tumor mass, extrinsic compression by malignant mediastinal adenopathy, or a combination of these pathologies. Bronchogenic carcinoma is the most common cause of malignant airway obstruction. The exact prevalence of airway obstruction in patients with lung cancer is not clear although it has been estimated that 20% to 30% of patients will develop large airway obstruction (80). Patients with other malignancies may also develop endobronchial metastases. Cancers of the thyroid, colon, breast, kidney, and esophagus as well as melanoma have been most commonly noted to cause endobronchial metastases. As a result of the airway obstruction, patients may experience respiratory distress, hypoxemia, or frank respiratory failure requiring mechanical ventilation. Postobstructive pneumonia may also occur.

Benign Airway Obstruction

Patients may develop tracheal stenosis or tracheal webs following endotracheal intubation. The reported incidence ranges from 10% to 22%, although only 1% to 2% of patients are symptomatic or have severe stenosis (81). Most stenoses occur at the site of the ET cuff, thought to be due to decreased regional blood flow as a result of pressure of the cuff on the tracheal wall. A similar incidence has been reported following tracheostomy tube placement (82). While patients may develop stenoses or webs at the site of the tracheostomy tube cuff, tracheal stenosis following tracheostomy tube placement typically occurs around the tracheal stoma site. This is thought secondary to abnormal wound healing with excess granulation tissue formation around the tracheal stoma (81). In addition, patients may develop focal tracheomalacia at the level of the stoma secondary to cartilaginous damage (dynamic A-shaped tracheal stenosis) (83). Patients may also develop airway obstruction secondary to granulation tissue above the tracheostomy tube and at the tip of the tracheostomy tube. Finally, patients may also develop tracheal or bronchial stenosis as a consequence of systemic disease, such as granulomatous polyangiitis (Wegener granulomatosis), relapsing polychondritis, sarcoidosis, and tracheobronchial...
amylloidosis. In about 3% to 5% of cases of tracheal stenosis, there is no known inciting process for the development of the stenosis and such cases are labeled idiopathic (84).

Expiratory central airway collapse—comprises two separate entities: tracheobronchomalacia (TBM), characterized by weakness of the airway cartilages, and excessive dynamic airway collapse (EDAC)—which is defined as excessive bulging of the posterior membrane into the airway lumen during expiration without cartilage collapse—may produce significant airway obstruction (85). Focal tracheomalacia may result from prolonged intubation, tracheostomy tube placement, vascular abnormalities such as vascular rings, and space occupying lesions of the mediastinum such as a large thyroid goiter. Focal tracheomalacia and bronchomalacia can occur following radiation therapy; diffuse TBM can result from relapsing polychondritis, and EDAC is associated with COPD, asthma, and obesity.

**History and Evaluation**

Symptoms depend on the location and degree of airway narrowing as well as concurrent thoracic pathology. Dyspnea is the most common symptom. Dyspnea on exertion typically occurs when the tracheal diameter is reduced to 8 mm; stridor may be noted when the tracheal diameter decreases to 5 mm (86). Airway narrowing of this degree increases susceptibility to acute obstruction from mucus plugs or blood clots. This is thought to be the reason why 50% of these patients present with acute respiratory distress. CT may be used as the initial evaluation to be the reason why 50% of these patients present with acute respiratory distress. CT may be used as the initial evaluation to determine the precise anatomical location, the characteristics of the lesion, and the extent of disease, including distal airway patency and local vascular anatomy (87,88). Multiplanar reformations in sagittal, coronal, or oblique planes eliminate the known limitation of axial images including detection of subtle airway stenoses, underestimation of the longitudinal extent of narrowing, inadequate evaluation of the airways oriented obliquely to the axial plane, and the difficulty in displaying complex three-dimensional anatomy of the airways. In addition, virtual bronchoscopy imaging can be performed using CT images constructed during postprocessing. Flexible bronchoscopy can be used as a diagnostic modality although ideally bronchoscopy, either flexible or rigid, is performed in conjunction with a bronchoscopic therapeutic intervention. Caution should be taken in performing bronchoscopy in high-grade tracheal or bilateral mainstem obstruction if an interventional pulmonologist/thoracic surgeon is not available as a diagnostic bronchoscopy may precipitate acute, complete airway occlusion.

**Interventional Bronchoscopy Techniques**

Various interventional bronchoscopy modalities are available to manage malignant or benign CAO. Mechanical debulking and dilatation may be performed with a rigid bronchoscope to relieve obstruction from endoluminal tumor as well as benign stenoses. Rigid bronchoscopists may also be used in conjunction with other ablative modalities, and has some advantages over interventions performed via flexible bronchoscopy including the ability to ventilate the patient through the rigid bronchoscope, use of larger suction catheters to manage blood in the airway, and use of larger forceps to remove tumor and debris. A variety of ablative techniques are available for relieving obstruction from endoluminal tumor (Table 116.3). Laser, electrocautery, and APC utilize heat thermal energy for tissue destruction. Microdebriders have been used in conjunction with rigid bronchoscopy for mechanical debulking; these

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<td>Nd:YAG laser</td>
<td>Noncontact, thermal energy from laser light</td>
<td>Deep tissue penetration and hemostasis, good for major tissue debulking</td>
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<td>Nd:YAP laser</td>
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<tr>
<td>Argon plasma coagulation</td>
<td>Noncontact, thermal energy from ionized argon gas</td>
<td>Good hemostasis, preferential flow to uncoagulated tissue, bends around corners</td>
<td>Shallow penetration, not ideal for major tissue debulking</td>
</tr>
<tr>
<td>Electrocautery</td>
<td>Contact, thermal electrical energy</td>
<td>Inexpensive, multiple accessory types for different situations, good hemostasis</td>
<td>Requires frequent cleaning of contact device, less precision than laser</td>
</tr>
<tr>
<td>Cryotherapy</td>
<td>Repeated cycles of freezing and thawing</td>
<td>Good for foreign body removal, no risk of airway fire</td>
<td>Not for acute airway use due to delayed tissue destruction, requires follow-up bronchoscopic tissue removal</td>
</tr>
<tr>
<td>Brachytherapy</td>
<td>Direct implantation of radiation source into/next to target lesion</td>
<td>Concentrated, long lasting, localized tissue effect</td>
<td>Not for acute airway use due to delayed tissue destruction, higher risk of hemorrhage and other complications</td>
</tr>
<tr>
<td>Photodynamic therapy</td>
<td>Preferential uptake of photosensitizer by malignant cells with nonthermal laser-activated phototoxic reaction</td>
<td>Potentially curative for early mucosal squamous cell cancers</td>
<td>Not for acute airway use due to delayed tissue destruction, requires follow-up bronchoscopic tissue removal, 4-6 wks of skin photosensitivity</td>
</tr>
<tr>
<td>Microdebrider</td>
<td>Mechanical removal of tissue by rotating blade and suction</td>
<td>No risk of airway fire</td>
<td>Device length limits to proximal airway use</td>
</tr>
</tbody>
</table>

debriders are composed of a serrated blade rotating at 1,000 to 3,000 rpm attached to a hollow suction tube (89). These modalities, as well as mechanical debulking with the rigid bronchoscope, have the advantage of achieving immediate airway patency. Either silicone or self-expanding metal stents will often be placed to maintain airway patency once the airway is de-obstructed. Other techniques such as standard cryotherapy, brachytherapy, and photodynamic therapy have the disadvantages of not achieving immediate airway patency and thus would not be the procedures of choice for patients in the ICU with CAO. A modification of the method of cryotherapy, referred to as cryorecanalization, is able to achieve immediate airway patency, however (90).

For benign tracheal or bronchial stenosis, dilatation with the rigid bronchoscope or balloon bronchoplasty via rigid or flexible bronchoscopy may be performed (91). In some cases, laser, electrocautery, or APC may be used as an adjunct to mechanical/balloon dilatation (92,93); some patients may require future repeat procedures. For patients who require repeated dilatation procedures, surgical intervention should be considered (94). If surgery is not feasible or contraindicated due to medical comorbidities, stent placement may be considered, although some clinicians prefer a trial of temporary stent placement before considering surgery. In general, silicone stents should be used for benign stenoses although the authors have had good success with the use of fully covered, self-expanding, metal stents that can be easily removed, similar to silicone stents.

For patients with TBM and EDAC causing respiratory failure, stent insertion can be considered to allow liberation from mechanical ventilation. Complications related to long-term stent placement in these patient populations are not insignificant however. Self-expanding metal stents can potentially fracture and cause granulation tissue. If uncovered metallic stents are used, removal at a later time if complications develop can be challenging. Silicone stents do not have issues with fracturing, but can cause granulation tissue and can migrate if a standard tracheal stent is used; a silicone Y stent is most commonly used in these patients. Consideration should be given for surgical management with tracheobronchoplasty once the patient is stabilized (87). Patients with focal tracheal cartilaginous malacia due to prolonged intubation or tracheostomy tube should typically not be managed with stent placement, as the need for the stent will be lifelong, and eventual complications are likely. Patients should be evaluated for resection of the pathologic segment and end-to-end anastomosis (95). If the patient is not a candidate for surgery, placement of a tracheostomy tube to bypass the segment may be required.

**Outcomes for Airway Interventions in the ICU**

Interventional bronchoscopy procedures can be successful in allowing patients to be liberated from mechanical ventilation (Table 116.4). Likewise, in a study by Noppen et al. 15 patients, who were ventilator/artificial airway dependent patients, were treated for airway obstruction after being referred for failed attempts at weaning from mechanical ventilation or from their tracheostomy cannula (96). All patients had benign disease, with most patients having postintubation tracheal stenosis or tracheomalacia. Median duration of mechanical ventilation prior to referral was 30 days (range, 7–105 days); 14 of the 15 patients were extubated/decannulated immediately following the intervention. Similarly, 36 patients with respiratory failure or impending respiratory failure due to malignant airway obstruction were treated emergently by Jeon et al. (97); of the 36 patients, 34 had a successful outcome. Overall survival ranged from 3 days to 69 months with a median of 23.6 months. Interventional bronchoscopy can have a significant impact on critically ill patients with CAO and may allow for successful withdrawal from mechanical ventilation, hospitalization in a lower level of care environment, relief of symptoms, and extended survival.

**Key Points**

- Massive hemothysis is defined as expectoration of blood exceeding 200 to 1,000 mL in 24 hours.
- Bleeding in massive hemothysis originates from bronchial and pulmonary arteries in 90% and 5% of cases respectively.
• CT scan with contrast helps localize bleeding in 63% to 100% of cases with massive hemoptysis and should be the investigation of choice if patients can tolerate the transport and are stable enough.
• The initial priority in management should be to protect the airway and stabilize the patient.
• Endobronchial balloon tamponade can be used to prevent aspiration of blood into the contralateral lung, preserve gas exchange, and can be used as a temporizing measure until more definitive management is instituted.
• Bronchial artery embolization is the most effective nonsurgical modality for treatment of massive hemoptysis, with success rates varying between 51% and 100%.
• Mortality rates for surgical management of massive hemoptysis range from 1% to 50% and should be considered only as a salvage therapy in the current era of minimally invasive approaches, that is, embolization and advanced bronchoscopic therapies.
• Caution should be taken in performing bronchoscopy in high-grade tracheal or bilateral mainstem obstruction if an interventional pulmonologist/thoracic surgeon is not available, as a diagnostic bronchoscopy may precipitate acute, complete airway occlusion.
• Interventional bronchoscopy can have a significant impact on critically ill patients with CAO and may allow for successful withdrawal from mechanical ventilation, hospitalization in a lower level of care environment, relief of symptoms, and extended survival.

References


