

Key Points

- With massive hemoptysis, death is usually caused by asphyxiation rather than by exsanguination.
- Urgent management focuses on the prevention of asphyxia while the source of bleeding is addressed.
- Endobronchial and/or angiographic control are usually possible.
- Bronchial artery embolization (BAE) is now the treatment of choice.
- There is now less indication for surgery, and surgical results are better in stabilized, “elective,” non-bleeding patient.
- Pulmonary artery injury is rare but associated with high mortality and is curable with BAE.

Introduction

Massive hemoptysis (MH) is a medical emergency that places the patient at risk of asphyxiation and death. Because of the explosive clinical presentation of MH, it is essential to respond quickly and appropriately. This is a potential lethal condition that deserves to be investigated thoroughly and brought under control promptly [1].

The definition of MH may vary depending of the publications. It is usually based on the volume of blood expectorated. In the literature, we can find large variations between

the definitions. They range from 100 to 1000 mL/24 h [2–4]. By consensus (Table 44.1 and Fig. 44.1), MH is defined as a rate of bleeding exceeding 600 mL/24 h, meaning 25 mL/h. Only 1.5–5% of hemoptysis are really massive [1]. Exsanguinating hemoptysis is defined as a bleeding rate exceeding 150 mL/h or blood loss over 1000 mL/24 h or over 300 mL during one expectoration [5].

Quantification of MH may be difficult, and from a clinical point of view, such criteria are not useful. Based on the fact that the anatomic dead space of the major tracheobronchial tree is about 200 mL in most adults [6], other definitions relying on the magnitude of the clinical effects have been proposed. MH can be defined as the volume of expectorated blood that is life-threatening mainly by virtue of airway obstruction and rarely by blood loss.

Table 44.1 Definition of hemoptysis

Hemoptysis	Massive	Exsanguinating
One expectoration (mL)		>300
mL by hour	>25	>150
mL by 24 h	>600	>1000



Fig. 44.1 Evaluation of the severity of hemoptysis. From left to right: a teaspoon = 5 cc, a medicine cup = 60 cc, a kidney basin = 650 cc

J. S. Bussi eres (✉)
 Department of Anesthesiology, Institut Universitaire de
 Cardiologie et de Pneumologie de Quebec – Universit e Laval,
 Quebec City, QC, Canada
 e-mail: jbuss@criucpq.ulaval.ca

M. Frenette
 Department of Anesthesiology and Critical Care, Universit e Laval,
 Quebec City, QC, Canada

Massive hemoptysis compared to moderate or minor hemoptysis represents a higher risk of mortality for the patient. Published series on patients presenting massive hemoptysis showed a mortality rate with medical management varying from 12% to 50%. Mortality rate was greater than 50% in patients not treated adequately [1].

Decision-making is a multidisciplinary process involving a critical care physician, pulmonary medicine bronchoscopist, interventional radiologist, thoracic surgeon, and anesthesiologist.

Historical Considerations

MH treatment was at first exclusively surgical. From the 1940s to the 1960s, different surgical approaches have been used to control and treat MH. In 1973, Remy and colleagues [7] changed management forever with the first report of bronchial artery embolization. Hiebert in 1974 [8] described the first successful use of a Fogarty balloon catheter through a rigid bronchoscope to tamponade bleeding in a moribund patient with massive bronchial hemorrhage. Subsequently, the use of the bronchoscope progressively changed from a rigid device to the flexible fiber-optic bronchoscopy (FOB). More recently, the role of early bronchoscopy, mainly with a FOB, is questioned since bronchial artery embolization (BAE) is easy to perform and so effective.

Since the introduction of radiological embolization, there are less frequent interventions by anesthesiologists for massive hemoptysis. With the rapid use of BAE, potentially massive hemoptysis is rapidly controlled, and there is less and less need for surgical interventions (emergent, semi-emergent, or elective post treatment), and therefore there is less anesthesia management. Consequently, anesthesiologists are nowadays only occasionally required to help with airway management, lung protection, and assistance during radiological intervention.

The literature usually refers to old publications, and there are only retrospective series or anecdotic reports that support recommendations for investigation and a treatment plan for MH. It is easy to understand that it is very difficult to design a prospective, randomized, controlled trial in this type of population.

Etiologies

Hemoptysis is defined by coughing of blood that originates from the tracheobronchial tree or pulmonary parenchyma. There are many potential causes of massive hemoptysis. It is very important to rapidly rule out any non-pulmonary bleeding since it can originate from the nasopharynx or from the upper gastrointestinal tract.

The lungs have a dual blood supply, the pulmonary circulation and the bronchial arteries. The bronchial circulation is a high-pressure system providing only 1% of the arterial supplies to the lungs, but the bronchial tree is implicated in MH in more than 90% of cases. Bronchial arterial bleeding is distinctively brisk and bright red. Bronchial arteries originate from the aorta that brings nutrients to the lung parenchyma and major airways. Seventy percent of the bronchial arteries typically arise from the descending thoracic aorta in regard to the fifth and the sixth vertebrae. The 30% remaining arise from other locations such as the subclavian arteries [9]. The same vessels that supply the bronchial arteries may also supply the esophagus, the mediastinal nodes, and, more importantly, the spinal cord, through a complex anastomotic network [10].

Pulmonary arterial circulation is responsible for gas exchanges and is involved in less than 5–10% of massive hemoptysis. The pulmonary bed is a high-compliance and usually a low-pressure system (15–20 mmHg systolic and 5–10 mmHg diastolic). However, if there is pulmonary artery hypertension, this low-pressure system may be modified to a high-pressure system, reaching sometimes the systemic level. Dark blood is more consistent with pulmonary artery bleeding since the blood is not sufficiently oxygenated.

Hemoptysis originates from systemic, namely, bronchial, or from pulmonary vessels. The most frequent causes of bronchial tree hemoptysis are inflammatory lung disease (bronchiectasis and tuberculosis) and neoplasia (Table 44.2). The most common causes of hemoptysis from pulmonary circulation are arteriovenous malformation (AVM) and Rasmussen's aneurysms (due to tuberculosis). Iatrogenic

Table 44.2 Possible causes of massive hemoptysis

Infectious
Bronchiectasis (including cystic fibrosis)
Chronic bronchitis
Tuberculosis
Non-tuberculous mycobacteria
Lung abscess
Necrotizing pneumonia
Mycetoma
Cardiovascular
Arteriovenous malformation
Pulmonary embolism or infarct
Mitral stenosis
Aortic aneurysm or bronchovascular fistula
Vasculitis, Wegener's granulomatosis
Neoplastic
Lung cancer
Bronchial adenoma
Pulmonary metastases
Miscellaneous
Aspirated foreign body
Pulmonary contusion, trauma
Idiopathic pulmonary hemosiderosis
Iatrogenic (transthoracic or transbronchial biopsy, pulmonary artery catheter)

pulmonary artery rupture from pulmonary catheter (Swan-Ganz) occurs rarely, but since anesthesiologists are frequently implicated in its causality, this subject will be discussed more deeply in another section of this chapter.

Clinical Manifestation

In the acute phase of hemoptysis, there is an accumulation of blood in the dependent parts of the lungs. Through the cough reflex, blood is expelled, producing the hemoptysis. During that time, blood can be dispersed bilaterally into the bronchial tree, and the evaluation can mislead to lateralization.

The degree of bleeding may be easily underrated because the volume of blood engulfing the involved lobes or lungs is not quantified and may be significant [1]. Many patients with hemoptysis have a medical history of compromised lung function, and even small quantities of blood into the bronchial tree can lead to significant respiratory distress. Expectored blood is often swallowed and cannot be measured. Asphyxia is the most life-threatening manifestation of MH, well before hemodynamic instability appears, and is the usual cause of death associated with MH.

Initial Management of MH

There is little consensus regarding the optimal management of patients presenting with MH. Moreover, from the beginning of the twenty-first century, there are few recent large series of patients studied [11]. In this section, conclusions of these studies, some consensus from the literature, and also various controversies will be presented.

Initial management of massive hemoptysis needs to achieve a few objectives quickly and simultaneously. The initial step in management of hemoptysis is to differentiate between minor and massive hemoptysis. The approach to the patient presenting MH can be generally done in three steps [12]: airway protection, localization, and treatment. The initial approach to the patient should be dictated by the clinical presentation. Patients with rapid bleeding or severe functional decompensation need protection of the airway first, meaning that every effort should be made to protect the non-affected lung against blood spillage and to maintain adequate gas exchange to prevent asphyxia [13]. Hypoxia secondary to lung spillage or blood clots is the main cause leading to death. Secondly, it is essential to localize the source of bleeding, meaning finding as precisely as possible its origin or at least the side of the MH. Thirdly, the administration of a specific therapy is mandatory. Definitive treatment options include conservative medical therapy, endobronchial therapy, arterial embolization, or surgery.

All patients presenting MH should be admitted to an intensive care unit for further investigation and treatment or transferred to the radiological suite for CT or angiogram examination or, less frequently, directly to the operating room. While undergoing diagnostic procedures, the patient should be kept upright, and 100% oxygen should be administered. Appropriate venous access should be put in place, and blood should be available from the blood bank. A coagulation profile should be obtained to show any coagulopathy, including platelet dysfunction from drugs such as acetylsalicylic acid (Aspirin®), clopidogrel (Plavix®), and other antiplatelets and new anticoagulants. Every effort should be made to reverse anticoagulation, when possible. Invasive therapeutic measures are not indicated for the control of hemoptysis caused by anticoagulant therapy, blood dyscrasia, or Goodpasture's syndrome [1]. Blood loss is rarely massive enough to cause a great threat to hemodynamics. Slight hypotension can occur and may be treated with volume replacement.

Light sedation with anxiolytic drugs or cough suppression drugs is rarely useful in the acute phase of MH [13]. Once the immediate danger has passed and bleeding is settling, these drugs may be used to depress the excessive coughing that can aggravate or stimulate hemoptysis [6]. Bronchodilators cannot be administered since they can have a vasodilation effect and may precipitate renewed bleeding [14].

Life-Threatening Intervention

When facing MH, many strategies to prevent airway contamination should be available. If lung isolation is delayed or not available, the patient intubated or not should be placed in the lateral decubitus position with the bleeding lung on the dependent (inferior) side to prevent spillage to the unaffected lung (nondependent, superior side).

Lung isolation may be used to avoid spillage to the unaffected lung by blood from the bleeding lung. Lung isolation can be performed with different techniques including a selective endobronchial intubation with a standard endotracheal tube, the use of a bronchial blocker (BB), or the insertion of a double-lumen tube (DLT). The two last lung isolation techniques are not specific to hemoptysis as they are regularly used during anesthesia for thoracic surgery [15] (see Chap. 16).

Main stem bronchial selective intubation with a large uncut endotracheal tube is facilitated using FOB, if the visualization is good enough to allow the guidance of the tube. Unfortunately, blood and clots can obstruct the view from FOB. Blood highly absorbs the light of the FOB and consequently alters its capacity to identify the tracheal bifurcation and the guidance of the endobronchial tube. Blind endobronchial insertion may be attempted and verified by auscultation.

However, the presence of large amount of blood in the main airway may confuse the sound perceived unilaterally.

Bronchial blockers (BBs) may be used for lung isolation or act as a therapeutic avenue when facing MH. It can be a temporary measure in life-threatening situations until a more specific treatment is applied. Bronchial blockers may be inserted into a bleeding bronchus under the control of a FOB to induce an endobronchial tamponade. Bronchial blocking device is useful for lung separation when the DLT is not immediately available or when there is some difficulty in inserting the DLT (e.g., percutaneous tracheotomy) [16]. As for any other lung isolation devices, the use of FOB is mandatory, but the airway visualization may be difficult because of the presence blood and blood clots. BB can be used in a non-intubated (through nostrils) or intubated patient. It can be positioned and stabilized alongside or inside the lumen of the endotracheal tube or DLT. In some situations, the BB may be introduced through a DLT. At that time, the use of very small FOB (2.8 mm) may be necessary, mainly with a smaller-sized DLT (35–37 Fr). The Uniblocker BB (Fuji Systems Corporation, Tokyo, Japan) is easy to use compared to other models, and it is now the golden standard for that procedure.

It is an excellent method to achieve control of the bleeding and to protect the contralateral lung and potentially the ipsilateral non-bleeding lobes or segments. The technique of endobronchial tamponade for bleeding control in MH was first introduced by Hiebert in 1974 [8]. The author occluded a bleeding bronchus with a balloon catheter inserted through a rigid bronchoscope. Different catheters have been used for this application, Foley catheter, Fogarty catheter, Swan-Ganz catheter, specific double balloon catheter, and more recently bronchial blockers. In addition to the tamponade effect, the administration of vasoactive drugs is possible through the inner channel [17]. This gives time to proceed with a therapeutic and more definitive intervention. If possible, the BB should be replaced by a DLT or a simple lumen tube to allow further evaluation and suctioning of the bleeding lung. The bronchial blocker should always be deflated under FOB vision. In 2006, Giannoni et al. [18] described a bilateral concurrent massive hemoptysis successfully controlled with the placement of more than one balloon catheter. In 2017, Caddell reported a case of an emergency surgical pulmonary embolectomy complicated by an acute massive hemoptysis [19, 20]. The authors used a double bronchial blocker system with a sequential inflate and deflate technique to localize the hemorrhage and provide lung isolation and ventilation.

A third alternative for the management of the airway during MH is the placement of a double-lumen tube (DLT) or endobronchial tube. These DLTs are specially designed for selective intubation of the right or left main stem bronchi [21]. For MH, selective intubation of the left lung is preferable. The left-sided tube is easier to position than the right-

sided tube which carries the risk obstructing the right upper lobe bronchus. Double-lumen tubes have a bad reputation in the literature when used in the context of MH. Nevertheless, some of these observations were published before [22] the introduction of the polyvinyl DLTs whose positioning can be verified with the FOB. Other publications [1] refer to the use of DLTs for other conditions than MH, and their conclusions cannot be used to determine the safety of DLTs during MH [23]. However, it has been demonstrated that anesthesiologists with limited experience in thoracic anesthesia frequently fail to successfully place lung isolation devices, DLT, or bronchial blockers [24].

It is the authors' opinion that inserting a left-sided double-lumen tube is a good strategy. Many problems with DLT were described with older nondisposable model of DLT or with older models of FOB. When confronted with massive hemoptysis, the use of polyvinyl DLT and new FOB with larger suction channel can be helpful. The lumen inserted into the non-bleeding lung is used to ventilate the patient. The other lumen, connected to the bleeding lung, allows the insertion of a specific FOB. These FOBs, with a diameter less than 4.2 mm, have a working channel allowing suction of blood and blood clots from the bleeding site. The lumen directed to the bleeding lung may be used to carry out a relatively "blind" and very careful catheter aspiration. With attentive care, the lumen may be cleared of blood or clots. The application of CPAP or mechanical ventilation with PEEP on the bleeding lung may help to decrease the bleeding and/or to improve the patient's gas exchange.

Nevertheless, insertion of a DLT may be difficult, even for an experienced anesthesiologist, because large amounts of blood can make the visibility poor with FOBs. Also, a well-positioned DLT may be easily displaced during the frequent transfers of the patient from ICU to radiologic suites or frequent patient's repositioning in bed. The authors prefer to use left-sided DLT with a carinal hook to stabilize the DLT onto the carina and fix it to the maxillary bone to minimize its displacement. Sometime, when facing a massive hemoptysis, the use of a carinal hook helps to "blindly" position a DLT. It is important to note that BBs are at higher risk of dislodgement with movement or transfer of the patient than DLTs. Consequently, patients with a DLT or a BB usually should not be moved unless in absolute necessity, and sometime these patients should be with muscle relaxants to prevent coughing until the hemoptysis has been treated [25, 26].

Once adequate lung isolation is achieved, the patient can be placed in the lateral decubitus position with the bleeding lung on the nondependent (superior) side. The dependent non-bleeding lung will thus receive most of the pulmonary blood flow, and this will help to control the hemorrhage, as it decreases the perfusion in the upper lung. This position will also improve ventilation/perfusion (V/Q) ratio. The patient should be ventilated with 100% oxygen, and PEEP should be

applied on the inferior lung to improve gas exchange. PEEP or CPAP may also be used on the upper bleeding lung acting as a hemostatic effect and help gas exchange.

Diagnostic Tools and Therapeutic Approaches

There is a controversy regarding the sequence of bronchoscopic and radiological interventions. Initial computed tomographic scan (CT) is thought to shorten examination time in critical patients, but in a patient with massive hemoptysis, etiologic diagnosis is less important than immediate interruption of the bleeding process [27].

Chest radiography is readily available and is an important diagnostic tool in finding the cause of the bleeding and localizing pulmonary pathology. High-definition computed tomographic (HDCT) scanning angiography is also an excellent diagnostic tool. Except for life-threatening situations, CT scan should be performed before the bronchoscopic exploration. It has superior diagnostic capacity over bronchoscopy and chest radiography for demonstrating underlying pathology and the site of bleeding in hemoptysis, especially in bronchiectasis, bronchogenic carcinoma, and aspergilloma cases. Vascular pathologies such as arteriovenous malformation or aneurysm, which are rare causes of hemoptysis, are also depicted very clearly in contrast-enhanced CT scan examinations [13]. With recent developments in multidetector CT scan technique, it is now possible to scan the whole thorax into very thin slices (1.25 mm) in a very short time (12–15 s) [14, 15]. Both the lesion causing hemoptysis and bronchial or nonbronchial systemic feeding arteries are detected during the same study using 80–100 mL of contrast medium. In angiography-controlled studies, 86–87% of the pathologic vessels detected by angiography were discovered with CT angiography (CTA) [14, 15].

Bronchoscopy

For a patient presenting MH, the medical team may choose to proceed with early or late bronchoscopy. This bronchoscopy may be rigid or flexible, and its primary goal is to localize the site or at least lateralize the side of the bleeding source. The secondary goal is to clear the airway of gross blood. Finally, the third goal may be to use a therapeutic agent to control the bleeding. If the situation is not critical, a quick trial of fiber-optic bronchoscopy (FOB) can be performed to determine the origin or at least the side of the bleeding. If the patient's oxygenation is significantly compromised or the bleeding continues at a brisk pace, elective oral intubation with an endotracheal tube (8.0 mm or larger)

should be performed; this may be done simultaneously with the bronchoscopy [28].

Timing

Although most authorities advocate bronchoscopy to help localize bleeding during MH, the moment when to use the bronchoscopy is still controversial [12]. In the literature, it is frequently mentioned that patients with MH require urgent bronchoscopy. The argument for this assertion is that bleeding will increase with time, making visualization difficult. It is reported that bronchoscopy helps to detect the bleeding site in a lung or lobe in patients with diffuse pulmonary disease [10].

More recently a new option emerged. Patients presenting with MH are immediately directed to the angiographic suite to get a diagnostic angiogram and bronchial artery embolization (BAE) at the same time. Patients at risk of asphyxia prior to the BAE benefit from lung isolation techniques. The FOB is then carried out a few days later. The main argument for this sequence is that during the acute phase of severe MH, airways are filled with large volumes of blood restricting the use of bronchoscopy and consequently invalidating endobronchial treatment [7, 29]. Sometimes, endoscopic examination may aggravate bleeding and delay more effective treatment. Hsiao et al. reported in 2001 [30] that bronchoscopy was not a prerequisite in the treatment process considering risk of airway compromised from sedation, delay in definitive treatment, hypoxemia, and high cost [11]. In this study, bronchoscopy findings were taken into consideration whenever they were available. Not having to perform a bronchoscopy did not affect the progress of endovascular treatment. Bronchoscopy findings have not altered the course of angiography and endovascular treatment in any of their patients. In this observational retrospective study of 28 patients, there was no need for emergency bronchoscopy during active and abundant bleeding.

In a retrospective study of 28 patients with massive hemoptysis, flexible bronchoscopy successfully localized the site of bleeding in 26 patients [2]. In comparison, chest radiographs localized the bleeding site in 23 patients. This suggests that flexible bronchoscopy is effective at identifying the site of bleeding in patients with massive hemoptysis, but localizing a radiographic abnormality is sufficiently accurate to warrant proceeding to bronchial artery embolization without bronchoscopy [28].

Despite the “lack of proof” showing that early bronchoscopy is beneficial, the general expert consensus favors an early bronchoscopy, especially for patients with massive hemoptysis [3]. The early procedure provides clinicians with the maximal amount of information upon which to base future decisions, particularly in patients who develop sudden recurrence or acceleration of their bleeding. Nevertheless,

early bronchoscopy has not been strictly proven to improve outcome [28].

Practically speaking, early bronchoscopy should be done within the first 12–18 h for the patient who is clinically stable or for whom bleeding has become quiescent. Alternatively, bronchoscopy is performed as early as it is safely feasible on the unstable, decompensating patient [28]. In some institutions, in patients with MH who are unstable, diagnostic angiography is the imaging method of choice to localize the bleeding site because it allows for immediate treatment [31].

Type of Bronchoscopy

Depending on each institution's local practice, rigid or flexible bronchoscopes are used to evaluate and to stabilize any patient presenting MH. The selection is likely to reflect the institution's or the user's experience [12]. No study addressed this issue.

Rigid

Rigid bronchoscopy has been, until recently, the procedure of choice after initial chest radiography. Many surgeons and much of the older literature strongly advocate the use of the rigid bronchoscope. Rigid bronchoscopy is preferred because of its ability to suction large quantities of liquid and clotted blood, to use a great variety of therapies during bronchoscopy, such as direct cauterization or packing of bronchial lesions, and to continuously provide ventilation. Obviously, operating room setting and general anesthesia are required. By contrast, the visual range of inspection is significantly reduced compared to the fiber-optic bronchoscopy.

Changing over time, the technique of rigid bronchoscopy is being less and less used and, as a result, is not available in many institutions [13]. Consequently, a rigid bronchoscope is usually used for patients with ongoing massive hemoptysis after an unsuccessful bedside fiber-optic bronchoscopy. The flexible bronchoscope can also be used in conjunction with the rigid bronchoscope by passing it through its lumen. This allows a better examination of the more distal and upper lobe airways [28].

A survey in 1998 noted that 79% of physicians treating massive hemoptysis favored the flexible optic bronchoscope (FOB) as the initial technique compared to 48% in a similar survey performed in 1988 [32, 33].

Flexible

FOB has become more acceptable as an initial procedure for intubated or not intubated patients. The main limitation of FOB compared to the rigid bronchoscope is its limited ability to produce adequate suction through its smaller port. Also, with massive hemoptysis, the presence of blood in the inferior airway may absorb the light transmitted by the FOB and consequently decrease the capacity of visualization [13]. As described above, FOB can be used through the rigid bronchoscope for further examination.

For maximal safety, most patients should be intubated when bronchoscopy is indicated in presence of massive hemoptysis. If bleeding increases or recurs during the procedure, the bronchoscope can be removed, and suction can be applied while controlling the airway. The perception obtained through the bronchoscope is commonly obscured by clots on the tip of the scope; thus, it is important to be able to safely remove the scope, clean the tip, and suction the channel to continue the examination [28]. The disadvantages of bronchoscopy in an acute bleeding situation include poor visibility due to endobronchial blood and frequent ineffective therapeutic options [1].

Endobronchial Therapy

Laser photocoagulation or resection, electrocauterization, and cryotherapy are useful tools for minor or moderate hemoptysis. Unfortunately, these techniques are rarely efficient against massive hemoptysis [13]. If bronchoscopy allows visualization of a localized bleeding mucosal lesion, laser therapy or electrocauterization may be considered, if available. Both techniques can be used through a flexible or rigid bronchoscope. Since excellent visualization of the bleeding site is required, the rigid bronchoscope may be preferred because of its better suction capability [11].

Pharmacologic Adjuncts

Some pharmacologic adjuncts may be used through the FOB. Topical agents such as warm saline may initially help to break down gross clots and identify the bleeding site [13]. After identification of the bleeding side, initial control may be obtained by using 50 mL sequential aliquots of up to 500 mL ice-saline lavage [12]. Topical epinephrine (1:20,000) is also used to act locally as a vessel's vasoconstrictor to stop bleeding. Thrombin, fibrinogen-thrombin, or fibrin precursor solutions, such as hemostatic agents, can be injected via intrabronchial infusion through a catheter inserted into a FOB wedged against the bleeding bronchus [34, 35].

Mechanical Therapy

When a lesion is not treatable by embolization, as there is no feeding vessel and if surgical resection is not thought to be a viable option, the protection of the contralateral lung can be achieved by the insertion of a lung isolation device. But this measure may offer only a temporary relief. At that time, the use of self-expanding airway stent to cover the bleeding segmental bronchial orifice may act as both tamponade and isolation of the bleeding source [36].

Systemic Therapy

Intravenous vasopressin has been used to treat massive hemoptysis, in a similar way as its use in gastrointestinal

hemorrhage [37]. Therefore, its administration may prevent successful BAE [38–40]. Other therapies that may promote coagulation and that have been used successfully for massive hemoptysis include intravenous estrogens (Premarin®), desmopressin (DDAVP®), ADH (vasopressin), tranexamic acid (Cyklokapron®) [41–44], and recombinant activated coagulation factor VII [45].

Bronchial Artery Embolization (BAE)

First reported by Remy et al. in 1973 [7], the use of BAE for management of MH has become widespread. It has become the main option for the treatment of MH, either at first presentation or in case of recurrence. Development of BAE has been a huge advance in treatment of patients with MH, both as a temporizing measure and as a definitive treatment for some patients [46]. After several improvements, BAE is now considered the procedure of choice in cases of both massive and recurring hemoptysis and should be undertaken promptly [29]. It is also now considered the most effective nonsurgical treatment in MH [1]. This approach reduces the systemic arterial perfusion pressure from the fragile bronchial arteries within the diseased lung parenchyma [47]. In the hands of experienced angiographers, embolization successfully stops bleeding more than 85% of the time, especially if the bronchial circulation and the systemic arterial supply are carefully defined [11, 48].

Multiple imaging modalities are used to confirm the diagnosis and to locate the bleeding site in stable patients. These include plain chest radiography, chest computed tomography, and bronchoscopy. But in patients with MH who are unstable, diagnostic angiography is the preferred imaging method for localizing the bleeding site because it allows for immediate treatment [31].

The initial step for transcatheter embolization is performing a thoracic angiogram to visualize and localize all the main systemic arteries to the lung(s). Once the feeding arteries are localized, selective bronchial arteriography is performed to characterize the bleeding vessel. When the bleeding vessel is identified, an embolic agent is used. There are numerous options regarding material used for BAE. They all have different characteristics as being particles or coils or having irregular or spherical form or different sizes [49].

Postembolization, bronchial arteriogram, and thoracic aortogram are performed to ensure the complete block of all the feeding arteries with no further bleeding from vessels. Immediate recurrent hemoptysis often occurs due to missed feeding arteries that went untreated, whereas later recurrence may take place as a result of collateralization or recanalization of either the feeding artery or new bleeding vessels [31].

Multiple publications (10 series including 609 patients, from 1983 to 2007) have demonstrated an immediate suc-

cessful rate of controlled bleeding varying from 70% to 95% with a recurrence rate ranging between 13% and 43%. These studies also report a minimal immediate complication rate of less than 1% [31]. The most serious complication of BAE is the accidental embolization of the anterior spinal artery (Adamkiewicz) either by contrast material or the embolizing particles causing ischemic injuries [1]. The anterior spinal artery originates from a bronchial artery in about 5% of patients. The reported prevalence of this complication has been described as 1% [50]. This risk has been decreased by superselective embolization techniques using smaller catheters that can be placed distally [51]. Renal dysfunction resulting from the contrast load is a concern, especially in patients who are hemodynamically unstable due to blood loss [6].

Correct clinical evaluation and ventilation stabilization of the patient are mandatory before BAE in massive hemoptysis [27]. Intubating a patient with a single- or double-lumen tube helps to monitor, with the help of a FOB, the interruption of bleeding through a radiologic intervention and to clean the inferior airways from any residual blood and blood clots.

Surgery

Historically, pulmonary resection has been the most effective method to control and prevent recurrent bleeding [6]. Comparing the results with those of medical or surgical management is difficult for several reasons. The criteria of eligibility for surgery differ among institutions and seem to be subject to surgical or institutional bias [1]. The primary problem in selection bias is that patients who are more likely to die are less likely to be operated on [6].

Patients with lateralized, ideally well-localized, uncontrollable bleeding should be assessed early for possible surgery in case the bleeding remains brisk and unresponsive to other measures. Surgery is reserved as an absolute last resort for operative candidates not salvageable by BAE [6].

Patients presenting MH are too ill for physiologic testing; historical data are therefore used to estimate the patient's ability to undergo lung resection. Relative contraindications to surgery include severe underlying pulmonary disease, active TB, diffuse underlying lung disease (cystic fibrosis, multiple arteriovenous malformations, multifocal bronchiectasis), and diffuse alveolar hemorrhage [11].

Morbidity and mortality are significantly greater with emergent surgery for persistent massive bleeding compared to elective surgery in non-bleeding patients. In most series of emergent therapy, surgical mortality for treatment of massive hemoptysis is approximately 20%, ranging from 10% to 38% for series published between 2000 and 2003 [52–54], with morbidity occurring in an additional 25–50% of patients; however, most of these series are more than 20 years old.

The reasons for such high mortality and morbidity may be related to the ongoing bleeding in unstable hemodynamic conditions, together with soiling of the remaining healthy bronchopulmonary segments before and during the operation. Contamination of the contralateral lung before, during, and after surgery is the main cause of postoperative respiratory failure leading to prolonged ventilation, nosocomial pneumonia, and death [6]. One solution is to delay the surgery with BAE, to obtain hemodynamic stability preoperatively, and to perform bronchial toilet pre- and postoperatively. Before the critical decision to perform surgery is taken, the surgeons should make sure that available interventional modalities such as balloon bronchial blockers, rigid bronchoscopy, or BAE can be used in an optimal manner to buy time to delay surgery for a better surgical outcome.

Iatrogenic Pulmonary Artery Rupture (IPAR)

The use of a pulmonary artery catheter (PAC) is becoming less frequent in the operating room since the general use of transesophageal echocardiography. Nevertheless, PAC remains a useful tool for diagnosis and management of many patients with cardiac or lung diseases. Sicker patients may need the insertion of a PAC, and these sicker patients are usually the patients most at risk for catheter-induced pulmonary artery rupture. Prevention is the first approach to develop when confronted with an iatrogenic complication. The first step of prevention is the judicious selection of patients. Risk factors for catheter-induced pulmonary artery rupture include female gender, age over 60 years old, improper catheter placement, and pre-existing pulmonary hypertension. The second is the appropriate use and management of the PAC. Nevertheless, when a catheter-induced pulmonary artery rupture occurs, the physician needs to have a clear scheme of intervention to deal with this severe complication.

The incidence of rupture is not very high, with an average of 0.01–0.47%. In a large retrospective study of patients with a Swan-Ganz catheter, Kearney et al. [55] found an incidence of pulmonary artery rupture (PAR) of 0.031% and a mortality of 70% [55]. The mortality rate of pulmonary artery rupture averages 50% but can be as high as 75% in anticoagulated patients. Death occurs most often secondary to asphyxia. If a delay before the appropriate management is instituted, it will contribute to a higher mortality rate.

The initial presentation may be as obvious as massive pulmonary hemorrhage or as subtle as a minor hemoptysis associated with cough, or it may be totally asymptomatic [56]. Moreover, any hemoptysis in the presence of a PAC should be investigated because of the high suspicion of PAR or false aneurysm formation. Hemothorax may be the mode of presentation when blood enters the pleural space instead of the airway.

The proposed mechanisms for catheter-induced PAR include catheter tip lodged in the vessel wall when the PAC is advanced while the balloon is not inflated or when eccentric balloon inflation exposes the catheter tip and guides it into the arterial wall or migration of catheter in a smaller arteriole with subsequent rupture caused by balloon inflation. Primary management of catheter-induced pulmonary artery rupture focuses on the prevention of asphyxia. Asphyxia secondary to lung spillage or blood clots is the main factor leading to death. Prevention of contamination of the unaffected lung is essential. As for other types of MH, blood loss is rarely massive enough to cause a great threat in hemodynamics, and slight hypotension may be treated with volume replacement.

The management of IPAR is a three-pronged approach, with targeted therapy derived from the basic “ABC” principles of resuscitation, airway, breathing, and circulation. The goals are (A) lung isolation, (B) maintaining appropriate gas exchange and oxygen delivery, and (C) volume resuscitation. The decision to leave the PAC in place may be critical at that time for the next steps in the radiologic management of this complication. It is essential not to inflate the balloon without radiologic imaging support (see later discussion). Management differs depending on the clinical presentation, mainly in which setting it is presenting, intensive care unit, operating room, or radiology suite.

Intensive Care Unit Setting

When a pulmonary artery catheter is inserted and there is hemoptysis, whether it is massive or negligible, a chest radiograph is usually obtained and will show infiltration around the catheter tip or pleural effusion. The side of a PAC can serve as a guide to determine from which side the hemorrhage may come from. Since most PACs are located in the right lung (90%), mainly the right lower lobe, it can be assumed that hemorrhage comes from the right side if the situation is critical [20, 57].

While diagnostic procedures are performed, 100% oxygen should be administered to the patient. If the lungs are not isolated, the patient should be placed with the bleeding lung on the dependent (inferior) side to prevent spillage to the unaffected side. If the situation is not critical, a short fiber-optic bronchoscopy can be performed to determine the origin of bleeding.

The patient must undergo selective intubation to obtain lung isolation. Lung isolation can be performed with various techniques, including selective intubation with a standard endotracheal tube, bronchial blocker, or DLT. As mentioned earlier, it is our opinion that the best strategy is to place a DLT, but a bronchial blocker can be used for lung separation if a DLT is not immediately available or is difficult to insert.

After lung isolation, fiber-optic bronchoscopy can be useful to confirm the good positioning of the device used for the lung isolation and to identify the bleeding site. It is frequently difficult to get a good view of the structures because the

blood in the tracheobronchial tree highly absorbs the light of the fiber-optic bronchoscope.

It has been suggested that the PAC could be deflated, withdrawn of a few centimeters, and left in the pulmonary artery. The balloon may be inflated to compress the bleeding vessels or to temporarily obstruct the feeding artery [58, 59]. We recommend that this technique should be used only under fluoroscopy and angiographic control to finely adjust the position of the balloon, in order to avoid malpositioning of the balloon. Improper positioning could augment the bleeding by increasing the vascular laceration or by diverting the pulmonary blood flow to the injured vessel.

Pulmonary angiogram generated through a PAC may be difficult to realize because of the very small inner lumen of the Swan-Ganz catheter. With a stable patient or if the diagnosis remains unclear, a contrast-enhanced CT scan may be performed and is a valuable diagnostic tool. It can confirm the possibility of a pulmonary artery false aneurysm (PAFA) but also exclude any other causes of hemoptysis. CT scan-

ning is usually followed by an angiography with embolization, if indicated and feasible (Table 44.3).

Operating Room Setting

Most cases of life-threatening hemoptysis described during cardiac surgery result from a catheter-induced pulmonary artery perforation [1]. If a hemoptysis happens during surgery, lung isolation can be rapidly achieved, and the diagnostic and therapeutic procedures started, while the patient is still under anesthesia. If the hemoptysis happens before the scheduled surgery, elective surgery must be postponed until the PAR is investigated and stabilized. During cardiac surgery, when a hemorrhage happens after cardiopulmonary bypass (CPB) but before heparin reversal, CPB should be restarted to bypass the lung circulation and stop the bleeding, giving time to the anesthesia team to identify the side of the bleeding, to isolate the lungs, and to maximize oxygenation (see Fig. 44.2). If the hemoptysis begins after protamine administration, the best conduct is to finish the surgery as quickly as possible, isolate the lung, and proceed to a definite investigation and treatment of the pulmonary artery rupture.

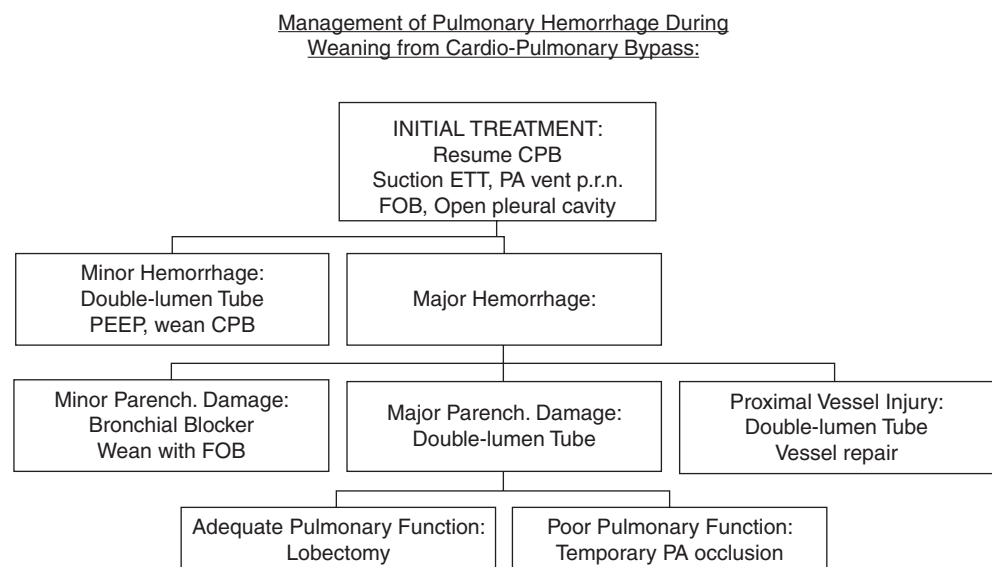
Iatrogenic pulmonary artery rupture can be localized at the proximal trunk of the pulmonary artery (PA). As this perforation occurred outside the lung parenchyma, it explains the absence of hemoptysis [60]. Surgical repair is then indicated.

After ruling out the implication of PAC as the cause of MH during cardiac surgery, other causes, including surgical trauma, traumatic intubation, or concomitant disease (neoplasia, pulmonary edema) amplified by heparin and anticoagulation, should be considered. When no pre-existing disease and no lesion can be identified by bronchoscopy or by the surgeons to explain unilateral bleeding and that the patient is too instable to be transferred to the angiographic

Table 44.3 Management of pulmonary artery catheter-induced pulmonary hemorrhage

1. Initially place the patient with the non-isolated, bleeding lung in the inferior position
2. Endotracheal intubation, oxygenation, airway toilet
3. Lung isolation: endobronchial double- or single-lumen tube or bronchial blocker
4. Withdraw the pulmonary artery catheter several centimeters, leaving it in the main pulmonary artery. Do not inflate the balloon (except with fluoroscopic guidance).
5. Position the patient with the isolated bleeding lung nondependent. PEEP to the bleeding lung if possible
6. Transport to medical imaging for diagnosis and embolization if feasible

Fig. 44.2 Flow diagram of management of massive hemoptysis during weaning from cardiopulmonary bypass. CPB cardiopulmonary bypass, FOB fiber-optic bronchoscopy, ETT endotracheal tube, PA pulmonary artery, Parench. lung parenchyma



room, temporary pulmonary vascular exclusion by thoracotomy is an effective alternative to radiologic embolization for managing unilateral hemoptysis during heart surgery [61].

Radiological Setting

During cardiac catheterization, the PAC can be used to evaluate pulmonary vascular resistance and the wedge pressure. When catheter-induced pulmonary artery rupture happens in this setting, it is relatively easy to pull back the PAC a few cm and to reinflate the balloon under direct visualization [59, 62]. Thus, it may be possible to stop the bleeding pending further radiological intervention. However, this measure does not always enable physician to contain the hemorrhage [63]. This is the reason why the authors recommend using fluoroscopy and contrast injection to confirm that the PAC is still proximal to the injured vessel and that balloon inflation impedes flow through the lacerated vessel. Diagnostic angiography and embolization can be easily performed at that point. This may help to avoid intubation, lung isolation, and post-procedure ventilation in some circumstances [59]. Since the use of a FOB to initially position a DLT or BB in a patient with MH can be difficult due to obstruction of the view by blood, one option in the radiology suite is to use fluoroscopy to help guide the lung isolation device (see Fig. 44.3) [62].

Pulmonary Artery False Aneurysm (PAFA)

PAFA formation is secondary to the accumulation of blood in an aneurismal sac compressed by the lung parenchyma. While there is no intact vessel wall lining containing the bleeding, the lung parenchyma may prevent further extravasation.

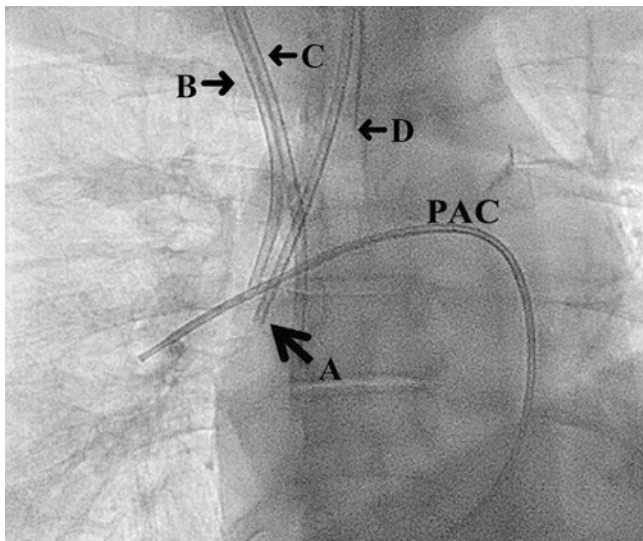


Fig. 44.3 An Arndt bronchial blocker (arrow A) has been positioned under fluoroscopic guidance into the right main stem bronchus of a patient who developed massive hemoptysis during right heart catheterization. The bronchial blocker can be seen between the central venous catheter (arrow B), proximal pulmonary artery catheter (arrow C), distal end of the endotracheal tube (arrow D). PAC distal pulmonary artery catheter (Modified with permission from Addante et al. [62])

The presence of a PAFA requires intervention, because one can never be certain that spontaneous healing will occur. Delayed pulmonary hemorrhage occurs in 30–40% of cases of a PAFA caused by a previous catheter-induced pulmonary artery rupture. Rebleeding can occur as late as 2 weeks to 7 months after the initial event [64].

With the development of interventional cardiology and different vascular devices, novel therapeutic approaches to IPAR have been recently developed. For instance, vascular plug (Amplatzer® AGA Medical Corp., North Plymouth, MN) has been used with success [65, 66].

If there is suspicion of a PAFA on the CT scan, an angiogram should be done. When the clinical suspicion of pulmonary artery rupture is high or when the patient is unstable, angiography remains the procedure of choice because it allows both diagnostic and therapeutic intervention (see Fig. 44.4) [67]. If diagnosis of a PAFA is confirmed, selective embolization helps to reduce morbidity and mortality. Embolization is successful in 75% of cases, with a rebleeding rate of about 20%. Sometimes, it can be deleterious to embolize the PAFA regarding global lung function. In these cases, conservative treatment can be tried. Follow-up of this type of patients with repeat contrast CT scan is required.

Other Causes

Some other causes or presentations of IPAR have been described. Its occurrence is extremely rare during insertion of a thoracic percutaneous drainage tubes but may be devastating due to the large-bore chest tube typically used and sometimes may be rapidly fatal [68]. An idiopathic bilateral bronchial hemorrhage during cardiac surgery has been reported in the literature. Despite a very aggressive treatment, the patient died on day 6 [19].

Management Post Hemoptysis

Following massive hemoptysis, treated either by endoscopy, radiological intervention or surgery, it is important to perform a bronchoscopy to clean the tracheobronchial tree and to remove any blood and clots in the distal airway. This action will help promote better and faster patient recuperation.

Tracheostomy Hemorrhage

Another clinically challenging scenario involving massive airway bleeding is tracheostomy hemorrhage. Hemorrhage in the immediate postoperative period following a tracheostomy is usually from local vessels in the incision such as the anterior jugular or inferior thyroid veins. Massive hemorrhage 1–6 weeks postoperatively is most common due to tracheo-innominate artery fistula [69]. A small sentinel bleed occurs in most patients before a massive bleed.

Fig. 44.4 (a) Radiographic contrast dye injection showing a false aneurysm of the pulmonary artery of the right lower lobe following massive hemoptysis induced by pulmonary artery catheter rupture. (b) A coil has been placed by interventional radiology in the false aneurysm of the right lower pulmonary artery in the same patient. Dye injection shows that the aneurysm has embolized with no further leakage

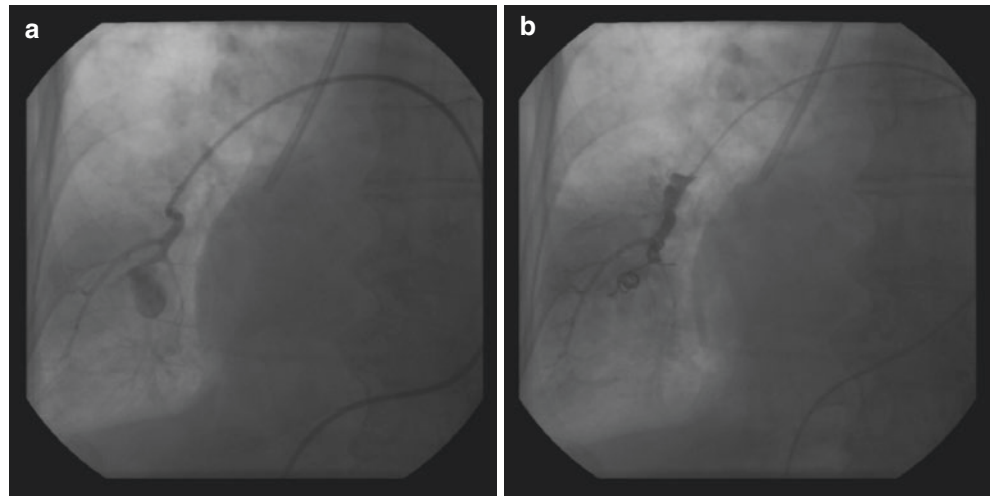


Table 44.4 Management of tracheo-innominate artery fistula hemorrhage

1. Overinflate the tracheostomy cuff to tamponade the hemorrhage. If this fails:
2. Replace the tracheostomy tube with an oral endotracheal tube. Position the cuff with FOB guidance just above the carina
3. Digital compression of the innominate artery against the posterior sternum using a finger passed through the tracheostomy stoma. If this fails:
4. Slow withdrawal of the ETT and overinflation of the cuff to tamponade
5. Then proceed with definitive therapy: sternotomy and ligation of the innominate artery

The management protocol for tracheo-innominate artery fistula is outlined in Table 44.4.

Conclusion

Endobronchial control measures and artery embolization have radically changed the management of massive hemoptysis. In experienced hands, BAE is an effective therapeutic tool and plays a pivotal role in the management of life-threatening massive hemoptysis. With control of hemorrhage, nonsurgical patients can be identified and surgical candidates accurately assessed to allow an elective operation, with lower morbidity and mortality, if conservative measures are unsuccessful [6].

Based on the above information, the following approach is a reasonable way to manage a patient with massive hemoptysis. First, stabilize the patient's oxygenation, ventilation, and hemodynamic status. Early correction of coagulopathy and consultation with critical care physician, pulmonary medicine (bronchoscopist), interventional radiologist, thoracic surgery, and anesthesiologist are essential. Perform early bronchoscopy along with other appropriate diagnostic evaluations. If the patient

continues to bleed aggressively, arteriography is most reasonable for localization and therapy. If bleeding persists despite embolization or if the patient is too ill to undergo an angiography, blockade therapy or insertion of a double-lumen tube should be considered in preparation for rigid bronchoscopy in the operating room with possible lung resection if warranted. While surgery remains the only truly definitive therapy, it should not be used in the acute emergent setting unless it cannot be avoided [11]. Surgery, including pulmonary artery ligation, segmentectomy, lobectomy, or pneumonectomy, is reserved for extreme cases since it is technically challenging and associated to high morbidity [64].

Clinical Case Discussion

A 26-year-old woman is well known in the authors' institution for Eisenmenger's syndrome secondary to a complex congenital heart disease. She had a patent ductus arteriosus for which no surgical option was available when diagnosed at 4 years old. She was referred to our center 4 years ago for pulmonary hypertension. At that time, it was observed that the right pulmonary artery originates directly from the aorta. She has been treated with epoprostenol (Flolan) for 3 years.

A few months ago, she presented some episodes of moderate hemoptysis treated with BAE. Someday, she presented a new moderate hemoptysis necessitating BAE. She was admitted to the intensive care unit for 5 days, and no bleeding was observed. She was then transferred to the bronchoscopy suite to search for a blood occluding the right inferior lobe bronchus. With the aid of sedation and local anesthesia, the area was easily reached. The blood clot was partially dislodged without any problem. While trying to dislodge the remaining of the clot, coughing was provoked, and it induced a massive bleeding from the inferior lobe.

Questions

Which immediate procedure should be undertaken?

1. Nasal oxygen was already in place for the FOB exam.
2. Right lateral decubitus position to protect the left lung from blood spillage.
3. Irrigation of the origin of bleeding with cold saline.

Bleeding continues and becomes a MH. What is the next step?

4. Left-side endobronchial intubation with single-lumen tube with the assistance of the FOB.
5. Left lateral decubitus position to improve left lung gas exchange and minimize bleeding from the right lung.
6. Exchange the endobronchial simple lumen tube for a double-lumen tube.

The patient was directed, with anesthesia assistance, to the radiological suite for angiography and BAE as needed. Following diagnostic angiography and therapeutic embolization, the radiologist wanted to know about the bleeding in the right lung. What we can do to help her?

7. Fiber-Optic Bronchoscopy Examination

Following this procedure, the right-side bronchial tree was suctioned. At that time, active bleeding was identified originating from the right inferior super dorsal bronchus. Following a new BAE, the bleeding ceased, and the cleaning of the bilateral bronchial tree was completed without finding any other bleeding site.

The patient was transferred to the ICU with the DLT in place. She was sedated and ventilated until the next morning when she was extubated. She did not present any recurrence, and she was transferred to another center for evaluation for lung transplantation.

References

1. Jean-Baptiste E. Clinical assessment and management of massive hemoptysis. *Crit Care Med*. 2000;28(5):1642–7.
2. Amirana M, Frater R, Tirschwell P, Janis M, Bloomberg A, State D. An aggressive surgical approach to significant hemoptysis in patients with pulmonary tuberculosis. *Am Rev Respir Dis*. 1968;97(2):187–92.
3. Bobrowitz ID, Ramakrishna S, Shim YS. Comparison of medical v surgical treatment of major hemoptysis. *Arch Intern Med*. 1983;143(7):1343–6.
4. Corey R, Hla KM. Major and massive hemoptysis: reassessment of conservative management. *Am J Med Sci*. 1987;294(5):301–9.
5. Garzon AA, Cerruti MM, Golding ME. Exsanguinating hemoptysis. *J Thorac Cardiovasc Surg*. 1982;84(6):829–33.
6. Wigle DA, Waddell TK. Chapter 38: Investigation and management of massive hemoptysis. In: Pearson's thoracic & esophageal surgery. J. D., Meyerson, S. L., A, P. (Ed.), & JD, C. (Ed.). Pearson's Thoracic and Esophageal Surgery 3rd edition. Philadelphia: Churchill Livingstone. 2008
7. Remy J, Voisin C, Ribet M, Dupuis C, Beguery P, Tonnel AB, et al. Treatment, by embolization, of severe or repeated hemoptysis associated with systemic hypervascularization. *Nouv Press Med*. 2060;2(31):1973.
8. Hiebert CA. Balloon catheter control of life-threatening hemoptysis. *Chest*. 1974;66(3):308–9.
9. Cauldwell EW, Siekert RG, et al. The bronchial arteries; an anatomic study of 150 human cadavers. *Surg Gynecol Obstet*. 1948;86(4):395–412.
10. Fraser KL, Grosman H, Hyland RH, Tullis DE. Transverse myelitis: a reversible complication of bronchial artery embolisation in cystic fibrosis. *Thorax*. 1997;52(1):99–101.
11. Ingbar DH. Causes and management of massive hemoptysis in adults. Post TW, ed. UpToDate. Waltham, MA: UpToDate Inc. <http://www.uptodate.com>; 2009.
12. Dweik RA, Stoller JK. Role of bronchoscopy in massive hemoptysis. *Clin Chest Med*. 1999;20(1):89–105.
13. Karmy-Jones R, Cuschieri J, Vallieres E. Role of bronchoscopy in massive hemoptysis. *Chest Surg Clin N Am*. 2001;11(4):873–906.
14. Wedzicha JA, Pearson MC. Management of massive haemoptysis. *Respir Med*. 1990;84(1):9–12.
15. Campos JH. Progress in lung separation. *Thorac Surg Clin*. 2005;15(1):71–83.
16. Spicsek-Macan J, Hodoba N, Nikolic I, Stancic-Rokotov D, Kolaric N, Popovic-Grle S. Exsanguinating tuberculosis-related hemoptysis: bronchial blocker introduced through percutaneous tracheostomy. *Minerva Anesthesiol*. 2009;75(6):405–8.
17. Freitag L, Tekolf E, Stamatis G, Montag M, Greschuchna D. Three years experience with a new balloon catheter for the management of haemoptysis. *Eur Respir J*. 1994;7(11):2033–7.
18. Giannoni S, Buti G, Allori O, Conti D, Ferri L. Bilateral concurrent massive hemoptysis successfully controlled with double endobronchial tamponade. A case report. *Minerva Anesthesiol*. 2006;72(7–8):665–74.
19. Uzuka T, Nakamura M, Nakajima T, Kusudoh S, Usubuchi H, Tanaka A, et al. Idiopathic bronchial hemorrhage: a rare but catastrophic complication in cardiac surgery. *J Cardiothorac Surg*. 2016;11(1):78.
20. Caddell B, Yelverton B, Tippett JC, Ravi Y, Sai-Sudhakar CB, Culp WC Jr. Management of massive hemoptysis after pulmonary thromboembolectomy using a double bronchial blocker system. *J Cardiothorac Vasc Anesth*. 2017;31(2):633–6.
21. Jardin M, Remy J. Control of hemoptysis: systemic angiography and anastomoses of the internal mammary artery. *Radiology*. 1988;168(2):377–83.
22. Gourin A, Garzon AA. Operative treatment of massive hemoptysis. *Ann Thorac Surg*. 1974;18(1):52–60.
23. Klein U, Karzai W, Bloos F, Wohlfarth M, Gottschall R, Fritz H, et al. Role of fiberoptic bronchoscopy in conjunction with the use of double-lumen tubes for thoracic anesthesia: a prospective study. *Anesthesiology*. 1998;88(2):346–50.
24. Campos JH, Hallam EA, Van Natta T, Kernstine KH. Devices for lung isolation used by anesthesiologists with limited thoracic experience: comparison of double-lumen endotracheal tube, Univent torque control blocker, and Arndt wire-guided endobronchial blocker. *Anesthesiology*. 2006;104(2):261–6, discussion 5A
25. Campos JH. Which device should be considered the best for lung isolation: double-lumen endotracheal tube versus bronchial blockers. *Curr Opin Anaesthesiol*. 2007;20(1):27–31.
26. Narayanaswamy M, McRae K, Slinger P, Dugas G, Kanellakos GW, Roscoe A, et al. Choosing a lung isolation device for thoracic surgery: a randomized trial of three bronchial blockers versus double-lumen tubes. *Anesth Analg*. 2009;108(4):1097–101.
27. de Gregorio MA, Medrano J, Laborda A, Higuera T. Hemoptysis workup before embolization: single-center experience with a 15-year period follow-up. *Tech Vasc Interv Radiol*. 2007;10(4):270–3.

28. Ingbar DH. Diagnostic approach to massive hemoptysis in adults. Post TW, ed. UpToDate. Waltham, MA: UpToDate Inc. <http://www.uptodate.com>; 2009
29. Poyanli A, Acunas B, Rozanes I, Guven K, Yilmaz S, Salmaslioglu A, et al. Endovascular therapy in the management of moderate and massive haemoptysis. *Br J Radiol.* 2007;80(953):331–6.
30. Hsiao EI, Kirsch CM, Kagawa FT, Wehner JH, Jensen WA, Baxter RB. Utility of fiberoptic bronchoscopy before bronchial artery embolization for massive hemoptysis. *AJR Am J Roentgenol.* 2001;177(4):861–7.
31. Lee EW, Grant JD, Loh CT, Kee ST. Bronchial and pulmonary arterial and venous interventions. *Semin Respir Crit Care Med.* 2008;29(4):395–404.
32. Haponik EF, Chin R. Hemoptysis: clinicians' perspectives. *Chest.* 1990;97(2):469–75.
33. Lippmann ML, Walkenstein MD, Goldberg SK. Bronchoscopy in hemoptysis. *Chest.* 1990;98(6):1538.
34. Tsukamoto T, Sasaki H, Nakamura H. Treatment of hemoptysis patients by thrombin and fibrinogen-thrombin infusion therapy using a fiberoptic bronchoscope. *Chest.* 1989;96(3):473–6.
35. Bense L. Intra-bronchial selective coagulative treatment of hemoptysis. Report of three cases. *Chest.* 1990;97(4):990–6.
36. Brandes JC, Schmidt E, Yung R. Occlusive endobronchial stent placement as a novel management approach to massive hemoptysis from lung cancer. *J Thorac Oncol.* 2008;3(9):1071–2.
37. Magee G, Williams MH Jr. Treatment of massive hemoptysis with intravenous pitressin. *Lung.* 1982;160(3):165–9.
38. Mal H, Rullon I, Mellot F, Brugiere O, Sleiman C, Menu Y, et al. Immediate and long-term results of bronchial artery embolization for life-threatening hemoptysis. *Chest.* 1999;115(4):996–1001.
39. Remy J, Arnaud A, Fardou H, Giraud R, Voisin C. Treatment of hemoptysis by embolization of bronchial arteries. *Radiology.* 1977;122(1):33–7.
40. Stoller J. Diagnosis and management of massive hemoptysis: a review. *Respir Care.* 1992;37:564–81.
41. Bilton D, Webb AK, Foster H, Mulvenna P, Dodd M. Life threatening haemoptysis in cystic fibrosis: an alternative therapeutic approach. *Thorax.* 1990;45(12):975–6.
42. Chang AB, Ditchfield M, Robinson PJ, Robertson CF. Major hemoptysis in a child with cystic fibrosis from multiple aberrant bronchial arteries treated with tranexamic acid. *Pediatr Pulmonol.* 1996;22(6):416–20.
43. Graff GR. Treatment of recurrent severe hemoptysis in cystic fibrosis with tranexamic acid. *Respiration.* 2001;68(1):91–4.
44. Popper J. The use of premarin IV in hemoptysis. *Dis Chest.* 1960;37:659–60.
45. Tien HC, Gough MR, Farrell R, Macdonald J. Successful use of recombinant activated coagulation factor VII in a patient with massive hemoptysis from a penetrating thoracic injury. *Ann Thorac Surg.* 2007;84(4):1373–4.
46. Johnson JL. Manifestations of hemoptysis. How to manage minor, moderate, and massive bleeding. *Postgrad Med.* 2002;112(4):101–6, 8–9, 13
47. Marshall TJ, Jackson JE. Vascular intervention in the thorax: bronchial artery embolization for haemoptysis. *Eur Radiol.* 1997;7(8):1221–7.
48. Yoon W, Kim JK, Kim YH, Chung TW, Kang HK. Bronchial and nonbronchial systemic artery embolization for life-threatening hemoptysis: a comprehensive review. *Radiographics.* 2002;22(6):1395–409.
49. Ittrich H, Klose H, Adam G. Radiologic management of haemoptysis: diagnostic and interventional bronchial arterial embolisation. *RoFo.* 2015;187(4):248–59.
50. Zhang JS, Cui ZP, Wang MQ, Yang L. Bronchial arteriography and transcatheter embolization in the management of hemoptysis. *Cardiovasc Intervent Radiol.* 1994;17(5):276–9.
51. Tanaka N, Yamakado K, Murashima S, Takeda K, Matsumura K, Nakagawa T, et al. Superselective bronchial artery embolization for hemoptysis with a coaxial microcatheter system. *J Vasc Interv Radiol.* 1997;8(1 Pt 1):65–70.
52. Lee TW, Wan S, Choy DK, Chan M, Arifi A, Yim AP. Management of massive hemoptysis: a single institution experience. *Ann Thorac Cardiovasc Surg.* 2000;6(4):232–5.
53. Knott-Craig CJ, Oosthuizen JG, Rossouw G, Joubert JR, Barnard PM. Management and prognosis of massive hemoptysis. Recent experience with 120 patients. *J Thorac Cardiovasc Surg.* 1993;105(3):394–7.
54. Endo S, Otani S, Saito N, Hasegawa T, Kanai Y, Sato Y, et al. Management of massive hemoptysis in a thoracic surgical unit. *Eur J Cardiothorac Surg.* 2003;23(4):467–72.
55. Kearney TJ, Shabot MM. Pulmonary artery rupture associated with the Swan-Ganz catheter. *Chest.* 1995;108(5):1349–52.
56. Poplousky MR, Rozenblit G, Rundback JH, Crea G, Maddineni S, Leonardo R. Swan-Ganz catheter-induced pulmonary artery pseudoaneurysm formation: three case reports and a review of the literature. *Chest.* 2001;120(6):2105–11.
57. Stratmann G, Benumof JL. Endobronchial hemorrhage due to pulmonary circulation tear: separating the lungs and the air from the blood. *Anesth Analg.* 2004;99(5):1276–9.
58. Dopfner UR, Braun JP, Grosse J, Hotz H, Duveneck K, Schneider MB. Treatment of severe pulmonary hemorrhage after cardiopulmonary bypass by selective, temporary balloon occlusion. *Anesth Analg.* 2004;99(5):1280–2; table of contents
59. Fortin M, Turcotte R, Gleeton O, Bussières JS. Catheter-induced pulmonary artery rupture: using occlusion balloon to avoid lung isolation. *J Cardiothorac Vasc Anesth.* 2006;20(3):376–8.
60. Booth KL, Mercer-Smith G, McConkey C, Parissis H. Catheter-induced pulmonary artery rupture: haemodynamic compromise necessitates surgical repair. *Interact Cardiovasc Thorac Surg.* 2012;15(3):531–3.
61. Fortin J, Vaillancourt R, Vigneault L, Laflamme M, Simon M, Bussières JS. Unusual cause of life-threatening hemoptysis during cardiac operation: surgical management revisited. *Ann Thorac Surg.* 2017;104:e251.
62. Addante RA, Chen J, Goswami S. Successful management of a patient with pulmonary artery rupture in a catheterization suite. *J Cardiothorac Vasc Anesth.* 2016;30(6):1618–20.
63. Gottwalles Y, Wunschel-Joseph ME, Hanssen M. Coil embolization treatment in pulmonary artery branch rupture during Swan-Ganz catheterization. *Cardiovasc Intervent Radiol.* 2000;23(6):477–9.
64. Mullerworth MH, Angelopoulos P, Couyant MA, Horton AM, Robinson SM, Petring OU, et al. Recognition and management of catheter-induced pulmonary artery rupture. *Ann Thorac Surg.* 1998;66(4):1242–5.
65. Kalra A, Heitner S, Topalian S. Iatrogenic pulmonary artery rupture during Swan-Ganz catheter placement--a novel therapeutic approach. *Catheter Cardiovasc Interv.* 2013;81(1):57–9.
66. Rudzinski PN, Henzel J, Dzielinska Z, Lubiszewska BM, Michalowska I, Szymanski P, et al. Pulmonary artery rupture as a complication of Swan-Ganz catheter application. Diagnosis and endovascular treatment: a single centre's experience. *Postepy Kardiologii Interwencyjnej.* 2016;12(2):135–9.
67. Utsumi T, Kido T, Ohata T, Yasukawa M, Takano H, Sakakibara T. Swan-Ganz catheter-induced pseudoaneurysm of the pulmonary artery. *Jpn J Thorac Cardiovasc Surg.* 2002;50(8):347–9.
68. Bozzani A, Arici V, Bellinzona G, Pirrelli S, Forni E, Odero A. Iatrogenic pulmonary artery rupture due to chest-tube insertion. *Tex Heart Inst J.* 2010;37(6):732–3.
69. Grant CA, Dempsey G, Harrison J, Jones T. Tracheo-innominate artery fistula after percutaneous tracheostomy: three case reports and a clinical review. *Br J Anaesth.* 2006;96:127–30.