

## Endobronchial Hemorrhage Due to Pulmonary Circulation Tear: Separating the Lungs and the Air from the Blood

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This issue of *Anesthesia & Analgesia* contains a report by Doepfmer et al. (1) of endobronchial hemorrhage caused by perforation of a pulmonary vein because of a misplaced left ventricular vent catheter. This case is important because it illustrates well the need for properly managing the immediate mechanisms of death, namely, extravasation of blood and extravasation of air. The former causes either asphyxiation from endobronchial hemorrhage or, less frequently, exsanguination, whereas the latter can cause systemic air embolism (2–4). Until the Doepfmer et al. case (1), virtually all knowledge of catheter-related perforations of the pulmonary vasculature was derived from perforations of a pulmonary artery (PA) by a PA catheter (PAC).

How does blood gain access to the airway after perforation of the pulmonary circulation by a catheter? PA perforation caused by a PAC is rare ( $\approx 0.1\%$ ), presents with endobronchial hemorrhage in  $>90\%$  of cases, and has a mortality of 40%–60% (2,3). Perforation is usually of a right (2,5) lobar, segmental, or subsegmental PA and is caused either directly by the PAC tip or as a consequence of balloon inflation (which either can directly damage the PA or, if the balloon inflates excentrically, can force the tip of the catheter into the wall of the PA) (6,7). Extravasated blood appears to gain access to the airway at the point where the PA perforation occurred. Mayr et al. (8) report an autopsy-proven case of right lower lobe PA-to-bronchus perforation by a PAC. Also at autopsy, Yellin et al. (9) demonstrated a direct communication between a subsegmental PA and its bronchus. Barash et al. (6) fiberoptically identified the bleeding points in the lobar bronchi of two patients. In two other cases of PA perforation, a PA-to-bronchus fistula was demonstrated by injection of contrast medium

into the distal lumen of the PAC (10,11). Four other reports (12–15) suggested a communication between the corresponding PA and bleeding bronchus (two lobar and two segmental), but the communication was not objectively proven. Finally, and as demonstrated by Doepfmer et al. (1), the occurrence of severe air embolism to the left heart precisely when injured lung airway pressure became greater than pulmonary vascular pressure is very consistent with the presence of direct communication between a bronchus and a pulmonary vessel.

Perforation of a PA and bronchus by a PAC at the lobar to subsegmental level makes sense for 2 reasons. First, at these levels, the PA courses to the periphery along the posterior surface of the bronchial tree (16). The posterior membrane of the bronchial tree is the part of the bronchus most susceptible to disruption by either a vascular catheter from the outside or by a ventilating catheter from the inside (17,18). Second, the size of the vessels and bronchi at these levels corresponds to the outside diameter of the PAC.

Doepfmer et al. (1) convincingly demonstrate a direct communication between a right lower lobe pulmonary vein and bronchus by pulmonary wedge angiography. Because the lobar veins also come into close relation to the bronchial tree at this level, the Doepfmer et al. case (1) is entirely consistent with the commonly ascribed mechanism of injury via perforation of the PA and bronchus by a PAC with respect to both the lobar to subsegmental location and the expected size of the injured vessel and bronchus.

Others have suggested that blood gains access to the airway via ruptured alveoli (19,20), but good evidence for this assertion is lacking. Parenchymal hemorrhage is a common finding of PA rupture, but whether it precedes or is a consequence of endobronchial hemorrhage is not known. With respect to exactly where blood distributes after a pulmonary circulation tear, several more observations are pertinent. First, in some cases, the application of positive pressure ventilation (PPV) and positive end-expiratory pressure (PEEP) has caused cessation of endobronchial hemorrhage

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(19,21,22). This observation suggests that the alveoli might be a route of entry of blood into the airway. However, because an increased alveolar to vascular pressure gradient (i.e., PPV plus PEEP) does not ordinarily cause air embolism, either the alveoli do not represent a route of entry or air bubbles cannot readily gain access to the pulmonary vasculature via the alveolar route. Second, the amount of bleeding into the airway seems to be independent of whether blood enters the airway via a direct communication between the pulmonary vessel and the conducting airway or via the parenchyma. Small herald bleeds are compatible with a discrete bronchovascular fistula (6,9,10), and massive bleeding can occur in the absence of a demonstrable lesion in either the airway or the PA (19,23). Third, if blood does not enter the airway, it can enter the pleural space (2,3) or be contained by the lung parenchyma (24), ultimately forming a PA-pseudoaneurysm that may or may not rebleed (25). Rupture of a pseudoaneurysm can subsequently occur into a bronchus (9), the lung parenchyma, or the pleural space (26).

Cardiopulmonary bypass (CPB) increases the risk of PA perforation by a PAC and the severity of endobronchial hemorrhage by several mechanisms: hypothermia causes stiffening of the PA and PAC, cardiac manipulation causes peripheral migration of a PAC, PA hypertension and old age are associated with fragile PA (3), and anticoagulation increases bleeding (12,13,19). However, mortality is not necessarily increased in this setting (3), because death from asphyxiation or exsanguination after CPB may be prevented by rapid reinstatement of CPB and lung separation. Most of the factors cited above for promoting PA perforation by a PAC during CPB are probably operative for pulmonary venous perforation by a pulmonary venous catheter.

Separation of the lungs is essential with endobronchial hemorrhage. CPB allows for suctioning and cleansing of the airway and full application of advanced diagnostic and therapeutic modalities, including fiberoptic confirmation of double-lumen tube (DLT) intubation, as is well demonstrated by the Doepfmer et al. case (1). If CPB is not available, lung separation may still be achieved by inserting a DLT or by advancing a single-lumen tube (SLT) into the nonbleeding bronchus (see below). The DLT offers the great advantages of selective lung suctioning and direct visualization of the bleeding site to confirm the absence of rebleeding, as described by Doepfmer et al. (1).

Given that deliberate attempts at SLT intubation of the right and left mainstem bronchus (rotate the head of the patient to the right and the SLT 180° so that the SLT bevel faces to the right) are nearly 100% and 92% successful, respectively (27,28), and that the location of the PAC is known (right versus left lung), blind intubation of the nonbleeding mainstem bronchus with an

SLT should be successful in separating the lungs 92%–100% of the time. PACs locate in the right lung approximately 90% of the time (2,5); this fact provides a theoretical basis for attempting SLT intubation of the left lung when the location of the PAC has not been radiologically verified. Nevertheless, there is a significant residual 10%–20% risk of intubating the bleeding lung. There are also reports of endobronchial hemorrhage being controlled by passage of a bronchial blocker into the mainstem bronchus of the bleeding lung (2,12,13,29).

Control of endobronchial hemorrhage has been attempted from both the pulmonary vascular and the bronchial side of the bronchovascular communication. The concept of temporary balloon occlusion of the artery feeding the bleeding site, as described in the case of Doepfmer et al. (1), is not novel (13,30–32). With PAC perforation of a PA, we believe that it is logical to deflate the PAC balloon, withdraw the PAC by a few centimeters, and then reinflate the balloon to cause a significant dampening of the phasic PA pressure wave form; by definition, blood flow to the bleeding area should be significantly decreased. However, reports or recommendations of complete PAC removal or withdrawal of the PAC without balloon reinflation abound (2,9,22,23,26,33–35). In three of these reports, the endobronchial hemorrhage occurred immediately after PAC withdrawal (9,23,35).

Attempts to control endobronchial hemorrhage from the alveolar-bronchial side of the airway-vascular communication consist mainly of applying PEEP or high continuous positive airway pressure (CPAP) (2,12,19,22,29,30) to tamponade the endobronchial hemorrhage. Doepfmer et al.'s report (1) illustrates an interesting dilemma inherent in this concept; if blood can enter an airway, then air can enter the circulation (4,30–32), especially when airway pressure exceeds the pressure in the pulmonary vessel (positive bronchovascular pressure gradient). Indeed, Doepfmer et al. (1) describe massive air embolism to the left atrium visualized by transesophageal echocardiography (TEE) when PPV was instituted while blood was still being diverted from the pulmonary circulation by the venous return cannulas of the CPB circuit (creating a positive bronchovascular gradient).

Gas insufflated into the PA can accumulate and backtrack to the right cardiac chambers, where it can compromise ventricular ejection (4) or gain access to the systemic circulation via an intracardiac shunt (36). However, for PA air to embolize to the systemic circulation, an intracardiac shunt is not required (37). Although the lung is usually a good filter for air, this function can be exhausted by the administration of larger quantities of air. Indeed, even when there is no obvious bronchovascular communication, high peak airway pressure can cause systemic gas embolism (38). In dogs, as much as  $0.3 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$  (39,40), or

20 mL total (39), of air is enough to saturate the pulmonary filter function. Air in the PA increases PA pressure mainly by causing intense PA vasoconstriction, which further promotes the transpulmonary passage of gas (39). Increased PA pressure opens intrapulmonary arteriovenous shunts, thus permitting passage of air bubbles as large as 500  $\mu\text{m}$  into the systemic circulation (40). When aminophylline is administered before PA gas embolism, the pulmonary filter function is completely abolished, permitting free transfer of gas into the systemic circulation (39). The effect of inotropic drugs often used to assist separation from CPB on the pulmonary filter function for air is unknown, but these drugs could similarly reduce the lung's capacity to filter gas bubbles. Once air enters the left heart chambers of piglets, it reaches the carotid arteries in 75% of cases (41). This can cause a spectrum of neurologic symptoms, including strokes.

Considering that endobronchial hemorrhage from PA perforation complicates 0.05%–0.2% of PAC insertions and given the frequent use of positive airway pressure in an attempt to tamponade endobronchial hemorrhage (2,12,19,22,29,30), reports of massive air embolism are remarkably rare (2,4,36). This problem is probably underdiagnosed, and its clinical consequences are probably masked by the frequent mortality rate of PA perforation (2). It is unclear to what extent systemic gas embolism contributes to this frequent mortality, but reports of sudden bradycardia or asystolic cardiac arrest after endobronchial hemorrhage may represent the clinical correlate of gas in the right coronary circulation (9,13,19). As little as 0.5–1.0 mL of air injected into a left-sided cardiac chamber can cause cardiac arrest (39), and 2–3 mL of intracerebral air can cause a fatal stroke (42).

When air in the left-sided cardiac chambers is detected by TEE, the fraction of inspired oxygen should be 1.0, and the patient should be placed in a head-down and right-side-down position, which may prevent air from entering the cerebral circulation (36). The right coronary artery territory is at greatest risk for myocardial infarction due to systemic gas embolism, because gas tends to rise toward the right coronary ostium in a supine patient. Pharmacologic increases in systemic arterial blood pressure may force small amounts of air through the capillary bed, but the risk of myocardial afterload increase and intracerebral hypertension has to be balanced against an unproven benefit. If end-organ manifestations of systemic gas embolism develop, expedient transfer to a hyperbaric facility can resolve gas embolism and should be considered.

We have addressed the anatomy of injury and the principles of diagnosis and treatment of endobronchial hemorrhage and systemic gas embolism caused by pulmonary circulation tear. Nevertheless, it cannot be stressed enough that prevention obviates the need

for treatment. With respect to avoiding catheter-induced pulmonary venous tears, correct positioning of vent catheters should be confirmed by the surgeon palpating the vent in the left ventricle, although migration of the vent may subsequently occur when the heart is manipulated. With respect to PA tears due to PAC, it is important to avoid distal migration of the PAC and frequent inflation of the PAC balloon, and the PAC should be withdrawn into the main PA before manipulation of the heart. If a catheter does cause a pulmonary circulation tear and endobronchial hemorrhage, then lung separation and attempts at control of the hemorrhage from both the airway side (PEEP and CPAP) and the vascular side (balloon inflation in the feeding PA) of the tear, with monitoring of effects from both the airway (fiberoptic bronchoscopy) and vascular side (TEE), may permit a successful resolution of the problem.

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