Review Article

Anesthetic management during bronchoscopy of patients with pulmonary hypertension

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Abstract: This article is intended to provide an overview of the anesthetic management for bronchoscopic procedures in patients with pulmonary hypertension (PH). This includes the classifications of PH, diagnosis with severity, and treatment modalities as detailed knowledge of this condition is imperative for proper anesthetic management. Preoperative evaluation and optimization of the patient is discussed as well. Bronchoscopic procedures require airway manipulation and compromise ventilation for lengths of time. As hypoxemia and hypercarbia trigger decompensation in PH, this may lead to a downward spiral if control is lost. Therefore a discussion of the intraoperative anesthetic management and the critical concerns are detailed in this article, as well as treatment modalities and management for said concerns.

Keywords: Anesthetic management; bronchoscopy; pulmonary hypertension (PH); pulmonary arterial hypertension (PAH); pulmonary vascular resistance (PVR)

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Introduction

Patients with pulmonary hypertension (PH) pose a significant challenge for anesthesiologists. This condition places them at increased intraoperative risk and they may be difficult to manage. Traditionally PH was considered a relative contraindication to anesthesia. With new treatments patients are living longer functional lives. Elective surgical procedures with these patients may not pose unacceptable risks (1). This article will discuss proper anesthetic management for those with PH during bronchoscopic procedures.

Definition, diagnosis, and prognosis

Prior to understanding proper management, a precise definition must be made.

At the 6th World Symposium on Pulmonary Hypertension a consensus was reached for resting mean pulmonary arterial pressure (mPAP) ≥20 mmHg as a criterion required for the diagnosis of PH. For the diagnosis of pre-capillary PH, the pulmonary wedge pressure must be ≤15 mmHg and the pulmonary vascular resistance (PVR) ≥3 WU (2).

The gold standard for diagnosis requires an invasive measurement via right heart catheterization with a pulmonary artery catheter (PAC). Estimates via echocardiography are imprecise and frequently underestimated but may be used as a screening tool (2). When advanced therapy is indicated to target PH directly (rather than the underlying etiology) a right heart catheterization should be performed.

There are multiple classification categories for PH. The most common etiology is left heart disease however idiopathic pulmonary arterial hypertension (PAH), lung disease, and pulmonary embolism are all causes.

Regardless, the primary concern is the development of right sided heart failure which can be acute or chronic in its presentation. In an acute presentation, a sudden increase

in pulmonary pressure could cause the right ventricle (RV) to fail. In a chronic presentation, the RV attempts to compensate with hypertrophic changes. However, over time it dilates and will ultimately fail.

PH is a progressive disease with continuous deterioration over time. Data prior to the introduction of therapeutic treatments showed 2.8 years as the median survival time.

Preoperative evaluation

It is essential to determine the severity of PH and right heart function. The World Health Organization functional classifications are determined based on actual functional status graded in increasing severity from class I to class IV (3).

(I) Grade I: no limitation of usual physical activity; ordinary physical activity does not cause dyspnea, fatigue, chest pain, or presyncope.

(II) Grade II: mild limitation of physical activity; no discomfort at rest; but normal activity causes increased dyspnea, fatigue, chest pain, or presyncope.

(III) Grade III: marked limitation of activity; no discomfort at rest but less than normal physical activity causes increased dyspnea, fatigue, chest pain, or presyncope.

(IV) Grade IV: unable to perform physical activity at rest; may have signs of RV failure; symptoms increased by almost any physical activity.

Additional symptoms that may be elicited on physical exam include peripheral edema, jugular venous distension, and ascites. A history of syncope portends a poor prognosis (4).

Further preoperative evaluation may include an electrocardiogram, chest x-ray, and a recent echocardiogram especially if symptoms have been increasing in severity. If the patient is on diuretic therapy a basic metabolic panel at minimum should be obtained for a potassium level.

Optimization

Due to the risk of decompensation with anesthetic agents, positive pressure ventilation, and surgical stimulation, non-invasive alternatives should always be explored. If that is not possible then it is important to optimize the patient prior to procedural intervention.

Patients in RV failure have a narrow therapeutic range of acceptable volume status. Therefore, it is critical to optimize their fluid status. Invasive monitoring with central venous catheter (CVC), PAC, or transesophageal echocardiogram (TEE) may be beneficial to this end.

Home medications in general should be continued throughout the perioperative window (5). Chronic medications for PAH, especially if parenteral, should not be discontinued. With increasing use of oral prostacyclin therapy, Orenitram (oral treprostinil taken 3 times a day) must be taken with at least 250 calories to aid with bioavailability, which will impact the patient's ability to be NPO for a procedure. In cases where strict NPO or procedures lasting longer than 4 hours are anticipated, transitioning from an oral prostacyclin to a parenteral form may be needed. For those on parenteral prostacyclin therapy, the dedicated central line (frequently, a Hickman catheter) should not be used during the procedure for IV anesthetic agents due to risk of bolusing the patient with prostacyclin. Alternative access should be established in such cases.

Medications utilized for heart failure should be taken as well, though ACE inhibitors/ARBs may be held. If a patient is on Coumadin for pulmonary embolism this should be switched to low molecular weight heparin for this time period.

Patients with heart failure may be on continuous oxygen at home. This should be continued as well. If there is contributing lung disease then bronchodilators or steroids may be useful. If obstructive sleep apnea is a concern then bilevel positive airway pressure (BiPAP) may be of use.

Primary concerns

As mentioned above, the primary concern in patients with symptomatic PH is to avoid decompensation. This is quite significant as such patients can rapidly progress into a chain reaction where reduced pulmonary blood flow from RV failure causes hypoxemia which then increases PVR. This further worsens right heart failure and decreased the ability to deliver oxygenated blood to the left ventricle. With the reduction in left ventricle cardiac output (CO), coronary artery blood flow decreases potentially leading to cardiac arrest.

When initially sedating such a patient a delicate balance is sought. Lowering sympathetic tone from anxiety is desirable. However, one must also avoid hypoventilation, as hypoxemia and hypercarbia both increase PVR.

General hemodynamic goals (6)

(I) Avoid increasing PVR. Hypoxemia, hypercarbia, acidosis, and increased sympathetic tone from pain

or anxiety all increase PVR. Supplementary oxygen is beneficial to this end.

(II) Systemic vascular resistance (SVR) must be maintained. If SVR is decreased, then CO decreases as well.

(III) Myocardial contractility must be maintained. Avoid myocardial depressants.

(IV) Sufficient preload is necessary to maintain CO.

(V) Sinus rhythm should be preserved with arrhythmias promptly treated.

Anesthetic management

Depending on the PH severity and procedure the following should be considered: an arterial line prior to induction especially if PH/RV disease is significant. Aside from the prompt recognition of hypotension this also allows for intraoperative blood gas sampling.

A CVC may be useful if inotropes or vasopressors are needed.

PAC/TEE if available and depending on the disease severity on the patient. TEE is especially useful for evaluation and diagnosis during acute decompensation.

Choice of anesthetic

Intubation and extubation cause physiological changes that may increase PVR. For this reason, some prefer sedation rather than general anesthesia. Others feel that the advantages of a secured airway allowing controlled ventilation outweigh those risks due to the fact that hypoxia and hypercarbia are more easily avoided. In addition, this allows for muscle relaxation and appropriate depth of anesthesia that would reduce sympathetic responses that would otherwise increase PVR.

General anesthesia

The critical points are to avoid hypoxia with supplemental oxygen and to avoid hypercarbia that can be caused by preoperative sedation.

Induction should be titrated slowly with opioids and etomidate for hemodynamic stability. A rapid acting muscle relaxant would also be beneficial. It is important to maintain oxygenation during the induction process as hypoxia can be the trigger for decompensation.

Maintenance of anesthesia during bronchoscopic procedures requires sufficient anesthetic depth to prevent sympathetic stimulation. Ideally inhalational agents should be avoided due to air leaks during bronchoscopy. It should be noted that nitrous oxide should be avoided as it both increases PVR and may lower the fraction of inspired oxygen (FiO2) causing hypoxia. Therefore, a total intravenous anesthetic (TIVA) would be ideal. A balanced anesthetic with a sedative such as propofol and an opioid should be combined for this regimen. Of note, ketamine is a direct myocardial depressant (7), and should be used cautiously as its role in patients with PH is controversial.

Ventilation

As mentioned above, avoiding hypoxemia and hypercarbia is crucial to avoid increasing PVR and thereby decreasing RV function which can trigger decompensation. This may be challenging during bronchoscopy as ventilation can easily become insufficient. Positive pressure ventilation also may reduce venous return and thereby reduce CO. High peak pressures and high PEEP should be avoided.

100% oxygen should be utilized and if pulse oximetry shows desaturation, the procedure should be paused to allow oxygenation to improve prior to resuming. Hyperventilation may be useful prior to apneic periods. Additionally, end-tidal CO2 (ETCO2) is difficult to accurately measure during bronchoscopy due to adapter leak. Intermittently the bronchoscopy should be paused with the leak closed in order to obtain accurate ETCO2 values and increase ventilation as needed to prevent hypercarbia.

Volume status

Optimal intravascular volume status has a narrow range to avoid overfilling the RV causing distension, or underfilling and thereby reducing preload. One may use central venous pressure (CVP) as a relative indicator but it should be based on a patient's historical CVP values as it can be unreliable. One should avoid acute changes in volume status.

Chronic medications intraoperatively (5)

(I) Those with PAH on therapies like prostaglandins: These should be continued either intravenously or via inhalation. If PVR increases and hypoxemia, hypercarbia, or PE have been ruled out as causes then nitric oxide (NO) or Milrinone can be used to reduce PVR.

(II) Those with PH due to left heart disease: PAH

therapy should not be used as it can cause pulmonary edema from backflow of the non-compliant left heart (8). Patients should be optimized with diuresis or milrinone and NO if necessary. A PAC should be used for monitoring.

Vasopressors and inotropes (5)

If evidence of worsening RV function or PH occurs then vasopressors and inotropes may be needed. As vasopressors increase both SVR and PVR, caution is needed. The PVR to SVR ratio is decreased to a greater extent by norepinephrine and vasopressin than by phenylephrine which produces bradycardia and pulmonary vasoconstriction. Vasopressin causes less of an increase in PA pressure than even norepinephrine. However, while low doses of vasopressin decrease PVR (9), it also causes coronary vasoconstriction especially at higher doses. Inodilators such as milrinone and dobutamine help RV function. Epinephrine increases SVR and PVR as well. It causes tachycardia and increases oxygen consumption. For these reasons it is best avoided if possible (10).

Vasodilators

Nitroglycerin, sodium nitroprusside and nicardipine all reduce PH. They blunt hypoxic pulmonary vasoconstriction and cause systemic hypotension, and therefore should be used with caution.

Rhythm

It is crucial to avoid arrhythmias. The atrial kick is very helpful with a potentially 20% boost in CO. Therefore, sinus rhythm should be kept if at all possible, with immediate treatment for atrial fibrillation if it occurs as that may precipitate decompensation.

Temperature

Normothermia should be maintained to prevent sympathetic stimulation and increases in myocardial oxygen demand from shivering.

Decompensation

Finally, if medical management fails then extracorporeal membrane oxygenation (specifically VA-ECMO) may be an emergent necessity.

Emergence and extubation

A smooth emergence is critical to avoid increased sympathetic tone. To this end pain must be properly controlled. In addition, hypoxia and hypercarbia must be avoided with appropriate ventilation. Weaning ventilation with pressure support would be helpful during this critical time (5).

Postoperative period

Postoperatively the same hemodynamic concerns discussed above remain. It is important not to quickly discontinue infusions running throughout the case, rather careful titration should be employed. This also applies to sedatives as well as mechanical ventilation. Especially if hemodynamically unstable, careful monitoring may be warranted in an intensive care unit.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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