Severe Pulmonary Hypertension: A Noncardiac, Nonobstetric Surgical Case Study

Jennifer E. Andrews, CRNA, MSN, BSN, BSEd

This report describes the noncardiac, nonobstetric surgical case of a 29-year old woman with idiopathic pulmonary arterial hypertension. To safely manage a patient with pulmonary hypertension, the anesthesia provider must have a thorough understanding of the disease and associated risks. This case study briefly summarizes the surgical case and then discusses history, current classifications, epidemiology, pathophysiology, contemporary treatments, and basic anesthetic management related to pulmonary hypertension.

Keywords: Anesthesia management, noncardiac surgery, nonobstetric surgery, pulmonary hypertension.

Pulmonary hypertension (PH) is a progressive disease with no known cure and a complex pathophysiology. The disease increases pulmonary arterial pressures (PAP) and pulmonary vascular resistance (PVR). Ultimately, if untreated, these elevated pressures can lead to right ventricular (RV) failure and death. The perioperative management of patients with PH is challenging and requires knowledge of the disease, treatments, and risks to ensure a high level of patient safety.1,2

This case study reviews the successful course of a noncardiac, nonobstetric surgery for a patient with idiopathic pulmonary arterial hypertension (IPAH). The discussion includes the history, pathophysiology, some current treatments, and anesthetic management related to PH.

Case Summary
A 29-year old, 79-kg woman presented to a university hospital with right upper quadrant pain, which had worsened over the course of a week. She subsequently received a diagnosis of acute cholecystitis and was scheduled for a laparoscopic cholecystectomy. Her past history included surgical repairs of an anterior cruciate ligament tear and ankle fracture, longstanding asthma, and postoperative nausea and vomiting. She had no drug allergies, drank alcohol socially, and had quit smoking 1 year ago. Nevertheless, this healthy-looking patient carried an ASA physical class status of 3 because of a recent diagnosis of IPAH. Her daily medications included a continuous intravenous (IV) infusion of treprostinil (Remodulin) at 50 ng/mL, through a mediport.

Results of her preoperative blood and urine tests were within normal physiologic defined limits. She had a current blood type and crossmatch. Her most recent transthoracic echocardiogram showed the following cardiac changes: mild tricuspid and pulmonic valve regurgitation, mild left and right atrial enlargement, mild RV dilatation with mild dysfunction, and left ventricular ejection fraction of 60% to 65%. The patient’s most recent right heart catheterization demonstrated an elevated PAP of 87/45 mm Hg.

Preoperative vital signs were as follows: blood pressure, 111/72 mm Hg; heart rate, 82/min in normal sinus rhythm; respiratory rate, 18/min; and pulse oximeter reading, 100% on room air. Results of the physical examination revealed a clean, well-groomed woman with clear lung sounds, and neither jugular venous distension nor peripheral edema was noted. Findings of her airway examination revealed a Mallampati class 2 with normal cervical range of motion, thyromental distance, oral opening, and intact dentition. She had had nothing by mouth for an estimated 12 hours.

Because of the severity of the patient’s PH, the surgery was not a typical laparoscopic cholecystectomy. She was in a high-risk category for perioperative morbidity and mortality.3 Before surgery, several precautionary measures were taken to avoid an acute pulmonary hypertensive crisis. There was a discussion between the surgical and anesthesia teams regarding the relationship between abdominal insufflation pressures and the development of hypercarbia. Pneumoperitoneum can decrease preload and increase afterload as well, which are poorly tolerated in the patient with PH. The surgeon agreed to minimize his insufflation pressures as well as temporarily deflate the abdomen if necessary. Two units of packed red blood cells were available to be brought into the operating room upon the start of the case, because patients with PH do not tolerate acute blood loss. An epinephrine drip was ready and programmed on the infusion pump, to be immediately available if needed. Additionally, the respiratory therapy department was made aware of the possible need for intraoperative nitric oxide in case a rapid pulmonary vasodilator was needed to reduce PAP.

After entering the operating room, the patient was positioned supine, and a low dose of 0.375 μg/kg/min of milrinone, a phosphodiesterase-3 inhibitor, was initiated.
IV. The patient was preoxygenated for approximately 8 minutes with 8 L/min of 100% oxygen by a face mask while the following monitors were applied: an electrocardiogram, an automated noninvasive blood pressure cuff to the left upper arm, and a pulse oximeter probe to the right index finger. During the preoxygenation period, and after an Allen test, a right radial artery blood pressure monitoring line was placed. The arterial line was verified with blood return and “zeroed” to atmospheric pressure, and a good waveform was noted. The patient’s baseline vital signs were checked and were similar to her preoperative values. Anesthesia was then induced intravenously with 2 mg of midazolam, 100 μg of fentanyl, 24 mg of etomidate, and 140 mg of succinylcholine through a 16-gauge peripheral IV catheter in her left hand. Using a modified rapid sequence technique, the trachea was intubated with a cuffed 7.0-mm endotracheal tube. The patient was initially given volume-controlled ventilation. Anesthesia was maintained with inhaled isoflurane in 100% oxygen. A prophylactic antibiotic was given IV, 3 g of ampicillin and sulbactam (Unasyn). The surgical team placed a Foley catheter.

After induction of anesthesia, a 9F introducer with pulmonary artery (PA) catheter was placed in the patient’s right internal jugular vein, and a transesophageal echocardiogram (TEE) probe was placed in the esophagus, both without difficulty. An upper-body, forced-air warming device was used to maintain normothermia. Upon the return of a full train-of-four twitch via neuromuscular monitoring of the facial nerve, neuromuscular blockade was maintained with IV boluses of rocuronium.

When the surgeons initiated 15 mm Hg of abdominal insufflation for the laparoscopy, the patient was administered pressure-controlled ventilation because the peak inspiratory pressure elevated above 25 cm of water. Ventilation parameters during the case were adjusted to avoid factors that increase PVR such as hypoxemia and hypercarbia. Maximization of oxygenation and ventilation were achieved by providing the patient with low tidal volumes, avoidance of hypercarbia, and maintaining peak inspiratory pressures below 25 cm of water.

Throughout the surgery, the patient’s vital signs remained stable. Her adjusted ventilatory rate was 8 to 14/min; pulse oximetry was 93% to 100%; sinus rhythm was 43 to 65/min; arterial blood pressure ranged from 100/50 to 140/72 mm Hg; end-tidal carbon dioxide was 32% to 37%; core temperature was stable at 36.5°C; central venous pressures were 9 to 13 cm of water; cardiac output, measured by thermodilution, was 5.2 L/min; and PA systolic pressures ranged from 60 to 80 mm Hg.

Continuously throughout the case, a trained provider monitored the TEE. The intraoperative TEE examination showed the following findings: dilated left and right ventricles, ejection fraction of 50% with mild decrease in global function.

Hypokalemia (potassium, 3.0 mM/L) and hypocalcemia (ionized calcium, 1.10 mM/L), determined via intraoperative arterial blood gas sampling, were treated with 20 mEq potassium chloride and 0.5 g of calcium chloride, respectively. The low potassium level may have been the result of an intracellular shift due to increased circulating catecholamine levels. The potassium level eventually corrected to 4.0 mM/L. Postoperative nausea and vomiting was prophylactically treated with 8 mg of ondansetron and 8 mg of dexamethasone, 45 minutes before the end of the case. Fluids for the case totaled 900 mL of 0.9% normal saline; 375 mL of urine output; and 10 mL of estimated blood loss.

When the surgeons finished the cholecystectomy, neuromuscular blockade was reversed with a combination of 2.5 mg of neostigmine and 0.6 mg of glycopyrrolate, and the patient was weaned from mechanical ventilation. During emergence and extubation, the patient experienced a peak PA systolic pressure of 115 mm Hg and a peak systolic arterial blood pressure of 170 mm Hg. These pressures were immediately treated with 3 doses of nitroglycerine, for a total of 240 μg, and 2 doses of 50 μg of fentanyl over 5 minutes. The patient’s systolic PAP and systolic arterial blood pressure returned to the baseline of 85 and 125 mm Hg, respectively. Postoperatively, the patient began shivering. She was treated with a total of 20 mg of meperidine and the application of warm blankets. Total surgical time was 2 hours and 45 minutes.

The patient was transported to the cardiothoracic intensive care unit with full hemodynamic monitoring, 8 L/min of oxygen via a simple face mask, and the initial IV milrinone infusion continued at 0.375 μg/kg/min, without incidence. The patient was discharged home 2 days later.

At her 2-month follow-up visit, the patient reported that she was continuing to work on a full-time basis, that her energy level remained good, and that further symptoms of her IPAH had not worsened.

Discussion

Although mild PH usually does not influence anesthesia,4 moderate to severe forms of the disease are predictors of adverse outcomes in noncardiac surgery.5 It is imperative that anesthesia providers are prepared to manage these patients. Even though there have been major advances in monitoring and treatments of PH, according to Price et al,6 patients with PH are of the highest risk cohorts to undergo noncardiac, as well as cardiac, surgical procedures. It is essential that the anesthesia professional be familiar with PH, as well as the effects of anesthetic drugs on the pulmonary circulation.4 The following discussion includes history, current classifications, epidemiology, pathophysiology, contemporary treatments, and anesthetic management related to PH.
History and Classification. Ernst von Romberg made the first mention of pulmonary vascular sclerosis in 1891.2 But it was not until 1973 that the World Health Organization held the first international conference on PH.7 Since that time, the classification system for PH has been through many changes, with the latest revisions made in 2008 in Dana Point, California.8 According to Simonneau et al.,8 the goal of the classification system is to differentiate pathophysiology, presentation, and therapies for the many diseases under the umbrella of PH. The current classification system is divided into 5 main groups based on the cause (Table 1).8

In addition to the classification system that describes the individual PH diseases, patients are divided into 4 functional classes according to the New York Heart Association based on their degree of disability (Table 2).9

Epidemiology. Various national and international agencies maintain registries of people with PH.2 An estimated 15 to 52 people in 1 million have PAH worldwide.7,9 Most people with IPAH are female, but the reasons for this are unclear.2 Most patients with PAH are older than 45 years and have a body mass index greater than 29 kg/m².2 Unfortunately, the prognosis of PAH is poor, and untreated patients often die within 2 to 3 years of diagnosis.9 Price et al8 report that despite contemporary medical treatments, there is a 15% mortality rate at 1 year after diagnosis.

Pathophysiology. The definition of PH is persistent elevation of the mean PAP greater than 25 mm Hg at rest or greater than 35 mm Hg with exercise.4 Further parameters include the following: mild PH, mean PAP of 25 to 40 mm Hg; moderate PH, mean PAP of 41 to 55 mm Hg; and severe PH, mean PAP of greater than 55 mm Hg.1 As discussed earlier, PH is progressive and there is no cure. For years, it was thought that PH was a disease of imbalance between vasoconstriction and vasodilation; however, today it is understood that the cause is more complex. Current theories suggest that increased pulmonary pressures are due to vascular remodeling that results from excessive abnormal proliferation of cells and reduced apoptosis, as well as thrombosis and the dominance of vasoconstriction.1,2,9,10

Eventually in PH, the PAP and PVR are increased. These higher pressures in the pulmonary system lead to an increased RV afterload that in turn causes RV hypertrophy and dilation. It is these RV changes that result in RV dysfunction and, ultimately, in RV failure.2,7,11

Symptoms that the patient may experience with PH are often nonspecific, such as dyspnea on exertion, chest pain, and syncope.1,4 Signs that may be seen on physical examination are tachypnea, tachycardia, jugular venous distention, ascites, and lower extremity edema.2 Factors that aggravate PH are hypoxemia, hypercarbia, acidosis, hypothermia, hypervolemia, increased intrathoracic

<table>
<thead>
<tr>
<th>Main group</th>
<th>Classification</th>
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<tbody>
<tr>
<td>1. Pulmonary arterial hypertension</td>
<td>1.1 Idiopathic</td>
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<tr>
<td></td>
<td>1.2 Heritable</td>
</tr>
<tr>
<td></td>
<td>1.3 Drug/toxin induced</td>
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<tr>
<td></td>
<td>1.4 Associated with various comorbidities</td>
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<tr>
<td></td>
<td>1.5 Persistent PH of the newborn</td>
</tr>
<tr>
<td>2. PH from left heart disease</td>
<td>2.1 Systolic dysfunction</td>
</tr>
<tr>
<td></td>
<td>2.2 Diastolic dysfunction</td>
</tr>
<tr>
<td></td>
<td>2.3 Valve disease</td>
</tr>
<tr>
<td>3. PH from lung disease and/or hypoxia</td>
<td>3.1 COPD</td>
</tr>
<tr>
<td></td>
<td>3.2 Interstitial lung disease</td>
</tr>
<tr>
<td></td>
<td>3.3 Other pulmonary diseases</td>
</tr>
<tr>
<td></td>
<td>3.4 Sleep-disordered breathing</td>
</tr>
<tr>
<td></td>
<td>3.5 Alveolar hypoventilation disorders</td>
</tr>
<tr>
<td></td>
<td>3.6 Chronic exposure to high altitude</td>
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<tr>
<td></td>
<td>3.7 Developmental abnormalities</td>
</tr>
<tr>
<td>4. Chronic thromboembolic PH</td>
<td>None</td>
</tr>
<tr>
<td>5. PH with unclear or multifactorial causes</td>
<td>5.1 Hematologic disorders</td>
</tr>
<tr>
<td></td>
<td>5.2 Systemic disorders</td>
</tr>
<tr>
<td></td>
<td>5.3 Metabolic disorders</td>
</tr>
<tr>
<td></td>
<td>5.4 Others</td>
</tr>
</tbody>
</table>

Table 1. Classification of Pulmonary Hypertension
Abbreviations: COPD, chronic obstructive pulmonary disease; PH, pulmonary hypertension.
(Adapted from Simonneau et al.9)
Abbreviation: PAH, pulmonary arterial hypertension.

<table>
<thead>
<tr>
<th>Main class</th>
<th>Functional description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>PAH without physical activity limitations. No dyspnea or fatigue, chest pain, or syncope.</td>
</tr>
<tr>
<td>2</td>
<td>PAH causes minor limits on physical activity. Rest is comfortable, but regular physical activity causes dyspnea or fatigue, chest pain, or syncope.</td>
</tr>
<tr>
<td>3</td>
<td>PAH causes major limits on physical activity. Rest is comfortable, but minor physical activity causes dyspnea or fatigue, chest pain, or syncope.</td>
</tr>
<tr>
<td>4</td>
<td>PAH causes symptoms with any physical activity. Evidence of right-sided heart failure, and patient may have symptoms at rest.</td>
</tr>
</tbody>
</table>

Table 2. Classification of Heart Failure
Abbreviation: PAH, pulmonary arterial hypertension. [Adapted from Burt et al.]

Pressure, and pain.4,7 Patients with inexplicable dyspnea necessitate further inquiry.2 In advanced PH, presentation includes a high degree of disability as well as right heart failure, RV enlargement, and dysfunction. Surgery for patients with advanced PH should only be considered if necessary for lifesaving measures.9,10

- Contemporary Treatments of Pulmonary Hypertension

Over the past 10 years, the research, understanding, and treatment options for PH have increased dramatically.9 With this expansion in knowledge base have come improved therapeutic strategies. The aim of drug therapies is to improve quality of life by reducing symptoms and increasing years of survival.7 The management of PH is currently treated with many oral, IV, and inhaled drugs. There are 4 main pharmacologic classes of oral and IV drugs for treating PH: calcium channel blockers, prostanoids, endothelin-1 receptor antagonists, and phosphodiesterase inhibitors.2,9,10

Calcium channel blockers work to reduce available calcium to cardiac myocytes and arterial smooth muscle cells, which results in vasodilation, negative inotropism and negative chronotropism.9 It is important to note that while calcium channel blockers are often used in chronic PH they are not recommended in the acute setting, as many patients with PAH do not respond to them.2,9 Common calcium channel blockers include nifedipine, diltiazem, and amlodipine. Verapamil is not recommended because of its greater negative inotropism and relative cardiac selectivity.9,10

Prostanoids, also known as prostacyclin analogs or prostaglandins, are synthetic versions of prostacyclin.12 Prostacyclin is a potent vasodilator, which works on systemic and pulmonary vessels. Prostacyclin is also an inhibitor of platelet aggregation, helping to reduce the incidence of thrombosis.9,10 Because of possible coagulation issues, extra caution is necessary when administering neuroaxial anesthesia in patients who have been receiving the IV form of these drugs long term.10,13 The IV form of prostanoids can cause systemic hypotension because they are not selective to the pulmonary circulation.4 These drugs have a short half-life and are often delivered as continuous infusions.9,10 Predominate prostanoids are epoprostenol, iloprost, and treprostinil.2

Endothelin-1 receptor antagonists exert action by blocking endothelin receptors on endothelial cells to limit the powerful vasoconstrictor endothelin-1.9,10 These drugs are not used in the operative setting but can have a positive effect on the vasodilation of the pulmonary vasculature of patients with PH, increasing their exercise tolerance.7,9 Monthly blood and liver function tests are recommended while a patient is receiving endothelin antagonists because of possible side effects such as hepatotoxicity, teratogenicity, anemia, and peripheral edema.2,10,12 Some currently used oral endothelin-1 receptor antagonists are bosentan, sitaxsentan, and ambrisentan.9

Phosphodiesterase inhibitors selectively target the enzymes that break down cyclic guanosine monophosphate (cGMP) and cyclic adenosine monophosphate, allowing a buildup of intracellular concentrations, which overall increases cardiac inotropism while also providing vasodilation.13 These drugs also inhibit the growth of vascular smooth muscle cells.9 Phosphodiesterase-5 is exclusive to the pulmonary vasculature, whereas phosphodiesterase-3 is selective to cardiac and vascular tissue.12 Both types of phosphodiesterase inhibitors can cause systemic hypotension.12 The primary phosphodiesterase-5 inhibitor is sildenafil, and the main phosphodiesterase-3 inhibitor is milrinone.2

Additional medications used in the management of PH include nitroglycerin and sodium nitroprusside.4 Both agents produce vasodilation by relaxing smooth muscle by releasing nitric oxide from tissues, resulting in systemic hypotension.4,12 These agents must be used with caution because of the risks associated with hypotension causing RV ischemia and failure in patients with PH.4

In addition to the oral and IV drugs already mentioned, inhaled vasodilators are drugs that should be considered for the intraoperative decompensation of PH.2,4,10 These inhaled vasodilators decrease RV afterload and improve cardiac output, but do not produce systemic hypotension.13 However, inhaled vasodilators can cause rebound PH when discontinued.2

To provide specific localized pulmonary vascular dilation, inhaled nitric oxide (INO) is delivered as a gas.4,10 Inhaled nitric oxide increases cGMP production, which
relaxes pulmonary vascular smooth muscle leading to
vasodilation.\textsuperscript{10,12} It has the ability to increase arterial
oxygenation by reducing intrapulmonary shunting and
improving ventilation.\textsuperscript{4} Two advantages of INO include
a reliable dose delivery system and it does not produce
systemic hypotension.\textsuperscript{8} Unfortunately, inhaled INO is
costly and requires specialized equipment for delivery.\textsuperscript{2}
A common dosage range is 20 to 40 parts per million.\textsuperscript{10}

Inhaled prostaglandins (prostanoids) are also used in
the management of acute PH. Epoprostenol and iloprost
provide effective pulmonary arterial vasodilation without
systemic effects.\textsuperscript{2} The inhaled prostaglandins are less
costly than INO and are delivered via a nebulizer atta-
tached to the ventilator circuit.\textsuperscript{3} One of the downsides to
inhaled prostaglandins is their short half-life, and some
require treatments of 6 to 9 times daily.\textsuperscript{9} Additionally,
even the inhaled form of prostaglandins can inhibit plate-
let aggregation, as mentioned previously.\textsuperscript{12}

• Anesthetic Management. Preoperative evaluations
for the patient with PH should include electrocardiogra-
phy, chest radiography, arterial blood gas analysis, echo-
cardiography, right heart catheterization.\textsuperscript{1,7} Right-sided
heart catheterization is the gold standard for diagnosis of
PH. Ideally before surgery, mean PAP should be reduced
to a normal of 25 mm Hg.\textsuperscript{1,7} If substantial RV dysfunction
is present, the advisability of surgery should be reexam-
inied.\textsuperscript{3} Any chronic pulmonary hypertensive therapies
that patients are currently taking should be continued perioperatively.\textsuperscript{1,2,7,11}

Preexisting PH is a major cause of morbidity and
mortality irrespective of anesthetic methods in surgical
patients.\textsuperscript{1,3,9,6} Nonetheless, in patients with stable forms
of PH, surgical procedures can be undertaken as long as
the surgery is managed by clinicians experienced in the
treatment of PH.\textsuperscript{6,9}

There is controversy regarding the effects of specific
inhahalional agents and their effects on pulmonary cir-
culation. Questions also have arisen in published studies
with regard to optimal anesthetic technique in PH. For
example, Lai et al\textsuperscript{5} state that the “pulmonary-specific
vasoactive effects are incompletely known” with isoflu-
rane, desflurane, sevoflurane, propofol, and bupivacaine
despite their known peripheral vasodilation properties.
Lai et al\textsuperscript{5} report that no clear clinical benefit has been
found when using one anesthetic instead of another in
patients with PH. On the other hand, Price et al\textsuperscript{6} cite 2
studies that have shown worse pulmonary vascular
effects with desflurane than isoflurane. Price et al\textsuperscript{6} also
suggest in their study that the use of general anesthesia
in PH was linked to worse patient outcome than regional
anesthesia. However, Lai et al\textsuperscript{5} report that no significant
difference was found in their study between various
anesthetics or techniques in relation to intraoperative
outcomes.

Regardless of the particular anesthetic or technique
used, there are clear goals for managing patients with
PH. Intraoperative management must be geared toward
preventing factors that worsen PH and increase PVR:
hypoxemia, hypercarbia, acidosis, hypothermia, hyper-
volemia, increased intrathoracic pressure, and pain.\textsuperscript{4,7}
The priorities in any surgical procedure for a patient with
PH involve hemodynamic stability and optimization of
RV function by maintaining preload and contractility.\textsuperscript{7,10}

Hypotension needs to be quickly corrected because a
decrease in RV perfusion pressure can lead to RV failure
due to worsening of PH and finally cardiac arrest.\textsuperscript{7,10}
Regarding the use of inotropes and vaspressors, there
are conflicting studies. Because of the lack of clinical
trials, many authors are reluctant to give specific recom-
endations.\textsuperscript{4} For example, Subramaniam and Yared\textsuperscript{4}
discover that one study found vasopressin to be a pul-
monary vasoconstrictor whereas another study claimed
it dilated the pulmonary arteries. However, Fox et al\textsuperscript{11}
state that vasopressin is recommended for hypotension.
In regard to phenylephrine, Pritts and Pearl\textsuperscript{7} endorse
its use, whereas Subramaniam and Yared\textsuperscript{4} suggest using
norepinephrine and vasopressin over phenylephrine.
Among 5 different authors, ephedrine was not mentioned
in the treatment options for hypotension in patients with
PH.\textsuperscript{1,2,4,7,10} Another point of agreement is that hypoten-
sion must be treated aggressively and without hesitation
according to the cause.

Advanced monitoring during the surgery is essential.
Optimal monitoring includes continuous arterial blood
pressure monitoring, serial blood gas analysis, pulmonary
artery catheter measurements, central venous pressure
assessment, cardiac output evaluation, and TEE.\textsuperscript{1,10,11}

Many interventions can be used to treat intraoperative
elevations of PAP. Ensure that the patient is adequate-
ly anesthetized. Check for acid/base disturbances and
correct imbalances.\textsuperscript{11} Maintain normothermia and treat
hypothermia.\textsuperscript{1} During positive pressure ventilation, aim
for optimal ventilation and oxygenation.\textsuperscript{1} Recommended
positive pressure parameters include the following: a
high oxygen concentration, low tidal volumes of 6 mL/
kg, a respiratory rate to attain mild hypocarbia, and a low
positive end-expiratory pressure of 5 to 10 cm water.\textsuperscript{1,10}
Pulmonary vasodilator therapy, both in the IV and
inhaled forms, is indicated when PH persists.\textsuperscript{1,4,10}

In the postoperative period, it is recommended that
patients with PH be closely monitored in the intensive
care unit because as intraoperative medications are elimi-
nated from the body, there can be an acute aggravation
describe the general characteristics of PH in noncardiac, nonobstetric surgery.5,6 This case study was written in the hope that the nurse anesthesia community might expand its knowledge about this disease and gain insight into ways to address basic perioperative management of these high-risk patients.

REFERENCES


AUTHOR

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### Table 3. Anesthetic Implications of Severe Pulmonary Hypertension

<table>
<thead>
<tr>
<th>Period</th>
<th>Anesthetic implication</th>
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<tbody>
<tr>
<td>Preoperative</td>
<td>Thoroughly assess PH severity</td>
</tr>
<tr>
<td></td>
<td>Avoid anxiety, pain, and sympathetic stimulation</td>
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<td></td>
<td>Avoid oversedation and hypoventilation</td>
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<td></td>
<td>Continue all PH-specific, long-term therapy perioperatively</td>
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<tr>
<td>Intraoperative</td>
<td>Use appropriate invasive monitoring</td>
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<td></td>
<td>Provide adequate anesthesia</td>
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<td></td>
<td>Positive pressure ventilation parameters</td>
</tr>
<tr>
<td></td>
<td>• High oxygen concentration</td>
</tr>
<tr>
<td></td>
<td>• Low tidal volumes (6 mL/kg)</td>
</tr>
<tr>
<td></td>
<td>• Respiratory rate to attain mild hyperventilation</td>
</tr>
<tr>
<td></td>
<td>• Low PEEP (5-10 cm H₂O)</td>
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<tr>
<td></td>
<td>Aggressively treat hypotension</td>
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<tr>
<td></td>
<td>Avoid heart rate extremes</td>
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<tr>
<td></td>
<td>Optimize RV function and CO with adequate preload, SVR, and contractility</td>
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<tr>
<td></td>
<td>Monitor and treat RV dysfunction</td>
</tr>
<tr>
<td></td>
<td>Avoid factors that worsen PH and increase PVR</td>
</tr>
<tr>
<td></td>
<td>• Hypoxemia</td>
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<tr>
<td></td>
<td>• Hyperventilation</td>
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<tr>
<td></td>
<td>• Increased intrathoracic pressure</td>
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<td></td>
<td>• Pain</td>
</tr>
<tr>
<td></td>
<td>Consider pulmonary vasodilators to decrease RV afterload</td>
</tr>
<tr>
<td>Postoperative</td>
<td>Avoid pain and shivering</td>
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<tr>
<td></td>
<td>Monitoring</td>
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</table>

of PH and RV ischemia, leading to death.1 Both Lai et al1 and Kaw et al3 reported that after general anesthesia, patients with PH were more likely to have a higher incidence of major adverse events postoperatively, including prolonged intubation and heart failure. Additionally, it is critical to gradually wean the patient from mechanical ventilation to avoid inducing elevation of PAP and to control hypoxia, acidosis, and pain. Extubate the patient only when conditions are optimal (Table 3).1

### Conclusion

Regrettably, many anesthesia texts have limited content regarding PH.14,15 Unfortunately, few studies exist that