Postoperative Care of the Patient With Pulmonary Hypertension

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Summary: Pulmonary hypertension (PH) is a known contributor to increased morbidity and mortality in cardiac and noncardiac surgery.1 Careful postoperative management is necessary to prevent worsening right ventricular (RV) dysfunction and failure. Attention must be paid to volume status (both hypervolemia and hypovolemia are deleterious), oxygen levels, and acid-base status. Continued administration of pulmonary arterial hypertension (PAH)-specific medications is imperative in the postoperative period; alternative delivery of outpatient medications, adjustment of dosing, or even changes in medication class may be required based on the clinical situation (eg, ability to swallow, dexterity and coordination for inhaled therapy, renal and hepatic functions, etc). Adequate pain control is also important as activation of the sympathetic nervous system can cause worsening PH; however, this

CASE: SH is a 46-year-old woman with idiopathic pulmonary arterial hypertension (IPAH) (World Health Organization [WHO] Group 1), New York Heart Association functional class (NYHA FC) I, treated with bosentan and sildenafil as well as with anticoagulation. She was diagnosed with IPAH 10 years prior and her most recent right heart catheterization (RHC) showed a right atrial pressure of 7 mm Hg, mean pulmonary artery pressure (mPAP) of 48 mm Hg, cardiac output of 5.3 L/min, and pulmonary vascular resistance (PVR) of 7.1 Wood units. She was transferred to our intensive care unit with vaginal bleeding significant enough to cause systemic hypotension associated with a cardiac troponin leak and requiring multiple units of transfusions. Uterine artery embolization was attempted by interventional radiology at our institution, but the patient continued to have significant bleeding postprocedure. The patient ultimately was urgently taken to the operating room and underwent dilation and curettage while receiving general anesthesia. The patient tolerated the procedure well, was extubated, and transferred to the intensive care unit on 100% oxygen delivered by face mask.

The surgical team requests consultation regarding postoperative management. What advice would you give?

must be balanced with the potential for systemic effects of analgesics to negatively affect cardiopulmonary function. While these and other factors likely impact the clinical course and outcomes of patients with PH who undergo surgery, there are scant data in the literature to guide therapy. Taken together, these issues highlight the challenges that exist in the postoperative care of patients with PH. In this manuscript, we present our approach to the postoperative management of PH patients. A general overview of this approach is presented in Figure 1.

LOGISTICS

Good postoperative care of a PH patient starts in the preoperative period. As discussed in detail elsewhere in this issue of *Advances in Pulmonary Hypertension*, preoperative assessment is critically important in PH patients. Postoperative

complications are likely related to preoperative factors concerning the patient, planned surgery, and anesthesia (see below). One practical aspect of preoperative assessment pertains to postoperative logistics. Before a PH patient is scheduled for surgery, we have found it helpful to determine in which unit(s) the patient will likely reside postoperatively and whether the staff in these units are trained in inhaled, subcutaneous, and intravenous prostacyclin administration. It is imperative to ensure that the patient will be cared for in a unit where nursing staff are experienced in both taking care of PH patients and their medical regimen. If there is a lack of experience with the management of these medications, unit staff should be in-serviced prior to the patient's scheduled surgery. In our hospital, we often arrange to bypass the postoperative recovery unit for inpatient procedures and return patients to our intensive care unit. Further, we often admit patients overnight for monitoring even for procedures that are generally performed on an outpatient basis.

We ask that our PH faculty and staff

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that would influence postoperative management. Further, we discuss with the surgeon whether it is feasible to avoid PATIENT CONSIDERATIONS A myriad of patient factors may also influence complications postoperatively. However, there are few studies exampostoperative complications in PH. In a recent prospective study of 114 patients with PAH undergoing noncardiac and nonobstetric surgery, Meyer and colleagues reported a 3.5% perioperative mortality rate, considerably less than the 7%-18% suggested in smaller retrospective studies.⁴ The risk factors for major complications (bleeding greater than 1 liter, systemic inflammatory response or sepsis requiring vasopressor therapy, right heart failure [RHF] requiring inotropic support, or death) after surgery were a pre-operative 6-minute walk distance of less than 399 meters and a right atrial pressure of greater than 7 mm Hg. Our patient's

most recent RHC, 2 years prior, showed a right atrial pressure of 7; her 6-minute walk distance was 516 meters. Ramikrishna et al did not find that 6-minute walk distance affected short-term morbidity after noncardiac surgery, but did find that NYHA FC II

Hemodynamically Stable Hemodynamically Unstable **Requiring Mechanical Ventilation** Liberated From Mechanical Ventilation Somnolent V Intensive Care Unit Postoperative Care Unit/Recovery Management Principles **Management Principles** • Avoid IV fluid boluses ≈8 mL/kg tidal volume, PEEP≤10 Avoid hypoxia/hypercarbia Avoid IV vasopressors with Avoid fluid boluses/transfusions** predominantly peripheral vasoconstrictor activity* Avoid IV vasopressors with predominantly Continue oral/IV/SC PAH medications peripheral vasoconstrictor activity* Administer diuretics Continue oral/SC/IV PAH medications • Pain control Inhaled NO Administer diuretics Pain control DVT prophylaxis . Early mobilization Monitored Telemetry Bed Management Principles Avoid IV fluid continuous infusion Continue oral PAH medications *See text for details. Administer diuretics **Transfusions of fresh frozen plasma or Low-salt diet/fluid restriction platelets should be avoided; transfusions of Chest physiotherapy/incentive packed red blood cells should be followed spirometer/flutter valve with intravenous diuretics. See text for details Early mobilization . DVT prophylaxis

Postoperative Pulmonary Hypertension Patient

Figure 1. Overview of postoperative approach for patients with pulmonary hypertension.

are notified as soon as our patients are out of the operating room; one member of our team then sees and evaluates the patient as soon as possible, within a maximum of 30 minutes. In addition to reviewing the operative note and anesthesia flow sheets, the PH team member assesses the patient, confirms that PH medications are ordered correctly and immediately postoperatively, and ensures that medications have been given appropriately. Further, he or she reviews management principles (see Figure 1) with the nursing staff and house staff to prevent common therapeutic errors. A member of our PH team sees the patient daily during the admission. We ask our surgical colleagues to defer assessment of medical readiness for discharge and often will assume primary care of the patient when the acute surgical issues have resolved.

SURGICAL AND ANESTHESIA CONSIDERATIONS

The type of surgery and anesthesia used influence the perioperative risk and postoperative management. Multiple factors in each of those realms influence this risk, as shown in Table 1. For example, as one would expect, emergency surgeries are associated with greater mortality than nonemergent procedures. To mitigate this risk in our patient's case, we recommended a shorter stabilizing procedure (dilation/curettage) for urgent control of bleeding. The hysterectomy, the definitive but more extensive surgery, was

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Table 1. Surgical and Anesthetic Risk Factors for Postoperative Complications

Higher Risk
Vascular, Cardiothoracic, Abdominal
Emergency
Greater Than 3 Hours
Propofol vs Etomidate
Nitrous or Halothane
Higher vs Lower Tidal Volume
Нурохіа
Hypercapnia

or higher, history of pulmonary embolism, RV hypertrophy, RV systolic pressure to systolic blood pressure ratio >0.6, and RV index of myocardial performance index (Tei index, calculated by dividing the sum of RV isovolumic relaxation and contraction times by the ejection time interval) >0.75 were associated with increased morbidity.

We also consider non-PH-specific patient factors when assessing perioperative risk. Multiple groups have looked at patient factors that influence outcomes in patients admitted for RHF. High respiratory rate, renal dysfunction, hyponatremia, severity of tricuspid regurgitation, systolic blood pressure <100, and the presence of connective tissue disease have each been independently found to be associated with mortality. While some of these factors are immutable and admissions for RHF are certainly different than for surgery, we use these factors as a guideline when considering operative risk preoperatively along with postoperative management and risk assessment.

Based on these studies and our clinical experience, our approach is to assess NYHA FC, 6-minute walk distance, N-terminal brain natriuretic peptide levels (NT-proBNP), renal function, and echocardiography in all PH patients prior to any procedure that requires general anesthesia. If a patient demonstrates significant differences from prior evaluations (typically, we collect these data yearly for clinical purposes), then we augment therapy by increasing diuresis (if clinically indicated) or adding PAH therapy. If a patient requires higher risk surgery (Table 1), we prefer to repeat the RHC to directly assess PH severity and to guide preprocedure interventions (such as increasing diuresis or adding PAH-specific therapy). Occasionally, we will repeat the RHC to determine response to these interventions prior to the planned procedure, particularly if the patient has severe PH. We look for any improvement in pulmonary artery pressures or cardiac output, or decrease in right atrial pressure and PVR. We do not routinely look for a nitric oxide response unless the patient has demonstrated a prior response to nitric oxide and we want to ensure that this response has endured. Due to the urgent nature of the required intervention, we did not perform a RHC prior to our patient's procedure. However, we did obtain NT-proBNP and an echocardiogram prior to surgery; the NT-proBNP was significantly elevated at 10,500 pg/mL (normal range 0-135 pg/mL), and the echocardiogram demonstrated RV pressure and volume overload with significantly reduced systolic function when compared to the echocardiogram from 1 year earlier. Thus, based upon these data, we recommended a shorter, stabilizing procedure due to our concerns for peri- and postoperative complications.

PULMONARY HYPERTENSION MEDICATIONS

Our patient was on sildenafil and bosentan prior to surgery, both oral medications. Other PH patients may be on continuous intravenous, subcutaneous, or inhaled prostacyclin analogues. Sudden withdrawal of pulmonary vasodilators may result in rebound PH.

Further, perturbations in cardiopulmonary function resulting from the effects of anesthesia, MV, and infusions of fluid can adversely impact RV function. Thus, maintenance of preoperative PAH medications is of paramount importance. We review and potentially change the time schedule of our patients' PH medications to ensure that daily PH medications are taken on the morning of surgery rather than the night before, and inhaled therapy times are adjusted to allow for a dose to be taken on-call to the operating room. If a patient is on subcutaneous or intravenous prostacyclin therapy, close monitoring and collaboration with the anesthesiology team is required. Additionally, close attention must be paid to the dosing of PH medications if a patient experiences systemic hypotension. Because 1) systemic hypotension may be related to multiple potential etiologies including sepsis, hemorrhage, and acute RHF (among others), and 2) reduction or cessation of pulmonary vasodilators may further exacerbate the systemic hypotension, we ask that any changes to PAH-specific medications be approved by our PH faculty and staff after careful evaluation of the patient. Additionally, issues of potentially increased toxicity or decreased bioavailability related to acute liver, kidney, or gastrointestinal injury must be considered; often we will consult with critical care pharmacists to help with medication dosing in these situations.

Before surgery, a backup plan should be made if a patient is unstable or unable to take oral medications; we often use inhaled nitric oxide (iNO) for pulmonary vasodilation in place of oral agents as a temporizing measure. Availability of this agent varies by hospital and should therefore be confirmed prior to an individual patient's surgery. Intravenous sildenafil is available commercially, but may not be on formulary at a particular institution. Dosing of intravenous sildenafil is 10 mg 3 times a day, which is equivalent to 20 mg orally 3 times a day. Also due to variations in hospital formularies and availability of medications in general, we ask all patients to bring their PH medications to the hospital, even if the medication is on formulary. We ask

families to hold the patient's medications until the patient is admitted to the postoperative unit to minimize risk of misplacement. Our patient was maintained on her oral therapies perioperatively and did not require iNO.

BLEEDING AND THROMBOSIS

Many patients with PH are prescribed prostacyclin analogues, which carry a theoretical risk of bleeding. Observational studies have demonstrated a higher prevalence of thrombocytopenia in patients on intravenous prostacyclin.⁵ Additionally, patients with PAH have abnormalities in platelet function that may influence bleeding risk.⁶ However, studies have not shown any increased risk of postsurgical bleeding in PAH patients in general or in those using prostacyclin analogues.

If a patient develops a postoperative bleed, it is important to assess the severity of the bleed and if a surgical intervention is warranted. Transfusion of packed red blood cells (PRBC) should be used with an appropriate degree of caution, balancing the risks of bleeding with risks of volume overload and subsequent RV dysfunction. Typically, for the patient who develops hypotension in the setting of a bleed, we stabilize the patient with intravenous vasopressors while determining whether the bleeding site can be identified and corrected. We transfuse patients with life-threatening bleeding, but typically follow each unit of PRBC with intravenous diuretic to mitigate the volume effects on the pulmonary vasculature and RV. For patients who are anticoagulated, or have another bleeding diathesis such as thrombocytopenia and develop bleeding, a similar approach is employed. However, we have a very high threshold for correcting such coagulopathies with either fresh frozen plasma (FFP) or platelet transfusions due to concerns about acute worsening of PH. Both of these substances contain thromboxane A2, a potent pulmonary vasoconstrictor, which could theoretically increase RV afterload acutely.⁶ While purely anecdotal, we have had several patients experience poor outcomes immediately following transfusion of either FFP or platelets and thus avoid these transfusions if possible. Fortunately, our patient did not have a coagulopathy and did not require these blood products.

The majority of postoperative patients are at increased risk of pulmonary embolism. Patients with right heart dysfunction can decompensate with even a small pulmonary embolism. We therefore recommend immediate noninvasive mechanical deep venous thrombosis (DVT) prophylaxis and pharmaceutical prophylaxis when it is safe to do so. If a patient is anticoagulated for PH, anticoagulation should be restarted when safe from a surgical perspective; such patients do not need to be bridged. (The exception is patients with chronic thromboembolic PH, WHO Group 4 disease, who should be bridged with intravenous or subcutaneous anticoagulation.) Early mobilization strategies should also be employed.

ARRHYTHMIAS

Postoperative arrhythmias are common complications after surgery, affecting up to 60% of postcardiac surgery patients and up to 30% of noncardiothoracic surgery patients.⁷ In one study of PH patients undergoing noncardiac surgery, 12% developed cardiac dysrhythmia, defined as new onset atrial fibrillation, supraventricular tachycardia, bradycardia with conduction block, or ventricular tachycardia/fibrillation.8 Atrial arrhythmias, even when rate controlled, are poorly tolerated in patients with PH. The inability to restore sinus rhythm in this population is associated with high 1-year mortality;⁹ however, the short-term impact of new onset atrial arrhythmias is unknown. Management of PH patients with atrial arrhythmias postoperatively is challenging. Standard first-line medical therapy for rate control (calcium channel blockers and betablockers) is poorly tolerated in patients with significant PH due to potential negative inotropic effects on the RV and should be avoided in the emergent or urgent setting.

If a patient develops a new onset atrial (or other) arrhythmia, management principles do not vary from traditional practice beyond avoidance of betablocker and calcium channel blocker therapy. If a patient is hemodynamically

unstable with the new onset arrhythmia, then advanced cardiac life support protocols should be employed.¹⁰ If the patient is hemodynamically stable, then identification of potentially reversible causes of new onset atrial arrhythmias should be sought. Commonly, electrolyte imbalances, volume overload, or hypoxia may be the precipitant for these arrhythmias; treatment of these may resolve the problem. Other potential causes, such as pulmonary embolism, should be considered, evaluated, and treated if other etiologies are ruled out or if the clinical picture supports this diagnosis. In any case, our patients with atrial arrhythmias all receive cardiac monitoring. We tend to involve our electrophysiology team early in the process, as elective cardioversion may be indicated given the limited ability of medical therapies to safely return the patient to sinus rhythm. We treat our patients with digoxin and amiodarone (standard loading protocol followed by a maintenance dose of generally not more than 200 mg/day) if digoxin alone is not effective.

GENERAL POSTOPERATIVE CARE

As for any patient, good postoperative care in PH patients should focus on prevention of complications. Thus, DVT prophylaxis (as mentioned above) should be maintained. Early mobilization with the assistance of physical therapists if needed is also important. We ask for all of our patients to have daily standing (not in bed) weights and strict collection of intake and output to follow trends. Commonly, patients with PH will experience weight gain 24-48 hours postoperatively that likely reflects mobilization of third-spaced fluid. Augmentation of diuretic dosing is often required and is dictated by symptoms, examination findings of volume overload, and trends in weights and output. Removal of arterial lines and pulmonary arterial catheters should be done as soon as medically appropriate. Pulmonary toilet, with use of incentive spirometer and sometimes flutter-or a capellavalves, is useful to promote airway clearance and to reverse or prevent atelectasis. As discussed below, supplemental oxygen should be used to maintain high

oxygen saturations as hypoxia can worsen PH. We recommend checking ambulatory oxygen saturations as a patient nears discharge to see if he or she may require higher flow rates of supplemental oxygen than baseline or if he or she demonstrates a new oxygen requirement. New or higher flow requirements are often found in our PH patients postoperatively, but the need tends to resolve within a few weeks of discharge with appropriate convalescence. Our patient did develop a new oxygen requirement while in the hospital and was discharged with supplemental oxygen; she was able to discontinue this within 3 weeks of her surgery.

THE CRITICALLY ILL POSTOPERATIVE PH PATIENT Mechanical Ventilation

There is a paucity of evidence regarding ventilation strategies for MV in patients with PH. While increasing intrathoracic pressure by positive pressure ventilation may improve left ventricular (LV) function by reducing LV wall stress,² attendant increases in alveolar pressure lead to acute increases in mPAP and PVR. In the setting of rapid sequence intubation and subsequent MV, these effects on mPAP and PVR are exacerbated by the systemic effects of anesthetics, analgesics, and sedatives. Subsequent increases in RV afterload lead to decreased pulmonary blood flow and LV preload that will eventually cause systemic hypotension. As hypotension progresses, myocardial perfusion pressure to the RV drops as driving pressure falls and mean ventricular pressure rises; this may lead to RV ischemia, further compromising RV function¹¹ and exacerbating systemic hypotension. Therefore, minimizing the deleterious effects of MV in PH patients is an important consideration in the management of postoperative complications.

Physiologic studies suggest that PVR is lowest at functional residual capacity (FRC). We therefore recommend a low tidal volume ventilation (around 8 cc/kg ideal body weight) strategy to avoid hyperinflation and decreasing FRC, which increases PVR.¹² While these lower tidal volumes have traditionally been used in the acute respiratory distress syndrome (ARDS), other aspects of ARDS ventilator management should not be routinely employed in patients with right heart dysfunction. High level of positive end expiratory pressure (PEEP) should be avoided, as this can compress alveolar capillaries and cause an increase in PVR. Permissive hypercapnia is not well tolerated in PH patients as carbon dioxide can directly increase PVR; Viitanen et al studied 18 patients with hypercapnia after coronary artery bypass graft surgery and found that hypercapnia increased PVR by 54% and mPAP by 30%.¹³ Other studies have shown significant changes in echomeasured maximum tricuspid pressure gradient in response to hypercapnia, even in healthy volunteers.¹⁴ Similarly, hypoxia can induce increased PVR and RV afterload through pulmonary hypoxic vasoconstriction.¹⁵ Thus, whereas oxygen saturations of 90% are well tolerated by the majority of the population, at our center we aim for oxygen saturations of 95% in PH patients. Therefore, we often maintain PH patients on supplemental oxygen during the postoperative period to minimize the risk of hypoxic vasoconstriction. When on MV, we aim for high saturations while balancing the potential for oxygen toxicity.

It is our practice to minimize sedation in our mechanically ventilated patients to quicken liberation from the ventilator. Additionally, many sedatives cause systemic hypotension, which is poorly tolerated in this population. However, inadequate control of pain may lead to sympathetic nervous system activation that increases PVR.¹⁶ Optimal use of sedation and analgesia requires vigilant and careful management.

If a PH patient who was recently liberated from the ventilator develops respiratory distress, we first attempt noninvasive ventilation to prevent the need for invasive ventilation. Similarly, due to the negative effects of invasive positive pressure ventilation on RV function, we will liberate patients from the ventilator who may otherwise be borderline candidates for extubation and immediately place them on noninvasive bilevel support. In our experience, noninvasive support may be better tolerated from a hemodynamic perspective, allowing patients to improve clinically.

Hypotension and Vasopressors

Systemic hypotension is a common postoperative occurrence. However, patients with PH may have systemic hypotension at baseline; therefore, it is important to review outpatient records to determine baseline blood pressure. If hypotension is truly new in onset and if there are no overt signs of bleeding as the cause of the hypotension, then one must consider RV volume and/or pressure overload as the etiology of the systemic hypotension. Correction of hypoxia, hypercapnia, arrhythmia, and adequate pain control should be pursued along with other potential causes of hypotension in the ICU such as tension pneumothorax, cardiac tamponade, or sepsis. Unfortunately, frequently the hypotension is refractory to interventions to address these ancillary problems. Aggressive fluid resuscitation in patients with RV dysfunction should be avoided as this can cause right heart dilation and failure, so we often use vasopressors in our patients with PH and systemic hypotension.

At our center, we recommend the use of dopamine for hypotension.¹⁷ Dopamine use can be limited by the high incidence of arrhythmias associated with its use; however, at low "renal" doses (less than 5 mg/kg/minute infusion), we have found it useful to facilitate diuresis in the setting of RV failure and maintain systemic blood pressure.¹⁸ However, there remain limited randomized controlled data to support the use of dopamine for this purpose. When patients require additional vasopressor support, norepinephrine is often used in this capacity or as initial therapy if there is profound systemic hypotension. Norepinephrine stimulates α_1 and β_1 adrenergic receptors. While this medication does increase both mPAP and PVR, it does so to a lesser degree than other vasopressors while producing similar inotropic effects and supporting myocardial perfusion.

Recent animal studies suggest that dobutamine may offer better inotropic

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support for the RV than dopamine, and some experts recommend this agent as first-line therapy for right heart failure in PH.¹⁹ We find progressive systemic hypotension limits the utility of dobutamine in many of our patients; therefore, we tend to use it in combination with norepinephrine, particularly if RHF (and not volume depletion) is the major impetus for the hypotension. The majority of dobutamine's effect is seen at lower doses (5 mcg/kg/min or less). We prefer dobutamine to milrinone, a phospohdiesterase-3 inhibitor, as the latter causes more systemic hypotension and has a longer half-life. However, if dobutamine is not tolerated (ie, arrhythmia), milrinone at lower doses (ie, <0.375 mcg/kg/min) can be considered if the patient has relatively stable blood pressure and preserved renal function (since milrinone is renally excreted). Phenylephrine, a purely α_1 -adrenergic agent, should be avoided in patients with significant PH. This agent increases mPAP and PVR but decreases cardiac output thereby worsening right heart function.²⁰ It may also cause a reflex bradycardia, which can further lower cardiac output. There is a lack of data on the use of epinephrine in patients with PH; at our center we recommend avoiding its use. Vasopressin has historically been avoided because of increased mPAP and PVR with decreased cardiac output in animal models receiving high doses of this medication; however, lower doses may be safe and have been advocated for use by other PH experts as well tolerated and effective in the setting of severe PH, RHF, and hypotension.¹⁹

INHALED NITRIC OXIDE

The physical stress of surgery and anesthesia can contribute to RV failure in patients with PH. The keys to treating such failure are optimizing fluid status and reducing RV afterload. We liberally use iNO for the latter. Nitric oxide increases the production of cyclic guanosine monophosphate (cGMP), which reduces intracellular calcium and therefore relaxes smooth muscles. Inhaled nitric oxide acts on the pulmonary vasculature and in theory improves V/Q matching,²¹ as opposed to systemic vasodilators such as nitroprusside, which can worsen V/Q matching and cause systemic hypotension. Nitric oxide has been shown to decrease PVR, increase cardiac output, and increase the PaO_2 to FiO_2 ratio in patients with PH.²² The dose of iNO used ranges from 5 to 80 parts per million. If a patient has a pulmonary artery catheter, we titrate iNO dose according to cardiac output and PVR data from the catheter, but we do not require a catheter for its use. We follow daily methemoglobin levels in our patients receiving iNO as iNO can oxidize the heme iron to the ferric state; however, this complication tends to occur in patients who have prolonged exposure at high doses. We also advocate for weaning the iNO once a patient has stabilized. While reductions in dose from 80 ppm to 40 ppm are generally well tolerated, we have found slower weaning once the dose falls below 20 ppm to be a prudent strategy. We recommend reducing the dose by no more than 50% every 3 to 4 hours once the dose is below 20 ppm.

CONCLUSION

In general, good postoperative management of patients with PH depends on 1) a thorough preoperative evaluation (as dictated by urgency of surgery) to appropriately risk stratify patients; 2) good communication between the surgeon, anesthesiologist, and PH provider before, during, and after the surgery; 3) prior planning for postoperative care that includes level of care (ICU vs monitored bed), service (medical or surgical), delivery of PAH medications (type, route of administration, and schedule); and 4) institution of usual best practice postoperative care for non-PH patients (DVT prophylaxis, early mobilization, etc). Complications such as development of postoperative arrhythmias and hypotension require thoughtful management as typical interventions, such as intravenous calcium channel blocker therapy for atrial arrhythmia or intravenous phenylephrine infusion for hypotension, may be particularly harmful in the PH patient.

The recommendations proposed in this manuscript are based on experience at our center and are unencumbered by data (ie, there are no guidelines or randomized controlled trial data to which to refer). Additionally, these recommendations pertain to patients with PAH; patients with other forms of PH who require ICU care, for instance, may have specific considerations that would alter management (see Figure 2^{23}). For



Pulmonary hypertension in the ICU

WHO categories/disease aetiology

WHO: World Health Organization; RV: right ventricular; LV: left ventricular; BNP: brain natriuretic peptide; iNO: inhaled nitric oxide; PEEP: positive end-expiratory pressure; CVP: central venous pressure; SvO2: mixed venous oxygen saturation; PAH: pulmonary arterial hypertension; CO: cardiac output; SVR: systemic vascular resistance;

Figure 2. Pulmonary hypertension in the ICU. Reprinted with permission.²³

further reading regarding the ICU management of PH patients in general, we refer the reader to several excellent reviews on the subject.^{19,23-25}

OUR PATIENT'S OUTCOME

As noted throughout the article, our patient with severe PAH experienced a good outcome despite a significant hemodynamic insult from the massive vaginal bleed. Postoperatively, she did not require reinstitution of invasive MV or institution of noninvasive ventilation. She received her PAH-specific medications orally and did not require iNO. Her postoperative course was otherwise unremarkable and she was discharged home 3 days after the procedure; however, she did require supplemental oxygen for desaturation noted with ambulation. She returned for a total vaginal hysterectomy 3 months later under general anesthesia. She again tolerated this procedure well, was extubated prior to transfer to the intensive care unit, and was discharged home on the second postoperative day.

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