A Patient with Severe Pulmonary Hypertension for Hip Arthroplasty

Michael K. Urban, MD, PhD • Kethy M. Jules-Elysee, MD

Received: 21 February 2014/Accepted: 21 February 2014 /Published online: 15 May 2014
© Hospital for Special Surgery 2014

Original Release Date: April 30, 2014

Expiration Date: April 30, 2015

Overview

The objective is to increase knowledge and understanding of pulmonary arterial hypertension (PAH) as well as greater competency in managing and treating patients with PAH. This disease has serious potential complications including death in the perioperative period. Proper planning and clinical management are crucial to a good outcome.

Learning Objectives

Hospital for Special Surgery professional education activities are intended to improve knowledge, competence, and performance of our learners and to lead to better patient care. At the conclusion of the activity, the participant should be able to:

- Describe the types of complications that may occur in their patients with pulmonary arterial hypertension in a perioperative setting, such as dyspnea and right heart failure which can lead to significant morbidity and mortality.
- Properly manage and treat patients with pulmonary arterial hypertension, such as with aggressive use of diagnostic hemodynamic monitors.

Target Audience

This activity is targeted at primary care physicians, specialty physicians, orthopaedic surgeons, general medicine, residents, and fellows.

Accreditation

Hospital for Special Surgery is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

Credit Designation

Hospital for Special Surgery designates this journal-based CME activity for a maximum of *1.0 AMA PRA Category 1*

*Credit(s)*TM. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Commercial Support

This journal-based activity did not receive commercial support.

Faculty Disclosure:

In accordance with the Accreditation Council for Continuing Medical Education's Standards for Commercial Support, all CME providers are required to disclose to the activity audience the relevant financial relationships of the Activity Directors, planning committee members, presenters, authors and staff involved in the development of CME content. An individual has a relevant financial relationship if he or she has a financial relationship in any amount occurring in the last 12 months with a commercial interest whose products or services are discussed in the CME activity content over which the individual has control.

It is the policy of Hospital for Special Surgery to request all financial relationships that Activity Directors, planning committee members, presenters, authors and staff have with commercial interests, but to disclose to the activity audience only the **relevant** financial relationships.

Activity Directors Disclosure:

Kethy M. Jules-Elysee, MD has disclosed no relevant financial relationships.

Michael K. Urban, MD, PhD has disclosed no relevant financial relationships.

Planning Committee Disclosure:

Charles Cornell, MD has disclosed no relevant financial relationships.

Antonia Costello, LCSW has disclosed no relevant financial relationships.

Natanya Gayle, MPH has disclosed no relevant financial relationships.

Allison Goldberg, MPA has disclosed no relevant financial relationships.

OCME/CME Committee Disclosure:

Hospital for Special Surgery Office of CME Staff and CME Committee members have no relevant financial relationships to disclose regarding this activity.

Activity Faculty

Activity Directors:

Michael K. Urban, MD, PhD
Associate Attending Anesthesiologist
Hospital for Special Surgery
Clinical Associate Professor of Anesthesiology,
Weill Cornell Medical College
New York, NY

Kethy M. Jules-Elysee, MD
Assistant Attending Anesthesiologist
Hospital for Special Surgery
Clinical Assistant Professor of Anesthesiology,
Weill Cornell Medical College
New York, NY

Planning Committee

Charles N. Cornell, MD
Clinical Director of Orthopaedic Surgery
Attending Orthopaedic Surgeon
Hospital for Special Surgery
Professor of Clinical Orthopaedic Surgery
Weill Cornell Medical College
New York, NY

Antonia Costello, LCSW
Professional Education Program Coordinator
Education & Academic Affairs
Hospital for Special Surgery
New York, NY

Natanya Gayle, MPH
Managing Editor, HSS Journal
Education & Academic Affairs
Hospital for Special Surgery
New York, NY

Allison Goldberg, MPA
Director, Professional Education
Education & Academic Affairs
Hospital for Special Surgery
New York, NY

Technical Requirements

Windows

- Microsoft Windows XP, Windows Server 2003, Windows Vista, Windows Server 2008, Windows 7 operating system

- Microsoft Internet Explorer 6.0 SP1 or later, Firefox 2.0 or later, or Google Chrome 1.0 (Chrome is only supported on Mediasite version 5.0.3 and later) web browser
- Windows Media Player 9 or later
- For Firefox and Chrome playback, Microsoft Silverlight 1.0 or later (viewers are prompted to install this plug-in when attempting to view a presentation)
- Broadband internet connection (256 Kbps or more)

Mac—Requires Mediasite 4.3 and later:

- Mac OS X 10.4.8 or later operating system
- Safari 2.0.4 or later or Firefox 2.0 or later web browser
- Microsoft Silverlight 1.0 or later (viewers are prompted to install this plug-in when attempting to view a presentation).
- Broadband internet connection (256 Kbps or more)

For more information, please visit [Sonic Foundry](http://www.sonicfoundry.com/who-we-are/contact-us) <http://www.sonicfoundry.com/who-we-are/contact-us>.

Please view our [privacy policy](http://www.hss.edu/privacy-policy.asp) <http://www.hss.edu/privacy-policy.asp>.

Instructions for Post-test, Course Evaluation, and CME Credit:

In order to earn CME credit, you must complete an online or print posttest and evaluation following the completion of this activity. There is a passing requirement of 100%. Once you complete the post test and subsequent evaluation, a certificate will be available for you to print. Please note that you will be able to re-take the post test with no penalties until a 100% score is achieved.

For questions related to the post test and subsequent evaluation, please contact the HSS Office of Continuing Medical Education at professionaleducation@hss.edu.

Option 1: take the post-test online.

1. Go to the HSS Journal homepage at www.springer.com/hss.
2. Click on CME articles.
3. Click on “A Patient with Severe Pulmonary Hypertension for Hip Arthroplasty” to view the full-text pdf article.
4. After you have reviewed the article, click on ‘Complete the Current CME Test Online’ to register and complete the test.

Option 2: take the post-test via hard copy printed in the Journal.

You may complete the hard copy of the post test. Please mail the test to:

Journal CME Post-Test
Office of Continuing Medical Education
Education & Academic Affairs
535 East 70th Street
New York, NY 10021

A Patient with Severe Pulmonary Hypertension for Hip Arthroplasty

Michael K. Urban, MD, PhD · Kethy M. Jules-Elysee, MD

Received: 21 February 2014/Accepted: 21 February 2014 /Published online: 15 May 2014
© Hospital for Special Surgery 2014

Introduction

Pulmonary arterial hypertension (PAH) is defined as a mean PA pressure ≥ 25 mmHg at rest or 30 mmHg with exercise without left ventricular failure (left ventricular end diastolic pressure (LVEDP) ≤ 15) [7]. Most patients present with symptoms of progressive exertional dyspnea, which may also include angina and syncopal episodes. If these patients are not treated, the disease will progress to right heart failure, with peripheral edema, ascites, and hepatomegaly.

Pulmonary hypertension is a known risk factor for morbidity and mortality during noncardiac surgery [9]. Stress, pain, acidosis, and hypoxemia which are often encountered during surgery may increase PAH and precipitate right ventricular decompensation. In addition during total hip arthroplasty, the embolization of bone marrow debris and fat to the lungs will further increase pulmonary arterial pressures and acute cor pulmonale [6]. This is a case report of a young patient with severe pulmonary hypertension who died after a noncemented total hip arthroplasty.

Case Report

A 22-year-old female with avascular necrosis of her femoral head was scheduled for a left total hip arthroplasty. Her past medical history was significant for a mixed connective tissue disease complicated by vasculitis, thrombocytopenia,

pulmonary infiltrates, and pulmonary hypertension. An echocardiogram performed 3 months prior to surgery revealed a peak pulmonary artery systolic pressure of 105 mmHg with right ventricular (RV) dilatation, diminished RV function, a flattened intraventricular septum, severe tricuspid regurgitation, and moderate pulmonary insufficiency. The left ventricle demonstrated normal contractile function with no regional wall abnormalities. The patient was not short of breath at rest, but did complain of dyspnea with limited exertion. She was treated with sildenafil to reduce her PAH and improve her exercise tolerance. Additional preoperative medications included metoclopramide, hydromorphone, and iron supplements. The patient had recently completed a short course of prednisone for exacerbation of her connective tissue disease.

The patient's preoperative laboratory and diagnostic studies were significant for a mild anemia with a hematocrit (Hct) of 34 mg%, bicarbonate (HCO_3) of 18 meq/l, platelets 167 K, PT INR 1.06, and a slightly elevated aspartate aminotransferase (AST) of 56 U/l. The ECG revealed a normal sinus rhythm with a pattern suggestive of right ventricular hypertrophy and ST-segment depression in leads I, II, and V1-3. Cardiomegaly was prominent on the chest radiograph.

Vital signs immediately before surgery included a blood pressure (BP) of 104/60, heart rate (HR) 90, O_2 saturation of 95%, and respiratory rate of 15. The patient was monitored for surgery with the standard ASA monitoring including a radial arterial catheter. The anesthetic consisted of a combined lumbar spinal-epidural with spinal 3 cc 1.5% mepivacaine. Sedation included an initial dose of midazolam 5 mg, intermittent fentanyl 25 mcg, and an intravenous infusion of propofol. An epinephrine infusion was titrated to maintain the mean arterial blood pressure (MAP) in the 50's and HR in the 60's. With 3 l/min of oxygen via nasal cannula, the oxygen saturation remained about 95%. The surgery lasted 105 min. A noncemented press-fit femoral prosthesis and an uncemented hemispherical acetabular component were implanted. During the procedure, the patient received 600 ml of lactated ringers (LR) and lost an estimated 150 ml of blood.

The patient was brought to the PACU in stable condition; BP 150/90, HR 100, and O_2 saturation of 100%. Initial laboratory analysis included an arterial blood gas (ABG) pH

Electronic supplementary material The online version of this article (doi:10.1007/s11420-014-9383-y) contains supplementary material, which is available to authorized users.

M. K. Urban, MD, PhD (✉) · K. M. Jules-Elysee, MD
Department of Anesthesiology, Hospital for Special Surgery,
535 East 70th St,
New York, NY 10021, USA
e-mail: urbanm@hss.edu

M. K. Urban, MD, PhD · K. M. Jules-Elysee, MD
Weill Cornell Medical College,
1300 York Avenue,
New York, NY 10065, USA

7.30, paCO_2 33, paO_2 266 on NC O_2 5 l/min, BE-9.4, Hb 10.7 g/dl, K 4.2 mEq/l, BUN 12, creatinine (Cr) 0.8, and HCO_3^- 16. The patient remained hemodynamically stable over the first 12 post-op hours, but then became increasingly hypotensive and tachycardic with reduced O_2 saturation and urine output. The hypotension was initially treated with intravenous boluses of crystalloid and colloid, but her condition continued to deteriorate with increasing metabolic acidosis, hypotension, decreased urine output, and rising creatinine. On the morning of POD 1, her ABG revealed a worsening metabolic acidosis with compensated respiratory alkalosis: pH of 7.43, paCO_2 of 17, and paO_2 135 on a 40% FIO_2 face mask with a BE -19.

The patient was then transferred from the PACU to the medical intensive care unit (MICU) where she experienced an acute decompensation which required endotracheal intubation, controlled ventilation, and pharmacological blood pressure support. At that time, her ABG on 100% FIO_2 was pH 7.24, paCO_2 16, paO_2 195 with a Hb 9.1, platelet 89 K, WBC 14 K, BUN 17, Cr 1.7, and BS 403.

A pulmonary artery catheter (PAC) was inserted through the right internal jugular; initial PA pressures were 75/45, pulmonary capillary wedge pressure (PCWP) of 20, cardiac output (CO) 2.4, mixed venous oxygen saturation (SVO_2) 30%, and a calculated pulmonary vascular resistance (PVR) of $1,233 \text{ dyn s cm}^{-5}$. Several different therapies were then instituted in an attempt to restore adequate tissue perfusion and oxygenation: inhaled nitric oxide at 35 ppm and intravenous prostacyclin reduced the PA pressures to 55/40 and PVR to 554. A sodium bicarbonate infusion was started to correct the metabolic acidosis and a dobutamine infusion increased the cardiac output to 3.0 l/min and SVO_2 51%. Despite these interventions, however, the metabolic acidosis persisted (lactate 28.7 mmol/l) indicative of poor tissue perfusion. In response, the patient was placed on continuous venovenous hemodialysis (CVVHD). On POD 3, the patient again became acutely hypotensive with persistent oliguria and acidosis, initiating the institution of a vasopressin infusion. Despite the added therapy, her hemodynamic condition continued to deteriorate and a pulse dose of methoprednisolone was administered assuming the development of a systemic inflammatory response syndrome. On POD 5, the patient expired after a ventricular fibrillation arrest.

Discussion

Primary pulmonary hypertension is a rare disease (1–3 per million). Most cases of PAH are associated with disease states which have been classified into five categories by the World Health Organization [10] (Table 1). Although there are many etiologies, they all result in similar pathological findings of smooth muscle and endothelial cell proliferation in response to chronic vasoconstriction and elevated pressures. Hypoxia induces vasoconstriction in the pulmonary vasculature in part through the effects of endothelial-derived endothelin, thromboxane, and serotonin. The stressed endothelium produces reduced levels of nitric oxide synthase, resulting in a reduction in the

Table 1 World Health Organization classification of pulmonary hypertension

Pulmonary arterial hypertension: idiopathic, collagen vascular disease, Eisenmenger
Pulmonary venous hypertension: elevated LVEDP
PAH associated with hypoxemia: COPD, OSA, interstitial lung dz
PAH associated with chronic thrombotic disease
Miscellaneous: schistosomiasis, sarcoidosis

synthesis of the vasodilator nitric oxide. These changes result in an imbalance in the regulation of pulmonary vasodilators and vasoconstrictors. These changes also promote platelet aggregation leading to an increase in pulmonary thrombosis. Together, these changes ultimately lead to chronic luminal narrowing and irreversible PAH.

The severity of the disease is assessed using the patient's functional clinical classification. Patients with no impairment are class I; dyspnea with ordinary activity are class II; dyspnea causing severe restriction of activity is class III, and class IV implies dyspnea at rest. The degree of functional impairment is then used to assess the success of treatment and the suitability (and risks) of the patient for noncardiac surgery [8, 14].

Doppler echocardiography is essential in the assessment of PAH. There is reasonably good correlation between Doppler and pulmonary artery catheter-derived pulmonary pressures [14]. In addition, the echocardiogram provides information with regard to right and left ventricular functions and valvular abnormalities which will aid in diagnosis and management. The electrocardiogram may also demonstrate evidence of pulmonary hypertension including right ventricular hypertrophy, right axis deviation, and/or right atrial enlargement. Chest radiographic evidence of PAH may include cardiomegaly, enlarged hilar pulmonary shadows, and attenuation of peripheral vascular markings (pruning). Pulmonary function studies (PFTs) are appropriate in the initial evaluation of patients with PAH to characterize underlying respiratory causes and to assess the severity of parenchymal disease. Many of the patients will exhibit a restrictive defect and most will have reduced diffusing capacity for carbon monoxide. However, a poor correlation between lung function measures and the severity of PAH has been reported [11]. Ultimately, right heart cardiac catheterization is required to confirm the diagnosis of PAH, assess the severity of the disease, and guide therapy. Using PA systolic pressures, the severity of PAH is grouped into mild (PAS 25–40 mmHg), moderate (PAS 41–55 mmHg), and severe (>55 mmHg).

There is no cure for PAH, but treatment modalities exist which reduce symptoms and prolong survival. Therapeutic agents include vasodilators, anticoagulants, antiplatelet agents, anti-inflammatory, and vascular remodeling drugs [3, 7, 14]. The American College of Chest Physicians in 2007 issued practice guidelines for the medical therapy of patients with PAH [3]. Before instituting vasodilator therapy, patients with PAH should undergo vasoreactivity testing. In the absence of right heart failure, a favorable acute response to a vasodilator is defined as a decrease in mean

PA pressures of at least 10 mmHg and overall PA systolic pressure of ≤ 40 mmHg with increased or unchanged cardiac output. In addition, patients with PAH should undergo serial determinations of their New York Heart Association (NYHA) classification and their exercise capacity assessed by the 6-min walk (6 MW) test in order to evaluate the efficacy of their current therapeutic regimen.

Calcium channel blockers (CCB) are considered the first line of therapy for patients with PAH; however, only a small percentage of patients will respond favorably. CCBs with negative inotropic effects, such as verapamil, should be avoided. Prostanoids, such as epoprostenol (prostacyclin), a potent vasodilator with platelet inhibitor and anti-inflammatory properties, have been shown to improve exercise capacity and survival in patients with PAH. Epoprostenol must be administered as a continuous i.v. infusion. Treprostinil a prostanoid with a longer half life (3 h.) must also be administered as a continuous i.v. or subcutaneous infusion (s.c.). A newer stable prostacyclin analogue, iloprost, can be administered by inhalation. In a recent trial, iloprost at six inhalations per day improved exercise tolerance, pulmonary hemodynamics, and two-year survival in NYHA II and III PAH patients by 29% [12].

Sildenafil, a potent and specific phosphodiesterase-5 inhibitor, has become first-line therapy for PAH because of its demonstrated efficacy in NYHA II and early III patients and ease of administration. Those patients who have ocular disease, recurrent bleeding problems, or are unresponsive to sildenafil are now treated with endothelin receptor antagonists. Endothelin 1 is a potent vasoconstrictor and stimulates smooth muscle cell proliferation. Bosentan, an endothelin receptor antagonist, has been shown to improve performance by PAH patients in the 6 MW test. Bosentan will produce an increase in hepatic aminotransferases and potentially long-term liver damage. Two additional endothelin receptor antagonists, ambrisentan and sitaxsentan, are in clinical trials. Many NYHA III patients are now on combination therapy, and most patients with NYHA IV disease require iv epoprostenol in addition to another therapy. Since microscopic pulmonary thrombosis is an integral part of this disease process, anti-coagulation is also recommended for PAH patients [10].

In patients with advanced PAH and a failing right ventricle, atrial septostomy may be performed to create a right to left shunt, which will improve cardiac hemodynamics but worsen the hypoxemia. The final solution requires bilateral lung transplantation, which still only affords a ~70% survival.

There is a significant risk of perioperative morbidity and mortality in patients with PAH regardless of the anesthetic technique [8]. Predictors of perioperative mortality and morbidity are presented in Table 2 [4, 8]. In an analysis of 145 patients with PAH undergoing elective noncardiac surgery, the 30-day morbidity was 42% and mortality 9.7% [13]. Preoperative evaluation of these patients must include an assessment of right ventricular function and PVR with a recent echocardiogram, and if worse, a right heart catheterization. Evidence of worsening right ventricular function, progressive dyspnea, metabolic acidosis (low cardiac output), hypoxemia, peripheral edema, hepatomegaly, and syncope

Table 2 Predictors perioperative morbidity and mortality

History of pulmonary emboli

NYHA class $\geq$ II
 Cannot travel 332 m during a 6 MW test
 Intermediate or high risk surgery
 Duration of surgery >3 h
 Right ventricular hypertrophy; right axis deviation on ECG
 Pericardial effusion
 Right ventricular systolic pressure/systemic systolic pressure ≥ 0.66
 Intraoperative vasopressor use

MacKnight B, Martinez EA, Simon BA. *Sem Cardiothoracic and Vasc Anesthesia* 12:91-96, 2008, copyright© 2008 by Sage Publications. Adapted with permission of Sage Publications

should warrant postponing the procedure until RV function is further evaluated and the patient's condition maximized and/or the risk/benefit of the surgical procedure is reevaluated [4, 6]. Right ventricular failure was a contributing cause of postoperative mortality in 50% of the patients [13].

Intraoperative monitoring should in most cases include an arterial catheter and central venous pressure monitoring in order to provide adequate preload. In patients with RV dysfunction, a PAC or transesophageal (TEE) monitor will guide the management of cardiac output. However, a PAC is associated with an increased risk of PA rupture and poorly tolerated dysrhythmias in PAH patients. A specific anesthetic technique is probably less important than the maintenance of hemodynamic stability. The reduction in systemic vascular resistance which occurs with neuraxial anesthesia may be poorly tolerated in patients with severe PAH and RV dysfunction. If general anesthesia is chosen, it should include a balanced technique with lower levels of potent inhalational agents and avoidance of nitrous oxide. The intraoperative use of milrinone may be beneficial since it reduces both PVR and SVR and increases myocardial contractility. However, in patients with RV dysfunction, decreases in systemic pressures may result in RV ischemia and cardiac failure. In all cases, a strategy for the continuation of PAH treatment should be formulated preoperatively. This may include the use of intravenous prostacyclin and/or inhaled iloprost or nitric oxide. Postoperatively, these patients should be monitored in an ICU to insure the adequacy of oxygenation, perfusion, pain management, and the continuation or initiation of pulmonary vasodilator therapy. The highest incidence of mortality occurs several days after surgery from worsening PVR and RV failure.

In summary, a patient with severe PAH scheduled for elective arthroplasty will have an increased risk for major perioperative morbidity and mortality. A team of care givers should be assembled prior to the surgery to discuss the risks and develop a perioperative management plan for the patient. This committee should include the surgeon, anesthesiologist, a pulmonologist with experience treating patients with severe PAH, the ICU attending and an ICU with the capability of treating severe PAH, and an ethicist. Full discussion of the surgical procedure including risks and benefits should be thoroughly explored with the patient and their family prior to surgery. The hip pain from avascular necrosis in this patient had become intolerable and significantly limited her mobility.

The risk of respiratory depression and hypoxemia exacerbating her PAH prevented further increases in narcotic analgesics. Hence, despite the risks, a THA probably represented the best option for our patient. Optimization of the patient's cardiopulmonary status should be attempted prior to surgery, with the institution of additional therapeutic agents when appropriate. A preoperative echocardiogram should be repeated in combination with an ABG and a 6 MW test to assess the degree of progression of the disease. This information would then be used to assess the efficacy of adding further therapeutic agents including perioperative intravenous prostacyclin or inhaled iloprost. Regional anesthesia is acceptable choice for this procedure, as long as the patient's oxygen saturation remained above 90%. Intraoperative monitoring with a PAC permits assessment of the cardiac output and PVR and provides the ability to institute therapeutic measures to reverse deleterious trends. Although the hip prosthesis was not cemented, the contents of the femoral canal still embolize to the lung with the potential for acute increases pulmonary pressures and systemic hypotension [1]. Pulmonary vasodilators, such as prostacyclin, must be readily available to treat these events. The preferred agent for the treatment of acute hypotension may be vasopressin since it will reduce pulmonary pressures while supporting systemic pressure [5]. Despite the best care patients with severe PAH are at risk for major perioperative complications including death. Therefore, it is imperative to weigh the risks and benefits of the procedure with the patient, their family, and the medical care team.

Disclosures

Conflict of Interest: Michael K. Urban, MD, PhD and Kethy M. Jules-Elysee, MD have declared that they have no conflict of interest.

Human/Animal Rights: All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008 [5].

Informed Consent: Informed consent was waived from all patients for being included in the study.

References

1. Apostolou CD, Skourtas CE, Tsifetakis SD, Papagelopoulos PJ. Fat embolism after uncemented total Hip arthroplasty. *Clin Orthop Relat Res.* 2002; 398: 153-156.
2. Badesch DB, Abman SH, Ahearn GS, Barst RJ, McCoy DC, Simonneau G. Medical therapy for pulmonary arterial hypertension: ACCP evidence-based clinical practice guidelines. *Chest.* 2004; 126: 35S-65S.
3. Badesch DB, Abman SH, Simonneau G, Rubin LJ, McLaughlin VV. Medical therapy for pulmonary arterial hypertension updated ACCP evidence-based clinical practice guidelines. *Chest.* 2007; 131: 1917-1928.
4. Bapoje SR, Whitaker JF, Schulz T, Chu ES, Albert RK. Preoperative evaluation of the patient with pulmonary disease. *Chest* 2007;132.
5. Braun EB, Palin CA, Hogue CW. Vasopressin during spinal anesthesia in a patient with primary pulmonary hypertension treated with intravenous epoprostenol. *Anesth Analg.* 2004; 99: 36-37.
6. Ereth MH, Weber JG, Abel HD, et al. Cemented versus Non-cemented total Hip arthroplasty-embolism, hemodynamics, and intrapulmonary shunting. *Mayo Clin Proc.* 1992; 67(11): 1066-1074.
7. Farber HW, Loscalzo J. Pulmonary arterial hypertension. *N Eng J Med.* 2004; 351: 1655-1665.
8. Fox C, Kalarickal PL, Yarborough MJ, Jin JY. Perioperative management including new pharmacological vistas for patients with pulmonary hypertension for noncardiac surgery. *Curr Opin Anesth.* 2008; 21: 467-472.
9. Lai HC, Lai HC, Wong KY, Lee WL, Ting CT, Liu TJ. Severe pulmonary hypertension complicates postoperative outcome of cardiac surgery. *Br J Anaesth.* 2007; 126: 1-7.
10. MacKnight B, Martinez EA, Simon BA. Anesthetic management of patients with pulmonary hypertension. *Sem Cardiothoracic Vasc Anesthesia.* 2008; 12: 91-96.
11. Nathan SD, Oksana AS, Shahzad A, Urbanek S, Barnett SD. Pulmonary hypertension and pulmonary function testing in idiopathic pulmonary fibrosis. *Chest* 2007;131.
12. Opitz CF, Wensel R, Winkler J. Clinical efficacy and survival with first-line inhaled iloprost therapy in patients with idiopathic pulmonary arterial hypertension. *Eur Heart J.* 2005; 174: 1257-1263.
13. Ramakrishna G, Sprung J, Ravi BS. Impact of pulmonary hypertension on the outcomes of noncardiac surgery: predictors of perioperative morbidity and mortality. *J Am Coll Cardiol.* 2005; 45: 1691-1699.
14. Rubin LJ, Badesch DB. Evaluation and management of the patient with pulmonary arterial hypertension. *Am Intern Med.* 2005; 143: 282-292.

A Patient with Severe Pulmonary Hypertension for Hip Arthroplasty

Michael K. Urban, MD, PhD • Kethy M. Jules-Elysee, MD

Received: 21 February 2014/Accepted: 21 February 2014 /Published online: 15 May 2014
© Hospital for Special Surgery 2014

CME Questions

- 1) Definition of pulmonary arterial hypertension (PAH)
 - a) PAP > 25 mmHg at rest
 - b) PAP > 30 mmHg at rest
 - c) PCWP < 15 mmHg
- 2) This group of patients with PAH and connective tissue disorders have the worse prognosis
 - a) A. Systemic lupus erythematosus
 - b) Rheumatoid arthritis
 - c) Systemic sclerosis
- 3) Most common cause of death in patients with mixed connective tissue disorder
 - a) PAH
 - b) Renal disease
 - c) Cerebral involvement
- 4) Cellular mechanism involved in development of PAH
 - a) Increased prostacyclin synthase
 - b) Increased nitric oxide synthase
 - c) Increased endothelin
- 5) Calcium channel blockers should be given empirically
 - a) True
 - b) False
- 6) Double lung transplant preferred over single lung transplant
 - a) True
 - b) False
- 7) Class of PAH patients requiring IV prostacyclin
 - a) WHO Class I and II
 - b) WHO Class III
 - c) WHO Class IV
- 8) The following are all possible causes of primary PAH except
 - a) BMPR2 mutation and vascular smooth muscle cell proliferation
 - b) Inhibition or down-regulation of the Kv channel in pulmonary artery smooth muscle cell
 - c) Monoclonal proliferation of endothelial cells in plexiform lesions
 - d) Decreased productions of constrictors
- 9) Effect of Viagra (sildenafil) in patients with PAH
 - a) Increase exercise time on treadmill
 - b) Decreased PAP
 - c) Increase cardiac output
 - d) Worsens quality of life
- 10) Variables considered independent predictors of short term morbidity after non-cardiac surgery in patients with PAH except
 - a) History of pulmonary embolism
 - b) Low risk surgery
 - c) Duration of anesthesia > 3 hours
 - d) New York Heart functional class

CME Registration Form

The Accreditation Council for Continuing Medical Education requires that accredited CME providers evaluate and assess the effectiveness of all Continuing Medical Education accredited activities. This evaluation will help us document our efforts to address physicians' practice gaps and educational needs.

Should you have any questions, please contact the Office of Continuing Medical Education at professionaleducation@hss.edu.
Thank you.

Expires April 30, 2015

Please complete the following contact information:

First Name:

Last Name:

Title (i.e. MD, PhD, NP)

Organization

Street Address

City, State, Zipcode

Country

Email Address

Please indicate your profession:

- Family Medicine
- Fellows
- General Medicine
- Medical Students
- Orthopaedic Surgeons
- Residents
- Rheumatologist
- Other

Please give us your feedback by completing this evaluation. **All fields are required.**

Enhanced practitioner

(Scale: 1= Not At All Certain; 7= Very Certain)

1 2 3 4 5 6 7

How certain are you that your practice will be enhanced by participating in this course?

□ □ □ □ □ □ □

If you do not feel that your skills have been enhanced, please comment.

How certain are you to implement the following strategies in your practice:

(Scale: 1= Not At All Certain; 7= Very Certain)

How certain are you to implement the following strategies in your practice:

Describe the types of complications that may occur in their patients with pulmonary arterial hypertension in a perioperative setting, such as dyspnea and right heart failure which can lead to significant morbidity and mortality.

[illegible]

Properly manage and treat patients with pulmonary arterial hypertension, such as with aggressive use of diagnostic hemodynamic monitors.

[illegible]

Please respond to the following statement:

As a result of what I have learned, I will make the following change(s) in my practice:

1. _____
2. _____
3. _____

Overall, the content covered in this journal article has been useful and relevant to my scope of practice.

- ☐ Strongly Agree ☐ Agree ☐ Strongly Disagree ☐ Disagree

If you disagree, please explain why

Do you feel this activity was free of commercial bias?

- ☐ Yes ☐ No

If no, please comment

Please respond to the following statement: Using this technology was easy.

- ☐ Strongly Agree ☐ Agree ☐ Strongly Disagree ☐ Disagree

If you disagree, please explain why

How likely are you to read more journal articles offered in this format?

- ☐ Very likely ☐ Neutral ☐ Very unlikely

If unlikely, please explain why

Was the learning format for this activity appropriate for the content being presented?

- ☐ Yes ☐ No

If no, please suggest other format that would be more effective

Please provide at least one topic to be covered in future journal articles that would help you in your practice and/or improve the health of your patients:

Please provide at least one suggestion for improving this journal article so that it will be more effective: