QUESTIONs

Before continuing, consider the following scenario and question. The answers can be found at the end of the article, together with an explanation.

You are to anaesthetise a 65-year-old woman for laparoscopic sigmoid colectomy. She has recently been diagnosed with colorectal carcinoma. The patient has been a heavy smoker in the past and has severe chronic obstructive pulmonary disease (COPD), with secondary pulmonary hypertension. She also has essential hypertension. Medications include a beta-blocker, ACE inhibitor, inhaled steroid/beta-agonist and aspirin.

What are your concerns in anaesthetising this patient?

INTRODUCTION

The disease spectrum of Pulmonary Hypertension (PH) has received greater interest in the past decade, as specific therapies have been developed and survival has improved. More patients with PH are now presenting for surgery, and this poses a challenge to the anaesthetist. Knowledge of the underlying physiology is paramount in preventing the feared complication of right heart failure.

DEFINITION AND CLASSIFICATION

Pulmonary Hypertension is defined as a mean pulmonary artery pressure (PAP) >25mmHg at rest with a pulmonary capillary wedge pressure <12mmHg. Pulmonary hypertension is considered moderately severe when mean PAP >35mmHg. Right ventricular failure is unusual unless mean PAP is >50mmHg.

The World Health Organisation classifies pulmonary hypertension by aetiology into five groups. The disease, including its classification, was comprehensively reviewed at the 4th World Symposium on Pulmonary Hypertension in 2008.

Table 1: Clinical classification of pulmonary hypertension

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<tbody>
<tr>
<td>1</td>
<td>Pulmonary Hypertension (PAH)</td>
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<td>2</td>
<td>Pulmonary hypertension owing to left heart disease</td>
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<tr>
<td>3</td>
<td>Pulmonary Hypertension owing to lung disease</td>
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<tr>
<td>4</td>
<td>Chronic thromboembolic pulmonary hypertension (CTEPH)</td>
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<td>5</td>
<td>Pulmonary hypertension with unclear multifactorial mechanisms</td>
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Group 1 includes the disease idiopathic pulmonary hypertension (formerly known as primary pulmonary hypertension), as well as PH associated with connective tissue disorders. This group of diseases share similar pathological findings and clinical appearance. The incidence of idiopathic PH is higher than previously thought, although remains relatively rare at 15 per million.

Of greater interest to the anaesthetist are the more common forms of PH: those due to left heart disease (group 2) and those due to lung disease (group 3). Cardiac anaesthetists have long been familiar with PH due to left heart disease, which often occurs in patients undergoing cardiac surgery. Examples would include patients with mitral valve disease undergoing valve replacement, or patients with severe LV failure undergoing coronary bypass surgery.

Non-cardiac anaesthetists are more likely to encounter PH in patients with lung disease. Underlying diseases include COAD, interstitial lung disease, and sleep disordered breathing. The majority of patients in this group have modest PH.

**PITFALLS IN DIAGNOSIS**

Pulmonary hypertension may be suspected after patient assessment based on history, examination, ECG and chest x-ray. The symptoms of PH are non-specific, and diagnosis can be delayed.

If PH is suspected, transoesophageal echocardiography (TTE) is usually the first investigation undertaken. TTE utilizes Doppler across a tricuspid regurgitant jet, to estimate pulmonary artery pressure. This technique has been shown to under or over estimate PAP in up to half of patients at risk of PH, and therefore as a diagnostic test has limitations in accuracy.

Right heart catheterization is required to confirm the diagnosis. A vasodilator challenge forms part of this assessment.

**UNDERSTANDING THE PHYSIOLOGY**

Providing anaesthesia to patients with PH poses some challenges. An underlying knowledge of the cardiovascular pathophysiology is paramount to providing safe anaesthesia in these patients.

Right ventricular output is dependent on preload, afterload, contractility and heart rate. Consideration must be given to optimizing each of these parameters.

Raised pulmonary vascular resistance (PVR) places an additional pressure load on the right ventricle. The right heart is poorly designed to deal with these increases in afterload. A rise in PVR and hence right ventricular afterload can put the right heart into failure. Left ventricular failure can then ensue, due to both reduced volume reaching the left heart, and septal interdependence.

Factors which can raise PVR include hypoxia, hypercarbia, hypothermia, acidaemia, and pain. Anaesthetic technique is aimed at preventing these occurrences.

The coronary circulation to the right heart is dependent on perfusion pressure at the aortic root, which in turn is dependent on cardiac output and systemic vascular resistance (SVR).
SVR must be aggressively defended in order to maintain coronary perfusion to the right heart. Ischaemia to the right ventricle can put in place a downward spiral of right heart failure, with ensuing cardiovascular collapse.

**ANAESTHETIC TECHNIQUES**

Many anaesthetic techniques have been employed in anaesthetising patients with pulmonary hypertension. The actual technique chosen is probably less important than the manner in which it is executed.

Extended monitoring will be useful. Invasive blood pressure monitoring is ideal as it will aid early and aggressive treatment of systemic hypotension. A pulmonary artery catheter can be useful in monitoring trends in PAP. A rising PAP may indicate a rising PVR or a failing right ventricle (RV). Transoesophageal echocardiography or other cardiac output monitors can also be useful.

An effort to obtund the response to laryngoscopy should be made.

A variety of anaesthetic induction drugs have been safely employed in patients with PH. One technique would be to use a combination of midazolam and fentanyl. Etomidate has been described as an ideal agent in PH. Propofol and thiopentone have also been used without problems. Concern has been raised that ketamine may raise PVR, however it too has been utilized safely in humans with PH.

Non-depolarising and depolarising muscle relaxants can be used safely, and should be chosen based on airway management issues.

A balanced anaesthetic of volatile agent and opioids can be used as maintenance. All of the commonly used modern volatile agents have been safely used in PH, and there is no evidence to recommend one over the other. Nitrous oxide should be used with caution as it may raise PVR.

A systemic vasoconstrictor such as phenylephrine, noradrenaline or metaraminol should be on hand to treat reductions in systemic blood pressure. A carefully titrated infusion may be commenced at induction.

Inotropes can be employed to improve right heart contractility but they are often more beneficial to the left heart than the right heart. The inodilators such as milrinone and
dobutamine will cause systemic vasodilation and hence reduce coronary perfusion pressure, which limits their utility.

RV failure and raised PVR can be targeted with inhaled selective pulmonary vasodilators. Agents such as nitric oxide and prostacyclin are increasingly being employed perioperatively in these patients.

Neuraxial anaesthesia and analgesia, can be used safely in PH, however the anaesthetist must be vigilant about the cardiovascular consequences of sympathetic blockade. There are no alpha-1 adrenoreceptors in the pulmonary circulation, hence it is unlikely that neuraxial blockade has a direct effect on PVR. Systemic vasodilation however, will reduce aortic coronary perfusion pressure, as well as venous return to the right heart. A decrease in right atrial filling can reduce stretch on atrial receptors, resulting in reflex bradycardia. Loss of the cardio-accelerator fibres at T1-T4 may result in bradycardia and loss of inotropy. Bradycardia and hypotension can cause right heart failure and can be lethal in patients with PH.

**SUMMARY OF ANAESTHETIC TECHNIQUES IN PH**

Avoid any stimulus which can increase PVR including: nitrous oxide, adrenaline, dopamine, protamine, serotonin, thromboxane A₂, prostaglandins such as PGF₂α and PGE₂, hypoxia, hypercarbia, acidosis, PEEP and lung hyperinflation, cold, anxiety and stress.

If PVR is increased it can be reduced by: hypocarbia (via hyperventilation), nitric oxide, morphine, glyceryl trinitrate, sodium nitroprusside, tolazoline, prostacycline (PGI₂), isoprenaline, and aminophylline.

Aims are to avoid marked decreases in venous return (correct blood and fluid loss quickly), avoid marked decreases in SVR, avoid drugs which cause myocardial depression, and to maintain a normal heart rate.
SUMMARY

- A feared complication of pulmonary hypertension is right heart failure
- Understanding the physiology of right ventricular function and coronary perfusion are important when anaesthetising patients with pulmonary hypertension
- Anaesthetic aims in PH are to avoid increased PVR, to avoid marked decreases in venous return or SVR, to avoid myocardial depression and to maintain normal heart rate.

ANSWERS TO QUESTIONS

Your concerns in anaesthetising this patient may include:
1. The presence of severe systemic disease in a patient undergoing intermediate risk surgery. Optimisation of the patient’s chronic obstructive pulmonary disease and pulmonary hypertension is warranted pre-operatively.
2. The cardiovascular and respiratory sequelae of pneumoperitoneum, trendelenburg position, and potentially prolonged surgery.
3. Anticipation and prevention of perioperative right heart failure in the patient with pulmonary hypertension.
4. Provision of excellent perioperative pain relief, especially in the patients with lung disease, to reduce the risk of respiratory complications.

REFERENCES and FURTHER READING


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