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# Perioperative management of pulmonary vascular resistance

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## Introduction

Raised pulmonary vascular resistance, its sequelae of pulmonary hypertension and right-sided heart failure, presents the highest risk of morbidity and mortality during anaesthesia. Pulmonary vascular resistance (PVR) is increased by increases in the sympathetic output driven by stress-related perioperative circumstances such as hypothermia, pain, surgery-related inflammatory response and ventilation techniques. Additionally, several intraoperative factors such as fluid shifts, medications, systemic hypotension, arrhythmias,[1] hypoxaemia, hypercapnia, acidosis, hypervolaemia, and insufficient anaesthesia may exacerbate pulmonary hypertension.[2] These may precipitate acute right ventricular ischaemia and failure. Although it is important to understand the underlying pathology, this manuscript will address these very briefly, as the aim is to discuss the perioperative management of PVR. Table I depicts the 2022 ESC/ERS classification of pulmonary hypertension.[3]

## Definition

The 2022 ESC/ERS definition of pulmonary hypertension based on haemodynamic assessment by right heart catheterisation is a mean pulmonary artery pressure (mPAP) > 20 mmHg at rest (Table II). In defining pulmonary hypertension, it is recommended that PVR and pulmonary arterial wedge pressure (PAWP) are included in the definition of precapillary PH, in order to differentiate between elevated PAP due to pulmonary vascular disease (PVD) from that due to left heart disease (LHD), elevated pulmonary blood flow, or increased intrathoracic pressure.[3] Precapillary pulmonary hypertension may be from various causes, whereas postcapillary disease is caused by left-heart disease.

## Functional capacity and preoperative assessment

Preoperative assessment will elicit the history of functional capacity, medications such as anticoagulants, antihypertensives, drugs used to reduce pulmonary hypertension (e.g. sildenafil, bosentan) and antiarrhythmic agents. A physical examination will inform the anaesthetist on how well compensated the physiology is. A cardiac catheterisation report gives information on tests of reversibility, among other information. Poor exercise tolerance is indicated by fatigue and dyspnoea should be elicited. The information on devices such as pacemakers and automated defibrillators is important. Laboratory investigations should be tailored accordingly. The World Health Organisation (WHO) has defined a functional classification system, the WHO-FC. It is deemed a very strong predictor of survival, both at diagnosis and follow-up.[3] Table III depicts the WHO-FC classification.

**Table I:** Classification of pulmonary hypertension[3]

| | |
|---|---|
| **Group 1: Pulmonary arterial hypertension (PAH)** | |
| 1.1 | Idiopathic |
| 1.1.1 | Non-responders at vasoreactivity testing |
| 1.1.2 | Acute responder at vasoreactivity testing |
| 1.2 | Heritable |
| 1.3 | Associated with drugs and toxins |
| 1.4 | Associate with: |
| 1.4.1 | Connective tissue disease |
| 1.4.2 | HIV infection |
| 1.4.3 | Portal hypertension |
| 1.4.4 | Congenital heart disease |
| 1.4.5 | Schistosomiasis |
| 1.5 | PAH with features of venous/capillary (PVOD/PCD) involvement |
| 1.6 | Persistent PH of the newborn |
| **Group 2: PH associated with left heart disease** | |
| 2.1 | Heart failure |
| 2.1.1 | with preserved ejection fraction |
| 2.1.2 | with reduced or mildly reduced ejection fraction |
| 2.2 | Valvular heart disease |
| 2.3 | Congenital/acquired cardiovascular conditions leading to postcapillary PH |
| **Group 3: PH associated with lung diseases and/or hypoxia** | |
| 3.1 | Obstructive lung disease or emphysema |
| 3.2 | Restrictive lung disease |
| 3.3 | Lung disease with mixed restrictive/obstructive pattern |
| 3.4 | Hypoventilation syndromes |
| 3.5 | Hypoxia without lung disease (e.g. high altitude) |
| 3.6 | Developmental lung disorders |
| **Group 4: PH associated with pulmonary artery obstructions** | |
| 4.1 | Chronic thrombo-embolic PH |
| 4.2 | Other pulmonary artery obstruction |
| **Group 5: PH with unclear and/or multifactorial mechanisms** | |
| 5.1 | Haematological disorders |
| 5.2 | Systemic disorders |
| 5.3 | Metabolic disorders |
| 5.4 | Chronic renal failure with or without haemodialysis |
| 5.5 | Pulmonary tumour thrombotic microangiopathy |
| 5.6 | Fibrosing mediastinitis |

### *Intraoperative monitoring*

The key to monitoring is early recognition of factors that may precipitate episodes of increased pulmonary pressure. Episodes

of systemic hypotension which risk right ventricular (RV) ischaemia, acute elevations in pulmonary vascular resistance, and fluid overload with risks of RV failure should be identified early.[1] The extent of monitoring should be decided in relation to the severity of the disease and the extent and length of the surgical procedure, and may range from standard monitoring to invasive monitoring including echocardiography. Cardiac haemodynamic monitors that reflect stroke volume (SV) and SV variation may also be helpful in identifying changes timeously. SV has been found to be the best haemodynamic parameter reflecting right ventricular function in response to its load.[4]

**Table II:** Haemodynamic parameters in the definition of pulmonary hypertension[5]

| Definition | Haemodynamic characteristics |
|---|---|
| PH | mPAP > 20 mmHg |
| Precapillary PH | mPAP > 20 mmHg<br>PAWP ≤ 15 mmHg<br>PVR > 2 WU |
| CpcPH | mPAP > 20 mmHg<br>PAWP > 15 mmHg<br>PVR > 2 WU |
| IpcPH | mPAP > 20 mmHg<br>PAWP > 15 mmHg<br>PVR > 2 WU |
| Exercise PH | mPAP/CO slope between rest and exercise > 3 mmHg/L/min |

CO – cardiac output, CpcPH – combined post- and precapillary pulmonary hypertension, IpcPH – isolated postcapillary pulmonary hypertension, mPAP – mean pulmonary arterial pressure, PAWP – pulmonary arterial wedge pressure, PH – pulmonary hypertension, PVR – pulmonary vascular resistance, WU – wood units

## *Anaesthetic management*

Anaesthetic technique depends on functional capacity, preoperative medication history (especially anticoagulants), type of surgery, and the pathophysiology of pulmonary hypertension.

### *Regional anaesthetic techniques*

In surgical procedures where mechanical ventilation and muscle paralysis are not required, regional techniques may offer a greater benefit of profound analgesia and absence of impairment of spontaneous breathing and avoidance of elevated pulmonary pressures.[6] A titrated epidural technique is superior as it avoids significant decrease in systemic vascular resistance, reduction of coronary perfusion, and right-heart failure. It also affords the patient prolonged analgesia in the perioperative period. A single dose bolus spinal anaesthetic may, however, lead to uncontrolled drops in blood pressure, leading to a compromise in myocardial perfusion. For procedures where nerve blocks can be performed, such as sciatic or femoral nerve plexus blocks, catheters are recommended. Management of anticoagulation should be adhered to according to the guidelines.[7] Combinations of general and regional anaesthesia techniques are also advantageous.

### *General anaesthesia*

Most intravenous anaesthetic agents, when used carefully in combination with opioids, are safe. There is conflicting evidence on the safe use of ketamine. Generous preoxygenation should be employed. Intubation, and similarly extubation, necessitate special attention to attenuate responses. Histamine-releasing relaxants (atracurium, mivacurium) are best avoided in these patients. Nearly all inhalational anaesthetics can be administered without adverse event.[8] They may have an added pulmonary vasodilatory effect through their block of the ATP-dependent potassium channels that induces vascular relaxation. Nitrous oxide is not recommended for patients with pulmonary hypertension.

### *Management of intraoperative pulmonary artery pressure elevation*

The benefits of general anaesthesia (GA) are in managing inducers of the sympathetic response such as the intubation response. A generous premedication with an anxiolytic attenuates the stress response to surgery. It also affords the anaesthetist opportunity to manipulate inducers of pulmonary vascular hypertension through ventilatory manoeuvres. Hypoxia, hypercarbia, and acidosis can be managed by targeting higher $FiO_2$, moderately lower $PCO_2$ and avoiding alveolar overinflation and atelectasis by performing recruitment manoeuvres intraoperatively (Table III). The pH should, however, not be allowed to fall below 7.4.

Depth of anaesthesia, temperature management and analgesia should be managed to avoid exacerbations of raised pressures. Fluid management should be targeted to response using stroke volume variation monitoring where feasible to maintain changes below ± 15–20%. Maintenance of coronary perfusion pressure with the use of vasoactive agents such as norepinephrine and vasopressin during periods of low systemic blood pressure has been recommended.[9]

**Table III:** Intraoperative manoeuvres to attenuate increases in pulmonary arterial pressure[2]

1. "Luxury" oxygenation with inspiratory $FiO_2$ 0.6–1.0
2. Moderate hyperventilation (goal: $PaCO_2$ 30–35 mmHg
3. Avoidance of metabolic acidosis (pH > 7.4)
4. Recruitment manoeuvre to avoid ventilation/perfusion mismatch
5. Low-tidal-volume ventilation to avoid overinflation of alveoli (goal: 6–8 ml/kg ideal body weight)
6. Temperature management to maintain body temperature of 36–37 °C
7. "Goal-directed" fluid and volume therapy with haemodynamic monitoring

Inodilator agents (milrinone, dobutamine), where necessary, can be given to support the right ventricle. Sildenafil has been approved for intravenous therapy of pulmonary arterial hypertension and can therefore be used in the perioperative period.[10] These need an assembly of a nebuliser in the ventilatory circuit. Specific pulmonary vasodilators can be administered both intravenously (iloprost, prostacyclin) and by inhalation (Table IV). Nitric oxide (NO) administration requires a specialised delivery system which needs a knowledgeable operator. During laparoscopic procedures, care should be taken to avoid extremes of pressure and positions during pneumoperitoneum employment.[11]

**Table IV:** Interventions for reduction of right-ventricular afterload[2]

| | |
|---|---|
| **Intravenous vasodilation** | |
| Milrinone | 50 µg/kgBW bolus, followed by 0.5–0.75 µg/kgBW/min continuously |
| Dobutamine | 2–5 µg/kgBW/min continuously |
| Prostacyclin | 4–10 ng/kgBW/min continuously |
| Na-nitroprusside | 0.2–0.3 µg/kgBW/min continuously |
| Nitroglycerine | 2–10 µg/kgBW/min continuously |
| **Pulmonary-selective inhaled vasodilatation** | |
| Iloprost | 5–10 µg for 10–15 min (by ultrasonic nebuliser) |
| Nitrogen monoxide | 0.5–20 ppm continuously |
| Prostacyclin | 30–40 ng/kgBW/min continuously |
| Milrinone | 2 mg (–5 mg) for 10–15 min (diluted in 10–15 ml NaCl 0.9%) |

Pain management through multimodal means, including patient-controlled devices, is advisable. High-dose opioids as single doses could lead to respiratory depression with hyperaemia and hypercarbia with undesirable effects.

### *Postoperative considerations*

A multidisciplinary approach in preparation of the patient for surgery and their postoperative destination, with good planning, understanding of the physiology and anatomy, coupled with clinical experience, can reduce untoward complications. Most of the patients will need a high/intensive care and monitoring environment as a minimum. This environment is also important for continued postoperative pain management.[12]


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