Identifying and treating postanesthesia emergencies

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Emergencies are serious, unanticipated events that require immediate action. Evaluation of an emergency in the postanesthesia care unit (PACU) is complicated by the presence of residual drugs given by anesthesia providers. The overall PACU complication rate is approximately 23%, with cardiovascular and respiratory events being the most emergent problems.[1-3] Patients who undergo orthopedic and abdominal surgeries have the highest rate of PACU complications. Intraoperative hypertension or hypotension is associated with a higher rate of PACU complications, and patients who undergo general anesthesia have a higher rate of complications than patients who receive regional anesthesia. Critical respiratory events after general anesthesia have a reported rate of 1.3%.[4]

Three of the most critical events that PACU nurses may face requiring emergency intervention are:

- residual neuromuscular blockade
- acute postoperative hypertension
- acute hypotension.[5]

Nurses who practice in this setting should be able to recognize and manage these emergencies.

Residual neuromuscular blockade

Neuromuscular blocking drugs (NMBDs) are commonly used in the OR to assist with tracheal intubation, improve surgical exposure, and ensure immobility during surgery. Common NMBDs used during surgery include rocuronium, vecuronium, pancuronium, atracurium, and cisatracurium. However, these agents often have residual effects that are detrimental to recovery.

Residual neuromuscular blockade is the continuation of muscle weakness after administration of an NMBD.[6] The use of nondepolarizing NMBDs presents an independent risk factor for reintubation in the PACU.[7] Among NMBDs, the odds of reintubation in the PACU are the highest with the use of aminosteroid NMBDs (Odds Ratio [OR] 7.26, 95% CI 2.67 to 19.8) versus benzylisoquinoline NMBDs (OR 3.89, 95% CI 1.46 to 10.3), even when reversal drugs are given.[7] In addition, patients who require early postoperative intubation have a much higher 30-day mortality rate than those who do not (15% versus 1.9%, *P* less than 0.001) and carry a 90-fold increased risk for in-hospital mortality.[7-9] To minimize residual neuromuscular blockade, intermediate-acting drugs are preferred over longer-acting agents unless the postoperative plan includes mechanical ventilation. (See *Classification of NMBDs.*) However, even the use of intermediate-acting NMBDs is associated with a postoperative decline in oxygen saturation and a prolonged PACU stay.[6]

The degree of neuromuscular blockade should be monitored intraoperatively and antagonized with acetylcholinesterase inhibitors (neostigmine or edrophonium) based on this assessment and prior to emergence from general anesthesia.[10] An antimuscarinic agent (glycopyrrolate or atropine) is commonly coadministered with reversal agents to counter adverse cholinergic effects. Despite appropriate monitoring and drug reversal, the proportion of patients exhibiting residual neuromuscular blockade ranges from 33% to 64%.[11] Complications such as upper airway obstruction, pharyngeal dysfunction, and aspiration are associated with residual muscle paralysis.[10,11] However, it may be difficult to separate the postoperative impact of residual neuromuscular blockade from the continued effects of opioids, benzodiazepines, and inhaled anesthetic agents in the immediate postoperative period.[6]

The odds of reintubation in the PACU are the highest with the use of aminosteroid NMBDs, even when reversal drugs are given.

Currently, the definitive technique used to isolate residual neuromuscular blockade from other factors is the use of a peripheral nerve stimulator (PNS) to measure the train-of-four ratio (TOFR). Using this technique, a ratio is determined by comparing a series of four electrically elicited muscular responses or twitches. For example, if the fourth muscular response is half the strength of the first muscular response, the TOFR is 0.5. Clinicians cannot accurately discern this level of measurement by tactile or visual assessment, making the use of a specialized PNS the only quantitative method of assessing for residual NMBD effects.[6,10,12]

Residual neuromuscular blockade is defined as a TOFR less than 0.9, and this degree of recovery is necessary to avoid related complications. The importance of PNS monitoring is emphasized by the fact that 90.5% of patients experiencing critical respiratory events in the postoperative period demonstrate a TOFR less than or equal to 0.9.[11]

Treatment of residual neuromuscular blockade is dependent upon a quantitative assessment and the patient's signs and symptoms. With adequate recovery from a neuromuscular blockade, a patient should be able to normally ventilate, protect, and maintain the upper airway and talk and cough effectively.[6] Unfortunately, these common clinical tests have a low reliability unless the TOFR is less than 0.5.[13] However, patient variability exists and many patients tolerate a mild level of residual neuromuscular blockade without treatment.[10]

Patients who exhibit respiratory compromise (inability to maintain their own airway, hypoxemia, or hypercapnia) require immediate intervention.

Classification of NMBDs

Classification	Examples of generic drugs	Duration of action	Risk of residual muscle weakness
Depolarizing	Succinylcholine	Very short	Low
Nondepolarizing			
Aminosteroid	Rocuronium	Intermediate	High
	Vecuronium	Intermediate	High
	Pancuronium	Intermediate	High
Benzylisoquinoline	Atracurium	Intermediate	Moderate
	Cisatracurium	Intermediate	Moderate

Endotracheal intubation with positive pressure ventilation is the gold standard for securing a compromised airway and/or treating respiratory failure; however, noninvasive ventilation (NIV) should also be considered. While the choice of ventilatory support depends on the specific situation, early intervention with NIV is key to postoperatively improving gas exchange.[14] Bag-valve-mask ventilation should be used to bridge the time from recognition of respiratory compromise until the method of ventilation is chosen.

From a pharmacologic standpoint, the duration of action of some NMBDs may exceed that of standard reversal agents. Only after sufficient monitoring of neuromuscular status and review of previously administered reversal agents should additional doses of reversal drugs be considered. The administration of neostigmine, along with sufficient recovery from a neuromuscular blockade or an excessive dose of neostigmine, may result in a paradoxical compromise of neuromuscular function and muscle weakness.[8]

Acute postoperative hypertension

Acute postoperative hypertension (APH) occurs in 1% to 2% of general PACU patients and in over 50% of cardiac surgery patients.[2,3] Hypertension typically occurs as the patient is emerging from the cardiodepressant effects of anesthesia within the first 1 to 2 hours in the PACU.[5,15,16] Acute hypertension in the PACU is a systolic BP greater than 20% of baseline or diastolic BP greater than 110 mm Hg. Chronically hypertensive patients who have intraoperatively labile BP are more likely to experience postoperative hypertension. This history is an important part of the transfer of care.

Acutely hypertensive patients are at high risk for dysrhythmias, myocardial infarction, heart failure, or stroke. An immediate search for the cause is crucial. Risk of surgical bleeding also increases in APH and is more common after cardiac, vascular, head and neck, and neurovascular procedures. Nurses caring for postoperative patients after carotid endarterectomy, abdominal aortic surgery, radical neck dissection, and intracranial surgery have a high probability of dealing with APH.[17] Pain, anxiety, hypoxia, hypercapnia, hypothermia, volume status, increased intracranial pressure, and distended viscus should be ruled out and/or treated prior to instituting antihypertensive therapy.[15,16] Abrupt withdrawal of chronic antihypertensive medication as a result of preoperative instructions—especially centrally acting agents such as clonidine—may also cause a hypertensive crisis in the PACU.[15]

There are no definitive guidelines on the treatment of APH; however, adjustable parenteral therapy is indicated in postoperative hypertensive emergencies. BP should be reduced by a maximum of 20% within the first hour and slowly controlled to 160/100 over the next 2 to 6 hours.[3,16] The more immediate goal is a reduction in the diastolic pressure to less than or equal to 110 mm Hg over only 30 to 60 minutes.[3] Volume expansion may be required during therapy to prevent brain and kidney hypoperfusion. Continuous BP monitoring with attention to neurologic status and urine output is necessary. It is wise to administer a

Drugs of choice for APH

Drug* Mechanism of action/ therapeutic actions	Clinical considerations	Adverse reactions
Clevidipine Dihydropyridine L-type calcium channel blocker Acts on peripheral arterioles to decrease SVR	Onset 2 to 4 min Duration 5 to 15 min Not dependent on renal or hepatic function; metabolized by RBC esterases Contraindicated in: • Soy allergy • Egg allergy • Diseases of lipid metabolism • Severe aortic stenosis Discard within 12 hours of puncturing stopper	Reflex tachycardia and hypotension if titrated rapidly; do not treat this adverse reaction with beta-blockers May exacerbate heart failure Headache Nausea and vomiting
Esmolol $Beta_1$-adrenergic receptor blocker Acts preferentially on cardiac cells to slow heart rate and reduce contractility; decreases cardiac output At higher doses, $beta_2$-blockade may occur	Rapid onset 60 seconds Duration 10 to 20 minutes Contraindicated in: • Severe sinus bradycardia • Heart block greater than first degree • Sick sinus syndrome • Decompensated heart failure • Cardiogenic shock • Pulmonary hypertension • Coadministration of an I.V. cardiodepressant calcium channel blocker with esmolol is contraindicated Not dependent on renal or hepatic function; metabolized by RBC esterases	Severe bradycardia Heart block May exacerbate heart failure Bronchospasm May mask signs of hypoglycemia Infusion site reactions
Labetalol $Alpha_1$ and nonselective beta-adrenergic blocker Decreases SVR, possible reduction in HR; maintains CO	Onset 2 to 5 minutes Duration of action 2 to 4 hours Contraindicated in: • Bronchial asthma • Severe sinus bradycardia • Heart block greater than first degree • Overt heart failure • Cardiogenic shock • Conditions associated with severe and prolonged hypotension Metabolized in the liver	Hypotension Nausea and vomiting Dizziness Paresthesias
Nicardipine Short acting Dihydropyridine L-type calcium channel blocker Preferentially acts on coronary and cerebral arterioles reducing cardiac and cerebral ischemia Increases SV and coronary blood flow	Onset 5 to 15 minutes Duration 4 to 6 hours Contraindicated in: • Advanced aortic stenosis Metabolized in the liver	Hypotension Nausea and vomiting Dizziness Tachycardia Headache

Legend: CO, cardiac output; HR, heart rate; IOP, intraocular pressure; RBC, red blood cell; SV, stroke volume; SVR, systemic vascular resistance.
*Consult the manufacturer's complete prescribing information for dosing and other important information including contraindications and warnings.

drug with a rapid onset and short half-life that can be titrated to achieve a BP close to normal within minutes to an hour.

Since APH is associated with an increased sympathetic outflow that causes peripheral vasoconstriction and tachycardia, treatment with sympathetic alpha- and beta-blockers or calcium channel blockers is a common approach. The drugs of choice for treatment of APH include clevidipine, esmolol, labetalol, and nicardipine. (See *Drugs of choice for APH.*)[15,17,18]

Acute postoperative hypotension

Hypotension occurs in up to 2.7% of PACU patients and has detrimental effects that make emergency treatment necessary.[2] Postoperative hypotension is defined as a systolic BP less than 20% of baseline for more than 15 minutes.[5] Acute organ hypoperfusion due to hypotension leads to organ dysfunction, anaerobic metabolism, and harmful endocrine and inflammatory responses. Mean arterial BP (MAP) should be optimized at 60 to 65 mm Hg in most patients with MAP as high as 90 mm Hg in traumatic brain injury patients.[5] Overall, the MAP must be kept sufficient to maintain cerebral, cardiac, and renal perfusion.[19]

Residual anesthetics and opioids are common causes of early hypotension in the PACU; vasodilation causes a relative hypovolemia that recovers with the duration of time. Relative hypovolemia may also be caused by intra-abdominal hypertension that decreases venous return, depresses cardiac contractility, and increases systemic vascular resistance.[20] Other common causes include actual hypovolemia, acute hemorrhage, cardiac failure, and metabolic derangements, such as acidosis or severe electrolyte imbalance. It is imperative that the cause is identified and treated rapidly to avoid the development of a shock state.

Differentiating the cause of hypotension is relatively straightforward when systematically performed. Hypovolemia is generally signified by hypotension accompanied by tachycardia, pulse oximetry waveform variability with respiration, and concentrated or decreased urine output. Hypotension due to hypovolemia is a significant cause of reduced kidney perfusion and acute kidney injury.[19] Excessive or unrecognized intraoperative blood loss can be assessed with evaluation of the hematocrit. In most cases, treatment with volume (crystalloid, colloid, blood products) will elevate MAP sufficiently, although in about 20% vasopressors may be indicated.[5]

The presentation of postoperative anaphylaxis, sepsis, or vasoplegia mimics hypovolemic hypotension with tachycardia and tachypnea; temperature may or may not be elevated. Treatment is symptomatic in order to maintain organ perfusion (fluids, vasopressors, corticosteroids, antihistamines); severe hypotension refractory to epinephrine may respond to methylene blue administration.[21]

Hypotension due to inadequate cardiac output may be due to intraoperative myocardial infarction, heart failure, cardiac tamponade, or pulmonary emboli. A physical examination and ECG are necessary to assess peripheral perfusion and identify underlying dysrhythmias. Volume replacement must be cautious if cardiac hypotension is suspected. Hypotension associated with tachycardia or loss of atrial kick may require rate control and/or antiarrhythmic agents.

Summary

Although PACU emergencies are rare, early recognition and treatment by PACU nurses is necessary for patient care. Residual neuromuscular blockade is a significant cause of respiratory complications in the PACU; ventilatory support should be instituted while muscle function with a PNS is assessed. Reversal agents should not be administered prior to this quantitative exam. APH is frequent after cardiovascular surgeries and can cause substantial morbidity. Short-acting I.V. agents should be immediately available to reduce MAP in a controlled fashion. Assessment of acute hypotension should be systematic, and include a differential diagnosis of relative or actual hypovolemia, cardiac causes, and systemic metabolic derangements should be considered. Any of these three serious postoperative complications can be effectively managed in the PACU. **OR**

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