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REVIEW

Emergence Issues - not so simple

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Introduction

Emergence from anaesthesia is by definition the process of return to baseline physiological function of all organ systems after cessation of administration of general anaesthesia and is the stage from general anaesthetic that includes a return to spontaneous breathing, voluntary swallowing and normal consciousness.¹ It is a time of great physiological stress for many patients and should ideally happen smoothly in a controlled environment but, unfortunately, can happen on the way to the recovery room.¹

Emergence from general anaesthesia depends on:

- Inhalational agents: alveolar ventilation, uptake/ solubility of the agent, duration of the anaesthetic and the concentration used.
- Intravenous agents: pharmacokinetics of the drugs: duration of infusion and the elimination and context sensitive half-life.

The incidence of complications in the recovery room can be up to 23 %.² Therefore, patients should not leave theatre unless they have a stable patent airway, adequate ventilation and oxygenation and they should be haemodynamically stable before transfer to the recovery room. Timely recognition and management of issues that arise in the immediate postoperative period saves lives, time and money.

Common problems that occur during emergence include:

- · Postoperative nausea and vomiting (PONV)
- Respiratory complications
- · Cardiovascular complications
- Emergence delirium and postoperative cognitive dysfunction
- · Delayed emergence
- Hypothermia

The most common complications in recovery room are PONV, followed by upper airway problems and then hypotension.²

Postoperative nausea and vomiting (PONV)

PONV is the most common complication in the immediate postoperative period in the first world and this may lead to delay in discharge, unplanned admissions and poor patient satisfaction. Patient factors, anaesthetic factors and type of surgery need to be taken into account. The Apfel scoring system and other scoring systems are used to predict which patient is likely to develop PONV.²

Prophylaxis before the development of PONV significantly reduces the incidence of PONV.

Drug classes available for prophylaxis are:

- Serotonin-receptor antagonists 5-HT₃: Ondansetron, dolasetron and granisetron. These are the most commonly used agents because they have fewer side effects and they have intravenous preparations. Because of their effect on the QT interval these agents should be avoided in patients with a prolonged QT interval and those with cardiac dysrhythmias.
- Glucocorticoids: a meta-analysis found that dexamethasone reduces the incidence of PONV by 50%. Dose: 4 mg at induction and this dose had similar clinical effects to 8- 10 mg in reducing PONV.
- Anticholinergic: Scopolamine transdermal patch has a slow onset of action (2-4 hours) but the effect is prolonged for up to 24 hours postop. The patch should be removed 24 hours after surgery. Its side effects are drowsiness, blurred vision and dry mouth.
- Neurokinin-receptor antagonist: Aprepitant. It is a selective neurokinin (pro-emetic) antagonist that penetrates blood brain barrier. It has shown efficacy for chemotherapy induced nausea and vomiting. It has a long duration of action and its use is limited because of cost considerations.

Approach to therapy using patient risk factors:²

- Low risk for PONV (no risk factor): no prophylaxis unless for surgical indications
- Moderate risk (1 risk factor): single prophylactic agent
- High risk (> 1 risk factor): 2 or three agents used and avoidance of opioids and volatile agents.

Rescue therapy is not as effective as prophylaxis and a different class of agents should be used. The $5HT_3$ are the most commonly used for rescue therapy.

Respiratory complications

Respiratory problems are the second most common complications in the postoperative period.⁶ Respiratory complications after extubation are three times more common than during intubation and induction of anaesthesia. There remains a controversy of where extubation should take place: in the UK 65-91% of extubations are done in theatre. Although many hospitals now use recovery room to perform extubations the ultimate responsibility for the patient is the anaesthetist.³ In the immediate postoperative period the most common cause of hypoventilation include: airway obstruction, effects of anaesthetics and poorly controlled pain.² Impaired oxygen exchange in the postoperative period is rare but can occur as a result of intrapulmonary shunting, pulmonary oedema and pulmonary embolism.

Common causes of airway obstruction:

- Pharyngeal muscle weakness
- Laryngospasm
- Vocal cord paralysis
- Airway oedema
- · Postoperative cervical haematoma
- · Foreign bodies

Pharyngeal muscle weakness

Loss of pharyngeal motor tone leading to muscular laxity can be due to residual neuromuscular blockade, residual anaesthetic effects and opioids. Preoperative signs and symptoms of OSA need to be actively sought out as a risk factor for upper airway obstruction. Decrease pharyngeal muscle tone causes the base of the tongue and tissues of the posterior oropharynx to obstruct the supraglottic inlet. The patient present with retraction of sternal notch and paradoxical motion of the abdominal muscle. Treatment is jaw thrust and CPAP via face mask.

Laryngospasm

Most common cause of airway obstruction in paediatric patients but can also occur in adults. Occurs mostly during extubation in a patient with secretions and light anaesthesia. Laryngospasm is common in children during intubation without muscle relaxation.

Management includes:³

- 100% oxygen
- · Visualise and clear pharynx/airway
- Jaw thrust with bilateral digital pressure behind temperomandibular joint, oral/nasal airway
- Mask CPAP/IPPV
- Deepen anaesthesia with propofol (20% induction dose)

- Succinylcholine 0.5 mg/kg to relieve laryngospasm (1.0– 1.5 mg/kg i.v. or 4.0 mg/kg i.m. for intubation). Be aware of contraindications, for example, neuromuscular problems
- Intubate and ventilate

Vocal cord paralysis

Rare cause of upper airway obstruction. Usually due to trauma to the vagus nerve or recurrent laryngeal nerve after surgery involving the head and neck or thoracic cavity. The incidence of vocal cord paralysis is 1-3% after thyroid surgery. Unilateral vocal cord paralysis present as hoarseness while bilateral vocal cord injury will present like laryngospasm requiring even tracheostomy if intubation is not successful.

Airway oedema

The airway can become oedematous due to direct tissue trauma from multiple intubation attempts or surgical manipulations.

An endotracheal tube leak test can be performed if airway oedema is suspected and if airway patency is compromised then the patient should not be extubated.

Postoperative cervical haematoma

Following surgery of the neck the incidence of cervical haematoma is 0.2- 1.9%. The lumen of the trachea can be narrowed to less than 5 mm before the patient becomes clinically symptomatic. After this there is a rapid deterioration made difficult by distorted anatomy and the potential for catastrophic outcome.

Management is to remove the sutures, evacuate the haematoma, secure the airway which will be challenging and explore the wound.

Foreign bodies

Rare cause of airway obstruction during emergence. Dentures, teeth and throat packs need to be removed before emergence.

A systematic approach to emergence and extubation is outlined in Figure 1.

Cardiovascular complications

The most common circulatory complications during emergence are hypertension, hypotension and arrhythmias. These complications may be related to cardiovascular comorbidities e.g. coronary artery disease, hypertension and blood loss.

- Hypotension from hypovolaemia, myocardial failure, sepsis, spinals and epidurals need to be corrected before discharge to the ward.
- **Hypertension**: Patients who develop hypertension in recovery room are at higher risk for postoperative complications. The most common cause during emergence is pre-existing hypertension. Other frequent causes of hypertension in recovery room are inadequate pain control, urinary retention and anxiety. Alcohol and drug withdrawal are rare causes of hypertension in the recovery room.
- Arrhythmias: the most common arrhythmias in the postoperative period are premature ventricular contractions and atrial fibrillation. AF is common following cardiac surgery and pneumonectomy. Management must exclude hypoxia, hypercarbia, ischaemia and electrolyte abnormalities.

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Figure 1: Emergence and extubation

Emergence delirium (ED)

A dissociated state of consciousness in which a child is irritable, uncompromising, uncooperative, incoherent, inconsolable crying, moaning, kicking and thrashing.⁴ It occurs in all patients, but there is an increased incidence in children. There is a wide range of incidence from 2-80 % due to the rating systems and the wide range of surgical procedures that were included in the studies.⁴ ED is very distressing to the anaesthetist, nurses

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and the parents. The phenomenon itself is self-limiting with duration of 15-30 minutes. ED can cause disruption of surgery, self-injury, removal of drains and injury to nursing staff.

Aetiology

The introduction of short-acting volatile agents has been historically associated with the increase in ED but it's worth noting that ED can occur with halothane.⁵

The sudden emergence from anaesthesia into a disordered state of consciousness or unfamiliar environment has been proposed as the cause of ED.

This theory of sudden awakening does not offer complete explanation, as propofol doesn't cause ED despite rapid awakening profile.

Elevated postoperative pain was also suggested to contribute to ED but this cannot be true because patients who had MRI with sevoflurane had an increased incidence compared to halothane. Inhalational anaesthetic agents are thought to have an underlying mechanism of action triggering ED by possibly acting on the GABA receptor affecting balance between excitation and inhibition.⁶

Risk factors for ED⁴

- Rapid emergence from anaesthesia
- · Use of short acting volatile anaesthetic agents
- Postoperative pain
- Type of surgery (head and neck/ ENT)
- Age (1-5) years
- · Preoperative anxiety
- · Child temperament
- Scales for evaluation of severity and incidence ED Cravero scale:
- The PAED scale is validated but difficult to use because of many variables and grading
- The Watcha scale: has fewer variables and easier to use.

The PAED scale has been validated but it is difficult to use clinically. It is much more practical to use a simple scale and then the PAED scale for severity.

Preventative strategies: 5,6

Premedication

Midazolam has been used to have a calmer child but has shown varying results on ED with the majority showing an increase in ED after sevoflurane. Ketamine has been used at a dose of 6 mg/kg orally with success

- Analgesia and sedatives
 - Fentanyl: 1 $\mu g/kg$ 10 min before the end of the procedure
 - Propofol : 1 mg/kg at the end of the procedure
 - Ketamine: 0.25 mg/kg at the end of procedure
 - $\alpha_{2}\text{-}$ adreno-receptor agonist: clonidine (2-3 $\mu g/kg$ iv or 1-3 $\mu g/kg$ caudally) and dexmedetomidine (0.15- 0.3 $\mu g/kg)$
 - Perioperative analgesia

The PAED scale: A scale of > 10 has a sensitivity of 86%

Behaviour	Not at all	Just a bit	Quite a bit	Very much	Extremely
Makes eye contact with care giver	4	3	2	1	0
Purposeful actions	4	3	2	1	0
Aware of surroundings	4	3	2	1	0
Restless	0	1	2	3	4
Inconsolable	0	1	2	3	4

Cravero scale: A score of \geq 4 for 5 or more minutes despite calming, is ED2⁵

Behaviour	Score
Obtunded with no response to stimulation	1
Asleep but responsive to movement or stimulation	2
Awake and responsive	3
Crying for more than 3 minutes	4
Thrashing behavior that requires restraint	5

The Watcha scale: Defines ED at level 3-4

Behaviour	Score
Asleep	0
Calm	1
Crying but can be consoled	2
Crying but cannot be consoled	3
Agitated and thrashing around	4

Parental presence both at induction and awakening

Treatment of ED

- Fentanyl
- Propofol
- Reuniting with parents and reassurance

Postoperative agitation and delirium in adults

This phenomenon is very common in the elderly and they can present a challenge in recovery room. The precise aetiology is not known. The risk factors are those older than 70 years, bladder catheterisation, visual and hearing defects.⁷ Organic causes also need to be excluded: alcohol withdrawal, hypertensive encephalopathy, hypoperfusion, hypogly-caemia , hypoxaemia and medications.

Management is to treat the cause and symptomatic treatment.

Delayed emergence

Failure of a patient to regain consciousness 30-60 minutes following a general anaesthesia. The time taken to full consciousness is affected by patient factors, anaesthetic factors, duration of surgery and painful stimulation.⁸ Delayed recovery is usually multifactorial.

Causes of delayed emergence:8

- Residual anaesthetic drugs:
 - Opioids: respiratory depression and direct sedation via opioid receptors
 - Benzodiazepines: respiratory depression in combination with opioids.

- Neuromuscular blockers and this include drug interactions with CCB, acidosis and hypomagnesaemia.
- Metabolic effects and electrolytes abnormalities:
 - Hypoglycaemia
 - Hyperglycaemia
 - Hyponatraemia : < 120 mmol/l confusion and irritability and <110 mmol/l patients get seizures and coma.
 - Hypernatraemia cause dehydration, ruptured vessels and coma
- Respiratory failure:
 - Hypercapnia initially stimulates respiratory center but thereafter depress the respiratory failure causing hypoventilation and apnoea.
 - Hypoxia causes cerebral hypoxia depressing cerebral function and finally cell death.
- Hypothermia has widespread effects with neurological and respiratory depression at decreasing temperatures. These are compounded by the effects of hypothermia on drug metabolism.
- Neurological causes
 - Hemorrhage, thrombus and emboli
 - Hypotension and hypoperfusion of the brain
 - Hypoxia

Clinical assessment and assessment is important in approach to delayed emergence and the diagram that follows is a guide to management. (Figure 2)

Hypothermia

Decrease of body temperature by 2 °C slows drug metabolism and decrease platelet function. Large number patients become hypothermic on admission to recovery room. Shivering during emergence cause patient discomfort and increases myocardial oxygen consumption and should be treated with pethidine (12.5-25 mg) or α_2 -agonists. Emergence from anaesthesia is usually uneventful but unexpected complications are costly and can have adverse outcome. Vigilance and proper monitoring during this period to avoid unnecessary complications.

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Figure 2: A stepwise approach to the patient with prolonged unconsciousness⁸

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Further reading

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