

Delayed Awakening From Anaesthesia

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Abstract

Ideally, on completion of surgery and anaesthesia, the patient should be awake or easily arousable, protecting the airway, maintaining adequate ventilation and with their pain under control. Time to emerge from anaesthesia is variable, and depends on many factors related to the patient, the type of anaesthetic given and the length of surgery. Unexpected delayed emergence after the use of general anaesthesia has a plethora of causes.

EMERGENCE FROM ANAESTHESIA

Following an inhalational-based anaesthetic, the speed of emergence is directly proportional to alveolar ventilation and inversely proportional to the agent's blood solubility. As the duration of anaesthesia increases, emergence also becomes increasingly dependent on total tissue uptake of the anaesthetic, which is a function of the agent's solubility, the average concentration used and the duration of exposure.

Emergence from an IV anaesthetic is a function of its pharmacokinetics. Recovery from most IV agents is dependent chiefly on redistribution rather than elimination half-life. This changes once the total administered dose increases and the tissue becomes saturated. Once this occurs, the termination of action becomes increasingly dependent on the elimination or metabolic half-life. Hence, advanced age, renal or hepatic disease can prolong emergence. Pre-operative medications can also influence the speed of emergence, particularly if its duration of action outlasts the duration of the procedure.

CAUSES OF DELAYED AWAKENING

- Residual drug effect – which may be due to overdosage, undue susceptibility of the patient or delayed drug metabolism.
- Duration and the type of anaesthetic given - agents with low blood gas solubility are eliminated more rapidly.
- Potentiation by other drugs – sedatives, anxiolytics and tranquilizers are known to potentiate the depth of anaesthesia.
- Prolonged NM blockade – secondary to overdosage or incomplete reversal, can mimic unconsciousness by preventing motor response to stimuli. Prolonged apnoea following suxamethonium due to an abnormal or absent plasma cholinesterase enzyme may lead to delayed emergence.
- Metabolic and endocrinal disorders – decreased drug metabolism in hypothyroid patients may lead to prolonged unconsciousness. Similarly severe hypo / hyperglycaemia are common causes of post-operative coma.
- Acid-base and electrolyte imbalance - hyperphosphataemia following administration of sodium phosphate for bowel preparation, hyponatraemia following TURP are common causes of delayed emergence.
- Hypothermia – severe hypothermia can lead to reduced level of consciousness. A core temperature of less than 33°C, itself has a marked anaesthetic effect and will potentiate the CNS depressant effect of anaesthetic drugs by reducing the MAC value of inhalational agents. It also potentiates NM blockade and limits drug metabolism.
- Neurological complication – cerebral hypoxia as a result of any intraoperative hypoxic insult and intracerebral events like haemorrhage, embolism or thrombosis may all lead to delayed awakening. Another rare cause is “Factitious disorder” which is defined by Diagnostic and Statistical Manual of

Mental Disorders as the “intentional production of physical or psychological symptoms in order to assume the sick role”. The patients' achieve a trance like state, and have the ability to ignore pain. They often have a period of amnesia. It is a diagnosis of exclusion that should be considered only after other medical conditions have been ruled out as a cause of altered consciousness.

ASSESSMENT

The evaluation of prolonged unconsciousness after anaesthesia requires an organized analysis:

- Level of pre-operative responsiveness – Is there unrecognized intoxication with drugs or alcohol? Was there any preexisting mental dysfunction?
- Pre-operative and intra-operative medications – The time and amount of all medications given should be noted and any unusual intra-op event should be reviewed.
- Stimulus – Firm tactile stimulus, which is often more effective than verbal Stimulation, should be used to elicit arousal.
- Ventilation – The rate and character of spontaneous ventilation can indicate the depth of anaesthesia.
- Autonomic tone – Heart rate, rhythm and systemic blood pressure can indicate the level of autonomic tone and the adequacy of cerebral perfusion.

PRACTICE POINTS

- Plan anaesthesia according to –
 - Duration of surgery
 - Type of surgery – an abdominal surgery will need a denser NM blockade as compared to limb surgery.
 - Coexisting diseases – Hypothyroidism, Myasthenia Gravis, hepatic or renal disease.
- Peripheral nerve stimulator guided NM blockade.
- Bispectral index monitoring for depth of anaesthesia - 40 – 60 is recommended for general

anaesthesia.

- Maintain normothermia, normocarbida and avoid hypoxia.

MANAGEMENT

- Immediate care
 - Airway – maintain a clear airway and oxygenation. Re-intubate if indicated.
 - Breathing – ensure adequate respiration. If indicated, ventilate the patient via ETT.
 - Circulation – assess heart rate, BP, ECG and peripheral perfusion.
 - Temperature – use body warmer.
- Intensive monitoring of all haemodynamic parameters, EtCO₂, SpO₂, CVP, intake and output is mandatory.
- Review the history, investigations and peri-operative management including the anaesthesia chart and the timings of drug administration.
- Assess for persisting NM blockade using a nerve stimulator and repeat reversal if needed. In case of succinylcholine apnoea prolonged ventilation (12 – 36 hrs), and Fresh frozen plasma transfusion is required.
- Arrange for antidotes
 - Inj. Naloxone - for suspected opioid narcosis.
 - Inj. Flumazenil – for Benzodiazepine overdosage.
- Check blood glucose and treat accordingly.
- Correct acid – base and electrolyte imbalance if indicated.
- If no other cause found, an intracerebral event may be suspected and a full neurological evaluation should be performed. Radiological imaging (CT or MRI) is often required.

CONCLUSION

Delayed awakening of varying degree is not uncommon after

anaesthesia and may have a number of different causes, individual or combined, which may be both drug or non-drug related thus causing a diagnostic dilemma. The primary management is always in support of airway, breathing and circulation, whilst the cause is sought and treated as outlined above.

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