

# Anaesthetic drugs: effects on pulmonary function

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### ABSTRACT

The fundamental function of the lungs is to keep the blood oxygenated and remove carbon dioxide via the network of capillaries next to the alveoli. Utilizing ventilatory reserve capacity and alterations to lung mechanics help to maintain this. The induction of anaesthesia affects pulmonary functioning by causing unconsciousness, reflex depression, rib cage alterations, and hemodynamic abnormalities. All anaesthetic medications, including those administered through inhalation, have an impact on pulmonary functions either directly by acting on the respiratory system or indirectly by acting on other systems. Comparatively to intravenous induction drugs, volatile

anaesthetics have more prominent effects on pulmonary functions, resulting in hypercarbia and hypoxia. The patient's position also causes significant modifications in lung functioning. Neuromuscular blockers and anticholinergics have negligible impact. Combining volatile anaesthetics and induction agents with analgesics and sedatives may make them more potent. The final result may differ from when individual agents are used alone since many ones are employed during anaesthesia.

**KeyWords:** *General anaesthesia; Spinal anaesthesia; Analgesics; Airway*

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### INTRODUCTION

The lungs' principal job is to exchange enough gases so that the blood's oxygen level stays normal and to expel carbon dioxide. In order to satisfy the increased metabolic demand during the perioperative period, lung capacity must be optimised. As a whole, General Anaesthesia (GA) impairs breathing, which affects both oxygenation and carbon dioxide removal. The effects of anaesthetic agents and medications used during anaesthesia on respiratory smooth muscles and secretions are among the factors affecting pulmonary function, along with loss of consciousness, method of ventilation (spontaneous or mechanical), patient position, and anaesthesia.

### LITERATURE REVIEW

#### Effects of general anaesthesia

The pharyngeal and jaw muscles relax as a result of general anaesthesia, which also causes the tongue to shift posteriorly. Airway blockage, laryngospasm, and bronchospasm are caused by the loss of the cough reflex and excessive secretions. Patients are more vulnerable to problems if their airways are hyper-reactive [1]. Delivery of dry gases may compromise pulmonary function, particularly in younger patients, while tracheal intubation preserves the airway but

increases dead space.

#### Effect on hypoxic pulmonary vasoconstriction

In order to increase oxygenation, pulmonary capillaries in poorly ventilated zones respond by constricting blood flow to better-ventilated parts (Hypoxic Pulmonary Vasoconstriction, or HPV). Due to the fact that an increase in FiO<sub>2</sub> weakens HPV, there is some dependence on FiO<sub>2</sub>. HPV is suppressed by all volatile anaesthetics in a dose-dependent manner. At two minimum alveolar concentrations, isoflurane and halothane may reduce HPV by 50%. (MAC). Agents used for IV induction do not appear to have this effect [2]. In a patient with normal lung function, sodium nitroprusside and nitroglycerin-induced hypotension enhances pulmonary shunting and decrease Pulmonary Arterial Pressure (PAP) and Pulmonary Vascular Resistance (PVR). As damaging vascular alterations raise PAP and prevent vasodilators from lowering PVR in individuals with Chronic Obstructive Pulmonary Disease (COPD), pulmonary gas exchange is not impacted.

#### Effects of regional anaesthesia

The central neuraxial inhibition has no impact on pulmonary functioning in healthy people. Due to the paralysis of the abdominal muscles under high spinal anaesthesia, the vital capacity and expiratory reserve volume both decrease, which may make it difficult to cough and forcefully exhale. When spinal anaesthesia is administered above T<sub>6</sub>, there is a considerable decrease in FEV<sub>1</sub>, Forced Vital Capacity (FVC), and forced expiratory flow 25–75 in

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elderly patients and individuals with weak respiratory reserves. [3] Except in cases of phrenic nerve obstruction, VT is typically unaffected. In patients with crippling respiratory diseases, inspiratory muscles are enough to keep the patient breathing, but paralysis of the expiratory muscles may make it difficult for the patient to cough effectively and empty their lungs of secretions. However, these effects are so negligible in comparison to postoperative lung function after abdominal or thoracic surgery without epidural anaesthesia that the positive effects nonetheless result in an improvement in postoperative lung function. Thoracic epidural anaesthesia causes a 10% decrease in VC and FEV1 and no increase in bronchial reactivity in patients with severe asthma [4].

#### Effects of drugs used during anaesthesia

Due to cerebral depression, barbiturates cause dose-dependent respiratory depression. Transient apnoea occurs after IV injection and correlates well with MV and electroencephalography suppression. After receiving an induction dose of 3.5 mg/kg, the MV generally reaches its peak depression after 1 minutes-1.5 minutes, and after 15 minutes, it rebounds to its pre-drug level [5]. Thiopentone-induced "dual apnoea" is the term used to characterise the ventilatory pattern. During barbiturate anaesthesia, it is necessary to offer respiratory assistance via jaw holding and bag-mask ventilation since the initial apnoea lasts for a short period of time before being followed by a few breaths of normal breathing (VT) and then again followed by protracted apnoea. Barbiturates, more frequently methohexitone than thiopentone, induce induction by causing excitatory symptoms such as coughing, hiccoughing, tremors, and twitching because of an increase in muscular tone.

The central respiratory depression caused by methylhexitone lasts about as long as that caused by thiopentone. Patients awaken after around 5 minutes, but the ventilatory response to CO<sub>2</sub> and VT peaks at 30 s and 60 s after the induction dosage and returns to baseline after 15 minutes.

#### Inhalational agents

The VT and MV of all volatile anaesthetics decrease with increasing dose, and this respiratory depression may be partially offset by an increase in respiratory rate. When compared to isoflurane, the concurrent rise in respiratory rate is more pronounced with halothane, desflurane, and sevoflurane. Desflurane maintains MV up to 1.6 MAC alveolar concentrations when compensatory tachypnoea is present. The impact of various anaesthetic drugs on the severity of alterations varies. PaCO<sub>2</sub> at rest indicates the level of respiratory depression. Enflurane is more effective at raising PaCO<sub>2</sub> than desflurane, isoflurane, sevoflurane, halothane, and nitrous oxide in that order.

#### Anticholinergic agents

The most widely used anticholinergic drugs during anaesthesia are atropine and glycopyrrolate, which are generally utilised as premedication drugs to lessen bronchial secretions. By inhibiting muscarinic receptors, they work. Both medications reduce airway resistance by widening both large and small airways, increasing specific airway conductance, and increasing maximum expiratory flow rates, however, glycopyrrolate's action is more long-lasting. When used in conjunction with anticholinesterases for reversal of Neuromuscular Blocking Agents (NMBAs), both medications effectively block the muscarinic actions. Lung elastic recoil is decreased over the full range of lung volume. [6] As a result of bronchodilation, there is an increase in anatomical and physiological dead space, FEV1, FVC, FEV1/FVC ratio, and peak expiratory flow

#### Analgesics

Opioids reduce the sensitivity of peripheral chemoreceptors to carbon dioxide and directly affect the brain stem respiratory centre to cause dose-dependent respiratory depression. However, the patient may be awoken when directed and can breathe in verbal order. The respiratory rate is lowered by prolonging the expiratory duration (gasping respiration), and spontaneous respiration can be eliminated by high doses. Reduced hypoxic drive and CO<sub>2</sub> ventilation response. As repeated or heavy doses of opioids (like morphine) might be deposited being extremely lipophilic, delayed respiratory depression may occur as a result. Additionally, buprenorphine depresses MV, which has a ceiling effect at greater doses.

#### CONCLUSION

The effects of anaesthesia on pulmonary functioning may last well after surgery. Changes in body position required by the need for surgery and changes in respiratory mechanics brought on by the loss of consciousness are two ways that GA manifests its effects. The effects of agents and medications used during anaesthesia also have an impact on the lungs' ability to function, and these effects are reflected in changes in lung volume, airway resistance, and respiratory compliances, all of which influence the V/Q ratio. Even if newer medications and agents have a better safety record and fewer side effects, GA's negative effects cannot entirely be eradicated. Potential issues during anaesthesia and the post-operative period can be avoided with a thorough grasp of pulmonary functioning.

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