Emergencies and Treatment of Complications

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Introduction

The emergencies associated with the nonoperating room may be higher compared to those occurring in the operating room. These emergencies may be prevented better with monitoring of hemoglobin saturation by pulse oximeter and end-tidal carbon dioxide (SpO2 and ETCO2), as the more damaging events are inadequate oxygenation and ventilation. Robbertze et al. [1] compared 24 nonoperating room anesthesia closed claims with 1,927 operating room claims from 1990 to 2001. The most common emergencies during gastrointestinal endoscopy are difficulty controlling the airway and aspiration (and therefore inadequate ventilation and oxygenation), as well as hemodynamic instability.

In the gastrointestinal unit, appropriate monitoring and equipment must be available. Oxygen analyzer to monitor inspired oxygen concentration, SpO2, ECG, arterial pressure monitoring as well as oxygen supply failure and disconnection alarms should be available. All these may give an early warning of an emergency situation. Suction, an anesthesia cart including anesthetic and resuscitation drugs and equipment, immediate access to a defibrillator, and a fully stocked emergency cart should be available as well [2].
Respiratory Complications

The most frequent respiratory complications during gastrointestinal endoscopies arise from depression of respiration and/or airway patency problems. The patient’s physical status must be assessed before drug administration. Oxygen should be given routinely so that in case of an emergency, desaturation will be delayed and the anesthesia provider ‘can buy time’. With the use of the circle absorber system, tidal volume breathing for 3–5 min is effective in achieving maximal preoxygenation before induction of anesthesia. The 4 deep breaths per 0.5 min method results in suboptimal preoxygenation; therefore, it should be reserved only for emergency situations where time is limited. Neither tidal volume breathing nor the 4 deep breaths per 0.5 min technique improves preoxygenation at higher fresh gas flows, end-tidal O₂ values of 90% are not attained. When the duration of deep breaths is increased to 1.5 or 2 min with a high fresh gas flow (10 l/min), preoxygenation is further improved and end-tidal O₂ values equivalent to tidal volume breathing for 3 min are obtained [3].

Sedatives, opioids and general anesthetics depress respiration by either decreasing tidal volume or respiratory rate, or both. These drugs also depress the hypercapnic and hypoxic drive in a dose-dependent manner. Respiratory depression due to benzodiazepines is reversible by intravenous administration of flumazenil 0.2 mg every 3–4 min up to 1 mg, and respiratory depression due to opioids is reversible by intravenous administration of naloxone with an initial dose 0.2 mg and repeated doses of 0.1 mg every 2–3 min. However, due to their pharmacokinetic profile, both flumazenil and naloxone may wear off earlier than the sedative or opioid administered, which could result in re-sedation.

Airway obstruction is a serious emergency situation as it is followed by oxygen desaturation, hypoxemia and possible acute pulmonary edema. It may be an upper airway obstruction due to the tongue falling backwards, relaxation of the laryngopharyngeal muscles and/or presence of a foreign material in the mouth and pharynx. The measures to be taken in case of an upper airway obstruction should be examination of the mouth for foreign material, back tilt of the head, chin lift and jaw thrust [4].

A laryngospasm also causes airway obstruction and unless treated early will result in severe hypoxia. Application of IPPV (intermittent positive pressure ventilation) using the bag-mask-ventilation technique relaxes the vocal cords and allows air to pass through them. If laryngospasm persists, 0.1–0.2 mg/kg of succinylcholine may be given additionally to the bag-mask-ventilation. In critical circumstances, when intubation of the trachea is expected, 1–2 mg/kg of succinylcholine is given intravenously. However, the intubating dose of succinylcholine should be given only by those providers who are well trained to manage deep sedation and general anesthesia. Epinephrine 0.3–0.5 mg given intramuscularly, or in increments of 0.1 mg intravenously in severe cases, causes vasoconstriction and decongests the mucosa.

Lower airway obstruction may be caused by a bronchospasm elicited by a hyperactive airway or by an anaphylactic or anaphylactoid reaction. Treatment consists of a β₂-agonist, like albuterol, which is given via a metered inhaler and 0.1 mg of intravenous epinephrine every 3–5 min. Other drugs included in the treatment of bronchospasm are corticosteroids and aminophylline.

Hypotension

General anesthetics, even in subhypnotic doses, and sedatives depress the sympathetic outflow to the cardiovascular system. The provider must take care to increase venous return of blood. This increase can be achieved by increasing the myocardial contractility using β₁-agonists, increasing the venous return by administering intravenous fluids and/or vasoconstrictors. Finally if the patient has a heart rate below 60, this should be increased by administering atropine. Ephedrine acts directly on α- and β-adrenergic receptors and stimulates epinephrine release from the sympathetic nerve endings resulting in tachycardia, increased myocardium contractility and vasoconstriction. It is administered intravenously in increments of 5–10 mg every 3–5 min, and its effect lasts 60–90 min. In cases of hypovolemia or dehydration, the patient becomes tachycardic and an α-adrenergic agonist like phenylephrine is the vasoconstrictor of choice. Phenylephrine constricts the veins increasing the preload and systolic arterial pressure and the arteries increasing the diastolic pressure. The dose is 0.1 mg every 3 min to 0.5 mg [4].

Hypertension

A symptomatic hypertensive crisis with diastolic arterial pressure higher than 120 mm Hg needs immediate treatment involving emergency medical service. In the mean time, the provider must start treatment as soon as possible. Esmolol is a selective β₁-receptor antagonist and is given intravenously as a bolus dose of 1 mg/kg followed by an infusion 150 µg/kg/min. For severe hypertension, labetalol, a nonselective β₁- and β₂-receptor antagonist...
and α₁-antagonist, is given in a dose of 5–20 mg by slow intravenous injection. Finally, nitroglycerin infusion may be given in a dose of 0.5–10 μg/kg/min titrated to effect [4].

Sinus Tachycardia
Hypoxia, hypotension, pain and stress, all of which the patient may experience during gastrointestinal endoscopy, are factors capable of provoking sinus tachycardia. Treatment consists of intravenous fluids to support blood pressure in case of hypotension. If tachycardia continues, a selective β₁-receptor antagonist can be given intravenously to decrease the heart rate [4].

Angina, Myocardial Infarction
If the patient experiences chest pain, 0.15–0.6 mg of nitroglycerin every 5 min is administered sublingually. The total number of doses should not exceed the three sublingual doses. Blood pressure and heart rate should be assessed before each dose of nitroglycerin. If there is no relief of symptoms, the patient is transferred to emergency medical service. Aspirin (325 mg) is given as well. Its maximal antiplatelet influence is achieved within 1 h of administration [4].

Conclusion
In his article ‘Anesthesia and Sedation Outside the Operating Room’, Dr. Melloni concludes, ‘No anesthesia or sedation performed outside the operating room should be considered minor; it requires skill, experience, and organization’ [5, 6]. Patient preparation, patient consent, sedation and analgesia should be performed using the same standards with the standards applied to operating room cases.

References