Undesirable side effects of Succinylcholine

Undesirable Effects

Hyperkalemia

Depolarization of the postjunctional membrane results in extracellular movement of potassium ions. Under standard conditions, this produces an average increase in the serum potassium concentration of 0.5 to 1.0 mEq/L. Upregulation of junctional and extrajunctional cholinergic receptors may result in the release of a higher amount of potassium that is unpredictable and may result in severe hyperkalemia, to the point of cardiac arrest. This occurs in multiple clinical situations, including trauma, burn, immobility, and upper motor neuron disease. The increase in potassium is not tempered by administration of a defasciculating dose of a nondepolarizing muscular blocker.

Rhabdomyolysis

Rhabdomyolysis occurs after succinylcholine administration in patients with Duchenne muscular dystrophy and has also been associated with masseter muscle rigidity. The resulting hyperkalemia may provoke cardiac arrest, and myoglobinemia may result in renal failure. It is typically advised to avoid the regular use of succinylcholine in boys younger than 5 years because of the possibility of undiagnosed Duchenne muscular dystrophy, except for cases of refractory laryngospasm or emergent intubation.

Malignant Hyperthermia

Malignant hyperthermia (MH) is triggered by succinylcholine administration, and a known history of MH is an absolute contraindication. Clinicians should also strongly consider avoiding it in patients with a known family history or with conditions known to be associated with MH. A recent position statement by the Society for Ambulatory Anesthesia stated that although succinylcholine is a known trigger of MH, even outpatient surgical centers that do not stock dantrolene should maintain an emergency inventory of succinylcholine for laryngospasm. This recommendation is based on the much higher incidence of laryngospasm compared with MH, and a patient who is given succinylcholine can be closely observed for signs of MH and transferred to a center with dantrolene if needed (see Chapter 232 for more information on MH).

Postoperative Myalgia

A 2005 meta-analysis demonstrated that as many as 50% of patients have skeletal muscle myalgia 24 hours after succinylcholine administration. Techniques that were significantly effective in reducing this rate include administration of larger doses of succinylcholine (1.5 mg/kg rather than 1 mg/kg), lidocaine, NSAIDs, and nondepolarizing muscle relaxant (10%–35% of ED_{95}) administered in advance as a defasciculating dose. This should be done with caution, however, because the study patients who received nondepolarizing muscle relaxant had side effects, including diplopia, eyelid drooping, breathing difficulties, and dysphagia.

Cardiac Arrhythmias

Succinylcholine has sympathetic and parasympathetic activity because of its structural similarity to acetylcholine, and it may interfere with either pathway to the sinus node. This predisposes children with higher vagal tone to bradycardia and adults with less vagal tone to tachycardia with the first dose. Subsequent doses given within 10 min may result in sinus bradycardia or junctional rhythm because of the accumulation of metabolites (primarily succinylmonocholine and choline).

Increased Intraocular Pressure

Succinylcholine causes a modest transient increase in intraocular pressure that persists for 5 to 10 min after administration. Possible mechanisms include choroidal vascular dilation and a decrease in drainage secondary to elevated central venous pressure. Although patients with treated glaucoma are at minimal risk, administration of succinylcholine to patients with recent ocular incisions or penetrating eye injuries may result in vitreous expulsion and visual loss. Many case reports have been published and studies have been conducted to identify a pretreatment agent to obviate this adverse effect; results have been mixed with the use of nondepolarizing muscle relaxants, opioids, propranolol, lidocaine, and others.

Increased Intragastric Pressure

Succinylcholine can cause, on average, a 40-cm H_2O increase in intragastric pressure, presumably as a result of abdominal muscle contraction. Lower esophageal sphincter pressure also increases after the administration of succinylcholine, resulting in maintained gastroesophageal barrier pressures. Whether the administration of succinylcholine during induction causes increased susceptibility to esophageal reflux and possible pulmonary aspiration (secondary to increased intragastric pressures) remains debatable. Studies have shown that pretreatment with a nondepolarizing neuromuscular blocking agent decreases this rise in pressure.

Increased Intracranial Pressure

Several studies have suggested that succinylcholine may increase intracranial pressure, whereas others have been unable to demonstrate this phenomenon. This ambiguity has spawned a variety of clinical recommendations and considerable debate. The proposed mechanisms include decreased venous effluent from the brain as a result of fasciculation-induced increases in intrathoracic pressure, contraction of the neck muscles with resultant jugular venous compression, and succinylcholine-induced increases in afferent muscle spindle activity that cause increased cerebral blood flow, cerebral blood volume, and intracranial pressure. However, succinylcholine should not be deleted from the therapeutic armamentarium for emergency airway management based solely on concerns about increased intracranial pressure.

Suggested Readings

Joshi GP, Desai MS, Gayer S, Vila H. Succinylcholine for emergency airway rescue in class B ambulatory facilities: the Society for Ambulatory Anesthesia position statement. *Anesth Analg*. 2017;124(5):1447–1449.

Tran DT, Newton EK, Mount VA, Lee JS, Wells GA, Perry JJ. Rocuronium versus succinylcholine for rapid sequence induction intubation. *Cochrane Database Syst Rev*. 2015;(10) [CD002788].

Dysrhythmias

SCh activates muscarinic receptors, leading to bradycardia, junctional rhythms, and even sinus arrest – in children this an occur after one dose, but in adults is most common when a second dose is administered 5 minutes after the first . Risk can be decreased by pretreating with non-depolarizing NMBDs or by administering atropine. Interestingly, SCh can mimic ACh at other sites and also cause increased SBP and HR.

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Normal increase is 0.5 – 1.0 mEq/mL. Risk of serious hyperkalemia usually peaks 7-10 days after insult, but increased K+ release may occur as soon as 2-4 days after denervation injury, or after several days of immobility. Duration of risk has not been adequately characterized but is suspected to be for 3-6 months. renal failure itself does not increase the risk of SCh administration as long as hyperkalemia is well-controlled