

[Episode 139: Keywords Part 5: Succinylcholine and Interscalene Block](#)

On this episode: Dr. Jed Wolpaw and Dr Gillian Isaac

In this 139th episode I welcome Dr. Gillian Isaac back to the show to discuss another 2 ABA keywords, succinylcholine and interscalene brachial plexus block.

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Succinylcholine

- Topics tested: mechanism of action, pharmacokinetics, pharmacodynamics, prolongation of action and synergism, metabolism, side effects and toxicity, indications and contraindications, antagonism and blockade, drug interactions
- Commonly tested topics: indications, contraindications, termination of action, effect on fetus, side effects, pseudocholinesterase deficiency, dibucaine number

Succinylcholine Mechanism of Action

- Depolarizing drugs are agonists at Ach receptor → succinylcholine is only one in clinical use; other one was decamethonium which is no longer used or tested
- Succinylcholine is two Ach molecules joined together
- Succinylcholine mechanism of action: quaternary ammonium radicals of succinylcholine bind to two alpha subunits of nicotinic receptor → voltage sensitive Na⁺ channels sense membrane depolarization → channels open and close → depolarization → channels become inactivated
 - o Ach gets hydrolyzed by acetylcholinesterase very quickly → Na⁺ channels reactivated
 - o Succinylcholine does not get hydrolyzed by acetylcholinesterase in NMJ → prolonged activation of receptors → Na⁺ channels at endplate and perijunctional zone remain inactivated → junctional transmission blocked → flaccid muscle → called phase I block
- Succinylcholine diffuses away and is metabolized in blood stream by pseudocholinesterase aka plasma cholinesterase
- Prolonged exposure either via repeated dose or drip → result in phase II block
- Question: the effect of succinylcholine is terminated at postsynaptic effector cells by:
 - o A) binding and uptake by effector cells
 - o **B) diffusion into capillaries** → has to diffuse away from junction first
 - o C) hydrolysis by junctional cholinesterases → Ach
 - o D) hydrolysis by pseudocholinesterase
 - o E) spontaneous degradation into succinylmonocholine

Effects on Fetus

- Question: a parturient receives ketamine 2mg/kg and succinylcholine 1.5mg/kg for induction prior to elective caesarean delivery. Which of the following is most likely to be present in the newborn infant:
 - o **A) normal muscle tone** → succinylcholine does NOT cross placenta
 - o B) bradycardia
 - o C) opisthotonus → means severe muscle spasms related to tetanus
 - o D) respiratory depression
 - o E) seizures
 - Four classes of drugs that don't cross placenta: insulin, glycopyrrolate, heparin, neuromuscular blocking drugs (including succinylcholine)
 - Question: administration of succinylcholine 1mg/kg to a pregnant women rarely causes fetal neuromuscular blockade. Which characteristic of succinylcholine best explains this phenomenon:
 - o A) high protein binding → usual reason why drugs don't cross
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- **B) ionization** → succinylcholine is one of few that don't cross because of ionization, same for glycopyrrolate
 - C) lack of passive placenta diffusion
 - D) lipid solubility
 - E) metabolism in the fetal liver

Indications for Succinylcholine

- Question: A 28 y.o. patient has severe laryngospasm after extubation of trachea following general anesthesia. Administration of 100% oxygen using CPAP does not improve symptoms. Now the saturation is 75%. Which of the following is most appropriate immediate management:
 - A) LMA → already tried positive pressure with CPAP
 - B) lidocaine
 - C) racemic epinephrine
 - **D) succinylcholine** → key is saturation at 75%; if patient satting 98%, could try deepening anesthesia with propofol
 - E) cricothyroidotomy
- Two main indications for succinylcholine: tracheal intubation, laryngospasm
- Question: 40 y.o. man requires brief surgical relaxation after administration of neostigmine and glycopyrrolate for reversal of vecuronium induced neuromuscular blockade. Compared with a patient who has not had prior reversal of neuromuscular blockade, which of the following characterizes a succinylcholine blockade in this patient?
 - A) greater antagonism with calcium chloride
 - B) slower onset
 - C) less profound
 - D) less likelihood of phase II NMB
 - E) prolonged duration → giving anticholinesterase medication for reversal of non-depolarizing is going to have anti-pseudocholinesterase activity which prolongs duration of action; could be seen in setting of laryngospasm after reversal
- Question: 16 y.o. girl receiving isoflurane, NO, O₂, pancuronium for insertion of Harrington rod. In order to perform a wake-up test, the muscle relaxant was antagonized with neostigmine and atropine. The patient moved all extremities and was given thiopental and succinylcholine rapidly. After 45 minutes, no twitch could be elicited with a nerve stimulator. The most likely explanation is:
 - A) a dibucaine number of 20
 - B) incomplete antagonism of pancuronium
 - **C) prolongation of action of succinylcholine by neostigmine**
 - D) spinal cord damage caused by the abrupt arousal
 - E) synergism between succinylcholine and pancuronium

Contraindications for Succinylcholine

- Question: one week after sustaining third degree burns over 40% of the body surface area, a patient receives general anesthesia for debridement and skin grafting. Which of the following response to neuromuscular blockers is most likely?
 - A) clinically insignificant of serum potassium concentration after administration of succinylcholine
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- **B) increased risk of hyperkalemia after administration of succinylcholine** → 24 to 48 hours after significant burn start to develop immature extra junctional Ach receptors → no longer safe to give succinylcholine because could have significant hyperkalemic response
 - Not supposed to use it for at least one year
- C) increased sensitivity to vecuronium
- D) laudanosine toxicity after administration of atracurium
- E) normal serum potassium concentration if administration of succinylcholine is preceded by tubocurarine
- Question: succinylcholine can be administered safely to a patient with:
 - A) amyotrophic lateral sclerosis → any time there is denervation accident, you cannot give succinylcholine
 - **B) cerebral palsy**
 - C) pelvic crush injury sustained two weeks ago
 - D) 20% body surface area burns sustained 10 days ago
 - E) hemiparesis 1 month after a cerebral vascular accident
- Use of succinylcholine in patients with myasthenia gravis does not cause hyperkalemic response
 - Patients with myasthenia gravis are resistant to succinylcholine → use bigger dose
- Question: which of the following conditions is a contraindication to the use of succinylcholine
 - A) burns of 50% body surface area occurring 12 hours ago
 - B) cirrhosis
 - **C) myotonic dystrophy** → denervation type of disease
 - D) seizure disorder
 - E) spinal cord transection within the past 6 hours
- Question: hyperkalemia in response to the administration of an intubating dose of succinylcholine is associated with each of the following disorders except:
 - A) poliomyelitis
 - B) multiple sclerosis
 - C) hemiplegia
 - **D) acute cervical cord transection**
 - E) familial periodic paralysis

Side Effects of Succinylcholine

- Question: compared with similar use in adults, routine use of succinylcholine in children is hazardous because of the increased risk of which of the following:
 - A) anaphylactoid reactions
 - B) phase II blockade
 - C) pseudocholinesterase deficiency
 - D) pulmonary aspiration
 - **E) undiagnosed myopathy** → especially in boys, there is risk of undiagnosed X-linked myotonic dystrophy
- Question: anesthesia is induced with sevoflurane in a three year old girl. Sixty seconds after administration of succinylcholine 1mg/kg IV, heart rate decreases rapidly from 120bpm to 60bpm. What is the most likely cause?
 - A) acute hyperkalemia

- B) failure to pretreat with non-depolarizing relaxant
- C) sevoflurane overdose
- **D) muscarinic activity** → succinylcholine commonly causes bradycardia in kids due to structural similarity
- E) sympathetic ganglionic blockade
- Other side effects: increased gastric pressure, increased lower esophageal sphincter tone, elevated ICP, elevated intraocular pressure

Pseudocholinesterase Deficiency

- Question: A 15kg three year old child is anesthetized with halothane and NO. Trachea is intubated after administration of 30mg succinylcholine. At the conclusion of the 45 minute procedure, the child is not breathing. A peripheral nerve twitch monitor indicates no response to a TOF. Further investigation is most likely to show:
 - A) a normal response to non-depolarizing muscle relaxants
 - **B) a low dibucaine number** → abnormal function of pseudocholinesterase
 - C) a low plasma cholinesterase concentration
 - D) an underlying myopathy
 - E) a positive halothane caffeine contracture test → testing for malignant hyperthermia
- Pseudocholinesterase deficiency is rare; 96% population have normal plasma cholinesterase activity; 4% are heterozygous; 0.04% of population are homozygous atypical
 - Normal cholinesterase is inhibited by dibucaine
 - Normal people have dibucaine number of 80 → 80% of pseudocholinesterase will be inhibited
 - Heterozygous people have dibucaine number of 40
 - Homozygous atypical people have dibucaine number of 20
- Question: a 23 y.o. man who is receiving his first anesthetic has not resumed spontaneous ventilation two hours after receiving succinylcholine. TOF shows no twitch response. Which of the following is the most likely pseudocholinesterase genotype in this patient?
 - **A) atypical, atypical** → likely homozygous because it has been two hours; heterozygous prolongs by 30 mins compared to normal
 - B) fluoride resistant, fluoride resistant
 - C) fluoride resistant, silent
 - D) normal, normal
 - E) normal, silent

Phase II Block

- Question: 60kg 38 y.o. woman undergoes laparoscopic tubal ligation, paralysis is maintained for one hour by infusion of succinylcholine at a rate of 10mg per minute. At the end of the procedure, respirations are shallow and tetanic fade is noted on neuromuscular stimulation. In addition to continued mechanical ventilation, which of the following is the most appropriate next step in management?
 - **A) observe until the patient recovers spontaneously** → support patient until they recover for phase II block
 - B) monitor until the EtCO₂ reaches 50
 - C) determine the dibucaine number

- D) administer FFP
- E) administer glycopyrrolate and neostigmine
- For a phase II block, patient has to receive succinylcholine for prolonged period of time or repeated doses → 5mg/kg is dose at which consider phase II block

Interscalene Brachial Plexus Block

- Topics tested: anatomy, complications, side effects, technique
 - Using more ultrasound images on written exam and OSCE

Indications and Anatomy for Interscalene Brachial Plexus Block

- Indications for block: distal shoulder, arm, and elbow surgery
- Brachial plexus is nerve network that supplies upper extremity → formed by C5 to T1
- Exits cervical spine and travels between anterior and middle scalene muscles → space between scalene muscles called interscalene groove
- Palpable behind lateral head of sternocleidomastoid muscle and adjacent to C6 lateral tubercle (aka Chassaignac's tubercle) → same level as cricothyroid cartilage
- Under ultrasound visualization, brachial plexus looks like “stoplight” with three circles corresponding to superior, middle and inferior trunks (may be difficult to visualize with thicker muscle)
- Question: With an interscalene brachial plexus block:
 - A) more local anesthetic drug is required than for axillary block
 - B) the biceps and brachialis muscles are blocked last
 - C) the intercostal brachial nerve is usually blocked → this is a cutaneous nerve that innervates upper medial arm and must be supplemented with any brachial plexus block
 - D) the lateral antebrachial cutaneous nerve is usually spared → from posterior cord and receives innervation from all three trunks
 - **E) the ulnar is most likely spared** → ulnar distribution is most commonly missed because most inferior
- Question: the last muscle to be affected by an interscalene brachial plexus block is:
 - A) brachialis → supplied by musculocutaneous nerve which is missed with axillary block
 - B) brachioradialis → supplied by radial nerve
 - C) biceps → musculocutaneous nerve
 - D) flexor carpi radialis → median nerve
 - **E) interosseous of hand** → exception of 1st and 2nd lumbricals, rest are innervated by deep branch of ulnar nerve
- Question: which of the following statements regarding innervation of the upper extremity is true?
 - A) blockade of radial nerve decreases the patients ability to spread the fingers apart → palmar interosseous muscles responsible for this; innervated by ulnar nerve
 - B) the brachial plexus receives preganglionic sympathetic fibers arising from C5 to T2 → it receives it from T1 to L2/3
 - C) interscalene injection of the brachial plexus at C6 is likely to spare the axillary nerve → axillary nerve comes from posterior cord which is C5 to C8/T1
 - **D) the musculocutaneous nerve receives contributions from C5 and C6**

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- E) the vertebral artery lies posterior to the nerve roots of the brachial plexus
 - Question: which surface area of the upper extremity is most likely to be not anesthetized by an interscalene block?
 - **A) hypothenar eminence** → on ulnar side
 - B) thenar eminence
 - C) dorsal lateral surface of hand
 - D) lateral aspect of forearm
 - E) lateral surface of upper arm
 - Question: when performing an interscalene nerve block with a peripheral nerve stimulator, you note diaphragmatic movement, you should now:
 - A) inject the local anesthetic as the needle is in the appropriate location
 - B) redirect the needle in an anterior direction
 - **C) redirect the needle in a posterior direction** → phrenic nerve runs anterior to the brachial plexus
 - D) advance the needle about 0.5cm and inject

Complications

- Complications (bad, but rare): intervascular injection, pneumothorax, hypotension from sympathetic blockade, intrathecal injection via intervertebral foramen causing high spinal, hematoma if hit vertebral artery, Bezold–Jarisch reflex (severe hypotension and bradycardia)
 - Side effects (common):
 - Ipsilateral phrenic nerve blockade causing diaphragmatic paralysis corresponding with 25% reduction of pulmonary function
 - Horner’s syndrome due to spread → ptosis, anhidrosis, miosis
 - Hoarse voice due to spread to recurrent laryngeal nerve
 - Question: which of the following statements concerning interscalene brachial plexus block is true:
 - A) the three trunks of the plexus are in the same facial plane as the internal jugular vein
 - B) distal spread of anesthetic past humeral head is accelerated by adduction of the arm
 - **C) anesthetic solution can spread up the facial sheet to involve the stellate ganglion**
 - D) ipsilateral diaphragmatic paralysis results from epidural spread → results from phrenic nerve, not epidural spread
 - E) rich vascularity from the sheath promotes rapid vascular uptake of anesthetic
 - Question: an obese 75 y.o. woman is scheduled for ORIF of the arm. Thirty minutes after successful interscalene block using 20mL of 2% lidocaine, she becomes dyspneic. The dyspnea is most likely related to:
 - A) cervical epidural block
 - B) cervical post sympathetic block with bronchospasm
 - C) chylothorax
 - **D) elevation of the left hemidiaphragm**
 - E) recurrent laryngeal nerve block
 - Question: a 30 y.o. woman has difficulty talking 15 minutes after initiation of interscalene block for closed reduction of dislocated shoulder. Most likely cause is:
 - A) cervical sympathetic block
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- B) delayed systemic toxic reaction
 - C) phrenic nerve paralysis
 - D) pneumothorax
 - **E) recurrent laryngeal nerve block**
- Question: which of the following is the most important disadvantage of interscalene brachial plexus block compared with other approaches?
- A) large volumes of local anesthetics are required
 - **B) frequent sparing of the ulnar nerve**
 - C) frequent sparing of the musculocutaneous nerve
 - D) high incidence of pneumothorax
- Question: during the placement of an interscalene block, the patient becomes hypotensive, bradycardic, apneic, and cyanotic. The most likely cause is:
- Answer: total spinal in subarachnoid space

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Notes by [April Liu](#)