

Neuromuscular blockers

Paralytic agents that act at the nicotinic receptor of the neuromuscular junction, used only where airway control and ventilation are already secured, because the drugs paralyse the muscles of breathing along with every other skeletal muscle.

Online guide: <https://www.medguideglobal.com/pharmacology/analgesia-and-anaesthesia/neuromuscular-blockers>

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Quick revision

One Liner: Neuromuscular blockers act at the nicotinic acetylcholine receptor of the neuromuscular junction - either by depolarising it persistently (succinylcholine) or by competitively blocking it (rocuronium and relatives) - and must never be given without airway control and ventilation already in place.

1. Depolarising agents (succinylcholine) bind the nicotinic receptor and cause sustained depolarisation, producing brief fasciculations followed by paralysis as sodium channels become refractory. [1]
2. Nondepolarising agents (rocuronium, vecuronium, atracurium, cisatracurium, pancuronium, mivacurium) competitively block acetylcholine from binding the receptor without activating it, producing paralysis without preceding fasciculation. [1]
3. Succinylcholine carries an FDA boxed warning for rare but fatal hyperkalaemic cardiac arrest in apparently healthy paediatric patients, often later found to have an undiagnosed skeletal muscle myopathy such as Duchenne muscular dystrophy. [2]
4. Succinylcholine is contraindicated after the acute phase of major burns, multiple trauma, extensive denervation, or upper motor neuron injury, because it can cause severe, potentially fatal hyperkalaemia in these settings. [2]
5. Succinylcholine can trigger malignant hyperthermia, and that risk rises with concomitant volatile anaesthetic administration. [2]
6. Atracurium and mivacurium can cause histamine release, producing bronchospasm, hypotension and tachycardia. [1]
7. Reversal: sugammadex encapsulates aminosteroid agents (rocuronium, vecuronium) for rapid reversal; neostigmine inhibits acetylcholinesterase and requires co-administration of an anticholinergic to offset muscarinic side effects. [1]
8. Neuromuscular blocking agents must never be given without personnel and facilities for resuscitation and life support, and an antagonist should be readily available. [1]

Overview

1. Neuromuscular blocking agents act at the postsynaptic nicotinic acetylcholine receptors of the neuromuscular junction, producing complete skeletal muscle paralysis. They are frequently used in anaesthesia to facilitate endotracheal intubation, optimise surgical conditions, and assist mechanical ventilation in patients with reduced lung compliance - always alongside general anaesthesia or deep sedation, since paralysis carries no analgesic or amnestic effect of its own. [1]
2. The class divides mechanistically into depolarising and nondepolarising agents. Succinylcholine, the only depolarising agent in routine use, binds the receptor and causes sustained depolarisation - producing brief fasciculations before the membrane becomes refractory and paralysis sets in, with an onset of about 1 minute and duration of about 6 minutes. Nondepolarising agents such as rocuronium, vecuronium, atracurium, cisatracurium, pancuronium and mivacurium instead compete with acetylcholine for the receptor without ever activating it, producing paralysis without preceding fasciculation and lasting considerably longer. [1]
3. Succinylcholine carries the class's most serious labelled risks: an FDA boxed warning for rare but fatal paediatric hyperkalaemic cardiac arrest, contraindication after the acute phase of major burns, trauma, denervation or upper

motor neuron injury because of severe hyperkalaemia risk, and association with malignant hyperthermia. Nondepolarising agents carry their own risks - notably histamine release with atracurium and mivacurium - and are reversed differently: sugammadex for the aminosteroid agents, neostigmine (with an anticholinergic) more broadly. [2][1]

Classification and drug examples

Intro: Best learned by mechanism first, then by chemical family within the nondepolarising group, since chemical family determines which reversal agent will actually work.

1. Name: Depolarising agents

1. Note: A single agent in routine clinical use, distinguished by rapid onset, short duration, and a distinct set of serious labelled risks.

Name: Succinylcholine

Suxamethonium

Route: IV/IM

Note: Onset about 1 minute, duration about 6 minutes; carries an FDA boxed warning for paediatric hyperkalaemic cardiac arrest and is a malignant hyperthermia trigger. [1][2] [1]

2. Name: Nondepolarising agents: aminosteroids

2. Note: Reversible with sugammadex, which physically encapsulates the drug molecule.

1. Name: Rocuronium

1. Route: IV

1. Note: A commonly used aminosteroid nondepolarising agent, reversible with sugammadex. [1]

2. Name: Vecuronium

2. Route: IV

2. Note: An aminosteroid nondepolarising agent, reversible with sugammadex or neostigmine. [1]

3. Name: Pancuronium

3. Route: IV

3. Note: A longer-acting aminosteroid agent. [1] [1]

3. Name: Nondepolarising agents: benzyloquinoliniums

3. Note: Not meaningfully reversed by sugammadex; neostigmine is used instead. Associated with histamine release.

1. Name: Atracurium

1. Route: IV

1. Note: Can cause histamine release with bronchospasm, hypotension and tachycardia. [1]

2. Name: Cisatracurium

2. Route: IV

2. Note: A stereoisomer of atracurium with less histamine release. [1]

3. Name: Mivacurium

3. Route: IV

3. Note: Shorter-acting; can also cause histamine release. [1] [1]

Mechanism of action

Summary: Neuromuscular blockers act at the postsynaptic nicotinic acetylcholine receptor of the neuromuscular junction. Succinylcholine causes sustained depolarisation and eventual receptor refractoriness; nondepolarising agents competitively occupy the receptor without activating it. Both routes end in the same clinical result - skeletal muscle paralysis - by different molecular means.

Target: Postsynaptic nicotinic acetylcholine receptor at the neuromuscular junction

Cidal Static: Not applicable - this class produces paralysis, not antimicrobial killing.

Kill Kinetics: Not applicable in the antimicrobial sense; onset and duration depend on the specific agent, dose and

route.

1. Step: 1

1. Title: Depolarising route: succinylcholine binds and activates the receptor

1. Detail: Succinylcholine adheres to the postsynaptic nicotinic receptor and, unlike acetylcholine, is not rapidly hydrolysed there, producing sustained depolarisation of the motor endplate. [1]

2. Step: 2

2. Title: Depolarising route: transient fasciculations, then refractory paralysis

2. Detail: Sustained depolarisation first causes brief, disorganised muscle fasciculations, then inactivates the adjacent voltage-gated sodium channels, leaving the membrane refractory and producing flaccid paralysis. [1]

3. Step: 3

3. Title: Nondepolarising route: competitive receptor occupancy

3. Detail: Nondepolarising agents compete with acetylcholine for the alpha subunit of the nicotinic receptor, occupying the binding site without activating the channel, so no depolarisation and no fasciculation occurs. [1]

4. Step: 4

4. Title: Sufficient receptor occupancy prevents muscle contraction

4. Detail: Once enough receptors are blocked, acetylcholine released from the presynaptic terminal cannot generate an adequate endplate potential, and skeletal muscle contraction fails. [1]

5. Step: 5

5. Title: Reversal restores normal transmission

5. Detail: Sugammadex physically encapsulates aminosteroid agents, removing them from the receptor; neostigmine instead raises synaptic acetylcholine by inhibiting acetylcholinesterase, allowing acetylcholine to outcompete the blocking drug at the receptor. [1]

Major clinical uses

1. Drug: Succinylcholine

1. Indication: Rapid-sequence induction and intubation, where fast onset and short duration are wanted

1. Line: first-line for rapid-sequence intubation in eligible patients

1. Note: Avoided beyond the acute phase of burns, trauma, denervation or upper motor neuron injury, and in malignant hyperthermia susceptibility. [2]

2. Drug: Rocuronium

2. Indication: Intubation and maintenance of surgical paralysis, including rapid-sequence induction when succinylcholine is contraindicated

2. Line: first-line nondepolarising agent in many settings

2. Note: Reversible with sugammadex for rapid recovery when needed. [1]

3. Drug: Cisatracurium

3. Indication: Maintenance of paralysis for mechanical ventilation, particularly in hepatic or renal impairment

3. Line: targeted

3. Note: Organ-independent (Hofmann) elimination makes it useful when hepatic/renal clearance of other agents is unreliable. [1]

4. Drug: Neuromuscular blockers as a class

4. Indication: Facilitating mechanical ventilation in patients with reduced lung compliance

4. Line: adjunct in intensive care

4. Note: Requires deep sedation/analgesia alongside paralysis, since paralysis provides neither. [1]

Pharmacokinetics

1. Drug: Succinylcholine

1. Route: IV/IM

1. Absorption: Not applicable (parenteral)
1. Csf Penetration: Not applicable - acts peripherally at the neuromuscular junction
1. Metabolism: Plasma pseudocholinesterase
1. Elimination: As metabolites
1. Half Life: Very short; onset approximately 1 minute, duration approximately 6 minutes
1. Dose Adjust: Prolonged block in pseudocholinesterase deficiency; avoid beyond acute phase of burns/trauma/denervation [1]
2. Drug: Rocuronium / vecuronium (aminosteroids)
2. Route: IV
2. Absorption: Not applicable (parenteral)
2. Csf Penetration: Not applicable - acts peripherally
2. Metabolism: Hepatic
2. Elimination: Hepatic/renal
2. Half Life: See prescribing reference
2. Dose Adjust: Hepatic/renal impairment prolongs effect; reversible with sugammadex or neostigmine [1]
3. Drug: Cisatracurium (benzylisoquinolinium)
3. Route: IV
3. Absorption: Not applicable (parenteral)
3. Csf Penetration: Not applicable - acts peripherally
3. Metabolism: Organ-independent Hofmann elimination and ester hydrolysis
3. Elimination: Not primarily dependent on hepatic or renal function
3. Half Life: See prescribing reference
3. Dose Adjust: Useful in hepatic/renal impairment because of organ-independent elimination [1]
1. This page gives no dose regimens by design. Dosing depends on the specific agent, indication, hepatic/renal function and depth of block required, and must come from a current prescribing reference and the treating clinician.
2. Neuromuscular blockers must be given only with personnel and equipment for airway management, ventilation and resuscitation already in place, and an antagonist should be readily available. [1]

Adverse effects

Effect: Prolonged block in susceptible patients

Detail: Pseudocholinesterase deficiency can prolong succinylcholine's effect well beyond the expected short duration. [1]

1. Effect: Hyperkalaemic cardiac arrest (succinylcholine, paediatric)
1. Detail: Rare but fatal cases of acute rhabdomyolysis with hyperkalaemia, ventricular dysrhythmias and cardiac arrest in apparently healthy children, frequently later explained by an undiagnosed skeletal muscle myopathy such as Duchenne muscular dystrophy.
1. Action: Immediate treatment for hyperkalaemia - IV calcium, bicarbonate, glucose with insulin, and hyperventilation - if cardiac arrest occurs within minutes of administration and is not explained by inadequate ventilation, oxygenation or anaesthetic overdose. [2]
2. Effect: Severe hyperkalaemia (succinylcholine, beyond the acute injury phase)
2. Detail: In burns, multiple trauma, extensive denervation or upper motor neuron injury beyond the acute phase, succinylcholine can cause severe hyperkalaemia potentially resulting in cardiac arrest.
2. Action: Avoid succinylcholine in this setting; use a nondepolarising agent instead. [2]
3. Effect: Malignant hyperthermia
3. Detail: Succinylcholine can trigger acute malignant hyperthermia, a potentially fatal hypermetabolic state, with risk increased by concomitant volatile anaesthetic use. The underlying defect is a hereditary abnormality of the skeletal muscle ryanodine receptor, which releases calcium uncontrollably once triggered.

- 3. Action: Stop the triggering agent, give IV dantrolene, and manage temperature, acidosis and hyperkalaemia. [2][3]
- 4. Effect: Histamine release (atracurium, mivacurium)
- 4. Detail: Can produce bronchospasm, hypotension and tachycardia.
- 4. Action: Monitor haemodynamics and consider a different agent in patients with reactive airway disease. [1]
- Drug: Succinylcholine
- Effect: Bradycardia, particularly in paediatric patients. [1]

Contraindications, precautions and interactions

- 1. Succinylcholine beyond the acute phase of major burns, multiple trauma, extensive denervation of skeletal muscle, or upper motor neuron injury. [2]
- 2. Succinylcholine in patients with known or strongly suspected malignant hyperthermia susceptibility. [2]
 - 1. Known or suspected pseudocholinesterase deficiency, which prolongs succinylcholine's effect. [1]
 - 2. Reactive airway disease, with atracurium or mivacurium, because of histamine release risk. [1]
 - 3. Hepatic or renal impairment, which can prolong the effect of aminosteroid nondepolarising agents. [1]
- 1. With: Volatile inhalational anaesthetics, with succinylcholine
 - 1. Effect: Increases the risk of triggering malignant hyperthermia. [2][3]
- 2. With: Other CNS/respiratory depressants
 - 2. Effect: Additive respiratory compromise, underscoring the requirement for airway control whenever these agents are used. [1]

Comparison tables

Title: Which reversal agent works for which drug

Caption: Reversal is not interchangeable across the class. A prescribing reference and the treating anaesthetist govern actual therapy.

- 1. Key: drug_family
 - 1. Label: Drug family
 - 1. Is Row Header: true
 - 2. Key: example
 - 2. Label: Example
 - 3. Key: reversal
 - 3. Label: Effective reversal
- 1. Drug Family: Depolarising
 - 1. Example: Succinylcholine
 - 1. Reversal: No specific antagonist; effect wears off with plasma pseudocholinesterase metabolism [1]
 - 2. Drug Family: Nondepolarising aminosteroid
 - 2. Example: Rocuronium, vecuronium
 - 2. Reversal: Sugammadex (encapsulation) or neostigmine (with anticholinergic) [1]
 - 3. Drug Family: Nondepolarising benzyliisoquinolinium
 - 3. Example: Atracurium, cisatracurium, mivacurium
 - 3. Reversal: Neostigmine (with anticholinergic); sugammadex is not effective [1]

High-yield exam pearls

- 1. Pearl: Fasciculations are the tell for depolarising block.
 - 1. Why: Succinylcholine's initial receptor activation causes transient, visible fasciculations before paralysis sets in, because it opens the channel before eventually leaving it refractory. Nondepolarising agents never cause this - they block the receptor without ever activating it. [1]

2. Pearl: Succinylcholine's paediatric boxed warning is about an unmasked disease, not a drug flaw.
2. Why: The fatal hyperkalaemic cardiac arrests reported in apparently healthy children were frequently later explained by an undiagnosed skeletal muscle myopathy such as Duchenne muscular dystrophy - the drug did not create a new risk so much as reveal an existing, silent one. [2]
3. Pearl: Burns and denervation turn succinylcholine from routine to dangerous, over time.
3. Why: The contraindication applies after the acute phase of injury - upregulated extrajunctional nicotinic receptors in burned, denervated or upper-motor-neuron-injured muscle can release massive potassium in response to succinylcholine, a risk that is not present immediately after the injury. [2]
4. Pearl: Sugammadex only works on one chemical family.
4. Why: Sugammadex encapsulates aminosteroid nondepolarising agents (rocuronium, vecuronium) directly; it has no meaningful effect on benzyisoquinolinium agents (atracurium, cisatracurium, mivacurium) or on succinylcholine, which is why neostigmine remains relevant for those agents. [1]
5. Pearl: Neostigmine never travels alone.
5. Why: Because neostigmine inhibits acetylcholinesterase everywhere, not just at the neuromuscular junction, it must be co-administered with an anticholinergic (such as glycopyrrolate) to prevent bradycardia and other muscarinic side effects. [1]

Common exam traps

1. Trap: "Succinylcholine is safe to use routinely in any burn patient because burns are treated urgently."
1. Correction: The contraindication specifically applies after the acute phase of a major burn injury, once extrajunctional receptor upregulation has had time to develop - this is a delayed risk window, not an immediate one, and it must still be respected once it applies. [2]
2. Trap: "Sugammadex reverses any nondepolarising neuromuscular blocker."
2. Correction: Sugammadex is selective for aminosteroid agents (rocuronium, vecuronium), which it physically encapsulates. Benzyisoquinolinium agents such as atracurium and mivacurium are not meaningfully reversed by sugammadex; neostigmine is used for those instead. [1]
3. Trap: "A neuromuscular blocker can be given whenever intubation is planned, before airway equipment is confirmed ready."
3. Correction: The essential safety rule is the reverse: these drugs are given only with personnel and facilities for resuscitation and life support already in place and an antagonist readily available, precisely because they paralyse the muscles of breathing along with everything else. [1]
4. Trap: "Fasciculations mean something has gone wrong with the drug."
4. Correction: Fasciculations are an expected, mechanistically predictable feature of depolarising block from succinylcholine's initial receptor activation, not a sign of drug error or malfunction. [1]

Self-test questions

1. Question: What distinguishes the mechanism of a depolarising neuromuscular blocker from a nondepolarising one?
1. Depolarising agents block the receptor without ever activating it; nondepolarising agents activate it repeatedly
2. A depolarising agent (succinylcholine) binds the nicotinic receptor and causes sustained depolarisation and eventual refractoriness; a nondepolarising agent competitively blocks acetylcholine from binding the receptor without activating it
3. Both classes work identically at the molecular level and differ only in duration
4. Depolarising agents act at the presynaptic membrane; nondepolarising agents act only postsynaptically
1. Explanation: Succinylcholine, the depolarising agent, binds the postsynaptic nicotinic receptor and causes sustained depolarisation, producing transient fasciculations followed by paralysis as sodium channels become refractory. Nondepolarising agents instead competitively occupy the receptor, preventing acetylcholine binding without ever activating the channel. [1]
2. Question: The succinylcholine boxed warning for pediatric hyperkalaemic cardiac arrest is most closely associated

with what underlying finding?

1. An unrelated cardiac arrhythmia present from birth
2. An undiagnosed skeletal muscle myopathy, such as Duchenne muscular dystrophy, in an apparently healthy child
3. A known allergy to eggs
4. Concurrent use of an ACE inhibitor

2. Explanation: Reports of ventricular dysrhythmias, cardiac arrest and death from acute rhabdomyolysis with hyperkalaemia after succinylcholine in apparently healthy paediatric patients were frequently later explained by an undiagnosed skeletal muscle myopathy such as Duchenne muscular dystrophy, whose clinical signs had not yet been recognised. [2]

3. Question: Why is succinylcholine contraindicated after the acute phase of a major burn injury?

1. It has no relevant risk in burns and the contraindication is outdated
2. Beyond the acute phase, upregulated extrajunctional receptors can respond to succinylcholine with severe, potentially fatal hyperkalaemia
3. Burn patients metabolise succinylcholine too slowly, prolonging paralysis only
4. Succinylcholine causes burns to worsen directly

3. Explanation: After the acute phase of major burns, multiple trauma, extensive denervation, or upper motor neuron injury, succinylcholine can cause severe hyperkalaemia through its action on upregulated extrajunctional nicotinic receptors, which can result in cardiac arrest - this is why the label specifically contraindicates it in that setting. [2]

4. Question: Which reversal agent is selective for aminosteroid nondepolarising blockers such as rocuronium and vecuronium?

1. Neostigmine
2. Sugammadex, which physically encapsulates aminosteroid agents
3. Naloxone
4. Dantrolene

4. Explanation: Sugammadex encapsulates aminosteroid nondepolarising agents (rocuronium, vecuronium), providing rapid, targeted reversal. It is not effective for benzylisoquinolinium agents such as atracurium, cisatracurium or mivacurium, for which neostigmine remains the relevant reversal option. [1]

5. Question: Why must neostigmine always be given together with an anticholinergic such as glycopyrrolate?

1. To speed up neostigmine's onset of action
2. Because neostigmine inhibits acetylcholinesterase systemically, not only at the neuromuscular junction, and would otherwise cause muscarinic side effects such as bradycardia
3. Because neostigmine alone has no effect on neuromuscular blockade
4. To prevent malignant hyperthermia

5. Explanation: Neostigmine's acetylcholinesterase inhibition is not confined to the neuromuscular junction. Left unopposed, it produces muscarinic effects such as bradycardia, so an anticholinergic is co-administered specifically to offset those effects while the nicotinic reversal at the neuromuscular junction is retained. [1]

6. Question: What is the essential safety requirement for administering any neuromuscular blocking agent?

1. The patient must be conscious and cooperative throughout
2. Personnel and facilities for resuscitation and life support must be available, with an antagonist readily accessible, because these drugs paralyse the muscles of respiration along with every other skeletal muscle
3. The drug must be given only by mouth to avoid overdose
4. No monitoring is required once the drug has been given

6. Explanation: Because neuromuscular blockers paralyse the muscles of breathing along with all other skeletal muscle, administration should never occur without personnel and facilities for resuscitation and life support, and a reversal agent readily available. [1]

Frequently asked questions

1. Question: Why can't neuromuscular blockers ever be given without securing the airway first?

1. Answer: These drugs paralyse every skeletal muscle, including the diaphragm and other muscles of breathing, and provide no sedation or pain relief on their own. That is why they are used only alongside general anaesthesia or deep sedation and only with airway management and ventilation equipment immediately available. [1]

2. Question: Why does succinylcholine carry a specific warning for children?

2. Answer: Rare but fatal cases of hyperkalaemic cardiac arrest have occurred in apparently healthy children given succinylcholine, and many were later found to have an undiagnosed skeletal muscle disease such as Duchenne muscular dystrophy. The warning reflects that succinylcholine can unmask a serious, previously silent condition. [2]

3. Question: Can sugammadex reverse any neuromuscular blocker?

3. Answer: No. Sugammadex works by physically encapsulating aminosteroid agents like rocuronium and vecuronium. It does not meaningfully reverse benzyisoquinolinium agents such as atracurium or mivacurium, or succinylcholine, which are managed differently. [1]

4. Question: What is malignant hyperthermia and how does it relate to succinylcholine?

4. Answer: Malignant hyperthermia is a potentially fatal hypermetabolic reaction in genetically susceptible patients, and succinylcholine is a recognised trigger, with risk rising further when a volatile anaesthetic is also used. It is treated as a medical emergency with intravenous dantrolene and supportive care. [2]

References

[1] Neuromuscular Blocking Agents (StatPearls). StatPearls Publishing / NCBI Bookshelf 2025.

<https://www.ncbi.nlm.nih.gov/books/NBK537168/>

[2] Succinylcholine Chloride Injection - FDA-approved label (DailyMed). DailyMed, U.S. National Library of Medicine 2026.

<https://dailymed.nlm.nih.gov/dailymed/lookup.cfm?setid=81412f5a-493a-276d-e053-2a91aa0a21df>

[3] Malignant Hyperthermia (StatPearls). StatPearls Publishing / NCBI Bookshelf 2025.

<https://www.ncbi.nlm.nih.gov/books/NBK430828/>

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