

Succinylcholine in modern anesthesia

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LEARNING OBJECTIVES

The purpose of this issue is to:

- Present the main complications of succinylcholine and their underlying mechanisms of action;
- Determine which complications are diminished or eliminated by precurarization;
- Summarize the pharmacological characteristics of succinylcholine;
- Review the effects of precurarization on fasciculations and myalgia;
- Make recommendations on the type of neuromuscular blocking agent to be used for precurarization, the dose, and the time of injection.

Since entering the market over 50 years ago, succinylcholine has continued to fuel controversy, but its usefulness was never doubted. Indeed, it is the only neuromuscular blocking agent with a rapid onset and a short duration, which makes it particularly appropriate for use in facilitating tracheal intubation. However, succinylcholine has frequent side effects. Most of the adverse effects are minor, but there are situations where succinylcholine is contraindicated because of serious side effects.

Efforts have been made, with success, to limit the impact of minor effects such as fasciculations and myalgia, notably by precurarization. The major side effects, which involve severe hyperkalemia and cardiac arrest, cannot be avoided with certainty unless succinylcholine is not used. Replacement solutions, at least partial ones, have appeared over the years. Nondepolarizing short- to intermediate-acting neuromuscular blocking agents are now available and have replaced succinylcholine for some of its indications. The judicious use of short-onset opioids make it possible to decrease the dose of neuromuscular blocking agents used for intubation, and the laryngeal airway mask has also decreased the number of cases where tracheal intubation is necessary.

Even though alternatives are more plentiful than they were, succinylcholine remains the neuromuscular blocking agent of choice in certain situations, particularly in emergency cases, and is still indicated in many others, notably in short-duration elective surgery. The purpose of this article is to summarize the pharmacological properties of succinylcholine, as well as its side effects and contraindications. The different ways of using succinylcholine will be reviewed with an emphasis on the optimal use of precurarization.

MECHANISMS OF ACTION (TABLE 1)

Succinylcholine's mechanisms of action are still poorly understood. These mechanisms depend on the species studied, and in humans, there are many sites and many distinct mechanisms. In order to understand the principal effects of succinylcholine and its most relevant side effects, 3 mechanisms and 4 distinct sites must be considered. First, it should be mentioned that succinylcholine is a cholinergic receptor agonist, and these receptors are found at the neuromuscular junction. Its effect is similar to that of acetylcholine, that is, it induces the opening of the receptor channel, allowing the ions to pass freely, thereby depolarizing the membrane in which the receptor is found. It is important to note, however, that succinylcholine, unlike acetylcholine, is not hydrolyzed by the acetylcholinesterase present in the synaptic cleft. Succinylcholine remains bound to the receptor for a relatively long period of time, this causes desensitization of the receptor, and therefore, decreased sensitivity to acetylcholine. Furthermore, a depolarization lasting more than a few milliseconds causes inactivation of the sodium channels concentrated at the endplate. This inactivation prevents the spread of an action potential in the muscle fibre, and no contraction occurs. However, sustained depolarization may produce a contracture, that is, increased tension in the muscle fibre. The effect of succinylcholine is therefore a complex interaction between activation on the one hand, and inhibition on the other, caused by the desensitization of the receptor and the inactivation of the sodium channels. These interactions take place at four

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TABLE 1: Succinylcholine: mechanisms of Action

Manifestations	Mechanism	Site	Effect of small doses of nondepolarizing drug
Fasciculations	Depolarization	Presynaptic	Reduces incidence and severity
Myalgia	Fasciculations	Presynaptic	Reduces incidence and severity
Myoglobinemia Increase in creatinine kinase	Muscle lesion	Presynaptic	Reduces increase
Increase in O ₂ consumption and CO ₂ production	Muscle contractions	Presynaptic	Eliminates increase
Release of catecholamines	Muscle contractions	Presynaptic	Eliminates increase
Increase in intra-gastric pressure	Muscle contractions	Presynaptic	Reduces increase
Increase in intraocular pressure	Contractures	Postsynaptic receptors	Little or no effect
Neuromuscular block	Desensitization, inactivation of sodium channels	Postsynaptic receptors	Reduces block. Succinylcholine dose must be increased
Bradycardia	Activation	Vagus nerve	No effect
Hyperkalemia	Opening of channels	Postsynaptic and extra-junctional receptors	Little or no effect
Myoclonic response Masseter spasm	Contractures	Postsynaptic and (?) extrajunctional receptors	Little or no effect
Rhabdomyolysis	Membrane fragility	Membrane of muscle fibre	No proven effect. May reduce damage to fragile membranes.

sites: (1) presynaptic, (2) postsynaptic, (3) on the extra-junctional receptors, (4) on the muscle membrane.

Presynaptic effects

The nerve fibre terminals contain cholinergic receptors that control neurotransmission by adjusting the number of available acetylcholine vesicles. These receptors are activated by succinylcholine and blocked by small doses of nondepolarizing neuromuscular blocking agent.¹ Succinylcholine depolarizes these receptors enough to generate action potentials that spread to all the muscle fibres innervated by the nerve involved. This is the accepted mechanism for explaining involuntary contractions, called fasciculations, that occur a few seconds after the injection of succinylcholine. These fasciculations, even if they do not last long, are sufficiently severe to cause muscle damage. This phenomenon also produces biochemical manifestations (increases in myoglobin and creatinine kinase) and clinical ones (myalgia or muscle pain in the 24 to 48 hours after the administration of succinylcholine).² Furthermore, the intensity of the contractions is enough to increase oxygen consumption and CO₂ production. The increase in pCO₂ may lead to vasodilation in the brain, and thus an increase in intracranial pressure in subjects at risk. The fasciculations are also accompanied by a release of catecholamines, with tachycardia and hypertension.³ All of these manifestations are eliminated or greatly diminished by the administration of a small dose of nondepolarizing neuromuscular blocking agent.

Postsynaptic effects

At the postsynaptic level, the principal pharmacological effect of succinylcholine occurs at the endplate where succinylcholine depolarizes the membrane enough to desensitize the receptor or inactivate the

sodium channels (present there in large amounts), or both. Thus, there is a block in neurotransmission, and therefore paralysis, and its intensity depends directly on the number of available receptors. Succinylcholine will be less effective if the number of receptors is reduced, for example, in cases of myasthenia gravis, or when a nondepolarizing drug has already been administered. On the other hand, the initial effect of succinylcholine is to open the cholinergic receptor channels, thereby allowing potassium to leave the muscle fibre. Therefore, succinylcholine causes hyperkalemia, but the increase in extracellular potassium is ordinarily modest, usually less than 0.5 mEq/L.²

Effects on extrajunctional receptors

A normal muscle fibre has a receptor density a thousand times greater at the endplate than elsewhere on the muscle membrane (10,000/μm² compared with 10/μm²). Yet, the endplate represents only a small part of the surface of the muscle fibre membrane. Therefore, the total number of extrajunctional receptors is comparable, and possibly higher than the number of junctional receptors, even in normal individuals. Hence, the extrajunctional receptors are a target for succinylcholine, and the magnitude of the hyperkalemia depends directly on the number of these receptors. In cases of denervation or lack of endplate activity, these extrajunctional receptors proliferate and there is a possibility of severe hyperkalemia. But the extrajunctional receptors do not contribute to the paralyzing effect of succinylcholine, since they are not involved in the transmission between nerve and muscle.

The hyperkalemia is counteracted only by doses of nondepolarizing neuromuscular blocking drugs large enough to prevent the access of succinylcholine to the receptor. Small doses are insufficient. Obviously,

succinylcholine is not indicated in patients already paralyzed with a nondepolarizing agent.

Muscle damage

Succinylcholine may cause muscle damage, either because of the fasciculations, with the associated uncontrolled contractions, or because of the contractures it causes. A contracture is an increase of tension in the depolarized muscle fibres without neuromuscular transmission. Certain muscles, especially the masseter, and certain individuals, particularly those suffering from myotonia, are more susceptible to contractures. In normal subjects, there are no serious consequences to muscle damage. However, some individuals have fragile muscle membranes and are subject to rhabdomyolysis, usually accompanied by hyperkalemia. The probable mechanism of this hyperkalemia is the appearance of breaks in the membrane, with a loss of intracellular potassium. Patients with muscular dystrophies or those receiving long-term corticotherapy are particularly subject to this type of complication. A thorough review of the literature led to the conclusion that patients with succinylcholine-induced rhabdomyolysis have less chance of being resuscitated following asystole than subjects with a pathology compatible with receptor proliferation (trauma to the spinal cord, burns) and who also suffer cardiac arrest following succinylcholine.⁴ This difference in prognosis is explained by the more permanent nature of hyperkalemia produced by membrane fragility.

PHARMACOLOGY

The succinylcholine dose that produces, on average, a 95% decrease in twitch height of the thumb (effective dose₉₅ or ED₉₅) is 0.3 to 0.5 mg/kg for an adult.^{5,6} It is approximately 0.5 mg/kg for children, but for infants less than a year old, it is 0.7-0.8 mg/kg. In newborns, this value is 0.6 mg/kg and could be even lower in the premature infant.⁷ The intubation doses are 2 to 3 times greater than the ED₉₅, therefore, approximately 1 mg/kg for an adult. A dose of 1 mg/kg has an onset time of 1 to 1.5 min, and a duration of action until twitch recovery of the thumb, of 8 to 12 minutes in the adult; it is shorter in the child. Duration is only 5 minutes at the diaphragm; in most cases, spontaneous breathing resumes before the reserve of oxygen accumulated during pre-oxygenation has been exhausted. Theoretically, an adult with a functional residual capacity of 2 L and an oxygen consumption of 250 mL has a reserve of 8 minutes (2000 mL/250 mL per minute). However, pre-oxygenation is not always 100% effective, the functional residual capacity is sometimes reduced and the effect of succinylcholine in all patients is not uniform. Nevertheless, succinylcholine is the only neuromuscular blocking agent where return of spontaneous breathing is likely to take place before hypoxia occurs, if intubation is unsuccessful.⁸

The block produced by succinylcholine is depolarizing in nature, that is, there is no fade or decrease in the force of the contraction following a train-of-four or tetanic stimulation. Thus, one can use a frequency of 1 Hz to monitor the onset of block. Furthermore, since onset is rapid and relatively uniform from one muscle to another, twitch depression of the thumb may be

considered good evidence for paralysis of the other muscles. The prolonged administration of succinylcholine is associated with a change in the nature of the block. After 45-60 minutes of continuous infusion, the succinylcholine-induced block becomes similar to that of a nondepolarizing neuromuscular blocking agent, and is usually called a phase-II block. In the clinical setting, this type of block is rarely seen since the practice of giving succinylcholine infusions has been almost entirely abandoned.

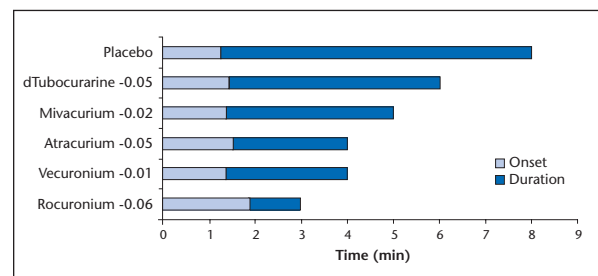
Until recently, it was known that succinylcholine was rapidly metabolized by plasma cholinesterase, but the pharmacokinetics had never been measured. Recent data show that the mean elimination half-life of succinylcholine in normal subjects is only 47 seconds.⁹ Thanks to molecular biology, many types of plasma cholinesterase have been identified and most of these have limited activity in terms of hydrolyzing succinylcholine. These atypical cholinesterases are seen at a frequency of only 1:2500. After a normal dose of succinylcholine, these patients' lungs must be ventilated mechanically for 3 to 4 hours.

The prior administration of nondepolarizing agents decreases the effectiveness of succinylcholine (Figure 1). A dose representing one-tenth of the ED₉₅ (ie, d-tubocurarine 0.05 mg/kg) pushes the dose-response curve of succinylcholine to the right, doubling its ED₉₅ from 0.5 mg/kg to 1.0 mg/kg.⁶

Side effects

The undesirable effects of succinylcholine on muscle are more easily understood if their specific mechanism of action is considered. The presynaptic activity of succinylcholine produces fasciculations. These involuntary contractions produce muscle damage, manifested as myalgia,¹⁰ myoglobinemia, an increase in creatinine kinase, an increase in oxygen consumption, a rise in CO₂ production leading to an increase in intracranial pressure, the release of catecholamines with repercussions on the heart rate and blood pressure, and an increase in intragastric pressure. All of these effects may be counteracted by the presynaptic effect of small doses of nondepolarizing blocking agents. Another important category of side effects has an origin in the junctional and extrajunctional postsynaptic receptors. Hyperkalemia is one major manifestation. Succinylcholine also produces contractures in any muscle, especially the

FIGURE 1. The administration of nondepolarizing blocking agent decreases the effectiveness of a succinylcholine dose of 1.5 mg/kg, administered 3 min after, by prolonging the onset of action and decreasing the duration. Doses in mg/kg. From Martin et al.¹⁴



masseter; it increases intraocular pressure; it may help to precipitate malignant hyperthermia in susceptible individuals, and it causes severe hyperkalemia in cases involving burns or denervation. These effects do not respond to small doses of nondepolarizing blocking agents. Finally, there are the cardiovascular and pulmonary effects of succinylcholine that, in practice, are less important than would be expected from the cholinergic-receptor agonistic properties of succinylcholine. Bradycardia is frequent in children, but rare in adults. However, severe bradycardia is common after a second dose of succinylcholine, regardless of the patient's age. Also, an increased incidence of bronchospasm could be expected following the administration of succinylcholine, due to its cholinergic effects, but frequently, this effect does not seem to occur.

CONTRAINDICATIONS

The cases where succinylcholine is contraindicated or must be administered cautiously are many, but fortunately they are not common. Absolute contraindications are damage to the spinal cord, extensive burns, malignant hyperthermia, previous anaphylactic reactions to the drug, and muscular dystrophies. The presence of atypical cholinesterases is not a contraindication per se, but succinylcholine presents no advantage in these cases. Nevertheless, the product must be administered cautiously or alternatives must be strongly considered in cases of severe trauma, cerebrovascular accident, neurological disease originating in the central nervous system, sepsis, hyperkalemia, malnutrition, prolonged immobilization, and/or prolonged corticotherapy.

In the 1990s, a controversy arose over the administration of succinylcholine to children going for elective surgery, following reported cases of cardiac arrest, of which some were fatal. In most cases, it seems that these accidents occurred in children with muscular dystrophy not yet diagnosed. Usually, it was found that a halogenated agent, most often halothane, had been given with succinylcholine.⁴ Fortunately, the incidence of these events is rare, but the risk must be taken into account, nevertheless. Succinylcholine is still recommended for children in emergency situations, especially if intravenous agents are used for induction.

PRECURARIZATION

The method of choice for decreasing the severity and incidence of fasciculations is still the administration of a small dose of a nondepolarizing agent before giving succinylcholine. Even though some studies have not managed to prove the effectiveness of this technique against myalgia, a meta-analysis has gathered sufficient data to convince the skeptics that at least d-tubocurarine, gallamine, pancuronium, and atracurium are effective in this respect.¹⁰ It is more difficult to demonstrate a pretreatment effect on myalgia than it is on fasciculations, because pain is a subjective symptom and involves several confounding factors. Fasciculations

do not occur unless a person has received succinylcholine. However, 15% to 20% of subjects will complain of myalgia, even if they have not been given succinylcholine.¹¹

The drug most widely used to prevent fasciculations and myalgia was, until recently, d-tubocurarine. Its effectiveness, in doses of 0.05 mg/kg or 3 mg for an adult of average weight, 3 minutes before the administration of succinylcholine, is unquestioned. Unfortunately, d-tubocurarine is no longer available in most hospitals, and a safe and effective alternative must be found. All of the nondepolarizing drugs have been studied with respect to their effectiveness against fasciculation and myalgia. The accepted criteria for assessing the safety and effectiveness of a product are: (1) the precurarizing dose must be small enough not to cause any unpleasant symptoms of muscle weakness in almost all patients; (2) it must prevent fasciculations; (3) it must prevent myalgia; and (4) the effectiveness of succinylcholine with respect to its onset, intensity of block, and duration of action must be maintained.

Dose of nondepolarizing agent

In the surge of enthusiasm that accompanied the introduction of several nondepolarizing agents on the market in the last 30 years, there was little concern about using doses that were equivalent to d-tubocurarine, that is, 3 mg or 0.05 mg/kg, which is one-tenth of the ED₉₅. The ED₉₅ of d-tubocurarine is 0.5 mg/kg. The ED₉₅ and the intubation dose were confused. A habit developed of using higher and higher doses of nondepolarizing agents for intubation because the new compounds were short-acting and had no cardiovascular effects. Thus, the recommended precurarization doses were unjustifiably inflated (Table 2). In fact, the ideal dose, one that produces no symptoms in the conscious patient, has nothing to do with the dose used for intubation. It depends on the potency of the agent, hence on the ED₉₅. An elegant study supported the wisdom of recommendations to limit the precurarizing dose to one-tenth of the ED₉₅; it predicted that certain patients would experience unpleasant symptoms if higher doses were used.¹² In fact, it was discovered that muscle weakness, particularly difficulty in swallowing, occurred following the administration of pancuronium 1 mg (0.2 x ED₉₅), atracurium 0.05 mg/kg (0.2 x ED₉₅),¹³ vecuronium 0.01 mg/kg (0.2 x ED₉₅; Figure 2), mivacurium 0.02 mg/kg (0.3 x ED₉₅),¹⁴ or rocuronium 0.06 mg/kg (0.2 x ED₉₅).² A recent study recommended that the precurarization dose not be administered, because 80% of patients receiving rocuronium in doses of 0.06 mg/kg presented with symptoms of muscle weakness (Figure 3).¹⁵ It would have been more logical to recommend a decrease in the dose of rocuronium.

Which nondepolarizing drug for precurarization?

The meta-analysis published in 1990 on the then available nondepolarizing agents demonstrated the effectiveness of d-tubocurarine, gallamine, atracurium, and pancuronium in preventing myalgia.¹⁰

TABLE 2: Precurarization doses

Drug	Recommended dose* (mg/kg)	Doses used† (mg/kg)	Doses where symptoms appear‡ (mg/kg)	Effectiveness
d-Tubocurarine	0.05	0.04-0.06		+++
Gallamine	0.2	0.15-0.25		+++
Pancuronium	0.007	0.01-0.015	0.015	++
Vecuronium	0.005	0.007-0.015	0.01	+
Atracurium	0.025	0.05-0.07	0.05	++
Mivacurium	0.01	0.02	0.02	+
Cisatracurium	0.005	0.01-0.02	0.01	+
Rocuronium	0.03-0.04	0.03-0.1	0.06	+++

* Is equal to 0.1 x ED95 of the product

† Doses that have been the subject of published studies.

‡ Doses for which symptoms of muscle weakness have already been described. The true threshold is probably lower than the value cited.

Vecuronium did not prove to be effective beyond all doubt. Given the above discussion concerning doses, the doses of atracurium and pancuronium used in most of the studies were too high. Since then, cisatracurium, mivacurium, and rocuronium have been studied, all three in doses of up to 0.2, even 0.3 times the ED₉₅. Of these, only rocuronium was not shown to be less effective than d-tubocurarine.¹⁴ It seems that the logic suggesting that the short-acting agents are the most suitable, is proven by the facts. Therefore, among the drugs now available, rocuronium and, to a lesser extent, atracurium are the best candidates. The usual dose of atracurium given during the studies was 0.05 mg/kg, and, therefore, too high. A more appropriate dose (ie, 0.025 mg/kg), was the subject of another study and proved to be effective. As for rocuronium, most of the studies were conducted using doses that were too high (0.06 to 0.1 mg/kg) or borderline (0.05 mg/kg). All these studies demonstrated an effect on the incidence and severity of fasciculations, and many demonstrated a decrease in myalgia.¹⁶ Only one study was conducted on lower doses (0.03 mg/kg), and these proved to be effective,

FIGURE 2. Function of the geniohyoid, a muscle of the upper airway, 3 and 6 minutes after injection of vecuronium, 0.015 mg/kg, (typically 1 mg) in conscious adults. Each line represents a patient. The patient who had a decrease to zero needed emergency intubation. From d'Honneur et al.¹³

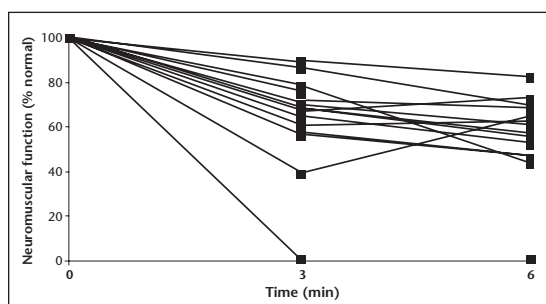
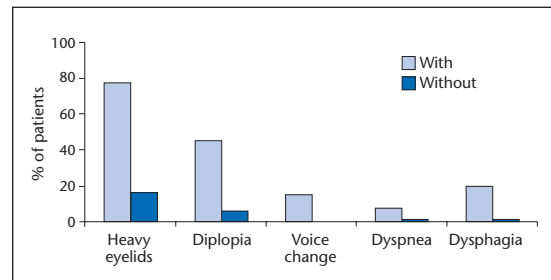


FIGURE 3: Percentage of patients with symptoms of muscle weakness 4 minutes after administration of rocuronium, 0.06 mg/kg, (with) or a placebo (without).



at least in reducing the incidence of fasciculations (Figure 4).¹⁷

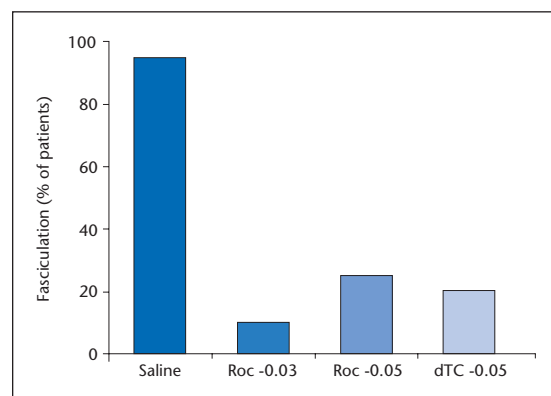
What interval?

The time interval between precurarization and the administration of succinylcholine is usually about 3 minutes. The effectiveness of the precurarization increases over time, but in practice, more than 3 minutes could be too constraining. With the use of rocuronium, 1.5 minutes could be sufficient.¹⁸

What dose of succinylcholine?

It has long been recognized that the dose of succinylcholine must be increased if given after a nondepolarizing agent. The recommended dose of 1.5 mg/kg is based on old studies where a similar duration of action was noted for succinylcholine (1.0 mg/kg) alone and succinylcholine (1.5 mg/kg) preceded by d-tubocurarine (3 mg). More recent studies have demonstrated that the block was not as deep when succinylcholine in doses of 1.5 mg/kg was preceded by precurarization (Figure 1),¹⁴ and that the duration of action of a dose of 2 mg/kg, with precurarization, was the

FIGURE 4. Incidence of fasciculations observed in patients who did not receive precurarization (saline), or received rocuronium or d-tubocurarine in the indicated doses, (mg/kg). The succinylcholine dose was 1.5 mg/kg. From Harvey et al.¹⁷



same as that of a dose of 1 mg/kg, without precurarization.¹⁸ After precurarization, a dose of 2 mg/kg of succinylcholine is recommended, since a lower dose may be insufficient.

Other pretreatments

Among the numerous medications tested to decrease the incidence of fasciculations and myalgia, the only one that really stands out is lidocaine, in doses of 1.5 mg/kg. Lidocaine has little effect on fasciculations, but it reduces the incidence of myalgia. Combined with a nondepolarizing agent, lidocaine helps decrease the incidence of myalgia to an extent comparable to that reported for nondepolarizing agents.^{17,19}

CONCLUSION

Despite the explosion of new techniques and medications in anesthesia, the use of succinylcholine is indicated in emergency situations (full stomach) and for elective surgery where brief paralysis is necessary. Precurarization is a technique that diminishes certain side effects of succinylcholine, but not all. Among the nondepolarizing agents now available, rocuronium (0.03-0.04 mg/kg) administered 2 to 3 minutes before succinylcholine, and atracurium (0.025 mg/kg), are more effective than the other drugs. Higher doses can cause symptoms of neuromuscular weakness in the conscious patient. When used following precurarization, the intubation dose of succinylcholine must be at least 1.5 mg/kg, preferably 2 mg/kg.

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