

Muscle Relaxants

[MB01](#) [Mar96] [Jul97] With regard to tetanic stimulation by a nerve stimulator:

- A. Used to determine residual curarisation **maybe**
- B. Degree of fade is independent of stimulus duration
- C. Degree of fade is dependent on stimulus intensity
- D. Used to check depth of anaesthesia

[MB02](#) [Mar96] [Apr01] [Hyperkalaemia](#) with [suxamethonium](#) is associated with:

- A. Abdominal infection
- B. Parkinson's disease
- C. Meningomyelocele
- D. Cerebral palsy
- E. Myotonic dystrophy

[MB03](#) [Mar96] [Jul96] [Jul97] [Mar98] [Mar99] [Jul99] [Feb00] Which of the following is NOT metabolised by plasma cholinesterase?

- A. [Procaine](#)
- B. Cocaine **although major metabolic process is hepatic cholinesterase**
- C. [Dibucaine](#)
- D. [Suxamethonium](#)
- E. Esmolol
- F. [Mivacurium](#)

[MB03b](#) [Mar98] [Apr01] Which of the following is metabolised by plasma cholinesterase?

- A. [Remifentanyl](#)
- B. Procaine
- C. [Esmolol](#)
- D. ?

E. All of the above

[MB03c](#) [Jul98] [Feb00] Esterases metabolise all EXCEPT:

- A. [Remifentanil](#)
- B. [Dibucaine](#)
- C. [Pyridostigmine](#) - metab by choline-esterases
- D. ?

[MB03d](#) [Feb04] Which drug has a significantly prolonged duration of action in plasma cholinesterase deficiency?

- A. [Remifentanil](#)
- B. [Procaine](#)
- C. [Mivacurium](#)
- D. [Rocuronium](#)
- E. [Cocaine](#)

[MB04](#) [Mar96] [Jul02] The action of nondepolarising neuromuscular blocking agents is PROLONGED by:

- A. [Respiratory acidosis](#)
- B. Increased temperature
- C. Increased calcium
- D. Increased potassium
- E. Decreased magnesium

[MB05](#) [Mar96] Agents prolonging nondepolarising NMBA by desensitising the post-junctional membrane :

- A. [Phenytoin](#)
- B. [Halothane](#)
- C. [Lignocaine](#)
- D. [Verapamil](#) maybe??

[MB06](#) [Mar96] [Jul98] Which drugs (?competitively) inhibit acetylcholinesterase?

- A. [Neostigmine](#)
- B. [Pyridostigmine](#)
- C. [Physostigmine](#)
- D. [Edrophonium](#)
- E. All of the above

[MB06b](#) [Jul00] [Apr01] The activity of plasma cholinesterase is decreased by the following drugs except:

- A. [Neostigmine](#)
- B. Organophosphates
- C. [THA](#) = tacrine = a weak acetylcholinesterase inhibitor
- D. [Metoclopramide](#)
- E. [Cimetidine](#)

- Severe hepatic disease
- Atypical plasma cholinesterase (dibucaine number 20 greatly prolongs the action of suxamethonium)
- Drugs
 - Neostigmine
 - Organophosphates
 - Chemotherapeutics - Nitrogen mustard and cyclophosphamide
 - Metoclopramide
- High oestrogen - Parturients at term

Increased plasma cholinesterase activity:

- Obesity

Resistance to suxamethonium:

- Myasthenia gravis
- Juvenile hyaline fibromatosis

[MB06c](#) [Jul04] Which decrease plasmacholinesterase activity? (remembered options from 2 questions)

- A. Hepatic disease

B. [Cyclophosphamide](#)

C. Six weeks post partum ??

D. Hyperthyroidism ??

E. Obesity - ↑ in fat people

F. Cytotoxic drugs

G. Pregnancy

E. Dibucaine number of 20

[MB07](#) [Mar97] [Jul98] [Jul99] [Feb00] [Apr01] Regarding [vecuronium](#):

A. It accumulates in renal failure - vec & roc both accumulate. atrac will not

B. Is a benzylisoquinolinium

C. Is a bisquaternary amine - monoquaternary structure

D. Is more lipid soluble than pancuronium

E. Is predominantly renally excreted 75% biliary

[MB08](#) [Jul97] [Jul98] [Mar99] [Jul02] [Mar03] In reversing neuromuscular blockade, which of the following combinations is best matched with respect to time of onset?

A. [Atropine](#) & neostigmine

B. Atropine & [glycopyrrolate](#)

C. Atropine & edrophonium

D. Atropine & physostigmine

E. Glycopyrrolate and edrophonium

Edrophonium	1-2	minutes
Neostigmine	7-11	minutes
Pyridostigmine	16	minutes
Atropine	1	minute
Glycopyrrolate	2-3	minutes

[MB09](#) [Jul97] [Jul98] [Mar99] [Jul99] [Jul00] [Mar03] Plasma cholinesterase:

A. Metabolises [dibucaine](#) = amide LA which inhibits plasma cholinesterase. dibucaine no 80% = normal people

B. Metabolises [esmolol](#)

C. Hydrolyses mivacurium at 80% the rate of suxamethonium **88% rate**

D. Is unaffected by [neostigmine](#) - **reduces plasma cholinesterase activity by 50% & lasts 30mins**

[MB09b](#) [Jul01] [Jul04] [Suxamethonium](#)

A. Bigger molecule than [vecuronium](#)

B. Needs to occupy 80% of nicotinic receptors to get effect **20%**

C. Resistant to hydrolysis by acetylcholinesterase

D. ??Is an antagonist at nicotinic receptors

E. Increasing dose produces similar block

[MB10](#) [Jul97] [Jul98] With regard to the nerve stimulator in competitive blockade:

A. Fade is dependent on stimulating frequency **Fade in response to tetanic stimulation is dependant on degree of NMB, frequency of stimulation, length of stimulation and how often tetanic stimuli applied**

B. TOFC of four is a sign of adequate reversal. **not count. need ratio ie 0.7-0.9**

C. ?

D. ?

[MB11](#) [Jul97] Anticholinesterase agents:

A. Carbamates duration of action is related to the time required for dissociation from the anionic site.

B. Carbamates act by acetylation of the esteratic site.

C. ?

(See also [\[\[MB11b](#), [\[\[MD28](#))

[MB11b](#) [Jul00] [Apr01] [Jul02] [Jul04] Carbamylation of acetylcholinesterase: (Jul02: Phosphorylation of acetylcholinesterase:)

A. Ionic bonding at anionic site

- B. Ionic bonding at esteratic site
- C. Covalent bonding at anionic site
- D. Covalent bonding at esteratic site
- E. None of above

(see also [MB27](#) for similar Q)

[MB12](#) [dgj] [Jul00] [Jul02] [Jul04] [Mivacurium](#):

- A. Is metabolised at 80% the rate of suxamethonium 88%
- B. Takes 15 mins from ED95 dose to recovery of 95% twitch height onset 2-3min, lasts 12-20mins
- C. Has an ED95 of 1.5 mg/kg = 0.067mg/kg
- D. Trigger for [malignant hyperthermia](#)
- E. ? Duration of action is increased in renal failure

July 2000 version: [Mivacurium](#):

- A. Twice the ED95 dose is 1.5mg/kg ED95 = 0.067mg/kg
- B. is metabolised at 80 to 90% the rate of suxamethonium
- C. After 2 x ED95 dose 95% return of twitch height after 15mins >20mins

July 2002]] version included the following options:

- C. Does not usually require reversal
- D. Duration of action may be prolonged by anti-cholinesterases

[MB12b](#) [Jul00] [Mivacurium](#) administered at a dose of 2 times the ED95 dose produces relaxation for: ie 0.15mg/kg

- A. 10 mins
- B. 15 mins
- C. 20 mins
- D. 25 mins
- E. None of the above

[MB13](#) [Mar98] [Jul99] [Jul01] The Recovery Index 25% to 75% is 7 minutes for which drug?

- A. [Vecuronium](#)
- B. [Rocuronium](#)
- C. [Mivacurium](#)
- D. [Suxamethonium](#)

recovery index:

- Mivacurium: 6.6 mins.
- Vecuronium: 14-30 mins according to dose.
- Rocuronium: 8-17 mins.

Also recalled as: A muscle relaxant is administered at twice ED95 for a short dental case. Return of normal TOF ratio occurred at 7minutes. The muscle relaxant used was:

- A. [Suxamethonium](#) only one to return in 7mins but ratio should remain normal for depolariser!!!
- B. [Vecuronium](#)
- C. [Atracurium](#)
- D. [Rocuronium](#)
- E. [Mivacurium](#)

Duration of Return to TOF > 0.9

- Mivacurium - 25-40 mins
- Vecuronium - 50-80 mins
- Atracurium - 55-80 mins
- Rocuronium - 55-80 mins

[MB14](#) [Mar98] [Jul00] [Mar03] Release of acetylcholine at the motor endplate:

- A. ?? [gentamicin](#) - cause ↓release by competing with calcium
- B. Botulinum toxin works by ?? - blocks ACh release by cleaving enzymes which allow Ach vesicles to fuse with presynaptic membrane
- C. ?

D. ?

July 2000 version: Release of acetylcholine at motor endplate:

A. Hemicholinium directly interferes with release - **interferes with synthesis**

B. Only in response to action potential

C. Decreased by aminoglycosides / ?? prejunctional effect **has effect at both**

D. Is Ca²⁺ dependent process

E. Always causes an action potential

[MB15](#) [Mar98] Gentamicin potentiates non-depolarising neuromuscular block by:

A. Interfere with Ca⁺⁺ influx for exocytosis

B. ?

C. ?

[MB16](#) [Jul98] [Mar99] [Feb00] [Jul01] [Mar03] [Rocuronium](#):

A. Monoquatary at physiological pH

B. More lipid soluble than pancuronium **??maybe**

C. 30% metabolised (?deacetylated) in the liver **excreted unchnaged in bile**

D. Rapid onset is due to its high potency **due to low**

E. Fastest onset is with 2 times ED₉₅ dose

F. Is bisquatary

[MB17](#) [Mar96] Plasma cholinesterase is inhibited 80% by 10⁻⁵ molar dibucaine:

A. In late pregnancy - **preg causes acquired defect in plasma cholinestrace**

B. ?

C. ?

[MB18](#) [Mar99] Which of the following do NOT prolong neuromuscular blockade?

A. Volatile anaesthetics

B. Antibiotics

- C. Phenytoin
 - D. Beta-blockers
 - E. Hyperthermia
- (see also [MB26](#))

[MB19](#) [Jul98] Malignant hyperthermia causes:

- A. Hypertension
- B. Whole body rigidity
- C. Tachyphylaxis with a suxamethonium infusion
- D. ?

[MB20](#) [Jul99] [Jul01] Edrophonium:

- A. Longer half-life than neostigmine quicker onset, same elim half life ie around 80mins, but distribution half life is v short
- B. Onset slower than neostigmine
- C. ?Pyridostigmine
- D. Binds to anionic site of cholinesterase as well as esteratic site
- E. Relieves symptoms of myasthenia gravis - used for diagnosis
- F. ? Is reliable in reversing a Phase 2 block

[MB20b](#) [Apr01] ("[Edrophonium](#) Q about elimination half times and metabolism")

- A. ?
- B. ?

[MB21](#) [Jul99] . . ? . . with return of $\frac{3}{4}$ TOF ratio:

E. ?Neostigmine may prolong the action of Mivacurium

Neostigmine profoundly decreases plasma cholinesterase activity and could thus interfere with the normal rapid spontaneous recovery from mivacurium-induced NMB

[MB22](#) [Jul99] [Apr01] [Atracurium](#):

- A. Has an active metabolite
- B. Ester metabolism is a minor pathway of elimination = major pathway
- C. Metabolism is by Hofmann elimination which is pH dependent ('Did not include temperature') but is also
- D. ?
- E. ?

[MB23](#) [Feb00] [Jul04] What muscle relaxant has an active metabolite with a half-life twice that of the parent compound?

- A. [Rocuronium](#)
- B. [Vecuronium](#)
- C. [Pancuronium](#)
- D. [Atracurium](#) or [Cisatracurium](#)
- E. None of the above
- F. [Mivacurium](#)

vecuronium has active metabolite 80% as active, but elimination half life 116min c.f. vec t 1/2 of 34, > 2 x duration

Pancuronium has active metabolite 50% to 66% as active, with similar elimination half life to pancuronium

[MB23b](#) [Jul04] Which of these NDNMB has a metabolite that's 50-70% as active as its parent drug

- A. [Atracurium](#)
- B. [Vecuronium](#)
- C. [Rocuronium](#)
- D. dTC
- E. None of the above

Both pancuronium (though 80% is excreted unchanged) and vecuronium have active metabolites that are more than 50% active at the NMJ.

[MB24](#) [Feb00] Succinylcholine can cause: **dirty drug anything possible**

- A. Bradycardia
- B. Histamine release
- C. Tachycardia
- D. Hypertension
- E. **All of the above**

[MB25](#) [Feb00] Neostigmine reversal of nondepolarising neuromuscular block

- A. Not affected by enflurane at 2 MAC
- B. **Varies depending on use of NDNMA by bolus or infusion**
- C. Is/isn't affected by age
- D. ?

[MB26](#) [Feb00] Which of the following is associated with a decrease in duration or effect of nondepolarising neuromuscular blocking drugs:

- A. Volatile anaesthetic alkanes
- B. Volatile anaesthetic ethers
- C. Aminoglycoside antibiotics
- D. **Aminopyridine derivatives = potassium channel blocker \Rightarrow \uparrow release at NMJ**
- E. Local anaesthetic esters

(see also [MB18](#))

Alt version: Which of the following decreases the duration/depth of neuromuscular blockade?

- A. [Enflurane](#) at 2 MAC
- B. Aminoglycosides
- C. Bolus doses versus infusion
- D. **Aminopyridines**

[MB26b](#) [Jul01] Neuromuscular blockade NOT prolonged by:

- A. Hyperthermia
- B. [Gentamicin](#)
- C. Volatile agents
- D. Hypothermia
- E. ?

[MB27](#) [Jul00] [Apr01] [Jul04] [Neostigmine](#)'s mechanism of action:

- A. Binds covalently to esteric site on AChEsterase
- B. Binds electrostatically to esteric site on AChEsterase
- C. Binds to anionic site
- D. Forms complex with AChEsterase with a shorter halflife than acetylcholine
- E. ("Some other long winded explanation requiring 30 seconds to read and impossible to remember.")

Neostigmine binds to the anionic site, but is then transferred to the esteratic site and hydrolysed. This is a much slower process with neostigmine (minutes) than with acetylcholine (milliseconds). Neostigmine is an ester.

[MB28](#) [Jul00] With depolarising neuromuscular blocker:

- A: Is competitively antagonised by NDMR
- B: ("Something about tetany & fade")
- C. ?
- D. ?
- E: Shows post tetanic potentiation

[MB29](#) [Jul00] Rocuronium administered in 2 times the ED95 dose: = 0.6mg/kg

- A. Rapid onset, short duration
- B. Rapid onset, Intermediate duration
- C. Slow onset, intermediate duration
- D. Slow onset, long duration

E. ("some other combination.")

[MB30](#) [Apr01] Anticholinesterase drugs

A. ?

B. ?

C. Used in treatment of Glaucoma

- 1. Antagonist - assisted reversal of neuromuscular blockade produced by non depolarising neuromuscular blocking agents.

In addition treatment of:

- 2. myasthenia gravis
- 3. glaucoma
- 4. paralytic ileus and atony of the urinary bladder
- 5. CNS effects of certain drugs
- 6. May be useful in alzheimers disease.

[MB31](#) [Apr01] [Neostigmine](#):

A. Tertiary ammonium compound

B. quaternary ammonium compound

C. ?

physostigmine - tertiary & ∴ able to cross bbb

[MB32](#) [Jul01] [Jul04] The [dibucaine number](#) for a normal person is:

A. 20

B. 40

C. 60

D. 80

E. 100

The [dibucaine number](#) in a patient with **Ea:Ea** genotype is: *repeat*

A. 0

B. 20

C. 35

D. 50

E. 60

Genotype	Dibucaine Number	Response to Sux	Population Frequency
$E^u:E^u$	80	normal	96%
$E^a:E^a$	20	Greatly Prolonged	1 in 3200
$E^u:E^a$	60	Slightly Prolonged	2 in 480
$E^u:E^f$	75	Slightly Prolonged	1 in 200
$E^f:E^a$	45	Greatly Prolonged	1 in 20000

MB33 [Jul01] Muscle relaxants are less likely to cause [anaphylaxis](#) if:

- A. Injected slowly
- B. Suxamethonium is the most common cause - but depends on location
- C. H1 and H2 blockers prevent anaphylaxis
- D. Always fatal
- E. ?

MB34 [Jul01] [Laudanosine](#):

- A. ?
- B. ?
- C. ?
- D. ?

MB35 All of the following result in prolongation of Vecuronium block except:

- A. Concomitant insulin and dextrose infusion
- B. Prior suxamethonium blockade - will ↑ magnitude of response but not duration

MB36 [Feb04] Post suxamethonium myalgia:

- A. Preceded by transient myoglobinuria
- B. More common in the elderly
- C. Can be prevented by pre-treatment with 0.04 mg/kg of D-tubocurarine
- D. Is invariably associated with increased intra-ocular pressure
- E. Is associated with hypokalaemia

[MB37](#) [Feb04] Regarding anticholinesterases:

- A. [Pyridostigmine](#) is a tertiary amine
- B. Quaternary ammonium anticholinesterases have a larger volume of distribution than non-depolarising muscle relaxants
- C. [Edrophonium](#) has a slower onset of action than neostigmine
- D. [Neostigmine](#) has a longer duration of action than [pyridostigmine](#)
- E. Edrophonium binds covalently to the esteratic site of acetylcholine

[MB37b](#) [Jul04] Regarding Antiacetylcholinesterase

- A. Given orally to treat glaucoma - **used topically**
- B. Edrophonium is a long acting AChE inhibitor - **half life longer than neostigmine**
- C. [Physostigmine](#) is quaternary ammonium = **tertiary**
- D. ?

[MB38](#) [Jul04] Which is the best indicator of adequate reversal?

- A. TOF Count of 4 = **ratio is impt**
- B. No fade on DBS
- C. No fade to 200 Hz tetanus - **not good indication**
- D. Head lift??
- E. Evidence of post-tetanic facilitation

[MB38b](#) [Jul04] Residual curarization is best evaluated with:

- A. TOF 1:4 > 50%

B. Equal twitch height on DBS or ratio >0.9

C. ?Degree of fade is independent on stimulus intensity

D. ?Used to check depth of anaesthesia

E. ?

[MB39](#) [Jul07] Sugammadex binds most avidly to:

A. Pancuronium - may also partly reverse

B. Rocuronium

C. Vecuronium - will adequately reverse

D. Atracurium

E. Cisatracurium