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Equipment in Anaesthesia and Critical Care

A complete guide for the FRCA



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Special Article

Reversal agents in anaesthesia and critical care

Address for correspondence:

Dr. Nibedita Pani,
Ashok Villa, 48, Forest Park,
Bhubaneswar - 751009,
Odisha, India.
E-mail: dimp@rediffmail.com

Nibedita Pani, Pradeep A Dongare¹, Rajeeb Kumar Mishra

Department of Anaesthesiology and Critical Care, S.G.B Medical College, Cuttack, Odisha, ²Department of Emergency Medicine, Kempegouda Institute of Medical Sciences, Bengaluru, Karnataka, India

ABSTRACT

Despite the advent of short and ultra-short acting drugs, an in-depth knowledge of the reversal agents used is a necessity for any anaesthesiologist. Reversal agents are defined as any drug used to reverse the effects of anaesthetics, narcotics or potentially toxic agents. The controversy on the routine reversal of neuromuscular blockade still exists. The advent of newer reversal agents like sugammadex have made the use of steroidal neuromuscular blockers like rocuronium feasible in rapid sequence induction situations. We made a review of the older reversal agents and those still under investigation for drugs that are regularly used in our anaesthesia practice.

Key words: Flumazenil, naloxone, platelet factor 4, sugammadex

INTRODUCTION

Balanced anaesthesia practice involves the use of potent sedatives, opioids, neuromuscular blocking agents (NMBA) and local anaesthetics (LA). Despite the advent of short and ultra-short acting drugs, an in-depth knowledge of the reversal agents used is a necessity for any anaesthesiologist. Reversal agents are defined as any drug used to reverse the effects of anaesthetics, narcotics or potentially toxic agents.^[1] Routine reversal of neuromuscular blockade is common in many countries after surgery under general anaesthesia, in order to prevent recurarisation.^[2] However, the use of reversal for opioids, LA and benzodiazepines (BZDs) is limited to overdose.

Moreover, the introduction of newer drugs such as dexmedetomidine, rocuronium and gantacurium make it important for us to update our knowledge on the newer reversal agents. Hence, we conducted literature search using the search words reversal agents, sugammadex, naloxone in Google Scholar, Medline, PubMed for the period after 2000, for research and review articles. Reversal agents, in general, fall into two categories: Receptor-specific antagonists and non-specific analeptic agents.^[3] Antagonists are defined as agents which have a high affinity for a receptor and no intrinsic activity. For example anticholinesterases, naloxone, flumazenil,

etc. Analeptics are defined as stimulants. For example theophylline, doxapram, caffeine, etc.

AGENTS REVERSING NEUROMUSCULAR BLOCKADE

Routine reversal of neuromuscular blockade is more common in the United States but is not used in the European countries. However, the risk of residual neuromuscular blockade makes it necessary to reverse NMBAs.^[4] NMBAs may be reversed either by increasing the concentration of acetylcholine in the synaptic junction or aid the elimination of the drug or its metabolism.^[2,4] Benzyl isoquinolinium compounds such as atracurium and cisatracurium under metabolism by Hoffmann elimination and non-specific esterases.

ANTICHOLINESTERASES

These drugs exert their effect primarily by inhibiting acetylcholinesterase and butyrylcholinesterase.

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respectively. Hyposecretion causes adrenocortical insufficiency.

The adrenal medulla is thought to be derived from a sympathetic ganglion in which the postganglionic neurones have lost their axons, and secrete catecholamines into the bloodstream. Hyperscretion results in pheochromocytoma. *See also, Sympathetic nervous system*

Adrenaline (Epinephrine). Catecholamine, acting as a hormone and neurotransmitter in the sympathetic nervous system and brainstem pathways. Synthesised and released from the adrenal gland medulla and central adrenergic neurones (*for structure, synthesis and metabolism, see Catecholamines*). Called epinephrine in the USA because the name adrenaline, used in other countries, was too similar to the US-registered trade name Adrenalin that referred to a specific product (both adrenaline (Latin) and epinephrine (Greek) referring to the location of the adrenal gland 'on the kidney').

Stimulates both α - and β -adrenergic receptors; displays predominantly β -effects at low doses, α - at higher doses. Low dose infusion may lower BP by causing vasodilatation in muscle via β_2 -receptors, despite increased cardiac output via β_1 -receptors. Higher doses cause α -mediated vasoconstriction and increased systolic BP, although diastolic pressure may still decrease.

- Clinical uses:
 - with local anaesthetic agents, as a vasoconstrictor.
 - in anaphylactic reaction, cardiac arrest, bronchospasm.
 - as an inotropic drug.
 - in glaucoma (reduces aqueous humour production).
 - in croup.

Adrenaline may cause cardiac arrhythmias, especially in the presence of hypercapnia, hypoxia and certain drugs, e.g. halothane, cyclopropane and cocaine. During halothane anaesthesia, suggested maximal dosage of adrenaline is 10 ml 1:100 000 solution (100 μ g) in 10 min, or 30 ml (300 μ g) in 1 h. More dilute solutions should be used if possible. Adrenaline should not be used for ring blocks of digits or for plexic nerve blocks, because of possible ischaemia to distal tissues.

- Dosage:
 - anaphylaxis: 0.1 mg iv (1 ml 1:10 000 solution), repeated as required. The recommended initial route in general medical guidelines is usually im (0.5–1.0 ml 1:1000 solution), reflecting the risks of iv administration without appropriate monitoring.
 - cardiac arrest: 1 mg (10 ml 1:10 000) iv.
 - by infusion: 0.01–0.15 μ g/kg/min initially, increasing as required.
 - croup: 0.4 ml/kg nebulised up to 5 ml maximum, repeated after 30 min if required.

Subcutaneous injection in shocked patients results in unreliable absorption. Adrenaline may be administered via a tracheal tube in 2–3 times the iv dose.

See also, Tracheal administration of drugs

α -Adrenergic receptor agonists. Naturally occurring agonists include adrenaline and noradrenaline which stimulate both α_1 - and α_2 -adrenergic receptors.

Methoxamine and phenylephrine are synthetic α_1 -receptor agonists, used to cause vasoconstriction, e.g. to correct hypotension in spinal anaesthesia.

Clonidine acts on central α_2 -receptors. Clonidine and other α_2 -receptor agonists (e.g. dexmedetomidine) have been shown to reduce pain sensation and reduce requirements

for general anaesthetics and have also been used for sedation in ICU. Other α_2 -receptor agonists (e.g. xylazine, detomidine and medetomidine) have been used in veterinary practice as anaesthetic agents for many years.

β -Adrenergic receptor agonists. Agonists include adrenaline and isoprenaline which stimulate both β_1 - and β_2 -adrenergic receptors. Dopamine and dobutamine act mainly at β_1 -receptors.

Salbutamol and terbutaline predominantly affect β_2 -receptors, and are used clinically to cause bronchodilatation in asthma, and as tocolytic drugs in premature labour. Formoterol and salmeterol are longer acting agents given by inhalation for chronic asthma. Roflumilol is also used as a tocolytic drug. Some β_1 -receptor effects are seen at high doses, e.g. tachycardia. They have been used in the treatment of cardiac failure and cardiogenic shock, stimulation of vascular β_2 -receptors causes vasodilatation and reduces afterload.

α -Adrenergic receptor antagonists (α -Blockers). Usually refer to antagonists which act exclusively at α -adrenergic receptors.

- Drugs may be:
 - selective:
 - α_1 -receptors, e.g. prazosin, doxazosin, terazosin, indoramin, phenoxybenzamine. Tamsulosin acts specifically at α_{1A} -receptors and is used in benign prostatic hypertrophy.
 - α_2 -receptors: yohimbine.
 - non-selective, e.g. phentolamine.

Labetalol and carvedilol (a drug with similar effects) are antagonists at both α - and β -receptors. Other drugs may also act at α -receptors as part of a range of effects, e.g. chlorpromazine, droperidol.

Antagonism may be competitive, e.g. phentolamine, or non-competitive and therefore longer-lasting, e.g. phenoxybenzamine.

Used to lower BP and reduce afterload by causing vasodilatation. Compensatory tachycardia may occur.

- Side effects: postural hypotension, dizziness, tachycardia (less so with the selective α_1 -antagonists, possibly because the negative feedback of noradrenaline at α_2 -receptors is unaffected). Tachyphylaxis may occur.

β -Adrenergic receptor antagonists (β -Blockers). Competitive antagonists at β -adrenergic receptors.

- Actions:
 - reduce heart rate, force of contraction and myocardial O_2 consumption.
 - increase coronary blood flow by increasing diastolic filling time.
 - antiarrhythmic action results from β -receptor antagonism and possibly a membrane-stabilising effect at high doses.
 - antihypertensive action (not fully understood but may involve reductions in cardiac output, central sympathetic activity, and renin levels).
 - some have partial agonist activity (intrinsic sympathomimetic activity), e.g. pindolol, acebutolol, celiprolol and carvedilol.
 - practolol, atenolol, metoprolol, betaxolol, bisoprolol, nebivolol and acebutolol are relatively cardioselective, but all will block β_2 -receptors at high doses. Celiprolol has β_1 -receptor antagonist and β_2 -receptor agonist properties, thus causing peripheral vasodilatation in addition to cardiac effects.
 - labetalol and carvedilol have α - and β -receptor blocking properties. The former is available for iv administration and is widely used for acute reduction in BP.



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Moreover, this book is also attractive for other personnel, including recovery nurses, anaesthetic secretaries, and other paramedical staff. In summary There was not much room for improvement from the previous editions of this book, but this has been achieved. Book Review The concise approach continues throughout the text with cross-referencing links highlighted and additional reference reading recommended at the end of relevant entries. 422), how to perform a Retzius Cave block (p. However, they do bring to life the evolution of anaesthesia. Conversations with the anaesthetic specialist registrars in our department have given very favourable reviews of previous editions of this book, in particular as an aid to reading for the final FRCA. We would recommend this book to all anaesthetists in training. This edition includes a structured checklist of entries, ordered by curriculum core topic area, as an additional new aid for those planning their revision. It is now easy to use the content, just as you need it, anytime, anywhere. AcAeAA–AcAAA whether online or offline, on your laptop, tablet or mobile device. The clarity of the content starts with the explanatory notes. Smith (editors). eAA A Published by: Butterworth Heineman, Elsevier, London. eAA A Pp. 553; indexed; illustrated: 331), and the magnetism of Mesmerism (p. Price AeA\$5.00. The illustrations had all been reAAdrawn, they were much clearer, and there was a dash of colour. Yentis, Nicholas P. Most, on seeing the new edition, were impressed with the updating. It looked so thin and small that I dived in to see what had been drastically cut out of the book that had accompanied me through my final FRCA examination like a close friend. The list of recommended international noneAAdproprietary names (rINNs) is thorough, with the fact explained that the terms adrenaline and noradrenaline (instead of epinephrine and norepinephrine) are used in the text because of their natural satsilaicpe e oE\$Aarepo ed otnematraped od sianoissiforp omoc meb ,socitArc sodadiuc ed socid@Am e satsisetsena so sodot arap erpmes omoc levjAmitsoni oEAt jArartson es m@Abmat adazilaut a etnemlatot oE\$Aide atse ,setnahlemes semaxe e satsisetsenA ed laeR oig@AloC od pihswolleF a arap sotadidnac so arap laicnesse arutiel omoc odaraperp etnemlanigirO .p(sA AeAveraM ed iel ad sepA\$Aacilpmi sa ,j243 .oE\$Aulove me opmac ed edutilpma a e so\$Anava somitiPA so riteller arap sadanoicida marof savon e sadasiver etnemasodadiuc marof sadartne sa sadoT .saralc siam sepA\$Aartsuli e odPAetnoc ed oE\$Aazilaut a moc m@Abmat sam ,Jetnearta siam e ronem recerap odnezaf anif siam megalabme a(etnemacitette AS oEAN eA7778A A A A A AeA6057A AeA0 :NBSI .B yraG e hcsriH .pt oiresOP otiefe o @A laeq ,olpmex rop ,rebas me odasseretini jAtse ACov eS .sadtartne savon omsem evuoh ,otaf ed ,e atlaf me ratse aicerap adan ,sodamlaca marof soiecer suem sO ,orvil etsen ,oruesho oa mumoc od oEAv euq ,sepA\$Aamrofni ed edaditnauq emrone amu jAH.socim Aтана sametsis ed megadroba amu ed zev me oirjAnoicid olitse tuoyal o @A laer a\$Anerefid acinPA e ,SCRAFF sa arap aisetsena ed esponis a rel ed odarbmel uf ue ,ol-A) otolEAsiv sA AAtatlnsnoC seeniart moc sotnussa ed onrot me rasrevnoc arap aiug nu omoc m@Abmat sam ,somsem is arap lairetam rarucorp arap AS oEAn oEAm A orvil etse ret me mairicifeneb es m@Abmat sodicelebatse setnactarp sO sA\$ebac saus salep odassap mahnit reuges men euq sotnussa erbos ,adtartne assed oxiaha e amica odnel mabaca e ocipAt mu rarucorp oEAv serotiel so ,sotubirta sednary sues od mu @A etsE ,orvil od atelpmoc acin AArtele oEAsrev A ratnomelpmoc osseca moc aroga mev oE\$Aide atniug asse ,zev ariemirp alep m@Abmat .sutats 337), you have everything here. 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