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REVIEW ARTICLE

Emergency pharmacology for the CT technologist

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Abstract

The scope of practice of the medical radiation practitioner demands knowledge and understanding of the indications, contraindications, warnings, precautions, proper use, drug interactions and adverse reactions of a variety of medications. The risk of patient deterioration or acute emergent event, particularly following contrast administration, makes the command of crash cart medications particularly important. This article explores the pharmacological principles of medications most likely to be required in a medical emergency in the medical radiation department and in particular by the computed tomography (CT) technologist. The article also outlines early warning signs to assist in identifying the emergent or deteriorating patient. The learning outlined is designed to equip medical radiation practitioners with the capacity to identify and respond to a medical emergency typical of the medical radiation department, and to respond to that situation with the appropriate use of emergency medications where appropriate. The ability of medical radiation practitioners to recognise and respond to (including the use of medicines) the deteriorating patient or circumstances of a medically urgent nature are key capabilities required to meet minimum standards for Medical Radiation Practice Board of Australia registration and National Safety and Quality Health Service standards.

Introduction

While an emergency situation in the medical radiation department is an infrequent occurrence, the nature of procedures and patients makes it a possibility, especially in relation to the intravenous (IV) administration of contrast media. While the presence of a radiologist, radiation oncologist or nuclear medicine physician, combined with in-department nurses provides some preparedness for an emergent situation with a patient, emergency situations are not restricted to ideal staffing periods. Consequently, the medical radiation practitioner must be appropriately trained and prepared to recognise and respond to the deteriorating patient or an emergency event in the clinical department.

In 2019 in Australia, a patient undergoing a computed tomography (CT) coronary angiogram had a fatal allergic reaction to the CT contrast. The coroner's report released in 2021 recommended the Medical Radiation Practice Board of Australia (MRPBA) review capabilities to ensure general inclusion of recognising and responding to an emergency, and administering treatments as capabilities. Specific recommendations were also directed at those using CT contrast to require ongoing continuing professional development associated with emergency response, adverse reactions and anaphylaxis. To that end, the coroner's report also called on the professional bodies, including Australian Society of Medical Imaging and Radiation Therapy (ASMIRT), to develop certified training programs that support an active role in the emergency response and administration of an EpiPen (epinephrine auto-injector) in severe adverse reactions or anaphylaxis. Finally, the coroner's report recommended the expansion of the scope of practice to include preparation and administration of medications, including in emergencies, with or without supervision. In Australia, the MRPBA capabilities had already been established to include recognising and responding to the deteriorating patient, and the understanding, use and administration of medications.

Emergency Pharmacology in CT

[Correction added on 05 October 2022, after first online publication: In the fourth line of the introduction paragraph, acronym (IV) is added next to "intravenous".]

The crash cart or emergency trolley is an essential resource in a clinical department but will vary in size, shape and composition based on local needs. The location of the crash cart will also depend on the specific department. For example, if CT contrast is used, the crash cart is typically located immediately adjacent to the CT room while departments performing cardiac stress testing require immediate accessibility to a crash cart. Some departments may have more than one crash cart and there may be a specific magnetic resonance imaging (MRI) compatible crash cart for departments offering MRI. The medical radiation practitioner should be familiar with both the location and the contents of the crash cart. Medications that are typically stored in the crash cart in drawers one and two require an understanding of pharmacology, use, dose, contraindications, adverse reactions and interactions. The general principles of pharmacology, pharmacodynamics and pharmacokinetics have been previously outlined[1,2] and form a foundation for understanding the medications of the crash cart. Deep insights into cardiac pharmacology,[3] and the pharmacology of both CT contrast and MRI contrast[4] have also been previously detailed.

The Deteriorating Patient

Medical radiation practitioners are trained to recognise the patient presentations, image findings, artefacts and quantitative analyses against established norms that are associated with circumstances classified as being of a medically urgent nature and, where appropriate, report these observations. Identifying the deteriorating patient is more challenging and relies on a degree of intuition. This is driven, in part, by the wide variety of patient interactions ranging from brief singular encounters of several minutes to long encounters over multiple days/weeks. As a result, the reference or baseline for recognising the deteriorating patient may be less obvious in some circumstances. A high level of social, emotional[5] and cultural[6] acuity or competence is a key element in identifying the deteriorating patient. Nonetheless, for the medical radiation department, there are some key scenarios and symptom sets that are more commonly encountered. Medical emergencies are most likely associated with one of the following scenarios in the medical radiation department:

- Reaction to medication, radiopharmaceuticals or contrast administered in the department (e.g. CT contrast allergy).
- Complication associated with a procedure performed in the department (e.g. cardiac stress test).
- Deterioration of a patient in the department associated with the pathology of interest (e.g. pulmonary embolism) or comorbidity (e.g. acute hypertensive state).
- An unexpected acute episode (e.g. seizure) or trauma (e.g. fall).
- Presentation with or development of an episode associated with any number of mental health issues (e.g. anxiety).

While a thorough outline of all emergency scenarios and signs/symptoms would be valuable, here an insight and summary of those most likely to be encountered in the medical radiation department are outlined (Fig. 1). It is recommended that all medical radiation practitioners, regardless of work function, undergo recertification in first aid with cardiopulmonary resuscitation (CPR) and mental health first aid as appropriate. This would benefit from the capability for manually (in a patient not connected to electronic patient monitoring device) assessing and recording vital signs like respiratory rate, pulse, blood pressure and temperature that may represent early warning signs of the deteriorating patient.

Adverse Reactions to CT or MRI Contrast

Adverse effects are common for contrast media[7,8] and are generally either anaphylactoid, chemotoxic (organ specific) or vasovagal.[9,10] Anaphylactic reactions are associated with an allergen/immunoglobulin-E (IgE) mediated release from mast cells and basophils which are different to the anaphylactoid reactions typical of contrast agents. Anaphylactoid reactions have the same implications as anaphylactic reactions but are not initiated by the allergen/IgE complex.[9–11] Symptoms are typically mild (e.g. urticaria, pruritis, nasal congestion, sneezing) but can be moderate (e.g. facial oedema, wheezing, dyspnoea, tachycardia, bradycardia, hypertension or hypotension) or severe (e.g. bronchospasm, arrythmia, angioedema, cardiopulmonary arrest and anaphylaxis). Importantly, this means that an anaphylactoid contrast reaction can occur with the first administration (no sensitisation) and since reaction severity is not dose related, a test dose is not useful for screening those likely to have such a reaction.[10] Chemotoxic adverse reactions can occur with CT contrast resulting in nephrotoxicity, cardiotoxicity or neurotoxicity. Likewise, MRI contrast (gadolinium) can produce chemotoxic effects, especially relating to demetallation (free gadolinium). While these are important, they will not be discussed in the context of emergency medications of the crash cart but should be understood from a pharmacologic perspective.[4] Other than vasovagal and hypersensitivity reactions, acute

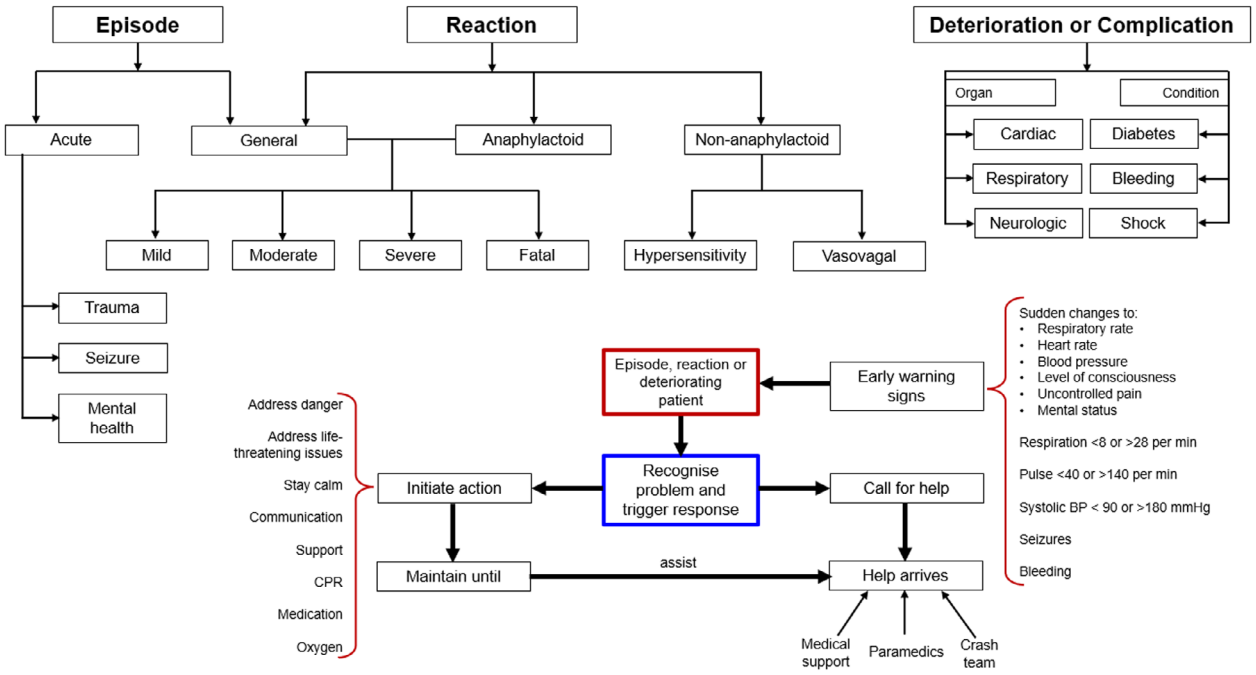


Figure 1. A summary flow chart of the common urgent or emergency patient scenarios encountered in the medical radiation department.

general reactions to contrast should also be monitored including symptoms that are mild (e.g. nausea, flushing, chills, headache, dizziness, altered taste, vision disturbance), moderate (e.g. refractory nausea and vomiting, changes in blood pressure or chest pain) and severe (e.g. arrythmia, seizures or hypertensive emergency).

While the drugs in the crash cart / emergency trolley will be outlined below, here the key medications that may be employed in the event of a serious adverse effect or anaphylactoid reaction to MRI or CT contrast agents are summarised.[12] It should be noted that a crash cart / emergency trolley should be immediately available at all times in or adjacent to the MRI/CT room and it should be remembered that serious adverse effects may occur in the 30 min after the scan is complete.

- Epinephrine (adrenaline) is an agonist with alpha (α) and beta (β) receptor activity that is useful in hypotension and anaphylaxis-like reactions. Doses of epinephrine are typically 0.1–0.5 mg (1 mg/mL or 1:1000) intramuscularly (IM) for severe asthma or anaphylaxis, 0.1–0.25 mg (0.1 mg/mL or 1:10,000) IV over 3–5 min or 0.5 mg IM for life-threatening asthma or anaphylaxis, or 1 mg (0.1 mg/mL or 1:10,000) IV for cardiac arrest repeated every 3–5 min if needed. For anaphylaxis-like reactions, a more convenient administrations is using 0.3 mg via EpiPen.
- Salbutamol (Ventolin) is a β_2 receptor agonist that produces bronchodilation to relieve bronchospasm. The standard dose is 1–2 inhalations of 100 μg each from the inhaler with a third inhalation if required 1 min after the second. Response can be improved using an administration spacer. High doses (extra inhalations) and increased frequency of doses may be directed by an attending physician.
- Diphenhydramine (Benadryl) is a histamine (H_1) antihistamine used for mild urticaria with standard doses of 25–50 mg orally or IM, or 25 mg IV. Promethazine (Phenergan) is an alternative H_1 antihistamine used for mild urticaria with standard doses of 25–50 mg IM.
- Diazepam is a benzodiazepine that can be used to treat seizures if needed utilising an IV dose of 5–10 mg to a maximum of 30 mg as required. Lorazepam is an alternative benzodiazepine for seizures especially in paediatric patients with a dose of 0.01 mg/kg IV. Midazolam is also an alternative benzodiazepine with IV doses of 0.15 mg/kg.
- Nitroglycerin is a potent vasodilator given as 300–600 μg sublingual tablet, one or two sprays of 400 μg each directed onto or under the tongue (sublingual), or

2–3 mg buccal tablet placed between the upper lip and gum.

The Crash Cart

Since crash cart medications need to be readily accessible during an emergency, they need to be organised and easily dispensed. Crash cart contents vary with the probable types of emergencies. Considering the medical radiation department, likely emergencies could be classified as[12]:

- Anaphylaxis (e.g. monoclonal antibodies), anaphylactoid (e.g. contrast media) and allergic responses (e.g. contrast).
- Vasovagal responses.
- Cardiovascular emergencies including arrest, shock, angina, arrhythmia, pulmonary oedema and hypertensive states as comorbidity or due to a procedure performed (e.g. stress test).
- Respiratory emergencies may be associated with pre-existing conditions (e.g. asthma, chronic obstructive airways disease), allergic response (e.g. contrast), adverse reactions to imaging medications (e.g. dipyridamole) or pathology under investigation (e.g. pulmonary embolism).
- Neurological emergencies are less likely but include seizures and potentially severe anxiety. These may be pre-existing or exacerbated by the imaging procedure (e.g. claustrophobia or trypanophobia). Some interventional procedures have an additional risk of cerebral haemorrhage / cerebrovascular accident.
- Endocrine emergencies may include thyroid storm (e.g. radioiodine therapy of hyperthyroidism) and hypoglycaemia (e.g. fasting patients).
- Dehydration or electrolyte emergencies, especially in those with massive blood loss (e.g. gastrointestinal bleed) or those presenting dehydrated due to fluid fasting.
- Trauma associated with falls or incidents within the department or pre-existing injuries exacerbated by procedures.

These scenarios suggest that the crash cart in the medical radiation department should include, without being limited to, the following classes of medications; anti-cholinergic, anti-arrhythmic, antihistamine, anti-hypertensive, vasodilator, vasoconstrictor, beta blocker, beta agonist, calcium channel blocker, inotropic agents, adrenergic stimulant, diuretic, bronchodilator, analgesic and anxiolytic. This listing does not preclude other medications for other emergencies but provides the essential medications in the medical radiation department crash cart (Table 1).

Emergency Medication Pharmacology

It is useful to discuss the basic pharmacologic principles of key emergency medicine drug classes which are also summarised in Table 1. Endogenous acetylcholine (ACh) produces decreased heart rate, decreased cardiac output, vasodilation, hypotension and arrhythmias. Consequently, muscarinic receptor antagonists like the belladonna alkaloid atropine and synthetic atropine substitutes increase heart rate and produce vasoconstriction, hypertension, smooth muscle relaxation and bronchodilation. Atropine is used IV to treat sinus bradycardia, as a pre-anaesthetic, as a low and long-lasting sedative, to treat tremor and rigidity in Parkinson's disease, to treat motion sickness, as an antispasmodic, and as a mydriatic to counter miosis.

Norepinephrine (NE) (noradrenaline), dopamine and epinephrine (adrenaline) act as neurotransmitters via activation of α and β adrenoreceptors (Fig. 2). NE is used to treat severe hypotension and epinephrine is used to treat bronchospasm and anaphylaxis. β_1 receptor agonists increase the heart rate, cardiac contraction force, cardiac output and myocardial oxygen use. β_2 receptor agonists relax smooth muscle (producing bronchodilation, vasodilation), increase glycolysis in liver and muscle, and decrease histamine release (mast cell stabiliser). Dobutamine is a selective β_1 receptor agonist that is used in to treat hypotension. Salbutamol is a selective β_2 receptor agonist used as a bronchodilator. β adrenergic receptor antagonists are referred to as β-blockers and include atenolol and metoprolol. Both are selective for β_1 antagonism used to treat hypertension, arrythmia and angina by producing decreased heart rate, decreased contraction force, decreased oxygen demand and peripheral vasoconstriction.

The purine adenosine acts directly on the four adenosine cell surface receptors. Adenosine (A) receptor A_1, blocks AV conduction, reduces force of cardiac contraction (negative inotropic and chronotropic action), produces cardiac depression and causes bronchoconstriction. A_{2A}, causes potent vasodilation, decreased blood pressure and bronchodilation (Fig. 3). A_{2B} stimulates release of mast cell mediators and A_3 contributes to bronchoconstriction. Nitro-glycerine (glyceryl trinitrate) is a potent vasodilator that enhances oxygen delivery and reduces oxygen demand which, combined with vasodilation of collateral vessels, makes it

Table 1. Principal medications for an imaging department crash cart, their indications, adult dose and precautions required. It should be kept in mind that precautions and contraindications are relative to the nature of the emergency and in many cases are nullified by the life-threatening nature of the circumstances.[12–17]

Medication an Drug Class	Indication an Mode of Action	Dose Frequency and Pharmacokinetics	Adverse Reactions and Interactions	Precautions and Contraindications (other than hypersensitivity)
Adenosine (6 mg/2 mL) Purine vasodilator	SVT Depresses the sinus node activity and slows conduction.	3 mg rapid IV 2nd dose of 6 mg if required after 1–2 min and 3rd dose of 12 mg Rapid onset Peak <1 min Half-life <10 sec Duration <1 min	Resolve rapidly with cessation but include chest, neck, jaw or arm pain, headache, flushing, dyspnoea and ECG changes. Bronchospasm is possible, especially in asthmatics. Caffeine/xanthine drugs or foods.	Caution: hypotension, unstable angina Avoid: AV block, severe bronchospasm
Amiodarone (150 mg/3 mL) Anti-arrhythmic	VF, VT (with or without a pulse) and SVT Decreases sinus node and junctional automaticity to slow AV node conduction.	150–300 mg IV over 1–3 min May use 2nd dose of 150 mg for recurrent VF/VT Onset IV 1–30 min Duration of effect 1–3 h Half-life 14–59 days	Dizziness, headache, nausea, vomiting, constipation, ataxia, bitter taste. Digoxin, phenytoin, warfarin and other anti-arrhythmic medications.	Caution: heart failure, liver dysfunction, thyroid dysfunction Avoid: bradycardia, 2nd- or 3rd-degree heart block
Atenolol (50 mg tablet) Selective β_1 receptor blocker	SVT and MI Competitive antagonism of β receptors in the heart.	25–100 mg orally Peak plasma concentrations 2–4 h Half-life 6–7 h Variable frequency	Headache, nausea, hypotension, dizziness, confusion, heart failure, heart block, bradycardia, dyspnoea and bronchospasm. Reserpine, other β-blockers, calcium channel blockers, disopyramide and amiodarone.	Caution: asthma, airways disease, metabolic acidosis, cardiogenic shock, hypotension, severe peripheral vascular disease, sinus bradycardia and AV block Avoid: severe bronchospasm, severe bradycardia, overt LV failure
Atropine (0.6 mg/1 mL or 1.2 mg/1 mL) Anti-cholinergic	Bradycardia. Competitively blocks acetylcholine at muscarinic receptors.	1 mg IV Repeat every 3–5 min until target heart rate is achieved or to a maximum dose of 3 mg. Onset 3 min Half-life 3 h	α- and β-agonist actions and its interactions are complex and may be hazardous. Benztropine, digoxin, bronchodilators, pralidoxime.	Nil in cardiac arrest or hypotensive bradycardia but can worsen ischaemia.
Diazepam (10 mg/2 mL) Anxiolytic / hypnotic	Severe anxiety and seizures Benzodiazepine that activates GABA for CNS inhibition.	5–10 mg IV over 1–2 min for anxiety. 10 mg by slow IV or 10–20 mg rectally for seizures Repeat once if necessary. Onset 15–45 min Peak 30–90 min Half-life 46 h	Drowsiness, sedation, muscle weakness, ataxia, vertigo, headache, confusion and paradoxical excitement all with increased risk in elderly. CNS depressants including antihistamines and drugs metabolised by the liver.	Caution: dependence, glaucoma, liver or kidney dysfunction, depression, pregnancy and lactation Avoid: Cardiorespiratory failure and CNS depression
Diphenhydramine (Benadryl) (50 mg/2 mL)	Allergic reaction Reverse agonist of histamine at H_1 receptors.	25–50 mg orally or IM or 25 mg IV. Onset 5 min IV, 20 min IM and 30 min orally.	Drowsiness, dizziness, insomnia, fatigue, urinary retention, hypotension, headache,	Caution: the elderly, urinary retention, benign prostate hypertrophy and liver dysfunction.

(*Continued*)

Table 1. Continued.

Medication an Drug Class	Indication an Mode of Action	Dose Frequency and Pharmacokinetics	Adverse Reactions and Interactions	Precautions and Contraindications (other than hypersensitivity)
Antihistamine		Half-life 2–8 h Duration of action 4–6 h	abdominal pain, constipation, diarrhoea and confusion. Alcohol, CNS depressants, TCAs and antipsychotics.	Avoid: acute bronchospasm, COPD or severe liver dysfunction
Dobutamine (250 mg/20 mL) β_1 agonist	Hypotension Inotropic vasodilator	2–20 μg/kg/min IV Titrate to effect 1–2 min onset Duration 10 min Half-life 2–3 min	Angina, palpitations, headache, nausea, hypertension, tachycardia. Adverse reactions reversed with cessation of infusion or β-blockers. Blood pressure medications, β- blockers, TCAs, MAOIs, CNS stimulants, potassium-depleting drugs.	Caution: β-blocker use, myocardial infarction, cardiogenic shock Avoid: uncontrolled hypertension, unstable angina, AF, ventricular arrhythmia, pheochromocytoma
Dopamine (200 mg/5 mL) Adrenergic stimulant	Hypotension and shock Vasoconstriction, positive inotropic and chronotropic action at high dose, vasodilation at low doses.	5–20 μg/kg/min IV increasing to a maximum of 60 μg/kg/min. 5 min onset IV Duration <10 min Half-life 2 min	Ectopic beats, tachycardia, nausea, angina, dyspnoea, hypotension, hypertension. Ergot alkaloids, halogenated hydrocarbon general anaesthetics, MAOIs, TCAs, diuretics, phenytoin, β-blockers.	Caution: pulmonary hypertension, vascular disease Avoid: pheochromocytoma, tachycardia
Epinephrine (adrenaline) (1 mg/1 mL or 1:1000 and 1 mg/10 mL or 1:10,000) Adrenergic stimulant	Any pulseless arrhythmias. Anaphylaxis Positive inotropic and chronotropic action, vasodilation at low doses but vasoconstriction at high doses, mast cell stabiliser, relaxes bronchial smooth muscle.	0.5–1.0 mg slow IV for cardiac arrest 0.1–0.5 mg IM for severe or life-threatening anaphylaxis	Anxiety, dyspnoea, hyperglycaemia, restlessness, palpitations, tachycardia (sometimes with anginal pain), tremors, sweating, hypersalivation, weakness, dizziness, headache and coldness of extremities. α- and β-agonist, digitalis, diuretics, anti-arrhythmics, TCAs, MAOIs, sodium levothyroxine, chlorpheniramine, tripelennamine and diphenhydramine. Patients taking β-blockers may also have an impaired response to epinephrine if it is needed for anaphylaxis.	Caution: tachyarrhythmia and pulmonary hypertension Avoid: nil in cardiac arrest and anaphylaxis
EpiPen (300ug/0.3 mL) EpiPen Junior (150ug/0.3 mL) Adrenergic stimulant	Anaphylaxis Positive inotropic and chronotropic action, vasodilation at low doses but vasoconstriction at high doses, mast cell stabiliser, relaxes bronchial smooth muscle.	0.3 mg IM > 20 kg Repeat every 3–5 min if no response Rapid onset IM Short duration Half-life <5 min	Transient effects include anxiety, over-stimulation, restlessness, tremor, weakness, dizziness, sweating, tachycardia, palpitations, pallor, nausea, headache and respiratory difficulties. Ventricular arrhythmias can occur. α- and β-agonist, digitalis, diuretics, anti-arrhythmics, TCAs, MAOIs, sodium levothyroxine, chlorpheniramine, tripelennamine and diphenhydramine. Patients taking β-blockers may also have an impaired	Caution: not to be administered into buttock, digits, hands, feet or IV Avoid: There are no absolute contraindications in a life-threatening allergic situation

(*Continued*)

Table 1. Continued.

Medication an Drug Class	Indication an Mode of Action	Dose Frequency and Pharmacokinetics	Adverse Reactions and Interactions	Precautions and Contraindications (other than hypersensitivity)
			response to epinephrine if it is needed for anaphylaxis.	
Frusemide (20 mg/2 mL) Loop diuretic	LV failure, acute pulmonary oedema and hypertension Inhibition of sodium and chloride reabsorption in the ascending loop of Henle.	20–40 mg IV or IM 5 min onset Peak 15–30 min Half-life 1.5–2 h Duration 2–3 h	Tinnitus, allergic reaction, nausea, vomiting, dizziness, blurred vision, headache, hypotension and dehydration. Aspirin, diuretics, digoxin, lithium, antihypertensives and NSAIDs.	Caution: kidney or liver disease, diabetes, gout, SLE, pregnancy and after urologic procedures Avoid: Sulphonamide allergy, anuria and sodium or fluid depletion
Glucagon (1 mg powder for solution) Pancreatic hormone	Hypoglycaemia, bradycardia and hypotension. Stimulates release of glucose from liver glycogen stores and inhibits smooth muscle tone.	1 mg SC or IM for hypoglycaemia. 50ug/kg IV for bradycardia and hypotension. 5 min onset Peak 15–30 min Half-life 1.5–2 h Duration 2–3 h	Nauseas, abdominal pain, hypotension, tachycardia, hyperglycaemia, hypoglycaemia. Insulin, indomethacin, warfarin, βblockers.	Caution: the elderly and drug interactions Avoid: hypersensitivity and pheochromocytoma
Lidocaine / Lignocaine (100 mg/ 5 mL) Anti-arrhythmic	VF, pulseless VT and SVT Reduces automaticity without changing contractility (class 1b).	1 mg/kg IV over 1–2 min Repeat after 5 min if required. Onset of action 1 min IV or 3-15 min IM Duration 10–20 min IV Half-life is biphasic at 0.5 and 2 h	Serious adverse effects including the potential to worsen arrhythmia and cardiac failure. Drowsiness, confusion, dyspnoea, nausea and hypotension. Flecainide, disopyramide, phenytoin, propranolol, β-blockers, procainamide and cimetidine.	Caution: bradycardia, heart failure, severe renal impairment and severe hepatic dysfunction Avoid: 2nd- or 3rd-degree block
Metoclopramide (10 mg/2 mL) Dopamine antagonist	Nausea and vomiting. Antagonises dopamine receptors.	10 mg IV or IM for 60 kg and over, 5 mg for less than 60 kg. Maximum of 0.5 mg/kg per day. 10–15 min onset Duration 1–2 h Half-life 4–7 h	Drowsiness, restlessness, diarrhoea, insomnia, depression, tardive dyskinesia. Can increase prolactin levels to stimulate lactation. Alcohol, TCAs, antipsychotics and CNS depressants. Can accelerate gut absorption of some drugs.	Acute complete bowel obstruction
Metoprolol (5 mg/5 mL) Selective β_1 receptor blocker	SVT and MI Competitive antagonism of β receptors in the heart.	5 mg IV over 3 min. Repeat every 5 min as required to a maximum dose of 20 mg. Onset 10–20 min Half-life 3–4 h	Headache, nausea, hypotension, dizziness, confusion, heart failure, heart block, bradycardia, dyspnoea and bronchospasm. Reserpine, other β-blockers, calcium channel blockers, disopyramide and amiodarone.	Caution: asthma, airways disease, metabolic acidosis, cardiogenic shock, hypotension, severe peripheral vascular disease, sinus bradycardia and AV block Avoid: severe bronchospasm, severe bradycardia, overt LV failure
Morphine sulphate (15 mg/1 mL or 30 mg/ 1 mL)	Severe pain Opioid receptor agonist.	SC or IM based on age; <40 7.5 mg, 40–59 5 mg, 60–85 2.5 mg, over 85 2 mg. 5 min onset Peak 20 min	Respiratory depression, hypotension, vomiting, dysphoria, urinary retention, dizziness, sedation, nausea and constipation. Narcotic analgesics, CNS depressants, benzodiazepines and MAOIs.	Caution: renal or liver impairment, pregnancy, seizures, head injuries, asthma, hypotension, hypothyroidism,

(*Continued*)

Table 1. Continued.

Medication an Drug Class	Indication an Mode of Action	Dose Frequency and Pharmacokinetics	Adverse Reactions and Interactions	Precautions and Contraindications (other than hypersensitivity)
Analgesic		Half-life 2 h		pheochromocytoma, addiction and dyspnoea Avoid: respiratory or CNS depression
Naloxone (2 mg/ 5 mL) Opioid antagonist	Opioid overdose Opioid antagonist displaces agonists at the receptor due to higher affinity for mu receptors.	0.4–0.8 mg IV, IM or SC. Repeat as required. 2 min onset IV, 5 min IM Half-life 1–1.5 h Duration 30–60 min	Hypotension, hypertension, ventricular tachycardia, ventricular fibrillation, dyspnoea, pulmonary oedema. Buprenorphine, methohexital	Nil (other than known hypersensitivity) during emergency (opioid overdose)
Nitroglycerin spray (glyceryl trinitrate or GTN) (400 μg/dose) Vasodilator	Acute angina, hypertensive crisis and acute LV failure. Increases exogenous nitric oxide for venodilation.	1–2 sprays of 400 μg each onto or under the tongue. 300–600 μg sublingual tablet or 2–3 mg buccal tablet are alternatives. Repeat after 5 min if required to a maximum of 3 sprays. 1–3 min onset Half-life 2–3 min Duration 30–60 min	Flushing, dizziness, tachycardia and headache. Phosphodiesterase inhibitors, alcohol, antihypertensives and vasodilators.	Caution: phosphodiesterase 5 inhibitors (e.g. Viagra) potentiate the effects of nitric oxide in the corposa cavernosa Avoid: hypotension, hypovolaemia, increased intracranial pressure
Nor-epinephrine (2 mg/2 mL or 1:1000) Adrenergic vasoconstrictor	Hypotension and shock Vasoconstrictor without major cardiac effects.	Start at 8–12 μg/min, then titrate to 2–4 μg/min for maintenance; maximum dose of 30 μg/min if hypotension unresponsive to lower doses. Titrate to effect. Rapid onset IV Short duration Half-life <5 min	Anxiety, palpitations, headache. Cyclopropane, halothane, MAOIs, triptyline or imipramine antidepressants.	Caution: hypovolaemia, hyperthyroidism Avoid: hypertension
Procainamide (100 mg/mL or 500 mg/mL) Anti-arrhythmic	VT Class 1a anti-arrhythmic inhibits post repolarisation recovery to decrease conduction velocity and excitability.	17 mg/kg IV slow bolus (20–30 min) at maximum rate of 50 mg/min. Continue infusion (4 mg/min) until QRS widening >50%, dysrhythmia terminated, onset of hypotension or 17 mg/kg infused. Peak 15–60 min IM Half-life 2.5–5 h	Rapid intravenous dosage may result in severe hypotension, ventricular fibrillation and asystole. High plasma concentrations are also associated with impaired cardiac conduction. Enhance the effects of antihypertensives, other antiarrhythmics, arrhythmogenic drugs, antimuscarinics and neuromuscular blockers, and diminish those of para-sympathomimetics.	Caution: heart failure and hypotension Avoid: heart block and SLE
Promethazine (Phenergan) (50 mg/2 mL) Antihistamine	Allergic reaction Reverse agonist of histamine at H_1 receptors.	25–50 mg IM at 25 mg per minute. Single dose. Onset 5 min IV and 20 min IM Half-life 9–10 h Duration of action 4–6 h	Pain and inflammation at injection site, dizziness, sedation, hypotension, confusion and can cause paradoxical stimulation. CNS depressants, epinephrine, anticholinergics, MAOIs.	Caution: epilepsy, respiratory depression, closed-angle glaucoma Avoid: elderly (reduce dose)

(*Continued*)

Table 1. Continued.

Medication an Drug Class	Indication an Mode of Action	Dose Frequency and Pharmacokinetics	Adverse Reactions and Interactions	Precautions and Contraindications (other than hypersensitivity)
Salbutamol (100ug/dose inhaler or 5 mg/2.5 mL nebuliser) Bronchodilator β_2 receptor agonist	Bronchospasm Anaphylaxis Activates B_2 receptors in the lungs to produce smooth muscle relaxation.	4 inhalations of 100 μg each via spacer (same). Repeat every 4 min as required. 2.5-5 mg by nebuliser. Repeat of required. 5 min onset Peak 60 min Half-life 4–6 h	Tremor, palpitations, tachycardia, anxiety, headaches, peripheral vasodilation, muscle cramps, hyperglycaemia and hypersensitivity. Other β_2 agonists, corticosteroids, diuretics, xanthines, β-blockers and antidepressants.	Nil in emergency
Vasopressin (20 IU/mL) Anti-diuretic	Hypotension Hormone that increases water reabsorption in the tubules.	0.25-1 mL (5–20 units) SC or IM Duration 2–8 h Half-life 10–20 min	Anaphylaxis, hyperhydration, headache, tremor, chest pain, cardiac arrest, peripheral ischaemia, pallor, hypertension, bronchospasm, nausea. Amiodarone, haloperidol, ketamine, diuretics.	Caution: hypertension, peripheral vascular disease. Avoid: hypersensitivity, coronary artery disease.
Verapamil (5 mg/2 mL) Calcium channel blocker	SVT Blocks calcium influx to reduce cardiac conduction and contractility.	5 mg over 2–3 min Repeat after 5–10 min. 3–6 min onset after IV bolus Half-life biphasic at 4 min and 2–5 h	Peripheral oedema, rash, headache, dizziness, nausea, bradycardia and constipation. Other antiarrhythmics, β-blockers, dabigatran, lithium, cimetidine, rifampin, phenobarbital, cyclosporin, carbamazepine.	Caution: heart failure, severe LV dysfunction Avoid: severe bradycardia, sick sinus syndrome, 2^{nd}- and 3^{rd}-degree block, hypotension

SVT is supraventricular tachycardia; VF is ventricular fibrillation; VT is ventricular tachycardia; MI is myocardial infarction; AV is atrioventricular; LV is left ventricular; SLE is systemic lupus erythematosus; CNS is central nervous system; IV is intravenous; IM is intramuscular; SC is subcutaneous; ECG is electrocardiogram; NSAID is non-steroidal anti-inflammatory drug; TCA is tricyclic antidepressant, CNS is central nervous system; MAOI is monoamine oxidase inhibitor; SLE is systemic lupus erythematosus.

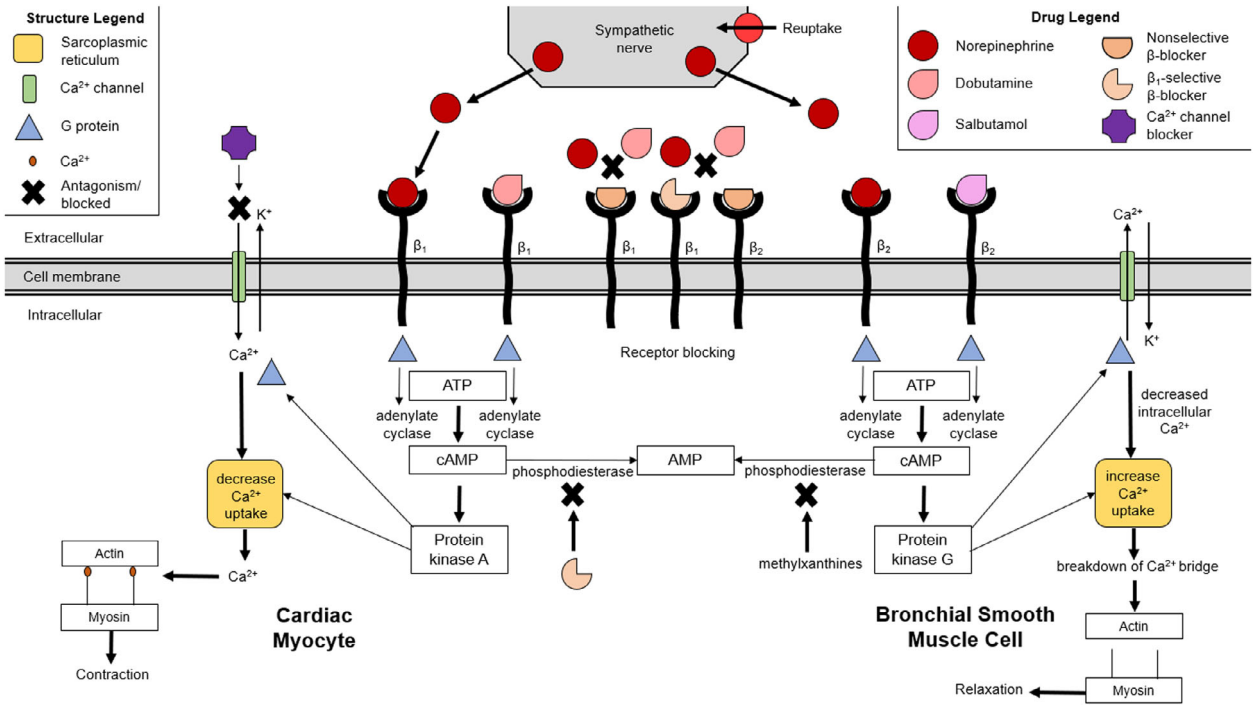


Figure 2. Endogenous norepinephrine and exogenous dobutamine agonise β_1 receptors in a cardiac myocyte. β_2 agonism in a bronchial smooth muscle by norepinephrine or salbutamol produce relaxation. Inhibition of phosphodiesterase conversion of cAMP to AMP by methylxanthines or a β-blocker influences intracellular calcium. β-blockers can be either non-selective (e.g. propranolol or labetalol) or selective (e.g. atenolol for β_1 or butoxamine for β_2). Calcium channel blockers (e.g. verapamil) act to antagonise the voltage-dependent calcium channel to block the inotropic and chronotropic contraction response. Adapted with permission 12.

suitable for acute ischaemic events (Fig. 3). Care should be exercised with use of phosphodiesterase 5 inhibitors (e.g. sildenafil) as it also potentiates the effects of nitric oxide in the corposa cavernosa.

Histamine release from the mast cell is mediated by an interaction between the antigen and the IgE antibody or in response to substance-P. H_1 causes type 1 sensitivity (e.g. urticaria) and bronchoconstriction. Antihistamine refers to H_1 medications that are inverse agonists rather than antagonists. Inflammation and other symptoms of allergic response are managed because antihistamines block the vasodilatory and increased vascular permeability effects of histamine. First-generation antihistamines like diphenhydramine (Benadryl) and promethazine (Phenergan) are often referred to as sedating antihistamines because they cross the blood–brain barrier to cause sedation. Second-generation antihistamines like loratadine, cetirizine and fexofenadine are less-sedating antihistamines because they do not cross the blood–brain barrier.

Furosemide is a loop diuretic (action in the thick ascending loop of Henle) that increases urine volume by inhibiting reabsorption of sodium, potassium and chloride. The value of loop diuretics in an emergency is that they have rapid and potent onset of action but short duration of effect. Since furosemide acts inside the lumen, efficacy is reliant on adequate glomeruli filtration and, therefore, is less effective in severe renal dysfunction/failure. Furosemide has a clinical role in managing symptoms associated with congestive heart failure (e.g. oedema) and in the acute setting, can manage an episode of hypertension.

There are four classes of anti-arrhythmic medications (Fig. 4). Sodium channel blockers (e.g. procainamide and lignocaine) are class I anti-arrhythmics that decrease arterio-ventricular (AV) conduction and decrease contractility. They can be further classified as class IA which have a moderate effect, class IB which have a small effect and class IC which have a pronounced effect. β blockers are the class II anti-arrhythmics that act to

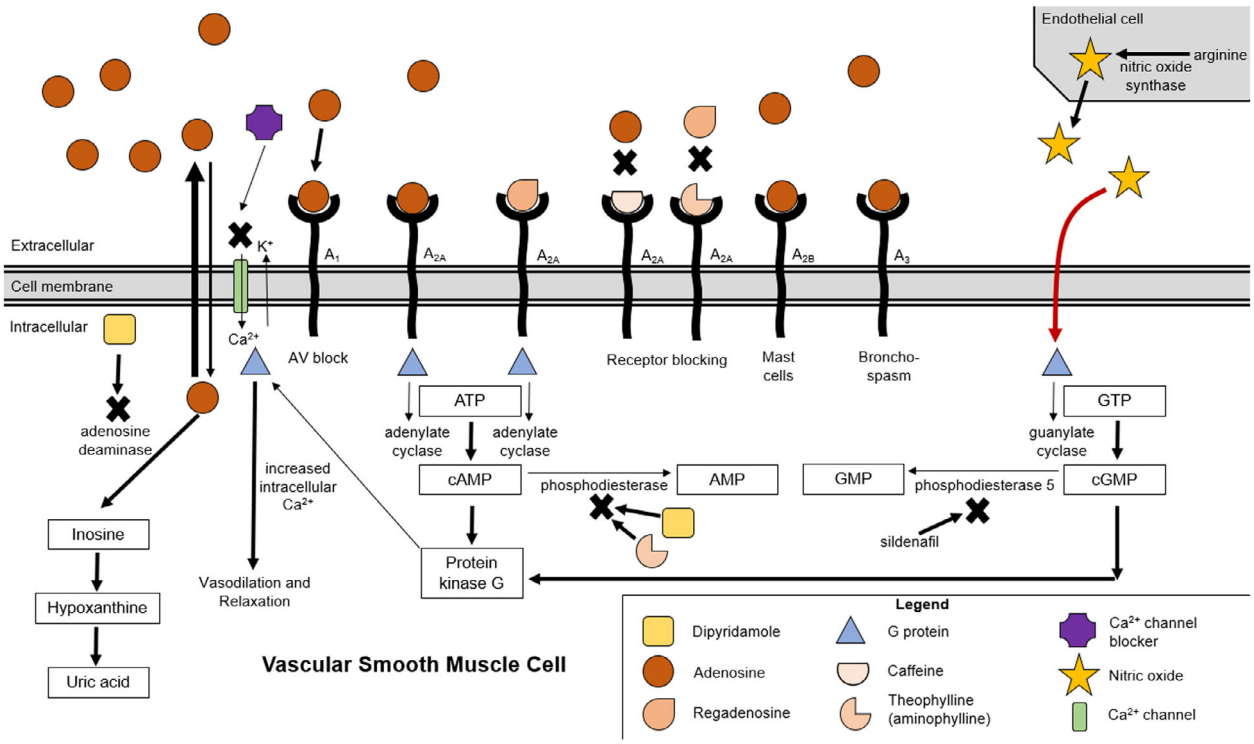


Figure 3. Schematic representation of the action of vasodilating agents adenosine and nitric oxide in a vascular smooth muscle cell. Dipyridamole indirectly produces vasodilation by antagonising adenosine metabolism causing increased availability of adenosine. Inhibition of phosphodiesterase potentiates vasodilation. Adapted with permission 12.

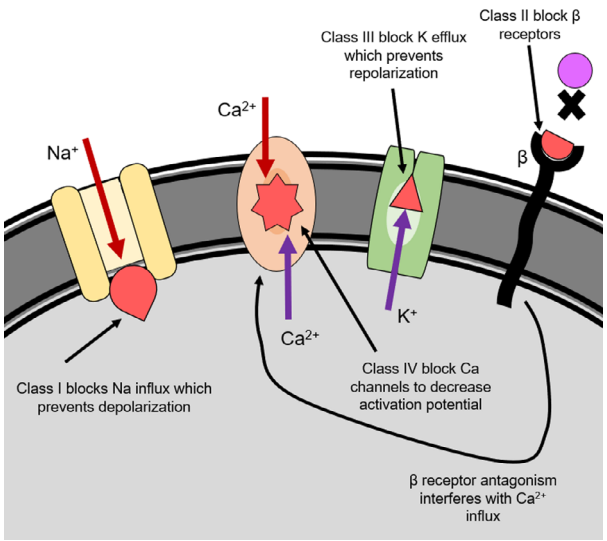


Figure 4. Schematic representation of the effects of anti-arrhythmia drugs with class I blocking the open sodium channel, class II reducing calcium influx by blocking β receptors, class III preventing opening of the potassium channel and class IV blocking the calcium channel. Adapted with permission 12.

decrease AV conduction and decrease the rate and force of contraction (e.g. atenolol, metoprolol). Potassium channel blockers (e.g. amiodarone) are class III anti-arrhythmics that increase the effective refractory period and decrease AV conduction. Calcium channel blockers are the class IV anti-arrhythmics (e.g. diltiazem) that act to decrease the action potential duration, decrease AV conduction and decrease contractility.

Morphine is the prototype opioid analgesic but there are a number of morphine derivatives or other opioid medications used routinely (Table 2). Morphine acts on the opioid receptor (OP) (Fig. 5) and is mostly selective for the μ receptor (OP_3) but at high doses can interact with other opioid receptors (κ and δ). Naloxone and naltrexone are antagonists. The central effects of opioids provide the analgesia but can also suppress the cough reflex, produce respiratory depression, and cause sedation, euphoria/dysphoria, cause nausea, hypotension and bradycardia. The peripheral effects of opioids include constipation, sphincter spasm, suppression of spinal reflexes and release of histamine. Opioids are known for the risk of rapid development of tolerance and dependence.

Table 2. Comparison of various opioid medications.[12–17]

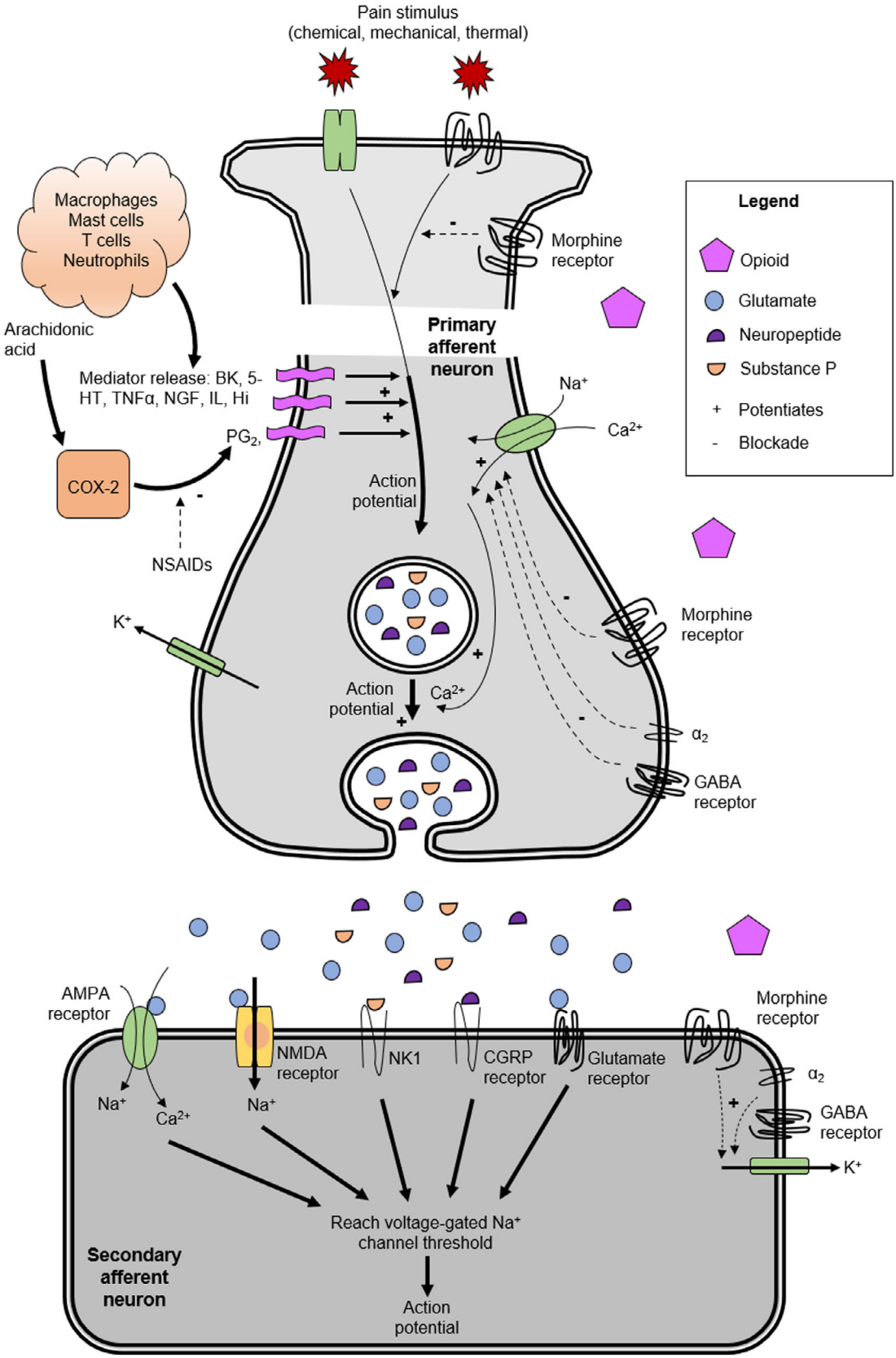
Medication	Administration	Use	Comment	Plasma Half Clearance	Potency Relative to Morphine
Codeine	Oral, IM	Mild pain	Preparation with cough medicines, and with paracetamol or ibuprofen are sub-analgesic	3 h	0.1x
Pethidine	IV, IM, SC	Renal and colic pain or in labour	Can cause paradoxical excitement	3–4 h	0.125x
Tramadol	Oral, IM	Neuropathic pain	Lower abuse risk	4–6 h	0.2x
Morphine	Oral, IM, IV, SC, epidural	Moderate to severe acute and chronic pain	Sustained release formulations available	2–3.5 h	**1x**
Oxycodone	Oral, SC, IV, rectal	Moderate to severe acute and chronic pain including in renal impairment	Oral is controlled release for longer action	2–3 h	1.5–2x
Hydromorphone	Oral, IM, SC, IV	Severe pain	Less sedation	2–3 h	5–7.5x
Methadone	Oral, IM, IV	Severe chronic pain	Long half-life so daily for narcotic rehabilitation	24 h	5–10x
Buprenorphine	IM, sublingual, transdermal patch	Moderate to severe pain		3–5 h	60–80x
Fentanyl	SC, IV, IM, transdermal patch, lozenge	Palliation, moderate to severe acute and chronic pain including in renal impairment	Lozenge for breakthrough pain	3–4 h	100–150x

Benzodiazepines are widely used hypnotics / anxiolytics with diazepam being the prototype drug. Diazepam is a central nervous system (CNS) depressant that has specific functions to reduce anxiety by acting on the gamma-aminobutyric acid (GABA) receptor to increase the inhibitory effects of GABA and further reduce neuron excitability. In emergency scenarios, it is useful to decrease anxiety if required to gain compliance in management of the emergent condition, as an anticonvulsant, and as a muscle relaxant.

Conclusion

The ability of medical radiation practitioners to recognise and respond to (including the use of medicines) the deteriorating patient or circumstances of a medically urgent nature are key capabilities required to meet minimum standards for MRPBA registration and National Safety and Quality Health Service standards. This article, in part, aids the medical radiation practitioner in meeting those standards and enhancing patient safety. Hypersensitivity reactions to contrast media are the most frequent reaction among patients. Recognising and responding to patient reactions significantly reduces risk and morbidity. In the medical emergency, while less frequent, medications form a fraction of the response and management protocol. While this article provides an insight into the pharmacology of emergency medications, a broader mastery of patient care and advanced life support is necessary.

Figure 5. Schematic representation of the response to pain stimulus. Afferent receptors produce an action potential in the neuron that is subsequently enhanced by mediators and sensitising agents. Calcium influx potentiates the action potential and release of glutamate, neuropeptides and substance P into the synaptic cleft where activation of a number of receptors occurs. Activation of opioid receptors in the neuron reduces the initial action potential associated with the primary stimulus and reduces calcium influx in the primary afferent neuron reducing release of neurotransmitters from the vesicle. Activation of the morphine receptor on the secondary afferent neuron increases potassium efflux which reduces sodium and calcium influx to further truncate the pain sensation. It is worth noting the limited action of NSAID medications on pain control. Adapted with permission 12.



Conflict of Interest

There are no funding or conflicts of interest to declare.

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