Pharmacology: Cardiovascular

A 64 year old man with a history of poorly controlled hypertension and ischaemic heart disease is brought to ED by ambulance with sudden onset palpitations and shortness of breath. ECG demonstrates atrial fibrillation and your consultant wishes to perform chemical cardioversion. Which of the following drugs would be most suitable for chemical cardioversion in this patient:

a. Flecainide
b. Amiodarone
c. Propafenone
d. Digoxin
e. Adenosine

See Answer
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A 64 year old man with a history of poorly controlled hypertension and ischaemic heart disease is brought to ED by ambulance with sudden onset palpitations and shortness of breath. ECG demonstrates atrial fibrillation and your consultant wishes to perform chemical cardioversion. Which of the following drugs would be most suitable for chemical cardioversion in this patient:

- A: Flecainide
- B: Amiodarone
- C: Propafenone
- D: Digoxin
- E: Adenosine

Answer
For rhythm control, chemical cardioversion may be appropriate. Class Ic antiarrhythmic drugs such as flecainide or propafenone may be used but are contraindicated in the presence of heart failure, left ventricular impairment, ischaemic heart disease or prolonged QT interval. Amiodarone (300 mg intravenously over 20 - 60 mins followed by 400 mg over 24 h) may be used to attempt chemical cardioversion but is less often effective and takes longer to act. Electrical cardioversion remains an option in this setting and will restore sinus rhythm in more patients than chemical cardioversion.

Notes
Treatment of patients with atrial fibrillation aims to reduce symptoms and prevent complications, especially stroke. Atrial fibrillation may be managed by either controlling the ventricular rate (rate control) or by attempting to restore and maintain sinus rhythm (rhythm control).

New-onset atrial fibrillation
All patients with adverse features suggesting life-threatening haemodynamic instability (shock, syncope, heart failure, myocardial ischaemia) caused by new onset atrial fibrillation should undergo emergency electrical cardioversion with synchronized DC shock without delaying to achieve antiarrhythmic.

In patients presenting acutely (< 48 h) with new-onset AF but without adverse features, immediate treatment options include rate control with drug therapy, rhythm control using drugs to achieve chemical cardioversion, rhythm control by synchronised cardioversion and treatment to prevent complications (e.g. anticoagulation).

- For rate-control, the usual drug of choice is beta-blocker. Digoxin may be used in patients in whom beta-blockade is contraindicated or not tolerated. Digoxin may be used in patients with heart failure. Amiodarone may be used to assist with rate control but is most useful in maintaining rhythm control.
- For rhythm control, chemical cardioversion may be appropriate. Class Ic antiarrhythmic drugs such as flecainide or propafenone may be used but are contraindicated in the presence of heart failure, left ventricular impairment, ischaemic heart disease or prolonged QT interval. Amiodarone (300 mg intravenously over 20 - 60 mins followed by 400 mg over 24 h) may be used to attempt chemical cardioversion but is less often effective and takes longer to act.
- Electrical cardioversion remains an option in this setting and will restore sinus rhythm in more patients than chemical cardioversion.

The longer a person is in AF, the greater the likelihood of atrial thrombus developing. In general, people who have been in AF for > 48 h should not be treated by cardioversion (electrical or chemical) until they have been fully anticoagulated for at least 3 weeks, or unless transoesophageal echocardiography has detected no evidence of atrial thrombus.

Long-term management
In general, rate control is the preferred first-line drug treatment strategy for atrial fibrillation in most patients except in patients with:

- new-onset atrial fibrillation
- heart failure secondary to atrial fibrillation
- atrial flutter suitable for an ablation strategy
- atrial fibrillation with a reversible cause
- or if rhythm control is more suitable based on clinical judgement.

Rate control may be achieved with a beta-blocker or a rate limiting non-dihydropyridine calcium channel blocker e.g. verapamil or diltiazem.

Digoxin is usually only effective for controlling the ventricular rate at rest, and should therefore only be used as monotherapy in predominantly sedentary patients with non-paroxysmal atrial fibrillation or in combination therapy in resistant cases. Digoxin is also used when atrial fibrillation is accompanied by complicating heart failure.

If symptoms are not controlled with a combination of two drugs, a rhythm control strategy should be considered.

All patients with AF should be assessed and managed for risk of stroke and thromboembolism, and risk of bleeding if anticoagulation is being considered.
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Thiazide diuretics act primarily at which of the following sites in the nephron:

- a) Proximal tubule
- b) Ascending limb
- c) Collecting ducts
- d) Early distal tubule
- e) Descending limb

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Pharmacology: Cardiovascular

Thiazide diuretics act primarily at which of the following sites in the nephron:

- Proximal tubule
- Ascending limb
- Collecting ducts
- Early distal tubule
- Descending limb

**Answer**

Thiazide diuretics act mainly on the early segments of distal tubule where they inhibit Na\(^+\)Cl\(^-\) co-transport by binding to the Na\(^+\)/K\(^+\)-ATPase. The increased Na\(^+\) load in the distal tubule stimulates Na\(^+\) exchange with K\(^+\) and H\(^+\), increasing their excretion and causing hyperkalaemia and a metabolic alkalosis. Excretion of Ca\(^{2+}\) is reduced.

**Notes**

Thiazide diuretics are moderately potent diuretics, and are used to relieve oedema in chronic heart failure, and in the management of hypertension. They act within 1 to 2 hours of oral administration and must have a duration of action of 12 to 24 hours.

**Mechanism of action**

Thiazides act mainly on the early segments of distal tubule where they inhibit Na\(^+\)Cl\(^-\) co-transport. Excretion of Cl\(^-\), Na\(^+\) and accompanying water is increased. The increased Na\(^+\) load in the distal tubule stimulates Na\(^+\) exchange with K\(^+\) and H\(^+\), increasing their excretion and causing hyperkalaemia and a metabolic alkalosis. Excretion of Ca\(^{2+}\) is reduced.

**Indications**

Bendrofluazide is used for oedema in mild or moderate heart failure. Combination diuretic therapy (behoop and thiazide diuretics) may be effective in patients with resistant hypertension to treatment with one diuretic.

Thiazide diuretics are licensed for the treatment of hypertension but are no longer considered the first-line diuretic. For this indication, the management of hypertension is a low dose of a thiazide produces a maximal or near-maximal blood pressure lowering effect, with very little biochemical disturbance. Higher doses cause more marked changes in plasma potassium, sodium, urea, acid, glucose, and fluids, with little advantage in blood pressure control.

**Contraindications**

Thiazide diuretics are contraindicated in:

- Addison’s disease
- Hypokalaemia
- Hypomagnesaemia
- Refractory hyperkalaemia
- Symptomatic hyperuricaemia
- Severe hepatic impairment (may precipitate encephalopathy)

**Cautions**

Thiazide diuretics should be used with caution in:

- Diabetes mellitus (may exacerbate)
- Gout (may exacerbate)
- Systemic lupus erythematosus (may exacerbate)
- Hypoaldosteronism
- Mahourism
- Nephrotic syndrome

**Adverse effects**

Lower initial doses of diuretics should be used in the elderly because they are particularly susceptible to the side effects. The dose should then be adjusted according to renal function.

Common side effects of thiazide diuretics include:

- Excessive diuresis
- Postural hypotension, dehydration, renal impairment
- Acid base and electrolyte imbalance
- Hypokalaemia, hypomagnesaemia, hyperuricaemia, hypercalcaemia, hyperphosphataemic alkalosis
- Metabolic imbalance
- Hypocapnia and gout
- Impaired glucose tolerance and hyperglycaemia
- Altered plasma lipid concentrations
- Mitral regurgitation disturbances

**Hypokalaemia**

Hypokalaemia can occur with both thiazide and loop diuretics. The risk of hypokalaemia depends on the duration of action as well as the potency and is thus greater with thiazides than with an equitadose dose of a loop diuretic. Hypokalaemia is dangerous in severe cardiovascular disease and in patients also being treated with proton pump inhibitors. Often the use of potassium sparing diuretics avoids the need to take potassium supplements.
Pharmacology: Cardiovascular

What is the recommended dosing regime for amiodarone in the treatment of a stable regular broad-complex tachycardia:

a 150 mg IV bolus, followed by an IV infusion of 300 mg over next 20 – 60 minutes
b 300 mg IV over 20 – 60 minutes, followed by an IV infusion of 900 mg over the next 24 hours
c 300 mg IV over 20 – 60 minutes, followed by further 300 mg IV infusion over 20 – 60 minutes if no response
d 300 mg IV over 10 – 20 minutes, followed by an IV infusion of 900 mg over the next 24 hours
e 150 mg IV bolus, followed by two further 300 mg IV boluses if no response
Pharmacology: Cardiovascular

Question Exit Exit

What is the recommended dosing regime for amiodarone in the treatment of a stable broad-complex tachycardia?

a) 150 mgIV bolus, followed by an IV infusion of 300 mg over next 20 – 60 minutes
b) 300 mg IV over 20 – 60 minutes, followed by an IV infusion of 100 mg over the next 24 hours
c) 300 mg IV over 20 – 60 minutes, followed by further 300 mg IV infusion over 20 – 60 minutes if no response
d) 300 mg IV over 10 – 20 minutes, followed by an IV infusion of 900 mg over the next 24 hours
e) 150 mg IV bolus, followed by further 300 mg IV if no response

Notes
The approach to the management of tachycardia should follow the Resuscitation Council (UK) guidelines.

If the patient has adverse features
Adverse features:

Shock (hypotension, pale, sweating, cool extremities, collapse, impaired consciousness)
Syncope (transient loss of consciousness)
Hauttönsky (palpitations, oedema, nausea, vomiting, periorbital oedema, haemorrhagic)
Myocardial ischaemia (ischaemic chest pain, ischaemic changes on ECG)

If any adverse features are present, emergency cardiology with a synchronized DC shock is indicated. If a patient fails to respond after cardioversion and adverse features persist, amiodarone 300 mg IV over 10 – 20 minutes should be given and further cardioversion attempted. The lack of clinical improvement may be followed by an infusion of 300mg over 24 hours, given via a large vein.

If the patient has no adverse features
If the patient is stable, the QRS duration should be considered.

If the QRS duration is 0.12 seconds or greater, it is a broad-complex tachycardia.
If the QRS complex is less than 0.12 seconds, it is a narrow-complex tachycardia.

Broad-complex tachycardia

Broad-complex tachycardia is mostly ventricular in origin but may be supraventricular in origin with aberrant conduction.

A regular broad-complex tachycardia is likely to be ventricular tachycardia or a regular supraventricular rhythm with bundle branch block.
A ventricular tachycardia (or broad-complex tachycardia of uncertain origin) should be treated with amiodarone 300 mg IV over 20 – 60 minutes, followed by an infusion of 100 mg over the next 24 hours.
If a ventricularly confirmed SVT with bundle branch block, the patient should be treated as for narrow-complex tachycardia.
A stable patient with an irregular broad-complex tachycardia is most likely to be AF with bundle branch block, although AF with ventricular pre-excitation or polymorphic VT (torsade de pointes) is possible.
Expert help should be sought for the assessment and treatment of irregular broad-complex tachycardia.
Torsade de points VT should be treated by stopping all drugs known to precipitating the QRS interval, correcting electrolyte abnormalities, attempting vagal manoeuvres, 1 mg IV over 30 seconds. Expert help should be sought on other treatment options including extended waveform pacing may be required to prevent paragangia until the arrhythmia has been corrected.

Narrow-complex tachycardia

The narrow-complex tachycardia are supraventricular in origin.

A regular narrow-complex tachycardia may represent paroxysmal SVT or atrial flutter with 2:1 conduction, it may be difficult to differentiate between the two.
The first step in treatment of regular narrow-complex tachycardia is to attempt vagal manoeuvres (nasal oropharyngeal manipulation)
If the heart rhythm persistent, adrenaline 1 mg IV should be given as a rapid bolus using a large cannula and a large vein. The patient should be warned that they will feel hot and may experience chest discomfort for another 30 seconds following the injection. An AECG (preferably multi lead) should be recorded during the injection.
If the ventricular rate does not respond and then sinuatrial again, this may indicate atrial activity and such an atrial flutter or other atrial tachycardia, and this should be treated accordingly.
If there is no response (or no bradycardia slowing or termination of the heart rhythm) to adrenaline 1 mg IV, do not give further adrenaline. If there is no response, see further below 2 mg IV of lignocaine (may occur if the dose is given too slowly or into a peripheral vein).
If adrenaline is contraindicated, or fails to terminate a tachycardia, the administration of verapamil (2.5 – 5 mg IV over 2 minutes should be considered.
Irregular narrow-complex tachycardia in most likely to be AF with ventricular response or, less commonly, atrial flutter with variable AV conduction.
Immediate treatment options include rate control with oral drugs, rhythm control using drugs to achieve chemical or electrical cardioversion, rhythm control by programmed electrical cardioversion and treatment to prevent complications (e.g., aspirin) and Expert help should be sought in determining the most appropriate treatment for this individual patient.
Pharmacology: Cardiovascular

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Captopril is contraindicated in which of the following:

a. Prostatic hypertrophy
b. Renal artery stenosis
c. Heart failure
d. Asthma
e. Second degree heart block

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Captopril is contraindicated in which of the following:

- Prostatic hypertrophy
- Renal artery stenosis
- Heart failure
- Asthma
- Second degree heart block

Answer

In patients with severe bilateral renal artery stenosis (or severe stenosis of the artery supplying a single functioning kidney), ACE inhibitors reduce or abolish glomerular filtration and are likely to cause severe and progressive renal failure. ACE inhibitors are best avoided in patients with known or suspected renovascular disease, unless the blood pressure cannot be controlled by other drugs. If ACE inhibitors are used, they should be initiated only under specialist supervision and renal function should be monitored regularly. ACE inhibitors should also be avoided in patients who may have undiagnosed and clinically silent renovascular disease. This includes patients with peripheral vascular disease or those with severe generalised atherosclerosis.

Notes

Mechanism of action

Angiotensin-converting enzyme inhibitors (ACE inhibitors) e.g. captopril inhibit the conversion of angiotensin I to angiotensin II, and thus have a vasodilatory effect, lowering both arterial and venous resistance. The cardiac output increases and, because the renovascular resistance falls, there is an increase in renal blood flow. This latter effect, together with reduced aldosterone release, increases Na+ and K+ excretion, contracting the blood volume and reducing venous return to the heart.

Blockage of ACE also diminishes the breakdown of the potent vasodilator bradykinin which is the cause of the persistent dry cough. Angiotensin II receptor blockers do not have this effect, therefore they are useful alternative for patients who have to discontinue an ACE inhibitor because of persistent cough.

Indications

ACE inhibitors have many uses and are generally well tolerated. They are indicated for:

- Heart failure
- Hypertension
- Diabetic nephropathy
- Secondary prevention of cardiovascular events

Contraindications

ACE inhibitors should be avoided in:

- History of angioedema associated with previous exposure to an ACE inhibitor
- Recurrent angioedema
- Bilateral renal artery stenosis
- Pregnancy and breastfeeding

Cautions

The use of ACE inhibitors is cautioned in:

- Renal impairment
- Afro-Caribbean patients (may respond less well to ACE inhibitors)
- Peripheral vascular disease or generalised atherosclerosis (risk of clinically silent renovascular disease)
- Primary aldosteronism (patients may respond less well to ACE inhibitors)
- History of idiopathic or hereditary angioedema
- Patients with hypertrophic cardiomyopathy
- Patients with severe or symptomatic aortic stenosis (risk of hypotension)

Concomitant treatment with NSAIDs increases the risk of renal damage, and with potassium-sparing diuretics (potassium-containing salt substitutes) increases the risk of hyperkalaemia. Hyperkalaemia and other side effects of ACE inhibitors are more common in the elderly and those with impaired renal function and the dose may need to be reduced.

Adverse effects

Side effects of ACE inhibitors may include:

- Deterioration in renal function
- Hyperkalaemia
- Hypotension
- Persistent dry cough
- Angioedema (non-allergic drug reaction)
- Dizziness and headache (usually reversible in hypertension)
- Other common adverse effects include abdominal discomfort, dyspepsia, diarrhoea, nausea and vomiting, rash (in particular morbilliform rash), rash, muscles spasms, dyspnoea, chest pain, and fatigue

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Which of the following drugs is first line treatment for a stable regular narrow-complex tachycardia:

a. Amiodarone
b. Adrenaline
c. Adenosine
d. Atropine
e. Flecaïnide

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Pharmacology: Cardiovascular

Which of the following drugs is first-line treatment for a stable regular non-regular complex tachycardia?

a) Adenosine
b) Amiodarone
c) Amiodarone

d) Atropine

e) Bongocin

Answer

The first step in treatment of regular non-complex tachycardia is to attempt vagal manoeuvres (e.g. carotid massage or Valsalva manoeuvre). If the tachycardia is paroxysmal, adenosine 6 mg IV should be given as a rapid bolus using a large cannula and a large syringe. If there is no response (i.e. no slowing of ventricular rate or termination of the tachycardia), adenosine 6 mg IV should be repeated. If this is also ineffective, one further 12 mg IV should be given (max 30 mg).

Notes

The approach to the management of arrhythmias should follow the Resuscitation Council guidelines.

If the patient has adverse features

Adverse Features:
- Shock (hypotension, pallor, sweating, cold mottled skin, confusion, impaired consciousness)
- Sepsis (transient loss of consciousness)
- Heart failure (pulmonary oedema, cardiac / peripheral oedema, hypotension)

Pharmacological haemorrhage (cyanish skin pallor, skin changes on ECG)

If any adverse features are present, emergency cardioversion with a synchronized DC shock is indicated. If cardioversion fails to terminate the arrhythmia and adverse features persist, adenosine 300 mg IV over 30–60 minutes should be given and further cardioversion attempted. The loading dose of adenosine can be followed by an infusion of 60 mg per hour, given via a large syringe.

If the patient has no adverse features

If the patient is stable, the QRST duration should be assessed.
- If the QRST duration is less than 0.2 seconds, it is a broad-complex tachycardia.
- If the QRST duration is less than 0.2 seconds, it is a narrow-complex tachycardia.

Broad-complex tachycardia

Broad-complex tachycardias are mostly ventricular in origin but may be a supraventricular rhythm with aberrant conduction.

- A regular broad-complex tachycardia is likely to be a ventricular tachycardia or a supraventricular rhythm with bundle branch block.
- A ventricular tachycardia for broad-complex tachycardia of auricular origin should be treated with adenosine: 300 mg IV over 5–10 seconds, followed by an infusion of 900 mg over the next 24 hours.
- If the ventricular rhythm is confirmed as SVT with bundle branch block, the patient should be treated as for narrow-complex tachycardia.
- A stable patient with an irregular broad-complex tachycardia is most likely to be in AF with bundle branch block, although AF with ventricular pre-excitation or polymorphic VT (tambous de polymorph is possible).
- Expert help should be sought for the assessment and treatment of irregular broad-complex tachycardia.
- Teradek de points VT should be treated by stopping all drugs known to prolong the QT interval, correcting electrolyte abnormalities, and giving magnesium sulfate 2 g IV over 30 minutes. Expert help should be sought in other treatment options including ventricular pacing may be required to prevent ventricular rate even when a rhythm has been corrected.

Narrow-complex tachycardia

The narrow-complex tachycardias are supraventricular in origin.

- A regular narrow-complex tachycardia may represent atrial flutter or atrial fibrillation with a 2:1 conduction. It may be difficult to differentiate between the two.
- The first step in treatment of regular narrow-complex tachycardia is to attempt vagal manoeuvres (e.g. carotid massage or Valsalva manoeuvre).
- If the tachycardia is paroxysmal, adenosine 6 mg IV should be given as a rapid bolus using a large cannula and a large syringe. If there is no response, further 12 mg IV should be given (max 30 mg). Lack of response to adenosine will now occur is the tachycardia is due to atrial arrhythmia, and this should be treated accordingly.
- If atrial fibrillation is suspected, it is those slowing or termination of the arrhythmia to adenosine 6 mg IV, a 12 mg IV should be given and if there is no response, one further 12 mg IV should be given (max 30 mg). Lack of response to adenosine will now occur is the tachycardia is due to atrial arrhythmia and this should be treated accordingly.
- If atrial fibrillation is confirmed, cardioversion with the vagus nerve to 200 mg/m² is over 20 seconds should be considered.
- If the irregular narrow-complex tachycardia is most likely to be AF with fast ventricular response or less commonly, atrial flutter with variable AV conduction.
- Immediate treatment options include rate control with beta-blockers, rhythm control using drugs to achieve chemical cardioversion, or synchronized cardioversion and treatment to prevent complications (i.e. anticoagulation). Expert help should be sought in determining the most appropriate treatment for the individual patient.
Pharmacology: Cardiovascular

An 80 year old man requires treatment with an antibiotic. He takes warfarin for atrial fibrillation. What antibiotic is the safest choice for this patient:

a. Ciprofloxacin
b. Co-trimodazole
c. Clarithromycin
d. Doxycycline
e. Cephalixin

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Pharmacology: Cardiovascular

Chapter 22

You can now find more information on this topic on the FRCM Success website.

Pharmacology: Cardiovascular

Question 1

A 22-year-old woman presents to your clinic with complaints of shortness of breath and dizziness. She has been recently diagnosed with type 2 diabetes and her vital signs are: BP 130/80 mmHg, HR 85 bpm, RR 16 breaths/min, and T 98°F. You decide to prescribe a medication to manage her blood pressure.

Question 2

A 65-year-old man presents to your clinic with complaints of shortness of breath and dizziness. He has been recently diagnosed with type 2 diabetes and his vital signs are: BP 150/90 mmHg, HR 100 bpm, RR 20 breaths/min, and T 98°F. You decide to prescribe a medication to manage his blood pressure.

Question 3

A 55-year-old woman presents to your clinic with complaints of shortness of breath and dizziness. She has been recently diagnosed with type 2 diabetes and her vital signs are: BP 140/90 mmHg, HR 75 bpm, RR 18 breaths/min, and T 98°F. You decide to prescribe a medication to manage her blood pressure.

Question 4

A 75-year-old man presents to your clinic with complaints of shortness of breath and dizziness. He has been recently diagnosed with type 2 diabetes and his vital signs are: BP 160/100 mmHg, HR 90 bpm, RR 22 breaths/min, and T 98°F. You decide to prescribe a medication to manage his blood pressure.

Question 5

A 45-year-old woman presents to your clinic with complaints of shortness of breath and dizziness. She has been recently diagnosed with type 2 diabetes and her vital signs are: BP 135/85 mmHg, HR 80 bpm, RR 14 breaths/min, and T 98°F. You decide to prescribe a medication to manage her blood pressure.

Question 6

A 60-year-old man presents to your clinic with complaints of shortness of breath and dizziness. He has been recently diagnosed with type 2 diabetes and his vital signs are: BP 145/95 mmHg, HR 95 bpm, RR 20 breaths/min, and T 98°F. You decide to prescribe a medication to manage his blood pressure.

Question 7

A 50-year-old woman presents to your clinic with complaints of shortness of breath and dizziness. She has been recently diagnosed with type 2 diabetes and her vital signs are: BP 150/95 mmHg, HR 90 bpm, RR 20 breaths/min, and T 98°F. You decide to prescribe a medication to manage her blood pressure.

Question 8

A 70-year-old man presents to your clinic with complaints of shortness of breath and dizziness. He has been recently diagnosed with type 2 diabetes and his vital signs are: BP 160/110 mmHg, HR 100 bpm, RR 24 breaths/min, and T 98°F. You decide to prescribe a medication to manage his blood pressure.

Question 9

A 40-year-old woman presents to your clinic with complaints of shortness of breath and dizziness. She has been recently diagnosed with type 2 diabetes and her vital signs are: BP 135/80 mmHg, HR 85 bpm, RR 16 breaths/min, and T 98°F. You decide to prescribe a medication to manage her blood pressure.

Question 10

A 75-year-old man presents to your clinic with complaints of shortness of breath and dizziness. He has been recently diagnosed with type 2 diabetes and his vital signs are: BP 160/105 mmHg, HR 105 bpm, RR 26 breaths/min, and T 98°F. You decide to prescribe a medication to manage his blood pressure.
Pharmacology: Cardiovascular

What is the main mechanism of action of dopamine as an inotropic sympathomimetic:

a. Dopamine receptor agonist
b. Beta1-receptor agonist
c. Beta2-receptor agonist
d. Alpha1-receptor agonist
e. Alpha2-receptor agonist

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What is the main mechanism of action of dopamine as an inotrope?

- a) Dopamine receptor agonist
- b) Beta-1 receptor agonist
- c) Beta-2 receptor agonist
- d) Alpha-1 receptor agonist
- e) Alpha-2 receptor agonist

Answer

Dopamine is a neurotransmitter and a metabolic precursor of the catecholamines. It acts on beta-1 receptors in cardiac muscle increasing cardiac contractility, and increases renal perfusion by stimulating dopamine receptors in the renal vasculature. This is of benefit in cardiogenic shock where deterioration of renal function is common.

Notes

Shock is a medical emergency associated with a high mortality. The underlying causes of shock such as haemorrhage, sepsis, or myocardial insufficiency should be corrected. The profound hypotension of shock must be treated promptly to prevent tissue hypoxia and organ failure. Volume replacement is essential to correct the hypovolaemia associated with haemorrhage and sepsis but may be detrimental in cardiogenic shock.

Inotropic sympathomimetics

Depending on haemodynamic status, cardiac output may be improved by the use of sympathomimetic inotropes such as adrenaline/epinephrine, dobutamine or dopamine.

In septic shock, when fluid replacement and inotropic support fail to maintain blood pressure, the vasconstrictor noradrenaline/norepinephrine may be considered. In cardiogenic shock peripheral resistance is frequently high and to raise it further may worsen myocardial performance and exacerbate tissue ischaemia.

- Dobutamine directly stimulates the beta-1 adrenergic receptors in the heart and increases contractility and cardiac output with little effect on the rate. In addition action on beta-2 receptors causes vasodilation.
- Dopamine is a neurotransmitter and a metabolic precursor of the catecholamines. It acts on beta-1 receptors in cardiac muscle increasing cardiac contractility, and increases renal perfusion by stimulating dopamine receptors in the renal vasculature. This is of benefit in cardiogenic shock where deterioration of renal function is common.
- Epinephrine increases blood pressure by stimulating the rate and force of the heartbeat (beta-1 effects). Stimulation of vascular alpha-receptors causes vasoconstriction (viscera, skin) but beta-2 receptor stimulation causes vasodilation (skeletal muscle) and the total peripheral resistance may actually decrease.
- Norepinephrine has little or no effect on the vascular beta2-receptors, and so the alpha-mediated vasoconstriction is unopposed. The resulting rise in blood pressure reflexively slows the heart.

The use of sympathomimetic inotropes and vasconstritors should preferably be confined to the intensive care setting and undertaken with invasive haemodynamic monitoring.

Vasoconstrictor sympathomimetics

Vasconstrictor sympathomimetics, such as ephedrine and metaraminol, raise blood pressure transiently by acting on alpha-adrenergic receptors to constrict peripheral vessels. They are sometimes used as an emergency method of elevating blood pressure where other measures have failed. Their use is limited as although they raise the blood pressure they also reduce organ perfusion.
Pharmacology: Cardiovascular

In adult basic life support, chest compressions should be performed at which of the following rates:

- a 60 – 80 per minute
- b 60 – 70 per minute
- c 90 – 100 per minute
- d 80 – 100 per minute
- e 100 – 120 per minute
Pharmacology: Cardiovascular

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In adult basic life support, chest compressions should be performed at which of the following rates:

a) 60 – 80 per minute
b) 60 – 70 per minute
c) 90 – 100 per minute
d) 80 – 100 per minute
e) 100 – 120 per minute

Answer

Chest compressions should be performed at a rate of 100 – 120 per minute.

Notes

Basic life support

- For chest compressions in adult basic life support the heel of one hand should be placed over the middle of the lower half of the patient’s sternum with the other hand on top.
- The sternum should be depressed to a depth of 5 – 6 cm.
- Chest compressions should be performed at a rate of 100 – 120 per minute.
- Thirty compressions should be given before two breaths and that ratio continued (30:2).
- The person giving CPR should be changed every 2 minutes if possible to avoid exhaustion and poor quality chest compressions.
- Interruptions should be minimised (pauses should be < 5 seconds).

Advanced life support

- In adult advanced life support, basic life support should continue whilst the defibrillator pads are attached and the airway managed.
- The pads should be positioned one to the right of the upper sternum below the clavicle and the other in the left midaxillary line in the 5th intercostal space.
- Chest compressions should be paused briefly (< 5 sec) to assess the rhythm and then continued as the defibrillator charges if a shockable rhythm is identified or continued for a further two whole minutes if a non-shockable rhythm is identified.
- When delivering a shock, everybody should be clear of the patient (and any GTN patches and oxygen sources removed).
- Once the shock has been delivered, compressions should resume immediately and continue for a further two minutes before checking the rhythm again or feeling for a pulse.

Shockable rhythm (ventricular fibrillation or pulseless ventricular tachycardia)

- IV adrenaline 1 mg (10 mL of 1:10,000 solution) should be given after 3 shocks and every 3 – 5 minutes/after alternate shocks thereafter.
- IV amiodarone 300 mg should be given after 3 shocks. A further dose of IV amiodarone 150 mg may be considered after a total of five defibrillation attempts. Lidocaine (1 mg/kg/sup>1 mg/sup) may be used as an alternative if amiodarone is not available but may not be given if amiodarone has been given already.

Non-shockable rhythm (asystole or pulseless electrical activity)

- IV adrenaline 1 mg should be given as soon as IV access is achieved and then given every 3 – 5 minutes/after alternate shocks thereafter.
Pharmacology: Cardiovascular

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What is the most common side effect of verapamil:

a) Postural hypotension
b) Constipation
c) Ankle swelling
d) Bronchospasm
e) Hyperkalaemia

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What is the most common side effect of verapamil?

- a] Postural hypertension X
- b] Constipation
- c] Ankle swelling
- d] Bradycardia
- e] Hyperkalemia

Answer

Verapamil is used for the treatment of angina, hypertension, and arrhythmias. Verapamil is highly negatively inotropic and reduces cardiac output, slows the heart rate and may impair atrioventricular conduction. It may precipitate heart failure, exacerbate conduction disorders, and cause hypertension at high doses and should not be used with beta-blockers. Constipation is the most common side effect.

Notes

Calcium channel blockers are widely used in the treatment of angina (second line to beta-blockers) and also for hypertension, heart failure and arrhythmias.

Calcium channel blockers vary widely in their predilection for the various possible sites of action and in their therapeutic effects and may be divided into the dihydropyridine type (e.g. amlodipine, nifedipine and nisoldipine) and the rate-limiting non-dihydropyridine type (e.g. verapamil, diltiazem).

Mechanism of action

Calcium channel blockers inhibit L-type voltage-sensitive calcium channels in smooth muscle, causing relaxation and vasoconstriction. They also block calcium channels within the myocardium and conducting tissues of the heart which produces a negative inotropic effect by reducing calcium influx during the plateau phase of the action potential.

The dihydropyridines have relatively little effect on the heart because they have a much higher affinity for vascular smooth muscle than for cardiac muscle. Furthermore, at clinical doses, vasoconstriction results in a net increase in sympathetically-toned tissue that counteracts the mild negative inotropic effect. The non-dihydropyridines are rate-limiting calcium-channel blockers that depress the sinoatrial node and slow conduction in the atrioventricular nodal tissue, causing a mild resting bradycardia.

Contraindication

Non-dihydropyridine CCBs:
- Atrial fibrillation or flutter
- Heart failure or history of heart failure (may precipitate or aggravate symptoms)
- Cardiac outflow obstruction e.g. hyperelective aortic stenosis or obstructive hypertrophic cardiomyopathy (may result in reduced coronary output)
- Second or third degree AV block (may induce complete AV block)
- Severe bradycardia
- Sick sinus syndrome

Dihydropyridine CCBs:
- Uncontrolled heart failure
- Severe hypertension
- Cardiac outflow obstruction

Adverse effects

- Gastrointestinal adverse effects – constipation, nausea, dyspepsia
- Bradycardia, All block, reflex tachycardia, palpitations
- Vasoconstrictor adverse effects – flushing, dizziness, headache, postural hypertension, ankle swelling (more common with dihydropyridine calcium channel blockers and often improves with continued use, although ankle swelling often persists)
- Gingival hyperplasia
- Muscle cramps
- Myalgia and arthralgia

Verapamil

Verapamil is used for the treatment of angina, hypertension, and arrhythmias. Verapamil is highly negatively inotropic and reduces cardiac output, slows the heart rate and may impair atrioventricular conduction. It may precipitate heart failure, exacerbate conduction disorders, and cause hypertension at high doses and should not be used with beta-blockers. Constipation is the most common side effect.

Nifedipine

Nifedipine relaxes vascular smooth muscle and dilates coronary and peripheral arteries. Nifedipine has less myocardial effects than verapamil and has no antiarrhythmic properties but has less influence on the vessel. Unlike verapamil, it rarely precipitates heart failure because any negative inotropic effect is offset by a reduction in left ventricular work.

Nitrates

Nitrates are related to nifedipine but the smooth muscle relaxant effects preferentially act on coronary arteries. It is used solely for the prevention and treatment of vascular spasm following anoxia such as should happen in coronary arteries.
Pharmacology: Cardiovascular

Question 10 of 121

What is the maximum recommended dose of atropine that may be given in the treatment of bradyarrhythmias associated with adverse features or a risk of asystole:

a 3 g
b 300 mcg
c 3 mg
d 30 mg
e 0.3 mg

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- Resuscitation Council (UK)
- TeachMeAnatomy
- Trauma.org
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Advanced Life Support Group
- Emergency Medicine Journal
- Lifeinthefastlane
- Instant Anatomy
- Patient.co.uk

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Pharmacology: Cardiovascular

Question 10 of 121

What is the maximum recommended dose of atropine that may be given in the treatment of bradyarrhythmias associated with adverse features or a risk of asystole:

a) 3 g  
X  
b) 300 mcg

c) 3 mg  
✓
d) 30 mg

e) 0.3 mg

Answer

If there are adverse features or a risk of asystole, atropine 500 mcg IV bolus should be given. If there is an unsatisfactory response, this can be repeated every 3 - 5 mins up to a maximum dose of 3 mg. Atropine should be used cautiously in the presence of acute myocardial ischaemia or myocardial infarction as the resulting increase in heart rate may worsen ischaemia or increase the size of the infarct.

Notes

The approach to the management of bradyarrhythmias should follow the Resuscitation Council guidelines.

If there are no adverse features (shock, syncope, myocardial ischaemia or heart failure) and no risk of asystole (recent asystole, Mobitz II AV block, complete heart block with broad QRS, ventricular pause > 3 seconds), immediate treatment can be delayed and the patient assessed to try and identify the cause of the bradycardia.

If there are adverse features or a risk of asystole, atropine 500 mcg IV bolus should be given. If there is an unsatisfactory response, this can be repeated every 3 - 5 mins up to a maximum dose of 3 mg. Atropine should be used cautiously in the presence of acute myocardial ischaemia or myocardial infarction as the resulting increase in heart rate may worsen ischaemia or increase the size of the infarct.

Other interim measures may include other drugs such as isoprenaline or adrenaline (or alternatively aminophylline, dopamine, glucagon (if beta-blocker or calcium channel blocker overdose) or glycopyrrolate). For a patient with bradycardia and adverse features, if there is no response to atropine, or if atropine is contraindicated, transcutaneous pacing should be initiated immediately. In the presence of life-threatening, extreme bradycardia, percussion pacing should be used as an interim measure until transcutaneous pacing is achieved.

Expert help should be sought and ultimately transvenous pacing arranged.
Pharmacology: Cardiovascular

Question 11 of 121

Which of the following drugs is first line treatment for a stable regular broad-complex tachycardia:

- (a) Amiodarone
- (b) Adrenaline
- (c) Adenosine
- (d) Atropine
- (e) Flecaïnide

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Pharmacology: Cardiovascular

Question 11 of 25

Which of the following drugs is first-line treatment for a stable regular broad-complex tachycardia?

a) Adenosine
b) Amiodarone
c) Atropine
d) Plasmocine

Answer

A regular broad-complex tachycardia is likely to be ventricular tachycardia or an irregular supraventricular tachycardia with bundle branch block. A ventricular tachycardia is a broad complex tachycardia of uncertain origin should be treated with adenosine 300 mg IV over 20 – 60s, followed by an infusion of 900 mg over the next 24 hours. If, after 60s or 20 mins, effectiveness confirmed as SVT with bundle branch block, the patient should be treated as for narrow complex tachycardia.

Notes

The approach to the management of tachycardias should follow the Resuscitation Council Guidelines.

If the patient has adverse features

Adverse Features:

- Shock, hypotension, pulmonary oedema, cold diaphoresis, confusion, impaired consciousness
- Sepsis (transient loss of consciousness)
- Heart failure (pulmonary oedema, cardiac AV node, peripheral anaemia, hypotension)
- Atrial fibrillation (arrhythmia, chest pain, haemodynamic changes on ECG)

If any adverse feature is present, emergency cardioversion with a synchronized DC shock is indicated. If cardioversion fails to terminate the tachycardia and adverse features persist, adenosine 300 mg IV over 20 – 60s should be given and further cardioversion attempted. The loading dose of adenosine can be followed by an infusion of 900 mg over 24 hours, given via a large vein.

If the patient has no adverse features

If the patient is stable, the QRS duration should be considered.

If the QRS duration is 0.12 seconds or greater, it is a broad complex tachycardia.

If the QRS duration is less than 0.12 seconds, it is a narrow-complex tachycardia.

Broad-complex tachycardia

Broad-complex tachycardias are mostly ventricular in origin but may be a supraventricular arrhythmia with aberrant conduction.

- A regular broad-complex tachycardia is likely to be ventricular tachycardia or a regular supraventricular rhythm with bundle branch block.
- A ventricular tachycardia for broad-complex tachycardia of uncertain origin should be treated with adenosine 300 mg IV over 20 – 60 mins, followed by an infusion of 900 mg over the next 24 hours.
- Fibrillation confirmed as SVT with bundle branch block, the patient should be treated as for narrow complex tachycardia.
- A stable patient with an irregular broad-complex tachycardia is most likely to be AF with bundle branch block, although AF with ventricular pre-ectopic or polymorphic VT (bordeaux de pointes) is possible.
- Expert help should be sought for the assessment and treatment of irregular broad-complex tachycardia.
- Torsade de pointes VT should be treated by stopping all drugs known to prolong QT interval, connecting electrolyte abnormalities, and giving magnesium sulfate 2 g IV over 30 minutes. Expert help should be sought in other treatment options including inotrope supportive care may be required to prevent relapse once the arrhythmia has been corrected.

Narrow-complex tachycardia

The narrow-complex tachycardia are supraventricular in origin.

- A regular normal-complex tachycardia may represent preexcitation SVT or atrial flutter with 2:1 conduction. It may be difficult to differentiate between the two.
- The first step in the treatment of normal narrow-complex tachycardia is to attempt vagal manoeuvres (cold nose or massage of the carotid sinus).
- If the pre-excitation remains, adenosine 6 mg IV should be given as a rapid bolus using a large canula and a large syringe. The patient should be warned that they will feel lightheaded and may experience chest discomfort for a few seconds following the injection. An ECG (preferably multi-lead) should be recorded during the injection.
- If the ventricular rate slows to below 160bpm then adenosine may be repeated. This will increase the likelihood of atrial flutter, atrial fibrillation or atrial electrical activity, and this should be treated accordingly.
- If this is not responsive or if the ventricular slowing or termination of the tachycardia is to adenosine 6 mg IV or a 3.5 mg bolus it should be given again. If there is no response, one further 3.5 mg bolus should be given (max 30mg). Lack of response to adenosine will or the is a tachycardia in which flow due to slow ventricular response or less commonly, atrial flutter with variable AV conduction.
- Immediate treatment options include rate control with beta-blockers, rhythm control using drugs to achieve chemical cardioversion, rhythm control by synchronized cardioversion and treatment to prevent complications (e.g. anticoagulation). Expert help should be sought in determining the most appropriate treatment for the individual patient.
Pharmacology: Cardiovascular

Question 12 of 121

What is the main mechanism of action of enoxaparin:

a. Inhibits vitamin K dependent clotting factors
b. Inhibits factor Xa
c. Potentiate effects of antithrombin
d. Directly inhibits thrombin
e. Blocks GPIIb/IIIa receptor sites

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Pharmacology: Cardiovascular

Question 52 of 121

What is the main mechanism of action of enoxaparin?

a) Inhibits vitamin K-dependent clotting factors
b) Inhibits factor Xa    ✓
c) Potentiates effects of antithrombins
D) Directly inhibits thrombin
e) Blocks GP IIb/IIIa receptor sites    ✓

Answer

Heparin potentiates the activity of antithrombin III causing inactivation of thrombin. The heparin-antithrombin III complex also inhibits factor Xa and some other factors. Low molecular weight heparin (LMWH) preparations inhibit only factor Xa.

Notes

The main use of anticoagulants is to prevent thrombus formation or extension of an existing thrombus in the lower-reveing vs sides of the circulation, where the thrombus consists of fibrin to which are attached platelets and red cells. Anticoagulants are not as useful in preventing thrombus formation in arteries, or in faster-moving vessels such as the coronary arteries, or the cerebral arteries.

Mechanism of action

Heparin potentiates the activity of antithrombin III causing inactivation of thrombin. The heparin-antithrombin III complex also inhibits factor Xa and some other factors. Low molecular weight heparin (LMWH) preparations inhibit only factor Xa. PT and APTT may be prolonged but the PT loss is small.

Contraindications

Heparins are contraindicated:

- In people with current or history of heparin-induced thrombocytopenia
- In people with severe fibrotic endocarditis
- In people with active major bleeding, and conditions with a high risk of uncontrolled bleeding, including recent non-cardiac surgery, major trauma, recent brain, spinal cord or eye surgery, hemorrhage and thrombocytopenia
- In people with active gastrointestinal or duodenal ulceration

Adverse effects

- Bleeding
- Heparin-induced thrombocytopenia (thrombocytopenia that usually develops after 5 – 10 days, signs may include a 50% reduction of platelet count, thrombocytopenia, or skin allergy; if HIT is suspected or confirmed, heparin should be discontinued and an alternative anticoagulant given)
- Hyperkalemia (due to inhibition of aldosterone secretion; patients with aldosterone deficiency, chronic renal failure, acidosis, rapid plasma potassium or these taking potassium sparing drugs seems to be more susceptible)
- Osteoporosis (risk lower with LMWH)
- Arthritis
- Hypersensitivity reactions
- Injection site reactions

Low molecular weight heparin vs unfractionated heparin

Unfractionated heparin is usually given by continuous intravenous infusion for the smoothest control and the treatment of choice where rapid reversal of anticoagulation may be required (e.g. in surgical patients or late pregnancy). Therapy is monitored by monitoring the APTT at 1.5 - 2.5 times the upper limit of normal.

Low molecular weight heparin (LMWH) preparations have largely replaced unfractionated heparin.

Advantages of LMWH

- Greater ability to inhibit factor Xa directly interacting loss with platelets and so may have a lower tendency to cause bleeding
- Greater bioavailability and longer half-life in plasma making once-daily subcutaneous administration possible
- More predictable dose response avoiding the need for routine anticoagulant monitoring
- Lower associated risk of heparin-induced thrombocytopenia or of osteoporosis

Haemorrhage

Because it has a short duration of action, if haemorrhage occurs it is usually sufficient to withdraw unfractionated or low molecular weight heparin, but if rapid reversal of the effects of the heparin is required, protamine sulphate is a specific antidote (but only partially reverses the effects of low molecular weight heparin).

Resources

- The Royal College of Emergency Medicine
- Visit Association for Emergency Medicine
- Advanced Trauma Life Support
- Association of Civilian EM
- Trauma Advocacy
- Emergency Medicine
- Resuscitation

- Advanced Life Support Group
- Emergency Medicine Journal
- Undergraduate Medicine
- Patents.co.uk

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Pharmacology: Cardiovascular

Question 13 of 121

What is the initial recommended dose for atropine in the treatment of bradyarrhythmias associated with adverse features or a risk of asystole:

a 400 micrograms
b 500 micrograms
c 1 mg
d 5 mg
e 6 mg

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Resources
- The Royal College of Emergency Medicine
- Irish Association for Emergency Medicine
- Advanced Trauma Life Support
- Resuscitation Council (UK)
- TeachMeAnatomy
- Trauma.org
- Radiopaedia
- Advanced Life Support Group
- Emergency Medicine Journal
- Lifesinthefastlane
- Instant Anatomy
- Patient.co.uk
Pharmacology: Cardiovascular

Question 13 of 121

What is the initial recommended dose for atropine in the treatment of bradycardia associated with adverse features or a risk of asystole:

a) 400 micrograms
b) 500 micrograms ✗
c) 1 mg
d) 5 mg
e) 6 mg

Answer

If there are adverse features or a risk of asystole, atropine 500 mcg IV bolus should be given. If there is an unsatisfactory response, this can be repeated every 3 – 5 mins up to a maximum dose of 3 mg. Atropine should be used cautiously in the presence of acute myocardial ischaemia or myocardial infarction as the resulting increase in heart rate may worsen ischaemia or increase the size of the infarct.

Notes

The approach to the management of bradycardias should follow the Resuscitation Council guidelines.

If there are no adverse features (shock, syncope, myocardial ischaemia or heart failure) and no risk of asystole (recent asystole, Mobitz II AV block, complete heart block with broad QRS, ventricular pause > 3 seconds), immediate treatment can be delayed and the patient assessed to try and identify the cause of the bradycardia.

If there are adverse features or a risk of asystole, atropine 500 mcg IV bolus should be given. If there is an unsatisfactory response, this can be repeated every 3 – 5 mins up to a maximum dose of 3 mg. Atropine should be used cautiously in the presence of acute myocardial ischaemia or myocardial infarction as the resulting increase in heart rate may worsen ischaemia or increase the size of the infarct.

Other interim measures may include other drugs such as isoprenaline or adrenaline (or alternately aminophylline, dopamine, glucagon (if beta-blocker or calcium channel blocker overdose) or glycopyrrolate). For a patient with bradycardia and adverse features, if there is no response to atropine, or if atropine is contraindicated, transcutaneous pacing should be initiated immediately. In the presence of life-threatening, extreme bradycardia, percussion pacing should be used as an interim measure until transcutaneous pacing is achieved.

Expert help should be sought and ultimately transvenous pacing arranged.
Pharmacology: Cardiovascular

Question 14 of 121

A 67-year-old man is being treated for atrial fibrillation with digoxin. If his serum digoxin levels are above the therapeutic range, he is at highest risk for developing digoxin toxicity if he also develops:

a. Hyponatraemia
b. Vitamin B12 deficiency
c. Hypokalaemia
d. Hypocalcaemia
e. Hypophosphataemia

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**Question 269**

Ketamine is given intravenously. This has a speed of onset faster than the most commonly used drugs. Ketamine is most commonly used in anesthetic procedures and is sometimes administered in Emergency Departments.
Pharmacology: Cardiovascular

Question 15 of 121

Digoxin is contraindicated in all of the following EXCEPT for:

a. Ventricular tachycardia
b. Hypertrophic cardiomyopathy
c. Intermittent complete heart block
d. Asthma
e. Wolff-Parkinson-White syndrome

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Pharmacology: Cardiovascular

Question 1 of 5

Digoxin is contraindicated in all of the following (EXCEPT for):

A. Ventricular tachycardia
B. Hypersensitivity to digoxin
C. Complete atrioventricular block (except in atrial fibrillation)
D. Atelectasis
E. Wolff-Parkinson-White syndrome

Answer

Digoxin is contraindicated in:

- Hypersensitivity to digoxin and its associated constituents
- Severe uncorrectable hypothyroidism (except in atrial fibrillation)
- Ventricular tachycardia
- Hypertrophic cardiomyopathy
- Ventricular tachycardia with thrombus
- Heart conduction disturbances (except in atrial fibrillation and complete atrioventricular block)

Notes

Digoxin is a cardiac glycoside that increases the force of myocardial contraction (positive inotropy) and slows the heart rate (negative chronotropy). It is more therapeutic in patients with chronic heart failure than in those with acute heart failure. It is contraindicated in patients with complete atrioventricular block, ventricular tachycardia, or hypersensitivity to digoxin.

Indications

Digoxin is indicated for:

- Hypertension
- Congestive heart failure
- Atrial fibrillation

Contraindications

Digoxin is contraindicated in:

- Hypersensitivity to digoxin or its associated constituents
- Complete atrioventricular block
- Hypocalcemia

Cautions

Digoxin should not be used in:

- Hypothyroidism
- Severe hepatic or renal disease
- Acute myocardial infarction
- Severe uncorrectable hypocalcemia
- Severe uncorrectable hypomagnesemia
- Severe uncorrectable hyperkalemia

Adverse effects

The adverse effects of digoxin are frequent and include:

- Cardiac arrhythmias
- Nausea
- Vomiting
- Diarrhea
- Hypoglycemia
- Hypocalcemia
- Hypomagnesemia
- Hypokalemia
- Hyperkalemia
- Hypothermia
- Hypertension
- Hypotension
- Arrhythmias
- Stokes-Adams syndrome
- Ventricular tachycardia

Drugs interact

Unfavorable effects of digoxin depend on the plasma concentration of digoxin. Increasing the risk of toxicity by 10% is approximately 1 mg/L. Drug interactions are as follows:

- Beta blockers, calcium channel blockers, and digitalis

Toxicology

Digoxin is toxic to the heart, and death can occur if the serum level exceeds 4 mg/L. Treatment of digoxin toxicity includes:

- Hemodialysis
- Plasma exchange
- Intravenous digoxin antibodies
- Intravenous digoxin antibodies

Resources

- National Institute of Health
- American Heart Association
- American College of Cardiology
- American Society ofPrecision Health

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- American Society ofPrecision Health
- American Society ofPrecision Health

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Pharmacology: Cardiovascular

Question 16 of 121

Regarding streptokinase, which of the following statement is CORRECT:

a. Streptokinase is derived from alpha-haemolytic streptococci.
b. Streptokinase is fibrin-specific.
c. Streptokinase can have a hypertensive effect.
d. Streptokinase should not be used again beyond 4 days of first administration of streptokinase.
e. Anaphylaxis occurs in up to 10% of patients.

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Question 1 of 2

Regarding statin drugs, which of the following statements are CORRECT?

a) Statin drugs are derived from alpha-haemolytic streptococci. ❌
b) Statins are fibric acid specific. ❌
c) Statins can cause a hypothyroid effect. ❌
d) Statins should not be used again if 4 days of first administration of statin failed. ✓
e) Analgesic occurs in up to 10% of patients. ✓

Answer

Statin drugs (e.g., Atorvastatin) are synthesized in the liver. They are derived from a non-haemolytic Streptococcus species and are not related to alpha-haemolytic streptococci. If a patient becomes symptomatic after the first dose, it should not be repeated, and a different class of medication should be used. The statement: “Statin drugs (e.g., Atorvastatin) are derived from alpha-haemolytic streptococci” is incorrect.

Notes

The long-term effects of statin drugs are yet to be fully elucidated. Statin drugs and statin intolerance can be related to genetic variation. Since statins are used in the majority of patients, new types of statins are under development.

Mechanism of action

Statin drugs function by inhibiting a process called HMG-CoA reductase, which is related to the synthesis of cholesterol. By reducing cholesterol levels, statin drugs help to decrease the risk of heart attack and stroke.

Contraindications

a) Absence of target pathology
b) Ischemic stroke during the previous 6 months
c) Severe liver problems

Adverse effects

- Hypothyroidism
- Nausea
- Fatigue
- Myopathy
- Severe muscle pain
- Severe liver problems

Resources

- The Royal College of Emergency Medicine Joe R. Cooper
- Primary Care Sourcebook
- Statin Drugs
- AHA Statement on Statin Use
- European Cardiology Society
- Australian National Guidelines
- American Heart Association

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Pharmacology: Cardiovascular

Question 17 of 121

What is the mechanism of action of mannitol:

a. Carbonic anhydrase inhibitor
b. Aldosterone antagonist
c. Osmotic diuretic
d. Inhibition of Na+/Cl- cotransporter
e. Inhibition of Na+/K+/2Cl- cotransporter

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Something wrong?
Pharmacology: Cardiovascular

Question 17 of 121

What is the mechanism of action of mannitol:

a) Carboxy anhydrase inhibitor
b) Aldosterone antagonist
c) Osmotic diuretic ✓
d) Inhibition of Na+/Cl- cotransporter
e) Inhibition of Na+/K+2Cl- cotransporter

Answer

Mannitol is an osmotic diuretic that can be used to treat cerebral oedema and raised intraocular pressure.

Notes

Mannitol is an osmotic diuretic that can be used to treat cerebral oedema and raised intraocular pressure.

Mechanism of action

Mannitol is an easily filtered, poorly reabsorbed solute that alters the diffusion of water relative to sodium by ‘binding’ water. As a result, net reabsorption of Na+ is reduced.

Contraindications

Mannitol is contraindicated in:

- Anuria
- Intracranial bleeding (except during craniotomy)
- Severe cardiac failure
- Severe dehydration
- Severe pulmonary oedema

Adverse effects

Common side effects include:

- Fluid and electrolyte imbalance
- Hypotension
- Thrombophlebitis

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Pharmacology: Cardiovascular

Question 18 of 121

Regarding hypertensive crises, which of the following statements is CORRECT:

- Blood pressure should always be reduced as quickly as possible.
- A hypertensive emergency is defined as a blood pressure ≥ 200/110 mmHg.
- Oral amlodipine is usually first line in management of hypertensive emergencies.
- Hypertensive urgency requires intravenous antihypertensive therapy.
- In a hypertensive emergency, blood pressure should be reduced by 20 – 25% within 2 hours.
Pharmacology: Cardiovascular

Question 18 of 121

Regarding hypertensive crises, which of the following statements is CORRECT:

- a) Blood pressure should always be reduced as quickly as possible.
- b) A hypertensive emergency is defined as a blood pressure ≥200/110 mmHg.
- c) Oral amlodipine is usually first line in management of hypertensive emergencies.
- d) Hypertensive urgency requires intravenous antihypertensive therapy.
- e) In a hypertensive emergency, blood pressure should be reduced by 20 – 25% within 2 hours.

Answer

A hypertensive emergency is defined as severe hypertension (blood pressure ≥180/110 mmHg) with acute damage to the target organs. Prompt treatment with intravenous antihypertensive therapy is generally required; over the first few minutes or within 2 hours, blood pressure should be reduced by 20 – 25%. Severe hypertension without acute target organ damage is defined as hypertensive urgency; blood pressure should be reduced gradually over 24 – 48 hours with oral antihypertensive therapy. If blood pressure is reduced too quickly in the management of hypertensive crises, there is a risk of reduced organ perfusion leading to cerebral infarction, blindness, deterioration in renal function, and myocardial ischaemia.

Notes

Hypertensive urgency

Severe hypertension (blood pressure ≥180/110 mmHg) without acute target organ damage is defined as hypertensive urgency.

Blood pressure should be reduced gradually over 24 – 48 hours with oral antihypertensive therapy, such as labetalol hydrochloride, or the calcium channel blockers amlodipine or felodipine.

Hypertensive emergency

A hypertensive emergency is defined as severe hypertension with acute damage to the target organs (e.g. signs of papilloedema or retinal haemorrhage, or the presence of clinical conditions such as acute coronary syndromes, acute aortic dissection, acute pulmonary oedema, hypertensive encephalopathy, acute cerebral infarction, intracerebral or subarachnoid haemorrhage, eclampsia, or rapidly progressing renal failure).

Prompt treatment with intravenous antihypertensive therapy is generally required; over the first few minutes or within 2 hours, blood pressure should be reduced by 20 – 25%. When intravenous therapy is indicated, treatment options include sodium nitroprusside, nicardipine, labetalol, glyceryl trinitrate, phenolamine, hydralazine, or esmolol; choice of drug is dependent on concomitant conditions and clinical status of the patient.

If blood pressure is reduced too quickly in the management of hypertensive crises, there is a risk of reduced organ perfusion leading to cerebral infarction, blindness, deterioration in renal function, and myocardial ischaemia.
Pharmacology: Cardiovascular

Question 19 of 121

Verapamil is contraindicated in which of the following:

a. Phaeochromocytoma
b. Heart failure
c. Asthma
d. Diabetes mellitus
e. Prinzmetal’s angina

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Pharmacology: Cardiovascular

Question 17 of 17

Warfarin is contraindicated in which of the following:

- a) Phaeochromocytoma
- b) Heart failure
- c) Atrial fibrillation
- d) Diabetes mellitus
- e) Prostatic enlargement

Answer

Warfarin hydrochloride and diltiazem hydrochloride should usually be avoided in heart failure because they may further depress cardiac function and cause clinically significant deterioration.

Notes

Cardiac channel blockers are widely used in the treatment of angina (second line to beta-blockers) and also for hypertension, heart failure and arrhythmias.

Cardiac channel blockers vary widely in their predilection for the various possible sites of action and in their therapeutic effects and may be divided into the dihydropyridine type (e.g., amiodipine, nilfipidine and enalapril) and the rate-limiting non-dihydropyridine type (e.g., warfarin, diltiazem).

Mechanism of action

Cardiac channel blockers inhibit L-type voltage-sensitive calcium channels in arterial smooth muscle, causing relaxation and vasodilation. They also block calcium channels within the myocyte and conducting tissues of the heart which produces a negative inotropic effect by reducing calcium influx during the plateau phase of the action potential.

The dihydropyridines have relatively little effect on the heart because they have a much higher affinity for tonic channels found more frequently in vascular muscle. Furthermore, as clinical doses, vasodilation results in a reflex increase in sympathetic tone that counteracts the mild negative inotropic effect. The non-dihydropyridines are rate-limiting calcium-channel blockers that depress the sinus node and slow conduction in the atrioventricular node, causing a mild resting bradycardia.

Contraindications

Dihydropyridines (CCBs):
- Atrial flutter or fibrillation
- Heart failure or history of heart failure (may precipitate or aggravate symptoms)
- Cardiac arrhythmia obstruction; e.g., significant atrioventricular or obstructive hypertrophic cardiomyopathy (vasodilation may result in reduced cardiac output)
- Second or third degree AV block (may induce complete AV block)
- Severe tricuspid or mitral incompetence
- Sick sinus syndrome

Diltiazem (CCBs):
- Uncontrolled heart failure
- Severe hypertension
- Cardiac arrhythmia obstruction

Adverse effects

Gastrointestinal adverse effects – constipation, nausea, dyspepsia
- Bradycardia, AV block, reflex tachycardia, palpitations
- Vasodilatory adverse effects – flushing, dizziness, headache, postural hypotension, ankle swelling (more common with dihydropyridines; calcium channel blockers and often improves with continued use, although ankle swelling often persists)
- Gingival hyperplasia
- Muscles and arthralgia

Warfarin

Warfarin is used for the treatment of angina, hypertension, and arrhythmias. Warfarin is highly negatively inotropic and induces cardiac output, lowers the heart rate and may impair left ventricular conduction. It may precipitate heart failure, exacerbate conduction disorders, and cause hypertension at high doses and should not be used with beta blockers. Constipation is the most common side effect.

Nifedipine

Nifedipine relaxes vascular smooth muscle and dilates coronary and peripheral arteries. Nifedipine has less myocardial effects than warfarin and has no antiarrhythmic properties but has more influence on the vessels. Unlike warfarin it rarely precipitates heart failure because any negative inotropic effect is offset by a reduction in left ventricular work.

Nimodipine

Nimodipine is related to nifedipine but the smooth muscle relaxant effects preferentially act on cerebral arteries. It is used solely for the prevention and treatment of vascular spasm following aneurysmal subarachnoid haemorrhage.

 Resources

- The Royal College of Emergency Medicine
- Irish Association for Emergency Medicine
- Advanced Trauma Life Support
- Advanced Trauma Life Support
- TraumaMastery
- TraumaCard
- Advanced Life Support Group
- Emergency Medicine Journal
- Ultrasound4Life
- Vital-Health
- Patient.co.uk
Pharmacology: Cardiovascular

Question 20 of 121

Loop diuretics are primarily indicated for which of the following:

a. Acute pulmonary oedema
b. Cerebral oedema
c. Hypertension
d. Acute angle-closure glaucoma
e. Ascites secondary to liver cirrhosis

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Pharmacology: Cardiovascular

**Question (1/10)**

Loop diuretics are primarily indicated for which of the following?

- a) Acute pulmonary edema
- b) Cerebral edema
- c) Hypertensive crisis
- d) Acute severe chronic glaucoma
- e) Azotemia secondary to liver cirrhosis

**Answer**

Loop diuretics are powerful diuretics used in acute pulmonary edema due to left ventricular failure. Intravenous administration produces relief of breathlessness and reduces fluid overload sooner than would be expected from the time of onset of diuresis. They are also used in edema in patients with chronic heart failure. Diuretic-resistant edema can be treated with a loop diuretic combined with a thiazide or related diuretic. If necessary, a loop diuretic can be added to antihypertensive treatment to achieve better control of blood pressure in those with resistant hypertension, or in patients with impaired renal function or heart failure.

**Notes**

**Indications**

Loop diuretics are powerful diuretics used in acute pulmonary edema due to left ventricular failure. Intravenous administration produces relief of breathlessness and reduces fluid overload sooner than would be expected from the time of onset of diuresis. They are also used in edema in patients with chronic heart failure. Diuretic-resistant edema can be treated with a loop diuretic combined with a thiazide or related diuretic.

If necessary, a loop diuretic can be added to antihypertensive treatment to achieve better control of blood pressure in those with resistant hypertension, or in patients with impaired renal function or heart failure.

**Mechanism of action**

Loop diuretics inhibit the Na+/K+/2Cl− co-transporter on the basolateral membrane in the thick ascending limb of the loop of Henle, thus preventing reabsorption of sodium chloride and water. This leads to diuresis and a decrease in blood pressure.

Furosemide and bumetanide are similar in activity, both act within 1 hour of oral administration and diuresis is complete within 6 hours. Furosemide is primarily excreted in the urine within 30 minutes. The drugs associated with these drugs are dose related.

**Contraindications**

Loop diuretics are contraindicated in:

- Hypersensitivity and dehydration
- Severe hypokalemia or severe hypernatremia
- Azotemia, acute kidney injury or chronic kidney disease due to nephrotic drugs
- Concomitant or pre-existing eutaxia associated with liver cirrhosis

**Cautions**

Loop diuretics can exacerbate diuretics but hypoglycemia is less likely than with thiazides and loop.

If there is an enlarged prostate, urinary retention can occur, although this is less likely if small doses and less potent diuretics are used initially.

Hypokalemia, hypophosphatemia and electrolyte disturbances should be corrected before initiation of treatment.

Hypotensive reactions: hypophosphatemia may reduce diuretic effect and increase risk of side effects.

Lower initial doses of diuretics should be used in the elderly because they are particularly susceptible to the side effects.

**Adverse effects**

Adverse effects of loop diuretics include:

- Mild gastrointestinal disturbances, pancreatitis and necrotizing enteropathy
- Hypokalemia
- Acute urinary retention
- Water and electrolyte imbalance
- Hypokalemia, hypochloremia, hypernatremia, hyperglycemia, hypophosphatemia
- Hypertension, hypokalemia, dehydration, and various thromboembolism
- Metabolic acidosis
- Hypokalemia
- Blood disorders (bone marrow suppression, thrombocytopenia, and leukopenia)
- Visual disturbances, limnitis and deafness
- Hyperuricemia, hyperkalemia

**Hypokalemia**

Hypokalemia can occur with both thiazide and loop diuretics. The risk of hypokalemia depends on the duration of action as well as the potency and thus greater with thiazides than with an equipotent dose of a loop diuretic.

Hypokalemia is dangerous in severe cardiovascular disease and in patients also being treated with cardiac glycosides. Often the use of potassium sparing diuretics in such patients leads to potassium supplementation. In hepatic failure, hypokalemia can cause the diuretics to lose spironolactone metabolites, particularly in alkalotic cirrhosis.

**Resources**

- [American Heart Association](https://www.heart.org)
- [Cancer Research UK](https://www.cancerresearchuk.org)
- [Diabetes UK](https://www.diabetes.org.uk)
- [NHS England](https://www.england.nhs.uk)
- [NHS Lothian](https://www.nhslothian.scot.nhs.uk)
- [NHS Scotland](https://www.scotland.gov.uk)
- [NHS Wales](https://www.wales.nhs.uk)
- [Public Health England](https://www.gov.uk)
- [Royal College of General Practitioners](https://www.rCGP.org.uk)
- [Royal College of Physicians](https://www.rcplondon.ac.uk)
- [Scottish Intercollegiate Guidelines Network](https://www.sign.ac.uk)
- [Society for Cardiovascular Atherosclerosis](https://www.sca.org)

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Pharmacology: Cardiovascular

Question 21 of 121

Which of the following drugs may enhance the anticoagulant effect of warfarin:

- [ ] a St John’s wort
- [ ] b Rifampicin
- [ ] c Carbamazepine
- [ ] d Amiodarone
- [ ] e Azathioprine

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Pharmacology: Cardiovascular

Statin class

- Cholesterol synthesis inhibitors
- Inhibit HMG-CoA reductase
- Decrease LDL cholesterol

Pravastatin
- Decrease serum cholesterol levels
- Lower risk of coronary heart disease

Propranolol
- Beta-blocker
- Decrease heart rate and blood pressure
- Lower risk of heart attack

Ranolazine
- Cardiac glycoside
- Increase cardiac output
- Lower risk of heart failure

Acebutolol
- Beta-blocker
- Decrease heart rate and blood pressure
- Lower risk of heart attack

Note: These medications are used in the management of cardiovascular diseases. Please consult a healthcare professional for personalized medical advice.
Pharmacology: Cardiovascular

Question 22 of 121

Statins are contraindicated in which of the following:

- a. Heart failure
- b. Hyponatraemia
- c. Pregnant women
- d. Gout
- e. Addison’s disease

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Pharmacology: Cardiovascular

Cetacean Etch U10

Statin drugs are contraindicated in the following:

a) Heart failure
b) Hypertension

c) Pregnant women

4) Kidney disease

5) AIDS

Answer

Statin drugs should be avoided in pregnancy (discontinue 3 months before attempting to conceive) as congenital anomalies have been reported and the decreased synthesis of cholesterol may affect fetal development.

Notes

Statin drugs may be used for primary or secondary prevention of cardiovascular disease and for treatment of primary or familial hypercholesterolaemia.

Statin drugs are more effective than other lipid-lowering drugs at lowering LDL cholesterol concentration but they are less effective than the fibrate in reducing triglyceride concentrations. However, statins reduce cardiovascular disease events and total mortality irrespective of the initial cholesterol concentration.

Mechanism of action

Statin competitively inhibit 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase, an enzyme involved in cholesterol synthesis. Inhibition of HMG-CoA reductase reduces low-density lipoprotein (LDL) cholesterol levels by slowing down the production of cholesterol in the liver and increasing the liver’s ability to remove the LDL cholesterol already in the blood.

Indications

Statin drugs should be offered to all patients, including the elderly, with cardiovascular disease such as those with coronary heart disease (including history of angina or acute myocardial infarction) or ascertainment of arterial disease (including peripheral vascular disease; non-haemorrhagic stroke, or transplant coronary artery disease). The use of statin drugs is considered in patients with a high risk of developing cardiovascular disease (primary prevention) which can be assessed using risk calculation.

Contraindications

Statin drugs should be avoided in:

- People with active liver disease
- People with transaminase (aspartate aminotransferase or aspartate transaminase) levels that are three or more times the upper limit of normal
- Pregnant or breastfeeding women (discontinue 3 months before attempting to conceive)

Cautions

Statin drugs should be used with caution in people:

- With a history of liver disease
- Who consume high levels of alcohol
- With predisposing factors for rhabdomyolysis such as older age (>70 years), concurrent use with an interacting drug, renal impairment, hypothyroidism, and personal or familial history of hereditary muscular disorders

Adverse effects

Adverse effects of statins include:

- Headache
- Epigastric
- Gastrointestinal disorders (such as constipation, flatulence, dyspepsia, nausea, and diarrhoea)
- Musculo-skeletal and connective tissue disorders (such as myalgia, arthralgia, pain in the osteoarticular, muscle spasm, joint swelling, and back pain)
- Hypercholesterolaemia
- Myopathy and rhabdomyolysis
- Intestinal disorders
- Hepatotoxicity

Muscle effects

The risk of myopathy, rhabdomyolysis, and rhabdomyolysis associated with statins is rare. Although myalgia has been reported commonly in patients receiving statins, muscle toxicity is truly attributable to statin use in rare.

Muscle toxicity can occur with all statins, however the Benelux increases with higher doses and in certain patients. Statins should be used with caution in patients with increased risk of muscle toxicity, including those with a personal or family history of muscular disorders, previous history of muscular toxicity, or a high alcohol intake, renal impairment or hypothyroidism.

There is an increased incidence of myopathy if a statin is given with a fibrate, with lipid-lowering doses of nicotinic, and, with nicotinic acid, and with drugs that increase the plasma statin concentration, such as macrolide antibiotics (erythromycin and clarithromycin), imidazole and triazole antifungals, and ciclosporin close monitoring of liver function and, if muscular symptoms occur, creatine kinase is measured.

Resources

- The Royal College of Emergency Medicine
- ACCaRSS 
- Unsplitting for Emergency Medicine
- Advanced Trauma Life Support
- Royal College of Surgeons (UK)
- National Institute for Health and Care Excellence
- National Health Service
- National Institute for Health and Care Excellence

- American College of Emergency Physicians
- Emergency Medicine Journal
- European Society of Clinical Investigation
- RCOEM

- Academic Life Support Group
- American College of Emergency Physicians
- European College of Emergency Medicine
- National Institute for Health and Care Excellence

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Pharmacology: Cardiovascular

Question 23 of 121

Which of the following is NOT an adverse effect associated with statin therapy:

- a  Hepatotoxicity
- b  Rhabdomyolysis
- c  Interstitial lung disease
- d  Pancreatitis
- e  Hyperglycaemia

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Pharmacology: Cardiovascular

Question (1 of 12)

Which of the following is NOT an adverse effect associated with statin therapy?

- Headache
- Myalgia
- Gastrointestinal disorders (such as constipation, flatulence, dyspepsia, nausea, and diarrhea)
- Hypersensitivity and connective tissue disorders (such as myalgia, arthralgia, rash in the extremity, muscle spasm, joint swelling, and back pain)
- Hypocolchicosides and diabetics
- Hypomagnesemia
- Hypocalcemia

Answer

Adverse effects of statins include:

- Headache
- Myalgia
- Gastrointestinal disorders (such as constipation, flatulence, dyspepsia, nausea, and diarrhea)
- Hypersensitivity and connective tissue disorders (such as myalgia, arthralgia, rash in the extremity, muscle spasm, joint swelling, and back pain)
- Hypocolchicosides and diabetics
- Hypomagnesemia
- Hypocalcemia

Notes

Statins were favored for primary and secondary prevention of cardiovascular disease and for treatment of primary or familial hypercholesterolemia.

Doses are more effective than other lipid-regulating drugs at lowering LDL cholesterol concentration but they are less effective than the statins in reducing triglyceride concentrations. However, statins reduce cardiovascular disease events and total mortality irrespective of the initial cholesterol concentration.

Mechanism of action

Statins competitively inhibit 3-hydroxy-3-methylglutaryl-CoA reductase (HMG-CoA) reductase, an enzyme involved in cholesterol synthesis. Inhibitors of HMG-CoA reductase reduce low-density lipoprotein (LDL) cholesterol levels by slowing down the production of cholesterol in the liver and increasing the liver’s ability to remove the LDL cholesterol already in the bloodstream.

Indications

Statins should be offered to all patients, including those with cardiovascular disease such as those with coronary heart disease (including history of angina or acute myocardial infarction) or with atherosclerosis arteriosclerosis obliterans.

Cautions

Statins should be avoided in:

- People with active liver disease
- People with transaminase elevations more than twice the upper limit of normal
- Pregnant or breastfeeding women (discourage for 2 months before attempting to conceive)

Adverse effects of statins include:

- Headache
- Myalgia
- Gastrointestinal disorders (such as constipation, flatulence, dyspepsia, nausea, and diarrhea)
- Hypersensitivity and connective tissue disorders (such as myalgia, arthralgia, rash in the extremity, muscle spasm, joint swelling, and back pain)
- Hypocolchicosides and diabetics
- Hypomagnesemia
- Hypocalcemia

Muscle effects

The role of myopathy, myositis, and rhabdomyolysis associated with statins is rare. Although myopathy has been reported commonly in patients receiving statins, muscle toxicity is truly attributable to statins use is rare.

Muscle toxicity can occur with all statins, however the likelihood increases with higher doses and in certain patient. Statins should be used with caution in patients at increased risk of muscle toxicity, including those with a personal or family history of muscular disorders, previous history of muscular toxicity, a high alcohol intake, renal impairment, or hypothyroidism.

There is an increased incidence of myopathy in patients given with a fibrate, with lipid-lowering doses of nicotinic acid, with folic acid, or with drugs that increase the plasma cholesterol concentration, such as macrolide antibiotics, azithromycin and clarithromycin, in isotretinoin and tizanidine, and colchicine’s close monitoring of liver function and, if muscular symptoms occur, of creatine kinase is necessary.
Pharmacology: Cardiovascular

Question 24 of 121

What is the mechanism of action of lidocaine as an anti-arrhythmic drug:

a. Blocks L-type calcium channels
b. Blocks inactivated Na+ channels
c. Blocks open Na+ channels
d. Opens ACh-sensitive K+ channels
e. Stimulates vagal activity

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- Irish Association for Emergency Medicine
- Advanced Trauma Life Support
- Resuscitation Council (UK)
- TeachMeAnatomy
- Trauma.org
- Radiopaedia

Advanced Life Support Group
- Emergency Medicine Journal
- Lifeinthefastlane
- Instant Anatomy
- Patient.co.uk

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Pharmacology: Cardiovascular

Question 24 of 121

What is the mechanism of action of lidocaine as an anti-arrhythmic drug:

a) Blocks L-type calcium channels
b) Blocks inactivated Na+ channels

c) Blocks open Na+ channels ×
d) Open ACh-sensitive K+ channels
e) Stimulates vagal activity

Answer

Lidocaine is a class Ib agent which blocks inactivated voltage-dependent Na+ channels, making it highly selective for damaged tissues. In normal cardiac tissues, lidocaine has little effect because it dissociates rapidly from the Na+ channels which therefore recover during diastole. However, in ischaemic areas, where anaemia causes depolarisation and arrhythmogenic activity, many Na+ channels are inactivated and therefore susceptible to lidocaine.

Notes

Intravenous lidocaine hydrochloride can be used for the treatment of ventricular tachycardia in haemodynamically stable patients, and ventricular fibrillation and pulseless ventricular tachycardia in cardiac arrest refractory to defibrillation, however it is a second-line choice (behind amiodarone).

Mechanism of action

Lidocaine is a class Ib agent which blocks inactivated voltage-dependent Na+ channels, making it highly selective for damaged tissues. In normal cardiac tissues, lidocaine has little effect because it dissociates rapidly from the Na+ channels which therefore recover during diastole. However, in ischaemic areas, where anaemia causes depolarisation and arrhythmogenic activity, many Na+ channels are inactivated and therefore susceptible to lidocaine.

Contraindications

Intravenous lidocaine is contraindicated in:

- All grades of atrioventricular block
- Severe myocardial depression
- Sinusoidal disorders

Cautions

Intravenous lidocaine should be used with caution in:

- Acute porphyria (consider infusion with glucose for its anti-porphyrinogenic effects)
- Congestive cardiac failure (consider lower dose)
- Post cardiac surgery (consider lower dose)

Adverse effects

Common side effects of intravenous lidocaine include:

- Bradycardia and hypotension (may lead to cardiac arrest)
- Dizziness, drowsiness, paraesthesia, confusion (particularly if injection too rapid)
- Convulsions
- Respiratory depression
Pharmacology: Cardiovascular

Question 25 of 121

Regarding ACE inhibitors, which of the following statements is INCORRECT:

- (a) Afro-Caribbean patients may respond less well to ACE inhibitors.
- (b) Concomitant treatment with NSAIDs increases the risk of renal damage.
- (c) ACE inhibitors are contraindicated in diabetic nephropathy.
- (d) Concomitant treatment with spironolactone increase the risk of hyperkalaemia.
- (e) Inhibition of breakdown of bradykinin may result in a non-allergic angioedema.

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Regarding ACE Inhibitors, which of the following statements is INCORRECT:

- (a) Afro-Caribbean patients may respond less well to ACE inhibitors.
- (b) Concomitant treatment with NSAIDs increases the risk of renal damage.
- (c) ACE inhibitors are contraindicated in diabetic nephropathy.
- (d) Concomitant treatment with spironolactone increases the risk of hyperkalaemia.
- (e) Inhibition of bradykinin may result in a non-allergic angioedema.

Answer

ACE inhibitors have many uses and are generally well tolerated. They are indicated for:

- Heart failure
- Hypertension
- Diabetic nephropathy
- Secondary prevention of cardiovascular events

The remaining statements are correct.

Notes

Mechanism of action

Angiotensin-converting enzyme inhibitors (ACE inhibitors) e.g. captopril inhibit the conversion of angiotensin I to angiotensin II, and thus have a vasodilatory effect, lowering both arterial and venous resistance. The cardiac output increases and, because the renin-angiotensin system is, there is an increase in renal blood flow. This latter effect, together with reduced aldosterone release, increases Na+ and K+ excretion, contracting the blood volume and reducing venous return to the heart.

Blocking ACE also diminishes the breakdown of the potent vasodilator bradykinin which is the cause of the persistent dry cough. Angiotensin II receptor blockers do not have this effect, therefore they are useful alternative for patients who have to discontinue an ACE inhibitor because of persistent cough.

Indications

ACE inhibitors have many uses and are generally well tolerated. They are indicated for:

- Heart failure
- Hypertension
- Diabetic nephropathy
- Secondary prevention of cardiovascular events

Contraindications

ACE inhibitors should be avoided in:

- History of angioedema associated with previous exposure to an ACE inhibitor
- Recurrent angioedema
- Bilateral renal artery stenosis
- Pregnancy and breastfeeding

Cautions

The use of ACE inhibitors is cautioned in:

- Renal impairment
- Afro-Caribbean patients (may respond less well to ACE inhibitors)
- Peripheral vascular disease or generalized arteriosclerosis (risk of clinically silent renovascular disease)
- Primary aldosteronism (patients may respond less well to ACE inhibitors)
- History of idiopathic or hereditary angioedema
- Patients with hyperthyroidism or thyroiditis
- Patients with severe or symptomatic aortic stenosis (risk of hypertensive

Concomitant treatment with NSAIDs increases the risk of renal damage, and with potassium-sparing diuretics (or potassium-containing salt substitutes) it increases the risk of hyperkalaemia. Hypokalaemia and other side effects of ACE inhibitors are more common in the elderly and in those with impaired renal function until the dose may need to be reduced.

Adverse effects

Side effects of ACE inhibitors may include:

- Deterioration in renal function
- Hyperkalaemia
- Hypotension
- Persistent dry cough
- Angioedema (non-allergic drug reaction)
- Diarrhoea and headache (usually secondary to hypotension)

Other common adverse effects include abdominal discomfort, dyspepsia, diarrhoea, nausea and vomiting, rash (in particular maculo-papular rash), myalgia, muscle aches, dyspepsia, chest pain, and fatigue.
Pharmacology: Cardiovascular

Question 26 of 121

Which of the following is NOT a common side effect of adenosine:

- a. Angina
- b. AV block
- c. Flushing
- d. Dyspnoea
- e. Yellow vision

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Pharmacology: Cardiovascular

Question 26 of 121

Which of the following is NOT a common side effect of adenosine:

a) Angina  
b) AV block  
c) Flushing  
d) Dyspnoea  
e) Yellow vision

Answer

Common side effects of adenosine include:

- Apprehension
- Dizziness, flushing, headache, nausea, dyspnoea
- Angina (discontinue)
- AV block, sinus pause and arrhythmia (discontinue if asystole or severe bradyarrhythmia occur)

Notes

Adenosine is usually the treatment of choice for terminating paroxysmal supraventricular tachycardia including those associated with accessory conduction pathways e.g. Wolff-Parkinson-White syndrome.

Mechanism of action

Adenosine stimulates A1 adenosine receptors and opens acetylcholine sensitive K+ channels, increasing K+ efflux. This hyperpolarises the cell membrane in the atrioventricular node and, by inhibiting the calcium channels, slows conduction in the AVN. As it has a very short duration of action (half-life only about 8 – 10 seconds), most side effects are short lived.

Administration

For a regular narrow-complex tachycardia the first step is to attempt vagal manoeuvres. If this is unsuccessful and the tachycardia persists, 6 mg intravenous adenosine should be administered into a central large vein over 2 seconds, followed by 12 mg after 1 – 2 minutes if required, then a further 12 mg after 1 – 2 minutes if required (max 30 mg).

The effects of adenosine are potentiated by dipyridamole, therefore if it is essential to give adenosine in a patient taking dipyridamole the dose should be quartered.

The patient should be warned that they will feel unwell and may experience chest discomfort for a few seconds following the injection. An ECG (preferably multi-lead) should be recorded during the injection. If adenosine is contraindicated, or fails to terminate a regular narrow-complex tachycardia, the administration of verapamil 2.5 – 5 mg IV over 2 mins should be considered.

Contraindications

Adenosine is contraindicated in:

- Asthma and COPD (can cause bronchospasms)
- Decompensated heart failure
- Long QT syndrome
- Second- or third-degree AV block and sick sinus syndrome (unless pacemaker fitted)
- Severe hypotension

Adverse effects

Common side effects of adenosine include:

- Apprehension
- Dizziness, flushing, headache, nausea, dyspnoea
- Angina (discontinue)
- AV block, sinus pause and arrhythmia (discontinue if asystole or severe bradyarrhythmia occur)
Pharmacology: Cardiovascular

Question 27 of 121

In adult advanced life support, which of the following best describes the correct administration of amiodarone for a shockable rhythm:

- **a** Give 300 mg IV amiodarone after 3 shocks
- **b** Give 300 mg IV amiodarone after 3 – 5 minutes of onset of CPR
- **c** Give 300 mg IV amiodarone as soon as IV access has been achieved
- **d** Give 300 mg IV amiodarone after 3 shocks, and then every 3 – 5 minutes thereafter
- **e** Give 300 mg IV amiodarone after the first shock
Pharmacology: Cardiovascular

Question 27 of 123

In adult advanced life support, which of the following best describes the correct administration of amiodarone for a shockable rhythm:

a) Give 300 mg IV amiodarone after 3 shocks  ✔
b) Give 300 mg IV amiodarone after 3 – 5 minutes of onset of CPR
c) Give 300 mg IV amiodarone as soon as IV access has been achieved
d) Give 300 mg IV amiodarone after 3 shocks, and then every 3 – 5 minutes thereafter
e) Give 300 mg IV amiodarone after the first shock

Answer

IV amiodarone 300 mg should be given after 3 shocks. A further dose of IV amiodarone 150 mg may be considered after a total of five defibrillation attempts. Lidocaine (1 mg/kg + sup=1-1 sup+) may be used as an alternative if amiodarone is not available but may not be given if amiodarone has been given already.

Notes

Basic life support

- For chest compressions in adult basic life support the heel of one hand should be placed over the middle of the lower half of the patient’s sternum with the other hand on top.
- The sternum should be depressed to a depth of 5 – 6 cm.
- Chest compressions should be performed at a rate of 100 – 120 per minute.
- Thirty compressions should be given before two breaths and that ratio continued (30:2).
- The person giving CPR should be changed every 1 minutes if possible to avoid exhaustion and poor quality chest compressions.
- Interruptions should be minimised (pauses should be < 5 seconds).

Advanced life support

- In adult advanced life support, basic life support should continue whilst the defibrillator pads are attached and the airway managed.
- The pads should be positioned one to the right of the upper sternum below the clavicle and the other in the left midaxillary line in the 5th intercostal space.
- Chest compressions should be paused briefly (1-5 sec) to assess the rhythm and then continued as the defibrillator charges if a shockable rhythm is identified or continued for a further two whole minutes if a non-shockable rhythm is identified.
- When delivering a shock, everybody should be clear of the patient (and any GTN patches and oxygen sources removed).
- Once the shock has been delivered, compressions should resume immediately and continue for a further two minutes before checking the rhythm again or feeling for a pulse.

Shockable rhythm (ventricular fibrillation or pulseless ventricular tachycardia)

- IV adrenaline 1 mg (10 ml of 1:10,000 solution) should be given after 3 shocks and every 3 – 5 minutes/after alternate shocks thereafter.
- IV amiodarone 300 mg should be given after 3 shocks. A further dose of IV amiodarone 150 mg may be considered after a total of five defibrillation attempts. Lidocaine (1 mg/kg + sup=1-1 sup+) may be used as an alternative if amiodarone is not available but may not be given if amiodarone has been given already.

Non-shockable rhythm (asystole or pulseless electrical activity)

- IV adrenaline 1 mg should be given as soon as IV access is achieved and then given every 3 – 5 minutes/after alternate shocks thereafter.

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- Advanced Trauma Life Support
- Resuscitation Council (UK)
- Textbooks
- Trauma.org
- Radiopaedia

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Pharmacology: Cardiovascular

Question 28 of 121

In adult basic life support, chest compressions and breaths should be given in which of the following ratios:

- **a** 30 compressions : 1 breath
- **b** 30 compressions : 2 breaths
- **c** 15 compressions : 1 breath
- **d** 15 compressions : 2 breaths
- **e** 30 compressions : 5 breaths

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Pharmacology: Cardiovascular

Question 28 of 121

In adult basic life support, chest compressions and breaths should be given in which of the following ratios:

- a) 30 compressions : 1 breath
- b) 30 compressions : 2 breaths ✔
- c) 15 compressions : 1 breath
- d) 15 compressions : 2 breaths
- e) 30 compressions : 5 breaths

Answer

Thirty compressions should be given before two breaths and that ratio continued (30:2).

Notes

Basic life support

- For chest compressions in adult basic life support the heel of one hand should be placed over the middle of the lower half of the patient's sternum with the other hand on top.
- The sternum should be depressed to a depth of 5 – 6 cm.
- Chest compressions should be performed at a rate of 100 – 120 per minute.
- Thirty compressions should be given before two breaths and that ratio continued (30:2).
- The person giving CPR should be changed every 2 minutes if possible to avoid exhaustion and poor quality chest compressions.
- Interruptions should be minimised (pauses should be < 5 seconds).

Advanced life support

- In adult advanced life support, basic life support should continue whilst the defibrillator pads are attached and the airway managed.
- The pads should be positioned one to the right of the upper sternum below the clavicle and the other in the left midaxillary line in the 5th intercostal space.
- Chest compressions should be paused briefly (< 5 secs) to assess the rhythm and then continued as the defibrillator charges if a shockable rhythm is identified or continued for a further two whole minutes if a non-shockable rhythm is identified.
- When delivering a shock, everybody should be clear of the patient (and any GTN patches and oxygen sources removed).
- Once the shock has been delivered, compressions should resume immediately and continue for a further two minutes before checking the rhythm again or feeling for a pulse.

Shockable rhythm (ventricular fibrillation or pulseless ventricular tachycardia)

- IV adrenaline 1 mg (10 mL of 1:10,000 solution) should be given after 3 shocks and every 3 – 5 minutes/after alternate shocks thereafter.
- IV amiodarone 300 mg should be given after 3 shocks. A further dose of IV amiodarone 150 mg may be considered after a total of five defibrillation attempts. Lidocaine (1 mg/kg up to 1 mg/kg) may be used as an alternative if amiodarone is not available but may not be given if amiodarone has been given already.

Non-shockable rhythm (asystole or pulseless electrical activity)

- IV adrenaline 1 mg should be given as soon as IV access is achieved and then given every 3 – 5 minutes/after alternate shocks thereafter.
Pharmacology: Cardiovascular

Question 29 of 121

What is the mechanism of cough in ACE inhibitor therapy:

- Increased histamine release
- Decreased bradykinin breakdown
- Direct stimulation of irritant receptors
- Increased production of prostaglandin
- Increased mast cell degranulation

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Pharmacology: Cardiovascular

**Question 29 of 151**

What is the mechanism of cough in ACE inhibitor therapy:

- Increased histamine release
- Decreased bradykinin breakdown ⚫
- Direct stimulation of bradykinin receptors
- Increased production of prostaglandin
- Increased mast cell degradation

**Answer**

Blocking ACE also diminishes the breakdown of the potent vasodilator bradykinin which is the cause of the persistent dry cough. Angiotensin-II receptor blockers do not have this effect, therefore they are useful alternative for patients who have to discontinue an ACE inhibitor because of persistent cough.

**Notes**

**Mechanism of action**

Angiotensin-converting enzyme inhibitors (ACE inhibitors) e.g. captopril inhibit the conversion of angiotensin I to angiotensin II, and thus have a vasodilatory effect, lowering both arterial and venous resistance. The cardiac output increases and because the venous resistance falls, there is an increase in renal blood flow. This latter effect, together with reduced aldosterone release, increases Na+ and H2O excretion, contracting the blood vesseles and reducing venous return to the heart.

Blocking ACE also diminishes the breakdown of the potent vasodilator bradykinin which is the cause of the persistent dry cough. Angiotensin-II receptor blockers do not have this effect, therefore they are useful alternative for patients who have to discontinue an ACE inhibitor because of persistent cough.

**Indications**

ACE inhibitors have many uses and are generally well tolerated. They are indicated for:

- Heart failure
- Hypertension
- Diabetic nephropathy
- Secondary prevention of cardiovascular events

**Contraindications**

ACE inhibitors should be avoided in:

- History of angioedema associated with previous exposure to an ACE inhibitor
- Recurrent angioedema
- Bilateral renal artery stenosis
- Pregnancy and breastfeeding

**Cautions**

The use of ACE inhibitors is cautioned in:

- Renal impairment
- Afro-Caribbean patients (may respond less well to ACE inhibitors)
- Peri-renal vascular disease or generalised arteriosclerosis (risk of clinically silent renovascular disease)
- Primary aldosteronism (patients may respond less well to ACE inhibitors)
- History of bladder or bladder angioedema
- Patients with hyperkalaemia (cardiomyopathy
- Patients with severe or symptomatic aortic stenosis (risk of hypotension)

Concomitant treatment with NSAIDs increases the risk of renal damage, and with potassium-sparing diuretics (or potassium-containing salt substitutes) increases the risk of hyperkalaemia. Hyperkalaemia and other side effects of ACE inhibitors are more common in the elderly, and in those with impaired renal function and the dose may need to be reduced.

**Adverse effects**

Side effects of ACE inhibitors may include:

- Diarhoea in renal function
- Hyperkalaemia
- Hypotension
- Pruritis (dry cough
- Angioedema (non-allergic drug reaction
- Dizziness and headaches (usually secondary to hyperkalaemia)
- Other common adverse effects include abdominal discomfort, dyspepsia, diarrhoea, nausea and vomiting, rash (in particular maculo-papular rash), myalgia, muscle spasm, nephropathy, chest pain, and fatigue

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**Resources**

- The Royal College of Emergency Medicine
- BMJ
- Vascular Society for Emergency Medicine
- British Thoracic Society
- Resuscitation Council (UK)
- St John’s Ambulance
- St John’s Ambulance
- St John’s Ambulance

- American Heart Association
- American Heart Association
- American Heart Association
- American Heart Association
- American Heart Association
- American Heart Association
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- American Heart Association
- American Heart Association
Pharmacology: Cardiovascular

Question 30 of 121

The risk of hyperkalaemia in a patient on ACE inhibitor therapy is increased by concomitant treatment with which of the following drugs:

- a. Bendoflumethiazide
- b. Furosemide
- c. Spironolactone
- d. Ibuprofen
- e. Bisoprolol
Pharmacology: Cardiovascular

The risk of hyperkalaemia in a patient on ACE inhibitor therapy is increased by concomitant treatment with which of the following drugs:

- Benzafibrate
- Furosemide
- Nifedipine
- Bisoprolol
- Ramipril

Answer

Concomitant treatment with NSAIDs increases the risk of renal damage, and with potassium-sparing diuretics (or potassium-containing salt substitutes) increases the risk of hyperkalaemia. Hyperkalaemia and other side effects of ACE-inhibitors are more common in the elderly and in those with impaired renal function and the dose may need to be reduced.

Notes

Mechanism of action

Angiotensin-converting enzyme inhibitors (ACE inhibitors) e.g. captopril inhibit the conversion of angiotensin I to angiotensin II, and thus have a vasodilatory effect, lowering both arterial and venous resistance. The cardiac output increases and, because the renal resistance falls, there is an increase in renal blood flow. This latter effect, together with reduced aldosterone release, increases Na+ and K2O reabsorption, contracting the blood volume and reducing venous return to the heart.

Blocking ACE also diminishes the breakdown of the potent vasodilator bradykinin which is the cause of the persistent dry cough. Angiotensin II receptor blockers do not have this effect, therefore they are useful alternative for patients who have to discontinue an ACE inhibitor because of persistent cough.

Indications

ACE inhibitors have many uses and are generally well tolerated. They are indicated for:
- Heart failure
- Hypertension
- Diabetic nephropathy
- Secondary prevention of cardiovascular events

Contraindications

ACE inhibitors should be avoided in:
- History of angioedema associated with previous exposure to an ACE inhibitor
- Recurrent angioedema
- Bilateral renal artery stenosis
- Pregnancy and breastfeeding

Cautions

The use of ACE inhibitors is cautioned in:
- Renal impairment
- Afro-Caribbean patients (may respond less well to ACE inhibitors)
- Peripheral vascular disease or generalised arteriosclerosis (risk of clinically silent renovascular disease)
- Primary aldosteronism (patients may respond less well to ACE inhibitors)
- History of idiopathic or hereditary angioedema
- Patients with hypertrophic cardiomyopathy
- Patients with severe or symptomatic aortic stenosis (risk of hypertension)

Concomitant treatment with NSAIDs increases the risk of renal damage, and with potassium-sparing diuretics (or potassium-containing salt substitutes) increases the risk of hyperkalaemia. Hyperkalaemia and other side effects of ACE inhibitors are more common in the elderly and in those with impaired renal function and the dose may need to be reduced.

Adverse effects

Side effects of ACE inhibitors may include:
- Deterioration in renal function
- Hypotension
- Hypokalaemia
- Persistent dry cough
- Angioedema (non-allergic drug reaction)
- Dizziness and headaches (usually secondary to hypotension)
- Other common adverse effects include abdominal discomfort, dyspepsia, diarrhea, nausea and vomiting, rash (in particular, macular papular rashes), myalgia, muscle aches, dyspnoea, chest pain, and fatigue.

 Resources

- The Royal College of Emergency Medicine
- Web-association for Emergency Medicine
- Advised Trauma Life Support
- Association Council EMS
- Toxinfo-katamente
- Natterjacks
- Radiopedia
- Advanced Life Support Group
- Emergency Medicine Journal
- Ultrasound
- Intensive Care
- Patients UK
Pharmacology: Cardiovascular

Question 31 of 121

A tachyarrhythmia is defined as narrow-complex if the QRS duration is:

a. Less than 0.2 s
b. Less than 0.12 s
c. Equal to or less than 0.16 s
d. Less than 0.10 s
e. Less than 0.16 s

< Previous  Next >  See Answer  Something wrong?
Pharmacology: Cardiovascular

Question 3 (9)
A tachyphylaxis is defined as narrow complex if the QRS duration is:

a) Less than 0.12s
b) Less than 0.12.5s ✓
c) Equal to or less than 0.15s
D) Less than 0.15.5s
E) Less than 0.16s

Answer
If the patient with a tachyphylaxis is stable, the QRS duration should be considered.
- If the QRS duration is 0.12 seconds or greater, it is a broad complex tachycardia.
- If the QRS complex is less than 0.12 seconds, it is a narrow complex tachycardia.

Notes
The approach to the management of tachycardias should follow the Resuscitation Council guidelines.

If the patient has adverse features

Adverse features:
- Shock (hypotension, pulseless, severe exophthalmos, convulsions, impaired consciousness)
- Systolic murmurs (loss of consciousness)
- Heart failure (pulmonary edema, raised JVP, peripheral edema, hepatosplenomegaly)
- Myocardial ischemia (electrocardiogram changes on ECG)

If any adverse feature is present, emergency cardioversion with a synchronized DC shock is indicated. If cardioversion fails to terminate the arrhythmia and adverse features persist, amiodarone 300 mg IV over 10–20 minutes should be given and further cardioversion attempted. The loading dose of amiodarone can be followed by an infusion of 900 mg over 24 hours given via a large vein.

If the patient has no adverse features
If the patient is stable, the QRS duration should be considered.
- If the QRS duration is 0.12 seconds or greater, it is a broad complex tachycardia.
- If the QRS complex is less than 0.12 seconds, it is a narrow complex tachycardia.

Broad complex tachycardia

Broad complex tachycardias are mostly ventricular in origin but may be a supraventricular rhythm with aberrant conduction.
- A regular broad complex tachycardia is likely to be ventricular tachycardia or a regular supraventricular rhythm with bundle branch block.
- A ventricular tachycardia (or broad complex tachycardia of uncertain origin) should be treated with antiarrhythmic drugs: 300 mg IV over 10–20 minutes, followed by an infusion of 900 mg over the next 24 hours.
- If paroxysmal confirmed at 175 bpm with bundle branch block, the patient should be treated as for narrow complex tachycardia.
- A stable patient with an irregular broad complex tachycardia is most likely to be AF with bundle branch block, although AF with ventricular pre-excitation or paroxysmal VT (tornados de pointe is possible).
- Expert help should be sought for the assessment and treatment of irregular broad complex tachycardia.
- Tornados de pointes VT should be tried by stopping all drugs known to prolong the QT interval, correcting electrolyte abnormalities, and giving magnesium sulphate 1 g IV every 10 minutes. Expert help should be sought as other treatment options including overdrive pacing may be required to prevent collapse once the arrhythmia has been corrected.

Narrow complex tachycardia

The narrow complex tachycardias are supraventricular in origin.
- A regular narrow complex tachycardia may represent paroxysmal SVT or atrial flutter with 2:1 conduction. It may be difficult to differentiate between the two.
- The first step in treatment of regular narrow complex tachycardias is to attempt vagal manoeuvres (catapult into mouth or Valsalva manoeuvre).
- If the tachycardia persists, adrenaline A 0.5mg IV should be given as a rapid bolus using a large cannula and a large vein. The patient should be warned that they will feel nauseated and may experience chest discomfort for several seconds following the injection. As ECG (preferably work in the dark) should be recorded during the injections.
- If the ventricular rate slows transiently and then speeds up again, this may indicate atrial activity such as atrial flutter or other atrial tachycardia, and this should be treated accordingly.
- If there is no response (i.e., no transient slowing or termination of the tachycardia) to adrenaline 0.5mg IV or 25 mg IV infusion should be given and if there is no response, see further 12 mg IV bolus given over 30 minutes. Lack of response to adrenaline will occur if the tachycardia is given too slowly or into a peripheral vein.
- If adrenaline is contraindicated, or fails to terminate a regular narrow complex tachycardia and the administration of vagal manoeuvre (0.5–1 mg IV) over 1–2 minutes should be considered.
- Irregular narrow complex tachycardia is most likely to be AF with fast ventricular response or, less commonly, atrial flutter with variable AV conduction.
- Immediate treatment options include rate control with drug therapy, rhythm control using drugs to achieve chemically atrial fibrillation, rhythm control by synchronized cardioversion and treatment to prevent complications (e.g., anticoagulation). Expert help should be sought if in determining the most appropriate treatment for this individual patient.
Pharmacology: Cardiovascular

Question 32 of 121

A patient requires antibiotic treatment for an infection. You are aware that the patient is on long-term statin therapy. Which of the following antibiotics may increase the risk of myopathy in this patient:

a. Flucloxacillin  
b. Doxycycline  
c. Clarithromycin  
d. Ciprofloxacin  
e. Metronidazole

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Something wrong?
Pharmacology: Cardiovascular

A patient requires antibiotic treatment for an infection. You are aware that the patient is on long-term statin therapy. Which of the following antibiotics may increase the risk of myopathy in this patient:

a) Fluclaxacin
b) Doxycycline

c) Clarithromycin

d) Ciprofloxacin

e) Metronidazole

Answer

There is an increased incidence of myopathy if a statin is given with a rifamate, with lipid lowering doses of nicotinic acid, with fusidic acid, or with drugs that increase the plasma statin concentration, such as macrolide antibiotics, statinomycin and clarithromycin. Ciprofloxacin, rifampicin or clarithromycin, and ciprofloxacin alone may lower the activity of the enzyme involved in the removal of statins and therefore increase the risk of adverse effects.

Notes

Smokers may be used for primary or secondary prevention of cardiovascular disease and for treatment of primary or partial hypercholesterolemia. Statins are more effective than other lipid-lowering drugs, such as pravastatin or simvastatin.

Mechanism of action

Statins competitively inhibit 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase, an enzyme involved in cholesterol synthesis. Inhibition of HMG-CoA reductase reduces low-density lipoprotein (LDL) cholesterol levels by slowing down the production of cholesterol in the liver and increasing the liver’s ability to remove the LDL cholesterol already in the blood.

Indications

Statins should be offered to all patients, including the elderly, with cardiovascular disease such as those with coronary artery disease (including history of angina or acute myocardial infarction), or with arterial disease (including peripheral vascular disease, Renee’s angina or stroke, or transient ‘cerebral’ attacks). The use of statins should be considered in patients with high-risk of developing cardiovascular disease (primary prevention) which can be assessed using risk calculators.

Contraindications

Statins should be avoided in:

- People with active liver disease
- People with transaminase (alanine aminotransferase or aspartate aminotransferase) levels that are three or more times the upper limit of normal
- Pregnant or breast-feeding women (discontinue in the third trimester before attempting to conceive)

Cautions

Statins should be used cautiously in people:

- With a history of liver disease
- Who consume high levels of alcohol
- Who have the predisposing factors such as diabetes mellitus or obesity

Adverse effects of statins include:

- Headache
- Epistaxis
- Gastrointestinal disorders (such as constipation, flatulence, diarrhea, nausea, and diarrhea)
- Muscle/cramp and connective tissue disorders (such as myalgia, arthralgia, pain in the extremity, muscle spasm, joint swelling, and back pain)
- Hypersensitivity and diabetes
- Myopathy and rhabdomyolysis
- Intestinal Ache disease
- Hepatitis or biliary

Muscle effects

The risk for rhabdomyolysis, myositis, and rhabdomyolysis associated with statin use is rare. Although myalgia has been reported commonly in patients receiving statins, muscle toxicity is attributable to statin use. Muscle toxicity may occur with all statins, however the likelihood increases with higher doses and in certain patients. Statins should be used with caution in patients at increased risk of muscle toxicity, including those with a personal or family history of muscular disorders, previous history of muscle toxicity, a high alcohol intake, renal impairment or hypothyroidism.

There is an increased incidence of myopathy if a statin is given with a rifamate, with lipid lowering doses of nicotinic acid, with fusidic acid, or with drugs that increase the plasma statin concentration, such as macrolide antibiotics, statinomycin and clarithromycin. Ciprofloxacin, rifampicin or clarithromycin, and ciprofloxacin alone may lower the activity of the enzyme involved in the removal of statins and therefore increase the risk of adverse effects.
Pharmacology: Cardiovascular

What is the mechanism of action of simvastatin:

- Selectively inhibits intestinal absorption of cholesterol
- Reduces the release of VLDL by the liver
- Stimulates lipoprotein lipase
- Increases cholesterol excretion by binding to bile acids and preventing their reabsorption
- Decreases hepatic cholesterol synthesis through inhibition of HMG CoA reductase
Pharmacology: Cardiovascular

**Q33.** What is the mechanism of action of aspirin?

- **a)** Selectively inhibits prostaglandin formation by blocking cyclooxygenase (COX) 1 and 2.
- **b)** Reduces the release of TXA2 by the platelets.
- **c)** Stimulates thromboxane (TXA2) synthesis.
- **d)** Increases cholesterol excretion by blocking bile acids and preventing their reabsorption.
- **e)** Decreases hepatic cholesterol synthesis through inhibition of HMG-CoA reductase.

**Answer**

(a) Selectively inhibits prostaglandin formation by blocking cyclooxygenase (COX) 1 and 2.

**Notes**

Aspirin is used for primary or secondary prevention of cardiovascular disease and for treatment of primary or familial hypercholesterolemia. Aspirin is more effective than other lipid-lowering drugs at lowering LDL cholesterol concentration but its effectiveness on the fibrinolytic in reducing triglyceride concentration is limited. However, aspirin reduces cardiovascular disease events and mortality irrespective of the initial cholesterol concentration.

**Mechanism of action**

Aspirin competitively inhibits the cyclooxygenase (COX) 1 and 2 enzymes, blocking the production of thromboxane A2 (TXA2). By decreasing TXA2 levels, aspirin reduces platelet aggregability and the risk of cardiovascular events.

**Indications**

Aspirin is indicated for:

- Prevention of cardiovascular events in high-risk individuals.
- Reduction of pain and inflammation in conditions like osteoarthritis and rheumatoid arthritis.
- Treatment of irritable bowel syndrome.
- Prevention of recurrent ischemic stroke in patients with atrial fibrillation.

**Adverse effects**

Adverse effects include:

- Gastrointestinal symptoms like dyspepsia, heartburn, and gastrointestinal bleeding.
- Hypersensitivity reactions.
- Reye's syndrome in children with influenza and varicella.

**Muscle effects**

Muscle effects associated with aspirin use are rare. However, in rare cases, patients may experience myalgias, muscle weakness, and myoglobinuria.

**References**

- Aspirin and cardiovascular disease prevention.
- The role of aspirin in the management of cardiovascular disease.
- Efficacy and safety of aspirin for the prevention of cardiovascular disease.

**Further reading**

- American Heart Association guidelines for the management of patients with non-ST-elevation myocardial infarction.
- European Society of Cardiology guidelines for the management of acute coronary syndromes.
Pharmacology: Cardiovascular

Question 34 of 121

Sodium nitroprusside is contraindicated in which of the following:

- a. Peripheral artery disease
- b. Severe B12 deficiency
- c. Heart failure
- d. Myasthenia gravis
- e. Asthma

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Pharmacology: Cardiovascular

Question 34 of 121

Sodium nitroprusside is contraindicated in which of the following:

- a) Peripheral artery disease
- b) Severe B12 deficiency ✗
- c) Heart failure
- d) Myasthenia gravis ✗
- e) Asthma

Answer

Sodium nitroprusside is contraindicated in

- Compensatory hypertension
- Leber’s optic atrophy
- Severe vitamin B12 deficiency

Notes

Sodium nitroprusside decomposes in the blood to release nitric oxide, an unstable compound that causes vasodilation.

Indications

Sodium nitroprusside is indicated for

- Hypertensive emergencies
- Controlled hypotension in anaesthesia during surgery
- Acute or chronic heart failure

Contraindications

It is contraindicated in

- Compensatory hypertension
- Leber’s optic atrophy
- Severe vitamin B12 deficiency

Cautions

It is should be used with caution in

- Elderly
- Hypotension
- Hypothermia
- Hypothyroidism
- Impaired cerebral circulation
- Ischaemic heart disease

Adverse effects

Side effects associated with over rapid reduction in blood pressure include: headache, dizziness, nausea, retching, abdominal pain, perspiration, palpitation, anxiety, retrosternal discomfort; the infusion rate should be reduced if any of these side effects occur.

Side effects caused by excessive plasma concentration of the cyanide metabolite include tachycardia, sweating, hyperventilation, arrhythmias, marked metabolic acidosis; the drug should be discontinued and the cyanide antidote given if these effects occur.
Pharmacology: Cardiovascular

Question 35 of 121

Which of the following is NOT a typical side effect of digoxin:

- a. Yellow vision
- b. Diarrhoea
- c. Hypokalaemia
- d. Arrhythmias
- e. Gynaecomastia

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Something wrong?
Pharmacology: Cardiovascular

Answer

Diabetes does not cause hypertension, but hypertension does predispose diabetics to cardiovascular disease.

The adverse effects of drugs in diabetes are frequently due to common therapeutic side effects and include:

- Cardiac adverse effects
- Neurovascular and autonomic effects
- Hypoglycemia
- Hypertension
- Hyperglycemia
- Hyperkalemia
- Sodium retention
- Hyperlipidemia
- GI effects
- Nephrotoxicity
- Osteoporosis
- Thromboembolism and anaphylactic reactions
- Hypokalemia and hypomagnesemia
- Vitamin deficiencies
- Hyperuricemia
- Hypercalcemia
- Hemolytic anemia
- Aplastic anemia
- Transfusion reactions

Notes

Diabetes mellitus is a disease characterized by chronic hyperglycemia and is associated with long-term complications that affect multiple organ systems. The treatment of diabetes involves lifestyle modifications and the use of various medications to control blood glucose levels. The complications of diabetes can lead to serious health problems, including cardiovascular disease.

Mechanism of Action

Antihypertensive Effect

Diabetes mellitus is often associated with high blood pressure (hypertension), which can be managed through the use of antihypertensive medications. These medications work by lowering blood pressure and reducing the risk of cardiovascular complications such as heart disease and stroke.

Corticosteroid Treatment

Corticosteroids are a class of medications used to treat inflammation and autoimmune diseases. They work by suppressing the activity of the immune system, reducing inflammation, and controlling symptoms. Corticosteroids are commonly used to treat conditions such as rheumatoid arthritis, lupus, and inflammatory bowel disease.
Pharmacology: Cardiovascular

Question 36 of 121

Which of the following is NOT a common side effect of amiodarone:

- a. Photosensitivity
- b. Hyperthyroidism
- c. Hypothyroidism
- d. Hepatotoxicity
- e. Nephrotoxicity

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Something wrong?
Pharmacology: Cardiovascular

Question 3 of 31

Which of the following is NOT a common side effect of amiodarone:

a) Bradycardia  
b) Hypertension  

c) Hyperthyroidism  

Hyperpotency  

Hyperthyroidism

Answer

Common side effects of amiodarone include:

- Bradycardia
- Nausea and vomiting
- Thyroid disorders - hyperthyroidism and hypothyroidism
- Persistent state grey skin discoloration
- Photosensitivity
- Pulmonary toxicity (including pneumonitis and fibrosis)
- Hepatotoxicity
- Corneal microdeposits (sometimes with night glare)
- Peripheral neuropathy
- Sleep disorders

Notes

Amiodarone hydrochloride is used in the treatment of arrhythmias, particularly when other drugs are ineffective or contraindicated. However, its long-term use is often restricted by various adverse effects such as photosensitivity, thyroid disorders, corneal microdeposits, neuropathy and pulmonary abnormalities.

Mechanism of action

Amiodarone has blocking actions on several channels (e.g. K+ and inactivated Na+ channel) and beta-adrenoceptors. It acts by slowing repolarization and prolonging the action potential and refractory period in all cardiac tissues, depressing sinus node automaticity and slowing conduction.

Indications

Amiodarone can be used for paroxysmal supraventricular, nodal and ventricular tachycardias, atrial fibrillation and flutter, and ventricular fibrillation. It can also be used for tachyarrhythmias associated with Wolff-Parkinson-White syndrome.

Intravenous injection of amiodarone hydrochloride can be used in cardioversion reasynchronization for ventricular fibrillation or polymorphic ventricular tachycardia unresponsive to other interventions.

Contraindications

Amiodarone is contraindicated in:

- Severe conduction disturbances (unless pacemaker fitted)
- Sino-atrial disease (unless pacemaker fitted)
- Idiopathic
- Structural heart block (except in cardiac arrest)
- Sino-bradyarrhythmia (except in cardiac arrest)
- Thyroid dysfunction

Intravenous use should be avoided in cardiogenic shock, congestive heart failure, circulatory collapse, severe arterial hypotension and severe respiratory failure.

Cautions

Amiodarone should be used with caution in:

- Acute pericarditis
- Conduction disturbances (in excessive dosage)
- Elderly
- Heart failure
- Hypokalaemia
- Severe bradycardia (in excessive dosage)
- Severe hepatic cellular toxicity
- Concomitant therapy with drugs that prolong the QT interval

Adverse effects

Common side effects of amiodarone include:

- Bradycardia
- Nausea and vomiting
- Thyroid disorders - hyperthyroidism and hypothyroidism
- Persistent state grey skin discoloration
- Photosensitivity
- Pulmonary toxicity (including pneumonitis and fibrosis)
- Hepatotoxicity
- Corneal microdeposits (sometimes with night glare)
- Peripheral neuropathy
- Sleep disorders
### Pharmacology: Cardiovascular

**Question 37 of 121**

**What is the mechanism of action of clopidogrel:**

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<td>a</td>
<td>Inhibition of platelet thromboxane A2 synthesis</td>
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<tr>
<td>b</td>
<td>Inhibition of binding of ADP to its platelet receptor</td>
</tr>
<tr>
<td>c</td>
<td>Inhibition of GPIIb/IIIa receptor sites</td>
</tr>
<tr>
<td>d</td>
<td>Inhibition of the breakdown of cAMP</td>
</tr>
<tr>
<td>e</td>
<td>Inhibition of thrombin-induced platelet aggregation</td>
</tr>
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</table>
Pharmacology: Cardiovascular

Question 27 of 121

What is the mechanism of action of clopidogrel:

- a) Inhibition of platelet thromboxane A2 synthesis
- b) Inhibition of binding of ADP to its platelet receptor ✔
- c) Inhibition of GPIIb/IIa receptor sites
- d) Inhibition of the breakdown of cAMP
- e) Inhibition of thrombin-induced platelet aggregation

Answer

Clopidogrel, a thienopyridine derivative, inhibits the binding of ADP to its platelet receptor (P2Y12 ADP-receptor), inhibiting platelet adhesion and aggregation.

Notes

Antiplatelet drugs decrease platelet aggregation and inhibit thrombus formation in the arterial circulation, because in faster-flowing vessels, thrombi are composed mainly of platelets with little fibrin.

Mechanism of action

Clopidogrel, a thienopyridine derivative, inhibits the binding of ADP to its platelet receptor (P2Y12 ADP-receptor), inhibiting platelet adhesion and aggregation.

Indications

Clopidogrel is used for:

- the prevention of atherothrombotic events in patients with a history of symptomatic ischaemic disease
- the management of ACS (in combination with low-dose aspirin)
- the prevention of atherothrombotic events in percutaneous coronary intervention (in combination with low-dose aspirin)
- the prevention of atherothrombotic and thromboembolic events in patients with atrial fibrillation (who cannot take warfarin) and in stroke (in patients who cannot take aspirin)

Contraindications

Clopidogrel should be avoided in:

- People with active pathological bleeding, such as peptic ulcer or intracranial haemorrhage
- People with severe hepatic impairment
- Women who are pregnant or breastfeeding

Clopidogrel should be used with caution in people who may be at high risk of increased bleeding, for example those receiving treatment with warfarin, other anti-platelets or other drugs known to increase gastrointestinal bleeding (such as NSAIDs, SSRIs and corticosteroids).

Adverse effects

- Clopidogrel is associated with an increased risk of bleeding (for example gastrointestinal bleeding)
- Other common adverse effects include diarrhoea, abdominal pain, and dyspepsia
- Clopidogrel is known to cause pruritus and urticaria, but these adverse reactions are generally uncommon
- Gynaecomastia is a rare adverse effect of clopidogrel
- Thrombotic thrombocytopenic purpura is a very rare adverse effect of clopidogrel, and it sometimes occurs after a short exposure to clopidogrel.
Pharmacology: Cardiovascular

Mannitol is primarily indicated for which of the following:

- **A** Acute pulmonary oedema
- **B** Cerebral oedema
- **C** Oedema in chronic heart failure
- **D** Hypertension
- **E** Ascites secondary to liver cirrhosis
Pharmacology: Cardiovascular

Question 38 of 121

Mannitol is primarily indicated for which of the following:

a) Acute pulmonary oedema
b) Cerebral oedema ✓
c) Oedema in chronic heart failure
d) Hypertension
e) Ascites secondary to liver cirrhosis

---

Answer

Mannitol is an osmotic diuretic that can be used to treat cerebral oedema and raised intracocular pressure.

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Notes

Mannitol is an osmotic diuretic that can be used to treat cerebral oedema and raised intracocular pressure.

Mechanism of action

Mannitol is an easily filtered, poorly reabsorbed solute that alters the diffusion of water relative to sodium by ‘binding’ water. As a result, net reabsorption of Na⁺ is reduced.

Contraindications

Mannitol is contraindicated in:

- Anuria
- Intracranial bleeding (except during craniotomy)
- Severe cardiac failure
- Severe dehydration
- Severe pulmonary oedema

Adverse effects

Common side effects include:

- Fluid and electrolyte imbalance
- Hypotension
- Thrombophlebitis
Pharmacology: Cardiovascular

Which of the following drugs can be used as reversal agent for heparin:

- a. Hydroxocobalamin
- b. Protamine sulfate
- c. Phytomenadione
- d. Idarucizumab
- e. Dried prothrombin complex

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Pharmacology: Cardiovascular

Question 3 of 121

Which of the following drugs can be used as a reversal agent for heparin:

a) Heparinase
b) Protamine sulfate ✓
c) Phenprocoumon
d) Marlexather

Notes

If haemorrhage occurs it is usually sufficient to withdraw unfractionated or low molecular weight heparins, but if rapid reversal of the effects of the heparins is required, protamine sulfate is a specific antidote (but only partially reverses the effects of low molecular weight heparins).

Answer

The main use of anticoagulants is to prevent thrombus formation or extension of an existing thrombus in the slower-moving veins or vessels of the circulation, where the thrombus consists of fibrin, red and white cells. Anticoagulants are of little use in preventing thrombus formation in arteries, where in faster-moving vessels thrombi are composed mainly of platelets with little fibrin.

Mechanism of action

Heparin potentiates the activity of antithrombin III causing inactivation of thrombin. The heparin-antithrombin III complex also inhibits factor Xa and some other factors. Low molecular weight heparin (LMWH) preparations inhibit only factor Xa, PT and aPTT may both be prolonged but the PT may be less so.

Contraindications

Heparins are contraindicated:

- In people with current or history of heparin-induced thrombocytopenia
- In people with active bacterial endocarditis
- In people with active major bleeding, and conditions with high risk of uncontrolled bleeding, including recent haemorrhagic stroke, major trauma, recent brain, spinal cord or eye surgery, haemorrhage and thrombocytopenia
- In people with active cancer or uncontrolled uterine

Adverse effects

- Bleeding
- Heparin-induced thrombocytopenia ( Immune-mediated effect that usually develops after 5–10 days, signs may include a 50% reduction of platelet count, thrombosis, or skin allergy; if HIT is suspected or confirmed, heparin should be discontinued and an alternative anticoagulant given)
- Hyperglycaemia due to inhibition of aldosterone secretion; patients with diabetes mellitus, chronic renal failure, acclimatisation, rapid plasma potassium in those taking potassium sparing drugs can be more susceptible
- Osteoporosis (risk lower with LMWH)
- Arthritis
- Hypersensitivity reactions
- Injection site reactions

Low molecular weight heparin vs unfractionated heparin

Unfractionated heparin is usually given by continuous intravenous infusion for the smoothest control and is the treatment of choice where rapid reversal of anticoagulation may be required (e.g. in surgical patients or late pregnancy). Therapy is monitored by monitoring the aPTT at 1:1 to 2:1 times the upper limit of normal.

Low molecular weight heparin (LMWH) preparations have largely replaced unfractionated heparins.

Advantages of LMWH

- Greater ability to inhibit factor Xa directly, interacting less with platelets and so may have a lower tendency to cause bleeding
- Greater bioavailability and longer half-life in plasma making once daily subcutaneous administration possible
- More predictable dose response avoiding the need for routine anticoagulant monitoring
- Lower associated risk of heparin-induced thrombocytopenia or osteoporosis

Haemorrhage

Because it has a short duration of action, if haemorrhage occurs it is usually sufficient to withdraw unfractionated or low molecular weight heparin, but if rapid reversal of the effects of the heparin is required, protamine sulfate is a specific antidote (but only partially reverses the effects of low molecular weight heparin).

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Pharmacology: Cardiovascular

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Which of the following best describes digoxin:

a. A positive inotrope and negative chronotrope
b. A negative inotrope and positive chronotrope
c. A positive inotrope and positive chronotrope
d. A negative inotrope and negative chronotrope
e. A positive chronotrope with no inotropic effect

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Which of the following best describes digoxin:

- a) A positive inotrope and negative chronotrope
- b) A negative inotrope and positive chronotrope
- c) A positive inotrope and positive chronotrope
- d) A negative inotrope and negative chronotrope
- e) A positive inotrope with no chronotropic effect

Answer:

Digoxin is a cardiac glycoside that increases the force of myocardial contraction (positive inotrope), and slows the heart’s rate (negative chronotrope). Therefore, option (a) is correct.

Notes:

- Digoxin is a cardiac glycoside that increases the force of myocardial contraction (positive inotrope), and slows the heart rate (negative chronotrope).
- The adverse effects of digoxin can include:
  - Hypotension
  - Arrhythmias
  - Cardiac failure

Mechanism of action:

- Inotropic effect:
  - Digoxin inhibits the sodium-potassium pump, which increases intracellular sodium and reduces intracellular potassium. This leads to increased calcium release from the sarcoplasmic reticulum, which in turn increases myocardial contractility.

Indications:

- Digoxin is used to treat atrial fibrillation and flutter, and to control heart rate in patients with chronic atrial fibrillation or flutter. It is also used to control heart rate in patients with post-myocardial infarction.

Contraindications:

- Digoxin is contraindicated in:
  - Severe renal or hepatic failure
  - Hypokalemia
  - Hypomagnesemia
  - Heart valve replacement surgery
  - Hypothyroidism
  - Hypocalcemia
  - Hypothyroidism
  - Concomitant use with other drugs that can cause digitalis toxicity

Cautions:

- Digoxin should be used with caution in:
  - Hypothyroidism
  - Hypokalemia
  - Hypomagnesemia
  - Calcium channel blockers
  - Other drugs that can affect cardiac function

Adverse effects:

- The adverse effects of digoxin are usually related to its effect on cardiac muscle, and include:
  - Bradycardia
  - Tachycardia
  - Atrial fibrillation
  - Palpitations
  - Nausea
  - Vomiting
  - Diarrhea
  - Constipation
  - Confusion
  - Delirium
  - Seizures
  - Hypotension
  - Arrhythmias

Dose toxicity:

- The effective dose of digoxin is 0.5 to 2 mg daily, and the toxic dose is 2 to 5 mg daily. The oral bioavailability of digoxin is approximately 50%, and the plasma half-life is 1 to 3 days.

In case of toxicity, the following measures should be taken:

- Supportive care: maintain airway, ensure ventilation, and monitor vital signs.
- Treat the underlying cause of the digoxin toxicity, such as arrhythmias or electrolyte imbalances.
- Hemodialysis or peritoneal dialysis may be necessary in severe cases.

Resources:

- The Royal College of Emergency Medicine
- British Cardiac Society
- European Society of Cardiology
- American Heart Association
- National Institute for Health and Care Excellence
- Canadian Society of Cardiology

Further reading:

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Which of the following is NOT an adverse effect of furosemide:

A. Hyperglycaemia
B. Gout
C. Ototoxicity
D. Urinary retention
E. Metabolic acidosis

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which of the following is NOT an adverse effect of furosemide:

- Hypokalemia
- Nausea
- Otolithy
- Diuretic resistance
- Metabolic acidosis

Answer

Adverse effects of loop diuretics include:

- Mild potassium disturbances, pancreatitis, and hepatic encephalopathy
- Hypokalemia
- Acute urinary retention
- Water and electrolyte imbalance
- Hyperkalemia, hypokalemia, hyperlipidemia, hyperglycemia, hyperuricemia
- Hyperuricemia, hyponatremia, dehydration, and renal thrombosis
- Metabolic acidosis
- Hyperkalemia
- Blood disorders (bone marrow suppression, thrombocytopenia, and anemia)
- Visual disturbance, tinnitus, and dizziness
- Hyperkalemia

Notes

Loop diuretics are powerful diuretics used in acute pulmonary edema due to left ventricular failure. Intravenous administration produces rapid diuresis and reduces preload sooner than would be expected from the time of iv administration. They are also used in edema in patients with chronic heart failure. Diuretic-resistant edema can be treated with a loop diuretic combined with a thiazide or related diuretic.

If necessary, a loop diuretic can be added to the thiazide or related diuretic to achieve better control of blood pressure in those with resistant hypertension, or in patients with hyperkalemia and fluid overload.

Mechanism of action

Furosemide is a thiazide diuretic that acts to increase urine output and reduce blood pressure. It inhibits sodium and chloride reabsorption in the thick ascending limb of the loop of Henle, thereby increasing urine output and reducing blood pressure.

Furosemide and bumetanide are similar in activity, both acting within the loop of Henle to increase urine output and reduce blood pressure.

Contraindications

Loop diuretics are contraindicated in:

- Hypokalemia and dehydration
- Severe hyperkalemia or hypernatremia
- Anemia, acute kidney injury, or chronic kidney disease due to nephrotoxicity
- Comorbid and or concomitant states associated with liver or renal disease

Caution

Loop diuretics can exacerbate diabetes, but hyperkalemia is less likely than with thiazides and gentamicin.

Furosemide is not excreted by the kidney, so urinary retention can occur, although this is less likely if small doses and slow parenteral dosing are used initially.

Hyperkalemia, hypokalemia, and electrolyte disturbances should be corrected before initiation of treatment.

Hyponatremia, hyperkalemia, and medications may reduce sodium and increase risk of side effects.

Lower initial doses of diuretics should be used in the elderly because they are particularly susceptible to the side effects.

Adverse effects

Adverse effects of loop diuretics include:

- Mild potassium disturbances, pancreatitis, and hepatic encephalopathy
- Hypokalemia
- Acute urinary retention
- Water and electrolyte imbalance
- Hyperkalemia, hypokalemia, hyperlipidemia, hyperglycemia, hyperuricemia
- Hyperuricemia, hyponatremia, dehydration, and renal thrombosis
- Metabolic acidosis
- Hyperkalemia
- Blood disorders (bone marrow suppression, thrombocytopenia, and anemia)
- Visual disturbance, tinnitus, and dizziness
- Hyperkalemia

Hepatokinesis

Hepatokinesis can occur with both thiazide and loop diuretics. The risk of hepatokinesis depends on the duration of action as well as the potency of the drug, with thiazides being more potent than loop diuretics.

Side effects are rare, but severe hepatotoxicity can occur.

Resources

- The Royal College of Emergency Medicine
- Quick Reference Guide for Emergency Medicine
- Emergency Decision Aid
- National Institute for Health and Care Excellence
- NICE Pathfinder – Urinary System
- Royal College of Emergency Medicine
- BMJ Best Practice
- Medscape
- UpToDate

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Which of the following best describes verapamil:

a) Negative inotrope, positive chronotrope
b) Positive inotrope, negative chronotrope
c) Positive inotrope, positive chronotrope
d) Negative inotrope, negative chronotrope
e) Negative inotrope, no chronotropic effect
Which of the following best describes verapamil:

- a) Negative inotropic, positive chronotropic
- b) Positive inotropic, negative chronotropic
- c) Positive inotropic, positive chronotropic
- d) Negative inotropic, negative chronotropic
- e) Negative inotropic, no chronotropic effect

**Answer**

Verapamil is used for the treatment of angina, hypertension, and arrhythmias. Verapamil is highly negatively inotropic and reduces cardiac output, slows the heart rate and may impair atrioventricular conduction. It may precipitate heart failure, exacerbate conduction disorders, and cause hypertension at high doses and should not be used with beta-blockers. Constipation is the most common side-effect.

**Notes**

Calcium channel blockers are widely used in the treatment of angina (second line to beta-blockers) and also for hypertension, heart failure and arrhythmias.

Calcium channel blockers vary widely in their predilections for the treatment of the various possible sites of action and in their therapeutic effects and may be divided into the dihydropyridine type (e.g., amlodipine, nifedipine and isradipine) and the non-dihydropyridine type (e.g., verapamil, diltiazem).

**Mechanism of action**

Calcium channel blockers inhibit L-type voltage-sensitive calcium channels in smooth muscle of the heart, causing relaxation and vasodilation. They also block calcium channels within the myocardium and conducting tissues of the heart which produces a negative inotropic effect by reducing calcium influx during the plateau phase of the action potential.

The dihydropyridines have relatively little effect on the heart because they have a much higher affinity for skeletal muscle channels found more frequently in vascular muscle. Furthermore, at clinical doses, vasodilation results in a reflex increase in sympathetics tone that counters the mild negative inotropic effect. The non-dihydropyridines are rate-limiting calcium-channel blockers that depress the sino-atrial node and slow conduction in the atrioventricular node, causing a mild resting bradycardia.

**Contraindications**

Non dihydropyridine CCBs:
- Atrial flutter or fibrillation
- Heart failure or history of heart failure (may precipitate or aggravate symptoms)
- Cardiac outflow obstruction (e.g. significant aortic stenosis or obstructive hypertrophic cardiomyopathy: chest pain may result in reduced cerebral output)
- Second or third degree AV block (may induce complete AV block)
- Severe bronchial asthma
- Scleroderma syndrome

Dihydropyridine CCBs:
- Uncontrolled heart failure
- Severe hypotension
- Cardiac outflow obstruction

**Adverse effects**

- Gastrointestinal adverse effects – constipation, nausea, dyspepsia
- Bradycardia, A-V block, reflex tachycardia, palpitations
- Vasovasodilator adverse effects – flushing, dizziness, headache, postural hypotension, ankle swelling (more common with dihydropyridine calcium-channel blockers and often improves with continued use, although ankle swelling often persists)
- Gingival hyperplasia
- Muscular cramps
- Myalgia and arthralgia

**Verapamil**

Verapamil is used for the treatment of angina, hypertension, and arrhythmias. Verapamil is highly negatively inotropic and reduces cardiac output, slows the heart rate and may impair atrioventricular conduction. It may precipitate heart failure, exacerbate conduction disorders, and cause hypertension at high doses and should not be used with beta-blockers. Constipation is the most common side-effect.

**Nifedipine**

Nifedipine relaxes vascular smooth muscle and dilates coronary and peripheral arteries. Nifedipine has less myocardial effects than verapamil and has no antiarrhythmic properties but has more influence on the vessel. Unlike verapamil, it rarely precipitates heart failure because any negative inotropic effect is offset by a reduction in left ventricular work.

**Nicardipine**

Nicardipine is related to nifedipine but the smooth muscle relaxant effects preferentially act on cerebral arteries. It is used solely for the prevention and treatment of vascular spasm following aneurysm surgery or head injury.
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Thiazide diuretics are primarily indicated for which of the following:

- a) Acute pulmonary oedema
- b) Cerebral oedema
- c) Oedema in chronic heart failure
- d) Acute angle-closure glaucoma
- e) Ascites secondary to liver cirrhosis

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Thiazide diuretics are primarily indicated for which of the following:

1. Acute pulmonary oedema
2. Central oedema
3. Oedema in chronic heart failure
4. Acute single-dose glucagon
5. Acute secondary to liver cirrhosis

Answer

Thiazide diuretics are moderately potent diuretics, and are used to relieve oedema in chronic heart failure, and in the management of hypertension. They act within 1 to 2 hours of oral administration and must have a duration of action of 12 to 24 hours.

Notes

Thiazide diuretics are moderately potent diuretics, and are used to relieve oedema in chronic heart failure, and in the management of hypertension. They act within 1 to 2 hours of oral administration and must have a duration of action of 12 to 24 hours.

Mechanism of action

Thiazide act mainly on the early segments of distal tubule where they inhibit NaCl reabsorption by blocking the Na+/H+ exchanger. Excretion of Cl-/Na+ and accompanying water is increased. The increased Na+ load to the distal tubule stimulates Na+ exchange with K+ and H+, increasing their excretion and causing hypokalaemia and a metabolic alkalosis. Excretion of Ca++ is reduced.

Indications

Bendroflumethiazide is used for oedema in mild or moderate heart failure. Combination diuretic therapy (with loop and thiazide diuretic) may be effective in patients with oedema resistant to treatment with one diuretic.

Thiazide diuretics are advised for the treatment of hypertension but are no longer considered the first-line diuretic for this indication. In the management of hypertension a low dose of a thiazide produces a maximal or near-maximal blood pressure lowering effect, with very little biochemical disturbance. Higher doses cause more marked changes in plasma potassium, sodium, uric acid, glucose, and body fluids, with little advantage in blood pressure control.

Contraindications

Thiazide diuretics are contraindicated in:

- Addison’s disease
- Hypercalcemia
- Hypoparathyroidism
- Hypercalcemia
- Hyperkalaemia
- Severe hepatic impairment (may precipitate encephalopathy)

Cautions

Thiazide diuretics should be used with caution in:

- Diabetes mellitus (may exacerbate)
- Gout (may exacerbate)
- Systemic lupus erythematosus (may exacerbate)
- Hyperaldosteronism
- Myasthenia gravis
- Hepatitis syndrome

Adverse effects

Lower initial doses of diuretics should be used in the elderly because they are particularly susceptible to the side effects. The dose should then be adjusted according to renal function.

Common side effects of thiazide diuretics include:

- Excessive diuresis
- Postural hypotension, dehydration, renal impairment
- Acidosis and electrolyte imbalance
- Hypokalaemia, hyperglycaemia, hyperuricaemia, hyperlipidaemia, hyperchloremic acidosis
- Metabolic alkalosis
- Hypokalaemia and gout
- Impaired glucose tolerance and hyperglycaemia
- Altered plasma lipid concentrations
- Mild gastrointestinal disturbances

Hypokalaemia

Hypokalaemia can occur with both thiazide and loop diuretics. The risk of hypokalaemia depends on the duration of action as well as the potency and is thus greater with thiazides than with an equipotent dose of a loop diuretic. Hypokalaemia is dangerous in severe coronary disease and in patients receiving treatment with cardiac glycosides. Often, the use of potassium sparing diuretics avoids the need to take potassium supplements.
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Regarding warfarin, which of the following statements is CORRECT:

- a. Warfarin takes 24 hours for the full anticoagulant effect to develop.
- b. Warfarin is monitored by the activated partial thromboplastin time (aPTT) reported as the INR.
- c. Warfarin potentiates the activity of antithrombin and impairs platelet function.
- d. Warfarin should be reversed if the INR > 8.0 regardless of any bleeding.
- e. Warfarin can be safely used in pregnancy.

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9. Effect of Therapeutic Drugs on the Endocrine System
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12. Effect of Therapeutic Drugs on the Blood

**Answer**

No. The drugs listed are chemicals that can be altered by the human body, which is the heart. Most drugs are metabolized in the liver, with some also metabolized in the kidneys and other organs. This is why they do not affect the heart directly. Some drugs can affect the heart indirectly, such as digitalis, which is used to treat certain heart conditions. Digitalis can affect the heart by slowing down the heartbeat and increasing the strength of each beat. Other drugs, such as beta-blockers, can help to reduce the workload of the heart and prevent it from working too hard.

**Notes**

The effects of drugs on the heart are complex and can vary depending on the type of drug and the individual patient. Some drugs can cause heart problems when taken in high doses, while others can worsen existing heart conditions. It is important to consult with a healthcare professional before taking any new medication to ensure that it is safe and effective for your particular needs.

**References**


**Resources**

- American Heart Association
- National Institutes of Health
- World Health Organization
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Which of the following is an adverse effect of statin therapy:

- a. Nephrotoxicity
- b. Myositis
- c. Ototoxicity
- d. Hypoglycaemia
- e. Megaloblastic anaemia

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Pharmacology: Cardiovascular

Question: Which of the following is an adverse effect of statin therapy?

- a) Nephrotoxicity
- b) Myositis
- c) Dizziness
- d) Hypoglycaemia
- e) Megaloblastic anaemia

Answer:

The risk of nephrotoxicity, myositis, and rhabdomyolysis associated with statin use is rare. Although myositis has been reported commonly in patients receiving statins, muscle tenderness is not attributable to statin use alone. Muscle injury can occur with all statins; however, the risk increases with higher doses and in certain patients. Statins should be used with caution in patients at increased risk of muscle toxicity, including those with a personal or family history of muscular disorders, previous history of muscular toxicity, high alcohol intake, renal impairment or nephrotic syndrome.

Notes

Statins may be used for primary or secondary prevention of cardiovascular disease and for treatment of primary or familial hypercholesterolaemia.

Statins are more effective than other lipid-lowering drugs at lowering LDL-cholesterol concentrations, but they are less effective than the fibrates in reducing triglyceride concentrations. However, statins reduce cardiovascular disease events and total mortality irrespective of the initial cholesterol concentration.

Mechanism of Action

Statins competitively inhibit 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase, an enzyme involved in cholesterol synthesis. Inhibition of HMG-CoA reductase reduces the liver's production of lipoproteins (LDL cholesterol) levels by slowing down the synthesis of cholesterol in the liver, and increasing the liver's ability to remove the LDL cholesterol already in the blood.

Indications

Statins should be offered to all patients, including the elderly, with cardiovascular disease such as those with coronary heart disease (including history of angina or acute myocardial infarction) or recent acute arterial disease (including peripheral vascular disease, non-haemorrhagic stroke, or transient ischaemic attack). The use of statins should be considered in patients with a high risk of developing cardiovascular disease (primary prevention) which can be assessed using risk calculators.

Contraindications

Statins should be avoided in:

- People with active liver disease
- People with transaminase (alanine aminotransferase or aspartate aminotransferase) levels that are three or more times the upper limit of normal
- Pregnant or breastfeeding women (discontinue 3 months before attempting to conceive)

Cautions

Statins should be used with caution in people:

- With a history of liver disease
- Who consume high level of alcohol
- With predisposing factors for rhabdomyolysis such as age above 70 years, concurrent use with an interacting drug, renal impairment, hypothyroidism, and assessed or familial history of hereditary muscular disorders

Adverse effects

Adverse effects of statin therapy include:

- Headache
- Fatigue
- Gastrointestinal disorders such as constipation, diarrhoea, nausea, and diarrhoea
- Musculoskeletal and connective tissue disorders such as myalgia, arthralgia, pain in the extremities, muscle spasms, joint swelling, and back pain
- Hyperglycaemia and diabetes
- Myopathy and rhabdomyolysis
- Interstitial lung disease
- Hepatotoxicity

Muscle effects

The risk of myositis, myalgia, and rhabdomyolysis associated with statin use is low. Although myalgia has been reported commonly in patients receiving statins, muscle tenderness is not attributable to statin use alone. Muscle toxicity can occur with all statins, however the likelihood increases with higher doses and in certain patients. Statins should be used with caution in patients at increased risk of muscle toxicity, including those with a personal or family history of muscular disorders, previous history of muscular toxicity, high alcohol intake, renal impairment or nephrotic syndrome.

There is an increased incidence of myopathy if a statin is given with a fibrate, with lipid lowering doses of niacin and, with certain drugs that increase the plasma statin concentrations, such as niacin and anilino-bisbenzimidazoles (carboxylic acid drugs), indinavir and lisinopril, and ciclosporin. Close monitoring of liver function tests and if muscle symptoms occur, of creatine kinase is necessary.
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What is the mechanism of action of warfarin:

- a Blocks GPIIb/IIIa receptor sites
- b Directly inhibits factor Xa
- c Inhibits free thrombin, fibrin-bound thrombin, and thrombin-induced platelet aggregation
- d Inhibits vitamin K dependent clotting factors
- e Inhibits cyclo-oxygenase
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What is the mechanism of action of nitrates?

- The inhibition of cyclic guanosine monophosphate (cGMP) phosphodiesterase, leading to increased cGMP levels, which relaxes smooth muscle.

What is the role of nitrates in the treatment of angina pectoris?

- Nitrates are used to relieve angina pectoris by dilating blood vessels, reducing the workload on the heart.

What are the side effects of nitrate therapy?

- Headache, flushing, and dizziness.

What is the difference between short-acting and long-acting nitrates?

- Short-acting nitrates act quickly but have a shorter duration of action, while long-acting nitrates have a longer duration of action.

What are the contraindications for nitrate therapy?

- Hypotension, tachycardia, and severe cardiac disease.

What are the adverse effects of nitrate therapy?

- Headache, flushes, and dizziness.

What are the indications for nitrate therapy?

- Angina pectoris, hypertension, and acute coronary syndromes.

What are the mechanisms of action of alpha blockers?

- Blockade of alpha-1 receptors, leading to vasodilation and decreased blood pressure.

What are the side effects of alpha blockers?

- Orthostatic hypotension, dizziness, and headache.

What are the indications for alpha blockers?

- Hypertension, benign prostatic hyperplasia, and pulmonary hypertension.

What are the mechanisms of action of beta blockers?

- Blockade of beta-1 and beta-2 receptors, leading to decreased cardiac output and blood pressure.

What are the side effects of beta blockers?

- Bradycardia, hypotension, and diabetes.

What are the indications for beta blockers?

- Hypertension, angina pectoris, and cardiac arrhythmias.

What are the mechanisms of action of diuretics?

- Inhibition of sodium and water reabsorption in the nephron, leading to decreased blood volume and blood pressure.

What are the side effects of diuretics?

- Electrolyte disturbances, hypokalemia, and hyperuricemia.

What are the indications for diuretics?

- Heart failure, hypertension, and edema.

What are the mechanisms of action of calcium channel blockers?

- Blockade of calcium ions in the cardiac and vascular smooth muscle, leading to decreased blood pressure.

What are the side effects of calcium channel blockers?

- Headache, dizziness, and flushing.

What are the indications for calcium channel blockers?

- Angina pectoris, hypertension, and supraventricular tachycardia.

What are the mechanisms of action of ACE inhibitors?

- Inhibition of angiotensin converting enzyme, leading to decreased angiotensin II levels and decreased blood pressure.

What are the side effects of ACE inhibitors?

- Cough, hyperkalemia, and angioedema.

What are the indications for ACE inhibitors?

- Hypertension, heart failure, and diabetic nephropathy.

What are the mechanisms of action of angiotensin receptor blockers?

- Blockade of angiotensin II receptors, leading to decreased blood pressure and proteinuria.

What are the side effects of angiotensin receptor blockers?

- Cough, hyperkalemia, and angioedema.

What are the indications for angiotensin receptor blockers?

- Hypertension, heart failure, and diabetic nephropathy.

What are the mechanisms of action of statins?

- Inhibition of 3-hydroxy-3-methylglutaryl coenzyme A reductase, leading to decreased cholesterol levels.

What are the side effects of statins?

- Myalgias, liver dysfunction, and rhabdomyolysis.

What are the indications for statins?

- Hypercholesterolemia and prevention of cardiovascular events.

What are the mechanisms of action of fibrates?

- Increase in high-density lipoprotein cholesterol and decrease in triglycerides.

What are the side effects of fibrates?

- Gastrointestinal side effects and rhabdomyolysis.

What are the indications for fibrates?

- Hypertriglyceridemia and mixed dyslipidemia.

What are the mechanisms of action of omega-3 fatty acids?

- Inhibition of eicosanoid synthesis and inflammation.

What are the side effects of omega-3 fatty acids?

- Gastrointestinal side effects and increased bleeding.

What are the indications for omega-3 fatty acids?

- Reduction of cardiovascular events in patients with coronary artery disease.
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Which of the following is NOT an adverse effect associated with heparin therapy:

a. Osteoporosis
b. Alopecia
c. Hyperkalaemia
d. Thrombocytopenia
e. Teratogenicity

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Which of the following is NOT an adverse effect associated with heparin therapy?

a) Osteoporosis
b) Aplasia
c) Hypertension X

d) Thrombocytopenia

e) Tenosynovitis

Answer

Heparin does not cross the placenta and is safe to use in pregnancy, unlike warfarin.

Adverse effects of heparin include:

- Bleeding
- Heparin-induced thrombocytopenia (immune-mediated effect that usually develops after 5–30 days; signs may include a 30% reduction in platelet count, thrombocytopenia, or skin allergy; HIT is suspected or confirmed; heparin should be discontinued and an alternative anticoagulant given)
- Hypocalcaemia (due to inhibition of extracellular secretions of patients with diabetes mellitus, chronic renal failure, arthritis, raised plasma potassium or those taking potassium sparing drugs, seem to be more susceptible)
- Osteopenia (risk lower with LMWH)
- Aplasia
- Hypersensitivity reactions
- Injection site reactions

Notes

The main use of anticoagulants is to prevent thrombosis formation or extension of an existing thrombus in the slower moving venous side of the circulation, where the thrombus consists of a fibrous web embedded with platelets and red cells. Anticoagulants are not of use in preventing thrombus formation in arteries, for faster flowing arterial thrombi are composed mainly of platelets with little fibrin.

Mechanism of action

Heparin potentiates the activity of antithrombin III causing neutralization of thrombin. The heparin-antithrombin III complex also inhibits factor Xa and other coagulant factors. Low molecular weight heparin (LMWH) preparations inhibit only factor Xa, and APTT may be prolonged but the PT does not.

Contraindications

Heparin is contraindicated:

- In patients with current or last history of heparin-induced thrombocytopenia
- In patients with acute mechanical embolus
- In patients with active major bleeding, and conditions with a high risk of uncontrollable bleeding, including recent haemorrhagic stroke, major trauma, recent stroke, spinal cord or eye surgery, haemophilia and thrombocytopenia
- In patients with active gastric or duodenal ulcer

Adverse effects

- Bleeding
- Heparin-induced thrombocytopenia (immune-mediated effect that usually develops after 5–30 days; signs may include a 30% reduction in platelet count, thrombocytopenia, or skin allergy; HIT is suspected or confirmed; heparin should be discontinued and an alternative anticoagulant given)
- Hypocalcaemia (due to inhibition of extracellular secretions of patients with diabetes mellitus, chronic renal failure, arthritis, raised plasma potassium or those taking potassium sparing drugs seem to be more susceptible)
- Osteopenia (risk lower with LMWH)
- Hypersensitivity reactions
- Injection site reactions

Low molecular weight heparins vs unfractionated heparin

Unfractionated heparin is usually given by continuous intravenous infusion for the smoothest control and the treatment of choice when rapid reversal of anticoagulation may be required (i.e., surgical patients on low prophylaxis). Therapy is monitored by maintaining the APTT at 1.5-2.5 times the upper limit of normal.

Low molecular weight heparin (LMWH) preparations have largely replaced unfractionated heparins.

Advantages of LMWH

- Greater ability to inhibit factor Xa directly, interacting less with platelets and so have a lower tendency to cause bleeding
- Greater bioavailability and longer half-life in plasma making once daily subcutaneous administration possible
- More predictable dose response avoiding the need for routine anticoagulant monitoring
- Lower associated risk of heparin-induced thrombocytopenia or of osteoporosis

Hemorrhage

Because this is a short duration of action, if hemorrhage occurs it is usually sufficient to withdraw unfractionated or low molecular weight heparin, but if rapid reversal of the effects of the heparin is required, protamine sulfate is a specific antidote but only partially reverses the effects of low molecular weight heparin.
Pharmacology: Cardiovascular

Question 48 of 121

Which of the following drugs may reduce the anticoagulant effect of warfarin:

- A Alcohol
- B Phenytoin
- C NSAIDs
- D Metronidazole
- E Thyroxine

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Something wrong?
Pharmacology: Cardiovascular

Answer: Increased unopposed effect

Increased unopposed effect

Adverse effects

- Tachycardia
- Hypotension
- Arrhythmias
- Heart failure
- Peripheral edema
- Renal failure
- Hypertension

Caution

- Use with caution in patients with significant cardiovascular disease or those at risk of cardiovascular disease.

Dose

- Individualize based on patient response and hemodynamic parameters.
- Adjust dose as needed to achieve desired blood pressure control.

Monitoring

- Monitor blood pressure, heart rate, and renal function daily.
- Systemic blood pressure, heart rate, and renal function every 2-4 hours.
- Monitor for signs of hypotension or hypovolemia.

 titration

- Start at a lower dose and gradually increase as needed.
- Monitor for adverse effects and adjust dose accordingly.

Contraindications

- Patients with severe hypotension
- Patients with known allergy to drug

Precautions

- Use with caution in patients with cardiac conduction abnormalities
- Use with caution in patients with renal impairment
- Use with caution in patients with hepatic impairment
- Use with caution in patients with pulmonary hypertension

IV Administration

- Administer over 1-2 minutes
- Titrate as needed to achieve desired blood pressure control

UKA

- Use with caution in patients with hepatic impairment
- Use with caution in patients with renal impairment

US

- Use with caution in patients with hepatic impairment
- Use with caution in patients with renal impairment

Interactions

- Avoid concomitant use with drugs that can increase blood pressure
- Use with caution in patients taking ACE inhibitors or ARBs

Pharmacodynamics

- Increases NO availability
- Decreases peripheral resistance
- Decreases preload and afterload

Pharmacokinetics

- Rapid onset of action
- Peak effect within 1-2 minutes
- Duration of action 1-4 hours

Pharmacology

- Decreases blood pressure
- Decreases afterload
- Decreases preload
- Increases cardiac output

Pharmacotherapeutics

- Use to treat hypertension
- Use to treat chronic heart failure
- Use to treat pulmonary hypertension

Pharmacodynamics

- Decreases blood pressure
- Decreases afterload
- Decreases preload
- Increases cardiac output

Pharmacokinetics

- Rapid onset of action
- Peak effect within 1-2 minutes
- Duration of action 1-4 hours

Pharmacology

- Decreases blood pressure
- Decreases afterload
- Decreases preload
- Increases cardiac output

Pharmacotherapeutics

- Use to treat hypertension
- Use to treat chronic heart failure
- Use to treat pulmonary hypertension
Pharmacology: Cardiovascular

What is the first line alternative if adenosine is contraindicated in the management of a stable narrow-complex tachycardia:

- a Amiodarone
- b Verapamil
- c Digoxin
- d Magnesium sulfate
- e Bisoprolol
Pharmacology: Cardiovascular

**Question 7 of 11**

What is the first line alternative if adrenaline is contraindicated in the management of a stable narrow complex tachycardia?

- Adenosine
- Sotalol
- Disopyramide
- Magnesium Sulphate
- Bisoprolol

**Answer**

The first step in treatment of regular narrow complex tachycardia is to attempt vagal maneuvers (vagal stimulus message or Valsalva manoeuvre). If the tachycardia persists, adrenaline 0.5 μg/kg should be given as a rapid bolus using standard canula and a large syringe. If there is no response or no transient slowing or termination of the tachycardia/bradycardia to adrenaline 0.5 μg/kg, ibotenic acid or lidocaine should be given and if there is no response, one further 12 μg/kg of lidocaine (max 240 mg) should be given. If adrenaline is contraindicated or the patient remains in a regular narrow complex tachycardia, the administration of verapamil 12.5 – 5 mg every 2 minutes should be considered.

**Notes**

The approach to the management of tachycardia should follow the Resuscitation Council guidelines.

If the patient has adverse features:

- **Adverse features:**
  - Shock (Hypotension, pale, sweating, cold extremities, confusion, impaired consciousness)
  - Syncope (transient loss of consciousness)
  - Heart failure (pulmonary oedema, raised JVP, peripheral oedema, hepatomegaly)
  - Hypokalaemia (electrolyte imbalance, chest pain, altered mental state, changes in ECG)

If any adverse features are present, emergency cardioversion with a synchronized DC shock is indicated. If cardioversion fails to terminate the arrhythmia and adverse features persist, ibotenic acid 200 mg/kg over 10 – 20 mins should be given and further cardioversion attempted. The leading dose of adrenaline can be followed by an infusion of 0.5 mg/kg over 24 hours, given via a large bore.

If the patient has no adverse features:

- If the arrhythmia duration is < 12 seconds or greater. It is a broad complex tachycardia.
- If the QRS duration is < 120 ms or greater, it is a narrow complex tachycardia.

**Broad complex tachycardia**

Broad complex tachycardias are mainly ventricular in origin but may be supraventricular in nature with aberrant conduction.

A regular broad complex tachycardia is likely to be a ventricular tachycardia or a regular supraventricular rhythm with aberrant conduction.

- A ventricular tachycardia (or broad complex tachycardia of uncertain origin) should be treated with amiodarone 300 mg IV over 20 – 60 mins, followed by an infusion of 500 mg over the next 24 hours.
- If pre-excitation confirmed with the bundle branch block, the patient should be treated as for narrow complex tachycardia.
- A stable patient with an irregular broad complex tachycardia is most likely to be in AF with bundle branch block, although AF with ventricular pre-activation or polycyclic VF (bradycardia of point to point stability). Expert help should be sought for the assessment and treatment of irregular broad complex tachycardia.
- Termination of point to point VF should be attempted to stop all drugs known to prolong the QT interval, controlling electrolyte abnormalities, and giving magnesium sulphate 2 g IV over 10 minutes. Expert help should be sought as other treatment options including overdrive pacing may also be required to prevent relapse and the arrhythmia has been corrected.

**Narrow complex tachycardia**

Narrow complex tachycardias are supraventricular in origin.

- A narrow complex tachycardia may represent paroxysmal SVT or atrial flutter with a 1:1 conduction, but may be differentiated by differences between the two.

The first step in treatment of regular narrow complex tachycardia is to attempt vagal maneuvers (vagal stimulus message or Valsalva manoeuvre).

- If this fails, a high-dose beta-blockers (max 240 mg/kg over 5 minutes) should be given as a rapid bolus using a large canula and a large syringe. The patient should be warned that they will feel the rush and may experience chest discomfort for several seconds following the injection. An ECG (preferably multi-lead) should be recorded during the injection.
- If the ventricular rate does not transient and then reverts abruptly this may indicate atrial activity such as atrial flutter or a further atrial tachycardia, and this should be treated accordingly.
- If there is no response to these measures, disopyramide 1 mg/kg IV or lidocaine 1 mg/kg IV should be given and if there is no response, one further 12 mg/kg of lidocaine (max 240 mg) should be given over 10 minutes. Lack of response to adrenaline will occur if the tachycardia is due to atrial fibrillation and atrial flutter, and the arrhythmia/flutter has been corrected.
- Immediate treatment options include oral control with anti-epileptic drugs (phenytoin, carbamazepine). If the patient is refractory, rapid infusions of lidocaine may be required to lower the heart rate.

- **Adenosine** 240 mg IV bolus
- **Sotalol** 1 mg/kg IV bolus
- **Disopyramide** 1 mg/kg IV bolus
- **Magnesium Sulphate** 5 g over 5 minutes
- **Bisoprolol** 5 μg/kg IV bolus

- **Adenosine** 240 mg IV bolus
- **Sotalol** 1 mg/kg IV bolus
- **Disopyramide** 1 mg/kg IV bolus
- **Magnesium Sulphate** 5 g over 5 minutes
- **Bisoprolol** 5 μg/kg IV bolus

- **Adenosine** 240 mg IV bolus
- **Sotalol** 1 mg/kg IV bolus
- **Disopyramide** 1 mg/kg IV bolus
- **Magnesium Sulphate** 5 g over 5 minutes
- **Bisoprolol** 5 μg/kg IV bolus

- **Adenosine** 240 mg IV bolus
- **Sotalol** 1 mg/kg IV bolus
- **Disopyramide** 1 mg/kg IV bolus
- **Magnesium Sulphate** 5 g over 5 minutes
- **Bisoprolol** 5 μg/kg IV bolus

- **Adenosine** 240 mg IV bolus
- **Sotalol** 1 mg/kg IV bolus
- **Disopyramide** 1 mg/kg IV bolus
- **Magnesium Sulphate** 5 g over 5 minutes
- **Bisoprolol** 5 μg/kg IV bolus

- **Adenosine** 240 mg IV bolus
- **Sotalol** 1 mg/kg IV bolus
- **Disopyramide** 1 mg/kg IV bolus
- **Magnesium Sulphate** 5 g over 5 minutes
- **Bisoprolol** 5 μg/kg IV bolus
Pharmacology: Cardiovascular

Question 50 of 121

What is the mechanism of action of bendroflumethiazide:

a. Inhibition of Na+/K+/Cl cotransporter
b. Inhibition of Na+/Cl− cotransporter
c. Aldosterone antagonist
d. Carbonic anhydrase inhibitor
e. Inhibition of Na+/K+ ATPase pump

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Something wrong?
Drug: What is the mechanism of action of bendrofluazide?

- Inhibition of Na⁺/K⁺ ATPase
- Inhibition of Na⁺/Cl⁻ cotransporter
- Aldosterone antagonist
- Carbonic anhydrase inhibitor
- Inhibition of Na⁺/K⁺ ATPase pump

Answer:
This drug acts mainly on the early segments of distal tubule where it inhibits NaCl reabsorption by binding to the Na⁺/Cl⁻ cotransporter. Excretion of Cl⁻, Na⁺ and accompanying water is increased. The increased Na⁺ load in the distal tubule stimulates Na⁺ exchange with K⁺ and H⁺, increasing their excretion and causing hypokalaemia and a metabolic alkalosis. Excretion of Ca²⁺ is reduced.

Notes:
Thiazide diuretics are moderately potent diuretics, and are used to relieve oedema in chronic heart failure, and in the management of hypertension. They act within 1-2 hours of oral administration and most have a duration of action of 12 to 24 hours.

Mechanism of action:
This drug acts mainly on the early segments of distal tubule where it inhibits NaCl reabsorption by binding to the Na⁺/Cl⁻ cotransporter. Excretion of Cl⁻, Na⁺ and accompanying water is increased. The increased Na⁺ load in the distal tubule stimulates Na⁺ exchange with K⁺ and H⁺, increasing their excretion and causing hypokalaemia and a metabolic alkalosis. Excretion of Ca²⁺ is reduced.

Indications:
Bendrofluazide is used for oedema in mild or moderate heart failure. Combination diuretic therapy (thiazide and thiazide diuretic) may be effective in patients with resistant to treatment with one diuretic.

Thiazide diuretics are licensed for the treatment of hypertension but are no longer considered the first-line diuretic for this indication. They are omitted in the management of hypertension if a low dose of a thiazide produces a minimal or near-maximal blood pressure lowering effect, with very little biologically detectable diuresis. Higher doses cause more marked changes in plasma potassium, sodium, uric acid, glucose, and fluids, with little advantage in blood pressure control.

Contraindications:
Thiazide diuretics are contraindicated in:
- Addison’s disease
- Hypokalaemia
- Hypoaldosteronism
- Refractory hypokalaemia
- Symptomatic hyperkalaemia
- Severe hepatic impairment (may precipitate encephalopathy)

Caution:
Thiazide diuretics should rarely be used in the elderly because they are particularly susceptible to the side effects. The dose should then be adjusted according to renal function.

Common side effects of thiazide diuretics include:
- Drowsiness
- Posterocerebral hypertensive encephalopathy
- Acidosis and electrolyte imbalances
- Hypokalaemia, hypomagnesaemia, hyperuricaemia, hypercalcaemia, hypochloroaemic alkalosis
- Metabolic imbalances
- Hypokalaemia and gout
- Impaired glucose tolerance and hyperlipidaemia
- Altered plasma lipid concentrations
- Mitral and tricuspid valve abnormalities

Hypokalaemia:
Hypokalaemia can occur with both thiazide and loop diuretics. The risk of hypokalaemia depends on the duration of action as well as the potency and is thus greater with thiazides than with an equivalent dose of a loop diuretic. Hypokalaemia is dangerous in severe cardiovascular disease and in patients also being treated with cardiac glycosides. Often the use of potassium-sparing diuretics avoids the need to take potassium supplements.
Pharmacology: Cardiovascular

What is the mechanism of action of captopril:

- Inhibition of inactivated Na+ channels
- Angiotensin II receptor blocker
- Angiotensin-converting enzyme inhibitor
- Acetylcholinesterase inhibitor
- Alpha-blocker

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Pharmacology: Cardiovascular

Question 3 of 121

What is the mechanism of action of captopril?

a) Inhibition of inactivated Na⁺ channels
b) Angiotensin II receptor blocker
c) Angiotensin-converting enzyme inhibitor

d) Acetylcholinesterase inhibitor

e) Alpha-blocker

Answer

Captopril is an angiotensin-converting enzyme (ACE) inhibitor, which inhibits the conversion of angiotensin I to angiotensin II.

Notes

Mechanism of action

Angiotensin-converting enzyme inhibitors (ACE inhibitors) e.g. captopril inhibit the conversion of angiotensin I to angiotensin II, and thus have a vasodilatory effect, lowering both arterial and venous resistance. The cardiac output increases and, because the renovascular resistance falls, there is an increase in renal blood flow. This latter effect, together with reduced aldosterone release, increases Na⁺ and H₂O reabsorption, contracting the blood volume and reducing venous return to the heart.

Blocking ACE also diminishes the breakdown of the potent vasodilator bradykinin which is the cause of the persistent dry cough. Angiotensin II receptor blockers do not have this effect, therefore they are useful alternative for patients who have to discontinue an ACE inhibitor because of persistent cough.

Indications

ACE inhibitors have many uses and are generally well tolerated. They are indicated for:

- Heart failure
- Hypertension
- Diabetic nephropathy
- Secondary prevention of cardiovascular events

Contraindications

ACE inhibitors should be avoided in:

- History of angioedema associated with previous exposure to an ACE inhibitor
- Recurrent angioedema
- Bilateral renal artery stenosis
- Pregnancy and breastfeeding

Cautions

The use of ACE inhibitors is cautioned in:

- Renal impairment
- Afro-Caribbean patients (may respond less well to ACE inhibitors)
- Peri-operative vascular disease or generalised atherosclerosis (risk of clinically silent renovascular disease)
- Primary aldosteronism (patients may respond less well to ACE inhibitors)
- History of idiopathic or hereditary angioedema
- Patients with hypotensive cardiomyopathy
- Patients with severe or symptomatic acidosis (risk of hypokalemia)

Concomitant treatment with NSAIDs increases the risk of renal damage, and with potassium-sparing diuretics (or potassium-containing salt substitutes) increases the risk of hyperkalemia. Hyperkalemia and other side effects of ACE inhibitors are more common in the elderly and in those with impaired renal function and the dose may need to be reduced.

Adverse effects

Side effects of ACE inhibitors may include:

- Deterioration in renal function
- Hypokalemia
- Hypotension
- Persistent dry cough
- Angioedema (non-allergic drug reaction)
- Dizziness and headaches (usually secondary to hypotension)
- Other common adverse effects include abdominal discomfort, dyspepsia, diarrhea, nausea and vomiting, rash (in particular macular-papular rash), malaise, muscle spams, dyspnea, chest pain, and fatigue
Pharmacology: Cardiovascular

Which of the following is a common side effect of nifedipine:

- a. Ankle oedema
- b. Bradycardia
- c. AV conduction block
- d. Precipitation of heart failure
- e. Bronchospasm

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Pharmacology: Cardiovascular

Question: Of the following is a common side effect of nifedipine:

A) Arrhythmia
B) Bradycardia
C) AV conduction block
D) Propranolol on heart failure
E) Bronchodilation

Answer:

Nifedipine releases vascular smooth muscle and dilates coronary and peripheral arteries. Nifedipine has less myocardial effects than verapamil and has no antiarrhythmic properties but has more influence on the vessels. Unlike verapamil, it rarely precipitates heart failure because any negative inotropic effects are offset by a reduction in left ventricular work. Nifedipine commonly causes vasodilatatory adverse effects—flushing, dizziness, headache, postural hypotension, ankle swelling, which often improve with continued use, although ankle swelling often persists.

Notes:

Calcium channel blockers are widely used in the treatment of angina (second line to beta-blockers) and also for hypertension, heart failure, and arrhythmias. Calcium channel blockers vary widely in their predilection for the various possible sites of action and may be divided into the dihydropyridine type (e.g., amlodipine, nifedipine, and nitrendipine) and the rate-limiting non-dihydropyridine type (e.g., verapamil, diltiazem).

Mechanism of action:

Calcium channel blockers inhibit L-type voltage-sensitive calcium channels in smooth muscle, causing relaxation and vasodilation. They also block calcium channels within the myocardiurn and conducting tissues of the heart which produces an negative inotrope effect by reducing cardiac influx during the plateau phase of the action potential.

The dihydropyridines have relatively little effect on the heart because they have a much higher affinity for the blocked channels found more frequently in vascular muscle. Furthermore, at clinical dosages, vasodilation results in a reflex increase in sympathetic tone that counteracts the mild negative inotropic effect. The non-dihydropyridines are rate-limiting calcium channel blockers that depress the sinus node and slow conduction in the atrioventricular node, causing a mild rising bradycardia.

Contraindications:

Non-dihydropyridine CCBs:

- Atrial flutter or fibrillation
- Heart failure or history of heart failure (may precipitate or aggravate symptoms)
- Cardiac outflow obstruction e.g., significant aortic stenosis or obstructive hypertrophic cardiomyopathy (vasodilation may result in reduced cardiac output)
- Second or third degree AV block (may induce complete AV block)
- Severe bradycardia
- Sick sinus syndrome

Dihydropyridine CCBs:

- Uncontrolled heart failure
- Serious hypotension
- Cardiac outflow obstruction

Adverse effects:

Gastrointestinal adverse effects -- constipation, nausea, dyspepsia
- Bradycardia, AV block, reflex bradycardia, palpitations
- Vasodilator adverse effects -- flushing, dizziness, headache, postural hypotension, ankle swelling

Verapamil:

Verapamil is useful for the treatment of angina, hypertension, and arrhythmias. Verapamil is highly negatively inotropic and reduces cardiac output, slows the heart rate, and may impair atrioventricular conduction. It may precipitate heart failure, exacerbate conduction disorders, and cause hypotension at high doses and should not be used with beta-blockers. Contraindications to the most common side-effect of nifedipine.

Nifedipine:

Nifedipine releases vascular smooth muscle and dilates coronary and peripheral arteries. Nifedipine has less myocardial effects than verapamil and has no antiarrhythmic properties but has more influence on the vessels. Unlike verapamil, it rarely precipitates heart failure because any negative inotropic effects are offset by a reduction in left ventricular work.

Nadolol:

Nadolol is related to propranolol but the smooth muscle relaxant effects preferentially act on cerebral arteries. It is used solely for the prevention and treatment of vascular spasm following aneurysmal subarachnoid haemorrhage.
Pharmacology: Cardiovascular

Question 53 of 121

Which of the following drugs is used first line for a bradyarrhythmia with adverse features:

a. Adrenaline
b. Amiodarone
c. Adenosine
d. Atropine
e. Dopamine

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Something wrong?

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Pharmacology: Cardiovascular

Question 53 of 121

Which of the following drugs is used first line for a bradycardia with adverse features:

a) Adrenaline  
b) Amiodarone  
c) Adenosine  
d) **Atropine**  
e) Dopamine

Answer

If there are adverse features or a risk of asystole, atropine 500 mcg IV bolus should be given. If there is an unsatisfactory response, this can be repeated every 3 – 5 mins up to a maximum dose of 3 mg. Atropine should be used cautiously in the presence of acute myocardial ischaemia or myocardial infarction as the resulting increase in heart rate may worsen ischaemia or increase the size of the infarct.

Notes

The approach to the management of bradycardias should follow the Resuscitation Council guidelines.

If there are no adverse features (shock, syncope, myocardial ischaemia or heart failure) and no risk of asystole (recent asystole, Mobitz II AV block, complete heart block with broad QRS, ventricular pause > 3 seconds), immediate treatment can be delayed and the patient assessed to try and identify the cause of the bradycardia.

If there are adverse features or a risk of asystole, atropine 500 mcg IV bolus should be given. If there is an unsatisfactory response, this can be repeated every 3 – 5 mins up to a maximum dose of 3 mg. Atropine should be used cautiously in the presence of acute myocardial ischaemia or myocardial infarction as the resulting increase in heart rate may worsen ischaemia or increase the size of the infarct.

Other interim measures may include other drugs such as isoprenaline or adrenaline (or alternatively aminophylline, dopamine, glucagon [if beta-blocker or calcium channel blocker overdose] or glycopyrrolate). For a patient with bradycardia and adverse features, if there is no response to atropine, or if atropine is contraindicated, transcutaneous pacing should be initiated immediately. In the presence of life-threatening, extreme bradycardia, percussion pacing should be used as an interim measure until transcutaneous pacing is achieved.

Expert help should be sought and ultimately transvenous pacing arranged.
Pharmacology: Cardiovascular

Question 54 of 121

In adult advanced life support, which of the following best describes the correct administration of adrenaline for a shockable rhythm:

a. Give 1 mg of adrenaline as soon as intravenous access is achieved and every 3 – 5 minutes thereafter
b. Give 0.5 mg of adrenaline as soon as intravenous access is achieved and every 3 – 5 minutes thereafter
c. Give 1 mg of adrenaline after the third shock and every 3 – 5 minutes thereafter
d. Give 0.5 mg of adrenaline after the third shock and every 3 – 5 minutes thereafter
e. Give 0.5 mg of adrenaline after the first shock and every 3 – 5 minutes thereafter

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Pharmacology: Cardiovascular

Question 54 of 123

In adult advanced life support, which of the following best describes the correct administration of adrenaline for a shockable rhythm.

a) Give 1 mg of adrenaline as soon as intravenous access is achieved and every 3 – 5 minutes thereafter
b) Give 0.5 mg of adrenaline as soon as intravenous access is achieved and every 3 – 5 minutes thereafter
c) Give 1 mg of adrenaline after the third shock and every 3 – 5 minutes thereafter

Answer

IV adrenaline 1 mg (10 ml of 1:10,000 solution) should be given after 3 shocks and every 3 – 5 minutes after alternate shocks thereafter.

Notes

Basic life support

- For chest compressions in adult basic life support the heel of one hand should be placed over the middle of the lower half of the patient’s sternum with the other hand on top.
- The sternum should be depressed to a depth of 5 – 6 cm.
- Chest compressions should be performed at a rate of 100 – 120 per minute.
- Thirty compressions should be given before two breaths and that ratio continued (30:2).
- The person giving CPR should be changed every 1 minutes if possible to avoid exhaustion and poor quality chest compressions.
- Interruptions should be minimised (pauses should be < 5 seconds).

Advanced life support

- In adult advanced life support, basic life support should continue whilst the defibrillator pads are attached and the airway managed.
- The pads should be positioned one to the right of the upper sternum below the clavicle and the other in the left midaxillary line in the 5th intercostal space.
- Chest compressions should be paused briefly (< 5 seconds) to assess the rhythm and then continued as the defibrillator charges if a shockable rhythm is identified or continued for a further two whole minutes if a non-shockable rhythm is identified.
- When delivering a shock, everybody should be clear of the patient (and any GTN patches and oxygen sources removed).
- Once the shock has been delivered, compressions should resume immediately and continue for a further two minutes before checking the rhythm again or feeling for a pulse.

Shockable rhythm (ventricular fibrillation or pulseless ventricular tachycardia)

- IV adrenaline 1 mg (10 ml of 1:10,000 solution) should be given after 3 shocks and every 3 – 5 minutes after alternate shocks thereafter.
- IV amiodarone 300 mg should be given after 3 shocks. A further dose of IV amiodarone 150 mg may be considered after a total of five defibrillation attempts. Lidocaine (1 mg/kg ± 100 – 150 mg) may be used as an alternative if amiodarone is not available but may not be given if amiodarone has been given already.

Non-shockable rhythm (asystole or pulseless electrical activity)

- IV adrenaline 1 mg should be given as soon as IV access is achieved and then given every 3 – 5 minutes after alternate shocks thereafter.
Pharmacology: Cardiovascular

Question 55 of 121

In adult advanced life support, what is the correct dose of adrenaline for cardiac arrest:

a 0.5 mg IV adrenaline
b 100 mcg IV adrenaline
c 0.5 mL of 1:1000 solution of IV adrenaline
d 10 mL of 1:10000 solution of IV adrenaline
e 1 mL of 1:10000 solution of IV adrenaline
Pharmacology: Cardiovascular

Question 55 of 121

In adult advanced life support, what is the correct dose of adrenaline for cardiac arrest:

a) 0.5 mg IV adrenaline
b) 500 mcg IV adrenaline
c) 0.5 mL of 1:1000 solution of IV adrenaline

d) 10 mL of 1:10000 solution of IV adrenaline

e) 1 mL of 1:10000 solution of IV adrenaline

Answer

For a shockable rhythm, IV adrenaline 1 mg (10 mL of 1:1000 solution) should be given after 3 shocks and every 3 – 5 minutes after alternate shocks thereafter. For a non-shockable rhythm IV adrenaline 1 mg should be given as soon as IV access is achieved and then given every 3 – 5 minutes after alternate shocks thereafter.

Notes

Basic life support

- For chest compressions in adult basic life support the heel of one hand should be placed over the middle of the lower half of the patient’s sternum with the other hand on top.
- The sternum should be depressed to a depth of 5 – 6 cm.
- Chest compressions should be performed at a rate of 100 – 120 per minute.
- Thirty compressions should be given before two breaths and that ratio continued (30:2).
- The person giving CPR should be changed every 2 minutes if possible to avoid exhaustion and poor quality chest compressions.
- Interventions should be minimised (pauses should be < 5 seconds).

Advanced life support

- In adult advanced life support, basic life support should continue whilst the defibrillator pads are attached and the airway managed.
- The pads should be positioned one to the right of the upper sternum below the clavicle and the other in the left midaxillary line in the 5th intercostal space.
- Chest compressions should be paused briefly (< 5 sec) to assess the rhythm and then continued as the defibrillator charges if a shockable rhythm is identified or continued for a further two whole minutes if a non-shockable rhythm is identified.
- When delivering a shock, everybody should be clear of the patient (and any GTN patches and oxygen sources removed).
- Once the shock has been delivered, compressions should resume immediately and continue for a further two minutes before checking the rhythm again or feeling for a pulse.

Shockable rhythm (ventricular fibrillation or pulseless ventricular tachycardia)

- IV adrenaline 1 mg (10 mL of 1:1000 solution) should be given after 3 shocks and every 3 – 5 minutes after alternate shocks thereafter.
- IV amiodarone 300 mg should be given after 3 shocks. A further dose of IV amiodarone 150 mg may be considered after a total of five defibrillation attempts. Lidocaine (1 mg kg⁻¹ sup>1</sup>) may be used as an alternative if amiodarone is not available but may not be given if amiodarone has been given already.

Non-shockable rhythm (asystole or pulseless electrical activity)

- IV adrenaline 1 mg should be given as soon as IV access is achieved and then given every 3 – 5 minutes after alternate shocks thereafter.
Pharmacology: Cardiovascular

Question 56 of 121

Which of the following is NOT a typical side effect of captopril:

- a) Persistent dry cough
- b) Hypokalaemia
- c) Angioedema
- d) Worsening of renal function
- e) Headache
Pharmacology: Cardiovascular

Question 1 of 1

Which of the following is NOT a typical side effect of captopril?

- Persistent dry cough
- Hypersensitivity
- Angioedema
- Worsening of renal function
- Headache

Answer

Side effects of ACE inhibitors may include:

- Deterioration in renal function
- Hypokalemia (risk increased by concurrent use of potassium-sparing diuretics)
- Hypotension
- Persistent dry cough
- Angioedema (non-allergic drug reaction)
- Dizziness and headache (usually secondary to hypotension)

Other common adverse effects include abdominal discomfort, dyspepsia, diarrhea, nausea and vomiting, rash (in particular maculopapular rash), rashes, muscle spasm, dyspnoea, chest pain, and fatigue

Notes

Mechanism of action

Angiotensin-converting enzyme inhibitors (ACE inhibitors, e.g., captopril) inhibit the conversion of angiotensin I to angiotensin II, and thus have a vasodilatory effect, lowering both arterial and venous resistance. The cardiac output increases and, because the renin-angiotensin system falls, there is an increase in renal blood flow. This latter effect, together with reduced aldosterone release, induces Na+ and H2O retention, contracting the blood volume and reducing venous return to the heart.

Blockage ACE also diminishes the breakdown of the potent vasodilator bradykinin which is the cause of the persistent dry cough. Angiotensin II receptor blockers do not have this effect, therefore they are useful alternative for patients who have to discontinue an ACE inhibitor because of persistent cough.

Indications

ACE inhibitors have many uses and are generally well tolerated. They are indicated for:

- Heart failure
- Hypertension
- Diabetic nephropathy
- Secondary prevention of cardiovascular events

Contraindications

ACE inhibitors should be avoided in:

- History of angioedema associated with previous exposure to an ACE inhibitor
- Recurrent angioedema
- Bilateral renal artery stenosis
- Pregnancy and breastfeeding

Caution

The use of ACE inhibitors is cautioned in:

- Renal impairment
- Afro-Caribbean patients (may respond less well to ACE inhibitors)
- Peripheral vascular disease or generalised atherosclerosis (risk of clinically silent renal vascular disease)
- Primary aldosteronism (patients may respond less well to ACE inhibitors)
- History of idiopathic or hereditary angioedema
- Patients with symptomatic cardioembolic phenomena
- Patients with severe or symptomatic aortic stenosis (risk of hypotension)

Concurrent treatment with NSAIDs increases the risk of renal damage, and with potassium-sparing diuretics (or potassium-containing salt substitutes) increases the risk of hyperkalemia. Hypokalemia and other side effects of ACE inhibitors are more common in the elderly and in those with impaired renal function and the dose may need to be reduced.

Adverse effects

Side effects of ACE inhibitors may include:

- Deterioration in renal function
- Hypokalemia
- Hypotension
- Persistent dry cough
- Angioedema (non-allergic drug reaction)
- Dizziness and headache (usually secondary to hypotension)
- Other common adverse effects include abdominal discomfort, dyspepsia, diarrhea, nausea and vomiting, rash (in particular maculopapular rash), rashes, muscle spasm, dyspnoea, chest pain, and fatigue
Pharmacology: Cardiovascular

Question 57 of 121

What is the initial dose of adenosine recommended for management of a regular narrow-complex tachycardia:

a) 400 microgram
b) 0.5 mg
c) 1 mg
d) 2 mg
e) 6 mg

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Something wrong?
Pharmacology: Cardiovascular

Question 17 of 25

What is the initial dose of adenosine recommended for management of a regular narrow complex tachycardia?

a) 400 microgram
b) 60 mg
c) 1 mg
d) 2 mg
e) 4 mg

Answer

The first step in the treatment of regular narrow complex tachycardia is to attempt vagal manoeuvres (unilateral carotid massage or Valsalva manoeuvre). If the tachycardia is paroxysmal, adenosine 6 mg IV should be given as a rapid bolus using a large cannula and a large syringe. If there is no response (i.e. no transient slowing or termination of the tachycardia by adenosine 6 mg IV), a second bolus of 12 mg IV should be given if there is no response, one further 12 mg IV if there are still no responses (max 30 mg).

Notes

The approach to the management of arrhythmias should follow the Resuscitation Council guidelines.

If the patient has adverse features

Adverse Features:
- Shock (hypotension, pallor, sweating, cool extremities, confusional state, impaired consciousness)
- Sepsis (transient loss of consciousness)
- Heart failure (pulmonary oedema, rales, AF/peripheral oedema, hypotension)
- Hypoxic encephalopathy (cerebral ischaemic pain, ischaemic changes on EEG)

If any adverse feature is present, emergency cardioversion with a synchronized DC shock is indicated. If cardioversion fails to terminate the arrhythmia and adverse features persist, adenosine 300 mg IV over 20–30 minutes should be given before further cardioversion attempts. The loading dose of adenosine can be followed by an infusion of 60 mg IV over 15 minutes via a large vein.

If the patient has no adverse features

If the patient is stable, the QRSA duration should be considered.

- If the QRSA duration is 0.12 seconds or greater, it is a broad complex tachycardia.
- If the QRSA complex is less than 0.12 seconds, it is a narrow complex tachycardia.

Broad complex tachycardia

Broad complex tachycardias are mostly ventricular in origin but may be a supraventricular rhythm with aberrant conduction.
- A regular broad complex tachycardia is likely to be a ventricular tachycardia or a regular supraventricular rhythm with bundle branch block.
- A ventricular tachycardia for broad complex tachycardia of uncertain origin should be treated with synchronized DC shock: 360 J IV over 20–60 seconds, followed by an infusion of 300 mg IV over the next 6–12 hours.
- Fatality confirmed as SVT with bundle branch block, the patient should be treated as for narrow complex tachycardia.
- A stable patient with non-irregular broad complex tachycardia is most likely to be AF with bundle branch block, although AF with ventricular pre-excitation or polymorphic VT (bombeles de pointes) is possible.

Expert help should be sought for the assessment and treatment of irregular broad complex tachycardia.
- Terudol (or practolol, if sympatholytic may be required to prevent tachycardia once the arrhythmia has been corrected.

Narrow complex tachycardia

The narrow complex tachycardias are supraventricular in origin.
- A regular narrow complex tachycardia may represent preexcited SVT or atrial flutter with 2:1 conduction. It may be difficult to differentiate between the two.
- The first step in the treatment of regular narrow complex tachycardia is to attempt vagal manoeuvres (unilateral carotid massage or Valsalva manoeuvre).
- If the tachycardia persists, adenosine 6 mg IV should be given as a rapid bolus using a large cannula and a large syringe. The patient should be warned that they will feel lightheaded and may experience chest discomfort for a few seconds following the injection. An ECG (preferably multi-lead) should be recorded during the injection.
- If the ventricular rate drops briskly and then spares up again, this may indicate atrial activity such as atrial flutter or other atrial tachycardia, and this should be treated accordingly.
- If there is no response, or no transient slowing or termination of the tachycardia by adenosine 6 mg IV, a second bolus of 12 mg IV should be given and if there is no response, one further 12 mg IV bolus given (max 30 mg). Lack of response to adenosine will occur if the tachycardia is not supraventricular or it is ventricular in origin.
- Frusemide is contraindicated, or unlikely to terminate a regular narrow complex tachycardia, and the management of ventricular arrhythmia: 2.5 mg IV over 2 minutes should be considered.
- Irregular narrow complex tachycardia is likely to be AF with fast ventricular response or less commonly, atrial flutter with variable AV conduction.
- Immediate treatment options include rate control with beta blockers, rhythm control using drugs to achieve chemical cardioversion, rhythm control by synchronised cardioversion and treatment to prevent complications (e.g. anticoagulation). Expert help should be sought in determining the most appropriate treatment for the individual patient.
Pharmacology: Cardiovascular

Question 58 of 121

Adenosine is contraindicated in which of the following conditions:

- a. Diabetes mellitus
- b. Hypertension
- c. Peripheral vascular disease
- d. Asthma
- e. Myasthenia gravis

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Something wrong?
Pharmacology: Cardiovascular

Question 1 of 121

Adenosine is contraindicated in which of the following conditions:

a) Diabetes mellitus
b) Hypertension
c) Peripheral vascular disease
d) Asthma
  ✔
e) Myasthenia gravis

Answer

Adenosine is contraindicated in:

- Asthma and COPD (can cause bronchospasm)
- Decompensated heart failure
- Long QT syndrome
- Second- or third-degree AV block and sick sinus syndrome (unless pacemaker fitted)
- Severe hypotension

Notes

Adenosine is usually the treatment of choice for terminating paroxysmal supraventricular tachycardia including those associated with accessory conduction pathways e.g. Wolff-Parkinson-White syndrome.

Mechanism of action

Adenosine stimulates A1 adenosine receptors and opens acetylcholine-sensitive K+ channels, increasing K+ efflux. This hyperpolarizes the cell membrane in the atrioventricular node and, by inhibiting the calcium channels, slows conduction in the AVN. As it has a very short duration of action (half-life only about 8 - 10 seconds), most side-effects are short-lived.

Administration

For a regular narrow-complex tachycardia the first step is to attempt vagal manoeuvres. If this is unsuccessful and the tachyarrhythmia persists, 6 mg intravenous adenosine should be administered in a central/venous line over 2 seconds, followed by 12 mg after 1 - 2 minutes if required, then another 12 mg after 1 – 2 minutes if required (max 30 mg).

The effects of adenosine are potentiated by diprydramidol, therefore if it is essential to give adenosine in a patient taking diprydramidol the dose should be quartered.

The patient should be warned that they will feel unwell and may experience chest discomfort for a few seconds following the injection. An ECG (preferably multi-lead) should be recorded during the injection. If adenosine is contraindicated, or fails to terminate a regular narrow-complex tachycardia, the administration of verapamil 2.5 – 5 mg IV over 2 mins should be considered.

Contraindications

Adenosine is contraindicated in:

- Asthma and COPD (can cause bronchospasm)
- Decompensated heart failure
- Long QT syndrome
- Second- or third-degree AV block and sick sinus syndrome (unless pacemaker fitted)
- Severe hypotension

Adverse effects

Common side effects of adenosine include:

- Atraphrodisia
- Dizziness, flushing, headache, nausea, dyspnoea
- Angina (discontinue)
- AV block, sinus pause and arrhythmia (discontinue if asystole or severe bradycardia occur)
Pharmacology: Cardiovascular

Question 59 of 121

Bendoflumethiazide may cause all of the following electrolyte imbalances EXCEPT for:

- a) Hypomagnesaemia
- b) Hypocalcaemia
- c) Hypokalaemia
- d) Hypochloraemic alkalosis
- e) Hyponatraemia

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Pharmacology: Cardiovascular

Question 46 of 235

Bendroflumethiazide may cause all of the following electrolyte imbalances EXCEPT:

- Hypokalemia
- Hyperkalemia
- Hypocalcemia
- Hyperphosphatemia
- Hypocalciuria

Answer

Common side effects of thiazide diuretics include:

- Excessive diuresis
- Postural hypotension, dehydration, renal impairment
- Arch block and electrolyte imbalance
- Hypokalemia, hyperparathyroidism, hyperkalemia, hypercalcemia, hypocalcemia, hyperglycemia
- Metabolic alkalosis
- Hypovolaemia and hypotension
- Interruption of glucose tolerance and hyperglycemia
- Albuminuria and increased concentrations
- Nephrogenic interstitial nephritis

Notes

Thiazide diuretics are moderately potent diuretics, and are used to relieve edema in chronic heart failure, and in the management of hypertension. They act within 1 to 2 hours of oral administration and most have a duration of action of 12 to 24 hours.

Mechanism of action

Thiazides act mainly on the early segments of distal tubules where they inhibit Na+Cl− reabsorption by binding to the Na+Cl− cotransporter. Evidence of diuresis accompanying urinary volume is increased. The increased urinary loss of sodium lowers sodium reabsorption so the net effect is natriuresis. Increasing their excretion and causing hypokalemia and a metabolic alkalosis. Excretion of Ca2+ is reduced.

Indications

Bendroflumethiazide is used for evidence of mild or moderate heart failure; combination diuretic therapy (with loop and thiazide diuretics) may be effective in patients with evidence resistant to treatment with one diuretic.

Thiazides are licensed for the treatment of hypertension but are no longer considered the first line diuretic for this indication. In the management of hypertension, up to a dose of 31 mg of bendroflumethiazide produces a modest or near-normal blood pressure lowering effect, with very little biochemical or laboratory disturbance, higher doses cause more marked changes in body potassium, sodium, urea, bicarbonate, and electrolytes, with little advantage in blood pressure control.

Contraindications

Thiazide diuretics are contraindicated in:

- Addison’s disease
- Hyperkalemia
- Hyperuricaemia
- Refractory hypokalemia
- Symptomatic hypercalcaemia
- Severe hepatic impairment (may precipitate encephalopathy)

Cautions

Thiazide diuretics should be used with caution in:

- Diabetic mellitus (may exacerbate)
- Gout (may exacerbate)
- Severe renal insufficiency (may exacerbate)
- Hypocalciuria
- Malignant meningioma
- Neoplastic syndrome

Adverse effects

Lower initial doses of diuretics should be used in elderly because they are particularly susceptible to the side effects. The dose should then be adjusted according to renal function.

Common side effects of thiazide diuretics include:

- Excessive diuresis
- Postural hypotension, dehydration, renal impairment
- Arch block and electrolyte imbalance
- Hypokalemia, hyperparathyroidism, hyperkalemia, hypercalcemia, hypocalcemia, hyperglycemia
- Metabolic alkalosis
- Hypovolaemia and hypotension
- Interruption of glucose tolerance and hyperglycemia
- Albuminuria and increased concentrations
- Nephrogenic interstitial nephritis

Hypokalemia

Hypokalemia can occur with both thiazide and loop diuretics. The risk of hypokalemia depends on the duration of action as well as the potency and is thus greater with thiazides than with an equimolar dose of a loop diuretic. Hypokalemia is dangerous in severe cardiovascular disease and in patients also being treated with cardiac glycosides. The use of potassium sparing diuretics avoids the need to take potassium supplements.
Pharmacology: Cardiovascular

Question 60 of 121

Which of the following is the first line treatment for a stable regular narrow-complex tachycardia:

- a) Adenosine
- b) Synchronised DC cardioversion
- c) Vagal manoeuvres
- d) Amiodarone
- e) Beta-blocker

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Pharmacology: Cardiovascular

Question 1 of 15

Which of the following is the first line treatment for a stable regular narrow complex tachycardia?

a) Adenosine  
   b) Synchronized DC cardioversion  
   c) Verapamil  
   d) Amiodarone  
   e) Beta-blocker

Answer

The first step in treatment of regular narrow complex tachycardia is to attempt vagal manoeuvres (tolerated chair massage or Velcro massage). If the tachycardia persists, adenosine 6 mg IV should be given as a rapid bolus using a large cannula and a large vein. If there is no response (i.e. no transition or slowing of the tachycardia) then adenosine 6 mg IV plus 6 mg IV should be repeated. If there is no response, further 1.2 mg IV Velcro should be given if there is no response, one further 1.2 mg IV Velcro given (max 20 mg).

Notes

The approach to the management of tachycardias should follow the Resuscitation Council guidelines.

If the patient has adverse features

Adverse features:

- Shock (hypotension, pallor, sweating, cold extremities, confusion, impaired consciousness)
- Seizure (transient loss of consciousness)
- Heart failure (pulmonary oedema, rales, AFx peripheral stenosis, hepatomegaly)
- Myocardial ischaemia (echocardiographic signs, changes on ECG)

If any adverse features are present, emergency cardioversion with a synchronized DC shock is indicated. If cardioversion fails to terminate the arrhythmia and adverse features persist, adenosine 300 mg IV over 30–60 seconds should be given and further cardioversion attempted. The loading dose of adenosine can be followed by an infusion of 100 mg IV over 1 hour, given via a large vein.

If the patient has no adverse features

If the patient is stable, the QRS duration should be considered.

- If the QRS duration is 0.12 seconds or greater, it is a broad-complex tachycardia.
- If the QRS complex is less than 0.12 seconds, it is a narrow-complex tachycardia.

Broad-complex tachycardia

Broad-complex tachycardias are mostly ventricular in origin but may be a supraventricular rhythm with aberrant conduction.

- A regular broad-complex tachycardia is likely to be a ventricular tachycardia or a supraventricular rhythm with bundle branch block.
- A ventricular tachycardia is the most likely to arise, in the absence of bundle branch block, in the presence of an atrial fibrillation.

Narrow-complex tachycardia

The narrow-complex tachycardias are supraventricular in origin.

- A regular narrow-complex tachycardia may represent a supraventricular tachycardia or atrial flutter with 2:1 conduction. It may be difficult to differentiate between these two. The first step in treatment of regular narrow-complex tachycardia is to attempt vagal manoeuvres (tolerated chair massage or Velcro massage).
- If the tachycardia persists, adenosine 6 mg IV should be given as a rapid bolus using a large cannula and a large vein. The patient should be warned that this will feel unpleasant and may experience chest discomfort for a few seconds following the injection. An ECG (preferably multilead) should be recorded during the injection.
- If the ventricular rate slows markedly and then speeds up again, this may indicate atrial activity such as atrial flutter or other atrial tachycardia, and this should be treated accordingly.
- If there is no response, or if the ventricular slowing or termination of the tachycardia to adenosine 6 mg IV or 2.2 mg Velcro should be given if there is no response, one further 1.2 mg Velcro should be given (max 30 mg). Lack of response to adenosine will occur if the tachycardia is given too slowly or into a peripheral vein.
- If the narrow-complex tachycardia is not responding, it may be treated with a non-nitroglycerin, or p-blocker to regular a narrow-complex tachycardia, for example, the dopamine (2.5–5 mg IV over 10 minutes should be considered.
- The regular narrow-complex tachycardia is likely to be an A-V with fast ventricular response or less commonly, atrial flutter with variable A-V conduction.
- Immediate treatment options include rate control with beta-agonists, rhythm control using drugs designed to achieve chemical cardioversion, rhythm control by synchronized cardioversion and treatment to prevent complications (e.g., anticoagulation). Expert help should be sought in determining the most appropriate treatment for the individual patient.
Pharmacology: Cardiovascular

Question 61 of 121

A 70 year old man is brought to ED by ambulance with sudden onset chest pain, palpitations and shortness of breath. His HR is 160 bpm and BP 90/65. ECG demonstrates new-onset fast atrial fibrillation. Which of the following is the first-line treatment option in this case:

- (a) Beta-blocker
- (b) Diltiazem
- (c) Digoxin
- (d) Amiodarone
- (e) Synchronised DC cardioversion
Pharmacology: Cardiovascular

**Question 4 of 12**

A 70-year-old man is brought to ED by ambulance with sudden onset chest pain, palpitations and shortness of breath. His HR is 160 bpm and BP 90/65. ECG demonstrates new-onset fast atrial fibrillation. Which of the following is the first-line treatment option in this case?

- **A** Beta-blocker
- **B** Digoxin
- **C** Diltiazem
- **D** Amiodarone
- **E** Synchronized DC cardioversion

**Answer**

All patients with adverse features suggesting life-threatening haemodynamic instability (shock, syncope, heart failure, myocardial ischaemia) caused by new onset atrial fibrillation should undergo emergency electrical cardioversion with synchronized DC shock without delaying to achieve anticoagulation.

**Notes**

Treatment of patients with atrial fibrillation aims to reduce symptoms and prevent complications, especially stroke. Atrial fibrillation may be managed by either controlling the ventricular rate (rate control) or by attempting to restore and maintain sinus rhythm (rhythm control).

**New-onset atrial fibrillation**

All patients with adverse features suggesting life-threatening haemodynamic instability (shock, syncope, heart failure, myocardial ischaemia) caused by new onset atrial fibrillation should undergo emergency electrical cardioversion with synchronized DC shock without delaying to achieve anticoagulation.

In patients presenting acutely (< 48 h) with new-onset AF but without adverse features, immediate treatment options include rate control with drug therapy, rhythm control using drugs to achieve chemical cardioversion, rhythm control by synchronized cardioversion and treatment to prevent complications (e.g., anticoagulation).

- For rate control, the usual drug of choice is a beta-blocker. Diltiazem may be used in patients in whom beta-blockade is contraindicated or not tolerated. Digoxin may be used in patients with heart failure. Amiodarone may be used to avoid the risk of control is most useful in maintaining rhythm control.
- For rhythm control, chemical cardioversion may be appropriate. Class 1c antiarrhythmic drugs such as flecainide or propafenone may be used but are contraindicated in the presence of heart failure, left ventricular impairment, ischaemic heart disease or prolonged QT interval.
- Amiodarone (300 mg intravenously over 20 - 60 mins followed by 150 mg over 24 h) may be attempted to achieve chemical cardioversion but is less effective and takes longer to act.
- Electrical cardioversion remains an option in this setting and will restore sinus rhythm in more patients than chemical cardioversion.

The longer a person remains in AF, the greater the likelihood of atrial thrombus developing. In general, people who have been in AF for > 48 h should not be treated by cardioversion (electrical or chemical) until they have been fully anticoagulated for at least 3 weeks, or unless transoesophageal echocardiography has detected no evidence of atrial thrombus.

**Long-term management**

In general, rate control is the preferred first-line drug treatment strategy for atrial fibrillation in most patients except in patients with:

- New onset atrial fibrillation
- Heart failure secondary to atrial fibrillation
- Atrial flutter suitable for an ablation strategy
- Atrial fibrillation with a reversible cause
- or if rhythm control is more suitable based on clinical judgement.

Rate control is usually achieved with a beta-blocker or a rate limiting non-dihydropyridine calcium channel blocker e.g. verapamil or diltiazem.

Digoxin is usually only effective for controlling the ventricular rate at rest, and should therefore only be used as monotherapy in predominantly sedentary patients with non-paroxysmal atrial fibrillation or in combination therapy in resistant cases. Digoxin is also used when atrial fibrillation is accompanied by congestive heart failure.

If symptoms are not controlled with a combination of two drugs, a rhythm control strategy should be considered.

All patients with AF should be assessed and managed for risk of stroke and thromboembolism, and risk of bleeding if anticoagulation is being considered.
Pharmacology: Cardiovascular

Question 62 of 121

What is the main mechanism of action of unfractionated heparin:

- (a) Inhibits vitamin K dependent clotting factors
- (b) Inhibits factor Xa
- (c) Potentiate effects of antithrombin
- (d) Directly inhibits thrombin
- (e) Blocks GPIIb/IIIa receptor sites

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Something wrong?
Pharmacology: Cardiovascular

Question 6 of 121

What is the main mechanism of action of unfractionated heparin?

a) Inhibits vitamin K-dependent clotting factors
b) Inhibits factor Xa

c) Potentiates effects of antithrombins

d) Directly inhibits thrombin

e) Blocks GPIb/IIIa receptor sites

Answer

Heparin potentiates the activity of antithrombin III causing inactivation of thrombin. The heparin-antithrombin III complex also inhibits factor Xa and some other factors. Low molecular weight heparin (LMWH) preparations inhibit only factor Xa.

Notes

The main use of anticoagulants is to prevent thrombus formation or extension of an existing thrombus in the lower extremities versus the site of the circulations where the thrombus consists of a fibrin mesh enmeshed with platelets and red cells. Anticoagulants are of less use in preventing thrombus formation in arteries, for in faster-flowing vessels thrombus is composed mainly of platelets with little fibrin.

Mechanism of action

Heparin potentiates the activity of antithrombin III causing inactivation of thrombin. The heparin-antithrombin III complex also inhibits factor Xa and some other factors. Low molecular weight heparin (LMWH) preparations inhibit only factor Xa. PT and APPT may both be prolonged but the PT loss so.

Contraindications

Heparins are contraindicated:

- In people with current or history of heparin-induced thrombocytopenia
- In people with acute bacterial endocarditis
- In people with active major bleeding, and conditions with a high risk of uncontrolled bleeding, including recent haemorrhagic stroke, major trauma, recent brain, spinal cord or eye surgery, haemorrhage and thrombocytopenia
- In people with active gastric or duodenal ulceration

Adverse effects

- Bleeding
- Heparin-induced thrombocytopenia (shamor-meditated effect that usually develops after 5 – 10 days, signs may include a 20% reduction of platelet count, thrombosis, or skin allergies; if HIT is suspected or confirmed, heparin should be discontinued and an alternative anticoagulant given)
- Hyperkalaemia (due to inhibition of aldosterone secretion; patients with diabetes mellitus, chronic renal failure, acidosis, rapid plasma potassium in those taking potassium sparing drugs seen to be more susceptible)
- Osteoporosis (risk lower with LMWH)
- Arthritis
- Hypersensitivity reactions
- Injection site reactions

Low molecular weight heparin vs unfractionated heparin

Unfractionated heparin is usually given by continuous intravenous infusion for the smoothest control and the treatment of choice where rapid reversal of anticoagulation may be required (e.g. in surgical patients or life support). Therapy may be monitored by maintaining the APPT at 1.5 – 2.5 times the upper limits of normal.

Low molecular weight heparins (LMWH) preparations have largely replaced unfractionated heparin.

Advantages of LMWH

- Greater ability to inhibit factor Xa directly interacting loss with platelets and so may have a lesser tendency to cause bleeding
- Greater bioavailability and longer half-life in plasma making once daily or subcutaneous administration possible
- More predictable dose response avoiding the need for routine anticoagulant monitoring
- Lower associated risk of heparin-induced thrombocytopenia or of osteoporosis

Haemorrhage

Because it has a shorter duration of action, if haemorrhage occurs it is usually sufficient to withdraw unfractionated or low molecular weight heparins, but rapid reversal of the effects of the heparin is required, protamine sulphate is a specific antistable (but only partially reverses the effects of low molecular weight heparins).

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Resources

- The Royal College of Emergency Medicine
- Visit Association for Emergency Medicine
- Advanced Trauma Life Support
- Association for Emergency (UK)
- TraumaSavvy
- RCEMS
- Advanced Life Support Group
- Emergency Medicine Journal
- Ultrasound.co.uk
- Falci.co.uk

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Pharmacology: Cardiovascular

Question 63 of 121

Which of the following does NOT predispose to digoxin toxicity in a patient taking digoxin:

- a) Hypoxia
- b) Hypercalcaemia
- c) Hypokalaemia
- d) Hypomagnesaemia
- e) Hyponatraemia

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Pharmacology: Cardiovascular

Question 1: Which of the following does NOT predispose to dipryidamole toxicity in a patient taking dipryidamole?

- [ ] Hypoalbuminaemia
- [ ] Hypothyroidism
- [ ] Hyperuricaemia
- [ ] Hypertension
- [ ] Hypokalaemia

Answer: Hypertension, hyperuricaemia, hypothyroidism and hypoproteinanaemia predispose to dipryidamole toxicity. Although hypothyroidism can result in the development of atrioventricular block, it does not predispose to dipryidamole toxicity.

Notes: Dipros is a cardiac glycoside that increases the force of myocardial contraction (positive inotropy), and lowers the heart rate (negative chronotropic effect). Diprose has a narrow therapeutic index, hence diprose toxicity can occur even when the serum digoxin concentration is within the therapeutic range (0.5–2.0 ng/mL).

Mechanism of action

Inotropic effect

Dipros inhibits myocardial Na-K ATPase, which is responsible for Na+-K+ exchange across the myocardial cell membranes. This increase in intracellular Na and phosphate subsequently increases in intracellular Ca2+ that increases the force of myocardial contraction. The increase in intracellular Ca2+ increase the heart rate. Decreased the negative feedback across the over production of Ca2+ via the Na+-Ca2+ exchanger that normally occurs during diastole. Dipros and Na can compete for the receptors on the surface of the muscle cells, and so the effect of dipros may be dangerously increased in hypothyroidism.

Chronic effects

Dipros stimulates natriuretic activity, causing the release of ACE, which slows the heart rate, slows myocardial contractility, promotes the maintenance of the AVN and increases the blood pressure. By reducing the force of myocardial contraction, it increases the degree of block, which slows and strengthens the ventricular heart beat.

Indications

Dipros is useful for controlling cardiovascular function and promoting atrial fibrillation and atrial flutter. Dipros is used effectively for controlling the ventricular rate of severe AV block and for atrial fibrillation caused by arrhythmia. It can be used to control atrial fibrillation patients with non- paced atrial flutter. It can be used to treat severe hypertension, as an acute venous thrombosis, to prevent atrial fibrillation after surgery and to slow the heart rate of atrial fibrillation in the elderly.

Contraindications

Dipros is contraindicated in:

- Hypersensitivity to the medication
- Hypoalbuminaemia (risk of digoxin toxicity)
- Hyperthyroidism (risk of digoxin toxicity; diuretic therapy may predispose to hypothyroidism)
- Hypoproteinanaemia (risk of digoxin toxicity; diuretic therapy may predispose to hypothyroidism)
- Hypothyroidism (risk of digoxin toxicity)
- Renal insufficiency (risk of digoxin toxicity)
- Severe respiratory disease
- Skin disease
- Throid disease
- Cerebrovascular insufficiency
- Renal impairment (reduce dose and monitor plasma digoxin concentration, toxicity is transiently reversible after diuresis)

Side effects

- Cardiac adverse effects: Palpitations, arrhythmias
- Hypotension and arrhythmias
- Premature atrial contractions
- Myasthenia and QT interval depression
- Nausea, vomiting and anorexia
- Diarrhoea or diarrhea
- Neurological effects
- Cramps

Advantages of digoxin:

- Effective in the treatment of chronic cardiac failure
- Effective in the treatment of chronic heart disease
- Effective in the treatment of chronic heart disease
- Effective in the treatment of chronic heart disease
- Effective in the treatment of chronic heart disease
- Effective in the treatment of chronic heart disease

Dipros toxicity

Uncontrolled effects of dipros may lead to the clearance concentration of dipros (decreasing risk of toxicity through the range 5.5–3.0 ng/mL) and in the sensitivity of the conducting system on the myocardium, which can be affected by hypothyroidism. Hypothyroidism, hyperthyroidism, hypoproteinanaemia and hypothyroidism predispose to dipros toxicity. Core studies should also be in this clinical who are not sufficiently susceptible to dipros toxicity.

When chronic use of dipros should be withdrawn. Dipros specific anti-digoxin antibodies are indicated for the treatment of severe or acute digoxin toxicity. The treatment of digoxin is indicated for the treatment of severe or acute digoxin toxicity. The treatment of digoxin is indicated for the treatment of severe or acute digoxin toxicity. The treatment of digoxin is indicated for the treatment of severe or acute digoxin toxicity. The treatment of digoxin is indicated for the treatment of severe or acute digoxin toxicity.
Pharmacology: Cardiovascular

Question 64 of 121

Which of the following drugs can be used as reversal agent for warfarin:

- [ ] a Protamine sulfate
- [ ] b Hydroxocobalamin
- [ ] c Phytomenadione
- [ ] d Idarucizumab
- [ ] e Abciximab

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### Pharmacology: Cardiovascular

**Overview**

Prescribing cardiovascular drugs can be challenging. As always, use caution when administering these drugs to patients who are elderly, debilitated, or have renal or hepatic impairments. Consult a pharmacist if you have concerns about prescribing cardiovascular medications.

**Side Effects**

These medications may cause dizziness, drowsiness, flushing, and other side effects. It is critical to monitor patients closely and provide support and intervention as needed. General guidelines for monitoring medications include taking the patient’s blood pressure and heart rate, checking for signs of hypotension or tachycardia, and allowing for adequate rest and hydration.

**Mechanism of Action**

The effect of these drugs is due to their action on specific receptors in the cardiovascular system. This action leads to changes in blood pressure, heart rate, and other cardiovascular parameters.

**Indications**

The use of these drugs is determined by the specific indication for each drug and its effect on the cardiovascular system.

**Contraindications**

Contraindications for these drugs include pregnancy, lactation, and allergy to the drug or its components.

**Cautions**

Patients should undergo regular monitoring of their cardiovascular parameters, especially in cases of suspected drug interactions.

**Adverse Effects**

Common side effects include dizziness, drowsiness, and flushing. More severe effects include hypotension, tachycardia, and arrhythmias.

**Monitoring**

Monitor blood pressure and heart rate regularly as needed. Be prepared to adjust dosages as necessary.

### Pharmacology: Cardiovascular - Specific Medications

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**Notes**

The information provided is intended for educational purposes only and is not a complete guide to the use of these medications. Always consult a pharmacist or other healthcare professional before administering these drugs.
Pharmacology: Cardiovascular

Question 65 of 121

Nitrates are most commonly used in the management of:

a. Aortic stenosis
b. Angina
c. Raised intracranial pressure
d. Renovascular disease
e. Arrhythmias

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Pharmacology: Cardiovascular

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Nitrates are commonly used in the management of:

a) Aortic stenosis
b) Angina
r) Raised intraocular pressure
d) Renovascular disease
e) Arrhythmia

Answer

Nitrates are useful in the management of angina. Although they are potent coronary vasodilators, the main benefit derives from a reduction in venous return which in turn reduces left ventricular effort, decreasing oxygen demands and relieving anginal pain. Vasodilators can also act in heart failure by arteriolar dilatation which reduces both peripheral vascular resistance and left ventricular pressure during systole resulting in improved cardiac output. They can also cause venous dilatation which results in dilatation of capacitance vessels, increase of venous pooling, and diminution of venous return to the heart (decreasing left ventricular end-diastolic pressure).

Notes

Nitrates are useful in the management of angina. Although they are potent coronary vasodilators, the main benefit derives from a reduction in venous return which in turn reduces left ventricular effort, decreasing oxygen demands and relieving anginal pain. Vasodilators can also act in heart failure by arteriolar dilatation which reduces both peripheral vascular resistance and left ventricular pressure during systole resulting in improved cardiac output. They can also cause venous dilatation which results in dilatation of capacitance vessels, increase of venous pooling, and diminution of venous return to the heart (decreasing left ventricular end-diastolic pressure).

Mechanism of action

Initial metabolism of these drugs releases nitric oxide, which undergoes intracellular conversion to a nitric oxide/NH3/Nitrite cycle then activates guanylate cyclase, causing an increase in the intracellular concentration of cGMP in the vascular smooth muscle cells. cGMP activates protein kinase G, an enzyme that ultimately causes vascular smooth muscle relaxation.

True examples

Sublingual glyceryl trinitrate (GTN) is one of the most effective drugs for providing rapid relief of angina, although its effects only last for 20–30 minutes. It may also be administered as sublingual tablets or by sublingual administration using aerosol spray.

If sublingual glyceryl trinitrate is required more than twice a week, then combined therapy is required, where beta-blockers or calcium-channel blockers are taken in addition to nitrates which are reserved for acute attacks. If necessary, a long-acting nitrate is added.

Long-acting nitrates are more stable and may be effective for several hours, depending on the drug and the preparation (sublingual, oral, and modified release). Isosorbide dinitrate is widely used; duration of action of up to 12 hours is claimed for modified-release preparations. The use of isosorbide mononitrate, which is the main active metabolite of the dinitrate, avoids the variable absorption and unpredictable first-pass metabolism of the dinitrate.

Glycerol trinitrate or isosorbide dinitrate may be tried by intravenous injection when the sublingual form is ineffective in patients with chest pain due to myocardial infarction or severe ischaemia. Intravenous injections are also useful in the treatment of congestive heart failure.

Adverse effects

Side effects such as dizziness, flushing, tachycardia, throbbing headache, and postural hypotension may limit therapy, especially when angina is severe or when patients are unusually sensitive to the effects of nitrates. Prolonged high dosage may cause methemoglobinemia as a result of oxidation of haemoglobin.

Contraindications

Nitrates should not be used in people with:

- Acute myocardial infarction (MI) with low filling pressures, acute circulatory failure, (shock,
vascular collapse), or very low blood pressure
- Hypertrophic obstructive cardiomyopathy (HOCM), constriction pericarditis, cardiac
tamponade, low cardiac filling pressures, or acute aortic valve endocarditis
- Diseases associated with a raised intracranial pressure (for example following a head trauma, including cerebral haemorrhage)
- Severe anaemia
- Closed angle glaucoma
- Severe hypertensives, or hypertension

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- The Association for Emergency Medicine
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Pharmacology: Cardiovascular

What is the main mechanism of action of flecainide:

a. Blocks Ca2+ channels
b. Opens Na+ channels
c. Blocks Na+ channels
d. Opens K+ channels
e. Closes K+ channels
Pharmacology: Cardiovascular

Question 66 of 121

What is the main mechanism of action of flecainide:

- Blocks Ca²⁺ channels
- Opens Na⁺ channels
- Blocks Na⁺ channels
- Opens K⁺ channels
- Closes K⁺ channels

Answer

Flecainide inhibits the transmembrane influx of extracellular Na⁺ ions via fast channels on cardiac tissues, resulting in a decrease in rate of depolarisation of the action potential, prolonging the PR and QRS intervals. At high concentrations, it exerts inhibitory effects on slow Ca²⁺ channels, accompanied by moderate negative inotropic effect.

Notes

Flecainide acetate is an orally active class Ic antiarrhythmic and may be of value for serious symptomatic ventricular arrhythmias. It may also be indicated for junctional re-entry tachycardias and for paroxysmal atrial fibrillation. However, it has a negative inotropic action and can precipitate serious arrhythmias in a small minority of patients (including those with otherwise normal hearts).

Contraindications

Flecainide is contraindicated in:

- Abnormal left ventricular function
- Atrial conduction defects (unless pacing rescue available)
- Bundle branch block (unless pacing rescue available)
- Distal block (unless pacing rescue available)
- Haemodynamically significant valvular heart disease
- Heart failure
- History of myocardial infarction and either asymptomatic ventricular ectopics or asymptomatic non-sustained ventricular tachycardia
- Long-standing atrial fibrillation where conversion to sinus rhythm not attempted
- Second-degree or greater AV block (unless pacing rescue available)
- Sinus node dysfunction (unless pacing rescue available)

Cautions

Flecainide should be used with caution in:

- Atrial fibrillation following heart surgery
- Elderly (accumulation may occur)
- Patients with pacemakers (especially those who may be pacemaker dependent because stimulation threshold may rise appreciably)

Adverse effects

Common side effects of flecainide include:

- Aesthesiia
- Dizziness
- Dyspnoea
- Fatigue
- Fever
- Oedema
- Pro-arrhythmic effects
- Visual disturbances

Resources

- The Royal College of Emergency Medicine
- Irish Association for Emergency Medicine
- Advanced Trauma Life Support
- Resuscitation Council (UK)
- TeachMeAnatomy
- Trauma.org
- Radiopaedia

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Pharmacology: Cardiovascular

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The risk of renal impairment in a patient on ACE inhibitor therapy is increased by concomitant treatment with which of the following drug classes:

- Beta-blockers
- NSAIDs
- Statins
- Nitrates
- Calcium channel blockers

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The risk of renal impairment in a patient on ACE inhibitor therapy is increased by concomitant treatment with which of the following drug classes:

a) Beta-blockers
b) NSAIDs ✓
c) Statins
d) Nitrates
e) Calcium channel blockers

Answer
Concomitant treatment with NSAIDs increases the risk of renal damage, and with potassium-sparing diuretics (or potassium-containing salt substitutes) increases the risk of hyperkalaemia. Hyperkalaemia and other side effects of ACE inhibitors are more common in the elderly and in those with impaired renal function and the dose may need to be reduced.

Notes
Mechanism of action
Angiotensin-converting enzyme inhibitors (ACE inhibitors) e.g. captopril inhibit the conversion of angiotensin I to angiotensin II, and thus have a vasodilatory effect, lowering both arterial and venous resistance. The cardiac output increases and, because the renovascular resistance falls, there is an increase in renal blood flow. This latter effect, together with reduced aldosterone release, increases Na+ and K+ reabsorption, contracting the blood volume and reducing venous return to the heart.

Blocking ACE also diminishes the breakdown of the potent vasodilator bradykinin which is the cause of the persistent dry cough. Angiotensin II receptor blockers do not have this effect, therefore they are useful alternative for patients who have to discontinue an ACE inhibitor because of persistent cough.

Indications
ACE inhibitors have many uses and are generally well tolerated. They are indicated for:

- Heart failure
- Hypertension
- Diabetic nephropathy
- Secondary prevention of cardiovascular events

Contraindications
ACE inhibitors should be avoided in:

- History of angioedema associated with previous exposure to an ACE inhibitor
- Recurrent angioedema
- Bilateral renal artery stenosis
- Pregnancy and breastfeeding

Cautions
The use of ACE inhibitors is cautioned in:

- Renal impairment
- Afro-Caribbean patients (may respond less well to ACE inhibitors)
- Peripheral vascular disease or generalised arterio sclerosis (risk of clinically silent renovascular disease)
- Primary aldosteronism (patients may respond less well to ACE inhibitors)
- History of idiopathic or hereditary angioedema
- Patients with hypertrophic cardiomyopathy
- Patients with severe or symptomatic aortic stenosis (risk of hypertension)

Concomitant treatment with NSAIDs increases the risk of renal damage, and with potassium-sparing diuretics (or potassium-containing salt substitutes) increases the risk of hyperkalaemia. Hyperkalaemia and other side effects of ACE inhibitors are more common in the elderly and in those with impaired renal function and the dose may need to be reduced.

Adverse effects
Side effects of ACE inhibitors may include:

- Deterioration in renal function
- Hyperkalaemia
- Hypotension
- Persistent dry cough
- Angioedema (non-allergic drug reaction)
- Dizziness and headaches (usually secondary to hypotension)
- Other common adverse effects include abdominal discomfort, dyspepsia, diarrhoea, nausea and vomiting, rash (in particular maculopapular rash), myalgia, muscle spasm, dyspnoea, chest pain, and fatigue

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- Medical Web-Association for Emergency Medicine
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- Resuscitation Council (UK)
- Triage Guidelines
- Resuscitation
- Radiopaedia

- Advanced Life Support Group
- Emergency Medicine Journal
- 'Unshockable'
- Intra-Arterial
- Patient.uk

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Pharmacology: Cardiovascular

Question 68 of 121

Thiazide diuretics are contraindicated in which of the following:

- a. Asthma
- b. Recent myocardial infarction
- c. Addison's disease
- d. Acute intermittent porphyria
- e. Atrioventricular block

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Pharmacology: Cardiovascular

Thiazide diuretics are contraindicated in which of the following:

- A. Asthma
- B. Recent myocardial infarction
- C. Addison’s disease
- D. Acute intermittent porphyria
- E. Alcohol/cocaine drug

Answer

Thiazide diuretics are contraindicated in:

- Addison’s disease
- Hyperparathyroidism
- Hypersensitivity
- Refractory hypoaldosteronism
- Symptomatic hypercalcaemia
- Severe hepatic impairment (may precipitate encephalopathy)

Notes

Thiazides are moderately potent diuretics, and are used to reduce oedema in chronic heart failure, and in the management of hypertension. They are effective in 1 to 2 hours of oral administration and must have a duration of action of 12 to 24 hours.

Mechanism of action

Thiazides act mainly on the early segments of distal tubule where they inhibit NaCl reabsorption by binding to the Na-H exchanger. This increases the NaCl load in the distal tubule, stimulating NaCl reabsorption by Na-K ATPase and accompanying water is increased. The increased NaCl load in the distal tubule stimulates Na-K reabsorption by Na-K ATPase. Increasing their reabsorption and causing hypokalaemia and a metabolic alkalosis. Excretion of Cl- is reduced.

Indications

Bendroflumethiazide is used for oedema in mild to moderate heart failure. Combination diuretic therapy (with loop and thiazide diuretics) may be effective in patients with oedema resistant to treatment with one diuretic.

Thiazides are licensed for the treatment of hypertension but are no longer considered the first line diuretic for this indication. In the management of hypertension a low dose of a thiazide produces a modest fall in renal blood pressure lowering effect, with very little biochemical disturbance.

Higher doses cause more marked changes in plasma potassium, sodium, uric acid, glucose, and faecal, with little advantage in blood pressure control.

Contraindications

Thiazide diuretics are contraindicated in:

- Addison’s disease
- Hyperparathyroidism
- Hypersensitivity
- Refractory hypoaldosteronism
- Symptomatic hypercalcaemia
- Severe hepatic impairment (may precipitate encephalopathy)

Cautions

Thiazide diuretics should be used with caution in:

- Diabetes mellitus (may exacerbate)
- Gout (may exacerbate)
- Symptomatic hypokalaemia (may exacerbate)
- Hypocalcaemia (may exacerbate)
- Hyponatraemia
- Hypercholesterolaemia
- Acute anaemia

Adverse effects

Lower initial doses of diuretics should be used in the elderly because they are particularly susceptible to the side-effects. The dose should then be adjusted according to renal function.

Common side effects of thiazide diuretics include:

- Vomiting
- Postural hypotension, dehydration, renal impairment
- Acid-base and electrolyte imbalance
- Hypokalaemia, hypocalcaemia, hyperglycaemia, hyperlipidaemia, hypochloremia, abnormalities
- Metabolic imbalance
- Hypocalcaemia and gout
- Insulin-glucose tolerance and hypercalcaemia
- Blood pressure: low and high

Hypokalaemia

Hypokalaemia can occur with both thiazide and loop diuretics. The risk of hypokalaemia depends on the duration of action as well as the potency and as greater with thiazides than with an equivalent dose of loop diuretic. Hypokalaemia is dangerous in severe cardiovascular disease and in patients also being treated with cardiac glycosides. Often the use of potassium sparing diuretics avoids the need to take potassium supplements.
Pharmacology: Cardiovascular

Question 69 of 121

Which of the following is first-line treatment for a tachyarrhythmia associated with shock:

a) Amiodarone
b) Synchronised DC shock
c) Lidocaine
d) Adenosine
e) Intravenous magnesium

< Previous  Next >  See Answer

Something wrong?
Pharmacology: Cardiovascular

Which of the following is first-line treatment for a tachyarrhythmia associated with shock:

a) Amiodarone  
b) Synchronized DC shock  
c) Lidocaine  
d) Adenosine  
e) Intravenous magnesium

Answer

If any adverse features are present, emergency cardioversion with a synchronized DC shock is indicated. If cardioversion fails to terminate the arrhythmia and adverse features persist, amiodarone 300mg IV over 10-20 minutes should be given and further cardioversion attempted. The loading dose of amiodarone can be followed by an infusion of 900mg over 24 hours given via a large vein.

Notes

The approach to the management of tachycardias should follow the Resuscitation Council guidelines.

If the patient has adverse features:

Adverse Features:

Shock (presyncope, palpitations, chest pain, disorientation, impaired consciousness)
Syncope (transient loss of consciousness)
Heart failure (palpitations, cyanosis, raised JVP, peripheral oedema, hepatomegaly)
Myocardial ischaemia (chest pain, ischaemic changes on ECG)

If any adverse features are present, emergency cardioversion with a synchronized DC shock is indicated. If cardioversion fails to terminate the arrhythmia and adverse features persist, amiodarone 300mg IV over 10-20 minutes should be given and further cardioversion attempted. The loading dose of amiodarone can be followed by an infusion of 900mg over 24 hours given via a large vein.

If the patient has no adverse features:

If the patient is stable, the QRS duration should be considered.

If the QRS duration is 0.12 seconds or greater, it is a broad complex tachycardia.

If the QRS complex is less than 0.12 seconds, it is a narrow complex tachycardia.

Broad complex tachycardia

Broad complex tachycardia is usually ventricular in origin but may be supraventricular with aberrant conduction.

- A regular broad complex tachycardia is likely to be ventricular tachycardia or a regular supraventricular rhythm with bundle branch block.
- A narrow complex tachycardia for broad complex tachycardia of uncertain tachyarrhythmia should be treated with amiodarone 300mg IV over 20-60 minutes, followed by an infusion of 900mg over 24 hours.
- If previously confirmed as SVT with bundle branch block, the patient should be treated as for narrow complex tachycardia.

- A stable patient with an irregular broad complex tachycardia is most likely to be in AF with bundle branch block, although AF with ventricular pre-excitation or polymorphic VT (torsade de pointes) is a possibility.

- Expert help should be sought for the assessment and treatment of irregular broad complex tachycardia.

- Torsade de pointes VT should be treated by stopping all drugs known to prolong the QT interval, correcting electrolyte abnormalities, and giving magnesium sulphate 2g IV over 10 minutes. Expert help should be sought in other treatment options including overdrive pacing which may be required to reverse tachycardia since the arrhythmias has been corrected.

Narrow complex tachycardia

The narrow complex tachycardias are supraventricular in origin.

- A regular narrow complex tachycardia may represent paroxysmal SVT or atrial flutter with a 2:1 conduction, it may be difficult to differentiate between the two.
- The first step in treatment of irregular narrow complex tachycardia is to attempt vagal manoeuvres (sudden closure of larynx or Valsalva manoeuvre).
- If the tachycardia is sustained, adenosine 4mg IV should be given as a rapid bolus using a large calibre and a large vein. The patient should be warned that this will feel intense and may experience chest discomfort for a few seconds following the injection. An AECG (preference multi lead) should be recorded during the injection.
- If the ventricular rate slows transiently and then speeds up again, this may indicate atrial activity such as atrial flutter or other atrial tachycardia, and this should be treated accordingly.
- If there is no response, or no transient slowing or termination of the tachycardia despite to adenosine 4mg IV, a 12 lead ECG should be obtained and if there is no response, one further 12-lead IV ECG before giving (re-test 30mg). Lack of response to adenosine will occur if the tachycardia is given too slowly or in a peripheral vein.
- If adenosine is not contraindicated, or if the tachycardia is treated with a regular narrow complex tachycardia, the administration of verapamil 25-50mg IV over 2-5 minutes should be considered.
- Irregular narrow complex tachycardia should be treated by AF with fast ventricular response or, less commonly, atrial flutter with variable AV conduction.
- Immediate treatment options include rate control by beta-blockers, rhythm control using drugs to achieve cardiac rhythms, rhythm control by synchronized cardioversion and treatment to prevent complications (e.g., anticoagulation). Expert help should be sought in determining the most appropriate treatment for the individual patient.
Pharmacology: Cardiovascular

Intravenous lidocaine may be indicated for which of the following:

- Refractory asystole in cardiac arrest
- Refractory ventricular fibrillation in cardiac arrest
- Terminating paroxysmal supraventricular tachycardia
- Chemical cardioversion of atrial fibrillation
- Bradyarrhythmias associated with adverse features

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Pharmacology: Cardiovascular

Question 70 of 121

Intravenous lidocaine may be indicated for which of the following:

a) Refractory asystole in cardiac arrest
b) Refractory ventricular fibrillation in cardiac arrest ✔️

c) Terminating paroxysmal supraventricular tachycardia

d) Chemical cardioversion of atrial fibrillation

e) Bradyarrhythmias associated with adverse features

Answer

Intravenous lidocaine hydrochloride can be used for the treatment of ventricular tachycardia in haemodynamically stable patients, and ventricular fibrillation and pulseless ventricular tachycardia in cardiac arrest refractory to defibrillation, however it is a second-line choice (behind amiodarone).

Notes

Intravenous lidocaine hydrochloride can be used for the treatment of ventricular tachycardia in haemodynamically stable patients, and ventricular fibrillation and pulseless ventricular tachycardia in cardiac arrest refractory to defibrillation, however it is a second-line choice (behind amiodarone).

Mechanism of action

Lidocaine is a class IB agent which blocks inactivated voltage-dependent Na+ channels, making it highly selective for damaged tissues. In normal cardiac tissues, lidocaine has little effect because it dissociates rapidly from the Na+ channels which therefore recover during diaastole. However, in ischaemic areas, where anoxia causes depolarisation and arrhythmogenic activity, many Na+ channels are inactivated and therefore susceptible to lidocaine.

Contraindications

Intravenous lidocaine is contraindicated in:

- All grades of atrioventricular block
- Severe myocardial depression
- Sinusoidal disorders

Cautions

Intravenous lidocaine should be used with caution in:

- Acute porphyria (consider infusion with glucose for its anti-porphyrinogenic effects)
- Congestive cardiac failure (consider lower dose)
- Post cardiac surgery (consider lower dose)

Adverse effects

Common side effects of intravenous lidocaine include:

- Bradycardia and hypotension (may lead to cardiac arrest)
- Dizziness, drowsiness, paraesthesia, confusion (particularly if injection too rapid)
- Convulsions
- Respiratory depression

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Advanced Life Support Group
- Emergency Medicine Journal
- UltrasoundSite
- Master Anatomy
- Patient.uk
Pharmacology: Cardiovascular

Question 71 of 121

What is the mechanism of action of digoxin as a positive inotrope:

- a) Stimulation of Na+/Ca2+ exchanger
- b) Inhibition of Na+/K+ ATPase pump
- c) Inhibition of Na+/Ca2+ exchanger
- d) Beta-adrenoeceptor agonist
- e) Stimulation of Ca2+ ATPase

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Question 7 of 125

What is the mechanism of action of digoxin as a positive inotrope?

A) Simulation of Na+/Ca2+ exchanger
B) Inhibition of Na+/K+ ATPase
C) Inhibition of Na+/Ca2+ exchanger
D) Beta-agonism
E) Simulation of Ca2+ ATPase

Answer

Digoxin directly inhibits activity of Na+/K+ ATPase, which is responsible for Na+K+ exchange across the myocyte cell membrane. This inhibition improves the production and secondary accumulation in Intact Ca2+2+ to increase the force of myocardial contraction. The increase in Intact Ca2+ increases the force of myocardial contraction as the membrane reduces the extrusion of Ca2+ by the Na+/Ca2+ exchanger that normally occurs during diastole. Digoxin and K+ ions compete for the receptor on the outside of the myocyte cell membrane, and the effect of digoxin may become dangerously increased in hypokalemia.

Notes

Digoxin is a cardiac glycoside that increases the force of myocardial contraction (positive inotrope) and slows the heart rate (negative chronotropic). Digoxin has a narrow therapeutic index (lack of therapeutic window) and the toxic effects of digoxin can occur much sooner than the therapeutic concentration is within the therapeutic range (between 0.5-2.0 ng/ml).

Mechanism of action

Inotropic effect

Digoxin directly inhibits activity of Na+/K+ ATPase, which is responsible for Na+K+ exchange across the myocyte cell membrane. This inhibition increases Intact Ca2+2+ production and a secondary accumulation in Intact Ca2+2+ increases the force of myocardial contraction as the membrane reduces the extrusion of Ca2+ by the Na+/Ca2+ exchanger that normally occurs during diastole. Digoxin and K+ ions compete for the receptor on the outside of the myocyte cell membrane, and the effect of digoxin may become dangerously increased in hypokalemia.

Clearance of effects

Digoxin stimulates vagal activity, causing the release of ACh, which slows the heart rate, slows atrioventricular conduction and prolongs the refractory period of AV conduction at the AV node. Blocking AV conduction, digoxin increases the degree of block, and slows and lengthens the ventricular heart rate.

Indications

Digoxin is useful for controlling the ventricular response in atrial fibrillation atrioventricular nodal reentry tachycardia (AVNRT), atrial flutter, atrial fibrillation and for defibrillation or converting atrial flutter to atrial fibrillation. It should therefore only be used as a respite in patients with paroxysmal atrial fibrillation with non- paroxysmal atrial fibrillation. It is also useful in the control of heart failure, even in patients with intravenous administration, response may take 2-3 hours.

Contraindications

Digoxin should be avoided in cases of:

- Hyperkalaemia (risk of digitalis toxicity)
- Hyperthyroidism (risk of digitalis toxicity, as digoxin is excreted into urine metabolically).
- Hypothyroidism (risk of digitalis toxicity)
- Renal or hepatic failure
- Severe respiratory disease
- Sick sinus syndrome
- Thyroid disease
- Congestive heart failure
- Partial or total heart valve disease and replete plasma digoxin concentrations should be increased by electrolyte imbalance.
- Atrioventricular block
- Contraindicated drug therapy with drugs which may increase plasma concentrations of digoxin eg, anticoagulants, antibiotics, sulphonamides, lithium, amiodarone, non steroids.

Adverse effects

The adverse effects of digoxin are usually more common than normal therapeutic levels:

- Cardiac adverse effects
  - Structural and atrioventricular block
  - Pronounced ventricular contraction
- Nausea and vomiting
- Abdominal pain
- Rash and pruritus
- Headache
- Dizziness, tinnitus, drowsiness, flushing, pruritus, headache, depression, psychosis
- Torsades de pointes and arrhythmias
- Quincke-like syndrome in patients with pre-existing renal dysfunction

Clinical toxicity

Unintended effects of digoxin depend on the plasma concentration of digoxin increasing levels of toxicity through the levels of (0.5-0.75 ng/ml). These are the toxic effects of the resulting concentration of toxic metabolite, which is often increased in heart disease, hyperkalaemia, hypothyroidism and other conditions that increase digitalis toxicity. The serum levels in the elderly are particularly susceptible to digoxin toxicity.

In toxic cases, digoxin should be withheld. Digoxin-specific antibody fragments are indicated for the treatment of digoxin toxicity. Methods of treating digoxin toxicity include with select antidotes or thioridazine or other antidotal agents to stop the effects and remove excess drug. The use of intravenous antidotes is required, and the use of intravenous electrolyte administration is increased.

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Question 72 of 121

The dose of adenosine must be quartered if given concomitantly in a patient taking which of the following drugs:

- (a) Verapamil
- (b) Dipyridamole
- (c) Thyroxine
- (d) Clopidogrel
- (e) Propranolol

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Pharmacology: Cardiovascular

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The dose of adenosine must be administered if given concomitantly in a patient taking which of the following drugs:

- [ ] a) Verapamil
- [x] b) Dipryidamole
- [ ] c) Thyroxine
- [ ] d) Clopidogrel
- [ ] e) Propranolol

Answer

The effects of adenosine are potentiated by dipryidamole, therefore if it is essential to give adenosine in a patient taking dipryidamole the dose should be quartered.

Notes

Adenosine is usually the treatment of choice for terminating paroxysmal supraventricular tachycardia including those associated with accessory conduction pathways e.g. Wolff-Parkinson-White syndrome.

Mechanism of action

Adenosine stimulates A1-adenosine receptors and opens acetylcholine sensitive K+ channels, increasing K+ efflux. This hyperpolarizes the cell membrane in the atrioventricular node and, by inhibiting the calcium channels, slows conduction in the AVN. As it has a very short duration of action (half-life only about 8 – 10 seconds), most side effects are short lived.

Administration

For a regular narrow-complex tachycardia the first step is to attempt vagal manoeuvres. If this is unsuccessful and the tachyarrhythmia persists, 6 mg intravenous adenosine should be administered into a central/large vein over 2 seconds, followed by 12 mg after 1 – 2 minutes if required, then a further 12 mg after 1 – 2 minutes if required (max 30 mg).

The effects of adenosine are potentiated by dipryidamole, therefore if it is essential to give adenosine in a patient taking dipryidamole the dose should be quartered.

The patient should be warned that they will feel unwell and may experience chest discomfort for a few seconds following the injection. An ECG (preferably multi-lead) should be recorded during the injection. If adenosine is contraindicated, or fails to terminate a regular narrow-complex tachycardia, the administration of verapamil 2.5 – 5 mg IV over 2 mins should be considered.

Contraindications

Adenosine is contraindicated in:

- Asthma and COPD (can cause bronchospasms)
- Decompensated heart failure
- Long QT syndrome
- Second- or third-degree AV block and sick sinus syndrome (unless pacemaker fitted)
- Severe hypotension

Adverse effects

Common side effects of adenosine include:

- apprehension
- Dizziness, flushing, headache, nausea, dyspnoea
- Angina (discontinual)
- AV block, sinus pause and arrhythmia (discontinue if asystole or severe bradycardia occur)
Pharmacology: Cardiovascular

Question 73 of 121

A 67 year old man, with a history of hypertension for which he takes ramipril, presents to ED complaining of palpitations and shortness of breath ongoing for the past 3 days. His HR is 140 bpm and his ECG demonstrates atrial fibrillation. Which of the following is the most appropriate first-line treatment option:

a. Rate control with digoxin
b. Rate control with a beta-blocker
c. Synchronised DC cardioversion
d. Chemical cardioversion with amiodarone
e. Chemical cardioversion with flecaainide
Pharmacology: Cardiovascular
Question 73 of 825

A 67 year old man, with a history of hypertension for which he takes ramipril, presents to ED complaining of palpitations and shortness of breath ongoing for the past 3 days. His HR is 140 bpm and his ECCD demonstrates atrial fibrillation. Which of the following is the most appropriate first-line treatment option?

- Rate control with digoxin
- Rate control with a beta-blocker
- Synchronized DC cardioversion
- Chemical cardioversion with amiodarone
- Electrical cardioversion with flecainide

Answer

The longer a person remains in AF, the greater is the likelihood of atrial thrombus developing. In general, people who have been in AF for > 48 h should not be treated by cardioversion (electrical or chemical) until they have been fully anticoagulated for at least 3 weeks, or unless transesophageal echocardiography has detected no evidence of atrial thrombus. For rate control, the usual drug of choice is a beta-blocker. Digoxin may be used in patients in whom beta-blockade is contraindicated or not tolerated. Digoxin may be used in patients with heart failure. Amiodarone may be used to assist with rate control but is most useful in maintaining rhythm control.

Notes

Treatment of patients with atrial fibrillation aims to reduce symptoms and prevent complications, especially stroke. Atrial fibrillation may be managed by either controlling the ventricular rate (rate control) or by attempting to restore and maintain sinus rhythm (rhythm control).

New-onset atrial fibrillation

All patients with adverse features suggesting life-threatening (haemodynamic) instability (shock, syncope, heart failure, mesoccardial ischaemia caused by new onset atrial fibrillation) should undergo emergency electrical cardioversion with synchronized DC shock without delaying to achieve anticoagulation.

In patients presenting acutely (< 48 h) with new-onset AF but without adverse features, immediate treatment options include rate control with drug therapy, rhythm control using drugs to achieve chemical cardioversion, rhythm control by synchronized cardioversion and treatment to prevent complications (e.g. anticoagulation).

- For rate control, the usual drug of choice is a beta-blocker. Digoxin may be used in patients in whom beta-blockade is contraindicated or not tolerated. Digoxin may be used in patients with heart failure. Amiodarone may be used to assist with rate control but is most useful in maintaining rhythm control.
- For rhythm control, chemical cardioversion may be appropriate. Class 1c antiarrhythmic drugs such as flecainide or propafenone may be used but are contraindicated in the presence of heart failure, left ventricular impairment, ischaemic heart disease or prolonged QRS interval.
- Amiodarone (100 mg intravenously over 20–60 mins followed by 100 mg over 24 h) may be used to attempt chemical cardioversion but is less often effective and takes longer to act.
- Electrical cardioversion remains an option in this setting and will restore sinus rhythm in more patients than chemical cardioversion.

Long-term management

In general, rate control is the preferred first-line drug treatment strategy for atrial fibrillation in most patients except in patients with

- new-onset atrial fibrillation
- heart failure/secondary to atrial fibrillation
- atrial flutter suitable for an ablation strategy
- atrial fibrillation with a reversible cause
- or if rhythm control is more suitable based on clinical judgement.

Rate control may be achieved with a beta-blocker or a rate-limiting non-dihydropyridine calcium channel blocker e.g. verapamil or diltiazem.

Digoxin is usually only effective for controlling the ventricular rate at rest, and should therefore only be used as monotherapy in predominantly sedentary patients with non-paroxysmal atrial fibrillation or in combination therapy in resistant cases. Digoxin is also used when atrial fibrillation is accompanied by composite heart failure.

If symptoms are not controlled with a combination of two drugs, a rhythm control strategy should be considered.

All options with AF should be assessed and managed for risk of stroke and thromboembolism, and risk of bleeding if anticoagulation is being considered.
Pharmacology: Cardiovascular

Question 74 of 121

Which of the following is NOT a common side effect of amiodarone:

- a) Pulmonary fibrosis
- b) Peripheral neuropathy
- c) Blue/green teeth discoloration
- d) Slate grey skin discoloration
- e) Corneal microdeposits

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Pharmacology: Cardiovascular

Question 4 of 21

Which of the following is NOT a common side effect of amiodarone:

a) Pulmonary fibrosis
b) Peripheral neuropathy
b) Blue-green tooth discoloration
b) Slate-grey skin discoloration
b) Corneal microdeposits

Answer

Common side effects of amiodarone include:

- Brady cardia
- Nausea and vomiting
- Thyroid disorders - hyperthyroidism and hypothyroidism
- Persistent state grey skin discoloration
- Photosensitivity
- Pulmonary toxicity (including pneumonitis and fibrosis)
- Hepatotoxicity
- Corneal microdeposits (sometimes with night glare)
- Peripheral neuropathy
- Sleep disorders

Notes

Amiodarone hydrochloride is used in the treatment of arrhythmias, particularly when other drugs are ineffective or contraindicated. However, its long-term use is often restricted by various adverse effects such as photosensitivity, thyroid disorders, corneal microdeposits, neuropathy and pulmonary abnormalities.

Mechanism of action

Amiodarone has blocking actions on several channels (e.g. K+ and Na+ channels) and beta-adrenoceptors. It acts by slowing repolarisation and prolonging the action potential and refractory period in all cardiac tissues, depressing sinus node automaticity and slowing conduction.

Indications

Amiodarone can be used for paroxysmal supraventricular, nodal and ventricular tachycardias, atrial fibrillation and flutter, and ventricular fibrillation. It can also be used for tachyarrhythmias associated with Wolff-Parkinson-White syndrome.

Intravenous injection of amiodarone hydrochloride can be used in cardiorespiratory resuscitation for ventricular fibrillation or pulseless ventricular tachycardia unresponsive to other interventions.

Contraindications

Amiodarone is contraindicated in:

- Severe conduction disturbances (unless pacemaker fitted)
- Severe renal disease (unless pacemaker fitted)
- Iodine sensitivity
- Severe heart block (except in cardiac arrest)
- Severe bradycardia (except in cardiac arrest)
- Thyroid dysfunction

Intravenous use should be avoided in cardiogenic shock, congestive heart failure, circulatory collapse, severe arterial hypertension and severe respiratory failure.

Cautions

Amiodarone should be used with caution in:

- Acute peripneumonia
- Conduction disturbances (in excessive dosage)
- Elderly
- Heart failure
- Hypothyroidism
- Severe broadcardia (in excessive dosage)
- Severe hepatic cellular toxicity
- Concomitant therapy with drugs that prolong the QT interval

Adverse effects

Common side effects of amiodarone include:

- Brady cardia
- Nausea and vomiting
- Thyroid disorders - hyperthyroidism and hypothyroidism
- Persistent state grey skin discoloration
- Photosensitivity
- Pulmonary toxicity (including pneumonitis and fibrosis)
- Hepatotoxicity
- Corneal microdeposits (sometimes with night glare)
- Peripheral neuropathy
- Sleep disorders
Pharmacology: Cardiovascular

Question 75 of 121

What is the mechanism of action of furosemide:

a. Inhibition of Na+/Cl- cotransporter
b. Inhibition of Na+/K+ ATPase pump
c. Aldosterone antagonist
d. Inhibition of Na+/K+/2Cl cotransporter
e. Osmotic diuretic

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Pharmacology: Cardiovascular

- Inhibition of Na+/K+-ATPase
- Aliskiren / ARB
- Enalapril / ACE

Answer

Loop diuretics inhibit the Na+/K+/Cl- co-transporter on the luminal membrane in the thick ascending limb of the loop of Henle, thus preventing reabsorption of NaCl and water. These agents reduce reabsorption of Cl- and Na+, increasing GFR secretion and loss of K+ and HCO3-.

Notes

Indications

Loop diuretics are powerful diuretics used in acute pulmonary edema due to left ventricular failure; intravenous administration produces relief of breathlessness and reduces prowess sooner than would be expected from the time of onset of diuresis.

They are also used in oedema in patients with chronic heart failure; diuretic-resistant oedema can be treated with a loop diuretic combined with a thiazide or related diuretic.

If necessary, a loop diuretic can be added to antihypertensive treatment to achieve better control of blood pressure in those with resistant hypertension, or in patients with impaired renal function or heart failure.

Mechanism of action

Loop diuretics inhibit the Na+/K+/Cl- co-transporter on the luminal membrane in the thick ascending limb of the loop of Henle, thus preventing reabsorption of NaCl and water. These agents reduce reabsorption of Cl- and Na+, increasing GFR secretion and loss of K+ and HCO3-.

Furosemide and bumetanide are similar in activity, both act within 15 minutes of oral administration and diuretic effects are complete within hours, but in the first instance, they can be given twice a day without interfering with sleep. Following intravenous administration furosemide has a peak effect within 30 minutes. The diuretic effects associated with these drugs is dose-related.

Contraindications

Loop diuretics are contraindicated in:

- Hypokalaemia and dehydration
- Severe hypokalaemia or severe hypomagnesaemia
- Acute, acute kidney injury or chronic kidney disease due to nephrotoxic drugs
- Comorbid and pre-morose states associated with liver cirrhosis

Cautions

Loop diuretics can exacerbate diabetes mellitus (but hypoglycaemia is less likely than with thiazides) and gout.

If the patient is an enlarged prostate, urinary retention can occur, although this is less likely if small doses and low potency diuretics are used initially.

Hypertension, hypokalaemia and electrolyte disturbances should be corrected before initiation of treatment.

Hypertensive nephropathy: hypokalaemia and electrolyte disturbances may reduce diuretic effect and increase risk of side-effects.

Lower initial doses of diuretics should be used in the elderly because they are particularly susceptible to the side effects.

Adverse effects

Adverse effects of loop diuretics include:

- Mild gastrointestinal disorders, pancreatitis and hepatic encephalopathy
- Hyperuricaemia
- Acute urinary retention
- Water and electrolyte imbalance
- Hypokalaemia, hypomagnesaemia, hypophosphataemia, hypocalcaemia
- Hypertension, hypokalaemia, dehydration, and various hormones imbalance
- Metabolic alkalosis
- Hyperuricaemia
- Blood disorders (bone marrow suppression, thrombocytopenia, and leukopenia)
- Visual disturbance, dizziness and weakness
- Hypersensitivity reactions

Hypokalaemia

Hypokalaemia can occur with both thiazide and loop diuretics. The risk of hypokalaemia depends on the duration of action as well as the potency and is thus greater with thiazides than with an equivalent dose of a loop diuretic. Hypokalaemia is dangerous in severe cardiovascular disease and in patients also being treated with cardiac glycosides. Often the use of potassium-sparing diuretics avoids the need to take potassium supplements, in hepatic failure, hypokalaemia caused by diuretics can precipitate encephalopathy, particularly in alcoholic cirrhosis.
Pharmacology: Cardiovascular

Question 76 of 121

Which of the following drug classes may cause bronchoconstriction:

a) Beta-agonists
b) Beta-blockers
c) Calcium-channel blockers
d) Alpha-blocker
e) Alpha-agonists

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12 Answered

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Answer

Beta-blockers, including those considered to be cardioselective, functionally block all beta receptors in patients with coronary artery disease and a history of MI. Beta-blockers are also important in the management of heart failure and angina. Long-acting beta blockers can be used for larger doses in patients with coronary artery disease and can significantly reduce mortality in these patients.

Notes

Beta-blockers block both beta 1 and beta 2 receptors in the heart, peripheral vasculature, brain, pancreas, and lung.

Therapeutic effects:

- Cardiovascular actions
  - Reduces preload
  - Reduces afterload
  - Increases cardiac contractility
  - Decreases heart rate

- Antiarrhythmic actions
  - Prevents ventricular fibrillation
  - Reduces risk of atrial fibrillation

- Anti-inflammatory actions
  - Decreases inflammation

- Anti-lipidemic actions
  - Reduces cholesterol levels

Indications:

- Hypertension
- Angina pectoris
- Migraine
- Premature labor
- Post-MI
- Refractory heart failure
- Inotropic agents
- Ventricular arrhythmias
- Malignant hypertension
- Malignant hyperthermia

Contraindications:

- Beta-blockers are absolute contraindications in patients with asthma, chronic obstructive pulmonary disease, and bronchial asthma.

- Beta-blockers are absolute contraindications in patients with bronchial asthma, chronic obstructive pulmonary disease, and bronchial asthma.

- Beta-blockers are absolute contraindications in patients with asthma, chronic obstructive pulmonary disease, and bronchial asthma.

- Beta-blockers are absolute contraindications in patients with asthma, chronic obstructive pulmonary disease, and bronchial asthma.

- Beta-blockers are absolute contraindications in patients with asthma, chronic obstructive pulmonary disease, and bronchial asthma.

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- Beta-blockers are absolute contraindications in patients with asthma, chronic obstructive pulmonary disease, and bronchial asthma.

- Beta-blockers are absolute contraindications in patients with asthma, chronic obstructive pulmonary disease, and bronchial asthma.
Pharmacology: Cardiovascular

Question 77 of 121

Which of the following is NOT a predisposing factor for rhabdomyolysis in a patient being considered for statin therapy:

a) High alcohol intake
b) Concomitant fibrate therapy
c) Elderly
d) Hyperthyroidism
e) Renal impairment
Pharmacology: Cardiovascular

Question 77 of 125

Which of the following is NOT a predisposing factor for rhabdomyolysis in a patient being considered for statin therapy?

a) High alcohol intake
b) ConcomitantNNRT therapy

c) Diabetes
d) Hyperlipidemia

e) Renal replacement

Answer

Statins should be used with caution to people:

- With a history of liver disease
- Who consume high levels of alcohol
- With predisposing factors for rhabdomyolysis such as older age (> 70 years)
- Concomitant use with an interacting drug, renal impairment, hypothyroidism, and personal or familial history of hereditary muscular disorders

There is an increased incidence of rhabdomyolysis in a patient with statins, with high flushing doses of nicotinic acid and, with fucidic acid, or with drugs that increase the plasma-sterol concentration, such as monastral and antibiotics, anticonvulsant, and dopamine antagonists, and clindamycin, close monitoring of liver function test. If muscle symptoms occur, of creatine kinase is necessary.

Notes

Statins may be used for primary or secondary prevention of cardiovascular disease under the use of a primary or for statins to cholesterol.
Pharmacology: Cardiovascular

Question 78 of 121

Which of the following is NOT a pharmacological effect of beta-blockers:

a) Reduced heart rate
b) Reduced blood pressure
c) Reduced intraocular pressure
d) Reduced cardiac contractility
e) Reduced AV conduction time

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Pharmacology: Cardiovascular

**Question Navigator**

- **What is the effect of ACE inhibitors on blood pressure?**
- **List the adverse effects of beta blockers.**
- **What is the mechanism of action of thiazide diuretics?**
- **Describe the use of angiotensin receptor blockers.**
- **Define the role of statins in cardiovascular disease.**

**Answer**

**Effects of ACE inhibitors**
- **Reduction in blood pressure**
- **Reduction in cardiovascular risk**
- **Improvement in heart failure outcomes**

**Notes**

- **Beta-blockers** lower the heart rate and reduce oxygen demand.

**Therapeutic effects**
- **Cardiovascular**
  - **Reduces heart rate**
  - **Reduces heart contractility and coronary vasoconstriction**
  - **Improves insulin sensitivity and glucose control**

**Tachycardia**

- **Hyperkalemia**
- **Pharmacokinetics**
- **Agranulocytosis**
- **Nephrotoxicity**
- **Gastrointestinal**
- **Heart failure**
- **Thrombocytopenia**
- **Atrial fibrillation**
- **Glucose**

**Indications**

- **Hypertension**
- **Preventive therapy (used in a prophylactic manner)**
- **Angina**
- **Nonischemic post-MI**
- **Gastrointestinal**
- **Heart failure**
- **Thromboembolism**
- **Atrial fibrillation**
- **Glucose**

**Contraindications**

- **Asthma**
- **Hypersensitivity to beta blockers**
- **Refractory heart failure (CHF)**
- **Severe or unstable angina**
- **Sick sinus syndrome (usually no bradycardia)**
- **Peripheral vascular disease**
- **Pulmonary hypertension**
- **Symptomatic bradycardia**
- **Nonischemic post-MI**

**Side effects**

- **Dizziness**
- **Diaphoresis**
- **Atrial fibrillation**
- **Increased heart rate in small children**
- **Cough**
- **Hypotension**
- **Blurred vision**

**Dosages**

- **Beta-blockers** should be used cautiously in people with:
  - **Heart failure with chronic heart failure (CHF)**
  - **Hypertension**
  - **Cardiac arrhythmias**
  - **Peripheral vascular disease**
  - **Respiratory**
  - **Chronic obstructive pulmonary disease (COPD)**
  - **Pulmonary hypertension**
  - **Sick sinus syndrome**
  - **Atrial fibrillation**
  - **Chronic obstructive pulmonary disease (COPD)**

**Adverse effects**

- **Decreased exercise performance**
- **Hypotension**
- **Bradycardia**
- **Dizziness**
- **Bradycardia**
- **Diaphoresis**
- **Cough**
- **Hypotension**
- **Blurred vision**
- **Increased heart rate in small children**
- **Cough**
- **Hypotension**
- **Blurred vision**

**References**

- **The American Heart Association.**
- **The American College of Cardiology.**
- **The American College of Physicians.**

**Sources**

- **FDA website.**
- **WHO website.**
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**Image credit**

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Pharmacology: Cardiovascular

In adult advanced life support, which of the following best describes the correct administration of adrenaline for a non-shockable rhythm:

- **a** Give 1 mg of adrenaline as soon as intravenous access is achieved and every 3 – 5 minutes thereafter
- **b** Give 0.5 mg of adrenaline as soon as intravenous access is achieved and every 2 – 4 minutes thereafter
- **c** Give 1 mg of adrenaline after 2 minutes of compressions and every 3 – 5 minutes thereafter
- **d** Give 1 mg of adrenaline as soon as intravenous access is achieved and every 10 minutes thereafter
- **e** Give 0.5 mg of adrenaline as soon as intravenous access is achieved and every 3 – 5 minutes thereafter
Pharmacology: Cardiovascular

Question 79 of 121

In adult advanced life support, which of the following best describes the correct administration of adrenaline for a non-shockable rhythm:

- Give 1 mg of adrenaline as soon as intravenous access is achieved and every 3 – 5 minutes thereafter
- Give 0.5 mg of adrenaline as soon as intravenous access is achieved and every 2 – 4 minutes thereafter
- Give 1 mg of adrenaline after 2 minutes of compressions and every 3 – 5 minutes thereafter
- Give 1 mg of adrenaline as soon as intravenous access is achieved and every 10 minutes thereafter
- Give 0.5 mg of adrenaline as soon as intravenous access is achieved and every 3 – 5 minutes thereafter

Answer

IV adrenaline 1 mg should be given as soon as IV access is achieved and then given every 3 – 5 minutes after alternate shocks thereafter.

Notes

Basic life support

- For chest compressions in adult basic life support the heel of one hand should be placed over the middle of the lower half of the patient’s sternum with the other hand on top.
- The sternum should be depressed to a depth of 5 – 6 cm.
- Chest compressions should be performed at a rate of 100 – 120 per minute.
- Thirty compressions should be given before two breaths and that ratio continued (30:2).
- The person giving CPR should be changed every 2 minutes if possible to avoid exhaustion and poor quality chest compressions.
- Interruptions should be minimized (pauses should be < 5 seconds).

Advanced life support

- In adult advanced life support, basic life support should continue whilst the defibrillator pads are attached and the airway managed.
- The pads should be positioned one to the right of the upper sternum below the clavicle and the other in the left midaxillary line in the 5th intercostal space.
- Chest compressions should be paused briefly (< 5 sec) to assess the rhythm and then continued as the defibrillator charges if a shockable rhythm is identified or continued for a further two whole minutes if a non-shockable rhythm is identified.
- When delivering a shock, everybody should be clear of the patient (and any GTN patches and oxygen sources removed).
- Once the shock has been delivered, compressions should resume immediately and continue for a further two minutes before checking the rhythm again or feeling for a pulse.

Shockable rhythm (ventricular fibrillation or pulseless ventricular tachycardia)

- IV adrenaline 1 mg (10 mL of 1:10,000 solution) should be given after 3 shocks and every 3 – 5 minutes after alternate shocks thereafter.
- IV amiodarone 300 mg should be given after 3 shocks. A further dose of IV amiodarone 150 mg may be considered after a total of five defibrillation attempts. Lidocaine (1 mg/kg x 1/10 x 1/10 x 1/10) may be used as an alternative if amiodarone is not available but may not be given if amiodarone has been given already.

Non-shockable rhythm (asystole or pulseless electrical activity)

- IV adrenaline 1 mg should be given as soon as IV access is achieved and then given every 3 – 5 minutes after alternate shocks thereafter.
Pharmacology: Cardiovascular

Regarding alteplase, which of the following statements is INCORRECT:

- a. Alteplase is a recombinant tissue-type plasminogen activator.
- b. Alteplase has selectivity for activation of fibrin-bound plasminogen.
- c. Alteplase has a half-life of about 3–4 minutes.
- d. Alteplase is commonly associated with hypotensive effects.
- e. Alteplase must be given by continuous intravenous infusion.
Pharmacology: Cardiovascular

Chapter 412

Regarding alopepsia, which of the following statements is INCORRECT?

a) Alopepsia is a recumbent tissue-plateauing activation.

b) Alopepsia has a density for activation of fibrin-bound platelets.

c) Alopepsia has a half-life of about 3–4 minutes.

d) Alopepsia is commonly associated with hypovolaemic effects.

e) Alopepsia must be given by continuous intravenous infusion.

Answer

Alopepsia is a recumbent tissue-plateauing anticoagulant (WHP), a naturally occurring fibrin-specific enzyme that has selectivity for activation of fibrin-bound platelets. It has a short half-life of 3–4 minutes and must be given by continuous intravenous infusion but is not associated with antihypovolaemic effects, and can be used in patients when recent streptokinase infusion or recent use of stentplasty contraindicates the use of stentplasty.

Notes

The value of thrombolytic drugs for the treatment of myocardial infarction has been established. Streptokinase and alopepsia have been shown to reduce mortality. Retropulse and thrombolysis are also licensed for acute myocardial infarction. Pharmacologic therapy carries a risk of bleeding, including cerebral haemorrhage, and not all patients can be given this treatment safely.

Mechanism of action

Fibrinolytic drugs act as thrombolitics by activating platelets to form re-occlusions, which degrades fibrin and so breaks up thrombi.

Alopepsia should be given within 6–12 hours of symptom onset, reocclusion and stentplasty within 12 hours of symptom onset, but ideally all should be given within 1 hour; use after 12 hours requires specialist advice.

Contraindications

- Absolute
  - Previous haemorrhagic stroke
  - Ischemic stroke during the previous 6 months
  - Central nervous system damage or necrosis
  - Recent (within 3 weeks) major surgery, head injury or other major trauma
  - Active internal bleeding or gastrointestinal bleeding within the past month
  - Known bleeding disorder

- Relative
  - Retained hyperhydration (BP > 180 mmHg)
  - Transient ischaemic attack during the previous 4 months
  - Oral anticoagulant treatment
  - Pregnancy or less than 4 weeks postpartum
  - Traumatic CPR
  - Non-compressible vascular puncture
  - Active peptic ulcer disease
  - Advanced liver disease
  - Infective endocarditis
  - Previous allergic reaction to fibrinolytic drugs to be used

Adverse effects

- Bleeding (including bleeding calls for discontinuation of the thrombolytic and may require administration of coagulation factors and antihaemostatic drugs)
- Nausea and vomiting
- Further embolisation (either due to clots that break away from the original thrombus or to cholesterol crystal emboli)
- Hypertension
- Hypersensitivity reactions

Streptokinase

Streptokinase (SK) is a single-chain polypeptide, derived from beta haemolytic streptococcus. Its lack of fibrin specificity makes it less desirable thrombolytic drug than tPA compound because it produces more fibrinolytic activity. Streptokinase is antigenic, and as such not be given to patients who have already been exposed, due to the development of antibodies. After about 4–5 days, prolonged persistence of antibodies to streptokinase can reduce the effectiveness of subsequent treatment; therefore, streptokinase should not be used again beyond 4 days of first administration of streptokinase. Minor allergic reactions may occur in up to 10% of patients – streptokinase occurs in less than 0.5% of cases. Hypersensitivity re-occurs during infusion which usually responds to fell or slowing of the infusion.

Alopepsia

Alopepsia is a recombinant tissue-plateauing anticoagulant (WHP), a naturally occurring fibrin-specific enzyme that has selectivity for activation of fibrin-bound platelets. It has a short half-life of 3–4 minutes and must be given by continuous intravenous infusion but is not associated with antihypovolaemic effects, and can be used in patients when recent streptokinase infusion or recent use of stentplasty contraindicates the use of stentplasty.

Retropulse and thrombolysis

Retropulse and thrombolysis are genetically engineered forms of human tPA and have a longer half-life, higher specificity for fibrin, and greater resistance to platelet activation inhibitor. 10 times native tPA. The increase in half-life permits administration in a bolus rather than by continuous infusion.

Resources

- The Royal College of Emergency Medicine
- International Liaison Committee on Resuscitation
- Advanced Trauma Life Support
- British Thoracic Society
- British Thoracic Society
- British Hypertension Society
- American Life Support Group
- Emergency Medical Journal
- International Liaison Committee on Resuscitation
- British Thoracic Society
- British Hypertension Society

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Pharmacology: Cardiovascular

Question 81 of 121

Which of the following is NOT an adverse effect of bendroflumethiazide:

- Hyperglycaemia
- Hyperlipidaemia
- Adrenal suppression
- Postural hypotension
- Hyperuricaemia

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Something wrong?
Pharmacology: Cardiovascular

Which of the following is NOT an adverse effect of bendrofluazide?

a) Hypokalaemia
b) Hypertension
   c) Asthenic suppression
   d) Postural hypotension
c) Hyperkalaemia

Answer

Common side effects of thiazide diuretics include:

- Excessive diuresis
- Postural hypotension, dehydration, renal impairment
- Acid-base and electrolyte imbalance
  - Hypokalaemia, hyperuricaemia, hyperglycaemia, hypercalcaemia, hypercholesterolaemic
  - Metabolic alkalosis
- Hypertension and pain
- Impaired glucose tolerance and hyperglycaemia
- Adrenal plasma-aldosterone concentrations
- Mitro-intestinal disturbances

Notes

Thiazide diuretics are moderately potent diuretics, and are used to relieve edema in chronic heart failure, and in the management of hypertension. They act within 6 to 2 hours of oral administration and must have a duration of action of 12 to 24 hours.

Mechanism of action

Thiazides act mainly on the early segments of distal tubule where they inhibit NaCl reabsorption by binding to the NaCl-transport protein on the Na+K+-ATPase pump. Excretion of Na+ and accompanying water is increased. This increases the load on the distal tubule stimulates ion exchange with Cl- in amiloride, increasing its excretion and causing hypokalaemia and a metabolic alkalosis. Excretion of Ca2+ is increased.

Indications

Bendrofluazide is used for oedema in mild or moderate heart failure. Combination diuretic therapy (with loop and thiazide diuretics) may be effective in patients with oedema resistant to treatment with one diuretic.

Thiazide diuretics are licensed for the treatment of hypertension but are no longer considered the first line diuretic for this indication. In the management of hypertension, a low dose of a thiazide diuretic produces a modest or minor maximal blood pressure lowering effect, with very little bradycardiac disturbance.

Higher doses cause more marked changes to plasma potassium, sodium, uric acid, glucose, and lipids, with little advantage in blood pressure control.

Contraindications

Thiazide diuretics are contraindicated in:

- Allergic disease
- Hyperuricaemia
- Hypersensitivity
- Refractory hypokalaemia
- Symptomatic hyperuricaemia
- Severe hepatic impairment (may precipitate encephalopathy)

Cautions

Thiazide diuretics should be used with caution in:

- Diabetes mellitus (may exacerbate)
- Gout (may exacerbate)
- Tumours: lupus erythematosus (may exacerbate)
- Hyperosmolar states
- Malignancies
- Nephrotic syndrome

Adverse effects

Lower initial doses of diuretics should be used in the elderly because they are particularly susceptible to the side effects. The dose should then be adjusted according to renal function.

Common side effects of thiazide diuretics include:

- Excessive diuresis
- Postural hypotension, dehydration, renal impairment
- Acid-base and electrolyte imbalance
- Hypokalaemia, hyperuricaemia, hyperglycaemia, hypercalcaemia, hypercholesterolaemic
- Metabolic alkalosis
- Hypertension and pain
- Impaired glucose tolerance and hyperglycaemia
- Adrenal plasma-aldosterone concentrations
- Mitro-intestinal disturbances

Hypokalaemia

Hypokalaemia can occur with both thiazide and loop diuretics. The risk of hypokalaemia depends on the duration of action as well as the potency and is thus greater with thiazides than with an equipotent dose of a loop diuretic. Hypokalaemia is dangerous in severe cardiovascular disease and in patients being treated with cardiac glycosides. Often the use of potassium-sparing diuretics avoids the need to take potassium supplements.
Pharmacology: Cardiovascular

Question 82 of 121

Unfractionated heparin therapy is monitored using which of the following:

a. International normalised ratio (INR)
b. Prothrombin time (PT)
c. Activated partial thromboplastin time (APTT)
d. Activated clotting time (ACT)
e. Bleeding time

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Pharmacology: Cardiovascular

Question 62 of 121

Unfractionated heparin is monitored using which of the following:

- a) International normalised ratio (INR)
- b) Prothrombin time (PT)
- c) Activated partial thromboplastin time (aPTT)
- d) Activated clotting time (ACT)
- e) Bleeding time

Answer

Unfractionated heparin is usually given by continuous intravenous infusion for the smoothest control and is the treatment of choice where rapid reversal of anticoagulation may be required (e.g. in surgical patients or late pregnancy). Therapy is monitored by maintaining the activated partial thromboplastin time (aPTT) at 1.5 – 2.5 times the upper limit of normal.

Notes

The make use of anticoagulants is to prevent thrombus formation or extension of an existing thrombus in the slower-moving veins of the circulation, where the thrombus consists of a fibrin with embedded platelets and red cells. Anticoagulants are of less use in preventing thrombus formation in arteries, for in faster-flowing vessels thrombi are composed mainly of platelets with little fibrin. Mechanism of action

Heparin potentiates the activity of antithrombin III, causing inactivation of thrombin. The heparin-antithrombin III complex also inhibits factor Xa and some other factors. Low molecular weight heparin (LMWH) preparations inhibit only factor Xa, PT and aPTT may be prolonged but the PT less so.

Contraindications

Heparin is contraindicated:

- In people with current (or history of) heparin-induced thrombocytopenia
- In people with acute bacterial endocarditis
- In people with active major bleeding, and conditions with a high risk of uncontrolled bleeding, including recent haemorrhagic stroke, major trauma, recent brain, spinal cord or eye surgery, haemophilia and thrombocytopenia
- In people with active peptic or duodenal ulceration

Adverse effects

- Bleeding
- Heparin-induced thrombocytopenia (immune-mediated effect that usually develops after 5 – 10 days, signs may include a JAK3 reduction of platelet count, thrombocytopenia, or skin allergy. If HIT is suspected or confirmed, heparin should be discontinued and an alternative anticoagulant given)
- Hypersensitivity (due to inhibition of administration solutions. Patients with diabetes mellitus, chronic renal failure, acidosis, raised plasma potassium or those taking potassium sparing drugs seem to be most susceptible)
- Osteoporosis (risk lower with LMWH)
- Aleopecia
- Hypersensitivity reactions
- Injection site reactions

Low molecular weight heparin vs unfractionated heparin

Unfractionated heparin is usually given by continuous intravenous infusion for the smoothest control and is the treatment of choice where rapid reversal of anticoagulation may be required (e.g. in surgical patients or late pregnancy). Therapy is monitored by maintaining the aPTT at 1.5 – 2.5 times the upper limit of normal.

Low molecular weight heparin (LMWH) preparations have largely replaced unfractionated heparins.

Advantages of LMWH

Greater ability to inhibit factor Xa directly, interacting less with platelets and so may have a lower tendency to cause bleeding

Greater bioavailability and longer half life in patients making once daily subcutaneous administration possible

More predictable dose response avoiding the need for routine anticoagulant monitoring

Lower associated risk of heparin-induced thrombocytopenia or of osteoporosis

Hemorrhage

Because it has a short duration of action, if hemorrhage occurs it is usually sufficient to withdraw unfractionated or low molecular weight heparins, but rapid reversal of the effects of the heparins is required, protamine sulfate is a specific antidot (but only partially reverses the effects of low molecular weight heparins).
Pharmacology: Cardiovascular

Which of the following is NOT a typical side effect of beta-blockers:

a. Bronchospasm
b. Tachycardia
c. Cold extremities
d. Hyperglycaemia
e. Hypoglycaemia
Pharmacology: Cardiovascular

**Pharmacology: Cardiovascular**

**Answer:**

**Side effects of the different drugs:**

- **Ischemic symptoms:**
  - Decreased symptoms of heart failure (such as symptoms of fluid overload and fatigue)
  - Hypertension and heart failure
  - Acute kidney injury
  - Hypertension and heart failure
  - Increased risk of heart attack
  - Decreased ejection fraction
  - Increased risk of heart attack and stroke

- **Hypertension symptoms:**
  - Sodium and fluid retention
  - Hypertension and heart failure
  - Increased risk of heart attack
  - Increased risk of heart attack
  - Increased risk of heart attack and stroke

- **Hypotensive symptoms:**
  - Decreased symptoms of heart failure
  - Hypertension and heart failure
  - Increased risk of heart attack
  - Increased risk of heart attack and stroke

**Notes:**

- **Beta-blockers:**
  - Decreased symptoms of heart failure
  - Hypertension and heart failure
  - Increased risk of heart attack
  - Increased risk of heart attack and stroke
  - Sodium and fluid retention

- **Angiotensin-converting enzyme inhibitors (ACE-inhibitors):**
  - Decreased symptoms of heart failure
  - Hypertension and heart failure
  - Increased risk of heart attack
  - Increased risk of heart attack and stroke
  - Sodium and fluid retention

- **Aldosterone antagonists:**
  - Decreased symptoms of heart failure
  - Hypertension and heart failure
  - Increased risk of heart attack
  - Increased risk of heart attack and stroke
  - Sodium and fluid retention

**Therapeutic effects:**

- **Cardiovascular system:**
  - Decreased heart rate
  - Decreased heart rate in heart failure
  - Increased risk of heart attack
  - Increased risk of heart attack and stroke

- **Sodium and fluid retention:**
  - Decreased symptoms of heart failure
  - Hypertension and heart failure
  - Increased risk of heart attack
  - Increased risk of heart attack and stroke

- **Angiotensin-converting enzyme inhibitors (ACE-inhibitors):**
  - Decreased symptoms of heart failure
  - Hypertension and heart failure
  - Increased risk of heart attack
  - Increased risk of heart attack and stroke
  - Sodium and fluid retention

- **Aldosterone antagonists:**
  - Decreased symptoms of heart failure
  - Hypertension and heart failure
  - Increased risk of heart attack
  - Increased risk of heart attack and stroke
  - Sodium and fluid retention

**Side effects of the different drugs:**

- **Ischemic symptoms:**
  - Decreased symptoms of heart failure
  - Hypertension and heart failure
  - Acute kidney injury
  - Hypertension and heart failure
  - Increased risk of heart attack

- **Hypertension symptoms:**
  - Sodium and fluid retention
  - Hypertension and heart failure
  - Increased risk of heart attack

- **Hypotensive symptoms:**
  - Decreased symptoms of heart failure
  - Hypertension and heart failure
  - Increased risk of heart attack
  - Increased risk of heart attack and stroke
Pharmacology: Cardiovascular

Question 84 of 121

Regarding fibrinolytics, which of the following statements is INCORRECT:

a. Serious bleeding may occur which may require the administration of coagulation factors and antifibrinolytic drugs.

b. Retepase is a genetically engineered form of human tPA.

c. Tenecteplase has a longer half-life than alteplase allowing for bolus administration.

d. Fibrinolytic drugs act as thrombolytics by directly degrading the fibrin mesh and so breaking up thrombi.

e. Further embolism may occur either due to clots that break away from the original thrombus or to cholesterol crystal emboli.

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Pharmacology: Cardiovascular

Question 1 of 5:

Regarding fibrinolytics, which of the following statements is INCORRECT?

a) Serious bleeding may occur which may require the administration of coagulation factors and anti-fibrinolytic drugs.
b) Replectase is a genetically engineered form of human rPA.
c) Tenecteplase has a shorter half-life than alteplase allowing for bolus administration.
d) Fibrinolytic drugs act as thrombolytics by directly degrading the fibrin mesh and are breaking up thrombs.
e) Further embolism may occur either due to clots that break away from the original thrombus or to cholesterol crystal emboli.

Answer

Fibrinolytic drugs act as thrombolytics by activating plasminogen to form plasmin, which degrades fibrin and so breaks up thrombs.

Notes

The value of thrombolytic drugs for the treatment of myocardial infarction has been established. Streptokinase and urokinase have been shown to reduce mortality. Replectase and tenecteplase are also licensed for acute myocardial infarction. Fibrinolytics therapy carried a risk of bleeding, including cerebral haemorrhage, and not all patients can be given this treatment safely.

Mechanism of action

Fibrinolytic drugs act as thrombolytics by activating plasminogen to form plasmin, which degrades fibrin and so breaks up thrombs.

Alteplase should be given within 6 – 12 hours of symptom onset, replectase and streptokinase within 12 hours of symptom onset, but ideally all should be given within 1 hour; use of alteplase requires specialist advice.

Contraindications

- Absolute
  - Precipitous haemodynamic shock
  - Ischaemic stroke during the previous 6 months
  - Central nervous system damage or neuropathy
  - Recent (within 3 weeks) major surgery, head injury or other major trauma
  - Active internal bleeding or gingival/intestinal bleeding within the past month
  - Known bleeding disorder
- Relative
  - Pre-existing hypertension (SBP > 150 mmHg)
  - Transient ischaemic attack during the previous 6 months
  - Oral anticoagulant treatment
  - Pregnancy or less than 3 week postpartum
  - Traumatic CVA
  - Non-compressible vascular puncture
  - Active peptic ulcer disease
  - Advanced liver disease
  - Intestinal obstruction
  - Previous allergic reaction to fibrinolytic drug to be used

Adverse effects

- Bleeding (serious bleeding calls for discontinuation of the thrombolytic and may require administration of coagulation factors and anti-fibrinolytic drugs)
- Nausea and vomiting
- Further embolus (either due to clots that break away from the original thrombus or to cholesterol crystal emboli)
- Hypotension
- Hypersensitivity reactions

Streptokinase

Streptokinase (SK) is a single chain polypeptide, derived from beta-haemolytic streptococcus. It lacks of fibrin specificity and is less powerful fibrinolytic drug than tPA compound because it produces more fibrinogenolytic activity. Streptokinase is antigenic, and so should not be given to patients who have already been exposed due to the development of antibodies (after about 6 – 12 days). Prophylactic pretreatment of antibodies to streptokinase can reduce the effectiveness of subsequent treatment; therefore, streptokinase should be used only once. After 4 days of continuous administration of streptokinase, minor reactions may occur in up to 10% of patients – anaphylactic reactions in less than 0.5% of cases. Hypotension may occur during infusions which usually responds to fluids or slowing of the infusions.

Alteplase

Alteplase is a recombinant tissue-type plasminogen activator (tPA), a naturally occurring fibrin-specific enzyme that has selectivity for activation of fibrin-bound plasminogen. It has a shorter half-life of 2 – 4 minutes and must be given by continuous intravenous infusion but is not associated with antihypertensive effects; it can be used in patients where more thrombolytic drugs or invasive use of streptokinase contraindicates the use of streptokinase.

Replectase and tenecteplase

Replectase and tenecteplase are genetically engineered forms of human tPA and have longer half-lives, higher specificity for fibrin, and greater resistance to plasminogen activator inhibitor I than native tPA. The increase in half-life permits administration as a bolus rather than by continuous infusion.
Pharmacology: Cardiovascular

Question 85 of 121

Regarding nitrates, which of the following statements is INCORRECT:

a. The effects of sublingual GTN last for about 20 – 30 minutes.
b. Nitrates act as potent coronary vasodilators.
c. Nitrates act to decrease venous return.
d. Prolonged high dosage may cause methaemoglobinemia as a result of oxidation of haemoglobin.
e. Nitrates act by decreasing intracellular cGMP levels causing vascular smooth muscle relaxation.

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Pharmacology: Cardiovascular

Quarter #1 #121

Regarding nitrates, which of the following statements is INCORRECT:

a) The effects of sublingual GTN last for about 20 – 30 minutes.

b) Nitrates act as potent coronary vasodilators.

c) Nitrates act to increase venous return.

d) Prolonged high dosage may cause methaemoglobinemia as a result of oxidation of haemoglobin.

e) Nitrates act by decreasing intracellular cGMP levels causing vascular smooth muscle relaxation.

Answer

Initial metabolism of these drugs releases nitric oxide, which undergoes intracellular conversion to nitric oxide (NO). Nitric oxide then activates guanylate cyclase, causing an increase in the intracellular concentration of cGMP in the vascular smooth muscle cells. cGMP activates protein kinase G, an enzyme that ultimately causes vascular smooth muscle relaxation.

Notes

Nitrates are useful in the management of angina. Although they are potent coronary vasodilators, the main benefit derives from a reduction in venous return which in turn reduces left ventricular effort, decreasing oxygen demands and relieving anginal pain.

Vasodilators can also act in heart failure by arteriolar dilatation which reduces both peripheral vascular resistance and left ventricular pressure during systole resulting in improved cardiac output. They can also cause venous dilatation which results in dilatation of capacitance vessels, increase of venous pooling, and diminution of venous return to the heart (decreasing left ventricular end-diastolic pressure).

Mechanism of action

Initial metabolism of these drugs releases nitric oxide, which undergoes intracellular conversion to nitric oxide (NO). Nitric oxide then activates guanylate cyclase, causing an increase in the intracellular concentration of cGMP in the vascular smooth muscle cells. cGMP activates protein kinase G, an enzyme that ultimately causes vascular smooth muscle relaxation.

Types examples

Sublingual glyceryl trinitrate (GTN) is one of the most effective drugs for providing rapid relief of angina, although its effects only last for 20 – 30 minutes. It may be administered as sublingual tablets or by sublingual administration using aerosol spray.

If sublingual glyceryl trinitrate is required more than twice a week, then combined therapy is required, where beta-blockers or calcium-channel blockers are taken in addition to nitrates which are reserved for acute attacks. If necessary, a long-acting nitrate is added.

Long-acting nitrates are more stable and may be effective for several hours, depending on the drug and the preparation (sublingual, oral, modified release). Isosorbide dinitrate is widely used duration of action of up to 12 hours is claimed for modified-release preparations. The use of isosorbide mononitrate, which is the main active metabolite of the dinitrate, avoids the variable absorption and unpredictable first-pass metabolism of the dinitrate.

Glyceryl trinitrate or isosorbide dinitrate may be tried by intravenous injection when the sublingual form is ineffective in patients with chest pain due to myocardial infarction or severe ischaemia. Intravenous injections are also useful in the treatment of congestive heart failure.

Adverse effects

Side effects such as dizziness, flushing, tachycardia, throbbing headache and pericardial hypotension may limit therapy, especially when angina is severe or when patients are unusually sensitive to the effects of nitrates. Prolonged high dosage may cause methaemoglobinemia as a result of oxidation of haemoglobin.

Contraindications

Nitrates should not be used in people with:

- Acute myocardial infarction (MI) with low filling pressure, acute circulatory collapse, (shock, vascular collapse, or very low blood pressure.
- Hypertrophic obstructive cardiomyopathy (HOCM)
- Congestive heart failure
- Severe peripheral vascular disease
- Severe aortic stenosis
- Closed angle glaucoma
- Severe hypertension, or hypoaldosteronism

Resources

The Royal College of Emergency Medicine

- Web Association for Emergency Medicine
- Advanced Trauma Life Support
- Resuscitation Council UK
- Prehospital Medicine
- Trauma
- Radiopaedia

- Advanced Life Support Group
- Emergency Medicine Journal
- Journal of the American College of Emergency Physicians
- Resuscitation
- Prehospital and Disaster Medicine

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Pharmacology: Cardiovascular

Question 86 of 121

Regarding heparin, which of the following statements is CORRECT:

a. Unfractionated heparin must be monitored using the prothrombin time (PT).

b. Unfractionated heparin is usually given subcutaneously.

c. Heparin is contraindicated in pregnancy.

d. Protamine sulphate completely reverses the effects of low molecular weight heparin (LMWH).

e. Heparin is associated with a risk of hyperkalaemia due to inhibition of aldosterone secretion.
Pharmacology: Cardiovascular

Corrected by A-I

Regarding heparin, which of the following statements is CORRECT:

- a) Unfractionated heparin must be monitored using the partial thromboplastin time (PTT).
- b) Unfractionated heparin is usually given subcutaneously.
- c) Heparin is contraindicated in pregnancy.
- d) Prosthetic subcutaneous completely reverses the effects of low molecular weight heparin (LMWH).
- e) Heparin is associated with a risk of hyperkalaemia due to inhibition of aldosterone secretion.

Answer
Heparin can cause hyperkalaemia due to inhibition of aldosterone secretion, patients with diabetes mellitus, chronic renal failure, accident, related plasma protein or those taking potassium sparing drugs seem to be more susceptible. Plasma potassium concentration should be measured in patients at risk of hyperkalaemia before starting the heparin and monitored regularly thereafter. Partial treatment of heparin is to be continued for longer than 7 days.

Prosthetic subcutaneous only partially reverses the effects of LMWH. Heparin does not cross the placenta and is not contraindicated in pregnancy. Unfractionated heparin therapy is usually given by continuous intravenous infusion for the smoothest control and monitored using the aPTT.

Notes
The main use of anticoagulants is to prevent thrombus formation or extension of an existing thrombus in the slower-moving vessels of the circulation, where the thrombus consists of a fairly well-stranded material with platelets and red cells. Anticoagulants are of less use in preventing thrombus formation in arteries, for in faster-moving vessels thrombus is composed mainly of platelets with little fibrin.

Mechanism of action
Heparin potentiates the activity of antithrombin III, it acts by neutralizing thrombin. The heparin- antithrombin III complex also inhibits Factor Xa and some other factors. Low molecular weight heparins (LMWH) preparation inhibit only factor Xa. PTT and aPTT may not be prolonged for the LMWHs.

Contraindications
Heparin is contraindicated:
- In patients with current or history of heparin-induced thrombocytopenia.
- In patients with acute bacterial endocarditis.
- In patients with active major bleeding, and conditions with a high risk of uncontrolled bleeding, including recent haemorrhagic stroke, major trauma, recent visceral surgery for any surgery, haemophilia and thrombocytopenia.
- In patients with active ulcers or duodenal ulceration.

Adverse effects
- Bleeding
  - Heparin-induced thrombocytopenia (premature mediated effect that usually develops after 5 - 10 days, signs may include a 30% reduction of platelet count, thrombosis, on skin allergic if HIT II is suspected or confirmed, heparin should be discontinued and an alternative anticoagulant given)
  - Heparin-induced thrombosis is a delay of inhibition of aldosterone secretion, patients with diabetes mellitus, chronic renal failure, accident, related plasma protein or those taking potassium sparing drugs seem to be more susceptible.
- Osteoporosis (risk lower with LMWH)
- Allergies
- Hypersensitivity reactions
- Injection site reactions

Low molecular weight heparin vs unfractionated heparin
Unfractionated heparin is usually given by continuous intravenous infusion for the smoothest control and the treatment of choice where rapid reversal of anticoagulation may be required (i.e. in surgical patients or late pregnancy). Therapy is monitored by maintaining the aPTT at 1.5 - 2.5 times the upper limit of normal.

Low molecular weight heparin (LMWH) preparations have largely replaced unfractionated heparins

Advantages of LMWH
- Greater ability to inhibit factor Xa directly, interacting less with platelets and so has a lesser tendency to cause bleeding
- Greater bioavailability and longer half-life in plasma making once daily subcutaneous administration possible
- More predictable dose response avoiding the need for routine anticoagulant monitoring
- Lower associated risk of heparin-induced thrombocytopenia or of osteoporosis

Hemorrhage
Because this is a short duration of action, if hemorrhage occurs it is usually sufficient to withdraw unfractionated or low molecular weight heparins, but if rapid reversal of the effects of the heparins is required, protamine sulfate is a specific antidote but only partially reverses the effects of low molecular weight heparin.
Pharmacology: Cardiovascular

Question 87 of 121

What is the mechanism of action of captopril:

a. Inhibition of the conversion of angiotensinogen to angiotensin
b. Inhibition of the breakdown of angiotensin II
c. Inhibition of the conversion of angiotensin I to angiotensin II
d. Direct inhibition of aldosterone release
e. Blockage of angiotensin II receptors
Pharmacology: Cardiovascular

Question 87 of 151

What is the mechanism of action of captopril?

- a) Inhibition of the conversion of angiotensinogen to angiotensin
- b) Inhibition of the breakdown of angiotensin II
- c) Inhibition of the conversion of angiotensin I to angiotensin II ✗
- d) Direct inhibition of aldosterone release
- e) Blockage of angiotensin II receptor

Answer

Angiotensin-converting enzyme inhibitors (ACE inhibitors) e.g. captopril inhibit the conversion of angiotensin I to angiotensin II, and thus have a vasodilatory effect, lowering both arterial and venous resistances. The cardiac output increases and, because the renovascular resistance falls, there is an increase in renal blood flow. This latter effect, together with reduced aldosterone release, increases Na+ and H2O retention, contracting the blood volume and reducing venous return to the heart.

Notes

Mechanism of action

Angiotensin-converting enzyme inhibitors (ACE inhibitors) e.g. captopril inhibit the conversion of angiotensin I to angiotensin II, and thus have a vasodilatory effect, lowering both arterial and venous resistances. The cardiac output increases and, because the renovascular resistance falls, there is an increase in renal blood flow. This latter effect, together with reduced aldosterone release, increases Na+ and H2O retention, contracting the blood volume and reducing venous return to the heart.

Blocking ACE also diminishes the breakdown of the potent vasodilator bradykinin which is the cause of the peripheral dry cough. Angiotensin II receptor blockers do not have this effect, therefore they are useful alternative for patients who have to discontinue an ACE inhibitor because of persistent cough.

Indications

ACE inhibitors have many uses and are generally well tolerated. They are indicated for:

- Heart failure
- Hypertension
- Diabetic nephropathy
- Secondary prevention of cardiovascular events

Contraindications

ACE inhibitors should be avoided in:

- History of angioedema associated with previous exposure to an ACE inhibitor
- Recurrent angioedema
- Bilateral renal artery stenosis
- Pregnancy and breastfeeding

Cautions

The use of ACE inhibitors is cautioned in:

- Renal impairment
- Afro-Caribbean patients (may respond less well to ACE inhibitors)
- Peripheral vascular disease or generalised arteriosclerosis (risk of clinically silent renovascular disease)
- Primary aldosteronism (patients may respond less well to ACE inhibitors)
- History of idiopathic or hereditary angioedema
- Patients with hypertrophic cardiomyopathy
- Patients with severe or symptomatic aortic stenosis (risk of hypotension)

Concomitant treatment with NSAIDs increases the risk of renal damage, and with potassium-sparing diuretics (or potassium-containing salt substitutes) increases the risk of hyperkalaemia. Hyperkalaemia and other side effects of ACE inhibitors are more common in the elderly and in those with impaired renal function and the dose may need to be reduced.

Adverse effects

Side effects of ACE inhibitors may include:

- Deterioration in renal function
- Hyperkalaemia
- Hypotension
- Persistent dry cough
- Angioedema (non-allergic drug reaction)
- Dizziness and headaches (usually secondary to hypotension)
- Other common adverse effects include abdominal discomfort, dyspepsia, diarrhoea, nausea and vomiting, rash (in particular macula, popular, rash), myalgia, muscle spasms, dysarthria, chest pain, and fatigue.
Pharmacology: Cardiovascular

In adult advanced life support, the defibrillator pads should be placed at which of the following positions:

- **a** One to the left of the upper sternum below the clavicle and one in the left midaxillary line in the 5th intercostal space
- **b** One to the right of the upper sternum below the clavicle and one in the left midaxillary line in the 3rd intercostal space
- **c** One to the right of the upper sternum below the clavicle and one in the left midaxillary line in the 5th intercostal space
- **d** One to the left of the upper sternum below the clavicle and one in the left midaxillary line in the 3rd intercostal space
- **e** One to the right of the lower sternum and one in the left midaxillary line in the 4th intercostal space
Pharmacology: Cardiovascular

Question 6 of 121

In adult advanced life support, the defibrillator pads should be placed at which of the following positions:

a) One to the left of the upper sternum below the clavicle and one in the left midaxillary line in the 5th intercostal space

b) One to the right of the upper sternum below the clavicle and one in the left midaxillary line in the 3rd intercostal space

c) One to the right of the upper sternum below the clavicle and one in the left midaxillary line in the 5th intercostal space

d) One to the left of the upper sternum below the clavicle and one in the left midaxillary line in the 3rd intercostal space

e) One to the right of the lower sternum and one in the left midaxillary line in the 4th intercostal space

Answer

The pads should be positioned one to the right of the upper sternum below the clavicle and the other in the left midaxillary line in the 5th intercostal space.

Notes

Basic life support

- For chest compressions in adult basic life support the heel of one hand should be placed over the middle of the lower half of the patient’s sternum with the other hand on top.
- The sternum should be depressed to a depth of 5 – 6 cm.
- Chest compressions should be performed at a rate of 100 – 120 per minute.
- Thirty compressions should be given before two breaths and that ratio continued (30:2).
- The person giving CPR should be changed every 2 minutes if possible to avoid exhaustion and poor quality chest compressions.
- Interruptions should be minimized (pauses should be < 5 seconds).

Advanced life support

- In adult advanced life support, basic life support should continue whilst the defibrillator pads are attached and the airway managed.
- The pads should be positioned one to the right of the upper sternum below the clavicle and the other in the left midaxillary line in the 5th intercostal space.
- Chest compressions should be paused briefly (< 5 sec) to assess the rhythm and then continued as the defibrillator charges if a shockable rhythm is identified or continued for a further two minutes if a non-shockable rhythm is identified.
- When delivering a shock, everybody should be clear of the patient (and any GTN patches and oxygen sources removed).
- Once the shock has been delivered, compressions should resume immediately and continue for a further two minutes before checking the rhythm again or feeding for a pulse.

Shockable rhythm (ventricular fibrillation or pulseless ventricular tachycardia)

- IV adrenaline 1 mg (10 ml of 1:10,000 solution) should be given after 3 shocks and every 3 – 5 minutes afterwards until shocks thereafter.
- IV amiodarone 300 mg should be given after 3 shocks. A further dose of IV amiodarone 150 mg may be considered after a total of five defibrillation attempts. Lidocaine (1 mg kg sup=1.5 kg sup) may be used as an alternative if amiodarone is not available but may not be given if amiodarone has been given already.

Non-shockable rhythm (asystole or pulseless electrical activity)

- IV adrenaline 1 mg should be given as soon as IV access is achieved and then given every 3 – 5 minutes afterwards until shocks thereafter.

Something wrong?
Pharmacology: Cardiovascular

Question 89 of 121

What is the mechanism of action of atropine in the management of bradyarrhythmias:

a. Increases cAMP
b. Nicotinic receptor agonist
c. Muscarinic receptor antagonist
d. Alpha-adrenergic agonist
e. Beta-adrenergic agonist

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Something wrong?
Pharmacology: Cardiovascular

Question 89 of 121

What is the mechanism of action of atropine in the management of bradyarrhythmias:

a) Increases cAMP  
b) Nicotinic receptor agonist  
c) Muscarinic receptor antagonist ✓  
d) Alpha-adrenergic agonist  
e) Beta-adrenergic agonist

Answer

Atropine antagonises the action of the parasympathetic neurotransmitter acetylcholine at muscarinic receptors. Therefore it blocks the effect of the vagus nerve on both the sinoatrial and atrioventricular node, increasing sinus automaticity and facilitating AV node conduction.

Notes

Atropine is used in sinus, atrial or nodal bradycardia or AV block, when the haemodynamic condition of the patient is unstable because of the bradycardia. The dose in this case is 500 micrograms intravenously, repeated if necessary to a maximum of 3 mg. Doses greater than 3 mg can cause a paradoxical slowing of the heart rate.

Asystole during cardiac arrest is usually caused by primary myocardial pathology rather than excessive vagal tone and there is no evidence that routine use of atropine is beneficial in the treatment of asystole or PEA.

Mechanism of action

Atropine antagonises the action of the parasympathetic neurotransmitter acetylcholine at muscarinic receptors. Therefore it blocks the effect of the vagus nerve on both the sinoatrial and atrioventricular node, increasing sinus automaticity and facilitating AV node conduction.

Contraindications

Antimuscarinics should be avoided in:

- Gastrointestinal obstruction, intestinal atony or paralytic ileus
- Myasthenia gravis
- Prostatic enlargement, significant bladder outflow obstruction or urinary retention
- Severe ulcerative colitis or toxic megacolon

Adverse effects

Side effects of atropine are dose-related and include:

- Dilatation of pupils with loss of accommodation
- Blurred vision
- Dry mouth
- Urinary retention
- Constipation
- Drowsiness
- Acute confusion
- Skin dryness and flushing
- Tachycardia, palpitations and arrhythmias
Pharmacology: Cardiovascular

Question 90 of 121

Adenosine is primarily indicated for which of the following:

a. Non-shockable rhythm in cardiac arrest
b. Ventricular tachycardia
c. New onset fast atrial fibrillation
d. Paroxysmal supraventricular tachycardia
e. Bradyarrhythmias

< Previous  Next >  See Answer  Something wrong?
Pharmacology: Cardiovascular

Question 10 of 121

Adenosine is primarily indicated for which of the following:

a) Non-shockable rhythm in cardiac arrest
b) Ventricular tachycardia
c) New onset fast atrial fibrillation
d) Paroxysmal supraventricular tachycardia

e) Bradycardias

Answer

Adenosine is usually the treatment of choice for terminating paroxysmal supraventricular tachycardia including those associated with accessory conduction pathways e.g. Wolff-Parkinson-White syndrome.

Notes

Adenosine is usually the treatment of choice for terminating paroxysmal supraventricular tachycardia including those associated with accessory conduction pathways e.g. Wolff-Parkinson-White syndrome.

Mechanism of action

Adenosine stimulates A1-adenosine receptors and opens acetylcholine sensitive K+ channels, increasing K+ efflux. This hyperpolarises the cell membrane in the atrioventricular node and, by inhibiting the calcium channels, slows conduction in the AVN. As it has a very short duration of action (half-life only about 6 – 10 seconds), most side effects are short lived.

Administration

For a regular narrow-complex tachycardia the first step is to attempt vagal manoeuvres. If this is unsuccessful and the tachycardia persists, 6 mg intravenous adenosine should be administered into a central/large vein over 2 seconds, followed by 12 mg after 1 – 2 minutes if required, then a further 12 mg after 1 – 2 minutes if required (max 30 mg).

The effects of adenosine are potentiated by dipyridamole, therefore if it is essential to give adenosine in a patient taking dipyridamole the dose should be quartered.

The patient should be warned that they will feel unwell and may experience chest discomfort for a few seconds following the injection. An ECG (preferably multi-lead) should be recorded during the injection. If adenosine is contraindicated, or fails to terminate a regular narrow-complex tachycardia, the administration of verapamil 2.5 – 5 mg IV over 2 mins should be considered.

Contraindications

Adenosine is contraindicated in:

- Asthma and COPD (can cause bronchospasm)
- Decompensated heart failure
- Long QT syndrome
- Second or third-degree AV block and sick sinus syndrome (unless pacemaker fitted)
- Severe hypotension

Adverse effects

Common side effects of adenosine include:

- Apprehension
- Dizziness, flushing, headache, nausea, dyspnoea
- Angina (discontinue
- AV block, sinus pause and arrhythmia (discontinue if any side or severe bradycardia occur)
Pharmacology: Cardiovascular

Question 91 of 121

Sodium nitroprusside is used therapeutically for which of the following effects:

- Alpha-blockade
- Vasodilation
- Negative inotropic effect
- Negative chronotropic effect
- Bronchodilation

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- Irish Association for Emergency Medicine
- Advanced Trauma Life Support
- Resuscitation Council (UK)
- TeachMeAnatomy
- Trauma.org
- Radiopaedia

Advanced Life Support Group
Emergency Medicine Journal
Lifeinthefastlane
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Pharmacology: Cardiovascular

Question 93 of 121

Sodium nitroprusside is used therapeutically for which of the following effects:

- Alpha-blockade
- Vasodilation ✓
- Negative isotropic effect
- Negative chronotropic effect
- Bronchodilation

Answer

Sodium nitroprusside decomposes in the blood to release nitric oxide, an unstable compound that causes vasodilatation. Sodium nitroprusside may be used to lower blood pressure in hypertensive emergencies or to relieve symptoms in congestive cardiac failure.

Notes

Sodium nitroprusside decomposes in the blood to release nitric oxide, an unstable compound that causes vasodilatation.

Indications

Sodium nitroprusside is indicated for:

- Hypertensive emergencies
- Controlled hypotension in anesthesia during surgery
- Acute or chronic heart failure

Contraindications

It is contraindicated in:

- Compensatory hypertension
- Leber’s optic atrophy
- Severe vitamin B12 deficiency

Cautions

It is should be used with caution in:

- Elderly
- Hyponatraemia
- Hyperthermia
- Hypothyroidism
- Impaired cerebral circulation
- Ischaemic heart disease

Adverse effects

Side effects associated with over rapid reduction in blood pressure include: headache, dizziness, nausea, retching, abdominal pain, perspiration, palpitation, anxiety, retrosternal discomfort; the infusion rate should be reduced if any of these side effects occur.

Side effects caused by excessive plasma concentration of the cyanide metabolite include tachycardia, sweating, hyperventilation, arrhythmias, marked metabolic acidosis; the drug should be discontinued and the cyanide antidote given if these effects occur.
Pharmacology: Cardiovascular

Question 92 of 121

Which of the following is NOT a benefit of low molecular weight heparin (LMWH) compared to unfractionated heparin:

- a  Longer plasma half-life
- b  Lower risk of thrombocytopaenia
- c  Lower risk of osteoporosis
- d  Readily reversed with specific antidote
- e  More predictable dose-response

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Pharmacology: Cardiovascular

Question No: 211

Which of the following is NOT a benefit of low molecular weight heparin (LMWH) compared to unfractionated heparin?

A) Longer plasma half-life
B) Lower risk of neurotoxicity
C) Lower risk of osteoporosis
D) Readily reversed with specific antidote
E) More predictable dose response

Answer

Unfractionated heparin is less readily reversible as it has a shorter duration of action. If hemorrhage occurs it is usually sufficient to withdraw an unfractionated or low molecular weight heparin. But if rapid reversal of the effects of the heparin is required, an agent with a specific antidote (but only partially reverses the effects of low molecular weight heparin).

Advantages of LMWH

- Greater ability to inhibit factor Xa directly, interacting with platelets and so may have a lower tendency to cause bleeding.
- Greater bioavailability and longer half-life in planar compared to once daily subcutaneous administration possible.
- More predictable dose response avoiding the need for routine anticoagulant monitoring.
- Lower associated risk of heparin-induced thrombocytopenia or of osteoporosis.

Notes

The main use of anticoagulants is to prevent thrombosis formation or extension of an existing thrombus in the lower moving venous side of the circulation, where the thrombus consists of a fibrin network enmeshed with platelets and red blood cells. Anticoagulants are less used in preventing thrombosis in arteries, for fast moving vessels thrombosis are compared mainly of platelets with little fibrin.

Mechanism of action

Heparin inhibits the activity of antithrombin III, causing inactivation of thrombin. The heparin-antithrombin III complex also inhibits factor Xa and some other factors. Low molecular weight heparin (LMWH) preparations inhibit only factor Xa, PT and aPTT may both be prolonged but the PT less so.

Contraindications

Heparins are contraindicated:

- In people with current (or history of) heparin-induced thrombocytopenia.
- In people with acute bacterial endocarditis.
- In people with active major bleeding, and conditions with a high risk of uncontrolled bleeding, including recent hemorrhagic stroke, major trauma, recent lower, spinal cord or eye surgery, haemoptysis and hemorrhage.
- In people with active gastric or duodenal ulceration.

Adverse effects

- Bleeding.
- Heparin-induced thrombocytopenia (LMWH are a reduced risk factor that usually develops within 1 - 3 days, signs may include a 30% reduction of platelet count, thrombosis or mimic HIT is suspected or confirmed, heparin should be discontinued and an alternative anticoagulant given).
- Hypokalemia (due to inhibition of aldosterone secretion that interferes with renal effect).
- Chronic renal failure, acidosis, raised plasma potassium or those taking potassium sparing drugs seem to be more susceptible.
- Osteoporosis (risk lower with LMWH).
- Allergies.
- Hypersensitivity reactions.
- Injection site reactions.

Low molecular weight heparin vs unfractionated heparin

Unfractionated heparin is usually given by continuous intravenous infusion for the smoothed control and the treatment of a site where rapid reversal of anticoagulation (e.g., in surgical patients or late pregnancy). Therapy is monitored by maintaining the aPTT at 1.5 - 2.5 times the upper limit of normal.

Low molecular weight heparin LMWH preparations have largely replaced unfractionated heparin.

Advantages of LMWH

- Greater ability to inhibit factor Xa directly, interacting with platelets and so may have a lower tendency to cause bleeding.
- Greater bioavailability and longer half-life in planar compared to once daily subcutaneous administration possible.
- More predictable dose response avoiding the need for routine anticoagulant monitoring.
- Lower associated risk of heparin-induced thrombocytopenia or of osteoporosis.

Haemorrhage

Because it has a short duration of action, if hemorrhage occurs it is usually sufficient to withdraw an unfractionated or low molecular weight heparin, but if rapid reversal of the effects of the heparin is required, antithrombin is a specific antidote (but only partially reverses the effects of low molecular weight heparin).
Pharmacology: Cardiovascular

Question 93 of 121

Which of the following is NOT an adverse effect associated with warfarin therapy:

- a. Hepatic dysfunction
- b. Calciphylaxis
- c. Skin necrosis
- d. Pancreatitis
- e. Renal impairment

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**Answer**

Adequate effect of methyldopa:
- **Thermoregulation:** adequate effect of methyldopa in lowering arterial pressure
- **Cough suppression:** adequate effect of methyldopa in reducing cough
- **Skin rashes:** adequate effect of methyldopa in preventing rashes

**Notes**

Pharmacological actions relevant to the treatment of arterial hypertension include:
1. **Decreased cardiac output:** due to reduced venous return and arterial pressure
2. **Increased renal excretion:** of sodium and water
3. **Decreased peripheral vascular resistance:** leading to decreased arterial pressure

**Mechanism of action**

Methyldopa is a competitive inhibitor of decarboxylase, an enzyme that is involved in the biosynthesis of dopamine. It functions by blocking the conversion of L-tyrosine to dopamine, which is a precursor of norepinephrine and epinephrine.

**Indications**

Methyldopa is indicated for:
- **Hypertension:** in the management of mild to moderate hypertension
- **Cough suppression:** in the treatment of cough associated with respiratory conditions

**Contraindications**

Methyldopa is contraindicated in:
- **Known hypersensitivity:** to methyldopa or any of its excipients
- **Severe renal impairment:** with creatinine clearance less than 30 mL/min

**Cautions**

Methyldopa should be used with caution in patients with:
- **Liver disease:** due to increased methyldopa levels
- **Pregnancy:** due to the risk of fetal toxicity

**Adverse effects**

The most common adverse effects of methyldopa are:
- **Nausea and vomiting:** may occur in up to 20% of patients
- **Drowsiness:** may occur in some patients

**Management**

If adverse effects occur:
- **Dose adjustment:** may be required
- **Symptomatic treatment:** may be necessary

**Drug interactions**

Methyldopa may interact with:
- **Alcohol:** may increase sedative effects
- **Cimetidine:** may increase plasma levels

**Resources**

- [Pharmacology Textbook](https://example.com/pharmacology)
- [Methyldopa Information](https://example.com/methyldopa)

**FRCM Success**

No cholesterol and no stress during the exam preparation phase. Good luck!

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**Footnotes**

Pharmacology: Cardiovascular

Adenosine has a half-life of approximately:

a  8 – 10 seconds
b  8 – 10 minutes
c  30 minutes
d  1 hour
e  6 – 8 hours

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- Resuscitation Council (UK)
- TeachMeAnatomy
- Trauma.org
- Radiopaedia

Advanced Life Support Group
- Emergency Medicine Journal
- Lifeinthefastlane
- Instant Anatomy
- Patient.co.uk

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Pharmacology: Cardiovascular

Question 94 of 121

Adenosine has a half-life of approximately:

a) 8 – 10 seconds ✅
b) 8 – 10 minutes ×
c) 30 minutes ×
d) 1 hour

e) 6 – 8 hours

Answer

Adenosine stimulates A1-adenosine receptors and opens acetylcholine sensitive K+ channels, increasing K+ efflux. This hyperpolarises the cell membrane in the atrioventricular node and, by inhibiting the calcium channels, slows conduction in the AVN. As it has a very short duration of action (half-life only about 8 – 10 seconds), most side effects are short lived.

Notes

Adenosine is usually the treatment of choice for terminating paroxysmal supraventricular tachycardia including those associated with accessory conduction pathways e.g. Wolff-Parkinson-White syndrome.

Mechanism of action

Adenosine stimulates A1-adenosine receptors and opens acetylcholine sensitive K+ channels, increasing K+ efflux. This hyperpolarises the cell membrane in the atrioventricular node and, by inhibiting the calcium channels, slows conduction in the AVN. As it has a very short duration of action (half-life only about 8 – 10 seconds), most side effects are short lived.

Administration

For a regular narrow-complex tachycardia the first step is to attempt vagal manoeuvres. If this is unsuccessful and the tachyarrhythmia persists, 6 mg intravenous adenosine should be administered into a central/large vein over 2 seconds, followed by 12 mg after 1 – 2 minutes if required, then a further 12 mg after 1 – 2 minutes if required (max 30 mg).

The effects of adenosine are potentiated by dipryidamole, therefore if it is essential to give adenosine in a patient taking dipiridamole the dose should be quartered.

The patient should be warned that they will feel unwell and may experience chest discomfort for a few seconds following the injection. An ECG (preferably multi-lead) should be recorded during the injection. If adenosine is contraindicated, or fails to terminate a regular narrow-complex tachycardia, the administration of verapamil 2.5 – 5 mg IV over 2 mins should be considered.

Contraindications

Adenosine is contraindicated in:

- Asthma and COPD
- Compromised heart failure
- Long QT syndrome
- Second- or third-degree AV block and sick sinus syndrome (unless pacemaker fitted)
- Severe hypotension

Adverse effects

Common side effects of adenosine include:

- Apprehension
- Dizziness, flushing, headache, nausea, dyspnoea
- Angina (discontinue)
- AV block, sinus pause and arrhythmia (discontinue if asymptotic or severe bradycardia occur)
Pharmacology: Cardiovascular

Question 95 of 121

All of the following are indications for beta-blockers EXCEPT for:

- a. Prinzmetal’s angina
- b. Thyrotoxicosis
- c. Heart failure
- d. Atrial fibrillation
- e. Essential tremor

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Something wrong?
Pharmacology: Cardiovascular

Answer

Data for selected indications in Processed in samples. No data foruner in labeled indications.

Hypertension
Phenothiazines block with an alpha blocker
Antihypertensives

Concerns with uncontrolled side effects, including orthostatic hypotension

Hypotension

Reduced blood pressure
Reduced heart rate

Tretinoin
Gastrointestinal

Increased risk of bleeding

Pancreatitis

Decrease transaminase

Bone metastases block the fatty acid synthetase in the tumors, peripheral vasodilatation by benzylamine

Time-line

More data available on these areas and regions of the are equally effective. There is no absolute difference between these, and the way they affect the increasing arterial pressure in individual arteries.

Baseline failure in non-coplanar vessel intake with additional data on testosterone levels. It is currently recommended to treat arterial hypertension in all cases of hypertension. Some patients treated with antihypertensives had no significant effect on metabolism. This may be due to the absence of the effect of metabolic drug interactions with antihypertensives. It is recommended for patients with severe and moderate hypertension.

Labeling is in addition to have blockers affecting, an antihypertensive or dietary regimen in use, and the levels may remain hypertensive. It is unsuitable for patients with moderate hypertension. It is currently recommended to treat these areas and regions of the are equally effective. There is no absolute difference between these, and the way they affect the increasing arterial pressure in individual arteries.

Indications

Data blocks may be indicated to

Hypertension
Phenothiazines block with an alpha blocker

Antihypertensives

Concerns with uncontrolled side effects, including orthostatic hypotension

Hypotension

Reduced blood pressure
Reduced heart rate

Tretinoin
Gastrointestinal

Increased risk of bleeding

Pancreatitis

Decrease transaminase

Bone metastases block the fatty acid synthetase in the tumors, peripheral vasodilatation by benzylamine

Contradictions

Bone metastases are contraindicated in patients with

Allergy to anticoagulants, hemostatic agents, and sulfonamides.

Rheumatoid arthritis (RA) and lupus erythematosus are in labeled indications.

Contraindications: Inoperable hyperthyroidism and on labeled indications.

Pancreatitis

Decrease transaminase

Bone metastases block the fatty acid synthetase in the tumors, peripheral vasodilatation by benzylamine

Contraindications

Hypertension

Reduced blood pressure
Reduced heart rate

Tretinoin
Gastrointestinal

Increased risk of bleeding

Pancreatitis

Decrease transaminase

Bone metastases block the fatty acid synthetase in the tumors, peripheral vasodilatation by benzylamine

Adverse effects

Detecting symptoms of ischemia (such as chest pain, shortness of breath, or cold hands)

Hypertension

Orthostatic hypotension

Ectopic beat and arrhythmias

Miscellaneous reactions in patients

Effect on appetite, weight, and blood pressure

Fatigue and increase in energy and weight

Sleep disturbance with depression

Blood pressure

Elevation of blood pressure (may cause fainting and anxiety in some cases)

Questions and answers

1. What is the role of hydrochlorothiazide in the management of hypertension?

2. What are the common adverse effects of hydrochlorothiazide?

3. How does hydrochlorothiazide work in the body to lower blood pressure?

4. What is the difference between hydrochlorothiazide and other diuretics?

5. Is hydrochlorothiazide safe for patients with diabetes or kidney disease?

6. What is the appropriate dosage of hydrochlorothiazide, and how often should it be taken?

7. Can hydrochlorothiazide be taken with other medications, or are there any interactions to be aware of?

8. Are there any specific populations for whom hydrochlorothiazide is contraindicated?

9. What are the potential long-term effects of using hydrochlorothiazide for the treatment of high blood pressure?

10. How does hydrochlorothiazide compare to other blood pressure-lowering medications in terms of effectiveness and side effects?
Pharmacology: Cardiovascular

Question 96 of 121

Clinical features of digoxin toxicity include all of the following EXCEPT for:

- Visual disturbance
- Gastrointestinal disturbance
- Hyperkalaemia
- Hypoglycaemia
- Arrhythmias

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Question 1
Clinical features of digoxin toxicity include all of the following EXCEPT:

- A) Visual disturbance
- B) Gastrointestinal disturbances
- C) Cardiac disturbances
- D) Hypokalemia
- E) Hypngeytropinemia

Answer
Digoxin has a narrow therapeutic index. Features of toxicity include gastrointestinal effects (nausea, vomiting, anorexia) and arrhythmias (atrial fibrillation, supraventricular tachycardia). Digoxin toxicity has been reported in children and adults. Common symptoms include nausea, vomiting, diarrhea, constipation, abdominal pain, headache, and palpitations. Common cardiovascular symptoms include atrial fibrillation, supraventricular tachycardia, cardiac arrhythmias, and congestive heart failure. Treatment of digoxin toxicity includes discontinuation of the drug, intravenous potassium chloride, and digoxin-specific Fab antibodies. In case of severe toxicity, hemodialysis or plasmapheresis may be considered. Prevention of digoxin toxicity includes monitoring serum digoxin levels and adjusting the dose as needed. 

Clinical features of digoxin toxicity include:

- Visual disturbances
- Gastrointestinal disturbances
- Cardiac disturbances
- Hypokalemia
- Hypogeitoninemia

Question 2
Digoxin is a cardiac glycoside that increases the force of myocardial contractility (positive inotropy), and slows the heart rate (negative chronotropic effect). Digoxin is primarily used to treat atrial fibrillation and control the ventricular response rate in patients with atrial fibrillation. The positive inotropic effect of digoxin increases the force of myocardial contraction, which can lead to increased cardiac output and improved oxygen delivery to tissues. The negative chronotropic effect of digoxin reduces the heart rate, which can help to control the ventricular response rate in patients with atrial fibrillation.

Mechanism of action
Inotropic effect
Digoxin inhibits the enzyme Na+/K+ ATPase, which is responsible for Na+/K+ exchange across the cardiac cell membrane. This inhibition decreases the accumulation of Na+ inside the cell and increases the concentration of K+ outside the cell. The increased K+ concentration can lead to an increase in intracellular Ca2+ levels, which can increase cardiac contractility. Digoxin also has a negative chronotropic effect, which reduces the heart rate and decreases the ventricular response rate in patients with atrial fibrillation.

Indications
Digoxin is used to treat atrial fibrillation, control the ventricular rate in patients with atrial fibrillation, and reduce the risk of thromboembolic events in patients with atrial fibrillation. It is also used to treat congestive heart failure, reduce the risk of sudden cardiac death in patients with a history of myocardial infarction, and improve the symptoms of chronic heart failure.

Contraindications
Digoxin is contraindicated in patients with:

- Hypersensitivity to digoxin or any of its components
- Severe renal impairment
- Severe hepatic impairment
- Advanced atrial fibrillation
- Congestive heart failure

Adverse effects
The adverse effects of digoxin are frequent and include:

- Cardiovascular: Bradycardia, AV block, and increased ventricular arrhythmias
- Gastrointestinal: Nausea, vomiting, anorexia, and abdominal pain
- Central nervous system: Dizziness, headache, and confusion
- Other: Hypothyroidism, hyperkalemia, and hypomagnesemia

Digoxin toxicity is rarely encountered, but when it occurs, it can be severe. The most common manifestations of digoxin toxicity include:

- Bradycardia
- Atrioventricular block
- Hypokalemia
- Hypomagnesemia
- Hypothyroidism
- Hypomagnesemia
- Hypokalemia

Treatment of digoxin toxicity includes:

- Discontinuation of digoxin
- Intravenous potassium chloride
- Calcium gluconate
- Cardiac pacing
- Dialysis

Resources
- American Heart Association
- Heart Rhythm Society
- American College of Cardiology
- American College of Physicians
- European Society of Cardiology

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What is the maximum dose of adenosine recommended for management of a regular narrow-complex tachycardia:

- a) 6 mg
- b) 12 mg
- c) 24 mg
- d) 30 mg
- e) 36 mg

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Question ID: 251

What is the maximum dose of adenosine recommended for administration of a single dose of aortic stenosis in aortic stenosis patients?

a) 6 mg
b) 12 mg
c) 24 mg
d) 30 mg

Answer

The first step in treatment of regular narrow complex tachycardia is to attempt vagal manoeuvres (uncontrolled diastolic ventricular tachycardia or aortic stenosis). If the tachycardia is not induced by vagal manoeuvres, adenosine 6 mg IV should be given as a rapid bolus using a large canula and a large syringe. If there is no response (i.e. no transient slowing or termination of the tachycardia), adenosine 6 mg 12 mg IV should be given and if there is no response, one further 12 mg IV should be given (max 30 mg).

Notes

The approach to the management of tachycardia should follow the Resuscitation Council guidelines.

If the patient has adverse features

Adverse features:

- Shock (hypotension, paleness, sweating, cold extremities, confusion, impaired consciousness)
- Sense (transient loss of consciousness)
- Heart failure (muffled sounds, cardiac failure, pericardial, hypotensive, hypoplastic)
- Hypothermia (cardiac arrest, hypothermic changes on ECG)

If any adverse features are present, emergency cardioversion with a synchronized DC shock is indicated. If cardioversion fails to terminate the tachycardia and adverse features persist, intravenous adenosine 300 mg IV over 10–20 minutes should be given and further cardioversion attempted. The loading dose of adenosine can be followed by an infusion of 100 mg over 10–20 minutes, given via a large syringe.

If the patient has no adverse features

If the patient is in stable QRs the duration should be considered.

If the QRs duration is less than 5 seconds, it is a broad-complex tachycardia.

If the QRs duration is more than 5 seconds, it is a narrow-complex tachycardia.

Broad-complex tachycardia

Broad-complex tachycardias are mostly ventricular in origin but may be a supraventricular arrhythmia with aberrant conduction.

- A regular broad-complex tachycardia is likely to be a ventricular tachycardia or a regular supraventricular rhythm with bundle branch block.
- A ventricular tachycardia for broad-complex tachycardia of uncertain origin should be treated with adenosine 300 mg IV over 10–20 minutes, followed by an infusion of 100 mg over the next 24 hours.
- Fibrillation confirmed as SVT with bundle branch block, the patient should be treated as for narrow-complex tachycardia.
- A stable patient with an irregular broad-complex tachycardia is most likely to be in AF with bundle branch block, which, if AF with ventricular pre-contraction or polymorphic VT (a normal condition) is possible.
- Expert advice should be sought for the assessment and treatment of irregular broad-complex tachycardia.
- Torsade de pointes VT should be treated by stopping all drugs known to prolong the QT interval, correcting electrolyte abnormalities, and giving magnesium sulfate 2 g IV over 30 minutes. Expert advice should be sought in other treatment options including intravenous sympathetic may be required to prevent fibrillation once the arrhythmia has been controlled.

Narrow-complex tachycardia

The narrow-complex arrhythmias are supraventricular in origin.

- A regular narrow-complex tachycardia may represent paroxysmal SVT or atrial flutter with 2:1 conduction. It may be difficult to differentiate between the two.
- The first step in treatment of regular narrow-complex tachycardia is to attempt vagal manoeuvres (uncontrolled diastolic ventricular tachycardia or aortic stenosis).
- If the tachycardia is not induced by vagal manoeuvres, adenosine 6 mg IV should be given as a rapid bolus using a large canula and a large syringe. The patient should be warned that they will feel lightheaded and may experience chest discomfort for a few seconds following the injection. An ECG (preferably with lead V6) should be recorded during the injection.
- If the ventricular rate does not increase and then speeds up again, this may indicate atrial activity such as atrial flutter or other atrial tachyarrhythmia, and this should be treated accordingly.
- If the rate is not expected to decrease, even after a slow rate and/or the effect of vagal manoeuvres, adenosine 6 mg IV should be given as a rapid bolus using a large canula and a large syringe. If there is no response, one further 12 mg IV should be given (max 30 mg). Lack of response to adenosine will occur if the tachycardia is not a ventricular tachycardia.
- If the ventricular rate does not increase and then speeds up again, this may indicate atrial activity such as atrial flutter or other atrial tachyarrhythmia, and this should be treated accordingly.
- If the tachycardia is not induced by vagal manoeuvres, adenosine 300 mg IV over 10–20 minutes should be given and if there is no response, one further 12 mg IV should be given (max 30 mg).
- Immediate treatment options include rate control with beta-blockers, rhythm control using drugs to achieve chemical cardioversion, and treatment with beta-blockers, and/or the use of other antiarrhythmic agents.
- Torsade de pointes VT should be treated by stopping all drugs known to prolong the QT interval, correcting electrolyte abnormalities, and giving magnesium sulfate 2 g IV over 30 minutes. Expert advice should be sought in other treatment options including intravenous sympathetic may be required to prevent fibrillation once the arrhythmia has been controlled.
Pharmacology: Cardiovascular

Question 98 of 121

Regarding ACE inhibitors, which of the following statements is CORRECT:

- **a** They are contraindicated in diabetic nephropathy due to risk of worsening renal impairment.
- **b** They are recommended first line treatment for hypertension in patients of Afro-Caribbean descent.
- **c** ACE inhibitors are used first line for hypertension in pregnancy.
- **d** Angiotensin-II receptor blockers are a useful alternative in patients who cannot tolerate ACE-inhibitors due a persistent cough.
- **e** ACE-inhibitors cause a increase in histamine release which can result in a persistent dry cough.
Pharmacology: Cardiovascular

Gordon Hill D21

Regarding ACE inhibitors, which of the following statements is CORRECT?

- They are contraindicated in diabetic nephropathy due to risk of worsening renal impairment.
- They are recommended first-line treatment for hypertension in patients of African Caribbean descent.
- ACE inhibitors are used first-line for hypertension in pregnancy.
- Angiotensin II receptor blockers are a useful alternative in patients who cannot tolerate ACE inhibitors due to persistent cough.
- ACE inhibitors cause a decrease in histamine release which can result in a persistent dry cough.

Answer

ACE inhibitors should be used with caution in patients of African Caribbean descent who may respond less well. Calcium channel blockers are first-line for hypertension in these patients. ACE inhibitors have a role in the management of diabetic nephropathy. ACE inhibitors are contraindicated in pregnant women. ACE inhibitors inhibit the breakdown of the potent vasoconstrictor bradykinin which is the cause of the persistent dry cough. Angiotensin II receptor blockers do not have this effect, therefore they are useful alternatives for patients who have to discontinue an ACE inhibitor because of persistent cough.

Notes

Mechanism of action

Angiotensin-converting enzyme inhibitors (ACE inhibitors) e.g. captopril inhibit the conversion of angiotensin I to angiotensin II, and thus have a vasodilatory effect, lowering both arterial and venous resistance. The cardiac output increases and, because the renovascular resistance falls, there is an increase in renal blood flow. This latter effect, together with reduced aldosterone release, increases Na+ and K+ excretion, contracting the blood volume and reducing venous return to the heart. Blocking ACE also diminishes the breakdown of the potent vasoconstrictor bradykinin which is the cause of the persistent dry cough. Angiotensin II receptor blockers do not have this effect, therefore they are useful alternatives for patients who have to discontinue an ACE inhibitor because of persistent cough.

Indications

ACE inhibitors have many uses and are generally well tolerated. They are indicated for:

- Heart failure
- Hypertension
- Diabetic nephropathy
- Secondary prevention of cardiovascular events

Contraindications

ACE inhibitors should be avoided in:

- History of angioedema associated with previous exposure to an ACE inhibitor
- Recurrent angioedema
- Bilateral renal artery stenosis
- Pregnancy and breastfeeding

Cautions

The use of ACE inhibitors is cautioned in:

- Renal impairment
- African Caribbean patients (may respond less well to ACE inhibitors)
- Peripheral vascular disease or generalised atherosclerosis (risk of clinically silent renal vascular disease)
- Primary aldosteronism (patients may respond less well to ACE inhibitors)
- History of idiopathic or hereditary angioedema
- Patients with hypertrophic cardiomyopathy
- Patients with severe or symptomatic aortic stenosis (risk of hypertensive crisis)

Concomitant treatment with NSAIDs increases the risk of renal damage, and with potassium-sparing diuretics (or potassium-containing salt substitutes) increases the risk of hyperkalaemia. Hyperkalaemia and other side effects of ACE inhibitors are more common in the elderly and in those with impaired renal function and the dose may need to be reduced.

Adverse effects

Side effects of ACE inhibitors may include:

- Deterioration in renal function
- Hypokalaemia
- Hypotension
- Persistent dry cough
- Angioedema (non-allergic drug reaction)
- Dizziness and headaches (possibly secondary to hypotension)
- Other common adverse effects include abdominal discomfort, dyspepsia, diarrhoea, nausea and vomiting, rash (in particular, maculopapular rash), rashes, muscle spasms, dizziness, chest pain, and fatigue.
Pharmacology: Cardiovascular

Question 99 of 121

Which of the following is NOT a typical side effect of glyceryl trinitrate:

a. Flushing
b. Tachycardia
c. Hyperkalaemia
d. Postural hypotension
e. Throbbing headache

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Pharmacology: Cardiovascular

Question P7 of 121
Which of the following is NOT a typical side effect of glyceryl trinitrate:

a) Flushing
b) Tachycardia
c) Hypertension

Answer
Side effects such as dizziness, flushing, tachycardia, throbbing headache and postural hypotension may limit therapy, especially when angina is severe or when patients are unusually sensitive to the effects of nitrates. Prolonged high dosage may cause methemoglobinemia as a result of oxidation of haemoglobin.

Notes
Nitrates are useful in the management of angina. Although they are potent coronary vasodilators, the main benefit derives from a reduction in venous return which in turn reduces left ventricular effort, decreasing oxygen demands and relieving anginal pain.

Vasodilators can also act in heart failure by arteriolar dilatation which reduces both peripheral vascular resistance and left ventricular pressure during systole resulting in improved cardiac output. They can also cause venous dilatation which results in dilatation of capacitance vessels, increase of venous pooling, and diminution of venous return to the heart (decreasing left ventricular end diastolic pressure).

Mechanism of action
Initial metabolism of these drugs releases nitric oxide, which undergoes intracellular conversion to nitric oxide (NO). Nitric oxide then activates guanyl cyclase, causing an increase in the intracellular concentration of cGMP in the vascular smooth muscle cells. cGMP activates protein kinase G, an enzyme that ultimately causes vascular smooth muscle relaxation.

Type examples
Sublingual glyceryl trinitrate (GTN) is one of the most effective drugs for providing rapid relief of angina, although its effects only last for 20–30 minutes. It may be administered as sublingual tablets or by sublingual administration using aerosol spray.

If sublingual glyceryl trinitrate is required more than twice a week, then combined therapy is required, where beta-blockers or calcium-channel blockers are taken in addition to nitrates which are reserved for acute attacks. If necessary a long-acting nitrate is added.

Long-acting nitrates are more stable and may be effective for several hours, depending on the drug and the preparation (sublingual, oral, modified release). Isosorbide dinitrate is wildly used, duration of action up to 12 hours is claimed for modified-release preparations. The use of inosorbide mononitrate, which is the main active metabolite of the dinitrate, avoids the variable absorption and unpredictable first-pass metabolism of the dinitrate.

Glyceryl trinitrate or isosorbide dinitrate may be tried by intravenous injection when the sublingual form is ineffective in patients with chest pain due to myocardial infarction or severe ischaemia. Intravenous injections are also useful in the treatment of congestive heart failure.

Adverse effects
Side effects such as dizziness, flushing, tachycardia, throbbing headache and postural hypotension may limit therapy, especially when angina is severe or when patients are unusually sensitive to the effects of nitrates. Prolonged high dosage may cause methemoglobinemia as a result of oxidation of haemoglobin.

Contraindications
Nitrates should not be used in people with:

- Acute myocardial infarction (MI) with low filling pressure, acute circulatory failure (shock, vascular collapse), or very low blood pressure
- Hypertrophic obstructive cardiomyopathy (HOCM), constrictive pericarditis, cardiac tamponade, low cardiac filling pressures, or aortic/mitral valve stenosis
- Diseases associated with a raised intracranial pressure (for example following a head trauma, including cerebral haemorrhage)
- Severe anaemia
- Closed angle glaucoma
- Severe hypotension, or hypovolaemia

Resources
- The Royal College of Emergency Medicine
- UK Association for Emergency Medicine
- Advanced Trauma Life Support
- Resuscitation Council (UK)
- ‘Stich’ for the future
- ‘Yoelmung’
- Radiopedia
- Adventures In Support Group
- Emergency Medicine Journal
- Resuscitation
- Instant Anatomy
- Emtalks
Pharmacology: Cardiovascular

Question 100 of 121

Your consultant wishes to chemically cardiovert a patient who has presented to ED with new onset atrial fibrillation (AF). Which of the following would be an absolute contraindication to the use of flecainide:

- a. Asthma
- b. Thyroid dysfunction
- c. Acute porphyrias
- d. Heart failure
- e. Hypokalaemia

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Question 60 of 121.

Your consultant wishes to chemically cardiovert a patient who has presented to ED with new onset atrial fibrillation (AF). Which of the following would be an absolute contraindication to the use of flecainide:

a) Asthma
b) Thyroid dysfunction
c) Acute periphraxis
d) Heart failure

e) Hypokalaemia

Answer

Sinus rhythm can be restored by electrical cardioversion, or pharmacological cardioversion with an oral or intravenous antiarrhythmic drug e.g. flecainide or amiodarone. Flecainide should not be given when there is known ischaemic or structural heart disease. Consider amiodarone in patients with left ventricular impairment or heart failure.

Notes

Flecainide acetate is an orally active class III antiarrhythmic and may be of value for serious symptomatic ventricular arrhythmias. It may also be indicated for junctional re-entry tachycardias and for paroxysmal atrial fibrillation. However, it has a negative inotropic action and can precipitate serious arrhythmias in a small minority of patients (including those with otherwise normal hearts).

Contraindications

Flecainide is contraindicated in:

- Abnormal left ventricular function
- Atrial conduction defects (unless pacing rescue available)
- Bundle branch block (unless pacing rescue available)
- Distal block (unless pacing rescue available)
- Haemodynamically significant valvular heart disease
- Heart failure
- History of myocardial infarction and either asymptomatic ventricular ectopics or asymptomatic non-sustained ventricular tachycardia
- Long-standing atrial fibrillation where conversion to sinus rhythm not attempted
- Second-degree or greater AV block (unless pacing rescue available)
- Sinus node dysfunction (unless pacing rescue available)

Cautions

Flecainide should be used with caution in:

- Atrial fibrillation following heart surgery
- Elderly (accumulation may occur)
- Patients with pacemakers (especially those who may be pacemaker dependent because stimulation threshold may rise appreciably)

Adverse effects

Common side effects of flecainide include:

- Anaemia
- Dizziness
- Dyspnoea
- Fatigue
- Fever
- Oedema
- Pro-arrhythmic effects
- Visual disturbances
Pharmacology: Cardiovascular

What is the initial dose of amiodarone recommended for treatment of a stable regular broad-complex tachycardia:

a 50 mg
b 150 mg
c 200 mg
d 300 mg
e 400 mg

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Something wrong?
Pharmacology: Cardiovascular

Question 1
What is the initial dose of amiodarone recommended for treatment of a stable regular broad-complex tachycardia?

a) 50 mg
b) 150 mg
c) 200 mg
d) 300 mg

e) 400 mg

Answer
A ventricular tachycardia (or broad-complex tachycardia of uncertain origin) should be treated with amiodarone 300 mg IV over 10-20 minutes followed by an infusion of 1 mg/mg over the next 24 hours.

Notes
The approach to the management of tachycardias should follow the Resuscitation Council guidelines.

If the patient has adverse features:

Adverse features:
- Shock
- Hypotension, palpitations, cold extremities, confusion, impaired consciousness
- Syncope (transient loss of consciousness)
- Heart failure (peripheral oedema, raised JVP, peripheral oedema, hepatomegaly)
- Ischaemic tachycardia (electrocardiographic signs of ischaemic heart disease and ECG)

If any adverse features are present, immediate cardioversion with a synchronised DC shock is indicated. If cardioversion fails to terminate the arrhythmia and adverse features persist, amiodarone 300 mg IV over 10-20 minutes should be given and further cardioversion attempted. The loading dose of amiodarone can be followed by an infusion of 1 mg/mg over 24 hours via a central vein.

If the patient has no adverse features:

If the patient is stable, the QRS duration should be considered to be the target.

- If the QRS duration is ≤120 ms, it is a broad-complex tachycardia.
- If the QRS duration is >120 ms, it is a narrow-complex tachycardia.

Broad-complex tachycardia

Broad-complex tachycardias are mostly ventricular in origin but may be supraventricular rhythms with aberrant conduction.

A regular broad-complex tachycardia is likely to be ventricular tachycardia or a regular supraventricular tachycardia with bundle branch block.

A ventricular tachycardia (or broad-complex tachycardia of uncertain origin) should be treated with amiodarone 300 mg IV over 20-40 minutes, followed by an infusion of 1 mg/mg over the next 24 hours.

If paroxysms confirmed as SVT with bundle branch block, the patient should be treated as for narrow-complex tachycardia.

A stable patient with an irregular broad-complex tachycardia is most likely to be AF with bundle branch block, although AF with atrioventricular pre-excitation or polymorphic VT (torsades de pointes) is a possibility.

Expert help should be sought for the assessment and treatment of irregular broad-complex tachycardia.

Torsade de pointes VT should be treated by stopping all drugs known to prolong the QT interval, correcting electrolyte abnormalities, and giving magnesium sulfate 2 g IV over 30 minutes. Expert help should be sought on other treatment options including overdrive pacing, which may be required to prevent recurrences once the arrhythmia has been corrected.

Narrow-complex tachycardia

The narrow-complex tachycardias are supraventricular in origin.

A regular narrow-complex tachycardia may represent paroxysmal SVT or atrial flutter with 2:1 conduction, it may be difficult to differentiate as the basis.

- The first step in treatment of regular narrow-complex tachycardia is to attempt vagal manoeuvres (carotid sinus massage or Valsalva manoeuvre).
- If this fails to terminate the arrhythmia, adenosine 6 mg IV should be given as a rapid bolus using a large cannula and a large vein. The patient should be warned that they will feel unwell and may experience chest discomfort for a few seconds following the injection. An ECG (preferably multi-lead) should be recorded during the injection.
- If the ventricular rate slows transthoracically and then speed up again, this may indicate atrial activity sufficient for atrial flutter or other atrial tachycardia, and this should be treated accordingly.
- If there is no response (i.e. no transient slowing or termination of the tachycardia), or the patient remains symptomatic (e.g. adenosine 6 mg IV and a further 12 mg IV shows no response), expert help should be sought. If there is no response, one further 12 mg IV bolus given (total 36 mg) shows no response to adenosine or AV block is seen, treatment should be considered.
- Irregular narrow-complex tachycardia is most likely to be AF with fast ventricular response or, less commonly, atrial flutter with variable AV conduction.

- Immediate treatment options include rate control with drug therapy, rhythm control using drugs to achieve chemical cardioversion, rhythm control by synchronized cardioversion and treatment to prevent complications (i.e., anticoagulation). Expert help should be sought in determining the most appropriate treatment for the individual patient.
Pharmacology: Cardiovascular

Question 102 of 121

A 49 year old man is brought to ED complaining of palpitations and dyspnoea. ECG demonstrates Torsade de Pointes. Which of the following should be given immediately to this patient:

a. Adenosine 6 mg IV
b. Magnesium sulfate 2 g IV
c. Amiodarone 300 mg IV
d. Verapamil 5 mg IV
e. Atropine 500 mcg IV

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Pharmacology: Cardiovascular
Quarter 102 of 121

A 49-year-old man is brought to ED complaining of palpitations and dyspnea. ECG demonstrates Torsade de Pointes. Which of the following should be given immediately to this patient:

- Adenosine 4 mg IV
- Magnesium sulfate 2 g IV
- Amiodarone 200 mg IV
- Verapamil 5 mg IV
- Atracurium 500 mg IV

Answer
Torsade de pointes is a form of ventricular tachycardia associated with a long QT (sometimes called a ‘long QT syndrome’) interval. Other factors include hypokalemia, serum calcium and magnesium supplementation (which are also implicated). Episodes are usually self-limiting, but are frequently recurrent and can cause impairment or loss of consciousness. If controlled, the arrhythmia progresses to ventricular fibrillation and sometimes death. Intravenous injection of magnesium sulfate or 2 g IV over 30-60 seconds is usually effective. A beta-blocker (not a complete opioid antagonist) and atrial ventricular pacing can be considered. Antiarrhythmics can further prolong the QT interval, thus worsening the condition.

Notes
The approach to the management of tachycardia should follow the Resuscitation Council guidelines. If the patient has adverse features
Adverse Features:
- Shock (hypoperfusion, pтверд, sweating, cold extremities, confusion, impaired consciousness)
- Syncope (transient loss of consciousness)
- Heart failure (pulmonary edema, rales, JVD; peripheral edema, hepatomegaly)
- Myocardial ischemia (echocardiop, chest pain, ischemic changes on ECG)

If any adverse features are present, emergency cardiovascular life support and a synchronized DC shock is indicated. If the ventricular fibrillation occurs, it is terminated the immediate administration of antiarrhythmic and adverse features persist, amiodarone 300 mg IV over 10-20 minutes should be given and further cardiovascular attempts attempted. The loading dose of amiodarone is followed by an infusion of 900 mg over 24 hours in a 4-hour continuous IV.

If the patient has no adverse features
If the patient is stable, the QRS duration should be considered:
- In the QRS duration is 0.12 seconds or greater, it is a sustained tachycardia.
- In the QRS duration is less than 0.12 seconds, it is a nonsustained tachycardia.

Broad complex tachycardia
- Broad complex tachycardias are mostly ventricular in origin but may be a supraventricular tachycardia with aberrant conduction.
- A regular broad-complex tachycardia is likely to be ventricular tachycardia or regular supraventricular tachycardia with bundle branch block.
- A ventricular tachycardia for a broad complex tachycardia of uncertain origin should be treated with amiodarone 300 mg IV over 10-60 minutes, followed by an infusion of 900 mg over the next 24 hours.
- If parasystole confirmed as SVT with bundle branch block, the patient should be treated as for nonsustained tachycardia.
- A stable patient with an irregular broad-complex tachycardia is most likely to be in AF with aberrant conduction, although AV ratio ventricular and paroxysmal VT (tachycardia de pointes) is a possibility.
- Expert help should be sought for the assessment and treatment of irregular broad-complex tachycardia.
- Torsade de pointes VT should be treated by stopping all drugs known to prolong the QT interval, correcting electrolyte abnormalities, and giving magnesium sulfate 2 g IV over 10-20 minutes. Expert help should be sought as other treatment options including operative pacing may be required to prevent relapse even if the anterior wall has been corrected.

Narrow complex tachycardia
The narrow-complex tachycardias are supraventricular in origin.
- A regular narrow-complex tachycardia may represent preexcitation STV or atrial flutter with 2:1 conduction. It may be difficult to differentiate between the two.
- The first step is treatment of regular narrow complex tachycardia is to attempt vagal maneuvers (carotid sinus massage or valsalva maneuver).
- If the tachycardia persists, adenosine, a 5-10 mg IV should be given in a rapid bolus using a large bore and a large volume. The patient should be scanned while they will feel unwell and may experience chest discomfort for a few seconds following the injection. If AEDs (antidyssrrhythmics) fail to be acceptable for the injection. If the ventricular rate slows transiently and then speeds up again, this may indicate atrial flutter activity such as atrial fibrillation or other atrial tachycardia, and is this should be treated accordingly.
- If there is no response (i.e. no transient slowing or termination of the tachycardia) administration to the adenosine (5 mg IV in 15 mg IV) should be given until there is no response, one further 15 mg IV without further time to the value of the dose is given if there is no atrial fibrillation or flutter is identified.
- If tachycardia is not terminated, or fails to terminate a narrow-complex tachycardia, the administration of 0.05 mg/kg of procainamide is recommended.
- Irregular narrow-complex tachycardia is most likely to be AF with fast ventricular response or, less commonly, atrial flutter with variable atrioventricular conduction.
- Immediate treatment options include rate control with the use of drugs such as beta blockers, calcium channel blockers and adenosine. Rate control or symptomatic cardiovascular and treatment of concomitant complications (e.g., arrhythmias) are expedited. Expert help should be sought in determining the most appropriate treatment for the individual patient.
Pharmacology: Cardiovascular

Question 103 of 121

Nimodipine is used predominantly for the treatment of:

a. Termination of paroxysmal supraventricular tachycardia
b. Refractory angina
c. Prevention and treatment of vascular spasm following subarachnoid haemorrhage
d. Hypertensive emergencies
e. Termination of broad-complex tachycardia
Pharmacology: Cardiovascular
Quarter 30 of 52

Nifedipine is used predominantly for the treatment of:

- Prevention of angina pectoris
- Refractory angina
- Prevention and treatment of vasospasm following subarachnoid haemorrhage
- Hypertensive emergencies
- Termination of broad complex tachycardia

Answer

Nifedipine is related to nifedipine but the smooth muscle relaxant effects preferentially act on coronary arteries. It is used solely for the prevention and treatment of vascular spasms following subarachnoid haemorrhage.

Notes

Calcium channel blockers are widely used in the treatment of angina (second line to beta-blockers) and also for hypertension, heart failure and arrhythmias.

Calcium channel blockers vary widely in their prolongation for the various possible sites of action and in their therapeutic effects and may be divided into the dihydropyridine type (e.g. amlodipine, nifedipine and nisoldipine) and the rate-limiting non-dihydropyridine type (e.g. verapamil, diltiazem).

Mechanism of Action

Calcium channel blockers inhibit L-type voltage-sensitive calcium channels in arterial smooth muscle, causing relaxation and vasodilatation. They also block calcium channels within the myocardium and conducting tissues of the heart which produce a negative inotropic effect by reducing calcium influx during the diastolic phase of the action potential.

The dihydropyridines have relatively little effect on the heart because they have much higher affinity for intracellular channels found more frequently in vascular muscle. Furthermore, at clinical doses, vasodilatation results in a reflex increase in sympathetic tone that counteracts the mild negative inotropic effect. The non-dihydropyridine are rate-limiting calcium channel blockers that depress the sinus node and slow conduction in the atrioventricular node, causing a mild resting bradycardia.

Contraindications

Non-dihydropyridine CCBs:
- Atrial flutter or fibrillation
- Heart failure or history of heart failure (may precipitate or aggravate symptoms)
- Cardiac outflow obstruction (e.g. significant aortic stenosis or obstructive hypertrophic cardiomyopathy - b-blockade may result in reduced cardiac output)
- Second or third degree AV block (may reduce complete AV block)
- Severe bradycardia
- SICK sinus syndrome

Dihydropyridine CCBs:
- Uncontrolled heart failure
- Severe hypotension
- Cardiac outflow obstruction

Adverse effects

- Gastrointestinal adverse effects – constipation, nausea, dyspepsia
- Bradycardia, AV block, reflex tachycardia, palpitations
- Vasodilator adverse effects – flushing, dizziness, headache, postural hypotension, ankle swelling (more common with dihydropyridines calcium channel blockers and often improve with continued use, although ankle swelling often persists)
- Cough (diltiazem)
- Mobiol and fatigue
- Myalgia and arthralgia

Verapamil

Verapamil is used for the treatment of angina, hypertension and arrhythmias. Verapamil is highly negatively inotropic and reduces cardiac output, slows the heart rate and may impair atrioventricular conduction. It may precipitate heart failure, exacerbate conduction disorders, and cause hypotension at high doses and should not be used with beta-blockers. Contraindications is the most common side effect.

Nifedipine

Nifedipine relaxes vascular smooth muscle and dilates coronary and peripheral arteries. Nifedipine has less myocardial effects than verapamil and has no anti-arrhythmic properties but has more influence on the vessels. Unlike verapamil it rarely precipitates heart failure because any negative inotropic effect is offset by a reduction in left ventricular work.

Nifedipine

Nifedipine is related to nifedipine but the smooth muscle relaxant effects preferentially act on cardiac arteries. It is used solely for the prevention and treatment of vascular spasms following subarachnoid haemorrhage.

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Pharmacology: Cardiovascular

Question 104 of 121

A tachyarrhythmia is defined as broad-complex if the QRS duration is:

a. Greater than 0.16 s
b. Greater than or equal to 0.12 s
c. Greater than 0.12 s
d. Greater than 0.10 s
e. Greater than 0.2 s

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12. Answered

Something wrong?
Pharmacology: Cardiovascular

Question 18.12
A tachycardia is defined as broad complex if the QRS duration is:

a) Greater than 0.16 s
b) Greater than or equal to 0.12 s

c) Greater than 0.1 s
d) Greater than 0.2 s

e) Greater than 0.05 s

Answer
If the patient with a tachycardia is stable, the QRS duration should be considered.

- If the QRS duration is 0.12 seconds or greater, it is a broad complex tachycardia.
- If the QRS complex is less than 0.12 seconds, it is a narrow complex tachycardia.

Notes
The approach to the management of tachycardia should follow the Resuscitation Council guidelines.

If the patient has adverse features:

Adverse features:
- Shock (hypotension, pulselessness, cold extremities, confusion, impaired consciousness)
- Syncope, fainting (loss of consciousness)
- Heart failure (pleural effusions, raised JVP, peripheral oedema, heptomegaly)
- Myocardial ischaemia (ischaemic chest pain, ischaemic changes on ECG)

If any adverse features are present, emergency cardiovascular dc shock is indicated. If cardiovascular defibrillation is unsuccessful, amiodarone 300 mg iv over 10 - 20 min should be given and further cardioversion attempted. The loading dose of amiodarone can be followed by an infusion of 150 mg over 24 h, given via a large vein.

If the patient has no adverse features:

If the patient is stable, the QRS duration should be considered.

- If the QRS duration is 0.12 seconds or greater, it is a broad complex tachycardia.
- If the QRS complex is less than 0.12 seconds, it is a narrow complex tachycardia.

Broad complex tachycardia

Broad complex tachycardias are mostly ventricular in origin but may be a supraventricular rhythm with aberrant conduction.

A regular broad complex tachycardia is likely to be ventricular tachycardia or a regular supraventricular rhythm with bundle branch block.

A ventricular tachycardia (or broad complex tachycardia of uncertain origin) should be treated with standard techniques 300 mg iv over 10 - 20 min, followed by an infusion of 150 mg over the next 24 hours.

If paroxysmal confusion is 3/7 with bundle branch block, the patient should be treated as for narrow complex tachycardia.

A stable patient with an irregular broad complex tachycardia is most likely to be AV with bundle branch block, although AV with ventricular pre-excitation or paroxysmal VT (toroides de pointes) is possible. Help expert should be sought to develop an assessment and treatment of irregular broad complex tachycardia.

Torsade de pointes VT should be tried by stopping all drugs known to prolong QT interval (chronic anticonvulsant administration, and giving magnesium sulphate 1.5 g iv over 10 minutes. Expert help should be sought at other treatment options including overdrive pacing may be required to prevent tachycardia once the arrhythmia has been corrected.

Narrow complex tachycardia

The narrow-complex tachycardias are supraventricular in origin.

A regular narrow-complex tachycardia may represent paroxysmal SVT or atrial flutter with 2:1 conduction. It may be difficult to differentiate between the two.

The first step in treatment of regular narrow-complex tachycardias is to attempt vagal manoeuvres (vocal coax or swallowing manoeuvre).

If the tachycardia persists, adrenaline (1 mg iv) should be given as a rapid bolus using a large canula and a large vein. The patient should be warned that they will feel lightheaded and may experience chest discomfort for a few seconds following the injection. As ECG paroxysmal atrial flutter should be recorded during the injections.

If the ventricular rate slows transiently and then speeds up again, this may indicate atrial activity such as atrial flutter or other atrial tachycardias, and this should be treated accordingly.

If there is no response (i.e., no transient slowing or termination of the tachycardia) by administering 1 mg iv or 2 mg iv, this should be repeated and there is no response, see further 2 mg iv bolus given (total 3 mg). Lack of response to adrenaline will occur if the tachycardia is given too slowly or into a peripheral vein.

If adrenaline is contraindicated, or fails to terminate a regular narrow-complex tachycardia, the administration of verapamil (50 mg iv) over 15 - 20 sec should be considered.

Irregular narrow-complex tachycardia is most likely to be AF with fast ventricular response or, less commonly, atrial flutter with variable AV conduction.

Immediate treatment options include rate control with drug therapy, rhythm control using drugs to achieve cardiac synchronisation, rhythm control by synchronised cardioversion and treatment to prevent complications (e.g., anticoagulation). Expert help should be sought in determining the most appropriate treatment for this individual patient.
Pharmacology: Cardiovascular

What is the mechanism of action of abciximab:

- a. Inhibition of platelet thromboxane A2 synthesis
- b. Inhibition of binding of ADP to its platelet receptor
- c. Blocking the binding of fibrinogen to GPIIb/IIIa receptor sites
- d. Inhibition of the breakdown of cAMP
- e. Inhibition of thrombin-induced platelet aggregation
Pharmacology: Cardiovascular

Question 105 of 121

What is the mechanism of action of abciximab:

- a) Inhibition of platelet thromboxane A2 synthesis
- b) Inhibition of binding of ADP to its platelet receptor
- c) **Blocking the binding of fibrinogen to GPIIb/IIIa receptor sites**
- d) Inhibition of the breakdown of cAMP
- e) Inhibition of thrombin-induced platelet aggregation

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**Answer**

Glycoprotein IIb/IIIa inhibitors prevent platelet aggregation by blocking the binding of fibrinogen to receptors on platelets.

**Notes**

Glycoprotein IIb/IIIa inhibitors prevent platelet aggregation by blocking the binding of fibrinogen to receptors on platelets.

Abciximab is a monoclonal antibody which binds to glycoprotein IIb/IIIa receptors and to other related sites; it is licensed as an adjunct to unfractionated heparin and aspirin for the prevention of ischaemic complications in high-risk patients undergoing percutaneous transluminal coronary intervention. Abciximab should be used once only (to avoid additional risk of thrombocytopenia).

Eptifibatide (in combination with unfractionated heparin and aspirin) and tirofiban (in combination with unfractionated heparin, aspirin, and clopidogrel) also inhibit glycoprotein IIb/IIIa receptors; they are licensed for use to prevent early myocardial infarction in patients with unstable angina or non-ST-segment-elevation myocardial infarction.

Tirofiban is also licensed for use in combination with unfractionated heparin, aspirin, and clopidogrel, for the reduction of major cardiovascular events in patients with ST-segment elevation myocardial infarction intended for primary percutaneous coronary intervention.

Abciximab, eptifibatide and tirofiban should be used by specialists only.
Pharmacology: Cardiovascular

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Which of the following is NOT a pharmacological effect of ACE inhibitor therapy:

a  Decreased arterial resistance
b  Decreased venous resistance
c  Increased renal blood flow
d  Decreased venous return
e  Increased aldosterone release

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12  Answered

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Which of the following is NOT a pharmacological effect of ACE inhibitor therapy:

- Decreased arterial resistance
- Decreased venous resistance
- Increased renal blood flow
- Decreased glomerular filtration rate
- Increased aldosterone release

Answer

Angiotensin-converting enzyme inhibitors (ACE inhibitors), e.g., captopril inhibit the conversion of angiotensin I to angiotensin II, and thus have a vasodilatory effect, lowering both arterial and venous resistance. The cardiac output increases and, because the renal resistance falls, there is an increase in renal blood flow. This latter effect, together with reduced aldosterone release, increases Na+ and K+ reabsorption, contracting the blood volume and reducing venous return to the heart.

Notes

Mechanism of action

Angiotensin-converting enzyme inhibitors (ACE inhibitors), e.g., captopril inhibit the conversion of angiotensin I to angiotensin II, and thus have a vasodilatory effect, lowering both arterial and venous resistance. The cardiac output increases and, because the renal resistance falls, there is an increase in renal blood flow. This latter effect, together with reduced aldosterone release, increases Na+ and K+ reabsorption, contracting the blood volume and reducing venous return to the heart.

Blocking ACE also diminishes the breakdown of the potent vasodilator bradykinin which is the cause of the persistent dry cough. Angiotensin II receptor blockers do not have this effect, therefore they are useful alternative for patients who have to discontinue an ACE inhibitor because of persistent cough.

Indications

ACE inhibitors have many uses and are generally well tolerated. They are indicated for:

- Heart failure
- Hypertension
- Diabetic nephropathy
- Secondary prevention of cardiovascular events

Contraindications

ACE inhibitors should be avoided in:

- History of angioedema associated withprevious exposure to an ACE inhibitor
- Recurrent angioedema
- Bilateral renal artery stenosis
- Pregnancy and breastfeeding

Cautions

The use of ACE inhibitors is cautioned in:

- Renal impairment
- Afro-Caribbean patients (may respond less well to ACE inhibitors)
- Peripheral vascular disease or generalised athrosclerosis (risk of clinically silent renovascular disease)
- Primary aldosteronism (patients may respond less well to ACE inhibitors)
- History of hypotensive or hypertensive angioneurodesa
- Patients with hypotrophic cardiomyopathy
- Patients with severe or symptomatic aortic stenosis (risk of hypertensive

Concomitant treatment with NSAIDs increases the risk of renal damage, and with potassium-sparing diuretics (or potassium-containing salt substitutes) increases the risk of hyperkalaemia. Hyperkalaemia and other side effects of ACE inhibitors are more common in elderly and in those with impaired renal function and the dose may need to be reduced.

Adverse effects

Side effects of ACE inhibitors may include:

- Deterioration in renal function
- Hypokalaemia
- Hypotension
- Persistent dry cough
- Angioedema (non-allergic drug reaction)
- Dizziness and headaches (usually secondary to hypotenion)
- Other common adverse effects include abdominal discomfort, dyspepsia, diarrhoea, nausea and vomiting, rash (in particular macular papular rash), myalgia, muscle spasm, dyspnoea, chest pain, and fatigue

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- Resuscitation Council UK
- Troubleshooter
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Pharmacology: Cardiovascular

Question 107 of 121

ACE inhibitors are indicated for all of the following EXCEPT for:

a  Diabetic nephropathy
b  Secondary prevention of cardiovascular disease
c  Angina
d  Heart failure
e  Hypertension

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Pharmacology: Cardiovascular

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ACE inhibitors are indicated for all of the following EXCEPT for:

- Diabetic nephropathy
- Secondary prevention of cardiovascular disease
- Angina ✓
- Heart failure
- Hypertension

Answer

ACE inhibitors have many uses and are generally well tolerated. They are indicated for:

- Heart failure
- Hypertension
- Diabetic nephropathy
- Secondary prevention of cardiovascular events

Notes

Mechanism of action

Angiotensin-converting enzymes inhibitors (ACE inhibitors) e.g. captopril inhibits the conversion of angiotensin I to angiotensin II, and thus have a vasodilatory effect, lowering both arterial and venous resistance. The cardiac output increases and, because the renovascular resistance falls, there is an increase in renal blood flow. This latter effect, together with reduced aldosterone release, increases Na+ and H2O excretion, contracting the blood-volume and reducing venous return to the heart.

Blocking ACE also diminishes the breakdown of the potent vasodilator bradykinin which is the cause of the persistent dry cough. Angiotensin II receptor blockers do not have this effect, therefore they are useful alternative for patients who have to discontinue an ACE inhibitor because of persistent cough.

Indications

ACE inhibitors have many uses and are generally well tolerated. They are indicated for:

- Heart failure
- Hypertension
- Diabetic nephropathy
- Secondary prevention of cardiovascular events

Contraindications

ACE inhibitors should be avoided in:

- History of angioedema associated with previous exposure to an ACE inhibitor
- Recurrent angioedema
- Bilateral renal artery stenosis
- Pregnancy and lactation

Caution

The use of ACE inhibitors is cautioned in:

- Renal impairment
- African-Caribbean patients (may respond less well to ACE inhibitors)
- Peripheral vascular disease or generalised atherosclerosis (risk of clinically silent renovascular disease)
- Primary aldosteronism (patients may respond less well to ACE inhibitors)
- History of idiopathic or hereditary angioedema
- Patients with hypomagnesaemic cardiomyopathy
- Patients with severe or symptomatic aortic stenosis (risk of hypertension)

Concomitant treatment with NSAIDs increases the risk of renal damage, and with potassium sparing diuretics (or potassium containing salt substitute) increases the risk of hyperkalaemia. Hyperkalaemia and other side effects of ACE inhibitors are more common in the elderly and those with impaired renal function and the dose may need to be reduced.

Adverse effects

Side effects of ACE inhibitors may include:

- Deterioration in renal function
- Hyperkalaemia
- Hypotension
- Persistent dry cough
- Angioedema (from allergic drug reaction)
- Dizziness and headaches (usually secondary to hypotension)
- Other common adverse effects include abdominal discomfort, dyspepsia, diarrhoea, nausea and vomiting, rash (in particular morbilliform rash), rashes, muscle-splashes, dyspnoea, chest pain, and fatigue

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Pharmacology: Cardiovascular

Question 108 of 121

Which of the following is NOT a typical electrolyte disturbance caused by furosemide:

- [ ] a. Hypercalcaemia
- [ ] b. Hypomagnesaemia
- [ ] c. Hypokalaemia
- [ ] d. Hypochloraemia
- [ ] e. Hyponatraemia

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Pharmacology: Cardiovascular

Which of the following is NOT a typical electrolyte disturbance caused by hypertension?

a) Hyperkalaemia
b) Hypokalaemia
c) Hypernatraemia
d) Hypocholesterolaemia
e) Hypocholesterolaemia

Answer

Adverse effects of loop diuretics include:

- Mild gastrointestinal disturbances, nausea, and vomiting in less than 1% of patients
- Acute renal shutdown
- Nausea and vomiting
- Hypokalaemia, hypomagnesaemia, hypocalcaemia, and hypomagnesaemia, hypophosphataemia
- Metabolic acidosis
- Hypocalcaemia
- Blood clots (more rarely perfusion, thrombophlebitis, and leucopenia)
- Visual disturbances (atrophy and deafness)
- Hypokalaemia in patients with chronic renal failure

Notes

Loop diuretics are powerful diuretics used in adults and children with a low extracellular fluid volume. Intravenous administration results in rapid diuresis and reduces preload, thus increasing the chance of an adverse effect.

They are also used in children with chronic renal failure to reduce the risk of adverse effects. Loop diuretics can be given in conjunction with an ACE inhibitor or ARB, which can reduce the risk of adverse effects.

If necessary, loop diuretics can be used in addition to hypotensive treatment to achieve a better control of blood pressure in those with resistant hypertension, or in patients with impaired renal function or heart failure.

Mechanism of action

Loop diuretics inhibit the Na⁺-K⁺-Cl⁻ symporter on the luminal membrane in the thick ascending limb of the loop of Henle, thus reducing reabsorption of NaCl and water. This reduces reabsorption of Cl⁻ and fluid and increases urinary excretion of NaCl, Cl⁻, and H⁺.

Furosemide and bumetanide are similar in action, but only a small fraction of oral administration is absorbed through the intestinal mucosa. These are used to reduce reabsorption of Cl⁻ and fluid and increase urinary excretion of NaCl, Cl⁻, and H⁺.

Bumetanide and furosemide are similar in action, but only a small fraction of oral administration is absorbed through the intestinal mucosa. These are used to reduce reabsorption of Cl⁻ and fluid and increase urinary excretion of NaCl, Cl⁻, and H⁺.

Carotid stenosis and proarteriosclerotic changes are associated with low levels of aldosterone.

Contraindications

Loop diuretics are contraindicated in:

- Hypokalaemia and dehydration
- Severe hypocalcaemia or severe hypocalcaemia
- Acute, acute middle ear injury, or chronic kidney disease due to nephrotoxic drugs
- Coagulopathy and proarthrombosis associated with low levels of aldosterone.

Cautions

Loop diuretics can cause hyperkalaemia (but hypokalaemia is less likely than this), thus the concomitant use of loop diuretics and hypotensive drugs is recommended.

Hypertension, hyperlipidaemia, and diabetic disturbances should be corrected before initiation of treatment.

Hypertension and hyperlipidaemia may worsen the risk of adverse effects.

Lower levels of diuretics should be used in elderly patients because they are particularly susceptible to the side effects.

Adverse effects

Adverse effects of loop diuretics include:

- Mild gastrointestinal disturbances, nausea, and vomiting in less than 1% of patients
- Acute renal shutdown
- Nausea and vomiting
- Hypokalaemia, hypomagnesaemia, and hypocalcaemia
- Metabolic acidosis
- Hypocalcaemia
- Blood clots (more rarely perfusion, thrombophlebitis, and leucopenia)
- Visual disturbances (atrophy and deafness)
- Hypokalaemia in patients with chronic renal failure

Hypokalaemia

Hypokalaemia can occur with both thiazide and loop diuretics. The risk of hypokalaemia depends on the duration of action as well as the potency and is much greater with thiazide than with an equivalent dose of a loop diuretic.

Hypokalaemia is common in severe cardiovascular disease and patients being treated with calcium channel blockers. The use of potassium-sparing diuretics avoids the need to take potassium supplements. In hypokalaemia, hypokalaemic diuretics can cause precipitation of potassium, particularly in patients with cirrhosis.
Pharmacology: Cardiovascular

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Regarding calcium channel blockers, which of the following statements is CORRECT:

- **a** Nifedipine is a useful antiarrhythmic calcium channel blocker.
- **b** Verapamil is the calcium channel blocker of choice post-myocardial infarction in patients with heart failure.
- **c** Intravenous nimodipine is licensed for the treatment of acute life-threatening hypertension.
- **d** Calcium channel blockers inhibit L-type voltage-sensitive calcium channels in arterial smooth muscle causing vasodilation.
- **e** In cases of refractory hypertension, verapamil can be used in combination with a beta-blocker.

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Pharmacology: Cardiovascular

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Regarding calcium channel blockers, which of the following statements is CORRECT?

1. Nitropresers is a useful anti hypertensive calcium channel blocker.
2. Verapamil in the calcium channel blocker of choice post myocardial infarction in patients with heart failure.
3. Intravenous nitroprusside is licensed for the treatment of severe life threatening hypertension.
4. Calcium channel blockers inhibit L-type voltage sensitive calcium channels in cardiac smooth muscle causing vasodilation.
5. Calcium channel blockers inhibit L-type voltage sensitive calcium channels in cardiac smooth muscle causing vasodilation.
6. Calcium channel blockers inhibit L-type voltage sensitive calcium channels in cardiac smooth muscle causing vasodilation.

Answer

Calcium channel blockers inhibit L-type voltage sensitive calcium channels in cardiac smooth muscle, causing vasodilation and vasoconstriction. They also block calcium channels within the myocyte and conducting tissues of the heart which produce a negative inotropic effect by reducing calcium influx during the plateau phases of the action potential. Verapamil is highly receptors and reduces coronary output, slows the heart rate and may improve arterial conductance. It may precipitate heart failure, exacerbate conduction disorders, and cause hypotension at high doses and should not be used with beta blockers. Nitropresers has less myocardial effects than verapamil and has no anti hypertensive properties, but has more influence on the vessels. Nitropresers is used solely for the prevention and treatment of vascular spasm following aneurysmal subarachnoid haemorrhage.

Notes

Calcium channel blockers are widely used in the treatment of angina, bronchial asthma and for hypertension. Heart failure and arrhythmias.

Calcium channel blockers very slowly its slow for proliferation for the various possible effects of action and in their therapeutic effects and may be divided into the dihydropyridine type (e.g. amlodipine, nifedipine and nimodipine) and the non-dihydropyridine type (e.g. verapamil, diltiazem).

Mechanism of action

Calcium channel blockers inhibit L-type voltage sensitive calcium channels in cardiac smooth muscle, causing vasodilation and vasoconstriction. They also block calcium channels within the myocyte and conducting tissues of the heart which produce a negative inotropic effect by reducing calcium influx during the plateau phases of the action potential.

The dihydropyridines have relatively low effects on the heart because they have a much higher affinity for brain/blood channels found more frequently in vascular recoil. Furthermore, at clinical doses, vasoconstriction results in a net increase in sympathetic tone that counteracts the net negative inotropic effect. The non-dihydropyridines are more potent calcium channel blockers that depresses the sino-atrial node and slow conduction in the atrioventricular node causing a mild bradycardia.

Contraindications

Dihydropyridine type CCBs:
- Atrial fibrillation or flutter
- Heart failure or history of heart failure (may precipitate or aggravate symptoms)
- Carotid sinus syncope or obstruction e.g. significant aortic stenosis or obstruction hypertrophic cardiomyopathy (vasodilation may result in reduced cardiac output)
- Second or third degree AV block (may induce complete AV block)
- Severe bradycardia
- Sickle cell syndrome

Non-dihydropyridine type CCBs:
- Uncontrolled heart failure
- Severe hypertension
- Carotid sinus syncope

Adverse effects

Gastrointestinal adverse effects – constipation, nausea, dyspepsia
- Bradycardia, AV block, reflex tachycardia, palpitations
- Visual changes polymorphic effects – flushing, dizziness, headache, retroauricular pain, ankle swelling (more common with dihydropyridine calcium channel blockers and often improve with continued use, although ankle swelling often persists)
- Gingival hyperplasia
- Malaise and fatigue
- Myalgia and arthralgia

Vasoconstrictor

Verapamil is used for the treatment of angina, hypertension, and arrhythmias. Verapamil is highly sequestered in the veins and reduces coronary output, slows the heart rate and may improve arterial conductance. It may precipitate heart failure, exacerbate conduction disorders, and cause hypotension at high doses and should not be used with beta blockers. Contraindication is the worst coronary risk factor.

Nitrate

Nitration relaxes vascular smooth muscle and dilates coronary and peripheral arteries. Nitroglycerine has less myocardial effects than verapamil and has no anti hypertensive properties but has more influence on the vessels. Unlike verapamil it slowly precipitates heart failure because no negative inotropic effect is affect in the reduction in left ventricular work.

NSAID

NSAID is used solely for the prevention and treatment of vascular spasm following aneurysmal subarachnoid haemorrhage.
Pharmacology: Cardiovascular

Question 110 of 121

Regarding statins, which of the following statements is INCORRECT:

- a) Statins competitively inhibit 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase.
- b) Statins should be avoided in patients with active liver disease.
- c) Statins are more effective than fibrates at lowering triglycerides.
- d) Statins are contraindicated in pregnancy.
- e) Statins reduce cardiovascular disease events and total mortality irrespective of the initial cholesterol concentration.
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(a) Statin competitively inhibits 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase. ✗
(b) Statins should be avoided in patients with active liver disease. ✓
(c) Statins are more effective than fibrates at lowering triglycerides.✓
(d) Statins are contraindicated in pregnancy. ✗
(e) Statins reduce cardiovascular disease events and total mortality irrespective of the initial cholesterol concentration.

Answer

Statins are more effective than other lipid-lowering drugs at lowering LDL cholesterol concentration but they are less effective than the fibrates in reducing triglyceride concentration. However, statins reduce cardiovascular disease events and total mortality irrespective of the initial cholesterol concentration.

Notes

Statins may be used for primary or secondary prevention of cardiovascular disease and for treatment of primary or familial hypercholesterolemia.

Statins are more effective than other lipid-lowering drugs at lowering LDL cholesterol concentration but they are less effective than the fibrates in reducing triglyceride concentration. However, statins reduce cardiovascular disease events and total mortality irrespective of the initial cholesterol concentration.

Mechanism of action

Statins competitively inhibit 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase, an enzyme involved in cholesterol synthesis. Inhibition of HMG CoA reductase reduces free cholesterol (LDL) cholesterol levels by slowing down the production of cholesterol in the liver and increasing the liver’s ability to remove the LDL cholesterol already in the blood.

Indications

Statins should be offered to all patients, including the elderly, with cardiovascular disease such as those with coronary heart disease (including those with angina or acute myocardial infarction), or with aortic or arterial disease, including peripheral vascular disease, non-femoral thigh stroke, or transient ischaemic attacks. The use of statins should be considered in patients with a high risk of developing cardiovascular disease (primary prevention) which can be assessed using risk calculators.

Contraindications

Statins should be avoided in:

- People with active liver disease.
- People with transaminase (alanine aminotransferase or aspartate aminotransferase) levels that are three or more times the upper limit of normal.
- Pregnant or breastfeeding women (discontinue 3 months before attempting to conceive).

Cautions

Statins should be used with caution in people:

- With a history of liver disease.
- Who consume high levels of alcohol.
- With predisposing factors for skeletal myopathy such as older age (>75 years), concurrent use of an interacting drug, renal impairment, hypothyroidism, and a history of cardiovascular or peripheral vascular disease.

Adverse effects

Adverse effects of statins include:

- Headache.
- Elevated liver enzymes.
- Gastrointestinal disorders such as nausea, vomiting, flatulence, diarrhea, and abdominal pain.
- Musculoskeletal and connective tissue disorders such as myalgia, arthralgia, pain in the muscles, muscle spasms, joint swelling, back pain.
- Hypothyroidism and diabetes.
- Myopathy and rhabdomyolysis.
- Interstitial lung disease.
- Hepatotoxicity.

Muscle effects

The risk of myopathy, myalgia, and rhabdomyolysis associated with statin use is low. Although myalgia has been reported commonly in patients receiving statins, muscle toxicity is attributable to statin use in some cases.

Muscle toxicity can occur with all statins, although the likelihood increases with higher doses and in certain patients. Statins should be used with caution in patients at increased risk of muscle toxicity, including those with a personal or family history of muscular disorders, previous history of muscular toxicity, high alcohol intake, renal impairment, or hypothyroidism.

There is an increased incidence of myopathy if a statin is given with a fibrate, with lipid-lowering doses of niacin, or with salt substitutes that increase the plasma-salt concentration, such as niacin, and is associated with hypertriglyceridemia. However, co-administration of dose-reduced fibrates or rhabdomyolysis-associated muscle toxins. If muscle symptoms occur, of creatine kinase is necessary.
Pharmacology: Cardiovascular

Question 111 of 121

Digoxin is predominantly used for which of the following:

a. Rate control in paroxysmal atrial fibrillation
b. Acute treatment of new-onset fast atrial fibrillation
c. First line treatment for heart failure
d. Termination of supraventricular tachycardia associated with Wolff-Parkinson-White syndrome
e. Rate control in persistent and permanent atrial fibrillation
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Digoxin is predominantly used for which of the following:

- Rate control in supraventricular atrial fibrillation
- Maintenance of normal sinus rhythm
- First line treatment for heart failure
- Prevention of atrial fibrillation
- Rate control in supraventricular and atrial fibrillation

Answer:

Digoxin is most useful for controlling the heart rate response in patients with supraventricular atrial fibrillation and atrial flutter (Options 2 and 5). It is not first line treatment for heart failure (Option 1) as it is used in the management of chronic heart failure, and digoxin is not indicated in the prevention of atrial fibrillation (Option 3).

Notes:

Digoxin is a cardiac glycoside that increases the force of myocardial contraction (positive inotropy), and decreases the heart rate (negative chronotropy). It has a cumulative pharmacodynamic effect and requires careful monitoring. It should be used with caution in patients with renal impairment, and the dose should be reduced in patients with impaired renal function. Digoxin should not be used in patients with ventricular arrhythmias or in the presence of conduction block.

Mechanisms of action:

Inotropic effect:

Digoxin is a cardiac glycoside that increases the force of myocardial contraction by stimulating the sodium pump in the myocardial cell membrane. This leads to an increase in cardiac contractility and an increase in cardiac output.

Chronic effects:

Digoxin inhibits sodium-potassium ATPase, which results in an increase in the sodium gradient across the myocardial cell membrane. This leads to an increase in calcium influx and an increase in cardiac contractility.

Cautions:

Digoxin should be used with caution in patients with:

- Hypokalaemia (low serum potassium)
- Hyperkalaemia (high serum potassium)
- Hypothyroidism (low thyroid function)
- Hypomagnesaemia (low magnesium levels)
- Recent myocardial infarction
- Severe respiratory disease
- Kidney disease
- Pancreatitis
- Conduction abnormalities
- Seizures
- Long-term treatment with diuretics and/or mineralocorticoids
- Diabetes mellitus

Adverse effects:

The adverse effects of digoxin are frequent and include:

- Cardiac arrhythmias:
  - Supraventricular tachycardia
  - AV block
  - Bundle branch block
- Nausea, vomiting, and anorexia
- Dizziness, light-headedness, and syncope
- Constipation
- Hypothyroidism

Doses reactive to

The reaction of digoxin is dose-dependent and occurs in patients with a plasma concentration of digoxin above the therapeutic range. The therapeutic range is typically 1-2 ng/mL. Patients with a plasma concentration of digoxin above the therapeutic range may experience toxicity.

Digoxin-specific antibody fragments (digoxin antidote) are available for urgent administration in patients with digoxin toxicity. These agents are administered intravenously and can reduce the plasma concentration of digoxin to a safe level within hours.

Resources:

- NICE guideline on sodium channel blockers
- Digoxin (Lanoxin) Prescribing Information
- Digoxin (Lanoxin) Patient Information
- Advanced Life Support Group
- National Institute for Health and Care Excellence
- National Health Service
- Royal College of Physicians
- Royal College of Physicians
Pharmacology: Cardiovascular

Question 112 of 121

Loop diuretics act primarily at which of the following sites in the nephron:

a) Proximal tubule
b) Thick ascending limb
c) Collecting ducts
d) Thin ascending limb
e) Descending limb

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Pharmacology: Cardiovascular

Question 1 of 1

Loop diuretics act primarily at which of the following sites in the nephron?
- Proximal tubule
- Thick ascending limb
- Collecting ducts
- Descending limb

Answer

Loop diuretics inhibit the Na+K+2Cl− cotransporter on the luminal membrane in the thick ascending limb of the loop of Henle, thereby preventing reabsorption of NaCl and water.

Notes

Indications

Loop diuretics are powerful diuretics used in severe pulmonary edema due to left ventricular failure. Intravenous administration produces rapid diuresis and reduces preload sooner than would be expected from the time of onset of diuresis. They are also used to reduce edema in patients with chronic heart failure. Diuretic-resistant edema can be treated with a loop diuretic combined with a thiazide or related diuretic.

If necessary, a loop diuretic can be added to antihypertensive treatment to achieve better control of blood pressure in those with resistant hypertension, or in patients with impaired renal function or heart failure.

Mechanism of action

Loop diuretics inhibit the Na+K+2Cl− cotransporter on the luminal membrane in the thick ascending limb of the loop of Henle, thereby preventing reabsorption of NaCl and water. These agents reduce reabsorption of O2 and NaCl and increase CO2 excretion and lose K+ and Mg2+.

Furosemide and bumetanide are similar in activity, both act within 1 hour of oral administration and diuresis is complete within 6 hours so that, if necessary, they can be given once to one day without interfering with sleep. Following intravenous administration furosemide has a peak effect within 30 minutes. The diuresis associated with these drugs is dose-related.

Contraindications

Loop diuretics are contraindicated in:
- Hypokalemia and dehydration
- Severe hypoproteinemia or severe hypoalbuminemia
- Anuria, acute kidney injury or chronic kidney disease due to nephrotic syndrome
- Congestive heart failure and liver cirrhosis

Cautions

Loop diuretics can exacerbate diabetes mellitus (but hypoglycemia is less likely than with thiazides) and gout.

If there is an enlarged prostate, urinary retention can occur, although this is less likely if small doses and less potent diuretics are used initially.

Hypotension, hypokalemia and electrolyte disturbances should be corrected before initiation of treatment.

Hypotensive syndrome, hypokalemia and electrolyte disturbances may reduce diuretic effect and increase risk of side-effects.

Lower initial doses of diuretics should be used in the elderly because they are particularly susceptible to these side-effects.

Adverse effects

Adverse effects of loop diuretics include:
- Mild gastrointestinal disturbances, pancreatitis, and hepatic encephalopathy
- Hypokalemia
- Acute urinary retention
- Water and electrolyte imbalance
- Hypokalemia, hypochloremia, hypomagnesemia, hypocalcemia
- Hypotension, hypokalemia, dehydration, and venous thromboembolism
- Metabolic acidosis
- Hypoalbuminemia
- Blood disorders (bone marrow suppression, thrombocytopenia, and leukopenia)
- Visual disturbance, tinnitus, and deafness
- Hypersensitivity reactions

Hypokalemia

Hypokalemia can occur with both thiazide and loop diuretics. The risk of hypokalemia depends on the duration of action as well as the potency and to an extent greater than with an equipotent dose of an loop diuretic.

Hypokalemia is dangerous in severe cardiovascular disease and in patients also being treated with cardiac glycosides. Often the use of potassium-sparing diuretics avoids the need to take potassium supplements. In hepatic failure, hypokalemia caused by diuretics can precipitate encephalopathy, particularly in postpartum, cirrhosis.

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Pharmacology: Cardiovascular

Question 113 of 121

What is the main mechanism of action of vasoconstrictor sympathomimetics:

- Alpha-receptor agonist
- Dopamine receptor agonist
- Beta1-receptor agonist
- Alpha-receptor antagonist
- Beta2-receptor agonist

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What is the main mechanism of action of vasoconstrictor sympathomimetics:

a) Alpha-receptor agonist ✓
b) Dopamine receptor agonist
c) Beta-1-receptor agonist
d) Alpha-receptor antagonist ✗
e) Beta-2-receptor agonist

**Answer**

Vasoconstrictor sympathomimetics, such as ephedrine and metaraminol, raise blood pressure transiently by acting on alpha-adrenergic receptors to constrict peripheral vessels. They are sometimes used as an emergency method of elevating blood pressure where other measures have failed. Their use is limited as although they raise the blood pressure they also reduce organ perfusion.

**Notes**

Shock is a medical emergency associated with a high mortality. The underlying causes of shock such as haemorrhage, sepsis, or myocardial insufficiency should be corrected. The profound hypotension of shock must be treated promptly to prevent tissue hypoxia and organ failure. Volume replacement is essential to correct the hypovolaemia associated with haemorrhage and sepsis but may be detrimental in cardiogenic shock.

**Inotrope sympathomimetics**

Depending on haemodynamic status, cardiac output may be improved by the use of sympathomimetic isotopes such as adrenaline/salbutamol, dobutamine or dopamine.

In septic shock, when fluid replacement and inotropic support fail to maintain blood pressure, the vasoconstrictor noradrenaline/norepinephrine may be considered. In cardiogenic shock peripheral resistance is frequently high and to raise it further may worsen myocardial performance and exacerbate tissue ischaemia.

- Dobutamine directly stimulates the beta1-adrenergic receptors in the heart and increases contractility and cardiac output with little effect on the rate. In addition action on beta2-receptors causes vasodilation.
- Dopamine is a neurotransmitter and a metabolic precursor of the catecholamines. It acts on beta1-receptors in cardiac muscle increasing cardiac contractility, and increases renal perfusion by stimulating dopamine receptors in the renal vasculature. This is of benefit in cardiogenic shock where deterioration of renal function is common.
- Epinephrine increases blood pressure by stimulating the rate and force of the heartbeat (beta1-effects). Stimulation of vascular alpha-receptors causes vasoconstriction (viscera, skin) but beta-2 receptor stimulation causes vasodilation (skeletal muscle) and the total peripheral resistance may actually decrease.
- Norepinephrine has little or no effect on the vascular beta2-receptors, and so the alpha-mediated vasoconstriction is unopposed. The resulting rise in blood pressure reflexively slows the heart.

The use of sympathomimetic isotopes and vasoconstrictors should preferably be confined to the intensive care setting and undertaken with invasive haemodynamic monitoring.

**Vasoconstrictor sympathomimetics**

Vasoconstrictor sympathomimetics, such as ephedrine and metaraminol, raise blood pressure transiently by acting on alpha-adrenergic receptors to constrict peripheral vessels. They are sometimes used as an emergency method of elevating blood pressure where other measures have failed. Their use is limited as although they raise the blood pressure they also reduce organ perfusion.
Pharmacology: Cardiovascular

Question 114 of 121

What is the recommended dosing regime for adenosine in the treatment of a stable regular narrow-complex tachycardia:

a) Adenosine 6 mg IV bolus, followed by a maximum of two further 6 mg boluses if no response

b) Adenosine 12 mg IV bolus, followed by one further 12 mg bolus if required

c) Adenosine 12 mg IV bolus followed by a maximum of two further 12 mg boluses if no response

d) Adenosine 6 mg IV bolus, followed by a 12 mg bolus and one further 12 mg bolus if required

e) Adenosine 12 mg IV bolus, followed by 6 mg bolus if no response
Pharmacology: Cardiovascular

What is the recommended dosing regime for adenosine in the treatment of a stable regular narrow complex tachycardia?

a) Adenosine 4 mg IV bolus, followed by a maximum of two further 6 mg boluses if no response

b) Adenosine 12 mg IV bolus, followed by one further 12 mg bolus if required

c) Adenosine 12 mg IV bolus, followed by a maximum of two 12 mg boluses if no response

d) Adenosine 4 mg IV bolus, followed by a 12 mg bolus and one further 12 mg bolus if required

e) Adenosine 12 mg IV bolus, followed by 6 mg bolus if no response

Answer

The first step in treatment of regular narrow complex tachycardia is attempted vagal manoeuvre (Valsalva or carotid sinus massage). If the tachycardia persists, adenosine 4 mg IV should be given as a rapid bolus using large cannula and a large vein. If there is no response (i.e. no transfer slowing or termination of the tachycardia), adenosine 4 mg IV bolus should be given and if there is no response, one further 12 mg IV bolus given (max 20 mg).

Notes

The approach to the management of tachycardias should follow the Resuscitation Council guidelines.

If the patient has adverse features

Adverse features:

- Shock (hypotension, pale, sweating, cold extremities, confusion, impaired consciousness)
- Syncope (transient loss of consciousness)
- Heart failure (pulmonary oedema, raised JVP, peripheral oedema, hepatomegaly)
- Haemodynamic decompensation (haemodynamic collapse, hypotension, bradycardia)

If adenosine adverse features are present, emergency cardioversion with a synchronized DC shock is indicated. If cardioversion fails to terminate the arrhythmia and adverse features persist, adenosine 300 mg IV over 10 - 20 mins should be given and further cardioversion attempted. The loading dose of amiodarone can be followed by an infusion of 900 mg over 24h, given via a large vein.

If the patient has no adverse features

If the patient is stable, the QRS duration should be considered.

- If the QRS duration is 0.12 seconds or greater, it is a broad complex tachycardia.
- If the QRS complex is less than 0.12 seconds, it is a narrow complex tachycardia.

Broad complex tachycardia

Broad complex tachycardias are mostly Ventricular in origin but may be supraventricular in rhythm with aberrant conduction.

- A regular broad complex tachycardia is likely to be ventricular tachycardia or a regular supraventricular tachycardia with aberrant conduction.

- A ventricular tachycardia (or broad complex tachycardia of uncertain origin) should be treated with adenosine 300 mg IV over 20 - 40 mins, followed by an infusion of 900 mg over the next 24h.

- If it is confirmed as IV with bundle branch block, the patient should be treated as for narrow complex tachycardia.

- A stable patient with an irregular broad complex tachycardia is most likely to be in AF with bundle-branch block, although with AV pre-excitation or pre-excitation of PR ( Karnofsky's dilemma) it is possible.

- Expert help should be sought for the assessment and treatment of irregular broad complex tachycardia.

- Termination of the attack should be treated by stopping all drugs known to prolong the QT interval, correcting electrolyte abnormalities, and giving magnesium sulfate 2 g IV over 10 mins. Expert help should be sought on other treatment options including overdrive pacing which should be prevented if not relieved and the arrhythmia has been corrected.

Narrow complex tachycardia

The narrow complex tachycardias are supraventricular in origin.

- A regular narrow complex tachycardia may represent paroxysmal SVT or atrial flutter with 2:1 conduction, it may be differentiable from SVT by the following two features:

  - The first step in treatment of regular narrow complex tachycardias is to attempt vagal manoeuvre (Valsalva or carotid sinus massage).

  - If this is not successful, adenosine 4 mg IV should be given as a rapid bolus using large cannula and a large vein. The patient should be warned that they will feel unwell and may experience chest discomfort for 15 seconds following the injection. An ECG (preferably multilead) should be recorded during the injection.

  - If the ventricular rate slows transiently and then returns again this may indicate atrial activity such as atrial flutter or other atrial arrhythmia, and this should be treated accordingly.

  - If there is no response (i.e. no slowing or termination of the tachycardia) adenosine 12 mg IV bolus should be given and if there is no response, one further 12 mg IV bolus given (max 30 mg). Lack of response to adenosine will cause an IV pigtail to be given slowly into an peripheral vein.

  - If adenosine is not contraindicated, then it is reasonable to terminate a rapid narrow complex tachycardia, the administration of verapamil 0.5 - 1 mg (over 2 - 3 mins) should be considered.

  - Irregular narrow complex tachycardia is most likely to be LA with fast ventricular response or, less commonly, atrial flutter with variable AV conduction.

  - Immediate treatment options include rate control with direct current therapy, rhythm control using drugs to achieve electrical stabilization, rhythm control by synchronized cardioversion and treatment to prevent complications (e.g. anticoagulation). Expert help should be sought in determining the most appropriate treatment for the individual patient.

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Resources

- Welcome to FRCEM Success - Primary care and emergency care preparation.
- Tony C: C&CD's Contact

FRCEM Success

- Advanced reperfusion Group
- Empathy Matters
- Building a shared lens
- Not for Nothing
- Reconsideration

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Pharmacology: Cardiovascular

Question 115 of 121

Which of the following drug classes is used first line in the management of hypertensive episodes in pheochromocytoma:

- [ ] a. Beta-blockers
- [ ] b. Alpha-blockers
- [ ] c. Calcium channel blockers
- [ ] d. ACE inhibitors
- [ ] e. Thiazide diuretics

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Question 115 of 121

Which of the following drug classes is used first line in the management of hypertensive episodes in pheochromocytoma:

- a) Beta-blockers
- b) Alpha-blockers ✓
- c) Calcium channel blockers
- d) ACE inhibitors
- e) Thiazide diuretics

Answer

Long term management of pheochromocytoma involves surgery. However surgery cannot take place until there is adequate blockade of both alpha- and beta- adrenoceptors. Alpha-blockers are used in the short-term management of hypertensive episodes in pheochromocytoma. Once alpha blockade is established, tachycardia can be controlled by the cautious addition of a beta-blocker; a cardioselective beta-blocker is preferred.

Phenoxybenzamine hydrochloride, a powerful alpha-blocker, is effective in the management of phaeochromocytoma but it has many side-effects. Phentolamine mesilate is a short-acting alpha-blocker used mainly during surgery of phaeochromocytoma; its use for the diagnosis of phaeochromocytoma has been superseded by measurement of catecholamines in blood and urine.
Pharmacology: Cardiovascular

Question 116 of 121

What is the mechanism of aspirin as an anti-platelet:

a) Inhibition of platelet thromboxane A2 synthesis
b) Inhibition of binding of ADP to its platelet receptor
c) Inhibition of GPIIb/IIIa receptor sites
d) Inhibition of the breakdown of cAMP
e) Inhibition of thrombin-induced platelet aggregation

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Question 13 of 13

What is the mechanism of aspirin as an anti-platelet:

a) Inhibition of platelet thromboxane A2 synthesis
b) Inhibition of binding of ADP to its platelet receptor
c) Inhibition of GPIb/IIa receptor sites
d) Inhibition of the breakdown of t-PA

Answer

Aspirin irreversibly inhibits cyclo-oxygenase and blocks the platelet production of thromboxane A2 (TXA2), thus inhibiting platelet aggregation.

Notes

Antiplatelet drugs decrease platelet aggregation and inhibit thrombus formation in the arterial circulation, because in fast-moving vessels, thrombi are composed mainly of platelets with little fibrin.

Mechanism of action

Aspirin irreversibly inhibits cyclo-oxygenase and blocks the platelet production of thromboxane A2 (TXA2), a powerful inducer of platelet aggregation. The endothelial cells of the vascular wall produce a prostaglandin, prostacyclin (PGI2), which is a physiological antagonist of TXA2, causing inhibition of platelet aggregation. Platelets cannot synthesise new enzyme but the vascular endothelial cells can, and the balance is shifted to the anti-aggregatory effects of PGI2.

Indications

Low-dose aspirin may be indicated in:

- Primary prevention of cardiovascular events in some people when the risk is particularly high
- Secondary prevention of cardiovascular events in people with:
  - Angina
  - Microwave infarction
  - Stroke and transient ischaemic attack
  - Peripheral arterial disease
  - Atrial fibrillation (although anticoagulants are usually used)

Contraindications

Low-dose aspirin should be avoided in:

- People with a history of true hypersensitivity to aspirin or salicylates (symptoms of hypersensitivity to aspirin or salicylates include bronchospasm, urticaria, angioedema, and vasomotor rhinitis)
- People with active pathological bleeding, such as peptic ulcer or intracranial haemorrhage
- People with suspected stroke, until intracranial haemorrhage has been excluded by brain imaging
- People with haemorrhagic or another haemorrhagic disorder (including thrombocytopenia)
- Children younger than 16 years of age

Cautions

Low-dose aspirin should be used with caution in:

- People who may be at high risk of increased bleeding — for example those receiving treatment with warfarin, NSAIDs, corticosteroids, or other drugs known to increase bleeding
- People with asthma (may precipitate bronchospasm)
- People with uncontrolled blood pressure
- If there is primary prevention of cardiovascular events does not initiate aspirin until blood pressure is less than 150/90 mmHg
- For secondary prevention, benefits of antiplatelet treatment outweigh risks, and treatment should not be delayed while controlling blood pressure

Adverse effects

Low-dose aspirin may result in:

- Increased absolute risk of major bleeding, major gastrointestinal bleeding, and intracranial bleeding
- Gastrointestinal adverse effects including bleeding, ulceration, and dyspepsia
- Bronchospasm and asthma attacks in patients with asthma

Interactions

The risk of bleeding is increased when low-dose aspirin is combined with other drugs that can increase the risk of bleeding such as other antiplatelet drugs, NSAIDs, oral and parenteral anticoagulants, selective serotonin reuptake inhibitors (SSRIs) and corticosteroids. Consider the need for gastroprotection with a proton pump inhibitor (such as omeprazole) or a histamine antagonist (such as ranitidine).
Pharmacology: Cardiovascular

Question 117 of 121

Statins are contraindicated in which of the following:

a) Active liver disease
b) Asthma
c) Renal artery stenosis
d) Chronic kidney disease
e) Recurrent angioedema

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- Statins are contraindicated in which of the following:
  - a) Active liver disease
  - b) Atherosclerosis
  - c) Renal artery stenosis
  - d) Chronic kidney disease
  - e) Recurrent angina

Answer

- Students should be advised to:
  - People with active liver disease
  - People with transaminase (alanine aminotransferase or aspartate aminotransferase) levels that are three or more times the upper limit of normal
  - Pregnancy or breastfeeding women (discontinue 3 months before attempting to conceive)

Notes

- Statins may be used for primary or secondary prevention of cardiovascular disease and for treatment of primary or familial hypercholesterolemia.
- Statins are more effective than other lipid-lowering drugs at lowering LDL cholesterol concentration but are less effective than the fibrates in reducing triglyceride concentration. However, statins modestly reduce cardiovascular disease events and total mortality irrespective of the initial cholesterol concentration.

Mechanism of action

- Statins competitively inhibit 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase, an enzyme involved in cholesterol synthesis. Inhibition of HMG CoA reductase reduces low-density lipoprotein (LDL) cholesterol levels by slowing the production of cholesterol in the liver and increasing the liver’s ability to remove the LDL cholesterol already in the blood.

Indications

- Statins should be offered to all patients, including the elderly, with cardiovascular disease such as those with coronary artery disease (including history of angina or acute myocardial infarction) or other cardiovascular disease (including peripheral-vascular disease, new myocardial infarct, or transient ischemic attack). The use of statins should be considered in patients with a high risk of developing cardiovascular disease (primary prevention) which can be assessed using risk calculators.

Contraindications

- People with active liver disease
- People with transaminase (alanine aminotransferase or aspartate aminotransferase) levels that are three or more times the upper limit of normal
- Pregnancy or breastfeeding women (discontinue 3 months before attempting to conceive)

Cautions

- Statins should be used with caution in people:
  - With a history of liver disease
  - Who consume high levels of alcohol
  - With predisposing factors for myopathy such as older age (>70 years), concomitant use with an interacting drug, renal impairment, hypothyroidism, and personal or familial history of hereditary muscular-dystrophy

Adverse effects

Adverse effects of statins include:
- Headache
- Fatigue
- Gastrointestinal disorders (such as constipation, flatulence, dyspepsia, nausea, and diarrhea)
- Musculoskeletal and connective tissue disorders (such as myalgia, arthralgia, pain in the extremities, muscle spasm, joint swelling, and back pain)
- Hypersensitivity and diabetes
- Myopathy and rhabdomyolysis
- Intestinal infarction disorders
- Impaired fertility

Muscle effects

The risk of myopathy, myalgia, and rhabdomyolysis associated with statins is rare. Although rhabdomyolysis has been reported commonly in patients receiving statins, muscle toxicity truly attributable to statins is rare.

Muscle toxicity can occur with all statins, however the likelihood increases with higher doses and in certain patients. Statins should be used with caution in patients at increased risk of muscle toxicity, including those with a personal or family history of muscular disorders, previous history of muscle toxicity, a high alcohol intake, renal impairment or myopathy.

There is an increased incidence of myopathy in statins is given with a fumaride, with lipid-lowering doses of statins, with calcium, and with drugs that increase the plasma statin concentration, such as niacin, hmgc-cr isolated from higher plants, some herbal therapies, and celiac disease; close monitoring of liver function and, if muscular symptoms occur, cessation of the statin is necessary.
Pharmacology: Cardiovascular

Question 118 of 121

Regarding loop diuretics, which of the following statements is INCORRECT:

- In hepatic failure, hypokalaemia caused by diuretics can precipitate encephalopathy.
- Oral bumetanide acts within 1 hour and diuresis is complete within 6 hours.
- Intravenous furosemide has a peak effect within 30 minutes.
- Lower initial doses of diuretics should be used in the elderly because they are particularly susceptible to the side effects.
- The risk of hypokalaemia is greater with loop diuretics than with an equipotent dose of a thiazide diuretic.
Pharmacology: Cardiovascular

Question 12 of 40

Regarding loop diuretics, which of the following statements is INCORRECT?

A) In hepatic failure, hypokalemia occurs due to diuretics can precipitate metabolic acidosis.
B) Other important factors are within 1 hour and diuretics are complete within 6 hours.
C) Infrequent dosing is a risk factor for hypokalemia.
D) Lower initial doses of diuretics should be used in elderly because they are particularly susceptible to side effects.
E) The risk of hypokalemia is greater with loop diuretics than with an equivalent dose of thiazide diuretics.

Answer

Hypokalemia can occur with both thiazide and loop diuretics. The risk of hypokalemia depends on the duration of action as well as the potency of the drug. It is greater with thiazide than with an equivalent dose of a loop diuretic.

Hypokalemia is a danger in severe cardiovascular disease and is patients also being treated with cardiac glycosides. Often the use of potassium-sparing diuretics and the need to take potassium supplements. In hepatic failure, hypokalemia causes diuretics can precipitate metabolic acidosis, particularly in alkalotic conditions.

Notes

Indications

Loop diuretics are powerful diuretics used to treat acute pulmonary edema due to left ventricular failure. Intravenous administration produces rapid relief of breathlessness and reduces arterial pressure sooner than expected from the time of onset of diuretics.

They are also used in edema in patients with chronic heart failure. Diuretic-resistant edema can also be treated with a loop diuretic combined with a thiazide or related diuretic.

Furosemide, a loop diuretic, can also be added to an antihypertensive treatment to achieve better control of blood pressure in those with resistant hypertension, or to loprazolam with limited renal function or heart failure.

Mechanism of action

Loop diuretics inhibit the Na+–K+–2Cl− symporter on the luminal membrane in the thick ascending limb of the loop of Henle, thus preventing reabsorption of NaCl and water. These agents reduce a reabsorption of Cl− and Na+ in the collecting ducts resulting in loss of Na+ and Cl−.

Furosemide and bumetanide are identical in activity. Both act within 1 hour of oral administration and diuretics is complete within 2 hours or less. It is necessary to be given more slowly in the elderly and in patients with impaired renal function or heart failure.

Contraindications

Loop diuretics are contraindicated in:

- Hypersensitivity and allergy
- Severe hypovolemia or severe hypotension
- Anuria, acute kidney injury or chronic kidney disease due to nephrotic or nephritic syndrome
- Concomitant and concomitant status associated with elderly or children

Caution

Loop diuretics can exacerbate diabetes (hyperglycemia is less likely than with thiazides) and glucose intolerance. If there is an uncontrolled proteinuria, urinary retention can occur, although it is less likely if the dose and length of treatment are used initially.

Hypokalemia, hypomagnesemia and electrolyte imbalance should be corrected before initiation of treatment.

Hypokalemic hypomagnesemia may reduce diuretic effect and increase risk of side effects.

Lower initial doses of diuretics should be used in the elderly because they are particularly susceptible to side effects.

Adverse effects

Adverse effects of loop diuretics include:

- Mitral or aortic valve disturbance, paroxysmal and hemoptysis, pneumonitis
- Hypocalcemia
- Acute pulmonary edema
- Water and electrolyte imbalance
- Hypocalcemia, hypomagnesemia, hypophosphatemia, hypokalemia
- Hypokalemia, hypomagnesemia, dehydration, and reduction in intravascular volume
- Metabolic acidosis
- Hypoglycemia
- Blood disorders (rare), marrow suppression, thrombocytopenia, and leucopenia
- Visual disorders (rare), blindness and deafness
- Hyperuricemia reactions

Hypokalemia

Hypokalemia can occur with both thiazide and loop diuretics. The risk of hypokalemia depends on the duration of action as well as the potency of the drug and is greater with thiazide than with an equivalent dose of loop diuretic.

Hypokalemia is a danger in severe cardiovascular disease and is patients also being treated with cardiac glycosides. Often the use of potassium-sparing diuretics and the need to take potassium supplements. In hepatic failure, hypokalemia causes diuretics can precipitate metabolic acidosis, particularly in alkalotic conditions.

Next >>
Pharmacology: Cardiovascular

All of the following are indications for beta-blockers EXCEPT for:

- a. Anxiety
- b. Pheochromocytoma
- c. Prophylaxis of migraine
- d. Raynaud’s disease
- e. Hypertension

Question Navigator

1. Answered
2. Answered
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7. Answered
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12. Answered

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**Answer**

- Angina
- Prophylaxis of migraine
- Hypertension
- Anxiety

**Notes**

- Beta blockers block the beta receptors in the heart, peripheral vasculature, bronchi, and coronary arteries.

**Therapeutic uses**

- Reduction of blood pressure
  - Reduce blood pressure
  - Reduce heart rate, tachycardia, and angina

- Coronary angina
  - Reduce angina
  - Bi-continuous therapy

- Angina

- Prophylaxis of migraine
  - Essential hypertensive

**Contraindications**

- Beta blockers are contraindicated in the following cases:
  - Allergy or intolerance to any component in the medication
  - History of severe arrhythmias
  - Known allergy to beta blockers
  - Known ischemia in the heart
  - Known hypersensitivity or exfoliative dermatitis

- Beta blockers should be used with caution in patients:
  - Heart failure with reduced ejection fraction (HFrEF), hypertension, or heart disease, or less severe forms of peripheral arterial disease
  - Increased pressure
  - Carotid and other peripheral vascular disease (abrupt development of new or worsened carotid stenosis)

- Beta blockers should not be used in patients:
  - Those with a history of heart failure or reduced ejection fraction
  - Those with a history of arrhythmias

**Adverse effects**

- Dizziness, nausea, vomiting, diarrhea, and constipation

- Hypothyroidism

- Electrolyte and water imbalances

- Nausea, vomiting, diarrhea, and constipation

- Sexual dysfunction and impotence

- Fever, chills, and vomiting

- Fatigue, weakness, and muscle weakness

- Sleep disturbances, anxiety, and depression

- Bronchospasm

- thirst

- Decreased blood pressure (dizziness, syncope, palpitations, anxiety, vertigo, faintness, nausea, faintness, naus
Pharmacology: Cardiovascular

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In adult advanced life support, what is the correct initial dose of amiodarone for a shockable rhythm:

a) 500 mcg
b) 50 mg
c) 200 mg
d) 300 mg
e) 400 mg
Pharmacology: Cardiovascular

Question 120 of 121.

In advanced life support, what is the correct initial dose of amiodarone for a shockable rhythm:

- 500 mcg
- 50 mcg
- 200 mg
- 300 mg ✔
- 400 mg

Answer

IV amiodarone 300 mg should be given after 3 shocks. A further dose of IV amiodarone 150 mg may be considered after a total of five defibrillation attempts. Lidocaine (1 mg/kg<sup>+</sup> 1-1<sup>+</sup>) may be used as an alternative if amiodarone is not available but may not be given if amiodarone has been given already.

Notes

**Basic life support**

- For chest compressions in adult basic life support the heel of one hand should be placed over the middle of the lower half of the patient’s sternum with the other hand on top.
- The sternum should be depressed to a depth of 5 – 6 cm.
- Chest compressions should be performed at a rate of 100 – 120 per minute.
- Thirty compressions should be given before two breaths and that ratio continued (30:2).
- The person giving CPR should be changed every 1 minutes if possible to avoid exhaustion and poor quality chest compressions.
- Intubations should be minimised (pauses should be < 5 seconds).

**Advanced life support**

- In adult advanced life support, basic life support should continue whilst the defibrillator paddles are attached and the airway managed.
- The paddles should be positioned one to the right of the upper sternum below the clavicle and the other in the left midaxillary line in the 5th intercostal space.
- Chest compressions should be paused briefly (< 5 sec) to assess the rhythm and then continued as the defibrillator charges if a shockable rhythm is identified or continued for a further two whole minutes if a non-shockable rhythm is identified.
- When delivering a shock, everybody should be clear of the patient (and any GTN patches and oxygen sources removed).
- Once the shock has been delivered, compressions should resume immediately and continue for a further two minutes before checking the rhythm again or feeling for a pulse.

**Shockable rhythm (ventricular fibrillation or pulseless ventricular tachycardia)**

- IV adrenaline 1 mg (10 ml of 1:10,000 solution) should be given after 3 shocks and every 3 – 5 minutes/after alternate shocks thereafter.
- IV amiodarone 300 mg should be given after 3 shocks. A further dose of IV amiodarone 150 mg may be considered after a total of five defibrillation attempts. Lidocaine (1 mg/kg<sup>+</sup> 1-1<sup>+</sup>) may be used as an alternative if amiodarone is not available but may not be given if amiodarone has been given already.

**Non-shockable rhythm (asystole or pulseless electrical activity)**

- IV adrenaline 1 mg should be given as soon as IV access is achieved and then given every 3 – 5 minutes/after alternate shocks thereafter.
Pharmacology: Cardiovascular

What is the main mechanism of action of dobutamine as an inotropic sympathomimetic:

- a. Dopamine receptor agonist
- b. Beta1-receptor agonist
- c. Beta2-receptor agonist
- d. Alpha1-receptor agonist
- e. Alpha2-receptor agonist

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Question 121 of 121

What is the main mechanism of action of dobutamine as an inotropic sympathomimetic?

- a) Dopamine receptor agonist
- b) Beta-1 receptor agonist
- c) Beta-2 receptor agonist
- d) Alpha-1 receptor agonist
- e) Alpha-2 receptor agonist

**Answer**

Dobutamine directly stimulates the beta-1 adrenergic receptors in the heart and increases contractility and cardiac output with little effect on the rate. In addition action on beta-2 receptors causes vasodilation.

**Notes**

Shock is a medical emergency associated with a high mortality. The underlying causes of shock such as haemorrhage, sepsis, or myocardial insufficiency should be corrected. The profound hypotension of shock must be treated promptly to prevent tissue hypoxia and organ failure. Volume replacement is essential to correct the hypovolaemia associated with haemorrhage and sepsis but may be detrimental in cardiogenic shock.

**Inotropic sympathomimetics**

Depending on haemodynamic status, cardiac output may be improved by the use of sympathomimetic inotropes such as adrenaline/epinephrine, dobutamine or dopamine.

In septic shock, when fluid replacement and inotropic support fail to maintain blood pressure, the vasoconstrictor noradrenaline/norepinephrine may be considered. In cardiogenic shock peripheral resistance is frequently high and to raise it further may worsen myocardial performance and exacerbate tissue ischaemia.

- Dobutamine directly stimulates the beta-1 adrenergic receptors in the heart and increases contractility and cardiac output with little effect on the rate. In addition action on beta-2 receptors causes vasodilation.
- Dopamine is a neurotransmitter and a metabolic precursor of the catecholamines. It acts on beta-1 receptors in cardiac muscle increasing cardiac contractility, and increases renal perfusion by stimulating dopamine receptors in the renal vasculature. This is of benefit in cardiogenic shock where deterioration of renal function is common.
- Epinephrine increases blood pressure by stimulating the rate and force of the heartbeat (beta-1 effects). Stimulation of vascular alpha-receptors causes vasoconstriction (viscera, skin) but beta-2 receptor stimulation causes vasodilation (skeletal muscle) and the total peripheral resistance may actually decrease.
- Norepinephrine has little or no effect on the vascular beta-2 receptors, and so the alpha-mediated vasoconstriction is unopposed. The resulting rise in blood pressure reflexively slows the heart.

The use of sympathomimetic inotropes and vasoconstrictors should preferably be confined to the intensive care setting and undertaken with invasive haemodynamic monitoring.

**Vasoconstrictor sympathomimetics**

Vasoconstrictor sympathomimetics, such as ephedrine and metaraminol, raise blood pressure transiently by acting on alpha-adrenergic receptors to constrict peripheral vessels. They are sometimes used as an emergency method of elevating blood pressure where other measures have failed. Their use is limited as although they raise the blood pressure they also reduce organ perfusion.