

CVS Drugs

[CD01](#) [Mar96] [Mar98] [Mar99] [Jul01] [Milrinone](#):

- A. Decreases pulmonary vascular resistance
- B. Increases systemic vascular resistance
- C. Is poorly absorbed when given orally - causes ↑ed mortality
- D. Chronic use causes thrombocytopenia

Alt version: Milrinone causes:

- A. Chronic use causes thrombocytopenia
- B. Pulmonary vasoconstriction
- C. Not effective orally
- D. ?
- E. ?

[CD01b](#) [Mar97] [Milrinone](#):

- A. Cannot be given orally
- B. Is a phosphodiesterase III inhibitor that decreases cyclic AMP
- C. Decreases peripheral vascular resistance
- D. Increases pulmonary vascular resistance

[CD01c](#) [Feb00] [Milrinone](#):

- A. Is structurally related to thyroid hormone
- B. Is arrhythmogenic
- C. Has its effects via cAMP mediated increase in intracellular Ca²⁺
- D. Increases myocardial oxygen consumption

[CD02](#) [Mar96] [Mar03] [Sodium nitrite](#) used in cyanide toxicity:

- A. Increases methaemoglobinaemia
 - B. To produce increased hepatic sulphhydryl groups Na thiosulphate
 - C. Increases conversion to cyanocobalamin (?hydroxycobalamin) hydroxycobalamine
 - D. Displaces cyanide from haemoglobin
 - E. Enhances oxidative phosphorylation only indirectly
- (see also [CD06](#), [CD37](#))

[CD03](#) [Mar96] [Jul96] [Jul98] [Jul99] [Feb00] [Apr01] [Mar03] [Jul04] Ephedrine:

- A. Is resistant to metabolism by [MAO](#) resistant in GIT, is metab slightly by MAO in liver
- B. Is metabolised by COMT
- C. Action is totally indirect
- D. Acts via direct & indirect beta effect & alpha
- E. Action is purely alpha agonist

(Alternative versions) [Ephedrine](#):

- A. Has direct alpha actions only
- B. Has direct beta actions only
- C. Has indirect (alpha) actions only
- D. ?
- E. Has both indirect & direct actions on alpha & beta receptors

Ephedrine:

- A. Alpha 1 and 2 and beta 1 & 2 & 3
- B. More alpha than beta
- C. *Indirect this and direct that..*
- D. *Direct this and indirect that.. (etc)..*

[CD03b](#) [Apr01] [Mar02] [Ephedrine](#):

- A. ?Increases/?decreases skeletal muscle blood flow
- B. Acts only by indirect effects
- C. Not metabolised by GIT MAO
- D. Not metabolised by COMT
- E. Increase renal blood flow

[CD03c](#) [Jul01] [Jul04] Ephedrine has:

- A. Direct agonist on alpha receptors
- B. Direct and indirect effects on alpha and beta receptors
- C. Indirect actions on alpha receptors

- D. Direct actions on beta receptors
- E. Indirect actions on beta receptors

[CD04](#) [Mar96] [Jul98] The principal (?urinary) metabolite of [adrenaline](#) is:

- A. Normetanephrine
- B. Metanephrine
- C. 3,4-dihydroxy-mandelic acid
- D. 3-methoxy, 4-hydroxymandelic acid otherwise known as VMA
- E. 3-Methoxy 4-hydroxy phenylalanine

[CD05](#) [Mar96] [Jul97] [Jul98] [Mar99] [Feb00] [Apr01] [Jul01] [Feb04] Thiazide diuretics:

- A. Work mainly on PCT
- B. Not effective if severely sodium depleted
- C. Action is independent of acid-base balance
- D. Increase GFR immediately
- E. Decrease BP by decreasing contractility
- F. Cause hypoglycaemia
- G. Interferes with kidney concentrating mechanisms
- H. Causes hypocalcaemia
- I. Used to treat hypercalcaemia
- J. Potentiate hyperglycaemia - ↓glucose tolerance & may unmask DM. Not totally understood but involves ↓insulin secretion
- K. Are effective as antihypertensives by decreasing cardiac output
- L. Cause hypernatraemia
- M. Washes out the medullary concentration gradient
 - (*Multiple options remembered so possibly an amalgam of 2 or more questions*)

SEs:

↑Ca, ↑uric acid

↓Mg, ↓Cl, ↓Na, ↓K

hypochloraemic hypokalaemic alkalosis

MCQ-17 on July 2001 paper:

[Thiazide diuretics](#):-

- A. Increase calcium excretion in the urine.

- B. Decreased efficacy in sodium depletion.
- C. Main site of action is the proximal tubule.
- D. Cause equivalent amount of diuresis to [frusemide](#) less. **only ↑5% filtered Na output because 90% of filtered load is reabsorbed before DCT**
- E. ?

July 2004 version [Frusemide](#) (furosemide), not thiazides

[CD06](#) [Mar96] [Sodium nitroprusside](#) in healthy patient:

- A. Decreases venous more than arterial resistance SNP **unlike GTN causes direct venous & arterial vasoD**
- B. Has no effect on control of pulmonary vascular resistance
- C. Decreases cerebral blood flow
- D. Causes uterine relaxation**
- E. Does not inhibit hypoxic pulmonary vasoconstriction

[CD07](#) [Mar96] [Mar97] [Jul97] [Mar98] [Jul98] [Jul99] [Feb00] [Apr01] [Mar03] Which one of the following statements about [clonidine](#) is correct?

- A. Increase MAC requirements
 - B. Cause transient hypertension with IV administration
 - C. With IV bolus causes hyper- then hypo-tension**
 - D. Causes hypotension immediately
 - E. Is not (?administered/absorbed) transdermally
- (see also [\[\[CD12](#), [\[\[CD36\)](#)

[CD08](#) [Mar97] [Mar99] Regarding Digoxin:

- A. The aglycone portion causes the cardiac effects**
- B. The glycone portion causes the cardiac effects
- C. ?
- D. ?

[CD09](#) [Mar97] [Jul99] [Digoxin](#):

- A. Decreases ventricular response due to vagal stimulation in AF
- B. Decreases myocardial oxygen consumption. **inhibit NaKATPase \Rightarrow \uparrow intracellular Na \Rightarrow \downarrow drive for NCX pump \Rightarrow \uparrow contractility \Rightarrow \uparrow O₂ consumption**
- C. Increases the R-T interval **shortern QT**
- D. Decreases AV conduction

[CD10](#) [Jul97] [Jul00] [Apr01] [Jul02] Which of the following ECG changes would be most likely in [digoxin](#) toxicity:

- A. Increased PR interval - **possibly**
- B. Increased QT interval
- C. Peaked T waves
- D. ST elevation
- E. Ventricular extrasystoles

toxicity more likely if \downarrow k, \downarrow Mg, \uparrow Ca

July 2000 version: Digoxin toxicity:

- A. Inverted T waves
- B. Prolonged PR interval
- C. Xanthopsia
- D. Prolonged PT interval

[CD11](#) [Jul97] [Jul98] Regarding [digoxin](#) overdose/toxicity:

- A. Serum level > 2.1 ng/ml is toxic **>2 (definitely >3) 0.5-2 (considered therapeutic)**
- B.
- C. Causes a long PR interval
- D. Causes xanthopsia (OR: 'causes yellow vision')
- E. Causes a long QT interval and bigeminy

[CD12](#) [Jul97] [Mar02] [Jul02] [Jul04] Clonidine:

- A. Elimination half-life of 3 hours (??or 3 to 6 hrs) 6-23hrs
- B. Excreted 50% unchanged in the urine (or 50% renally excreted)
- C. Oral bioavailability 50% 75-100%
- D. Cannot be absorbed topically
- E. Is highly protein bound 20%

[CD13](#) -Deleted- same Q as [CD05](#)

[CD14](#) [Jul97] [Jul98] [Jul00] [Jul04] [Adenosine](#):

- A. Slows conduction velocity and increases refractory period
- B. Is metabolised in plasma rbc
- C. Decreases urate levels cause ↑10-20%
- D. Methylxanthines increase response inhibit response. dipyrimadole cause ↑response by competing with rbc for metabolism

(see also [CD34](#))

[CD16](#) [Mar96] [Jul96] [Jul97] [Jul98] [Esmolol](#):

- A. Active at beta-1 & beta-2 receptors β_1 selective but does have residual β_2 activity
- B. Half-life < 2 minutes 10min
- C. Has methanol as a metabolite
- D. Is metabolised by (?acetyl/?plasma) cholinesterase rbc and plasma esterases NOT cholinesterase
- E. Is excreted unchanged in the urine
- F. Is a non-selective beta-1 receptor antagonist

[CD16b](#) [Feb04] [Jul04] [Esmolol](#)

- A. Is a non-selective beta antagonist
- B. Has intrinsic sympathomimetic activity
- C. Does not have membrane stabilising activity
- D. ?

CD16c [Feb12]

Esmolol:

A is broken down by plasma cholinesterases.

B has significant membrane stabilising effects

C has active metabolites **methanol & major acid metabolite which weakly active with long elim half life**

D has intrinsic sympathomimetic properties

E is a non-selective agent

CD17 [Jul97] [Jul98] [Mar99] [Jul99] [Jul01] [Jul04] Osmotic diuretics (?Mannitol):

A. Less sodium delivered to distal tubule

B. Hypotonic medulla - **isotonic**

C. **Increased sodium loss**

D. Urine osmolality > plasma osmolality **same**

E. Increased sodium reabsorption / ?causes hytpernatraemia

F. ?MW greater than 600 **182**

G. **Washes out the medullary interstitial gradient**

(see also MD07)

MCQ-16 on July 2001]] paper:

Osmotic diuretics:

A. Include mannitol and the dextrans.

B. **Wash out the medullary osmotic gradient.**

C. Cause sodium retention

D. ?

E. Have a molecular weight >600

CD18 [Jul97] Guanethidine:

A. Causes sedation as a side effect

B. **Postural hypotension occurs**

C. Decreases reuptake of catechols presynaptically

D. ?

CD19 [Jul97] [Jul99] Labetalol:

- A. Alpha agonist & beta agonist
- B. Alpha agonist & beta antagonist
- C. Alpha antagonist & beta antagonist
- D. Is a more potent alpha blocker than [phenoxybenzamine](#)
- E. Alpha > beta effect

"Labetalol is formulated as a racemic mixture of four isomers... Two ...are inactive, a third (S,R) is a potent alpha-blocker, and the last (R,R) is a potent beta-blocker... Labetalol has 3:1 ratio of beta:alpha antagonism"

does have partial agonist activity at β_2

[CD20](#) [Mar98] [Jul98] [Jul99] [Feb00] [Apr01] [Jul04] [Frusemide](#):

- A. 30% plasma protein binding
- B. ??% absorption
- C. Elimination half-life less than one hour
- D. Promotes active secretion
- E. Affects the uricosuric effect of probenecid
- F. Effects not decreased until large decrease in GFR
- G. Causes a diuresis which is dependant on GFR over a wide range

Apr 2001 version: [Frusemide](#):

- A. Has 30% (?35%) protein binding
- B. Has an elimination half-life less than 1 hour
- C. 90% excreted in bile
- D. Increases rate of secretion in the renal tubules

[CD20b](#) [Jul00] [Jul02] [Frusemide](#) does NOT cause:

- A. Hyponatremia
- B. Hypokalemia
- C. Hypouricemia

- D. Hypomagnesemia
- E. Hypocalcemia

[CD21](#) [Mar98] [Jul98] The antiarrhythmic effect of lignocaine:

- A. Because it increases the refractoriness of in cardiac muscle
- B. Therapeutic level 2-5ng/ml **mcg**
- C. ?

[CD22](#) [Jul98] [Feb04] [Jul04] The effects of beta blockers – the following is not true

- A. Relax uterine muscle
- B. Increased AV conduction
- C. Decreased lipolysis
- D. Increased SVR
- E. Mask hypoglycaemia
- F. Negative inotropy
- G. Opposing effects of insulin
- H. ↑ Lipolysis

[CD23](#) [Mar96] [Jul96] [Jul00] [Apr01] Phentolamine: = non selective competitive α antagonist
phenoxybenzamine = non-competitive antagonist

- A. Is a selective alpha-1 antagonist
- B. Binds covalently to the alpha receptor
- C. Causes bradycardia
- D. Is a selective alpha-2 antagonist
- E. Increases cardiac output - \downarrow SVR \Rightarrow \uparrow CO

[CD24](#) [Mar96] [Feb00] [Mar03] A non-selective beta-blocker with low extraction ratio, long half-life and ISA:

- A. [Atenolol](#) β 1 selective

- B. [Propranolol](#) non selective but lacks ISA
- C. [Metoprolol](#) β_1 selective
- D. [Labetalol](#) ISA for β_2 , non selective
- E. ?

[CD24b](#) [Mar02] [Jul02] Which ONE of the following is water soluble, half life 6-8hrs, (“and something else”)?

- A. [Esmolol](#) t1/2 10mins
- B. [Metoprolol](#) 3-4hr
- C. Propranolol 3-4
- D. ?
- E. [Atenolol](#) elim unchanged renally, t1/2 6-9hr, low lipid soluble

Which one of the following selective beta blockers has a low extraction ratio and is predominantly excreted in urine?

- A. Propranolol
- B. Esmolol
- C. [Atenolol](#)
- D. Metoprolol

[CD26](#) [Jul98] [Mar99] [Mar03] [Feb04] [Jul04] [Sotalol](#):

- A. Non-selective beta-blocker
- B. Contraindicated in long QT
- C. Increases K⁺ conductance class III K channel blocker
- D. Used in the treatment of torsades
- E. Class II anti-arrhythmic drug class II, & class III (usually under III)
- F. Is a selective beta 1 antagonist
- G. Blocks K⁺ channels

[CD27](#) [Mar99] [Trimetaphan](#): = ganglion blocking drug used in hypotensive anaesthesia.

- A. Crosses the blood-blood barrier
- B. [Incompatible with thiopentone](#)
- C. ?

[CD28](#) [Mar99] [Diazoxide](#):

- A. Has diuretic activity - **causes Na & water retention**
- B. Opens ATP-dependent K channels
- C. Not absorbed orally **can Rx hypoglycaemia**
- D. ?

[CD29](#) [ghj] [Jul00] [Phenylephrine](#):

- A. Metabolised by COMT
- B. Causes mydriasis
- C. Metabolised by MAO
- D. Effect lasts (?same time as/?longer than) noradrenaline **longer**
- E. Acts by indirect method only

[CD30](#) [Jul98] Regarding [hydrallazine](#):

- A. Fast acetylators have shorter half lives than slow acetylators
 - B. Acts via SNS mechanism
 - C. Slow acetylators decrease half-life
 - D. Has diuretic action
 - E. Clearance > 50ml/kg/min
- (see also [[CD32, [[CD35)

[CD31](#) [Mar99] Which ONE of the following beta-blockers is selective fore beta-1 receptors?

(No other details)

= **atenolol(6-7), bisoprolol(9-12), esmolol(0.15), metoprolol(3-7)**

[CD32](#) [Jul99] Which of the following statements about [hydrallazine](#) is (?true/false)?:

- A. Acts via alpha 1 receptors **is a relaxant!**
- B. ?

C. ?

D. ?

E. Has a duration of action of 1-2 hours. **onset 10-20min, lasts 3-6hrs**

[CD33](#) [Jul99] Concerning [Dobutamine](#)

A. Levo has alpha 1 antagonist and beta agonist effects

B. Levo has partial alpha agonist effect and beta effects

C. Is a pure beta agonist

D. ?

my notes & ganong state is pure β_1 >> β_2 agonist.

black bank blather on about Levo isomer = α agonist & dextro isomer = α antagonist \therefore cancelling each other out. Gay

[CD34](#) [Feb00] [Apr01] [Jul01] [Adenosine](#)

A. Causes AV block via action at A1 receptors

B. Causes bronchoconstriction via A2 receptors

C. Causes renal vasodilation - **is a vasodD so maybe**

D. Causes profound depression of the SA node

E. Decreases AV transmission - **it does but a bit generic**

(see also [[CD14)

[CD35](#) [Feb00] Mechanism of action of [hydralazine](#)

A. Selective cerebral, coronary, renal vasodilator **is it selective???**

B. Alpha agonist

C. None of the above **???** maybe

Hydralazine is a general vasodilator (arterioles) which reduces TPR causing a compensatory increase in HR -> increase in CO and blood flow to brain, heart, kidneys especially (but not exclusively - also get skin flushing due to increase skin blood flow) Hence C (which is

probably D)

[CD36](#) [Jul00] [Jul04] [Clonidine](#):

A. Causes hypertension and tachycardia

B. Causes bradycardia - due to ↓SNS output ⇒ ↓HR, ↓bp & ↓CO

C. A single dose given orally is significantly less effective than an intravenous dose **rapid absorbed**

D. Counteracts the hypertensive response in [phaeochromocytoma](#)

E. ?

(see also [\[\[CD07](#), [\[\[CD12](#))

[CD36b](#) [Jul04] [Clonidine](#) can cause these, except

A. Bradycardia

B. Apnoea

C. Sedation

D. ?

[CD37](#) [Jul00] [Jul04] The first sign of sodium [nitroprusside](#) toxicity is:

A. Cyanide toxicity

B. Tachyphylaxis **ie therapeutic ceiling & impending toxicity**

C. Hypotension

D. ?

(see also [\[\[CD02](#), [\[\[CD06](#))

other signs of toxicity

a. **Increased mixed venous PO₂ indicating paralysis of cytochrome oxidase and inability of tissues to use O₂**

b. **metabolic acidosis (secondary to anaerobic metabolism). Monitor blood lactate, which correlates well with increasing blood cyanide concentrations.**

c. c. CNS dysfunction in awake pts

[CD38](#) [Apr01] [Dexmedetomidine](#):

- A. Alpha-1 antagonist
- B. ?
- C. Decrease in intraocular pressure
- D. Partial alpha2 agonist
- E. Less selective than [clonidine](#)

[CD39](#) [Jul01] [Jul04] [Amiloride](#):

- A. Potassium sparing antidiuretic which blocks the aldosterone receptor - **block Na channel which have been inserted in response to aldosterone**
- B. Blocks luminal sodium channels in the collecting tubules
- C. Increases potassium excretion.
- D. Is metabolised by the liver. **excreted unchanged**
- E. Has a short elimination half time. **18-24hrs**

[CD40](#) [Jul01] With regard to sodium nitrite in cyanide (CN) toxicity:

- A. Causes MetHb
- B. Used to create more hydrocobalamin
- C. Used to displace CN from Hb
- D. Creates more sulfhydryl groups

[CD41](#) [Jul01] Methylxanthines:

- A. (Something about Ca⁺⁺ currents)
- B. (Something about K⁺ currents)
- C. Inhibit adenosine receptors = A1 receptor antagonists & phosphodiesterase inhibitors ⇒
↑cAMP
- D. Decrease plasma glucose level ↑BSL

E.?? Cause diuresis by acting on renal tubules - inhibitor ADh secretion & inhibition of prox tubular Na reabsorption

F. Physically addictive

[CD42](#) [Feb04] [Jul04]

Which is the initial drug to use in the treatment of [ventricular fibrillation](#)?

A. [Amiodarone](#)

B. [Lignocaine](#)

C. Adrenaline

D. Magnesium

E. [Sotalol](#)

[CD43](#) [Feb04] All are side effects of [Thiazides](#) except:

A. Hypokalaemia

B. Hypernatraemia

C. Impaired carbohydrate tolerance

D. Pancreatitis

[CD44](#) [Feb04] Why do you give adrenaline for VF?

A. To coarsen fine VF

B. To improve coronary blood flow

C. Increase chronotropy

[CD45](#) [Feb04] [Nitroprusside](#) toxicity:

A. Treat with ???

B. ?

[CD46](#) [Jul04] Which of the following is a sign of [SNP](#) toxicity?

A. Tachyphylaxis

B. Decreased mixed venous PO₂

C. Sudden decrease in arterial PO₂

D. ?Hypotension

[CD47](#) [Jul04] Dihydropyridine Ca channel blocker causes peripheral oedema due to

Dihydropyridines end with -dipine

A. vasodilator causing redistribution of ECF

B. has a mild antidiuretic effect, and therefore easily treatable with diuretic **actually weak intrinsic pro-diuretic**

C. salt and water retention due to hypotension

D. ?

[CD48](#) [Jul04] [Isoprenaline](#)

A. can be used as a substitute to [Metaraminol](#) for treatment of hypotension

B. used extensively to treat ischaemic heart disease - **↓DBP ∴ ↓coronary blood flow & ↑contractility ∴ ↑O₂ requirement**

C. cause decrease SVR

D. cause bradycardia - **tachy**

E. ?

[CD49](#) Which one of the following is NOT an adverse effect of [amiodarone](#)?

A. Pulmonary fibrosis **5-15%, mortality 5-10%**

B. Photosensitive rash **up to 10%**

C. Corneal microdeposits **all pts after few weeks of Rx**

D. cardiomyopathy - but does cause brady/block/arrhythmias

E. thyrotoxicosis does cause

[CD50](#) The beta blocker with the greatest oral bioavailability is:

A. [Atenolol](#) **50**

B. [Metoprolol](#) **10**

C. [Sotalol](#) **100**

D. Labetalol **90**

E. Carvedilol **30**

- Others: ??Propranolol ??Esmolol

[CD51 Dexmedetomidine](#)

- A. MAC sparing for isoflurane by maximal 30%
- B. can cause bradycardia & sinus arrest
- C. increases CBF - smooth mm relax or ↓CBF
- D. ?
- E. ?

[CD52 \[Jul08\] Acetazolamide:](#)

- A. maximum increase in urine pH 8 hours after oral dose 2hours post
- B. maximum safe dose causes complete absence of HCO₃ reabsorption
- C. maximum safe dose decreases HCO₃ reabsorption (?to) 45% of whole kidney
- D. causes hypochloraemic acidosis hyperchloraemic acidosis
- E. is a potassium sparing diuretic distal tubule excrete K in exchange for Na
 - Carbonic anhydrase inhibition causes a rapid rise to excretion of ~35% filtered HCO₃-load
 - Even with a "high degree" of inhibition, ~65% of filtered load reabsorbed by unknown, downstream, non-carbonic anhydrase related mechanisms

[CD53 \[Mar09\] Acetazolamide](#)

- A. Structurally related to procainamide and may have anti-arrhythmic activity at high doses
- B. *Something about metabolism*
- C. It is not metabolised and is excreted unchanged in the urine.
- D. ?

[CD54 \[Mar09\] Pharmacokinetics of amiodarone:](#)

- A. Oral bioavailability is reliable poor & variable
- B. Doses must be reduced in renal and hepatic failure only in liver failure. renal failure apparently not a problem as half life of active metabolite can be weeks anyway
- C. Omission of 1 or 2 doses can lead to severe consequences
- D. Metabolism is via ?hydroxylation/demethylation?
- E. ?Increases/?decreases refractory period

[CD55 Sympathomimetics:](#)

- A. Phenylephrine acts only on alpha receptors
- B. [Metaraminol](#) acts only on alpha receptors
- C. Methoxamine in high doses acts on beta receptors high doses has inhibitory effect on β receptors - may \Rightarrow bradycardia
- D. Pseudoephedrine is an isomer of ephedrine = stereo isomer
- E. ?

[CD56](#) Which ONE of the following is True about [vasopressin](#)?

- A. Slowly metabolized by renal peptidase liver & kidneys
- B. Does not cause coronary vasoconstriction
- C. Causes mesenteric vasoconstriction - [terlipressin](#) an ADH analogue
- D. Increases plasma level of factor VIII [DDAVP](#)
- E. Is an orally active derivative of ADH [0% OBA](#)

[CD57](#) [Clonidine](#):

- A. Dry mouth and agitation are very common side effects
- B. Half life is 24-48 hours
- C. ?
- D. Can cause severe hypertension if withdrawn abruptly after long term therapy with large doses
- E. Therapeutic dose is 2-5mg per day

[CD58](#) Beta adrenergic receptor antagonists

- A. Seldom causes inhibition of lipolysis
- B. Causes inhibition of gluconeogenesis caused by adrenergic stimulation following hypoglycaemia
- C. Does not mask the signs of hypoglycaemia
- D. Sudden cessation is not associated with rebound effects
- E. There is no evidence of cardiac protection for high risk patients pre-operatively

[CD59](#) [Labetalol](#):

- A. Beta and alpha antagonism with partial agonist activity at alpha 2 receptors
- B. Beta and alpha 1 antagonist
- C. Alpha agonist and beta 1 antagonist
- D. ?
- E. ?

[CD60 GTN](#) is helpful myocardial infarction by:

- A. Decreasing left ventricular pressure and mean arteriolar pressure
- B. Producing methaemoglobinaemia
- C. improving coronary blood flow by dilating the small arterioles **dilates large arterioles**
- D. ?
- E. ?

[CD61](#) Which of the following could cause significant adverse reactions with the MAO-i selegiline?

- A. [Dopamine](#)
- B. [Phenylephrine](#)
- C. **Ephedrine**
- D. [Metaraminol](#)
- E. None of the above

[CD62 Mannitol](#):

- A. **Causes loss of medullary tonicity**
- B. Urine hyperosmolar compared to plasma
- C. Site of action is PCT and DCT
- D. Tubular fluid is isotonic in descending loop of Henle

[CD63 Clonidine](#) side effects

- A. **Sedation**
- B. **Nausea and vomiting**
- C. ?
- D. ?
- E. **delirium**

[CD64](#) With regards to SNP toxicity:

- A **treated with sodium thiosulphate - \uparrow SCN >100x less toxic**
- B treated with GTN
- C one SNP molecule releases four CN⁻ ions **5**
- D. (Edit: *think this also contained an incorrect answer about minimising methaemoglobin levels or something like that*)

[CD65](#) A blood test is taken on a patient who is on a diuretic drug. It shows hyperkalaemia and hyponatraemia. The drug is most likely to be:

- A. Triamterene
- B. Hydrochlorothiazide
- C. [Frusemide](#)
- D. Indapamide
- E. **Spironolactone**

[CD66](#) Vasopressin is used in all except:

- A. Variceal bleed
- B. To improve coronary and cerebral perfusion in cardiac arrest
- C. **Nephrogenic diabetes insipidus**
- D. Septic Shock

[CD67](#) Amphetamine is:

- A. A catecholamine **synthetic non-catecholamine**
- B. **Similar presentation to cocaine toxicity**
- C. **Can cause tolerance** (or ?withdraw) **tachyphylaxis is prominent**
- D. ?
- E. Presents often as coma

[CD68](#) Ventricular fibrillation (VF) in an adult:

- A lignocaine 1.5mg/kg is first line treatment
- B high dose adrenaline (100mcg/kg) improves outcome in "in hospital cardiac arrest"
- C bicarbonate indicated if $\text{pH} < 7.2$
- D vasopressin increase coronary and cerebral perfusion via its action at V1 receptors**
- E caution with use of amiodarone as it may cause Torsades