Quiz for Week 16 - Questions

Question 1. List the mechanisms involved in blood pressure control.

Question 2. By what mechanisms does the renin-angiotensin-aldosterone system act as an effector arm in the control of systemic BP?

Question 3. What are the principal sites of action and corresponding effects of Angiotensin II?

Question 4. How does the sympathetic nervous system elevate blood pressure?

Question 5. List five causes of secondary hypertension.

Question 6. Why does hypertension develop when blood flow into one kidney is restricted by renal artery disease?

Question 7. Name some lifestyle factors which have been proven to be risk factors for the development of hypertension

Question 8. Name three organs which are major targets for tissue damage from uncontrolled hypertension.

Question 9. Name five categories of antihypertensive drugs, citing a potential adverse effect or complication of therapy in each case.

Question 10. How do diuretics reduce blood pressure?

Question 11. A 52-year-old woman is referred for assessment of resistant hypertension. She has had raised blood pressure for 5 years. During this time a variety of medications have been used to control her blood pressure but with limited success. Treatment with a thiazide diuretic was partly effective but was discontinued when her potassium fell to 2.4 mmol/l. Even in the absence of diuretics, her potassium is in the range of 2.9-3.6 mmol/l. Currently, imperfect blood pressure control has been achieved using a beta-blocker (atenolol) and an ACE-inhibitor (enalapril).

What possible cause of secondary hypertension is suggested by this history? What clinical features support this diagnosis?

STOP!
DON'T TURN THE PAGE UNTIL YOU HAVE WORKED ON THESE QUESTIONS IN MONDAY'S PBL TUTORIAL, OR ATTEMPTED WRITTEN ANSWERS YOURSELF.
Quiz for Week 16 – Answer Guides

Question 1. List the mechanisms involved in blood pressure control.

- Baroceptor reflex,
- chemoceptor reflex,
- cerebral ischaemic response,
- atrial (low pressure baroceptor reflex) giving ANP and vasopressin,
- stress-relaxation in capacitance vessels,
- RAS giving A II,
- RAS giving aldosterone,
- Starling forces fluid shifts,
- renal pressure diuresis.

Question 2. By what mechanisms does the renin-angiotensin-aldosterone system act as an effector arm in the control of systemic BP?

Angiotensin II acts as a direct peripheral vasoconstrictor as well as enhancing sodium chloride reabsorption in the proximal tubule, thereby acting to expand ECF volume. Aldosterone further contributes to volume expansion by stimulating sodium reabsorption in the distal nephron.

Question 3. What are the principal sites of action and corresponding effects of Angiotensin II?

1. Vascular smooth muscle of resistance vessels (arterioles): vasoconstriction, so increased TPR
2. Adrenal Cortex, G layer: promotes aldosterone secretion
3. Myocardium: increases force of contraction
4. Brain, hypothalamus: promotes thirst and release of vasopressin (antidiuretic hormone)
5. Renal tubules and renal vasculature: increases salt resorption

Question 4. How does the sympathetic nervous system elevate blood pressure?

Increased total peripheral resistance, increased inotropic state of the heart and ejection fraction (rate is less important, since increasing rate without other changes merely reduces stroke volume) and activation of the Renin Angiotensin System. In the long term (years), there are probably changes in the arterial system and heart.

Question 5. List five causes of secondary hypertension.

1. renal disease in general (e.g. glomerulonephritis often presents with hypertension);
2. renal artery stenosis;
3. coarctation of the aorta;
4. phaeochromocytoma (adrenal medullary type tissue tumour producing catecholamines); Conn’s syndrome (adrenal cortex tumour producing aldosterone or other salt retaining steroid);
5. iatrogenic – side effects of glucocorticoid treatment
Question 6. Why does hypertension develop when blood flow into one kidney is restricted by renal artery disease?

In the early phase of renal artery stenosis, the affected kidney becomes ischaemic, with the release of renin and subsequent activation of angiotensin II in the circulation, leading to systemic vasoconstriction. Later in the disease, hypertensive nephrosclerosis develops in the unaffected kidney, overall GFR is reduced and a volume-retention pattern of hypertension supervenes.

Question 7. Name some lifestyle factors which have been proven to be risk factors for the development of hypertension

- obesity
- excessive salt intake
- excessive alcohol intake
- lack of exercise

Question 8. Name three organs which are major targets for tissue damage from uncontrolled hypertension. For each, describe how damage may occur.

- The brain: cerebrovascular disease develops as a result of hypertensive effects in small and large vessels;
- The heart: is affected by left ventricular hypertrophy as well as accelerated coronary artery disease;
- The kidneys: are affected by progressive glomerular ischaemia (nephrosclerosis) with ultimate progression to chronic renal failure.

Question 9. Name five categories of antihypertensive drugs, citing a potential adverse effect or complication of therapy in each case.

Any of the following:
1. diuretics: electrolyte / metabolic disturbances; allergies
2. beta-blockers: fatigue, insomnia, peripheral vascular disease, worsening of asthma, 1st degree heart block
3. Calcium-channel blockers: constipation, oedema, flushing, dizziness
4. ACE inhibitors: cough, angio-oedema, reduced GFR in renal artery stenosis
5. direct vasodilators: reflex tachycardia, oedema
6. centrally acting hypotensive drugs: drowsiness, depression
7. Angiotensin II receptor blockers: angio-oedema, reduced GFR in renal artery stenosis
8. Alpha-blockers: postural hypotension

Question 10. How do diuretic drugs reduce blood pressure?

Diuretics cause a loss of salt (and water) thus reducing the total extracellular fluid volume and hence, by the Starling forces equilibrium, the blood volume. Decreased blood volume gives decreased RAP and EDV, and reduces SV, which tends to reduce CO and therefore BP. A more sophisticated way to understand this is to see that diuretics move the renal function curve to the left (the curve of output of salt on the y axis, versus mean arterial BP on the x axis) so that the intersection of the curve with a constant salt intake moves to a lower BP.
**Question 11.** A 52-year-old woman is referred for assessment of resistant hypertension. She has had raised blood pressure for 5 years. During this time a variety of medications have been used to control her blood pressure but with limited success. Treatment with a thiazide diuretic was partly effective but was discontinued when her potassium fell to 2.4 mmol/l. Even in the absence of diuretics, her potassium is in the range of 2.9-3.6 mmol/l. Currently, imperfect blood pressure control has been achieved using a beta-blocker (atenolol) and an ACE-inhibitor (enalapril).

What possible cause of secondary hypertension is suggested by this history? What clinical features support this diagnosis?

The most likely diagnosis is a high mineralocorticoid secretion state (e.g. primary aldosteronism). Supporting features are:
- the combination of persistent hypokalaemia and hypertension
- the relatively short history in a middle-aged patient

Other possible, although less likely, options are renal artery stenosis or phaeochromocytoma.