CHAPTER 4 - OEDEMA

IMPORTANT BACKGROUND PHYSIOLOGY

Capillary Filtration. The important basic physiology is Starling's Law of the capillary whereby along the capillary the forces tending to produce capillary filtration (hydrostatic pressure i.e. capillary pressure minus intestitial fluid pressure), are normally balance by the inward oncotic force exerted by proteins (mostly by albumin, being the smallest M.W. protein). There is normally a net outward flow of fluid from the arteriolar end of the capillary, where pressure about 32 mm Hg, and about. 12 mm Hg at the venous. The resultant mean capillary pressure filtration pressure (MCP) of 20 mm Hg is normally balanced by an inward oncotic. This figure of approx. 20 mm Hg only holds for tissues with tight junctions between the capillary cells, such as skin and skeletal muscle, and varies enormously between different vascular beds. This leads us to the concept of:

The capillary filtration coefficient. This is the volume of fluid filtered at the capillary/unit time/unit pressure/unit weight of tissue. Its large variation between tissues is at least partly due to the fact that many capillary beds have intercellular pores that allow large molecules, even albumin, to be filtered. The liver in particular has such large pores and behaves as if almost freely permeable to protein. In between, the kidney has approx 5% of such pores. The skin and skeletal muscle capillaries have only about 0.1%.

These tissue differences in capillary pores would be expected to produce vast differences in the net amount of capillary for filtration versus reabsorption. In the extreme case, the liver would seem to have no inward oncotic drive to subserve reabsorption at all. However there is a low capillary filtration pressure in the portal venous capillaries, as as low as 4 mm Hg

On the other hand, there can be genuine and important imbalance in local Starling capillary forces in some tissues. For example, in the lung, it is vital that tissue fluid does not accumulate in the alveoli, and again that the MCP is low (approx. 8 mm Hg). Were this not so, there would be the constant risk of pulmonary oedema, because pulmonary capillaries have rather loose junctions. To save the day, there are additional factors protecting against alveolar oedema, first a rich lymphatic system capable of carrying substantial quantities of interstitial filtered fluid from the capillary back to the circulation, and second tight junctions between the alveolar epithelial cells to protect the *alveolus* from invasion by fluid.

Normally, approx 20 litres of fluid are filtered across the body capillaries per day, and of this quantity approx. 18 litres are reabsorbed into the circulation direct (at the venous end of the capillary). The remaining two litres are returned indirectly via the lymphatics. Compare this with the 8-10,000 litres per day of blood flowing through the vascular bed. This leads us to the next concept.

CAPILLARY FUNCTION

You might think this is obvious, namely to supply the tissues with oxygen and nutrients. However, the diffusion of oxygen and at least small molecular weight nutrients is a much more rapid process than capillary filtration, and hence the vital point is this.
Diffusion and not capillary filtration determines tissue delivery of these substances. Filtration is required for the delivery of larger molecular weight molecules, and of course the continued diffusion of even small molecular weight substances over time is also highly dependent on the rate of blood supply to the capillary, i.e. is a blood-flow limited process. That said, overall, filtration itself is of minor importance to tissue nutrition.

In this respect, we should consider the primary function of capillary filtration to be the provision of a system capable of rapid adjustment for the purposes of maintaining a relatively normal circulating blood volume. Thus in states of "shock" e.g. due to blood loss, hydrostatic MCP falls substantially, and as a result fluid is rapidly withdrawn into the capillaries to help offset this. On the other side of the coin, any expansion of the circulating blood volume can be rapidly off-loaded into the interstitial space so as to maintain blood pressure relatively normal.

CLINICAL OEDEMA

Clearly, disturbance of any capillary Starling forces can result in oedema, whether that disturbance be a general or local one. In practice, Starling forces must be disturbed substantially before actual clinical oedema occurs. For example, we need an average outward driving pressure along the capillary of at least 15 mm Hg before hydrostatic oedema is clinically manifest - for several reasons. First, the interstitial fluid volume has to increase by approx. 2 litres before oedema becomes clinically apparent; second, this early retention of interstitial fluid will be associated with an increase in interstitial hydrostatic pressure and reduction in interstitial oncotic pressure, both tending to oppose any net outward filtration from the capillary; and third, the lymph drainage has a great reserve of function, being capable of increasing its flow up to approx. 7 fold. These physiological compensatory mechanisms normally stabilize against any change from the capillary fluid balance.

Let us now go through the various factors in turn and see how they may lead to oedema

I. TRANSUDATES & EXUDATES

TRANSUDATES are brought about by simple alteration of the various Starling capillary forces.

(A) Retention of Sodium Chloride

This is one of the potent causes of oedema. Normally, of course, the kidney compensates for any undue sodium and water retention, so usually for oedema via this means only occurs via significant impairment of renal function. With normal renal function, it is almost impossible for salt intake to be high enough to produce oedema. Even in cases of primary aldosteronism where there is a functional increase in the re-absorption of sodium from the distal convoluted tubule (in exchange for $K+$ and/or $H+$), oedema is rare. This is partly for the reasons discussed above, but there are others as well. The first is that dilution of plasma albumin from the retained sodium chloride results in a reduction in plasma oncotic pressure and, at the level of the glomerular capillaries, this increases glomerular filtration (by alteration of local Starling's capillary forces), and hence tends to limit the amount of renal sodium retention. Release of atrial natriuretic peptides from plasma (atria) volume expansion is another compensatory mechanism.

Structural renal disturbances can lead to sodium retention of sufficient degree to cause clinical oedema, although because this organ has a vast reserve of function, not usually until the disease/ dysfunction widespread. Primary sodium retention in renal disease may occur in several ways. The most obvious is a reduction in renal blood flow and glomerular filtration rate. Less obviously, oedema may occur at the glomerular level in the presence of a reduced renal blood flow even with a normal GFR. This situation arises from efferent arteriolar constriction which results in increased glomerular capillary filtration, so that blood leaving the efferent arteriole to perfuse the tubules has a high oncotic pressure. Hence more of the glomerular ultrafiltrate is reabsorbed at that level, resulting in partial compensation.

In theory, proximal tubular disease should readily lead to renal sodium loss, but distal tubular reabsorption of the increased $Na+$ load can normally compensate for this. Disal renal tubular disease/ dyfunction, on the other hand, is more likely to be associated with alteration to sodium balance, e.g. $Na+$ loss in Addison's disease, where absence of circulating aldosterone causes sodium wasting; and in Conn syndrome, where there is mineralocorticoid excess, sodium retention occurs to cause expansion of the ECF.

The kidney can also cause oedema through excessive protein loss. This will be discussed under C. below.

One of the commonest causes of oedema due to sodium retention in hospital wards is over-infusion of IV fluids, particularly in the elderly with impaired GFR/renal function.

An important point is that, when sodium is retained it is adjusted to isotonicity in the vascular and interstitial compartment, due to the release of anti-diuretic hormone. Plasma sodium concentration remains therefore normal. The corollary is that:

Plasma Na+ level is not a guide to total body Na+ balance

Distribution of Na+ across the ECF. Initial absorption of Na+ must be in the vascular compartment. But three mechanisms determine rapid equilibration with the interstitial fluid. First, the increased blood volume which leads to an increased venous volume and pressure, and therefore an increased "back pressure" on the capillary, so increasing the hydrostatic filtration. Second, this increased venous pressure will be manifest centrally as an elevated JVP, so tending to increase ventricular filling pressure and therefore cardiac output, so as to raise arterial and arteriolar pressure, again increasing capillary filtration.Third, sodium and water retention will lead to a dilution of plasma proteins, with a reduction in plasma oncotic pressure, again facilitating sodium and water equilibration with the interstitial fluid.

A point to puzzle over. Why is it that five litres of isotonic sodium chloride retained in the body causes severe oedema and vascular congestion, yet 10 litres of retained water causes no trace of oedema at all, much more a disturbance of organ function, particularly confusion and other cerebral disturbances, even epileptic seizures?

Answer: Because Na+ is mostly distributed across the ECF, not the intracellular comparment, and so Na+ retention does not, unlike H2O retention, cause intracellular swelling.

Diuretic-withdrawal oedema. A not uncommon cause of oedema from sodium retention is diuretic withdrawal after chronic abuse (e.g. for slimming). The explanation for the subsequent oedema is as follows. During long-term inappropriate administration of diuretics such as frusemide, a depletion of total body sodium occurs. Compensatory juxtaglomerular apparatus (JGA) release of renin follows and then increased angiotensin II stimulates aldosterone to limit sodium loss. In the early stages, this stimulated renin release is readily reversible on withdrawal of the diuretic. But after many years the JGA undergoes hyperplasia, and the increased renin release becomes autonomous, so that when the diuretics are withdrawn, Na+ retention continues. This hyperplasia of the JGA will gradually resolve and oedema will settle, but this may take months, even years, so can be difficult to manage.

Odema of Heart Failure. This is also primarily due to renal sodium retention (Ch. 3). So when you see a patient with oedema and an elevated JVP, think about whether this is primary right heart failure, or whether you are dealing with circulatory overload from primary renal impairment.

Pulmonary oedema can occur very quickly with acute left heart failure (Ch. 3) *before there is any time for sodium retention*. This is probably because the sympathetic discharge which arises to compensate the "forward" failure in this situation causes not only constriction of splanchnic, renal, and skin vascular arterioles, but venular constriction, so shifting blood centrally. Then, if the right heart is functioning normally, the increased right ventricular filling pressure will increase the strength of right ventricular contraction and R.V. output, so as to displace the blood to the lungs. This rapidly causes pulmonary congestion and oedema, because the failing left ventricle just cannot keep pace with the increased input delivered to it.

(B) Other Causes of Increased Capillary Pressure

First, a local elevation of venous pressure, (e.g. by venous obstruction, failure of the venous muscle pump as in muscle paralysis) may cause local oedema.

Second, an increased hydrostatic capillary driving pressure can come about in other ways than through increased venous "back-pressure", for example by arteriolar dilatation. Such dilatation will also produce a shift towards salt and water retention, which occurs even normally in pre-menstrual females, especially in warm weather. Also seen in patients with heart faiure or hypertension treated with the arteriolar dilatoring dihydropyridine Ca++ channel blockers like nifedipine, felodipine and amlodipine.

Arteriolar dilatation also plays a role in the oedema of local inflammation, but this is associated with increased capillary permeability so will be considered separately. (See Exudates).

(C) Hypoproteinaemia

When plasma albumin falls, plasma oncotic pressure falls, and with it the forces tending to limit the flow of fluid to the interstitial compartment under the normal influence of the capillary hydrostatic pressure. Hence, any condition which results in a reduction of circulating albumin will produce an expansion of the interstitial fluid compartment. Note, first, however, that the vascular compartment in this situation is not expanded but contracted (contrast primary Na+ retention) Second, because of the various compensatory mechanisms discussed, it will not usually be until the plasma albumin falls below levels of approximately 30 grams/litre (normal 35-50 g/l) that significant clinical oedema will occur by this means alone.

HIERARCHIC DISSECTION OF HYPOPROTEINAEMIC OEDEMA

(i) Liver Disease

Significant hypoproteinaemia may occur in liver disease, due to reduced hepatic production of albumin, and this may lead to oedema. However, additional factors are sometimes at work. For example alcoholics, who frequently have liver disease, may also have a reduced protein intake contributing to the lowered albumin level. In addition, we sometimes see ascites out of proportion to peripheral oedema in patients with liver disease, due to narrowing of portal venules from portal fibrosis, with resultant increase in venous back-pressure in the splanchnic bed. A further factor which

may contribute to the oedema of liver disease is sodium retention, due to lack of aldosterone clearance by the diseased liver causing increased circulatory aldosterone levels.

Measurement of oedema protein levels. In any clinical situation of oedema, it is useful to aspirate and analyse the oedema fluid. A simple rule of thumb is that when the oedema is due entirely to alteration of Starling's capillary forces (transudate), the albumin concentration will be at least 50% less than that of circulating plasma. By contrast, with an (inflammatory) exudate, where capillaries are damaged and their permeabilities altered, albumin concentration will usually be more than half that of plasma (there may also be red cells, white cells). This is important in diagnosis.

Of course, where capillarie are more porous, as in the lever, so that even normal hepatic interstitial fluid will contain a high protein content. Because of this, liver disease distorting hepatic sinusoids to increase portal venous pressure will result in the transudation of fluid into the interstitial space - and peritoneal cavity - of a fluid which contains a protein content almost as high as plasma. So in any such ascites, high protein concentration does not necessarriiy mean that we are dealing with an exudate. This is particularly true in the so-called Budd-Chiari syndrome where the hepatic sinusoids are thrombosed, leading to a high back pressure and the transudation of vast quantities of ascitic fluid of very high protein content. Similar high protein ascitic fluid accumulation may also occur in severe chronic right heart failure e.g. from gross tricuspid incompetence.

(ii) Nephrotic Syndrome:

Oedema occurs when urinary protein excretion exceeds 3.5 g/L. and plasma albumin < 30 g /L. This condition occurs where glomerular disease causes not primary sodium retention, but capillary leakage of protein, particularly albumin, and so eventually leads to hypo-albuminaemia with secondary fluid retention. Actually, a great deal of protein (up to 10 g/day) may be lost in the urine in this way without significant oedema, due to both an increased increased proximal tubuar reabsorption of any filtered protein, and to a high liver reserve capacity to increase albumin production.

(iii) Starvation

Clearly, to build plasma albumin in the liver requires an intake of the appropriate amino acids etc., so that starvation can lead to quite severe oedema, ascites etc., as in Kwashiorkor.

(iv) Steatorrhoea

Malabsorption - and/or maldigestion may lead to hypoproteinaemia, because complex proteins have to be both digested and absorbed before they can be handed on to the liver in the form of appropriate building blocks (amino acids) for albumin production.

<u>(v) Protein-losing enteropathy</u>.

Some gastorintestinal conditions, particularly the inflammatory ones like chronic ulcerative colitis, are associated with vast quantities of plasma protein exudation at the site of inflamed and ulcerated areas, and this contributes to oedema. In other situations (e.g. Whipple's disease), local lymphatics become blocked, resulting in a similar effect (see also Exudates).

2. EXUDATES

These are typically seen in inflammation, where there is not only gross local capillary (and arteriolar) dilatation, but also capillary damage with increased capillary pore size - even to the extent that red and white blood cells may exude across the capillary wall. Examples of inflammatory oedema include local trauma, burns and infection,and some of the allergic oedemas such as bee stings, hives, etc. Typically, the oedema fluid has a protein concentraion of greater than 50% of plasma. It may also contain inflammatory cells, so fluid analysis is particularly useful in diagnosis.

3. LYMPHATIC OBSTRUCTION

This can also give rise to oedema. "Lymphoedema" tends to be rich in protein. This is because, under the increase in interstitial pressure, the water and electrolyte elements of the oedema reach a new equilibrium across the capillary wall to facilitate their reabsorption, but the same cannot occur with the larger protein molecules. Chronic lymphoedema therefore characteristically shows an increased protein concentration, which eventually tends to become organised in the interstitial tissue to give a "wooden" feel i.e. it does not "pit" on finger pressure. However, this organisation takes time, so that acute lymphatic obstruction usually does pit in the same way as exudative or transudative oedema. Exudates have a high protein concentration, but so too do some transudates (e.g. liver disease), so distinction can be difficult on occasions. In thoracic duct obstruction, the lymphatic drainage also subserves the function of fat absorption from the intestine (via the lacteals) so as well as a high protein content, thoracic duct obstruction fluid will typically contain fat globules giving the associated ascites (and even urine) a milky appearance, readily verified by fat staining, and thus readily distinguishable from transudation.

DETERMINING THE ANATOMICAL SITE OF THE PROBLEM IN A PATIENT WITH OEDEMA

As usual, always ask BROAD questions first, and narrow down hierarchically, as follows:

1. <u>Is it local or general?</u> In this respect, oedema of any general cause is usually localised, at least initially, by gravity (to the ankles).

2. <u>If general, then is there increased circulating blood volume with raised central venous pressure?</u>

(a) If so, are we dealing with primary cardiac failure, or with some renal cause of sodium retention?

(b). <u>If the central venous pressure is not raised</u>, is the plasma protein albumin low, i.e. is there evidence of hypoproteinaemia. In this respect, significant clinical oedema will not usually occur until plasma albumin concentration is less than 25 g/l (normal greater than 35 g/l). This is important, because in many conditions associated with oedema, such as sodium chloride retention (ECF expansion), there is some secondary dilution of the plasma proteins and this can trap you into thinking that the moderate reduction in plasma albumin is the primary cause.

3. <u>Hypoproteinaemia</u>

If there is hypoproteinaemia, is it due to reduced protein intake, maldigestion, malabsoprtion, gastro-intestinal protein loss, liver disease, or increased renal loss of protein?

If ascites is out of proportion to oedema elsewhere in this context, is there evidence of cirrhosis or other liver disease?

Analyse the oedema fluid directly if possible, e.g. pleural or acitic fluid to distinguish transudates from exudates.

4. <u>If the JVP and plasma proteins are normal</u>, think again whether you might be dealing with a central obstruction either high in the inferior vena cava, or high lymphatic duct obstruction.

PATHOLOGICAL DIAGNOSIS

There are no special additional points, but again the time-intensity relationship, weight loss, fever, give you the clues; also the consistency of any palpable organs (e.g. the "hard" liver edge of hepatic cirrhosis). Examination of the the relevant fluid may also help determine the general pathological nature of the condition

FUNCTIONAL DIAGNOSIS

We have really used our functional diagnosis, as we so often do, to determine our Anatomical diagnosis, and little further needs to be said, except that in this category we should always give an indication of the *degree* of functional impairment of the organ concerned.

AETIOLOGICAL DIAGNOSIS

As usual, look for this in the background history; for example, in a patient with local oedema, particularly when associated with erythema without trauma or infection, is there a history of background allergy in the past or family history, or of medications like amlodipine in the background. In difficult cases of generalised oedema, remember diuretic withdrawal, and other drugs, particularly vasodilators such as the calcium channel blockers.

MCQs & PROBLEM SOLVING

Mechanisms in Disease MCQs

A. Which of the following are well-recognised features of oedema and ascites associated with chronic generalised liver cell disease?

1. Proteinuria due to a high back-pressure on the renal veins.

2. The ascitic fluid often has an albumin concentration of more than half that of plasma, despite being a transudate.

3. Substantial clinical oedema occurs at protein levels of 30 g/l.

4. The ascitic fluid will usually contain large numbers of fat globules.

5. Ascitic fluid out of proportion to peripheral oedema normally only occurs when there is associated sub-hepatic inferior vena caval obstruction as well.

6. A reduced aldosterone clearance rate typically contributes to the oedema.

7. A high protein intake is often associated with, and will aggravate, the degree of oedema.

8. Clinically, other signs of chronic liver dysfunction are unusual.

9. The oedema of chronic liver disease is characteristically "wooden" and difficult to pit at the ankles.

Answers in last section of chapter.

Clinical Problem-Solving [Online tutorial available at this site under 'Tutorials']

A 42 year old (1) boiler maker (2) presents with moderate bilateral leg swelling (3) and abdominal swelling (4), increasing over a period of about four months (5). Some loss of appetite (6), but other symptoms have been vague (7). Smokes 20 cigarettes/day for 20 years (8) and drinks a "moderate" amount of alcohol (9). No past history of hypertension (10), kidney disease (11), chest pain (12), diarrhoea (13), or jaundice (14).

Examination: Palmar erythema (15), bruising (16), finger clubbing (17), Dupuytren's contracture (18), spider naevi (19), mild jaundice (20), parotid enlargement (21), gynaecomastia (22), reduced bodily hair with female distribution (23), testicular atrophy (24), and abdominal swelling which is dull in the flanks with a shifting postural percussion dullness of 10 cm (25). A firm mass in the right upper quadrant with an edge running running across to the left upper epigastrium (26); moves downwards on inspiration (27) and is dull to percussion anteriorly (28). No overlying bruits (29). A notched mass under the left costal margin which moves downwards and medially on inspiration (30); dull to percussion anteriorly (31); not ballottable from the loin (32). Prominent veins around the umbilicus (33). Jugular venous pressure normal (34). Bilateral moderate pitting ankle oedema (35). Heart examination NAD (36). Blood pressure 140/90 mm Hg (37). Right lower chest is dull to percussion posteriorly where breath sounds are diminished (38). The upper edge of liver dullness is in the fourth right intercostal space anteriorly in the mid-clavicular line (39). Temp. 37 deg. C(40). Urine NAD (41).

Solving the Problem. Now draw up your usual four columns (widest for "How?" column), and work through to a solution of this problem linking inferences leading to like conclusions. 'Rule off' and make sub-conclusions where you can and then make a final overall diagnosis before answering the MCQs below.

Graphic Solution: Available in next section as a jpeg. When viewing, centre the picture so that all 4 columns are able to be seen at the same time. The solution is available in two parts, in the sections: An overall solution, and a more detailed view.

Diagnostic Dissertation. A Diagnostic Dissertation about the case follows. Make one yourself for comparison before viewing.

MCQs. Think about the following questions about the case before turning to the graphic solution. Answers are available in the final section of this chapter.

MCQ: Which of the following are likely to be correct?

1.This patient has evidence consistent with portal venous hypertension.

2.The shifting abdominal dullness might well have been produced by an increased back-pressure on the renal vein (reducing GFR) from increased portal venous pressure.

3.He is likely to have hypoproteinaemia, with a plasma albumin of less than 25 g/l.

4.There is clinical evidence of chronic liver dysfunction.

5.There is clinical evidence of fluid in the right pleural space.

6.There is evidence of hepatic enlargement.

7.There is left renal enlargement.

8.There is evidence of secondary feminisation.

9. Alcoholic congestive cardiac failure might well be contributing to this man's oedema.

10.There is good evidence that cigarette smoking is involved in the pathogenesis of fibrotic hepatic disease.

11. Alcohol is well recognised as an important underlying aetiological cause for such clinical features.

12. Pathologically, this can be classified as an acute inflammatory disorder.

PROBLEM SOLUTION-1

Oedema — Diagnostic Problem

Where?	What?	How?	Why
Hepatic	5. 4 /12.= **Chronic** 6. Loss of appetite ?wt. loss as well. 7. Vaguely unwell = ? generalised process or organ dysfunction. — non-speecific. 40. Afebrile i.e. not a generalised inflammatorry process i.e. **Prob.** **Non-inflamm.** 26. Liver firm rather than hard; thus prob. not neoplastic, more fibrotic. i.e. prob. **cirrhotic** .	3. Bilat. leg swelling (?oedema). 4. Abdominal swelling (?ascites). 15. Palmar erythema 16. Bruising — (?prothrombin defic.) 17. Finger clubbing 19. Spider naevi 20. Jaundice 22. Gynaecomastia 23. ↓ body hair, female distrib ^{n}. 24. Testicular atropy. — all compat. with (chronic) **liver dysfunction** including secondary feminisation (22-24) 18. Dupuytrens. 21. Bilat parotid swelling. Compatible with alcohol excess 25. Abdominal swelling with shifting dullness = Ascites 26. R.upper quadrant mass,.firm 27. attached to diaphragm, 28. anterior mass, 29. non-vascular 26. — 29. **Liver** ?enlarged non-vascular **Firm** 30. L.upper quadrant mass, attached to diaphragm, 31. dull anteriorly 32. ballottable from loin = anterior mass. Enlarged spleen 33. Prominant veins around umbilicus,prob. Caput medusa; **Portal hypertension** 34. J.V.P.normal Thus,oedema not due to primary fluid retention / heart failure. 35. Pitting **oedema** of legs = probably not due to lymphatic obstruction (present for 3/12). 36. Heart N.A.D., 37. B.P = normal oedema non cardiac. Oedema & ascites therefore probably due to **Hypoproteinaemia** 38. Dullness at right base ? R.pleural effusion or enlarged liver 39. Upper border of liver is high. Thus, with evid. above (26 - 28), Liver enlarged 41. No evidence of renal disease.	1. Aet. 42 = rel. young. 2. Boilermaker ? alcohol intake. 8. Smoker 20 / day 9. **? High alcohol** **intake.** 10-14. No obvious background of kidney, heart, intestinal or liver disease.

PROBLEM SOLUTION: FINAL DIAGNOSIS

Oedema Diagnostic Problem: Final Diagnosis

ANATOMICAL DIAGNOSIS
Hepatic.

PATHOLOGICAL DIAGNOSIS
Chronic process to sub-acute, prob. not inflammatory

FUNCTIONAL DIAGNOSIS
Hepatic dysfunction with secondary feminisation, ascites, oedema (hypo-proteinaemia), jaundice, prothrombin deficiency, portal hypertension (splenomegaly, caput medusa)

No firm evidence of R. pleural effusion. Percussion dullness at R. base probably due to liver enlargement

AETIOLOGICAL DIAGNOSIS
Alcohol

MCQ ANSWERS

Answers to Mechanisms in Disease MCQ:
Only 1 & 6 correct.

Answers to Problem Solving MCQs:
1, 3, 4, 6, 8 & 11 correct. All others false