REN FAQs

1. Can you explain about how you determine its respiratory or metabolic acidosis by the blood gas values again?
   First step is to realise its acidosis because the pH is below the normal range. Next, you can look at either the CO_2 or the bicarb values. If the CO_2 is above the normal range, we know that is it respiratory acidosis because CO_2 is acidic when dissolved in blood. In this scenario, we would expect to see a high bicarbonate as a metabolic compensation – if the bicarbonate was the problem we would be in metabolic alkalosis. On the other hand, if bicarbonate is low with a low pH then we can determine it is a metabolic acidosis. In this scenario, we would expect to see low CO_2 as a respiratory compensation – if this was the problem we would be in respiratory alkalosis

2. ATII causes afferent vasoconstriction in the volume-receptor reflex to lower GFR and increase arterial BP so does ATII increase or decrease GFR? Or does it depend on the situation?
   I've consulted my CVS notes for this one. ATII causes all arterioles to constrict. In CVS, the information regarding central receptors describes that ATII will systemically vasoconstrict which as we know will lead to reduced renal blood flow and GFR. Now referring back to REN, I have doubled checked and it specifically talks about ATII causing efferent vasoconstriction to increase GFR. I think an important consideration here is that ATII also increases Na^+ absorption in the proximal tubule which will help to increase GFR. To be honest I'm a bit puzzled on the 2 conflicting pieces of information myself. ATII can be released systemically or locally in the kidneys so this may explain the 2 alternative answers but I'm not 100% confident so sorry I can't be more help here but I don't want to give false information out.

3. Are granular cells and juxtaglomerular cells the same?
   Yes

4. How is adenosine a vasoconstrictor but in functional hyperaemia it is a vasodilator?
   Again, I had to check my CVS notes on this. In most vessels, adenosine is a vasodilator so you are correct but in renal vasculature it acts as a vasoconstrictor in tubuloglomerular feedback. I have just read a journal article to check this and the understanding of how and why this occurs is not fully understood as it can act as a vasodilator for other scenarios in the kidney. For the exam, I would just accept that in CVS it is a vasodilator and in the tubuloglomerular feedback in REN it is vasoconstrictor. But a great question it's made me think!

5. Is caffeine another diuretic in the proximal tubule?
   Caffeine is an adenosine receptor antagonist. As adenosine acts to vasoconstrict the afferent caffeine causes it to vasodilate and as such can be considered a weak diuretic. This is an action at the glomerulus. It also reduces Na^+ absorption in the proximal tubule so I suppose you could argue it is a proximal tubule diuretic but if I got a question asking me to pick a diuretic and describe it, I'd probably go for one of the more conventional ones ☺

6. Would hyponatraemia be a side effect of every diuretic?
   No. Consider the aquaretic drug class for example – they have no effect on ion movements and only decrease water absorption therefore there is no risk of hyponatraemia. Most diuretics cause hyponatraemia however, as the majority block a channel which is used for reabsorbing sodium.

7. Which space would be inside the cell? Would it be interstitial?
   No it is intracellular. Interstitial is part of the extracellular space. It is the space between cells within a tissue, outside of the blood vessels.