

1. E and yes you will lose 2 pts if not E, or E is not in fact true in your case)
2. C
3. E Ppl must reach a large neg ; Palv-Ppl must be positive (~20-30) at high lung volume. Palv varies from 0 to drive inspiratory and expiratory flow
4. C flow is independent of further effort in this range.
5. D Need to match perfusion to ventilation.
6. C .
7. D $3 \times 760 = 2280$; $-47 = 2233$; $x.21 = 469$; $-40/0.8 = 419$
8. C Hypoxia has little effect (prob negative effect) on central chemoreceptors.
9. E This would create a R-L shunt equivalent (deoxygenated blood to left heart)
10. B With hypoventilation (increasing PCO_2) the reaction is going the other direction.
11. A .
12. A
13. A
14. A Trachea is filled with gas which has been equilibrated in alveoli.
15. B No, some oxygen has been extracted into the blood.
16. A This gas has been warmed and humidified (but may be slight cooling).
17. A
18. B Not above the minor fissure.
19. A RLL extends quite high posteriorly.
20. A Either posterior or anterior, parietal or visceral pleural nodule is possible..
21. A to improve oxygen delivery, compensating for the decreased content.
22. B partial pressure is still equilibrated to (normal) alveolar gas.
23. B potential O_2 saturation sites are filled by CO instead.
24. A this is the key abnormality
25. B $150 - 64/0.8 = 150 - 80 = 70$; $70 - 40 = 30$
26. B correcting pH to 7.4 will lower bicarbonate by 1 to 31; $31 - 24 = 7$
27. B respiratory acidosis and metabolic alkalosis are present.
28. A appropriately compensated respiratory acidosis.
29. A Vessels are dilated by radial traction (interdependence).
30. A Capillaries tend to be stretched and flattened, also compressed as vascular pressures, influenced by pleural pressure, fall relative to alveolar pressure.

Decreasing ventilation relative to perfusion (low V/Q) will increase the amount of oxygen extracted from incoming fresh air, decreasing the alveolar PO_2 . (The PCO_2 will rise a few mmHg, but is limited by the level in the incoming blood, about 46-47 mmHg). The exiting blood will be equilibrated to this lower PAO_2 and its decreased content will contribute to widening the $(A-a)\Delta O_2$ after downstream mixing.

Decreasing perfusion relative to ventilation (high V/Q) will decrease the amount of oxygen extracted from incoming fresh air, increasing the alveolar PO_2 toward that of inspired air. The amount of CO_2 delivered to these alveoli will decrease so the local alveolar PCO_2 level will fall until the output via ventilation matches the input. The lower exhaled CO_2 from these alveoli will contribute to alveolar dead space and wasted ventilation. Again the exiting blood will be equilibrated to the local alv gas.

Absence of ventilation with continued perfusion is intrapulmonary shunt. The exiting blood will be unchanged from the incoming (systemic venous) blood with PO_2 of about 40 and content about 5 ml/dl less than arterial blood. This will contribute to widening the $(A-a)\Delta O_2$ after downstream mixing.

Blood flow should decrease, in response to hypoxic vasoconstriction, reducing shunt.