Q1: Explain the negativity of intraplural pressure?

Due to dynamic harmonious antagonism between the chest wall and the lung

Q2: Discuss the physiological and pathological factors that affect the negativity of intraplural pressure?

-Physiological : at birth (zero), elasticity of the lung and chest wall, surfactant factor, vaslalva maneuver(positive)
Pathological:
- Less negative (emphysema)
- Zero (stab wound without valve)
- Positive (stab wound tension pneumothorax with valve)

Q3: Explain the factors that affect the resistance of the airway?

Resistance and diameter (radius) are inversely proportional
*Asthma patients have problem in bronchi not bronchioles because the radius in bronchi is less than in bronchioles thus more resistant during expiration in brochi

Q4: Explain how the effect of inspiration and expiration on heart murmurs?

-During inspiration:
Inspiration will increase the venous return of lower extremities
->accentuates the Rt. Side murmurs
-During expiration:
Expiration will increase the venous return from pulmonary veins
accentuates the Lt. Side murmurs

Q5: If you want to examine the chest wall during expiration what is the best location to put the stethoscope?

Above the clavicle , trachea
* the expiration will be shorter than insp if we examine on the chest wall
* Expiration will be longer if we examine on trachea

Q6: Explain the physiological, clinical and forensic significance of residual volume?

- Physiological:
Maintain aeration of blood, prevent sudden flotation of blood gases
- Clinically:
Increasing in RV/TL > 30%, this indicates emphysema
- Forensically:
To differentiate between stillborn death and child homicide by putting lung autopsy in water
FLOATING-> Child homicide, SINKING ->Stillborn

Q7: Explain how FEV1/FC affected during restrictive and obstructive lung disease?

- Obstructive :
In obstructive diseases the problem will be in expiration ,so FEVI will decrease more than FUC -> low ratio
- Restrictive:
Problem will be in inspiration, so equal decrease in FEVI and FVC -> high

Q8: What is the primary function of lung? Why?

Getting rid of C02,
Because it will cause acidosis thus affecting metabolic pathways and disrupt homeostasis

Q9: Explain why the deposition of pollutant and dust is higher in the terminal bronchioles?

At upper respiratory tract the velocity is the only factor to move particles
Then at generation 16 (terminal bronchioles), particles will move by diffusion , and diffusion will be hard
So particles will deposit at terminal bronchioles

Q10: what is the normal value of A-a gradient and explain physiological and pathological factors that affect A-a gradient?

It's normally being between [5-15 mmHg]
•Pathological -> intrinsic restrictive lung disease (E.g. Lung fibrosis), increase A-a gradient (refractory O2 therapy).
•A-a gradient is normal and there's hypoxia , due to :
•Hypoventilation
•High attitude
•Defect in Hgb
-> giving this patient 100% O2 is a good therapy

Q11: what is the best way achieve more alveolar ventilation?

By increasing tidal volume (volume rate)
Not by FiO2 / pressure/ flow rate

Q12: after surgical removing several arteries the lungs still adequately oxygenated?

Yes, Due to presence of dual circulation
Pulmonary circulation + bronchopulmonary circulation

Q13: how the resistance of the pulmonary vasculature of pressure at high attitude differs from that of pressure at sea level?

In high altitude(low Pb , low FiO2) pulmonary resistance will increase due to :
- pulmonary vessels will be constricted as a response to hypoxemia
- Other systemic arteries will be dilated

At sea level compared to high altitude resistance will decrease