Case 27

Pneumothorax

Serena Cervantes and her boyfriend left their senior prom and were en route to the post-prom party when a limousine carrying other students slammed broadside into their sport utility vehicle. Serena was not wearing a seatbelt, and she was thrown from the vehicle and landed on a fence. When the emergency medical crew arrived, it was clear that she had multiple injuries, including a penetrating chest trauma that caused a pneumothorax. She was having difficulty breathing, and pulse oximetry showed an $O_2$ saturation of 85%. In the emergency department, a chest x-ray confirmed that her left lung had collapsed, and a large-bore chest tube was placed in her thoracic cavity.

QUESTIONS

1. Following a traumatic pneumothorax, the pressure in the intrapleural space becomes zero. What is the normal intrapleural pressure, and what does this pressure of zero mean?

2. Why did the pneumothorax cause her left lung to collapse?

3. Pneumothorax also causes the chest wall to “spring out.” Why?

4. The chest tube was connected to a vacuum pump. What is the purpose of creating a vacuum in the thoracic cavity?

5. Serena’s $O_2$ saturation of 85% was much lower than normal. What is the significance of this number, and what caused it to be low?
1. Normal intrapleural pressure is negative, or less than atmospheric pressure. Negative intrapleural pressure is created by the elastic forces of the lungs and chest wall pulling in opposite direction on the intrapleural space. (Note that the intrapleural space is not a literal space, but a virtual space between the visceral and parietal pleura.) When the system is at equilibrium (i.e., at functional residual capacity, FRC), the lungs, with their elastic properties, are naturally inclined to collapse, and the chest wall, with its elastic properties, is inclined to spring out. These two equal and opposite forces pulling on the intrapleural space create a vacuum, or negative pressure, in the space.

When Serena sustained a penetrating chest wound in the accident, her chest wall was punctured and her intrapleural space was opened to the atmosphere. Her intrapleural pressure was "zero," meaning that intrapleural pressure was equal to atmospheric pressure. (By convention, lung pressures are always expressed relative to atmospheric pressure.)

2. Pneumothorax caused her lung to collapse because the injury eliminated the normal, negative intrapleural pressure. Normally, the lungs are held open by the negative intrapleural pressure outside of them. Without this negative outside pressure, the lungs follow their natural tendency to collapse (owing to their elastic properties), as shown in Figure 3-12.

3. The elastic properties of the chest wall are such that it is naturally inclined to "spring out" (like a compressed coil). This tendency of the chest wall is normally opposed by the negative intrapleural pressure. (Just as the negative intrapleural pressure keeps the lungs from collapsing, it also keeps the chest wall from springing out.) When the negative intrapleural pressure is eliminated by a traumatic pneumothorax, the chest wall springs out because there is no longer a force opposing its natural tendency (also shown in Figure 3-12).

4. A large-bore tube connected to vacuum was inserted in Serena's chest. The vacuum restored the negative pressure that is normally present in the intrapleural space, which would have the effect of reinflating her collapsed lung.

5. While Serena's left lung was collapsed, pulse oximetry estimated her O₂ saturation at 85%. This measurement refers to percent saturation of hemoglobin by O₂; a value of 85% means that 85% of heme groups are bound to O₂, and 15% are not bound. Percent saturation of hemoglobin is a way of approximating arterial Pₒ₂ according to the hemoglobin–O₂ dissociation curve shown in Figure 3-13. Eighty-five percent saturation corresponds to an arterial Pₒ₂ of approximately 50 mm Hg.
Serena's estimated arterial $P_{O_2}$ of 50 mm Hg is significantly lower than the normal value of 100 mm Hg—she had severe hypoxemia, which is caused by a ventilation–perfusion (V/Q) defect. Secondary to the pneumothorax, her left lung collapsed and was not being ventilated; consequently, the blood flow to her left lung became a shunt, in which there is perfusion of lung regions with no ventilation. The blood perfusing her left lung, the shunt, had the same $P_{O_2}$ as mixed venous blood, typically 40 mm Hg. This shunted blood from the left lung mixes with blood flow to the ventilated right lung, and dilutes the overall $P_{O_2}$ of systemic arterial blood (venous admixture).

### Key topics

- Hypoxemia
- Intrapleural pressure
- $O_2$–hemoglobin dissociation curve
- $O_2$ saturation
- Pneumothorax
- Venous admixture
- Ventilation–perfusion (V/Q) defect