Kidney

- 25% compensation activity
- much slower than ventilation

proximal tubule is involved in the reabsorption of the HCO3-

distal tubule is involved in fine tuning the pH

Proximal tubule-

No energy

lumen or apical side, H+/Na+ antiport (H+ out, Na+ in) NH4+/Na+ antiport (NH4+ out, NA+ in)

inside the cell- CO2 + H2O $\leftarrow \rightarrow$ H2CO3 $\leftarrow \rightarrow$ H+ + CO3-

Basolateral side- HCO3-/Na+ symporter

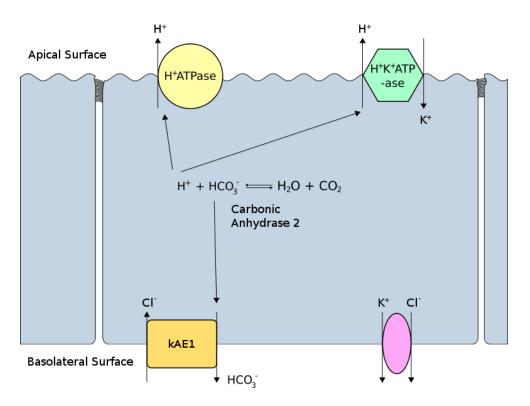
Glutamine can be converted into ammonia and alpha-ketoglutarate. Alpha-ketoglutarate can go into the blood as the ammonium ion is a buffer that can combine with H+ to form the NH4+/Na+ antiport.

Distal tubule-

- regulate pH homeostasis

During acidosis- secrete H+ and absorb HCO3- (intercalated type A cell)

During alkalosis- secrete HCO3-, adsorb H+ (intercalated type B cells)



Once inside the lumen, H+ will bind to NH3, HPO4, and HPO2, which act as buffers and decrease the concentration of H+

Hyperkalemia- too much K+ in the blood. Therefore, you must secrete K+, which is accomplished by type B cells, which secretes K+ by absorbing back H+ Hypokalemia- too little K+ in the blood. Therefore you must absorb K+, which is accompoished by type A cells, which absorbs K+ by secreting H+

Think of type B cells as the exact opposite of type A cells. In the apical side is a Cl-/HCO3- antiport and on the basolateral side is a H+ atpase and the H+/K+ antiport ATPase

COPD- renal compensation is satisfactory, but not complete. Therefore, you can see elevated H+ levels in the blood

Acidosis (much more predominant)

- respiratory acidosis \rightarrow renal compensation
 - o due to decreased alveolar ventilation
 - depress ventilation by drugs- alcohol
 - \circ \uparrow resistance (allergies or infection)
 - $\circ \downarrow$ gas exchange- fibrosis, emphysema
 - \downarrow muscle strength (motor neuron degeneration or weakening of the respiratory muscles)
 - $\circ \quad \uparrow CO2 + H2O \longleftrightarrow H2CO3 \longleftrightarrow \uparrow H+ + \uparrow HCO3-$

- metabolic acidosis \rightarrow respiratory + renal compensation
 - o intake of toxins- methanol, anti-freeze, aspirin
 - diarrhea- due to too much secretion of HCO3-, leading to acid base imbalance.
 - Too much glycolysis- during maximal exercise, pyruvate is converted to lactic acid, which decreases blood pH
 - $\circ \quad \uparrow \text{CO2} + \text{H2O} \longleftrightarrow \text{H2CO3} \longleftrightarrow \uparrow \text{H} + \downarrow \text{HCO3} -$

Alkalosis

- respiratory alkalosis- hyperventilation
 - o artificial ventilation is too high
 - o anxiety attack
 - $\circ \quad \downarrow CO2 + H2O \longleftrightarrow H2CO3 \longleftrightarrow \downarrow H+ + \downarrow HCO3-$
- metabolic alkalosis
 - o excess vomiting
 - o ingestion of too much antacids, which \uparrow HCO3-
 - $\circ \quad \downarrow \text{CO2} + \text{H2O} \longleftrightarrow \text{H2CO3} \longleftrightarrow \downarrow \text{H} + \uparrow \text{HCO3}$
 - hypoventilation can only go on for a certain extent or you will suffer hypoxia. Therefore, renal compensation is more prevalent.

In respiratory acidosis and alkalosis, you are changing your CO2 concentrations in your blood through changes in ventilation rates. As CO2 concentrations change, this drives either the formation of H+ or the decrease in H+, leading to respiratory acidosis or respiratory alkalosis.

In metabolic acidosis and alkalosis, you are changing your H+ and HCO3- concentrations directly in your blood through various metabolic reactions. Therefore, in order to compensate for this change, your body utilizes changes in CO2 concentrations through either hyper or hypoventilation to compensate for changes in your blood pH.