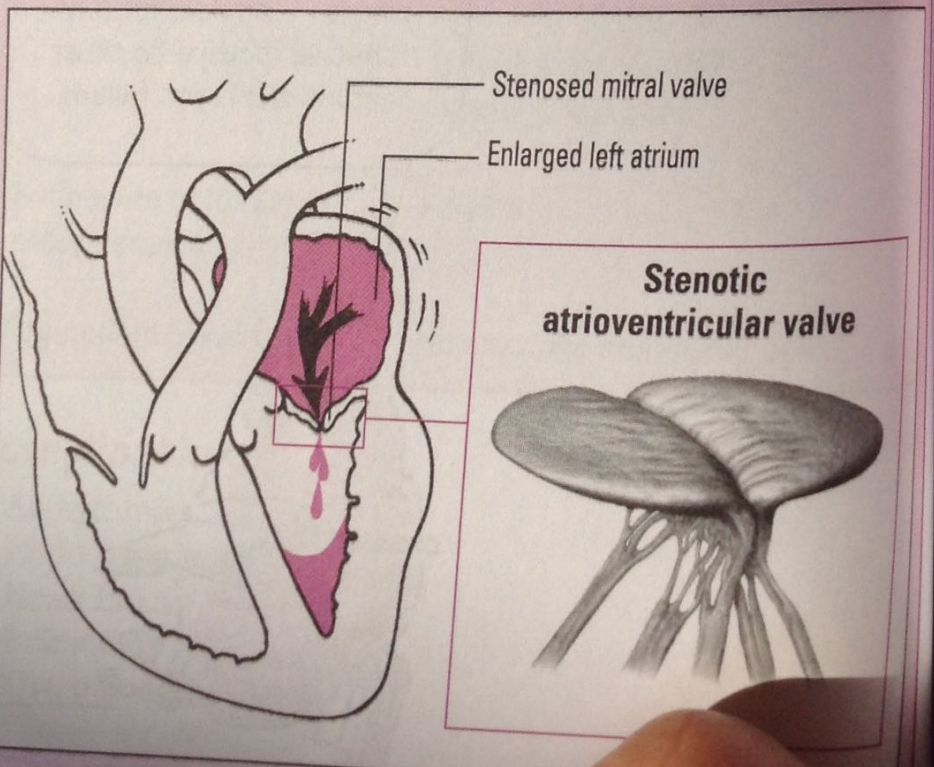
Mitral stenosis

- Narrowing of mitral valve orifice
- Valve leaflets thickened by fibrosis and calcification

I see, I see

Understanding mitral stenosis

In mitral stenosis, narrowing of the mitral valve by valvular abnormalities, fibrosis, or calcification obstructs blood flow from the left atrium to the left ventricle. Left atrial volume and pressure rise and the chamber dilates. Greater resistance to blood flow causes pulmonary hypertension, right ventricular hypertrophy, and right-sided heart failure. Inadequate filling of the left ventricle results in low cardiac output.



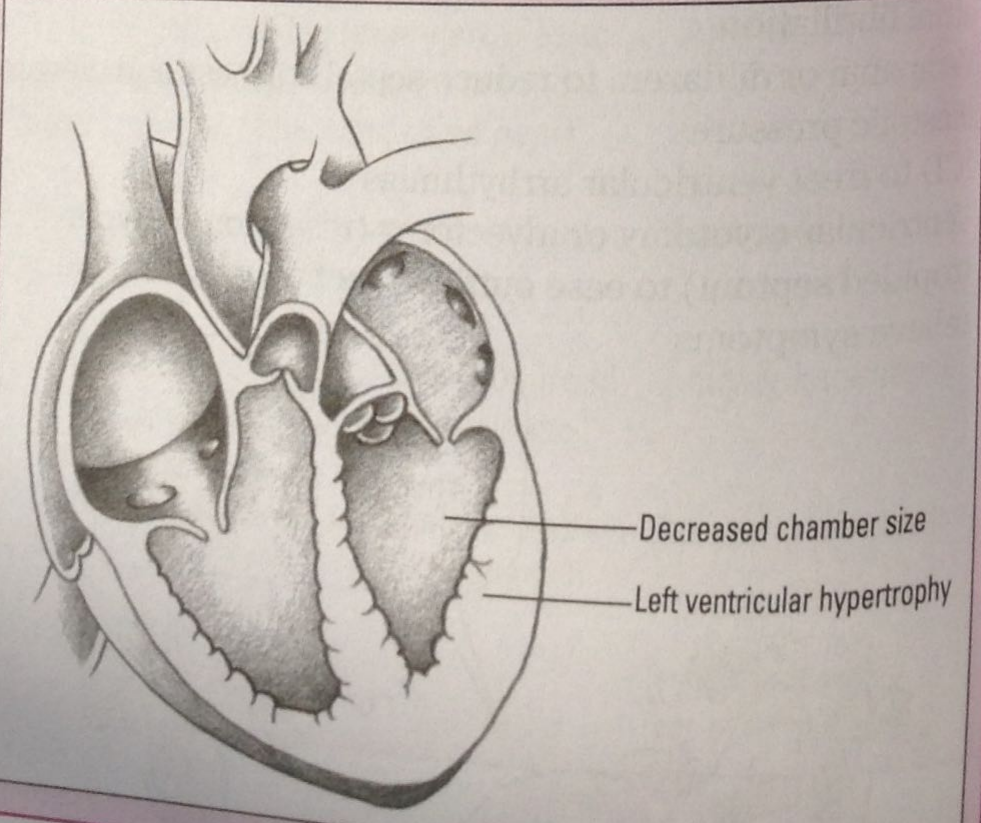
Cardiomyopathy, restrictive

- Disease of heart muscle fibers
- Irreversible if severe

I see, I see

Understanding restrictive cardiomyopathy

Restrictive cardiomyopathy is characterized by stiffness of the ventricle caused by left ventricular hypertrophy and endocardial fibrosis and thickening, which reduces the ability of the ventricle to relax and fill during diastole. The rigid myocardium fails to contract completely during systole. As a result, cardiac output falls.



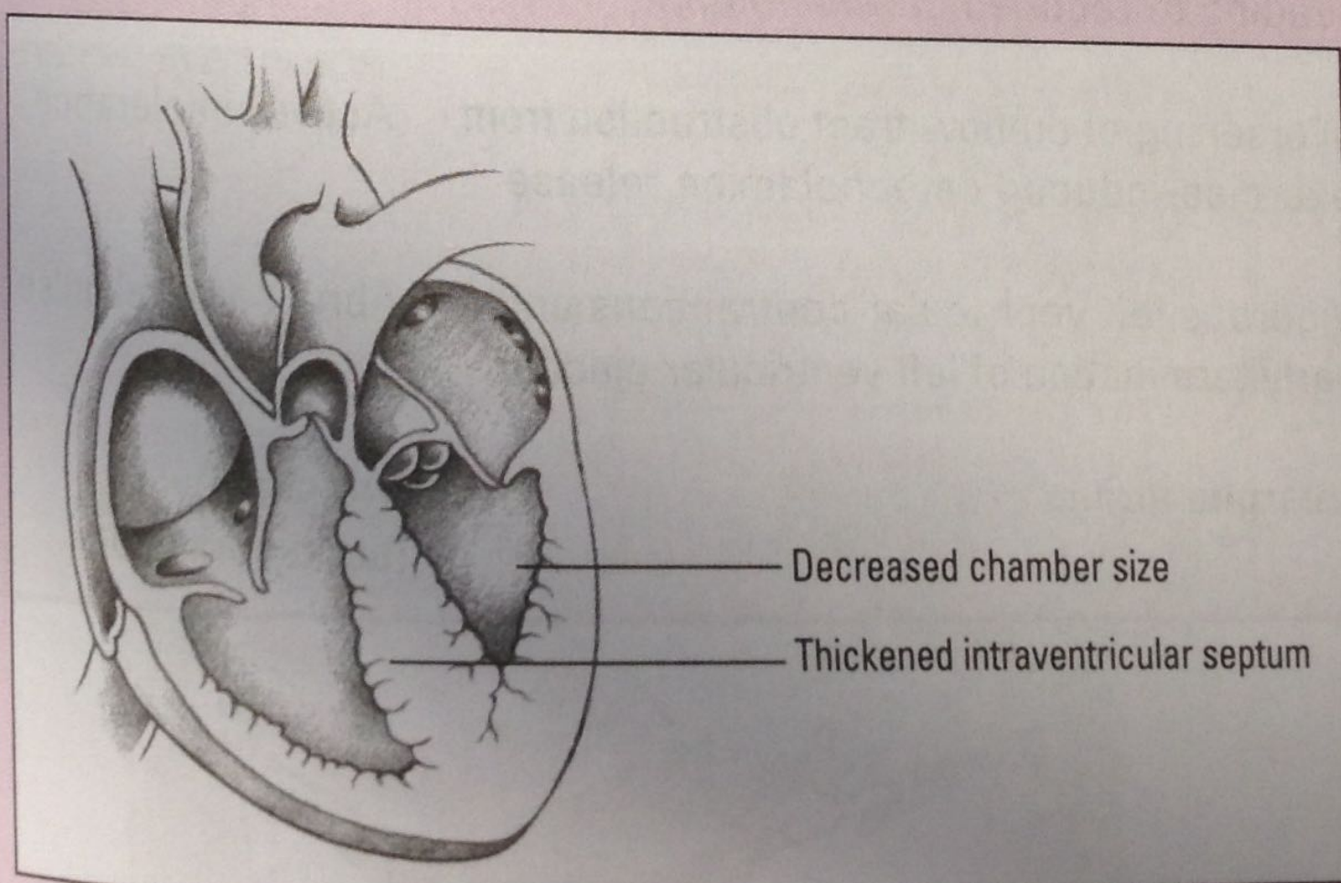
Cardiomyopathy, hypertrophic obstructive

- Primary disease of cardiac muscle and intraventricular septum
- 50% of all sudden deaths in competitive athletes are due to hypertrophic obstructive cardiomyopathy (HOCM)

I see, I see

Understanding HOCM

HOCM affects diastolic function. The left ventricle and intraventricular septum hypertrophy and become stiff, noncompliant, and unable to relax during ventricular filling. Ventricular filling decreases and left ventricular filling pressure rises, causing a rise in left atrial and pulmonary venous pressures. This rise in pressure leads to rapid, forceful contractions of the left ventricle and impaired relaxation. The forceful ejection of blood draws the anterior leaflet of the mitral valve to the intraventricular septum, which causes early closure of the outflow tract, decreasing ejection fraction.



Cardiomyopathy, dilated

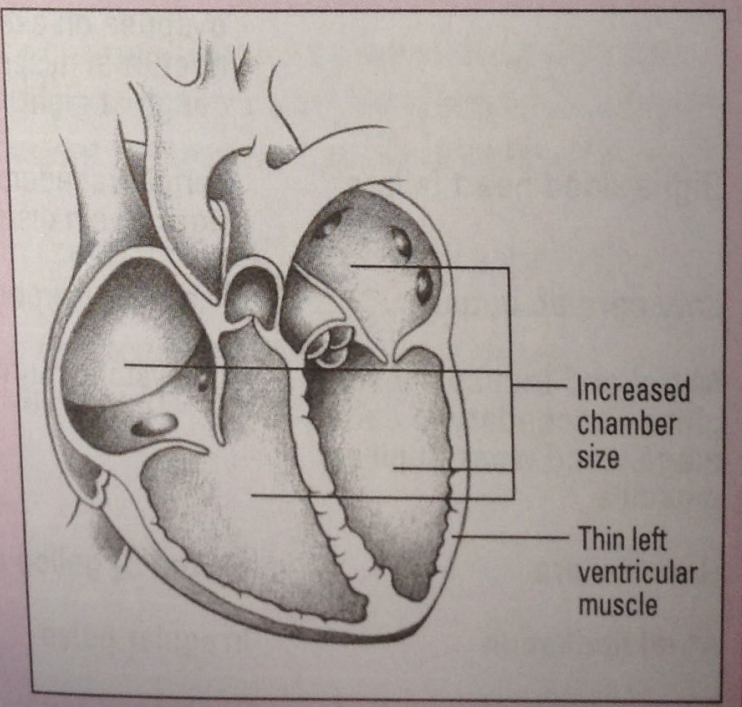
- Disease of heart muscle fibers
- Usually not diagnosed until advanced stage; prognosis generally poor

I see, I see

Understanding dilated cardiomyopathy

In dilated cardiomyopathy, extensively damaged myocardial muscle fibers reduce contractility of the left ventricle. As systolic function declines, stroke volume, ejection fraction, and cardiac output fall. The sympathetic nervous system is stimulated to increase heart rate and contractility. The kidneys are stimulated to retain sodium and water to maintain cardiac output, and vasoconstriction also occurs as the renin-angiotensin system is stimulated.

When compensatory mechanisms can no longer maintain cardiac output, the heart begins to fail. Left ventricular dilation occurs as venous return and systemic vascular resistance rise. Eventually, the atria also dilate as more work is required to pump blood into the full ventricles. Cardiomegaly occurs due to dilation of the atria and ventricles.



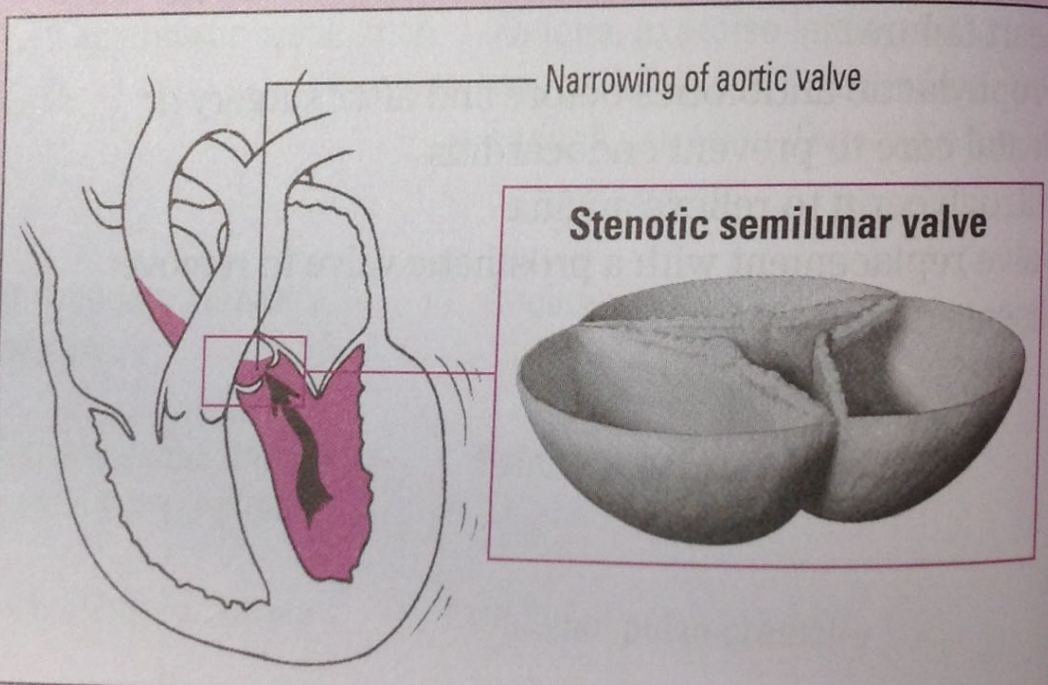
Aortic stenosis

- Narrowing of aortic valve
- Classified as acquired or rheumatic
- Classic triad of angina pectoris, syncope, and dyspnea

I see, I see

Understanding aortic stenosis

Stenosis of the aortic valve results in impedance to forward blood flow. The left ventricle requires greater pressure to open the aortic valve. The added workload increases the demand for oxygen, and diminished cardiac output causes poor coronary artery perfusion, ischemia of the left ventricle, left ventricular hypertrophy, and left-sided heart failure.



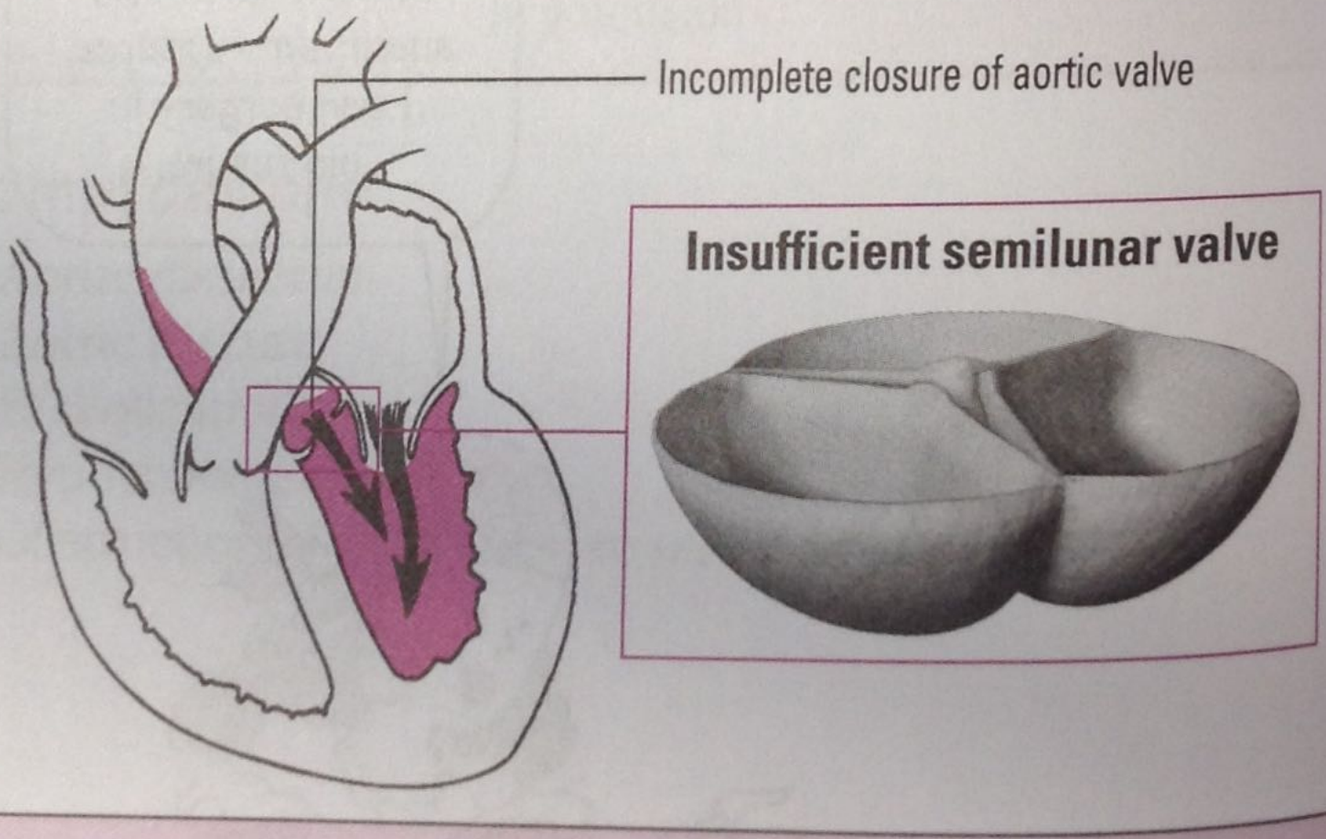
Aortic insufficiency

- Incomplete closure of aortic valve
- Usually caused by scarring or retraction of valve leaflets

I see, I see

Understanding aortic insufficiency

In aortic insufficiency, blood flows back into the left ventricle during diastole, causing fluid overload in the ventricle, which dilates and hypertrophies. The excess volume causes fluid overload in the left atrium and, lastly, the pulmonary system. Left-sided heart failure and pulmonary edema eventually result.



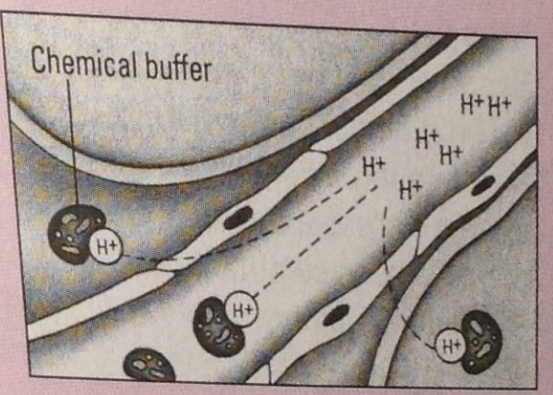
Metabolic acidosis

Acid-base imbalance characterized by excess acid and deficient HCO_3^-

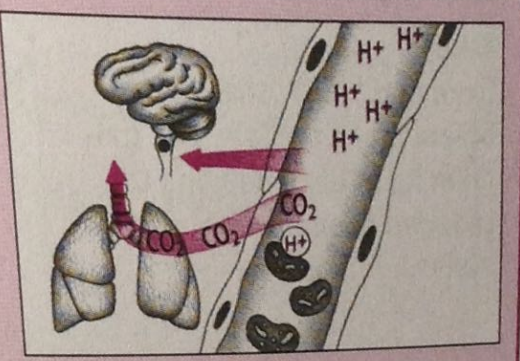
I see, I see

Understanding metabolic acidosis

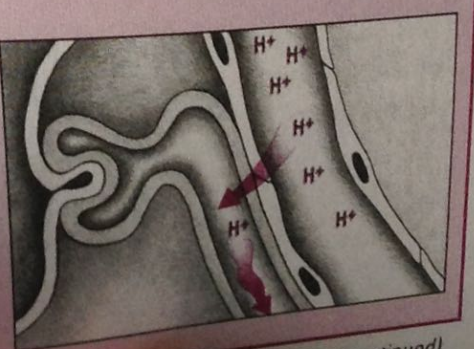
Step 1
As H^+ start to accumulate in the body, chemical buffers in the cells and extracellular fluid bind with them.



Step 2
Excess H^+ that the buffers can't bind with decrease the pH and stimulate chemoreceptors in the medulla to increase the respiratory rate. This increased rate lowers the partial pressure of arterial carbon dioxide ($Paco_2$), which allows more H^+ to bind with HCO_3^-.



Step 3
Healthy kidneys try to compensate for acidosis by secreting excess H^+ into the renal tubules.

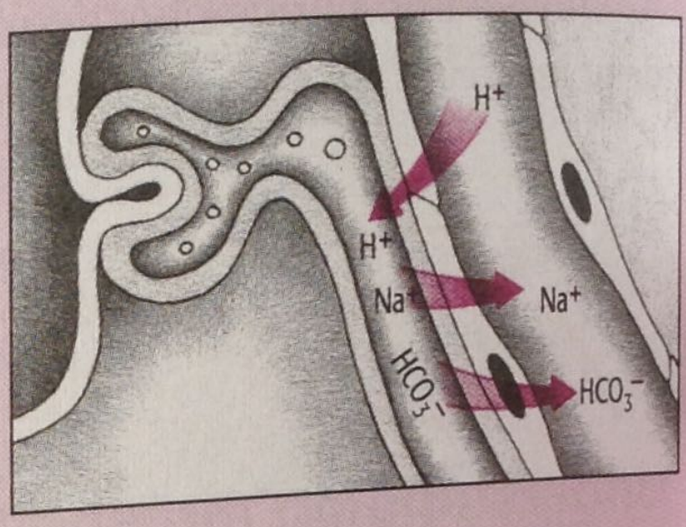


(continued)

Understanding metabolic acidosis *(continued)*

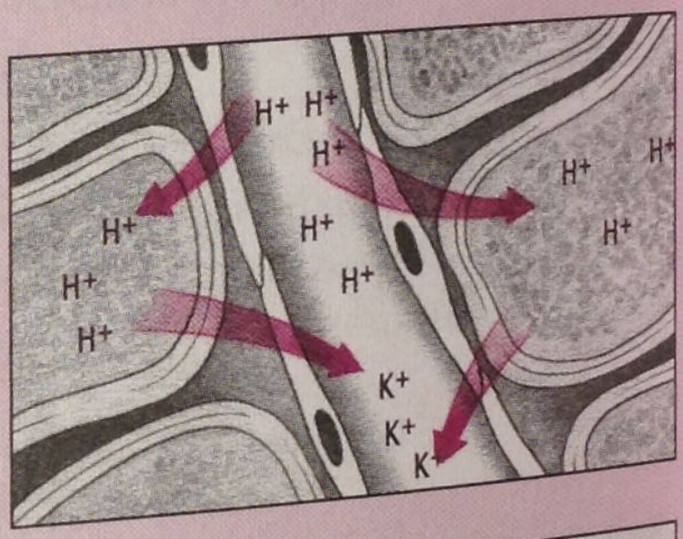
Step 4

Each time an H^+ is secreted into the renal tubules, a sodium ion (Na^+) and an HCO_3^- are absorbed from the tubules and returned to the blood.



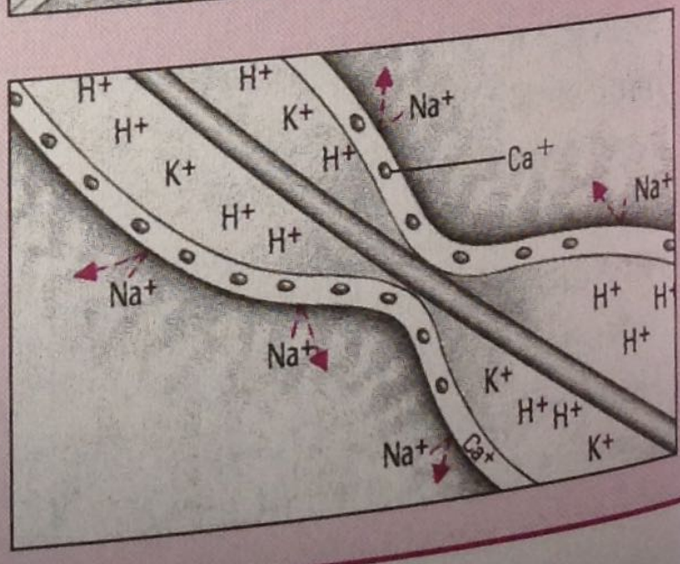
Step 5

Excess H^+ in the extracellular fluid diffuse into cells. To maintain the balance of the charge across the membrane, the cells release potassium (K^+) into the blood.



Step 6

Excess H^+ alter the normal balance of K^+, Na^+, and calcium ions (Ca^+), leading to reduced excitability of nerve cells.



What causes it

- Excessive production of metabolic acids: diabetic ketoacidosis, lactic acidosis, malnutrition, starvation, chronic alcoholism

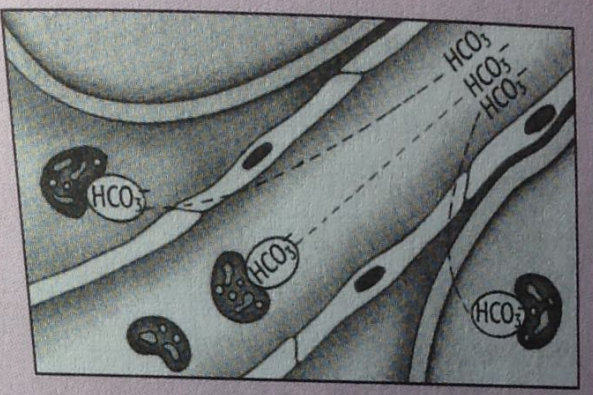
Metabolic alkalosis

Acid-base imbalance characterized by decreased amounts of acid and increased amounts of base HCO_3^-

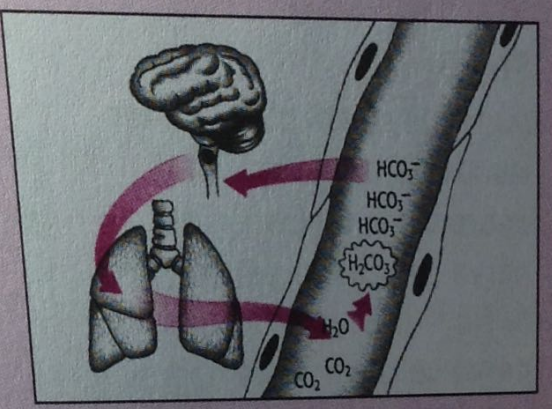
I see, I see

Understanding metabolic alkalosis

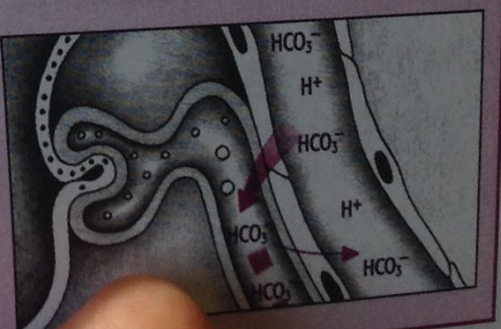
Step 1
As HCO_3^- start to accumulate in the body, chemical buffers bind with the ions.



Step 2
Excess HCO_3^- that don't bind with chemical buffers elevate serum pH levels, which in turn depress chemoreceptors in the medulla. This causes a decrease in respiratory rate, which increases the $Paco_2$. The additional CO_2 combines with water to form carbonic acid (H_2CO_3).



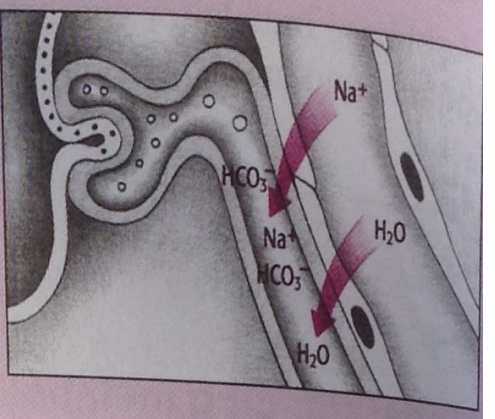
Step 3
When the HCO_3^- level exceeds 28 mEq/L, the renal glomeruli can no longer reabsorb excess HCO_3^-. That excess HCO_3^- is excreted in the urine; H^+ are retained.



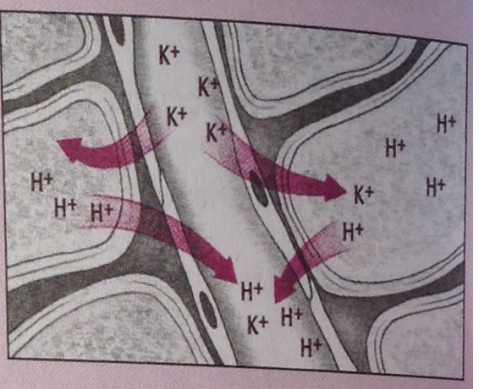
(continued)

Understanding metabolic alkalosis *(continued)*

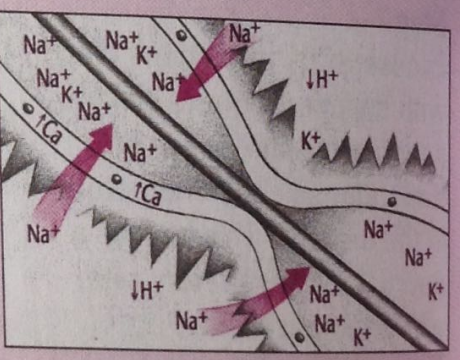
Step 4
To maintain electrochemical balance, the kidneys excrete excess Na^+, H_2O, and HCO_3^-.



Step 5
Lowered H^+ levels in the extracellular fluid cause the ions to diffuse out of the cells. To maintain the balance of charge across the cell membrane, extracellular K^+ move into the cells.



Step 6
As H^+ levels decline, Ca ionization decreases. That decrease in ionization makes nerve cells more permeable to Na^+. Na^+ moving into nerve cells stimulate neural impulses and produce overexcitability of the peripheral system and CNS.



What causes it

- Loss of acid, retention of base, or renal mechanisms associated with low serum levels of K and chloride
- Critical acid loss
  - Chronic vomiting

asogastric tube drainage or lavage without adequate
ctrolyte replacement
Fistulas
Use of steroids and certain diuretics (furosemide, thiazides,
acrynic acid)
Massive blood transfusions
Cushing's disease, primary hyperaldosteronism, and
artter's syndrome
Excessive HCO_3^- retention (causing chronic hypercapnia)
- Excessive intake of bicarbonate of soda or other
ntacids or absorbable alkali
- Excessive amounts of I.V. fluids with high concentrations
of HCO_3^- or lactate
- Alterations in extracellular electrolyte levels
- Low plasma K, causing increased H^+ excretion by kidneys

What's happening?!

What happens in metabolic alkalosis

Change	Signs and symptoms
Decreased cerebral perfusion	Irritability, picking at bedclothes (carphology), twitching, seizures, confusion
Hypokalemia	Arrhythmias
Compensatory response when severe respiratory alkalosis leads to respiratory failure	Slow, shallow respirations
Diminished peripheral blood flow (during repeated blood pressure checks)	Carpal spasm in hand (Trousseau's sign, possible sign of impending tetany)

Complications

- Coma
- Seizures

How it's treated

- Potassium chloride and normal saline solution (except in heart failure) to replace losses from gastric drainage
- Discontinuation of diuretics and supplementary potassium chloride to prevent further electrolyte loss
- Oral or I.V. acetazolamide to enhance renal HCO_3^- excretion and correct metabolic alkalosis without rapid volume expansion