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838K
-  **Human Biology Volume 71 issue 3 1999 [doi 10.2307_41465747] M.L. CHOONG, S.K. SETHI and E.S.C. KOAY -- Effects of Intragenic Variability at 3 Polymorphic Sites of the Apolipoprotein B Gene on Serum .pdf**
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-  **Interdisciplinary Sciences- Computational Life Sciences Volume issue 2017 [doi 10.1007_s12539-017-0258-z]
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Guyton and Hall Textbook of Medical Physiology-Elsevier (2020).pdf

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figures-14

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Table 31-1 pH and H⁺ Concentration of Body Fluids

	H ⁺ Concentration (mEq/L)	pH
Extracellular Fluid		
• Arterial blood	• 4.0 × 10 ⁻⁸	• 7.40
• Venous blood	• 4.5 × 10 ⁻⁸	• 7.35
• Interstitial fluid	• 4.5 × 10 ⁻⁸	• 7.35
Intracellular fluid		
	1 × 10 ⁻³ to 4 × 10 ⁻⁵	6.0-7.4
Urine	3 × 10 ⁻² to 1 × 10 ⁻⁵	4.5-8.0
Gastric HCl	160	0.8

cmk2.jpg
36K

following formula (H⁺ concentration [H⁺] is expressed in equivalents per liter):

$$\text{pH} = \log \frac{1}{[\text{H}^+]} = -\log [\text{H}^+]$$

For example, normal [H⁺] is 40 nEq/L (0.00000004 Eq/L). Therefore, the normal pH is as follows:

$$\text{pH} = -\log[0.00000004]$$

$$\text{pH} = 7.4$$

cmk1.jpg
27K

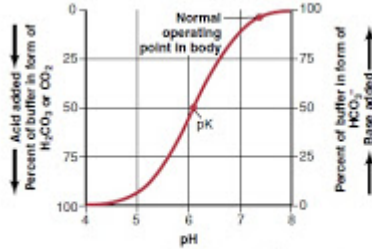
cmk3.jpg
34K

$$pH = pK + \log \frac{HCO_3^-}{(0.03 \times P_{CO_2})} \quad (7)$$

For the bicarbonate buffer system, the pK is 6.1, and Equation 7 can be written as follows:

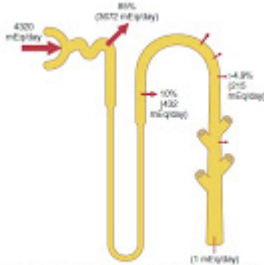
$$pH = 6.1 + \log \frac{HCO_3^-}{0.03 \times P_{CO_2}} \quad (8)$$

Equation 8 is the Henderson-Hasselbalch equation and, with it, one can calculate the pH of a solution if the molar concentration of HCO_3^- and the P_{CO_2} are known.



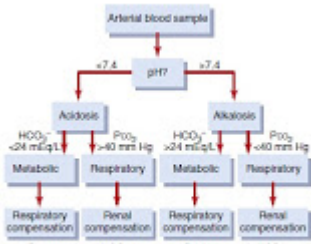
cmk4.jpg
45K

Figure 31-1. Titration curve for bicarbonate buffer system showing the pH of extracellular fluid when the percentages of buffer in the form of HCO_3^- and CO_2 (or H_2CO_3) are altered.



cmk5.jpg
51K

Figure 31-4. Reabsorption of HCO_3^- in different segments of the renal tubule. The percentages of the filtered load of HCO_3^- absorbed by the various tubular segments are shown, as well as the number of milliequivalents reabsorbed per day under normal conditions.



cmk7.jpg
63K

Figure 31-10. Analysis of simple acid-base disorders. If the compensatory responses are markedly different from those shown at the bottom of the figure, one should suspect a mixed acid-base disorder.

Table 31-3 Extracellular Fluid Characteristics of Primary Acid-Base Disturbances*

	pH	H ⁺	P _{CO₂}	HCO ₃ ⁻
Normal	7.4	40 mEq/L	40 mm Hg	24 mEq/L
Respiratory acidosis	I	I	II	I
Respiratory alkalosis	I	I	II	I
Metabolic acidosis	I	I	I	II
Metabolic alkalosis	I	I	I	II

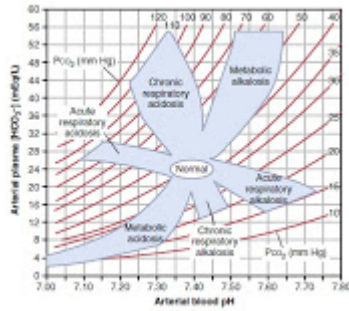
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*The primary event is indicated by the double arrows (I) or (II). Note that respiratory acid-base disorders are initiated by an increase or decrease in P_{CO_2} , whereas metabolic disorders are initiated by an increase or decrease in HCO_3^- .

cmk10.jpg
48K

Table 31-4 Metabolic Acidosis Associated With Normal or Increased Plasma Anion Gap

Increased Anion Gap (Normochloremia)	Normal Anion Gap (Hyperchloremia)
Diabetes mellitus (ketoacidosis)	Diarrhea
Lactic acidosis	Renal tubular acidosis
Chronic renal failure	Carbonic anhydrase inhibitors
Aspirin (acetylsalicylic acid) poisoning	Addison disease
Methanol poisoning	
Ethylene glycol poisoning	
Starvation	



cmk8.jpg
92K

Use of Anion Gap to Diagnose Acid-Base Disorders

The concentrations of anions and cations in plasma must be equal to maintain electrical neutrality. Therefore, there is no real anion gap in the plasma. However, only certain cations and anions are routinely measured in the clinical laboratory. The cation normally measured is Na⁺, and the anions are usually Cl⁻ and HCO₃⁻. The anion gap—which is only a diagnostic concept—is the difference between unmeasured anions and unmeasured cations and is estimated as follows:

$$\text{Plasma anion gap} = [\text{Na}^+] - [\text{HCO}_3^-] - [\text{Cl}^-]$$

$$= 144 - 24 - 108 = 12 \text{ mEq/L}$$

The anion gap will increase if unmeasured anions rise or if unmeasured cations fall. The most important unmeasured cations include calcium, magnesium, and potassium, and the major unmeasured anions are albumin, phosphate, sulfate, and other organic anions. Usually, the unmeasured anions exceed the unmeasured cations, and the anion gap ranges between 8 and 16 mEq/L.

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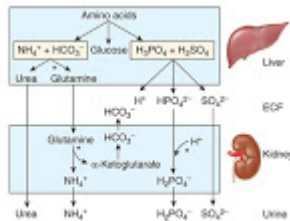


FIGURE 39-5 Role of the liver and kidneys in the handling of metabolically produced acid loads. Sites where regulation occurs are indicated by asterisks.

cmk2.jpg
105K

	H ⁺ Concentration		
	mEq/L	mol/L	pH
Gastric HCl	150	0.15	0.8
Maximal urine acidity	0.03	3 × 10 ⁻⁴	4.5
Plasma	Extreme acidosis	0.0001	1 × 10 ⁻⁷
	Normal	0.00004	4 × 10 ⁻⁸
	Extreme alkalosis	0.00002	2 × 10 ⁻⁸
Pancreatic juice	0.00001	1 × 10 ⁻⁸	8.0

cmk1.jpg
55K

TABLE 39-2 Principal buffers in body fluids.

Blood	H ₂ CO ₃ ⇌ H ⁺ + HCO ₃ ⁻ HProt ⇌ H ⁺ + Prot ⁻ HHb ⇌ H ⁺ + Hb ⁻
Interstitial fluid	H ₂ CO ₃ ⇌ H ⁺ + HCO ₃ ⁻
Intracellular fluid	HProt ⇌ H ⁺ + Prot ⁻ H ₂ PO ₄ ⇌ H ⁺ + HPO ₄ ²⁻

cmk3.jpg
40K

cmk4.jpg
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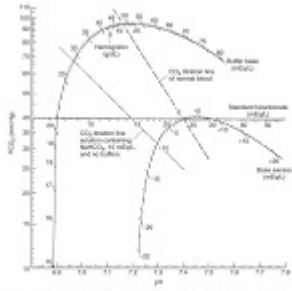


FIGURE 20-6 Sigmoidal-Andersen curve concept. (Adapted with permission of H. Siggard-Andersen and K. Andersen, Copenhagen, Denmark.)



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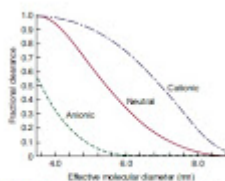
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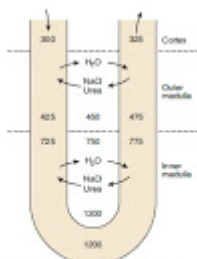
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FIGURE 38-5 Effect of electric charge on the fractional clearance of dextran molecules of various sizes in rats. The negative charges in the glomerular membrane retard the passage of negatively charged molecules (anionic, dashed line) and facilitate the passage of positively charged molecules (cationic, dotted line). (Reproduced with permission from Borner SK, Bornerin R. The renal circulation. *Resp Phys* (1978) 13:252)



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33K

FIGURE 38-17 Operation of the vasa recta as countercurrent exchangers in the kidney. H_2O and urea diffuse out of the ascending limb of the vessel and into the descending limb, whereas water diffuses out of the descending and into the ascending limb of the vasa recta loop.

TABLE 38-3 Agents causing contraction or relaxation of mesangial cells.

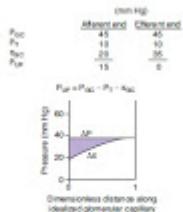
Contraction	Relaxation
Endothelin	AMP
Angiotensin II	Dopamine
Vasopressin	PGI ₂
Norepinephrine	cAMP
Platelet-activating factor	
Platelet-derived growth factor	
Thromboxane A ₂	
PGI ₂	
Leukotrienes C ₄ and D ₄	
Histamine	

2.jpg
26K

TABLE 38-4 Factors affecting the GFR.

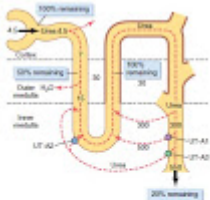
Changes in renal blood flow
Changes in glomerular capillary hydrostatic pressure
Changes in systemic blood pressure
Afferent or efferent arteriolar constriction
Changes in hydrostatic pressure in Bowman's capsule
Urinary obstruction
Edema of kidney inside tight renal capsule
Changes in concentration of plasma proteins: dehydration, hypoproteinemia, etc. (minor factors)
Changes in $f_{\text{H}_2\text{O}}$
Changes in glomerular capillary permeability
Changes in effective filtration surface area

3.jpg
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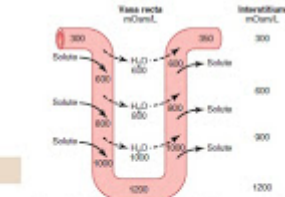
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FIGURE 38-6 Hydrostatic pressure (P_{GC}) and oncotic pressure (P_{GC}) in a glomerular capillary in the rat. P_{GC} pressure in Bowman's capsule, P_{BC} net filtration pressure. It is normally negligible, so $\Delta\pi = P_{GC} - P_{GC} - P_{GC}$ (reproduced with permission from Moore JE, Malins SB, Sirois JM. Current concepts of sodium chloride and water transport by the mammalian nephron. *Wald J Med Biol* 1973;13:22).



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Figure 28-6. Reabsorption of water and NaCl from the tubules. The reabsorption of water and NaCl from the tubules is the major mechanism for conserving water and electrolytes. The reabsorption of water and NaCl from the tubules is the major mechanism for conserving water and electrolytes. The reabsorption of water and NaCl from the tubules is the major mechanism for conserving water and electrolytes.



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Figure 28-7. Countercurrent exchange in the vasa recta. Plasma flowing down the descending limb of the vasa recta becomes more hyperosmotic because of diffusion of water out of the blood and diffusion of solutes from the renal interstitial fluid into the blood. In the ascending limb of the vasa recta, solutes diffuse back into the interstitial fluid and water diffuses back into the vasa recta. Large amounts of solute would be lost from the renal medulla without the U shape of the vasa recta capillaries. (Numerical values are in milliosmoles per liter.)

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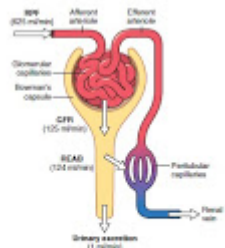
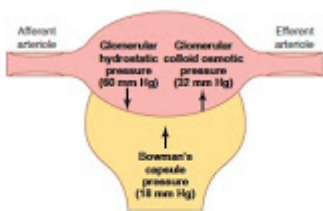


Figure 27-3. Average values for renal plasma flow (RPF), glomerular filtration rate (GFR), tubular reabsorption (TRAB), and urine flow rate. RPF is equal to renal blood flow \times 0.91 (hematocrit). Note that GFR averages about 20% of the RPF, while urine flow rate is less than 1% of the GFR. Therefore, more than 99% of the fluid filtered is normally reabsorbed. The filtration fraction is the GFR/RPF.

Table 27-1 Filterability of Substances by Glomerular Capillaries Based on Molecular Weight

Substance	Molecular Weight	Filterability
Water	18	1.0
Sodium	23	1.0
Glucose	180	1.0
Inulin	5500	1.0
Myoglobin	17,000	0.75
Albumin	69,000	0.005

9.jpg
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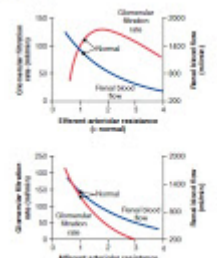


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Net filtration pressure (10 mm Hg) = Glomerular hydrostatic pressure (90 mm Hg) - Bowman's capsule pressure (18 mm Hg) - Glomerular oncotic pressure (32 mm Hg)

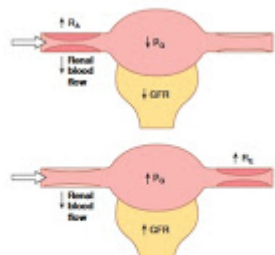
Figure 27-4. Summary of forces causing filtration by the glomerular capillaries. The values shown are estimates for healthy humans.

Table 27-2 summarizes the factors that can decrease GFR.



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Figure 27-3. Effect of change in afferent arteriole resistance or efferent arteriole resistance on glomerular filtration rate and renal blood flow.



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Figure 27-4. Effect of increases in afferent arteriole resistance (R_a , top panel) or efferent arteriole resistance (R_e , bottom panel) on renal blood flow, glomerular hydrostatic pressure (P_G), and glomerular filtration rate (GFR).

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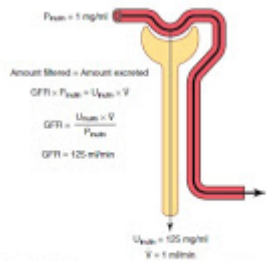


Figure 28-20. Measurement of glomerular filtration rate (GFR) from the renal clearance of inulin. Inulin is freely filtered by the glomerular capillaries but is not reabsorbed by the renal tubules. P_{inulin} , plasma inulin concentration; U_{inulin} , urine inulin concentration; V , urine flow rate.



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