

# Fiziologia singelui -I

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2008/2009

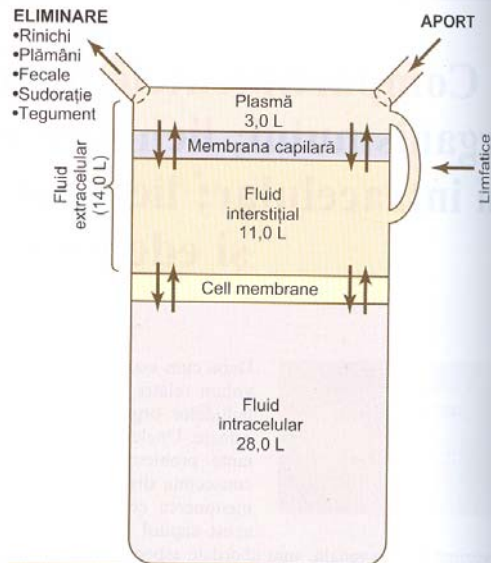


Figura 25-1

Privire de ansamblu asupra reglării echilibrului hidric al organismului, cu ilustrarea principalelor compartimente lichidiene, precum și a membranelor care le delimitează. Valorile indicate corespund unui individ cu greutatea de 70 kg.

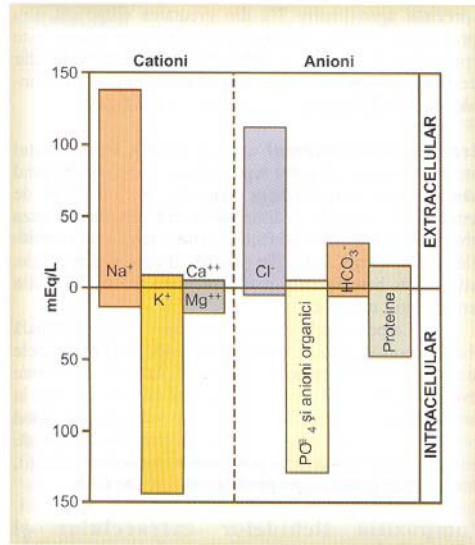


Figura 25-2

Principali cationi și anioni din lichidele intracelular și extracelular. În cazul ionilor de calciu și de magneziu se iau în considerare concentrațiile însumate. Concentrațiile prezentate corespund ionilor liberi și ionilor complecși considerați împreună.

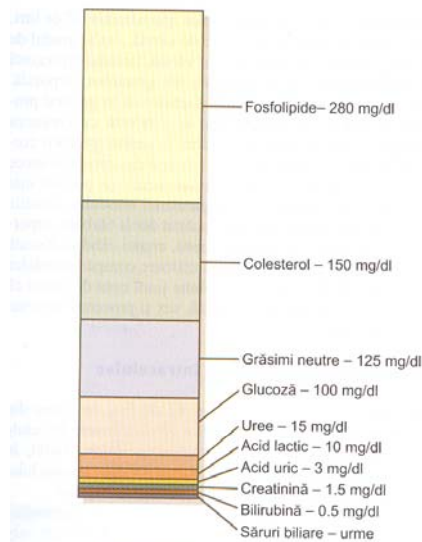
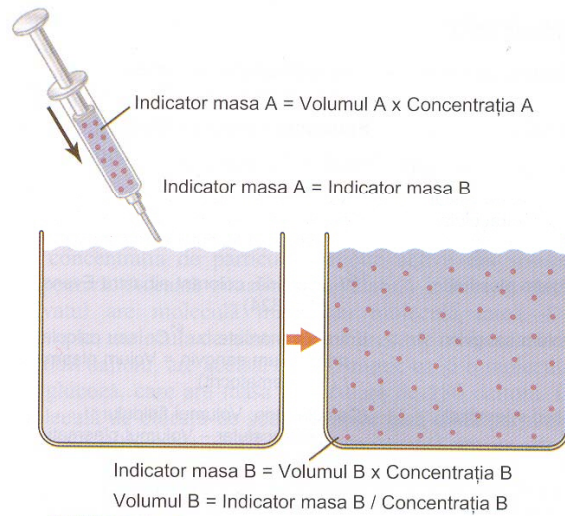


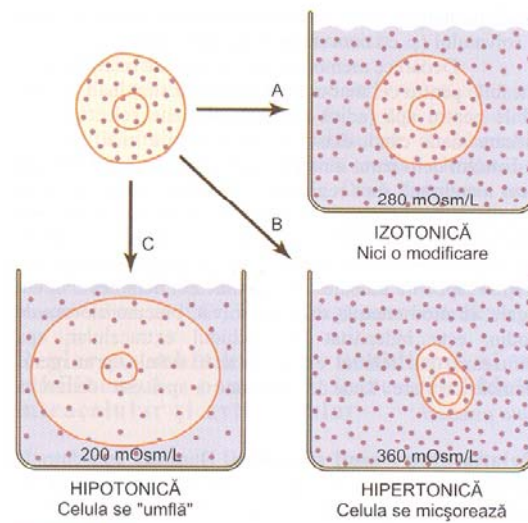
Figura 25-3

Substanțe plasmatică care nu sunt electroliti.



**Figura 25-4**

Măsurarea volumelor de lichid cu ajutorul metodei diluției substanței indicatoare.



**Figura 25-5**

Efectul soluțiilor izotonice (A), hipertonică (B) și hipotonice (C) asupra volumului celular.

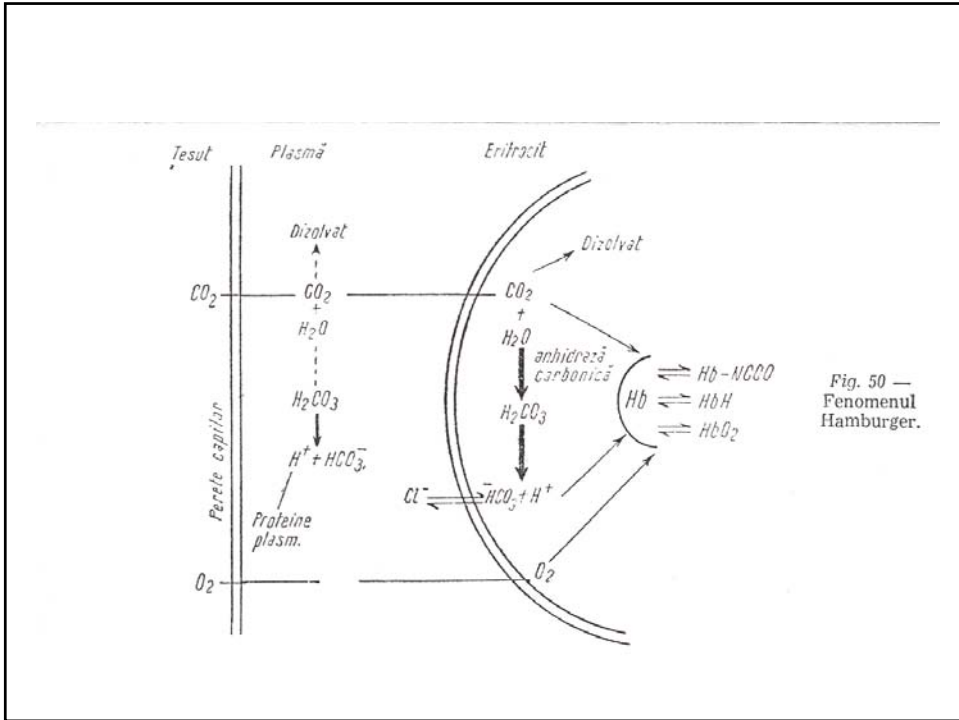


Fig. 50 — Fenomenul Hamburger.

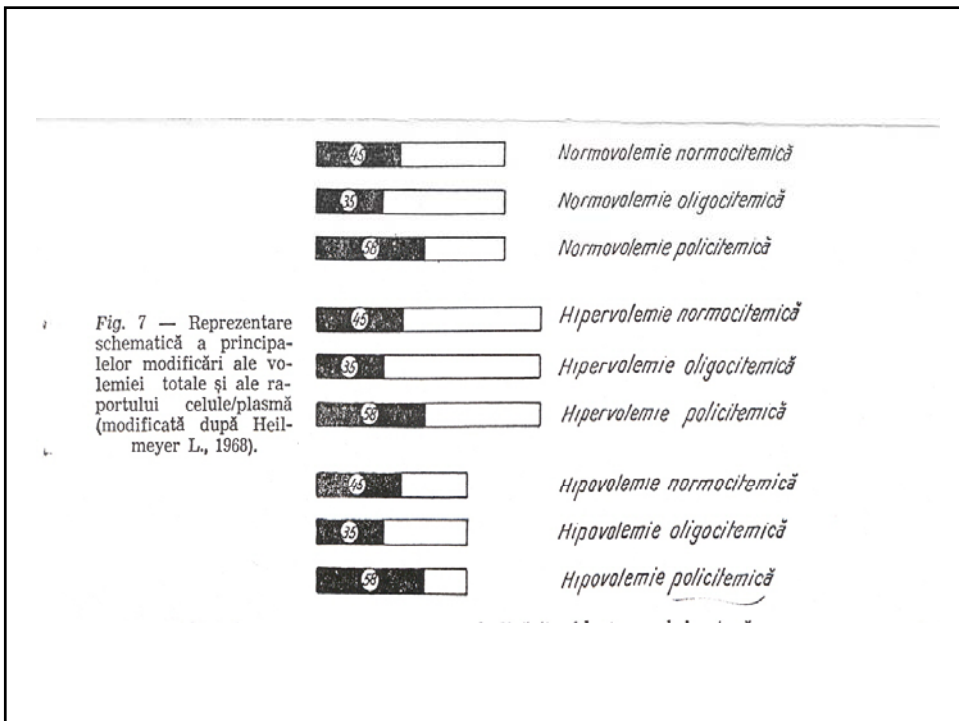
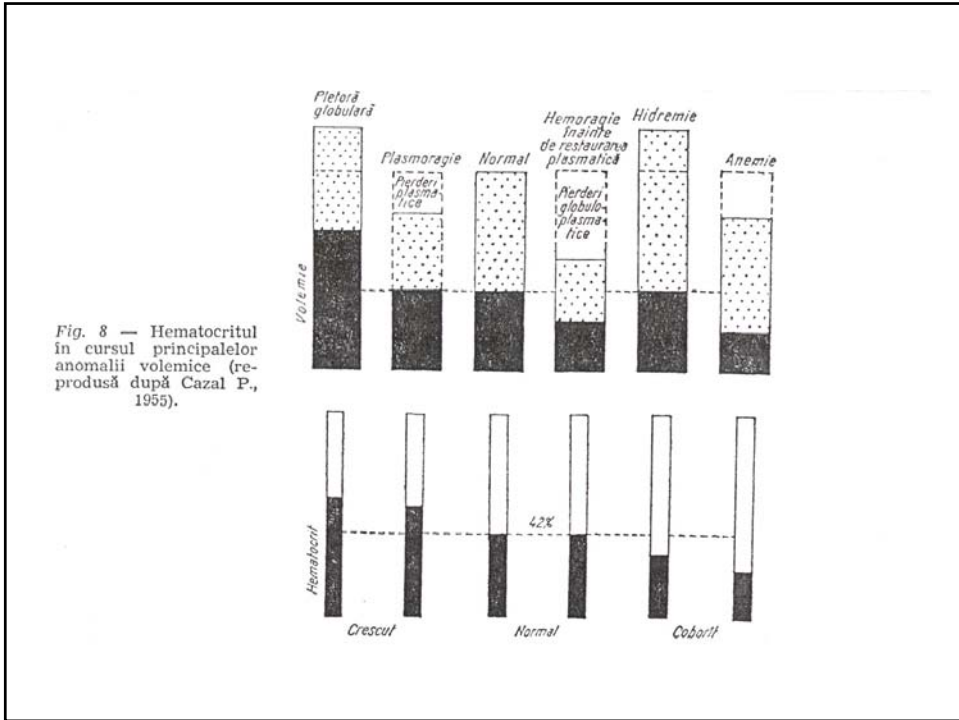
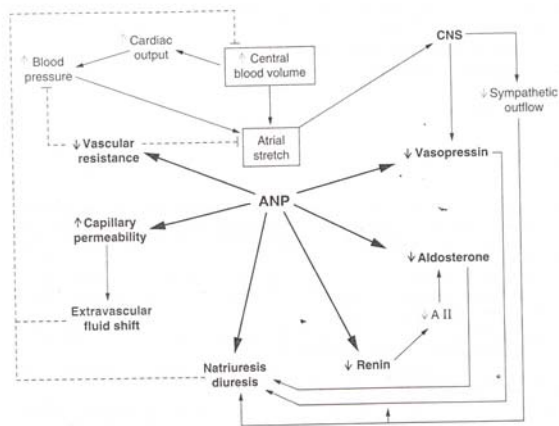


Fig. 7 — Reprezentare schematică a principalelor modificări ale volumiei totale și ale raportului celule/plasmă (modificată după Heilmeyer L., 1968).

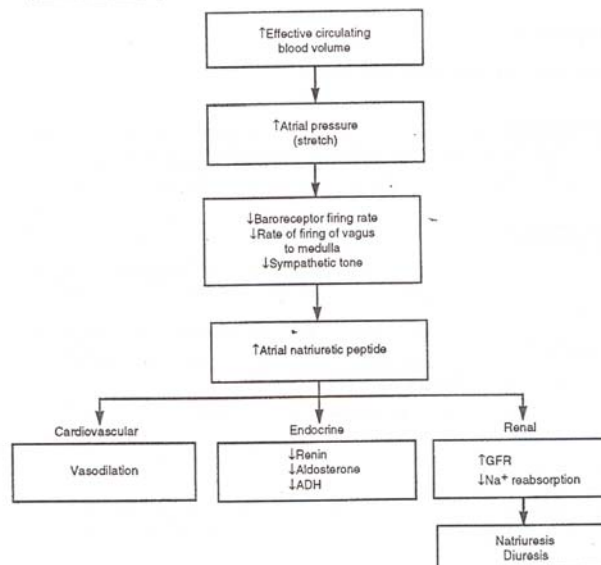


	Lichid extracelular	Lichid intracelular
Na <sup>+</sup>	142 mEq/L	10 mEq/L
K <sup>+</sup>	4 mEq/L	140 mEq/L
Ca <sup>++</sup>	2,4 mEq/L	0,0001 mEq/L
Mg <sup>++</sup>	1,2 mEq/L	58 mEq/L
Cl <sup>-</sup>	103 mEq/L	4 mEq/L
HCO <sub>3</sub> <sup>-</sup>	28 mEq/L	10 mEq/L
Fosfați	4 mEq/L	75 mEq/L
SO <sub>4</sub> <sup>-</sup>	1 mEq/L	2 mEq/L
Glucoză	90 mg/dl	0 pînă la 20 mg/dl
Aminoacizi	30 mg/dl	200 mg/dl ?
Colesterol		
Fosfolipide	} 0,5 gm/dl	} 2 pînă la 95 gm/dl
Grăsimi neutre		
Po <sub>2</sub>	36 mm Hg	20 mm Hg ?
Pco <sub>2</sub>	46 mm Hg	50 mm Hg ?
pH	7,4	7,0
Proteine	2 gm/dl (5mEq/L)	16 gm/dl (40mEq/L)

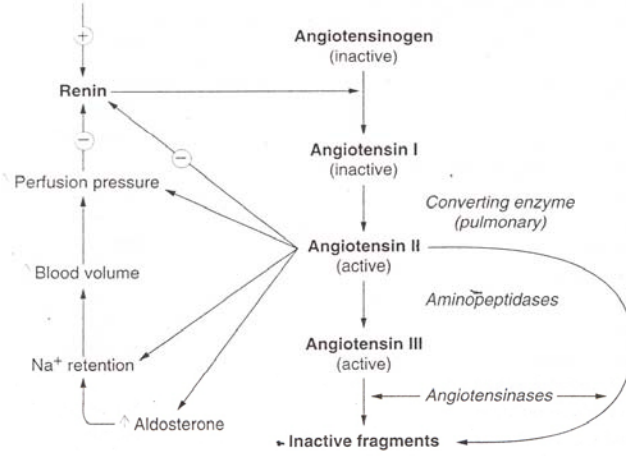
Fig.4-1. Compoziția chimică a lichidului extracelular și a celui intracelular.



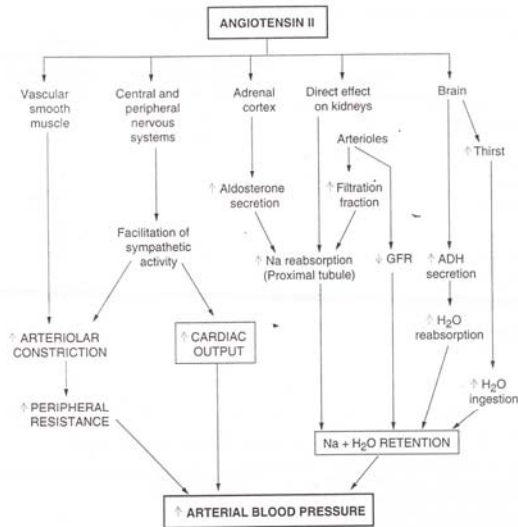
**FIGURE 31-2.** Schematic representation of the regulation of atrial natriuretic peptide (or factor)[ANP(ANF)] secretion, its major target organ actions, and its interrelationship with reflex inhibition of central sympathetic outflow during expansion of central blood volume. *Dashed lines* indicate probable negative feedback signals. Although unproved, it is likely that ANP-induced inhibition of aldosterone and renin release, natriuresis, and extravascular fluid shifts occur at near physiologic concentrations of the hormone. The latter two effects, by diminishing venous return to the heart, would provide negative feedback signals for ANP secretion. Inhibition of vasopressin release and decreased vascular resistance are probably not major effects under normal physiologic conditions. Antagonism of vasoconstrictor action is probably significant in certain pathologic states, and the resultant decrease in vascular resistance would tend to counter the rise in blood pressure due to increased cardiac output and might also have negative feedback effects on atrial stretch. *A II* = angiotensin II. (Reprinted with permission from Windhager EE (ed): *Handbook of Physiology*, vol II, section 8, Renal Physiology, Chart 33, Atrial Natriuretic Factor. New York, Oxford University Press, 1992, p 1632.)



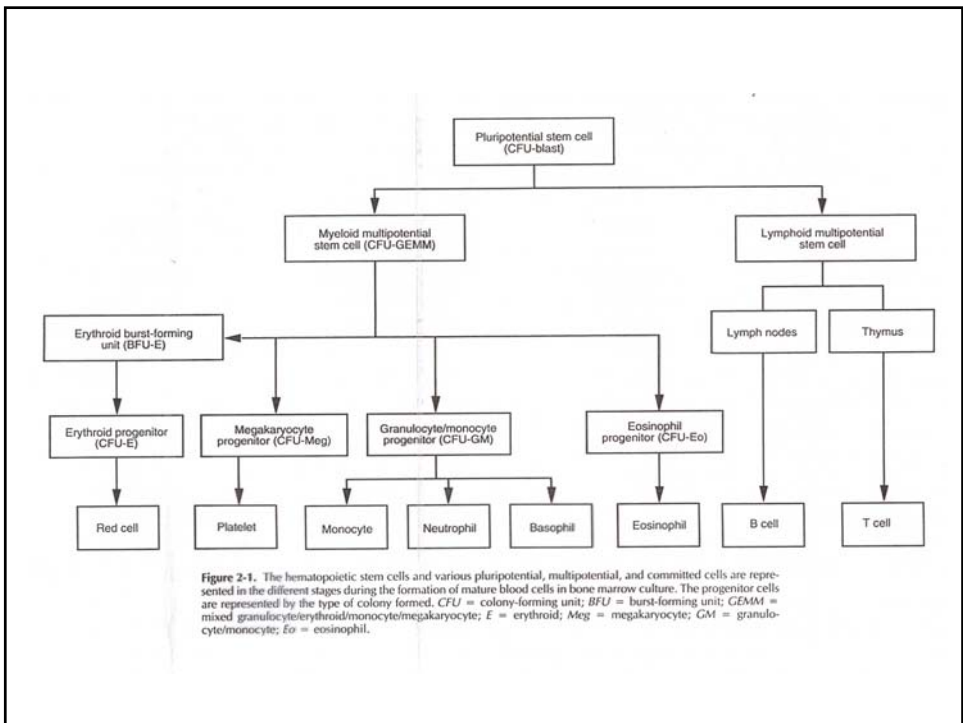
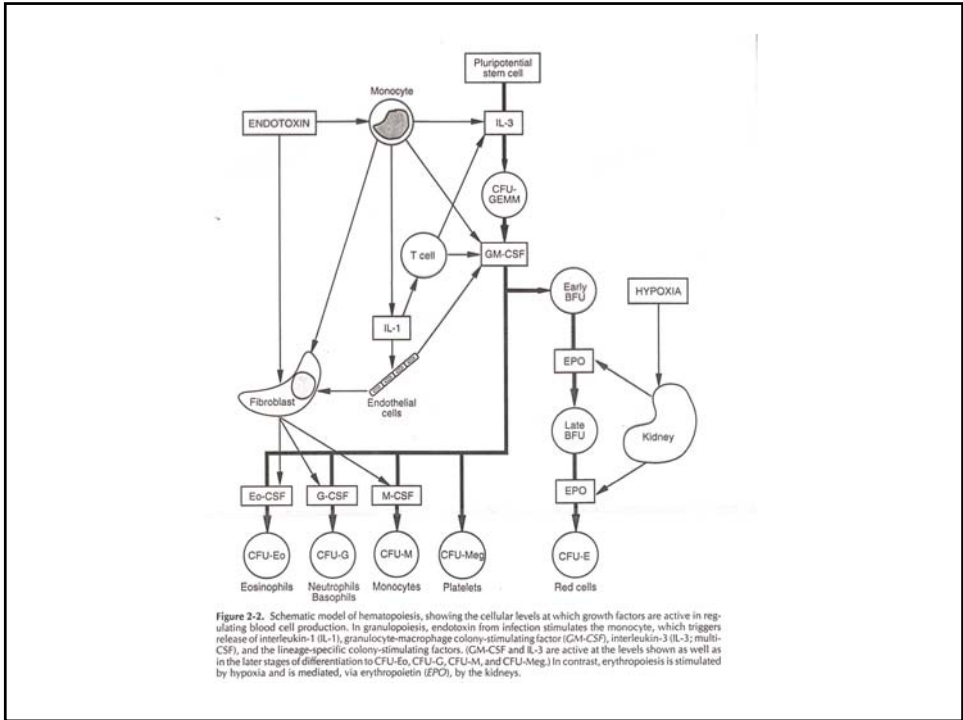
**FIGURE 31-1.** Activation of atrial natriuretic peptide (ANP) via low-pressure baroreceptors in the atria (especially the left atrium) leads to cardiovascular, endocrine, and renal effects. *ADH* = antidiuretic hormone; *GFR* = glomerular filtration rate.



**FIGURE 30-4.** Components of the renin–angiotensin–aldosterone system, stimuli for renin secretion: ↓ macula densa NaCl, ↓ blood volume, ↓ perfusion pressure, ↑ β<sub>1</sub>-adrenergic activity, and ↑ prostaglandins (PGI<sub>2</sub>). + = indicates stimulation; - = indicates inhibition.



**FIGURE 30-5.** Summary of those angiotensin-mediated actions that facilitate fluid retention and elevate the arterial blood pressure. The arrow connecting “Na and H<sub>2</sub>O retention” to “arterial blood pressure” represents a simplified shortcut—of course, fluid retention affects arterial blood pressure only by altering cardiac output and peripheral resistance. (Modified with permission from Vander AJ: *Renal Physiology*, 4th edition. New York, McGraw-Hill, 1991, p 138.)



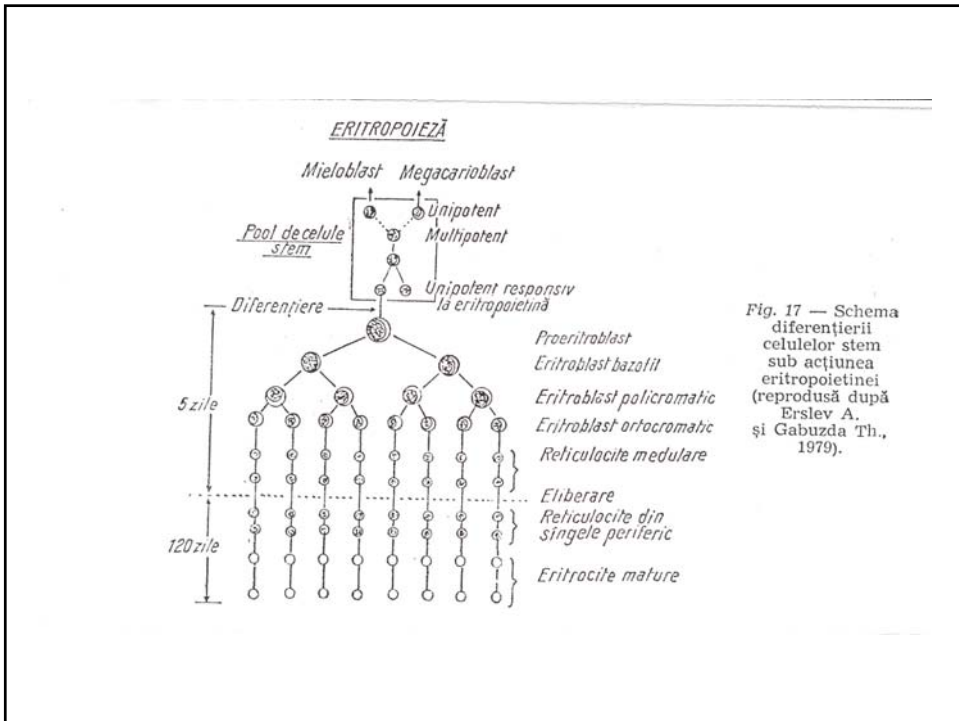
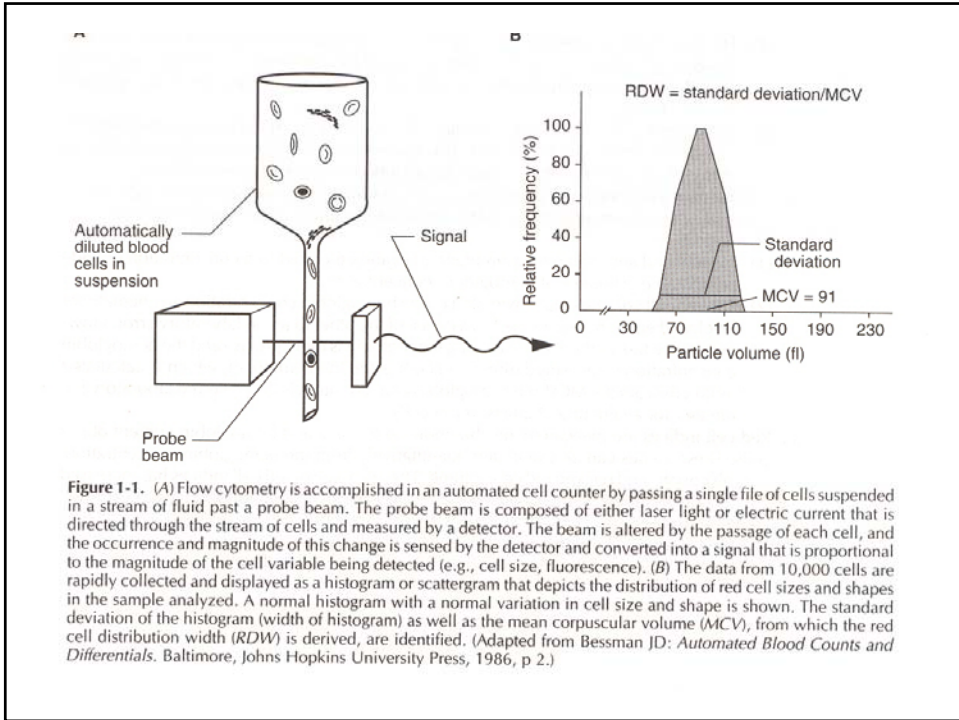


Fig. 13 — Evoluția și sediul eritropoezei pre-natale (reprodusă după Miale G. B., 1972).

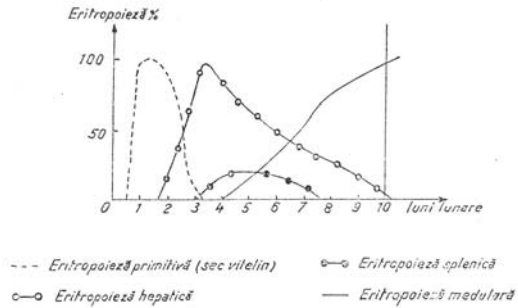
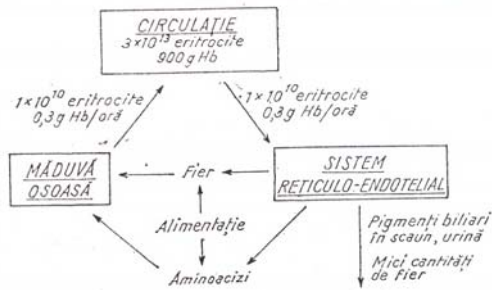
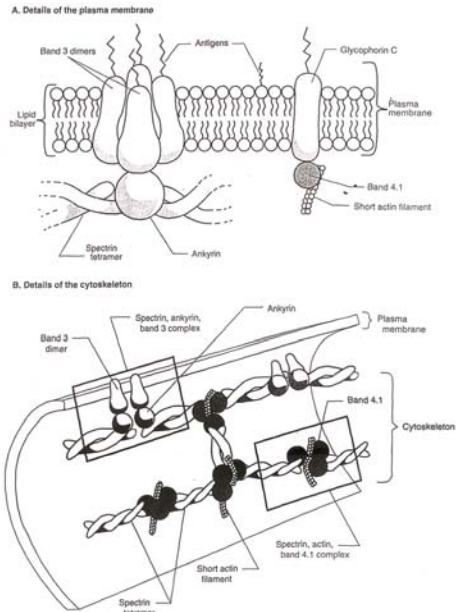


Fig. 12 — Formarea și distrugerea eritrocitelor (reprodusă după Ganong W. F., 1981).





**Figure 7-1.** The red cell membrane is composed of a plasma membrane and a cytoskeleton. (A) The plasma membrane is a bipolar lipid layer penetrated by certain membrane proteins (band 3 and glycophorin C). (B) The cytoskeleton, located beneath the plasma membrane, is composed of four major proteins: ankyrin, spectrin, band 4.1, and actin. Ankyrin anchors the cytoskeleton to the plasma membrane by coupling with band 3. Spectrin, which is composed of entwined alpha ( $\alpha$ ) and beta ( $\beta$ ) chains, forms complexes with ankyrin and band 3 and with actin and band 4.1.