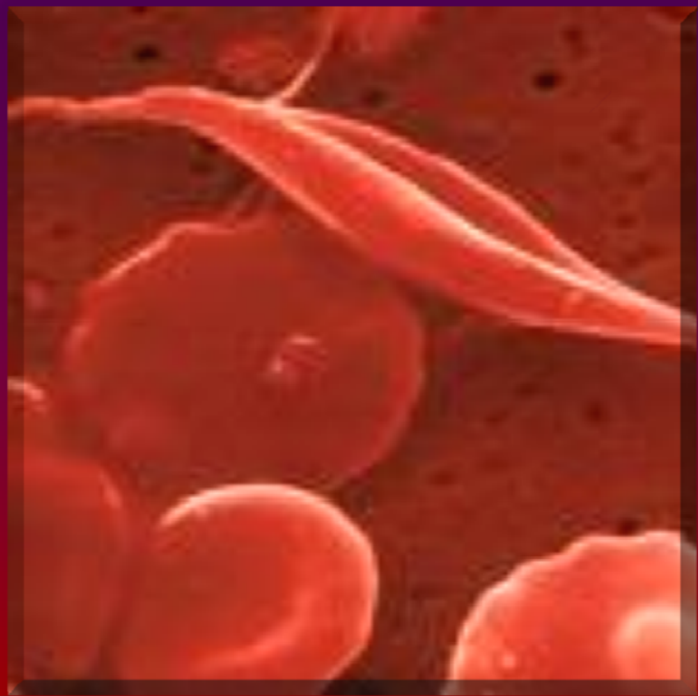
Valutazione del profilo ematologico dell’atleta

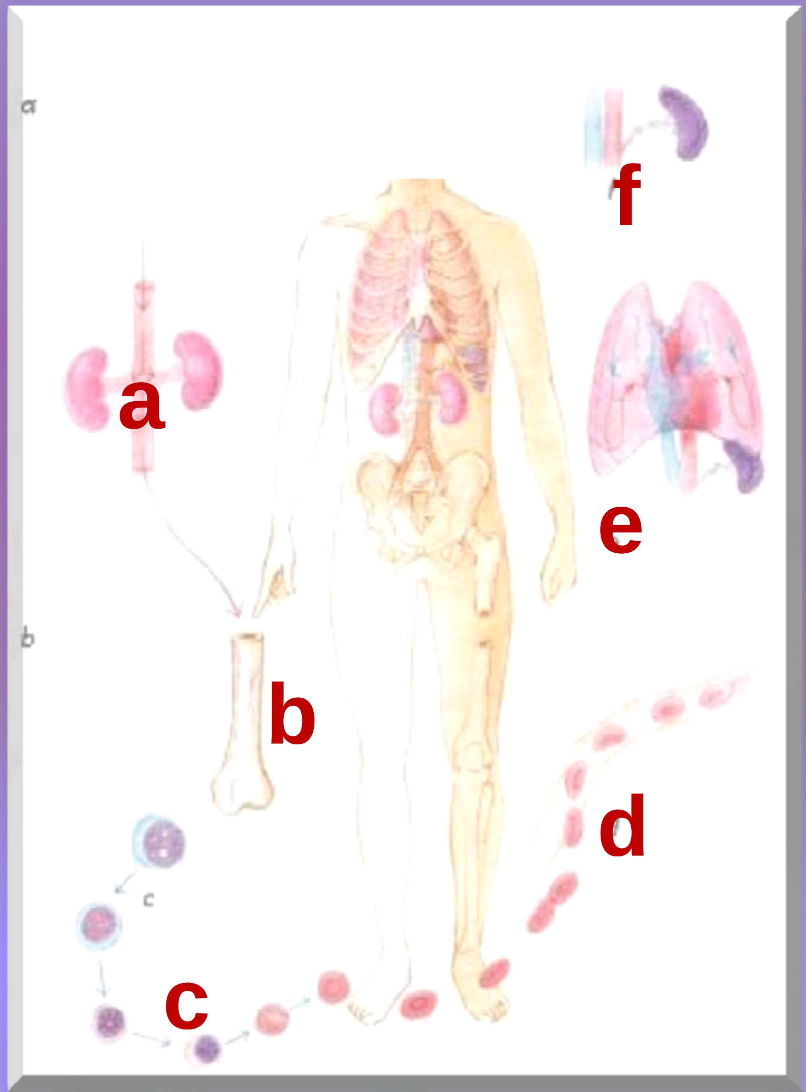
Giorgio Galanti

AMS
AGENZIA DI MEDICINA DELLO SPORT
CENTRO DI RIFERIMENTO DI METODICHE DIAGNOSTICHE
E DI RICERCA IN MEDICINA DELLO SPORT

Effetti dell'allenamento sull'apparato emopoietico



The life cycle of a red blood cell



a) Kidneys respond to a lower than normal oxygen concentration in the blood by releasing the hormone erythropoietin.

b) Erythropoietin travels to the red bone marrow and stimulates an increase in the production of red blood cells (RBCs).

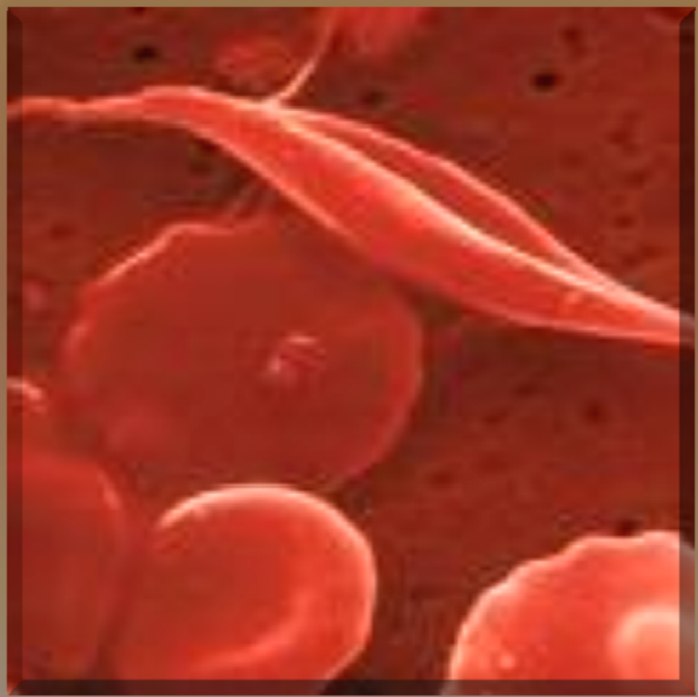
c) The red bone marrow manufactures RBCs from stem cells that live inside the marrow.

d) RBCs squeeze through blood vessel membranes to enter the circulation.

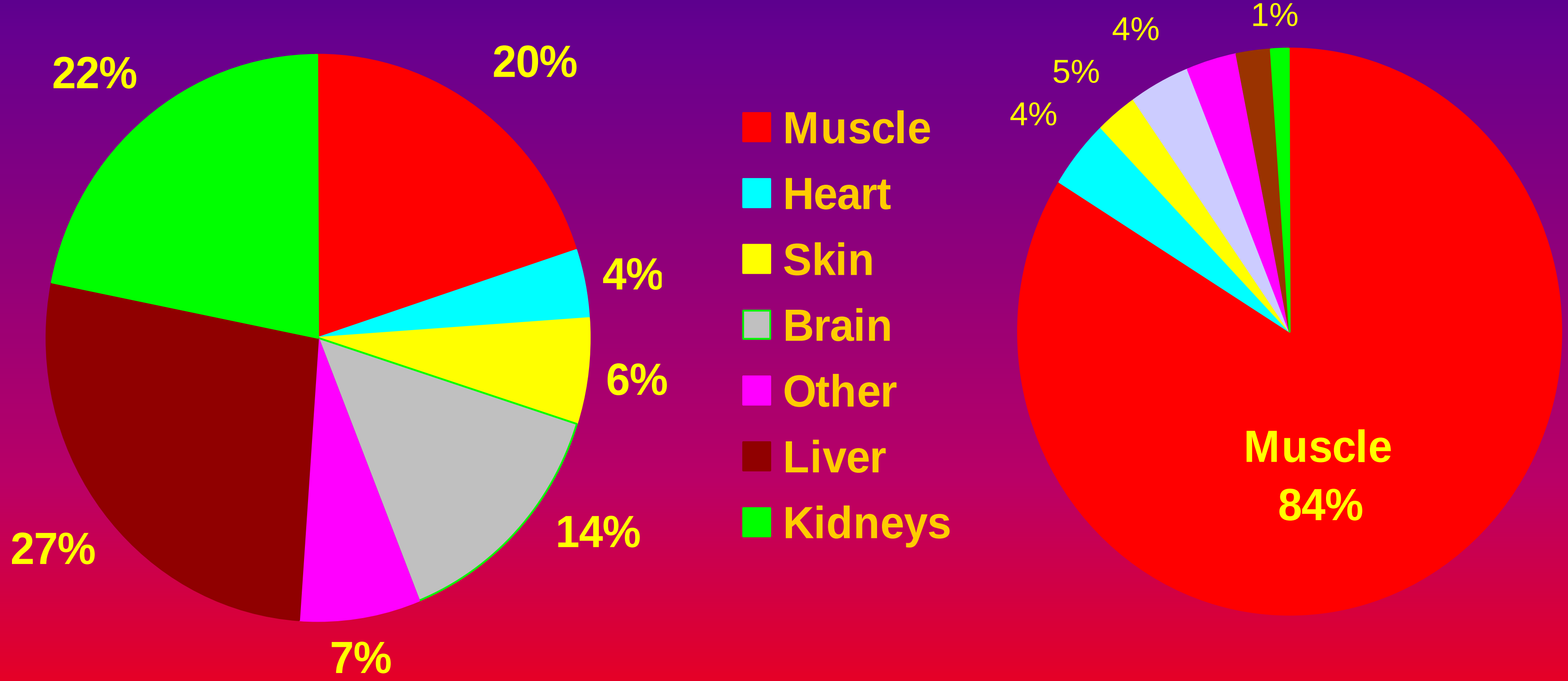
e) The heart and lungs work to supply continuous movement and oxygenation of RBCs.

f) Damaged or old RBCs are destroyed primarily by the spleen.

RBC Adaptation to Exercise
acute



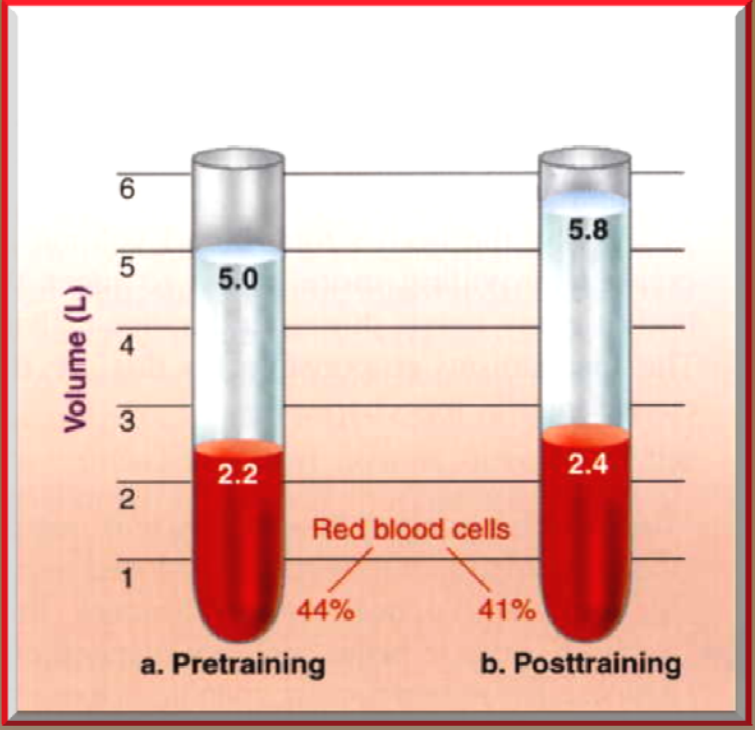
Distribution of Flow at rest and during Acute Exercise



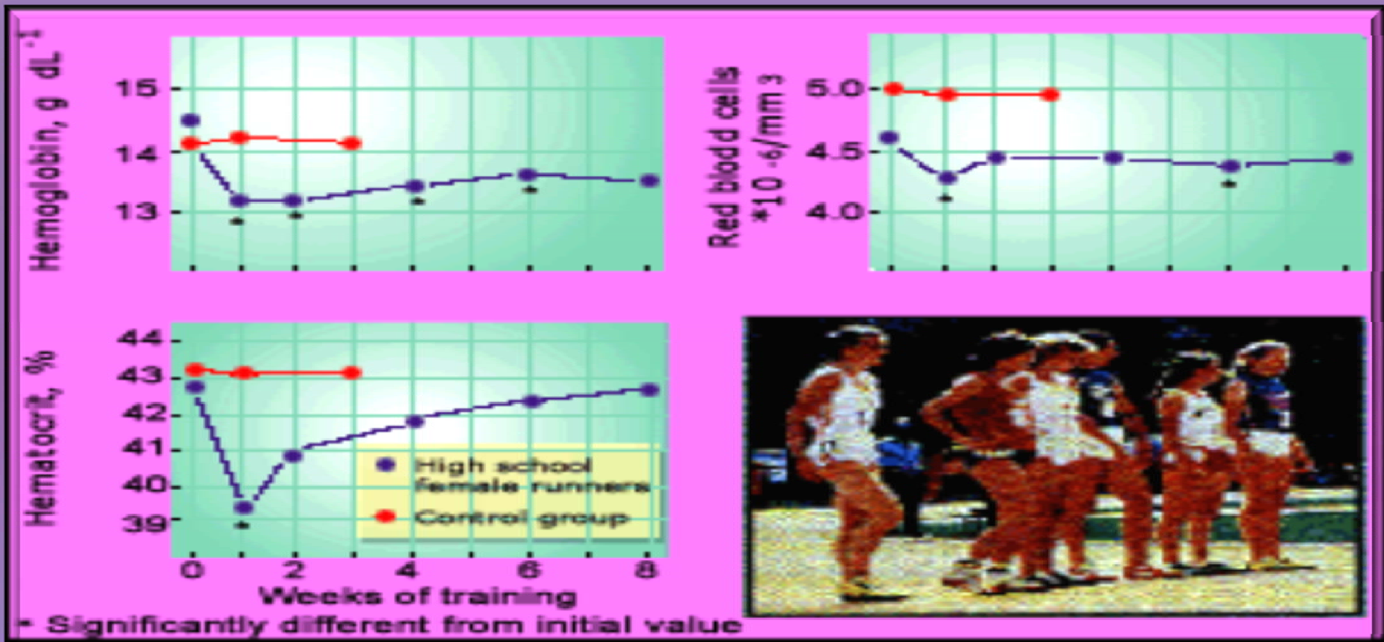
Rest
3-5 Lmin

Exercise
25-30 Lmin

RBC Adaptation to Exercise
chronic



RBC Adaptation to Exercise
chronic



Hemoglobin, red blood cell count, and hematocrit in high school female cross-country athletes and a comparison group during the competitve season. Significant changes occur during training in the high school athletes levels.

Is it anemia?

No

It is expanded blood volume

How does execise expand the plasma volume?

1. Increasing mean arterial blood pressure and thus capillary hydrostatic pressure
2. Generating lactic acid and other metabolites in working muscle that increases tissue osmotic pressure producing
3. Producing sweat

Water loss at rest and during prolonged exercise

Source of loss	Resting		Exercise	
	ml/h	% total	ml/h	% total
Skin	14,6	15	15	1,1
Respir	14,6	15	100	7,5
Sweat	4,2	5	1.200	90,6
Urine	58,3	60	10	0,8
Feces	4,2	5	0	0
Total	95,9 ml/h		1325 ml/h	

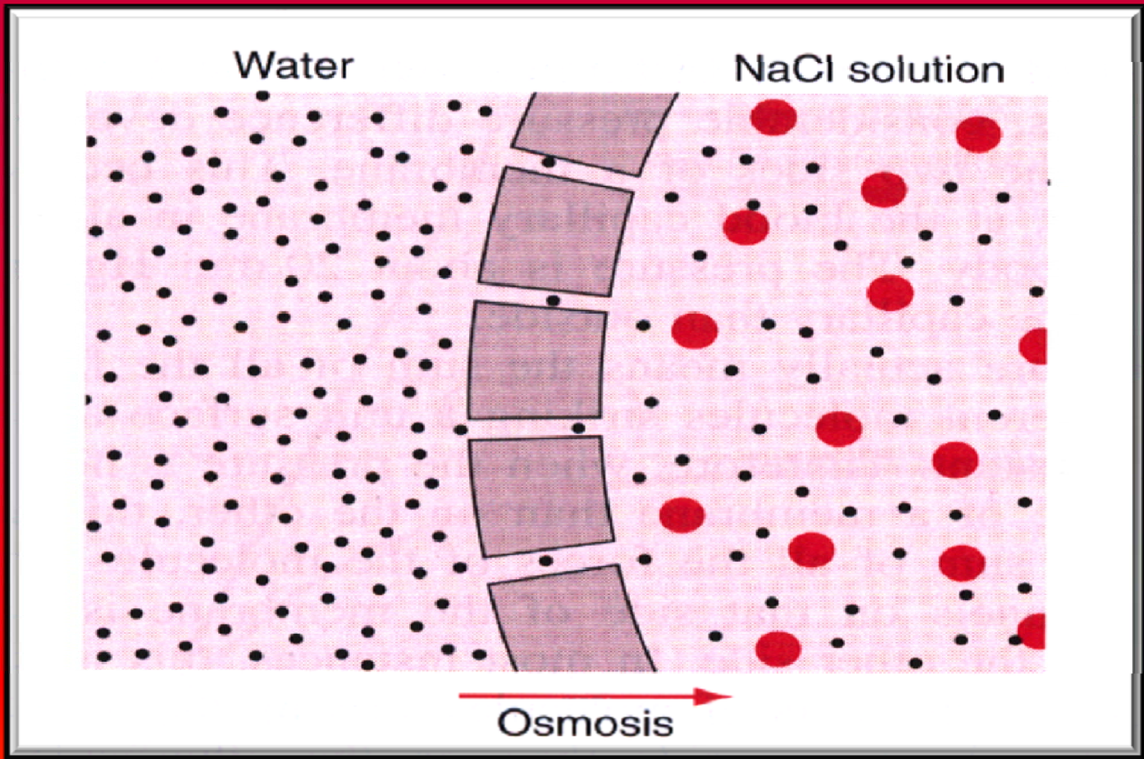
Plasma decrease
Htc increase

Barr et al 1991

Osmolarity and Volumes variation effects: Local

Volumes Reduction

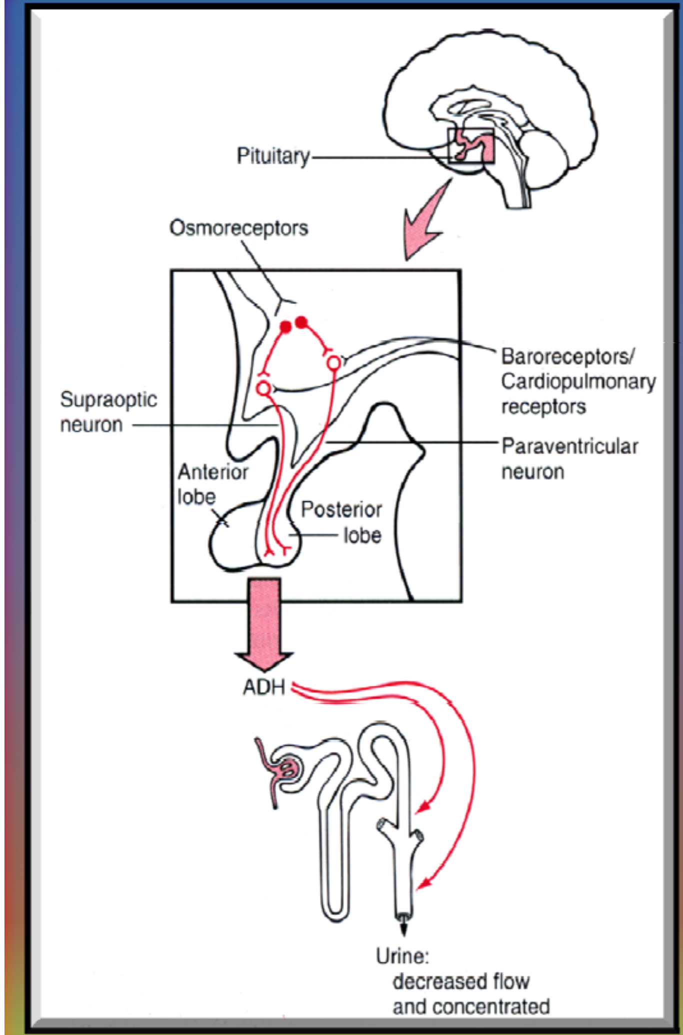
+ Extracellular Na^+ Concentration



Electrolites and Osmolarity following 2 h of exercise

	Electrolite mEq/l				Osmol (mOsm/l)
	Na^+	Cl^-	K^+	$Mg2^+$	
Sweat	40-60	30-50	4-6	1.5-5	80-185
Plasma	140	101	4	1.5	295
Muscle	9	6	162	31	205

Osmolarity and Volumes: Systemic effects



Volumes Reduction

Extracellular Na^+ Concentration

+ Osmolarity extracellular liq

↓

Osmoceptors Hypotalamus

↓

ADH release

↓

Renal water retention

Osmolarity and Volumes variation effects: *Systemic*

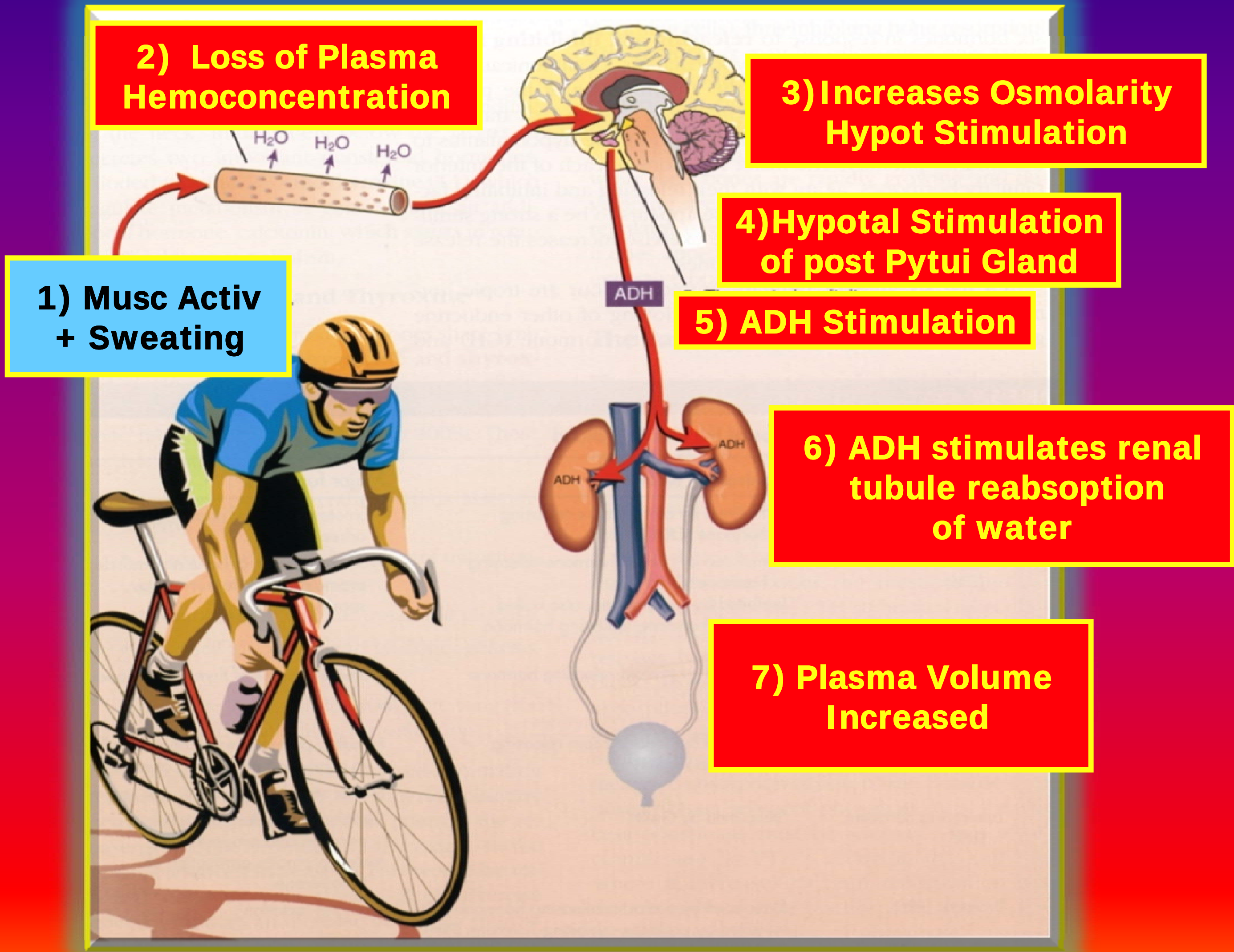
Volumes Reduction

renal flow reduction

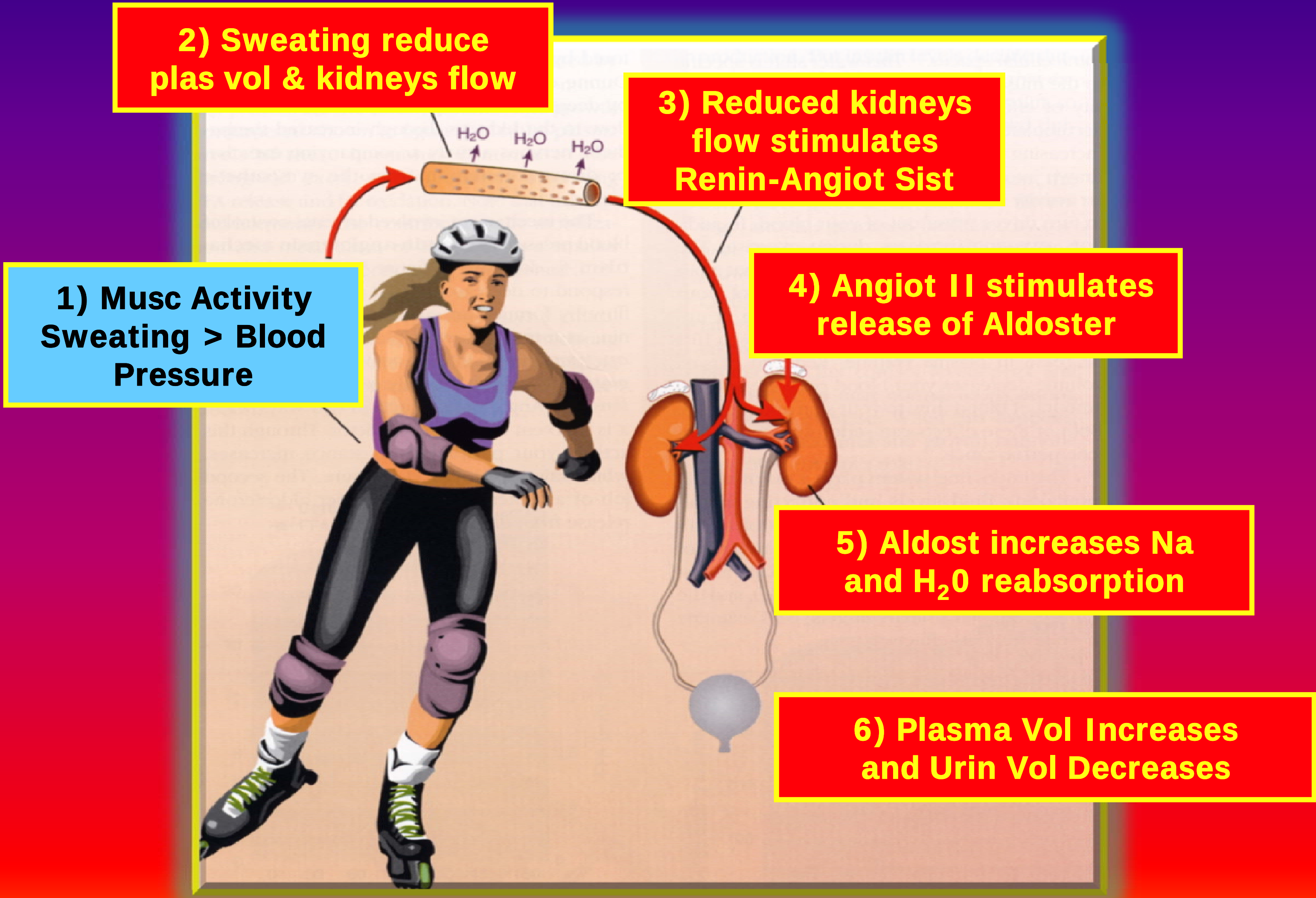
Renin-angiotensin Sistem & Aldosterone

Aldosterone increases Na^+ H_2O reabsorption

Prolonged Exercise & ADH



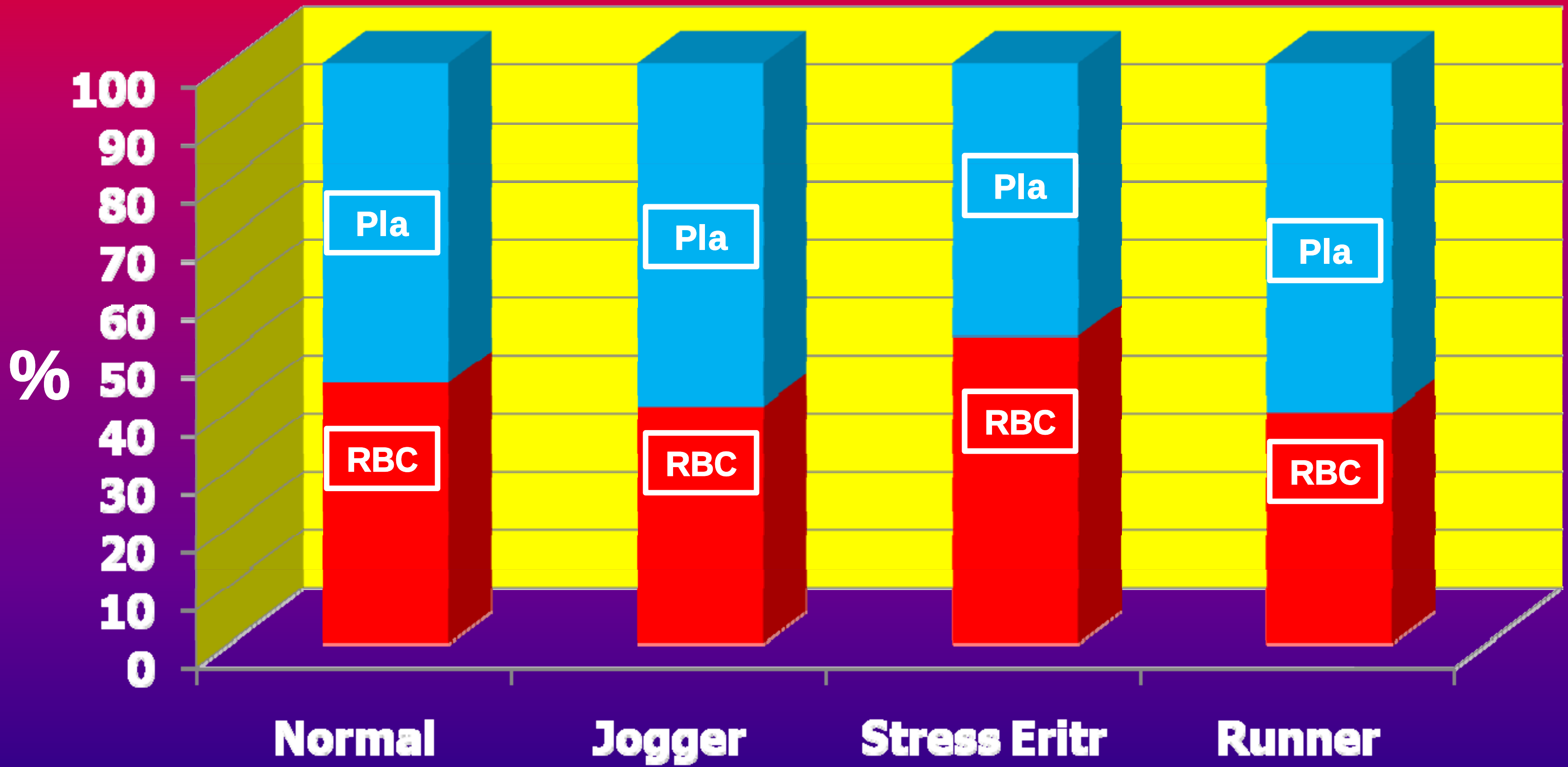
Prolonged Exercise & Aldost



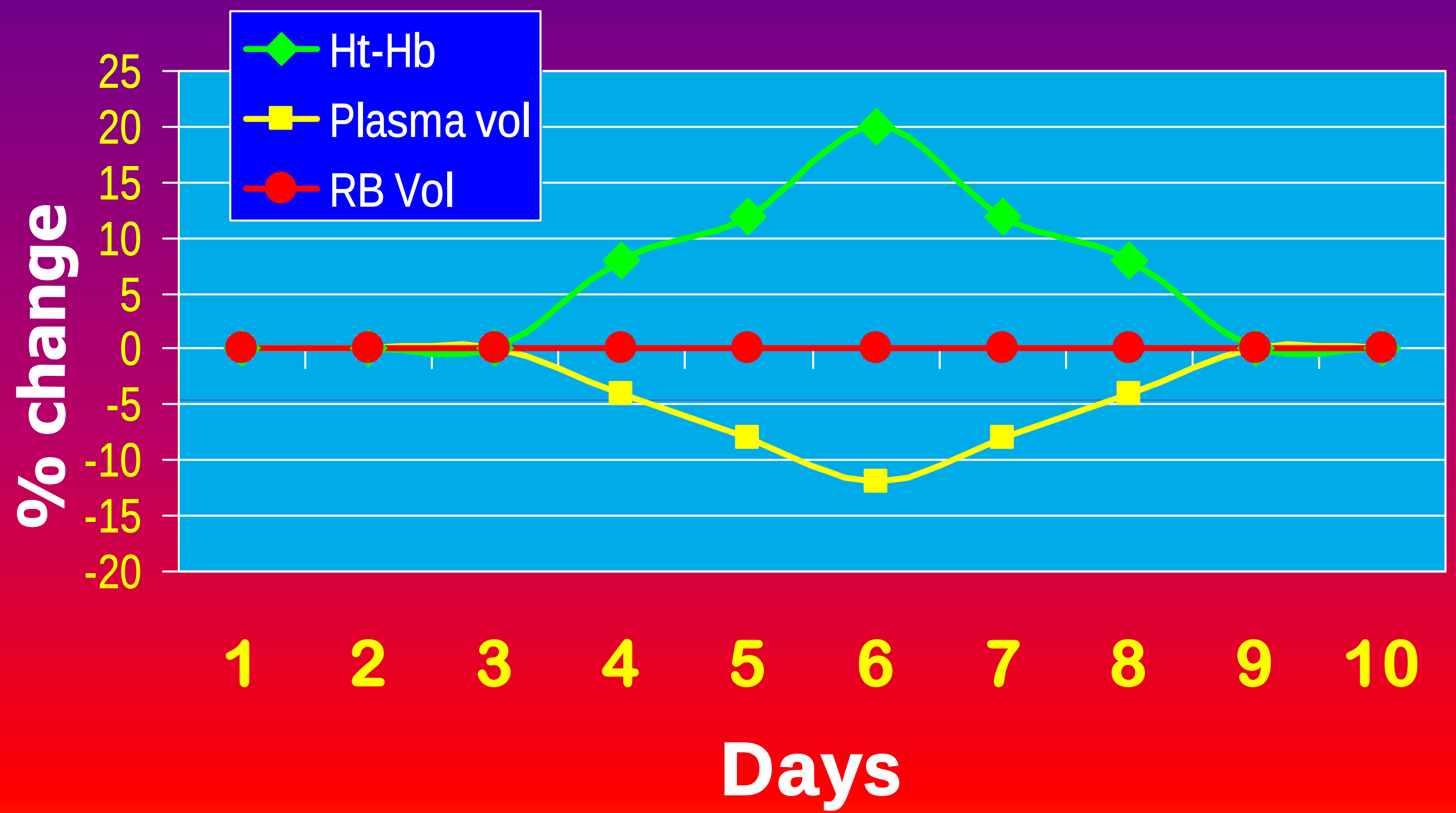
What good is the expanded plasma volume?

1. The rise in plasma volume increases the cardiac stroke volume.
2. This increase more than offsets the fall in hemoglobin concentration per unit of blood so that more oxigen is delivered to muscles
3. The athlete’s blood is “thinned” in a healthful way;both hemoglobin and fibrinogen are diluted.This makes blood flow more easily and clot less readily and thus helps prevent heart attack and stroke

Schematic Hematocrit of different subjects



Change in Plasma Vol during 3 days of repeated exercise & dehydration

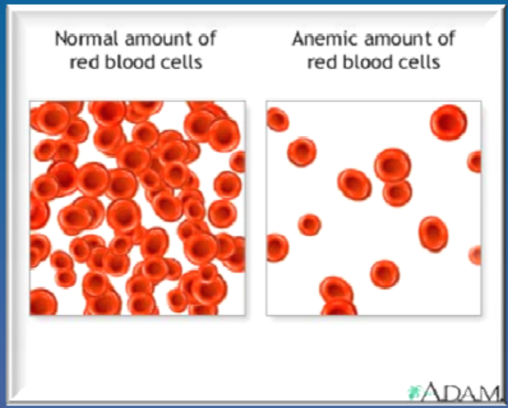


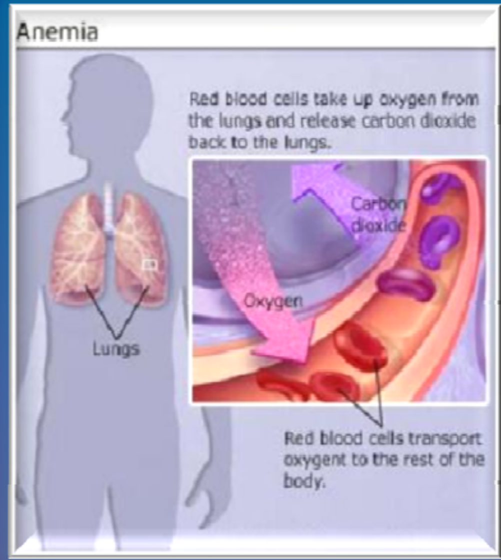
Anemia

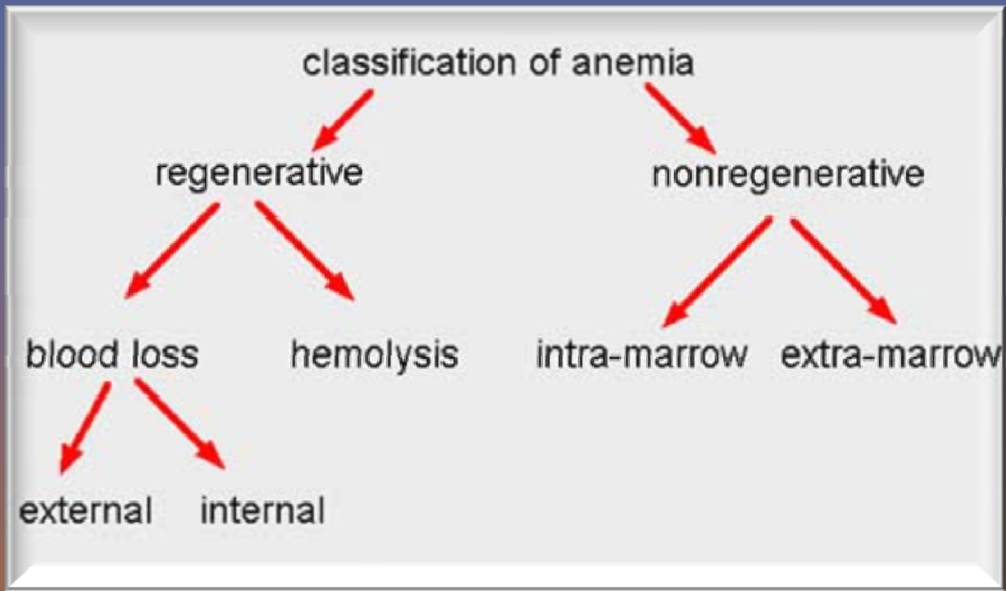
Anemia (a decrease in the number of RBCs, Hb content, or Hct) can result from decreased RBC production (erythropoiesis), increased RBC destruction, or blood loss.

Anemias due to decreased erythropoiesis are recognized by reticulocytopenia, which is usually evident on the peripheral smear.

The RBC indices, mainly the MCV, narrow the differential diagnosis of deficient erythropoiesis and determine what further testing is necessary.


Normal amount of red blood cells
Anemic amount of red blood cells
ADAM


Anemia
Red blood cells take up oxygen from the lungs and release carbon dioxide back to the lungs.
Carbon dioxide
Oxygen
Lungs
Red blood cells transport oxygent to the rest of the body.


classification of anemia
regenerative
nonregenerative
blood loss
hemolysis
intra-marrow
extra-marrow
external
internal

CHARACTERISTICS OF COMMON ANEMIAS

Etiology or Type	Morphologic Changes	Special Features
Blood loss, acute	Normochromic-normocytic, with polychromatophilia Hyperplastic marrow	If severe, possible nucleated RBCs and left shift of WBCs Leukocytosis Thrombocytosis
Blood loss, chronic	Same as iron deficiency	Same as iron deficiency
Folate deficiency	Same as vitamin B_{12} deficiency	Serum folate < 5 ng/mL (< 11 nmol/L) RBC folate < 225 ng/mL RBCs (< 510 nmol/L) Nutritional deficiency and malabsorption (in sprue, pregnancy, infancy, or alcoholism)
Hereditary spherocytosis	Spheroidal microcytes Normoblastic erythroid hyperplasia	Increased mean RBC Hb level Increased RBC fragility Shortened survival of labeled RBCs Increased radioactivity of spleen (exceeds that of liver)
Hemolysis, acute	Normochromic-normocytic Reticulocytosis Marrow erythroid hyperplasia	Increased serum bilirubin and LDH Increased stool and urine urobilinogen Hemoglobinuria in fulminating cases Hemosiderinuria
Hemolysis, chronic	Normochromic-normocytic Reticulocytosis Marrow erythroid hyperplasia Basophilic stippling (especially in lead poisoning)	Increased serum bilirubin and LDH Shortened RBC life span Increased radioiron turnover Hemosiderinuria
Infection or chronic inflammation	Normochromic-normocytic early, then microcytic Normoblastic marrow Normal iron stores	Decreased serum iron *Decreased total iron-binding* capacity Normal serum ferritin Normal marrow iron content
Iron deficiency	Microcytic, with anisocytosis and poikilocytosis Reticulocytopenia Hyperplastic marrow, with delayed hemoglobination	Possible achlorhydria, smooth tongue, and spoon nails Absent stainable marrow iron Low serum iron *Increased total iron-binding* capacity Low serum ferritin Low RBC ferritin
Marrow failure	Normochromic-normocytic (may be macrocytic) Reticulocytopenia Failed marrow aspiration (often) or evident hypoplasia of erythroid series or of all elements	Idiopathic (> 50%) or secondary *to exposure to toxic drugs or* chemicals (eg, chloramphenicol, quinacrine, hydantoins, insecticides)

CHARACTERISTICS OF COMMON ANEMIAS—Continued

Etiology or Type	Morphologic Changes	Special Features
Marrow replacement (myelophthisis)	Anisocytosis and poikilocytosis Nucleated RBCs Early granulocyte precursors Marrow aspiration may fail or may show leukemia, myeloma, or metastatic cells	Marrow infiltration with infectious granulomas, tumors, fibrosis, or lipid histiocytosis Possible hepatomegaly and splenomegaly Possible bony changes Radioiron uptake greater over spleen and liver than over sacrum
Paroxysmal cold hemoglobinuria	Normochromic-normocytic	Follows exposure to cold Results from a cold agglutinin or hemolysin Often associated with syphilis or other infections
Paroxysmal nocturnal hemoglobinuria	Normocytic (may be hypochromic because of iron deficiency) Marrow may be hypercellular or hypocellular	Dark morning urine Hemosiderin Positive acid hemolysis (Ham's test) and sugar-water tests (mostly replaced by flow cytometric testing)
Sickle cell anemia	Anisocytosis and poikilocytosis Some sickle cells in smear All RBCs sickle in preparation with hypoxia or hyperosmolar exposure	Largely limited to blacks Urinary isosthenuria Hb S on electrophoresis Possibly painful vaso-occlusive crises and leg ulcers Bony changes on x-ray
Sideroblastic anemia	Usually hypochromic but dimorphic with normocytes and macrocytes Hyperplastic marrow, with delayed hemoglobination Ringed sideroblasts	Inborn or acquired metabolic defect Stainable marrow iron (plentiful) Response to vitamin B_6 administration (rare) Commonly part of myelodysplastic syndrome
Thalassemia	Microcytic Thin cells Target cells Basophilic stippling Anisocytosis and poikilocytosis Nucleated RBCs in homozygotes	Decreased RBC fragility Elevated Hb A_2 and Hb F (often) Mediterranean ancestry (common) Anemic homozygotes from infancy Splenomegaly Bone changes on x-ray
Vitamin B_{12} deficiency	Oval macrocytes Anisocytosis Reticulocytopenia Hypersegmented WBCs Megaloblastic marrow	Serum B_{12} < 180 pg/mL (< 130 pmol/L) Frequent GI and CNS involvement Positive Schilling test (although no longer done) Elevated serum bilirubin Increased LDH Antibodies to intrinsic factor in serum (common) Absent gastric intrinsic factor secretion

Diagnosis of Anemia

- CBC with WBC and platelets
- RBC indices and morphology
- Reticulocyte count
- Peripheral smear
- Sometimes bone marrow aspiration and biopsy

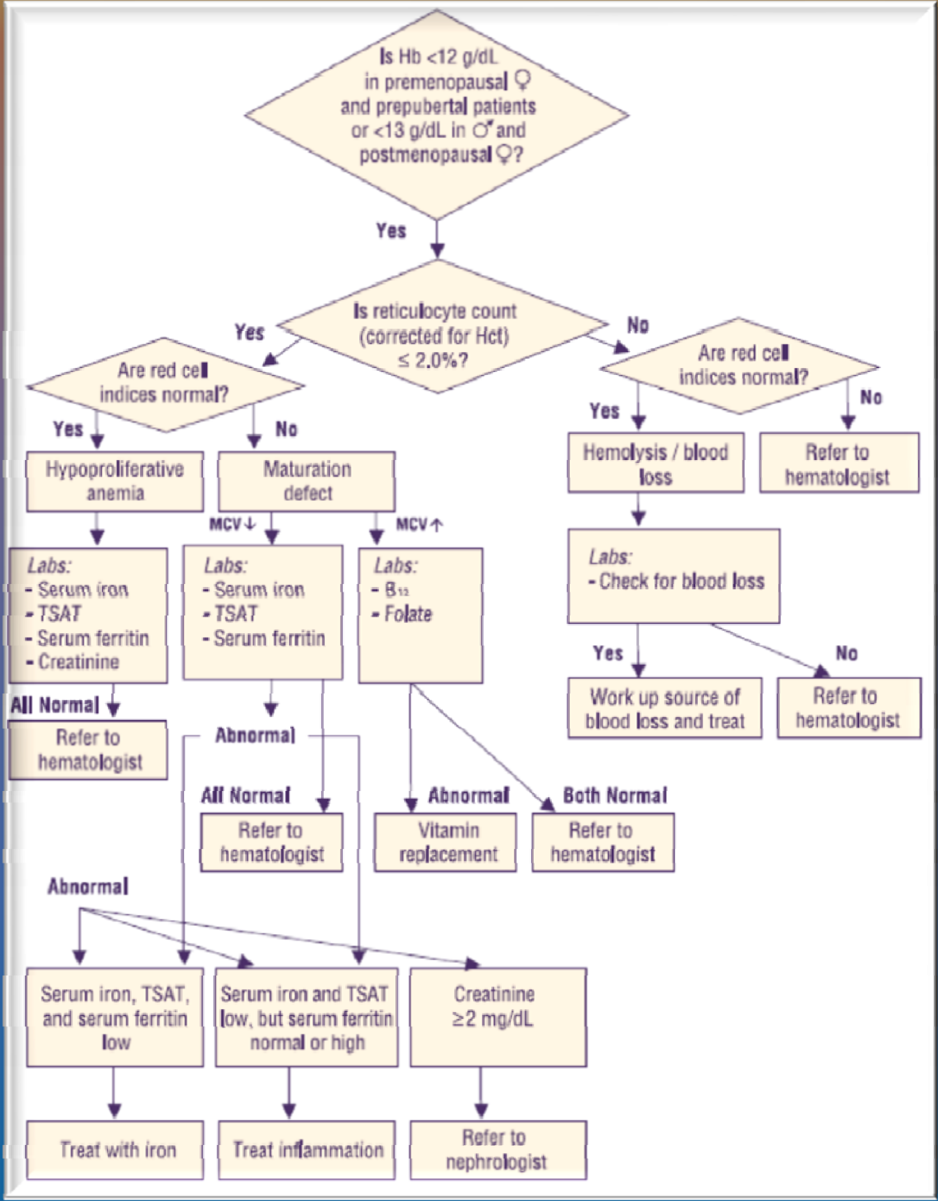
- Laboratory evaluation begins with a CBC, including WBC and platelet counts, RBC indices and morphology (MCV, MCH, MCHC, RBC volume distribution width [RDW]), and examination of the peripheral smear.
- Reticulocyte count demonstrates how well the bone marrow compensates for the anemia. Subsequent tests are selected on the basis of these results and on the clinical presentation. Recognition of general diagnostic patterns can expedite the diagnosis

- The automated CBC directly measures Hb, RBC count, and MCV (a measure of RBC size).
- Hct (a measure of the percentage of blood made up of RBCs)
- MCH (a measure of the Hb content in individual RBCs)
- MCHC (a measure of the Hb level in individual RBCs) are calculated values.
- The diagnostic criterion for anemia in men is Hb < 14 g/dL, Hct <42%, or RBC <4.5 million/L; for women, Hb <12 g/dL, Hct < 37%, or RBC < 4 million/L. For infants, normal values vary with age, necessitating use of age-related tables.
- RBC populations are termed microcytic (small cells) if MCV is < 80 fL, and macrocytic (large cells) if MCV is > 100 fL.

- **However, because reticulocytes are also larger than mature red cells, large numbers of reticulocytes can elevate the MCV and not represent an alteration of RBC production.**
- **Automated techniques can also determine the degree of variation in RBC size, expressed as the RDW. A high RDW may be the only indication of simultaneous microcytic and macrocytic disorders (or simultaneous microcytosis and reticulocytosis); such a pattern may result in a normal MCV, which measures only the mean value.**
- **The term hypochromia refers to RBC populations in which MCH is < 27 pg/RBC or MCHC is < 30%. RBC populations with normal MCH and MCHC values are normochromic.**

Causes of Anemia

- Iron deficiency is the most common cause of anemia and usually results from blood loss. Symptoms are usually nonspecific.
- RBCs tend to be microcytic and hypochromic, and iron stores are low as shown by low serum ferritin and low serum iron levels with high serum total iron binding capacity.
- If the diagnosis is made, occult blood loss is suspected. Treatment involves iron replacement and treatment of the cause of blood loss.


Is Hb <12 g/dL in premenopausal ♀ and prepubertal patients or <13 g/dL in ♂ and postmenopausal ♀?
Yes
Is reticulocyte count (corrected for Hct) ≤ 2.0%?
Yes
No
Are red cell indices normal?
Yes
No
Hypoproliferative anemia
Maturation defect
MCV ↓
MCV ↑
Labs:
- Serum iron
- TSAT
- Serum ferritin
- Creatinine
Labs:
- Serum iron
- TSAT
- Serum ferritin
Labs:
- B12
- Folate
All Normal
Refer to hematologist
Abnormal
All Normal
Refer to hematologist
Abnormal
Vitamin replacement
Both Normal
Refer to hematologist
Abnormal
Serum iron, TSAT, and serum ferritin low
Serum iron and TSAT low, but serum ferritin normal or high
Creatinine ≥2 mg/dL
Treat with iron
Treat inflammation
Refer to nephrologist
Are red cell indices normal?
Yes
No
Hemolysis / blood loss
Refer to hematologist
Labs:
- Check for blood loss
Yes
No
Work up source of blood loss and treat
Refer to hematologist

Athletes and Iron Deficiency

A combination of the following factors place athletes at risk of iron deficiency:

- Inadequate supply of dietary iron. Athletes who avoid red meat have difficulty meeting the body's iron needs.
- Increased demands for iron. Hard training stimulates an increase in red blood cell and blood vessel production, and increases the demand for iron. (Iron turnover is highest for endurance athletes training at high intensity).
- High iron loss. Blood loss through injury, or menstruation. In endurance athletes, 'foot strike' damage to red blood cells in the feet due to running on hard surfaces with poor quality shoes leads to iron loss. Finally, because iron is lost in sweat, heavy sweating leads to increased risk of

ACSM Clinicians' News and Highlights

Should we screen for anemia? A seminal study of the value of the blood count (CBC) concluded that screening hemoglobin level is useful .

Yet screening in the preparticipation evaluation is not recommended. This policy needs pondering.

- **Not screening male athletes makes sense, because the yield is low and most men with a low hemoglobin level have sports anemia.**
- **Sports anemia is a false anemia— the erythrocyte mass is normal. The hemoglobin is low because aerobic exercise expands the baseline plasma volume and dilutes hemoglobin. Sports anemia is a benefit—a cardinal feature of aerobic fitness.**

- **Not screening female athletes, however, makes no sense. The cost is low, the yield is high, and the fix is easy.**
- **Many studies find that about 10% of young women have iron deficiency anemia; yet, less than half of National Collegiate Athletic Association (NCAA) Division I-A schools screen female athletes.**

- Iron deficiency is a common problem for women athletes. Studies have routinely found that athletes, especially female athletes, are often iron-deficient or anemic. Iron is essential for athletic performance.
- One of its major functions is to carry oxygen to and carbon dioxide away from all the cells in your body. The brain also relies on oxygen transport and without enough iron you will find it hard to concentrate and feel tired and irritable.
- Iron is also needed to maintain a healthy immune system. If you don't have enough iron you may be prone to more frequent infections.

ACSM Clinicians' News and Highlights: Dietary iron supply can be increased by

1) eating more lean red meat;
2) avoiding tea or coffee with meals;
3) drinking orange juice with breakfast;
4) cooking in cast-iron cookware;
5) eating mixed meals, so the protein factor in meat, fish, and poultry will enhance iron absorption from grains, beans, and legumes.

A Pearl in Diagnosis

In female athletes only two common anemias are microcytic; only one of them benefits from treatment, and they can be differentiated by CBC and serum ferritin alone; no other testing is needed. The first is iron deficiency anemia, in which the erythrocyte count is low and erythrocyte distribution width (RDW) is high. Ferritin is also low. The second is thalassemic trait, commonly alpha thalassemic trait in a black athlete. Here erythrocyte count is normal or even high and RDW is normal, as is serum ferritin.

- The pearl of a normal or high erythrocyte count in thalassemic trait is also a paradox. The athlete is anemic (low hemoglobin) yet has a normal or elevated number of erythrocytes. The hemoglobin is low because the erythrocytes are microcytic, but the body compensates by cranking out more of the little rascals. Thalassemic trait is the only anemia with normal or high numbers of erythrocytes. It can not be cured with any therapy, but incredibly, one NCAA football player with thalassemic trait was treated with erythropoietin, raising his hemoglobin in 1 month to 16 g/dL from his lifelong baseline of 12 g/dL. However, this is not a good idea.

ACSM Clinicians' News and Highlights

- If iron stores are normal, footstrike or exertional hemolysis does not cause anemia.
- This is because the intravascular hemolysis from this mechanism is trivial, easily compensated by a slight increase in erythrocyte production.
- In other words, we normally destroy (and produce) up to 25 mL of erythrocytes a day, and exertional hemolysis may increase destruction by 10% (at most). All it takes to compensate for this is increasing the reticulocyte count 10% (eg, from a normal of 1% to a new level of 1.1%).
- Footstrike hemolysis is jazzy but not key. It may even be a benefit, by culling out the older, stiffer erythrocytes and replacing them with young, pliable ones .

“Ergogenic”Drugs

- ↑Alcohol
- ↑Amphetamines
- ↑Epinephrine
- ↑Caffeine
- ↑Marijuana
- ↑Cocaine
- ↑Steroids
- ↑Growth Hormon
- ↑Red cell reinfusion
- ↑Erythropoietin

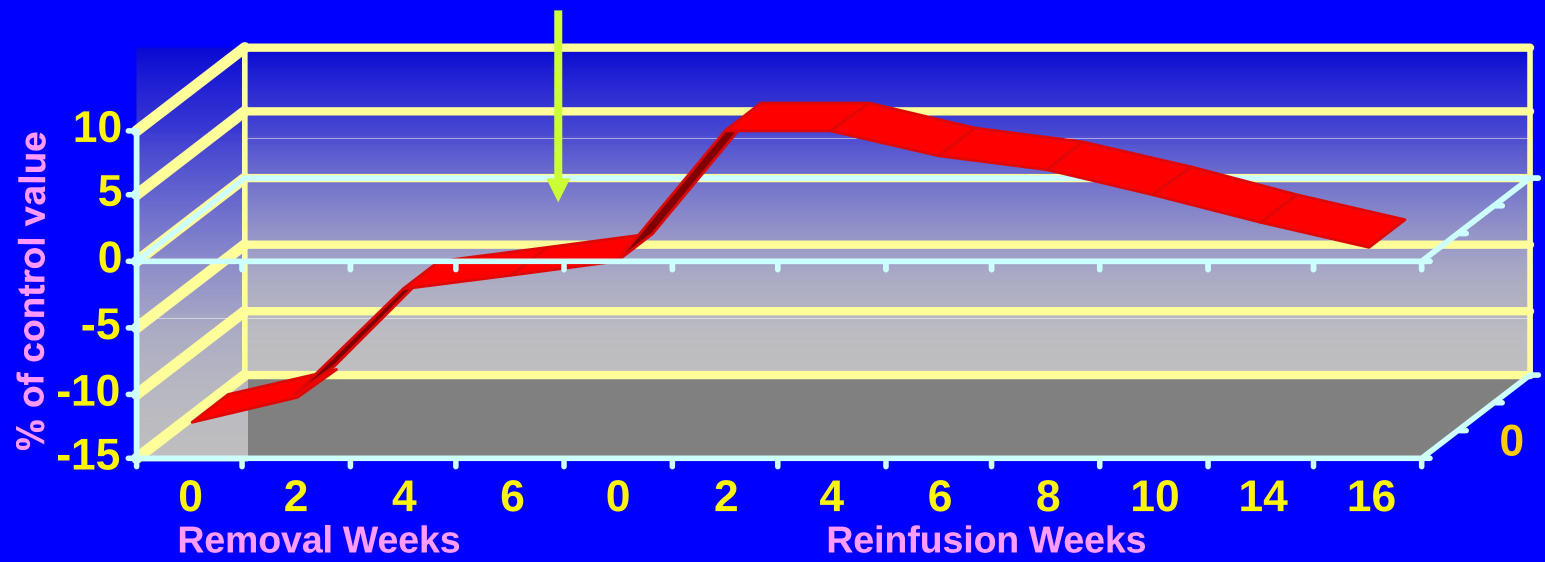
WADA's Prohibited List

(1) Scientific evidence or experience demonstrates that the method or substance has the potential to enhance, or enhances sport performance.

(2)Medical evidence or experience suggests that the use of the substance or method represents an actual or potential health risk to the athlete.

(3) The use of the substance or method violates the spirit of sport

Time course of hematology changes after the removal and reinfusion of 900 ml of freeze-preserved blood

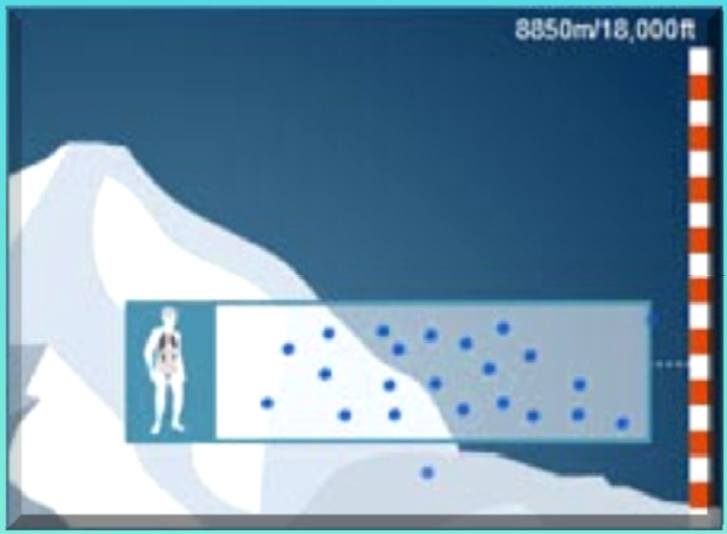


Gledhill Med.Sci.Sport.Exer. 1982

The Effects of Altitude

In a normal healthy body, red blood cells deliver oxygen to the tissues, including all the muscles. This oxygen is used by the mitochondria in the muscle cells to convert carbohydrates and fats into energy.

The Effects of Altitude



Oxygen : Whether you're at sea level or high on a mountain, the atmosphere holds the same level of oxygen: 21%. As people climb higher, however, they experience less atmospheric (barometric) pressure, and the oxygen molecules are farther apart, making it harder to breathe.

The Effects of Altitude

Acclimatisation :At sea level, atmospheric pressure helps force oxygen from the lungs into the blood and tissues. At higher altitudes, as the pressure decreases, this process slows down.

The body responds by increasing the number of red blood cells, which carry oxygen, as well as increasing production of an enzyme that transfers oxygen to the tissues.

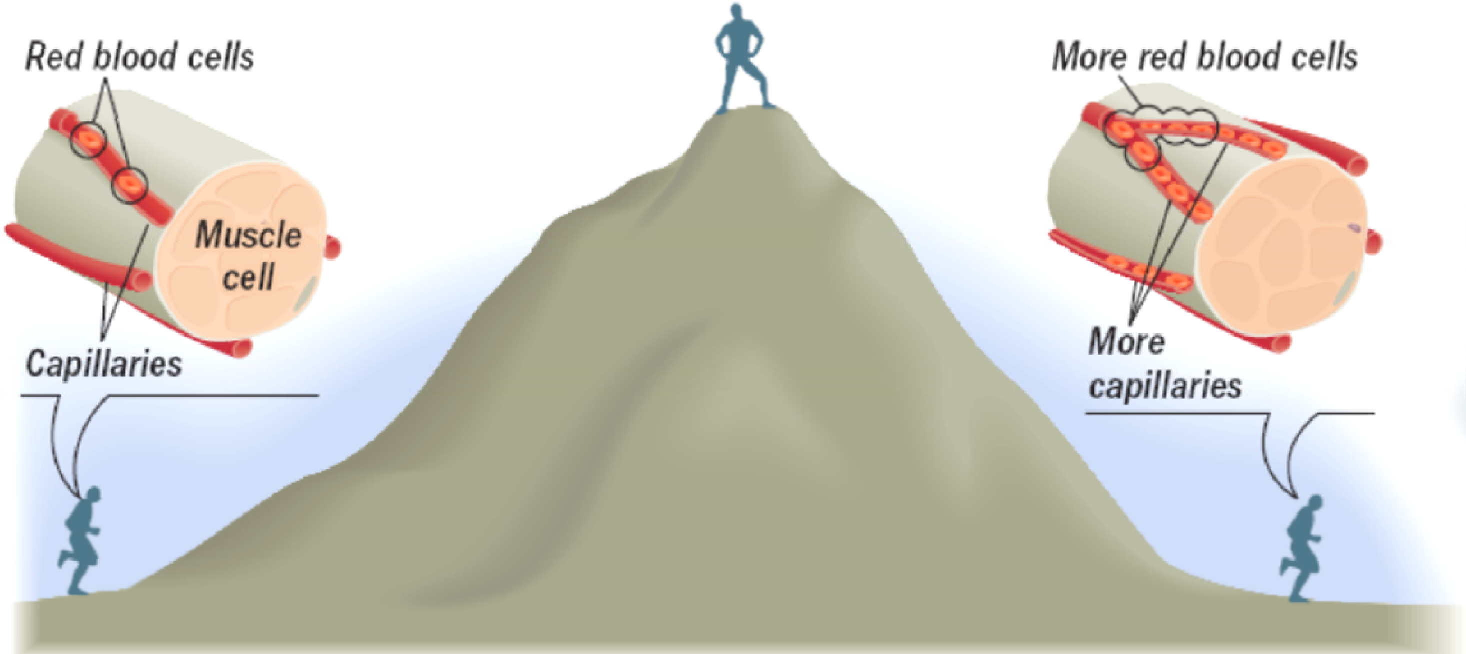
Living High/Training Low

Spending time at high altitude before training at low altitude results in marked improvement in athletic performance.

Normal
Red blood cells deliver oxygen to the muscles, where oxygen is used to convert sugar to energy.

Living High
The number of blood vessels and red blood cells increase to compensate for the decrease in the amount of oxygen available.

Training Low
The increased red blood cells and capillaries allow more oxygen to be delivered.



CVAC™

Cyclic Variations in Altitude Conditioning™

Enhancing Performance Beyond High-Altitude Acclimatization

In a normal healthy body, red blood cells deliver oxygen to the tissues, including all the muscles. This oxygen is used by the muscles to convert sugar (fuel) to energy. At high altitude, less oxygen is available. The body compensates by increasing the number of blood vessels and red blood cells. The increased red blood cells and capillaries allow more oxygen to be delivered to the body's cells—'living high/training low'.

Extensive research into the high-altitude adaptation response reveals that it results in the kidneys producing *erythropoietin* (EPO), a hormone that up-regulates red blood cell (RBC) production. This increase provides more oxygen available to the target cells. Additionally, high-altitude conditioning has been proven to increase the number of capillaries, providing more surface area for oxygen exchange. When a high-altitude-acclimatized individual exercises at lower altitudes, mitochondrial density increases in response to the additional availability of oxygen, resulting in increased *strength*, *stamina*, and numerous other benefits.

> **CVAC increases the oxygen-carrying capacity of the blood. Early results from an ongoing study at the University of Hawaii seem to validate this observation in as little as four weeks of use.**

Over and Above High Altitude Conditioning: CVAC—How It Works

CVAC essentially consists of various altitudes arranged into precise patterns. Each session contains a number of maneuvers involving multiple transits to target pressures; and consists of 300-500 cyclic altitude changes in a 20-minute period. The targets and consequent transits, pressure and temperature changes are arranged to fit the specific requirements of an individual's body via pulse-sequence profiles resembling the tonal variations of music. These pulse-sequence profiles are tailored to an individual's stress, stimulation and relaxation responses, because the human body appears to respond differently to varied pressure and temperature just as humans respond individually to different sequences of tones in music.

A high-performance blower pulls air out of the instrument while a patent-pending robotic valve controls incoming fresh air. This continuously changes the air pressure to simulate different altitudes. By using dynamic changes in oxygen concentration, air pressure and temperature, CVAC stimulates the entire body and increases the oxygen-carrying capacity of the blood. Early results from an ongoing study at the University of Hawaii seem to validate this observation in as little as four weeks of use.

The CVAC process is expected to remove the lactic acid and dead or injured cell parts built up during physical exertion. Flushing of such waste products may explain the universal claim by CVAC users of significantly enhanced 'recovery' after workouts, competition, or injury. Any level of exercise into the anaerobic range produces quantities of waste material (most notably, lactic acid). Very dramatic pressure changes create a squeezing and expanding action, or 'total-body vasopneumatic compression' (TBVPC). This causes more fluid to be returned to the heart, triggering the production of alpha-atrial natriuretic peptide (ANP) and, hence, removes excess fluid through increased urine production.

In 2003 CVAC Systems, Inc. applied for a patent on the concept, application and unique methods of this breakthrough technology. The patent effectively makes numerous claims. Additional patents are currently in the filing process.

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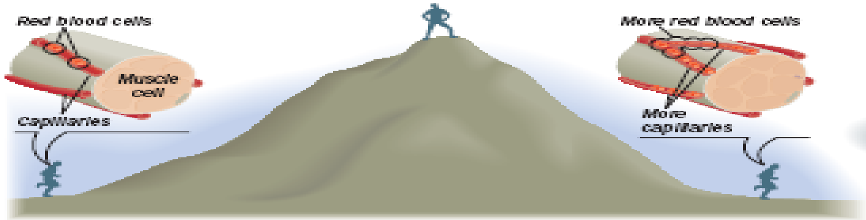
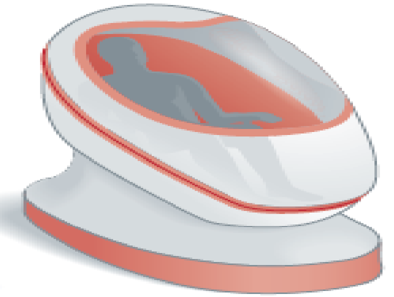


The CVAC Solution

In a safe, time-efficient dynamic session, CVAC not only offers superior benefits to those of altitude acclimatization, but also offers benefits without associated negative side effects.



- A high-performance blower pulls air out of the instrument while a patent-pending robotic valve controls incoming fresh air, continuously changing the air pressure to simulate different altitudes.
- Sessions are customized to the user's body type. A computer program precisely controls the pressure changes which may range from 300 to 500 changes in 20 minutes.
- The varied altitude changes enhance tissue oxygenation, and the constantly changing air pressure also improves circulation of blood. Moreover, stimulation of lymphatic system function results in enhanced removal of toxins.

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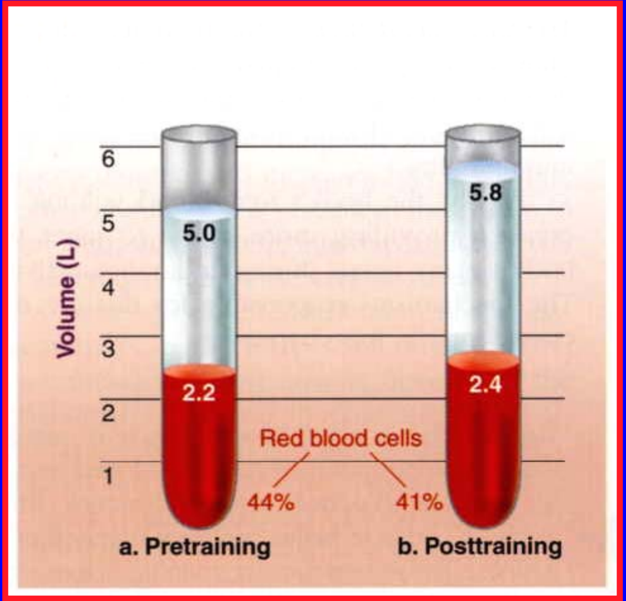
The Effects of Altitude

<u>Blood clotting</u> Red blood cell production increases at altitude over one to two months, causing blood to thicken, which may cause clotting in some chambers. Another likely source of clotting is dehydration at high altitudes.

ERITROPOIETINA

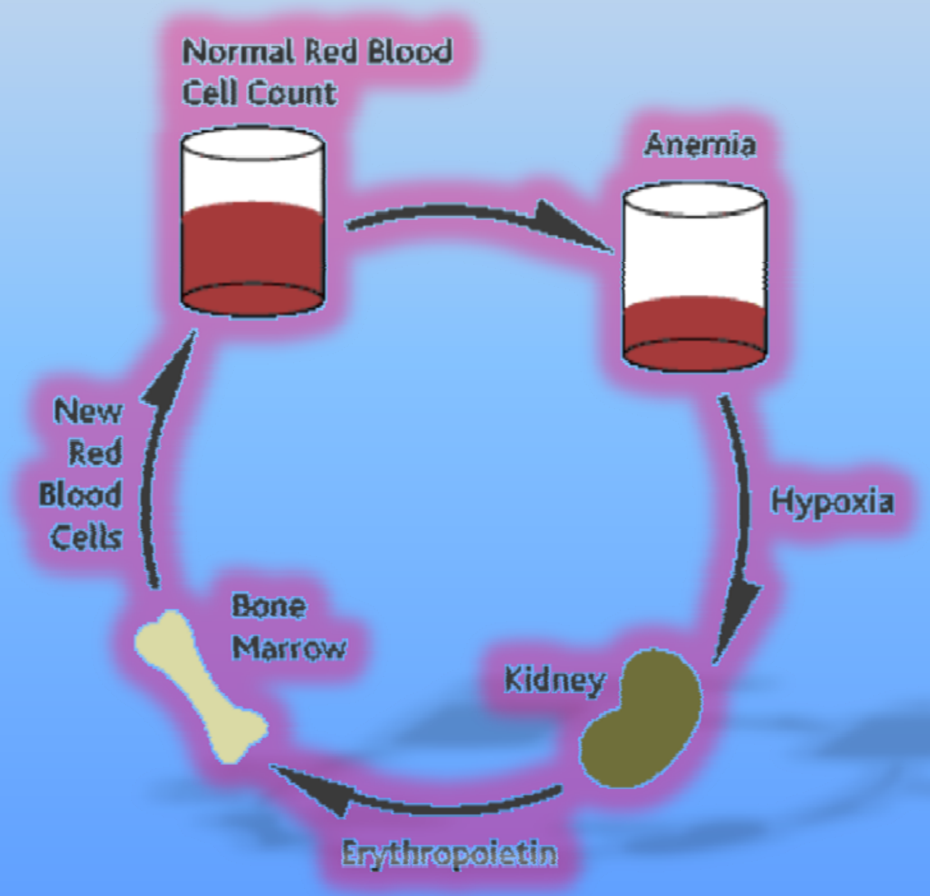


L'EPO È UN ORMONE PROTEICO PRODOTTO FISIOLOGICAMENTE DAL RENE IN RISPOSTA ALL'IPOSSIA.

L'EPO AGISCE A LIVELLO MIDOLLARE STIMOLANDO LA DIFFERENZIAZIONE DEI PRECURSORI DEI GLOBULI ROSSI CON CONSEGUENTE AUMENTO DELLA MASSA ERITROCITARIA E DEI LIVELLI DI EMOGLOBINA

ERYTHROPOIETIN (EPO) IS A NATURALLY OCCURRING PROTEIN HORMONE PRODUCED BY SPECIALIZED CELLS IN THE KIDNEYS. THESE CELLS ARE SENSITIVE TO THE OXYGEN CONCENTRATION IN THE BLOOD, AND INCREASE THE RELEASE OF EPO WHEN THE OXYGEN CONCENTRATION IS LOW (HYPOXIA). SINCE OXYGEN IS CARRIED BY RED BLOOD CELLS, TOO FEW RED BLOOD CELLS (ANEMIA) WILL RESULT IN ERYTHROPOIETIN RELEASE. EPO IS A CYTOKINE FOR STEM CELLS IN THE BONE MARROW CAUSING THEM TO INCREASE THE PRODUCTION OF ERYTHROCYTES (RED BLOOD CELLS).

RECOMBINANT ERYTHROPOIETIN IS A THERAPEUTIC AGENT PRODUCED BY DNA TECHNOLOGY IN MAMMALIAN CELL CULTURE.



Eritropoietina ricombinante r-HuEPO

- Nel 1987 è stata introdotta l'EPO ricombinante (r-HuEPO) con struttura ed azione sovrapponibili a quella endogena.

- La somministrazione di r-HuEPO consente di aumentare la massa eritrocitaria e i livelli di emoglobina per 3-4 settimane, con aumento del VO2max pari al 10%.

- La somministrazione di r-HuEPO consente quindi di migliorare la capacità aerobica dell'atleta. Gli effetti sono additivi a quelli dell'allenamento, che consente di aumentare il VO2max fino ad un massimo del 20%.
(International Olympic Committee World Congress on Sports Science 1990)

Recombinant Human Erythopoietin

A. Erythopoietin alfa (Eprex Jansen) Chinese hamster ovary cells <u>*detectable within 7 days*</u>
B. Erythopoietin Beta (Neorecormon Roche) Chinese hamster ovary cells <u>*detectable within 7 days*</u>
C. Erythopoietin Darbepoietin alfa (Nespo Dompe') Chinese hamster ovary cells <u>*detectable within 7 days*</u>
D. Erythopoietin omega or (Epomax or Hemax) Chinese hamster ovary cells <u>*detectable within 7 days*</u>
E. Erythopoietin delta or Dynepo TKT Human cells <u>*Undetectable*</u>

Varlet-Marie E,Sport Med 2003

Effetti ematologici della sommistrazione acuta di Eritropietina

A. Aumento Ematocrito
B. Aumento dei G.R.
C. Aumento del HB
D. Aumento dei Reticolociti

L’EPO viene spesso utilizzata insieme ad altre sostanze ‘doping’
Dati NCAA 1997: il 2,5% degli atleti che assumevano steroidi dichiarava di assumere contemporaneamente EPO

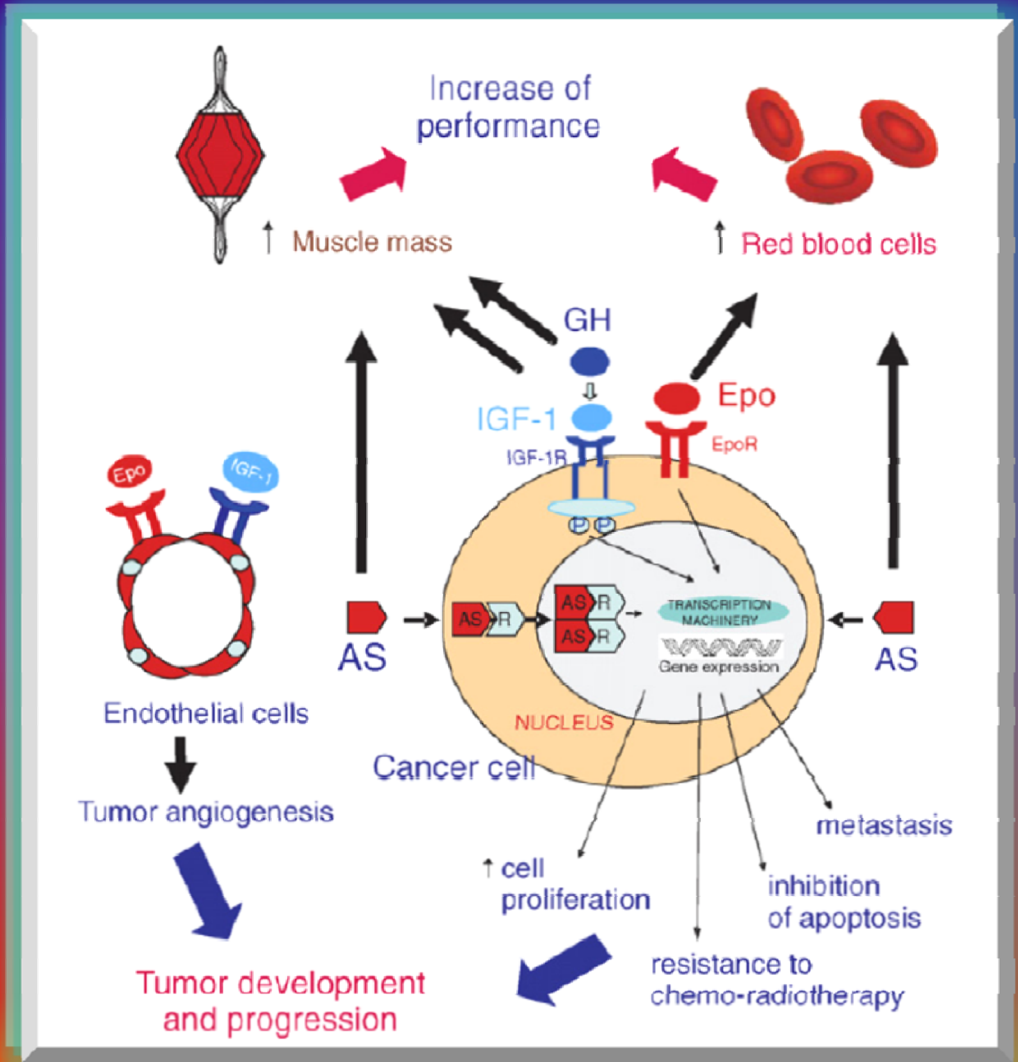
The Effects of EPO

<u>Blood clotting</u> **Red blood cell production increases over one to two months, causing blood to thicken, which may cause clotting in some chambers. Another likely source of clotting is dehydration .**

Rischi dell'Eritropoietina

Tra il 1987 e il 1990 si sono verificati 18 casi di morte in ciclisti tedeschi e belgi che facevano uso di EPO.
La spiegazione di questi drammatici eventi risiede nella elevata viscosità ematica indotta dagli effetti combinati della aumentata massa eritrocitaria (conseguente all'uso di EPO) associata alla disidratazione indotta dall'esercizio, che avrebbe provocato eventi trombotici nel circolo coronarico e celebrale

Schematic drawing of the biochemical mechanisms underlying the potential cancer risk of doping agents.



Medical literature on the association between AS, GH/IGF-1 or Epo and cancer

A. Cancer cases attributed to doping					
AS doping	Liver tumours: hepatic adenomas and hepatocarcinoma [8,9,11,17,18,20,21]	Renal tumours: nephroblastoma and renal cell carcinoma [7,12,15,19]	Prostate cancer [10,13]	Testicular leiomyosarcoma [14]	Non-Hodgkin's lymphoma [16]
GH/IGF-1 doping	Hodgkin's lymphoma [59]				
Epo doping	No cases reported to date				
B. Clinical studies on AS, GH/IGF-1, Epo and risk of cancer					
Long-term AS therapy, high serum androgen levels	Liver [18,31,75]	Breast [81]			
High plasma levels of GH/IGF-1	Breast [24-26]	Prostate [27]	Lung [28]	Colorectal [29]	
rGH replacement therapy	Colorectal [46,58,60,61]	Hodgkin's lymphoma [58]			
rEpo to prevent or treat malignancy-associated anaemia	Dubious impact on progression-free survival time [107–113]				

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Commissione Organizzativa Sanitaria
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Tel.: 06 36856507
Fax mail 06 62276223
commissione.sanitaria@fip.it

PASSAPORTO DELL'ATLETA

N° ________________

Cognome: ________________________ Nome: ________________________

Data di nascita: ______________________ Luogo di nascita: ________________

Data di compilazione: ___________

Medico curante : Dr. __________________ Tel.: ______________________

La pratica dello sport rappresenta un elemento importante per il benessere psico-fisico dell' uomo ed ormai sin dai primi anni di vita i bambini vengono avviati ad attività sportive le più varie (nuoto, calcio, ginnastica artistica, tennis, basket ecc.) per le quali sono indispensabili delle certificazioni sanitarie che comprovino lo stato di salute e quindi la cosiddetta idoneità all'attività sportiva prescelta. Il passaporto dell'atleta nasce dalla esigenza di tutelare la salute di coloro che praticano lo sport attraverso il controllo "periodico" di alcuni parametri del sangue di facile rilievo che consentano di : 1) scoprire eventuali anomalie congenite o acquisite con importanti informazioni anche di carattere epidemiologico migliorandone la prevenzione e la cura precoce; 2) stabilire per ogni soggetto dei valori a carico di pochi parametri del sangue che rappresentino dei veri e propri valori di riferimento per quell'individuo che, proprio come il comune passaporto , consentono di "individuarlo" sotto il profilo ematologico.

Tale iniziativa ha pertanto lo scopo di tutelare la salute dell'atleta sin dall'inizio dell'attività agonistica. In quest'ottica riteniamo essenziale il contributo e la collaborazione dei medici curanti nel pianificare i controlli. I dati ematici dovranno essere inviati alla Commissione Organizzativa Sanitaria della F.I.P. in forma anonima nel pieno rispetto della normativa sulla privacy (la presente pagina non deve essere inviata). Eventuali anomalie riscontrate saranno tempestivamente comunicate al medico curante per gli eventuali approfondimenti clinici del caso. I dati potranno essere elaborati solo sotto il profilo statistico e/o scientifico.

Classification criteria for blood samples

v.n. Hb 14.8+0.8 Reticul 1.90+0.52

- Hemoglobin :High abnormal Hb >17.2 or Htc > 50%
- Hemoglobin:Abnormal Hb 16.4 Htc >48%
- Reticulocites Hemoglobin: highly accelerated>3.40
- Reticulocites hemoglobin : Accelerated >2.90

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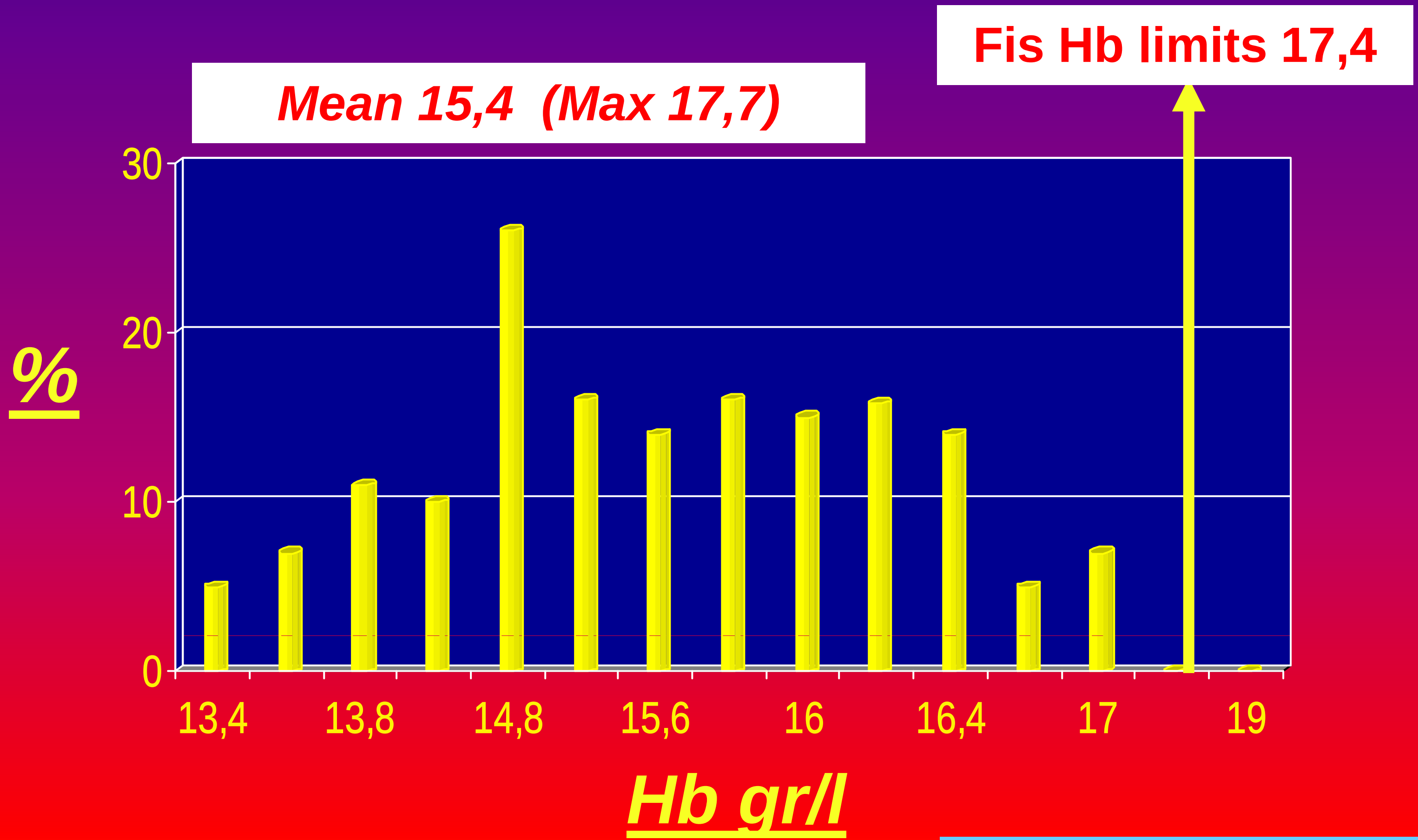
Hemoglobin concentration for male competitors in the 2001 Lathi World Nordish Championship



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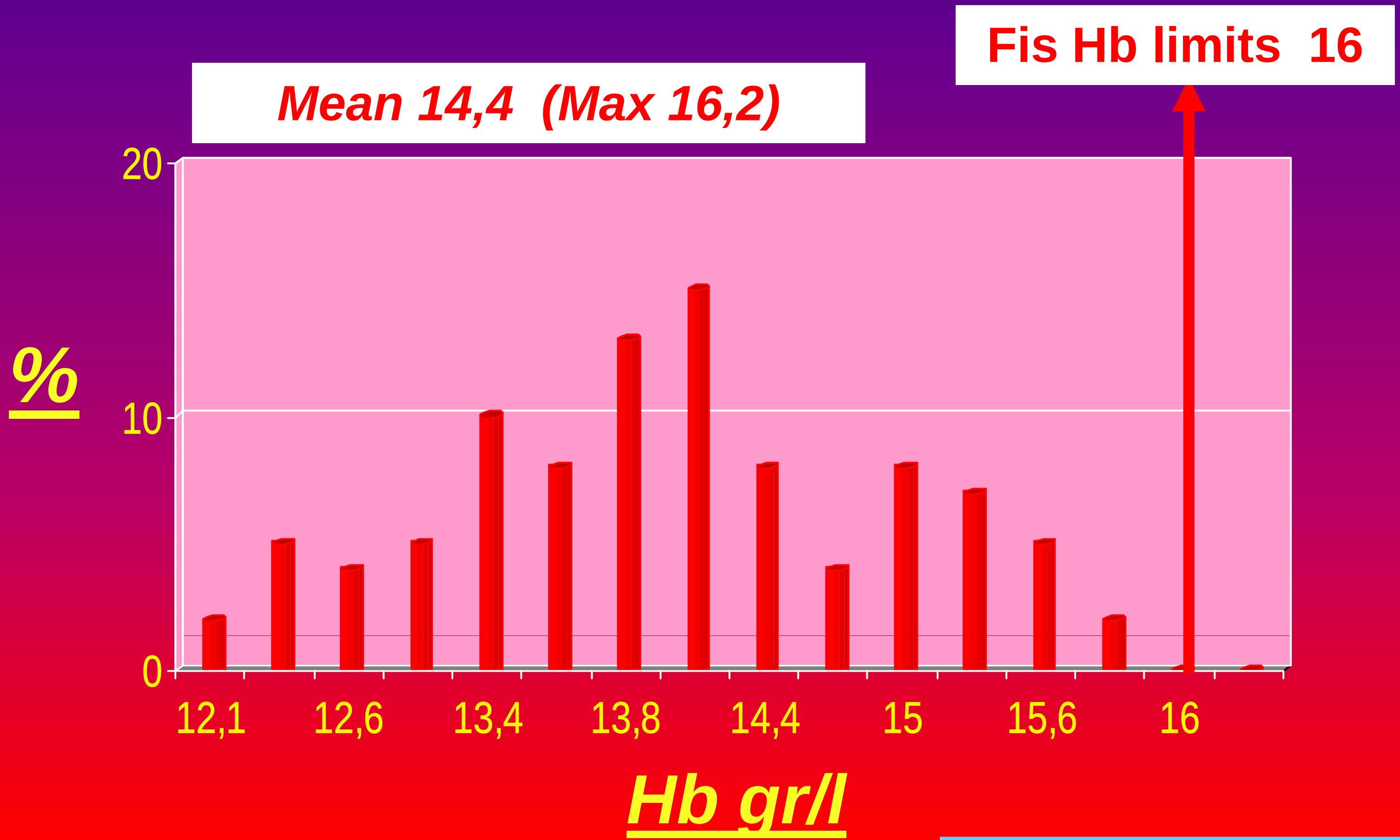
Hemoglobin concentration for femal competitors in the 2001 Lathi World Nordish Championship



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