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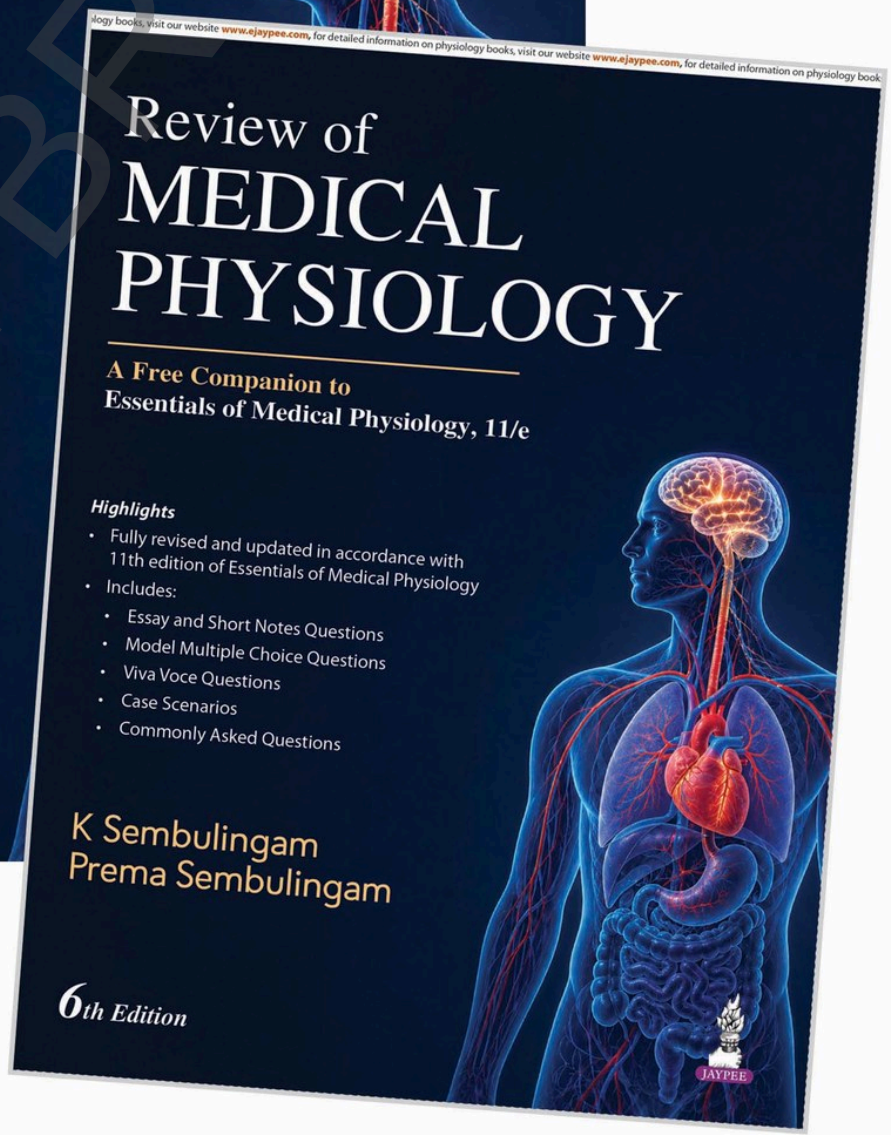


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Chapter 8

Red Blood Cells

CHAPTER OUTLINE

- RED BLOOD CELLS
  - NORMAL RBC COUNT
  - MORPHOLOGY OF RED BLOOD CELLS
- PROPERTIES OF RED BLOOD CELLS
- LIFESPAN RED BLOOD CELLS
- FATE OF RED BLOOD CELLS
- FUNCTIONS OF RED BLOOD CELLS
  - TRANSPORT OF OXYGEN
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  - USE IN BLOOD GROUP DETERMINATION
- APPLIED PHYSIOLOGY: VARIATIONS IN RED BLOOD CELLS
- VARIATIONS IN NUMBER OF RBC
  - PHYSIOLOGICAL VARIATIONS
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- VARIATIONS IN SIZE OF RBC
- VARIATIONS IN SHAPE OF RBC
- VARIATIONS IN STRUCTURE OF RBC
- CLINICAL PHYSIOLOGY OF RBC
  - RBC DISORDERS
  - DIAGNOSIS OF RBC DISORDERS
  - TREATMENT FOR RBC DISORDERS

RED BLOOD CELLS

Red blood cells (RBCs) are the **non-nucleated** formed elements in the blood. Red blood cells are also called **erythrocytes**. Erythro is derived from Greek word erythrós meaning red. RBCs are **larger in number** compared to the other two blood cells, namely white blood cells and platelets.

Red color of RBCs is due to its coloring pigment, **hemoglobin**. RBCs play a vital role in the transport of respiratory gases.

NORMAL RBC COUNT

RBC count ranges between 4 and 5 million/cu mm of blood.

In adult males : 5 million/cu mm.
In adult females : 4.5 million/cu mm.

MORPHOLOGY OF RED BLOOD CELLS

Normal Shape of RBC

Normally, RBC is disk-shaped and biconcave (dumbbell-shaped). The central portion is thinner, and periphery is thicker.

Advantages of biconcave shape of RBC

Biconcave contour of RBC has some mechanical and functional advantages which are given in **Box 8.1**.

Normal Size of RBC

Diameter : 7.2 µ (6.9 to 7.4 µ)
Thickness : At the periphery it is thicker with 2.2 µ and at the center it is thinner with 1 µ **(Fig. 8.1)**. This difference in thickness is because of the biconcave shape

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BOX 8.1: Advantages of biconcave shape of RBCs.

Advantages of biconcave shape of RBCs

1. Biconcave shape helps in equal and rapid diffusion of oxygen and other substances into the interior of cell.
2. Large surface area is provided for absorption or removal of different substances from the cell.
3. Minimal tension is offered on the membrane when volume of cell alters.
4. Because of biconcave shape, while passing through minute capillaries, RBCs squeeze through the capillaries very easily without getting damaged.

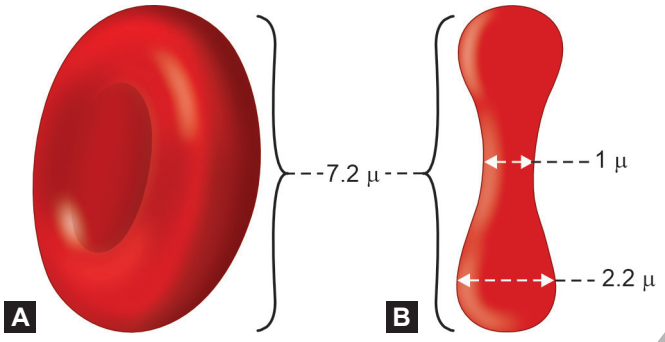


FIGURE 8.1: Dimensions of RBC.
A. Surface view. **B.** Sectioned view.

Surface area : 120 sq μ
Volume : 85 to 90 cu μ

Normal Structure of RBC

Red blood cells are **non-nucleated** (**prokaryotic cells**). Camel is the only mammal, which has nucleated RBC.

Because of the absence of nucleus in human RBC, DNA is also absent. Other organelles such as mitochondria, endoplasmic reticulum and Golgi apparatus also are absent in RBC. Because of absence of mitochondria, the energy is produced from glycolytic process.

RBC has a special type of **cytoskeleton**, made up of proteins **actin** and **spectrin**. Both the proteins are anchored to transmembrane proteins by means of another protein called **ankyrin**. Absence of spectrin results in **hereditary spherocytosis**. In this condition, the cell is deformed, loses its biconcave shape and becomes globular (spherocytic). **Spherocyte** is very fragile and easily ruptured (hemolyzed) in hypotonic solutions.

PROPERTIES OF RED BLOOD CELLS

1. ROULEAUX FORMATION

Rouleaux (singular = rouleau) formation is the piling up of RBCs one above another like a pile of coins, when blood is taken out of blood vessel and allowed to stand without movement **(Fig. 8.2)**. Rouleaux formation is accelerated by plasma proteins globulin and fibrinogen.

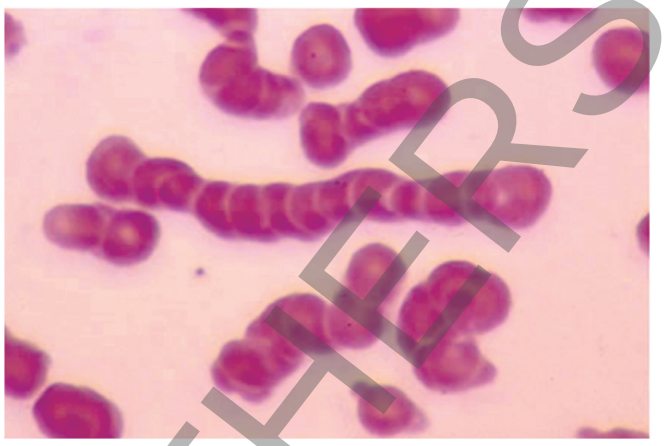


FIGURE 8.2: Rouleaux formation.
(*Courtesy:* Dr Nivaldo Medeiros)

2. PACKED CELL VOLUME

Packed cell volume (PCV) or **hematocrit value** is the proportion of blood occupied by RBCs expressed in percentage. PCV is 45% of the blood and the plasma volume is 55% (Chapters 6 and 11).

3. SPECIFIC GRAVITY

Specific gravity of RBC is 1.092 to 1.101.

4. SUSPENSION STABILITY

It is the ability of RBCs to remain suspended uniformly in the blood during circulation.

LIFESPAN RED BLOOD CELLS

Average lifespan of RBC is about 120 days.

DETERMINATION OF LIFESPAN OF RBC

Lifespan of RBC is determined by radioisotope method. RBCs are tagged with radioactive substances like **radioactive iron** or **radioactive chromium**. The life of RBC is determined by studying the rate of loss of radioactive cells from circulation.

FATE OF RED BLOOD CELLS

After lifetime, **senile RBCs** (old RBCs) are destroyed by macrophages present in spleen and liver. Most of RBCs are destroyed in **spleen.** When RBCs cells become older (120 days), cell membrane becomes more fragile. Diameter of capillaries is less or equal to that of RBC. Younger RBCs can pass through capillaries easily. However, because of fragile nature, the older cells are destroyed while trying to squeeze through capillaries.

Destruction of old cells occurs mainly in capillaries of **red pulp of spleen** because the diameter of splenic capillaries is very small. So, the spleen is called

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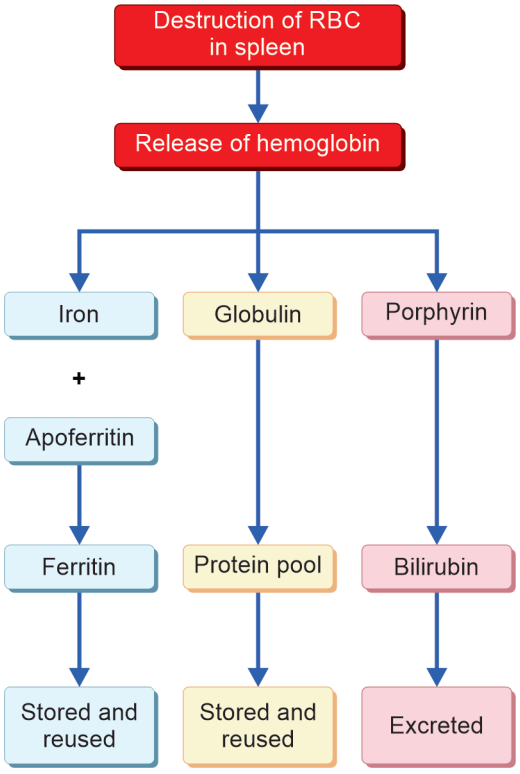


FIGURE 8.3: Fate of RBC.

graveyard of RBCs. Destroyed RBCs are fragmented and hemoglobin is released from fragmented parts.

Hemoglobin is immediately phagocytized by macrophages of the body, particularly macrophages present in liver **(Kupffer cells)**, spleen and bone marrow. Hemoglobin is degraded into iron, globin and porphyrin. Iron combines with protein called **apoferritin** to form **ferritin**, which is stored in the body and reused later. Globin enters the protein depot for later use **(Fig. 8.3)**. Porphyrin is degraded into bilirubin, which is excreted by liver through bile (Chapter 38).

Daily Rate of Destruction of Senile RBCs

Daily 10% RBCs, which are senile, are destroyed in normal young healthy adults. It causes the release of about 0.6 g/ dL of hemoglobin into the plasma. From this 0.9 to 1.5 mg/ dL bilirubin is formed.

Significance of Destruction of Senile RBCs

Destruction of senile RBCs helps in recycling process of iron in the body.

FUNCTIONS OF RED BLOOD CELLS

Major function of RBCs is transport of respiratory gases.

1. TRANSPORT OF OXYGEN FROM LUNGS TO TISSUES

Hemoglobin in RBC combines with oxygen to form **oxyhemoglobin**. About 97% of oxygen is transported in blood in the form of oxyhemoglobin (Chapter 122).

2. TRANSPORT OF CARBON DIOXIDE FROM TISSUES TO LUNGS

Hemoglobin combines with carbon dioxide and form **carbhemoglobin**. About 30% of carbon dioxide is transported in in blood in the form of carbhemoglobin.

RBCs contain a large amount of **carbonic anhydrase**. This enzyme is necessary for formation of bicarbonate from water and carbon dioxide (Chapter 122). Thus, it helps to transport carbon dioxide in the form of bicarbonate from tissues to lungs. About 63% of carbon dioxide is transported in this form.

3. BUFFERING ACTION

Hemoglobin present in RBCs functions as a good buffer. By this action, it regulates the hydrogen ion concentration. And thereby blood plays a major role in the maintenance of **acid-base balance** (Chapter 52).

4. USE IN BLOOD GROUP DETERMINATION

RBCs carry **blood group antigens** like A antigen, B antigen and Rh factor. This helps in determination of blood group of the person and enables to prevent reactions due to incompatible blood transfusion (Chapter 19).

APPLIED PHYSIOLOGY: VARIATIONS IN RED BLOOD CELLS

I. Variations in number of RBC
II. Variations in size of RBC
III. Variations in shape of RBC
IV. Variations in structure of RBC

VARIATIONS IN NUMBER OF RBC

PHYSIOLOGICAL VARIATIONS IN NUMBER OF RBC

Physiological Increase in RBC Count: Physiological Polycythemia

Increase in RBC count is known as **polycythemia**. It occurs in both physiological and pathological conditions. When it occurs in physiological conditions it is called physiological polycythemia. The increase in number during this condition is marginal and temporary.

Physiological polycythemia occurs during the following conditions:

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1. *Age*

At birth, RBC count is 8 to 10 million/cu mm of blood. The count decreases within 10 days after birth due to destruction of RBCs. Due to excess destruction of RBCs and liberation of bilirubin, **physiological jaundice** develops in some newborn babies between 2 to 3 days after birth. Such type of jaundice lasts only for few days.

However, in infants and growing children, RBC count is more than in adults.

2. *Sex*

Before puberty and after menopause in females the RBC count is similar to that in males. During reproductive period of females, the count is less than that of males (4.5 million/ cu mm).

3. *High altitude*

Inhabitants of mountains (above 10,000 feet from mean sea level) have an increased RBC count of more than 7 million/ cu mm. It is due to **hypoxia** (decreased oxygen supply to tissues) at high altitude. Hypoxia stimulates the kidney to secrete a hormone called **erythropoietin**. Erythropoietin in turn stimulates the bone marrow to produce more RBCs **(Fig. 8.4)**.

4. *Muscular exercise*

There is a temporary increase in RBC count after exercise. It is because of mild hypoxia and contraction of spleen. Spleen stores RBCs (Chapter 23). Hypoxia increases sympathetic activity resulting in secretion of adrenaline from adrenal medulla. Adrenaline contracts spleen and RBCs are released into blood **(Fig. 8.5)**.

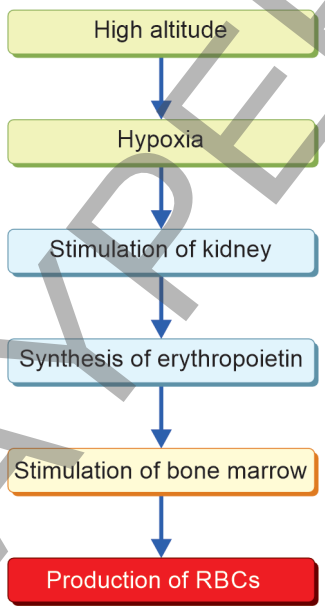


FIGURE 8.4: Physiological polycythemia in high altitude.

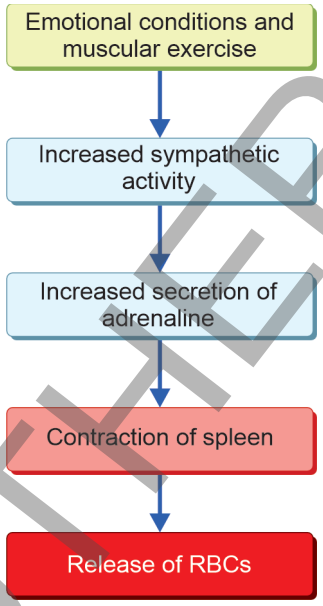


FIGURE 8.5: Physiological polycythemia in emotional conditions and exercise.

5. *Emotional conditions*

RBC count increases during emotional conditions such as anxiety. It is because of an increase in sympathetic activity as in the case of muscular exercise **(Fig. 8.5)**.

6. *Increased environmental temperature*

Increase in atmospheric temperature increases RBC count.

7. *After meals*

There is a slight increase in the RBC count after taking meals because of need for more oxygen for metabolic activities.

Physiological Decrease in RBC Count

Decrease in RBC count occurs during the following physiological conditions:

1. *High barometric pressures*

At high barometric pressures as in deep sea, when oxygen tension of blood is higher, RBC count decreases.

2. *During sleep*

RBC count decreases slightly during sleep and immediately after getting up from sleep.

3. *Pregnancy*

In pregnancy, RBC count decreases because of increase in ECF volume. Increase in ECF volume, increases the plasma volume resulting in **hemodilution**. So, there is a relative reduction in the RBC count.

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PATHOLOGICAL VARIATIONS IN NUMBER OF RBCs

Pathological Increase in RBC Count: Pathological Polycythemia

Pathological polycythemia is the abnormal increase in RBC count above 7 million/cu mm of the blood.

Pathological polycythemia is of **two types**:

1. Primary polycythemia.
2. Secondary polycythemia.

Primary polycythemia: Polycythemia vera

Primary polycythemia or **polycythemia vera** is a disease characterized by persistent increase in RBC count above 14 million/cu mm of blood. It is always associated with increased white blood cell count above 24,000/cu mm of blood. Polycythemia vera occurs in **myeloproliferative disorders** like malignancy of red bone marrow.

Secondary polycythemia

This is secondary to some of pathological conditions such as:

i. Respiratory disorders like **emphysema**.
ii. Congenital heart disease.
iii. **Ayerza's disease:** Condition characterized by polycythemia, cyanosis, dyspnea and sclerosis (hardening of) pulmonary artery.
iv. Chronic carbon monoxide poisoning.
v. Poisoning by chemicals like phosphorus and arsenic.
vi. Repeated mild hemorrhages.

All the above conditions lead to hypoxia which stimulates the release of erythropoietin. Erythropoietin stimulates the bone marrow resulting in increased RBC count.

Pathological Decrease in RBC Count: Anemia

An abnormal decrease in RBC count is called anemia which is described in Chapter 12.

VARIATIONS IN SIZE OF RBC

Under physiological conditions size of RBCs in venous blood is slightly larger than those in arterial blood.

During pathological conditions, variations in size of RBCs are:

A. Microcytes (smaller cells).
B. Macrocytes (larger cells).
C. Anisocytes (cells with different sizes).

MICROCYTES

Microcytes are present in:

1. Iron deficiency anemia.
2. Prolonged forced breathing.
3. Increased osmotic pressure in blood.

MACROCYTES

Macrocytes are present in:

1. Megaloblastic anemia.
2. Decreased osmotic pressure in blood.

ANISOCYTES

Anisocytes are red blood cells without uniform size. Anisocytosis occurs in pernicious anemia.

VARIATIONS IN SHAPE OF RBC

Shape of RBCs is altered in many conditions including different types of anemia:

1. **Crenation:** Shrinkage as in hypertonic conditions.
2. **Spherocytosis:** Globular form as in hypotonic conditions.
3. **Elliptocytosis:** Elliptical shape as in certain types of anemia.
4. **Sickle cell:** Crescentic shape as in sickle cell anemia.
5. **Poikilocytosis:** Unusual variations in shape due to deformed cell membrane. The shape will be of flask, hammer or any other unusual shape.

VARIATIONS IN STRUCTURE OF RBC

1. *Punctate Basophilism*

Striated appearance of RBCs by the presence of dots of **basophilic materials** (porphyrin) is called punctate basophilism. It occurs during conditions like **lead poisoning**.

2. *Ring in RBCs or Goblet Ring*

Ring or twisted strands of basophilic material appear in the periphery of RBCs. This appears in RBCs during some types of anemia.

3. *Howell-Jolly Bodies*

In certain types of anemia, some nuclear fragments called Howell-Jolly bodies are present in ectoplasm of RBCs.

CLINICAL PHYSIOLOGY OF RBC

RBC DISORDERS

Red blood cell disorders involve conditions that impair structure or function of red blood cells.

Common Categories of RBC Disorders

1. **Polycythemia**: Polycythemia (primary) vera and secondary polycythemia. See above.
2. **Anemia**: Different types of anemia ae explained in Chapter 12.
3. **Hemoglobinopathies**: A group of inherited genetic blood disorders affecting production or structure of

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hemoglobin in RBC leading to anemia, fatigue and structural issues like sickling. Refer Chapter 10 for details.

4. **RBC membrane disorder**: An inherited disorder caused by genetic mutations affecting membrane proteins. This leads to formation of spherocytes (round and fragile RBCs) which are prematurely destroyed in spleen.
5. **Enzyme deficiency disorders**: Inherited chronic metabolic disorders characterized by premature destruction of RBCs (hemolytic anemia). These disorders often lead to jaundice, splenomegaly and gallstones.

   Most common disorders:

   i. Glucose-6-phosphate dehydrogenase deficiency.
   ii. Pyruvate kinase deficiency.

DIAGNOSIS OF RBC DISORDERS

Following are the core diagnostic tests for RBC disorders:

i. **Complete blood count (CBC)**: To check RBC count, hematocrit value, hemoglobin content and blood indices such as MCV, MCH, MCHC and CI (Chapter 11).
ii. **Reticulocyte count**: To determine production of sufficient new RBCs form bone marrow. Low count indicates decreased production and high count indicates bleeding or hemolysis.
iii. **Peripheral blood smear**: Microscopic examination to identify morphology, size and shape of RBCs. Very essential to diagnose spherocytosis or sickle cell.
iv. **Osmotic fragility**: To diagnose hereditary spherocytosis.
v. **Iron and nutrient analysis**: Measurement of iron, ferritin, vitamin B_{12} and folate to diagnose anemia.
vi. **Bone marrow biopsy**: To check bone marrow disorder.
vii. **Coombs test**: Antiglobulin test to detect antibodies that destroy RBCs. It indicates of hemolysis and hemolytic anemia.

TREATMENT FOR RBC DISORDERS

i. **Polycythemia vera**: Regular therapeutic phlebotomy to reduce RBC count and administration of blood thinner to prevent blood clotting.
   Therapeutic phlebotomy is a procedure to remove specific amount of blood from a vein, similar to blood donation.
ii. **Medications**: Antibiotics for infections and modulators of immune system.
iii. **Dietary modifications**: Diet with adequate nutrition to improve blood health.
iv. **Anemia**: Refer Chapter 12 for treatment for different types of anemia.

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K Sembulingam PhD is a renowned person in the field of Physiology. He has a long and vast experience of teaching Physiology to undergraduate and postgraduate medical students in various institutions in India and abroad. He has served JJM Medical College and Hospital, Davanagere, Karnataka, MR Medical College, Kalaburagi, Karnataka, India; Sri Ramachandra Medical College and Research Institute, Chennai, Tamil Nadu, India; School of Health Sciences, Universiti Sains Malaysia, Kelantan, Malaysia; Sri Lakshmi Narayana Institute of Medical Sciences, Puducherry, India; Sri Manakula Vinayagar Medical College and Hospital, Puducherry; Shri Sathya Sai Medical College and Research Institute, Nellikuppam, Tamil Nadu; and Madha Medical College and Research Institute, Chennai, Tamil Nadu. In addition to this textbook *Essentials of Medical Physiology*, he has published *Essentials of Physiology for Dental Students* and *Review in Medical Physiology*, which is like a pocketbook for quick revision before the examination. His interest and knowledge in research are commendable. He has published many papers in indexed scientific journals. He was the guide and supervisor for many PhD and postgraduate students in India and Malaysia. His fields of interest in research are Neuroscience, Stress Physiology, Antistressor Effects of Indigenous Plants, Fertility Control, and Evaluation of Autonomic Functional Status.

Prema Sembulingam PhD started her career as Lecturer in MR Medical College, Kalaburagi, Karnataka, India, and step by step gained a long and vast experience of teaching Physiology to undergraduate and postgraduate medical students by serving in various Institutions in India and abroad. She worked as Professor of Physiology in Sri Ramachandra Medical College and Research Institute, Chennai, Tamil Nadu, India; School of Health Sciences, Universiti Sains Malaysia, Kelantan, Malaysia; Sri Lakshmi Narayana Institute of Medical Sciences, Puducherry, India; Sathyabama University Dental College, Chennai, Tamil Nadu; Sri Manakula Vinayagar Medical College and Hospital, Puducherry; Shri Sathya Sai Medical College and Research Institute, Nellikuppam, Tamil Nadu; and Madha Medical College and Research Institute, Chennai, Tamil Nadu. She has coauthored all the books with Dr K Sembulingam. Her research experience is also commendable with many publications to her credit in indexed scientific journals. Her major field of interest in research is evaluation of autonomic functional status. Her other fields of interest are Neuroscience, Stress Physiology, and Antistressor Effects of Indigenous Plants.

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