ALTERATIONS OF ERYTHROCYTE FUNCTION

(CHAPTER 21)

QUESTION 1

Does "anemia" always refer to a decrease in the number of red blood cells (RBCs?)

No. While anemia **can** be associated with a decreased number of RBCs, often it is the **function** of the erythrocytes that is impaired, rather than a decrease in the number present.

Regardless of the cause, the result is the same: the **same volume** of blood will carry **less oxygen** than normal.

QUESTION 2

What do the suffixes -cytic and -chromic refer to?

The various **types of anemia** are classified according to two criteria: the **size** of the RBCs and their **percent hemoglobin content** (MCHC.)

The **size** of the RBCs can described in one of three ways:

microcytic – RBCs are **abnormally small**

normocytic – RBCs are **normal-sized**

macrocytic – RBCs are **abronmally large**

The **hemoglobin content** of the RBCs can described in one of two ways:

hypochromic – **low** hemoglobin content

normochromic – **normal** hemoglobin content

(but decreased **number**)

All five of these criteria occur in **at least one** type of anemia that we will talk about in this unit.

QUESTION 3

Why do the heart rate and respiratory rate increase in anemic patients?

As we mentioned before, anemia prevents the blood from holding (and thus transporting) the normal amount of **O_2** and **CO_2**.

As a result, the body **compensates** by attempting to **bring in more oxygen**, **expel more CO_2**, and **circulate more blood** to keep up.

QUESTION 4

Describe pernicious anemia.

To understand the concept of **pernicious anemia**, we need to review the concept of **intrinsic factor** from A&P.

Intrinsic factor is a protein produced by the **parietal cells** of the stomach, which aids the absorption of vitamin B_{12} in the ileum.

In **pernicious anemia**, a disease process (usually autoimmune or as a sequela of gastric surgery) results in diminished production of **intrinsic factor** in the stomach.

As a result, B_{12} absorption is impaired, which can result in **vitamin B_{12} deficiency**.

Vitamin B_{12} plays an important role in the **maturation** of red blood cells. Without it, new RBCs become **abnormally large** but lack the **hemoglobin content** of healthy RBCs.

This means that pernicious anemia is classified as a **macrocytic hypochromic** anemia.

QUESTION 5

Why is vitamin B_{12} injected for pernicious anemia, when iron is supplemented orally for iron-deficiency anemia?

Vitamin B_{12} physically **cannot** be absorbed enterically in the absence of **intrinsic factor**. As such, if there is a deficit of IF, absorption of PO vitamin B_{12} supplements will be diminished.

Because of this, **parenteral administration** is sometimes required to restore normal, healthy B_{12} levels.

QUESTION 6

Why is iron-deficiency anemia also called microcytic hypochromic anemia?

In contrast to vitamin B_{12} which plays a role in the maturation of RBCs, **iron** is a direct component used in the **synthesis of hemoglobin**.

When there is **insufficient iron** in the body, new RBCs produced are **smaller than normal** and contain **less hemoglobin** because the hemoglobin production is impaired.

Thinking back to our terms earlier, this means that iron-deficiency anemia can be described as **microcytic** and **hypochromic**.

QUESTION 7

What hematological malignancy might develop in sideroblastic anemia?

Sideroblastic anemia is a form of **microcytic hypochromic** anemia in which immature erythrocytes (called **sideroblasts**) **fail to produce hemoglobin** and instead retain deposits of "raw" unused iron.

Sideroblastic anemia can occur as a result of **myelodysplastic syndrome**, a bone marrow disease which is known to progress to **acute myeloblastic leukemia** (AML) in some cases.

QUESTION 8

What are aplastic anemia and post-hemorrhagic anemia?

Aplastic anemia and **post-hemorrhagic** anemia are two examples of **normocytic normochromic** anemias.

As this classification suggests, the RBCs being produced are **fine**; there just aren't **enough** of them.

Aplastic anemia can be inherited, but is often idiopathic. It results in the bone marrow producing less of **all blood cells**, including WBCs and platelets, not just RBCs.

There isn't anything wrong with the RBCs that **are** produced, but the slowed-down bone marrow can't produce enough to keep up with demand.

Post-hemorrhagic anemia is super simple: it describes the loss of RBCs due to **acute bleeding**.

In this case, nothing is wrong with production at all; the RBCs have simply **left the body**.

A transfusion might be necessary, or if the anemia is minor, fluids and time can be enough to return the RBCs to normal levels.

QUESTION 9

What is the underlying problem in hemolytic anemia? Contrast intrinsic and extrinsic mechanisms.

We've talked about the underproduction, malformation, and loss of RBCs; **hemolytic anemia** describes anemia due to the **destruction** of RBCs.

Again, this is a category of **normocytic** anemias, although hemoglobin content may vary depending on the cause.

Intrinsic causes of hemolytic anemia refer to **inherited** or **lifelong** diseases such as **thalassemia** (a congenital decrease in Hgb production) and **sickle-cell disease** (misshapen, "pointy" RBCs.)

Extrinsic causes of hemolytic anemia refer to **non-inherited** causes, such as **infection**, **autoimmune disease**, **lead poisoning**, etc.

QUESTION 10

What is autoimmune hemolytic anemia?

Autoimmune hemolytic anemia is a relatively rare condition in which the body develops **antibodies** against its own red blood cells.

The result is that the immune system improperly views the RBCs as a **foreign threat** and **destroys them**, leading to a sometimes drastic **decrease in RBC count**.

QUESTION 11

Describe polycythemia vera (PCV.)

In contrast to normochromic anemias, **polycythemia vera** is a condition in which the body produces **too many** red blood cells.

There are often no symptoms, but patients with PCV may experience **pain**, **pruritus**, and **erythema** of the extremities.

QUESTION 12

What are the consequences of polycythemia?

The main consequence of polycythemia is "**thick**" blood, which clinically presents as increased **RBC count** and **hematocrit**.

There may be compensatory **hepatosplenomegaly** as the spleen and liver enlarge to speed up the breakdown of RBCs and hemoglobin.