



# Back to Basics: The Complete Blood Count

Understanding the clinical implications behind the numbers.

**ABSTRACT:** In this latest article in the *AJN* series *Back to Basics*—developed to improve nurses' understanding and application of common laboratory diagnostic tests—the author delineates the meaning and function of complete blood count components, highlighting the important pathophysiological evidence they provide and using composite patient scenarios to assist nurses in recognizing findings that can inform their plan of care and produce the best patient outcomes.

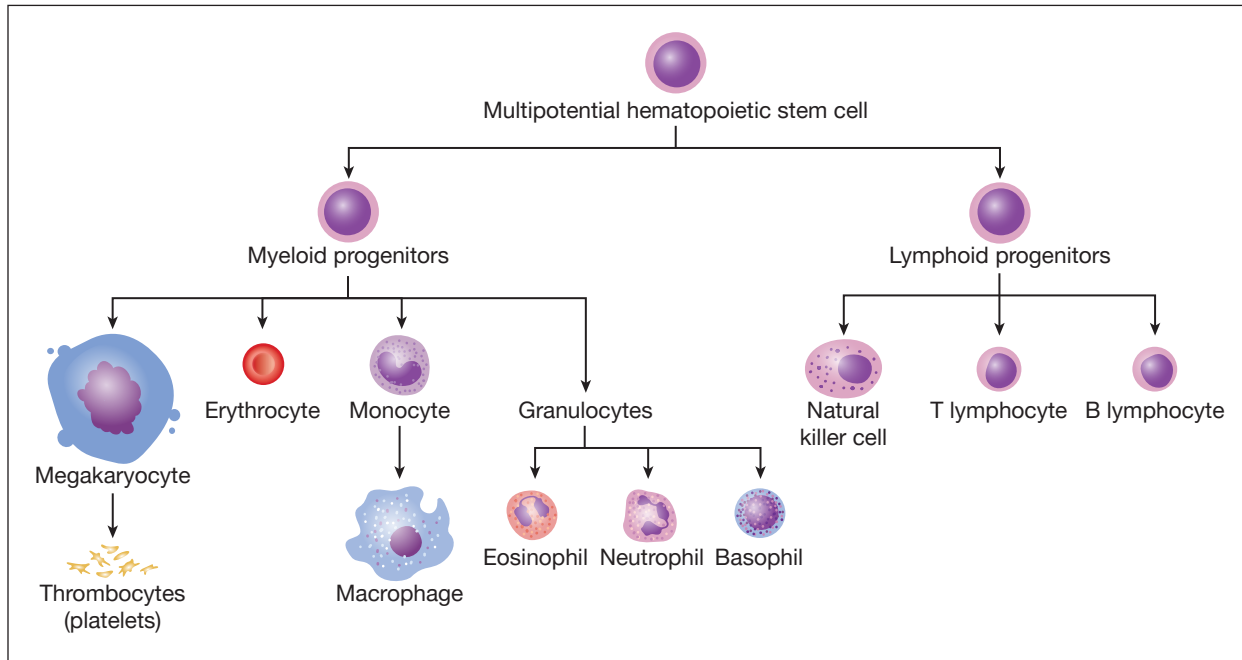
**Keywords:** anemia, complete blood count, hematopoiesis, leukocytosis, leukopenia, neutropenia, red blood cell indices, thrombocytosis, thrombocytopenia, white blood cell

Imagine you're caring for Alison Phillip, an 86-year-old woman who was admitted from a long-term care facility the previous night presenting with fever, chills, and confusion. (This case is a composite based on my experience.) Ms. Phillip has had a long-term indwelling urinary catheter and is being treated for a urinary tract infection. The night nurse reported that after a difficult catheter replacement, the patient's night was uneventful. Morning laboratory values reveal abnormalities in the complete blood count (CBC). The white blood cell (WBC) count is down to 2,800/mm<sup>3</sup>, whereas the previous night it was within the normal range of 5,000 to 10,000/mm<sup>3</sup>. Her hemoglobin has dropped from 10.5 to 8.1 g/dL (normal range for women: 12 to 16 g/dL); the platelet count has been nearly cut in half from 185,000 to 95,000/mm<sup>3</sup> (normal range: 150,000 to 400,000/mm<sup>3</sup>). Did the phlebotomist draw the sample proximal to the forearm IV line running normal saline? Or is something else going on here?

There won't always be a call from the hospital laboratory to warn you if your patient's CBC values have changed significantly. While you'll almost certainly get a call if a CBC value is critical, what

do you do when you review values at the start of your shift and see notable differences from the last shift? Do you wait for rounds, send another sample for verification, or page the attending provider because your patient needs urgent intervention? Nurses who understand blood cell production and function recognize the significance of abnormal findings and CBC patterns and can integrate this knowledge with the patient's history and findings on physical examination in order to make the best clinical decisions. Before considering the specifics of Ms. Phillip's case, let's review hematopoiesis and the pathophysiological conditions that need to be taken into account when making these clinical decisions.

This article continues the theme of *AJN's Back to Basics* series, developed to improve nurses' understanding and application of common laboratory diagnostic tests. Here, I'll discuss the process of hematopoiesis through which blood cells and platelets are produced; the various causes of anemia; other factors that can affect hemoglobin and hematocrit levels; red blood cell (RBC), WBC, and platelet counts; as well as factors that can precipitate thrombocytopenia.

**Figure 1.** The Cellular Components of Blood

Hematopoietic stem cells in the bone marrow differentiate into either myeloid or lymphoid progenitor cells, which in turn create specialized cell types. Illustration by Sara Jarret.

### HEMATOPOIESIS: WHERE IT ALL BEGINS

Erythrocytes (or RBCs), leukocytes (or WBCs), and thrombocytes (platelets) are cellular or formed blood components produced primarily in the bone marrow through the process of hematopoiesis. All formed blood components originate from pluripotent stem cells, which are capable of differentiating into committed progenitor cells (either lymphoid or myeloid stem cells). Lymphoid stem cells may become natural killer cells, T lymphocytes, or B lymphocytes; myeloid stem cells may become monocytes, granulocytes (including neutrophils, eosinophils, and basophils), megakaryocytes (which fragment to form platelets), or erythrocytes (see Figure 1). Control of this process of differentiation and maturation falls primarily on growth factors called cytokines, many of which are colony-stimulating factors, or on other mediators released as part of the inflammatory process (see *Select Mediators in the Regulation of Hematopoiesis* and *Normal CBC Component Values for Adults*).

### COMMON CAUSES OF ANEMIA REFLECTED IN THE CBC

Characterizing anemia can be challenging because of the many potential causes, acquired and hereditary, of both reduced RBC production and increased RBC loss, such as iron deficiency, vitamin deficiency,

### Select Mediators in the Regulation of Hematopoiesis

- **Colony stimulating factors (CSFs)** include granulocyte–monocyte CSFs, granulocyte CSFs, and macrophage CSFs; are produced primarily by macrophages, T cells, and fibroblasts in the marrow; and stimulate proliferation of associated hematopoietic stem cells.
- **Erythropoietin**, produced in the kidney in response to inadequate oxygen delivery, stimulates erythrocyte production.
- **Interferon gamma**, released from T cells and natural killer (NK) cells, stimulates proliferation of myeloid cell lines, and performs other functions.
- **Interleukins (ILs)** are produced by leukocytes for regulating immune responses.
  - IL-1, released from macrophages and monocytes, stimulates other macrophages, initiates inflammation, and produces fever.
  - IL-11, released from bone marrow, increases platelet production.
  - IL-18, released from macrophages in response to microbes, stimulates NK cell proliferation and activity, and performs other functions.
- **Thrombopoietin**, produced primarily in the liver in response to a reduced platelet count, stimulates proliferation and maturation of megakaryocytes, which are released into the circulation and fragment into platelets.
- **Tumor necrosis factor**, released from certain T cells, inhibits further proliferation of T and B lymphocytes.

## Normal CBC Component Values for Adults

### RBC values and indices.

- RBC count:  $4.7\text{--}6.1 \times 10^6$  per  $\text{mm}^3$  for men;  $4.2\text{--}5.4 \times 10^6$  per  $\text{mm}^3$  for women
- Hb: 14–18 g/dL for men; 12–16 g/dL for women
- Hct: 42%–52% for men; 37%–47% for women
- MCV: 80–95 fL/cell
- MCH: 27–31 pg/cell
- MCHC: 32–36 g/dL
- RDW: 11%–14.5%

**Platelet count:** 150,000–400,000/ $\text{mm}^3$

### WBC values.

- Total WBC count: 5,000–10,000/ $\text{mm}^3$
- Neutrophils: 55%–70%; 2,500–8,000/ $\text{mm}^3$
- Lymphocytes: 20%–40%; 1,000–4,000/ $\text{mm}^3$
- Monocytes: 2%–8%; 100–700/ $\text{mm}^3$
- Eosinophils: 1%–4%; 50–500/ $\text{mm}^3$
- Basophils: 0.5%–1%; 25–100/ $\text{mm}^3$

CBC = complete blood count; Hb = hemoglobin; Hct = hematocrit; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; RBC = red blood cell; RDW = red blood cell distribution width; WBC = white blood cell.

acute blood loss, chronic renal failure, and myelosuppression.

**Iron deficiency.** While our bodies efficiently recycle iron from erythrocytes at the end of their life cycle, iron deficiency can occur if there is inadequate dietary iron intake or chronic blood loss. Since iron is necessary to produce hemoglobin, iron deficiency reduces not only hemoglobin, but also hematocrit and the RBC count. In addition, the RBCs produced when iron is deficient are microcytic (small) and hypochromic (pale), causing RBC indices to demonstrate low levels of mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC). Patients with suspected iron deficiency anemia should have additional iron studies and possibly be investigated for chronic blood loss. Patients may be considered for dietary iron supplementation.

**Vitamin deficiency.** Folic acid (vitamin B<sub>9</sub>) deficiency can lead to megaloblastic anemia. In the absence of adequate folic acid, DNA synthesis of blast cells is impaired, resulting in macrocytic (large), normochromic RBCs, seen as elevated MCV and MCH with a normal MCHC. Vitamin B<sub>12</sub> deficiency produces similar RBC indices to folic acid deficiency, but the resultant anemia, called pernicious anemia, can produce serious neurologic symptoms, including ataxia, hallucinations, and delirium.

**Acute blood loss.** In acute blood loss, the RBC count, along with hemoglobin and hematocrit levels, can fall quickly. The RBC indices are all normal because the erythrocytes that remain were produced under normal circumstances. As erythropoietin begins to stimulate the bone marrow and immature RBCs are released into the circulation, additional findings may include a rapidly increasing reticulocyte count. Patients experiencing acute blood loss will likely require volume replacement with crystalloids or, in severe cases, blood transfusion.

**Chronic renal failure.** With chronic renal failure, erythropoietin, the RBC count, and hemoglobin and hematocrit levels are low. In the absence of associated iron deficiency or other impediments to RBC production, the RBC indices MCV, MCH, and MCHC would be normal. Patients with advanced kidney disease often require treatment with a recombinant erythropoietin medication, such as epoetin alfa (Procrit and others), in order to maintain normal hemoglobin levels. Chronic renal failure is also associated with uremia, which can cause myelosuppression (also known as bone marrow suppression). Patients with worsening uremia may need to be considered for dialysis.

**Myelosuppression** arises from the loss of hematopoietic tissue in the bone marrow. In patients with myelosuppression, the CBC reveals pancytopenia (also known as aplastic anemia), a reduction in all blood cell lines. Aplastic anemia may be acquired (as a result of certain infections, toxic exposures, or medications) or, less commonly, inherited. Patients with aplastic anemia are at elevated risk for infection and bleeding due to both leukopenia and thrombocytopenia. If removal of the offending agent doesn't initiate recovery, more advanced therapy, such as transfusion or bone marrow transplant, may be considered.

To test your knowledge of these common types of anemia, see *Differentiating Anemia Etiologies*.<sup>1</sup>

## HYDRATION STATUS

An important variable to consider when interpreting hemoglobin and hematocrit levels is hydration. Both hemoconcentration, which may result from dehydration or the use of diuretics, and hemodilution, a frequent consequence of excessive iv fluid administration or congestive heart failure, are often seen in hospitalized patients.<sup>2</sup> For example, a patient who has been vomiting frequently at home for three days is probably very dehydrated. A normal hemoglobin and hematocrit on presentation to the ED may not reflect the patient's true clinical picture. After rehydration with iv fluids and expansion of blood plasma volume, the patient's hemoglobin and hematocrit levels will likely fall, revealing the patient to be anemic.

Assessing hydration status is thus an essential part of accurately interpreting hemoglobin values and accounting for any sudden changes. In addition

## Differentiating Anemia Etiologies<sup>1</sup>

After reading about the following patients, try to determine the etiology of each patient's anemia and what you would expect their CBC to reveal, based on the laboratory values listed below.

- **Patient 1** presents with fatigue and dizziness upon exertion and reports having had melena two or three times a week for the past two months. A colonoscopy reveals a mass in the ascending colon, with a biopsy pending.
- **Patient 2** was admitted after a ground-level fall with left flank pain and no loss of consciousness. The patient currently takes an oral anticoagulant for a history of atrial fibrillation. Nothing in the patient's history suggests prior bleeding. A computed tomographic scan reveals left retroperitoneal hematoma.
- **Patient 3** has a history of chronic alcohol abuse but no known cirrhosis. Preoperative laboratory tests were obtained prior to elective total knee arthroplasty. No bleeding symptoms are recorded in the patient history.
- **Patient 4** has advanced chronic kidney disease due to diabetic nephropathy. Following routine laboratory tests ordered by the nephrologist, the patient was advised to come to the hospital for evaluation. No bleeding symptoms are reported in the patient history.
- **Patient 5** presents with shortness of breath and chest pain on exertion. The medical record indicates no risk factors for coronary artery disease, no elevation in cardiac isoenzymes, and no electrocardiographic changes suggesting ischemia. The medical history notes phenytoin was prescribed for a seizure disorder. The patient has no signs or symptoms of bleeding. The CBC indicates that the patient has not only anemia, but also leukopenia and thrombocytopenia.

### Laboratory values associated with the various anemias.

- Iron deficiency: low RBC count, low Hb, low Hct, low MCV, low MCH, low MCHC
- Vitamin deficiency: low RBC count, low Hb, low Hct, high MCV, high MCH, normal MCHC
- Acute blood loss: low RBC count, low Hb, low Hct, normal MCV, normal MCH, normal MCHC
- Chronic renal failure: low RBC count, low Hb, low Hct, normal MCV, normal MCH, normal MCHC
- Aplastic anemia: low RBC count, low Hb, low Hct, normal MCV, low MCH, low MCHC

### What the laboratory values reveal.

- **Patient 1:** Melena over a period of two months suggests chronic blood loss, which can lead to iron deficiency anemia, characterized by microcytic, hypochromic cells. The mass, which may be a colon cancer, is the likely source of blood loss. Until the mass is treated, the patient may require iron supplementation or even blood transfusions if the anemia is severe.
- **Patient 2:** Although the patient takes an anticoagulant, the history doesn't provide evidence of chronic blood loss. In addition, there is a large collection of blood in the retroperitoneal space, a result of acute bleeding after injury from the fall. Acute bleeding results in anemia similar to that seen in chronic renal failure: low RBC, Hb, and Hct levels with normal RBC indices. The patient may require volume replacement with crystalloids or a packed transfusion of RBCs, depending on the severity of the anemia.
- **Patient 3:** As someone with chronic alcohol abuse, this patient may have several problems. Chronic alcohol abuse may lead to a poor-quality diet and folic acid deficiency. Additional testing should include folic acid levels, as well as B<sub>12</sub> levels (low B<sub>12</sub> levels are also seen in chronic alcohol abuse). Although the patient history doesn't suggest iron deficiency, a generally poor diet and bleeding complications, which often occur in liver disease, may prompt iron studies to be ordered as well. Patients with chronic alcohol abuse are often prescribed folic acid and multivitamins due to dietary insufficiency.
- **Patient 4:** With chronic renal failure, there is decreased production of erythropoietin, which is essential to stimulate RBC production from bone marrow. As long as there are no other factors contributing to the anemia, RBC indices will be normal. The patient may require administration of a recombinant erythropoietin medication. Other potential contributing factors, such as worsening uremia, may also need to be considered.
- **Patient 5:** This patient has pancytopenia (anemia, leukopenia, and thrombocytopenia) due to myelosuppression, which is consistent with aplastic anemia. Treatment will typically require cessation of phenytoin and possibly a bone marrow biopsy.

CBC = complete blood count; Hb = hemoglobin; Hct = hematocrit; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; RBC = red blood cell.

to reviewing patient histories and intake–output records, nurses must repeatedly assess patients for any changes in vital signs, weight, condition of mucus membranes, urinary output, color and concentration of urine, and lung sounds, as well as for edema and flat or distended jugular veins. Integrating such assessment information with the trend in hemoglobin and hematocrit values is essential in determining whether hemodilution or hemoconcentration could be affecting these values.<sup>2</sup>

### HEMOLYSIS

Evaluating RBCs and hematocrit may also be challenging if specimens are hemolyzed. Although hemolysis may result from certain diseases, it can also occur with errors in collecting the blood sample, such as using a small-gauge needle, exposing the sample to the isopropyl alcohol used to clean the skin, shaking the sample, or subjecting it to excessive centrifugation. High degrees of hemolysis may produce falsely low RBC and hematocrit levels, and falsely high platelet levels and MCHC.<sup>3</sup> Careful attention to appropriate phlebotomy technique is important to ensure accurate results in the CBC.

### THROMBOCYTOPENIA

There are many etiologies for thrombocytopenia, some related to disease process and others to treatments administered by the health care team. Thrombocytopenia is concerning because of the associated increased risk of spontaneous bleeding. Generally, spontaneous bleeding doesn't occur until platelet counts fall below 10,000/mm<sup>3</sup>, but even minor trauma can cause bleeding if counts are below 30,000/mm<sup>3</sup>. Researchers conducting a prospective multicenter study to investigate the epidemiology of thrombocytopenia in ICU patients found that infections associated with sepsis, severe sepsis, and septic shock were the medical conditions most often associated with this condition.<sup>4</sup> Other important etiologies and related factors included viral infections; massive blood product transfusion; prior exposure to certain drugs that reduce platelet levels, including heparin; and cancer. Many participants had multiple etiologies contributing to low platelet counts.

**The role of infections.** Both bacterial and viral infections can precipitate thrombocytopenia. The viruses HIV, hepatitis B and C, and varicella-zoster can do so by suppressing the bone marrow. In sepsis and septic shock, whether the causative agent is viral or bacterial, thrombocytopenia may result from endothelial dysfunction, which precipitates platelet activation and aggregation. As platelets are “consumed” during the formation of microvascular thrombi, tissue perfusion is further impaired, exacerbating the organ dysfunction characteristic of sepsis.

Drug-induced thrombocytopenia can occur through either impaired production or increased

destruction of platelets. Some thrombocytopenia-inducing medications, including quinine, procainamide, and heparin, may result in immune system-mediated platelet destruction, whereas other medications and substances encourage apoptosis (programmed cell death). The latter include aspirin, vancomycin (Vancocin), tamoxifen, methotrexate (Otrexup and others), and (in chronic alcohol abuse) ethanol. Additionally, there are many medications, such as cytotoxic chemotherapy agents and the antibiotic linezolid (Zyvox), that result in myelosuppression.

Since hospitalized patients are frequently given medications that can contribute to thrombocytopenia, evaluation should include the timing of thrombocytopenia onset and patient response to drug discontinuation. Drug-induced thrombocytopenia typically occurs within five to 10 days of drug administration, and platelet counts should start improving one to two days after drug discontinuation.

**Heparin.** Nurses should be familiar with heparin-induced thrombocytopenia (HIT), which is a risk with both unfractionated heparin and low-molecular-weight preparations, such as enoxaparin (Lovenox).

*Type 1 HIT* is a transient, nonimmune-mediated reaction to heparin therapy, which initially encourages platelet aggregation, causing a mild decrease in the platelet count. Type 1 HIT occurs within two days of heparin initiation and resolves with no intervention.

*Type 2 HIT* is, by contrast, a dangerous process mediated by the immune system. In such cases, heparin exposure results not only in platelet aggregation, but also in antibody production. Type 2 HIT typically occurs five to 10 days after first exposure to heparin, though it may occur sooner in patients exposed to heparin within the previous 100 days. Type 2 HIT causes at least a 50% drop in the platelet count. Although platelet aggregation reduces the platelet count, possibly causing thrombocytopenia, the activated platelets actually increase the risk of thrombus formation (especially deep vein thrombosis, pulmonary embolism, and to a lesser extent, arterial thrombosis).<sup>5</sup>

Nurses must be attentive to platelet counts in patients receiving heparin products. A significant drop in the platelet count should prompt a discussion with the attending provider. If type 2 HIT is suspected, the patient should be assessed for related risks and additional laboratory testing, including antibody assays. Patients may need to be prescribed an alternative anticoagulant, which may need to be continued as long as thrombosis is present.

#### **Other common causes of thrombocytopenia.**

Various autoimmune disorders may result in thrombocytopenia. For example, idiopathic thrombocytopenic purpura involves both immune-mediated platelet



**Table 1.** Absolute vs. Differential WBC Counts in Common Conditions

	Allergic Asthma Exacerbation	Tuberculosis	Community-Acquired Pneumonia	Gram-Negative Septic Shock
WBCs	normal	high	high	low
Neutrophils	% and absolute count normal	% low; absolute count normal	% and absolute count high; elevated % bands	% and absolute count low; elevated % bands
Lymphocytes	% and absolute count normal	% and absolute count high	% low; absolute count normal	% high; absolute count normal
Monocytes	% and absolute count normal	% low; absolute count normal	% low; absolute count normal	% and absolute count normal
Eosinophils	% and absolute count high	% low; absolute count normal	% low; absolute count normal	% and absolute count normal
Basophils	% and absolute count normal	% low; absolute count normal	% low; absolute count normal	% and absolute count normal

% = differential; WBC = white blood cell.

destruction and impaired production of platelets, and may occur in isolation or secondary to other conditions, such as chronic liver disease, which decreases production of thrombopoietin; hypersplenism, in which platelets are sequestered in the spleen; or hemodialysis treatment for end-stage renal disease, which could bring it on either by uremia or by platelet trauma that occurs during hemodialysis. Other factors that contribute to platelet loss include hemorrhage, platelet destruction caused by mechanical injury from a prosthetic heart valve, and dilution caused by large-volume blood transfusions.

### LEUKOCYTOSIS AND LEUKOPENIA

Many conditions can affect the WBC count and differential, and the pattern of changes will often point to a specific etiology. Note that because the WBC report expresses each type of WBC as both a differential (percentage) and an absolute count, an increase in the differential of one cell type (such as neutrophils or lymphocytes) may decrease the differential of other cell types, though their absolute count remains normal. However, since monocytes, eosinophils, and basophils make up such a small proportion of the WBC count, increases or decreases in these cells may have only a negligible effect on the differential of other WBC types. See Table 1 for some examples of potential laboratory findings in common conditions.

The following patient scenarios demonstrate why each condition would be expected to produce these findings and the additional assessments or treatments that may be considered in each case. (All cases are composites based on my experience.)

**Status asthmaticus.** A patient with a history of severe allergic asthma has been unable to afford medications such as inhaled or systemic corticosteroids, or rescue inhalers, for the past two months.

The patient's CBC shows absolute eosinophilia, a common finding in allergy-related acute asthma exacerbations. Since eosinophils represent a small percentage of WBCs, the total WBC count and the differential of other WBCs are unlikely to be outside the normal range. If the patient has not previously undergone allergy testing, such testing would now be indicated. Because of the severity of the condition, the patient would be prescribed inhaled bronchodilators and probably systemic corticosteroids as well.

**Tuberculosis.** A patient presenting with hemoptysis, night sweats, and weight loss is later found to have cavitary lesions on chest radiograph. The patient is placed in airborne isolation for presumed tuberculosis, which is not as rare as we would hope. Immigrants from endemic areas; those who travel to areas with a high prevalence of tuberculosis; and those who are immunocompromised, incarcerated, or homeless are all at high risk. Since the immune response to *Mycobacterium tuberculosis* is mediated by T cells, lymphocytosis may be observed in patients with active tuberculosis. Severe lymphocytosis may increase the overall WBC count, reducing the relative percentage of other WBCs, though the absolute count of these WBCs would remain normal. In addition to a multidrug regimen for tuberculosis, this patient should have additional testing, including confirmative diagnostics (sputum cultures for acid-fast bacilli) as well as a risk assessment and possible testing for HIV.

**Community-acquired pneumonia.** A patient presenting with fever, chills, and a productive cough of two days' duration is diagnosed with community-acquired pneumonia. Vital signs and oxygen saturation level by pulse oximetry on room air are normal. Community-acquired pneumonia in a stable patient is likely to produce neutrophilia, an increase in the neutrophil count and differential, as these cells are the first responders to acute infection. As with lymphocytosis,

**Table 2.** Common Changes to the CBC in Cancer

Finding	Mechanism
Anemia—experienced by the majority (up to 60%) of patients with cancer <sup>6</sup>	<ul style="list-style-type: none"> <li>• Iron deficiency—primarily functional, in which inflammation traps iron in macrophages, rendering it unavailable for erythropoiesis despite adequate reserves, but it may also be absolute due to blood loss or inadequate dietary intake</li> <li>• Hypoplastic or aplastic—due to myelosuppression, which may occur with both chemotherapy and radiation therapy</li> <li>• Hemolysis—which results from some malignancies and occurs as an adverse effect of some chemotherapy agents</li> <li>• Folate or vitamin B<sub>12</sub> deficiency—anorexia, nausea, and vomiting may reduce dietary intake, and rapid tumor cell growth may increase demand. Treatment with folate antagonists may also lead to folate deficiency.</li> <li>• Bone marrow replacement—certain cancers may infiltrate the bone and replace healthy bone marrow</li> </ul>
Thrombocytosis—a common red flag for many types of cancer <sup>7</sup>	May be triggered by chemical mediators secreted by the tumor itself. Thrombocytosis can occur even before the cancer is recognized and treatment is initiated and may indicate a poorer prognosis, as it potentiates tumor growth and metastasis and increases risk of thromboembolic events such as deep vein thrombosis and pulmonary embolism.
Thrombocytopenia—the frequency of bleeding complications varies with the type of cancer <sup>8</sup>	May result from myelosuppression due to chemotherapy or radiation therapy or the cancer itself; other contributing factors include malnutrition, kidney disease, and hypersplenism
Leukocytosis	Commonly occurs in many hematologic cancers, though the type of cancer determines which types of white blood cells are affected. For example, neutrophilia may occur in myelocytic leukemias, while lymphocytosis may develop in lymphocytic leukemias and multiple myeloma. <sup>9</sup>
Neutropenia	Antineoplastic medications commonly cause myelosuppression, and in turn, neutropenia, which predisposes the patient to infection. <sup>10</sup>

neutrophilia may increase the overall WBC count, causing a relative reduction in the differential of other WBCs, though their absolute count would remain normal. Additional stimulation of bone marrow may cause the release of immature neutrophils (bands) as well, but an elevated absolute neutrophil count indicates that this patient's bone marrow is “keeping up” with the infection. Additional testing would include blood and sputum cultures to determine the causative organism and the most appropriate antibiotic therapy.

**Gram-negative septic shock.** A patient admitted to the hospital four days ago develops new lung infiltrates, fever, tachycardia, and hypotension with lactic acidosis requiring high-flow oxygen. The patient, who is becoming confused, is diagnosed with septic shock due to hospital-acquired pneumonia, increasing the risk of developing multidrug-resistant infections as well as gram-negative infections, which produce the bacterial endotoxins known to trigger the sepsis response. The patient's CBC and differential indicate leukopenia and neutropenia with “a shift to the left,” meaning there are increasing immature neutrophils in the circulation. A shift to the left with a low neutro-

phil count suggests a poor prognosis; the supply of neutrophils from the bone marrow is not keeping up with the demand.

### MALIGNANCIES

The effects of cancer on the CBC could fill volumes. Many of these effects are specific to particular types of cancer; others occur as adverse effects of cytotoxic chemotherapy regimens. (For changes to the CBC in cancer, see Table 2.<sup>6-10</sup>)

### IN THE CASE OF MS. PHILLIP: IS THERE A PROBLEM?

Let's revisit 86-year-old Ms. Phillip. It is certainly possible there was an error in sampling her blood. However, when you review the remainder of her laboratory values, you note other disturbing findings. The bands are at 15%, substantially above the normal range of 0% to 5%.<sup>11</sup> Her neutrophil differential is low and the absolute count is 1,200/mm<sup>3</sup>, but absolute values of the other leukocytes are all normal. MCV, MCH, and MCHC are slightly below normal as well. After calling the laboratory and verifying that the specimen was drawn out of the arm without an IV line, you suspect a real problem.

New leukopenia with bandemia (elevated bands) suggests severe infection, and possibly that the bone marrow is not keeping up with the demand for mature neutrophils. Given the abnormal RBC indices (microcytosis and hypochromia), you believe the patient may have a chronic anemia, but you're also concerned about the acute drop in hemoglobin. After reviewing her intake–output record, you note that Ms. Phillip received 2.5 liters of iv fluids and her weight is up about 2 kg. Her medication list includes ceftriaxone 1 g iv every 24 hours and subcutaneous enoxaparin 40 mg daily.

When you enter Ms. Phillip's room, you note that she's drowsy but is easily awakened. She remains confused and disoriented just as she was the previous night. Her vital signs are as follows:

- temperature, 95.9°F
- heart rate, 114 beats per minute
- respiratory rate, 24 breaths per minute
- blood pressure, 96/60 mmHg

Her oxygen saturation level by pulse oximetry is 92% on room air. On physical examination, her skin is cool and pale without bruising or lesions. Her heart rate is rapid but regular with 1+ peripheral pulses. Air entry in her lung bases is decreased. The urine in her catheter bag is cloudy and yellow.

Without obvious signs of bleeding (no hematuria from the new catheter insertion and no bruising), you doubt that the drop in her hemoglobin and platelet levels is due to blood loss. Furthermore, you realize that Ms. Phillip's hemoglobin may have dropped as a result of hemodilution. Although the patient is receiving enoxaparin, HIT is unlikely to develop less than a day after her first dose. Knowing Ms. Phillip has an infection with new leukopenia, a shift to the left, and thrombocytopenia, you decide to page the attending provider because you're concerned Ms. Phillip's sepsis is worsening and she is at risk for septic shock.

## THE IMPORTANCE OF THE CBC

Nurses are often the first providers to review a patient's laboratory values, including the CBC. Understanding the function and meaning of the CBC components enables nurses to recognize important patterns commonly seen in serious conditions and informs optimum clinical decision-making. ▼

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Lydia A. Bertschi is an assistant professor at Illinois Wesleyan University School of Nursing in Bloomington. Contact author: [lbertsch@iwu.edu](mailto:lbertsch@iwu.edu). The author and planners have disclosed no potential conflicts of interest, financial or otherwise. A podcast with the author is available at [www.ajnonline.com](http://www.ajnonline.com).

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This activity is also provider approved by the California Board of Registered Nursing, Provider Number CEP 11749 for 2 contact hours. LPD is also an approved provider of continuing nursing education by the District of Columbia, Georgia, and Florida, #50-1223. Your certificate is valid in all states.

### PAYMENT

The registration fee for this test is \$21.95.