CHAPTER SUMMARY

Pain serves as one of the body's defense mechanisms, resulting from stimulation of nociceptors by ischemia, chemical mediators, or distention of tissue.

- The pain pathway may be interrupted at many points, including the receptor site, a peripheral nerve, the spinal cord, or the brain.
- The gate control theory recognizes the role of synapses serving as open or closed gates at points in the pain pathway in the central nervous system. These gates may close under the influence of natural endorphins or other stimuli, thus inhibiting the passage of pain impulses to the brain.
- Descriptions of pain are subjective evaluations by an individual.
- Referred pain occurs when an individual locates the pain at a site other than the actual origin.
- An individual's perception of and response to pain depend on prior conditioning and experiences.
- Acute pain is usually sudden and severe but short term. Chronic pain is milder but long lasting. The person with chronic pain is often fatigued and depressed.
- There are many types of headaches, among them tension, sinus, and migraine, each with different characteristics.
- Analgesics are rated for the severity of pain controlled by the drug; for example, aspirin for mild pain and morphine for severe pain.
- Anesthesia may be classified as local, spinal or regional, or general.

STUDY QUESTIONS

1. Describe the characteristics and role of each of the following in the pain pathway:
   a. nociceptor
   b. C fibers
   c. spinothalamic tract
   d. parietal lobe
   e. reticular formation
   f. endorphins and enkephalins
2. Define and give an example of referred pain.
3. Differentiate the characteristics of acute and intractable pain.
4. List several factors that can alter the perception of pain and the response to pain.
5. Briefly describe six possible methods of pain control.

ADDITIONAL RESOURCES

Mosby's Drug Consult 2006, St. Louis, 2006, Mosby.

Web Sites

http://www.ampainsoc.org *American Pain Society*
http://www.mayoclinic.com *Mayo Clinic*
http://www.webmd.com/pain-management/default.htm
http://www.webmd.com/pain-management/guide/11-tips-for-living-with-chronic-pain
http://www.cancer.org/ssLINK/pain-control-toc

SECTION II
Defense/Protective Mechanisms

CHAPTER 5

Inflammation and Healing

CHAPTER OUTLINE

LEARNING OBJECTIVES

After studying this chapter, the student is expected to:

1. Explain the role of normal defenses in preventing disease.
2. Describe how changes in capillary exchange affect the tissues and the blood components.
3. Compare normal capillary exchange with exchange during the inflammatory response.
4. Describe the local and systemic effects of inflammation.
5. Explain the effects of chronic inflammation.
6. Discuss the modes of treatment of inflammation.
7. Describe the types of healing and the disadvantages of each.
8. List the factors, including a specific example for each, that hasten healing.
9. Identify the classifications of burns and describe the effects of burns.
10. Describe the possible complications occurring in the first few days after a burn.
11. Explain three reasons why the healing of a burn may be difficult.

KEY TERMS

abscess
adhesions
angiogenesis
anorexia
chemical mediators
chemotaxis
collagen
contracture
diapedesis
erythrocyte sedimentation rate (ESR)
exudate
fibrinogen
fibrinous
fibroblast
glucocorticoids
granulation tissue
granuloma
hematocrit
hematopoiesis
hydrostatic pressure
hyperemia
interferons
intra-articular
isoenzymes
leukocyte
leukocytosis
macrophage
malaise
neutrophil
osmotic pressure
perforation
permeability
phagocytosis
purulent
pyrexia
pyrogens
regeneration
replacement
resolution
scar
serous
stenosis
ulcer
vasodilation

Review of Body Defenses

Mechanical Barriers

Defense mechanisms used by the body to protect itself from any injurious agent may be specific or nonspecific. One nonspecific or general defense mechanism is the mechanical barrier such as skin or mucous membrane (often called the first line of defense) that blocks entry of bacteria or harmful substances into the tissues (Fig. 5-1). Associated with these mechanical barriers are body secretions such as saliva or tears that contain enzymes or chemicals that inactivate or destroy potentially damaging material.

Nonspecific Mechanisms

The second line of defense includes the nonspecific processes of phagocytosis and inflammation. **Phagocytosis** is the process by which **neutrophils** (a **leukocyte**) and **macrophages**," randomly engulf and destroy bacteria, cell debris, or foreign matter (see Fig. 5-2). Inflammation involves a sequence of events intended to limit the effects of injury or a dangerous agent in the body. **Interferons** are nonspecific agents that protect uninfected cells against viruses (see Chapter 6).

Specific Mechanisms

The third line of defense is the *specific* defense mechanism in the body (see Chapter 7). It provides protection by stimulating the production of unique antibodies or sensitized lymphocytes following exposure to specific substances. In recent years much effort has been expended on research on the immune system in an effort to increase understanding of the process of the immune response and to create ways to strengthen this defense mechanism.

APPLY YOUR KNOWLEDGE 5-1

Predict three ways that the normal defense systems in the body can fail.

APPLY YOUR KNOWLEDGE 5-2

Predict three factors that can change and interfere with normal capillary exchange.

Review of Normal Capillary Exchange

Usually all capillaries are not open in a particular capillary bed unless the cells' metabolic needs are not being met by the blood supply to the area, or an accumulation of wastes (byproduct of metabolism) occurs. Precapillary sphincters composed of smooth muscle restrict blood flow through some channels. Movement of fluid, electrolytes, oxygen, and nutrients out of the capillary at the arteriolar end is based on the *net* **hydrostatic pressure**. See Chapter 2 and Figure 2-1 for a detailed explanation of fluid shifts between body compartments. The net hydrostatic pressure is based on the difference between the hydrostatic pressure within the capillary (essentially arterial pressure) as compared with the hydrostatic pressure of the interstitial fluid in the tissues as well as the relative osmotic pressures in the blood and interstitial fluid (Fig. 5-2). Differences in concentrations of dissolved substances in the blood and interstitial fluid promote diffusion of electrolytes, glucose, oxygen, and other nutrients across the capillary membrane. Blood cells and plasma proteins (albumin, globulin, and fibrinogen) normally remain inside the capillary.

At the venous end of the capillary hydrostatic pressure is decreased due to the previous movement of fluid into the interstitial fluid space, and **osmotic pressure** in the vessels is relatively high because plasma proteins remain within the capillaries. This arrangement facilitates the movement of fluid, carbon dioxide, and other wastes into the blood. Excess fluid and any proteins are recovered from the interstitial area by way of the lymphatic system and eventually returned to the general circulation.

Physiology of Inflammation

The inflammatory response is a protective mechanism and an important basic concept in pathophysiology.

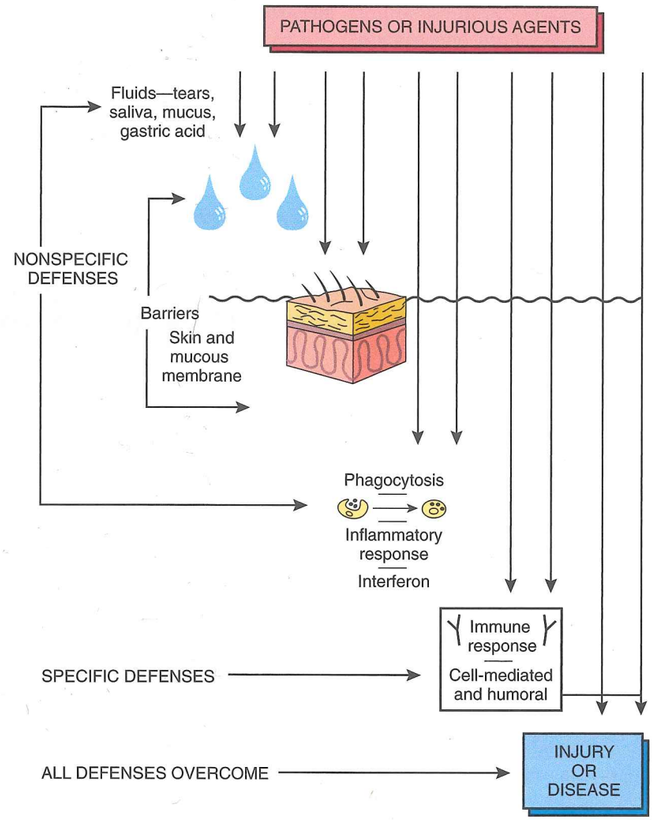


FIGURE 5-1 Defense mechanisms in the body.

Inflammation is a normal defense mechanism in the body and is intended to localize and remove an injurious agent, whatever it may be. You have probably observed the inflammatory process resulting from a cut, an allergic reaction, an insect bite, an infection, or a small burn on your body. The general signs and symptoms of inflammation serve as a *warning* of a problem, which may be hidden within the body.

Inflammation is not the same as infection, although infection is one cause of inflammation. With infection, microorganisms such as a bacteria, viruses, or fungi are always present at the site, causing the inflammation. This microbe can be identified and appropriate treatment instituted to reduce the infection, and the inflammation will subside. When inflammation is caused by an allergy or a burn, no microbes are present.

Definition

Inflammation is the body's nonspecific response to tissue injury, resulting in redness, swelling, warmth, and pain, and perhaps loss of function. Disorders are named using the ending *-itis* for inflammation. The root word is usually a body part or tissue; for example, pancreatitis, appendicitis, laryngitis, or ileitis.

THINK ABOUT 5-1

a. What term would indicate inflammation of the stomach? the liver? the large intestine? a tendon? the heart muscle?
b. Explain the relationship between inflammation and infection.

Causes

Inflammation is associated with many different types of tissue injury. Causes include direct physical damage such as cuts or sprains, caustic chemicals such as acids or drain cleaners, ischemia or infarction, allergic reactions, extremes of heat or cold, foreign bodies such as splinters or glass, and infection.

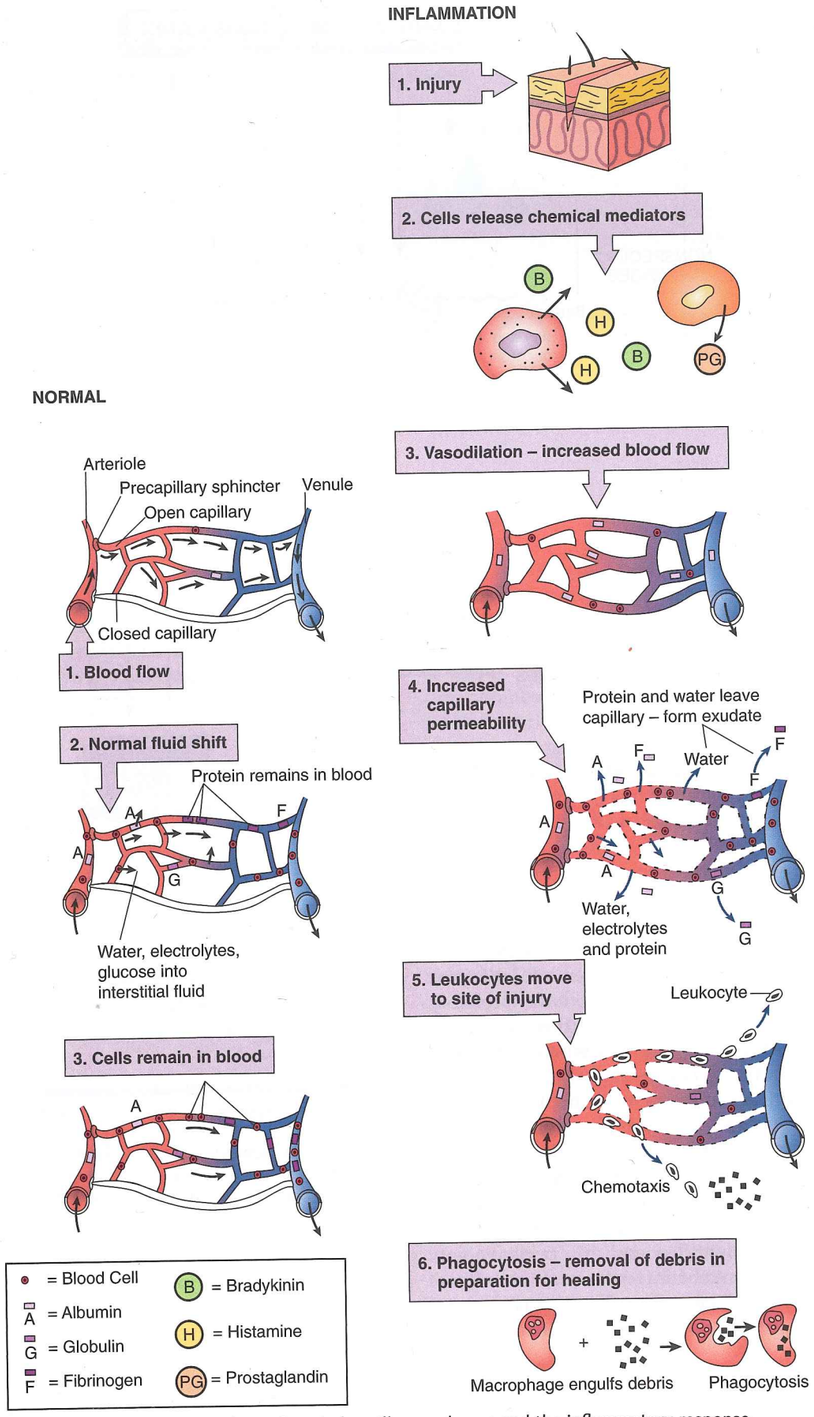


FIGURE 5-2 Comparison of normal capillary exchange and the inflammatory response.

Steps of Inflammation

An injury to capillaries and tissue cells will result in the following reactions:

- Bradykinin is released from the insured cells.
- Bradykinin activates pain receptors.
- Sensation of pain stimulates mast cells and basophils to release histamine.
- Bradykinin and histamine cause capillary dilation.
  - This results in an increase of blood flow and increased capillary permeability.
- Break in skin allows bacteria to enter the tissue.
  - This results in the migration of neutrophils and monocytes to the site of injury.
- Neutrophils phagocytize bacteria.
- Macrophages leave the bloodstream and phagocytose microbes.

Acute Inflammation

Pathophysiology and General Characteristics

The inflammatory process is basically the same regardless of the cause. The timing varies with the specific cause. Inflammation may develop immediately and last only a short time; it may have a delayed onset (e.g., a sunburn), or it may be more severe and prolonged. The severity of the inflammation varies with the specific cause and duration of exposure.

When tissue injury occurs, the damaged mast cells and platelets release **chemical mediators** including histamine, serotonin, prostaglandins, and leukotrienes into the interstitial fluid and blood (Table 5-1). These chemicals affect blood vessels and nerves in the damaged area. Cytokines serve as communicators in the tissue fluids, sending messages to lymphocytes and macrophages, the immune system, or the hypothalamus to induce fever.

Chemical mediators such as histamine are released immediately from granules in mast cells and exert their effects at once. Other chemical mediators such as leukotrienes and prostaglandins must be synthesized from arachidonic acid in mast cells before release and, therefore, are responsible for the later effects, prolonging the inflammation. Many of these chemicals also intensify the effects of other chemicals in the response. Note that many anti-inflammatory drugs and antihistamines reduce the effects of some of these chemical mediators.

Although nerve reflexes at the site of injury cause immediate transient vasoconstriction, the rapid release of chemical mediators results in local **vasodilation** (relaxation of smooth muscle causing an increase in the diameter of arterioles), which causes **hyperemia**, increased blood flow in the area. Capillary membrane **permeability** also increases, allowing plasma proteins to move into the interstitial space along with more fluid (see Fig. 5-2). The increased fluid dilutes any toxic material at the site, while the globulins serve as antibodies, and fibrinogen forms a fibrin mesh around the area in an attempt to localize the injurious agent. Any blood clotting will also provide a fibrin mesh to wall off the area. Vasodilation and increased capillary permeability make up the vascular response to injury.

During the cellular response, leukocytes are attracted by **chemotaxis** to the area of inflammation as damaged cells release their contents. Several chemical mediators at the site of injury act as potent stimuli to attract leukocytes. Leukocytes and their functions are summarized in Table 5-2. First neutrophils (polymorphonuclear

TABLE 5-1 Chemical Mediators in the Inflammatory Response

Chemical	Source	Major Action
Histamine	Mast cell granules	Immediate vasodilation and increased capillary permeability to form exudate
Chemotactic factors	Mast cell granules	For example, attract neutrophils to site
Platelet-activating factor (PAF)	Cell membranes of platelets	Activate neutrophils Platelet aggregation
Cytokines (interleukins, lymphokines)	T lymphocytes, Macrophages	Increase plasma proteins, ESR Induce fever, chemotaxis, leukocytosis
Leukotrienes	Synthesis from arachidonic acid in mast cells	Later response: vasodilation and increased capillary permeability, chemotaxis
Prostaglandins (PGs)	Synthesis from arachidonic acid in mast cells	Vasodilation, increased capillary permeability, pain, fever, potentiate histamine effect
Kinins (e.g., bradykinin)	Activation of plasma protein (kinogen)	Vasodilation and increased capillary permeability, pain, chemotaxis
Complement system	Activation of plasma protein cascade	Vasodilation and increased capillary permeability, chemotaxis, increased histamine release

TABLE 5-2 Function of Cellular Elements in the Inflammatory Response

Leukocytes	Activity
Neutrophils	Phagocytosis of microorganisms
Basophils	Release of histamine leading to inflammation
Eosinophils	Numbers are increased in allergic responses
Lymphocytes	**Activity**
T lymphocytes	Active in cell-mediated immune response
B lymphocytes	Produce antibodies
Monocytes	Phagocytosis
Macrophages	Active in phagocytosis. These are mature monocytes that have migrated into tissues from the blood.

leukocytes [PMNs]) and later monocytes and macrophages collect along the capillary wall and then migrate out through wider separations in the wall into the interstitial area. This movement of cells is termed **diapedesis**. There the cells destroy and remove foreign material, microorganisms, and cell debris by phagocytosis, thus preparing the site for healing. When phagocytic cells die at the site, lysosomal enzymes are released and damage the nearby cells prolonging inflammation. If an immune response (see Chapter 7) or blood clotting occurs, these processes also enhance the inflammatory response.

As excessive fluid and protein collects in the interstitial compartment, blood flow in the area decreases as swelling leads to increased pressure on the capillary bed, and fluid shifts out of the capillary are reduced. Severely reduced blood flow can decrease the nutrients available to the undamaged cells in the area and prevent the removal of wastes. This may cause additional damage to the tissue.

There are naturally occurring defense or control mechanisms in the form of enzymes that inactivate chemical mediators and prevent the unnecessary spread or prolongation of inflammation.

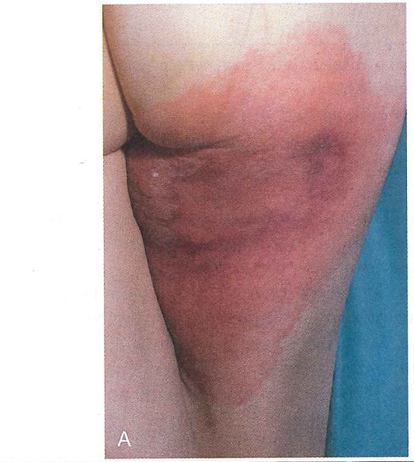
THINK ABOUT 5-2

a. List the local signs and symptoms of inflammation.
b. Consider the last time you experienced tissue injury. Describe the cause of the injury and how inflammation developed.

Local Effects

The *cardinal* signs of inflammation are redness (rubor or erythema), heat, swelling, and pain:

- Redness and warmth are caused by increased blood flow into the damaged area (Fig. 5-3).



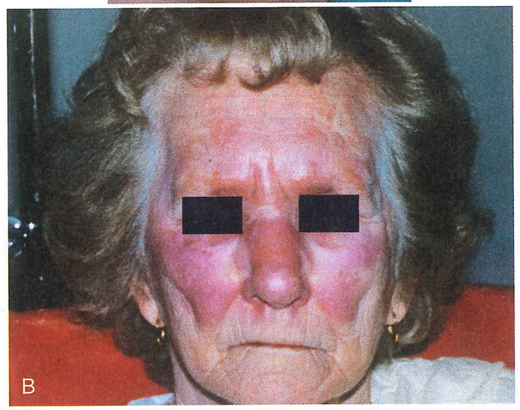


FIGURE 5-3 **A,** Erysipelas (cellulitis). *(From Lookingbill D, Marks J:* Principles of Dermatology, *ed 3, Philadelphia, 2000, WB Saunders.)* **B,** Erysipelas of the face caused by group A Streptococcus. *(From Mahon CR, Lehman DC, Manuselis G:* Textbook of diagnostic microbiology, *ed 3, St. Louis, 2007, Saunders.)*

- Swelling or edema is caused by the shift of protein and fluid into the interstitial space.
- Pain results from the increased pressure of fluid on the nerves, especially in enclosed areas, and by the local irritation of nerves by chemical mediators such as bradykinins.
- Loss of function may develop if the cells lack nutrients or swelling interferes mechanically with function, as happens in restricted joint movement.

Exudate refers to a collection of interstitial fluid formed in the inflamed area. The characteristics of the exudate vary with the cause of the trauma:

- **Serous** or watery exudates consist primarily of fluid with small amounts of protein and white blood cells. Common examples of serous exudates occur with allergic reactions or burns.
- **Fibrinous** exudates are thick and sticky and have a high cell and fibrin content. This type of exudate increases the risk of **scar** tissue in the area.
- **Purulent** exudates are thick, yellow-green in color, and contain more leukocytes and cell debris as well as microorganisms. Typically, this type of exudate indicates bacterial infection, and the exudate is often referred to as "pus."
- An **abscess** is a localized pocket of purulent exudate or pus in a solid tissue (e.g., around a tooth or in the brain).
- A hemorrhagic exudate may be present if blood vessels have been damaged.

Systemic Effects

Other general manifestations of inflammation include mild fever, **malaise** (feeling unwell), fatigue, headache, and **anorexia** (loss of appetite).

Fever or **pyrexia** (low grade or mild) is common if inflammation is extensive. If infection has caused the inflammation, fever can be severe, depending on the particular microorganism. However, high fever can be beneficial if it impairs the growth and reproduction of a pathogenic organism. Fever results from the release of **pyrogens**, or fever-producing substances (e.g., interleukin-1), from white blood cells (WBCs) or macrophages (Fig. 5-4). Pyrogens circulate in the blood and cause the body temperature control system (the thermostat) in the hypothalamus to be reset at a higher level. Heat production mechanisms such as shivering are activated to increase cell metabolism. Involuntary cutaneous vasoconstriction characterized by pallor and cool skin reduces heat loss from the body. Voluntary actions such as curling up or covering the body conserve heat. These mechanisms continue until the body temperature reaches the new, higher setting. Following removal of the cause, body temperature returns to normal by reversing the mechanisms.

THINK ABOUT 5-3

a. What are the physiologic changes that occur when the cause of a fever is removed?
b. Explain the differences among serous, fibrinous, and purulent exudates.

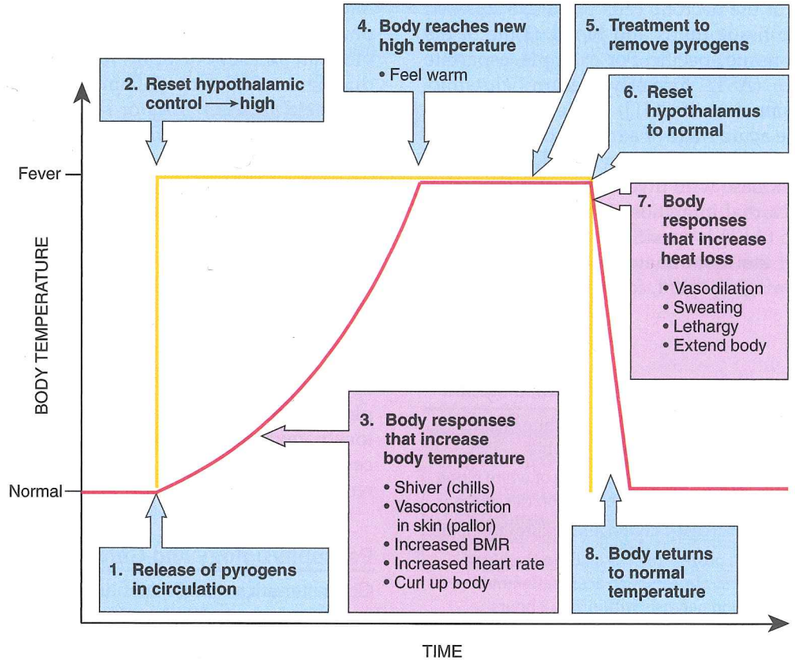


FIGURE 5-4 The course of a fever.

Diagnostic Tests

Refer to the *normal values* shown on the inside front cover of this book.

Leukocytosis (increased white blood cells in the blood), elevated serum C-reactive protein (CRP), an elevated **erythrocyte sedimentation rate or ESR**, and increased plasma proteins and cell enzymes in the serum are nonspecific changes (Table 5-3); they do not indicate the particular cause or site of inflammation. They provide helpful screening and monitoring information when a problem is suspected or during treatment. In patients with leukocytosis, there is often an increase in *immature* neutrophils, commonly referred to as "a shift to the left." A *differential* count (the proportion of each type of WBC) may be helpful in distinguishing viral from bacterial infection. Allergic reactions commonly produce eosinophilia. Examination of a peripheral blood smear may disclose significant numbers of abnormal cells, another clue as to the cause of a problem. Increased circulating plasma proteins (**fibrinogen**, prothrombin, and alpha-antitrypsin) result from an increase in protein synthesis by hepatocytes.

Cell enzymes and more specific **isoenzymes** may be elevated in the blood in the presence of severe inflammation and necrosis. These may be helpful in locating the site of the necrotic cells that have released the enzymes into tissue fluids and blood. Some of the enzymes are not tissue specific. For example, aspartate aminotransferase (AST, formerly serum glutamic-oxaloacetic transaminase [SGOT]) is elevated in liver disease and in the acute stage of a myocardial infarction (heart attack). However, the isoenzyme CK-MB (isoenzyme of creatine kinase with myocardial component) is specific for myocardial infarction. The enzyme alanine aminotransferase (ALT) is specific for the liver.

If the cause of the inflammatory response is a brief exposure to a damaging agent, for instance, touching a hot object, the response often subsides in approximately 48 hours. Vascular integrity is regained, and excess fluid and protein are recovered by the lymphatic capillaries and returned to the general circulation. The manifestations of inflammation gradually decrease. Otherwise inflammation persists until the causative agent is removed (Fig. 5-5).

The amount of necrosis that occurs depends on the specific cause of the trauma and the factors contributing to the inflammatory response. Extensive necrosis may lead to **ulcers** or erosion of tissue. For example, gingivitis or stomatitis in the oral cavity often leads to painful ulcerations in the mouth, and inflammation in the stomach may result in peptic ulcers.

TABLE 5-3 Changes in the Blood with Inflammation

Leukocytosis	Increased numbers of white blood cells, especially neutrophils
Differential count	Proportion of each type of white blood cell altered, depending on the cause
Plasma proteins	Increased fibrinogen and prothrombin
C-reactive protein	A protein not normally in the blood, but appears with acute inflammation and necrosis within 24-48 hours
Increased ESR	Elevated plasma proteins increase the rate at which red blood cells settle in a sample
Cell enzymes	Released from necrotic cells and enter tissue fluids and blood: may indicate the site of inflammation

Potential Complications

Local complications depend on the site of inflammation. For example, inflammation in the lungs may impair the expansion of the lungs, decreasing the diffusion of oxygen. Inflammation of a joint may affect its range of movement.

Infection may develop in an inflamed tissue because microorganisms can more easily penetrate when the skin or mucosa is damaged and the blood supply is impaired (see Fig. 5-14). Foreign bodies often introduce microbes directly into the tissue. Some microbes resist phagocytosis, and the inflammatory exudate itself provides an excellent medium for microorganism to reproduce and colonize the inflamed area.

Skeletal muscle spasms or strong muscle contractions may be initiated by inflammation resulting from musculoskeletal injuries such as sprains, tendinitis, or fractures. A spasm is likely to force the bones of a joint out of normal alignment, thus causing additional pressure on the nerves and increasing the pain.

Chronic Inflammation

Chronic inflammation may develop following an *acute* episode of inflammation when the cause is not completely eradicated. Or inflammation may develop insidiously owing to *chronic* irritation such as smoking, certain bacteria, or long-term abnormal immune responses.

Pathophysiology and General Characteristics

Characteristics of chronic inflammation include less swelling and exudate but the presence of more lymphocytes, macrophages, and **fibroblasts** (connective tissue cells) than in acute inflammation. Frequently more tissue destruction occurs with chronic inflammation. More collagen is produced in the area, resulting in more fibrous scar tissue forming. A **granuloma**, a small mass of cells with a necrotic center and covered by connective tissue, may develop around a foreign object such as a splinter, or as part of the immune response in some infections such as tuberculosis.

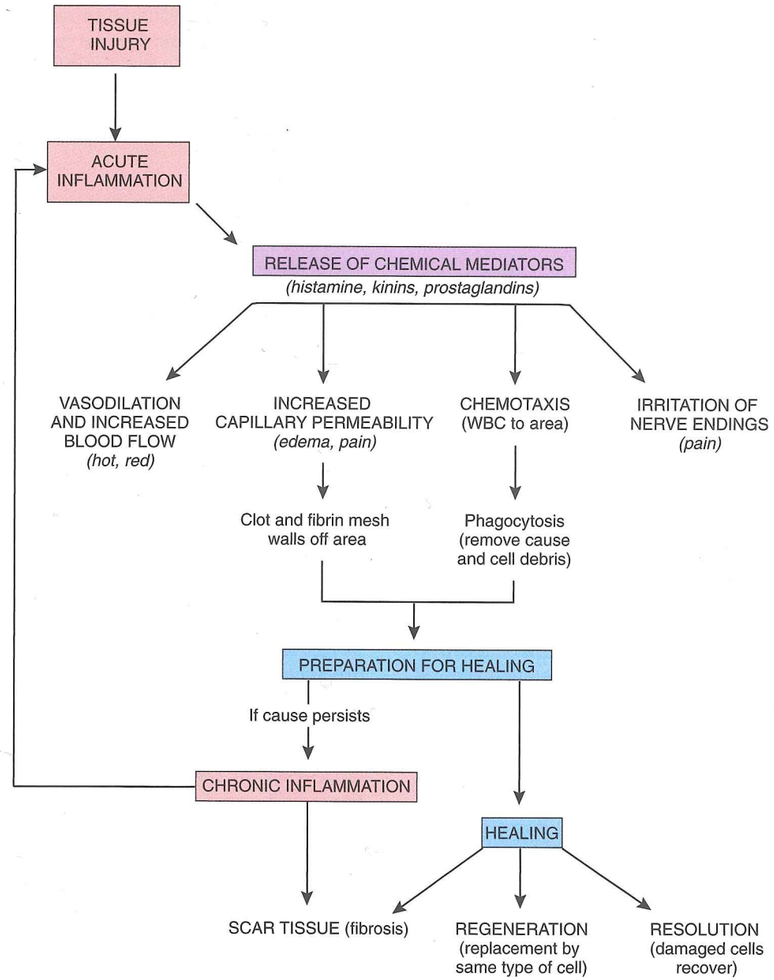


FIGURE 5-5 The course of inflammation and healing.

Potential Complications

Disorders such as rheumatoid arthritis are characterized by chronic inflammation with periodic exacerbations of acute inflammation. Deep *ulcers* may result from severe or prolonged inflammation because cell necrosis and lack of cell regeneration cause erosion of tissue. This in turn can lead to complications such as **perforation** (erosion through the wall) of viscera or development of extensive scar tissue.

THINK ABOUT 5-4

a. Describe three differences between acute and chronic inflammation.
b. Describe three changes in the blood with acute inflammation.

Treatment of Inflammation

Drugs

Acetylsalicylic acid (aspirin, ASA) has long been used as an anti-inflammatory agent, sometimes in very large doses (Table 5-4). This drug decreases prostaglandin

TABLE 5-4 Comparison of Drugs Used to Treat Inflammation

Actions	ASA	Acetaminophen	NSAID	Glucocorticoid	COX-2
Antiinflammatory	Yes	*No*	Yes	Yes	Yes
Analgesia	Yes	Yes	Yes	*No*	Yes
Antipyretic	Yes	Yes	Yes	*No*	*No*
Adverse Effects					
Allergy*	Yes	*No*	Yes	*No*	*Yes*
Delays blood clotting	Yes	*No*	Yes	*No*	*No*
Risk of infection	*No*	*No*	*No*	Yes	*No*
GI distress	Yes	*No*	Yes	Yes	May occur
Stomach ulceration	Yes	*No*	Yes	Yes	May occur
Edema or Increased BP	*No*	*No*	*No*	Yes	May occur
MI or CVA	*No*	*No*	*No*	*No*	May occur
Liver damage	*No*	*No*	*No*	*No*	May occur

*Note allergic reactions may occur with administration of any drug.

synthesis at the site of inflammation, reducing the inflammatory response. Acetylsalicylic acid reduces pain (analgesic effect) and fever (antipyretic effect), which are often helpful. However, ASA is never recommended for children with viral infections because the combination of ASA and a viral infection is believed to contribute to the development of Reye's syndrome, a serious complication involving the brain and liver, which may be fatal. Many individuals are allergic to ASA and similar anti-inflammatory drugs. For others, the drug may cause irritation and ulcers in the stomach. An enteric-coated tablet (the tablet coating does not dissolve until it reaches the small intestine) is available, as are drugs to reduce acid secretion in the stomach to reduce this risk. Anti-inflammatory drugs also interfere with blood clotting by reducing platelet adhesion, and therefore cannot be used in all conditions. Also it is usually necessary to discontinue taking ASA for 7 to 14 days before any surgical procedure to prevent excessive bleeding.

Acetaminophen (Tylenol or Paracetamol) decreases fever and pain, but does not diminish the inflammatory response.

THINK ABOUT 5-5

a. Based on your knowledge of the normal physiology of the stomach, explain why intake of food or milk with a drug reduces the risk of nausea and irritation of the stomach?
b. Why might an individual taking large quantities of ASA need to be monitored for the presence of blood in the feces?

Nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen (Advil or Motrin), piroxicam (Feldene), or diclofenac sodium (Arthrotec) are now used extensively to treat many types of inflammatory conditions. These drugs have anti-inflammatory, analgesic, and antipyretic activities. They act by reducing production of prostaglandins. They are used to treat inflammation in the musculoskeletal system, both acute injuries and long-term problems, such as rheumatoid arthritis. Also, they have become the treatment of choice for many dental procedures when an analgesic and anti-inflammatory are required. Ibuprofen has been recommended for many disorders, including menstrual pain and headache. The side effects are similar to those of aspirin, but are less severe. These drugs are available as oral medications, and some, such as ibuprofen, are available in small doses without a prescription.

A newer type of NSAID is celecoxib (Celebrex), which appears to be effective without unwanted effects on the stomach. This group of drugs (COX-2 inhibitors) is currently under further investigation following the withdrawal from the market of one drug in this class (rofecoxib, Vioxx). This followed reports of serious side effects such as increased incidence of heart attacks. This is an example of the necessity for long-term data collection from a large population to determine all the facts about new drugs or medical procedures.

Corticosteroids or steroidal anti-inflammatory drugs, are synthetic chemicals that are related to the naturally occurring glucocorticoids (hydrocortisone), hormones produced by the adrenal cortex gland in the body (see Chapter 16). These drugs are extremely valuable in the short-term treatment of many disorders, but also have significant undesirable effects that may affect health care.

The beneficial anti-inflammatory effects of glucocorticoids include:

- Decreasing capillary permeability and enhancing the effectiveness of the hormones epinephrine and norepinephrine in the system; thus, the vascular system is stabilized
- Reducing the number of leukocytes and mast cells at the site, decreasing the release of histamine and prostaglandins
- Blocking the immune response, a common cause of inflammation

The chemical structure of the drug has been altered slightly to enhance its anti-inflammatory action and reduce the other, less desirable effects of the hormone. These drugs can be administered as oral tablets, creams and ointments for topical application, or injections, both local and systemic. Examples include prednisone (oral), triamcinolone (topical), methylprednisolone (**intra-articular**—into joint), dexamethasone (intramuscular [IM] or intravenous [IV] injections), and beclomethasone dipropionate (Beclovent [inhaler]).

However, with long-term use and high dosages of glucocorticoids, marked *side effects* occur, similar to Cushing's disease (see Chapter 16). These side effects (or adverse effects) should be considered when taking a medical history from a patient because they may complicate the care of the person.

The adverse effects of glucocorticoids include:

- Atrophy of lymphoid tissue and reduced numbers of WBCs, leading to an increased risk of infection and a decreased immune response
- Catabolic effects (increased tissue breakdown with decreased protein synthesis and tissue regeneration), including osteoporosis (bone demineralization), muscle wasting, and a tendency to thinning and breakdown of the skin and mucosa (e.g., peptic ulcer)
- Delayed healing
- Delayed growth in children
- Retention of sodium and water, often leading to high blood pressure and edema
  - Increases gluconeogenesis causing a rise is blood sugar

THINK ABOUT 5-6

a. Explain why healing could be delayed in individuals taking glucocorticoids over a long period of time.

One additional consideration with the long-term use of steroids involves the effect of increased intake of glucocorticoids on the normal feedback mechanism in the body, leading to a reduction of the normal secretion of the natural hormones and atrophy of the adrenal gland. Therefore, sudden cessation of the administration of glucocorticoid drugs or the presence of increased stress may cause adrenal crisis (similar to shock) because insufficient glucocorticoids are available in the body.

To lessen the risk of serious side effects, it is best to limit use of glucocorticoids to minimal dosages in the treatment of acute episodes. Intermittent drug-free time periods (drug holidays) are recommended during long-term therapy. Whenever the drug is discontinued, the dosage should be gradually decreased over a period of days to allow the body's natural hormone secretions to increase to normal levels. Adrenocorticotropic hormone (ACTH) therapy is used for long-term therapy in some patients because it stimulates the patient's glands to produce more cortisol. The risk of adrenal shock is less because glandular atrophy does not occur.

A brief comparison of drugs used to treat inflammation is shown in Table 5-4.

Other drugs, such as analgesics for pain, antihistamines, and antibiotics to prevent secondary infection may be required, depending on the cause of the inflammation.

First Aid Measures

First aid directives for injury-related inflammation frequently recommend the RICE approach: Rest, Ice, Compression, and Elevation. Cold applications are useful in the early stage of acute inflammation. Application of cold causes local vasoconstriction, thereby decreasing edema and pain. The use of hot or cold applications during long-term therapy and recovery periods depends on the particular situation. In some instances, for example, acute rheumatoid arthritis, heat, and moderate activity may improve the circulation in the affected area, thereby removing excess fluid, pain-causing chemical mediators, and waste metabolites, as well as promoting healing.

Other Therapies

It is often helpful to keep an inflamed limb elevated to improve fluid flow away from the damaged area. Compression using elastic stockings or other supports may reduce the accumulation of fluid.

Mild-to-moderate exercise is useful in cases of many chronic inflammatory conditions in which improved blood and fluid flow is beneficial and mobility could be improved.

Other treatment measures, including physiotherapy and/or occupational therapy, may be necessary to maintain joint mobility and reduce pain, although splints may be required during acute episodes to prevent contractures, and fixed abnormal joint positions. Rest and adequate nutrition and hydration are also important.

Healing

Types of Healing

Healing of a wound area can be accomplished in several ways.

- **Resolution** is the process that occurs when there is minimal tissue damage. The damaged cells recover,

and the tissue returns to normal within a short period of time; for example, after a mild sunburn.

- **Regeneration** is the healing process that occurs in damaged tissue in which the cells are capable of mitosis. Some types of cells (e.g., epithelial cells) are constantly replicating, whereas other cells such as hepatocytes in the liver are able to undergo mitosis when necessary. The damaged tissue is thus replaced by identical tissue from the proliferation of nearby cells. This type of healing may be limited if the organization of a complex tissue is altered. For instance, sometimes fibrous tissue develops in the liver, distorting the orderly arrangement of cells, ducts, and blood vessels. Although nodules of new cells form, they do not contribute to the overall function of the liver.

THINK ABOUT 5-7

a. Which types of cells can regenerate? Name three types that cannot regenerate.
b. Explain why it is often advisable to elevate an inflamed limb.

- **Replacement** by connective tissue (scar or *fibrous tissue* formation) takes place when there is extensive tissue damage or the cells are incapable of mitosis; for example, the brain or myocardium. The wound area must be filled in and covered by some form of tissue. Chronic inflammation or complications such as infection result in more fibrous material.

Healing by first intention refers to the process involved when the wound is clean, free of foreign material and necrotic tissue, and the edges of it are held close together, creating a minimal gap between the edges. This type of healing is seen in some surgical incisions. Healing by second intention refers to a situation in which there is a large break in the tissue and consequently more inflammation, a longer healing period, and formation of more scar tissue. A compound fracture would heal in this manner.

The Healing Process

The process of tissue repair begins following injury when a *blood clot* forms and seals the area. Inflammation develops in the surrounding area (Fig. 5-6). After 3 to 4 days, foreign material and cell debris have been removed by phagocytes, monocytes, and macrophages, and then **granulation tissue** grows into the gap from nearby connective tissue.

Granulation tissue is highly vascular and appears moist and pink or red in color. It contains many new capillary buds from the surrounding tissue. This tissue is very fragile and is easily broken down by microorganisms or stress on the tissue (Fig. 5-7).

THINK ABOUT 5-8

What often happens if you pull a scab off a wound too early? Describe the appearance of the tissue.

At the same time as the wound cavity is being filled in, nearby *epithelial* cells undergo mitosis, extending across the wound from the outside edges inward. Shortly, fibroblasts, connective tissue cells, enter the area and produce **collagen**, a protein that is the basic component of scar tissue and provides strength for the new repair. As well, fibroblasts and macrophages produce growth factors (cytokines) in the area for the purpose of attracting more fibroblasts, which act as mitogens to stimulate epithelial cell proliferation and migration, and promote development of new blood vessels (**angiogenesis**) in the healing tissue.

Gradually cross-linking and shortening of the collagen fibers promote formation of a tight, strong scar. The capillaries in the area decrease, and the color of the scar gradually fades. It is important to remember that scar tissue is not normal, functional tissue, nor does it contain any specialized structures such as hair follicles or glands. It merely fills the defect or gap in the tissue. As scar tissue matures over time, it gains strength, but may also contract, causing increased tension on normal tissues.

THINK ABOUT 5-9

a. Which would heal more rapidly, a surgical incision in which the edges have been stapled closely together, or a large, jagged tear in the skin and subcutaneous tissue? Why?
b. Even after a long period of healing, explain how the scar tissue from a wound will be different from the surrounding undamaged tissue.

One area of current research is "tissue engineering," the search for new methods of replacing damaged tissue where regeneration is not possible; for example, extensive burns, deep ulcers, or cardiac muscle death. Biosynthetic skin is now available and research on other tissues uses a scaffold of synthetic material on which new cells can grow in an organized fashion. Growth factors and mitogens are released from the scaffolding as cells begin to metabolize nutrients in the scaffolding. Cells to populate the engineered tissue may be from a person's own stem cells, cord blood that has been stored, or a stem cell line maintained by the laboratory. Research is progressing, but to date (2012) no solid organs have been produced and used in clinical practice to replace a damaged organ. Ethical concerns regarding cost and access to commercially produced organs are important and need to be addressed before commencing therapies with this technology.

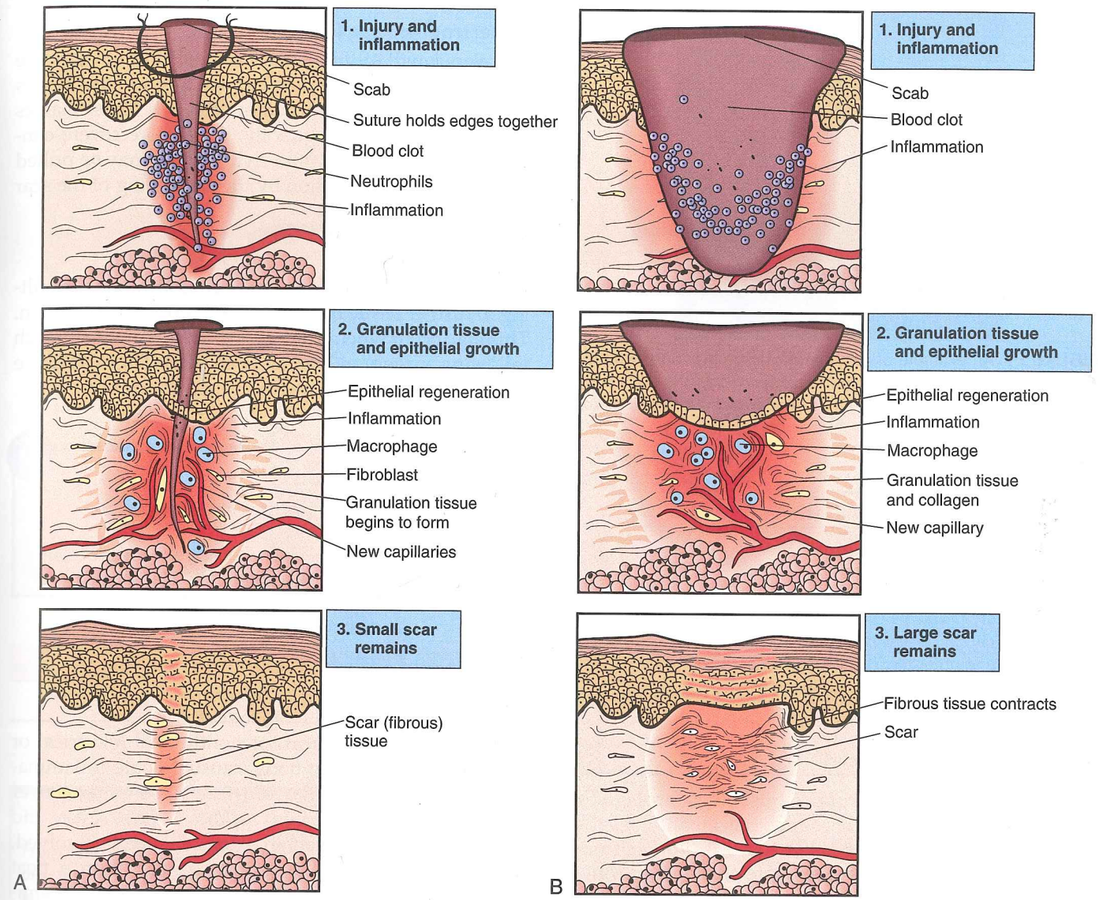


FIGURE 5-6 The healing process.

Factors Affecting Healing

A small gap in the tissue results in complete healing within a short period of time and with minimal scar tissue formation. A large or deep area of tissue damage requires a prolonged healing time and results in a large scar.

Many factors can promote healing or delay the process (Boxes 5-1 and 5-2).

BOX 5-1 Factors Promoting Healing

- Youth
- Good nutrition: protein, vitamins A and C
- Adequate hemoglobin
- Effective circulation
- Clean, undisturbed wound
- No infection or further trauma to the site

Complications due to Scar Formation

Loss of Function

Loss of function results from the loss of normal cells and the lack of specialized structures or normal organization in scar tissue. For example, if scar tissue replaces normal skin, that area will lack hair follicles, glands, and sensory nerve endings. In a highly organized organ such as the kidney, it is unlikely that the new tissue will fit the pattern of blood vessels, tubules, and ducts of the normal kidney; therefore the replacement tissue will not provide normal function.

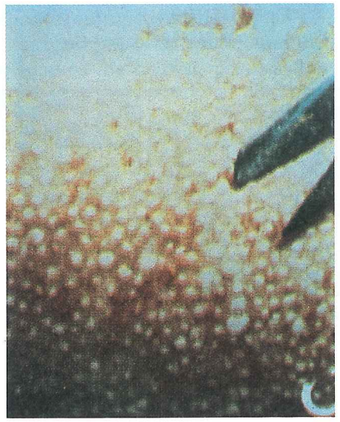


FIGURE 5-7 An example of granulation tissue in a burn wound. *(Courtesy of Judy Knighton, Clinical Nurse Specialist, Ross Tilley Burn Center, Sunnybrook and Women's College Health Center, Toronto, Ontario, Canada.)*

BOX 5-2 Factors Delaying Healing

- Advanced age, reduced mitosis
- Poor nutrition, dehydration
- Anemia (low hemoglobin)
- Circulatory problems
- Certain chronic diseases, such as diabetes
- Presence of other disorders such as diabetes or cancer
- Irritation, bleeding, or excessive mobility
- Infection, foreign material, or exposure to radiation
- Chemotherapy treatment
- Prolonged use of glucocorticoids

Contractures and Obstructions

Scar tissue is nonelastic and tends to shrink over time. This process may restrict the range of movement of a joint and eventually may result in fixation and deformity of the joint, a condition known as **contracture**. Fibrous tissue may also limit movement of the mouth or eyelids. Physiotherapy or surgery may be necessary to break down the fibrous tissue and improve mobility. Shrinkage of the scar tissue may also cause shortening or narrowing (**stenosis**) of structures, particularly tubes or ducts. For example, if the esophagus is shortened, malposition of the stomach (hiatal hernia) or a narrowed esophagus causing obstruction during swallowing (Fig. 5-8) can result.

Adhesions

Adhesions are bands of scar tissue joining two surfaces that are normally separated. Common examples are adhesions between loops of intestine (see Fig. 5-8*B*) or between the pleural membranes. Such adhesions usually result from inflammation or infection in the body cavities. Adhesions prevent normal movement of the structures and may eventually cause distortion or twisting of the tissue.

Hypertrophic Scar Tissue

An overgrowth of fibrous tissue consisting of excessive collagen deposits may develop, leading to hard ridges of scar tissue or keloid formation (Fig. 5-9). These masses are disfiguring and frequently cause more severe contractures. Skin and the underlying tissue may be pulled out of the normal position by the shortening of the scar tissue.

Ulceration

Blood supply may be impaired around the scar, resulting in further tissue breakdown and possible ulceration. This may occur when scar tissue develops in the stomach following surgery or healing of an ulcer. This scar tissue interferes with blood flow in nearby arteries.

THINK ABOUT 5-10

a. Describe three ways scar tissue on the thumb can interfere with normal function.
b. Explain how the characteristics of scar tissue can actually lead to new potential infections in the affected area.

Example of Inflammation and Healing

Burns

A burn is a thermal (heat) or nonthermal (electrical or chemical) injury to the body, causing acute inflammation and tissue destruction. Burns may be mild or cover only a small area of the body, or they may be severe and life threatening, as when an extensive area is involved. Burns may be caused by direct contact with a heat source such as flames or hot water (a scald), by chemicals, radiation, electricity, light, or friction. Most burns occur in the home. Any burn injury causes an acute inflammatory response and release of chemical mediators, resulting in a major fluid shift, edema, and decreased blood volume. Major burns constitute a medical emergency requiring specialized care as quickly as possible.

The severity of the burn depends on the cause of the burn, and the temperature, duration of the contact, as well as the extent of the burn surface and the site of the injury. Young children with their thin skin frequently receive severe burns from immersion in excessively hot water in a bathtub. The elderly also have thinner skin; therefore they can suffer much deeper burn injuries than younger adults. Skin thickness varies over the body, with facial skin being much thinner than the skin on the palms and soles. Thus, facial burns are more often more damaging than burns to the soles of the feet.

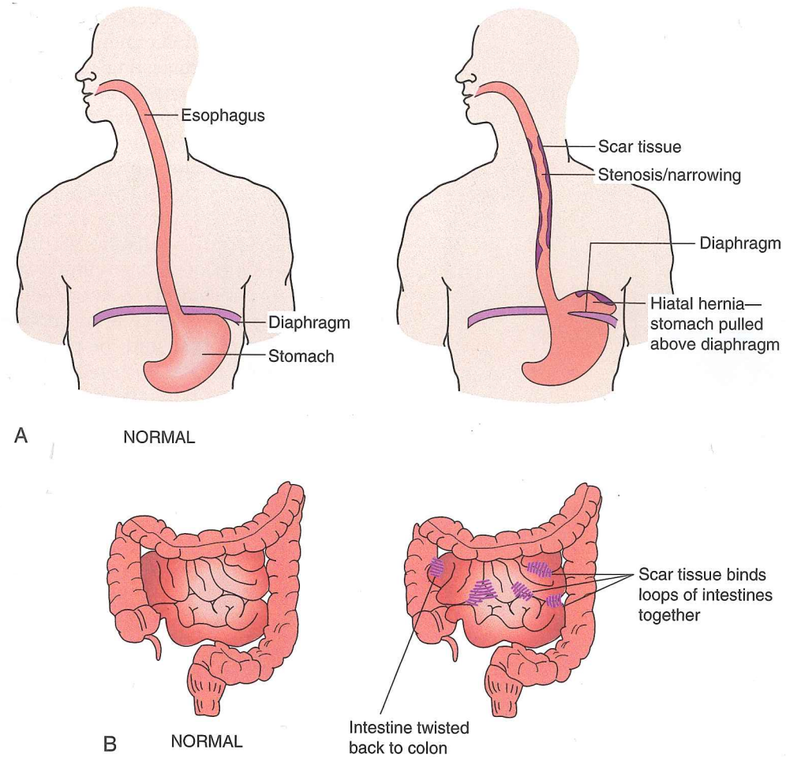


FIGURE 5-8 Effects of scar tissue. **A**, Esophageal scarring and obstruction. **B**, Adhesions and twisting of the intestines.

THINK ABOUT 5-11

From your own experience and the information just given, describe the appearance and sensation over time of a thermal burn (e.g., a burn resulting from touching a hot object).

Classifications of Burns

Burns are classified by the depth of skin damage and the percentage of body surface area involved. *Partial-thickness* burns involve the epidermis and part of the dermis (Fig. 5-10). Superficial partial-thickness burns (formerly known as first-degree burns) damage the epidermis and may involve the upper dermis. They usually appear red and painful but heal readily without scar tissue. Examples include sunburn or a mild scald.

Deep partial-thickness burns (formerly second-degree burns) involve the destruction of the epidermis and part of the dermis (Fig. 5-11). The area is red, edematous, blistered, and often hypersensitive and painful during the inflammatory stage. In severe cases, the skin appears waxy with a reddened margin. The dead skin gradually sloughs off, and healing occurs by regeneration from the edges of the blistered areas and from epithelium lining the hair follicles and glands. If the area is extensive, healing may be difficult, and complications occur. Grafts may be necessary to cover larger areas. These burns easily become infected, causing additional tissue destruction and scar tissue formation.

Full-thickness burns (formerly third- and fourth-degree burns) result in destruction of all skin layers and often underlying tissues as well (see Fig. 5-11*C*). The burn wound area is coagulated or charred and therefore is hard and dry on the surface. This damaged tissue (eschar) shrinks, causing pressure on the edematous tissue beneath it. If the entire circumference of a limb is involved, treatment (escharotomy—surgical cuts through this crust) may be necessary to release the pressure and allow better circulation to the area. This procedure may also be required when a large area of the chest is covered by eschar, impairing lung expansion. Initially the burn area may be painless because of destruction of the nerves, but it becomes very painful as adjacent tissue becomes inflamed due to chemical mediators released by the damaged tissues. Full-thickness burns require

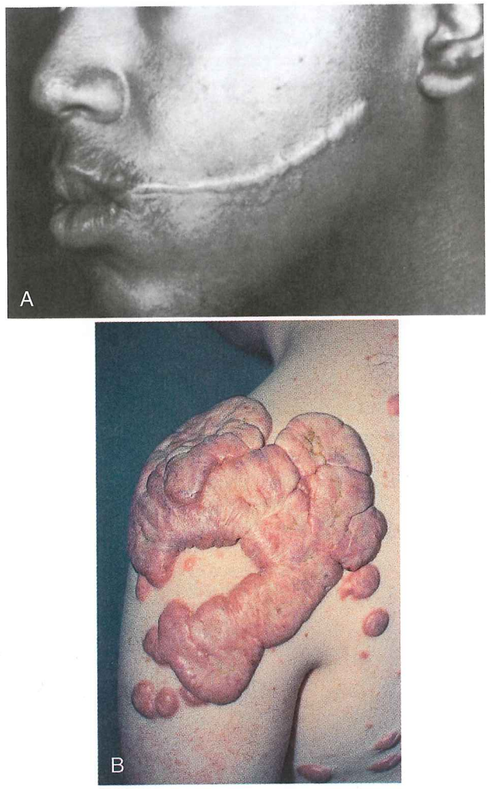
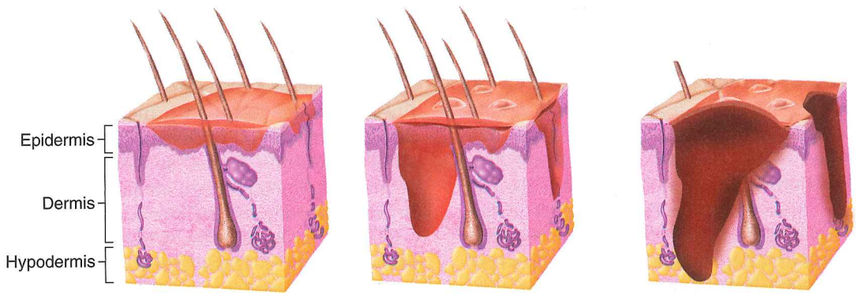


FIGURE 5-9 Complications of scar tissue. **A**, Example of scar tissue that may shrink and distort facial features in time. **B**, Example of a keloid. *(From Callen J, Greer K, Hood A, et al:* Color Atlas of Dermatology, *Philadelphia, 1993, WB Saunders.)*

skin grafts for healing because there are no cells available for the production of new skin. Many burn injuries are mixed burns, consisting of areas of partial burns mixed with full-thickness burns.

The percentage of *body surface area (BSA)* burned provides a guideline for fluid replacement needs as well as other therapeutic interventions. Complicated charts are provided in burn treatment centers for the accurate assessment of BSA. The "rule of nines" (Fig. 5-12) is a method for rapid calculation. In this estimate, body parts are assigned a value of nine or a multiple of nine. The head and each arm are estimated at 9%. Each leg is calculated at 18%. The anterior surface of the trunk is given a value of 18%, and the posterior surface is also 18%. The groin area at 1% brings the total BSA to 100%. The parts can be subdivided also; for example, the distal part of the arm (elbow to hand) accounts for 4.5% of the BSA. These figures are approximations and can be revised; for example, because a young child has a larger head and shorter limbs than an adult, an adjustment is required. The Lund and Browder chart provides a more detailed calculation for children.

Minor burns to a small area can be treated in a physician's office. Major burns, as classified by the American Burn Association, are best treated in a center specializing in burn wound care. Major burns include burns involving a large surface area, young children, or the elderly; burns to hands, feet, face, ears, or genitalia; inhalation injury; chemical burns; or cases in which other injuries or complications are present. Electrical injuries are always considered serious because there is immediate interference with the normal conduction of electrical impulses in the body, often causing cardiac arrest, and extensive unseen damage to blood vessels and organs. (An electric current travels on the path of



Partial-thickness burns	First-degree burn: Damaged epidermis and edema	Second-degree burn: Damaged epidermis and dermis	**Full-thickness burns**	Third-degree burn: Deep tissue damage

FIGURE 5-10 Classification of burns. Partial-thickness burns include first- and second-degree burns. Full-thickness burns include third-degree burns. Fourth-degree burns involve tissues under the skin, such as muscle or bone. *(From Patton KT, Thibodeau GA:* Anatomy & Physiology, *ed 8, St. Louis, 2013, Mosby.)*

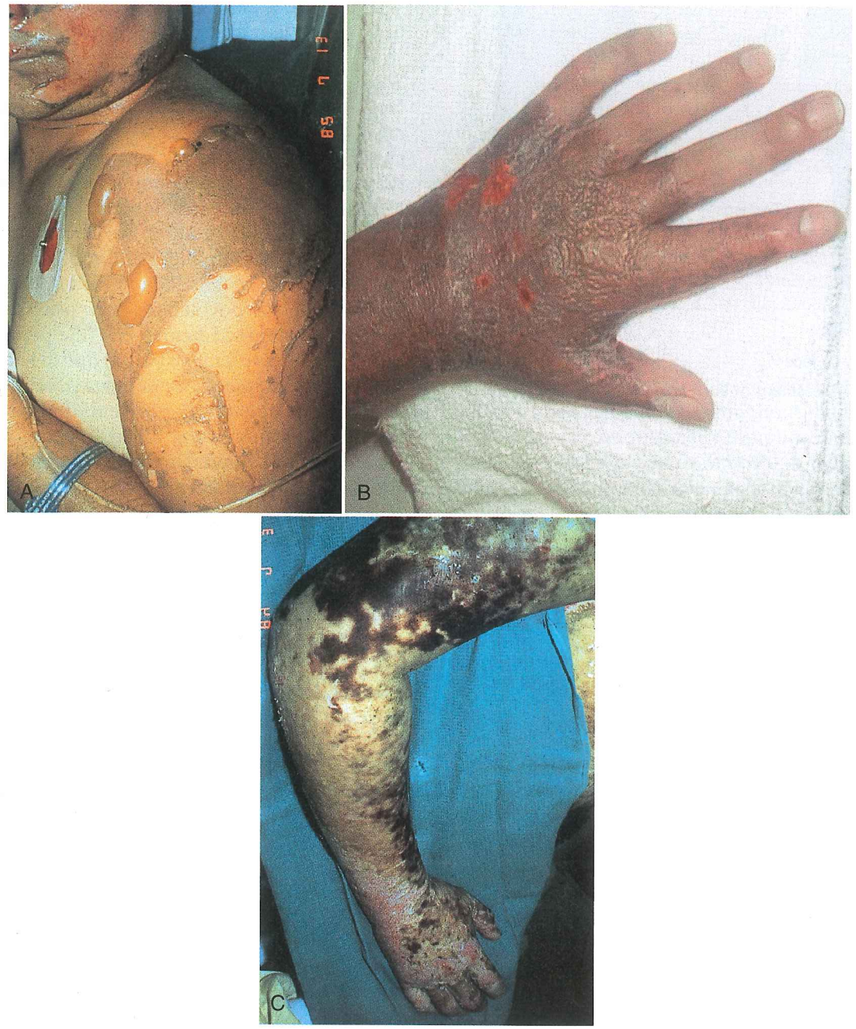


FIGURE 5-11 Examples of burns. **A**, Deep partial-thickness burn (note the blisters). **B**, Deep partial-thickness burn (note the edema). **C**, Full-thickness burn (note the dark color). *(All photos courtesy of Judy Knighton, Clinical Nurse Specialist, Ross Tilley Burn Center, Sunnybrook and Women's College Health Center, Toronto, Ontario, Canada.)*

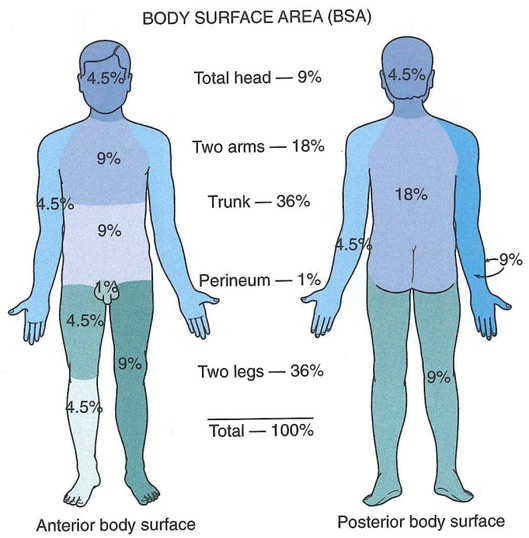


FIGURE 5-12 Assessment of burn area using the rule of nines.

least resistance, such as along the blood vessels, coagulating and obstructing blood supply.)

EMERGENCY CARE FOR BURNS

Stop, Drop, and Roll!

- When clothes are on fire, stop what you are doing, drop to the floor, cover up if possible, and roll to extinguish flames.
- Call emergency services (911) if the burn appears to be extensive or a major burn.
- Ensure that electrical power is off before caring for an electrical burn injury!
- Cool the burned area by soaking it with cool or tepid water. Remove nonsticking clothing if possible, and continue with cool water. Do not apply lotions, fats, or lubricants!
- Cover loosely with a clean cloth (e.g., the inside of a folded sheet) or sterile gauze.
- For a chemical burn, remove any affected clothing and flush the burn area well with cool water, then cover with a clean cloth.

THINK ABOUT 5-12

a. Using the rule of nines, calculate the approximate area of partial-thickness burn in an adult with burns to the right arm and chest area.
b. State two reasons why full-thickness burns are considered more serious than partial-thickness burns.
c. Why does sunburn usually heal readily?

Effects of Burn Injury

Serious burns have many effects, both local and systemic, in addition to the obvious damage to the skin. The burn wound is debrided during treatment, removing all foreign material and damaged tissue, in preparation for healing. A temporary covering is then applied.

Following is a brief description of additional effects, to be expanded upon in subsequent chapters.

Shock

No bleeding occurs with a burn injury (tissue and blood are coagulated or solidified by the heat). Under the burn surface an inflammatory response occurs. Where the burn area is large, the inflammatory response results in a massive shift of water, protein, and electrolytes into the tissues, causing fluid excess or edema (see Chapter 2) (Fig. 5-13). Loss of water and protein from the blood leads to decreased circulating blood volume, low blood pressure, and hypovolemic shock (see Chapter 12), as well as an increased **hematocrit** (the percentage of red blood cells in a volume of blood) due to hemoconcentration. The fluid imbalance is aggravated by the protein shift out of the capillaries and the resulting lower osmotic pressure in the blood, making it difficult to maintain blood volume until the inflammation subsides. Prolonged or recurrent shock may cause kidney failure or damage to other organs. Fluid and electrolytes as well as plasma expanders (a substitute for lost protein) are replaced intravenously using formulas designed to treat burn patients. In some cases of severe shock, particularly with extensive full-thickness burns, acute renal failure may develop (see Chapter 18).

THINK ABOUT 5-13

a. Explain how an increased hematocrit indicates a fluid shift.
b. How do reduced protein levels in the blood affect tissue metabolism and healing?
c. How does the reduction in blood flow through the burn promote infection and make an infection harder to treat should one develop?

Respiratory Problems

An immediate concern in the case of a burn patient is the inhalation of toxic or irritating fumes. Inspiration of carbon monoxide is dangerous because this gas preferentially binds to hemoglobin, taking the place of needed oxygen. The increasing presence of synthetic materials in the environment has increased the risk of exposure to toxic gases such as cyanide during a fire. These gases are particularly dangerous when an individual has been trapped in an enclosed space, such as a room or an automobile. High levels of oxygen are administered and the patient is observed for signs of respiratory impairment following such a burn.

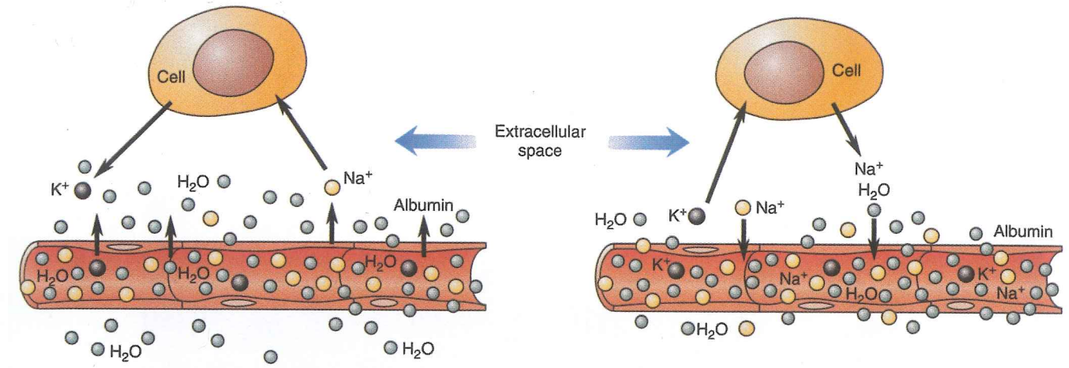


FIGURE 5-13 Direction of fluid and electrolyte shifts associated with burn shock. During burn shock, K^+ is moving out of the cell, and Na^+ and H_2O are moving in. After burn shock, K^+ moves in, and Na^+ and H_2O move out. *(From Copstead-Kirkorn LC:* Pathophysiology, *ed 4, St. Louis, 2009, Mosby.)*

If flame, hot air, steam, or irritating chemicals have been inhaled, damage to the mucosal lining of the trachea and bronchi may occur, and patients are observed for indications of inflammation and obstruction developing in the airway. Facial burns may be present, wheezing, and coughing up sputum containing black particles. Ventilation may be limited by eschar or pain. Pneumonia, a lung infection, is a threat, because of inflammation in the respiratory tract and immobility (see Chapter 13).

Pain

Burns are very painful injuries throughout the treatment process until healing is complete. The original injury, body movements, and application of grafts and other treatments contribute to pain. Analgesics (pain killers) are required.

Infection

Infection is a major concern in patients with burns. Infection of burn injuries increases tissue loss in the area, often converting a partial-thickness burn to a full-thickness burn. Because microbes are normally present deep in glands and hair follicles (see Chapter 8), there is a ready-made source of infection in the injured area. Also, opportunistic bacteria and fungi (see Chapter 6) are waiting to invade open areas, when defensive barriers and blood flow are reduced. Common microbes involved in burn injury infections include *Pseudomonas aeruginosa*, *Staphylococcus aureus* (including drug-resistant strains), *Klebsiella*, and *Candida* (Fig. 5-14). Antimicrobial drugs are usually administered only after specific microorganisms from the wound have been cultured and identified. Excessive or incorrect use of antimicrobial drugs increases the risk of emergence of drug-resistant microorganisms (see Chapter 6). When serious infection develops, there is risk of microorganisms or toxins spreading throughout the body, causing septic shock and other complications. Treatment involves rapid excision or removal of the damaged and infected tissue, application of antimicrobial drugs, and replacement with skin grafts or a substitute covering.

THINK ABOUT 5-14

a. Suggest three potential sources of infection in a burn patient.
b. Other than skin damage, explain what other dangerous effects can result from burns.

Metabolic Needs

Hypermetabolism occurs during the healing period, and increased dietary intake of protein and carbohydrates is required. There is considerable heat loss from the body until the skin is restored; the patient with burns tends to feel chilled and is sensitive to air movement. Therefore the ongoing need to produce more body heat and replace tissue demands increased nutrients. Also, protein continues to be lost in exudate from the burn site until healing is complete. The stress response contributes to an increased metabolic rate and demand for nutrients. Anemia or a low hemoglobin concentration in the blood develops because many erythrocytes are destroyed or damaged by the burn injury, and often bone marrow functioning is depressed by compounds released from damaged tissues, reducing **hematopoiesis** (the production of blood cells in bone marrow).

Healing of Burns

An immediate covering of a clean wound is needed to protect the burned area and prevent infection. Nonstick

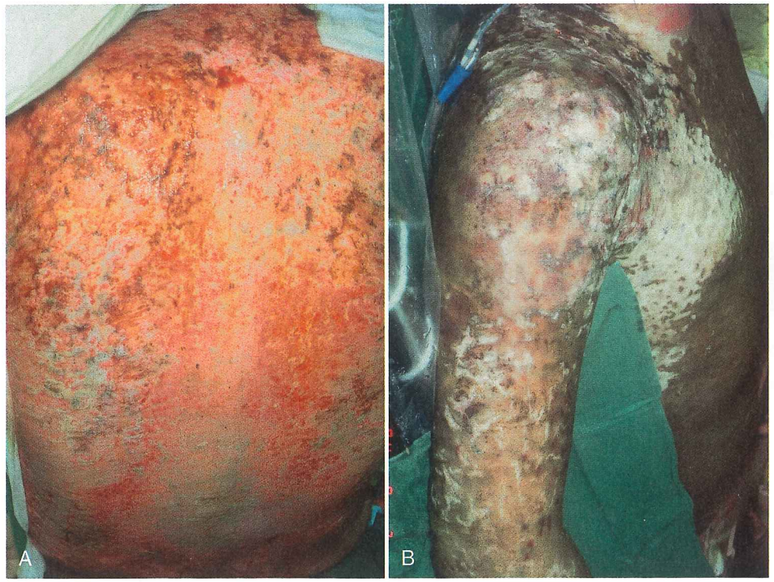


FIGURE 5-14 Infections in a burn wound. **A,** Purulent exudate (to be cultured to identify microbes). **B,** Blue-green color indicates infection by *Pseudomonas aeruginosa. (Courtesy of Judy Knighton, Clinical Nurse Specialist, Ross Tilley Burn Center, Sunnybrook and Women's College Health Center, Toronto, Ontario, Canada.)*

dressings are satisfactory for small areas or superficial burns. When a piece of skin is to be grafted over the burn wound, it may be "stretched" as a *mesh* to cover a greater area (Fig. 5-15A). In some cases, a small section of skin from the patient is cultured, producing a large piece of skin in several weeks. Alternative protection for the burn area may involve temporary substitute coverings, such as pig skin or cadaver skin, which will be rejected in time. In most serious burn cases, few epithelial cells are available in the burn area for healing.

Large burn centers are now using biosynthetic skin substitutes (e.g., Integra or TransCyte), particularly where severely burned patients lack sufficient healthy skin to graft (Fig. 5-15B). These coverings consist of two layers: A synthetic "epidermis" that can be removed easily as healing occurs, and underneath, a framework of proteins, collagen, and growth factors to stimulate healing. Some types contain living human cells. They are strong but elastic to conform to body structure. Research is proceeding to provide improvements to these materials. A very thin skin graft can then be applied with less scar tissue developing. Healing is more rapid, the number of surgical procedures and grafts are reduced, there is less risk of infection, and scarring is decreased when stable coverage of the burn wound can be quickly accomplished.

In a major burn, healing is a prolonged process, taking perhaps months. *Scar tissue* occurs even with skin grafting and impairs function as well as appearance. Hypertrophic scar tissue is common. Long-term use of elasticized garments and splints may be necessary to control scarring. In Figure 5-16, a burn survivor is being measured for an elastic pressure sleeve, a process that may be repeated many times.

Physiotherapy and occupational therapy are often necessary to reduce the effects of scar tissue and increase functional use of the area. In some cases, surgery may be necessary to release restrictive scar tissue or contractures. Severe burns require long-term team treatment because complications are frequent. The length of treatment has a major impact on a burn survivor, considering the psychological and practical effects on physical appearance and function, family, and job.

Children

The *growth of children* is often affected during the acute phase of burn recovery, when metabolic needs are compromised and stress is great. Often at a later time, additional surgery or grafts may be required to accommodate growth and ease the effects of scarring.

THINK ABOUT 5-15

a. Explain why healing is a particularly slow process in burn patients.
b. Explain what particular problems a child would encounter in any case where they have suffered an injury that has resulted in a considerable amount of scar tissue.

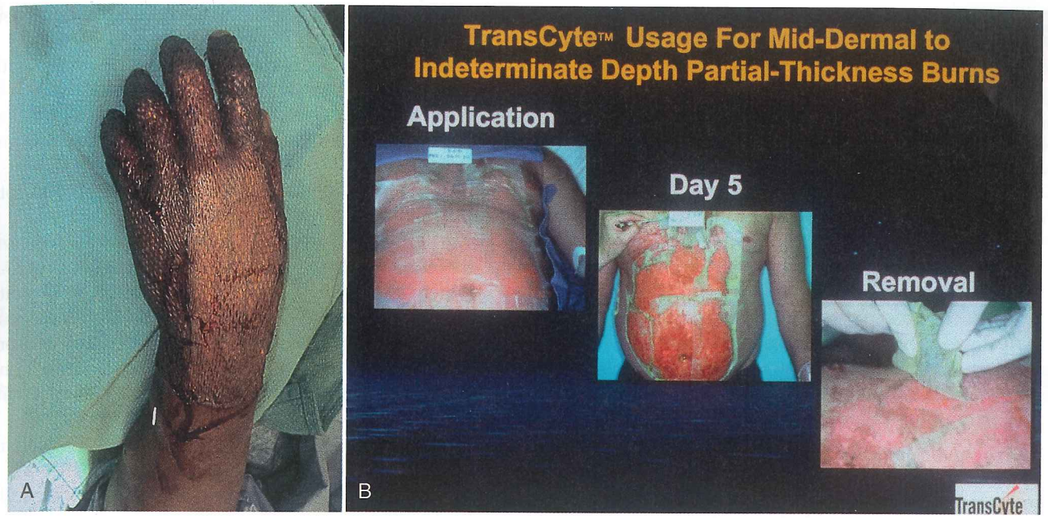


FIGURE 5-15 **A,** Example of a mesh skin graft. *(Courtesy of Judy Knighton, Clinical Nurse Specialist, Ross Tilley Burn Center, Sunnybrook and Women's College Health Center, Toronto, Ontario, Canada.)* **B,** Biosynthetic Covering (TransCyte). *Top:* A temporary dermal substitute "skin" is placed on a clean, partial-thickness burn wound. *Bottom:* The covering is removed after new epithelial tissue has formed. *(From Advanced Healing, Inc.)*

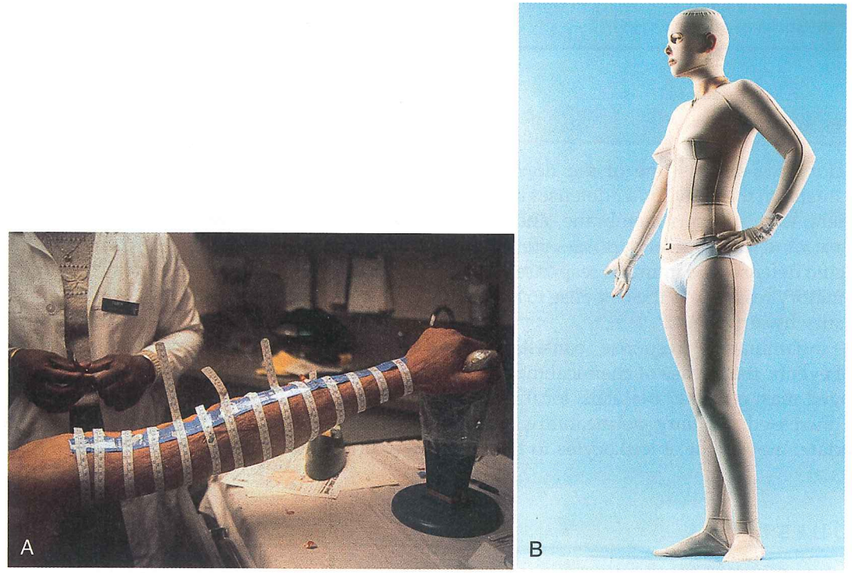


FIGURE 5-16 **A.** Measurement for an elastic garment to control scar tissue from a burn. *(Courtesy of Judy Knighton, Clinical Nurse Specialist, Ross Tilley Burn Center, Sunnybrook and Women's College Health Center, Toronto, Ontario, Canada.)* **B.** The custom-fitted antiscar support garment modeled here effectively provides pressure therapy over wounds, which helps to minimize the development of hypertrophic scarring. *(From Black JM, Matassarin-Jacobs E, editors:* Medical-Surgical Nursing: Clinical Management for Positive Outcomes, *ed 7, Philadelphia, 2005, Saunders, p 1356. Courtesy Medical Z, San Antonio, Texas.)*

CASE STUDY A

Trauma

M.H., age 6, fell while running down stairs and hurt his wrist and elbow. His arm was scraped and bleeding slightly, and the elbow became red, swollen, and painful. Normal movement was possible, although painful.

1. Explain why the elbow is red and swollen.
2. Suggest several reasons why movement is painful.
3. State two reasons why healing may be slow in the scraped area on the arm, and two factors that encourage healing in this boy.

CASE STUDY B

Burns

While trying to light a barbecue, the propane tank exploded, burning the face, arms, and chest of P.J., age 28. He had mixed burns to most areas except for his hands and face, which were full-thickness burns.

1. Why would this be considered a major burn?
2. Describe the process taking place in the burned area during the first hours after the injury.
3. P.J. was wheezing, coughing up mucus, and short of breath. Explain why this has likely developed.
4. P.J. developed a bacterial infection on his right hand. Explain three predisposing factors to this.
5. How will this burn injury affect P.J.'s ability to work? What are some of the social needs in this case?

CHAPTER SUMMARY

The inflammatory response is one of the nonspecific defense mechanisms in the body. Other defenses include: first, the barriers, skin, mucous membrane, and secretions such as tears and saliva; second, phagocytosis; and third, the specific defense, the immune response.

- The inflammatory response is the response to any cell or tissue injury by any agent.
- The acute inflammatory response consists of a sequence of events: the release of chemical mediators from damaged mast cells and platelets, local vasodilation and increased capillary permeability, formation of exudate, movement of leukocytes to the site, and phagocytosis for removal of the offending agent and debris.
- The signs of acute inflammation are redness, warmth, swelling, pain, and, frequently, loss of function.
- With extensive inflammation, systemic signs may present, including mild fever, headache, fatigue, and leukocytosis.
- Chronic inflammation results in formation of fibrotic or scar tissue.
- Anti-inflammatory drugs include aspirin (ASA) and the nonsteroidal anti-inflammatory drugs (NSAIDs), which block prostaglandin production at the site. These drugs also have antipyretic and analgesic activity. The glucocorticoids such as hydrocortisone are effective anti-inflammatory and antiallergenic agents, but significant adverse effects develop with long-term use.
- Healing may take place by regeneration, if cells are capable of mitosis and the damaged area is small.
- Fibrotic or scar tissue, consisting primarily of collagen fibers, replaces normal tissue when damage is extensive or cells are incapable of mitosis. Scar tissue lacks normal function and is nonelastic, tending to shrink over time, possibly causing contractures, deformity, or strictures at a later time.
- Factors promoting healing include youth, good circulation and nutrition, and lack of infection or other disease.
- Burns, an example of inflammation and healing, are classified by the percentage of body surface area damaged and the depth of the skin damage in the burn area. Partial-thickness burns involve the epidermis and part of the dermis. Full-thickness burns destroy all skin layers; thus, a skin graft is required for healing. In some cases, eschar restricts circulation or ventilation.
- Following severe burns, shock frequently occurs because of fluid and protein loss from the burn wound. Infection is a threat because the protective skin barrier has been lost. Inhalation of toxic or irritating fumes may cause respiratory impairment. Hypermetabolism and the increased demand for nutrients for healing require dietary supplements.
- Healing of burns is a prolonged process, and multiple skin grafts may be required. Biosynthetic wound coverings have promoted healing in many cases.

STUDY QUESTIONS

In answering these questions, the student is expected to use knowledge of normal anatomy and physiology.

Inflammation

1. a. Explain why a cast placed around a fractured leg in which extensive tissue damage has occurred might be too tight after 24 hours.
   b. Explain why such a cast might become loose in 3 weeks.
2. List specific reasons why the inflammatory response is considered a body defense mechanism.
3. a. Explain the rationale for each of the following with acute inflammation: (i) warmth, (ii) fever.
   b. State three systemic signs of inflammation.

4. Explain why leukocytosis, a differential count, and elevated ESR are useful data but are of limited value.
5. a. Explain how acute inflammation predisposes to development of infection.
   b. Classify each as inflammation or infection: (i) sunburn, (ii) skin rash under adhesive tape, (iii) common cold, (iv) red, swollen eye with purulent exudate.
6. How does the presence of thick, cloudy, yellowish fluid in the peritoneal cavity differ from the normal state?
7. If a large volume of fluid has shifted from the blood into the peritoneal cavity, how would this affect blood volume and hematocrit?
8. Explain how acute inflammation impairs movement of a joint.
9. Explain two mechanisms used to increase body temperature as a fever develops.
10. Why might a client be advised to avoid taking ASA a few days before extensive oral surgery (e.g., multiple tooth extractions)?
11. Explain why a young child taking prednisone (glucocorticoid) for chronic kidney inflammation is at high risk for infection and might need prophylactic antibiotics.

Healing

12. a. When part of the heart muscle dies, how does it heal?
    b. How would the new tissue affect the strength of the heart contraction?
13. Suggest several reasons why healing is slow in the elderly.
14. Explain how scar tissue could affect the function of the:
    a. small intestine
    b. brain
    c. cornea of the eye
    d. mouth
    e. lungs (try to find more than one point!)

Burns

15. a. Explain the rationale for pain and redness accompanying a burn.
    b. Explain three reasons why protein levels in the body are low after a major burn.
16. a. Explain why immediate neutralization or removal of a chemical spilled on the hand minimizes burn injury.
    b. Describe some of the factors that would promote rapid healing of this burn.
17. Describe three potential complications of a full-thickness burn covering 30% of the body, including the legs and back.
18. If the face receives a full-thickness burn, describe three ways function could be impaired after healing.

ADDITIONAL RESOURCES

Applegate EJ: *The Anatomy and Physiology Learning System*, ed 2, Philadelphia, 2006, WB Saunders.
Kumar V, Abbas AK, Fausto M: *Robbins and Cotran Pathologic Basis of Disease*, ed 7, Philadelphia, 2009, WB Saunders.
Mosby's Drug Consult 2006. St. Louis, 2006, Mosby.

Web Sites

http://www.nlm.nih.gov/medlineplus/burns.html *Medline Plus: Burns.*
http://www.aaem.org/jem/ *Journal of Emergency Medicine* (American Academy of Emergency Medicine).