

Table 3-7 Somatoform Disorders

Disorder	Presentation	Criteria
Somatization disorder (Briquet's syndrome)	Multiple focal symptom focus	Begins before age 30, extends over a period of years, characterized by a combination of pain, gastrointestinal, sexual, or pseudoneurologic symptoms.
Undifferentiated somatoform disorder	Diffuse symptom focus	Unexplained physical complaints that are below the threshold for the diagnosis of somatization disorder; complaints must be present for at least 6 months.
Hypochondriasis	Single symptom focus	Preoccupation with the fear of having, or the idea that one has, a serious disease based on the person's misinterpretation of body symptoms.
Somatoform pain disorder	Pain is the predominant focus	Pain is the prime complaint with psychologic factors having an important role in the onset, worsening, or maintenance of the disorder.
Body dysmorphic disorder	Body part distortion	Preoccupation with an imagined or exaggerated defect in physical appearance.
Conversion disorder	Motor/sensory symptoms	Involves unexplained symptoms affecting voluntary motor or sensory functions that mimic a general medical or neurologic condition; psychologic factors are associated with the presentation of the symptoms.

Modified from Woltersdorf MA: Hidden disorders: psychological barriers to treatment success, *Phys Ther* 3:58-66, 1995.

basis exists for the condition. The use of evidence-based guidelines is recommended in determining the appropriate extent of medical evaluation and the frequency with which medical tests are repeated.

Detecting exaggeration does not automatically indicate that the individual is malingering,¹¹⁶ and the differential diagnosis of malingering includes factitious disorder, the somatoform disorders, the dissociative disorders, and specific medical conditions without somatoform disorder.¹⁴⁴

Factitious disorders are differentiated from malingering by the goal that motivates the individual's behavior. The only apparent goal in factitious illness is to gain the sick role; the goal in malingering is to gain rewards, such as compensation, or to avoid the unwanted, such as military service or jail.²⁵⁷

TREATMENT. Medical management of psychophysiological disorders requires a collaborative alliance between the health care provider and the client making restoration of function (rather than elimination of symptoms) the goal of treatment. Accordingly, the treatment approach may vary depending on the clinical presentation and client profile. Medical management currently comprises palliative treatment (often including physical therapy), CBT, antidepressants, or other pharmacologic treatment. The individual may seek alternative therapies, which may benefit them, but empirical data on these interventions are not yet available.

PROGNOSIS. The course of psychophysiological disorders varies with each type of disorder. Within the somatoform disorders, somatization disorder and undifferentiated somatoform disorder are chronic and unpredictable and rarely remit with current, common treatment approaches. Additional research is needed to confirm the benefits and potential of more process-oriented treatment and right brain communication in medical care.²⁶⁶

Conversion is often of short duration, and hospitalized individuals have remission within 2 weeks in most cases. A good prognosis for conversion is associated with acute onset; presence of clearly identifiable stress at the time of onset; a short interval between onset and the initiation of treatment; above-average intelligence; and symptoms of paralysis, aphonia, and blindness. Poor prognostic indicators include symptoms of tremors and seizures.

Pain disorders resolve relatively quickly when associated with an acute episode, but a wide range of variability exists in the course of chronic pain (see the section Chronic Pain Disorders in this chapter). Hypochondriasis is usually chronic but can remit completely, especially in the absence of a personality disorder and secondary gain. Body dysmorphic disorder often has a continuous course once it is diagnosed. Symptoms are almost always present, although the intensity may vary over time.

The course of psychogenic pain disorder or factitious disorder may be limited to one or more brief episodes, but it is more often of a chronic nature with a lifelong pattern of hospitalization. In the case of malingering, painful symptoms and movement dysfunction resolve completely when the court case involved has been completed or the possibility of financial gain has been eliminated.

Borderline Personality Disorder

Overview and Incidence. BPD is a chronic psychiatric disorder, which can complicate medical care. BPD disorder affects more women than men and is estimated to affect from 1% to 14% of the adult population.²⁰⁷ Identifying this diagnosis is important to treatment planning to recognize somatic sequelae, as well as suicidal and other psychologic implications.

BPD is the focus here without an equally thorough discussion of narcissism or passive-aggressive personality disorders because BPD exhibits so many of the patho-

logic behaviors that impact medical care. If the provider can identify and deal with this diagnosis, the counselors can refine the diagnosis and treatment plan when the individual is referred. Identifying and managing a broad-scope, prevalent diagnosis, such as BPD, is the first and most important step for the therapist. Specialists can sort out the deeper specifics.

Etiology and Risk Factors. BPD is the result of type A and type B traumas, especially when occurring in childhood; 80% of physically and sexually abused victims demonstrate borderline personality symptoms. BPD usually manifests in adolescence or early adulthood and causes problems with most or all necessary skills for functional relationships.

Clinical Manifestations. People with BPD have stormy and unpredictable ways of relating to other people. This behavior covers up poor self-esteem and feelings of anger and of not deserving anything good (shame). BPD impacts the person's ways of thinking, feeling, and behaving, which causes many problems, socially and medically.

BPD has broad-scope characteristics; it can be mild-to-severe, short- or long-term, potentially resolving with extensive or minimal intervention. BPD can impact medical care in the following ways:

- Passive-aggressive behaviors (the clinician is great/awful)
- Playing one caregiver against another and manipulating to prevent accountability
- Poor compliance in self-care and appointment times
- As people reconnect with feelings, expect to see an increased urgency to regress in cognitive, emotional, and behavioral areas
- Misunderstanding of instructions, relationships, boundaries
- Prolonged treatment time required because of non-physical triggering and the development of extensive, subtle physical symptoms
- Self-persecution and egocentric perspective ("I caused it, it's my fault")
- Insurance challenges as a result of complexity of diagnoses/failed care

People who have this disorder may see the world in terms of extremes, black versus white, good versus bad, love versus hate, "your fault" versus "my fault." There is no middle ground. Other symptoms are listed in Box 3-11.

MEDICAL MANAGEMENT

DIAGNOSIS. There are no blood tests, physical examinations, or imaging studies that can diagnose BPD. The psychiatrist, other physicians, psychologists, or trained counselors/licensed social workers can make the diagnosis. They rely on history and clinical presentation and watch for red flags, such as unstable personal relationships, that switch back and forth between hate and love.²⁰⁷

TREATMENT. BPD is a difficult disorder to treat. Because symptoms create difficulties in relationship and may bring up unresolved hurts and issues in the caregiver, caregivers and affected individuals may feel frustration

Box 3-11

CLINICAL MANIFESTATIONS/DIAGNOSTIC CRITERIA FOR BORDERLINE PERSONALITY DISORDER

Clinical Manifestations

- Feeling hopeless, powerless, anxious, and depressed.
- Unable to have pleasure without feeling guilty about it.
- Poor self-awareness, disconnected from physical, emotional, and spiritual "self."
- Sexual dysfunction; uncertainty about which gender they are attracted to, or in some cases, even be confused about their own gender; they may have no sexual feelings at all, or may need to fill an inner sense of emptiness by engaging in sexual activity often.
- Difficulty understanding what others are feeling; they may often feel frightened that others do not like or respect them; they have difficulty trusting others.
- Feeling different, damaged, or flawed in some way.
- Struggling with very strong anger or rage at others.
- Difficulty with basic self-care, such as eating, bathing, and sleeping regularly, when under stress.
- Fearing the only hope of getting needs met is to manipulate others.
- Losing touch with reality; hearing voices at times or feeling paranoid; these symptoms usually are not permanent but come and go depending on stress levels.
- Frantic efforts to avoid being abandoned.
- An unstable sense of self.
- Acting without thinking, doing things on impulse that could be harmful to yourself like reckless driving, drug or alcohol abuse, and careless sexual behavior.
- Suicidal behavior.
- Intense ups and downs in mood.
- Feeling empty much of the time.
- Trouble controlling anger.
- Distrust of others, paranoia, and feeling separate from yourself.

Diagnostic Criteria

- Pervasive pattern of instability of interpersonal relationships, self-image, impulsivity, and affect, plus 5 or more of the following traits:
 - Frantic efforts to avoid real or imagined abandonment.
 - Extremes of idealization and devaluation in relationships.
 - Identity disturbance: unstable sense of self, self-image.
 - Recurrent suicidal behavior, gestures, threats, or self-mutilating behavior.
 - Affect/mood instability varying within hours to days, characterized by recurrent anxiety, irritability, and episodic dysphoria; chronic feelings of emptiness; feelings of and inappropriate expressions of anger.
 - Transient, stress-related paranoid ideation or severe dissociative symptoms ("magical thinking").
- Potentially damaging impulsivity (2 or more of the following):
 - Spending
 - Sex
 - Reckless driving
 - Binge eating
 - Substance abuse

Data from American Psychiatric Association: *Diagnostic and statistical manual of mental disorders-text revision (DSM-IV-TR)*, ed. 4, Washington, DC, 2000, The Association.

through the healing process. Clinicians can become even more overwhelmed by the diagnosis than the client and feed into a sense of helplessness in dealing with clients exhibiting BPD symptoms.

Healing is accomplished as the healthy clinician works with the individual diagnosed with BPD in the same way as with any other person suffering from brain injury. As the caregiver practices sensitive care, within individual client tolerances, and remains true to self through whatever the client presents, the individual with BPD benefits from consistent right and left hemisphere messages from the healthy provider.^{209,254,255}

Progressively, the client learns from the clinician's ability to appropriately respond to emotional changes while upholding healthy boundaries. The client learns necessary skills for functioning as a cohesive personality and to function in relationships, replacing destructive patterns with healthy ones, as regenerative areas of the brain experience increasing joy and strength of identity.^{254,255}

Dialectic behavior therapy is now being used to treat BPD. This type of therapy helps the individual learn to regulate emotions, tolerate distress, self-manage, and be more effective with other people. The following can help reduce symptoms such as impulsive behavior and unstable relationships²⁰⁷:

- Therapy that helps the person feel cared about and understood
- Learning healthy ways to cope when under stress (e.g., what to do besides self-mutilating)
- Day treatment programs, including structured activities and group therapy every day
- Short-term, structured time in the hospital for self-inflicting injuries, such as self-cutting, self-burning, or suicide attempts
- Medication, especially for symptoms of anxiety, panic, depression, or mood swings
- Treatment for any alcohol or drug abuse problems

SPECIAL IMPLICATIONS FOR THE THERAPIST 3-8

Psychophysiological Disorders

PREFERRED PRACTICE PATTERNS

Patterns will vary depending on clinical presentation of (real or feigned) signs and symptoms and the presence of impaired function.

The therapist's practice is often composed of individuals with somatoform disorders, personality disorders, malingering, or other psychophysiological disorders. The health care professional who can communicate a willingness to consider all aspects of illness, whether physiologic or psychologic, can foster a trusting relationship with the client that is foundational to healing. Such an attitude promotes client self-disclosure and a reliance on confidentiality. The presence of these diagnoses frequently requires behavioral and treatment modifications. As with all psychologically based illnesses, the therapist is encouraged to practice in cooperation with the other team members, espe-

cially when behavioral or psychologic approaches are the basis of medical treatment.

Somatoform Disorders

Somatoform disorders can account for 80% of all physician visits and make up a large portion of clients in a therapy setting. Personality disorders, although rare in the general population, are common in the medical arena and especially in the physical therapist's practice when medical treatment has been unsuccessful in improving a client's physical complaints. Somatoform clients are often described as whiners and can have a bewildering array of symptoms, all of which can be highly resistant to improvement through therapy or other medical or psychologic treatment.

The essential markers for the somatoform disorders are the absence or inadequacy of physical findings, insatiable complaints, excessive social and occupational consequences, preoccupation with problems, and lack of obvious secondary or material gain. This is not to say that adopting the sick role (although sometimes enjoyable) has no gain associated with it—just not material or monetary gain. However, symptoms of somatoform disorders are consistent with unresolved trauma and can coexist with a concurrent physical illness, emphasizing the need for ongoing evaluation.

These persons often seek treatment from several physicians concurrently, which may lead to complicated and sometimes hazardous combinations of treatment (polypharmacy). Frequent use of medications may lead to side effects and substance-related disorders. The therapist is encouraged to maintain close communication with the physician(s) if either of these situations is suspected or discovered.

Working with individuals who have somatoform disorders places the therapist at risk for personal and professional burnout. To preserve one's sanity and maintain a professional perspective, people with somatoform disorders must be identified. It may be helpful to affirm that somatic symptoms are not imaginary or feigned and describe the process of amplification, whereby sociocultural and psychologic stresses amplify symptoms and hinder recovery.

Working with individuals who have somatoform disorders places the therapist at risk for personal and professional burnout. To preserve one's sanity and health and maintain a professional perspective, the provider must (1) attend to his or her personal wounds and needs, (2) accept and treat highly involved clients in the same way as any other client, and (3) establish healthy boundaries in serving the most complex and needy individuals. It is also helpful to accept the person and his or her story as told, letting time and confirming evidence or lack thereof provide client feedback and treatment direction.

After the safe client-provider relationship is established, a discussion of the confirming or contradictory evidence, along with the client's needs and perspectives, can occur. Such a discussion can help give clients an explanatory model that focuses on processes and functioning rather than on structural or biomedical

abnormalities. At the same time, the therapist should discourage the client from assuming the sick role, minimize alarming expectations about the clinical course, and avoid making distressing symptom attributions.

Intervention includes the identification and alleviation of factors that amplify and perpetuate the person's symptoms and cause functional impairment. A discussion of current psychosocial stressors may be helpful, but physical therapists should not exceed their scope of practice.

Physical therapists trained in the therapeutic interventions that enable the person to move toward a state of physical and emotional well-being (e.g., somatoemotional release, myofascial release, craniosacral therapy, specific body work) may find more rapid clinical success with some individuals. Confronting him or her about the condition may only increase the problems (unconsciously) and exacerbate symptoms. It is better to stay supportive, conservative, and treat only what is objectively found and not what is subjectively reported.²⁶⁰

A brief summary of the somatoform disorders and possible clinical strategies are provided in Tables 3-7 and 3-8. The reader is referred to books and articles on these topics written specifically for the health care professional for more specific details.*

Factitious Disorder

In the case of a factitious disorder involving Munchausen syndrome, the MSBP parent fosters a close relationship with the medical team and pushes for findings not supported by the physical examination or laboratory tests. The perpetrator may even convince the therapy staff of the need for support in obtaining invasive diagnostic procedures.

The health care professional should be observant of the following red flags: (1) a parent with little formal education or training who has extensive knowledge of the child's medical condition, (2) a history of repeated hospitalizations or trips to the emergency department accompanied by an apparent lack of concern on the part of the parent, (3) inconsistent medical history, (4) clinical presentation does not fit the history and/or does not match any neuromuscular or musculoskeletal pattern of symptoms, and (5) the child's condition develops only when left alone with the parent in question.

Since these are also red flags for child abuse or ritual abuse, any of these findings requires a consultation with the physician and a competent counselor. It is not the physical therapist's role to confirm or prosecute abuse, but it is within the realm of the physical therapist's practice to recognize and track evidence of type A and type B trauma.

The child will rarely verbally report or confirm experiences of abuse due to fear, but careful observation of body language, facial expression, physical condition, and discerning palpation will allow the body to speak.

If time and red flags lead the clinician to suspect abuse, it is wise to confer with the attending physician and a knowledgeable counselor to report your findings and concerns, since local laws vary.

Malingering

The high base rate of malingering after events or incidents that lead to litigation (e.g., accidents, worker's compensation, disability claims, malpractice claims) requires assessment by the physical therapist.

A health care provider cannot rely on any single test to determine malingering, and symptoms of unresolved trauma can easily be confused with malingering tendencies, especially early on. A collection of information must be gleaned that documents consistent (or inconsistent) history. For example, the physical therapist may see that pain patterns and physical disability observed during treatment either change or disappear outside of the clinical setting as the client arrives or leaves the clinic or is seen outside of the professional relationship. Another example occurs when client abilities improve related to reward status, not personal change.

Test scores and measurements, including observations regarding effort, motivation, inconsistent behavior, and a host of other variables, also offer valuable feedback. Documentation of findings is important in all cases of suspected malingering.

The therapist does not make the determination or diagnosis of malingering but provides complete objective data for the client's record. This is important given the potential for a lawsuit against the therapist. Discussion of findings with the physician is essential because systemic disorders and unresolved trauma can masquerade as neuromusculoskeletal pathology and can present with apparent mismatching of disproportionate symptoms for the injury or pathology (e.g., symptoms last longer than the expected time for physiologic healing, symptoms are out of proportion for the type of injury).

If the physician has ruled out the possibility of an underlying systemic disorder accounting for the client's clinical manifestations, then the best approach is to discuss the therapist's concerns with the client over the lack of effort and/or inconsistent findings that have no apparent clinical meaning and consider referral to a competent counselor. The therapist should avoid confrontation or directly labeling the person as a malingerer but should remain focused on objective data and functional rehabilitation.

Personality Disorder

Personality disorders cannot be "fixed" but rather should be approached by the therapist with an eye to personal health, sensitive care, and accepting the client without bias or resistance. This can be accomplished through self-awareness and understanding of the disorder involved.

It is best to serve every individual as though he or she can be helped and attend to their physical and related needs. The healthy, sensitive, and insightful therapist who offers consistent professional help in his

*References 6, 16, 146, 258, 259, 261, and 266.

Table 3-8 Clinical Strategies for Somatoform Disorders

Disorder	Problem	Solution
Personality disorders	Rigid, inflexible, interpersonal style that has no correlation to the current personal relationship or needs of the setting of the moment	<i>Do:</i> Focus on the physical needs of the client, remind yourself that you are not there to satisfy all the needs of the client, remain professional, document objectively any troublesome exchanges. <i>Do not:</i> Try to be a friend, ever take any client's response personally, allow emotions to creep into your documentation.
Somatization disorder	Bewildering array of physical complaints that exceed findings	<i>Do:</i> Keep accurate records of all physical findings, assess regularly, mention his or her progress often, focus on what you can change (stiffness) and avoid what you cannot (nausea), praise his or her strengths. <i>Do not:</i> Tell the client that it is "in his or her head," even if you are right; do not confront the obvious contradictions. <i>Do:</i> Assess for physical findings only. <i>Do not:</i> Become more than a physical therapist for them; refer appropriately.
Undifferentiated somatoform disorder	Diffuse, ambiguous complaints	<i>Same as somatization disorder</i>
Hypochondriasis Somatoform pain disorder	Intense, single-symptom focus Pain, pain, pain	<i>Do:</i> Remember pain cannot be measured directly, focus on the indirect effects of pain, have a multidisciplinary approach, demand regular improvement, and set criteria early in treatment for what improvement looks like. <i>Do not:</i> Get angry with your clients; instead, gently confront their inconsistencies; document objectively and unemotionally. <i>Do:</i> Focus on a client's strengths, stay upbeat, downplay any undue attention to the actual area of disfigurement. <i>Do not:</i> Have the client talk about his or her feelings about the body part in question, tell him or her they are being unreasonable, say you know how they feel.
Body dysmorphic disorder	Excessive concerns about appearance	<i>Same as somatization disorder.</i>
Conversion disorder	Motor/sensory inconsistencies	

Modified from Woltersdorf AA: Hidden disorders: psychological barriers to treatment success, *PT Magazine* 3(12):58-66, 1995.

or her area of expertise will fare well. People with somatoform disorders and personality disorders cannot be helped unless they actually allow help and are equipped to receive the care they request. The therapist should be familiar with specific strategies for dealing with personality disorders.^{259,266}

more commonly associated with anxiety disorders. The associated mental disorders may precede the pain disorder (and possibly predispose the individual to it), co-occur with it, or result from it.⁶

The International Association for the Study of Pain has proposed a five-axis system for categorizing chronic pain (Box 3-12). This disease-based (etiological) classification approaches pain according to (1) anatomic region, (2) organ system, (3) temporal characteristics of pain and pattern of occurrence, (4) person's statement of intensity and time since onset of pain, and (5) etiologic factors. This five-axis system focuses primarily on the physical manifestations of pain but provides for comments on the psychologic factors on both the second axis where the involvement of a mental disorder can be coded and on the fifth axis where possible etiologic factors include psychophysiological and psychologic ones.

An alternate classification scheme has been proposed based on the possible mechanisms of pain and the accompanying physiologic characteristics (e.g., transient pain such as a pinprick, tissue injury pain, nervous system injury pain). This mechanism-based classification model

CHRONIC PAIN DISORDERS

Definition and Overview

Chronic pain has been recognized as pain that persists past the normal time of healing.²⁶ This pain may last more than 1 month, but more often is reported as being present for more than 6 months. The International Association for the Study of Pain has settled on 3 months as the dividing line between acute and chronic pain.¹⁶⁷

Chronic pain appears to be often associated with depressive disorders, whereas acute pain appears to be

Box 3-12**CLASSIFICATION OF CHRONIC PAIN****Axis I: Regions**

- Head, face, and mouth
- Cervical region
- Upper shoulder and upper limbs
- Thoracic region
- Abdominal region
- Low back: lumbar spine, sacrum, and coccyx
- Lower limbs
- Pelvic region
- Anal, perineal, and genital region
- More than three major sites

Axis II: Systems

- Nervous system (central, peripheral, and autonomic) and special senses; physical disturbance or dysfunction
- Nervous system (psychological and social)
- Respiratory and cardiovascular systems
- Musculoskeletal system and connective tissue
- Cutaneous and subcutaneous and associated glands (breast, apocrine, etc.)
- Gastrointestinal system
- Genitourinary system
- Other organs or viscera (e.g., thyroid, lymphatic, hematopoietic)
- More than one system

Axis III: Temporal Characteristics of Pain: Pattern of Occurrence

- Not recorded, not applicable, or not known
- Single episode, limited duration (e.g., ruptured aneurysm, sprained ankle)
- Continuous or nearly continuous, nonfluctuating (e.g., low back pain, some cases)
- Continuous or nearly continuous, fluctuating severity (e.g., ruptured intervertebral disk)
- Recurring irregularly (e.g., headache, mixed type)
- Recurring regularly (e.g., premenstrual pain)
- Paroxysmal (e.g., tic douloureux)
- Sustained with superimposed paroxysms
- Other combinations
- None of the above

Axis IV: Person's Statement of Intensity: Time Since Onset of Pain*

Not recorded, not applicable, or not known			
Mild	≤1 mo	1-6 mo	>6 mo
Medium	≤1 mo	1-6 mo	>6 mo
Severe	≤1 mo	1-6 mo	<6 mo

Axis V: Etiologic Factors

- Genetic or congenital disorders (e.g., congenital dislocation)
- Trauma, operation, burns
- Infective, parasitic
- Inflammatory (no known infective agent), immune reactions
- Neoplasm
- Toxic, metabolic (e.g., alcoholic neuropathy, anoxia, vascular, nutritional, endocrine), radiation
- Degenerative, mechanical†
- Dysfunctional (including psychophysiological)‡
- Unknown or other

- Psychologic origin (e.g., conversion hysteria, depressive hallucination)

Modified from Merskey H, ed: Classification of chronic pain: scheme for coding chronic pain diagnoses, *Pain* (suppl 3):S10-S11, 1986.

* Determine the time at which pain is recognized retrospectively as having started even though the pain may occur intermittently Grade

tFor example, a lumbar puncture headache would be mechanical.

‡For example, migraine headache, tension headache, or irritable bowel syndrome: syndromes where a pathophysiologic alteration is recognized are also included. Emotional causes may or may not be present.

is the result of the dramatic growth in the understanding of the molecular, cellular, and system's mechanisms responsible for nociception and pain.²⁶⁴

A mechanism-based classification could provide the basis for more reliable and valid tools for treatment and clinical investigation. Such an approach may lead to specific pharmacologic, surgical, or physical therapy interventions for each identified mechanism involved in a particular syndrome.

For example, groups of people with the same symptom but not necessarily the same disease (e.g., herpetic neuralgia versus diabetic neuropathy) could possibly be treated with a single drug effective across a variety of etiologies.²⁶⁵ This approach to the assessment and treatment of chronic pain has not been validated yet, and further testing is under way.

Incidence

Chronic pain disorders can occur at any age and are relatively common. For example, it is estimated that in any given year, 10% to 15% of adults in the United States have some form of work disability as a result of back pain alone and up to 80% of all adults will experience back pain. Females appear to experience certain chronic pain conditions such as headaches and musculoskeletal pain more often than do males.⁶

Etiologic Factors

Chronic pain disorders can be psychologically based (somatoform pain disorder); the result of a general medical condition; or a mixture of both. Among the most common general medical conditions associated with chronic pain are various musculoskeletal conditions (e.g., disk herniation, osteoporosis, osteoarthritis or rheumatoid arthritis, myofascial syndromes), neuropathies (e.g., diabetic neuropathies, postherpetic neuralgia), and malignancies (e.g., metastatic lesions in bone, tumor infiltration of nerves).⁶ An increasing number of studies now support the idea that most of back pain is triggered by biopsychosocial factors.

The most common chronic pain conditions encountered by the therapist are listed in Box 3-13. Chronic pain may be a form of self-defense as a result of domestic violence or abuse. (See the section on Somatoform Disorder in this chapter and Domestic Violence in Chapter 2.)

Chronic postoperative pain occurs in a small percentage of people after some procedures such as tumor resec-

Box 3-13**CHRONIC PAIN CONDITIONS**

- Arthritis
- Persistent neck/back pain
- Neuralgias
- Peripheral neuropathies
- Peripheral vascular disease
- Causalgia
- Chronic regional pain syndrome (CRPS), formerly reflex sympathetic dystrophy (RSD)
- Hyperesthesia
- Myofascial pain syndrome
- Fibromyalgia syndrome
- Phantom limb pain
- Cancer
- Postoperative pain
- Spinal stenosis

tion and subsequent regrowth or invasion of the chest wall, mastectomy with pain from interruption of the intercostobrachial nerve (branches from the brachial plexus to the thoracic region), surgical amputation followed by phantom limb pain, and chemotherapy when associated with neuropathies producing painful dysesthesias (abnormal sensations) of the feet and hands.

Physiologic, Psychologic, and Behavioral Response

Physiologic responses to chronic pain depend in part on the persistent (e.g., low back pain) or intermittent (e.g., migraine headache) nature of the pain. Intermittent pain produces a physiologic response similar to that of acute pain, whereas persistent pain allows for physiologic adaptation (e.g., normal heart rate, blood pressure, and respiratory rate) but can result in the detrimental sequelae of chronic cortisol dump.²⁰⁵ The traditional view conceptualizes pain as being directly associated with the extent of physical pathology, which has been found to be questionable (see earlier discussion in this chapter on Perceptions of Pain). However, since people report pain in the absence of physical pathology and individuals demonstrate objective physical pathology without symptoms, along with cases of low association between impairments and disability, it is suggested that factors other than physical pathology contribute to reports of pain.

Behavioral, cognitive, and affective factors have direct effects on the report of pain, adaptation to the pain, and response to treatment, as well as indirect effects by influencing sympathetic nervous system and neurochemical factors associated with nociception.²³⁹

Researchers have demonstrated that people with chronic pain may overemphasize their pain by reporting high levels of physical symptoms. They are less able to discriminate muscle tension levels compared with control subjects without pain. Biologic factors may initiate and maintain physical symptoms, whereas psychosocial factors influence pain perception.⁸⁶

At the same time, the presence of chronic pain can be associated with significant behavioral and psychologic changes. A constellation of life changes that produces

altered behavior in the individual and that persists even after the cause of the pain has been eradicated make up the chronic pain syndrome. Painful symptoms out of proportion to the injury or that are not consistent with the objective findings may be a red flag indicative of systemic disease or a psychogenic pain disorder. This can be differentiated from a chronic pain syndrome in that the syndrome is characterized by multiple complaints, excessive preoccupation with pain or physical symptoms, and often, excessive drug use.

The person exhibiting symptoms of a chronic pain syndrome may isolate himself or herself socially from other people and be fatigued, tense, fearful, and depressed. There are cases in which chronic pain occurs and a diagnosis is finally made (e.g., spinal stenosis or thyroiditis). In those cases, treatment is specific to the identified underlying cause and not simply a program of pain management.

Two polarizing stereotypes about how men and women cope differently with pain have been put forth over the years: (1) women are more likely to seek medical help and (2) men are more stoic about their pain and will avoid consulting a physician.

Researchers studying all types of chronic pain are currently collecting data on how men and women approach pain. Does it matter if the pain is the result of a chronic condition like fibromyalgia syndrome versus the pain associated with a life-threatening disease such as metastatic cancer?

Are men and women treated differently by the medical profession when their major symptom is pain? How do men and women respond to postsurgical pain? Thus far, numerous studies have found that the role of gender in chronic pain may be less important than psychologic and behavioral responses. In other words, gender does not appear to factor into how well people adapt to their chronic pain condition as much as coping mechanisms and strategies.²⁴⁰

People with chronic pain often are depressed, have sleep disturbances, and may become preoccupied with the pain. People with chronic pain often attempt to maintain their former lifestyle to appear as normal as possible, even denying pain and engaging in activities that exacerbate their painful symptoms. They may not report the full extent of their pain, for fear of being labeled a complainer, mentally ill, or a hypochondriac. The need to hide the pain may conflict with the need to have someone understand the pain. The result is emotional and psychologic conflicts.

Symptom Magnification Syndrome

Symptom magnification syndrome (SMS) is defined as a self-destructive, socially reinforced behavioral response pattern consisting of reports or displays of symptoms that function to control the life of the sufferer.^{155,157} At the present time, SMS is not listed in the DSM-IV-TR. It is likely that in the future, SMS will be categorized as a somatoform disorder, possibly a variant of somatoform pain disorder of a chronic nature.

Leonard N. Matheson first coined the term SMS in 1977 to describe people whose symptoms have reinforced their self-destructive behavior—that is, the symp-

toms have become the predominant force in the client's function, rather than the physiologic phenomenon of the injury determining outcome (unless physiologic changes occur leading to deconditioning or further disability). Conscious symptom magnification is referred to as malingering, whereas unconscious symptom magnification is labeled illness behavior or somatoform disorder.

SMS can fall into several categories and the reader is referred to Matheson¹⁵⁶ for an in-depth understanding of this syndrome. The following three signs indicate that a client may be exhibiting symptom magnification:

- Ineffective strategy for balancing symptoms against activities.
- Client acts as if the future cannot be controlled because of the presence of symptoms; limitation is blamed on symptoms: "My (back) pain won't let me ..."
- Client may exaggerate limitations beyond those that are reasonable in relation to the injury; client applies minimal effort on maximum performance tasks and overreacts to loading during objective examination.

Updated research on the identification of SMS has focused on screening for less than full effort performance during a functional capacity evaluation. Most methods of identification are not significant predictors of the syndrome and even the best methods frequently lead to misclassification.¹⁵⁸ Again, these symptoms can easily be associated with unresolved trauma and the related psychologic needs.

MEDICAL MANAGEMENT

Considering recent research discussing the impact of perception on pain and psychosocial impact on medical care, it may seem overwhelming to ferret out and attend to all of your client's physical and psychologic contributing factors. Remember that medical management and healing is a process wherein the client and provider(s) need to take one small step at a time—together. Addressing the client's basic developmental needs (through sensitive care habits) while treating the presenting physical complaints is a very effective and consistent way of caring for the whole person.

DIAGNOSIS. The clinical evaluation of pain currently involves identification or diagnosis of the primary disease/etiology factors considered responsible for producing/initiating the pain; placing the individual within a broad pain category, typically nociceptive, inflammatory, or neuropathic pain; and then identifying the anatomic distribution, quality, and intensity of pain. In conjunction with the proposed five-axis system for categorizing chronic pain (see Box 3-12), the physician may use the diagnostic criteria outlined in DSM-IV-TR (Box 3-14).

A newer approach to the problem of chronic pain is being evaluated in which the pain itself is considered the disease, and instead of emphasizing or categorizing the client on the basis of diagnosing the primary disease, an attempt is made to identify the mechanisms responsible for the pain.

Although identifying the disease is essential, especially when disease-modifying treatment is possible (e.g., acute herpes zoster, diabetes, tumor), the vast majority of

Box 3-14

DIAGNOSTIC CRITERIA FOR PAIN DISORDER

- A. Pain in one or more anatomic sites is the predominant focus of the clinical presentation and is of sufficient severity to warrant clinical attention.
- B. The pain causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- C. Psychologic factors are judged to have an important role in the onset, severity, exacerbation, or maintenance of the pain.
- D. The symptom or deficit is not intentionally produced or feigned (as in factitious disorder or malingering).
- E. The pain is not better accounted for by a mood, anxiety, or psychotic disorder and does not meet criteria for dyspareunia.

Modified from American Psychiatric Association: *Diagnostic and statistical manual of mental disorders-text revision (DSM-IV-TR)*, ed 4, Washington, DC, 2000, The Association.

people with persistent or chronic pain cannot be treated for the disease or pathology and/or the injury is not reversible (e.g., peripheral/segmental nerve lesions, brachial avulsion, spinal cord injury, poststroke central pain).¹⁶⁴

Valid criteria for assessing malingering and symptom embellishment do not exist, thereby requiring careful clinical judgment on the part of the physician and all other health care professionals.²³¹

PROGNOSIS. A wide range of variability exists in the course of chronic pain. In most cases, symptoms persist for many years, but function can improve when the individual follows a self-management program (usually prescribed by a therapist in conjunction with other health care professionals). Participation in regularly scheduled physical activity, exercise, and outside activities such as volunteer or paid work should be a part of the program whenever possible.

SPECIAL IMPLICATIONS FOR THE THERAPIST

3-9

Chronic Pain Disorders

PREFERRED PRACTICE PATTERNS

Musculoskeletal and Neuromuscular patterns may be present, depending on the underlying etiologic factors or pain mechanism present (e.g., myofascial syndrome, neuropathy) and the degree of impairment or disability.

It is counterproductive to speculate on whether the client's pain is real. Current data on the prevalence of malingering in cases of chronic pain are not consistent, and no reliable method for detecting malingering among those individuals diagnosed with chronic pain is available. Pain is real to the person, and acceptance of the pain as part of the clinical picture places the therapist in alliance with the client toward mutually

acceptable goals. Focusing on improving functional outcomes rather than on reducing the pain should be the underlying direction in therapy.

Chronic anxiety and depression may produce heightened irritability, overreaction to stimuli, and a heightened awareness of the symptoms. The person may become preoccupied with the details of anatomic function and how each movement or external event affects the symptoms. This self-focus must be redirected toward improving function and differs from the overdramatization of the discomfort sometimes helpful in alleviating the problem in certain cultures.

The cornerstone of a unified approach to chronic pain syndrome is a comprehensive behavioral program. Whenever possible, the therapist should reinforce the behavioral approaches used by the other members of the team. Some general guidelines are outlined in Box 3-15.

Pain may lead to inactivity and social isolation, which in turn can lead to additional psychologic problems (e.g., depression) and a reduction in physical endurance that results in fatigue and additional pain. People whose pain is associated with severe depression and those whose pain is related to a terminal illness, most notably cancer, appear to be at increased risk for suicide (see the section on Suicide in this chapter).

Psychosocial Factors and Chronic Pain

Many studies have now shown a link between psychosocial distress and chronic neck or back pain.^{107,151,233} Factors associated with chronic LBP may include job dissatisfaction, depression, and compensation issues.^{27,129} It may be necessary to conduct a social history to assess the client's recent life stressors and history of depression, drug, or alcohol abuse.

The presence of psychosocial risk factors does not mean the pain is any less real nor does it reduce the need for symptom control. The therapist concentrates on pain management issues and improving function. Tools to screen for emotional overlay and fear-avoidance behavior (FAB) are available.⁹¹

Fear-Avoidance Behavior

FABs can also be a part of disability from chronic pain. The Fear-Avoidance Model of Exaggerated Pain Perception (FAMEPP) was first introduced in the early 1980s.^{142,218} The concept is based on studies that show a person's fear of pain (not physical impairments) is the most important factor in how he or she responds to LBP.

Fear of pain commonly leads to avoiding physical or social activities. Screening for FAB can be done using the Fear-Avoidance Beliefs Questionnaire (FABQ).²⁴⁸ Elevated fear-avoidance beliefs are indicative of someone who has a poor prognosis for rehabilitation. They indicate psychosocial involvement, provide insight into the prognosis, and indicate the need to modify intervention with consideration for referral to a psychologist or behavioral counselor.

When the client shows signs of fear-avoidance beliefs, then the therapist's management approach should include education that addresses the client's

fear and avoidance behavior, while considering a graded approach to therapeutic exercise.⁸¹ The therapist can teach clients about the difference between pain and tissue injury. Chronic ongoing pain does not mean continued tissue injury is taking place. This common misconception can result in movement avoidance behaviors.

There are no known "cut-off" scores for referral to a specialist.⁸¹ Some researchers categorize FABQ scores into "high" and "low" based on the *physical activity scale* (score range 0-24). Less than 15 is a "low" score (low risk for elevated fear-avoidance beliefs) and more than 15 is "high." Higher numbers indicate increased levels of fear-avoidance beliefs. The distinction between these two categories is minor and arbitrary. It may be best to consider the scores as a continuum rather than dividing them into low or high.⁸¹ A cut-off score for the work scale indicative of having a decreased chance of returning to work has been proposed.

The work subscale of the FABQ is the strongest predictor of work status. There is a greater likelihood of return-to-work for scores less than 30 and less likelihood of return-to-work or increased risk of prolonged work restrictions for scores greater than 34.⁷⁵

Examination of fear-avoidance beliefs may serve as a useful screening tool for identifying clients who are at risk for prolonged work restrictions. Caution is advised when interpreting and applying the results of the FABQ work subscale to individual clients. This screening tool may be a better predictor of low risk for prolonged work restrictions. The work subscale may be less effective in identifying clients at high risk for prolonged work restrictions.⁷⁵

Symptom Magnification

Health care providers should recognize that we often contribute to SMS by focusing on the relief of symptoms, especially pain, as the goal of therapy. Reducing pain is an acceptable goal for some clients, but for those who experience pain after their injuries have healed, the focus should be restoration, or at least, improvement of function.

Instead of asking if the client's symptoms are better, the same, or worse, it may be more appropriate to inquire as to functional outcomes (e.g., what can the client accomplish at home that she or he was unable to attempt at the beginning of treatment, last week, or even yesterday?) Materials and courses on symptom magnification and functional capacity evaluations are available.

MOOD DISORDERS

Overview

Major mood disorders are generally divided into unipolar depression and bipolar (manic-depressive) disorders. Mood disorders are often but not always episodic. Symptoms may come and go in cycles, with a normal state in between. Schizophrenia, a form of mood disorder, is

Box 3-15**BEHAVIORAL GOALS AND GUIDELINES FOR CHRONIC PAIN SYNDROME**

- Identify and eliminate pain reinforcers.
- Decrease drug use.
- Use positive reinforcers that shift the focus from pain.
- Concentrate on abilities, not disabilities.
- Avoid the concept of cure: concentrate on control of pain and improved function.
- Avoid discussion of pain except as arranged by the team (e.g., only during monthly reevaluation, only with a designated team member).
- Use a home program to focus on function and functional outcome (e.g., self-help tasks within capabilities).
- The client should keep a log of accomplishments so that progress can be measured and remembered.
- Measure success by what the individual client can accomplish, not based on others' success.
- Take one day at a time. Direct energy toward solving today's problems rather than focusing on the future.
- Avoid negative reinforcers such as sympathy and attention to symptoms, especially pain.
- Encourage tolerance to increasing activity levels.
- Gradual progress is better than quick results with increased symptoms.
- Teach the client how and when to ask for and accept help when necessary. Do not offer help or yield to the demands of someone who does not need help.

sometimes characterized by periods when the symptoms are less severe, but people with schizophrenia rarely recover completely. Symptoms include apathy, emotional unresponsiveness, social withdrawal, limited or odd patterns of speech, and confused thinking with periodic outbreaks of psychotic symptoms such as hallucinations and delusions.

Many of the earlier models of mental disorders, their causes, and their neural substrates have been disproved. The concept that abnormal levels of one or more neurotransmitters could explain the pathogenesis of depression or schizophrenia appears to be a model that is too simple. The notion that a single gene can cause mental disorders or behavioral variations has been replaced by a complex genetic picture in which multiple genes act in concert with nongenetic factors to produce a risk of mental disorder. Current investigations are seeking a model that can explain the complex patterns of disease transmission within families and explain the expression of risk genes during brain development and of their function.¹¹¹

Psychoneuroimmunology (PNI) studies are attempting to determine and define the links among neural activity, the endocrine system, and altered immune responses in people with depressive disorders. Although the literature indicates some type of relationship exists between these systems, the exact mechanisms remain unknown.^{115,128}

Depression

Definition and Overview. Depression, also referred to as depression illness, is a morbid sadness, dejection, or a

sense of melancholy, distinguished from grief, which is a normal response to a personal loss.⁶ Mild, sporadic depression is a relatively common phenomenon experienced by almost everyone at some time, referred to as the common cold of emotions.

Depression is the most commonly seen mood disorder within a therapy practice, often associated with other physical illnesses (Box 3-16) and psychologic conditions. Depression is a normal response to (not a cause of) pain and may decrease the client's ability to cope with the pain. Although anxiety is more apparent in acute pain episodes, depression occurs more often in clients with chronic pain. When the pain is relieved, the depression usually abates.

Mood disorders can be classified into three broad types: major depressive disorder, organic mood disorder, and bipolar illness (manic depression). The most common type of depression observed is major depressive disorder encompassing several conditions, including depression, dysthymia, and SAD.

Major depressive disorder can occur as a single isolated episode lasting weeks to months or intermittently throughout a person's life. This type of depression may be seen as an adjustment disorder with depressive mood and occurs as a result of external circumstances (e.g., environmental stress, loss, or trauma). Profound depression may be an illness itself, considered as an affective or anxiety disorder, or it may be symptomatic of another psychiatric disorder such as schizophrenia.

Organic mood disorder is also biologically based; structural changes in the brain associated with disease (e.g., multiple sclerosis) or brain trauma (e.g., left-sided cerebrovascular accident, TBI) can cause depressive reactions, either on a short-term or recurring basis. Anticonvulsant medications, such as valproic acid (Depakote) and carbamazepine (Tegretol), are often effective, especially in the TBI population.

Bipolar disorder or *manic depressive disorder* is one of the most complex disorders in psychiatry, with multiple phases and varied presentations in each phase, making diagnosis and treatment difficult and challenging. Several epidemiologic studies have indicated that it is a much more prevalent disorder than has been previously thought.^{45,104}

Bipolar disorder is characterized by cyclical mood swings that often include intense outbursts of high energy and activity, elevated mood, a decreased need for sleep, and a flight of ideas (mania) followed by extreme depression (Box 3-17); each may last from days to months, switching back and forth quickly or with normal periods in between.

Bipolar disorder manifests itself as a manic episode with or without depression. Bipolar I is marked by severe manic episodes, grandiose thoughts, irritability, and decreased need for sleep, with depression often preceding or following the episode. Bipolar II is characterized by distinct periods of depression and a less severe form of mania called *hypomania*. The affected individual cycles through brief periods of heightened mood and irritability lasting several days. Frequent alterations of mood extremes (rapid cycling) called *cyclothymic disorder* affect some individuals. The cycles may only last a single day.

Box 3-16**PHYSICAL CONDITIONS COMMONLY ASSOCIATED WITH DEPRESSION*****Cardiovascular***

- Atherosclerosis
- Hypertension
- Myocardial infarction
- Angioplasty or bypass surgery

Central Nervous System

- Parkinson's disease
- Huntington's disease
- Cerebral arteriosclerosis
- Stroke
- Alzheimer's disease
- Temporal lobe epilepsy
- Postconcussion injury
- Multiple sclerosis
- Miscellaneous focal lesions

Endocrine, Metabolic

- Hyperthyroidism
- Hypothyroidism
- Addison's disease
- Cushing's disease
- Hypoglycemia
- Hyperglycemia
- Hyperparathyroidism
- Hyponatremia
- Diabetes mellitus
- Pregnancy (postpartum)

Viral

- Acquired immunodeficiency syndrome (AIDS)
- Hepatitis
- Pneumonia
- Influenza

Nutritional

- Folic acid deficiency
- Vitamin B6 deficiency
- Vitamin B12 deficiency

Immune

- Fibromyalgia
- Chronic fatigue syndrome
- Systemic lupus erythematosus
- Sjögren's syndrome
- Rheumatoid arthritis
- Immunosuppression (e.g., corticosteroid treatment)

Cancer

- Pancreatic
- Bronchogenic
- Renal
- Ovarian

Miscellaneous

- Pancreatitis
- Sarcoidosis
- Syphilis
- Porphyria
- Corticosteroid treatment

Box 3-17**CLINICAL MANIFESTATIONS OF BIPOLAR DISORDER*****Mania***

- Excessive high or euphoric feelings
- Sustained period of behavior different from usual
- Increased energy, activity, restlessness, racing thoughts, and rapid talking
- Decreased need for sleep
- Unrealistic beliefs in one's abilities and powers
- Extreme irritability and distractibility
- Uncharacteristically poor judgment
- Increased sexual drive
- Abuse of drugs, particularly cocaine, alcohol, and sleeping medications
- Aggressive, provocative, or intrusive behavior
- Denial that anything is wrong

Depression

- Persistent sad, anxious, or empty mood
- Feelings of hopelessness or pessimism
- Feelings of guilt, worthlessness, or helplessness
- Loss of interest or pleasure in ordinary activities, including sex
- Decreased energy, feeling fatigued, or being slowed down
- Difficulty concentrating, remembering, making decisions
- Restlessness or irritability
- Sleep disturbances
- Loss of appetite and weight, or weight gain
- Chronic pain or other persistent bodily symptoms that are not caused by physical disease
- Thoughts of death or suicide; suicide attempts
- Compulsive behavior
- Reckless behavior
- Violent behavior, anger
- Argumentative, oppositional
- Critical toward family members (fault finding)
- Preoccupation with self
- Unaffectionate with partner or spouse, withdrawn

Modified from National Institute of Mental Health (NIMH): *Bipolar disorder: manic-depressive illness*, Bethesda, MD, 2007, NIMH (www.nimh.nih.gov).

Heightened creativity and creative talent are sometimes associated with all phases of bipolar disorder.

Many studies suggest that bipolar disorder is frequently accompanied by alcoholism and/or other drug abuse. The comorbidity of alcohol abuse with bipolar mood disorder often delays early diagnosis of bipolar disorder, especially when the alcohol abuse has been the primary focus of intervention.⁴⁶ Other comorbidities may also exist (e.g., migraine headaches, asthma, anxiety and panic attacks, allergies, eating disorders).³⁵

SAD is a mood disorder with a consistent pattern of depressive symptoms that occurs with colder weather and shorter hours of daylight. It is characterized by feelings of fatigue, inability to maintain a regular lifestyle schedule, and lack of interest in social interactions or activities of enjoyment. Women are affected three times more often than men.

Some people report irritability, overeating and weight gain, and feelings of sluggishness, apathy, or "the blahs." It is more difficult to get up in the morning, and the level

of activity goes down. The symptoms go away in the spring and summer months and recur in the fall to winter months. With shorter days and less exposure to sunlight the body produces more melatonin, a hormone secreted by the pineal gland that is made almost exclusively at night to help us sleep and may help to synchronize other circadian rhythms.²⁵¹ It is possible that some people with SAD do not produce more melatonin but are hypersensitive to the hormone.

SAD is most prevalent in geographic areas north of 40-degrees latitude. Although Native Alaskans are affected, they are less likely to have SAD than people who move there.

Incidence and Prevalence. Major depressive disorder is the most common adult psychiatric disorder, beginning at any age with an average onset in the mid-20s, although it appears to be occurring at earlier ages for those born in the last decade. In the United States, an estimated 15 million people are diagnosed with mild-to-severe depression. Epidemiologic data from diverse cultures indicate the lifetime prevalence of major depression is twice as high in women as in men.^{53a} Rates in men and women are highest in the 25- to 44-year-old age group, although rates increase again in older adults. Bipolar disorder can occur at any age, but fully half of all cases begin before age 20, affecting men and women equally.

Etiologic and Risk Factors. Predisposing factors for the development of depression may be genetic, familial, biologic, or psychosocial (e.g., childhood history of sexual abuse, battery and rape during adulthood, other recent stressful life events, socioeconomic status, raising children without support). Given the fact that bipolar disorder occurs in families, research is focused on identifying a genetic basis. Linkage studies have implicated chromosome 18 or 21, but this has not yet been proved.^{178,198}

Depression may occur in association with past abuse or trauma, medical or chronic illness, or surgical procedures. Medical illness is the most consistently identified factor associated with the presence of late-life depression (see Box 3-16). A newly described condition called *vascular depression* accounts for 30% to 40% of all depression in people over the age of 65 and in people with a history of diabetes, hypertension, atherosclerosis, and angioplasty or bypass surgery. It appears to be a biologic alteration rather than a chemical one with black holes (lacunes) observed in the basal ganglia representing cerebral ischemia or silent strokes.¹³⁸ Major depression is considered a risk factor for cardiac morbidity and mortality; treating depression to reduce cardiac disease is under investigation.³⁸

Depression may occur as a result of medications, especially sedatives, hypnotics, cardiac drugs, antihypertensives, and steroids (Box 3-18); alcohol or drug abuse, especially cocaine dependence; or exposure to heavy metals or toxins (e.g., gasoline, paint, organophosphate insecticides, nerve gas, carbon monoxide, carbon dioxide); this type of depression may be labeled substance-induced mood disorder.¹⁰ Other causes of depression among the general population include prenatal and postpartum depression occurring in approximately 20% of all pregnancies and SAD, a dysfunction of circadian rhythms that

Box 3-18

DRUGS COMMONLY ASSOCIATED WITH DEPRESSION

Psychoactive Agents

- Amphetamines
- Cocaine
- Benzodiazepines
- Barbiturates
- Neuroleptics

Antihypertensive Drugs

- β-Blockers, especially propranolol (Inderal)
- α₂-Adrenergic antagonists
- Methyldopa (Aldomet)
- Hydralazine (Apresoline)

Analgesics

- Salicylates
- Propoxyphene (Darvon, Darvocet-N)
- Pentazocine (Talwin)
- Morphine
- Meperidine (Demerol)

Cardiovascular Drugs

- Digoxin (Lanoxin)
- Procainamide (Pronestyl)
- Disopyramide (Norpace)

Anticonvulsants

- Phenytoin (Dilantin)
- Phenobarbital

Hormonal Agents

- Corticosteroids
- Oral contraceptives
- Anabolic steroids

Miscellaneous

- Alcohol, illicit drugs
- Histamine H₂ receptor antagonists, especially cimetidine (Tagamet)
- Metoclopramide (Reglan)
- Levodopa (Dopar, Larodopa)
- Nonsteroidal antiinflammatory drugs (NSAIDs)
- Antineoplastic agents
- Disulfiram (Antabuse)
- Cytokines (interferons)

occurs more commonly in the winter as a result of decreased exposure to sunlight.

Pathogenesis. Researchers have examined several theories of pathogenesis based on etiologic factors. These include biochemical mechanisms, neuroendocrine mechanisms, sleep abnormalities, genetics, and psychosocial factors. Recent study of the biochemical basis of depression has centered on two primary neurotransmitters: norepinephrine and serotonin. Depression is associated with levels of norepinephrine, dopamine, and serotonin that are either produced in inadequate amounts or the receptor sites are not functioning properly; mania results from excessive levels of norepinephrine and dopamine.

As demonstrated in animal models, antidepressant drugs decrease the sensitivity of postsynaptic receptors by blocking the reuptake of the neurotransmitters into nerve endings. This change occurs 1 to 3 weeks after treatment, correlating with the delay seen clinically in the effectiveness of antidepressants. Neuroendocrine abnormalities, such as in the limbic hypothalamic-pituitary-adrenal (HPA) axis, have been implicated in the cause of depression, prompting a search for a reliable serum abnormality that could be used as a depression test. This abnormality is common in survivors of repeated abuse and the sequelae related to chronic sympathetic system stimulation.

Some examples of possible abnormalities include oversecretion of Cortisol, suppressed nocturnal secretion of melatonin, and decreased prolactin production in response to tryptophan administration. A known association exists between hormonal variations, such as low testosterone levels in men and basal levels of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) in women, and some depressions.

Sleep abnormalities are consistently associated with depression, including decreased REM latency (the time between falling asleep and the first REM period), longer first REM period, less continuous sleep, and early morning awakenings. Animal studies have shown that many antidepressants can reset the internal clock. Whether these sleep abnormalities represent causes or effects of depression remains unknown.

Genetic-based pathogenesis is suspected for bipolar depression based on a clear familial pattern and chromosomal linkage studies. Evidence exists that the key gene involved in the transmission of bipolar disease is X-linked. A familial pattern in the development of major depressive disorder is also evident, since this type of depression occurs up to three times more often in first-degree biologic relatives of people with this disorder.

Psychosocial factors, such as life events and perceived stress, are clearly associated with depression, but it is difficult to establish whether they cause depression or merely determine when a susceptible person will experience depression. Episodes of major depressive disorder often follow a severe psychosocial stressor such as the death of a loved one or divorce.

Psychosocial events as stressors may play a more significant role in the precipitation of the first or second episodes of major depressive disorder but less of a role in the onset of subsequent episodes. People hospitalized for any reason are particularly susceptible to feelings of depression and a sense of loss and despair.

Clinical Manifestations. Depressed mood and loss of interest in usually pleasurable activities are the hallmarks of depression (see Box 3-17). More than 95% of depressed people report decreased energy, even for minor daily tasks. The inability to accomplish new or challenging activities often results in occupational or school dysfunction; 90% report having problems with concentration and memory. Difficulty concentrating and marked forgetfulness is particularly common in depressed older adults and is called *pseudodementia*.

People with mood disorders, particularly depressive disorders, may present with somatic complaints, most commonly headache, gastrointestinal disturbances, or

Box 3-19

SOMATIC SYMPTOMS ASSOCIATED WITH MOOD DISORDERS*

- Weakness
- Headaches
- Joint pain (arthralgia)
- Muscle pain (myalgia)
- Excess perspiration
- Dizziness
- Dry mouth or excessive salivation
- Rapid breathing
- Blurred vision
- Constipation
- Tinnitus
- Dry skin
- Sexual dysfunction
- Flushing
- Slurred speech
- Chest pain
- Amenorrhea, polymenorrhea
- Difficulty with urination
- Sleep disturbance
- Confusion
- Back pain
- Fatigue
- Digestive problems

* Refers to nonmedicated persons.

unexplained pain (Box 3-19) (see earlier discussion in the section on Somatoform Disorder). Depression is also associated with elevated heart rate and reduced heart rate variability, which are known risk factors for cardiac disease.^{31,222} Family members who live with individuals with depressive illness report additional behavior symptoms previously unknown and unreported in the literature.^{214a}

Other mental disorders often co-occur with major depressive disorder such as anxiety, substance-related disorders, panic disorder, obsessive-compulsive disorder, anorexia nervosa, bulimia nervosa, and borderline personality disorder.

Almost 80% of depressed people report problems with sleep, including early morning and frequent nocturnal awakenings. Among older adults, depression is often the cause of sleep disturbances, but it may only be the first symptom of systemic illness. Often depression by itself or linked with acute confusion, falling, incontinence, or syncope signifies underlying disease requiring medical referral.

Bipolar disorder may be characterized by elation (mania), but mania is often accompanied by anxiety, intense irritability, or an uncomfortable feeling of being too energized. People with bipolar disorder may have bursts of creativity and productivity when they are manic, but they are more likely to be impulsive and make reckless decisions.

MEDICAL MANAGEMENT

DIAGNOSIS. Depression is often underrecognized and undertreated; primary care physicians detect only 33% to 50% of depressed outpatients. More than 50% of people

with depression present with somatic complaints or masked depression. Instead of telling the physician or health care worker, "I am sad and depressed," they are more likely to report physical symptoms, such as abdominal pain, headaches, joint pain, fatigue, or sleep disturbance, or any of the somatic symptoms listed in Box 3-19. The application criteria based on the DSM-IV-TR[®] remain problematic, with as many as 50% of people with depressive symptoms being unclassified using these diagnostic criteria.

The diagnosis of manic episodes associated with bipolar disorder has been difficult, relying on reports of the person's behavior during a manic episode as described in Box 3-17. Driven behaviors, such as late-night telephone calls, impulsive sexual liaisons, pathologic gambling, and excessively flamboyant dress and behavior, are also characteristic. New neuroimaging studies available in some areas are making it possible to identify those people with biochemical changes associated with depression.

The physician will use the history, laboratory findings, and physical examination to determine whether the depression is a mood disorder caused by a general medical condition (i.e., the direct physiologic consequence of a medical condition such as multiple sclerosis, stroke, or hypothyroidism) or if the depression is considered to be the psychologic consequence of having the general medical condition (e.g., myocardial infarct).

No etiologic relationship between the depression and the medical condition may be evident. The medical management of general medical conditions with accompanying major depressive disorder is more complex, with a less favorable prognosis than just the medical condition alone.

TREATMENT. Treatment for major depression includes psychosocial therapy (including process-oriented, right brain communication during sensitive care that empowers the individual), pharmacotherapy, and electroconvulsive therapy (ECT).

Shock therapy, or ECT, remains a viable treatment tool for depression. ECT uses an electrical stimulus to provoke a controlled seizure inside the brain to affect the brain in the same ways as antidepressants. It is a painless and safe procedure used for depressed people with dementia who do not improve with antidepressant therapy¹⁹⁴ or who are severely suicidal, self-mutilating, catatonic, or unable to eat or function.

National trials are under way to study the use of a vagal nerve stimulator, originally used to control epilepsy, with severe depression that is resistant to medications. The device, implanted under the clavicle with direct attachment to the vagus nerve, sends electrical pulses to the brain and improves mood.

In the case of SAD, light therapy using 10,000 lux is indicated along with lifestyle changes such as attention to nutrition and exercise. Lux is a measurement of light intensity equal to early morning sunlight; a bright summer day is equivalent to 100,000 lux. The light system uses white fluorescent light with a diffusing screen that filters out ultraviolet rays that can cause eye damage and skin cancer.

For the best results, light therapy should begin in the fall season several weeks before the start of daylight savings time. The lights must be set up or tilted at a 45-degree angle 18 to 24 (up to 3 feet away) inches from the eyes. The light must reach the retina or it will not produce any results; light therapy of this type is not absorbed by the skin. Brighter incandescent light bulbs brighten the room to as much as 1000 lux but do not provide enough intensity to treat SAD.

An alternate system called a *dawn simulator* uses a bedside timer to gradually increase the bedroom light in the morning to create an artificial early dawn.¹⁹⁵ Whatever method is used, experts agree that light therapy is best done in the early morning for 20 to 30 minutes. Some people use a standing unit while riding a stationary bike or walking on a treadmill at home. A smaller unit can be used while eating breakfast, reading the newspaper, or working at a desk. Light therapy should be avoided 3 to 4 hours before bed as the light suppresses melatonin, a natural sleep agent.

Light therapy alone helps but does not usually "cure" SAD. Antidepressant medication may be needed by some people with SAD. A program of self-care including exercise, stress reduction, social contact, and positive self-talk is advocated. Outdoor activities are encouraged. Exercise of any kind that increases the heart rate even mildly is advised by most experts in this field. Cardiovascular (aerobic) exercise 5 to 6 times a week for at least 30 minutes may be optimal. Benefits are derived even when working at the lower end of the target heart rate.

Bipolar disorders are treated with various medications (psychopharmacology), including antipsychotics (e.g., risperidone [Risperdal], olanzapine [Zyprexa]); anticonvulsants (seizure medication such as divalproex [Depakote]); and antidepressants (SSRIs), which are often combined with a mood stabilizer such as lithium carbonate. Some people choose to suffer the extremes of this disorder to avoid losing the creative edge that can occur when the medication balances the mood swings. Some programs advocate a predetermined plan of action to put in place if and when warning signs (called *prodromes*) of mania or depression develop. This may help delay or prevent the onset of acute mania.

Complementary therapies have gained increasing popularity as people seek out alternative strategies to cope with depression. Research is limited, but some evidence exists to support the beneficial effects of process-oriented treatment and right brain communication in medical care, trauma-sensitive body work²⁶⁶; exercise; herbal therapy (*Hypericum perforatum* [St. John's wort], S-adenosyl-L-methionine [SAM-e]); and to a lesser extent, acupuncture and relaxation therapies.²⁵³

PROGNOSIS. Depression is a chronic relapsing disorder associated with high morbidity and mortality; the severity of the initial depressive episode appears to predict persistence.²²⁰ Adolescent-onset depressive disorder may carry an increased risk for poor outcome.²⁵³

Chronic general medical conditions are also a risk factor for more persistent episodes. Up to 15% of people diagnosed with this mood disorder die by suicide. Epidemiologic evidence also suggests a fourfold increase in

death rates in people with major depressive disorder who are over age 55 years; from 1980 to 1992 the suicide rate among individuals age 65 and older increased by 9%.

In the same time span, the suicide rate for men and women age 80 to 84 increased 35%.¹⁷⁹ People with this condition admitted to nursing homes may have a markedly increased likelihood of death in the first year. Finally, considerable research investigates whether treating depression will improve medical prognosis in people who have a depressive disorder and a history of coronary artery disease or who have suffered an acute myocardial infarction.¹⁹

SPECIAL IMPLICATIONS FOR THE THERAPIST 3-10

Mood Disorders

PREFERRED PRACTICE PATTERNS

4A: Primary Prevention/Risk Reduction for Skeletal Demineralization

6A: Primary Prevention /Risk Reduction for Cardiopulmonary Disorders

Depression

Depression/depression illness occurs in more than 50% of people with Parkinson's disease and strokes. In these and other clients, the therapist may detect early signs of depression such as pessimistic statements about the illness and its prognosis (poor in the individual's mind) and passive noncompliance during therapy with minimal or no compliance in following a home program or in following (performing) postoperative or rehabilitative exercises.

If the therapist suspects the possibility of depression, baseline information can be obtained and provided to the physician when referring that client. Screening (not diagnostic) tests, such as the Beck Depression Index (BDI),¹⁸ the McGill Pain Questionnaire,¹⁶⁶ the Multidimensional Pain Inventory (MDI),¹⁹ and the Geriatric Depression Scale,²⁶⁵ are noninvasive, easy to administer, and do not require interpretation outside the scope of a therapist's practice.

Asking for permission to discuss symptoms of depression (or other mood or psychiatric disorders) with the referring practitioner or other appropriate health care professional is recommended; in the case of a minor, parental consent may be needed.

When depression is noted, it should be included as a problem in the care plan; the team can then develop strategies to help the person. A client who is not progressing in rehabilitation and who is moderately or severely depressed but not receiving intervention for the depression may need to delay rehab until the depression is under control. Under these circumstances, the therapist should not hesitate to refer a client for evaluation and treatment.

The client with mood disorders may cry easily and often for no apparent reason. Such a situation can be handled by offering reassurance and redirecting the person's attention toward the instructions, activity, or other more positive topics.

Using the acronym PLISSIT may help provide the therapist with some direction early on in the intervention: Permission: Acknowledge the presence of the depression and give the person permission to feel depressed. Limited information: "Of course you are feeling down. You broke your hip, it hurts, and you cannot get around." Again, this acknowledges and validates the person's experience. Specific suggestions: For example, knowing that depression often causes the person to avoid social contact and seek isolation, which then contributes to the depression, encourage the person to make telephone contact with at least one person every day or listen to upbeat music every morning and evening or make arrangements to exercise with someone else (even if only for 10 minutes once a week). Intensive therapy: The client is referred to appropriate specific therapy from other trained professionals.

For the client already taking antidepressants or other medication, the PLISSIT model may be inappropriate because the therapist may be observing "breakthrough" symptoms when the depression (or mania) breaks through the medicinal barrier. This type of situation requires medical reevaluation.

Depression may also lead to anger observed as outbursts of hostility, attempts to sabotage treatment efforts, or blame for the injury placed on the work site or employer. Sometimes the client sees the therapist as an extension of the employer and carries over anger into the rehabilitation process. The therapist can persist in asking questions and actively listening without communicating judgment when the client expresses despair, anger, or negative feelings. The therapist can offer encouragement by pointing out improvements in symptoms or function.

Although the physician must differentiate between pseudodementia of depression and dementia in the aging, the therapist's observations may be helpful toward this end. For example, characteristically, a person with dementia who does not know the answer to a question becomes tangential, changing the subject or resorting to confabulation. In contrast, a person with depression gives up easily and responds by saying, "I don't know."

With some encouragement and extra time, the depressed person often makes an appropriate response, but the person with dementia moves further from the topic with each comment. People with depression commonly have global memory loss, whereas dementia results in loss of recent memory but retention of detail for remote memory. It is possible for dementia and depression to coexist.

Additionally, studies have confirmed that depression is a risk factor for osteoporosis, especially among the aging population and for men. Researchers do not know how depression might cause a loss of bone mineral density, but increases in the stress hormone Cortisol could account for some of the loss. The therapist's intervention for people who are experiencing major depression should include fracture prevention.

Continued.

Exercise and Depression

Physical activity and exercise have a known benefit in the management of mild-to-moderate mental health diseases, especially depression and anxiety. The exact physiologic mechanism for this effect remains unknown, but researchers attribute aerobic exercise with the release of endorphins from the pituitary gland. Endorphins, which are neuropeptides, improve our mood and relieve pain. They also reduce the levels of Cortisol in the bloodstream; Cortisol is linked to stress and depression. Additionally, exercise appears to increase the sensitivity of serotonin neurotransmitter receptors much the same way that antidepressant drugs and therapies release serotonin to help a person relax or sleep.

Increased aerobic exercise or strength training has been shown to reduce mild-to-moderate depressive symptoms significantly in people less than 60 years of age, although habitual physical activity has not been shown to prevent the onset of depression. Studies of older adults and adolescents with depression have been limited, but physical activity and exercise appear beneficial in these populations as well.¹⁸² Numerous studies have shown that resistance training (weight lifting) can be both safe and appropriate for even the frailest individual, many of whom cannot take antidepressants because of the side effects.

In some cases, exercise can alleviate depression immediately, independent of achieving fitness, although some evidence exists that exercise must be continued to remain effective.¹⁴ Exertion appears to increase the levels of various neurotransmitters (e.g., dopamine and serotonin) in the same direction as antidepressants. Earlier research reporting that exercise increases levels of (3-endorphins (believed to be low in depressed people) is being reevaluated now that more highly specific radioimmunoassay tests are available.

Medications

Psychotropic drugs used to affect mood, behavior, and mental function include antidepressant, antianxiety, and antipsychotic drugs. Antipsychotics, also known as neuroleptics, are often used in long-term care settings to help normalize disturbances of thought. Antipsychotic medications are used in a number of conditions to treat psychotic symptoms, including hallucinations, delusions, paranoia, combativeness, agitation and hostility, insomnia, catatonia, hyperactivity, and poor grooming and self-care.¹¹⁸

They do not cure psychosis associated with acute mania and major depression but help manage signs and symptoms. Some of these medications help manage delusions, hallucinations, thought disorders, and persistently bizarre behavior. Other drugs are able to control fluency of ideas and language and alleviate the diminished ability to concentrate, express emotions, pursue goal-directed activity, and experience pleasure.²⁴

The therapist must be aware of anyone taking these medications because of the potential adverse side effects (Box 3-20), especially the extrapyramidal effects

Box 3-20

SIDE EFFECTS OF ANTIPSYCHOTIC MEDICATIONS

Dopaminergic Side Effects

- Pseudoparkinsonism
 - Cogwheel rigidity
 - Shuffling gait
 - Parkinsonian tremor
 - Masked facies
- Acute dystonias, such as opisthotonus, torticollis, and laryngospasm, which may cause acute airway obstruction
- Increased prolactin secretion that may lead to galactorrhea
- Akathisia—subjective or observable restlessness ("Thorazine shuffle")
- Tardive dyskinesia, tardive dystonia
- Neuroleptic malignant syndrome (NMS)

Anticholinergic Side Effects

- Dry mouth
- Blurred vision
- Constipation that may lead to adynamic ileus
- Urinary hesitancy or obstruction
- Memory and concentration difficulties, up to frank delirium

α -Adrenergic Blockade

- Hypotension; orthostatic hypotension

Antihistaminergic Side Effects

- Sedation, drowsiness
- Weight gain

Others

- Agranulocytosis
- ECG changes (prolonged QT interval)
- Elevated liver function tests
- Elevated creatine phosphokinase
- Fetal toxicity
- Photosensitivity
- Pigmentary retinopathy
- Seizures (decreased seizure threshold)
- Sexual dysfunction (erectile problems; impotency; delayed, absent, or retrograde ejaculation; priapism)
- Skin rashes

Data from Jacobson JL: *Psychiatric secrets*, ed 2, St. Louis, 2001, Hanley and Belfus.

or movement disorders commonly observed with their use. Dystonias, sustained abnormal postures, and disruption of movement caused by muscle tone alterations can develop within 5 days of administration. Other common extrapyramidal effects may include restlessness, anxiety, or pacing (akathisia) and Parkinson-like symptoms. Long-term use of antipsychotics can result in permanent involuntary choreoathetoid muscle movements of the face, jaw, tongue, and extremities.

For the client taking tricyclic antidepressants (TCAs), heart rate during peak exercise should be monitored (see Appendix B) because the anticholinergic effect of these medications significantly increases heart rate. Drugs used to treat mood disorders may cause a number of other side effects as a result of increased

Table 3-9 Side Effects of Antidepressants

Drug Class	Tricyclic Antidepressants (TCA)	Selective Serotonin Reuptake Inhibitors (SSRIs)	Monoamine Oxidase (MAO) Inhibitors
Examples	Amitriptyline (Elavil/Endep) Amoxapine (Asendin) Desipramine (Norpramin, Pertofrane) Doxepin (Adapin, Sinequan) Imipramine (Janamine, Tofranil)	Citalopram (Celexa) Fluoxetine (Prozac) Fluvoxamine (Luvox) Paroxetine (Paxil) Sertraline (Zoloft)	Phenelzine (Nardil) Tranylcypromine (Parnate) Selegiline (Deprenyl)
Function	Increase norepinephrine and serotonin levels	Block reuptake of serotonin resulting in higher circulating levels of active serotonin	Inactive MAO, the enzyme responsible for degradation of norepinephrine and serotonin
Effects	Anticholinergic effects: <ul style="list-style-type: none">• Dry mouth• Blurred vision• Nausea, vomiting• Abdominal bloating• Constipation• Confusion (older adults) Heart arrhythmia Tachycardia Orthostatic hypotension: <ul style="list-style-type: none">• Low blood pressure, or sudden drop• Dizziness• Weakness Sedation/drowsiness Sleep disturbance/nightmares Sexual dysfunction Weight gain Fine tremor (older adults) Skin rash/photosensitivity	Nervousness/jitteriness Gastrointestinal distress: <ul style="list-style-type: none">• Appetite loss• Nausea• Diarrhea Headache Insomnia/sleep disturbance Sexual dysfunction	Inactive MAO, the enzyme responsible for degradation of norepinephrine and serotonin Hypertensive crisis Postural hypotension Insomnia Headache Anemia Hyperreflexia Muscle weakness, tremors SIADH-like syndrome Sexual dysfunction Gastrointestinal disturbance

norepinephrine levels such as dry mouth, blurred vision, urinary retention, constipation, palpitations, and orthostatic hypotension (Table 3-9). This last symptom can be the source of dizziness and fainting, increasing the risk of falls and accidents, especially in older adults.

Older adults taking TCAs are at greater risk for heat stroke, especially in the summer. These people are not able to adjust easily to ambient air temperatures, which may affect their exercise program or pool therapy. Since some of these medications' side effects are dose-related, the therapist should encourage the client to report any symptoms to the prescribing physician.

The therapist may observe the breakthrough symptoms mentioned earlier when the depression, mania, personality changes, or psychosis break through the medicinal barrier, or alternately, symptoms may occur when the individual decides to stop taking the prescribed medication without notifying a health care professional.

Symptoms related to TCA withdrawal can occur immediately or up to 48 hours after withdrawal and can continue for as long as 14 days. Withdrawal symptoms may include mood fluctuations, sleep disturbance, gastrointestinal distress, palpitations, dry mouth, or tremors. Withdrawal from TCAs must be monitored by the physician; if the therapist is aware

that the client has decided to discontinue use of these (or other) medications without physician approval, appropriate counsel should be offered. Anyone demonstrating breakthrough symptoms must be referred to the prescribing physician.

Bipolar Disorder

The therapist should watch for warning signs of an impending manic episode, such as increased irritability, the "big plans," and fast talking, and strive to diffuse or deflate the tension. Personal attacks can be deflected by changing the subject or enlisting the client in solving whatever problem is at hand. The therapist may be instrumental in helping the client identify triggers and learn how to avoid them. Time pressure, fatigue, and challenging situations often present in a rehab setting can trigger an episode. The therapist can help create an environment that will reduce these factors as part of the plan of care. Try to work in an area that is quiet with the lights slightly lowered. Turn off the television, radio, or other extraneous background noise.

Suicide and homicide are potential consequences of bipolar disorder. The therapist should pay attention if the affected individual mentions killing himself or others and report such threats to the appropriate personnel.

SUICIDE

Overview and Incidence

Suicide is by far the most devastating outcome of depression; however, people do commit suicide who are not clinically depressed. In addition, there are people with significant mood disorders who do not end their lives.

At least 500,000 people attempt suicide annually; approximately 30,000 are successful, making suicide the fourth leading cause of adult deaths, second or third among youths ages 15 to 24 years old in the United States, and second among college students.¹²⁶

Between 25% and 50% of people with manic-depressive illness attempt suicide at least once. Most people who kill themselves have a treatable mental disorder but do not seek medical care because of social stigma or financial limitations. Many who do see a physician are misdiagnosed. New research in the area of the biologic basis for depression and suicide may result in better care and fewer deaths in the future.

Etiologic and Risk Factors

Great progress has been made in identifying the clinical, genetic, social, and biochemical factors that contribute to suicidal behavior. Positron emission tomography (PET) is now being used to pinpoint biologic markers commonly found in people who are at greatest risk of attempting or completing suicide.^{149,152} These imaging studies show impaired metabolic activity in the prefrontal cortex of the brains of people who have attempted suicide, compared with depressed individuals who have not attempted suicide. The prefrontal cortex is the area of the brain involved in mood regulation. An important factor in setting an individual's threshold for acting on suicidal impulses is brain serotonergic function; serotonin is the neurotransmitter that keeps impulsive and aggressive behaviors in balance.

People who are both impulsive or aggressive and depressed have a much higher likelihood of attempting suicide. Studies continue to examine the relationship of these (and other) variables. Indicators of suicide risk are listed in Box 3-21. Half of all successful suicide victims were described by family or friends as being depressed or suffering some other mental health problem just before their death.¹³⁷

Alcohol and other drug abuse is implicated in half of all cases tested.¹²⁶ In addition to these risk factors, males are five times more likely to take their own lives, and suicide rates are highest for young people under age 25 and white men over age 80. Chronic medical illness in older adults has been linked with increased rates of suicide. The risk is greatly increased in individuals with multiple illnesses.¹²³

Older adults do not necessarily attempt suicide more often than younger people. Instead they are more likely to succeed when they make an attempt. They are more likely to use a lethal method (e.g., gun) and less likely to tell anyone their intentions compared to younger people. Older adults living alone are less likely to be found in time to be saved.

Box 3-21

RISK FACTORS FOR SUICIDE

- Past history of attempted suicide
- Suicidal ideation, talking about suicide, determining a suicide method
- Mood disorders or mental illness:
 - Clinical depression, especially manic depressive illness
 - Schizophrenia
 - Personality disorders, especially borderline and antisocial
 - Chronic alcohol and other drug abuse
- Comorbidities (chronic pain and nonpsychiatric conditions)
- Circumstantial risk factors: stressful life events
- Exposure to suicide or suicidal behavior, especially in adolescents and young adults
- Genetic predisposition/family history of suicidal behaviors
- Decreased levels of serotonin
- Availability of firearms (most common method of completed suicide)
- Gender (males are five times more likely to commit suicide)
- Age (under age 40 or over age 65; risk increases 5 times in white males over age 80)

Courtesy of the American Foundation for Suicide Prevention, New York, 2000 [<http://www.afsp.org>; 888-333-AFSP].

Pathogenesis

Although the signaling and functional roles of serotonin have been implicated in the psychopathology of suicide, the exact physiologic phenomenon remains unknown. Fewer serotonin transporter sites with local reduction of serotonin binding may be associated with the predisposition to act on suicidal thoughts.^{148,149}

Warning Signs of Suicide

Warning signs of suicide, such as mood changes (e.g., irritability, sadness, difficulty getting along with others); loss of interest in family, work, or social activities; and significant changes in sleep pattern or appetite, are common signs of depression serious enough to lead to suicide (see Box 3-17). Suicide threats or previous suicide attempts and even statements revealing a desire to die are other warning signs of suicide. Making final arrangements, giving away prized possessions, saying goodbye to friends and family, and purchasing a gun or collecting prescription drugs are red flag signs of suicide. There may be subtle or overt signs of acute distress, expressions of hopelessness about the future, or a desire to "end it all."

MEDICAL MANAGEMENT

PREVENTION. Focus on suicide prevention has increased in the last 10 years. Age-appropriate tools to screen for depression are available. For example, the Center for the Advancement of Children's Mental Health at Columbia University has developed a Youth Depression Screening test.⁴⁹ The Geriatric Depression Scale can be used with older adults.

The National Institute of Mental Health¹⁸³ and the American Foundation for Suicide Prevention³ have a major focus on education, research, and health informa-

tion related to depression and suicide. Other organizations with a focus on suicide prevention target specific groups.¹¹⁹

It should be noted that for now the accuracy of methods to screen for high risk of suicide is unknown. Likewise, few studies have shown that screening reduces suicide attempts or mortality rates from suicide. More research is needed in this area.²⁴²

DIAGNOSIS AND TREATMENT. New diagnostic neuroimaging now offers an opportunity to visualize serotonin function in a more direct way than has previously been available. Although this technology may provide the possibility of timely therapeutic intervention in people at high risk for suicide, it is not available everywhere.

Major depression can be treated via counseling and trauma/stress resolution and/or pharmacologically, although it is often undertreated, even in the presence of a history of suicide attempt. Some suicide attempts may be preventable if diagnosed early and treated adequately. The need for psychoeducation for health professionals and the public is evident.²⁶⁰

PROGNOSIS. Treatment with SSRIs brings complete resolution of depressive symptoms for up to half of the people taking these agents. In addition, there is a lower risk of fatal overdose or serious heart arrhythmia reported with SSRIs compared with other antidepressants. Despite earlier concerns, there is no convincing evidence that SSRIs are linked with higher rates of suicide; however, tricyclic antidepressants are the leading cause of death by overdose after illicit drugs.^{84,121}

SPECIAL IMPLICATIONS FOR THE THERAPIST 3-11

Suicide

Treatment of depression (whether pharmacologic or psychotherapy) does not change or alleviate symptoms immediately; most drugs used to treat depression require 3 to 4 weeks before a true mood-elevating effect is perceived. The physical symptoms of sleep and appetite disturbances, fatigue, and agitation are the first to improve with medication. Cognitive and emotional symptoms, such as low self-esteem, guilt, uncertainty, pessimism, and suicidal thoughts, resolve more slowly but benefit significantly from modifying the health care approach to process-orientation rather than goal-orientation.^{209,266}

Side effects of antidepressant medications are common and may affect multiple systems. The therapist should be alert to any mention of these and encourage the affected person to continue taking the prescribed medications and to contact his or her physician before discontinuing or tapering dosage.

The therapist can offer some practical suggestions, such as an OTC artificial saliva spray for dry mouth; an education and prevention and management program for orthostatic hypotension (see the section on Postural Hypotension in Chapter 12); or reduced caffeine intake for people experiencing tremor.

Box 3-22

QPR FOR SUICIDE PREVENTION

Q—Question the person about suicide:

- Do you have thoughts of suicide? If yes, do you have a suicide plan in mind?
- Tell me about your plan.

Indirect questions:

- Are you unhappy enough to wish you were dead?
- Do you wish you could go to sleep and never wake up?
- Have you ever wanted to stop living?
- Do you feel your life is no longer worth living?

P—Persuade the person to get help. Listen carefully. Offer to help by making a referral or accompany the person to get help.

R—Refer for help. Contact the individual's physician, minister, rabbi, counselor, tribal leader, or call 1-800-SUICIDE (1-800-784-2433) for assistance in finding local agencies or services in your area.

NOTE: QPR is not intended as a form of counseling or prevention. It is a screening and prevention tool to help assess warning signs of suicide and potentially prevent a successful suicide. Asking questions about suicide does NOT increase the risk of suicide attempts or success.

Although some severely depressed people lack the energy necessary to complete an impulsive act such as suicide, close observation is required during the early weeks of pharmacologic treatment. As energy is restored but before a stable elevation of mood is achieved, the individual is at increased risk for suicide.

All suicidal thoughts and acts must be taken seriously and responded to appropriately. Three-fourths of all suicide victims give some warning of their intentions to a friend or family member. Many older adults who commit suicide have contact with a health care professional (usually their primary care physician) in the month before killing themselves; 40% are in contact with their physicians sometime in the week before taking their own lives.²⁴²

Observe for changes in client mood such as calmness or tranquility in a formerly hostile, angry, or depressed client. Such a behavior change may be a prelude to a suicidal event. Comments such as, "I won't be seeing you again," or "My family would be better off without me" may be a form of suicidal communication. Developing a contract with the client and identifying when and who should be contacted, as well as consequences of breach of contract, may be helpful to the caring relationship and to treatment success.

Use the QPR for Suicide Prevention model to potentially save a life (Box 3-22). Do not hesitate to ask whether a person is considering suicide or even if he or she has a plan or particular method in mind. Do not attempt to argue someone out of suicide, but rather let that person know that you care and understand and that depression can be treated. Avoid the temptation to offer reasons for living such as, "You have so much to live for," "You have come so far to

throw your life away," or "Your suicide will hurt your family, think about them."

People who are suicidal may also be manipulative; therefore therapy staff members need to be aware of and manage their own feelings while empathizing with the client's point of view. When in doubt, report concerns to the appropriate resource (e.g., physician or counselor when one is involved).

The therapist should chart accurately anything the client says or does that might suggest a suicide threat. It is better to meet the required standard of care and err on the side of caution through documentation and the referral process. In some cases, calling for emergency intervention may be necessary. A national suicide referral source is available by calling 1-800-SUICIDE (1-800-784-2433).

References

To enhance this text and add value for the reader, all references are included on the companion Evolve site that accompanies this textbook. The reader can view the reference source and access it online whenever possible. There are a total of 267 cited references and other general references for this chapter.

CHAPTER 4

Environmental and Occupational Medicine

CATHERINE C. GOODMAN

INTRODUCTION

Environmental medicine is a broad term that encompasses industrial and occupational medicine and environmentally induced illnesses and conditions. It is used throughout this chapter to refer to all three branches of study. Environmental medicine and a separate branch of medicine called *clinical ecology* both study the results of interaction between humans and the environment. *Occupational medicine* is a specialty involving the health of workers and workplaces and can be considered a special form of environmental medicine.

Clinical ecology encompasses little-understood health disorders and chronically fluctuating illnesses mainly attributed by clinical medicine to psychosomatic complaints. Considerable polarization occurs over the issues of environment-related illnesses. People affected by environment-related illnesses consider themselves victims of medical ignorance, and the medical community is skeptical of the physiologic basis for the often numerous and vague symptoms described.

The *environment* is defined as all agents outside the body, including infectious organisms, toxins, and food. Intrinsic factors include the genetic makeup of the host and the individual's underlying state of health and history of past illnesses. Cell injury and resultant disease result from interplay of the environment and these intrinsic factors when the host defenses are overcome. Whether at home, in the workplace, or in the community at large, chemical, physical, biologic, psychosocial-spiritual, and traumatic hazards exist.

Usually the focus of environmental medicine is on chemical and physical hazards in the environment. Many diseases, disorders, and defects (contact dermatitis, obstructive lung disease, nephropathy, neuropathy, autoimmune disorders, various cancers, and birth defects are a few examples) occur when the body is exposed to some agent or stressor in the environment.

Industrial, occupational, and environmental illnesses, injuries, and diseases widely affect the population. Hazardous waste sites, nuclear energy leaks, contaminated drinking water, low-level exposures to untested chemical compounds, and repeated exposure to electromagnetic waves and secondhand smoke are examples of problems the American public continues to face. However, it should

be noted that in comparison to all the possible hazards listed, morbidity and mortality from the voluntary intake of tobacco smoke, alcohol, and illicit psychoactive drugs far exceed effects from all other environmental hazards combined.

Many consumers are critical of the lack of an adequate, integrated, or holistic approach to health care, including the consideration of biopsychosocial-spiritual aspects. The challenge in the new millennium is to develop effective consumer protection against environmental illnesses for the general population and to bring evidence-based medical practice to the treatment of these illnesses for those people already affected.⁴⁵

Molecular Epidemiology

A new area of research called *molecular epidemiology* is specifically aimed at measuring biologic effects and the influence of individual susceptibility to carcinogens and mutagens. With the development of the Human Genome Project, increased genetic and population-based association studies are focusing on identifying underlying susceptibility genes and contributions from gene-environment interaction to common complex diseases.

Exposure to environmental contaminants can now be measured using biomarkers such as metabolites in urine, chromosomal aberrations, mutations in specific genes, or deoxyribonucleic acid (DNA) measure of exposure to hydrocarbons or tobacco smoke. Biomonitoring involves looking for "pollution in people" by testing bodily substances, usually blood and urine, for the presence of harmful substances such as dioxins, polychlorinated biphenyls (PCBs), and DDT.

Epidemiologic studies support the use of chromosomal breakage as a relevant biomarker of cancer risk. New ways of analyzing oxidative damage to DNA are now available.¹⁵²

Regulation of Environmental Health Care

Multiple agencies exist for the investigation and regulation of environmental health care. The National Institute for Occupational Safety and Health (NIOSH) is the federal research agency that conducts studies to develop

safety and health standards. It does not have legal authority to adopt or enforce regulations.

The Occupational Safety and Health Administration (OSHA) is the primary regulatory agency that determines which of the standards proposed by NIOSH are adopted and enforced. Its standards are law throughout the United States, and its compliance officers can inspect the workplace at any time to determine the status of health and safety. Many other government and private agencies and organizations are concerned with these regulatory issues.

Risk assessment used by the Environmental Protection Agency (EPA) in regulating new chemicals determines how much harm is acceptable to human health, animals, or the environment. Risk assessment determines how much hormone or pesticide residue is allowed in food, how much of a toxic substance can be discharged into a river, how much pollutant can be released as automobile exhaust, or how clean a Superfund site has to be before it is deemed "safe." Some scientists are advocating an alternative to this type of risk assessment by asking whether this toxin is necessary instead of how much is safe.¹¹⁰

Applying a precautionary principle and questioning whether a new substance is needed requires the industry to find alternatives when there is evidence of damage to the environment. For example, the industry did find an alternative to chlorofluorocarbons (CFCs) when damage to the ozone was identified. Many countries around the world have already established this approach by offering financial incentives for organic farming, resisting importation of beef treated with growth hormones, and seeking alternatives to new chemicals.

ENVIRONMENTAL MEDICINE

The rapid proliferation of new industrial materials, production methods, and commercial products in the twentieth century (particularly since World War II) has progressed with little known about their effects on the environment and human health. Only about 10,000 of the estimated 85,000 chemicals used commercially today have been tested for toxicity in animals. Although toxicity testing lags far behind the rate of new developments, the incidence of work- and environment-related illnesses in humans increases.

Each year in the United States, more than 2 million people experience permanent or temporary disability from various causes, including occupational illness and injury. Although the number of people with disabilities resulting from occupational illness is not known, it has been estimated that at least 390,000 new cases of disabling occupational illness and as many as 100,000 deaths from occupational diseases occur each year.

It is likely that because of the difficulty of diagnosis and the likelihood that occupational illness claims will be disputed by employers, these figures are most likely gross underestimates of the true incidence of environmentally induced illnesses. An estimated 25% of preventable illnesses worldwide can be attributed to poor environmental quality. In the United States, air pollution

alone is estimated to be associated with 50,000 premature deaths and an estimated \$40 to \$50 billion in health-related costs annually (preterm birth, infant mortality, lung disorders, or asthma).

The National Academy of Sciences estimates that 15% of the population experiences some degree of chemical sensitivity, and chemical-related injury and illness are dramatically on the rise. The Social Security Administration recognized chemical sensitivity as an environmental illness and disability in 1988.

The Clean Air Act, which was last amended in 1990, requires the EPA to set National Ambient Air Quality Standards for pollutants considered harmful to public health and the environment. The Clean Air Act established two types of national air quality standards. *Primary standards* set limits to protect public health, including the health of sensitive subgroups such as children, older adults, and anyone with conditions such as asthma or chronic obstructive pulmonary disease. *Secondary standards* set limits to protect public welfare, crops, vegetation, and buildings.¹⁶⁵

The federal government now has proposed stricter standards for particulates, which are pollutants that come from road dust, boilers, wood stoves, diesel fuel, traffic, and other sources. These standards, referred to as National Ambient Air Quality Standards, govern pollutants such as carbon monoxide, lead, nitrogen dioxide, particulate matter, ozone, and sulfur oxides.¹⁶⁵

Children are especially more likely to be adversely affected by environmental contaminants. From fetal development to early childhood, a greater risk of damage and impairment from exposure to environmental hazards is likely. Children are born with immature nervous, respiratory, reproductive, and immune systems. They absorb a greater proportion of substances through their intestinal tract and lungs and detoxify and excrete toxins differently than adults. Children are outdoors more often, engage in hand-to-mouth activity, and often play in the dirt or on the floor or carpet that places them closer to the source of many pollutants. Air pollution affects children more significantly because of the narrow airways and rapid rate of respiration, allowing inhalation of more pollutants per pound of body weight.

The EPA has established an Office of Child Care Protection to increase its studies on the welfare of children in its environment. Researchers are investigating the possible causal relationship between environmental exposure and the increased incidence of childhood onset of asthma, childhood cancers, autism, and learning disabilities or attention deficit disorders.

Of great concern is the recent finding of polybrominated diphenyl ethers (PBDEs) in human breast milk. PBDEs are flame retardants added to a multitude of products to reduce flammability. PBDEs have also been used in U.S. plastics for the last 30 years. Common consumer products that often include PBDEs are computers, electronics, clothes, and mattresses that are treated to be flame resistant. PBDEs have been found in breast milk samples from around the world and in both the northern and the southern hemispheres, indicating that PBDEs have become a major persistent organic pollutant.^{76,141,161,173}

The EPA is now conducting a biomonitoring study of breast milk. Many chemicals accumulate in the fat cells, making the breasts a prime target for these substances. Concerns about high levels of breast cancer in some areas of the United States have raised the suspicion of synthetic chemicals as an environmental risk factor for this disease. In 1940, a woman's lifetime risk of developing breast cancer was 1 in 22. Today that figure has jumped to 1 in 8. The increase cannot be explained simply by the fact that women live longer now.

The Environmental Working Group (EWG) also conducted a study of 10 umbilical cord samples from babies born in U.S. hospitals and found 287 industrial chemicals and pollutants. The blood harbored pesticides, chemicals from nonstick cooking pans and plastic wrap, long-banned PCBs, and wastes from burning coal, gasoline, and garbage. Each baby was exposed to an average of 200 chemicals. The blood sample with the lowest readings contained 154 chemicals. This study confirms that the placenta does not shield cord blood and the baby from chemicals and pollutants.⁷³

This was the first time a baby's "body burden" was identified and measured. The term *body burden* is used to describe the amount of pollutants buried in the bloodstream, organ tissues, and fat cells. Exposure to chemicals is a bigger concern with infants because the blood-brain barrier that keeps the contaminants from reaching the central nervous system (CNS) is not yet developed, putting the infants at risk for greater neurologic impairment. In addition, females are born with eggs that could become their own babies, meaning later generations may also be exposed.⁷³

As a result of this study, there has been a call for more publicly funded studies of the impact of chemical exposure to children and biomonitoring of exposure to environmental contaminants throughout the lifespan. The National Children's Study established in 2000 is examining the effects of environmental influences on the health and development of more than 100,000 children across the United States, following them from before birth until age 21. The goal of the study is to improve the health and well-being of children. It is anticipated that the preliminary results from the first years of the study will be available in 2009-2010.^{11,105}

Etiologic Factors

Chemical (organic and inorganic), physical, and biologic agents that can be considered environmental hazards are numerous (Box 4-1). Despite the many restrictions on industries placed by the EPA, according to the Toxic Release Inventory (TRI), the increased number of polluters in the United States (and worldwide) and under-reporting practices have resulted in the release of more toxic chemicals into the environment each year. The TRI is a publicly available EPA database that contains information on toxic chemical releases and other waste management activities reported annually by some industry groups, as well as federal facilities.⁴⁸

These agents, combined with psychosocial factors, can lower the body's resistance, making a person more susceptible to infectious diseases. Only chemical and physi-

Box 4-1

ENVIRONMENTAL HAZARDOUS AGENTS

Chemical Agents

Pollution or occupational exposure

- Air (carbon monoxide, smog, radon, acid rain, tobacco smoke, household cleaning products, sick building syndrome; see text for others)
- Water (industrial chemicals, pesticides, disease)
- Food (pesticide residues, hormone residues, irradiation, genetic modification, food additives, preservatives)
- Soil contamination

Asbestos

Manmade minerals

Aging polyvinyl chloride (PVC) (e.g., dolls, toys)

Fire and pyrolysis products

Heavy metals

Waste

- Solid waste
- Hazardous waste
- Incinerator waste
- Medical/infectious waste

Physical Agents

Electromagnetic fields

Vibration

Heat stress

High-altitude and aerospace medicine

Mechanical factors

- Cumulative or repetitive trauma
- Accidents/injury

Noise

Biologic Agents

Bacteria

Viruses

Allergens

Fungi (molds)

Psychosocial-Spiritual Factors

See Chapters 2 and 3

cal agents are discussed here; biologic agents are discussed in Chapter 7; behavioral, social, and lifestyle factors are presented in Chapter 2; and psychosocial-spiritual factors are discussed in Chapter 3.

Chemical Agents

Chemical agents can be classified by use (e.g., agricultural chemicals, automotive products, pharmaceutical agents, cleaning agents, paints, dyes, or explosives); mechanism of action (e.g., enzyme disruption, metabolic poison, irritants, or free radical formation); and target organ(s) (e.g., neurotoxins, hepatotoxins, or cardiotoxins). Although many toxic effects can occur, they can be broken down into three main categories: local acute effects, systemic effects, and idiosyncratic (unpredictable) effects.

Air Pollution

Many investigations of home and workplace environments have clearly documented the role of air pollutants in causing health complaints and disease. For example, construction and architectural modifications introduced

in the 1970s as a result of the worldwide energy crisis have resulted in better insulated and tighter buildings with reduced ventilation. Illnesses that develop from indoor air pollution in tight, energy-efficient homes and buildings with poor ventilation and reduced air-exchange rates are known as *sick building syndrome* or *building-related illness*.

Although exposure to air pollution is classified separately as indoor and outdoor, the concept of total personal exposure, whether exposure occurs in the home, office, outdoors, at home or at work, in a car or movie theater, and so on, is relevant to every individual. Anecdotal evidence and statistical studies have made a correlation between pollution and a variety of diseases, particularly asthma, heart disease, respiratory disorders, and cancer.

People considered especially susceptible to air pollution include cigarette smokers (or those exposed to secondhand smoke), older adults, infants and young children, and people with chronic obstructive pulmonary disease (COPD) or coronary heart disease (CHD). Increased rates of heart attacks and other cardiovascular events are reported with increased exposure to air pollution for individuals with known heart and blood vessel disease. Fine particulate matter that travels directly into the bloodstream, constricting arteries, is considered to be the mechanism for this effect.^{24,25}

Indoor Air Pollution. Other sources of indoor air pollution include tobacco smoke; fireplaces; space heaters; stoves; pilot lights; gas ranges; mothballs; cleaning fluids; glues; photocopiers; formaldehyde in foam, glues, plywood, particleboard, carpet backing, and fabrics; and infectious and allergic agents such as dust mites, cockroaches, bacteria, fungi, viruses, and pollen. Toxic chemicals found in every home, from drain cleaners to furniture polish, are three times more likely to cause respiratory distress than airborne pollutants.

The National Pollution Control Center estimates that the average home has approximately 62 different chemicals and that more than 2 million poisonings involving children age 6 and younger occur every year in the United States. Older children and adults account for another 900,000 poisonings.

Radon, a product of the breakdown of radium, poses an environmental risk because of its carcinogenic, especially lung cancer, properties. Exposure is predominantly naturally occurring rather than generated by human polluters and is present in poorly ventilated homes in the form of an odorless gas. Other sources include radioactive waste and underground mines; exposure to tobacco smoke multiplies the risk of concurrent exposure to radon.^{84,100}

Outdoor Air Pollution. As part of the Clean Air Act of 1990, the EPA set air quality standards to protect sensitive population groups from outdoor air pollutants. The Clean Air Act regulates oxide emissions, making these particles less available to react with volatile organic compounds that form ozone. *Healthy People 2010* set goals to reduce the proportion of people exposed to air that does not meet the EPA's standards for ozone and to reduce the proportion of nonsmokers exposed to environmental tobacco smoke. Preliminary research on pollutants indi-

cates that biofiltration technology used to clean up airborne waste stream removes 94% of total hazardous air pollutants. Scientists are working to identify microbes that will clean up more difficult-to-remove pollutants.⁵⁴

Carbon monoxide (CO), an odorless, tasteless, and colorless gas, is a common environmental pollutant from automobile exhaust emissions; the use of liquefied petroleum gas (LPG)-powered forklifts in inadequately ventilated warehouses and production facilities; fires; and in some areas, home heating systems (e.g., the incidence of CO poisoning in homes with faulty furnaces has become an increasing problem, especially in the Midwest). Inexpensive CO-monitoring devices have helped identify many previously undetected cases of high levels of CO in private homes.

CO is commonly recognized for its toxicologic characteristics, especially CNS and cardiovascular effects. CO combines 240 times more quickly with hemoglobin (or myoglobin affecting muscles) than oxygen, so when carbon dioxide is bound to hemoglobin, its oxygen-carrying capacity is decreased. In the presence of CO, oxygen is not released normally by the blood, resulting in tissue hypoxia.

Tissue hypoxia has serious functional consequences for organ systems that require a continuous supply of oxygen such as the brain and the heart. Exposure to CO also causes impaired visual acuity, headache, nausea, vomiting, fatigue, seizures, behavioral change, and ataxia. In addition, when tissue partial pressure of oxygen is low, CO binds to intracellular hemoproteins, such as myoglobin, inhibiting their function and thereby affecting muscle function.

More severe CO poisoning can produce metabolic acidosis, pulmonary edema, coma, and death. The classic clinical findings of cherry-red lips and nail bed cyanosis caused by the bright-red color of carboxyhemoglobin (COHb) may occur if the COHb concentration is above 40%, but this is rarely observed.

Other air pollutants include *smog*, a combination of smoke and fog that develops when vehicle emissions and exhaust fumes containing nitrous oxides and hydrocarbons are photochemically oxidized. Ozone and nitrogen, the components of smog, result from the action of sunlight on the products of vehicular internal combustion engines. Automobiles and trucks emit unburnt hydrocarbons and nitrogen dioxide. Ultraviolet irradiation of these compounds leads to complex chemical reactions that produce ozone, various nitrates, and other organic and inorganic compounds constituting smog.

Nitrogen dioxide and ozone are toxic byproducts of this reaction. Ozone is also produced in the welding process when oxygen is ionized. Both of these byproducts are toxic to the respiratory tract, damaging ciliated endothelial cells lining bronchioles and impairing the mucociliary clearance mechanism. Outdoor air pollution has long been associated with clinically significant adverse health effects. The very young, very old, heavy smokers, or those with preexisting lung disease are at increased risk in the presence of these toxins. Although it is unclear whether outdoor air pollution contributes to the development of asthma, it does trigger asthma episodes.¹

Growing evidence from around the world shows that the harmful effects of smog extend even to the unborn in utero. More than a dozen peer reviewed studies in the United States, Brazil, Europe, Mexico, South Korea, and Taiwan have linked smog to low birth weight, premature births, stillbirths, and infant deaths. In the United States, research has documented ill effects on infants even in cities with modern pollution controls. Although this research shows a correlation between air quality and infant illnesses, it does not establish a conclusive cause-effect connection.¹²⁵

Acid rain caused by the interaction of sulfur dioxide and nitrogen oxides in the atmosphere forms fine sulfate and nitrate particles transported by wind currents over long distances through the air. Outdoor sulfate and nitrate particles penetrate indoors and can be inhaled deep into the lungs. The northeastern United States experiences the greatest levels of acid and sulfate aerosols (up to 25% of the breathable particles) during the spring and summer months.^{64,151} No known correlation exists between elevated levels of these fine particles and bronchoconstrictive disorders such as asthma, emphysema, and bronchitis.

Water Pollution

Water pollution in the form of contamination of drinking water by toxic chemicals has become widely recognized as a public health issue since the late 1970s. Increased monitoring since then has shown that many pesticides and industrial chemicals can be detected in drinking water. The EPA, in conjunction with public health officials and the drinking water industry (e.g., Partnership for Safe Water), has worked diligently to survey and reduce waterborne-disease outbreaks, chemical contamination from leached industrial waste chemicals, and toxins released into recreational and drinking water.¹²

In 1996, the Safe Drinking Water Act was amended to require all community water systems to deliver an annual water quality report to their customers, including levels of any detected contaminants. The EPA has placed limits on the amount of certain contaminants in water provided by public water systems. Anyone with a private source of water (e.g., cistern or well water) does not come under this type of protection. Water that travels over the surface of the land or through the ground can pick up radioactive substances, naturally occurring minerals, or substances resulting from the presence of animals or human activity.

Contaminants that may be present in source water include microbial contaminants, such as viruses and bacteria, that come from sewage treatment plants, septic systems, agricultural livestock operations, and wildlife. Inorganic contaminants, such as salts and metals, may be present from urban stormwater runoff, industrial or domestic wastewater discharges, oil and gas production, mining, or farming.

Pesticides and herbicides from a variety of sources (e.g., agriculture, urban stormwater runoff, or residential uses) and organic chemical contaminants from by-products of industrial processes and petroleum production (including from gas stations) are additional source-water contaminants.

Some subgroups of people may be more vulnerable to contaminants in drinking water than the general population. Immunocompromised individuals, such as those with cancer who are undergoing treatment; organ transplant recipients; people with human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS) or other immune system disorders; some older adults; and infants are at increased risk from infections.

Disinfection with chlorine is the most common method to ensure drinking water safety in the United States. A dramatic decline in waterborne diseases, such as cholera and typhoid fever, occurs when water systems are disinfected this way. One potential downside of this disinfectant treatment is the increased genotoxicity that occurs with water treatment. Ways to evaluate the toxicity and genotoxicity of disinfected drinking water are under investigation.

A different form of water pollution has also raised concerns. Billions of gallons of treated sewage are released offshore into deep waters via long undersea pipelines called *outfalls*. Wastewater is filtered and processed, but many contaminants (especially estrogenic compounds) remain and settle into ocean sediment, where they are consumed by bottom-feeding organisms that become food for other ocean life. Evidence of abnormalities in animals and fish exposed to sewage and industrial contaminants has been reported, but the effect on overall health and abundance of fish populations and the rest of the marine ecosystem remains unknown.¹³²

In other areas of the United States, past abuses from mining thousands of tons of arsenic, copper, manganese, and other metals harmful to humans and aquatic life polluted the soil and groundwater, leaving areas barren and unable to support vegetation. Acid-generating deposits called *slickens* scattered throughout the floodplain continue to send toxic metals directly into rivers through runoff. Not all areas have undergone cleanup and restoration. The effect on health and potential for higher incidences of cancer and other diseases remains underdetermined at this time.

Food

Food as a pollutant is one of the major environmental agents to which people are exposed. In many documented cases, reversible and irreversible human and ecologic damage has occurred as a result of pollution-induced food contamination. As scientific and epidemiologic information accumulates, society is questioning to what degree these technologies and by-products contribute to the steadily rising incidence of certain cancers, autoimmune and other chronic diseases, birth defects, autism, learning disorders, and other health problems for which the cause is not well understood.

Pesticides, Insecticides, and Herbicides. Pesticide, insecticide, and herbicide residues in food; hormone residues; food irradiation (a method of preservation and protection from microbial contamination); genetically modified foods; and food additives and preservatives are major consumer concerns. Pesticide and herbicide exposure can cause many different health effects, from acute problems, such as dermatitis and asthma exacerbation, to chronic problems, such as COPD and cancer.¹³⁰ Acute

pesticide poisoning has been reported among food handlers (e.g., clerks, baggers, stockers, or shipping/receiving handlers) and janitors in retail establishments that sell food products, especially fruits and vegetables.³¹

Among the people most in danger from pesticide exposure are farmers and agricultural workers. Many studies of these groups have shown an increase in soft tissue sarcomas, presumably from herbicide exposure.⁵⁶ Children, especially very young children, are also at greater risk from exposures to pesticides and other environmental toxins compared to adults because, pound for pound of body weight, children drink more water, eat more food, and breathe more air than adults.

Childhood leukemia has also been linked with the use of home insecticides and garden fungicides during pregnancy and early childhood. The treatment of pediculosis (lice) with an insecticidal shampoo also may be associated with an increased risk of childhood leukemia.⁹⁴ School-aged children are also at increased risk for acute illnesses from repellants and pesticides applied within school grounds, pesticide drift exposure from farmland, and pesticide use at parks.³

In the United States, environmental exposure to chlorophenoxy herbicides used in wheat production has been linked to musculoskeletal and respiratory-circulatory birth defects, cancer, type 2 diabetes, and heart disease.^{135,136} Counties in which wheat is produced have a high rate of defects among infant boys conceived during April or June when herbicide application takes place. Boys conceived during other times of the year and born in counties with low wheat production have far fewer birth defects.¹³⁴

Pesticides that are not registered or are restricted for use in the United States can be imported in fruits, vegetables, and seafood produced abroad. Environmental quality is a global concern as increasing numbers of people and products cross national borders, transferring health risks such as infectious diseases and chemical hazards.

Contaminated Soil

Contaminated soil is often the main source of chemical exposure for humans, and an active interchange of chemicals occurs between soil and water, air, and food. Direct contact and ingestion of soil are important exposure pathways, and inhalation of volatile compounds or dust must also be considered. The movement of contaminants through soil is very complex, some moving rapidly and others slowly, eventually reaching and contaminating surface or ground water on which people rely for drinking and other purposes.

Asbestos

Asbestos continues to be a significant occupational hazard. It was not until the late 1960s and early 1970s that the public was made aware that asbestos used in products ranging from automotive brake linings to building insulation caused chronic respiratory illnesses, cancer, and other illnesses. Since then, commercial use of asbestos has decreased dramatically.

Abatement workers employed to remove asbestos in buildings wear protective clothing to decrease exposure

but still are considered at risk. Long latency (exposure occurring 30 or more years ago continues to affect former workers) and long-term, low-level exposure to the presence of indoor asbestos remain risk factors. It is not just asbestos but other long, thin mineral fibers in the workplace or in the environment that can have similar effects. See Chapter 15 for a discussion of asbestosis.

Manmade vitreous fibers containing mineral wool, glass wool or fiber, and ceramic fiber have replaced asbestos in the workplace. The nonoccupational exposure to manmade minerals does not put consumers at substantial risk; health issues related to these materials mainly occur among workers with long duration of exposure. Clinical consequences are similar to those of asbestos, including pulmonary fibrosis, bronchogenic carcinoma, mesothelioma, and possibly other types of cancers.

Other Chemical Compounds

Polyvinyl chloride (PVC), a type of plastic made flexible through the addition of a chemical, is used in a variety of products, including medical solutions stored in PVC medical devices such as saline bags. Vinyl chloride production has doubled in the last 20 years, with current production of 27 million tons per year worldwide. Concern exists over the possibility of chemical plasticizers leaching into the solutions used long-term by certain populations, including people on dialysis, individuals with hemophilia, or neonates exposed at critical points in development.

Additionally, measured changes in the acidity of intravenous (IV) solutions in PVC packaging have been reported.¹⁵⁵ Dioxin, a byproduct of PVC plastics manufacturing, was declared a carcinogen by the EPA in June 2000. Dioxin accumulates in fatty tissues of mammals and fish. The observed toxicities of these chemicals have been linked to birth defects and immune system disorders, resulting in the request for PVC-free medical devices and reduction of environmental contamination with these compounds to the lowest level possible.^{82,160} High levels of dioxin exposure are associated with chloracne, a distinctive form of acne (Fig. 4-1), and with porphyria cutanea tarda (Fig. 4-2).

Most adults tested also show the chemical perfluorooctanoic acid (PFOA), a chemical compound widely used in Teflon-coated cookware, water- and stain-resistant clothing, cosmetics, and many other products. There has been growing concern about the effects of this compound. Extensive scientific assessment of the perfluorochemical family is underway to determine whether the substances cause sexual reproductive and developmental damage to females. PFOA has also been linked to testicular, liver, and pancreatic cancer in animals.^{30,115}

Fire and Pyrolysis

Fire and pyrolysis directly affect 2 million people annually who are treated for burns, including civilians and firefighters. Pyrolysis, or incomplete combustion, of wood releases many highly toxic compounds that can react with other organic substances to produce new toxic and irritant chemicals. Incomplete combustion and fire-fighting water also produce highly acidic aerosols. Smoldering or partially controlled fires release many toxic products.



Figure 4-1

Chloracne. (From Bolognia JL, Jorizzo JL, Rapini RP: *Dermatology*, St Louis, 2003, Mosby.)



Figure 4-2

Porphyria cutanea tarda. Erosion, crusting, and vesicles on the dorsum of the hand in an individual with porphyria cutanea tarda. (From Goldman L: *Cecil textbook of medicine*, ed 22, Philadelphia, 2004, WB Saunders.)

The most common type of injuries is in the category of smoke inhalation and respiratory problems followed by lacerations, contusions, and falls. Death can occur as a result of smoke inhalation and myocardial infarction. See the section on Occupational Burns in this chapter.

Waste

Waste from solid, hazardous, and incinerator by-products is not likely to be encountered directly in a therapy practice. However, the effects of exposure to medical/infectious waste may be more problematic. Standard precautions for handling all medical/infectious waste are available (see Chapter 8 and Appendix A).

Heavy Metals

Heavy metals, such as lead, arsenic, and mercury, actually fall under the chemical agents category but are mentioned separately because of their former prevalence and uniqueness as classic occupational and environmental hazards. In the early 1990s, environmental concerns shifted attention away from lead, mercury, arsenic, and asbestos exposure despite continued high production volume chemical development, toxicology testing, and issues centered around environmental justice.⁸⁵

However, new findings from the TRI have resulted in a resurgence of interest and research in this area. The TRI is a publicly available EPA database that contains information on toxic chemical releases and other waste man-

agement activities reported annually by some industry groups, as well as federal facilities. The 2006 TRI findings report 4.24 billion pounds of on-site and off-site disposal or other releases of toxic chemicals. Total disposal or other releases of mercury and mercury compounds amounted to 4.8 million lbs in 2004.

Lead Poisoning

A normal blood lead level is 0. Apparent toxicity is not usually demonstrated until the blood serum lead levels exceed 24 mg/dl in adults and 10 mg/dl in children. Lower levels (5 mg/dl) have been identified for pregnant women. Any levels above these marks require intervention. OSHA prohibits workers with levels greater than 40 mg/dl from returning to the workplace where lead is present.³²

Lead poisoning is on the decline in the United States as a result of federal initiatives to end the use of lead in gasoline, lead solder in the seams of food cans (beware of foods in cans manufactured outside the United States), lead-based paints, and plumbing in homes. Consumers should also be aware that the porcelain glaze of old bathtubs and the glaze on imported dishware (ceramics, china, or porcelain) often contain lead and are a potential source of lead exposure. Internet purchases of dishware and ceramics on-line present a particular threat. Lead can leach out of dishware when the glaze is improperly fired or when the glaze has broken down because of wear from daily usage, especially after repeated use in a microwave or dishwasher. Chips and cracks in ceramic ware also allow leaching of lead. Children's risk for such exposures is increased with frequent consumption of acidic juices that promote lead leaching from ceramics.⁹⁹

Ingestion of lead paints found in older residential neighborhoods and exposure to lead dust particles during home renovation projects remain continuing problems among the pediatric population. Likewise, dust and soil containing lead particles too small to see expose children, who are more likely to be on the ground or outside and who engage in more hand-to-mouth activities.

Children. Lead is particularly toxic to infants and children for several reasons, including (1) the blood-brain barrier is immature before the age of 3, allowing lead to enter the brain more readily; (2) ingested lead has a 40% bioavailability in children, compared with 10% in adults; and (3) the behavioral hand-to-mouth habits previously mentioned. Lead is stored in the body predominantly in bone but may adversely affect many organ systems, including the CNS and the gastrointestinal, hemopoietic, reproductive, and renal systems.

Health effects in infants born to women with moderately elevated blood lead levels include preterm birth, decreased gestational maturity, lower birth weight, reduced postnatal growth, increased incidence of minor congenital anomalies, and early neurologic or neurobehavioral deficits. It remains unclear how long these neurologic effects persist, but some evidence suggests a link between prenatal elevated lead levels and decreased intelligence in children up to age 7 years.³²

Serum levels once thought to be safe have been shown to be associated with intelligence quotient (IQ) deficits, behavioral disorders, slowed growth, decreased compe-

tency in verbal performance and auditory processing, and impaired hearing. The impairment of cognitive function begins to occur at levels greater than 10 µg/100 ml, even though clinical symptoms may not be apparent; serum levels are required for diagnosis. Other studies show cognitive and delayed puberty at levels below 10 µg.^{33,126,140} Lead exposure has also been linked with reproductive dysfunction and toxicity to the kidneys, blood, and endocrine system.

Other risk factors for children include age under 6 years, low income, and urban dwelling. Adults are more likely to be exposed to lead in the manufacture of brass, batteries, bullets, solder, or glass; furniture refinishing; home renovations; stained-glass or pottery making; and prolonged exposure to the burning of metallic wick candles (e.g., home use, restaurant, religious, or ceremonial).^{109,149}

Arsenic

Arsenic is used in the manufacture of glass, pesticides, and wood preservatives and has been found to contaminate water, beer, and seafood. Arsenic binds to tissue proteins and is concentrated in the liver, skin, kidney, nervous system, and bone, with bone being affected to a lesser extent than with lead. The symptoms of acute inorganic arsenic poisoning may include severe burning of the mouth and throat, abdominal pain, nausea, vomiting, diarrhea, hypotension, and muscle spasms.

Epidemiologic studies have demonstrated a correlation between environmental or occupational arsenic exposure and a risk of vascular diseases related to atherosclerosis. It appears that arsenic induces endothelial dysfunction as a result of impaired nitric oxide (NO) balance, and inflammatory and coagulating activity. Arsenic may accelerate atherosclerosis, but the mechanism for this event remains under investigation.¹⁴⁵

In severe cases, cardiomyopathy, jaundice, renal impairment, red cell hemolysis, ventricular arrhythmias, coma, seizures, and intestinal hemorrhage are seen. Chronic arsenic poisoning is characterized by an irregular dusky pigmentation and hyperkeratosis of the skin that looks like raindrops on a dusty road. Painful dysesthesia in the hands and feet, bone marrow depression, transverse white striae of the nails, altered mentation, and occasionally garlicky perspiration odor may occur.

Cancers of the skin, kidney, bladder, and lungs have been associated with arsenic poisoning, but the mechanisms responsible for arsenic carcinogenesis have not been established. Increasing evidence indicates that arsenic acts at the level of tumor promotion by modulating the signaling pathways responsible for cell growth.^{146,170} The risk of arsenic-induced cancer is associated with 20 or 30 years of drinking polluted water, not from a brief or occupational exposure. The current standard in the United States is 10 parts per billion. Any water supply that has much higher arsenic levels is not considered safe for human consumption.

Mercury

Mercury is widespread and persistent in the environment and can be dangerous when ingested, inhaled, or absorbed through the skin. Most people have some amount of

mercury in their bodies, but random testing of individuals has revealed higher than the acceptable, safe limit of 1 part per million set jointly by the EPA and the Food and Drug Administration (FDA).

Exposures to women of childbearing age, pregnant and nursing women, and children younger than age 15 are of great concern because of the susceptibility of these groups and resultant adverse effects. According to analysis of data gathered by the National Health and Nutrition Examination Survey, between 200,000 and 400,000 children born in the United States each year have been exposed to mercury levels in their mothers' wombs high enough to impair neurologic development.¹⁰⁶

Sources of Mercury. Harmful mercury vapors can be transferred to water and soil where they can be introduced into the food chain, causing renal and neurologic disorders. Although eating contaminated fish is the leading cause of mercury accumulation in humans, elevated levels of emissions from coal-burning power plants and petroleum refineries, mining-related wastes, and the improper disposal of mercury products have resulted in increased mercury in the environment, with the trickle down effects on fish. The larger the fish, the higher the concentrations of mercury found in the fish.

Elemental or inorganic mercury is released into the air or water where it accumulates in animal tissues and increases in concentration through the food chain. The U.S. population is primarily exposed by eating fish, but mercury is also used in electrical products and as a fungicide. Industrial sources, such as coal-fired power plants, cement kilns, and refineries, also emit mercury into landfills and into the air and water. In water, mercury becomes methylmercury, a toxic substance that is consumed by fish.

American hospitals dispose of 17 tons of waste each year containing mercury found in medical supplies. In 1998 the American Hospital Association (AHA) and the EPA enacted a new initiative called *Hospitals for a Healthy Environment* (H2E), which outlined 10 steps for environment-friendly healthcare. Some of the top priorities are to reduce the total volume of hospital waste to 0% by 2010, with the total elimination of mercury products.^{67,72}

There is considerable controversy as to whether dental amalgams ("silver fillings") may cause significant health effects in humans. Dental amalgams still contain mercury as other materials have not been developed that are as strong or as long lasting. Amalgam restorations contain mercury, silver, tin, copper, and a trace amount of zinc. There is some evidence that a sustained release of mercury and other metals occurs from the amalgam into the body. Researchers have measured a daily release of mercury on the order of 10 µg from the amalgam into the body. Mercury is a toxic metal; the smallest amount damages cells.

In response to a need for a greater concentration of research in this area, a group of concerned dentists formed the International Academy of Oral Medicine and Toxicology (IAOMT) in 1984. One of their objectives was to scientifically explore the safety of amalgam restorations. Since that time, scientists at universities around the world have begun to research possible pathophysiological effects

associated with mercury leaking from amalgam restorations.

Consequently, there are a growing number of scientific studies that document pathophysiological effects associated with amalgam mercury. In the interest of protecting their citizens' health, Sweden, Norway, Germany, Denmark, Austria, Finland, and Canada have recently taken steps to limit and phase out the use of amalgam restorations. About one-third of the dentists in the United States no longer use mercury-containing products. Resin composite fillings, which match the tooth color, are used instead.

Despite consumer concerns about mercury exposure from dental fillings, clients and dental personnel are at greatest risk when amalgams are removed. The aerolization during removal creates greater mercury exposure than the hardened and intact filling in the mouth. It is advised by some that amalgams should only be removed when the filling (or tooth) is no longer intact, rather than to eliminate mercury exposure. Dental personnel are at greatest risk for this type of repeated exposure.

Clinical Manifestations (Mercury Exposure). The EPA reports that about 5 million women had at least 5.8 parts per billion of mercury in their blood as of 2000. They also noted that children born to women with blood concentrations of mercury above 5.8 parts per billion are at some risk of adverse health effects, especially decreased IQ and problems with motor skills.⁴⁷

Exposure to hazardous levels of mercury can cause permanent neurologic heart and kidney impairment. Neurologic or neurodegenerative diseases, mental retardation, cerebral palsy, seizures, memory loss, learning disabilities, developmental delays, autoimmune disorders, mental health disorders, and birth defects are among the many conditions blamed on mercury exposure.

Vaccines commonly given to children before 2001 contained a preservative (Thimerosal) that contained mercury. There were concerns raised over the total number of vaccinations given to children during the first 6 months of life that could lead to toxic levels of mercury. There was some suspicion that increased rates of autism could be the result of mercury poisoning from vaccines, but this has not been proved conclusively.

Although mercury is poorly absorbed from the gastrointestinal tract, mercury vapor is well absorbed through the lungs and from the gut. Mercury poisoning causes irritation of the mouth and pharynx and is accompanied by vomiting, dehydration, abdominal cramps, and bloody diarrhea. Death can occur from acute renal failure.

Chronic exposure to mercury may cause additional symptoms of gingivitis, speech defects, tremor, and a chronic personality disorder called the *Mad Hatter syndrome*, characterized by unusual shyness, labile affect, and decline in intellect. Mercury poison affects the nervous system, resulting in dysarthria, ataxia, paresthesias, and constricted visual fields.

Mercury Regulation. In 2005, the EPA took its first step toward reducing mercury pollution from coal-fired power plants with the Clear Air Mercury Rule designed to reduce mercury emissions by 70% over the next 20 years. The long-term strategy for reducing exposure to mercury is to cap mercury emissions from coal-burning

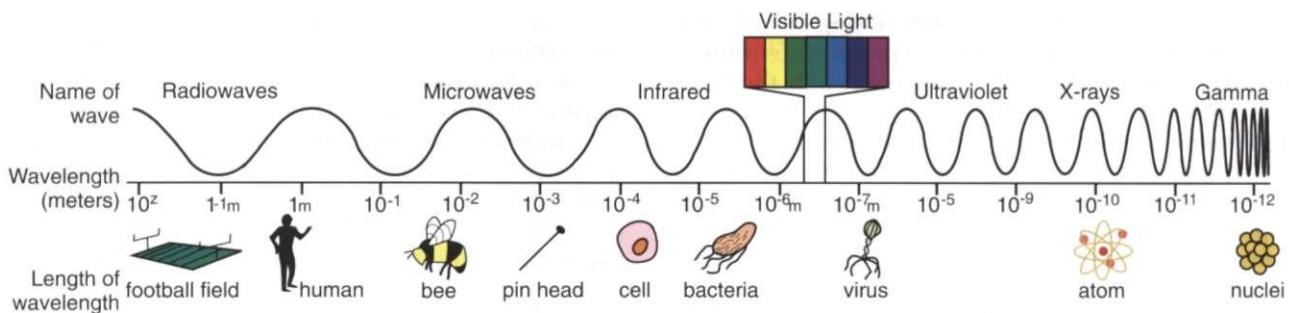


Figure 4-3

Electromagnetic (EM) spectrum. Different types of electromagnetic radiation have different frequency or wavelengths. Radio waves, television waves, and microwaves are all types of electromagnetic waves. The electromagnetic spectrum includes, from longest wavelength to shortest: radio waves, microwaves, infrared, optical, ultraviolet, x-rays, and gamma rays. Waves in the electromagnetic spectrum vary in size from very long radio waves the size of buildings, to very short gamma rays smaller than the size of the nucleus of an atom. The frequency is the rate at which the electromagnetic field goes through one complete oscillation (cycle) and is usually given in Hertz (Hz), where 1 Hz is one cycle per second. As the frequency rises, the wavelength gets shorter.

power plants nationwide, thereby lowering mercury releases into the atmosphere and lowering mercury concentrations in fish. Phasing out mercury-containing products, such as thermometers and thermostats, has already become a national trend.

The new rule does not require all plants to reduce emissions by the same amount. Some may be allowed to pollute more than others by buying "pollution credits" from other plants. This practice is called *cap and trade*. It is based on the assumption that mercury pollution disperses evenly in the environment, but in fact, mercury emitted by coal-fired power plants tends to remain near those plants. This creates what are referred to as geographical "hot spots" of mercury.

EPA regulations for waste incineration have resulted in decreased air emissions of mercury in the last two decades, and this trend is expected to continue. The FDA advises that children, nursing mothers, pregnant women, and those who may become pregnant should not eat fresh tuna, canned white (albacore) tuna, shark, swordfish, king mackerel, and tile fish known to contain elevated levels of mercury.⁹⁸

Xenoestrogens/Xenobiotics

Xenoestrogens are also part of chemical environmental exposure but are discussed separately because of their unique place as a hazardous agent. In the early 1970s, scientists from around the world met together to discuss the cumulative efforts of researchers investigating various endangered species. Together they identified that exposure to petrochemicals (previously called *xenoestrogens* but now referred to as *xenobiotics*, meaning "foreign to life") is the underlying cause of dwindling births in these species.

Petrochemicals, such as pesticides and insecticides, are the primary xenobiotics and constitute substances totally foreign to nature—that is, they are not found in the natural world but rather are synthesized chemicals. Other petrochemicals are present in commonly used items or products such as emollients in lotions and creams, spreaders in salad dressing, carpet glues, paints, solvents, automobile gasoline, plastics, and a multitude of other

common household objects. Researchers concluded that the effect of these residues is selective to the reproductive systems of the developing fetuses so that exposure in the developing fetuses resulted in infertility or sterility. Since that time, it has been recognized that these chemicals can affect other systems, including the thyroid, immune function, and nervous system.

The effect of these chemicals has been to create what is referred to as an *estrogen-dominant environment* because the chemicals have estrogenic activity. Estrogen dominance on humans (both men and women, although women are more susceptible) is the subject of intense scrutiny by scientists and researchers.

Studies from around the world continue to report broad human exposure to what are now called *estrogenic endocrine-disrupting chemicals* (EDCs) from environmental media such as food and water contaminated by dioxin and bisphenol A (BPA) widely used for the production of plastic products. BPA has been found in fetal serum and full-term amniotic fluid, confirming passage through the placenta.¹⁶⁴ This biologic phenomenon may be linked to autoimmune dysfunction, increased body fat, decreased sex drive and sperm production, altered blood clotting, early menarche, zinc deficiency associated with prostate dysfunction, endometriosis, and headaches associated with fluid retention.

Researchers are in agreement that such compounds in high doses may cause developmental, reproductive, and tumorigenic effects, but controversy remains regarding the risks associated with xenoestrogens under low exposure that are considered more realistic and how to assess the interaction of exogenous compounds with the endocrine system and its complex regulation.⁴¹

Physical Agents

Electromagnetic Radiation

The long-term effects of exposure to electromagnetic radiation or electromagnetic fields (EMF), including radiofrequency and microwave, ultraviolet light, x-ray, and gamma rays, remain under intense scrutiny (Fig. 4-3). Ionizing radiation is the result of electromagnetic waves entering the body and acting on neutral atoms or mole-

cules with sufficient force to remove electrons, creating an ion. The most common sources of ionizing radiation exposure in humans are accidental environmental exposure and medical, therapeutic, or diagnostic irradiation.

All living material is vulnerable to ionization by high-energy radiation because the disruption of atoms joined into molecules producing ions and free radicals (see Chapter 6 and Fig. 6-2) can result in further biochemical damage, including somatic effects, such as cell death, and genetic effects, including reproductive effects and cancer. Radiation-induced changes can cause genetic mutations and structural rearrangements in chromosomes that can be transmitted from generation to generation.^{16,163}

A wide range of other adverse health effects have been attributed to ionizing radiation, including visual, thermal, behavioral, CNS, and auditory effects; effects on the blood-brain barrier; and immunologic, endocrinologic (including effects on biorhythm), hematologic, developmental, and cardiovascular effects.

Exposure to nonionizing radiation (i.e., the electromagnetic wave does not have enough energy to strip an atom of its electron) occurs most commonly as a result of the use of a wide variety of industrial and electronic devices (e.g., microwave ovens, scanning lasers in stores, high-intensity lamps, video display terminals [VDTs], scanning radars, or electronic antitheft surveillance).

Chemical compounds from plastic wrap surrounding food or covering dishes used in a microwave can leach into the foods and affect the body. Packaging and plastic wraps that contain polyethylene are preferred for use in the microwave, since these do not have plasticizers (materials that make the wrap more pliable). Containers meant for cold foods, such as margarine or whipped topping, should also be avoided for microwave use, since these containers can melt, dispersing some of their components into the food.

Considerable speculation has gone on around the world that long-term exposure to EMFs is correlated with the development of breast cancer, leukemia, miscarriage, and neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis (ALS).⁶³

The unexplained high incidence of breast cancer in industrialized nations is suspected as being linked to electric power generation and consumption. The proposed biologic mechanism is the inhibition of melatonin caused by the products of electric power generation, EMFs, and light at night, but this has not been proved and further investigation is warranted.^{40,93,114}

Most exposures to electromagnetic interference are transient and pose no threat to people with pacemakers and implantable cardioverter defibrillators; however, magnetic resonance imaging (MRI) and prolonged exposure to EMFs are contraindicated in anyone who is pacemaker-dependent.¹¹⁸

Concerns that cellular telephone radiation is linked to brain tumors or causes a variety of serious problems (e.g., genetic damage, pacemaker or implantable cardioverter defibrillator disruption, interference with heart/lung monitors, or compromise to the blood-brain barrier) have not been substantiated or proven clinically important.¹⁶² Long-term studies of longer induction periods,

especially for slow-growing tumors with neuronal features, conclude that the data do not support the hypothesis that mobile phone use is related to an increased risk of brain tumors.^{86,127,138}

The lack of ionizing radiation and the low energy level emitted from cell phones and absorbed by human tissues make it unlikely that these devices cause cancer. The only health hazard of cell phones that has been confirmed is the increased risk of having an accident while driving and using a cell phone.³⁶ Likewise, a previous concern that living in close proximity to power lines was correlated to cancer has not been proved^{21,148} nor have reports linking VDTs to miscarriages been substantiated.⁸⁷

However, considerable evidence suggests that EMFs affect sleep and therefore affect mood, behavior, and cognitive abilities.¹⁴⁴ Exposure to EMFs has been suggested as the cause of a condition referred to as *electrosensitivity/screen dermatitis* in susceptible people using VDTs or artificial light. Cutaneous problems (e.g., itch, heat sensation, pain, erythema, papules, or pustules) and symptoms from internal organs (e.g., the heart) have been reported in association with EMFs.

From the results of recent studies, it is clear that EMFs affect mast cells releasing inflammatory substances, such as histamines, that result in these symptoms. Mast cells are also present in the heart tissue, and data from studies made on interactions of EMFs with cardiac function have demonstrated changes present in the heart after exposure to EMFs. However, the exact significance or cause of these changes remains unknown.⁵⁷

Vibration

Vibration is divided into two types: whole-body vibration (WBV) and hand-arm vibration (HAV). Truck, bus, and boat drivers; helicopter pilots; heavy equipment operators; miners; and others are at increased risk for WBV. Major clinical concerns of WBV exposure are chronic back pain and degenerative disk diseases, visual and vestibular changes, and circulatory and digestive system disorders.^{18,62,96} The risk for increased spinal loading and physiologic changes associated with WBV can be reduced by vibration damping, good ergonomic design, reducing exposure, and reducing other risks such as lifting.¹¹⁹

Vibration-induced white finger disease is the most common example of an occupational injury caused by vibration of the hands. This condition occurs secondary to the use of hand tools, such as power saws, grinders, sanders, pneumatic drills, and jackhammers, and other equipment used in construction, foundry work, machining, and mining. Interestingly, WBV is being used in a new neuromuscular training method for athletes called *vibration exercise* (VE) to increase the mechanical power output of muscles and improve neuromuscular efficiency; VE is also being investigated for the prevention and treatment of osteoporosis.^{23,95,124}

Heat Stress

Heat stress exceeding human tolerance can result in heat-related disorders (e.g., exertional heat stroke, exhaustion, cramps, dehydration, or prickly heat) and heat illnesses (e.g., chronic heat exhaustion, reduced heat tolerance, anhidrotic heat exhaustion, or exertional hyponatremia),

Table 4-1 Clinical Manifestations of Exertional Heat Illnesses

Heat Illness	Risk Event	Signs and Symptoms	Intervention
Dehydration	Fluids are not maintained or replenished.	Dry mouth, thirst, irritability, headache, dizziness, cramps, fatigue, decreased athletic performance.	Move to a cool environment, rehydration.
Heat cramps	Intense exercise, fluid deficiencies, electrolyte imbalance.	Intense muscle pain; prolonged muscle contraction.	Rehydration, replace sodium if needed, light activity (stretching, slow walking), massage, relaxation.
Heat syncope	Exposure to high environmental temperatures, first 5 days of acclimatization, prolonged standing.	Orthostatic dizziness, dizziness or fainting, tunnel vision, pallor, sweaty skin, decreased pulse rate.	Move to cool (shaded) area, adequate hydration, modified activity levels until acclimatized, elevate legs above level of heart, instruct person to increase venous return before standing (e.g., ankle pumps, arms over head, change positions slowly).
Heat exhaustion	Hot, humid conditions (e.g., indoor pool, sauna or hot tub, outdoor weather).	Unable to continue exercise or activity, unable to sustain cardiac output, loss of coordination, dizziness or fainting, profuse sweating, headache, nausea and vomiting, diarrhea, muscle cramps.	Move to cooler environment; remove excess clothing; recline and elevate legs above heart; cool with fans, ice towels, or ice bags; rehydration if possible (no nausea or vomiting).
Exertional heat stroke	Temperature regulation system is overwhelmed by excessive heat production or inhibited heat loss in challenging environmental conditions.	Elevated core body temperature (more than 40° C/104° F); tachycardia; hypotension; vomiting; sweating or dry skin; altered mental status, seizures, coma, death.	Whole body cooling (e.g., immersion), monitor body temperature recovery every 5 to 10 minutes, avoid overcooling, medical referral if physician is not on-site.

Data from Centers for Disease Control and Prevention (CDC): Extreme heat: a prevention guide to promote your personal health and safety. Available at www.bt.cdc.gov/disasters/extremeheat/heat_guide.asp. Accessed April 21, 2007.

some of which are fatal. Heat illness is more likely in hot, humid weather but can occur in the absence of hot and humid conditions.

More than 300 people die every year of heat-related illnesses, and many others require medical attention.³⁴ In a therapy setting, the groups of people most likely to experience heat stress include older adults during temperature extremes, industrial workers, construction workers, firefighters, outdoor sports participants, agricultural workers, pregnant women, and people taking mood-altering drugs (i.e., they lose touch with their environment).

Individuals receiving medications that interfere with salt and water balance are at increased risk for heat-related illness and death. Watch for diuretics, anticholinergic agents, and tranquilizers that impair sweating, as well as antidepressants, such as tricyclic antidepressants, that affect the body's ability to respond to temperature changes.

Other risk factors include obesity, age, mental illness, heart disease, poor circulation, prescription drug use, and alcohol and other drug use. People 65 years of age and older may not compensate for heat stress efficiently and are less likely to sense and respond to changes in temperature. Although older adults, the very young, and individuals with chronic diseases or mental health disorders are at greatest risk, anyone involved in strenuous physical activity during hot weather can be affected. Sudden

change in temperature is an additional risk factor to consider.

The signs and symptoms of exertional heat illnesses may vary from person to person but often include thirst, sweating, transient muscle cramps, fatigue, dizziness or lightheadedness, and dehydration (Table 4-1). Headache, nausea, loss of appetite, decreased urine output, chills, weakness, pallor, or cool and clammy skin may also occur, especially with exercise (heat) exhaustion.

Disorientation, staggering, seizures, loss of consciousness (coma), or emotional instability (even hysteria) occur with exertional heat stroke. Exertional hyponatremia is characterized by increased body-core temperature, low blood-sodium level, progressive headache, confusion, lethargy, significant mental compromise, seizures, swelling of the hands and feet, and even coma.¹⁹

High-Altitude

High-altitude environment (8000 to 14,000 feet) is characterized by atmosphere with decreasing partial pressure of oxygen and decreasing temperature. Hypoxia (reduced availability of oxygen to the body) appears to be the underlying cause of most of the physiologic changes of elevated altitude.

Acute altitude sickness includes *acute mountain sickness*, *high-altitude pulmonary edema*, and *high-altitude cerebral edema*. These three probably represent a continuum of

disease, but each has different symptom complexes, pathogenesis, and slightly different treatment interventions. With high-altitude pulmonary edema, fluid accumulates in the lungs when the arteries become constricted because of a lack of oxygen and the decrease in air pressure. Symptoms include fatigue; breathlessness at rest; fast, shallow breathing; cough that produces pink, frothy sputum; blue or gray lips or fingernails (cyanosis); chest tightness; and drowsiness.

High-altitude cerebral edema is brain swelling severe enough to interfere with brain function. The affected individual may experience confusion, inability to think or concentrate, confusion, and loss of physical coordination. Vision can become blurred if bleeding occurs from blood vessels at the back of the eye.

Not everyone gets sick at higher altitudes, but health risks increase the higher and faster one climbs, especially if early warning signs are ignored (e.g., headache, fatigue, dizziness or lightheadedness, nausea, or vomiting). People with cardiopulmonary and other diseases (e.g., sickle cell disease) are at increased risk for worsening of the medical disorder and possibly at increased risk for acute altitude illnesses with ascent to high altitudes. Aviation and aerospace illnesses are rarely encountered by the therapist and are beyond the scope of this book.

Risk Factors

Environmental pathogenesis requires an understanding of latency, the concept that a hazardous or toxic agent may initiate a series of internal reactions that do not manifest as overt disease for many years or even decades as the body strives to maintain a state of optimal health or homeostasis. Exposures to any of the agents discussed in the previous section on etiology are in fact risk factors. Many additional factors, such as route of exposure (e.g., inhalation, ingestion, or absorption through the skin); magnitude or concentration (dose) of exposure; duration (e.g., minutes, hours, days, lifetime); and frequency (e.g., seasonal, daily, weekly, or monthly), play into the development of progressive and overt disease.

Likewise, personal factors that vary from one person to another may affect pathogenesis and must be considered. These include age, gender, ethnicity, nutritional status, personal habits and lifestyle, genetic makeup and host susceptibility, and the strength of individual defense mechanisms. The host-agent-environment interactions are immensely complex and poorly understood at this time.

Pathogenesis

Once a hazardous substance is released into the environment, it may be transported and transformed in a variety of complex ways. For example, a chemical may be modified by the environment before entering the body; transformed by chemical or biochemical processes; or undergo vaporization, diffusion, dilution, or concentration by physical or biologic processes. Plants and animals may accumulate small doses of a chemical agent and bioconcentrate them to the degree that they become hazardous when consumed by humans.

All cells respond to a variety of different adverse environmental stimuli with a cellular defense response now commonly referred to as the *stress response*. Molecules released by the cells in response to stress (e.g., hyperthermic shock, radiation, toxins, or viral infections) are called *heat shock* or *stress proteins*. Increased levels of these proteins after a cellular injury from any of the environmental hazardous agents seem to act as molecular chaperones that facilitate the synthesis and assembly of new reparative proteins.

Cells that produce high levels of stress proteins seem better able to survive ischemic damage; stress proteins may be influential in certain immunologic responses and may also be a requirement for cells to recover from a metabolic insult. This finding may lead to further research investigating the role of pharmacology in raising the levels of stress proteins to provide additional protection to injured tissues and organs. This therapeutic approach could have other applications outside environmental medicine such as to reduce tissue damage from surgery-induced ischemia or to help protect isolated organs used for transplantation, which often experience ischemia and reperfusion injury.⁴⁹

Once some people are sensitized to chemicals, they develop increasingly severe reactions to more and more chemicals at smaller and smaller concentrations. The allergic response that occurs does not appear to be a typical response, perhaps suggesting altered immune system modulation. Immunologists have also discovered a possible connection between stress proteins and autoimmune disease, which may lead to preparations of specific protective vaccines.^{35,171}

Chronic exposure to air particulate matter leads to inflammation and oxidative stress, precursors to pulmonary and cardiovascular diseases and cancer.¹²³ Exposure to environmental pollutants has been linked with oxidative DNA damage in humans.¹⁴⁷ Exposures are genotoxic and interfere with DNA repair and inhibit the cellular apoptosis needed to prevent cancer. Bio-monitoring studies show that DNA damage is influenced by a variety of lifestyle and environmental exposures, including exercise, air pollution, sunlight, diet, and the chemical and physical agents discussed in this chapter.^{17,97,156}

Clinical Manifestations

An environmental illness may manifest in a variety of ways. The illness may present as a newly developed clinical syndrome or an aggravation or change in a preexisting condition. The EPA identifies the following seven categories of human health effects from hazardous exposures⁴⁶:

- *Carcinogenicity*: can cause cancer.
- *Heritable genetic and chromosomal mutations*: can cause mutations in genes and chromosomes that will be passed on to the next generation, such as caused by ionizing radiation.
- *Developmental toxicity*: can cause birth defects or miscarriages.
- *Reproductive toxicity*: can damage the ability of men and women to reproduce.

- Acute toxicity:** can cause death from even short-term exposure to the lungs, through the mouth, or the skin.
- Chronic toxicity:** can cause long-term damage other than cancer, such as liver, kidney, or lung damage.
- Neurotoxicity:** can harm the nervous system by affecting the brain, spinal cord, or nerves.

Local toxicities from exposure to environmental agents, such as ocular damage, mucous membrane complaints (eye, nose, and throat irritation), chemical burns to skin, noise-induced hearing loss, and vestibular disorders, can occur. Systemic toxicities can involve any organ system (Table 4-2). The clinical syndrome may mimic a wide range of psychiatric, metabolic, nutritional, inflammatory, and degenerative diseases.

Over the last 15 years, a new syndrome of environmental symptoms associated with chemicals, called *multiple chemical sensitivity* (MCS), has been observed both in the United States and in European countries. MCS is characterized by a chronic condition with symptoms that recur reproducibly in response to low levels of exposure to a wide variety of chemicals found in everyday substances, such as household cleaning agents, pesticides, fresh paint, new carpeting, synthetic building materials, newsprint, and perfume, and many other petrochemical products.

Symptoms occur in multiple organ systems and improve or resolve when irritants are removed.³⁷ Two to four times as many cases of MCS exist among Gulf War veterans compared with undeployed controls^{121,157} (see the section on Gulf War Syndrome in this chapter). Objective physical findings and consistent laboratory abnormalities or biomarkers associated with MCS are typically nonexistent, leading some of the medical community to call this condition *idiopathic environmental intolerance* (IEI), a psychosomatic or neuropsychiatric disorder. Reported symptoms range from runny nose to difficulty breathing and heart palpitations but also include fatigue, headaches, weakness, malaise, decreased attention/concentration, memory loss, disorientation, confusion, and mood changes.

The treatment focus of this philosophy is to overcome the affected individual's belief in a toxicogenic explanation for the symptoms,¹⁵⁴ whereas other health care professionals are calling for accurate diagnostic assessment, agreement on the use of specific questionnaires, clinical and technical diagnostic procedures, and prospective clinical studies of people with MCS, comparative groups, and experimental approaches.⁵ All in all, the concept of MCS has ignited considerable controversy in the fields of medicine, toxicology, immunology, allergy, psychology, and neuropsychology.⁸¹

Neurotoxicity

Of particular interest to the therapist may be the effects of hazardous or toxic agents on the nervous system. Neurologic symptoms are common presenting symptoms in people seen by occupational and environmental health professionals. Cognitive difficulties, headaches, fatigue, dizziness, and limb paresthesias are often experienced, but these are nonspecific and seldom point to a single disease or cause.

Table 4-2 Systemic Manifestations of Toxicity

Systems	Clinical Manifestations
Optic	Optic nephropathy, optic neuritis, optic atrophy
Integument	Atopic dermatitis Urticaria Pain, itching, erythema Pustules, papules Chemical burns
Cardiovascular	Cardiac arrhythmia Coronary artery disease Hypertension Myocardial injury Nonatheromatous ischemic heart disease Peripheral arterial occlusive disease
Respiratory system	Airway inflammation and hyperreactivity Bronchitis Asthma Hypersensitivity pneumonitis Pneumoconiosis Interstitial fibrosis Asbestosis Silicosis Granuloma formation Diffuse alveolar damage Cancer
Gastrointestinal (GI) tract	
Liver	Acute or subacute hepatocellular injury Cirrhosis Angiosarcoma Carcinoma Hepatitis
Kidney and urinary tract	Acute renal disease Chronic renal failure Tubulointerstitial nephritis Nephrotic syndrome Rapidly progressive glomerulonephritis Sensorimotor polyneuropathies (mild-to-severe weakness) Muscular fasciculations and weakness Reduced or absent reflexes Cranial neuropathy Prominent autonomic dysfunction
Central nervous system	Encephalopathy Cerebellar ataxia Aplastic anemia Hemolytic anemia Myelodysplastic syndromes Multiple myeloma Toxic thrombocytopenia Porphyria Allergic disease Allergic rhinitis Bronchial asthma GI allergy (food) Anaphylaxis Autoimmune diseases Neoplasia
Hematopoietic system	Menstrual disorders Altered fertility or infertility Spontaneous abortion or stillbirth Birth defects, low birth weight Cancer Reduced libido or impotence Altered or reduced sperm production Premature menopause
Immune system	
Reproductive system	

Many toxins manifest as a nonspecific syndrome of distal sensorimotor impairment that is indistinguishable from the neuropathy caused by common systemic diseases (e.g., diabetes mellitus, vitamin B₆ deficiency, alcoholism, or uremia). Toxins, such as lead, have a striking predilection for motor fibers and usually produce minimal sensory symptoms.

Neurologic symptoms that appear immediately after acute exposure are usually a result of the physiologic effects of the specific (usually chemical) agent. These symptoms subside with cessation of exposure and elimination of the compound from the body. By contrast, delayed neurologic disorders are generally a result of pathologic alterations of the nervous system.

Symptoms appear in a subacute manner over days or weeks after short-term exposure. In the case of long-term exposure, symptoms may appear insidiously and progress over many weeks or months. Recovery can be expected after cessation of exposure, but recovery is slow and depends on the extent of neuronal damage, the half-life of the chemical (i.e., continued exposure until the drug is out of the system), and the adverse effects of chelates used in the chemotherapy of metal poisoning.

Neurotoxicants do not cause focal (asymmetric) neurologic syndrome. Neurotoxins reach the nervous system by the systemic route and cause neurologic symptoms and deficits in a diffuse and symmetric manner, resulting in polyneuropathy. Any significant asymmetry in the presentation, such as weakness or numbness affecting one limb or one side of the body, is not likely to be attributed to neurotoxicity. Multiple neurologic syndromes are possible from a single toxin. Although the effects of neurotoxins are symmetric, neurons from different parts of the nervous system react differently to the agent.

Toxic polyneuropathy affects the distal limbs first, reflecting the greater vulnerability of the longest nerve axons. Sensory disturbances are usually reported as a tingling or burning sensation distributed in a stocking-and-glove pattern (see Fig. 39-5). The toes and the feet are affected first; hand symptoms are seldom present during the early stage. Involvement of the motor nerve fibers, if present, manifests first as atrophy and weakness of the intrinsic foot and hand muscles, bilaterally. More severe cases may present with footdrop or wristdrop, reflecting degeneration of motor axons to the lower leg and forearm muscles.

Neuropathic pain is commonly encountered in people with peripheral neuropathies regardless of the cause. In other words, pain patterns associated with chemically induced peripheral neuropathies do not differ significantly from the clinical picture of pain associated with neuropathy of other causes. Often this pain bears little relationship to the severity of neuropathy and may intensify during a period of recovery, or it may remit paradoxically as the neuropathy progresses, often with further loss of sensation. Pain is not a reliable indicator of neurologic progression or recovery.

MEDICAL MANAGEMENT

Clinical assessment may include assessing the details of exposure and correlating them with the medical condition. Various testing procedures may be developed on the

basis of the historical information provided by the client. The clinical presentation, environmental history, and results of laboratory tests assist the physician in demonstrating a correlation between exposure and the clinical manifestations. Nerve conduction velocity (NCV) studies and electromyography (EMG) are the primary tools for the laboratory evaluation of neuromuscular disorders. A toxic polyneuropathy is characterized by a diffuse and relatively symmetric pattern of NCV abnormalities.

Removal from exposure and decontamination of the exposed victim are essential in the treatment of exposure-linked toxicity. Specific intervention protocols depend on the agent involved (e.g., pesticide poisoning requires symptom-specific therapy such as IV anticonvulsants to halt a seizure; antihistamines are used for allergic reactions), the particular organ system involved, and the presenting pathologic condition.

SPECIAL IMPLICATIONS FOR THE THERAPIST

4-1

Environmental Medicine

PREFERRED PRACTICE PATTERNS

Various patterns may apply depending on the system(s) involved and the corresponding clinical manifestations (see Table 4-2).

5D: Impaired Motor Function and Sensory Integrity Associated With Nonprogressive Disorders of the Central Nervous System—acquired in adolescence or adulthood (hypoxia, vestibular disorders)

5G: Impaired Motor Function and Sensory Integrity Associated with Acute or Chronic Polyneuropathies

Environmental Hazards

Given the context of industrial, occupational, and environmental medicine and the single overriding factor of latency, health care professionals must view each client's health status holistically, as a composite of the individual's total life experience. Whenever symptoms present in the absence of a clearly identifiable history or cause, the client's past medical history must be carefully reviewed.

An environmental and occupational history includes dates of employment, a list of current and longest-held jobs, average hours worked per week, exposure to potential hazards in the workplace, common illnesses in coworkers, and personal protective equipment worn (or not worn) on the job.

Many healthcare providers use the mnemonic CH²OPD² (Community, Home, Hobbies, Occupation, Personal habits, Diet and Drugs) as a tool to identify an individual's history of exposures to potentially toxic environmental contaminants.⁹⁰ Specific questions for the therapist to ask are available.^{60,90} Any information elicited by the therapist but unknown to the physician must be documented and reported.

Each geographic area has its own specific environmental/occupational concerns. The therapist must find out about specific local exposures and community concerns. Overall, the chronic exposure to chemically based products and pesticides has escalated the inci-

Continued.

dence of environmental allergies and cases of multiple chemical sensitivity. Frequently, these conditions present in a physical therapy setting with nonspecific neuromusculoskeletal manifestations.

After the events of September 11, 2001, concern has increased about potential terrorist attacks involving the use of chemical agents, particularly the covert use of toxins (i.e., an unrecognized release in which the presence of signs and symptoms may be the first indicator of the event).

The therapist must be aware that chemically induced illness of this type may present as generalized muscle rigidity, peripheral neuropathy, or neurocognitive impairment as a result of neurotoxicity. Any unexplained muscle weakness and atrophy, sensory loss, depressed or absent deep tendon reflexes, memory loss, delirium, ataxia, or global change in muscle tone should raise a red flag for possible chemical etiology.¹¹⁶

Air Pollution

Vigorous exercise outdoors, which increases the dose of pollution delivered to the respiratory tract, should be avoided during periods of ambient air pollution.^{53,129} Health care providers can reasonably advise all clients, especially anyone with respiratory disorders, as well as athletes in training,¹²² to stay indoors during pollution episodes.

Respiratory protective equipment (RPE) has been developed for use in the workplace to minimize exposure to toxic gases and airborne particles. Many of these devices, particularly those likely to be most effective, add to the work of breathing and are not well tolerated by some people, especially those with respiratory disease. Much remains unknown about the efficacy of RPE, and concerns have been raised about the risk of dangerous carbon dioxide accumulation within the device, proper fit and inward leakage, resistance to airflow as the filter load increases, and individual breathing rates and filter replacement schedules. Research to answer these questions is necessary before specific recommendations can be made for the general population, as well as for individuals with known respiratory disease.

Studies have shown that high efficiency-filter air cleaners improve airway hyperresponsiveness and decrease peak flow amplitude in people with allergic asthma (studies to date have centered on children) who are sensitized and exposed to pets.¹⁶⁶ Future studies are needed to develop biologic markers to identify more accurately people who have a clinical improvement after allergen reduction.

Carbon Monoxide

Anyone with lung injury or reduced lung capacity may have a reduced ability to diffuse carbon monoxide when it is encountered. Individuals with low lung volumes for any reason (e.g., restrictive lung disease, sickle cell anemia, or lobectomy) who try to exercise may be at risk for CO poisoning under conditions a healthy individual would be able to tolerate.⁷⁴

The main symptoms of CO poisoning are dizziness, headache, nausea, weakness, and tachypnea, followed at higher amounts by loss of consciousness, coma,

convulsions, and death. As CO binds to hemoglobin to form carboxyhemoglobin, the reduced capacity of the blood to deliver oxygen to the tissues results in increased frequency of CHD and arrhythmias and stresses the immune system.^{59,70,143}

Acute myonecrosis (death of individual muscle fibers) has been associated with CO poisoning. Clinical studies of people with heart disease have been carried out to evaluate the effects of CO exposure on exercise capacity. During exercise, persons with coronary artery disease experience a decreased time to occurrence of myocardial ischemia when exposed to CO compared to healthy subjects.^{2,4}

Neurologic recovery in people with mild-to-moderate CO poisoning is good. The prognosis after severe poisoning is variable and correlates with the extent and duration of the insult. Short-term memory impairment, depression, and syndromes related to lesions of the basal ganglia are well known. A syndrome of delayed neurologic deterioration occurs in about 10% of victims of serious CO intoxication. Risk factors for the delayed syndrome include age older than 40 years, prolonged exposure, and abnormalities of the brain on computed tomography (CT).¹¹⁷

In a small study of 16 healthy ice hockey players exposed to CO from a faulty ice resurfacer, chronic cough and dyspnea persisted 6 months after the exposure. Conventional pulmonary function tests did not reveal airway abnormalities but impulse oscillometry (IOS) showed evidence of increased airway resistance and small-airway disease, which correlated with players' symptoms and reduced the players' ability to play effectively under exertion.⁷⁹

Lead

The brain is the target of lead toxicity in children, but adults usually present with manifestations of peripheral neuropathy. Typically, the radial and peroneal nerves are affected, resulting in wristdrop and footdrop, respectively. Anyone presenting with vague or nonspecific symptoms of myalgias, paresthesias, arthralgias accompanied by fatigue, irritability, lethargy, abdominal discomfort, poor concentration, headaches, tremors, and known risk factors may be suffering from lead poisoning.

Pica (compulsive chewing on nonnutritive objects such as dirt, paint, plaster, or clay) observed in children may be associated with lead toxicity and must be evaluated. Lead anemia and lead nephropathy may also occur (see the section on Neurotoxicity in this chapter.) For more information, contact the National Lead Information Center Clearinghouse at (800) 424-5323.

Vibration

Tools can be modified to reduce some of the dangerous levels of vibration. Grip kits provide grips that can be applied easily to any type of tool and dampening products made of Sorbothane to reduce shock and vibration.

Heat Stress

Even with a heat-illness prevention plan that includes medical screening, acclimatization, conditioning,

environmental monitoring, and suitable practice adjustments for the athlete, heat illness can and does occur. Monitoring vital signs in anyone at risk will help identify early signs of heat exhaustion (e.g., weak, rapid pulse; elevated body temperature; shallow and fast breathing; or changes in blood pressure).

Observe for or ask about heat rash, a red cluster of pimples or small blisters on the neck and upper chest, in the groin, under the breasts, and/or in the elbow creases. The therapist must be prepared to respond quickly to alleviate symptoms and minimize morbidity and mortality.¹⁹

Exercise-associated muscle (heat) cramps represent a condition that presents during or after intense exercise sessions as an acute, painful, involuntary muscle contraction. Muscle cramps and distal extremity edema, dehydration, and electrolyte imbalance are the most commonly observed phenomena associated with heat stress in a therapy practice. The implications surrounding these adverse effects are discussed fully in Chapter 5.

For athletes with spinal cord injuries, regulating heart rate, circulating blood volume, production of sweat, and transferring heat to the surface varies with the level and severity of the spinal cord lesion. The therapist must monitor these athletes closely for heat-related problems and be prepared to provide more fluids, lighter clothing, or cooling of the trunk, legs, and head.¹⁹

Individuals who experience heat stroke may have compromised heat tolerance for up to a year or more. For an athlete, this can affect training and competition. Gradual return to sports is advised with close monitoring during exercise. Older athletes have a decreased ability to maintain an adequate plasma volume during exercise, which may put them at risk for dehydration. Regular fluid intake is essential to avoid hyperthermia. The older athlete may need cardiovascular stress testing before participating in sports or strenuous activities in hot environments.¹⁹

The National Athletic Trainers' Association has published a position statement on exertional heat illnesses with helpful guidelines and recommendations for anyone working with athletes.¹⁹ Representatives from 18 leading medical, nutritional, and sports medicine-related organizations have formed the Inter-Association Task Force on Exertional Heat Illnesses with additional helpful information.¹⁰⁴

High Altitude

Many issues are related to altitude change (e.g., effects on fetal size and development, ultraviolet intensity with increases in altitude, sympathetic nervous system changes during acclimatization, air pollution at higher elevations, or physiologic changes and pathologic conditions occurring in military and aerospace personnel) that are being researched and reported in the literature. These are beyond the scope of this text. Implications here are confined to the more common issues in a therapy practice related to exercise capacity.

Chronic exposure to high altitude is known to result in changes in the mechanisms regulating oxygen delivery to the contracting muscles, but the underlying

cause of changes in exercise capacity associated with high altitude is not completely understood.⁶¹

The primary effect of altitude on exercise capacity is through effects on the cardiovascular system, with a decrease in maximum oxygen consumption ($VO_{2\max}$) and a decrease in maximum heart rate. Studies of oxygen saturation during submaximal exercise in natives of high-altitude areas compared with individuals born at sea level and acclimated to high altitudes suggest that oxygen saturation during exercise may be influenced by adaptation during growth and development and larger lung volume and pulmonary diffusion capacity for oxygen in the native high-altitude population.²⁶

With continued exposure to increased altitude, exercise capacity does seem to improve, but never reaches that which is attained by the native population at sea level.¹³³ People with congestive heart failure or coronary artery disease are more likely to be symptomatic at high altitudes. Those with either of these conditions are likely to experience reduced exercise capacity.^{75,101}

Mild sensory neuropathy may also occur at high altitudes, both as part of the burning feet/burning hands syndrome (distal limb burning and tingling paresthesias) associated with chronic mountain sickness and as a separate entity among control groups studied. This condition resolves with low-altitude sojourn (even for high-altitude natives), suggesting that a mechanism of altered axonal transport may be involved. Additionally, reduced thickness of microvessels observed implies that adaptive structural changes to hypobaric hypoxia may also occur in peripheral nerves and are similar to those reported in other tissues of high-altitude natives.¹⁵⁸

Neurotoxicity

Litigation and other potential sources of secondary gains often complicate environmental or occupational exposures that result in neurologic disorders. Psychologic factors may have profound effects on the client's perception of neurologic symptoms, even in those people with genuine organic disease. Emotional issues must be recognized and addressed throughout the rehabilitation process.

Coasting is the phenomenon of continuing clinical progression of neurologic deficits after removal of the offending toxin. Weakness or sensory deficits of these neuropathies often worsen for as long as 4 to 5 months after cessation of exposure, reflecting the delayed neuronal death or degeneration induced by the toxin.

Neurologic recovery is facilitated by the plasticity of the nervous system (i.e., its ability to adapt to injury). Peripheral sensory and motor nerve fibers have a remarkable capacity to regenerate after removal of the neurotoxin. Although the neurons in the CNS lack the ability to multiply, surviving neurons may eventually take over the function of degenerated neurons and partially restore neurologic function. Physical and occupational therapy is beneficial during the recovery time to facilitate this process. When given sufficient time (18 to 24 months), partial clinical improvement is demonstrable in the majority of cases.

OCCUPATIONAL INJURIES AND DISEASES

Overview

Each year, millions of the estimated 140 million U.S. workers are injured on the job or become ill from exposure to hazards at work. These work-related injuries and illnesses result in substantial human and economic costs for workers, employers, and society; estimated direct and indirect costs of work-related injuries and illnesses are approximately \$170 billion each year.¹⁵⁹

Data collected through a National Electronic Injury Surveillance System (NEISS) report an estimated 3.4 million nonfatal injuries and illnesses among workers of all ages. More than three-fourths of all nonfatal workplace injuries/illnesses were attributed to contact with objects or equipment (e.g., being struck by a falling tool or caught in machinery), sprain or strain, and falls. Male workers under the age of 25 have the highest rate of workplace injuries.⁴²

Approximately half of all injuries are sprains, strains, lacerations, amputations, punctures, and avulsions. Most sprains and strains affect the trunk (shoulder, back, chest, or abdomen) and lower extremities. The majority of lacerations, punctures, amputations, and avulsions affect the upper extremities. Dislocations and fractures account for approximately 7% of injuries and were attributed most often to falls.⁴²

The lifetime cost of all injuries (including occupational and others) occurring in a single year in the U.S. totals an estimated \$406 billion in medical expenses and productivity losses, including wages, fringe benefits, and the ability to perform normal household duties. The actual cost of these injuries is likely much higher when other related costs not included in the analysis are considered (e.g., police services, caregiver time, pain and suffering, decreased quality of life, or nonmedical expenditures such as wheelchair ramps or hand controls for vehicles).⁵¹

Computers and other time-saving devices have resulted in less physically demanding jobs, but new physical challenges and risk of impairments occur from incorrect ergonomics and prolonged (static) postures and positions, as well as repetitive motions. The prevalence of computers in modern society's workplace and leisure activities has also contributed to the increase in the "weekend warrior syndrome," or injuries to sedentary workers who go out on a weekend (or on an occasional basis during free time) and participate in sports or other strenuous physical activities. Overuse injuries and muscle strains are common, especially in the middle-aged and older adult. Activities, such as gardening, hiking, or household repairs, can be more strenuous than they seem in these age groups.⁷⁷

Faster travel for business or pleasure in smaller spaces for long periods of time may also be contributing to an increase in injuries, deep vein thrombosis (DVT), or neck and back strain. Some individuals are at increased risk for these problems. For example, people are more prone to DVT if they have had a previous history of DVT, stroke, heart disease, or cancer. Anyone who has a neurologic

disorder, lower extremity impairment, or mobility impairments may be at risk for DVT under these circumstances.⁷⁷

Healthy People 2010 continues to maintain and work toward the goal of preventing injuries and illnesses by identifying risk factors, providing education, and developing effective safety strategies, including public health surveillance programs. Data collected and reported from death certificates, cancer registries, and hospital discharge data are occupational health indicators (OHI) used to identify risk factors and populations at risk. Worker's compensation programs, the Bureau of Labor Statistics, and the U.S. Census provide additional information used in statistical analysis.

Risk Factors for Occupational Injury

Risk factors for musculoskeletal occupational injury have been identified by OSHA. If workers are exposed to two or more of these factors (Box 4-2) during their shift, they are at increased risk and require preventive intervention. Additionally, in April 2000, Congress adopted the Senior Citizens' Freedom to Work Act that allows retired seniors to continue working without losing their Social Security benefits. The growing silver collar work force (adults of the baby boomer generation working past the age of 65) may represent a unique risk factor, since aging is associated with a progressive decrement in various components of physical work capacity, including aerobic power and capacity, muscular strength and endurance, flexibility and coordination, and the tolerance of thermal stress.¹⁴² Aging may thus contribute to additional workplace injuries and accidents.

Other risk factors in the general population may include psychosocial stress, gender, and personality. For example, psychosocial stress increases the physical demands of lifting for people with certain personality traits, making those people more susceptible to spine-loading increases and suspected low-back disorder risk.⁸⁹

Obesity may also be a co-risk factor for the development of occupational asthma and cardiovascular disease that may modify the worker's response to occupational stress, immune response to chemical exposures, and risk of disease from occupational neurotoxins. The interrelationship of work, obesity, and occupational safety and health is under further investigation.¹³⁷

Ergonomics

Derived from the Greek terms *ergon*, meaning work, and *nomos*, meaning law, *ergonomics* is the study of work and of the relationship between humans and their working and physical environment. Over the last two decades, ergonomics has become a branch of industrial engineering that seeks to maximize productivity by minimizing worker discomfort and fatigue. Ergonomics is the science of fitting the task or the job to the worker.

Ergonomics is an interdisciplinary field of study that integrates engineering, medicine, and physical and behavioral management sciences and addresses issues arising from the interaction of humans in an increasingly technologic society. As a field of study, ergonomics deals with

Box 4-2**RISK FACTORS FOR OCCUPATIONAL INJURY****Worker Characteristics**

- Age
- Psychosocial stress
- Gender
- Personality
- Physical fitness, including aerobic capacity, endurance, strength, flexibility, range of motion
- Health status, including lifestyle and presence of pregnancy or disease(s) such as chronic fatigue, fibromyalgia, Raynaud's, diabetes, arthritis, coronary artery disease
- Individual anatomy and physiology (e.g., body capacity versus job requirements, tissue resilience, functional reach)
- Work experience and training

Occupational Risk Groups

- Manufacturing (e.g., assembly line work, meat packing, automobile plants)
- Health care workers, especially in hospitals, nursing and personal care facilities
- Lumber and building material retailing
- Trucking (over the road) and ground couriers (e.g., United Parcel Service, Federal Express)
- Sawmills, planing mills, millwork
- Construction
- Computer operators (keyboarding)
- Crude petroleum and natural gas extraction
- Retail store clerks and cashiers, especially grocery stores
- Musicians
- Agriculture production
- Beauty salons

Worksite Factors

- Lighting, temperature, noise
- Poor workstation ergonomics
- Poor ergonomic practices; inadequate injury prevention training
- Vibration
- Overtime, irregular shifts, length of workday; recovery time between shifts
- Infrequent or no breaks during work shift
- Continuing to work when injured or hurt (voluntarily or involuntarily)

Task-Specific Factors

- Performance of the same motions or motion pattern every few seconds for more than 2 hours at a time (repetition)
- Fixed or awkward work postures for more than a total of 2 hours (e.g., overhead work, twisted or bent back, bent wrist, kneeling, stooping, or squatting)
- Use of vibrating or impact tools or equipment for more than a total of 2 hours
- Unassisted manual lifting, lowering, or carrying of anything weighing more than 25 lbs more than once during the work shift
- Piece rate or machine-paced work for more than 4 hours at a time
- Using hands/arms instead of available tool(s)
- Improper positioning or use of tools
- Static or awkward postures
- Contact stress (placing the body against a hard or sharp edge)
- Computer keyboard usage more than 15 hours/week

For the Health Care Worker*

- Performing manual orthopedic techniques
- Assisting clients during gait activities
- Working with confused or agitated clients
- Unanticipated sudden movements or falls by client
- Treating a large number of clients in 1 day
- Rehab, acute care, long-term care facilities
- Working with TBI, SCI, stroke individuals (high physical demands)

*Adapted from Cromie JE, Robertson VJ, Best MO: Work-related musculoskeletal disorders in physical therapists: prevalence, severity, risks, and responses, *Phys Ther* 80(4):336-351, 2000.

TBI, Traumatic brain injury; SCI, spinal cord injury.

job design, work performance, health and safety, stress, posture, body mechanics, biomechanics, anthropometry (measurement of body size, weight, and proportions in relation to the task requirements), manual material handling, equipment design, quality control, environment, workers' education and training, and employment testing.

The goal is to provide an environment that allows the individual to adequately absorb and dissipate forces placed on the body. Fitting the work to the worker makes it possible to enhance productivity while controlling errors and reducing musculoskeletal strain and fatigue. Ergonomics reduces risk factors known to contribute to occupational ergonomic-related injuries.

Humans have limitations arising from factors such as gender differences; differences in size, weight, and body proportions; aging; physical fitness and lifestyle choices; diet; stress; and pain and injury. Our abilities (and limitations), combined with the necessary acquired skills, determine how well we perform our daily tasks. Ergonomics helps people recognize their abilities and limitations for safe and effective performance within the environment. Work environments are often designed without adequate consideration for the people who will use them. Inadequate workplace design can contribute to stress, injury, pain, job-related impairments, disabilities, and subsequently, lost productivity. If products are designed without considering the human factor, health and safety hazards can occur.

A substantial body of validated scientific research and other evidence (epidemiologic, biomechanical, pathophysiological studies) support the positive outcomes of ergonomic programs.^{103,107} The evidence strongly supports two basic conclusions: (1) a consistent relationship exists between musculoskeletal disorders and certain workplace factors, especially at higher exposure levels; and (2) specific ergonomic interventions (e.g., proper equipment, postural education, and use of correct body mechanics) can reduce these injuries and illnesses.

A new branch of ergonomists, *rehabilitation ergonomists*, are health care professionals who, in addition to functioning as an ergonomist practitioner, also use knowledge of the relationship between pathology and work to match the demands of the job to the capacity of the worker. Rehabilitation ergonomists work with people

who do not fit the normal standards but require modification to safely and productively perform their job or task. Concentrating on improved safety focuses on physiologic improvement, which in turn increases productivity.

Ergonomic Certification

Certification as an ergonomist practitioner is available through two national boards: the Board of Certified Professional Ergonomists (BCPE) and the Oxford Research Institute (ORI). The ORI and BCPE are nonprofit board certification programs that are nationally and internationally recognized. Competency is demonstrated through experience, work samples, and a passing grade on a board examination. Previously, board certification in professional ergonomics accredited engineering ergonomists through a certification examination. Today, psychologists, therapists, and others have joined engineers in the pursuit of ergonomics as a career.

A wide range of private certification programs are available to the health care professional seeking training and certification as an ergonomist. The Occupational Injury Prevention and Rehabilitation Society (OIPRS) supports the accreditation of therapists through the BCPE and ORI but recognizes other programs that meet the minimum criteria for certification as an ergonomist. These criteria and a listing of ergonomic certification options for therapists are available.^{13,68} The orthopedic section of the American Physical Therapy Association (APTA) also has an occupational health special-interest group (SIG) with plans for credentialing occupational health physical therapists (Certification as an Occupational Health Specialist).

Occupational Injuries

The most common occupational injuries, referred to as *musculoskeletal disorders* (MSDs), involve cumulative trauma disorders caused by prolonged static positioning while using force (e.g., exerting constant force with the thumb pressed in while holding a computer mouse or constant gripping of tools or handles) and forceful repetition of work (repetitive strain injury) while using incorrect muscle groups or posture (e.g., keyboarding, meat cutting, or repetitive lifting and turning). The use of the term *strain* may be a misnomer because the symptoms occur in response to static muscle overload or maintenance of constrained postures rather than repetitive or dynamic muscle load.

Back injuries account for 60% of all work-related cases, and upper extremity injuries account for a majority of the remaining percentage. Seventy percent of the repeated trauma cases are in manufacturing industries.²⁷ The shift in the U. S. economy to service industries, such as nursing homes and other long-term care facilities, in which staff members are required to perform heavy lifting, has contributed significantly to the number of back injuries.⁸³ Other commonly sustained workplace injuries include eye injuries, hearing impairment, fractures, amputations, and lacerations severe enough to require medical intervention.

Musculoskeletal Disorders

MSDs encompass both cumulative trauma disorders (CTDs) and repetitive strain injuries (RSIs) and are more accepted terminology in the fields of ergonomics and occupational medicine, although the use of CTD and RSI is still often used in the literature. *Work-related MSDs* (WMSDs) are defined as an injury or disorder of the muscles, tendons, ligaments, cartilage, or spinal disks as diagnosed by a health care professional, resulting in a positive physical finding sufficient to require medical intervention and/or days away from work or assignment (i.e., an "OSHA-recordable" injury).¹¹³

MSDs do not include injuries resulting from slips, trips, falls, or accidents. The disorder must be directly related to the employee's job and specifically connected to activities that form the core or a significant part of the job (e.g., a poultry processor might report tendinitis, but a back injury while occasionally changing the water bottle would not be covered).¹¹³

WMSDs account for more than one-third of all occupational injuries that are serious enough to result in days away from work. Back injuries and carpal tunnel syndrome (CTS) are the most prevalent, most expensive, and most preventable MSDs. Each year more than 100,000 women experience work-related back injuries that cause them to miss work. It is estimated that 300,000 injuries and \$9 billion in worker's compensation can be saved with improved industry safety and ergonomic practices.

Etiology and Risk Factors

Risk factors for MSDs are divided into four major categories: genetic, morphologic, biomechanical, and psychosocial. Among the various biomechanical risk factors, exposure to repetitive, static, and vibratory activities is known to result in MSDs.⁸⁰ Differences in physical, occupational, and physiologic factors may contribute to MSDs. For example, CTS is associated with pregnancy, as well as rheumatoid arthritis, which is a condition that affects women more often than men. Women comprise 70% of the CTS cases and 62% of tendinitis cases that are serious enough to warrant time off work.¹¹²

CTS accounts for more days away from work than any other workplace injury. In addition to workers who spend hours at the computer, CTS has been reported in meat packers, assembly line workers, jackhammer operators, athletes, physical and occupational therapists, and home-makers. In both genders, CTS can be associated with other medical conditions, such as thyroid problems, liver disease, multiple myeloma, and diabetes, as well as with other musculoskeletal disorders that may or may not be work-related (see Box 39-1). For all work-related CTS, poor worksite design, poor posture and body mechanics, and industrial equipment and computers that take out the automatic pauses of work must be evaluated as possible contributors. An in-depth discussion of CTS is included in Chapter 39.

Pathogenesis

The exact pathomechanisms of MSDs remain obscured because of the difficulty of analyzing tissues of individu-

als in the early stages of work-related MSDs. Tissue injury caused by repeated motion may involve an inflammatory response, but why one worker develops symptoms while others doing the same task do not remains unknown and a topic of discussion and study. The relationship between repetition rate, forcefulness of tasks, cellular responses to these activities, number of strains, and inflammatory response is under investigation. The role of genetics and psychosocial factors is also being considered.^{9,10,153}

It is likely that the development of MSDs is multifactorial, with variations in individual tissue tolerances. Each individual may have his or her own threshold below which tissue integrity is preserved and above which injury results. It is possible that the combined and/or accumulative effects of risk factors for MSD can exceed tissue tolerance capacity and cause injury. When continued task performance is superimposed on injured and inflamed tissues, a cycle of injury, inflammation, and motor dysfunction occurs.^{9,10}

MSDs in the lumbar spine may have a different mechanism. Static lumbar loading applied to ligaments results in creep (e.g., stretch of viscoelastic tissue over time that is not fully restored immediately after load removal). In theory, ligaments that remain stretched beyond their resting length may result in increased laxity of intervertebral joints and risk of instability and injury. In the spine, ligaments have a secondary role in maintaining intervertebral stability.¹⁵⁰

Static lumbar flexion under constant load results in long-lasting viscoelastic creep that does not fully recover after 7 hours of rest. The creep developed gives rise to a neuromuscular disorder with reduced reflexive muscle activity, muscle spasms during flexion, and hyperexcitability of muscle activity during rest that may last 24 hours or more. The viscoelastic creep and associated neuromuscular disorder can occur even with low loads, which may help explain how cumulative low back problems develop.¹⁵⁰

Clinical Manifestations

Workers suffering from MSDs, especially upper extremity MSDs (UEMSDs), may experience decreased grip strength and range of motion, impaired muscle function, and inability to complete activities of daily living (ADLs). Symptoms are persistent (although intermittent, they return and progress over time) and most commonly include pain (e.g., headache, neck, back, shoulder, wrist, hip, or knee); burning sensation, numbness, and/or tingling (hands or feet); Raynaud's phenomenon; and myalgias and arthralgias with spasm, stiffness, swelling, or inflammation.

Neural tissues at the cervical spine, carpal tunnel, cubital tunnel, or thoracic outlet can be compressed as a result of the swelling associated with the biomechanical microtrauma. The individual may perceive weakness and drop objects or have difficulty with handwriting. Common MSDs/UEMSDs are listed in Box 4-3.

A predictable sequence of events leads up to MSDs of a repetitive nature or those caused by static postures (e.g., some tasks such as prolonged writing or typing at a keyboard require co-contraction of the agonists and

Box 4-3

COMMON WORK-RELATED MUSCULOSKELETAL DISORDERS

- Carpal tunnel syndrome
- Carpet layers' knee
- Cubital tunnel syndrome
- de Quervain's disease
- Epicondylitis (medial or lateral tennis elbow)
- Focal hand dystonia
- Hand-arm vibration syndrome
- Herniated spinal disk
- Pronator syndrome
- Radial tunnel syndrome
- Raynaud's phenomenon
- Rotator cuff syndrome
- Sciatica
- Tendinitis (shoulder, elbow, wrist)
- Tenosynovitis (finger flexors or extensors; trigger finger)
- Tension neck syndrome, thoracic outlet syndrome, cervical radiculopathy
- Ulnar nerve syndrome

antagonists). Fatigue and the inability to recover from fatigue brought on by additional hours and pressured deadlines, combined with emotional stress and improper posture, improper use of tools, or an ergonomically inadequate workstation, result in muscle soreness.

Over time and without intervention or a change in the contributing factors, the body strains to keep up and pain develops, followed by injury or trauma. In the case of tendinitis or focal hand dystonia, it is possible that a sensory problem rather than just a motor problem occurs and is caused by a dysfunction in cortical sensory processing.²⁹ Evidence suggests that aggressive sensory discriminative training complemented by traditional exercises to facilitate musculoskeletal health can improve sensory processing and motor control.²⁸

SPECIAL IMPLICATIONS FOR THE THERAPIST

4-2

Occupational Injuries and Diseases

PREFERRED PRACTICE PATTERNS

4B: Impaired Posture

4C: Impaired Muscle Performance

4E: Impaired Joint Mobility, Motor Function, Muscle Performance, and Range of Motion Associated With Localized Inflammation

4F: Impaired Joint Mobility, Motor Function, Muscle Performance, Range of Motion, and Reflex Integrity Associated with Spinal Disorders (disk herniation, nerve root compression, synovitis, and tenosynovitis)

4G: Impaired Joint Mobility, Muscle Performance, and Range of Motion Associated With Fracture (Patterns 4H, 4I, and 4J may also apply depending on the outcome of the injury.)

Continued.

5F: Impaired Peripheral Nerve Integrity and Muscle Performance Associated with Peripheral Nerve Injury

5H: Impaired Motor Function, Peripheral Nerve Integrity, and Sensory Integrity Associated with Nonprogressive Disorders of the Spinal Cord (nerve root compression caused by lumbar radiculopathy; orthopedic or spinal instability)

OSHA has put into place a comprehensive plan designed to reduce ergonomic injuries through the development of guidelines, enforcement measures, workplace outreach, and research. OSHA has a targeting program that includes the approximately 2500 nursing or personal care facilities that reported injury and illness rates of eight or higher.

Under a new National Emphasis Program, OSHA will inspect approximately 1000 of those facilities focusing on specific hazards that account for the majority of nursing home staff injuries and illnesses. Those hazards include ergonomics (primarily back injuries from patient handling); bloodborne pathogens/tuberculosis; and slips, trips, and falls.¹¹¹

Therapists can have an important role in the development of health and safety programs that will accurately assess hazards in the workplace and reduce the risk of musculoskeletal injuries, amputations, and illnesses. For all clients with MSDs, questions related to occupation and exposure to toxins, such as chemicals or gases, are included because well-defined physical (e.g., cumulative trauma disorder) and health problems occur in people engaging in specific occupations.¹⁰⁸ For example, pesticide exposure is common among agricultural workers, who may also experience musculoskeletal problems from repetitive loading. Asthma and sick building syndrome are reported among office workers.

Ergonomics

The therapist can have a significant role in the prevention (e.g., worksite analysis and workstation redesign) and rehabilitation of occupational injuries. The role of ergonomics in injury management includes a prompt and safe return to work, cost savings, and prevention of injuries or reduction of injury progression or recurrence. There is evidence that workstation exercises can reduce musculoskeletal discomfort for workers sitting in front of a computer or other VDT. The therapist can devise easy-to-do, appropriate workstation exercises for individuals at risk for postural immobility and the resultant musculoskeletal discomfort.⁵⁰

With the increased and prolonged use of computers at home, school, and work, UEMSDs are becoming more prevalent. It is estimated that some individuals may use up to 100,000 keystrokes in an average day. Computer keyboard usage greater than 15 hours per week can contribute to MSDs affecting the upper extremity, shoulder, neck, and low back. The therapist can offer guidance to workers regarding keyboard workstation designs and features.⁸⁸

When conducting a job analysis, the therapist evaluates job duties and environmental factors that put physical stress on the worker; stressors most typically

include force (any weight that is lifted, pushed, or carried), repetition, and posture. The therapist will assess the amount of force needed to produce the necessary work, the number of repetitions, and the postural tolerances required by the job.

These variables are evaluated for both newly developing programs or job tasks and in industrial rehabilitation programs for cases of work conditioning and work hardening, a fairly recent innovation in rehabilitation specifically geared toward reemployment for previously injured or impaired workers. Unlike conventional programs, work conditioning/hardening does not focus on goals such as symptom reduction or increased physical capacity. Through graded work simulations conducted in a realistic industrial or office setting, injured people rebuild physical and psychologic fitness to work.⁹²

Quantifying the requirements for each job is essential in both prevention and return-to-work situations. Therapists can provide analysis and management of injury-related job hazards, injury prevention training, examination/evaluation management of MSDs, development of job/task alterations, and return-to-work program planning.⁸ Specific ways to prevent WMSDs are available.¹¹³ The APTA offers the following *Occupational Health Guidelines*:

- Evaluating functional capacity
- Physical therapist management of the acutely injured worker
- The physical therapist in occupational health
- Work conditioning and work hardening programs
- Work-related injury/illness prevention and ergonomics

Silver Collar Workers

Therapists also need to modify traditional intervention strategies for prevention and treatment of injuries in the silver collar work force previously mentioned. Although older workers may have lower injury rates than younger workers, their injuries are likely to be more severe with a longer recovery time.

Therapists can assist industries and job sites to adapt job duties to accommodate for age-related conditions such as reduced muscle strength and motion. Providing ergonomically correct worksites and work areas, implementing diagnostic and training programs to prevent specific conditions (e.g., CTS, tendinitis, or back injuries), and instituting wellness programs to include home- or gym-based exercise programs and organized stretch/walk breaks will help keep all employees, particularly seniors, in good health and injury free.¹⁴

There is a need for the development of distinct fall prevention programs specific to the older population in the workforce. Efforts should be made to minimize fall hazards in all occupational sectors. More attention must be paid to the sensory impairments of the older worker, especially in vision and hearing, with job modifications as needed. Motor learning theory suggests that older adults may need different types of safety training. Emphasizing task analysis and repeated practice may be needed in this population.⁵⁸

Physical Therapists with Work-Related Symptoms

Interventions employed by therapists can lead to WMSDs among themselves although little is known about this segment of the population. A summary of prevalence, severity, risks, and responses associated with MSDs in physical therapists suggests that therapists at greatest risk are more inexperienced therapists who may not know their limits (more than 50% have their first episode as a student or in their first 5 years of practice), those in neurology and rehabilitation, and those performing manual orthopedic techniques.³⁹

Researchers have demonstrated that knowledge of ergonomics, injury, and intervention strategies is not associated with a reduced risk of injury among therapists.^{39,71} For example, maintaining good body mechanics is not always protective when a client is starting to fall and pulls the therapist down. Lifting with sudden maximal effort, bending and twisting, repetitious movement, awkward postures maintained for a prolonged period of time, and using high levels of force are correlated with work-related injuries among therapists.^{22,71}

Therapists working in rehabilitation units with patients who have brain injuries, stroke, and spinal cord injuries with high physical demands are at increased risk of work-related injuries. Clients in long-term care facilities, skilled nursing facilities, or acute care who are immobile often put the greatest demands on therapists.¹⁶⁸

Other risk factors include heavy client loads, working with combative clients, increased number of hours performing manual therapy, and injuries that occur outside the workplace that are not treated or healed before returning to work. The cultural context in which therapists work might contribute to WMSDs. For example, the need to demonstrate hard work and care for patients/clients along with the need to appear knowledgeable and skilled by remaining injury-free may increase risk for therapists. The therapist may put the needs of the patient/client first, subsequently suffering an injury. They fail to report the injury to avoid being perceived as incompetent. There is also a tendency to try and manage their own condition, which can lead to delays in recovery.³⁸

Therapists are encouraged to maintain good body mechanics, change position often, ask for help, and report injuries when they occur. Using mechanical aids such as a lift is advised whenever available. The therapist should seek care and modify work or take time off when necessary. It is not a good idea to try to work through the injury.¹⁶⁸

Understanding risk factors, identifying what causes injury, and changing behaviors are the keys to preventing injuries to therapists in the workplace setting. Therapists frequently believe their knowledge of physical therapy and skills will prevent WMSDs from occurring. Further research is needed to identify aspects of therapy practice that place the therapist at greatest risk and ways to reduce that risk.¹⁶⁸

Table 4-3 Types of Fire-and-Rescue–Related Acute and Chronic Injury

Acute	Chronic
Lacerations, contusions	Chronic cardiovascular disease
Falls (including on site and from moving apparatus)	Chronic respiratory disease
Burns (superficial, deep, internal)	Noise-induced hearing loss
Dermal reactions to toxicants	Posttraumatic stress disorder
Eye irritation, injuries, and burns	Physical disability
Smoke inhalation	Hepatitis C
<ul style="list-style-type: none"> • Sore throat, hoarseness, cough • Exacerbated asthma • Dyspnea, tachypnea, wheezing • Headaches • Cyanosis 	
Cardiovascular strain	
Musculoskeletal trauma	
Heat stress and fatigue	
Neuropsychiatric effects	
Renal damage	
Death (motor vehicle accidents, falls, asphyxiation, burns)	

Modified from NIOSH Fire Fighter Fatality/Injury Investigation Reports (<http://www.cdc.gov/niosh/firehome.html>, 2001).

Occupational Burns

Of the more than one million firefighters employed in the United States, 300,000 are career firefighters. The rate of injury and death occurring on the fire ground or while responding to or returning from an incident has declined since the late 1980s with the mandatory use of gloves, self-contained breathing apparatus (SCBA), and full personal protective clothing. National trends for firefighter injuries are sprain/strain- and stress-related injuries. Over 50% of all injuries involve overexertion, lifting, pulling, or carrying hose and equipment. Studies support the long-standing assertion that the number of firefighters responding to a fire is a factor that affects injuries.¹⁶⁷

Aside from the acute injurious effects of fire, clinicians must be alert to the pathophysiologic changes associated with exposure to heat and smoke and to the chronic sequelae, both physical and psychologic (Table 4-3). In addition to the management of burns and trauma, it is necessary to evaluate clients for all acute systemic effects of exposure to smoke, heat, or toxic substances; recognize toxic effects that may be obscured by more serious traumatic effects; be alert for delayed consequences; and recognize acute and chronic exposure and health effects as a result of toxic chemicals in smoke, especially among firefighters.

Carbon monoxide is always present at fires. Smoldering fires with incomplete combustion of burning material can lead to significant levels of CO. For this reason, firefighters are required to wear SCBA at every incident. Respiratory responses of firefighters while wearing SCBA will reduce their breathlessness during exercise (exertion on the job).⁴³

Occupational Pulmonary Diseases

Materials inhaled in the workplace can lead to all the major chronic lung diseases, except those as a result of vascular disease. Exposure in office buildings and hospitals is now included as a known workplace-related cause of disease. As new industries are developed, new problems are reported. For example, obstructive lung disease has been reported in workers in the microwave popcorn and flavor-manufacturing business who have not been adequately protected from chemical exposures.⁹¹

Identifying the source of illness is important because it can lead to cure and prevention for others.¹⁵ Disorders caused by chemical agents are classified as (1) pneumoconioses, (2) hypersensitivity pneumonitis, (3) obstructive airway disorders, (4) toxic lung injury, (5) lung cancer, and (6) pleural diseases. These conditions are discussed more fully in Chapter 15.

Asbestos and other silicates, such as kaolin, mica, and vermiculite, can cause *pneumoconiosis*. Asbestos-induced diseases cause lung inflammation and fibrosis as a result of activation of alveolar macrophages. Coal worker's pneumoconiosis is another parenchymal lung disease caused by inhalation of coal dust.

Hypersensitivity pneumonitis has many other names, such as extrinsic allergic alveolitis, farmer's lung, and detergent worker's lung, and is characterized by a granulomatous inflammatory reaction in the pulmonary alveolar and interstitial spaces. Silicosis is a parenchymal toxic lung disease caused by inhalation of crystalline silica, a component of rock and sand. Workers at risk include miners, tunnelers, quarry workers, stonemasons, sandblasters, foundry workers, glass blowers, and ceramic workers.

The pathogenesis of these occupational lung diseases varies among different pneumoconioses, but the bottom line is that cilia and mucus-secreting cells are absent in the small bronchioles and alveoli. The body depends on macrophages to remove any of the tiny particles that lodge in these areas. The macrophages then carry them to the mucociliary elevator or dump them into the lymphatics. The process is often sabotaged because substances, such as silica dust, can destroy the macrophages. In the process, substances are released that trigger inflammation and pulmonary fibrosis. See Chapter 15 for more details about these diseases.

Exposure to allergens and irritants has resulted in the recognition of a new disease called *work-related upper airway disease* or *united airway disease*, which although not life-threatening, has been reported to cause sufferers to experience reduced quality of life. Occupational allergens identified are many and varied, including plants (e.g., tobacco leaf dust, grapes, asparagus, or flowers), insects (e.g., bees or locusts), powder paints, and others. Reports of upper airway disease in various occupational groups involved in rescue, recovery, and cleanup at the World Trade Center identified a new work hazard from irritants.¹⁶⁹

Health problems caused by these irritants range from runny nose to full-blown allergic rhinitis. Up to 40% of individuals in the workplace with allergic rhinitis also have asthma. The link between rhinitis and asthma is the

Box 4-4

ASTHMA-TRIGGERING SUBSTANCES IN THE HEALTH CARE SETTING

- Latex (primarily latex gloves)
- Glutaraldehyde (sensitizing agent used in cold sterilization)
- Ammonia and chlorine (cleaning and disinfecting solutions)
- Dust and irritating particles in the air (construction and remodeling projects)
- Mold and fungus (carpeting, ceiling tiles exposed to water)
- Perfumes, scented personal care products worn by clients/patients, coworkers, visitors
- Isocyanate (a class of extremely hazardous substances found in orthopedic casting materials)
- Pharmaceutical drugs (e.g., psyllium, rifampin, penicillin, tetracycline)
- Formaldehyde used in specimen preparation
- Diacetyl (ingredient in artificial butter and other flavoring)

From Bain EL: Perils in the air: Avoiding occupational asthma triggers in the workplace, AJN 100(6):88, 2000.

presence of inflammation of the nasal and bronchial mucosae.¹⁶⁹

Occupational asthma or work-related asthma (WRA) (*airway obstruction*) is asthma that is attributable to or is made worse by environmental exposures (e.g., inhaled gases, dusts, fumes, or vapors) in the workplace. The air in health care institutions may contain irritating and sensitizing chemicals and particles that can aggravate asthma (Box 4-4).

WRA has become the most prevalent occupational lung disease in developed countries, is more common than is generally recognized, and can be severe and disabling. The reactions can be immediate or delayed, sometimes hours after leaving the workplace. Identification of workplace exposures causing and/or aggravating the asthma and appropriate control or cessation of these exposures can often lead to reduction or even complete elimination of symptoms and disability.⁵⁵

OSHA requires employers to provide a safe and healthy work environment free from recognized hazards. In addition, the Americans with Disabilities Act of 1990 requires employers to accommodate workers with asthma. Suspected episodes of WRA should be documented, including symptoms, suspected exposures, visits to health services, and similar symptoms reported by other employees. Many effective and appropriate substitutions and controls are available that can be incorporated to eliminate or prevent airborne and topical exposures.⁷

Occupational Cancer

Despite increased knowledge of occupational risk for cancer, it is estimated that 30% to 40% of the population in the industrialized world will develop malignant disease during their lifetime. Changes in wood processing and decreased duration of occupational exposure because of more frequent job changes may have altered the picture somewhat.

Studies continue to provide evidence that cancer in humans has environmental causes (e.g., exposure to

arsenic is associated with increased risk of skin, urinary bladder, and respiratory tract cancers; chronic exposure to ultraviolet light is associated with skin cancer; vinyl chloride is associated with liver cancer; dry cleaning solvents are associated with kidney and liver cancer and non-Hodgkin's lymphoma). Research is ongoing to assess combined genetic and environmental contributions to risk.^{44,69,131}

Alteration or mutation in the genetic material (deoxyribonucleic acid [DNA]) may occur as a result of exposure to carcinogenic chemicals or radiation. Both experimental animal models of cancer and the study of human cancers with known causes have revealed the existence of a significant interval between first exposure to the responsible agent and the first manifestation of a tumor. This period is referred to as the *induction period*, *latency period*, or *induction-latency period*.

For humans, the length of the induction-latency period varies from a minimum of 4 to 6 years for radiation-induced leukemias to 40 or more years for some cases of asbestos-induced mesotheliomas. For most tumors, the interval ranges from 12 to 25 years; such a long period may easily obscure the relationship between a remote exposure and a newly discovered tumor.

In the future, individuals with a high environmental risk of developing cancer may benefit from immune stimulation as a means of cancer prevention by inducing specific immunity through the use of vaccines.⁵² Individual cancers and their treatment are discussed in organ-specific chapters in this text; see also Chapter 9.

Acute Radiation Syndrome

Acute radiation syndrome is caused by brief but heavy exposure of all or part of the body to ionizing radiation. The radiation disrupts chemical bonds, which causes molecular excitation and free radical formation. Highly reactive free radicals react with other essential molecules, such as nucleic acids and enzymes, and this in turn disrupts cellular function.

The clinical presentation and severity of illness depend on many factors, including volume of tissue treated, the dosage (fractionation), and other independent variables. Tissues with the most rapid cellular turnover are the most radiosensitive and include reproductive, hematopoietic, and gastrointestinal tissues. See the section on Physical Agents in this chapter and also the section on Radiation Injuries in Chapter 5.

Occupational Infections

Occupational infections are diseases caused by work-associated exposure to microbial agents, including bacteria, viruses, fungi, and protozoa. Occupational infections are distinguished by the fact that some aspect of the work involves contact with a biologically active organism. Occupational infection can occur after contact with infected people, as in the case of health care workers; infected animal or human tissue, secretions, or excretions, as in laboratory workers; asymptomatic or unknown contagious humans, as happens during business travel; or infected animals, as in agriculture (e.g., brucellosis).

Tuberculosis, herpes simplex and herpes zoster (shingles), hepatitis, and AIDS are the most likely occupational infections encountered in a therapy practice.

Occupational Skin Disorders

Accounting for 20% of all cases of occupational disease in the United States, 61,000 new cases of occupational skin disease are reported each year. It is likely that many cases of work-related skin disorders are underreported, since it is often not a life-threatening condition and never diagnosed or treated. The health care industry reports 4000 cases of skin illness each year, but the highest rates are in agriculture and manufacturing.

Dermatoses are more prevalent in some states such as California and Florida; contact dermatitis from plants, especially in combination with sunlight, and chemicals, such as pesticides or fertilizers, is common among agricultural workers. Contact dermatitis (acute, chronic, or allergic) is the most common of occupational skin disorders, but other types include contact urticaria, psoriasis, scleroderma, vitiligo (areas of depigmentation), chloracne (see Fig. 4-1), actinic skin damage known as farmer's skin or sailor's skin, cutaneous malignancy, and cutaneous infections.

Other agents in the workplace include irritating chemicals such as solvents, cutting oils, detergents, alkalis, and acids. Arsenic and tar products can increase the risk of cancer either alone or in combination with sunlight. Skin cancer is an important occupational illness and is most often the result of excessive exposure to ultraviolet light—farmers, fishermen, roofers, and road workers who continuously work in the sun are at greatest risk. For further discussion of specific skin disorders, see Chapter 10.

Rubber Latex Allergy

During the recent past, the incidence of natural rubber latex allergy (LA) has dramatically increased not only among the general population but also among health care workers, the latter because of repeated contact as a result of standard precautions and subsequent increased occupational exposure.

LA occurs predominantly in certain high-risk groups (Box 4-5); the estimated prevalence in health care workers varies widely (2.8% to 18%), and studies do not always distinguish between those who are positive in an assay for latex-specific immunoglobulin E (IgE) and those with clinical allergy.¹²⁰ The prevalence of LA in the general population ranges from 0.1% to 1.0%, compared with as high as 60% for those with spina bifida or other chronic medical conditions associated with repeated exposure to latex.¹⁰²

This occupational sensitivity to natural rubber latex (NRL) (i.e., latex proteins and in some cases the associated cornstarch glove powder serve as a carrier for the allergenic proteins from the NRL) has resulted in the following three types of reactions:

- Immediate hypersensitivity (type I hypersensitivity; IgE-mediated) with urticaria (hives), watery eyes, rhinitis, respiratory distress, and asthma or skin rash, which can spread from the hands, up the arms, and to the face (it can also cause swelling of the lips,

eyes, ears, and larynx [laryngeal edema can prevent the person from speaking]).

- Irritation or irritant contact dermatitis manifested as dry, crusty, hard bumps; sores; and horizontal cracks on the skin (Fig. 4-4)
- Mild-to-severe allergic contact dermatitis (delayed type IV hypersensitivity; cell-mediated) (Fig. 4-5)

The first two reactions are related to mechanical and chemical exposure, whereas LA is caused by sensitization to the proteins in NRL. These responses occur when items containing latex touch the skin, mucous membranes (eyes, mouth, nose, genitals, bladder, or rectum), or open areas.

Latex exposure has become one of the leading causes of occupational asthma. Once sensitized, some health care workers are at risk for severe systemic allergic reac-

Box 4-5

RISK FACTORS FOR LATEX ALLERGY

Repeated or frequent exposure to latex products via one or more of the following:

- Repeat or frequent catheterization or other urologic procedure(s)
- Occupation
 - Health care workers (dentists, nurses, surgeons, laboratory or operating room technicians, therapists, especially wound care specialists)
 - Rubber or latex industry workers
 - Doll manufacturing workers
 - Occupation requiring gloves (hair stylist, food handler, gardener or greenhouse worker, housekeeper)
- Immunocompromised individuals
- Individuals with spina bifida or myelomeningocele
- Spinal cord injury (presence of indwelling urinary catheter)
- History of multiple surgeries
- Individuals (including children) receiving home mechanical ventilation
- (Personal or family history of eczema, asthma, or atopy (allergies), including food allergies*)

*Cross-reactivity occurs between latex and avocado, kiwi fruit, papayas, chestnuts, brazil nuts, tomatoes, and bananas, probably because latex proteins are structurally homologous with other plant proteins.

Clinically, perioral itching and local urticaria occur; rarely food-induced anaphylactic shock occurs.



Figure 4-5

Latex allergy dermatitis. A, Latex glove allergy should be suspected in health care workers who present with eczema, blistering, or skin peeling anywhere on the hands. B, Allergy to the rubber band of underwear. Washing clothes with bleach may make the rubber allergenic. Similar skin reactions can be seen in women across the midback under the bra strap (not shown). (From Habif TP: *Clinical dermatology*, ed 4, St Louis, 2004, Mosby.)

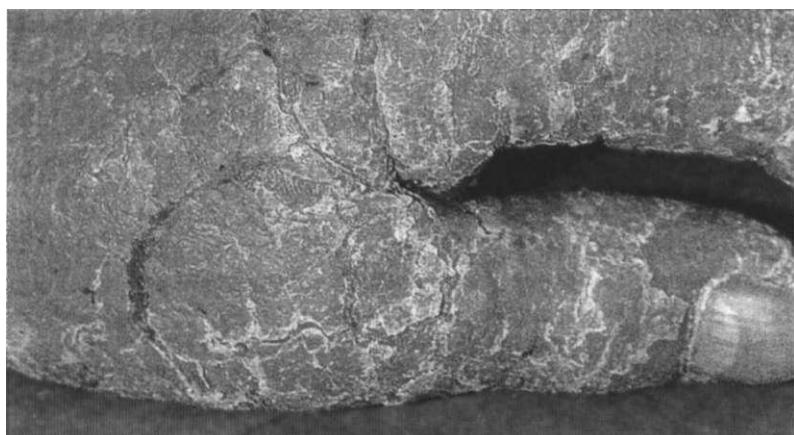


Figure 4-4

Rubber glove dermatitis. (From Raffle PAB, Adams PH: *Hunter's diseases of occupations*, ed 8, London, 1994, Edward Arnold.)

tions, which can be fatal in some cases. In susceptible individuals, airways react to low levels of a variety of sensitizers and irritants in the environment. The two major routes of exposure include dermal exposure and inhalation exposure. NRL protein absorption is enhanced when perspiration collects under latex clothing articles (e.g., elastic waistbands and leg bands in underwear). However, the elimination of wearing latex gloves has not been successful in eliminating latex sensitization, since latex allergens are airborne.

Exposure by the respiratory route occurs when the NRL protein becomes airborne, especially since glove powder becomes airborne, acting as a carrier for the NRL protein when gloves are donned or removed.

Latex-induced rhinitis and occupational asthma are new forms of occupational illness secondary to airborne latex allergens in operating rooms, intensive care units, and dental suites. Anyone with latex allergies should be treated as the first case of the day, whether in the operating room or in a therapy department, to avoid latex in the air and to avoid introducing any latex from clothes or materials from previous contacts.

SPECIAL IMPLICATIONS FOR THE THERAPIST 4-3

Rubber Latex Allergy

PREFERRED PRACTICE PATTERNS

7A: Primary Prevention/Risk Reduction for Integumentary Disorders

7B: Impaired Integumentary Integrity Secondary to Superficial Skin Involvement

Possible patterns related to latex-induced asthma: 6B, 6C, 6F, 6G, 6H, or 6I

All clients should be screened for known LA or risk factors on admission. It is not enough to ask if someone is allergic to latex; risk factors and past medical history must be assessed. This is especially important because anaphylaxis could be the first sign of LA. If anyone in the rehabilitation or therapy department develops symptoms in association with the use of latex gloves, emergency medical care may be required. The presence of hives, perioral itching, respiratory distress, watery eyes, and facial swelling may indicate a type I hypersensitivity response and requires immediate medical attention. In a hospital setting, a physician can be paged immediately; other locations may require an emergency medical team (calling 911 or an emergency medical service). Check with the facility for incident report requirements.

For the health care worker with a known sensitivity, a medical-alert bracelet should be worn, and the individual should have autoinjectable epinephrine (EpiPen) for use if another reaction occurs. Anyone experiencing the first reaction should not ignore the symptoms; further episodes must be avoided by developing a latex-safe environment and using nonlatex products.

All clients with myelomeningocele are to be treated as if latex allergic. The therapist, family members, and caregivers must avoid using toys, feeding utensils, pac-

ifiers, nipples, or other items made of latex that the infant or child might put in the mouth. Clothes and shoes with elastic anywhere must be avoided. Parents must be advised to read all labels and avoid all items containing latex. If no indication of latex content is evident, the manufacturer should be contacted for verification before purchase or use of the item. More information on this topic is available at the web site Exceptional Parent (<http://www.eparent.com/toys/latex.htm>) or the American Latex Allergy Association (www.latexallergyresources.org).

A latex-safe environment may be required for complete recovery for people with LA and is essential for all pediatric cases and anyone with known LA. A latex-safe environment, including the operating room, is described as one in which no latex gloves are used by any personnel, no direct client contact with latex devices (e.g., catheters, condoms, diaphragms, adhesives, tourniquets, rubber backing on bath mats or other materials, hot water bottles, or anesthetic equipment) occurs; and all medical and patient/client care items have been assessed for latex and labeled.

Handwashing before donning and after removing gloves must be carried out at all times with special care given to using a pH-balanced soap and rinsing well to remove all residue. All medical products containing NRL that could come in contact with clients must be labeled. Keep in mind that many latex-free supplies have packaging that contains latex (glue), and those workers in the production or packaging of these products may have worn latex gloves.

No latex balloons or toys containing latex should be allowed in health care facilities; crash carts should be latex free. Personnel in the therapy department must be aware of the many items in the department that contain latex and replace these with latex-free products or a latex-free barrier (Table 4-4). Almost all equipment, supplies, and personal protective equipment is available in latex-free form, although not by all manufacturers. Complete guidelines for prevention and protection are available through the American Nurses Association at (800) 274-4ANA.

Several potential sources of powder-free, natural hypoallergenic latex gloves may be tolerated by latex-sensitive individuals, but no single replacement glove has been found for all people affected. Cotton liners or barrier creams can be effective interventions. Vinyl gloves are generally less protective than latex and more prone to tearing. Some of the new synthetic materials, such as nitrile, neoprene, and thermoplastic elastomer, offer equal or superior barrier protection and durability and are a reasonable alternative to latex or vinyl, offer better protection than latex types when handling lipid-soluble substances and chemicals, and are reasonably priced.¹²⁸

However, like latex, synthetic glove products can cause allergic reactions because they may contain chemical additives similar to those found in latex and both are manufactured using the same process, called *vulcanization*. Additionally, synthetic gloves also provide a poorer fit than their latex counterpart and

Continued.

Table 4-4 Potential Sources of Latex in a Rehabilitation Department*

Item	Replacement Item
Personal Protective Equipment	
Gloves (sterile and nonsterile)	Nitrile, neoprene, or thermoplastic elastomer examination gloves
Goggles	
Hair covers	
Respirators	
Rubber aprons	
Shoe covers	
Surgical masks	
Equipment/Supplies	
Bandages	
Casting material	
Crash cart	
Crutch and walker handgrips	Cover with stockinette
Crutch axillary pads	Cover with stockinette
Dressings	
Elastic netting	
Electrode pads, especially disposable TENS	Band Net Latex Free (Western Medical, Ltd.) Stretch Net: Latex Safe (DeRoyal) The Net Works (Wells Lamont Medical)
Exercise balls	Cover ball with a towel
Exercise bands	Use the following latex-free brands: REP Band (Magister Corporation) Thera-Band Latex-Free Latex-Free CANDO exercise band (SPRI) Use free weights that are not covered with materials containing latex
Exercise mats	
Foam rubber lining splints, braces	Cover with sheet or blanket
Mini trampoline	Line with cloth, felt
Positioning supports and pads of foam rubber without complete coverings	Cover with stockinette
Reflex hammer	Cover with latex-free plastic bag
Rubber bands	String, paper clips
Shoe orthotics	
Stethoscope tubing	Cover with gauze or premade cover
Sphygmomanometer	Cover cuff or extremity with gauze
Tape (all kinds)	Cover skin first with gauze; tape over gauze
Toys; toys made from latex gloves	Toys made without latex
Vascular stockings	Cover with cloth
Wheelchair cushions	Propel with leather or cloth gloves
Wheelchair tires	

Courtesy Harriett B. Loehne, PT, DPT, CWS, FCCWS.

*Many manufacturers now make latex-free items. Any medical supply with latex must be so marked.
TENS, Transcutaneous electrical nerve stimulation.

come with environmental concerns (e.g., the production and disposal of vinyl gloves releases toxic substances, such as dioxins, into the environment).

Glove specifications (e.g., leak defect rate; American Society for Testing and Materials [ASTM] specifications for length, width, tensile strength, thickness; powder particulate weight per glove; and protein levels) are available and should be examined by each health care provider according to facility or client-based use.¹⁷² For more information, see NIOSH Alert: Preventing Allergic Reactions to Natural Rubber Latex in the Workplace (800-35-NIOSH) or OSHA: Technical Information Bulletin—Potential for Allergy to Natural Rubber Latex Gloves and Other Natural Rubber Products (http://www.osha.gov/dts/tib/tib_data/tib19990412.html)

Military-Related Diseases

Seven diseases (asthma, laryngitis, chronic bronchitis, emphysema, and three eye ailments) have been identified by the Department of Veterans Affairs for compensation as a result of exposure to toxic chemicals during World War II.

Survivors of the Vietnam War who have been exposed to a dioxin (2,3,7,8-tetrachlorodibenzo-para-dioxin [TCDD]) contained in the herbicide mixture Agent Orange (sprayed from the air, by boat, and on the ground in Vietnam to defoliate jungles from 1962 to 1971) are known to be at risk for diabetes and chronic lymphocytic leukemia. The risk for other types of cancer has never been conclusively proven, but as Vietnam veterans continue to age, additional research will yield more information about cancer risk.¹⁷³

There has been concern about reproductive effects of Agent Orange such as birth defects in the children of exposed veterans. Neural tube defects, neurotoxicity, neuropsychiatric dysfunction, deficits in motor function, and peripheral neuropathy may be linked to Agent Orange exposure but considerable uncertainty exists about these associations.⁵⁶

More recently, a group of symptoms presented by participants in the Gulf War have been identified. ALS, or Lou Gehrig's disease, has been identified among these military personnel. The U.S. government confirmed this link in December 2001. Anyone seeking more information about either Agent Orange or the Gulf War Syndrome can contact the Gulf War/Agent Orange Helpline at (800) PGW-VETS.

Gulf War Syndrome

Overview. Regardless of whether an actual Gulf War syndrome (GWS) exists, it remains a hotly debated topic. According to the Centers for Disease Control and Prevention (CDC), Americans who served in the Persian Gulf War are significantly more likely than others to experience more than a dozen disorders known generically as GWS. The CDC does not term this phenomenon GWS but reports that people who went to the Persian Gulf are experiencing problems (referred to as *Persian Gulf illness* [PGI]) that those who did not go are not experiencing. However, the Department of Defense does not support the existence of this illness, reporting only that the results of medical examinations of 10,000 veterans and family members affected revealed multiple illnesses with overlapping symptoms.⁷⁸

Incidence and Clinical Manifestations. Of the 700,000 troops dispatched to the Persian Gulf between August 1990 and June 1991, as of January 2007, more than 100,000 veterans have filed with the federal registry reports of symptoms that include (in order of frequency) fatigue, skin rash, headache, muscle and joint pain, memory loss, shortness of breath, sleep disturbances, diarrhea and other gastrointestinal symptoms, and depression. CDC data show that GWS affects 27% of veterans compared with 2% of nonveterans. Fatigue has been reported to affect 54% of Gulf War veterans compared with 16% of non-Gulf War veterans.

Etiologic Factors. No single cause has been identified, but possible factors include chemical or biologic weapons used on allied forces, insecticides, oil well fires in Kuwait, nerve agents from the demolition of Iraqi chemical weapons, parasites, pills protecting against nerve gas, and inoculations against petrochemical exposure administered by the military that had unexpected side effects or reacted with one another to create adverse symptoms.

In 1993, the Birmingham, Alabama Veterans Administration Center was designated as a national pilot center to study the possible neurologic effects of exposure to environmental agents in the Persian Gulf. Other designated environmental hazards research centers are located in Boston; East Orange, NJ; and Portland, OR.

Pathogenesis. The pathogenesis for GWS remains unknown but researchers are investigating the similarities between the underlying mechanisms of chronic fatigue syndrome, fibromyalgia, migraine headaches, and GWS. MRI studies of veterans with different GWS have biochemical evidence of neuronal damage in different distributions in the basal ganglia and brainstem, supporting the theory of neurologic toxicity related to chemically induced injury to dopaminergic neurons in the basal ganglia.^{65,66}

A new theory, referred to as *toxicant-induced loss of tolerance (TILT)*, has been suggested. Drug addiction and multiple chemical intolerance ("abduction") appear to be polar opposites (addiction is characterized by craving and dependency, abduction is characterized by aversion); however, when compared side-by-side, common underlying mechanisms are observed.

Both addiction and chemical intolerance involve a fundamental breakdown in innate tolerance, resulting in an amplification of various biologic effects, particularly withdrawal symptoms. Although addicts seek further exposures to avoid unpleasant withdrawal symptoms, chemically intolerant individuals avoid exposure to reduce unpleasant symptoms. The question of whether addictive drugs and environmental pollutants initiate an identical pathogenic process with triggered symptoms and cravings remains under investigation.⁷⁶

MEDICAL MANAGEMENT

No specific intervention beyond management and symptomatic measures exists for PGI. Focusing on triggering events rarely helps define treatment for people with syndromes such as GWS. Understanding the entire spectrum of illnesses from chronic fatigue syndrome to fibromyalgia to GWS in light of treatment must be the means to developing multidisciplinary treatment programs for affected people that includes allopathic, naturopathic, and alternative treatment.

Iraqi War

Veterans of Operations Enduring Freedom and Iraqi Freedom (OEF/OIF) have endured high combat stress and are at risk for posttraumatic stress disorder (PTSD) and psychosocial problems.¹³⁹ During deployment, there were reports of heat illness despite predeployment efforts to prepare soldiers. Extreme temperatures averaging 46° C still resulted in a heat illness rate of 50/1000 during the first 10 to 14 days of deployment.²⁰ There have been no reports of long-term results from this problem.

References

To enhance this text and add value for the reader, all references are included on the companion Evolve site that accompanies this textbook. The reader can view the reference source and access it on-line whenever possible. There are a total of 173 cited references and other general references for this chapter.

CHAPTER 5

Problems Affecting Multiple Systems

CELESTE PETERSON • CATHERINE C. GOODMAN

Many conditions and diseases seen in the rehabilitation setting can affect multiple organs or systems (Box 5-1). With the kinds of multiple comorbidities and system impairments encountered in the health care arena, the therapist must go beyond a systems approach and use a biopsychosocial-spiritual approach to client management. Chronic diseases and multiple system impairments require such an approach because risk factors correlate with health outcome; early intervention and intervention results are correlated with improved outcome.

Individual modifying (risk) factors (IMF) such as lifestyle variables and environment affect pathology and modify how a person responds to health, illness, and disease. For example, adverse drug reactions are correlated with increasing age and obesity, whereas fitness level has a profound impact on recovery from injury, anesthesia, and illness.

Additionally, a single injury, disease, or pathologic condition can predispose a person to associated secondary illnesses. For example, the victim of a motor vehicle accident in Chapter 6 (see Special Implications for the Therapist: Cell Injury) suffered a traumatic brain injury (TBI) and concomitant pelvic fracture then developed pneumonia and pulmonary compromise, subsequently experiencing a myocardial infarction.

This type of clinical scenario involving multiple organs and comorbidities is not uncommon. Also consider the medically complex person who needs a splint. The therapist must first review laboratory values (see Chapter 40) to determine albumin levels (nutritional status) and platelet levels (potential for bleeding), perform a skin assessment (see Chapter 10), and consult with both nursing staff and the nutritionist before providing an external device that could create skin breakdown and add to an already complex case.

Although medical conditions encountered in the clinic or home health care setting are discussed individually in the appropriate chapter, the health care provider must understand the systemic and local effects of such disorders. This chapter provides a brief listing of the systemic effects of commonly encountered pathologic conditions and a basic presentation of acid-base and fluid and electrolyte imbalances. The scope of this text does not allow for an in-depth discussion of each condition or disease and its related multiple systemic effects.

The importance of using the *Guide to Physical Therapist Practice*[®] is recognized throughout this text. However, identifying Preferred Practice Patterns in multisystem disorders depends on the presenting signs and symptoms and their influence on individual function and the rehabilitation process. For this reason, specific practice patterns are not listed with each section. The reader is referred to individual chapters discussing the underlying pathology or directly to the *Guide*.

SYSTEMIC EFFECTS OF PATHOLOGY

Systemic Effects of Acute Inflammation

Acute inflammation can be described as the initial response of tissue to injury, particularly bacterial infections and necrosis, involving vascular and cellular responses. Local signs of inflammation (e.g., redness, warmth, swelling, pain, and loss of function) are commonly observed in the therapy setting. Local inflammation can lead to abscesses when excessive suppuration (formation of pus) occurs.

Systemic effects of acute inflammation include fever, tachycardia, and a hypermetabolic state. These effects produce characteristic changes in the blood, such as elevated serum protein levels (C-reactive protein, serum amyloid A, complement, and coagulation factors) and an elevated white blood count (leukocytosis).⁷⁹ For a complete discussion of inflammation and its effects, see Chapter 6.

Systemic Effects of Chronic Inflammation

Chronic inflammation is the result of persistent injury, repeated episodes of acute inflammation, infection, cell-mediated immune responses, and foreign body reactions. The tissue response to injury is characterized by accumulation of lymphocytes, plasma cells, and macrophages (mononuclear inflammatory cells) and production of fibrous connective tissue (fibrosis). Fibroblasts and small blood vessels, along with collagen fibers synthesized by fibroblasts, constitute fibrosis. Grossly, fibrotic tissue is light gray and has a dense, firm texture that causes contraction of the normal tissue.