CHAPTER

16

Drugs Affecting Muscle Spasm and Spasticity

Learning Objectives

At the completion of this chapter the student will:

1. Correlate the pathophysiology of muscle spasm and spasticity with appropriate pharmacotherapy.
2. Identify core drug knowledge about pharmacologic therapies that affect muscle spasm and spasticity.
3. Identify core patient variables related to drugs that affect muscle spasm and spasticity.
4. Relate the interaction of core drug knowledge with core patient variables for pharmacologic therapies that affect muscle spasm and spasticity.
5. Generate a nursing plan of care from the interactions between core drug knowledge and core patient variables for pharmacologic therapies that affect muscle spasm and spasticity.
6. Describe nursing interventions to maximize therapeutic effects and minimize adverse effects for drugs that affect muscle spasm and spasticity.
7. Determine key points for patient and family education about drugs that affect muscle spasm and spasticity.

Key Terms

centrally acting

clonic

peripherally acting

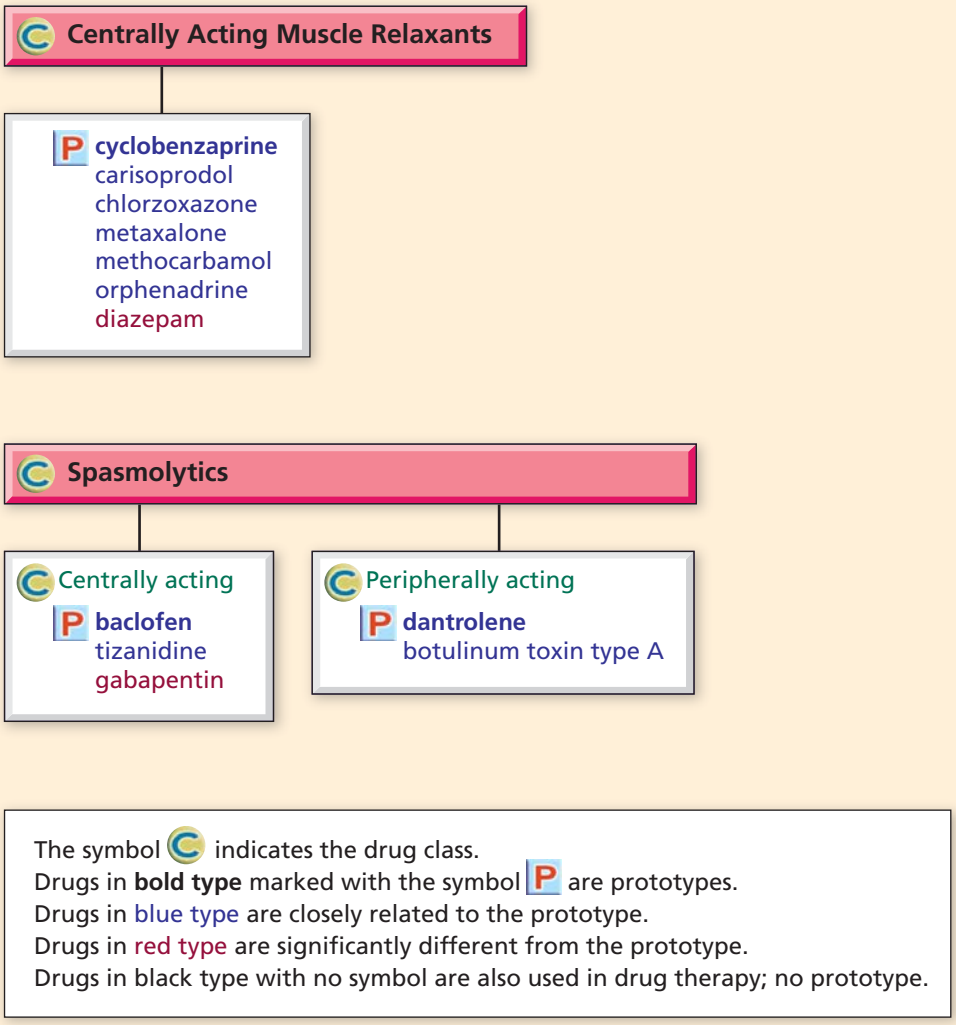
spasm

spasmolytics

spasticity

tonic

Drugs Affecting Muscle Spasm and Spasticity



The symbol C indicates the drug class.
Drugs in **bold type** marked with the symbol P are prototypes.
Drugs in blue type are closely related to the prototype.
Drugs in red type are significantly different from the prototype.
Drugs in black type with no symbol are also used in drug therapy; no prototype.

Drugs used to manage muscle spasm and spasticity can be divided into two major therapeutic groups: skeletal muscle relaxants and **spasmolytics.** Muscle spasms are treated with a combination of physical therapy, centrally acting muscle relaxants, and anti-inflammatory agents (see Chapter 24). Spasticity is treated with physical therapy and drugs called spasmolytics. Spasmolytics are categorized as **centrally acting** or **peripherally acting.** As their names imply, these agents act in the brain or in the peripheral muscles.

This chapter discusses the centrally acting muscle relaxant cyclobenzaprine (Flexeril), the centrally acting spasmolytic baclofen (Lioresal), and the peripherally acting spasmolytic dantrolene sodium (Dantrium). Table 16.1 presents a summary of these drugs. In addition, this chapter addresses core drug knowledge, core patient variables, nursing management practices, potential nursing diagnoses, and patient education guidelines related to the use of these drugs. For discussion of physical methods for managing spasm and spasticity, refer to an appropriate medical-surgical textbook.

PHYSIOLOGY

CONCEPTS in action ANIMATION

The human body contains approximately 600 skeletal muscles. Skeletal muscle is voluntary, meaning a person can contract it at will. Seen under a microscope, skeletal muscle fibers show a pattern of cross-banding, which gives rise to its other name: striated muscle. The striations are caused by the alignment of bands, the most prominent of which are the A bands, I bands, and Z lines. The unit between two Z lines is called the sarcomere (Figure 16.1).

Striated muscle is composed of two contractile proteins: actin and myosin. The thin filaments are made of actin, which is attached to the Z lines and is found in both A bands and I bands. The thick filaments, found in A bands, are made of myosin. In the process set forth in the sliding filament theory, the sarcomere shortens, and the Z lines move closer together when muscle contracts. The filaments slide together because myosin attaches to, and pulls on, actin. The myosin head attaches to the actin filament, forming a crossbridge. After formation of the crossbridge, the myosin head bends, pulling on the actin filaments and causing them to slide. The result is that the Z lines move closer together, the I band becomes shorter, and the A band stays the same (see Figure 16.1). Muscle contraction is like climbing a rope. The crossbridge cycle is one of grabbing, pulling, and releasing, repeated over and over.

Muscle contraction is triggered by a sudden inflow of calcium ion (Ca^{2+}). In the resting state, the protein tropomyosin winds around actin and covers the myosin-binding sites. The Ca^{2+} binds to a second protein, troponin; this action causes the tropomyosin to be pulled to the side, exposing the myosin-binding sites. With the sites exposed, muscle contracts in the presence of adenosine triphosphate (ATP). Muscle contraction stops when Ca^{2+} is removed from the immediate environment of the myofilaments.

PATHOPHYSIOLOGY

Muscle Spasm

A muscle **spasm** is a sudden violent involuntary contraction of a muscle or group of muscles. Spasm is usually related to a localized skeletal muscle injury from acute trauma. Spasms may also stem from disorders such as hypocalcemia, hypokalemia or hyperkalemia, chronic pain syndromes, or epilepsy. Pain and interference with function attend muscle spasm, producing involuntary movement and distortion. When a muscle goes into spasm, it freezes in contraction and becomes a hard knotty mass, rather than normally contracting and relaxing in quick succession. During spasm, the blood vessels that normally feed the muscles and supply oxygen constrict, further compounding the problem.

Tonic spasm, or cramp, is characterized by an unusually prolonged and strong muscular contraction, with relaxation occurring slowly. In the other form of spasm, called **clonic** spasm, contractions of the affected muscles occur repeatedly, forcibly, and in quick succession, with equally sudden and frequent relaxations.

Spasticity

Spasticity is a condition in which certain muscles are continuously contracted. This contraction causes stiffness or tightness of the muscles and may interfere with gait, movement, or speech. Damage to the portion of the brain or spinal cord that controls voluntary movement usually causes spasticity. Spasticity may be associated with spinal cord injury, multiple sclerosis (MS), cerebral palsy, anoxic brain damage such as a cerebrovascular accident (CVA), brain trauma, severe head injury, and some metabolic diseases, such as adrenoleukodystrophy and phenylketonuria. Symptoms may include hypertonicity (increased muscle tone), clonus (a series of rapid muscle contractions), exaggerated deep tendon reflexes, muscle spasms, scissoring (involuntary crossing of the legs), and fixed joints. The degree of spasticity varies from mild muscle stiffness to severe, painful, and uncontrollable muscle spasms. The condition can interfere with daily activities and with rehabilitation in patients with certain disorders.

CENTRALLY ACTING MUSCLE RELAXANTS

The centrally acting muscle relaxants are a group of drugs with similar pharmacologic properties. They act in the central nervous system (CNS). The prototype for centrally acting muscle relaxants is cyclobenzaprine (Flexeril), because evidence indicates that it is the most efficacious. Diazepam (Valium), a benzodiazepine, is mentioned because it is used in managing both muscle spasms and spasticity. Benzodiazepines are discussed in depth in Chapter 18.

TABLE 16.1 Summary of Selected Drugs That Affect Muscle Spasm and Spasticity

Drug (Trade) Name	Selected Indications	Route and Dosage Range	Pharmacokinetics
Centrally Acting Muscle Relaxants			
P cyclobenzaprine (Flexeril, *Canadian:* Apo-Cyclobenzaprine, Gen-Cyclobenzaprine, Novo-Cyclobenzaprine, Nu-Cyclobenzaprine, Flexitec)	Muscle spasms Muscle relaxation	*Adult:* PO, 10 mg tid; maximum 60 mg qid *Child <15 y:* not recommended	*Onset:* 1 h *Duration:* 12–24 h $t_{1/2}$: 8 h
carisoprodol (Soma)	Muscle spasms Muscle relaxation	*Adult:* PO, 350 mg qid *Child:* not recommended	*Onset:* 3–5 d *Duration:* 4–6 h $t_{1/2}$: 1–3 d
chlorzoxazone (Parafon Forte DSC, *Canadian:* Strifon Forte)	Muscle spasms Muscle relaxation	*Adult:* PO, 250–500 mg tid/qid *Child:* PO, 20 mg/kg/d in divided doses	*Onset:* 30–60 min *Duration:* 3–4 h $t_{1/2}$: 60 min
metaxalone (Skelaxin)	Muscle spasms Muscle relaxation	*Adult and child >12 y:* PO, 800 mg tid–qid	*Onset:* 1 h *Duration:* 4–6 h $t_{1/2}$: 2–3 h
methocarbamol (Robaxin)	Muscle spasms Muscle relaxation Tetanus	*Adult:* 1,500 mg qid; 1,000 mg qid for maintenance *Child:* not recommended *Adult:* IV, 2–4 g up to 3 g/d *Child:* not recommended	*Onset:* 30 min *Duration:* 8 h $t_{1/2}$: 1–2 h *Onset:* rapid *Duration:* unknown $t_{1/2}$: 1–2 h
orphenadrine (Norflex, *Canadian:* Orphenace, Rhoxal-orphenadrine)	Muscle spasms Muscle relaxation	*Adult and child >12 y:* PO, 200–250 mg/d in divided doses; IV, 60 mg q 12 h *Child <12 y:* not recommended	*Onset:* 1–2 h *Duration:* 4–6 h $t_{1/2}$: 14–16 h
Centrally Acting Spasmolytics			
P baclofen (Lioresal; *Canadian:* Apo-Baclofen, Gen-Baclofen, Liotec, Nu-Baclo, PMS-Baclofen)	Spasticity	*Adult:* PO, 5–20 mg tid; Intrathecal, 5–25 mcg *Child:* not recommended	*Onset:* 3–4 d *Duration:* 24–48 h $t_{1/2}$: 3–4 h
baclofen (Kemstro)		*Adult:* oral disintegrating tablet	
tizanidine (Zanaflex, *Canadian:* Apo-Tizanidine, Gen-Tizanidine)	Spasticity MS Muscle relaxation	*Adult:* 2–4 mg tid; maximum dose 36 mg/d *Child:* not recommended	*Onset:* 1 h *Duration:* 3–6 h $t_{1/2}$: 2.5 h
gabapentin (Neurontin, *Canadian:* Apo-Gabapentin, Gen-Gabapentin, Novo-Gabapentin, Nu-Gabapentin, BCI-Gabapentin)	Spasticity	*Adult:* PO, 600–1200 mg/d in divided doses *Child:* not recommended	*Onset:* 30 min *Duration:* 8 h $t_{1/2}$: 5–7 h
Peripherally Acting Spasmolytics			
P dantrolene (Dantrium)	Athetosis cerebral palsy, MS, hemiplegia, paraplegia, Parkinson disease, spasticity, CVA, spinal cord injury Prevention of malignant hyperthermia Malignant hyperthermia (adult and child) Post-crisis follow-up	*Adult:* PO, 25–100 mg 2–4 times qid *Child <5y:* not approved *Child >5y:* 0.5 mg/kg bid, maximum 100 mg qid IV: 2.5 mg/kg 1 h before surgery PO: 4–8 mg/kg in divided doses 1–2 d before surgery with last dose 3–4 h after surgery IV: 1 mg/kg PO: 4–8 mg/kg in four divided doses for 1–3 d	*Onset:* 4–7 d *Duration:* dose related $t_{1/2}$: 7–9 h
botulinum toxin type A (Botox)	Chronic spasticity	*Adult:* 1 m; extremely individualized	*Onset:* 3 d–2 wk *Duration:* 3 mo $t_{1/2}$: 10 h

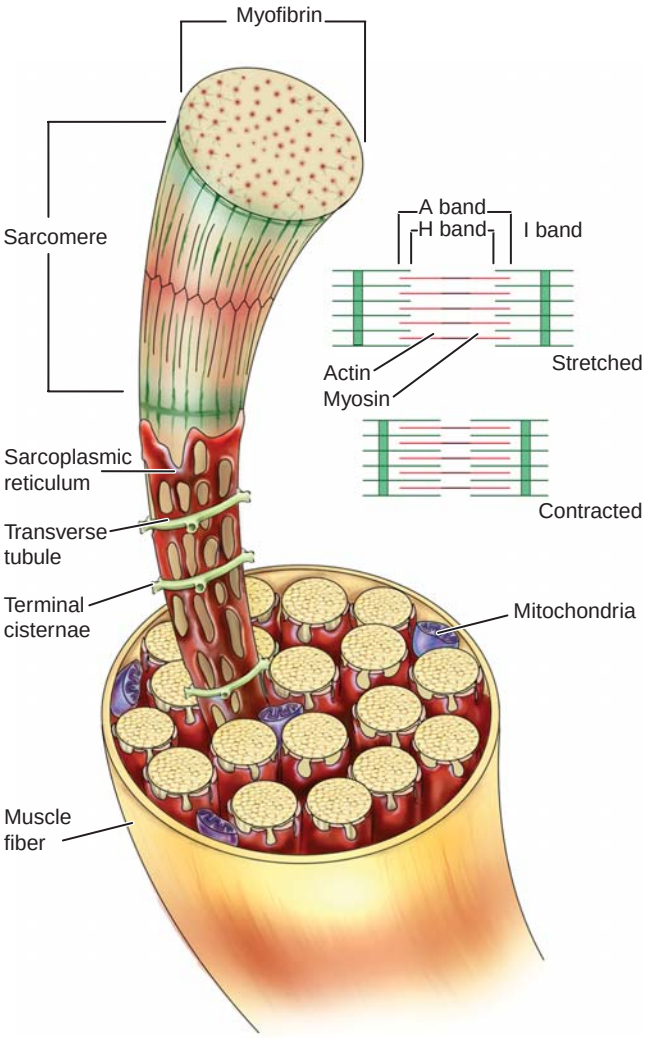


FIGURE 16.1 When stimulation stops, calcium ions are actively transported back into the sarcoplasmic reticulum, resulting in decreased calcium ions in the sarcoplasm. The removal of calcium ions restores the inhibitory action of troponin-tropomyosin; crossbridge action is impossible in this state.

Nursing Management of the Patient Receiving P Cyclobenzaprine

Core Drug Knowledge

Pharmacotherapeutics

Cyclobenzaprine is used to manage muscle spasms associated with acute musculoskeletal disorders, such as low back strain, muscle tenderness, or movement restriction due to musculoskeletal conditions. It is also used as supportive therapy in patients with tetanus or fibromyalgia.

Pharmacokinetics

Cyclobenzaprine is administered orally. It is well absorbed from the gastrointestinal (GI) tract and probably undergoes first-pass metabolism because plasma levels vary considerably among patients. Onset of skeletal muscle relaxation occurs in about 1 hour, and duration of action ranges from 12 to 24 hours. Optimal effects may take 1 to 2 days to fully develop. Cyclobenzaprine undergoes extensive metabolism and is excreted mainly as conjugated inactive metabolites in the urine and as unchanged drug through bile in the feces. Its half-life ranges from 1 to 3 days.

Pharmacodynamics

Cyclobenzaprine relieves muscle spasms through a central action, possibly at the level of the brain stem, with no direct action on the neuromuscular junction or the muscle involved. It reduces pain and tenderness and improves mobility. Because of its structural similarity to the tricyclic antidepressants (TCAs), cyclobenzaprine may reduce tonic somatic motor activity by influencing both alpha and gamma motor neurons. Cyclobenzaprine is ineffective for treating spasticity associated with cerebral or spinal cord disease or in children with cerebral palsy.

Contraindications and Precautions

Cyclobenzaprine is contraindicated for patients with hyperthyroidism because of a possible increased risk of arrhythmias or an exacerbation of tachycardia. Because of its similarity to the TCAs, cyclobenzaprine is also contraindicated for use within 14 days of administration of monoamine oxidase inhibitors (MAOIs).

In overdoses, TCAs cause conduction disturbances and have been associated with torsades de pointes (an atypical tachycardia) and death. Because it is closely related to the TCA amitriptyline, cyclobenzaprine should be used cautiously in patients with heart failure, cardiac arrhythmias, or atrioventricular block or other conduction disturbances, and in those who are in the acute recovery phase following myocardial infarction (MI).

Cyclobenzaprine possesses anticholinergic activity. Patients with increased intraocular pressure, angle-closure glaucoma, or urinary retention require careful monitoring. In addition, cyclobenzaprine should be used with caution in pregnant or breast-feeding women. Studies for determining safe use during pregnancy have not been performed.

Adverse Effects

The common adverse effects of cyclobenzaprine are related to its CNS depression and anticholinergic activity. The most common adverse effects are drowsiness, dizziness, and dry mouth. Other frequent adverse effects include fatigue, asthenia (loss of strength and energy), nausea and vomiting, constipation, dyspepsia, dysgeusia, blurred vision, headache, nervousness, and confusion. Serious adverse effects, including arrhythmias, seizures, and MIs, can occur because of cyclobenzaprine's similarity to the TCAs.

Drug Interactions

Interactions between cyclobenzaprine and CNS depressants or antimuscarinic drugs may be extensive. Cyclobenzaprine may also interact with tramadol, guanethidine, MAOIs, histamine-1 blocking agents, and various herbal remedies. These interactions are highlighted in Table 16.2.

Assessment of Relevant Core Patient Variables

Health Status

Assess the patient for a history of cyclobenzaprine hypersensitivity or pre-existing diseases such as hyperthyroidism, cardiac dysfunction, recent MI, and glaucoma. Also, assess

TABLE 16.2 Agents That Interact With P Cyclobenzaprine

Interactants	Effect and Significance	Nursing Management
CNS depressants • sedatives • tranquilizers • alcohol • opioids	Cyclobenzaprine, in combination with other CNS depressants, may induce additive effects, resulting in increased CNS depression.	Avoid this combination. Monitor for sedation and dizziness. Provide ambulatory assistance. Ensure patient's safety.
Tramadol	Cyclobenzaprine may react with tramadol, resulting in seizure activity.	Use of tramadol with cyclobenzaprine is not recommended. Monitor for seizure activity.
Guanethidine	Cyclobenzaprine may react with guanethidine, decreasing guanethidine's antihypertensive effect.	Use of guanethidine with cyclobenzaprine is not recommended. Monitor blood pressure. Discuss possible increased dosage of guanethidine with health care provider.
MAOIs	Although the mechanism of action is unknown, it is likely that concurrent administration of cyclobenzaprine and MAOIs enhances adrenergic activity, increasing the likelihood of oretic (hydrochlorothiazide) crisis, severe seizures, and death.	A minimum of 14 days should elapse between the discontinuance of MAOIs and the initiation of cyclobenzaprine therapy. Monitor for elevated temperature, confusion, or seizure activity.
Phytomedicinal herbs • Valerian • *Valeriana officinalis* • Kava kava • St. John's wort • gotu kola	Combination therapy can cause additive effects of sedation and dizziness, which can impair the patient's ability to undertake tasks that require mental alertness.	Monitor for sedation and dizziness. Provide ambulatory assistance. Ensure patient's safety. Dosage adjustments of either or both medications may be necessary.
Tricyclic antidepressants	Cyclobenzaprine is structurally similar to tricyclic antidepressants. When given concurrently, some of the more serious CNS reactions noted with the tricyclic antidepressants may occur.	Monitor for seizure activity. Monitor for arrhythmias.

for use of medications that may interact with cyclobenzaprine, such as MAOIs. Communicate positive findings to the prescriber before administering the drug. Also, assess for over-the-counter (OTC) drugs used for allergies or hay fever, because these drugs usually have an anticholinergic effect, and additive effects occur when anticholinergic drugs are given concurrently with cyclobenzaprine.

Life Span and Gender

In working with older adult patients, assess them for the anticholinergic and CNS depressant effects of cyclobenzaprine, because increased age makes a patient more susceptible to them.

In addition, it is important to evaluate the female patient's pregnancy and lactation status. Safety during pregnancy has not been documented, although cyclobenzaprine is a pregnancy category B drug. Distribution of the drug into breast milk has not been established, but TCAs are found in breast milk. Because of its structural similarity to the TCAs, cyclobenzaprine may also distribute into breast milk.

Lifestyle, Diet, and Habits

Assess for use of alcohol or any other CNS depressant, which increases the sedating effects of cyclobenzaprine. Advise the patient to assess the depth of sedation related to cyclobenzaprine use before driving a vehicle or attempting activities that require mental alertness or motor coordination. Long-term use of cyclobenzaprine may result in physical dependence. Assess for potential abuse of this drug.

Environment

Cyclobenzaprine may be administered in any environment, but it is most often given on an outpatient basis. Because it can cause sedation, discuss with the patient possible hazards in the home environment.

Nursing Diagnosis and Outcome

- Risk for Injury related to CNS depressant effects and potential cardiovascular effects.
  Desired outcome: *The patient will remain free from injury throughout therapy.*

Planning and Intervention

Maximizing Therapeutic Effects

Have the patient take cyclobenzaprine with a full glass of water at evenly spaced intervals. Coordinate physical modalities such as physical therapy, whirlpool, and cold or hot compresses to the affected area.

Minimizing Adverse Effects

While the patient is hospitalized, ensure the patient's safety by keeping the bed in the lowest position with the side rails up. Accompany the patient during ambulation until the degree of

sedation is ascertained. Also, caution the patient about the potential for orthostatic hypotension resulting in dizziness and teach the patient to change positions slowly. Finally, remind the patient of the potential for synergistic effects when cyclobenzaprine is taken with other CNS depressants, especially alcohol. Because cyclobenzaprine can cause physical dependence with long-term use, slowly withdrawing the drug over 1 to 2 weeks is important to prevent abstinence syndrome (see Chapter 9).

Providing Patient and Family Education

- Instruct patients to take cyclobenzaprine exactly as prescribed. If patients miss a dose, they should take it as soon as they remember but should never double the dose.
- Explain that adverse effects such as mild drowsiness, dizziness, or clumsiness may accompany cyclobenzaprine therapy. Although these symptoms will often improve with time, patients must refrain from driving or performing hazardous tasks until their level of sedation is ascertained. Advise patients to contact the prescriber if sedation persists or is bothersome. Also, emphasize the need to refrain from alcohol, which may exacerbate these adverse effects.
- Advise patients to contact the prescriber immediately if they experience severe headache, confusion, hallucinations, or sudden or increased weakness. Female patients should contact the prescriber immediately if they suspect they may be pregnant.
- Instruct patients to avoid using any OTC or prescription drugs without first consulting the prescriber. Many of these drugs can cause dangerous adverse effects when taken with cyclobenzaprine.
- Finally, instruct patients never to abruptly cease cyclobenzaprine if therapy has been with high doses or over a prolonged period; rather, they should decrease the dosage gradually over 2 weeks.

Ongoing Assessment and Evaluation

Constantly evaluate the patient's safety. Monitor the level of sedation and report to the prescriber if it is severe. In addition to CNS symptoms, monitor for GI symptoms, such as gastric distress or constipation. Provide analgesics for headache, offer small and frequent meals to decrease GI upset, and establish a bowel program if constipation becomes a problem. Successful cyclobenzaprine therapy is marked by decreased muscle spasms and no injury to the patient related to adverse effects.

Drugs Closely Related to P Cyclobenzaprine

Drugs that are similar to cyclobenzaprine include carisoprodol, chlorzoxazone, metaxalone, methocarbamol, and orphenadrine. Like cyclobenzaprine, they all work by affecting the CNS rather than the muscle itself. Thus, patient safety is a priority with all of these drugs.

Carisoprodol

Carisoprodol (Soma) is given orally. Although carisoprodol is itself a nonscheduled drug, it can be very addictive because it is metabolized into meprobamate, a Schedule IV controlled substance. Significant adverse effects include hypomania at higher than recommended doses, withdrawal syndrome, and hypersensitivity. Patients taking carisoprodol may also experience an idiosyncratic reaction inducing weakness, visual or motor disturbances, confusion, or euphoria. Hypersensitivity or idiosyncratic reactions generally occur within administration of the first 4 doses. Two factors have led to the diminished use of carisoprodol: its ability to produce dependence and the availability of newer agents.

Chlorzoxazone

Chlorzoxazone (Paraflex, Parafon Forte) has been used for many years. It is metabolized in the liver; therefore, it should be used with caution in patients with hepatic disease, because it may cause hepatotoxicity ranging from a mild elevation in hepatic enzymes to hepatic necrosis. In addition, a metabolite of chlorzoxazone is rapidly excreted in the urine, and the drug should be used with caution in patients with renal impairment because it may alter excretion and possibly cause toxicity. Chlorzoxazone requires multiple daily doses and is more expensive than cyclobenzaprine.

Metaxalone

Metaxalone (Skelaxin) is another oral muscle relaxant. It produces less sedation than cyclobenzaprine but requires 4 times a day dosing and is extremely expensive. Metaxalone is contraindicated in patients with severe renal and hepatic impairment. Before administration, baseline renal and hepatic function should be evaluated, especially if the patient will have long-term therapy. Periodic liver function tests (LFTs) should be performed throughout therapy.

Patients taking metaxalone may experience paradoxical muscle cramps or a mild withdrawal syndrome if the drug is discontinued abruptly.

Methocarbamol

Methocarbamol (Robaxin) may be administered by oral, intramuscular (IM), and intravenous (IV) routes. In addition to its use for muscle spasm, methocarbamol is used in managing tetanus. Methocarbamol may cause brown, brown-black, or green urine. Like chlorzoxazone, it causes less sedation than cyclobenzaprine. Contraindications to methocarbamol include hepatic or renal disorders, age younger than

MEMORY CHIP

P Cyclobenzaprine

- Centrally acting spasmolytic is used for muscle spasms; ineffective for spasticity
- Most common adverse effect: sedation; safety is a nursing priority
- Most serious adverse effects: occur with abrupt withdrawal and include agitation, auditory or visual hallucinations, seizures, or psychotic symptoms
- **Life span alert: Older adult patients are more prone to sedation and anticholinergic effects.**
- Maximizing therapeutic effects: Take medication exactly as prescribed.
- Minimizing adverse effects: Avoid use of other CNS depressants.
- Most important patient education: Never abruptly stop medication; withdraw medication over a 2-week period.

12 years or older than 60 years, and pregnancy. It is used with caution in patients with seizure disorders because it may exacerbate seizure activity.

When methocarbamol is given intravenously, the rate should not exceed 300 mg/min (3 mL of 10% injection). Because the solution is hypertonic, extravasation may occur, resulting in thrombophlebitis, sloughing, and pain at the injection site.

Orphenadrine

Orphenadrine (Norflex) is another centrally acting muscle relaxant. Administration is oral or parenteral. In addition to having CNS effects similar to those of other centrally acting skeletal muscle relaxants, orphenadrine may induce aplastic anemia or anaphylactic reaction. When given intravenously, it should not be diluted and should be given slowly over 5 minutes.

Orphenadrine should be used with caution in conditions that are affected by its anticholinergic and antihistaminic effects, which include bladder obstruction, prostatic hypertrophy, GI obstruction, peptic ulcer disease, gastroesophageal reflux disease, asthma, glaucoma, and myasthenia gravis. Orphenadrine should also be used with caution in older patients who do not tolerate anticholinergics well. Additionally, caution should be taken when administering to patients with cardiac insufficiency or thyrotoxicosis. The safety of orphenadrine in children or pregnant or breast-feeding women has not been established.

Orphenadrine interacts with haloperidol, worsening schizophrenic symptoms and possibly contributing to the development of tardive dyskinesia. It also interacts with amantadine, with resultant additive anticholinergic effects. Orphenadrine decreases the action of phenothiazines when given concurrently.

Drug Significantly Different From P Cyclobenzaprine

Diazepam (Valium) is a benzodiazepine that can produce any level of CNS depression required, including sedation, hypnosis, skeletal muscle relaxation, antiepileptic activity, or coma. Diazepam and baclofen are the only two centrally acting drugs that are used for spasticity as well as muscle spasm. Although diazepam is an extremely effective muscle relaxant, its use as a maintenance drug for spasms is limited because of its potential for physical and psychological dependence. Additionally, withdrawing diazepam abruptly may induce seizure activity. For further information on benzodiazepines, see Chapter 18.

C CENTRALLY ACTING SPASMOLYTICS

The centrally acting spasmolytics work in the CNS to reduce excessive reflex activity and to allow muscle relaxation. The prototype for the centrally acting spasmolytics is baclofen (Lioresal), which is very effective and inexpensive.

Nursing Management of the Patient Receiving P Baclofen

Core Drug Knowledge

Pharmacotherapeutics

Baclofen relieves some components of spinal spasticity—involuntary flexor and extensor spasms and resistance to passive movements. It is useful in MS, cerebral palsy, and traumatic injury to the spinal cord. Baclofen is not useful in treating spasms that follow a CVA or stroke, or those that occur in Parkinson disease or Huntington chorea (see Table 16.1). Surgically implanted pumps are used to deliver intrathecal baclofen to patients who have long-term needs or poor control with oral medications. Baclofen has been used in patients with focal dystonic movements, including torticollis (wry neck). It has been used with some success in Meige syndrome (blepharospasm-oromandibular dystonia) and stiff-man syndrome, also known as Moersch-Woltmann syndrome. Stiff-man syndrome occurs primarily in men. It is characterized by muscular rigidity accompanied by paroxysmal painful spasms precipitated by physical or emotional stimuli.

Baclofen has been effective in treating intractable hiccups. It may also be used to manage trigeminal neuralgia and various types of neuropathic pain, including migraine headaches.

Pharmacokinetics

Baclofen is rapidly absorbed orally and peaks in 2 to 3 hours. It is distributed throughout the body and crosses the blood–brain barrier. Baclofen also crosses the placenta and passes into breast milk. The half-life ranges from 2 to 4 hours. The kidneys excrete 70% to 85% of a dose as unchanged drug and active and inactive metabolites. The liver metabolizes the remainder, which is excreted through the feces.

Pharmacodynamics

Baclofen is a derivative of the neurotransmitter gamma-aminobutyric acid (GABA) and acts specifically at the spinal end of the upper motor neurons at $GABA_B$ receptors to cause hyperpolarization. This action reduces excessive reflex activity underlying muscle hypertonia, spasms, and spasticity and allows muscle relaxation. The mechanism of action explains why baclofen is not used for spasticity resulting from CVA or Parkinson disease, because these disorders involve lesional or functional impairment of basal ganglia coordination, in an area of the CNS above the spinal motor neurons.

Contraindications and Precautions

Baclofen is contraindicated in anyone who has demonstrated previous hypersensitivity to it. It is also contraindicated for spasticity of cerebral origin or for reducing the rigidity of parkinsonism or Huntington chorea because it is ineffective in these disorders.

Baclofen is classified as a pregnancy category C drug and should be used during pregnancy only when the benefits to the pregnant woman outweigh the risks to the fetus. Baclofen appears in small amounts in breast milk; therefore, caution should be used in administering it to breast-feeding women. Baclofen has not been approved for use in children younger than 12 years.

Baclofen is used with caution in patients who have seizure disorders. It has caused deterioration in seizure control and electroencephalographic changes in patients with epilepsy. When given to patients with CNS disorders, such as cerebral hemorrhage or a prior CVA, baclofen may increase the risk for developing CNS, respiratory, or cardiovascular depression and ataxia. Baclofen can increase blood glucose concentrations and should therefore be used cautiously in patients with diabetes mellitus. Patients with pre-existing psychiatric disorders are more likely to develop baclofen-induced psychiatric disturbances. Baclofen should also be used with caution in patients who have renal impairment, because most of the drug is excreted unchanged in the urine.

Adverse Effects

The most common adverse effects of baclofen therapy include drowsiness, weakness, dizziness and lightheadedness, headache, nausea and vomiting, hypotension, constipation, lethargy and fatigue, confusion, insomnia, and increased urinary frequency. Other effects in the CNS include euphoria, excitement, depression, and hallucinations. Baclofen may also cause paresthesias, myalgias, or tinnitus. The patient may experience difficulty with coordination, tremors, rigidity, or ataxia. The patient may also experience vision disturbances such as nystagmus, strabismus, miosis, mydriasis, or diplopia. Adverse effects in the GI system include xerostomia, anorexia, dysgeusia, abdominal pain, and diarrhea. Cardiovascular adverse effects, such as palpitations, angina, excessive diaphoresis, and syncope, are possible. Genitourinary (GU) effects may include urinary incontinence or retention, dysuria, erectile dysfunction, ejaculation dysfunction, and nocturia. Integumentary adverse effects may include rash and pruritus.

Drug Interactions

Baclofen has the potential to cause clinically important interactions with other CNS depressants or TCAs. Baclofen has also been reported to cause false-positive results on tests for occult blood in the stool. Additionally, baclofen may induce elevations in levels of aspartate aminotransferase (AST), alkaline phosphatase, or serum glucose (Table 16.3).

Assessment of Relevant Core Patient Variables

Health Status

Assess for a history of baclofen hypersensitivity or pre-existing disorders that contraindicate the use of baclofen. Also assess for muscle spasms and their causes. Baclofen therapy does not affect skeletal muscle spasms resulting from CVA or parkinsonism.

Perform a physical examination that includes baseline assessments of neurologic function, cardiac function, kidney function, and muscle strength and spasticity. Laboratory assessments should include baseline liver and kidney function values and blood glucose level.

Life Span and Gender

Older patients are more susceptible to baclofen-induced sedation and psychiatric disturbances, including hallucinations, excitation, and confusion. Therefore, continually assess older patients taking baclofen for such adverse effects. Evaluate the patient for pregnancy. Baclofen may be used during pregnancy when the benefits to the pregnant woman outweigh the risks to the fetus. Baclofen should not be used in children younger than 12 years.

Lifestyle, Diet, and Habits

Caution the patient about the concurrent use of alcohol and baclofen. Alcohol may increase the risk for CNS depression and other CNS adverse effects. Also, caution patients to

TABLE 16.3 Agents That Interact With P Baclofen

Interactants	Effect and Significance	Nursing Management
CNS depressants • alcohol • benzodiazepines • barbiturates • opioids	In combination with dantrolene, CNS depressant drugs may have additive effects, increasing CNS depression.	Monitor for increased sedation. Institute safety measures, especially with ambulation.
Nonsteroidal anti-inflammatory agents	Baclofen may decrease the clearance of NSAIDs and increase the potential for renal toxicity.	Monitor fluid intake and output.
Phytomedicinals • *Valeriana officinalis* • kava kava • St. John's wort • gotu kola	In combination with dantrolene, phytomedicinals may have additive effects, increasing CNS depression.	Monitor for increased sedation. Institute safety measures, especially with ambulation.
Tricyclic antidepressants	Baclofen and TCAs can potentiate muscle relaxation and enhance anticholinergic effects. This combination may result in severe weakness, memory loss, and loss of muscle tone.	Monitor for adverse effects. Provide ambulatory assistance. Increase fluids or consume sugarless candies for dry mouth. Monitor for urinary retention or constipation.

assess their level of alertness (which may be affected by CNS depression) before attempting to drive, use machinery, or perform activities that require concentration.

Environment

Oral baclofen can be given in any environment and is generally self-administered in the home. Intrathecal baclofen administration requires a surgical procedure to implant the device. The prescriber refills the reservoir every 4 to 12 weeks, depending on the daily dose.

Nursing Diagnoses and Outcomes

- Acute Pain related to headache, muscle pain, GI disturbances, or rash
  Desired outcome: *The patient will be provided with measures to decrease the discomfort of drug therapy and the possibility of nonadherence.*
- Risk for Disturbed Sensory Perception related to visual changes, vestibular dysfunction, and somatosensory changes
  Desired outcome: *The patient will be protected from injury if dizziness, weakness, visual changes, or perceptual changes occur.*

Planning and Intervention

Maximizing Therapeutic Effects

Have the patient take baclofen with a full glass of water at evenly spaced intervals. For the patient with GI distress, coordinate small, frequent meals.

Minimizing Adverse Effects

Ensure the patient's safety by keeping the bed in the lowest position with the side rails up. Assist ambulatory patients with locomotion because sedation and muscle weakness may increase. Advise the patient to change positions slowly to prevent dizziness. Remind the patient to assess his or her sedation level prior to driving or performing tasks that require concentration.

Withdrawing baclofen abruptly may result in agitation, auditory and visual hallucinations, seizures, psychotic symptoms, or, most commonly, acute exacerbations of spasticity. Ensure gradual reduction of the baclofen dosage over 1 to 2 weeks.

Abrupt cessation of intrathecal baclofen may cause severe spasticity and rigidity. In rare cases, rhabdomyolysis, a potentially fatal complication, may occur.

Providing Patient and Family Education

- Education for patients receiving baclofen is similar to that for patients receiving cyclobenzaprine. As with cyclobenzaprine, caution patients to avoid sudden cessation of the drug; instead, patients should taper doses of the drug over 2 weeks.
- Emphasize the importance of safety related to sedation. Be sure patients understand to avoid any activity that requires concentration, especially driving, until their level of sedation is established.
- Another major point is to remind patients to refrain from alcohol or any other CNS depressant agents. Advise patients that it may take them up to 1 month to experience the full benefit of baclofen therapy.
- Advise patients of the importance of contacting the prescriber should they experience severe headaches, confusion, hallucinations, or sudden or increased weakness. Advise all female patients to contact the prescriber immediately if they become pregnant.
- Additionally, instruct patients with diabetes to use capillary blood glucose monitoring because baclofen may cause blood and urine glucose levels to rise. These patients should notify their prescribers if serum glucose level elevations persist.
- For patients receiving intrathecal baclofen, teach the patient and family aseptic technique and how to assess the integrity of the catheter and infusion system.

Ongoing Assessment and Evaluation

To ensure safety, assess the patient for the CNS effects of baclofen throughout therapy. Monitor for the emergence of hallucinations or psychotic episodes and consult with the prescriber immediately about the possibility of reducing the dose or discontinuing the drug. Also, monitor the patient for integumentary, GI, or GU system complaints. Suggest approaches for dealing with minor symptoms, such as analgesics for headache or small, frequent meals for GI upset. Help establish a bowel program if constipation occurs. For GU effects such as erectile dysfunction, refer the patient to the prescriber and ensure that the patient does not abruptly stop the medication. Box 16.1 provides guidelines for ensuring successful baclofen therapy in the home.

Therapeutic monitoring during baclofen therapy will show improvement in symptoms of spasticity and a decrease in resistance to passive movement of limb joints.

Drug Closely Related to P Baclofen

Tizanidine (Zanaflex) is an oral agent used to treat spasticity related to spinal cord pathology and MS. It is structurally and pharmacologically similar to clonidine, an alpha-2 adrenergic agonist. Although tizanidine and baclofen are equally effective in treating spasticity, tizanidine is much more expensive.

Tizanidine is used cautiously in patients with hypotension, hepatic disease, psychosis, or renal impairment. An important interaction with tizanidine occurs with oral contraceptives, fluvoxamine, and ciprofloxacin because they are CYP1A2 inhibitors. Tizanidine also has additive effects when given with other antihypertensive agents, alpha-2 agonists, or ethanol. Adverse effects parallel those of other alpha-2 adrenergic agonists, including dry mouth, drowsiness, dizziness, GI disturbances, and liver function abnormalities. Because of its adrenergic action, hypotension and orthostatic hypotension may also occur. Tizanidine produces greater drowsiness and sedation than baclofen. Slow upward titration of the dose can minimize adverse effects. Patients on long-term therapy should have baseline LFTs performed prior to starting therapy and repeated at 3 and 6 months, and then periodically thereafter.

Drug Significantly Different From P Baclofen

Gabapentin (Neurontin) is a miscellaneous antiepileptic drug that has demonstrated efficacy in the management of neuropathic pain and spasticity. It is useful in managing

BOX 16.1
COMMUNITY-BASED CONCERNS

Intrathecal Baclofen Therapy

Surgically implanted drug delivery pumps are frequently used to deliver intrathecal infusions of baclofen in long-term treatment of patients with spasticity that is not adequately controlled by oral baclofen. These patients (particularly those with multiple sclerosis and traumatic spinal cord lesions) require an alternate route of administration because very little oral baclofen reaches the spinal fluid.

Intrathecal therapy is not appropriate for every patient. Prior to initiating intrathecal therapy, the patient is evaluated by a team of health care professionals, including a physician who specializes in rehabilitation (physiatrist), a physical therapist, an occupational therapist, a registered nurse, and a social worker. Once approved, the patient receives a test dose of intrathecal baclofen to evaluate the effect on the patient's spasticity. If the test dose positively affects the spasticity, the patient is then scheduled for surgical implantation of a small pump underneath the skin around the waistline. The pump is refilled by a health care provider every 2 to 3 months.

Benefits of intrathecal baclofen therapy include:

- Promotion of a more active lifestyle, better sleep, and reduced need for oral medicines.
- Decreased frequency and severity of adverse effects compared to those with oral baclofen.
- Ability to adjust infusion rates that vary over a 24-hour period.
- Pain and discomfort from spasms and spasticity are often reduced or eliminated.

The patient is monitored by the community health nurse. The nurse does the following: ensures that the infusion equipment remains patent and in good operating condition, evaluates the patient's understanding of the treatment, and reinforces patient teaching during each visit.

MEMORY CHIP

P Baclofen

- Centrally acting spasmolytic is used for muscle spasms or spasticity
- Major contraindication: patients who use spasticity to maintain posture or balance
- Most common adverse effect: sedation; safety is a nursing priority
- Most serious adverse effects: occur with abrupt withdrawal and include agitation, auditory or visual hallucinations, seizures, or psychotic symptoms
- **Life span alert: Older patients are more prone to sedation and other effects on the central nervous system.**
- Maximizing therapeutic effects: Administer at evenly spaced intervals.
- Minimizing adverse effects: Assist in changing positions slowly; withdraw medication over a 2-week period.
- Most important patient education: Never abruptly stop medication.

spasticity associated with MS; this is an off-label indication. Although its exact mechanism of action is not known, it is thought to interact with voltage-gated calcium channels to decrease pain and spasticity. Gabapentin is orally administered and rapidly absorbed. It is highly lipid soluble, crosses the blood–brain barrier, and is widely distributed in the CNS. Gabapentin is not metabolized and is excreted unchanged in the urine; therefore, monitoring renal function is important. Gabapentin is unusual in that it does not interact with other drugs. Common adverse effects include drowsiness, somnolence, nausea, and fatigue. For additional information regarding gabapentin, see Chapter 21.

PERIPHERALLY ACTING SPASMOLYTICS

Peripherally acting spasmolytics relax muscles through direct action on the skeletal muscle fibers. They neither interfere with neuromuscular communication nor have CNS effects. Dantrolene (Dantrium) is the most frequently used peripheral agent and the prototype for the peripherally acting spasmolytics.

Nursing Management of the Patient Receiving P Dantrolene

Core Drug Knowledge

Pharmacotherapeutics

IV dantrolene is the drug of choice, when accompanied by supportive measures, for acute treatment of malignant hyperthermia, a life-threatening complication of general anesthesia. Preoperatively, it can be used orally or intravenously to prevent malignant hyperthermia in patients considered at risk.

Dantrolene has several other pharmacotherapeutic uses. It has been effective in treating upper motor neuron disorders, such as hereditary spastic paraplegia. It has also been used to treat heatstroke and to prevent and treat the rigors associated with amphotericin B. It is useful in managing spasticity resulting from spinal cord and cerebral injuries, MS, cerebral palsy, and possibly CVA. Dantrolene is not effective in treating acute muscle weakness of local origin or muscle weakness resulting from rheumatoid spondylitis, arthritis, or bursitis.

Pharmacokinetics

Approximately 35% of an oral dose of dantrolene is absorbed; peak plasma concentrations are reached in approximately 5 hours. The liver metabolizes dantrolene to weakly active metabolites, which are excreted in the urine. The elimination half-life is reported to be about 9 hours in healthy adults and 7.3 hours in children. Therapeutic effects in patients being treated for upper motor neuron disorders may not appear for 1 week or more. Dantrolene crosses the placenta and enters breast milk.

Pharmacodynamics

Dantrolene reduces the force of contraction of skeletal muscle through a direct effect on muscle cells. It reduces the

amount of Ca^{2+} released from the sarcoplasmic reticulum, thereby uncoupling (relaxing) muscle contraction from excitation. Interference with the release of Ca^{2+} from the sarcoplasmic reticulum may prevent the increase in intracellular Ca^{2+}, which activates the acute catabolic events of malignant hyperthermia. Dantrolene has little or no effect on contraction of cardiac or intestinal smooth muscle. It may decrease hyperreflexia, muscle stiffness, and spasticity in patients with upper motor neuron disorders.

Contraindications and Precautions

Dantrolene is contraindicated in patients with active liver disease because of its associated liver toxicity.

Dantrolene causes weakness because of its generalized reduction of muscle contraction. Thus, it is given very cautiously in patients who rely on spasticity to maintain an upright posture and balance, such as patients with cerebral palsy. Dantrolene should be used with caution in patients with pre-existing myopathy or neuromuscular disease with respiratory depression. No contraindications apply to IV administration of dantrolene to prevent or acutely treat malignant hyperthermia crisis. The risk of perioperative complications is increased in patients with these conditions who receive dantrolene for prevention of malignant hyperthermia.

Dantrolene should also be used with caution in patients with cardiac disease and pulmonary dysfunction, particularly chronic obstructive pulmonary disease. For patients with cardiac disease, dantrolene can precipitate pleural effusions or pericarditis. In patients with pulmonary dysfunction, dantrolene can precipitate respiratory depression.

Dantrolene is classified as a pregnancy category C drug; therefore, pregnant or lactating women should avoid its use.

Adverse Effects

The most common adverse effect of dantrolene therapy is muscle weakness. Manifestations of such muscle weakness may include drooling, slurred speech, drowsiness, dizziness, malaise, and fatigue. Serious adverse effects seen with dantrolene therapy include potentially fatal hepatitis, seizures, and pleural effusion with pericarditis.

In the GI system, symptoms include diarrhea, constipation, GI bleeding, anorexia, difficulty swallowing, abdominal cramps, and nausea and vomiting. Diarrhea is usually dose-dependent and transient, but in some cases it can be severe, and the drug may have to be withheld. Hematologic adverse reactions with dantrolene therapy include aplastic anemia, leukopenia, and lymphocytic lymphoma.

Rash, acne, abnormal hair growth, and photosensitivity are possible integumentary effects. IV dantrolene may cause edema and thrombophlebitis. Rarely, IV administration may cause erythema and urticaria.

Drug Interactions

Drugs that interact with dantrolene include CNS depressants, clofibrate, estrogens, verapamil, and warfarin. Table 16.4 discusses these potential interactions.

Assessment of Relevant Core Patient Variables

Health Status

Elicit a comprehensive health history, including any history of active hepatitis, estrogen use in women older than 35 years, impaired cardiac or pulmonary function, any liver disease, or spasticity used to sustain upright posture and balance in locomotion or to obtain or maintain increased function. Communicate positive findings for any of these factors to the prescriber.

Perform a physical examination before initiating therapy. Assessment of the musculoskeletal system should include the patient's posture, ability to walk, reflexes, and muscle tone. Document the amount and location of spasticity. Other assessments should include the CNS and GI systems. Laboratory tests should include a complete blood count (CBC) and AST, alanine aminotransferase, alkaline phosphatase, and total bilirubin levels.

Life Span and Gender

Consider the patient's age relative to dantrolene therapy. Hepatotoxicity occurs most commonly in patients older than 30 years of age, especially women older than age 35 who are taking estrogens. It generally occurs 3 to 12 months after starting dantrolene therapy. Children younger than 5 years should not receive dantrolene. Older patients are more vulnerable to the adverse effects of dantrolene. Before giving dantrolene, assess the patient for pregnancy and breastfeeding, because the safety of dantrolene therapy has not been established for pregnant or lactating women.

Lifestyle, Diet, and Habits

Dantrolene capsules contain lactulose. Therefore, assess the patient for lactose intolerance before the drug is given. Assess for alcohol consumption because alcohol increases the sedative properties of dantrolene.

Environment

Caution the patient about the potential for photosensitivity. Advise patients to wear appropriate clothing and sunscreen whenever they are in direct sunlight. Because dantrolene causes muscle weakness, also discuss with the patient any barriers in the home (such as stairs) that may affect dantrolene therapy. In addition, caution the patient to assess the drug's effects before attempting to ambulate without assistance.

Nursing Diagnoses and Outcomes

- Risk for Injury related to muscular weakness
  Desired outcome: *The patient will be injury free despite muscular weakness.*
- Diarrhea or Constipation related to drug effects
  Desired outcome: *The patient will maintain baseline bowel habits.*
- Risk for Disturbed Sensory Perception: Kinesthetic, related to dizziness, malaise, and fatigue
  Desired outcome: *The patient will remain free of injury from adverse effects.*
- Disturbed Body Image related to drug-related dermatologic effects
  Desired outcome: *Any adverse effects will be resolved by the end of therapy.*

Planning and Intervention

Maximizing Therapeutic Effects

Administer dantrolene with food or milk to avoid gastric distress. For patients with difficulty swallowing, mix the

TABLE 16.4 Agents That Interact With P Dantrolene

Interactants	Effect and Significance	Nursing Management
Calcium channel blockers • diltiazem • verapamil	In combination with dantrolene, calcium channel blockers may increase hyperkalemia and myocardial depression.	Avoid coadministration, if possible. Monitor cardiac function. Monitor potassium levels.
Clindamycin	In combination with dantrolene, clindamycin may increase neuromuscular blockade.	Monitor for increased effects of dantrolene.
Clofibrate	Clofibrate may decrease plasma protein binding of dantrolene, resulting in decreased effects of dantrolene.	Monitor for efficacy of dantrolene therapy.
CNS depressant drugs • alcohol • benzodiazepines • barbiturates • opioids	In combination with dantrolene, CNS depressant drugs may have additive effects, increasing CNS depression.	Monitor for increased sedation. Institute safety measures, especially with ambulation.
Estrogens	Mechanism of interaction is unknown; women older than 35 years are at risk for hepatotoxicity when estrogens and dantrolene are coadministered.	Monitor for signs of hepatotoxicity. Coordinate periodic liver function tests for long-term therapy.
Phytomedicinals • *Valeriana officinalis* • kava kava • *Piper methysticum* • gotu kola	In combination with dantrolene, phytomedicinals may have additive effects, increasing CNS depression.	Monitor for increased sedation. Institute safety measures, especially with ambulation.
Psychotropic drugs • MAOIs • phenothiazines	In combination with dantrolene, psychotropic drugs may increase neuromuscular blockade.	Monitor for increased effects of dantrolene.
warfarin	Warfarin may decrease plasma protein binding of dantrolene, resulting in decreased effects of dantrolene.	Monitor for efficacy of dantrolene therapy.

contents of the capsule with fruit juice and administer immediately. If extended-release capsules or tablets are prescribed, do not open or crush them.

Minimizing Adverse Effects

To avoid injury, supervise the transfer or ambulation of patients taking dantrolene. Also, provide frequent skin care and hygiene measures to prevent skin breakdown and request treatment for acne if appropriate. Protecting the patient from exposure to ultraviolet light is important, as is providing sunscreen if exposure is inevitable.

Therapy is initiated at low doses and gradually increased to minimize dose-related side effects. This practice also determines the minimum effective dose and allows a smooth induction of antispastic effects.

Providing Patient and Family Education

- Before the drug is given, explain to patients and family that the drug is being used to relieve spasticity and that muscle weakness may occur. Family members should be instructed to assist patients with ambulation and ensure safety precautions.
- Inform patients that one of the most dangerous adverse effects of dantrolene therapy is hepatitis. Write down a list of symptoms for patients to report to the prescriber immediately, including loss of appetite, nausea, vomiting, yellowed skin or eyes, and changes in color of urine or stool. Also, explain that regular follow-up medical care, including blood tests, is necessary to monitor the effects of the drug on the body.
- Advise patients that other adverse effects may occur, such as drowsiness, dizziness, GI upset, diarrhea or constipation, or rash. Discuss self-care measures to alleviate common symptoms and urge patients to contact the prescriber if the symptoms do not abate. To avoid photosensitivity, advise patients to wear appropriate clothing and use sunscreen when in direct sunlight.

Ongoing Assessment and Evaluation

Monitor for improvement in symptoms of spasticity and decrease in resistance to passive movement of the limb joint. Beneficial effects in spasticity may take 1 week or more to appear. Assist ambulatory patients with locomotion, because muscle weakness may increase.

Monitor for signs of adverse effects, especially hepatitis and hematologic effects. Coordinate periodic laboratory tests to evaluate liver function and the CBC. Withhold dantrolene and contact the prescriber if clinical signs of hepatitis appear.

CRITICAL THINKING SCENARIO

Adjusting to Dantrolene Therapy

J.J. was born with cerebral palsy, which has been successfully managed medically for 18 years. He recently finished high school and enrolled in a community college. During his second week at college, he began having uncontrollable muscle spasms that were painful and embarrassing to him. The college health service recommended treating the spasms with dantrolene.

1. Prioritize the assessment factors that the college health service nurse must consider before and during therapy.
2. Suggest steps that may need to be taken at J.J.'s school as a result of the drug therapy.

Drug Closely Related to P Dantrolene

Botulinum toxin type A (Botox) is a neurotoxin used for its muscle-relaxing properties. It is a protein that is produced by the anaerobic bacterium *Clostridium botulinum*. As many as seven serotypes of botulinum neurotoxin exist, but only types A and B are in clinical use at this time. Botulinum toxin type B (Myobloc) is less potent and shorter-acting than botulinum toxin type A.

Botulinum toxin type A is produced under controlled laboratory conditions and is given in extremely small therapeutic doses (0.05–0.1 mL per injection site). It blocks neuromuscular conduction by binding to receptor sites on motor nerve terminals, entering nerve terminals, and inhibiting the release of acetylcholine. Conditions that are treated with botulinum toxin injections include muscle contraction headaches, chronic muscle spasms in the neck and back, torticollis (severe neck muscle spasms), myofascial pain syndrome, and spasticity from MS or stroke. Botulinum toxin type A is given as an IM injection. Gradual relaxation of muscle spasm develops 1 to 2 weeks after the injection. The reduction of muscle spasm lasts for 3 to 4 months, and pain relief can last even longer. Potential adverse effects from the injection may include temporary increase in pain, weakness in the muscles injected, body aches, dry mouth, hoarseness, and flu-like symptoms.

In 2002, the FDA approved botulinum toxin type A (Botox Cosmetic) for cosmetic use. It has been successfully used to treat severe glabellar (frown) lines and is approved for use in adult patients up to 65 years of age. When botulinum toxin type A is injected into the muscles in a particular area of the face, those muscles cannot "scrunch up" for a period of time because they are paralyzed. The effect lasts for 3 to 6 months. Teach your patient the importance of receiving this type of treatment in a medical facility rather than at a "Botox party."

MEMORY CHIP

P Dantrolene

- Peripherally acting spasmolytic is used for muscle spasms or spasticity.
- Drug of choice for preventing or treating malignant hyperthermia
- Major contraindications: patients who use spasticity to maintain posture or balance (such as patients with cerebral palsy) or who have active hepatic disorders
- Most common adverse effect: muscle weakness; safety is a nursing priority
- Most serious adverse effect: fatal hepatitis, especially in women older than 35 years who are taking estrogens
- Maximizing therapeutic effects: Give with food or milk to decrease GI distress.
- Minimizing adverse effects: Assist with ambulation.
- Most important patient education: Advise patients of symptoms of hepatitis and the importance of notifying the prescriber should any occur.

Chapter Summary

- Drugs used to manage muscle spasm and spasticity are divided into muscle relaxants and spasmolytics. Muscle spasm is a sudden, violent, involuntary contraction of a muscle or group of muscles.
- Cyclobenzaprine (Flexeril) is the prototype for centrally acting muscle relaxants.
- Centrally acting muscle relaxants do not act directly on painful muscles; rather, they work by their CNS depressant activity.
- Most centrally acting muscle relaxants are not effective in treating spasticity.
- In addition to their CNS depressant effects, centrally acting muscle relaxants have anticholinergic and antihistaminic effects.
- Safety is a primary concern for patients receiving centrally acting muscle relaxants and spasmolytics.
- Centrally acting muscle relaxants should be given with caution to older adults. They are not indicated for use in children.
- Spasticity is a prolonged increased tone in muscles that may lead to contraction.
- Baclofen (Lioresal) is the prototype centrally acting spasmolytic drug.
- Gabapentin (Neurontin) is a miscellaneous antiepileptic drug that also has spasmolytic properties.
- Dantrolene (Dantrium) is a peripherally acting spasmolytic that affects spasticity within the muscle fibers.
- Botulinum toxin type A (Botox) is used to manage chronic muscle spasms that do not respond to other treatment methods. It may also be used for cosmetic purposes.
- Spasmolytics should be used cautiously in patients who require spasticity to remain upright.

Questions For Study and Review

1. What is the difference between muscle spasm and muscle spasticity?
2. Your patient, who has a history of depression, rammed his car into a light pole, sustaining multiple contusions and abrasions. The patient is taking amitriptyline (Elavil) four times a day and diazepam (Valium) as needed. The patient is placed on cyclobenzaprine for muscle spasms of the neck and back. What precautions would you take with this patient?
3. Which drugs are effective in managing both muscle spasm and muscle spasticity?
4. Why is baclofen ineffective for spasms from CVA or Parkinson disease?
5. What symptoms suggest hepatitis in the patient who is on long-term dantrolene therapy?
6. Your patient was injured in a motor vehicle crash yesterday. The patient has severe musculoskeletal pain in the upper back. Is botulinum toxin a good choice for the management of this patient?

Need More Help?

Chapter 16 of the Study Guide to Accompany *Drug Therapy in Nursing,* 3rd Edition, contains NCLEX-style questions and other learning activities to reinforce your understanding of the concepts presented in this chapter. For additional information or to purchase the study guide, visit thePoint **http://thepoint.lww.com/aschenbrenner3e**

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