Muscle Relaxants and Antispasticity Agents

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Pain of muscle origin or myogenic pain is commonly observed among patients experiencing chronic pain. A 30% prevalence of myofascial pain has been observed within the setting of general internal medicine practice [1]. The incidence of spasticity-related muscle pain in patients with stroke, multiple sclerosis, or after spinal cord injury can reach as high as 65%, 74%, and 67%, respectively [2–4]. A variety of pharmacologic agents is available for the treatment of myogenic pain. These agents are typically referred to as muscle relaxants, muscle relaxers, or antispastic medications. One study showed that up to 91% of physicians report using muscle relaxants, and that up to 35% of patients visiting a primary care physician with the complaints of low back pain have them prescribed [5,6]. This pharmacologic class is typically subdivided into central and peripheral depolarizing/non-depolarizing muscle relaxants. The former category is generally used for the treatment of myogenic pain and the latter for the induction of muscle paralysis as part of perioperative general anesthesia.

Pathophysiology of muscle pain

The pathophysiology of muscle pain is fairly complex and cannot be comprehensively addressed within the confines of this article. Nevertheless, a concise overview is attempted herein as it specifically pertains to the use of muscle relaxants and antispastic medications.

Local muscle pain is clinically elicited after the thinly myelinated Aδ and unmyelinated C nerve fibers become activated. This activation can be induced by thermal or mechanical stimuli, local inflammation, or muscle ischemia. Localized muscle ischemia can occur with vascular claudication,
Various theoretical mechanisms have been proposed in an attempt to explain the chain of events leading up to the occurrence of chronic myogenic pain. One of the accepted explanations deals with the neuroplastic changes that occur at the level of the dorsal horn, where newly formed pathologic interneuronal synapses form. As a result, a previously nonpainful stimulus is interpreted as algesic or hyperalgesic owing to abnormal sensitization of the nociceptive neurons and the secondary neurons involved. Another explanation is pathologic functioning of the descending inhibitory pathways that, in the normal healthy state, serve as strong inhibitors of afferent stimulation. A third possibility is that constant peripheral sensitization of nociceptive intramuscular nerve endings occurs owing to the recurrent trauma or chronic local ischemic conditions. The local tissue ischemia is thought to occur as a result of vaso-occlusive events, as might be seen within trigger points, or owing to local neurogenic edema producing the same pathologic effect in the vascular supply. Perpetuating factors of a mechanical or systemic nature causing abnormal firing of the involved muscle groups can also lead to chronic muscle pain. Another theoretical mechanism is the well-documented mind-body connection in the context of chronic pain. Chronic pain patients with untreated depression or anxiety who are under significant stress tend to perceive pain at higher intensity. This effect can be explained by well-documented central nervous system connections involving ascending and descending nociceptive pathways and pathways relaying an emotional state of affairs.

Clinically, chronic muscle pain can be observed in individuals who sustain entities such as recurrent muscle spasms, spasticity, fibromyalgia, trigger points, and increased muscle tension. Typical complaints are a diffuse, aching, cramping pain of mild-to-severe intensity, which can be intermittent (ie, when induced by postural changes) or constant (ie, in fibromyalgia).

The pain of muscle spasms can be encountered as part of acute, subacute, or chronic pain states. It is defined as an involuntary muscular contraction that can be observed during electromyographic examination. A painful muscle spasm is typically referred to as a muscle cramp.

Spasticity, which is commonly seen in patients with previous stroke, multiple sclerosis, or spinal cord injury, is defined as a muscle tone disorder characterized by hyperactive tonic stretch reflexes. The exaggerated muscle tone is velocity dependent, producing progressive resistance to stretch with increased speed of applied passive movement. Other clinical findings in spasticity include hyperreflexia, clonus, the Babinski sign, and flexor spasms. Spasticity is thought to arise from an imbalance of excitatory and inhibitory inputs at the level of the spinal cord seen after injury to descending motor pathways.

Physiologic contracture, which is defined as a state of muscle contractile activity without the presence of electrical activity, can be found in disorders
with abnormal regulation of calcium at the level of sarcoplasmic reticulum as well as in commonly seen taut muscle bands that surround myofascial trigger points [8]. The physiologic contracture should be differentiated from structural contracture involving the surrounding fascia, connective tissue, and ligaments commonly seen in chronically shortened muscles.

Myofascial trigger points are frequently observed and contribute to the overall level of pain in individuals sustaining chronic musculoskeletal conditions. It has been proposed that sustained muscle fiber shortening, induced by an abnormal increase in the release of acetylcholine, produces localized ischemia and subsequent sensitization of nociceptive nerve fibers [8].

The pain of fibromyalgia, which some physicians still believe is a controversial clinical diagnosis, is described as diffuse and constant muscle pain with pathognomonic tactile allodynia on physical examination. This often incapacitating pain is thought to be due to the pathologic sensitization of central nociceptive pathways.

Muscle relaxants

Muscle relaxants make up a heterogeneous group of drugs that mainly exert their pharmacologic effect centrally at the level of the spinal cord, the brainstem, or the cerebrum, and that have an insignificant, if any effect, at the muscle fiber level. Their centrally mediated mechanism of action can exert a clinically significant peripheral therapeutic effect.

Cyclobenzaprine

Cyclobenzaprine is probably the most commonly used muscle relaxant for nonspasticity-related muscle pain. Structurally, it resembles tricyclic antidepressants and differs from amitriptyline by only one double bond. Its therapeutic effect is centrally mediated and carries no direct peripheral action on the affected muscles. Its main pharmacologic action occurs at the brainstem and spinal cord levels and is partially explained by a depressant effect on the descending serotonergic neurons [9]. It is extensively metabolized in the liver and excreted as a glucuronated metabolite through the kidneys. It possesses a fairly long half-life of approximately 18 hours and can continue to accumulate for up to 4 days when administered at a frequency of three times per day. Given its structural similarity to tricyclic antidepressants as well as potent anticholinergic properties, caution should be exercised when considering its use in the elderly or in patients with heart disease. Likewise, concomitant use with monoamine oxidase inhibitors is absolutely contraindicated because this combination can cause a hyperpyretic crisis or even death. The initial starting dose should be 5 mg three times per day on as needed basis and can be titrated up to 10 mg three times per day per therapeutic effect or side effect. In patients with hepatic or renal insufficiency, it should initially be administered once per day given its
relatively long half-life. Of note, one recent study showed equal efficacy of 5 and 10 mg doses, with the smaller dose showing a lower level of sedation [10]. The most common side effects are drowsiness, dry mouth, fatigue, and headache, followed by less often occurring adverse effects of diarrhea, dizziness, abdominal pain, nausea, nervousness, blurred vision, and confusion [11].

*Methocarbamol*

Methocarbamol is structurally related to the expectorant guaifenesin and the muscle relaxants chlorphenesin and mephenesin. It is a centrally acting muscle relaxant that suppresses spinal polysynaptic reflexes and has no direct effect on skeletal muscle. It is extensively metabolized in the liver and is excreted through the kidneys, with a small amount excreted with feces. Acutely, methocarbamol can be administered four times a day at a dose of 1500 mg. For maintenance therapy, 1500 mg can be given three times per day. This medication can also be administered via intravenous and intramuscular routes. Frequent side effects include drowsiness and dizziness, followed by nausea, anorexia, headache, blurred vision, muscular discoordination, and, in some instances, urine discoloration. Seizures have been reported with intravenous administration, especially with a history of epilepsy [11].

*Orphenadrine*

Orphenadrine is structurally related to diphenhydramine and carries relatively stronger anticholinergic and weaker sedative properties. It does not produce any direct effect on skeletal muscle, and its exact mechanism of action is unknown. Orphenadrine is mostly excreted through the kidneys. The typical adult oral dose is 100 mg administered at the frequency of twice per day owing to its relatively long half-life. The drug can also be administered intravenously as well as intramuscularly. Common side effects include drowsiness and dizziness, followed by other central nervous system effects such as agitation, hallucinations, and euphoria. The following adverse effects have been reported owing to orphenadrine’s anticholinergic properties: dry mouth, nausea, constipation, urinary retention, tachycardia, blurred vision, and mental confusion, especially in the elderly. Rare reports of aplastic anemia have been documented [11].

*Metaxalone*

Metaxalone is a centrally acting muscle relaxant with an unknown mechanism of action. It is metabolized in the liver and excreted through the kidneys in the form of metabolites. Typical adult dosing consists of 800 mg three to four times per day. It is recommended to monitor liver function tests after initiation of this agent. Common side effects include drowsiness,
dizziness, nervousness, nausea, and headache. The following rare but serious adverse reactions have been reported: leukopenia, hemolytic anemia, jaundice, and hypersensitivity reactions [11].

**Tizanidine**

Tizanidine is a centrally acting muscle relaxant that, through its alpha-2 adrenergic agonist properties, is thought to prevent the release of excitatory amino acids by suppressing polysynaptic excitation of spinal cord interneurons. Metabolism is through the liver, and excretion is 60% through the kidneys and 20% through the feces. Tizanidine should be administered through a gradual upward titration from an initial dose of 2 to 4 mg at bedtime up to the maximum of 8 mg three times per day. The bedtime dose can provide an analgesic effect as well as improve quality of sleep owing to the commonly occurring sedating side effect. Other common side effects are daytime drowsiness, hypotension, weakness, and dry mouth. Even though tizanidine’s pharmacologic effect is similar to another alpha-2 agonist, clonidine, it possesses only a fraction of its blood pressure–lowering effect. Less commonly reported side effects of tizanidine are palpitations, bradycardia, dizziness, headache, nausea, elevated liver enzymes, and several rare cases of fulminant liver failure that led to death. Serial monitoring of liver enzymes is strongly recommended [11].

**Quinine**

In the not so distant past, quinine was extensively prescribed for the treatment of nocturnal leg muscle cramps; however, it lost this Food and Drug Administration indication owing to the seriousness of potential side effects, with increased morbidity in the older population. Structurally, quinine is a stereoisomer of a class IA antiarrhythmic quinidine and possesses some of its cardiac effects. Quinine’s other pharmacologic properties include antimalarial, antipyretic, analgesic, and muscle relaxant effects. The last effect is achieved owing to its ability to increase the refractoriness of muscle and to decrease the excitability of the neuromuscular endplate. Quinine is extensively metabolized in the liver and is mainly excreted in the urine. The typical adult dosing for nocturnal leg cramps is 260 mg at bedtime. The more serious adverse effects include myelosuppression, thrombocytopenia, hemolysis, disseminated intravascular coagulation, torsades de pointes, angina, vasculitis, hypoglycemia, renal toxicity, hemolytic uremic syndrome, urine discoloration, liver toxicity, asthma, toxic epidermal necrolysis, and hearing loss. Less serious reported side effects are headache, confusion, dizziness, nausea, and blurred vision. If prescribed, regular checks of liver and kidney function tests, a complete blood count, and a performance of a 12-lead electrocardiogram are strongly recommended [11].
Carisoprodol

Carisoprodol is still a commonly prescribed muscle relaxant that should be dispensed with caution owing to the potentially addictive properties of its main metabolite, meprobamate. Carisoprodol produces its muscle relaxant effect by depressing the interneuronal activity at the spinal cord level as well as in the descending tracts of the reticular formation. It is not recommended for use in the pediatric age population. This drug is metabolized in the liver with meprobamate as its main metabolite. It is mainly excreted through the kidneys. The usual adult dosage is 350 mg four times per day. The most common side effect is drowsiness. Other central nervous system adverse effects that have been reported include ataxia, agitation, insomnia, and others. Adverse effects such as tachycardia, postural hypotension, nausea, erythema multiforme, and eosinophilia have also been seen [11].

Botulinum toxin type A, B

Clostridium botulinum is a spore-forming anaerobic bacillus that produces seven distinct antigenic types of botulinum toxin named from A to G. Toxins type A, B, and F are the only neurotoxins that have been used in the clinical arena. These neurotoxins exert their pharmacologic effect at the neuromuscular junction, where they prevent the calcium-dependent release of acetylcholine, producing a state of temporary drug-induced denervation. The therapeutic effect can take up to 1 week to take place fully and can last up to 3 months, at which point repeat injections can be considered. Injectable botulinum preparations are unstable and need to be stored and reconstituted with caution per the manufacturers’ recommendations. Each unit of the respective clinically available neurotoxin carries a different level of biologic potency and cannot be reliably interconverted. Relative muscle weakness of the injected musculature as well as of adjacent muscle groups is to some degree expected from local spread of the neurotoxin. Caution should be exercised in using appropriate botulinum dosing to avoid functionally limiting muscle weakness. Headache, dizziness, fever, flulike symptoms, nausea, biliary colic, injection site pain, edema, and erythema are some of the adverse effects encountered. Dysphagia, dry mouth, and dysphonia can be observed with injections in the cervical area. Rare instances of arrhythmia, myocardial infarction, hypertension, and botulismlike syndrome have been reported. Patients who receive repeated botulinum injections can develop antibodies and become resistant to the neurotoxin’s therapeutic effect. Trials of the other available antigenic types of the neurotoxin can be entertained in such cases [11].

Benzodiazepines (diazepam, lorazepam, clonazepam, etc)

Diazepam has commonly been used in the treatment of muscle spasm, especially in the acute setting. It belongs to a group of compounds called benzodiazepines, known for their potent anxiolytic, sedative, as well as muscle
relaxant effects. Their main mechanism of action is through central potentiation of the inhibitory $\gamma$-aminobutyric acid (GABA) effect through presynaptic facilitation of GABA release. Benzodiazepines are extensively metabolized in the liver into inactive and in some cases active metabolites. Compounds that lack active metabolites should be used as first-line agents in the elderly and in patients with liver or kidney insufficiency. Such compounds are lorazepam, clonazepam, temazepam, and oxazepam. Excretion principally occurs through the kidneys. Some of the common side effects are drowsiness, confusion, ataxia, cognitive impairment, memory loss, agitation, and disinhibition. Of note, withdrawal symptoms may occur after only 4 to 6 weeks of use, and their potential for abuse should be taken into consideration [11].

**Antispastic agents**

Antispastic drugs are principally used for the treatment of spasticity observed in disease states with upper motor neuron pathology, such as stroke, spinal cord injury, traumatic brain injury, and multiple sclerosis. All but two of these agents exert their clinical effect through centrally mediated mechanisms.

**Baclofen**

Structurally, baclofen is related to the centrally occurring inhibitory neurotransmitter GABA. Clinically, it has commonly been used for its muscle relaxant effects in the treatment of spasticity, as well as for its neuropathic analgesic properties in the treatment of trigeminal neuralgia pain. Baclofen is a GABA-B receptor agonist with presynaptic and postsynaptic effects leading to a decrease in the excitatory neurotransmitter release as well as in substance P, which is involved in transmission of nociceptive impulses [12]. It is metabolized in the liver and excreted in the urine. Baclofen can be administered orally as well as intrathecally via an implanted pump mechanism when significant adverse effects preclude further dose escalation to achieve therapeutic effect. Initial dosing of baclofen should be gradual, starting with 5 to 10 mg three times per day. The maximum recommended dose is 80 mg per day in divided doses; however, higher therapeutic doses in cases of refractory spasticity have been used without any significant untoward side effects. Common side effects are weakness, sedation, and dizziness. At higher doses, baclofen can cause seizures, ataxia, and hallucinations. Abrupt withdrawal should be avoided because it can precipitate seizures and hallucinations [11].

**Dantrolene**

Dantrolene reduces muscle spasms by inhibiting the release of calcium from the sarcoplasmic reticulum and does not directly affect the central
nervous system. It is metabolized in the liver and excreted through the kidneys. Because dantrolene tends to produce greater muscle weakness than baclofen, it should not be the first-line agent for patients who are capable of ambulation. Typically, this medication should be started at a relatively low dose of 25 mg per day and slowly titrated on a weekly basis per effect or side effect up to the maximum of 100 mg three times per day. Such slow titration can obviate the development of some of the common side effects: drowsiness, weakness, dizziness, and diarrhea. Less frequently observed side effects are constipation, nausea, insomnia, diplopia, tachycardia, depressed mood, anxiety, muscle pain, difficult urination/frequency, erectile dysfunction, and respiratory depression. More serious but relatively rare adverse effects are hepatitis, seizure, heart failure, aplastic anemia, leukopenia, thrombocytopenia, respiratory depression, pleural effusion, and pericarditis. Dantrolene should be used with caution in patients with heart failure, chronic obstructive pulmonary disease, and liver disease [11].

**Tizanidine**

For information on this agent, the reader is referred to the section on muscle relaxants.

**Benzodiazepines (diazepam, lorazepam, clonazepam, etc)**

For more information on the benzodiazepines, the reader is referred to the section on muscle relaxants. In the treatment of spasticity, the maximum daily doses of 40 to 60 mg and 10 to 20 mg of diazepam and clonazepam, respectively, can be gradually achieved as tolerated per therapeutic effect.

**Botulinum toxin type A, B**

For more information on botulinum toxin, the reader is referred to the section on muscle relaxants.

**Clonidine**

Clonidine is an antihypertensive agent available in an oral form as well as a transdermal preparation. It is an alpha-2 agonist that exerts its pharmacologic effect on the brainstem, leading to a reduced central sympathetic outflow. This effect in turn causes a reduction in the peripheral resistance, blood pressure, and heart rate. The oral preparation of clonidine is administered twice per day with a starting dose of 0.1 mg up to the maximum of 1.2 mg BID as tolerated. Transdermal clonidine patch, which is changed every 7 days, can be prescribed if compliance is an issue. It is metabolized in the liver with majority excretion through the kidneys. A small portion is eliminated through the biliary/fecal route. The most common side effects are drowsiness, dizziness, dry mouth, and constipation. Other adverse side effects include fatigue, headache, orthostatic hypotension, palpitations, syncope, Raynaud’s
phenomenon, congestive heart failure, conduction blocks, anxiety, insomnia, nausea, hepatitis, constipation, impotence, loss of libido, thrombocytopenia, muscle pain, and blurred vision [11].

Clinical implications

Available evidence

A large number of studies of varying quality and methodology have been conducted on the efficacy of muscle relaxants and antispastic agents in the treatment of muscle-related pain. Several systematic reviews and at least one meta-analysis have been performed, with three of these available through the Cochrane collaborative database.

The Cochrane review on the use of muscle relaxants for nonspecific low back pain was recently updated in May of 2005 and consists of 30 randomized, controlled and randomized, double-blinded, controlled clinical trials. The review concluded that there is strong evidence for statistically significant symptomatic relief within 1 week of therapy for non-benzodiazepine muscle relaxants in the treatment of acute low back pain; however, the evidence for benzodiazepines, specifically diazepam and tetrazepam, was less convincing. The various muscle relaxants were found to be equally effective (ie, tizanidine, cyclobenzaprine, orphenadrine, methocarbamol, metaxalone, carisoprodol), and there was a significantly higher prevalence of adverse effects in the muscle relaxant group than in the placebo group. Interestingly, antispasticity muscle relaxants (ie, baclofen, dantrolene) also showed clinical efficacy in the setting of acute low back pain. Likewise, muscle relaxants were found to be effective when used as adjunctive therapy in treating acute back pain. In regards to chronic low back pain, tetrazepam, flupirtine, and tolperisone, none of which are available in United States, showed short-term efficacy over placebo [13].

A meta-analysis by Browning and coworkers [14] evaluated the efficacy of cyclobenzaprine in the context of back pain. Fourteen randomized, placebo-controlled studies were included, three of which dealt with nonacute pain of spinal origin. The overall observation was that cyclobenzaprine was modestly more effective than placebo, with the greatest efficacy seen in the first 4 days of treatment, and that the agent showed a trend of declining effectiveness over time. Adverse effects were observed in more than 50% of patients, with drowsiness being the most common [14]. Three of the studies examined the muscle relaxant effects of cyclobenzaprine in patients with more of a chronic presentation. They included subjects with cervical as well as lumbosacral complaints. Basmajian [15] compared the effects of cyclobenzaprine, diazepam, and placebo in individuals with greater than a 30-day duration over the course of 18 days. Both treatment groups showed a statistically significant effect, more so in the cyclobenzaprine arm [15]. In a study by Bercel [16], cyclobenzaprine was compared with placebo in a population
with greater than a 30-day symptom duration over 14 days, again showing superiority of the treatment arm. The study by Brown and Womble [17] was the only study of a true chronic pain population with a minimum symptom duration of 12 months comparing cyclobenzaprine and diazepam versus placebo. Unfortunately, this study was of relatively poor quality with a short treatment period of 14 days. Both treatment arms showed a significantly positive therapeutic effect [17].

The most recent update of the Cochrane review on the treatment of spasticity after spinal cord injury was conducted in March of 2005 and included 55 studies that met the inclusion criteria. This review reported the clinical efficacy of intrathecal baclofen in reducing spasticity and improving performance of activities of daily living in a comparison with placebo, as well as a significant effect of tizanidine in improving the Ashworth score but not activities of daily living [18].

Another Cochrane review update (February, 2004) also dealt with the efficacy of antispasticity agents and was inconclusive in regards to their use in the setting of multiple sclerosis. Less than half of the placebo-controlled trials, which used the Ashworth score as one of its outcome measures, showed a statistically clinical significance over placebo. None of the comparative trials showed a significant difference of one antispastic agent over another [19].

**Clinical pearls**

Before initiating the prescription of muscle relaxants to patients with chronic pain, a comprehensive assessment should be performed to determine whether these agents are indicated or contraindicated for the clinical scenario at hand. The first question that should be addressed is whether the pain of muscle origin is one of the main contributors. Is there evidence of muscle spasm, trigger points with a referral pattern, spasticity, increased muscle tension, decreased range of motion, muscle tightness, cramping, or other factors?

Unfortunately, exact pathophysiologic factors of chronic pain are not always easily teased out owing to its multifactorial nature and the significant overlap of causative mechanisms. When one is uncertain about the putative mechanisms and when other pharmacologic options have been exhausted, an empiric trial of muscle relaxants should be considered.

Has the patient already tried a muscle relaxant, and what was the response? Was the dose therapeutic and was an adequately long trial attempted? Sometimes one needs to titrate the specific agent gradually up to the maximum daily allowable therapeutic dose and perform a trial for at least 4 to 7 days before concluding that it is ineffective. Failure or poor tolerance of one muscle relaxant should not preclude the use of others because the mechanisms of action and side-effect profiles are different. Clinically, some of the side effects dissipate over time with longer use of a specific
muscle relaxant. Because all of the muscle relaxants produce sedation to some degree, their introduction to the patient should begin at bedtime, especially if the patient is already suffering from insomnia.

What is the age of the patient? Are there any medical contraindications? In general, elderly patients do not tolerate muscle relaxants well, especially if they have anticholinergic properties, such as cyclobenzaprine, which can cause significant mental confusion. Initial doses should be minimal and titration gradual in this patient population.

Caution should be exercised in patients who have liver as well as kidney insufficiency, because all of the muscle relaxants undergo liver metabolism and principally are renally excreted. Patients with heart disease or conduction abnormalities should avoid using cyclobenzaprine and dantrolene owing to the proarrhythmic properties of the former and the cardiac muscle depressant effect of the latter.

Is the medication ineffective at the maximal oral dose, requiring an intrathecal route of administration? Do the adverse effects preclude ongoing use or prevent further dose escalation? Intrathecal infusion of the antispasticity drug baclofen allows administration of potent doses with a significantly improved side-effect profile [20].

Clinical scenarios

In Case 1 in the introductory article of this issue, that is, a 19-year-old male patient with a traumatic brain injury status post a motorcycle accident who presents with right-sided paresis, spasticity, and neuropathic pain involving his right lower extremity, the use of an agent with antispastic and neuropathic properties would be most advantageous. These agents include baclofen, which is one of the first-line agents used in the treatment of trigeminal neuralgia pain, as well as tizanidine and clonidine, which also possess antineuropathic pain properties [21,22]. Clonazepam could also be considered, because it might possess some analgesic properties for neuropathic pain [23]. Because all of these agents can affect the level of alertness and mentation, caution should be exercised in patients with a history of traumatic brain injury. Clonidine should not be used as the first-line agent owing to the possible hypotensive effects, especially if posttraumatic autonomic dysregulation is present.

A trial of bedtime cyclobenzaprine should be considered in Case 3, that is, the 35-year-old female patient with a diagnosis of posttraumatic fibromyalgia [24]. Initial dosing should start with 5 mg and be gradually titrated up to 30 to 40 mg at bedtime based on the therapeutic effects or side effects. Orphenadrine could also be tried, because it exhibited some therapeutic efficacy in at least one clinical study [25].

In Case 4, a 34-year-old roofer with an L1 vertebral fracture, an empiric trial of a muscle relaxant could be considered if a component of myogenic pain is present or if the other pharmacologic or nonpharmacologic therapies
have failed. Local trigger points might have formed as a result of the initial injury, or the patient might be experiencing intermittent muscle spasms owing to altered vertebral biomechanics. If the patient does not have any history of liver abnormalities, a trial of tizanidine can be undertaken because it is relatively well tolerated.

Summary

Muscle relaxants make up a heterogeneous group of agents and can have a clinically significant role in the treatment of chronic muscle pain. These medications are not without possible serious side effects; hence, care should be taken in deciding on their appropriateness and ongoing monitoring performed, if prescribed.

References


