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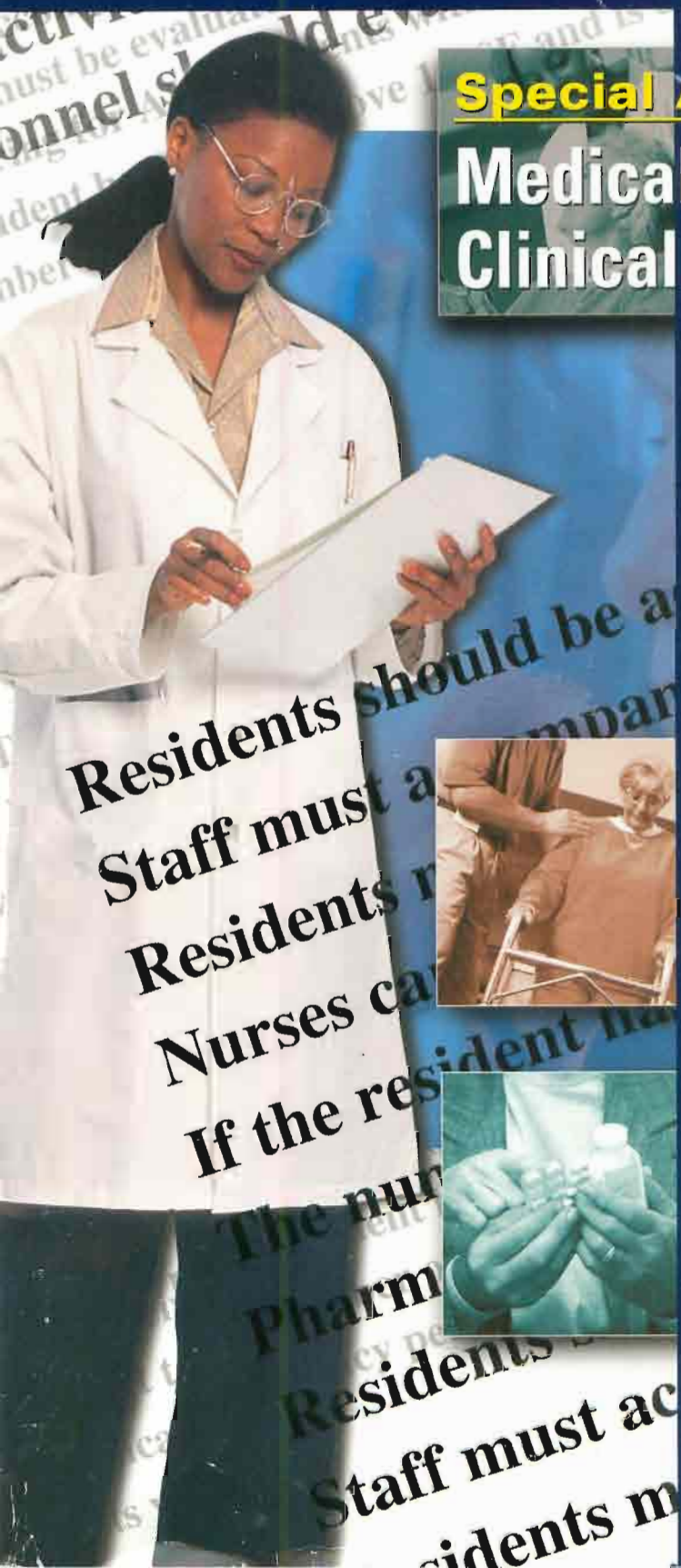

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SPASTICITY IN THE NURSING HOME: PRACTICAL CONSIDERATIONS

Michael I. Cheikin, MD

In addition to the typical issues of gerontological LTC residents, younger LTC patients with spinal cord injury, cerebral palsy, multiple sclerosis, and stroke, require additional considerations. These are the potential repercussions of interventions, occurring over the decades of a young person's life expectancy. Written to address practical issues for a busy nursing home clinician, the author offers this article not as a review of current state-of-the-art treatment but as an analysis based on many years of practice in this area.

CASE 1: PERSISTENT HAMSTRING SPASTICITY WITH BACLOFEN PUMP

A patient with multiple sclerosis has severe extensor tone of the thoracolumbar spine, with associated flexor tone of the hips and knees, and extensor tone of the ankles. After failing oral therapy, the patient had a successful trial with intrathecal baclofen and a pump was placed. After the pump dosage was titrated over the ensuing month, all that remained was persistent tone of the hamstrings, which was causing problems with positioning in bed and a wheelchair. The maximum extension of the knees was 110 degrees.

The therapy team performed alcohol blocks on each sciatic nerve, focusing on the medial and lateral hamstring muscles. Within one week of the blocks, the knees were easily extended to 75 degrees, enabling comfortable wheelchair positioning, and enabling the patient to lie supine in bed up to two hours at a time with proper knee bolsters provided by physical therapy.

CASE 2: NECK AND SHOULDER SPASTICITY

A patient with multiple sclerosis had spasticity in the neck and upper extremities. It was difficult for the staff to wash her underarms. Though noncommunicative, when the therapist tried to provide maintenance range of motion (ROM) of her shoulders, she would grimace and her tone would increase, indicating that she experienced pain. Her head listed to the left because of tone in the cervical paravertebral muscles. He head could not be brought to midline in a wheelchair or bed. She also had tone in her right biceps, causing extreme elbow flexion (130°), making it difficult to dress her and to clean her antecubital fossa.

Her neck muscle spasticity was successfully managed with botulinum A toxin 300 to 400 units every three to four months, as needed. Muscles affected included the sternocleidomastoid, upper trapezius, splenius capitis, and cervical paravertebral muscles. The pectoral and musculocutaneous nerves were each blocked with ethanol 100% 5 mL with good relief of tone, allowing adequate shoulder abduction and elbow extension.

After receiving the injection, she received aggressive ROM therapy, five times per week for one to two weeks. After stretching, therapy staff was able to bring her head to midline, abduct the shoulder to 60 degrees, and extend the elbow to 80 degrees.

Dr. Cheikin is Medical Director of Chestnut Hill Rehabilitation Hospital and the Center for Optimal Health, and Associate Medical Director of Inglis House, Philadelphia.

DEFINITION

Spasticity is a "syndrome," characterized by a collection of positive and negative findings on physical exam. The three positive findings not normally seen in healthy individuals are: (1) increased tone (resistance to movement), classically the extensor in lower extremities and the flexor in upper extremities; (2) exaggerated reflexes, which are stimulated at relatively low thresholds, are velocity-dependent, exhibit a larger response than normal, are generalized (radiated response), and demonstrate latent (such as Babinski, Hoffman, or crossed-extension) reflexes; and (3) spasms, which are groups of muscles firing at a same time. The two negative findings are (1) weakness and (2) loss of dexterity (coordination).

This definition of spasticity is helpful for evaluating patients, because when distinguishing between contracture and spasticity, one can look for the velocity-dependent nature of the stretch reflex. Even contracted tissues have some elastic features, so they will pull back like a rubber band if stretched. However, if a fast movement causes much greater resistance than a slow movement, this implies the presence of some spasticity.

Often, even joints that seem to have end-stage soft-tissue contracture have some spasticity present; after a nerve block, most joints will gain at least 15 to 25 degrees in range, despite the impression of end-stage contracture.

CAUSES AND PHYSIOLOGY OF SPASTICITY

The cause of spasticity is loss of central control over the interneurons that control the motor neuron (the final common pathway). Depending on the location of the damage to the nervous system, whether central in the motor cortex or cerebellum, or more distally in the spinal cord, the neurotransmitter balance and neurophysiology have slight differences, which then make the spasticity more or less responsive to various medications.

Muscle tissue contains spindles, specialized sense organs that convey information about fast and slow stretch of the muscle to the rest of the nervous system. Based on this sensory information, the motor side of the system modulates the drive of the motor neuron. By changing the control of these feedback loops, various spasticity patterns emerge. In addition, muscle fibers are recruited in a hierarchical manner, with the smaller, fine motor, longer endurance fibers being recruited first and the larger, gross motor, shorter endurance fibers being recruited last. In patients with spasticity, there seems to be a preferential loss of the smaller enduring fibers, which partially explains the weakness and poor coordination.

Spasticity can develop in any condition that causes loss of upper motor neurons. Diagnoses include stroke, multiple sclerosis, cerebral palsy, traumatic brain injury (TBI), familial spinocerebellar degeneration, Friedreich's ataxia, and Parkinson's disease or syndrome.

MEASUREMENT OF SPASTICITY

Numerous attempts have been made to formally measure spasticity, but none are easy or clinically useful. Generally, the affected limb is affixed to a device, a measured stimulus is provided, and a measurable response results. Time and force measures of the response do give some information. However, it can vary from day to day because of changes in baseline tone, other physiological parameters such as sympathetic/parasympathetic balance, emotional state of the subject, etc. In addition, other than the force and time data, it is very difficult to measure and characterize the overall pattern of the spasticity. In some patients, it spreads more, or quicker, to various muscle groups.

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Some electrophysiological correlates of spasticity can be measured on electromyography (EMG), but these have not proven useful clinically and are not utilized at present.

The Modified Ashworth scale quantifies the magnitude of the spasticity and how much it generalizes (ranging from 0, no increase in tone, to 4, rigid). It does not measure hypotonia, though, which is important as well (most patients with areas of hypertonia often have regions of hypotonia). One might use a simple, alternative scale (not validated, but constructed and used by the author), which simply rates spasticity from +3 for severe spasticity to –2 to totally flaccid (0 = normal).

BENEFITS AND RISKS OF SPASTICITY

Spasticity is not inherently bad. In fact, one might wonder about the evolutionary benefit of spasticity. For example, a spastic extended leg can bear

weight and may help maintain bone mass. A flexed upper extremity is kept close to the body and is less vulnerable to external trauma. Indeed, when one considers treating spasticity (as illustrated in some of the case examples), the usefulness of the tone and extremity is the highest priority.

The risks of spasticity include progressive contracture with loss of positioning ability; skin pressure and breakdown; difficulty of caretakers to have access to perineum, axilla, palms; and pain. The triad of tone–skin–positioning describes the areas most affected by spasticity. After the cognitive/safety concerns of dementia, it is usually this triad that causes families to place their loved ones in nursing homes. Implied in this logic, is if one adequately manages spasticity, patients can stay home with their families for most of their lives.

ASSOCIATED MEDICAL CONDITIONS

Besides the positioning, skin, activities of daily living (ADLs), and pain issues discussed above, secondary issues should be considered, most of which are the medical conditions associated with immobility (e.g., pneumonia and osteoporosis) that cannot be fully prevented in patients with advanced disease. Additionally, neck positioning issues and side effects of medication can cause problems with swallowing. Long-term spasticity often causes various patterns of kyphoscoliosis, which can further compromise cardiopulmonary and gastrointestinal function. Immobility can be a cause of obesity, type 2 diabetes, and hypercholesterolemia, leading to long-term medical consequences. Once patients no longer have swallow function, often resulting from the existence of both hypertonia and hypotonia, the weight gain one sees, while increasing skin integrity, can exacerbate the problems of overweightedness. Finally, the immobility of the extremities often puts the patient at risk for deep-vein thrombosis, pulmonary embolism, postphlebitic syndrome, and secondary lymphedema.

EVALUATION AND TREATMENT OPTIONS FOR SPASTICITY

Based on experience, the treatment of spasticity in the nursing home usually occurs too little, too late. Although subjecting a patient to a regimen of q6h medications for the rest of their lives or requiring staff to provide ROM therapy is not a minor decision, the costs to the patient and staff in not identifying spasticity early enough will be much higher in terms of the patient's quality of life, and in the staff and nursing home's resources.

Therapy staff must comprehensively and consistently evaluate the patient's ROM and muscle tone at least on admission and then periodically thereafter. It is important to discover changes in these areas before resource-consuming clinical situations develop that will also cause discomfort to the patient.

The main reasons to treat are the prevention, correction, and palliation of the effects of spasticity. These include (not in priority order) enabling positioning and function (such as hygiene, ambulation, wheelchair driving, swallowing), relieving and/or preventing pain and contracture, and preventing/treating skin breakdown.

The treatment of spasticity in the nursing home usually occurs too little, too late.

The Figure presents an algorithm that summarizes the considerations and the consequent strategies that one might use to evaluate and treat spasticity. The Table is a teaching tool for patients and their families, used to discuss the various options, risks, and benefits of treatments. The treatments are classified into four main categories: (1) physical interventions, (2) oral interventions, (3) simple/reversible procedures, and (4) end-stage procedures.

Physical Procedures. Good ROM therapy, restorative nursing, and family caregiving are essential. Often, a conflict arises as to who should perform ROM exercise (i.e., "ranging") for the patient, how often it should be performed, and what the goals should be. A good rule of thumb is treatment between one and three times per week, with some daily self-ranging or with the care staff is usually sufficient to maintain ROM. If a patient needs ranging more than two or three times per week by a professional, then the spasticity is likely progressing or suboptimally controlled; the patient then needs a more thorough evaluation and different treatment strategy. Good wheelchair and bed positioning must be an adjunct to ranging.

Medical Interventions. If the physical interventions are not sufficient and reversible causes have been

TABLE: SPASTICITY REVIEW

Treatment	Route	Where Done	Mechanism	Advantages	Disadvantages	Duration of Effect
Biofeedback	Phys. and occup. therapies	On site	Patient learns how to inhibit spasms	No medications	Requires concentration may not be sufficient	Long term
Wheelchair positioning	Phys. and occup. therapies	On site	Inhibits spasticity	6–12 hr/day	Expensive; skin risk if improperly placed	As long as in w/c plus long term
Physical and occupational therapy		On site	Stretches tissues; inhibits spasticity	No meds; professional staff can customize Rx and monitor	Subject to staffing; transport	1–7 days
CPM	Phys and occup. therapies	On site	Maintains ROM; inhibits spasticity	Does not need therapist, gentle; as necessary	Needs daily setup; less customized; skin risk; not yet covered	As needed
Baclofen	Oral med	On site	Blocks nerve transmission selectively	Rare side effects or toxicity; may cause some weakness/fatigue	Cannot increase or decrease suddenly; must take several times per day	~6 hr
Tizanidine	Oral med	On site	Blocks nerve transmission selectively	Rare side effects or toxicity; may cause some tiredness	Cannot increase or decrease suddenly; must take several times per day	~8 hr
Diazepam	Oral med	On site	Blocks nerve transmission selectively	Selectively inhibits spastic muscle	Dependency; sedation; must take several times per day	6–12 hr
Dantrolene	Oral med	On site	Blocks muscle transmission nonselectively	Always works	Affects all muscles—causes some weakness; liver toxicity blood test every month; must take for rest of life	6–12 hr
Lidocaine/ marcaine	Injection into muscle or nerve	On site	Temporarily blocks nerve and muscle	Reversible	Must be followed by permanent injection, which may have different effect	4–6 hr
Phenol	Injection into muscle or nerve	On site	"Kills" nerve along its path	Always works to some degree; oral meds not needed; can be done on site	May be permanent; can cause pain	6–52 wk

continued

ruled out, then oral medications are the next step, unless the tone is limited to one extremity (in which case, a local procedure may be preferred) (Figure). The oral agents work by modulating neurotransmitter balance; they have similar side effects: sedation, weakness/fatigue, and some cognitive impairment. Certain agents were thought to work uniformly well for certain types of spasticity, but, in practice, individual differences define which treatments are best for each patient. The approach is empirical, starting slow, observing positive and negative effects, and then moving forward.

The most commonly used medication is baclofen. It is best given at a q6h schedule (and initiated at a dose of 5 mg q6–12h, increased in 5-mg increments every 5–7 days). Patients need to be warned that they will usually experience some sedation with each dose increment, but it usually lasts only a few days. Although the manufacturer's recommended prescribing information states that baclofen's maximum dose is 80 mg/day, a maximum dosage of 160–240 mg/day is used commonly in patients with

TABLE *(cont'd)*: SPASTICITY REVIEW

Treatment	Route	Where Done	Mechanism	Advantages	Disadvantages	Duration of Effect
Alcohol	Injection into muscle or nerve	On site	"Kills" nerve along its path	Same as phenol	Same as phenol	mo–yr
Botulinum toxin A	Injection into muscle or nerve	On site	Blocks transmission of nerve to muscle	No oral meds; reversible	Only one muscle at a time; must be repeated; may become ineffective; not yet covered by all insurance; expensive; may affect swallowing	3–6 mo
Baclofen pump	Neurosurgery	Hospital (ambulatory surgery)	Blocks nerve transmission in spinal cord	Minimal oral meds; adjustable	Pump must be filled periodically; risk of pump failure; not covered by all insurance; not effective for arms	mo–yr
Dorsal column stimulator	Neurosurgery	Hospital (ambulatory surgery)	Sends alternate message to pain fibers	Relatively benign	Often not effective	None–yr
Radio-frequency neurolysis	Neurosurgery	Hospital (ambulatory surgery)	Damages nerve	Permanent; one time	Irreversible; requires hospitalization and anesthesia	mo–yr
Dorsal rhizotomy	Neurosurgery	Hospital (inpatient)	Cuts nerves	Permanent	Spasticity may recur or move elsewhere	mo–yr
Myeolotomy		Hospital (inpatient)	Cuts tracts of spinal cord	One time	Variable effectiveness	mo–yr
Flap surgery (skin or skin + muscle)	Plastic surgery	Hospital (inpatient)	Moves good skin and/or muscle to cover open wound	Quicker healing than nonflap	Requires clean wound; permanent risk of recurrence; not all wounds can be flapped	mo–yr
Tenotomy	Orthopedic surgery	Hospital (inpatient)	Cuts tendons	Permanent	May redevelop contractures in same or opposite side of joint	mo–yr

Phys. = Physical; Occup. = occupational; w/c = wheelchair; CPM = continuous passive motion; Rx = prescription; ROM = range of motion.

advanced spasticity. Even higher doses have been used with success in a few patients, as long as no evidence was seen of sedation or other lab abnormalities. Given the complexity of care once oral baclofen has failed, this approach may prove beneficial.

In addition, the clinician must always remember that although baclofen is relatively safe, abrupt decreases in dose can cause delirium, hallucinations, or both. This often occurs during transfer to or from the acute hospital, and has also occurred with changes in competing medications and initiation of the baclofen pump.

Once oral baclofen has failed, the next best option is tizanidine. A derivative of alpha-channel blockers, this medication may cause hypotension in addition to the side effects listed above. It is usually administered q8h, starting at 1 mg, increasing at 0.5- to 1.0-mg dose increments to a maximum of 36 mg/day. The dose is usually limited by side effects.

Other agents that can be tried include diazepam, gabapentin, and dantrolene. Diazepam can be effective, but it alters mood and cognition. It should be administered on a q6h schedule.

Gabepentin, an antiepileptic that sometimes works for pain, can sometimes be effective for spasticity. It tends to be very sedating.

Dantrolene is a nonspecific agent that weakens all muscles, both voluntary and spastic. It is also toxic to the liver; thus, monthly blood tests are required for life. Generalized weakness, including that of cough and swallowing, make it undesirable, owing to the risk of aspiration pneumonia.

Once monotherapy has been exhausted, then

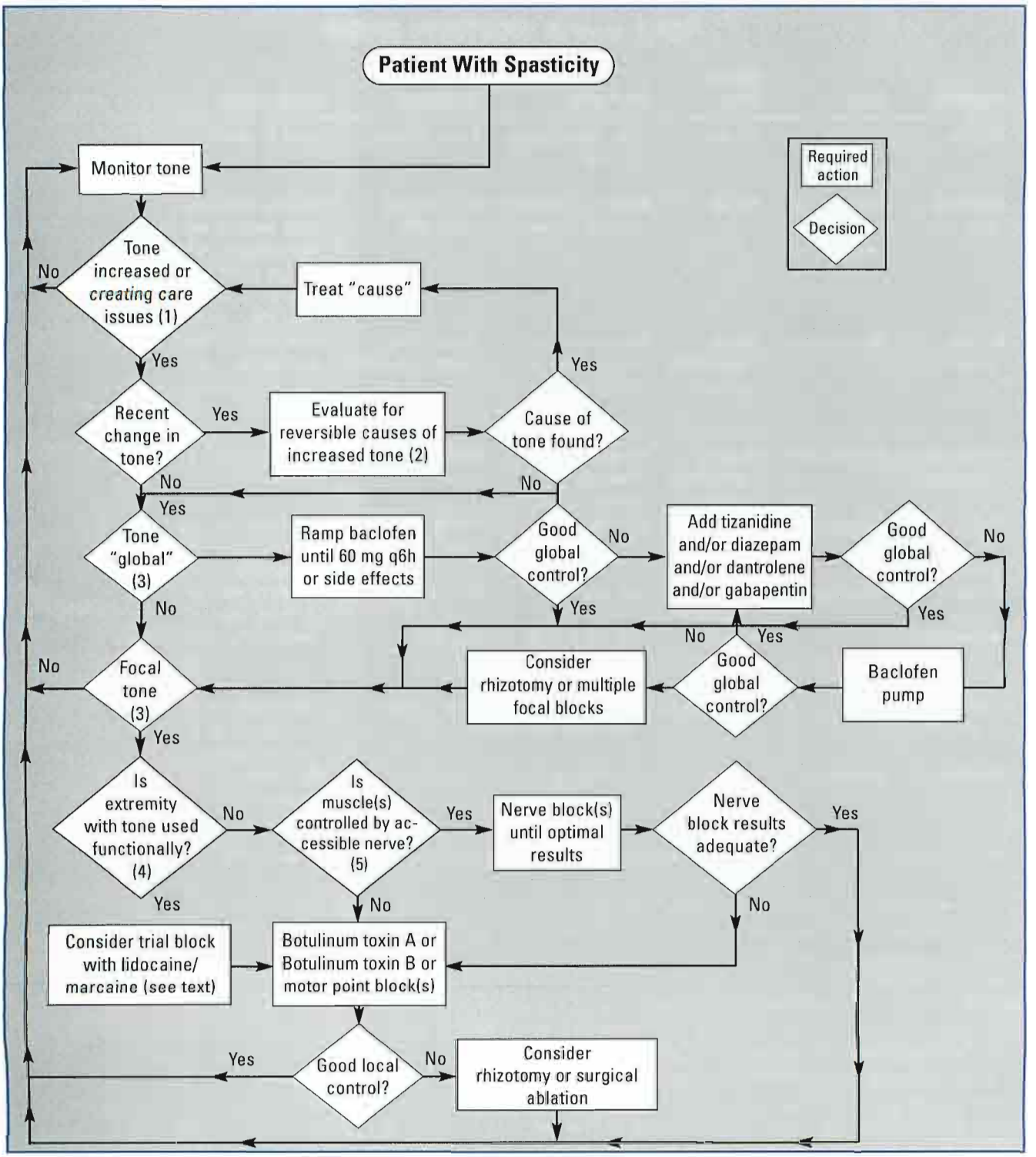

Figure. This algorithm illustrates the general logic in evaluating and treating spasticity. Notes: (1) Spasticity problems = Skin risk or wounds; progressive contracture; inability to position in bed or chair; limitations of mobility or activities of daily living (ADLs); and limited access to palms, underarms, or perineum by care staff. (2) "Reversible" causes of increased spasticity include urinary tract infection, urinary retention, constipation or impaction, wound or skin irritation, medication side effects, withdrawal from narcotics and anxiolytics, other acute illnesses, and synrinx in patients with spinal cord injury. (3) Extremity = Neck, arms, and legs (total of 5); "global" = ≥ 1/5 extremities or spine; "focal" = 1 extremity or 1 muscle group. (4) "Functional" = Neck for swallowing or driving; upper extremity for ADLs; lower extremity for positioning, transfer, or ambulation. (5) Muscles with "inaccessible" peripheral nerves include the paravertebrals, most neck muscles, and the iliopsoas.

using two agents at the same time may be helpful, starting with baclofen and adding tizanidine, increasing the dose of each drug alternately.

Simple/Reversible Procedures. If the oral agents have not proven effective, the next tier of choices includes the baclofen pump, botulinum toxin injection, and the use of peripheral nerve blocks. The dominant considerations are usually the focal or global nature of the muscle tone and how often the patient and staff want to repeat the treatment.

The baclofen pump is best used for global spasticity affecting the spine and lower extremities. Owing to the location of the catheter tip and the risk for respiratory depression, it does not work as well for the upper extremities and rarely works for spasticity of the neck muscles. It is fully reversible and removable. However, surgery is required for placement of the catheter, with the associated risk of infection (rare), the refilling and resetting the pump (every 2–3 mo), and the risk of failure over time (patients seem to need progressive dose increases and catheter issues are not rare). Baclofen can also be mixed with morphine, a nice alternative for patients suffering from high levels of chronic pain, in whom the sedation or other complications of a transdermal fentanyl patch or other oral pain strategies is to be avoided. The baclofen pump is adjusted for the average tone of muscles, so invariably, some muscles may become hypotonic and others remain hypertonic. For those muscles that remain too spastic after the baclofen pump has been adjusted to optimum, selective botulinum toxin or peripheral nerve blocks can be very effective.

Botulinum toxin is an excellent agent for use in small, focal areas.

Botulinum toxin is an excellent agent for use in small, focal areas, such as the neck, arms, and shoulders. Botulinum A is available in 100-unit vials, costing approximately $400 per vial. For neck and upper extremity muscles, one needs between 300 and 400 units every three months. It is rarely helpful in the lower extremities owing to the quantity that would be needed for the several large muscles usually causing lower extremity problems. This formulation must be kept frozen and used within four hours of mixing. Patients can develop antibodies to botulinum toxin over time. Recently, botulinum B has become available. Though comparable in cost to botulinum A, it does not need to be frozen. It has been found that antibodies to botulinum A do not affect botulinum B, therefore, they serve as good alternatives to the other. Other technical and procedural considerations exist, but these are beyond the scope of this article.

The nerve-blocking agents of choice are phenol and ethanol. They are both alcohols that work by causing destruction of the peripheral nerve by denaturaturation of protein. Phenol 5% is approximately equivalent to ethanol 100%. Ethanol is available for injection but phenol is not (and must be prepared by a willing pharmacist). On injection, these agents cause pain that subsides relatively quickly. The alcohol is injected under the guidance of a nerve stimulator, to insure that the agent is delivered as close to the nerve as possible. Whereas many of the nerves injected for spasticity (sciatic, obturator, tibial, median, ulnar, pectoral) are "mixed" nerves, proper site location, below where the sensory branches come off the trunk, minimizes the risk of pain after nerve injection. The alcohols cause a local inflammation, so ice and pain medication after the procedure are helpful. If the affected extremity is also used functionally (such as a leg for transfers or arm for power wheelchair driving), then a trial of a block with a mixture of lidocaine and bupivacaine will give the clinician and therapists a chance to evaluate the potential gains and losses of a long-term block.

End-Stage Procedures. If the above interventions are ineffective or clinically contraindicated (such as for patients taking anticoagulation medication), then more complex surgical procedures, such as rhizotomy or surgical nerve ablation, might be indicated. If the patient needs flap surgery, then an associated procedure to decrease the causative spasticity may be an effective strategy. However, the need for these interventions may represent a failure of the care team to provide timely spasticity care. These procedures tend to promote recurrence in associated regions. Sometimes, the care team admits a patient who has a preexisting spasticity/skin/contracture issue that is already at end stage, and such procedures are necessary and beneficial to get the patient back to a reasonable baseline.

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CASE 3: GLOBAL FLEXOR SPASTICITY

A man with quadriplegia caused by compressive cervical myelopathy was admitted from an acute hospital after decompressive laminectomy. All four extremities were contracted in a fetal-like position. His hips were fixed in adduction, allowing separation of the knees of only two inches, not enough for staff to care for his perineum. He also had a 3 x 3 cm sacral decubitus ulcer. Range of motion was painful in all extremities. He was given gabapentin 300 mg tid for pain management. When started on a dose of baclofen (as little as 10 mg q6h), he developed hypotensive episodes, with change of consciousness. He refused to be evaluated for any further surgery, though a baclofen pump was strongly recommended. He did consent to nerve blocks, however, performed in his room.

He was placed on an alternating air mattress, and his nutritional intake was increased, including Vitamin C and zinc supplementation. He was monitored weekly by the wound care team. The gabepentin dose was slowly tapered without worsening of his symptoms, and the baclofen was then restarted at a very slow ramp-up. His tone began to improve when the baclofen dosage reached 20 mg q6h, and he was adequately controlled globally at a dose of 40 mg q6h. He did not have any further hypotensive episodes. Concurrently, he received alcohol blocks to his obturator nerves, sciatic nerves, and musculocutaneous nerves. Each joint gained about 25 degrees in range, enough to allow him to sit comfortably in a wheelchair daily for two hours with a four-inch Roho brand cushion (www.rohoinc.com).

CASE 4: AMBULATOR WITH ADDUCTOR SPASTICITY

A young male with spastic quadriparetic cerebral palsy still had some ability to transfer, with stand-by assistance, and to ambulate therapeutically with moderate assistance of one. He had marked spasticity of his hip adductors and knee extensors. Owing to excessive adductor tone, the therapist suggested that an obturator block may enable a more functional transfer and gait. Since the legs were being used functionally, a trial block was suggested to assess the potential effects. A combination of lidocaine and bupivicaine was used to achieve a one- to six-hour therapeutic window. With loss of the adductor muscle tone, he also lost tone in his quadriceps muscles, and thus was unable to transfer or ambulate as he did with the adductor tone.

Though counterintuitive, it became clear that some of the strength of his quadriceps came through overflow from his adductor tone. Even though the quadriceps are innervated by a different nerve, they share the same myotome as the adductors, and thus through this overflow maintained his strength. If a trial block had not been done first, then a permanent nerve block would clearly have worsened his function.

REHABILITATION THERAPY ASSOCIATED WITH SPASTICITY INTERVENTIONS

Before and after any intervention for spasticity, the rehabilitation therapy team should be involved. After any intervention, a period of intensive therapy can maximize the range, tone, and functional consequences of the intervention, and give the clinician guidance as to additional procedures that might be warranted (as well as side effects). The rehabilitation therapy team can participate in peripheral blocks and decisions on whether to use the baclofen pump.

An acute inpatient rehabilitation admission for the purpose of assessment, serial blocks, aggressive ROM physical therapy, and prescription or wheelchair and/or bed positioning devices is a service that is generally underprovided to these patients.

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Address for correspondence: Michael I. Cheikin, MD, Medical Director, Chestnut Hill Rehabilitation Hospital, 8601 Stenton Avenue, Philadelphia, Pennsylvania 19038. E-mail: cheikinm@msn.com.