# Module 4: Optimizing Outcomes in Spasticity Treatment

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## Abstract

With many recent advancements in spasticity treatment, more patients are surviving critical illness and injury but are left with ongoing disability that needs constant treatment. Such treatment will change as the patient's condition evolves. Constant appraisal of treatment efficacy and patient progress is therefore an important component of spasticity management, and physicians need to be familiar with how to troubleshoot treatment regimens when outcomes of that regimen become suboptimal. This module considers how to optimize the use and outcomes of major treatment modalities and provides drug and device maintenance algorithms to guide the treating team.

**Keywords:** Troubleshoot- spasticity treatment regimens - suboptimal outcomes - Muscle spasticity - Treatment outcome - Baclofen/adverse effects - Implantable/adverse effects - Botulinum toxins/adverse effects - Muscle relaxants, central/adverse effects

## Learning Objectives

Upon completion of this module, the learner will be able to:

1. Identify reasons why botulinum toxin, intrathecal baclofen therapy, oral medications, or phenol/alcohol treatment are producing suboptimal outcomes
2. Discuss the need for constant reappraisal of the patient – goals, progress, and concomitant conditions
3. Enumerate ways to troubleshoot suboptimal outcomes of spasticity treatment.

## Addressing Suboptimal Outcomes of Spasticity Interventions

By its very nature, spasticity treatment is not a single, isolated, intervention but continues over months and years. Treatment will also progress over time and different treatment modalities will be employed as the patient's condition changes. It is not unusual for a seemingly effective treatment to start to show decreased efficacy and it is important therefore to be able to troubleshoot why efficacy is waning before making radical alterations to the care plan. Equally, treatments can produce over responses and these must be addressed before plans are changed. Side effects of treatments and other developing neurological conditions can also affect treatment outcomes.

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Suboptimal outcome following any treatment modality can have multiple etiologies. Concerns that the usual treatment is producing suboptimal outcomes may be raised by the patient, their family and/or carers, or the treating physician.

When a patient is reporting a poorer effect than expected it is important to:

- Listen to their concerns to help identify the problems
- Review previously agreed upon goals
- Ask if they think the treatment did not work at all
- Enquire if the effect wore off too fast
- Establish if the treatment is no longer working after experiencing good effects in previous cycles
- Elicit adverse effects, such as increased weakness
- Consider other side effects related to the treatment, among others.

Listening to patients' concerns expressed in their own terms may provide clues and help point to a real lack of efficacy of the spasticity treatment, or if another problem is impinging on the patient's condition and causing a temporary increase in the spasticity. A good first step is to review the previously agreed upon goals and the treatment objectives should be reviewed and documented. Goals of treatment should be reviewed using the SMARTER goal system (Specific Measurable Achievable Realistic Timely Ethical Recorded) (see Module 1 in this Supplement).

It should be considered whether the goals were appropriate and whether the patient and/or their carer were motivated to achieving these goals. Furthermore, patient expectations may change over time and it is important to assess how the patient views their progress and the future.

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The information previously discussed with the patient about their spasticity and how the treatment acts to address the problem should be re-iterated. It is important to use patient-friendly language and vocabulary; "patient-like wording" instead of medical terminology ensures clearer communication.[1]

It is telling that in a recent international survey of patients living with spasticity, 45% of respondents reported dissatisfaction with the information provided at diagnosis. Of these, 67% wanted more information; 36% felt the clinician used too many technical words or there were problems with communication; and 35% wanted more time with their doctor to better understand their diagnosis and its future consequences. Only 5% were overwhelmed by the information provided.[2]

For treatments that are repeated at regular intervals, such as botulinum toxin injections, it may be an issue of the frequency of treatment schedules. For continuous systems such as baclofen pumps that also rely on properly functioning equipment; there are other considerations.

## Treatment Algorithms

Table 1 is a comprehensive listing all the items that need to be considered when assessing under response or over response

**Table 1: Items for consideration when troubleshooting spasticity treatment**

| Item | Considerations |
|---|---|
| Goals | Unrealistic goals – no spasticity treatment can "undo" what has been done (i.e., neurotoxin cannot reverse the effects a spinal cord injury has on weakness, sensory changes, proprioception changes) |
| | Misaligned goals – if the treating physician has one set of goals, but the patient/caregiver/family has different goals, even the most successful treatment in the eyes of the physician can appear to be "ineffective" |
| | Previously met goals – goals can/will change over time. Periodic reassessment of goals is necessary over the course of treatment[3] |
| Injection technique | Knowledge of anatomy (including muscles, blood vessels, nerves) is key for proper injection technique |
| | Whenever possible (given local resources), guidance techniques, such as ultrasound, electrical stimulation, and/or electromyography should be used to assist in guidance for injection technique |
| Muscle selection | Proper knowledge of functional anatomy is essential to properly treat a problematic limb pattern due to spasticity |
| | For example – for wrist flexion, knowing that the long finger flexors also contribute to wrist flexion in addition to finger flexion is necessary to properly treat some cases of excessive wrist flexion |
| Not enough muscles | Need to consider all muscles responsible for a particular posture |
| | For example – when formulating a plan for ankle plantarflexion, you may need to also consider other muscles that cross the ankle and can contribute to plantarflexion such as flexor digitorum longus, flexor hallucis longus[4] |
| Dosing (per muscle) | Individual muscles may need different doses of toxin in different patients, depending on a number of factors |
| | Goals – passive functional goals may necessitate greater dose; active functional goals may need smaller doses |
| | Severity of spasticity |
| | Muscle size |
| | Dose ranges are published, but clinical practice falls outside these dosages |
| | Off-label muscles may not have as many published dose ranges |
| Disease progression | Majority of spasticity encountered is due to nonprogressive etiology (i.e., stroke, spinal cord injury, acquired brain injury, cerebral palsy), and is static in nature (although things like noxious stimulus can temporarily increase spasticity) |
| | Upper motor neuron syndromes due to multiple sclerosis or other potentially progressive conditions will lead to spasticity that can change over time due to the progression of the condition itself |
| Concomitant medical conditions | In most conditions resulting in spasticity, the underlying condition is nonprogressive, thus spasticity should not progress over time (after reorganization) |
| | Underlying medical conditions may increase spasticity due to the condition acting as a noxious stimulus to the central nervous system (constipation, urinary tract infection, ingrown toenail) |
| | Underlying medical conditions may increase spasticity such that the apparent effect of the injections is lesser than would be if the underlying condition was treated/not present |
| | Caution must be taken to account for the presence/absence of concomitant conditions when selecting dosages |
| Correct handling of the drug | Most neurotoxins require refrigeration |
| | Improper transportation/handling/storage could potentially lead to decreased effectiveness of the neurotoxin |
| | Improper reconstitution could lead to incomplete extraction of toxin from vial |
| Use of adjunctive treatments | Botulinum toxin injections alone may decrease spasticity but may not improve other factors, such as range of motion, goal attainment |
| | Optimal outcomes are typically seen when botulinum toxin injections are combined with therapy, bracing, ES, etc. |
| Correct timing for re-evaluation after injection | Evaluating response to botulinum toxin injections is best performed at peak effect of the botulinum toxin response – generally 4-6 weeks |
| | Doing evaluation prior to this time may be too early to see the magnitude of effect |
| | Doing evaluation at 12 weeks and beyond will be after effect has waned |
| | Opportunity for virtual assessment |

*Contd...*

**Table 1: Contd...**

| Item | Considerations |
|---|---|
| Unmasking weakness | Treating muscles that have spasticity will reduce the excessive activation of muscles |
| | These muscles frequently are weak in addition to hypertonic |
| | Some patients utilize the spasticity to substitute for underlying weakness |
| | This reduction of "useful spasticity" can result in an unmasking of excessive weakness, leading to decreased function |
| Local side effects | Physical effects |
| | Hematoma |
| | Infection |
| | Nerve injury |
| | Local effects of medication |
| | Muscle weakness |
| Systemic side effects | Neurotoxin side effects |
| | Distant spread of effect |
| | Flu-like syndrome |
| | Oral medication side effects |
| | Baclofen – sedation, constipation, caution in renal impairment |
| | Tizanidine – sedation, dizziness, hepatic dysfunction |
| | Dantrolene – nausea and vomiting, diarrhea, hepatic dysfunction |
| | Benzodiazepines – sedation, impaired memory and attention, impaired motor coordination |
| | Gabapentin – sedation, headache, nausea, and vomiting |
| | Cannabinoids – anxiety, psychosis, panic attacks |
| | Withdrawal syndromes from oral medications |
| Dilution | The literature is inconsistent, however, there is some evidence that increased volume can lead to increased diffusion (advantageous) but also increased spread (undesirable)[5–8] |
| | Can also decrease volume to limit diffusion and spread[9–11] |
| Spasticity was not the main issue | Contracture, other rheologic changes can contribute to disability. If muscles are appropriately treated and range of motion is unchanged, contracture may be primary issue |
| | May need to consider surgical intervention to improve range of motion |
| | Need to still manage spasticity after lengthening for example |
| | If using spasticity to advantage, weakness can be unmasked, decreasing function |
| Other neurological diseases (NMJ disease, etc.) | Neuromuscular junction diseases, motor neuron diseases can make patients more sensitive to typical dosages of neurotoxin |
| New medications | Medication that improves pain may indirectly improve spasticity by decreasing a noxious stimulus which was causing an increase in spasticity |
| | Medication that has a side effect that is seen as a noxious stimulus may cause a secondary increase in spasticity (constipation caused by opiates can increase spasticity) |
| | Interaction of medications |
| | Synergistic effect of aminoglycosides and botulinum toxins |
| | Inhibition of metabolism of tizanidine with concurrent administration of fluoroquinolones |
| Choice of the most appropriate toxin | Need to be mindful of noninterchangeability of toxins, no "conversion rates" |
| | Need to have knowledge of appropriate dosages and titration of each toxin |
| Change in patient's expectations | Need to continue to assess patient's goals, change goals if prior goals met |
| | Re-evaluating goals at each visit/periodically realign goals with patients or caregivers and physicians, therapists |
| Development of antibodies | Neutralizing antibodies can cause a secondary nonresponse to botulinum toxin a in a variety of settings[13] or antibodies against BoNT-A[12] |
| Change in soft tissue porperties | Contracture, other rheologic changes can contribute to disability. If muscles are appropriately treated and range of motion is unchanged, contracture may be primary issue |
| | May need to consider surgical intervention to improve range of motion |
| | Need to still manage spasticity after lengthening for example |
| Too long an interval between injections | Allowing effect of the injection to wear off completely and have patient get "back to baseline" can lead to excessive "up and down" in function[14] |
| Individual variability | The duration of effect of botulinum toxin can differ from an individual to another. Even if the mechanisms underlying this phenomenon are not completely known (muscle mass, muscle fibrosis, neutralizing antibodies, genetical variations, muscle activity), this has been reported for several years[15] |
| Compliance with medication | Inconsistent compliance with oral medications will lead to suboptimal change in spasticity and subsequent perceived treatment failure |

*Contd...*

**Table 1: Contd...**

| Item | Considerations |
|---|---|
| Changes following reinervation | Phenotype of spasticity may change when reinnervation occurs following neurolysis |
| | Under response to alcohol/phenol – it may be more difficult to localize the correct injection site after reinnervation since the anatomy may no longer be "typical" |
| | Over response for alcohol/phenol – neurolysis of more muscles than intended, more difficult to titrate |
| Pump problem or not | Refer to troubleshooting pump problems algorithm |

NMJ: neuromuscular junction, BoNT: Botulinum neurotoxin, ES: Electrical stimulation



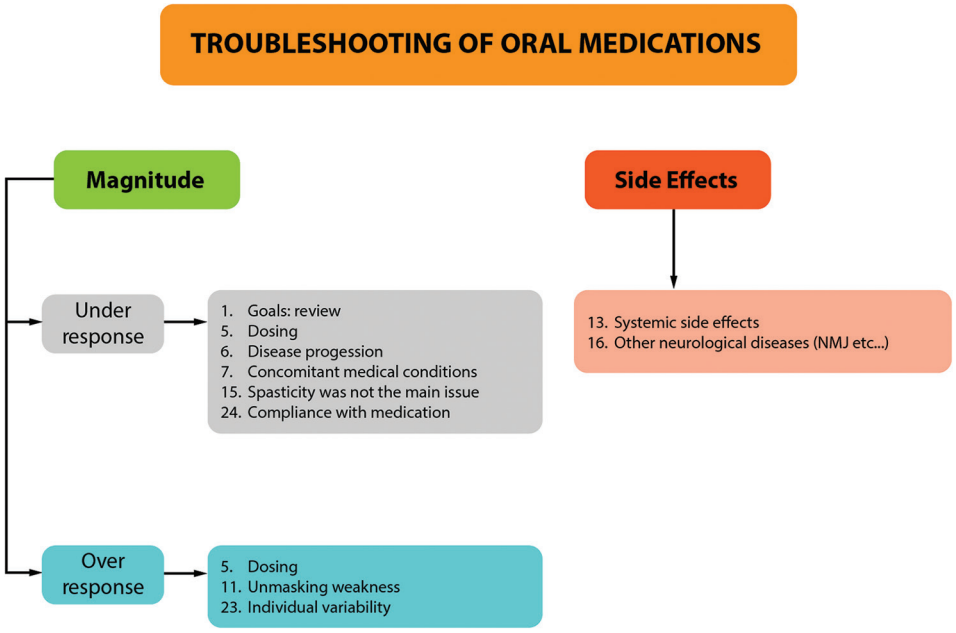


**Figure 1:** Algorithm for troubleshooting oral medications

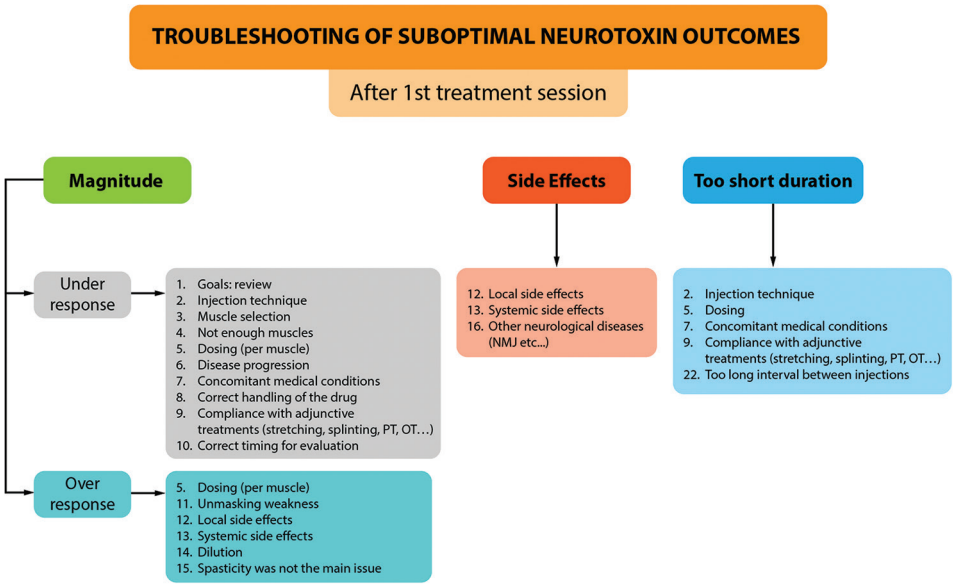


**Figure 2:** Algorithm for troubleshooting of suboptimal neurotoxin outcomes after the initial treatment session

to spasticity treatments.[3-12] The clinical treatment algorithms Figures 1-4 will provide guidance to which items need to be reviewed under different circumstances – considering both treatment modality and the nature of the problem.

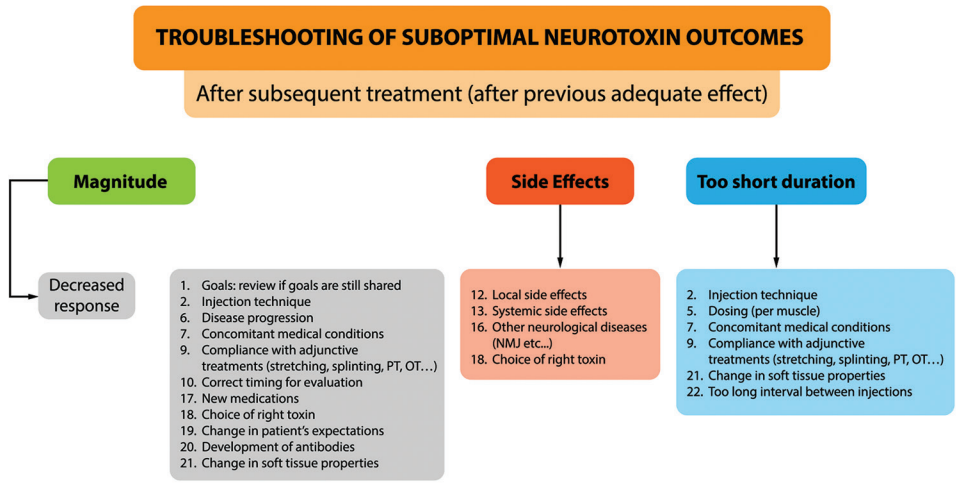


**Figure 3:** Algorithm for troubleshooting suboptimal neurotoxin outcomes after subsequent treatment sessions

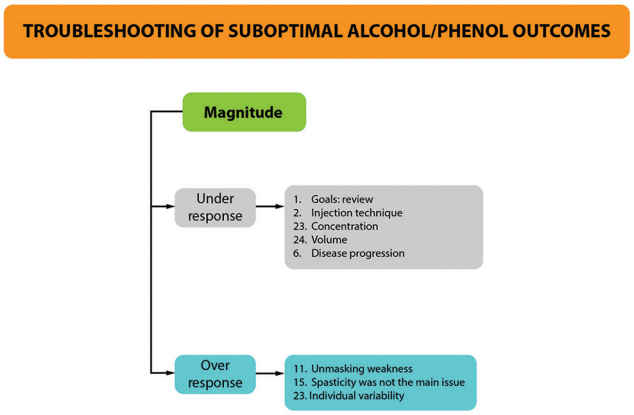


**Figure 4:** Algorithm for troubleshooting of suboptimal alcohol/phenol outcomes

In addition, there are special considerations for maintaining and troubleshooting intrathecal baclofen pumps and these are considered separately [Figures 5 and 6].

### Troubleshooting oral medications

Once the goals have been reviewed, it is necessary, as with any oral regimen, to ensure that the patient is showing compliance with the treatment and taking medication regularly and as prescribed. The dose and timing of the medication should be reviewed.

The patient's overall condition in terms of spasticity progression and other medical comorbidities should be assessed and their spasticity medication adjusted accordingly with referral or additional prescribing for comorbidities.

The algorithm provides guidance on items to review.

## Troubleshooting Botulinum Toxin A/Phenol/Ethanol Injections

BoNT-A and neurolytic agents are injected at multiple sites which raise issues such as: Were the muscles targeted correctly related to the goals; was the muscle localization technique adequate; were doses per muscle adequate or underdosed; was the adjunctive therapy carried out; was there any concurrent event that might have influenced a poor outcome?

In cases of suboptimal response to treatment important areas to address include;

- Goals
- Technique
- Concentration
- Volume
- Progression of disease
- Nerve/muscle selection.

Spasticity not the main issue in cases of exaggerated response (e.g., probable overdose), the areas to evaluate include:

- Goals
- Unmasking weakness
- Progression of disease
- Nerve/muscle selection
- Spasticity not the main issue.

The algorithms address troubleshooting after the first treatment session and after subsequent treatment sessions.

Troubleshooting after alcohol/phenol injections follows a similar pathway.

## Approach to Troubleshooting Intrathecal Baclofen Systems

Intrathecal baclofen is an effective means of managing severe problematic spasticity in patients. However, these systems can malfunction either acutely or over time. Knowing how to troubleshoot these systems can improve patient outcomes and minimize morbidity in a comprehensive spasticity management practice.

Decreased response, review:

- Pump problem or not?
- Goals

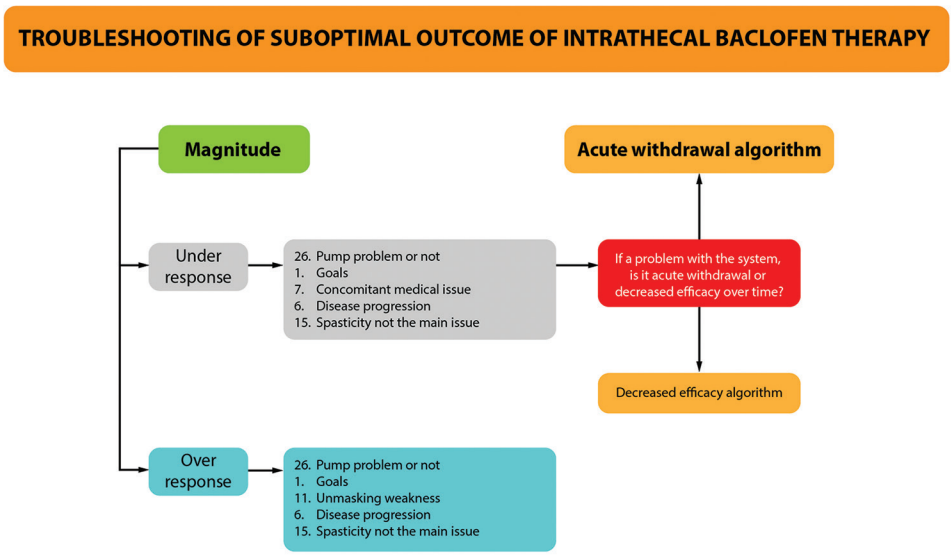


**Figure 5:** Algorithm for troubleshooting suboptimal outcomes of intrathecal baclofen therapy

- Concomitant medical issue
- Disease Progression
- Spasticity not the main issue.

Excessive response, review:

- Pump problem or not?
- Goals
- Unmasking weakness
- Progression of disease
- Spasticity not the main issue.

In addition:

- Acute withdrawal versus decreased control of spasticity over time.

## Assessing Pump Problems

Because of the nature of intrathecal baclofen systems, troubleshooting of these systems necessitates special consideration. Two best practice algorithms for troubleshooting intrathecal baclofen therapy were published in 2016.[16] The first was for loss-of-efficacy and applies to patients with previously well-controlled hypertonia on a stable dosing regimen who have increased spasticity. The second algorithm was for emergent care and describes treatment of overdose or withdrawal, which requires immediate care in a monitored setting and restoration of baclofen delivery. They also address suspected overdose or over-infusion.

These algorithms provide a useful basis for troubleshooting; however, local resources vary and each treatment center or practice will need to develop their own pathway for troubleshooting that uses their own resources to best advantage.

## Troubleshooting Intrathecal Baclofen Therapy-related Problems

Three main scenarios when troubleshooting is indicated are:

- When there is suspicion of medication overdose or pump over infusion
- Medication withdrawal or pump under infusion
- Loss of efficacy of the therapy over time.

However, many things can mimic baclofen overdose or underdose. The first step in any trouble setting algorithm is identifying other medical issues which weight may be masquerading as an intrathecal baclofen system malfunction.

Thankfully, baclofen overdose is a rare entity. The etiologies are typically programming error, using the wrong medication concentration, and very rarely, is the phenomenon of device over infusion.

Troubleshooting an intrathecal system is indicated when medication overdose or over-infusion is suspected, when withdrawal or under-infusion is suspected or when efficacy of the medication has decreased over time.[16]

As a first step conditions that mimic incorrect dosing must be eliminated to help confirm that the problem really lies in the pump or catheter delivery. Some conditions can confound identification of the problem as they may produce the same symptoms. For example, withdrawal or increased spasticity may be mimicked by autonomic dysreflexia, serotonin syndrome, noxious stimuli, urinary tract infections (UTIs), or constipation. Overdose or decreased spasticity may be mimicked by multiple sclerosis (MS) exacerbation, a new neurologic event (i.e., stroke), or excessive oral medication.

## Overdose

The signs or symptoms of baclofen overdose include dizziness, somnolence, hypotonia, respiratory depression, seizures, and coma. This can arise when the apparatus is programmed incorrectly by the user (e.g., entering 500 mcg/day instead of 50 mcg/day). Errors can also be made in drug concentration

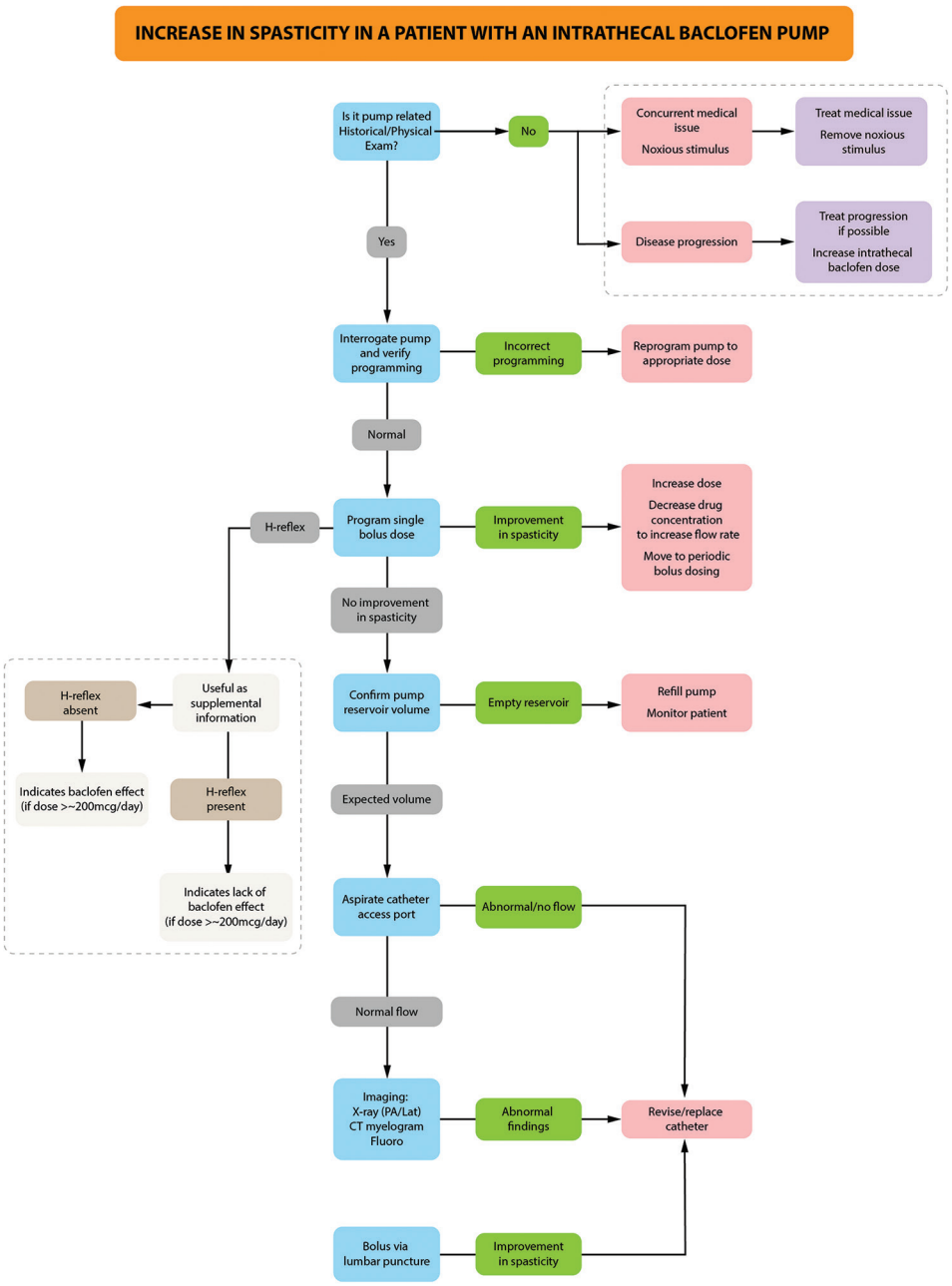


**Figure 6:** Algorithm for troubleshooting intrathecal baclofen pump malfunction[16]

(e.g., using medication of concentration 2000 mcg/mL rather than 500 mcg/mL). More rarely, the pump may deliver too much medication but this has been reported in < 0.16% of cases (Implantable Systems Performance Registry, August 2003 to July 2015, Medtronic).

The management of baclofen overdose is to maintain airway/breathing/circulation and decrease the intrathecal dose by programming the pump to minimum rate or withdrawal of large volume lumbar puncture (LP)/catheter access port (CAP). It should be noted, following a dramatic ITB dose reduction, the patient must be assessed and treated for symptoms of baclofen withdrawal. However, these symptoms should be self-limited.[16]

## Underdose

Withdrawal or under-infusion symptoms include rebound spasticity, pruritis without a rash, mental status changes, fever, or even death.

As with overdosing, a programing error may be responsible for underdosing (e.g., entering 50 mcg/day instead of 500 mcg/day) or errors can be made in the drug concentration (e.g., using medication with 500 mcg/mL concentration instead of 2000 mcg/mL).

If human error is not responsible, the device may have a malfunction (i.e., critical motor stall) or an empty pump

reservoir if the patient or caregiver has missed the alarm date or a refill appointment. The pump may be at the end of its useful service period. Typically, device batteries have a life of approximately 7 years and the device needs to be replaced before the end of battery life. Catheters may also be the source of system malfunction if they become occluded, kinked, or disconnected. One other source of underdosing can result from the so-called "pocket fill." In these cases, the refill needle may become dislodged from or never be properly located in the pump reservoir. When this happens, the medication gets delivered subcutaneously rather than into the pump.

A loss of efficacy may also present slowly as a change over time rather than acute withdrawal. This is seen when the ITB does not give the same effect on spasticity as was achieved with the test dose despite dose escalation. A diminishing effect of the medication on spasticity where there was previous good control can also indicate loss of efficacy.[16]

Apparent loss of efficacy could be due to disease progression or MS exacerbation or other medical issues such as constipation or a deep vein thrombosis.

Baclofen withdrawal or under-infusion should be managed by administering an intrathecal bolus of the medication. Restoring the intrathecal delivery of baclofen is first step if possible, using LP, external indwelling catheter, or pump (if known to be functioning).

Oral or enteral baclofen is another option; if the patient is able to take the medication oral administration is the simplest or, if a percutaneous endoscopic gastroscopy tube is present, this can be utilized. There is no intrathecal to oral conversion; typically, the patient should be given the preimplant dose. If the prior dose not known, 10–20 mg every 6 h should be considered.

Intravenous benzodiazepines are an option but these need cardiac/respiratory monitoring. They can also be prophylactic for seizures. Cyproheptadine may be given at 2–4 mg every 6 h.

Creatine kinase and renal function should be monitored and the patient assessed for rhabdomyolysis.

The "syndromes" associated with catheter malfunction are given in Table 2 and the incidence of catheter malfunction is given in Table 3.

Some of the important points to consider are outlined here.

Firstly, as the data above have shown, most of the time a patient's, caregiver's, or other provider's concern of increased spasticity is NOT a pump or a catheter problem. Hence, patients must be assessed for their recent medical history, paying attention to any illnesses, medication changes, or environmental changes. Compare the spasticity on examination to prior examinations. For many patients, intrathecal baclofen withdrawal is often described as "worse than before getting the pump." The patient should be assessed for changes in mental status and review whether there is a source of noxious stimuli.

While problems with the pump itself are rare, inaccurate programming is often the culprit. It is important to verify the

**Table 2: "Syndromes" associated with catheter malfunction**

| Catheter "syndrome" | Symptoms | Testing | Action | Comment |
|---|---|---|---|---|
| Microleak | Progressive loss of efficacy | Xray - NL<br>CAP - NL<br>Dye study - NL<br>CT - NL<br>Bolus - + response | Replace catheter/ segment (if segment can be identified) | Bolus doses may mask small leaks |
| Subdural catheter placement/migration | Vacillating response with alternating overdose and underdose | Xray - NL<br>CAP - NL<br>Dye study - NL<br>CT - Abnormal<br>Bolus - + response | Replace catheter/ segment (if segment can be identified) | Bolus doses may mask |
| Loculation at catheter tip | Progressive loss of efficacy | Xray - NL<br>CAP - abnormal<br>Dye study - NL<br>CT - abnormal<br>Bolus - + response | Replace catheter | Bolus doses may mask |
| Catheter migration | Acute loss of efficacy/ withdrawal | Xray - abnormal<br>CAP - abnormal<br>Dye study - abnormal<br>CT - abnormal<br>Bolus - response | Replace catheter | - |
| VP shunt malfunction | Progressive loss of efficacy | Xray – NL<br>CAP - NL<br>Dye study - NL<br>CT - NL<br>Bolus - + response | Revise shunt | Increase in CSF volume increases baclofen volume of distribution |

Taken from.[17] CAP: Catheter access port, CT: Computed tomography, CSF: cerebrospinal fluid, VP: Ventriculo-peritoneal, NL: Normal

**Table 3: The incidence of catheter malfunction**

| Event | Number of events (% of patient with event) |
|---|---|
| Catheter dislodgement | 255 (3.19) |
| Catheter occlusion | 242 (3.24) |
| Catheter leak | 32 (0.45) |
| Catheter disconnection | 39 (0.56) |
| Pump motor stall | 89 (1.18) |
| Overinfusion | 4 (0.16) |

Implantable systems performance registry August 2003 to July 2015. Medtronic

actual drug and concentration put in the pump. If the pump alarm is sounding, the pump may be empty, at the end of battery life, or experiencing motor stalling. The pump log should be checked.

The pump volume must be checked since it does not have a "fuel gauge." There may be too little drug in the reservoir due to "pocket fill." If this problem is leading to subcutaneous delivery of baclofen, it would be poorly absorbed and not lead to overdose. Downstream obstruction in the system could lead to a higher-than-expected volume.

The CAP must be checked using the specified kit to access the CAP. This is the only needle that can access the CAP – the refill needle cannot be used. A total of 2–3 mL of fluid should be aspirated, all the baclofen in the catheter will have been withdrawn after the first ~0.25–0.5 mL, and the system will be withdrawing cerebrospinal fluid (CSF). Inability to aspirate easily suggests obstruction downstream and the catheter MUST be replaced.

It is important to program a priming bolus if the system is flowing freely and drug/CSF are withdrawn. Forgetting to perform a priming bolus can lead to withdrawal since the spinal catheter will be empty.

Imaging is useful to confirm what components of the system are implanted. Some components (Medtronic Ascenda™ catheter) are less visible on plain films. A computed tomography (CT) myelogram/Dye Study may be necessary. It is important to make sure that the catheter is aspirated via CAP before instilling dye. Radiopaque dye may be instilled under fluoroscopy or dye may be instilled in the CT scanner immediately before scan.

When programming the bolus dose, the same dose as the test dose should be used if the patient is not currently receiving bolus medication. The clinical response to bolus administration should be assessed where soleus H-reflexes can be useful as they are sensitive to intrathecal baclofen.[18]

It must be remembered that the algorithm is only an example. Local resources must be employed and the algorithm modified to produce an acceptable way to troubleshoot the system. Consideration should be given to team members skill in accessing the CAP, who participates in managing baclofen pump patients, whether there should be baclofen pump clinic days and education for the local emergency department.

When "inheriting" a patient with a baclofen pump, it may be worth investing some time is checking the device, verifying dose concentration etc., and ensuring that the indication for intrathecal baclofen in the first place is clear; the information should be shared with the management team.[19]

## Case History

### Case 2

A 58-year-old male patient suffering from right sylvian ischemic stroke (right internal carotid artery dissection, happened at the age of 52) with left hemiplegia, left homonymous hemianopia, left neglect, and persistent anosognosia has been treated for severe hand spasticity with BoNT-A two times, 4 and 7 months after stroke.

He was lost at follow-up and came only 3 years later, due to a skin infection of the palm. His left hand had a severe clenched fist, and, after a new BoNT-A treatment and antibiotics, he recovered.

6 months after his treatment the patient comes back [Figures 7-9].

He was treated with incobotulinumtoxinA, under ultrasound guidance, to the following muscles:

- Interossei: 80 UI
- FDS: 190 UI
- FDP: 60 UI
- FPL: 20 UI.

Dilution was 1 ml 0.9% saline: 100 UI for interossei and FPL and 2 ml 0.9% saline:100 UI for the other muscles. The primary objective was hygiene ease (opening the hand alone to clean the palm).

## Competency Assessment

The answers to the Competency Assessments can be found at the end of the module before the references.

1. Which are the most frequent reason for a suboptimal outcome following BoNT-A?
2. What conditions can mimic apparent increased spasticity in a patient treated with intrathecal baclofen?
3. In the case history discussed above, what are the possible explanations for the result seen at 4 weeks after the last BoNT-A treatment?

## Competency Assessment Answers

1. Which are the most frequent reason for a suboptimal outcome following BoNT?

Expected content of answer:

The most frequent reasons can be summarized in 3 groups:

1. Goal-related: Revise goal appropriateness, formulation, and measurement
2. Procedure-related: Correct muscle selection, correct targeting, correct dose
3. Patient-related: Patient motivation, cooperation, concomitant health problems

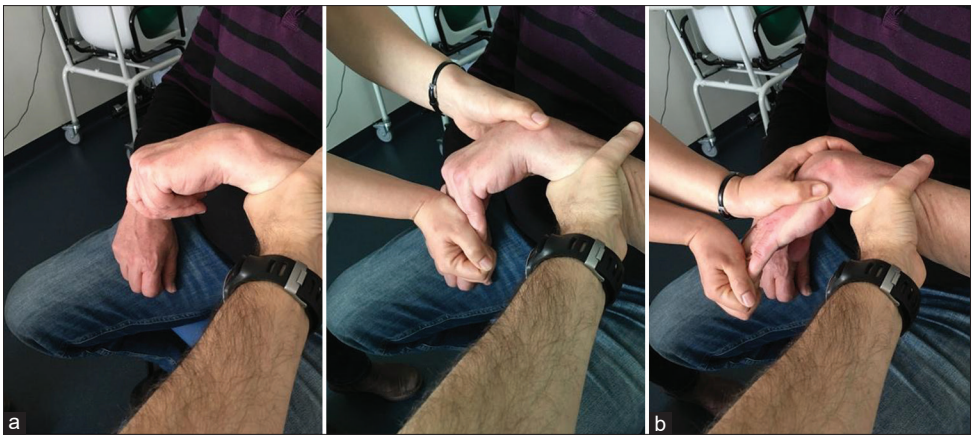


**Figure 7:** (a and b) Left-hand posture 6 months after last BoNT-A treatment

**Figure 8:** The result of this treatment 4 weeks after last BoNT-A treatment. The patient is quite disappointed about his hand posture

**Figure 9:** Four months after. The same patient, two weeks after having repeated the same BoNT-A treatment but this time with the addition of taping for 7 days

2. What conditions can mimic apparent increased spasticity in a patient treated with intrathecal baclofen?

Expected content of answer:

Withdrawal or increased spasticity may be mimicked by autonomic dysreflexia, serotonin syndrome, noxious stimuli, UTIs, or constipation.

3. In the case history discussed above, what are the possible explanations for the result seen at 4 weeks after the last BoNT-A treatment?

Expected content of answer:

Since the dose and dilution utilized are adequate, we should check for:

a. Goals: Is the goal realistic? If the patient had some contractures, the result achieved is the maximum possible. Anosognosia can make harder/impossible for the patient to evaluate his improvement
b. Procedure related: Did the patient have any adjuvant therapy?
c. Patient-related: Which was patient's compliance with correct positioning and how did he take care of his arm after the treatment?

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