PRACTICE PATTERNS FOR SPASTICITY MANAGEMENT WITH PHENOL NEUROLYSIS

Jay KARRI, MD, MPH, Manuel F. MAS, MD, Gerard E. FRANCISCO, MD and Sheng LI, MD, PhD From the Department of Physical Medicine and Rehabilitation, University of Texas Health Science Center at Houston (UTHealth) McGovern Medical School and The NeuroRecovery Research Center at TIRR Memorial Hermann, Houston, TX, USA

Objective: To present practice patterns for phenol neurolysis procedures conducted for spasticity management.

Design: A retrospective review of 185 persons with spasticity who underwent phenol neurolysis procedures (n = 293) at an academic rehabilitation hospital and clinic. Patient demographics, concomitant spasticity treatments, and procedure relevant information were collected.

Results: The cohort included 71.9% males and 61.6% inpatient procedures. Neurological diagnoses included stroke (41.0%), traumatic brain injury (28.6%) and spinal cord injury (24.3%). Musculoskeletal diagnoses included spastic hemiplegia or paresis (51.3%), tetraplegia (38.4) and paraplegia (9.2%). At the time of phenol neurolysis, most patients (77.5%) received concomitant pharmacological treatments for spasticity. Injection guidance modalities included electrical stimulation and ultrasound (69.3%) or ultrasound only (27.3%). A mean of 3.48 ml of phenol were injected per nerve and 10.95 ml of phenol were used per procedure. Most commonly injected nerves included the obturator nerve (35.8%) and sciatic branches to the hamstrings and adductor magnus (27.0%). Post-phenol neurolysis assessment was recorded in 54.9% of encounters, in which 84.5% reported subjective benefit. Post-procedure adverse events included pain (4.0%), swelling and inflammation (2.7%), dysaesthesia (0.7%) and hypotension (0.7%).

Conclusion: Phenol neurolysis is currently used to reduce spasticity for various functional goals, including preventing contractures and improving gait. Depending on the pattern of spasticity displayed, numerous peripheral nerves in the upper and lower extremities can be targeted for treatment with phenol neurolysis. Further research into its role in spasticity management, including studies exploring its cost-effectiveness and pharmacological and sideeffects compared with other treatment options are needed.

Key words: phenol; neurolysis; spasticity.

Accepted Apr 13, 2017; Epub ahead of print May 25, 2017

J Rehabil Med: 2017; 49: 482-488

Correspondence address: Sheng Li, Department of Physical Medicine and Rehabilitation, University of Texas Health Science Center at Houston, McGovern Medical School, Houston, TX, USA. E-mail: sheng.li@ uth.tmc.edu Characterized by hyperexcitable stretch reflexes that increase muscle tonicity and exaggerate tendon jerks, spasticity is a common motor disorder that follows a variety of central nervous system insults (1). Implicated neurological insults most often include stroke, traumatic brain injury (TBI) or spinal cord injury (SCI). Spasticity is often associated with various complications including joint contractures, muscle shortening and postural deformities (1) that lead to multiple impairments. Early goal-directed spasticity management is instrumental in helping increase the likelihood of good outcomes and limiting complications (1, 2). Unfortunately, a lack of universally standardized management and an abundance of therapeutic options make spasticity management a challenging task.

Currently, spasticity is frequently managed through a combination of therapeutic modalities, pharmaceutical options and surgical procedures (3). Pharmaceutical options include medications delivered orally, via local injections, or through intrathecal pumps. Oral medications, including baclofen and tizanidine, help decrease spasticity (3). However, systemic side-effects, such as generalized muscle weakness, sedation, confusion, and hypotension, preclude the use of higher dosages that might be warranted for control of moderate-to-severe spasticity (3, 4). Intrathecal baclofen pump (ITB) is often indicated in treating severe and/or diffuse spasticity as a means to deliver high-dosage baclofen with less concern for systemic side-effects (4). Although ITB treatment is very effective, numerous complications and the requirement for commitment to maintenance associated with this treatment makes it favourable only for some patients with severe spasticity (4, 5).

Chemoneurolysis via localized injections can help provide focal spasticity relief (1, 3, 6). In addition, the use of single-event multi-level chemoneurolysis helps treat several areas of muscle spasticity, each with varying severities (7). Medications used in chemoneurolysis procedures include botulinum neurotoxin (BoNT), phenol, and alcohol neurolysis (3–7). Compared with phenol and the understudied alcohol neurolysis, BoNT usage in treating spasticity is documented extensively in the literature with regards to pharmacodynamics, adverse effects and clinical benefits (7–9). However, the response to chemodenervation with BoNT often requires 3–5 days to generate JRM

spasticity benefit, which generally lasts approximately 3 months. Although clinical standards permit repeating chemodenervation every 3 months, the majority of patients with spasticity prefer an increased frequency for maintaining clinical benefit (10-12). BoNT injections are associated with significant costs, and repeated injections are often further restricted by financial feasibility. In the USA, depending on the insurance being used, the approved dosage of BoNT is only 400-600 units of every 3 months. These limitations prevent the sole utility of chemodenervation for a multi-pattern treatment, e.g. elbow flexion, clenched fist, stiff knee gait, and equinovarus of the foot. Consequently, phenol neurolysis (PN) and BoNT are used in complement, with PN frequently reserved for proximal nerves and BoNT used for distal musculature.

In contrast, PN produces an almost-immediate effect that manifests within minutes of injection, which may last as long as 6 months depending on the dosage used (1, 13). In addition, PN is significantly less expensive. PN may also be re-injected before 3 months, unlike BoNT. However, the safety and efficacy of PN is lesscommonly documented in the literature than BoNT chemodenervation. PN also requires a higher level of expertise to administer, and has a worse side-effect profile, which includes hypotension, prolonged pain, dysaesthesias, site inflammation, and joint fibrosis (1, 13, 14). These disadvantages for phenol usage are associated with safety concerns relative to neurotoxins, thus making BoNT a vastly more popular option for chemoneurolysis. Phenol is therefore being used increasingly less in the USA and is poorly documented in the spasticity literature. Given its advantages, PN may be superior to chemodenervation with BoNT in certain clinical scenarios. Thus, the primary purpose of the current study is to describe the utilization pattern of PN at a single site.

METHODS

Study design

This was a single-institution retrospective cohort study design. Data was compiled from 185 patients with spasticity, who collectively underwent 293 total PN procedures over a 3-year period at a free-standing rehabilitation hospital affiliated with a medical school. The study was approved by the University of Texas Health Sciences Center at Houston Committee for the Protection of Human Subjects.

Clinical parameters

All patients who underwent PN for spasticity management at least once from January 2013 through December 2015, and had documentation of all demographic variables, diagnoses, other concurrent spasticity treatments, clinical rationale, guidance modalities, nerves targeted, dosages used, reported subjective benefit, and adverse effects were included. Data for aforementioned variables were gathered from procedure notes and physician progress notes in the inpatient setting and clinic notes in the outpatient setting. Demographic variables collected include age, sex, body mass index (BMI), treatment setting, and source of insurance funding. Concomitant treatments included other anti-spasticity treatments that were concurrently being used at the time of PN; those treatments that were discontinued >1 week before the procedure were not considered. Clinical rationale, guidance modalities, nerves targeted and dosages were obtained from procedure notes. PN treatment outcomes were based on medical record entries reported by physicians, patients or caregivers. Adverse effects listed include any untoward reactions, but special attention was paid to prolonged pain, injection site inflammation, dysaesthesia, and/or hypotension, which are known side-effects of PN. Monitoring for these adverse effects occurred on a daily basis for inpatient procedures. Those patients receiving PN in the outpatient setting would have reported any adverse effects as they were actively invited to contact the physician and/or the clinic with any post-procedural concerns.

RESULTS

Demographics

Demographic data for the 185 patients who met inclusion criteria for our study are shown in Table I. A majority of the patients were middle-aged, male, with normal range BMI, privately funded, and treated in the inpatient setting.

Diagnoses, indications, and concomitant treatments

As depicted in Table II, a majority of patients had a neurological diagnosis of stroke. Collectively, stroke, TBI and SCI accounted for 94.05% of all patients; a small portion of our selected patients were diagnosed with anoxic brain injury, multiple sclerosis, or a brain neoplasm. Three subjects had more than one diagnosis. Approximately 90% of subjects had impairment listed as spastic hemiplegia, paresis, or tetraplegia; the remainder had paraplegia, limb dystonia, abnormal involuntary movement spastic monoplegia or monoparesis. A few (n=7) had more than one impairment reported.

Table I. Demographics and insurance status for patients (n = 185) who received phenol neurolysis. Continuous variables are reported as median (range) and categorical variables are reported as number (percentage)

47 (13-89)
133 (71.89)
23.9 (10.1-58.0)
114 (61.60)
99 (53.51)
47 (25.41)
35 (18.92)

BMI: body mass index; IQR: interquartile range

484 J. Karri et al.

Table II. Clinical profile of the study cohort. Displayed is the number (%) of diagnoses, neurological and musculoskeletal, for all reviewed patients (n = 185). Also shown is the number (%) of concomitant treatments, number and type, used at the time of phenol neurolysis for all reviewed procedures (n = 293)

Diagnosis	n (%)
Neurologic diagnosis	
Stroke	76 (41.08)
Traumatic brain injury	53 (28.65)
Spinal cord injury	45 (24.32)
Anoxic brain injury	10 (5.41)
Brain neoplasm	2 (1.08)
Multiple sclerosis	2 (1.08)
Musculoskeletal diagnosis	
Spastic hemiplegia/paresis	134 (45.73)
Tetraplegia	129 (44.03)
Paraplegia	27 (9.22)
Limb dystonia	4 (1.37)
Abnormal involuntary movement	7 (2.39)
Spastic monoplegia/paresis	1 (0.34)
Number of concomitant treatments	
0	69 (23.55)
1	156 (53.24)
2	62 (21.16)
3	6 (2.05)
Type of concomitant treatment	
Chemodenervation with BoNT	140 (47.78)
Oral Baclofen	105 (35.84)
Oral Tizanidine	26 (8.87)
Intrathecal Baclofen	23 (7.85)
Oral Dantrolene	4 (1.37)

BoNT; botulinum neurotoxin.

Almost all procedures had numerous indications for PN, the most common being "decrease spasms", "increase joint range of motion", "prevent contractures", and "improve positioning," collectively accounting for more than 90% of all procedures (Table III). The least common indications reported included "improve gait" (n=110, 37.54%) and "decrease clonus" (n=23, 7.85%).

In this review, it was found that physical therapy, occupational therapy, or at-home exercises were prescribed and/or encouraged in all patients. While a minority of patients at the time of neurolysis (n=68, 23.20%) did not receive additional spasmolytic treatments, most

Table III.	Clinical	rationales	for	phenol	neurolysis	procedures
(<i>n</i> = 293)						

Clinical rationale	n (%)
Decrease spasms	288 (98.29)
Increase joint range of motion	278 (94.88)
Prevent contractures, other complications	270 (92.15)
Improve positioning	268 (91.47)
Improve transfers and mobility	261 (89.08)
Improve hygiene and nursing care	258 (88.05)
Facilitate ADL performance	255 (87.03)
Increase active limb movement	252 (86.01)
Decrease spasm related pain	251 (85.67)
Prevent skin breakdown	248 (84.64)
Improve orthotic fit	231 (78.74)
Decrease abnormal movement, dystonia	213 (72.70)
Improve gait	110 (37.54)
Decrease clonus	23 (7.85)

ADL: activities of daily living.

W Journal of Rehabilitation Medicine

	n (%)	Mean (range) ml	SD
Guidance			
EStim Only	10 (3.41)		
US Only	80 (27.30)		
EStim and US	203 (69.28)		
Total	293 (100.00)		
Phenol Dose per Procedure			
EStim Only		8.75 (3.00-15.00)	3.79
US Only		10.89 (1.00-30.00)	6.35
EStim and US		11.08 (2.50-23.50)	4.51
Total		10.95 (1.00-30.00)	5.08
Phenol Dose per Nerve			
EStim Only		4.16 (1.75-9.00)	2.24
US Only		3.21 (0.75-10.00)	1.89
EStim and US		3.57 (1.00-18.00)	2.32
Total		3.48 (0.75-18.00)	2.23

EStim: electrical stimulation; US: ultrasound imaging; SD: standard deviation.

patients (n=225, 76.80%) received ≥ 1 pharmaceutical modality (Table II). The most commonly used concomitant treatments included chemodenervation with BoNT (n=140, 47.78%) and oral baclofen (n=105, 35.84%).

Procedure variables

The majority of PN treatments during the study period (n=289, 98.6%) were performed by 3 physicians, who had received training on PN at the same institution and, collectively, had 31 years of experience with PN. The injector experience ranged from 3 to 20 years. Most procedures (n=203, 69.28%) used both ultrasound imaging (US) and electrical stimulation (EStim) guidance to identify target nerves (Table IV). The only phenol preparation used was aqueous, 6%. The dosage used per nerve ranged from 0.75 to 18.00 ml, while total dosage per procedure ranged from 1.0 to

Table V. Nerves injected and phenol dosages for all considered procedures (n = 293). Phenol dosage in millilitres is reported as mean (range). A total of 146 (49.83%) of all procedures involved the upper extremity and 264 (90.10%) of all procedures involved the lower extremity

Nerve	n (%)	Phenol dose per nerve
Upper extremity		
Musculocutaneous	61 (20.82)	3.34 (1.00-7.50)
Pectoral Lateral	55 (18.77)	2.41 (1.00-6.00)
Pectoral Medial	33 (11.26)	2.30 (1.00-5.00)
Radial	12 (4.10)	1.96 (1.00-3.00)
Thoracodorsal	8 (2.73)	2.38 (2.00-3.00)
Median	6 (2.05)	2.17 (1.50-3.00)
Lower extremity		
Sciatic	161 (54.95)	5.48 (1.00-15.00)
Tibial	117 (39.93)	6.24 (1.00-18.00)
Obturator	105 (35.84)	5.62 (1.00-13.00)
Femoral	99 (33.79)	5.67 (1.00-13.50)
Superior Gluteal	4 (1.37)	3.75 (1.00-13.00)
Fibular	2 (0.68)	1.50 (1.00-2.00)
Piriformis	2 (0.68)	1.25 (1.00-1.50)
Total	293 (100.00)	4.06 (0.75-18.00)

JRM

30.00 ml. Overall, there were some slight differences in total phenol dosage used with regards to injection guidance. It was notable that procedures with US and EStim guidance used the highest total phenol dosage on average. Multiple nerves were injected for synergistic spastic patterns, e.g. PN of the musculocutaneous and pectoral nerves to address spasticity in the shoulder adductors and elbow flexors.

Approximately half of all procedures (49.83%) involved upper extremity nerves, while almost all procedures (90.10%) involved lower extremity nerves (Table V). The most commonly injected nerves in the upper extremity included the musculocutaneous, pectoral lateral, and pectoral medial nerves. The most commonly injected nerves in the lower extremity included the sciatic, tibial, obturator and femoral nerves. Mean phenol dose per nerve across all upper extremity nerves was limited, ranging from 1.96 to 3.34 ml. In contrast, the similar range amongst the lower extremity nerves was far greater, with means from 1.25 to 6.24 ml. Larger lower extremity nerves, such as the obturator, sciatic, femoral and tibial nerves, were injected more commonly and received larger doses and ranges of doses than did smaller nerves. Similar trends were not appreciated in upper extremity nerves, including the musculocutaneous, radial and median nerves.

Subjective benefit and adverse effects

Detailed pre- and post-injection evaluation was not documented, either by physicians or therapists. However, adverse effects and, sometimes, subjective benefits, were commonly reported. Subjective benefit, or lack of benefit, was reported in only approximately half of all procedures (n=161, 54.95%). In these procedures,

Table VI. Subjective benefit and adverse effects for all reviewed procedures with appropriate benefit documentation (n = 161). Also shown are the number of procedures associated with an adverse effect and type of adverse effect reported for all reviewed procedures (n = 293)

Subjective benefit	n (%)
Subjective spasticity improvement?	
Yes	136 (84.47)
No	25 (15.53)
Diagnoses associated with procedures of benefit	
Stroke	59 (36.65)
Traumatic brain injury	38 (23.60)
Spinal cord injury	27 (16.77)
Other	15 (9.32)
Number of adverse effects	
0	279 (95.22)
1	6 (2.05)
2	7 (2.39)
3	1 (0.34)
Туре	
Prolonged pain	11 (3.75)
Inflammation	8 (2.73)
Dysesthesia	2 (0.68)
Hypotension	2 (0.68)

subjective benefit attributed to PN was endorsed in a majority of cases (n=136, 84.47%) (Table VI). Most procedures (n=279, 95.22%) were not associated with any adverse effects (Table VI). However, patients in 14 procedures reported an adverse effect. The most commonly reported adverse events were pain (4.10%) and inflammation (2.73%), while dysaesthesia (0.68%) and hypotension (0.68%) were rarely reported. As shown in Table VII, most of the procedures with adverse effects involved PN to the tibial and femoral nerves. Overall, dysaesthesia followed PN to the tibial, femoral and obturator nerves only.

DISCUSSION

The aim of this study was to provide a description of PN clinical practice patterns in a single setting. Although limited to one institution, this study provides useful and comprehensive information, owing to a large cohort of patients (n=185) and total procedures (n=293). Moreover, this study is among the first to provide an extensive profile for numerous procedure variables in PN that are rarely mentioned in the spasticity literature.

Patient demographics, distribution and indications issues

As shown in Table II, the prevalence of stroke, TBI and SCI in our patient population was consistent with the diagnostic mix at our institution, which specializes in adult neurorehabilitation. The pattern we report may be unique to this diagnostic mix and our setting. Overall, the majority of our patients, being middle-aged and

Table VII. Nerves injected in phenol neurolysis procedures (n = 14) associated with an adverse event. The guidance modality used for these procedures was US Only (n = 7) and US with Estim (n = 7). Adverse events are presented as number of procedures (percentage of total reported adverse event procedures) and the type (number of procedures associated with specific adverse event)

	Procedures	
Nerve	n (%)	Adverse event type, (n)
Upper extremity		
Musculocutaneous	1 (6.67)	Pain (1)
Pectoral Lateral	1 (6.67)	Inflammation (1)
Pectoral Medial	0 (0.00)	-
Radial	0 (0.00)	-
Thoracodorsal	0 (0.00)	-
Median	0 (0.00)	-
Lower extremity		
Sciatic	3 (20.00)	Pain (3), inflammation (2)
Tibial	11 (73.33)	Pain (8), inflammation (7), dysesthesia (1), hypotension (2)
Obturator	2 (13.33)	Pain (2), dysesthesia (1)
Femoral	7 (46.67)	Pain (5), inflammation (3), dysesthesia (2), hypotension (2)
Superior Gluteal	0 (0.00)	-
Fibular	1 (6.67)	Inflammation (1)
Piriformis	0 (0.00)	-

male, are consistent with the epidemiology of TBI, stroke and SCI (15–16).

PN is currently used to reduce spasticity for various outcome goals, from improving gait to decreasing risk of developing contractures. The majority of clinical indications in this study were to "decrease spasms", "increase joint range of motion", "prevent contractures" and "improve positioning" (these indications were cited in >90 of precedures, Table III). Since PN produces almost-immediate effects for these indications and goals, PN may allow more time for inpatient therapies, compared with the BoNT chemodenervation effect, which peaks approximately 3 weeks post-injection. We believe that early usage of PN, in combination with the aforementioned inpatient therapies, will optimize the possible spasticity benefit and help propagate chronic neuromuscular recovery overall. It is therefore suggested that the use of PN could be emphasized more in inpatient settings. However, judicious and prudent use of PN is advised in the setting of potential and spontaneous neurological recovery in the acute/subacute phase.

The results of the current study show that the majority of PN procedures were conducted in the inpatient setting (n=114, 61.60%) compared with the outpatient setting. This disparity towards inpatient procedures may also be justified by insurance reimbursements (17), which are more easily processed for PN in the inpatient setting. The cost-effectiveness of chemoneurolysis for spasticity management has yet to be fully established (1, 3). Significant data exploring the benefits of PN are largely lacking. Future studies comparing PN with other therapies, such as chemodenervation with BoNT, with regards to injection timing, procedural parameters, and standard outcomes are necessary.

Procedure parameters: injection guidance, safety, phenol dosage, targeted nerves and side-effects

Ensuring the safety of PN procedures is often a clinical challenge, and sometimes is a limiting factor in pursuing this treatment (1, 3), compared with chemodenervation with BoNT. During chemodenervation with BoNT, needle placement is localized intramuscularly. In contrast, PN requires accurate needle placement to the target nerves to maximize efficacy. Due to anatomical proximity, PN is often associated with increased risks of damaging surrounding vasculature and untargeted nerves (18–21).

Different modalities are used to guide PN nerve blocks, including EStim and US. EStim of the target nerves, which causes contraction of the innervated muscles, can provide localization of needle placement over manual needle placement. However, its use independently for nerve localization is poorly documented, particularly for the purposes of PN (22–24). At our institution, 1 of 2 EStim devices, Clavis (Natus Medical Inc., San Carlos, CA, USA) or Myoguide (Inotronix Technologies, Bolton, ON, Canada), were used to guide the placement of a 23–27-gauge needle. EStim intensity, which is scenario-dependent and not recorded, ranged from 1 to 4 mA and the stimulus duration was 0.1 ms.

In contrast, US provides direct visualization of target nerves, thus increasing the safety of needle placement with minimal subjectivity. Using US guidance in chemoneurolysis has been evidenced to be more beneficial than EStim alone with regards to needle placement complications, notably in minimizing vascular involvement in anaesthetic studies (25, 26). Interestingly, the concurrent usage of US and EStim is suggested to be similar in efficacy to that of US alone (27-29). Nevertheless, use of US and EStim together does not afford any increased risk and may provide some physicians with a secondary level of certainty. In this study, almost all procedures (n=283, 96.59%) used US alone or in combination with EStim, while only a small minority used EStim alone (n=10, 3.41%). Furthermore, with regards to guidance and safety, there were no appreciable associations between guidance modality used and adverse reactions, which were low.

It is known that phenol has a dose-dependent effect in spasticity benefit (3). Moreover, there is a general guideline regarding the maximum limit of phenol dose per procedure (30). However, data regarding phenol dosage per procedure and per nerve and frequently injected nerves are lacking in the literature. In this study, detailed procedure parameters are reported (Table V). These parameters, such as mean phenol dose per procedure and per nerve, may be valuable for practitioners. Furthermore, the range of phenol dose is also important to provide a reference when patients with different levels of severity of their condition are treated. For example, one patient received up to 18 ml phenol for one nerve to reach clinical goals. It is recommended that that the total volume of phenol administered to an adult in a single treatment session should not exceed 1 g (approximately 17 ml of 6% phenol) (30). In the present study, it is reported that an adult patient received 30 ml in a single session without careful monitoring. It should also be reported that this patient tolerated the procedure well and with no adverse effects.

Adverse reactions specific to PN were lower in this study compared with others utilizing PN (9). The authors reported that 30% of the patients in the phenol group (n=10 subjects) developed dysaesthesia after the tibial nerve phenol block. In a recent Cochrane review study by Lindsay & Pandyan. (31), adverse

events of PN from this study and another by Kocabas et al. (32) involving phenol injection to the motor branches of tibial nerve were reviewed. It was reported that there was no significant difference between groups. It is known that the risk of phenol-specific side-effects, such as dysaesthesia, is relatively higher if distal mixed sensorimotor nerves are injected (30), e.g. neurolysis to upper extremity nerves, such as the median and radial nerves, and lower extremity nerves, such as the tibial nerve (3). This high rate of adverse effects (30%) is probably attributable to the fact that these nerves were (tibial nerve vs motor branch block) injected. Alternatively, it could be due to the sample size. Only 3 out of 10 subjects had this adverse event, while 293 procedures were reviewed. The majority of injected nerves were proximal nerves.

Study limitations

This study has several limitations, many of which are related to its retrospective nature. Because the reviewed cohort comprised patients from a free-standing rehabilitation hospital that focused on neurorehabilitation for adult patients, there was a unique diagnostic mix, including stroke, TBI and SCI. Thus, PN for spasticity management in other populations, such as cerebral palsy, is rarely performed at our facility.

A further limitation is the poor documentation of post-procedure spasticity assessment. Only half of procedures documented subjective outcomes, which suggested beneficial effects. This probably results from a lack of standardized clinical measurement of spasticity pre- and post-injection. This poor documentation may be associated with under-reporting of adverse events. For example, non-significant injection pain and post-injection pain may not be reported by patients or documented by the physicians or therapists. It may contribute to a under-reported rate of adverse events, though these were probably not clinically significant. Future research regarding PN in spasticity management should include quantitative assessment pre- and post-procedure to better characterize the benefit and adverse events of PN.

Conclusion

This study summarizes clinical data from a large cohort of patients (n=185) and their collective PN procedures (n=293) over a 3-year period from an academic institution. The results revealed that PN may be a useful treatment option for spasticity management in a variety of patients with a plethora of diagnoses. In addition, this extensive utilization of PN was shown to be associated with no serious procedure-related complications and only a small incidence of observed adverse effects.

ACKNOWLEDGEMENT

The study was in part supported by an NIH/NICHD/NCMRR grant (R21HD087128).

The authors declare no conflicts of interests.

REFERENCES

- Francisco GE, Li S. Spasticity. In: Cifu DX, editor. Physical medicine and rehabilitation, 5th edn. Philadephia: Elsevier; 2016, p. 487–489.
- Pierson SH. Outcome measures in spasticity management. Muscle Nerve 1997; 6: S37–S60.
- Gracies JM, Elovic E, McGuire J, Simpson D. Traditional pharmacological treatments for spasticity: part I-Local treatments. Muscle Nerve 1997; 6: S61–S91.
- Furr-Stimming, E, Boyle AM, Schiess MC. Spasticity and intrathecal baclofen. Semin Neurol 2014; 34: 591–596.
- Awaad Y, Rizk T, Siddiqui I, Roosen N, Mcintosh K, Waines GM. Complications of intrathecal baclofen pump: prevention and cure. ISRN Neurol 2012: 575168.
- Francisco GE, McGuire JR. Poststroke spasticity management. Stroke 2012; 43: 3132–3136.
- Ploypetch T, Kwon JY, Armstrong HF, Kim H. A retrospective review of unintended effects after single-event multi-level chemoneurolysis with botulinum toxin-A and phenol in children with cerebral palsy. PMR 2015; 31: 1073–1080.
- Rosales RL, Chua-Yap AS. Evidence-based systematic review on the efficacy and safety of botulinum toxin-A therapy in post-stroke spasticity. J Neural Transm 2008; 115: 617–623.
- Kirazli Y, On AY, Kismali B, Aksit R. Comparison of phenol block and botulinum toxin type A in the treatment of spastic foot after stroke: a randomized, double-blind trial. Am J Phys Med Rehab 1998; 77: 510–515.
- Bakheit AO, Badwan DH, McLellan DL. The effectiveness of chemical neurolysis in the treatment of lower limb muscle spasticity. Clin Rehabil 1996; 10: 40–43.
- Viel E, Pelissier J, Pellas F, Boulay C, Eledjam JJ. Alcohol neurolytic blocks for pain and muscle spasticity. Neuro-Chirurgie 2003; 49: 256–262.
- Bensmail D, Hanschmann A, Wissel J. Satisfaction with botulinum toxin treatment in post-stroke spasticity: Results from two cross-sectional surveys (patients and physicians). J Med Econ 2014; 17: 618–625.
- Halpern D, Meelhuysen FE. Duration of relaxation after intramuscular neurolysis with phenol. JAMA 1967; 200: 1152–1154.
- Superville-Sovak B, Rasminsky M, Finlayson MH. Complications of phenol neurolysis. Arch Neurol 1975; 32: 226–228.
- Sekhon LH, Fehlings MG. Epidemiology, demographics, and pathophysiology of acute spinal cord injury. Spine 2001; 26: S2–S12.
- Kraus, JF, McArthur DL. Epidemiologic aspects of brain injury. Neurol Clin 1996; 14: 435–450.
- Fu J, Gutiérrez C, Bruera E, Guo Y, Palla S. Use of injectable spasticity management agents in a cancer center. Support Care Cancer 2013; 21: 1227–1232.
- Kolaski K, Ajizian SJ, Passmore L, Pasutharnchat N, Koman LA, Smith BP. Safety profile of multilevel chemical denervation procedures using phenol or botulinum toxin or both in a pediatric population. Am J Phys Med Rehab 2008; 87: 556–566.
- Watanabe T. Assessment of spasticity. In: Brashear A, Elovic EP, editors. Spasticity: diagnosis and management. New York: Demos Medical Elsevier; 2011, p. 71–80.
- BOTOX Poststroke Spasticity Study Group. Repeated dosing of botulinum toxin type A for upper limb spasticity following stroke. Neurology 2004; 63: 1971–1973.
- Elovic EP, Esquenazi A, Alter KE, Lin JL, Alfaro A, Kaelin DL. Chemodenervation and nerve blocks in the diagnosis

Journal of Rehabilitation Medicine

488 J. Karri et al.

and management of spasticity and muscle overactivity. PMR 2009; 1: 842-851.

- Childers MK. The importance of electromyographic guidance and electrical stimulation for injection of botulinum toxin. Phys Med Rehabil Clin N Am 2003; 14: 781–792.
- Lim EC, Ong BK, Seet RC. Botulinum toxin-A injections for spastic toe clawing. Parkinsonism Relat Disord 2006; 1: 43–47.
- O'Brien CF. Injection techniques for botulinum toxin using electromyography and electrical stimulation. Muscle Nerve 1997; S6: 176–180.
- Chin KJ, Chan V. Ultrasound-guided peripheral nerve blockade. Curr Opin Anaesthesiol 2008; 21:624-31.
- Brull R, Perlas A, Cheng PH, Chan VW. Minimizing the risk of intravascular injection during ultrasound-guided peripheral nerve blockade. Anaesthesiology 2008; 109: 1142–1148.
- Casati A, Danelli G, Baciarello M, Corradi M, Leone S, Di Cianni S, et al. A prospective, randomized comparison between ultrasound and nerve stimulation guidance for multiple injection axillary brachial plexus block. Anaesthesiology 2007; 106: 992–996.

- Dhir S, Ganapathy S. Comparative evaluation of ultrasound-guided continuous infraclavicular brachial plexus block with stimulating catheter and traditional technique: a prospective-randomized trial. Acta Anaesthesiol Scand 2008; 52: 1158–1166.
- Dufour E, Quennesson P, Van Robais AL, Ledon F, Laloë PA, Liu N, et al. Combined ultrasound and neurostimulation guidance for popliteal sciatic nerve block: a prospective, randomized comparison with neurostimulation alone. Anesth Analg 2008; 106: 1553–1558.
- Stevenson VL, Jarrett L, editors. Spasticity management: a practical multidisciplinary guide. 2nd Ed. Boca Raton: CRC Press; 2016.
- 31. Lindsay C, Pandyan AD. Pharmacological interventions other than botulinum toxin for spasticity after stroke. Cochrane Database Syst Rev 2016; 10: CD010362.
- 32. Kocabas H, Salli A, Demir AH, Ozerbil OM. Comparison of phenol and alcohol neurolysis of tibial nerve motor branches to the gastrocnemius muscle for treatment of spastic foot after stroke: a randomized controlled pilot study. Eur J Phys Rehabil Med 2010; 46: 5–10.