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Original Research Article

A Cross Over Study to Establish Effectiveness of Phenol Blocks to Peripheral Nerve in Reducing Spasticity in Traumatic and Non-Traumatic Brain Injured and Spinal Cord Injured Patients

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Abstract

Aim: To Study phenol block to peripheral nerves, in patients with traumatic and non-traumatic brain injury and spinal cord injury, resulting in reduction in spasticity as measured by the modified Ashworth scale.

Material & Method: This study was conducted in the Department of Physical Medicine & Rehabilitation Tertiary Care Hospital, Telangana State, during the period March 2016 to January 2018.

Results: There were more male patients (18) compared to females (2), Spinal cord injury is the common etiology of neurological spasticity.

Conclusion: There is a significant improvement in the range of movement of joints after blockade of spasticity using phenol and there was a 5% incidence of adverse effect (pain) following administration of phenol during the period of this study.

Keywords: Phenol Blocks, Traumatic and Non-Traumatic Brain Injured, Spinal cord injured.

Introduction

Spasticity is one of the most disabling aspects of traumatic and non-traumatic brain injury and spinal cord injury. Spasticity is a component of the upper motor neuron syndrome and is a disorder characterized by a velocity-dependent increase in tonic stretch reflexes and exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex. Spasticity should be treated if it interferes with functions like gait or activities of daily living, comfort, and caregiving. Spasticity management is a challenge in practice because spasticity has many advantages and disadvantages [1]. The main advantages of spasticity are that it helps in standing and walking, maintains muscle mass and bone mineralisation, reduces dependant edema in paralysed

limbs, and lowers the risk of deep vein thrombosis. The disadvantages of spasticity are that it interferes in activities of daily living like standing and walking, interferes with positioning and personal hygiene, causes pain and disturbs sleep'. Long-term spasticity leads to the development of contractures [2].

The management of spasticity is a long-term process and needs to be undertaken 24 hours a day by patients, their caregivers and the multidisciplinary team. The common measures to control spasticity include passive stretching, serial casting, orthotics, medications, and interventions such as nerve blocks, intrathecal blocks," intrathecal medications, surgical neurectomies and tendon releases [3]. The initial assessment of spasticity includes screening for nociceptive and exteroceptive stimuli that can exacerbate the spasticity. The avoidance of noxious stimuli is an important initial management step. This includes such measures as prompt treatment of urinary tract complications (infections, stones), prevention of pressure sures and contractures, the release of tightly wrapped leg bags and tight garments, faecal impaction and heterotopic ossification [4,5].

Aims and Objectives

1. To Study phenol block to peripheral nerves, in patients with traumatic and non-traumatic brain injury and spinal cord injury, resulting in a reduction in spasticity as measured by the modified Ashworth scale.
2. To study the change in the range of motion in relevant joints after phenol block to peripheral nerves.
3. To study electrophysiological change after phenol block to peripheral nerves.
4. To Study change in gait analysis after phenol block to peripheral nerves.
5. To study phenol block side effects.
6. To study chemical neurolysis with phenol blocks is more cost-effective than standard orally administered systemic antispastic medications.

Materials and methods

This study was conducted in the Department of Physical Medicine & Rehabilitation of Tertiary Care Hospital, Telangana State. Patients admitted to the Rehabilitation unit following Brain injury and Spinal cord injury (traumatic and non-traumatic) were evaluated for disabling spasticity.

Inclusion criteria

1) Patients who were observed to have disabling spasticity 6 weeks after the onset of the neurological incident
2) Spasticity in the adductor group of muscles of the hip causes difficulty in performing activities of daily living (Toileting, Dressing) or spasticity in the Gastrosoleus group of muscles resulting in difficulty in walking or sitting in a wheelchair.

Exclusion criteria

1) Comatose patients
2) Patients who could not give informed consent.
3) Patients on Anti-coagulants.
4) Patients with other conditions which will aggravate spasticity (renal calculi, vesical calculi, in growing toenails, pressure ulcers, pneumonia).
5) Heterotopic ossification.

Measurements

For those patients who satisfied the inclusion criteria the following are.

1) Spasticity was measured by a modified Ashworth scale,
2) Range of motion was measured with a Goniometer before and one hour after the intervention.
3) Electromyographic changes were recorded by the investigator (H-Latency, H-Amplitude, M-Amplitude, H: M ratio) at the beginning and end of the study.

4) Gait analysis was done at the beginning of the study and the end of the study.
5) The cost of antispastic drugs that the patient is taking throughout the study was recorded.
6) Side effects of the injection were also recorded.
7) Functional independence measures (for activities of daily living) related to self-care and locomotion were scored at the beginning and the end of the study.

The protocol of the study was as follows:

Day 1

- Measurement of range of motion in relevant joints.
- Measurements of spasticity by Modified Ashworth scale.
- Electrophysiological examination (H-reflex latency, amplitude
- M-wave amplitude, H: M ratio)
- Gait analysis (Kinetics, Kinematics, PCI)

Day 7

- Measurement of range of motion in relevant joints.
- Measurements of spasticity by Modified Ashworth scale.

Intervention with solution A

- After 1 hour measurement of range of motion in relevant joints Measurements of spasticity by Modified Ashworth scale.

Day 14

- Measurement of range of motion in relevant joints
- Measurements of spasticity by Modified Ashworth scale.
- Intervention with solution B:

After 1 hour measurement of range of motion in relevant joints and measurement of spasticity by modified Ashworth scale.

Day 21

- Measurement of range of motion in relevant joints
- Measurements of spasticity by Modified Ashworth scale.
- Electrophysiological examination (H-reflex latency, amplitude M-wave amplitude, H: M ratio).

Electrophysiological examination

H-reflex: H-reflex was obtained by putting the patient in the prone position and the posterior tibial nerve was stimulated antidromically in the popliteal fossa and the recording electrode was placed on soleus muscle.

M-wave: M-wave was obtained by putting the patient in the prone position and the posterior tibial nerve was stimulated orthrodromically in the popliteal fossa and the recording electrode was placed on the soleus muscle.

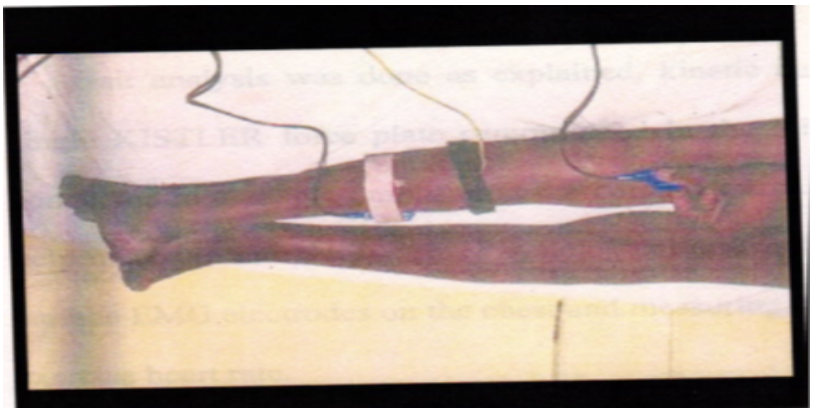

Figure 1: Method of eliciting H-reflex and M –wave in popliteal fossa

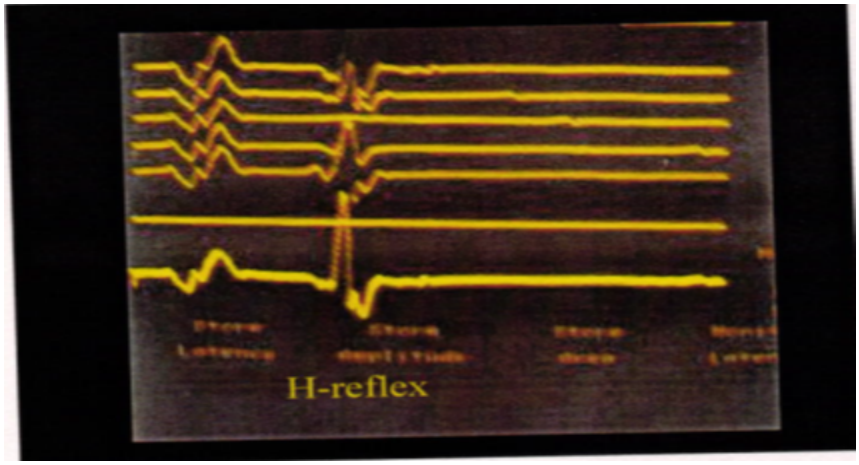

Figure 2: H-reflex

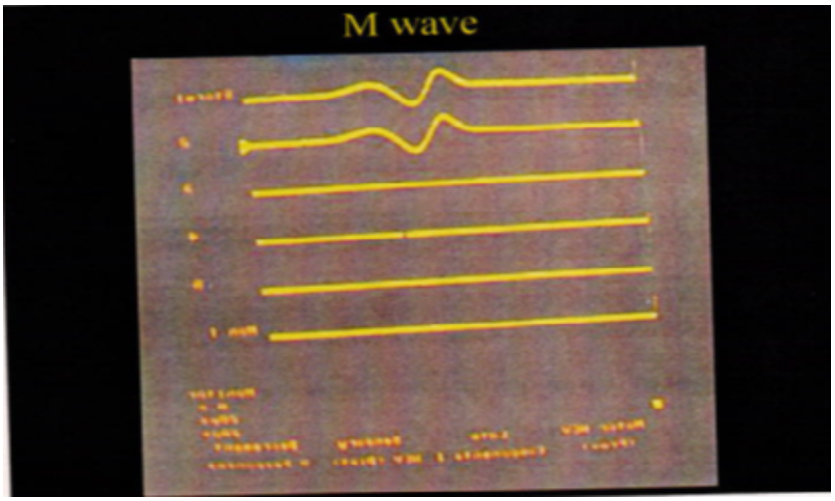

Figure 3: M- Wave

Gait Analysis

Gait analysis was done as explained, kinetic data collection was done from a single KISTLER force plate camouflaged in the middle of the walkway.

This is connected to a charge amplifier in the computer area. Kinematics was done with the help of an active marker system. The physiological cost index was obtained by placing the surface EMG electrodes on the chest and measuring the resting heart rate and the post-exercise heart rate.

The technique of the Procedure

The nerves to be blocked were located using a needle electrode connected to an electrical nerve stimulator.

Obturator nerve block

Position: The patient was positioned comfortably in the supine posture with the hip joint in maximum possible abduction.

Technique

To locate the anterior branch of the Obturator nerve, the Adductor longus tendon was identified and the needle was directed perpendicular to the coronal plane posterior to the Adductor longus muscle approximately 3 cm below the pubic tubercle.

To locate the posterior branch of the obturator nerve, the needle was directed posteriorly from the Adductor longus tendon towards the ischial tuberosity.

Obturator Nerve block

Position: The patient was positioned comfortably in the supine posture with the hip joint in maximum possible abduction.

Technique

To locate the anterior branch of the Obturator nerve, the Adductor longus tendon was identified and the needle was directed perpendicular to the coronal plane posterior to be Adductor longus muscle approximately 3 cm below the pubic tubercle. To locate the posterior branch of the obturator nerve, the needle was directed posterioly from the Addactor longus tendon towar4ds the ischial tuberosity.

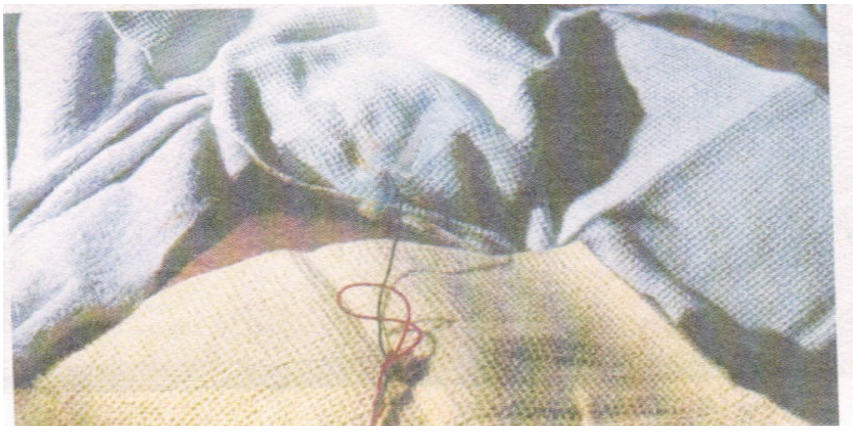

Figure 4: Obturator nerve block

Posterior tibial nerve

The patient was positioned comfortably in the prone position, with 15-20% of flexion at the knee joint

Technique

The surface landmark for identification of this nerve is along a perpendicular line drawn from the midpoint of the knee crease in the popliteal fossa, 3 cm proximally and 1 cm laterally. Once the nerve that needs injecting has been decided upon, the patient was appropriately positioned as described above. The skin was cleaned, and a needle electrode was introduced at this site to locate the nerve.

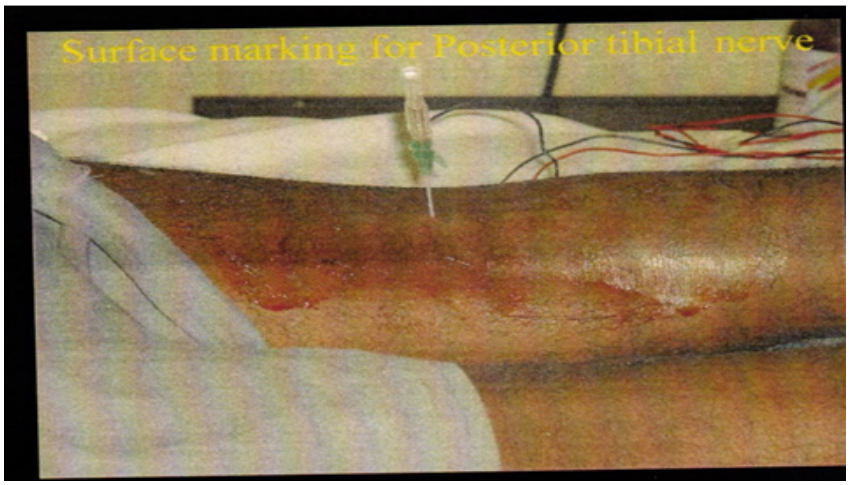

Figure 5: Tibial nerve block

The solution was administered through a hollow needle with an insulated shaft which was connected to the stimulator. For this purpose, an 18-gauge intravenous cannula was used. The removable plastic sheath of the intravenous cannula served as the insulator for the metallic needle, and the exposed tip of the needle provided stimulation of the peripheral nerve. The needle was connected to the stimulator using a connecting wire wound around the base of the needle. A direct current lasting 0.05 -0.1 m sec and current strength initially of 3-5mA was used at a rate of 0.5-3hz. Once the desired group of muscles were contracting in time to the needle stimulus, the nerve was further repositioned with the aim of obtaining a maximal response with a minimum stimulus, that is about 0.5mA. At this juncture, the 3 ml of solution was loaded onto a syringe which was connected to the intravenous cannula and aspiration was done to ensure no vessels were punctured and the contents in the vial were injected. Following administration of the solution at the predetermined peripheral nerve all the measurements were done as mentioned above namely range of motion in relevant joints, modified Ashworth scale, H: M ratio and Gait analysis in patients who were walking.

On completion of the study, the blinded solutions were revealed as solution A to be 0.5% Bupivacine and solution B to be 6% phenol.

Results

Table 1: Age distribution of the patients

Range (years)	Number
20 – 30	4
31 – 40	7
41 – 50	9

The age of the patients in this study varied from 20-50 years which are grouped into 3 categories: a) Age Group b) Sex Ratio c) Etiology

Table 2: Etiology of neurological lesion causing spasticity

Spinal cord injury (17)		Stroke (3)	
Traumatic	6	Cerebrovascular accident	3
Tuberculosis	4		
Transverse myelitis	2		
Epidural abscess	2		
Neoplasm	3		

Measurement of Spasticity

Measurement of spasticity was done by the investigator on the 1st day, 7th day (pre and post-intervention), 14th day (pre and post-intervention) and on 21st day.

Table 3: Measurement of spasticity by modified Asworth Scale of all Patients

Day 1		
Mean		
3.2000	.410	
Day 7		
Before intervention with 0.5% Bupivacaine	After intervention with 0.5% Bupivacaine	

Mean	SD	Mean	SD
3.2	.410	.2500	.444
Day 14			
Before Intervention with 6% Phenol		After intervention with 6% Phenol	
Mean	SD	Mean	SD
3.2000	.410	.8000	.523
Day 21		* = Statistically Significant	
Mean	SD		
.8000	.523		
P value .000*			

Table 4: Electrophysiological data before and after the intervention

Variables	Day 1		Day 21		P Value
	Mean	SD	Mean	SD	
H reflex (Latency)	28.6167	8.101	229.8778	3.906	.482
H reflex amplitude	416.3158	426.678	239.6842	294.485	.041*
M wave amplitude	6.9900	5.532	9.6350	18.733	.565
H: M Ratio	.1563	.168	.1102	.099	.286
* = Statistically Significant					

Gait Analysis

Only 3 patients were able to walk independently during the period of study. All these patients were hemiplegics and had an injection to the posterior tibial nerve. The kinetics, kinematic data and physiological cost index of these 3 patients before and after intervention are given below

Table 6: Range of motion in Hip Knee and Ankle joints in the patients before and after intervention

Patients	1		2		3	
Day	1st	21st	1st	21st	1st	21st
Hip ROM	10^0	9^0	9^0	13^0	11^0	10^0
Knee ROM	23^0	16^0	14^0	16^0	21^0	17^0
Ankle ROM	10^0	26^0	11^0	5^0	19^0	27^0

Patient 1 and 3 shows an increase in the range of motion in the Ankle joint but this was not seen in patient2 as showed. As the number of patients was small this data was not subjected to statistical analysis.

Table 7: Physiological cost index of 3 ambulant patients pre and post-intervention

Patients	1A	1B	Change	2A	2B	Change	3A	3B	Change
Physiological cost index	2.76	4.09	+1.33	2.32	0.49	-1.83	1.43	1.85	+0.42

1, 2, 3 are patients; A = Before intervention; B = After intervention.

The physiological cost index in patients 1 and 3 increased after intervention whereas the physiological cost index in patient 2 decreased. The number of ambulant patients was small, this data was not subjected to statistical analysis.

Discussion

Spasticity is a velocity-dependent increase in tonic stretch reflex activity. Spasticity is one of the most disabling aspects of traumatic and non-traumatic brain injury patients.

Spasticity can cause pain and muscle shortening which is a significant source of disability. The main goals of treatment are to reduce the deforming force as a result of spasticity, improve better function and prevent secondary complications due to spasticity.

Phenol blocks to peripheral nerves reduce spasticity in traumatic and non-traumatic spinal cord injury and brain injury patients. This study is to evaluate the cost, efficacy and side effect profile of phenol blocks to peripheral nerves.

In this study, 20 patients with disabling spasticity were included. In 17 patients spasticity was due to spinal cord injury and in 3 patients spasticity was due to cerebrovascular accidents. There were 6 tetraplegics, and 11 paraplegics in the spinal cord injured patient group. The benefits and possible side effects of local spasticity blockade were discussed in detail with the patients. Documentation was done as explained earlier in the materials and methods section.

The solutions for injection were prepared by the pharmacy department of our institution and these solutions were blinded to the patient and investigator until the end of the study. Solution A was 0.5% bupivacine and Solution B was 6% phenol, and they were injected on the 7th day and 14th day after including into the study.

All patients continued to receive physical modalities like stretching, standing, and orthotic appliances to reduce the tone during the study period. Spasticity was measured by a modified Ashworth scale, range of motion in relevant joints was measured by a goniometer, and Electrophysiological measures including H-reflex amplitude, M wave and HM ratio were documented. Gait analysis including kinetics, kinematics and physiological cost index was measured in 3 patients. The cost of antispastic medication which the patient was taking throughout the study, was documented.

Keenan et al [6]. reported an average duration of 5 months for musculocutaneous nerve. Chemodenervation of the musculocutaneous nerve with neurolytic agents is an effective treatment of spasticity of the elbow flexors. Although well-controlled trials make definitive statements about the duration of clinical benefit, a general average of 6 months seems to be shared by many authors. The complications following phenol injections are pain, dysasthesia and the most serious complication would be vascular from inadvertent direct infiltration of phenol into arterial or venous supply.

Herman *et al* [7]. notes that in the cerebral (or hemiplegic) model of spasticity, sinuosoisal stretching of calf muscles results in a rapid buildup of reflex activity.

Ethyl alcohol in low concentrations (5 to 10%) acts as a local anaesthetic by decreasing sodium and potassium conductance. At higher concentrations, ethyl alcohol non-selectively denatures protein and injures cells by precipitating and dehydrating protoplasm. The advantages of alcohol are it is easily available, and disadvantages include pain at the site of injection, skin irritation, permanent peripheral nerve palsy and painful muscle necroses.

Phenol (benzyl alcohol) is the major oxidized metabolite of benzene. The cell-damaging properties of phenol were first exploited in antispastic treatment with intrathecal administration. Khalili [8] and collaborators then performed perineural injections and Awad pioneered intramuscular injections.

Wallerian degeneration occurs in approximately 2 weeks following injection and eventually, there is re-growth of most axons. However, after the administration of

2% aqueous phenol, damage to the micro-circulation occurs around the nerves. This may lead to the occlusion of small blood vessels and fibrosis in the injected area and might account for long-term effects.

All patients who were included in the study showed a significant reduction of spasticity on the 7th day and 14th day onwards. There was a significant reduction in spasticity after giving 0.5% Bupivacaine on the 7th day but there was a recurrence of spasticity occurred from the 8th day onwards until the 14th day. After giving phenol injection the reduction in spasticity was observed from 14" days onwards and this continued till the end of the study. In a study by Khalili *et al* [8]. the clinical effect of phenol lasted for approximately 10 to 11 months. Similar observations were shared by many other authors who reported the duration of the effect of phenol to be 6 months. The observations in this study have also corroborated the long-term effects of phenol. Therefore, when the effect of a neurolytic agent on a complex task such as walking needs to be evaluated, 0.5% bupivacaine could be used as a diagnostic agent before giving 6% phenol which is a long-acting chemical neurolytic agent, to reduce spasticity.

As spasticity decreased there was the definite improvement in the range of motion in the neighbouring joints. Restricted range of motion interferes with functional activities. 13 patients showed consistent improvement in abduction range after giving 0.5% bupivacaine and phenol injections to the obturator nerve. The abduction range helped them in proper positioning, maintaining proper perineal hygiene and in self-care activities like toileting and lower half dressing. It also helped in proper ambulation by decreasing the scissoring of gait. The 7 patients who received 0.5% bupivacaine and 6% phenol injection to the posterior tibial nerve showed significant improvement in dorsiflexion range which helped them in sitting, walking and climbing stairs.

All patients who were included in the study were evaluated electrophysiologically (H-reflex wave amplitude, M wave amplitude and H: M ratio). H reflex is not a direct response of muscle to stimulation of its corresponding motor nerve but rather a reflex similar to a muscle stretch reflex. The H-reflex is usually elicited by delivering a submaximal stimulus to the tibial nerve in the popliteal fossa antidromically and recording over the soleus muscle. The generated nerve action potential propagates up to the spinal cord and then, via a predominantly monosynaptic reflex arc, passes down the efferent motor axon. The H-reflex differs from the muscle stretch reflex in that

1. The muscle spindle is bypassed

2. The afferent volley is temporarily less dispersed.

3. Tendon jerk involves significantly fewer lb fibres.

Several standard physiological tests are required to assess spasticity out of which H reflex amplitude M wave and H: M ratio showed significant changes in spasticity. Katz *et al* [9]. showed increased H (max) / M (max) ratios in spasticity following spinal cord injury and brain injury. In our study H amplitude was only significantly low compared to pre and post-phenol injections. The H/M ratio was not significant.

Quantitative gait analysis was done on 3 patients who were walking. It includes dynamic EMG recording from lower limb muscles as well as joint angle (Kinematic) data and ground reaction forces from the force plate. The physiological cost index was also calculated on 1 day and 21" days. The main significant change seen in kinematic data was the improvement in the

range of motion in the ankle after intervention with a posterior tibial nerve injection, but this was not statistically significant because only 3 patients could be evaluated.

The most common side effect reported from phenol injections is pain during the injection often described as a burning or stinging sensation and can be associated with edema several hours after injection. The side effect reported in this group of patients, related to phenol injections dysasthesia caused by the involvement of sensory nerve axons.

Many oral medications are currently available for the management of spasticity. Patients in this study were taking Tab. Diazepam, Tab. Tizanidine and Tab. Baclofen. The antispastic medication taken by the patients during this period of study was analysed, and it showed a significant reduction in the cost of antispastic medication following phenol administration.

Conclusions

1. Phenol blocks to peripheral nerves reduce spasticity among patients with spinal cord injury and brain injury as measured by the modified Ashworth scale.
2. There is a significant improvement in the range of movement of joints after blockade of spasticity using Phenol.
3. Following phenol blocks to peripheral nerves, there was a reduction in the amplitude of the H reflex. However, the fall in the H: M ratio was not significant.
4. Gait analysis demonstrated an increased range of dorsiflexion range at the ankle following posterior tibial nerve blockade however statistically significant conclusions could not be derived as only 3 patients in the study were able to walk.
5. There was a 5% incidence of adverse effects (pain) following the administration of phenol during the period of this study.
6. Chemical neurolysis with phenol was observed to be significantly cost-effective when compared to systemic antispastic medications.

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