Technical Report

Pulsed Radiofrequency of the Composite Nerve Supply to the Knee Joint as a New Technique for Relieving Osteoarthritic Pain: A Preliminary Report

Lakshmi Vas, MD, Renuka Pai, Diploma, Nishigandha Khandagale, MD, and Manorama Pattnaik, MD

From: Ashirvad Institute for Pain Management and Research, Mumbai, India

Address Correspondence:
Dr. Lakshmi Vas
M.D Anesthesia
Ashirvad Pain Relief Clinic
Plot. No. 117, Shubh Ashirvad, Road
No. 5
Hindu Colony, Dadar East,
Mumbai – 400 014
E-mail:
lakshmi@paincareindia.com

Disclaimer: There was no external funding in the preparation of this manuscript.

Conflict of interest: Each author certifies that he or she, or a member of his or her immediate family, has no commercial association (i.e., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted manuscript.

Manuscript received: 03-03-2014 Revised manuscript received: 06-20-2014 Accepted for publication: 07-29-2014

Free full manuscript: www.painphysicianjournal.com

We report a new technique for pulsed radiofrequency (PRF) of the entire nerve supply of the knee as an option in treating osteoarthritis (OA) of knee. We targeted both sensory and motor nerves supplying all the structures around the knee: joint, muscles, and skin to address the entire nociception and stiffness leading to peripheral and central sensitization in osteoarthritis. Ten patients with pain, stiffness, and loss of function in both knees were treated with ultrasonography (USG) guided PRF of saphenous, tibial, and common peroneal nerves along with subsartorial, peripatellar, and popliteal plexuses. USG guided PRF of the femoral nerve was also done to address the innervation of the quadriceps muscle. Assessment of pain (Numerical Rating Scale [NRS], pain DETECT, knee function [Western Ontario and McMaster Universities Osteoarthritis Index- WOMAC]) were documented pre and post PRF at 3 and 6 months. Knee radiographs (Kellgren-Lawrence [K-L] grading) were done before PRF and one week later. All the patients showed a sustained improvement of NRS, pain DETECT, and WOMAC at 3 and 6 months. The significant improvement of patellar position and tibio-femoral joint space was concordant with the patient's reporting of improvement in stiffness and pain. The sustained pain relief and muscle relaxation enabled the patients to optimize physiotherapy thereby improving endurance training to include the daily activities of life. We conclude that OA knee pain is a product of neuromyopathy and that PRF of the sensory and motor nerves appeared to be a safe, effective, and minimally invasive technique. The reduction of pain and stiffness improved the knee function and probably reduced the peripheral and central sensitization.

Key words: Osteoarthritis, knee pain, stiffness, knee innervation, femoral nerve supply, Hilton's law, peripheral sensitization, pulsed radiofrequency treatment of nerves to knee ioint

Pain Physician 2014; 17:493-506

ainful osteoarthritis of knee (OA) is a common condition with an estimated prevalence in 27 million Americans over 25 years of age (1). The pain in the OA knee is attributed to cartilage degeneration, reduced joint space, osteophytes, and loose bodies (2), even though self- reported pain intensity, disability, and its psychological impact correlate poorly with the peripheral joint damage assessed by the Kellgren-Lawrence scale (K-L scale) (3-

7). Recent evidence emphasizes the role of central sensitivity in the pathogenesis of the OA knee (8-13). We present a technique of pulsed radiofrequency (PRF) of the entire nerve supply of the knee. The PRF was performed to achieve 2 goals. One purpose was to achieve a pain-free and stiffness-free window for optimizing the knee function, and the other was to reduce the central sensitivity responsible for sustaining the pain and stiffness (14). We discuss the potential of

this technique in achieving both the goals in a small group of 10 patients.

METHODS

Ten patients who presented with pain, stiffness, and functional compromise in both knees were assessed with Numerical Rating Scale (NRS), pain DETECT scores (15), Kellgren-Lawrence (K-L) grading, and West-

ern Ontario and McMaster Universities Osteoarthritis Index (WOMAC) (5) as shown in Table 1, along with their demographic and clinical details. Assessments were done pre- and post-treatment at 3 and 6 months (Tables 2 and 3). Seven of the 10 patients had been on non-steroidal anti-inflammatory drugs (NSAIDs), paracetamol, and neuromodulators; one had received tramadol and one had received an intraarticular steroid

Table 1. Patient details regarding pain, limits of activities, X-ray findings, and treatment taken prior to presentation.

No./ Age in	Duratio	n (years)		Pain profi	le (NRS	S+)		N.P	Limits of	f activities in (min)	activities in minutes (min) K-L 'X-ray kn grades			Treatment taken prior to
years/ Sex of	Right	Left	Right		Left		S.D [!]	Score**	Standing	Walking	Climbing	Right	Left	consultation
patients	Rigili	Len	Rest	Activity	Rest	Activity			Standing	waiking	steps	Kight Left	Len	with us
1./61/M	2 years	5 years	0/10	5/10	3/10	8/10	+	13	60 min	60 min	Difficult (+)	Grade 1	Grade 2	NSAIDS
2./82/F	Many years; severe for 2 years	Many years; severe for 2 years	0/10	10/10	0/10	5/10	-	15	10 min	10 min	Difficult (++)	Grade 1	Grade 1	NSAIDS, gabapentin, intraarticular steroid
3./62/F	1 year	1 year	2/10	7/10	2/10	7/10	+	14	45 min	45 min	Difficult (+)	Grade 1	Grade 1	NSAIDS, duloxitene
4./52/F	15 years	15 years	3/10	5/10	6/10	10/10	+	18	10 min	10 min	Difficult (+++)	Grade 3	Grade 4	NSAIDS, gabapentin
5./60/F	15 years; severe for 2 years	15 years; severe for 3 years	3/10	10/10	2/10	8/10	+	16	10 min	10 min	Difficult (+++)	Grade 4	Grade 4	NSAIDS, amitrypltiline; replacement suggested, but patient unwilling
6./49/F	4 years	4 years	0/10	7/10	0/10	7/10	_	12	5-7 min	5-7 min	Difficult (++)	Grade 4	Grade 4	NSAIDS, weak opioids
7./57/F	Many years; severe for 7 years	Many years; severe for 7 years	5/10	9/10	2/10	5/10	-	17	5 min	5 min	Difficult (+++)	Grade 4	Grade 4	Alternative therapy (traditional Indian therapy of Ayurveda)
8./56/F	6 years; severe for 2 years	6 years; severe for 1 year	0/10	5/10	0/10	8/10	_	12	45 min	60 min	Difficult (+)	Grade 1	Grade 2	NSAIDS, pregabalin, muscle relaxants, intraarticular steroid
9./54/F	7 years	6 years	0/10	8/10	2/10	9/10	_	15	20 min	20 min	Difficult (+++)	Grade 2	Grade 2	NSAIDS, pregabalin, muscle relaxants, intraarticular steroid
10./77/F	Many years	Many years	5/10	10/10	5/10	10/10	+	14	2 min	2min	Difficult (+++)	Grade 4	Grade 4	NSAIDS

⁺ Numerical Rating Scale for pain intensity; * Kellgren-Lawrence radiological grading scale for OA knee;! Sleep Disturbances; ++ Neuropathic pain scoring by pain DETECT

Table 2. Patient data at 3 months.

		Pai	in		Limits of activities in minutes (min)			WOMAC score		
Patients	Ri	ght	I	eft	Limit	s of activities in	minutes (min)	WOMA	AC score	NP++ Score
	Rest	Activity	Rest	Activity	Standing	Walking	Climbing steps	Pre	Post	
1.	0/10	2/10	0/10	5/10	60 min	60 min	Fairly easy	71.2	77.8	10
2.	0/10	8/10	0/10	3/10	15min	15 min	Difficult (+)	42.4	50.2	11
3.	0/10	5/10	0/10	5/10	45min	45 min	Fairly easy	58.3	65.7	11
4.	0/10	4/10	3/10	8/10	10min	10 min	Difficult (++)	30.3	42.5	12
5.	0/10	7/10	0/10	6/10	15min	15min	Difficult (++)	34.1	45.7	12
6.	0/10	5/10	0/10	4/10	10 min	10 min	Difficult (+)	40.1	51.7	10
7.	2/10	5/10	0/10	4/10	7 min	7 min	Difficult (+++)	31.3	40.9	13
8.	0/10	4/10	0/10	6/10	45 min	60 min	Difficult (+)	70.8	78.6	11
9.	0/10	6/10	0/10	6/10	20 min	20 min	Difficult (+++)	56.4	64.3	11
10.	3/10	7/10	3/10	7/10	2 min	2 min	Difficult (+++)	24.6	32.5	11

⁺⁺ Neuropathic pain scoring by pain DETECT

Table 3. Patient data at 6 months.

		Pa	ain		Limits of activities in minutes			WOMAC score		NP++ Score
Patients	F	Right		Left						
	Rest	Activity	Rest	Activity	Standing	Walking	Climbing steps	Pre	Post	
1.	0/10	0/10	0/10	3/10	60 min	60 min	Fairly easy	71.2	87.9	5
2.	0/10	5/10	0/10	3/10	20 min	20 min	Difficult (+)	42.4	68.9	9
3.	0/10	4/10	0/10	4/10	60 min	60 min	Fairly easy	58.3	83.3	6
4.	0/10	2/10	3/10	5/10	15 min	15 min	Difficult (++)	30.3	59.8	10
5.	0/10	5/10	0/10	5/10	20 min	20min	Difficult (++)	34.1	66.7	10
6.	0/10	3/10	0/10	4/10	10 min	10 min	Difficult (+)	40.1	58.1	8
7.	0/10	3/10	0/10	1/10	10 min	10 min	Difficult (++)	31.3	48.9	9
8.	0/10	1/10	0/10	2/10	60 min	75 min	Fairly easy	70.8	86.4	6
9.	0/10	3/10	0/10	2/10	25 min	25 min	Difficult (+)	56.4	72.3	8
10.	1/10	4/0	1/10	4/10	5 min	5 min	Difficult (++)	24.6	39.3	7

⁺⁺ Neuropathic pain scoring by pain $\ensuremath{\mathsf{DETECT}}$

Patient 1 & 8 who had a good WOMAC to start with improved the maximum; patients 2, 3, 6 & 9 who had moderate scores improved considerably and patients 4, 5, 7 & 10 who had the lowest scores also showed improved activity and limits. Patients 2, 3, 8 & 9 with high pain scores and low K- L grades of 1 or 2 had the maximum benefit as at 12 weeks they had pain of 2-4 NRS on climbing up and down the stairs/ getting up from the chair/ getting out of a car. Patients 4, 5, 6, 7 & 10 with high pain scores as well as high K-L Grade of 3 or 4 also improved.

injection without any improvement in pain or stiffness. None had received viscosupplementation or chronic opioid therapy, as extreme restriction on the availability and licensing of opioids and non-availability of oxycodone and methadone makes opioid therapy uncommon in India for non-malignant chronic pain.

Of the above, pain DETECT is a simple, validated, patient-based, easy-to-use screening questionnaire to determine the prevalence of the neuropathic pain component. A score of 0 – 12 suggests the neuropathic component to be unlikely; a score of 12 – 18, suggests a

probable neuropathic component, and that of 18 – 38, suggests a greater than 90% likelihood of neuropathic pain). K-L grading is a radiograph-based method for assessing the deterioration of the joint in terms of severity as mild, moderate, and severe. It evaluates joint space narrowing, the presence of osteophytes, subchondral sclerosis, and bony deformity. WOMAC is a 24-item, patient-friendly, validated questionnaire for patients of OA knee/hip, used extensively in both observational/epidemiological studies and to examine changes following treatments such as pharmaco-

therapy, arthroplasty, exercise, physical therapy, knee bracing, and acupuncture. It has 3 subscales to assess pain (5 items), stiffness (2 items), and physical function (17 items).

The patients were given a thorough explanation of the scientific basis, the probable risks and benefits of the treatment, as well as the importance of maintaining a pain diary. Knee radiographs in the standing position were taken before and one week after PRF.

Informed consent was taken for PRF of 3 nerves and 3 plexuses around the knee. PRF was also administered to the femoral nerve that provides motor supply to the quadriceps muscle with its sesamoid bone patella

(Table 4 [16,17]).

PRF was performed in the operation theater with appropriate monitoring and aseptic precautions. Ultrasonography (USG) (Sonosite TM MSK, USA, linear 6-13 MHz transducer) guided the placement of a 10-cm, 22-gauge radiofrequency (RF) cannula, with a 10-mm active tip and confirmed the response to sensory (0.6 V at 50 Hz) and motor stimulation (2.0 V at 2 Hz) (COSMAN [cannula] RFK TM). Two millilitres of 2% lidocaine was injected before activation of the RF generator (COSMAN MEDICAL, INC. Burlington, MA, USA).

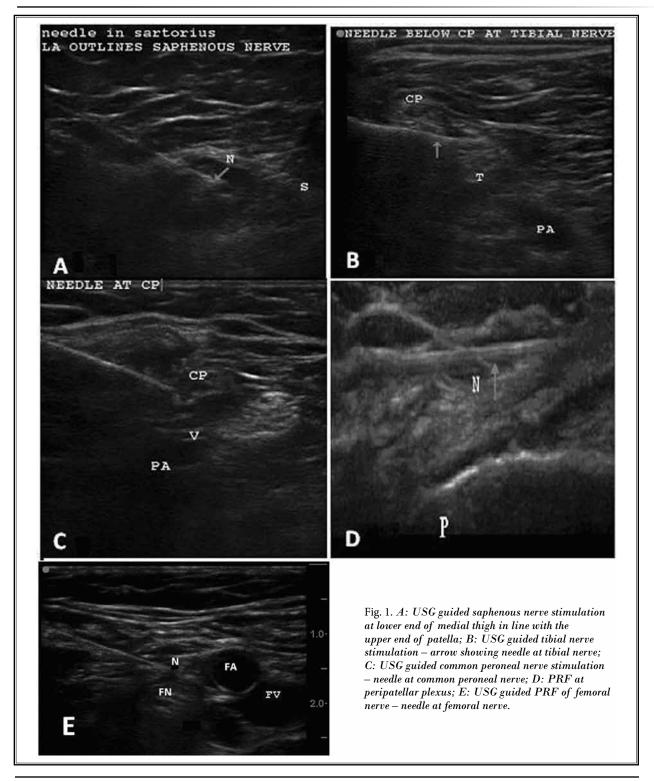
PRF was applied to the following nerves and plexuses for 8 minutes at 42°C:

Table 4. Nerve supply of the knee joint.

Nerve	Branches relevant to knee joint	Joint supply	Cutaneous supply °	Muscles supplied by the nerves	Contribution to plexus	Effects of PRF stimulation	
Saphenous nerve (L2, 3 roots)	Infrapatellar branch Descending branch	Antero inferior capsule of knee joint.	Anterior and medial side of leg, medial and dorsal side of foot up to great toe.	Sartorius; USG demonstrated sartorius twitches with infrapatellar branch of saphenous nerve stimulation implying that saphenous is a mixed nerve.	Peripatellar, subsartorial plexus supplying areas See 'Areas innervated by the plexuses around the knee'.	Paresthesia, pain on inferomedial aspect of patella. Motor twitches in sartorius concomitant with sensory response implying that saphenous is a mixed nerve	
Tibial nerve (I.4, 5 & S1, 2, 3 roots)	Articular (genicular) branches to knee medial superior medial inferior middle Capsular branches	Medial part of the capsule, retinaculum, collateral ligaments of knee joint, proximal and distal tibiofibular joint.	Lateral side and sole of foot, plantar aspect of toes.	Hamstrings, gastrocnemius, popliteus, soleus, plantaris.	Popliteal plexus supplying areas mentioned below	Motor twitches in plantar flexors.	
Common peroneal nerve (L4, 5 & S1, 2 roots)	Articular (genicular) branches to knee- Lateral superior Lateral inferior Recurrent	Inferolateral capsule of knee joint, proximal tibiofibular joint.	Anterior, posterior, lateral surface of leg, and foot, dorsum of foot and toes, first interdigital cleft	Short head of biceps femoris	Peripatellar plexus supplying areas mentioned below.	Motor twitches in biceps femoris, foot dorsiflexors and evertors.	
Plexuses		Contrib	ators to the plexus		Innervated areas		
Peripatellar	Femoral nerve: medi Saphenous nerve: inf Retinacular nerves: 1 branch of sciatic nerv Nerve to vastus inter	rapatellar branch medial (terminal bra ve)	nerves medius), lateral (direct	Skin anterior, superior, inferior, medial and lateral to patella; retinacula; collateral ligaments and capsule of knee joint.			
Subsartorial	Saphenous nerve: inf Obturator nerve: ant Medial femoral cutar Nerve to vastus medi	erior division neous nerve		Cutaneous to medial side of knee, retinaculum, collateral ligaments and capsule of knee joint.			
Popliteal	Tibial nerve Sciatic nerve Obturator nerve			Retinaculum, collateral ligaments and capsule of knee joint.			

[°] It is to be noted that peripheral nerves per se do not innervate the skin over the knee, instead contribute to the plexuses which innervate the skin as described in the lower half of this table. However, the cutaneous distribution of the peripheral nerves as described in this column enables us to understand the temporal radiation of pain in these areas as is frequently described by patients.

a) The saphenous nerve (Fig. 1A), towards the end of Hunter's canal in line with the upper border of patella where its infrapatellar branch emerges through the sartorius muscle. The needle was then directed deep to the sartorius for PRF of the subsartorial plexus.



- b) The tibial nerve above the popliteal crease (Fig. 1B). The effect of PRF was presumed to extend to the popliteal plexus which lies in close anatomical proximity to the nerve. The needle was then directed to common peroneal nerve for PRF (Fig. 1C).
- c) The peripatellar plexus: The needle was placed subcutaneously alongside the patella. PRF was administered for 8 minutes with a gradual withdrawal of the needle to cover the superior, lateral, inferior, and medial borders of the patella (Fig. 1D).
- d) The femoral nerve lateral to femoral artery just below the inguinal ligament (Fig. 1E).

The figure schematic that illustrates how the above mentioned nerves and plexuses were targeted is shown in Figs. 2 and 3.

The USG facilitated quick location of various nerves and the simultaneous PRF with 4 electrodes placed at different nerves to be targeted limited the total time taken to treat one knee to 45 – 50 minutes. After the procedure, patients were assessed for sensory as well as motor deficits prior to discharge. Patients were sent home with Paracetamol 500 mg thrice for a day. All patients received a customized physiotherapy regimen that included stretches initially, and strengthening and endurance training later by a certified physical therapist. Advice on lifestyle changes and dietary advice by a dietician were given as necessary.

RESULTS

Perusal of patient pain diaries showed that the effect of PRF was evident as relief in rest pain, stiff-

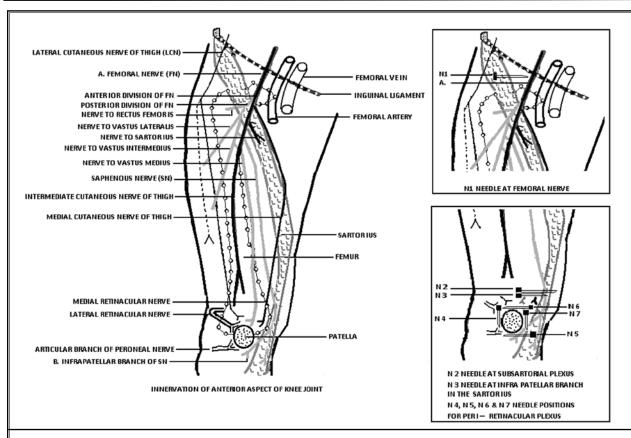


Fig. 2. Innervation of anterior aspect of knee joint. Sartorius forms the roof of Hunter's canal. The contents of the canal are saphenous nerve and nerve to vastus medialis. The infrapatellar branch of saphenous nerve pierces the sartorius at the level of superior end of patella before traveling to medial ankle. PRF targeted the nerves in the substance of the sartorius. The motor stimulation of this nerve specifically elicited twitches in sartorius indicating that it carries motor twigs to sartorius at its lower end. Inset 1 shows the in-plane approach to femoral nerve.

Inset 2 shows the needles placed perpendicular to each other in the subcutaneous plane around the patella.

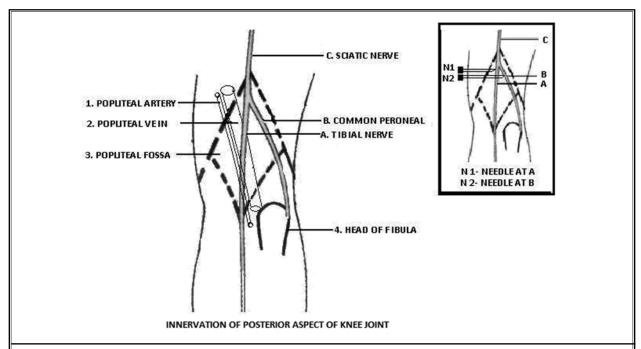


Fig. 3. Innervation of posterior aspect of knee joint. The needles are placed in an in-plane approach at the tibial and common peroneal nerves for PRF.

ness, swelling, and pain-induced sleep disturbances by the second day at the level of pre-block activity in all 10 patients. NRS, pain DETECT, and K-L grading indicated improvements at one week. Subtle but distinct changes were seen in both patellofemoral and tibiofemoral joints of the knee as seen in radiographs taken at one week in all 10 patients (Fig. 4). Despite this, it took 3 months for the physiotherapy to reverse this deconditioning with planned strengthening exercise programs and gradual introduction of weight-bearing stresses in these patients (especially in 2 of the patients who were obese) to regain the work ability. Sudden increase in activity at this time did produce pain, but this would subside mainly with rest, physical therapy modalities, and occasional NSAIDs, unlike in the preblock period. Activity-induced pain as well as the limits of activities like standing, walking, and climbing steps gradually improved over next 3 months, as indicated by the improving WOMAC scores (40% - 100% of the baseline). At 6 months, the patients had achieved a full range of motion at the knee as well as the ability to perform weight-bearing activities. However, they still had pain up to 2 – 5 NRS on stair climbing. No adverse effects or sensorimotor deficits secondary to PRF were observed in the early or late follow-up periods.

Statistical analysis: Data were analyzed using SPSS/

PC + statistical package (Statistical Package for Social Sciences Version 15.0, USA). Data were summarized as Mean + SD (Minimum, Maximum) for quantitative data and Number (%) for qualitative data. Friedman's test (nonparametric) was applied to data to compare overall trends at 3 time points (at presentation, 3 months, and 6 months post treatment). Friedman's test was preferred to parametric tests as the data were scores. Data on climbing steps were transformed to scores for statistical analysis purpose. All tests were 2 tailed. Level of significance was taken as P = 0.05 (where P was the probability value). The mean and SD for age-wise distribution was 61.00 ± 10.61 years; gender-wise distribution was females = 9 (90%), male = 1 (10%); duration of pain in the right knee was 5.70 ± 5.33 years; duration of pain in the left knee was 5.90 ± 5.20 years. The analysis of pain in both knees at rest and on activities, the WOMAC scores, the NP scores, and the limits of activities were all done at 3 and 6 months in comparison to that at presentation (Appendices 1 - 4).

There were statistically significant decreases in pain in both knees at rest and activities at the end of 6 months, statistically significant increase in WOMAC scores at the end of 6 months, statistically significant decrease in NP score at the end of 6 months, statistically significant increase in the standing and walking

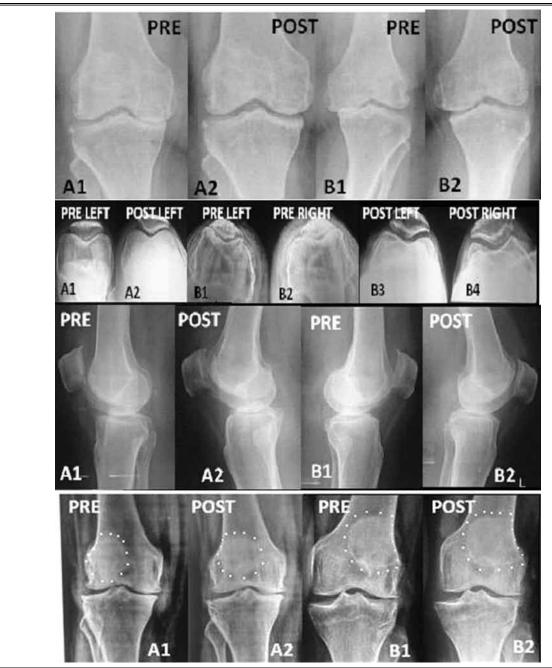
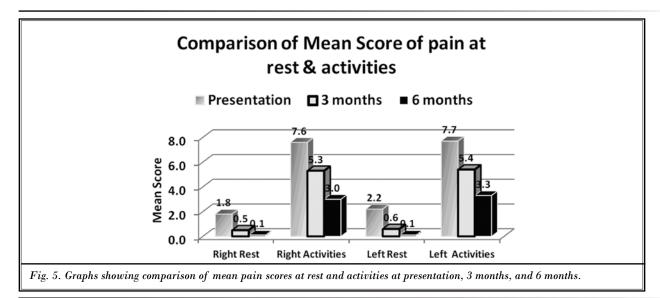


Fig. 4. Row 1: The femur and tibia in the medial compartment of both knees which were grinding against each other before PRF have shifted away from each other one week after PRF, with an obvious increase in the joint space between the femur and tibia. This implies the relaxation of the muscles (sartorius, semitendinosus, and gracilis) contributing to the pesanserine tendon that straddles the medial compartment from femur to the tibia. Row 2: Shows knee x rays of 2 patients referred as A and B. Patient A had no compromise of the right patellofemoral joint space at presentation. Hence, only the left x ray knee is shown. Note that the patella has moved more anteriorly from femur after PRF thereby increasing the joint space between femur and patella. This results in reducing the friction and facilitating smoother patellar movement over femur. In short, a relaxation of all the components of quadriceps muscle is implied. Row 3: Both the right and left patellae, which were pulled up before PRF, have shifted inferiorly after PRF with relaxation of quadriceps after femoral nerve PRF. Note also the change in the patellofemoral angles. This implies a relaxation of the rectus femoris and vastusintermedius muscle that control anteroposterior positioning of patella. Row 4: Note the shift of patella medially after PRF in 2 different patients, A and B. The x ray of patient B shows the patella to have moved from beyond the femoral border to the center. The patellar position is determined by the components of quadriceps muscle. Dominance of vastuslateralis over vastusmedius pulls the patella laterally and relaxation of vastus subsequent to PRF allows the patella to return to its anatomical position.

time, and a statistically significant decrease in difficulty in climbing steps at the end of 6 months. The graphs of mean pain scores, mean WOMAC scores, mean NP scores, and mean activity time of standing and walking along with ease of climbing steps are depicted in Figs. 5–9.



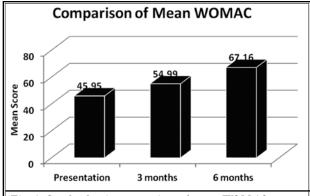


Fig. 6. Graphs showing comparison of mean WOMAC scores at presentation, 3 months, and 6 months.

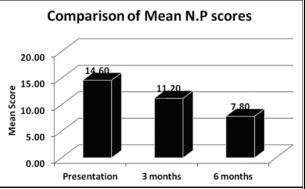


Fig. 7. Graphs showing comparison of mean pain DETECT scores at presentation, 3 months, and 6 months.

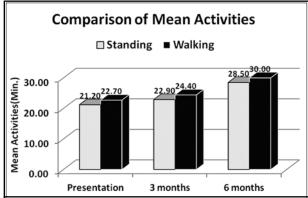


Fig. 8. Graphs showing comparison of mean increase in the level of activities at presentation, 3 months, and 6 months.

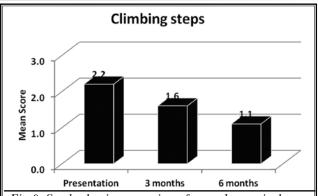


Fig. 9. Graphs showing comparison of mean decrease in the level of efforts in climbing steps at presentation, 3 months, and 6 months.

DISCUSSION

Persistent pain is one of the many manifestations of inflammation caused by the complex pathophysiological process involved in joint damage of the OA knee (18,19). Other accompanying phenomena include muscle spasm/stiffness that compromises the range of movements, the joint effusions manifesting as swelling around the joint, and most importantly, peripheral and central sensitization in the pain pathway (19). Quantitative sensory testing in OA has been reported to show a diffuse and persistent alteration of nociceptive (pain) pathways, irrespective of the level of activity of the underlying disease (20).

The innervation of the human knee follows Hilton's law, "all of the motor efferents serving muscles acting on the knee carry afferent branches from the knee capsular elements" (17,21). This implies that inflammation of the joint structures (such as capsule, synovium, and ligaments) activates the afferent limb of the reflex that initiates muscle contraction. The efferent limb of the reflex is formed by the motor nerves supplying the muscles that move the knee joint. Thus, inflammation of capsular elements causes reciprocal spasm of the muscles acting on the knee. The muscles in spasm (commonly perceived as "stiffness" by patients) could now compromise the situation in 2 ways. The stiff muscles further impede knee movements causing more inflammation, setting up a vicious cycle of "pain-spasmpain." Secondly, muscle pain is referred to the overlying skin. Thus, the joint movements necessary for the daily activities of life lead to stiffness and a wide-spread distribution of pain in the joint, overlying muscles, and skin. All these contribute to the peripheral sensitization, which in turn contributes to the development of central sensitization.

The overlap of the nerve supply around the knee that ensures a failsafe mechanism for pain transmission under physiological circumstances paves the way for a self-sustaining and self-perpetrating neuropathy with the onset of degenerative processes of OA. The neuropathy, especially that involving the motor nerves mediating Hilton's law, manifests clinically as a severe reduction of functional ability (11,22).

The choice of the nerves to be subjected to PRF was based on a report that described the innervation of the knee studied after cadaveric dissection of 45 knee specimens (17). This study was undertaken to provide surgeons with logical targets for ablative procedures considered as an option to relieve post total knee re-

placement (TKR) pain syndromes. We also included PRF of the femoral nerve with the express purpose of relaxing the guadriceps muscle so that the patellofemoral joint space could be optimized. All our patients had radiographs suggestive of compromise of patellofemoral as well as the tibiofemoral components of the knee joint before PRF. The radiographic changes indicating improved joint space at both the tibifemoral and patellofemoral joints after PRF supported our surmise of pain and spasm - joint inflammation - more pain and spasm; that reflex muscle spasm in response to joint inflammation was indeed holding the bones in closer proximity to each other, and that PRF would relieve this reflex spasm by reduction of the pain (the pain afferents from knee) as well as by a direct action through the PRF effect on motor fibers of femoral nerve. The net result was pain relief and relaxation of the muscles acting across the knee, with a resultant reduction of inflammatory effusion and swelling.

Choi et al (23) have described fluoroscopically guided radiofrequency neurotomy of the sensory nerves (genicular nerves) supplying the joint with the premise that ablating the nerve supply to a painful structure may alleviate pain and restore function. We performed PRF with USG guidance instead of conventional RF with fluoroscopy as we wished to avoid a neuroablative procedure, particularly in our younger patients. To this end, we extended our target to include the motor nerve supply as well as entire sensory supply around the knee joint, muscle, and skin. Our goal was to reduce pain, nullify the deleterious effects of Hilton's law causing the accompanying muscle stiffness that perpetuates the inflammation of joint structures, and most importantly, to reduce the entire peripheral input to central sensitization. Our premise was to reduce the afferent nociceptive input to the neuraxis as well as to produce relaxation of the muscles acting across the joint by reduction of the efferent outflow of motor response to pain and inflammation. The resultant pain relief and muscle relaxation improved the mechanics of joint function allowing the patients to perform effective strengthening and endurance training without the effort-induced exacerbations of pain that had restricted the lifestyle in these patients (9,24,25). Sustained relief of pain and stiffness deprived central sensitivity of its input. Attenuation of the "wind up" in the pain pathways presumably started the repair process restoring normal pain modulation in the neuraxis.

The initial pain DETECT scores were mild to moderate, reflecting a combination of inflammatory and neu-

ropathic symptoms. The most obvious improvements were seen in the more composite WOMAC assessments. It was indeed difficult to determine what caused the structural improvements in the knee demonstrated on radiographs which presumably explain the clinical relief in objective terms. The operation of Hilton's law complicates issues as it links synovial nociception with muscle spasm. There were 2 possibilities. One was that interruption of nociceptive traffic in the plexuses/sensory component of mixed nerves reduced the operation of Hilton's law, thereby reducing the muscle tensions approximating the bones. The other possibility was the direct reduction of the muscle tension mediated by motor component of mixed nerves following PRF. Selective PRF of the plexuses/sensory nerves versus PRF of only mixed nerves might provide the answer. Either way, it was the reduction of tension in the muscles approximating the bones that played an important role in the improvement of knee function. This allowed us to come to the following interesting surmise: the neuropathic processes involving both sensory and motor nerves makes OA knee pain a product of neuromyopathy rather than just neuropathy (14). Unlike neuropathic pains elsewhere in the body, the neuromyopathic component of the pain of OA has the ability to impede the free mobility of the knee joint. Embarrassed joint movement induces synovial irritation with the initiation of Hilton's law which alters the tensioning of muscle further, progressively impacting joint function, resulting in the manifestation of all the classical features of the OA knee. The motor neuropathy presumably has the potential to directly produce structural changes in the joint by altering tension in the muscles acting across the patellofemoral and tibiofemoral joints. PRF of these motor nerves reverses the neuromyopathy and its effect on the structural components of the joint. The net result, as seen in our study, is a normalization of joint contours after PRF. The clinical improvement in these 10 patients was probably a reflection of a complex interplay between reduction in the central sensitivity and

operation of Hilton's law.

PRF at 42°C was aimed at avoiding any neurological deficits that could lead to Charcot's joint. RF creates an alternating electric field with an oscillating frequency of 500,000 Hz to elicit heat production around the percutaneously introduced needle tip by the body tissue acting as the resistor (26). The output of the generator is interrupted to give 2 cycles/second each of 20-msec bursts followed by silent phases of 480 millisecond in PRF (27-29). The interval between the cycles allow for the dissipation of the heat maintaining the tissue temperature at 42°C, far below the irreversible tissue damage threshold range of 45 – 50°C (30). Thus PRF has no incidence of sensory or motor complications, unlike conventional RF ablation, which creates tissues temperatures of 70°C and above (31-34). PRF has been used successfully to treat myofascial trigger points, knee pain by intraarticular application, and various peripheral neuropathic pains (29,35-49). PRF appears to have genuine biological effects in cell morphology, synaptic transmission, and pain signalling, which are likely to be temperature independent (50-57).

CONCLUSION

PRF of peripheral nerves and plexuses supplying the knee joint appeared to be a safe, effective, and minimally invasive new technique that addresses the sensory, motor, and autonomic nerves to provide sustained relief of pain, stiffness, swelling, and the peripheral and central sensitivity in response to chronic pain in both knees from long-standing osteoarthritis in 10 patients. The x-ray changes appeared to correlate well with clinical improvement documented by WOMAC, a scale extensively used to examine changes following treatments. However, the efficacy of this technique requires further elucidation in a larger group with serial knee radiographs up to one year and also whether there is a reliable correlation between measured structural shifts in the patients who improve the most in term of pain and stiffness.

www.painphysicianjournal.com 503

APPENDIX 1:

Pain Analysis	Presentation/Basal	3 months	6 months	Friedman's test Chi sq. value, DF, Significance & P value
Right Rest	1.80 ± 2.10	0.50 ± 1.08	0.10 ± 0.32	9.3, DF=2,S,P=0.01
Right Activities	7.60 ± 2.12	5.30 ± 1.77	3.00 ± 1.63	20.0,DF=2, S,P<0.001
Left Rest	2.20 ± 2.04	0.60 ± 1.26	0.10 ± 0.32	13.1,DF=2, S,P=0.01
Left Activities	7.70 ± 1.77	5.40 ± 1.51	3.30 ± 1.34	19.2, DF=2,S,P<0.001

Overall Conclusion: There were statistically significant decreases among all variables at the end of 6 months.

APPENDIX 2:

WOMAC score

Friedman's test

Chi sq. value, DF, Significance &

Presentation/Basal 3 months 6 months P value

WOMAC 45.95 ± 17.06 54.99 ± 15.85 67.16 ± 16.11 20.0,DF=2,S,P<0.001

Overall Conclusion: There was statistically significant increase in WOMAC score at the end of 6 months.

APPENDIX 3:

NP score

Friedman's test

Chi sq. value, DF, Significance &

Presentation/Basal 3 months 6 months P value

NP score 14.60 ± 2.01 11.20 ± 0.92 7.80 ± 1.75 20.0, DF=2,S,P<0.001

Overall Conclusion: There was statistically significant decrease in NP score at the end of 6 months.

APPENDIX 4:

Limits of activities

Friedman's test

Chi sq. value, DF, Significance &

	Presentation/Basal	3 months	6 months	P value
Standing	21.20 ± 20.84	22.90 ± 19.75	28.50 ± 22.49	15.8,DF=2, S,P<0.001
Walking	22.70 ± 23.16	24.40 ± 22.05	30.00 ± 25.17	15.8, DF=2,S,P<0.001
Climbing steps#	2.20 ± 0.92	1.60 ± 1.17	1.10 ± 0.88	15.2, DF=2,S,P=0.001

Climbing steps score

(fairly easy=0, little difficult=1,moderate difficult=2,very difficult=3)

Overall Conclusions: .

- 1. There were statistically significant increases in Standing & walking at the end of 6 months
- 2. There was statistically significant decrease in difficulty in climbing score at the end of 6 months.

REFERENCES

- Lawrence RC, Felson DT, Helmick CG, Arnold LM, Choi H, Deyo RA, Gabriel S, Hirsch R, Hochberg MC, Hunder GG, Jordan JM, Katz JN, Kremers HM, Wolfe F. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States: Part II. Arthritis Rheum 2008; 58:26-35.
- www.niams.nih.gov/Health_Info/Osteoarthritis/default.asp. Online version updated July 2010.
- Bedson J, Croft PR. The discordance between clinical and radiographic knee osteoarthritis: A systematic search and summary of the literature. BMC Musculoskelet Disord 2008; 9:116-126.
- Kellgren JH, Lawrence JS. Radiological assessment of osteoarthritis of knee. Ann Rheum Dis 1957; 16:494-501.
- Goggins J, Baker K, Felson D. What WOMAC pain score should make a patient eligible for a trial in knee osteoarthritis? J Rheumatol 2005; 32:540-542.
- Hannan MT, Felson DT, Pincus T. Analysis of the discordance between radiographic changes and knee pain in osteoarthritis of the knee. J Rheumatol 2000; 27:1513-1517.
- Dieppe P, Basler HD, Chard J, Croft P, Dixon J, Hurley M, Lohmander S, Raspe H. Knee replacement surgery for osteoarthritis: Effectiveness, practice variations, indications and possible determinants of utilization. Rheumatology (Oxford) 1999; 38:73-83.
- Allen K. Central pain contributions in osteoarthritis: Next steps for improving recognition and changes. Arthritis Res & Ther 2011; 13:133.
- Murphy SL, Lyden AK, Phillips K, Clauw DJ, Williams DA. Subgroups of older adults with osteoarthritis based upon differing comorbid symptom presentations and potential underlying pain mechanisms. Arthritis Res Ther 2011; 13:R135.
- Hochman JR, French MR, Bermingham SL, Hawker GA. The nerve of osteoarthritis pain. Arthritis Care Res 2010; 62:1019-1023.
- 11. Mease PJ, Hanna S, Frakes EP, Altman RD. Pain mechanisms in osteoarthritis: Understanding the role of central pain and current approaches to its treatment. *J Rheumatol* 2011; 38:1546-1551.
- 12. Gwilym SE, Keltner JR, Warnaby CE, Carr AJ, Chizh B, Chessell I, Tracey I. Psychophysiological and functional imaging evidence supporting the presence

- of central sensitization in a cohort of osteoarthritis patients. *Arthritis Rheum* 2009; 61:1226-1234.
- Lee Y, Nassikas NJ, Clauw DJ. The role of the central nervous system in the generation and maintenance of chronic pain in rheumatoid arthritis, osteoarthritis and fibromyalgia. Arthritis Research & Therapy 2011; 13:211.
- 14. Vas L, Khandagale N, Pai R. Successful management of chronic post-surgical pain following total knee replacement. Pain Med 2014; 15:1781-1785.
- Freynhagen R, Baron R, Gockel U, Tölle T. painDETECT: A new screening questionnaire to identify neuropathic components in patients with back pain. Curr Med Res Opin 2006; 22:1911-1920.
- Mahadevan V (Section Editor). Pelvic girdle and lower limb. In: Gray's Anatomy – the Anatomical Basis of Clinical Practice. 40th ed. Churchill Livingstone, Elsevier, London 2008, 1383, 1393-1429.
- 17. Horner G, Dellon L. Innervation of the human knee joint and implications for surgery. Clinical orthopedics and related research 1994; 301:221-226.
- Amin AR. Regulation of tumor necrosis factor-alpha and tumor necrosis factor converting enzyme in human osteoarthritis. Osteoarthritis and Cartilage 1999; 7:392-394.
- Schaible HG, Ebersberger A, Von Banchet GS. Mechanisms of pain in arthritis. Annals of the New York Academy of Sciences 2002; 966:343-354.
- Kidd BL, Photiou A, Inglis JJ. The role of inflammatory mediators on nociception and pain in arthritis. Novartis Foundation Symposium 2004; 260:120-133.
- Hilton, J. Rest and Pain. A Course of Lectures. Ed. 2. P. W. Garfield, Cincinnati, 1891. Anatomy.
- Creamer P. Osteoarthritis pain and its treatment. Curr Opin Rheumatol 2000; 12:4505.
- Choi WJ, Hwang SJ, Song JG, Leem JG, Kang YU, Park PH, Shin JW. Radiofrequency treatment relieves chronic knee osteoarthritis pain: A double-blind randomized controlled trial. *Pain* 2011; 152:481-487.
- 24. Imamura M, Imamura ST, Kajiyama HHS, Targino RA, Hsing WT, de Souza LPM, Cutait MM, Fregni F, Camanho GL. Impact of nervous system hyperalgesia on pain, disability, and quality of life in patients with knee osteoarthritis: A controlled analysis. Arthritis Rheum 2008; 59:1424-1431.

- McAlindon TE, Cooper C, Kirwan JR, Dieppe PA. Determinants of disability in osteoarthritis of the knee. Ann Rheum Dis 1993; 52:258-262.
- 26. Sluijter M, Racz G. Technical aspects of radiofrequency. *Pain Practice* 2002; 2:195-200.
- Sluijter ME, Cosman E, Rittman W, van Kleef M. The effect of pulse radiofrequency fields applied to dorsal root ganglion: A preliminary report. *Pain Clin* 1998; 11:109-117.
- Abejon D, Reig E. Is pulsed radiofrequency a neuromodulation technique? Neuromodulation 2003; 6:1-3.
- Tamimi MA, McCeney MH, Krutsch J. A case series of pulsed radiofrequency treatment of myofascial trigger points and scar neuroma. *Pain Med* 2009; 10:1140-1143.
- Erdine S, Bilir A, Cosman ER, Cosman Jr ER. Ultrastructural changes in axons following exposure to pulsed radiofrequency fields. Pain Pract 2009; 9:407-417.
- Slujter ME. Pulsed radiofrequency. Anaesthesiology 2005; 103:1313.
- 32. Uematsu S. Percutaneous electrothermocoagulation of spinal nerve trunk, ganglion and rootlets. In: Schmidel HH, Sweet WS (eds). Current Techniques in Operative Neurosurgery. Grune and Stratton, New York, 1977. pp. 469-490.
- 33. Slappendel R, Crul BJJ, Braak GJJ, Geurts JWM, Booij LH, Voerman VF, Boo T. The efficacy of radiofrequency lesioning of cervical spinal dorsal root ganglion in a double blinded randomized study: No difference between 40°C and 67°C treatments. *Pain* 1997; 73:159-163.
- 34. Van Kleef M, Barendse GAM, Dingemans WAA, Winge C, De Lousberg R, Lange S, Sluijter ME. Effects of producing a radiofrequency lesion adjacent to the dorsal root ganglion in patient with thoracic segmental pain. Clin J Pain 1995; 11:325-332.
- West M, Wu H. Pulsed radiofrequency ablation for residual and phantom limb pain: a case series. *Pain Pract* 2010; 10:485-491.
- 36. Vanelderen P, Rouwette T, De Vooght P, Puylaert M, Heylen R, Vissers K, Van Zundert J. Pulsed radiofrequency for the treatment of occipital neuralgia: a prospective study with 6 months of follow-up. Reg Anesth Pain Med 2010; 35:148-151.
- Philip CN, Candido KD, Joseph NJ, Crystal GJ. Successful treatment of meralgia paresthetica with pulsed radiofrequency of the lateral femoral cutaneous nerve. Pain Physician 2009: 12:881-885.

www.painphysicianjournal.com 505

- Cohen SP, Foster A. Pulsed radiofrequency as a treatment for groin pain and orchialgia. *Urology* 2003; 61:645.
- 39. Higuchi Y, Nashold BS Jr., Sluijter M, Cosman E, Pearlstein RD. Exposure of the dorsal root ganglion in rats to pulsed radiofrequency currents activates dorsal horn lamina I and II neurons. Neurosurgery 2002; 50: 850-856.
- Tekin I, Mirzai H, Ok G, Erbuyun K, Vatansever D. A comparison of conventional and pulsed radiofrequency denervation in the treatment of chronic facet joint pain. Clin J Pain 2007; 23;524-529.
- Sluijter ME, Teixeira A, Serra V, Balogh S, Schianchi P. Intra-articular application of pulsed radiofrequency for arthrogenic pain—report of six cases. *Pain Pract* 2008; 8:57-61.
- Karaman H, Tufek A, Kavak GO, Yildirim ZB, Uysal E, Celik F, Kaya S. Intra-articularly applied pulsed radiofrequency can reduce chronic knee pain in patients with osteoarthritis. J Chin Med Assoc 2011; 74:336-340.
- Shah RV, Racz GB. Pulsed mode radiofrequency lesioning to treat chronic post-tonsillectomy pain (secondary glossopharyngeal neuralgia). Pain Pract 2003; 3:232-237.
- 44. Restrepo Garces CE, Marinov A, McHardy P, Faclier G, Avila A. Pulsed radiofrequency under ultrasound guidance for persistent stump-neuroma pain. *Pain Pract* 2011; 11:98-102.

- Liliang P, Lu K, Liang CL, Tsai YD, Hsieh C, Chen HJ. Pulsed radiofrequency lesioning of the suprascapularn for chronic shoulder pain: A preliminary report. Pain Med 2009; 10:70-75.
- Kane TPC, Rogers P, Hazelgrove J, Wimsey S, Harper GD. Pulsed radiofrequency applied to the suprascapular nerve in painful cuff tear arthropathy. *Journal of Shoulder and Elbow Surgery* 2008; 17:436-440.
- Kang KN, Park IK, Suh JH, Leem JG, Shin JW. Ultrasound-guided pulsed radiofrequency lesioning of the phrenic nerve in a patient with intractable hiccup. Korean J Pain 2010; 23:198-201.
- Lee JH, Kim TY, Ha SH, Kwon YE, Yoon CS. Pulsed radiofrequency lesioning of supraorbital and supratrochlear nerve in postherpetic neuralgia – A report of 2 cases. J Korean Pain Soc 2004; 17:239-242.
- Akural E, Järvimäki V, Korhonen R, Kautiainen H, Haanpää M. Pulsed radiofrequency in peripheral posttraumatic neuropathic pain: A double blind sham controlled randomized clinical trial. Scandinavian Journal of Pain 2012; 3:127-131.
- 50. Podhajsky RJ, Sekiguchi Y, Kikuchi S, Myers RR. The histologic effects of pulsed and continuous radiofrequency lesions at 42 degrees C to rat dorsal root ganglion and sciatic nerve. Spine (Phila Pa 1976) 2005; 30:1008-1013.
- 51. Bogduk N. Pulsed radiofrequency. Pain

- Med 2006; 7:396-407.
- Sluijter ME. Pulsed radiofrequency.
 In: Radiofrequency, Part 1. Fliovopress
 SA, Meggen (LU), Switzerland, 2001, p
 55-68.
- Hamann W, Abou-Sherif S, Thompson S, Hall S. Pulsed radiofrequency applied to dorsal root ganglia causes a selective increase in ATF3 in small neurons. Eur J Pain 2006: 10:171-176.
- 54. Hagiwara S, Iwasaka H, Takeshima N, Noguchi T. Mechanisms of analgesic action of pulsed radiofrequency on adjuvant-induced pain in the rat: roles of descending adrenergic and serotonergic systems. Eur J Pain 2009; 13:249-252.
- 55. Chua NHL, Vissers KC, Sluijter ME. Pulsed radiofrequency treatment in interventional pain management: Mechanisms and potential indications—a review. Acta Neurochir 2011; 153:763-771.
- 56. Tun K, Cemil B, Gurcay AG, Kaptanoglu E, Sargon MF, Tekdemir I, Comert A, Kanpolat Y. Ultrastructural evaluation of pulsed radiofrequency and conventional radiofrequency lesions in rat sciatic nerve. Surg Neurol 2009; 72:496-500; discussion 501.
- 57. Aksu R, Ugur F, Bicer C, Menku A, Guler G, Madenoglu H, Canpolat DG, Boyaci. The efficiency of pulsed radiofrequency application on L5 and l6 dorsal roots in rabbits developing neuropathic pain. Reg Anesth Pain Med 2010; 35:11-15.