



CLINICAL METHODOLOGY

Nerve conduction velocities in the lower extremity in patients with Raynaud's phenomenon and clinical applications

Dimitrios Kostopoulos, PT, PhD, DSc*, Konstantine Rizopoulos, PT, FABS, Nikolaos Vartholomeos, PT, DPT

5 Engineers Road, Roslyn Harbor, NY 11576, USA

Received 7 August 2007; received in revised form 17 October 2007; accepted 18 October 2007

KEYWORDS

Nerve conduction studies;
Raynaud's phenomenon;
Neural mobilization;
Electromyography

Summary

Background and purpose: The purpose of the study is to study the nerve conductivity of the tibial motor, peroneal motor, peroneal sensory, and sural nerves in patients with primary and secondary Raynaud's phenomenon (RP).

Subjects: Twenty each: primary RP, secondary RP, and normal controls.

Methods: Electromyography using distal latency (DL) and nerve conduction velocity (NCV) as dependent variables.

Results: Peroneal nerve DLs were slower and NCVs were weaker for the secondary RP group compared to the primary RP group and controls. Tibial motor nerve DLs from slowest to fastest were: primary RP, secondary RP, and controls. NCV strength order was: secondary RP weakest, primary RP, and controls.

Discussion: Patients with secondary RP generally had the slowest DLs and the weakest NCVs, with differences most pronounced in the motor nerves. With the exception of the tibial motor nerve, patients with primary RP had similar NCVs to the control group. Neural mobilization techniques can be applied to assist with patient symptoms.

© 2007 Elsevier Ltd. All rights reserved.

Introduction

Raynaud's phenomenon (RP) is a vascular disorder that most commonly affects the fingers, toes, ears,

and nose. It is characterized by episodic vasospastic attacks that cause the blood vessels in the digits to constrict. RP affects 5–10% of the general population in the United States and is more common in women than in men (Medical College of Wisconsin, 1999).

Vasospastic attacks are usually triggered by response to cold or stress and usually last anywhere

*Corresponding author. Tel.: +1 917 538 2242.

E-mail address: DIMIPT@aol.com (D. Kostopoulos).

from 5 min to a few hours. Normally, when the body is exposed to cold it will preserve its core temperature by moving the blood from the extremities towards the center of the body (peripheral vasoconstriction). In the normal population, warmth can still be maintained in the extremities due to thermoregulatory reflexes to protect against a total loss of blood flow to these areas. In patients with RP however, when the body responds to cold by shunting the blood towards the center of the body, spasmodic contractions of the small blood vessels occur in the extremities. This interferes with the normal thermoregulatory reflex and causes a loss of circulation to these areas. Typical symptoms of an attack include tingling and numbness in the fingers or toes, blanching or whitening, and pain, sometimes with redness, which accompanies the return of blood to the hands and feet. During the attack, three phases of skin color change may occur, known as the classic color triad. Pallor (whiteness) may occur in response to vasospasm. Cyanosis (blueness) may occur from lack of oxygen to the extremity. As blood returns, redness may occur in the affected area. The order of the color changes is not the same for all people and not everyone experiences all three-color changes (Medical College of Wisconsin, 1999; Canadian Centre for Occupational Health and Safety, 2004).

When RP occurs in the absence of a detectable underlying condition or without any other symptoms it is termed "Primary RP." Other names for this condition include idiopathic RP, Raynaud's disease, and Raynaud's syndrome. Primary RP occurs more commonly in females between the ages of 20 and 30 years old, and symptoms tend to be mild and symmetrical, usually attacking all digits. Primary RP rarely results in digital ulcerations; there is no evidence of tissue gangrene and nail fold capillaries appear normal. Systemic vasospasms rarely occur and there are seldom any findings of a secondary cause. Erythrocyte sedimentation rate is normal and serologic findings are negative for other rheumatologic conditions (Wrigley, 1998; Medical College of Wisconsin, 1999; Block and Sequeria, 2001).

Researchers have suggested that primary RP accounts for the vast majority of the cases of RP. Brand et al. (1997) in a 16-year-old follow up study of a cohort of 4182 men and women in Framingham Massachusetts, reported that primary RP accounted for 81.4% of the cases. Primary RP patients differed from non-RP patients by having lower blood pressures, higher incidence of coronary disease, and higher blood sugar counts. Subjects with primary RP were also more likely to be regular smokers.

Secondary RP, while less common than primary RP, tends to be more complex and serious. It often results in progressive digital damage and visceral organ involvement. Abnormal serologic findings and elevated erythrocyte sedimentation rates identify it. Nail fold capillaries have abnormalities such as dilated capillary loops, bushy capillaries, or a dropout of capillaries. Secondary RP is usually caused by an underlying condition and is associated with diverse physical symptoms. Usually, practitioners find evidence of rheumatic disease, especially systemic sclerosis, systemic lupus erythematosus, Sjorgren's syndrome or dermatomyositis. Investigators sometimes find evidence of vascular disease or diabetes mellitus. Other common causes of secondary RP may include vasculitis, repetitive trauma as in vibration syndrome, endocrine conditions including hypothyroidism, carcinoid syndrome, pheochromocytoma, and side effects of certain medications such as β -blockers, chemotherapeutic drugs, estrogen, narcotic analgesics, nicotine, and sympathomimetic drugs (Medical College of Wisconsin, 1999; Canadian Centre for Occupational Health and Safety, 2004).

Mondelli et al. (2000) conducted a study of upper limb nerve conduction velocity (NCV) in a sample of 39 subjects with primary RP, and 18 patients who had RP secondary to systemic sclerosis. They were compared with a control group of 42 healthy patients. The research team found that mean distal sensory conduction velocities of the median nerve were significantly lower in both RP groups when compared to the control group. In addition, median distal motor latency scores were higher in both patient groups in comparison to the controls. The secondary RP group had mean distal conduction scores of the ulnar nerve that were significantly slower than the primary RP group and the control group. The authors concluded that those patients with primary RP had a slowing of conduction of the distal part of the median nerve along the carpal tunnel. Those patients with secondary RP had slowing of the median and ulnar NCVs related to sub-clinical distal peripheral neuropathy. The findings suggest that among persons who have primary RP, the slowing of the conduction may affect only the median nerve, whereas those with secondary RP may have involvement of additional nerves in the hand.

The purpose of this experiment is to examine NCVs and latencies using three patient groups, and to extend the research finding of Mondelli et al. (2000) to study the relationship of NCVs and latencies in the upper extremities with the NCVs and latencies in the lower extremities. Previous research (Juntunen et al., 1983; Sakakibara et al.,

1988, 1991; Hirata et al., 1989, 1995) has demonstrated the effects VIWFS in the lower extremity. However, further research is needed to study the nerve conductivity of the lower extremity in patients with RP. Nerve conduction studies will be performed in the tibial motor, peroneal motor, peroneal sensory, and sural nerves.

The general research hypothesis of this study is that patients with RP (both primary and secondary) will have decreased NCVs and prolonged latencies when compared to normal controls. Specifically:

- (1) Patients with primary RP will have decreased NCVs and prolonged latencies on the peroneal motor, tibial motor, superficial peroneal sensory, and sural nerves when compared to normal controls.
- (2) Patients with secondary RP will have decreased NCVs and prolonged latencies on the peroneal motor, tibial motor, superficial peroneal sensory, and sural nerves when compared to persons with primary RP and normal controls.

Methods

Participants

Sixty patients were assigned to one of three groups with 20 participants in each group: the primary RP and secondary RP groups were clinically diagnosed by a rheumatologist. A control group was comprised of 20 normal individuals who had not been diagnosed with any form of RP. Thirty-eight (63.3%) of the participants were female and 22 (36.7%) were male. No significant gender differences were found among the three groups ($\chi^2 = 0.57$, ns). The mean age of the participants was 38.2 years (SD = 8.46). No significant differences were found among the three groups on age ($F_{[2, 57]} = 0.81$, ns).

The rheumatologist who referred these patients made a specific diagnosis of either primary RP or RP secondary to another condition. Certain specific inclusion and exclusion factors also were considered. Patients with entrapment syndromes, polyneuropathies or radiculopathies or other conditions (such as local edema) that may influence the NCV were excluded as well as any patients taking any medications that may influence the conduction velocities. General contraindications for NCV testing include the following: areas where active motion is contraindicated, patients wearing demand-inhibited cardiac pacemakers, stimulation directly over superficial metal implants, active

bleeding in the area to be tested, malignancies in the area to be tested, and disoriented patients. Patients who exhibited any of these contraindications were excluded from the study. All study participants were outpatients.

Prior to any NCV testing, patients were required to sign an informed consent document. Since this research project included human subjects, prior to any NCV testing, proper approval was obtained from an IRB committee.

NCV technique

NCV testing measures the speed of conduction of a nerve impulse along the course of a peripheral nerve. The procedure uses an electroneuromyograph that is equipped with an oscilloscope, amplifier, and a stimulator. All patients were tested using a Cadwell Sierra II¹ electromyography unit. All equipment was calibrated using the manufacturer's calibration procedure. All patients were positioned and tested according to the NIOSH standards manual.

The test was performed in a room maintained at a standard room temperature of approximately 22 °C. The patients' core body temperature was within the normal range of 37 °C measured orally. Initially, the patient was educated and instructed on the testing procedure. Both skin sensation and integrity were examined. Next, the patient was draped and positioned appropriately in order to remove slack from the nerve and allow for approximation of its true length. Once the site of reference and the muscle was determined, the ground electrode was placed on a bony prominence between the stimulating and recording electrodes.

The electromyograph was set at a frequency response of 10–10,000 Hz. The gain was set for 1000–5000 μ V per division, and the sweep for 2–5 μ s per division. The stimulator was set to produce a direct current stimulus of 0.1 μ s, at a rate of 1 s⁻¹. On the computer monitor, a stimulus artifact appeared at the moment of stimulation. The onset of the evoked compound action potential was recorded, followed by the measure of the latency from the stimulus artifact to the onset of the response. The time was recorded in milliseconds as Latency 1, and the site where the stimulating cathode was placed was marked with a skin pencil. Subsequently, the procedure was repeated in order to attain a second latency, which was found proximal to Latency 1, and a second stimulation site was marked. Ultimately, the equipment was

¹Cadwell Laboratories, 909 N. Kellogg St., Kennewick, WA 99336, www.cadwell.com.

removed, followed by an assessment of the subject's skin integrity, and the test was repeated on the subject's contralateral extremity. Calculations were performed in order to determine conduction velocities between the two stimulation cathode sites.

Right and left lower extremities were tested on each patient. For each measure, both the right and left values were averaged to create one mean value per patient. Distal latency (DL) and NCV were assessed for each of the four nerves. The dependent variables were tested for approximation to a normal distribution on each nerve using the Kolmogorov–Smirnov test. All eight assessments were nonsignificant ($p > .05$), indicating that all approximated a normal distribution and were therefore able to be used as dependent variables in analysis of variance models.

Results

The hypotheses of the study were assessed using multivariate analyses of variance. Descriptive statistics by group on DL and NCV are reported in Table 1; summaries of the analyses of variance are in Table 2. Paired comparisons using Fischer's least significant difference (LSD) method are presented in Table 3.

For the peroneal motor nerve, significant multivariate differences between groups were found (Wilks' $\lambda = .62$, $p < .01$). Between-group differences were found for DL ($F_{[2, 57]} = 7.62$, $p < .01$) and NCV ($F_{[2, 57]} = 15.03$, $p < .01$). Between-group differences accounted for 21% of the variance in DL and 35% of the variance in NCV. LSD post hoc comparisons indicated significant differences between the Reynaud's Secondary RP group and the

primary RP and normal control group on all three measures. Descriptive data show that the mean DL for the secondary RP group was 5.18 ms (SD = 0.67), which was significantly higher than for the primary RP group ($M = 4.41$ ms, SD = 0.75) and the control group ($M = 4.62$ ms, SD = 0.48). NCV was significantly lower for the secondary RP group ($M = 45.15$ m/s, SD = 5.38) when compared to the primary RP ($M = 50.63$ m/s, SD = 2.82) and control ($M = 51.28$ m/s, SD = 2.90) groups. No significant differences were found between the primary and control groups on either variable for the peroneal motor nerve.

For the tibial motor nerve, significant multivariate differences were found between groups (Wilks' $\lambda = .37$, $p < .01$). Between-group differences were found for DL ($F_{[2, 57]} = 15.22$, $p < .01$) and NCV ($F_{[2, 57]} = 37.21$, $p < .01$). Between-group differences accounted for 33% of the variance in DL and 55% of the variance in NCV. For DL, LSD post hoc comparisons indicated that the primary RP group was the slowest ($M = 5.91$ ms, SD = 0.95), which was significantly slower ($p < .01$) than both the secondary RP ($M = 5.08$ ms, SD = 0.88) and control ($M = 4.48$ ms, SD = 0.61) groups. In addition, the secondary RP group had significantly slower DLs than the control group ($p < .05$). For NCV, the primary RP group ($M = 41.73$ m/s, SD = 6.41) was significantly weaker than both the secondary RP ($M = 45.18$ m/s, SD = 4.85, $p < .05$) and control groups ($M = 55.11$ m/s, SD = 3.61, $p < .01$). In addition, the secondary RP group was significantly weaker than the normal controls ($p < .01$).

For the peroneal sensory nerve, significant multivariate differences were found between groups (Wilks' $\lambda = .84$, $p < .05$). No significant between-group differences were found in DL for the peroneal sensory nerve; however, significant differences were found in NCV ($F_{[2, 57]} = 3.95$, $p < .05$),

Table 1 Descriptive statistics by group and nerve.

Group	Nerve								
	Peroneal motor		Tibial motor		Peroneal sensory		Sural sensory		N
	M	SD	M	SD	M	SD	M	SD	
<i>DL (msec.)</i>									
Primary	4.41	0.75	5.91	0.95	3.09	0.45	2.94	0.53	20
Secondary	5.18	0.67	5.08	0.88	3.25	0.47	3.38	0.54	20
Normal controls	4.62	0.48	4.48	0.61	3.15	0.46	3.07	0.61	20
<i>NCV (m/sec.)</i>									
Primary	50.63	2.82	41.73	6.41	45.19	6.70	48.31	8.45	20
Secondary	45.15	5.38	45.18	4.85	40.99	5.17	42.10	8.66	20
Normal controls	51.28	2.90	55.11	3.61	46.32	6.94	46.04	7.61	20

Table 2 Summary of analyses of variance of group by nerve ($N = 60$).

Nerve	Source	SS	df	MS	F
<i>Peroneal Motor</i>					
DL (msec)	Between Groups	6.27	2	3.13	7.62**
	Within Groups	23.45	57	0.41	
	Total	29.71	59		
NCV (m/sec)	Between Groups	453.97	2	226.99	15.03**
	Within Groups	860.69	57	15.10	
	Total	1314.66	59		
<i>Tibial Motor</i>					
DL (msec)	Between Groups	20.68	2	10.34	15.22**
	Within Groups	38.72	57	0.68	
	Total	59.41	59		
NCV (m/sec)	Between Groups	1928.32	2	964.16	37.21**
	Within Groups	1476.88	57	25.91	
	Total	3405.20	59		
<i>Peroneal Sensory</i>					
DL (msec)	Between Groups	0.27	2	0.13	0.63
	Within Groups	12.06	57	0.21	
	Total	12.32	59		
NCV (m/sec)	Between Groups	315.05	2	157.53	3.95*
	Within Groups	2275.42	57	39.92	
	Total	2590.47	59		
<i>Sural Sensory</i>					
DL (msec)	Between Groups	2.03	2	1.01	3.24*
	Within Groups	17.83	57	0.31	
	Total	19.86	59		
NCV (m/sec)	Between Groups	395.38	2	197.69	2.90
	Within Groups	3881.11	57	68.09	
	Total	4276.48	59		

* $p < .05$. ** $p < .01$.

accounting for 12% of the between-groups variance. LSD post hoc comparisons for NCV indicated that the secondary RP group ($M = 40.99$ m/s,

Table 3 Paired contrasts of group by nerve.

Nerve/ Measure	Groups compared	Observed difference ^a
<i>Peroneal motor</i>		
DL (msec)	Primary vs. Secondary	-0.76**
	Primary vs. Control	-0.20
NCV (m/sec)	Secondary vs. Control	0.56**
	Primary vs. Secondary	5.48**
	Primary vs. Control	-0.65
	Secondary vs. Control	-6.13**
<i>Tibial motor</i>		
DL (msec)	Primary vs. Secondary	0.84**
	Primary vs. Control	1.43**
NCV (m/sec)	Secondary vs. Control	0.60*
	Primary vs. Secondary	-3.45*
	Primary vs. Control	-13.37**
	Secondary vs. Control	-9.93**
<i>Peroneal sensory</i>		
NCV (m/sec)	Primary vs. Secondary	4.20*
	Primary vs. Control	-1.13
	Secondary vs. Control	-5.33**
	<i>Sural sensory</i>	
DL (msec)	Primary vs. Secondary	-0.44*
	Primary vs. Control	-0.12
	Secondary vs. Control	0.31

* $p < .05$. ** $p < .01$.^aPaired comparisons using LSD computations.

$SD = 5.17$) had slower NCV than the primary RP group ($M = 45.19$ m/s, $SD = 6.70$, $p < .05$) and the normal controls ($M = 46.32$ m/s, $SD = 6.94$, $p < .01$).

Finally, for the sural nerve, multivariate statistics were conflicting (Wilks' $\lambda = .89$, ns; Roy's largest root = .12, $p < .05$), suggesting that whatever between-group differences that exist were

small. Further analysis indicated that significant between-group differences were found in DL ($F_{12, 571} = 3.24$, $p < .05$), accounting for 10% of the between groups of variance, but not for NCV. LSD post hoc analyses revealed a significant pair-wise difference ($p < .05$) in DL between the primary RP ($M = 2.94$ ms, $SD = 0.53$) and secondary RP groups ($M = 3.38$ ms, $SD = 0.64$).

Hypothesis 1, which stated that study participants with primary RP would have decreased NCVs and prolonged latencies on the peroneal motor nerve when compared to normal controls, was not supported by the findings. For the tibial motor nerve, participants with primary RP demonstrated decreased NCVs and prolonged latencies relative to the control group. On the peroneal and sural sensory nerves, the hypothesis was not supported.

The second hypothesis, which stated that study participants with secondary RP would have decreased NCVs and prolonged latencies on peroneal motor, the tibial motor, superficial peroneal sensory, and sural nerves when compared to persons with primary RP and normal controls, was supported for only the peroneal motor nerve. Findings on the tibial motor nerve were equivocal, in that the hypothesis was supported in relationship to the normal controls, but that on both measures, the primary RP group evinced slower DLs and weaker NCVs than the secondary RP group. Hypothesis 2 was partially supported for both sensory nerves. For the peroneal sensory nerve, patients with secondary RP had weaker NCVs than patients with primary RP and normal controls. For the sural sensory nerve, patients with secondary RP evidenced slower DLs than patients with primary RP.

4. Discussion

With respect to primary RP, the hypothesis of this was partially supported. Significant differences were found in DLs and NCVs on the tibial motor nerve when compared to normal subjects. These findings correlate with those found in the upper extremity study by [Mondelli et al. \(2000\)](#). They found that patients with primary RP only had abnormal nerve conduction problems in the median nerve along the carpal tunnel. This study's findings correlate with [Mondelli and associates'](#) findings. The tibial motor nerve was found to have significant abnormal nerve conduction in the area of the tarsal tunnel. With primary RP, there is no known underlying cause. It is known that in primary RP, NCVs slow on the median and tibial motor nerves in the areas of the carpal and tarsal tunnels. However, there is no known reason for this slowing to exist.

The possibility of connective tissue thickening in these tunnels cannot be excluded.

Secondary RP was hypothesized to involve all nerves in the lower extremities due to the systemic factors of the disease process. Findings on the peroneal motor and tibial motor nerves tend to support that hypothesis. However, the data are more equivocal on the sensory nerves. No significant differences were found on DLs with the peroneal sensory nerve, although NCVs were weaker than the primary RP and controls, which supports the hypothesis. On the sural sensory nerve, patients with secondary RP had slower DLs than those with primary RP.

First, both the sural and superficial peroneal nerves are both sensory, whereas the other two are motor nerves. In the lower extremity, secondary RP primarily affected the motor nerves. There is no known specific reason for this. Patients with secondary RP experience slowing of tibial and peroneal NCVs, possibly related to subclinical distal peripheral neuropathy. [Mondelli and associates \(2000\)](#) found that Secondary RP slowed NCVs of the median and ulnar nerves. The findings in combination of those of [Mondelli](#) may indicate that patients with Secondary RP may have subclinical distal peripheral polyneuropathy.

Second, both the peroneal motor nerve and the tibial motor nerve pass under a retinaculum at the ankle. The peroneal motor nerve passes under the extensor retinaculum, and the tibial motor nerve passes under the flexor retinaculum at the tarsal tunnel. Secondary RP has an underlying disease process. For example, a patient with scleroderma shows a systemic thickening of connective tissue. This thickening may occur in the area of the retinacula at the ankle and may cause compression of the peroneal motor and tibial motor nerves as they pass under the retinacula. This may have contributed to the abnormal NCVs found in this study.

Third, both the peroneal motor nerve and the tibial motor nerve travel with major arteries throughout their course down the lower extremity. The peroneal motor nerve travels with the anterior tibial artery/dorsalis pedis artery, and the tibial motor nerve travels with the posterior tibial artery. The superficial peroneal sensory nerve and the sural nerve do not travel with arteries during their course down the lower extremity. It is possible that vasospasm along the artery during a Raynaud's attack may cause decreased blood flow to the peroneal motor and tibial motor nerves, resulting in decreased nutrition for those nerve cells. It is also possible that local immune responses to the areas of vasospasm may cause damage to these

nerves, which are in close proximity to the artery. It is speculated that the underlying disease process is the cause of vasospasm, ischemia and the immune response, which causes abnormal nerve conduction. It is known that the vasospasm that occurs with RP is caused by an abnormal sympathetic response to temperature changes in the extremities (Medical College of Wisconsin, 1999; Canadian Centre for Occupational Health and Safety, 2004). This sympathetic response travels to the nerves via circulation and neural links. From this, it is also speculated that the underlying disease process causes problems along the nerves, which would then cause vasospasm. A third possibility is that both processes may be occurring simultaneously, and each would affect and worsen the other.

Fourth, the stage of the Raynaud's disease process may affect which nerves are affected by secondary RP. It is well understood that as most disease processes enter their latter stages, the body's systems begin to fail. Decreases in both nerve conductivity and blood flow will be magnified in Raynaud's patients. Therefore, future researchers may want to examine patients with a new onset of RP as compared to patients with a chronic history of RP. Research may focus on the degree to which the nerves are affected, or on which nerves may become affected.

Clinical solutions and correlates

As mentioned above, one of the main reasons for the electrodiagnostic findings and patient symptomatology can be a thickening of the retinacular structures, especially in cases of secondary RP with associated scleroderma. In such cases an entrapment neuropathy can take place. Neural mobilization can be an effective therapeutic approach for the treatment of entrapment neuropathies and thus applicable to patients with a diagnosis of primary or secondary RP (Kostopoulos, 2004). Neurodynamic testing of the corresponding nerve should take place before the actual treatment procedure is applied.

Should neurodynamic testing elicit positive results, then neural mobilization can be useful. A neurodynamic test can be considered positive if:

- (1) It produces the patient's symptoms of pain, numbness or tingling.
- (2) There is asymmetry when testing right and left sides (limitation in range of motion, resistance in the movement, production of symptoms during movement).

- (3) Test responses altered by movement of distant body parts (Butler, 2000; Kostopoulos, 2004).

Within the framework of neural mobilization are two different treatment approaches (Kleinrensink, 1995):

- (1) Mobilization of the nerve as an anatomical structure. This consists of "stretching" the nerve. The goal in this instance is to increase the flexibility of the collagen that maintains the integrity of the nerves (i.e., perineurium and epineurium).
- (2) Mobilization of the nerve in relation to its surrounding tissues. The goal in this case is to mobilize the nerve through its surrounding tissues and free up any adhesions formed by connective tissue.

In the cases of primary and especially secondary RP one of the suspected etiologies of neuropathic pain is the existence of adhesions around the various retinacula and thus creation of entrapment syndromes. Therefore, the main goal of neural mobilization in cases of RP should be to move the nerve through these narrow passages. Exerting simultaneous tension from both ends of the nerve will not result in a movement of this nerve in relation to the structures forming the narrow passageway at the site of the entrapment. In clinical terms the neural mobilization "flossing" technique would be the most applicable in this case.

Movement sequence for lower limb neurodynamic testing

(Hengeveld, Banks et al. 2005)

Superficial and deep peroneal nerve (Figure 1):

Foot inversion,
plantarflexion,
flexion of the toes,
knee in extension,
hip flexion,
may include head bias.

Posterior tibial nerve (Figure 2):

abduction of hind-foot,
dorsiflexion,
knee in extension,
hip flexion,
may include head bias.



Figure 1 Neural mobilization of the peroneal nerve.



Figure 2 Neural mobilization of the tibial nerve.

Sural nerve (Figure 3):
adduction of hind-foot,
dorsiflexion,
knee in extension,
hip flexion,
may include head bias.

The suggested protocol of the researchers of this paper includes three sets of 10 repetitions each set

for each treatment technique. Each repetition includes a distal stretch with a proximal slack followed by a distal slack with a proximal stretch. Complete and simultaneous proximal and distal stretch should be avoided for cases of entrapment neuropathies (Kleinrensink 1995). Further studies need to establish both symptomatic and electrophysiological changes with the application of neural mobilization techniques in patients with primary and secondary RP.



Figure 3 Neural mobilization of the sural nerve.

Clinical summary

Primary Raynaud's phenomenon

- Median motor and sensory affected,
- tibial nerve affected,
- neural mobilization of median and tibial nerves as indicated.

Secondary Raynaud's phenomenon

- Median motor and sensory affected,
- ulnar motor and sensory affected,
- peroneal and superficial peroneal affected,
- tibial nerve affected,
- sural nerve affected,

- neural mobilization of median, ulnar, peroneal, tibial and sural as indicated.

References

- Block, J.A., Sequeria, W., 2001. Raynaud's phenomenon. *The Lancet* 357 (9273), 2042–2048.
- Brand, F.N., Larson, M.G., et al., 1997. The occurrence of Raynaud's phenomenon in the general population: the Framingham study. *Vascular Medicine* 2 (4), 296–301.
- Butler, D.S., 2000. *The Sensitive Nervous System*. Noigroup Publications, Adelaide, Australia.
- Canadian Centre for Occupational Health and Safety, 2004. What is Raynaud's phenomenon? Retrieved 17 January, 2002, from <<http://www.ccohs.ca/oshanswers/diseases/raynaud.html>>.
- Hengeveld, E., Banks, K., et al., 2005. *Maitland's Peripherals and Manipulation*. Elsevier, Butterworth, Heinemann, New York.
- Hirata, M., Sakakibara, H., et al., 1989. Medial plantar nerve conduction velocities among patients with vibration syndrome due to chain-saw work. *International Archives of Occupational and Environmental Health* 72 (8), 551–554.
- Hirata, M., Sakakibara, H., et al., 1995. Nerve conduction velocities in the lower extremities among patients with vibration syndrome. *Central European Journal of Public Health* 3 (Supp.), 78–80.
- Juntunen, J., Matikainen, E., et al., 1983. Peripheral neuropathy and vibration syndrome. *International Archives of Occupational and Environmental Health* 52, 17–24.
- Kleinrensink, J.G., 1995. Mechanical tension in the median nerve—the effects of joint positions. *Clinical Biomechanics* 10 (5), 240–244.
- Kostopoulos, D., 2004. Treatment of carpal tunnel syndrome: a review of the nonsurgical approaches with emphasis on neural mobilization. *Journal of Bodywork and Movement Therapies* 8, 2–8.
- Medical College of Wisconsin, 1999. Raynaud's phenomenon. Retrieved 17 January 2002, from <<http://healthlink.mcw.edu/article/926055412.html>>.
- Mondelli, M., Romano, C., et al., 2000. Near conduction velocity study of the upper limit in Raynaud's phenomenon. *Rheumatology International* 19, 165–169.
- Sakakibara, H., Akamatsu, Y., et al., 1988. Correlation between vibration-induced white finger and symptoms of upper and lower extremities in vibration syndrome. *International Archives of Occupational and Environmental Health* 60, 285–289.
- Sakakibara, H., Hashiguchi, T., et al., 1991. Circulatory disturbances of the foot in vibration syndrome. *International Archives of Occupational and Environmental Health* 63, 145–148.
- Wrigley, F.M., 1998. Treating Raynaud's phenomenon and its underlying causes. *Journal of Musculoskeletal Medicine* 15 (1), 53–61.