International Journal of Neuroscience, 119:1105–1117, 2009 Copyright © 2009 Informa Healthcare USA, Inc. ISSN: 0020-7454 / 1543-5245 online DOI: 10.1080/00207450902834884

EFFECTS OF MANUAL LYMPH DRAINAGE ON CARDIAC AUTONOMIC TONE IN HEALTHY SUBJECTS

SUNG-JOONG KIM

Department of Physical Therapy Kangwon National University Kangwon-do, 245-711 Republic of Korea

OH-YUN KWON CHUNG-HWI YI

Department of Physical Therapy Yonsei University Wonju Kangwon-do, 220-710 Republic of Korea

This study was designed to investigate the effects of manual lymph drainage on the cardiac autonomic tone. Thirty-two healthy male subjects were randomly assigned to manual lymph drainage (MLD) (experimental) and rest (control) groups. Electrocardiogram (ECG) parameters were recorded with bipolar electrocardiography using standard limb lead positions. The pressure-pain threshold (PPT) was quantitatively measured using an algometer. Heart rate variability differed significantly between the experimental and control groups (p < 0.05), but the PPT in the upper trapezius muscle did not (p > 0.05). These findings indicate that the application of MLD was effective in reducing the activity of the sympathetic nervous system.

Address correspondence to Chung-Hwi Yi, PhD., P.T. Department of Physical Therapy, College of Health Science, Yonsei University Wonju, Kangwon-do, 220-710, Republic of Korea. E-mail: pteagle@yonsei.ac.kr



1105

Keywords heart rate variability, manual lymph drainage, autonomic tone, lymphedema, autonomic nervous system, stress

INTRODUCTION

Lymphedema arises when an intrinsic fault develops within the lymphconducting pathways (primary lymphedema) or when damage occurs from one or more factors originating outside the lymphatic system, such as surgical removal of a lymph node (secondary lymphedema) (Brennan, DePompolo, & Garden, 1996; Olszewski, 1991; Smeltzer, Stickler, & Schirger, 1985). Although the exact pathogenesis of lymphedema often is unclear, in most cases it is due to failure of the lymph pump (Mortimer, 1998).

Lymphedema is a common and troublesome problem: The cosmetic deformity can not be disguised with normal clothing; physical discomfort and extremities disability are associated with enlargement and recurrent episodes of cellulitis. Because of theses physical symptoms and its nonlethal nature, patients may experience distress (Petrek, Pressman, & Smith, 2000).

The method of choice for the treatment of lymphedema is complex decongestive physiotherapy (CDP), which includes manual lymph drainage (MLD) (Kasseroller, 1998). MLD affects the treatment of edema and acts on the body in various ways (Chikly, 2001), especially a calming effect on the sympathetic hyperactivity that results from daily stress, the environment, and other factors (Wittlinger & Wittlinger, 1998). This means that the appropriate application of MLD to patients who are in a state of disharmony, in which the sympathetic system dominates, makes them calmer and more relaxed, with MLD having a tonic effect on the smooth muscle of blood and lymph vessels that possess numerous nerve endings with connections to the autonomic nervous system (ANS) (Foldi & Foldi, 1993; Kasseroller, 1998; Weissleder & Schuchhardt, 2001; Wittlinger & Wittlinger, 1998). That is, MLD is one of the therapies useful for treating lymphedema patients with stress and also severe diseases such as myocardial infarction, panic disorder, and congestive heart failure that result from autonomic-function imbalance (Goldstein, Robertson, Esler, Straus, & Eisenhofer, 2002; Lombardi et al., 1987). However, but no previous study has investigated the impact of MLD on ANS.

Body functions will return to normal after a transiently applied stress, allowing the body to refocus on healing and growth. But prolongation of stress due to chronic lymphedema can cause distress throughout life (Petrek & Heelan, 1998). In this situation the sympathetic nervous system (SNS) arousal is never "turned off," which makes the individual more vulnerable to infection (Selye, 1978). Therefore, therapeutic interventions should aim at decreasing the SNS activity and increase the parasympathetic nervous system (PNS) activity.

In recent years noninvasive and sensitive indirect techniques have used heart rate variability (HRV) as a marker of changes in the activity of the ANS (Kay, 1987; Pagani & Malliani, 2000; Stein, Bosner, Kleiger, & Conger, 1994; Zefferino et al., 2003; Zhong, Jan, Ju, & Chon, 2006). Analysis of HRV can be used to identify cardiac autonomic disturbances (Kleiger, Miller, Bigger, & Moss, 1987; Pagani et al., 1988) and assess changes in sympathovagal tone in various psychological (Carney et al., 1995) and emotional (McCraty, Atkinson, Tiller, Rein, & Watkins, 1995; Rechlin, Weis, Spitzer, & Kaschka, 1994) states.

We are not aware of any reports on the effects of MLD on ANS using power spectrum analysis (PSA). In this study we investigated the effects of MLD on the cardiac autonomic tone and evaluated the pressure-pain threshold (PPT) as an index of psychological outcomes of the treatment.

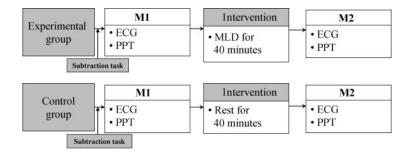
METHODS

Participants

The sample comprised of 32 normal, healthy male volunteers of Young-Dong University, Republic of Korea. The participants were randomly assigned to an experimental group (17 subjects) and a control group (15 subjects). Participants were screened for inclusion criteria. The inclusion criteria were (1) normal skin with no preexisting skin conditions, (2) no history of treatment for thrombosis, (3) no compromised skin, such as irritation, excessive dryness, known presence of eczema or psoriasis, or visible adverse conditions (rash or infection), and (4) no taking any prescription drugs known to affect the cardiovascular system and pain. Ethical committee agreement and written informed consent were obtained prior to testing.

Protocol

Subjects were allowed to rest comfortably for at least 5 min prior to the baseline recording procedure. Brief psychological stress (Kuriyama et al., 2005) was induced to change the sympathetic activity by performing a serial subtraction task for 2 min. Electrocardiogram (ECG) was performed for 5 min, and PPT data were acquired prior to performing MLD. Subjects in the experimental group received MLD to the neck and abdomen for 40 min, while the subjects in the control group relaxed by lying quietly for the same time period. Data acquisition and MLD took place in a quiet temperature-controlled environment



M1 = measurement before treatment, M2 = measurement after treatment, ECG = electrocardiography, PPT = pressure-pain threshold, MLD = manual lymph drainage

Figure 1. Experimental procedure for the determination of manual lymph drainage (MLD) effects on the heart rate variability and pressure-pain threshold (PPT).

 $(22 \sim 24)$. Conversation, phone calls, and noise that could increase the activity of the SNS were minimized, and the subject's body was covered with a soft and thin sheet so as to avoid discomfort from body exposure. ECG and PPT data were acquired immediately following MLD in the experimental group and in the control group (Figure 1).

Massage Procedure

Subjects were required to undress for MLD. Following baseline recordings, they remained supine on the massage table. In order to maintain consistency, an MLD therapist certified by Dr. Vodder's school and physical therapist with 10 years of experience in the treatment of lymphedema performed MLD by applying the same standardized procedures to the neck and abdomen. MLD procedure, which took 40 min, employed the technique as described by Dr. Vodder. The technique was applied in the areas of the neck and abdomen. MLD procedure used in this study can be found in detail in *the Textbook of Dr. Vodder's Manual Lymph Drainage* (Wittlinger & Wittlinger, 1998).

ECG Recording and Analysis

HRV was used to assess quantitatively the effect of MLD on cardiac autonomic tone, since this is an objective index of the emotional responses to stimuli (Pomeranz et al., 1985; Schumacher, 2004; Task force of the European Society of Cardiology and the North American Society of Pacing

and Electrophysiology, 1996). Heart rate data were acquired with bipolar electrocardiography using standard limb lead positions for 5 min at baseline and for 5 min immediately following MLD in a supine position. The ECG electrodes were connected to a four-channel monitoring system (QECG-3, LAXTHA, Korea) that satisfied the standard of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. A notebook computer was used for controlling the experiments and storing the output data.

The ECG signals were digitized by a 12-bit analog-to-digital converter at 256 Hz and processed on a personal computer with a software peak detection algorithm that located the R waves. This consisted of an automatic default filtering procedure contained within the TeleScan software package (LAXTHA) and also a careful inspection of the R-R interval tachogram. The tachogram was visually scanned to ensure the complete removal of abnormal data.

The assessed time-domain indices were the mean heart rate, R-R interval, the standard deviation of normal-to-normal (SDNN) cardiac interbeat (R-R) intervals, the root mean square of successive differences (RMSSD), and pNN50 (%), and the frequency-domain indices were the total power, low frequency (LF) power, high frequency (HF) power, and the LF/HF ratio. Data were computed off-line using TeleScan software.

Measuring the PPT

A pressure algometer was used (Algometer Commander, JTECH Medical, USA) to determine the changes in muscle tension associated with changes in ANS. Before the measurements, the subjects were asked to relax in a chair in an upright position as the investigator explained the procedure to them. To determine a baseline value, the investigator applied pressure to the upper trapezius muscle on the dominant side (which has received considerable attention in clinical fields due to the high prevalence of work-related shoulder and neck disorders (Madeleine, Farina, Merletti, & Arendt-Nielsen, 2000; Maeda, 1977; Schnoz, Laubli, & Krueger, 2000) until the subject experienced that the sensation of pressure changed to pain. The pressure was increased at a constant rate of approximately 0.5 kg/s. The recording was stopped immediately when the subject reported the sensation change. The entire procedure was repeated three times, with the mean value calculated for each muscle. Following MLD being performed for 40 min, the PPT of the upper trapezius muscle was immediately reassessed in the same manner as described above.

Statistical Analysis

Data are given as mean $\pm SD$ values. All variables were tested for normality using the one-sample Kolmogorov–Smirnov test and did have a normal distribution. An unpaired <u>t</u>-test was used to test for homogeneity and to compare the MLD and control groups for differences in HRV. The collected data were analyzed using a statistical package program (SPSS ver. 12.0). A two-tailed probability of p < 0.05 was considered statistically significant.

RESULTS

Demographics

The general characteristics and the time- and frequency-domain parameters did not differ statistically between the two groups at baseline (p > 0.05, Table 1).

ECG Measures

The postintervention R-R interval, SDNN, RMSSD, and pNN50 (%) value were significantly higher in the experimental group than in the control group (Table 2) (p < 0.05). Moreover, the heart rate was significantly reduced in the experimental group, as indicated by an increase in the average R-R interval length (p < 0.05). The postintervention total power and LF/HF ratio differed significantly between the groups (p < 0.05), with the latter being significantly lower in the MLD group.

PPT Measures

Pain threshold responses to treatment were assessed as changes in muscle tension as a result of changes to the ANS according to pressure algometer results. The mean ($\pm SD$) PPT were 24.56 \pm 5.11 for the experimental group and 22.45 \pm 3.80 for the control group. There were no significant differences between the groups after intervention (p > 0.05).

DISCUSSION

Effects of MLD on HRV

The PSA of HRV has recently been used as a sensitive index of autonomic nervous activity that provides quantitative information on autonomic control

Evennemental	Control		
		t	р
group(n=17)	group $(n = 15)$	i	P
21.16 ± 1.24	20.95 ± 1.37	0.46	0.65
176.10 ± 2.80	175.99 ± 2.43	0.11	0.91
73.78 ± 4.61	74.51 ± 3.963	-0.48	0.63
21.98 ± 4.44	22.37 ± 3.58	-0.27	0.79
72.71 ± 1.39	72.77 ± 2.03	-0.11	0.91
834.29 ± 40.69	827.75 ± 17.58	0.58	0.57
62.30 ± 2.77	60.50 ± 4.65	1.35	0.19
38.97 ± 1.43	37.47 ± 9.35	0.65	0.519
5.94 ± 0.50	5.93 ± 0.88	0.06	0.954
2912.59 ± 344.63	2824.20 ± 317.44	0.75	0.45
1512.94 ± 157.52	1555.87 ± 136.91	-0.82	0.42
633.47 ± 162.60	656.53 ± 173.61	-0.39	0.70
2.81 ± 0.39	3.01 ± 0.51	-1.32	0.19
	176.10 ± 2.80 73.78 ± 4.61 21.98 ± 4.44 72.71 ± 1.39 834.29 ± 40.69 62.30 ± 2.77 38.97 ± 1.43 5.94 ± 0.50 2912.59 ± 344.63 1512.94 ± 157.52 633.47 ± 162.60	rgroup $(n = 17)$ group $(n = 15)$ 21.16 ± 1.2420.95 ± 1.37176.10 ± 2.80175.99 ± 2.4373.78 ± 4.6174.51 ± 3.96321.98 ± 4.4422.37 ± 3.5872.71 ± 1.3972.77 ± 2.03834.29 ± 40.69827.75 ± 17.5862.30 ± 2.7760.50 ± 4.6538.97 ± 1.4337.47 ± 9.355.94 ± 0.505.93 ± 0.882912.59 ± 344.632824.20 ± 317.441512.94 ± 157.522824.20 ± 317.44633.47 ± 162.60656.53 ± 173.61	$rac{n}{group} (n = 17)$ $rac{n}{group} (n = 15)$ t 21.16 ± 1.24 20.95 ± 1.37 0.46 176.10 ± 2.80 175.99 ± 2.43 0.11 73.78 ± 4.61 74.51 ± 3.963 -0.48 21.98 ± 4.44 22.37 ± 3.58 -0.27 72.71 ± 1.39 72.77 ± 2.03 -0.11 834.29 ± 40.69 827.75 ± 17.58 0.58 62.30 ± 2.77 60.50 ± 4.65 1.35 38.97 ± 1.43 37.47 ± 9.35 0.65 5.94 ± 0.50 5.93 ± 0.88 0.06 2912.59 ± 344.63 2824.20 ± 317.44 0.75 1512.94 ± 157.52 1555.87 ± 136.91 -0.82 633.47 ± 162.60 656.53 ± 173.61 -0.39

 Table 1. Homogeneity test for general characteristics and time- and frequency-domain parameters of the subjects at baseline

mechanisms (Malliani, Pagani, Lombardi, & Cerutti, 1991; Pagani et al., 1986; Pomeranz et al., 1985; Task force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). This is the first study to examine the effects of MLD using HRV analysis to measure cardiac autonomic tone in healthy college students. The results of the study suggest that MLD for 40 min is highly effective in increasing HRV and cardiac parasympathetic activity in normal subjects, as demonstrated in the time domain by a decrease in heart rate and increases in SDNN and RMSSD, and in the frequency domain by an increase in the total power and a decrease in the LF/HF ratio.

	Experimental	Control group $(n = 15)$		р
Variables	Experimental group $(n = 17)$		t	
Heart rate (beats/min)	68.39 ± 2.11	71.15 ± 1.45	4.24	0.00
R-R interval (ms)	879.88 ± 51.41	830.35 ± 42.07	2.96	0.01
Standard deviation of	70.72 ± 7.52	65.02 ± 4.74	2.53	0.02
interbeat intervals				
(ms)				
Root mean square of	50.58 ± 6.31	38.77 ± 10.90	3.81	0.00
successive				
differences				
(ms)				
pNN50 (%)	7.54 ± 1.27	5.93 ± 0.98	3.98	0.00
Frequency-domain				
parameters				
Total power (ms ²)	3771.47 ± 538.37	3168.07 ± 443.94	3.43	0.00
Low-frequency power	1844.00 ± 279.50	1817.20 ± 300.77	0.26	0.79
(ms ²)				
High-frequency power	1459.18 ± 258.74	1268.73 ± 222.43	2.22	0.03
(ms ²)				
Low-frequency to	1.28 ± 0.17	1.46 ± 0.28	-2.28	0.03
high-frequency				
ratio				

 Table 2. Postintervention time- and frequency-domain results in the experimental and control groups

A decrease in the heart rate may be considered a sign of relaxation (without knowledge of the exact underlying mechanisms). An increase in parasympathetic activity or a decrease in sympathetic activity would decrease the heart rate. Lee, Kim, Song, Park, and Moon, 2004 have demonstrated that physical and psychological stresses can reduce the heart rate during experimental measurements. The results from this and previous studies show that physical stimuli such as MLD can induce relaxation through stabilization of the autonomic system.

It is generally accepted that the power of HF oscillations (between 0.15 and 0.4 Hz) is an indirect marker of efferent parasympathetic (vagal) input to the heart, and that the power of LF oscillations is generally accepted as a marker of SNS activity associated with components around 0.1 Hz corresponding to blood-pressure control oscillations known as Mayer waves (Benson, Beary, & Carol, 1974; Kay, 1987; Stein et al., 1994; Sztajzel, 2004). The LF/HF ratio

reflects the global sympathovagal balance (Pagani & Malliani, 2000). In this study MLD reduced the LF/HF ratio (p < 0.05), which again suggests a shift toward cardiac parasympathetic activity. An increase in PNS activity has been associated with the relaxation response (Benson et al., 1974).

Harker, Egekvist, and Bjerring, 2000 reported that the LF and HF powers increased following sensory stimulation by acupuncture. Delaney, Leong, Watkins, and Brodie, 2002 also reported a significant increase in HF power following trigger-point massage. The SNS and PNS responses to sensory stimulation in healthy persons may vary with the site of stimulation.

Effects of MLD on PPT

MLD may be able to decrease sympathetic responses and increase parasympathetic responses in the neuromuscular system (sympathicolytic action). By stimulating parasympathetic tone, MLD can cause relaxation, antispastic, and antalgic effects. Light rhythmic stimulation such as MLD of the skin nonnociceptive receptors may have a pain-inhibiting effect (Pagani et al., 1988). In the gate control theory of pain, Wall and Melzack, 1994 explain how nonnoxious sensory stimulation can help to reduce the intensity of pain.

Many clinical applications of the algometer have been documented, including evaluation (Fischer, 1988) and identification of trigger points, and evaluation of pain sensitivity (Fischer, 1987). The PPT of a muscle is the pressure level at which the patient reports that the feeling of pressure changes into a painful sensation (Fischer, 1987).

Not only has massage such as MLD to be used in this study recently become popular as part of the conservative treatment, but it also received empirical support for reducing pain and alleviating stress, depression, and anxiety in the context of various somatic and psychiatric disorders. Massage also has been described as positively affecting neurovegetative functions (e.g., lowering blood pressure and cardiac muscle) (Field, 1998; Field et al., 1996).

Autonomic nerves extend to all parts of the skin; blood vessels, lymphatic vessels, and soft connective tissue ground substance. Signals from the special senses acting via the limbic system and hypothalamus influence the responses of the ANS (Ebner, 1985; Wittlinger & Wittlinger, 1998). Although the PPT are not differed significantly between the experimental and the control groups, there was an improvement in pain threshold following MLD. These findings are probably due to an increased relaxation response and an overall reduction in the defense-arousal (stress) responses, and are possibly mediated by increased parasympathetic activity.

The present study shows that MLD is a safe and noninvasive technique that is effective in inducing relaxation in normal subjects. It is a relatively simple technique to learn, and under correct instruction could be taught to the caregivers of patients who could benefit from MLD. Whilst it is imprudent to generalize from a healthy population to those with disease, certain conditions are characterized by increased sympathetic tone and reduced HRV, and these conditions might show a relaxation response following MLD that may benefit the sympathovagal balance.

In conclusion, the data presented here suggest that MLD is highly effective in increasing HRV and cardiac parasympathetic activity in normal subjects, as demonstrated in the time domain by a decrease in heart rate and increases in SDNN, RMSSD, and pNN50 (%) and in the frequency domain by an increase in the total power and a decrease in the LF/HF ratio. These results suggest that MLD could help reduce stress and improve autonomic function by increasing HRV and parasympathetic activity.

REFERENCES

- Benson, H., Beary, J. F., & Carol, M. P. (1974). The relaxation response. *Psychiatry*, 37, 37–46.
- Brennan, M. J., DePompolo, R. W., & Garden, F. H. (1996). Focused review: Postmastectomy lymphedema. Archives of Physical Medicine and Rehabilitation, 77, s74–s80.
- Carney, R. M., Saunders, R. D., Freedland, K. E., Stein, P., Rich, M. W., & Jaffe, A. S. (1995). Association of depression with reduced heart rate variability in coronary artery disease. *The American Journal of Cardiology*, 76(8), 562–564.
- Chikly, B. (2001). *Theory of Practice of Lymph Drainage Therapy*. Arizona: International Health & Healing.
- Delaney, J. P., Leong, K. S., Watkins, A., & Brodie, D. (2002). The short-term effects of myofascial trigger point massage therapy on cardiac autonomic tone in healthy subjects. *Journal of Advanced Nursing*, 37(4), 364–371.
- Ebner, M. (1985). Connective Tissue Manipulation: Theory and Therapeutic Application. Florida: Krieger Pub Co.
- Field, T. M. (1998). Massage therapy effects. *The American Psychologist*, 53(12), 1270–1281.
- Field, T., Ironson, G., Scafidi, F., Nawrocki, T., Goncalves, A., Burman, I. et al. (1996). Massage therapy reduces anxiety and enhances EEG pattern of alertness and math computations. *The International Journal of Neuroscience*, 86(3–4), 197–205.
- Fischer, A. A. (1987). Pressure algometry over normal muscles. Standard values, validity, and reproducibility of pressure threshold. *Pain*, 30, 115–126.

RIGHTSLINK

- Fischer, A. A. (1988). Documentation of myofascial trigger points. *Archives of Physical Medicine and Rehabilitation*, 69(4), 286–291.
- Foldi, M., & Foldi, E. (1993). Lymphoedema: Methods of Treatment and Control. Australia: Lymphoedema Association of Victoria.
- Goldstein, D. S., Robertson, D., Esler, M., Straus, S. E., & Eisenhofer, G. (2002). Dysautonomias: Clinical disorders of the autonomic nervous system. *Annals of Internal Medicine*, 137(9), 753–763.
- Harker, E., Egekvist, H., & Bjerring, P. (2000). Effect of sensory stimulation (acupuncture) on sympathetic and parasympathetic activities in healthy subjects. *Journal of the Autonomic Nervous System*, 79(1), 52–59.
- Kasseroller, R. G. (1998). The Vodder school: The Vodder method. *Cancer*, 83, 2840–2842.
- Kay, S. M. (1987). Modern Spectral Estimation: Theory and Application. N.J.: Prentice-Hall Englewood Cliffs.
- Kleiger, R. E., Miller, J. P., Bigger, J. T., Jr., & Moss, A. J. (1987). Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. *The American Journal of Cardiology*, 59(4), 256–262.
- Kuriyama, H., Watanabe, S., Nakaya, T., Shiqemori, I., Kita, M., Yoshida, N. et al. (2005). Immunological and psychological benefits of aromatherapy massage. *Evidence-Based Complementary and Alternative Medicine*, 2(2), 179–184.
- Lee, M. S., Kim, H. J., Song, J., Park, K. W., & Moon, S. R. (2004). Effects of multifunctional fabrics on cardiac autonomic tone and psychological state. *The International Journal of Neuroscience*, 114(8), 923–931.
- Lombardi, F., Sandrone, G., Pernpruner, S., Sala, R., Garimoldi, M., Cerutti, S. et al. (1987). Heart rate variability as an index of sympathovagal interaction after acute myocardial infarction. *The American Journal of Cardiology*, 60(16), 1239–1245.
- Madeleine, P., Farina, D., Merletti, R., & Arendt-Nielsen, L. (2000). Upper trapezius muscle mechanomyographic and electromyographic activity in humans during low force fatigue and non-fatiguing contrations. *European Journal of Applied Physiology*, 87(4–5), 327–336.
- Maeda, K. (1977). Occupational cervicobrachial disorder and its causative factors. Journal of Human Ergology, 6(2), 193–202.
- Malliani, A., Pagani, M., Lombardi, F., & Cerutti, S. (1991). Cardiovascular neural regulation explored in the frequency domain. *Circulation*, 84(2), 482–492.
- McCraty, R., Atkinson, M., Tiller, W. A., Rein, G., & Watkins, A. D. (1995). The effects of emotions on short-term power spectrum analysis of heart rate variability. *The American Journal of Cardiology*, 76(14), 1089–1093.
- Mortimer, P. S. (1998). The pathophysiology of lymphedema. Cancer, 83, 2798-2802.
- Olszewski, W. L. (1991). Lymph Stasis: Pathophysiology, Diagnosis and Treatment. Florida: CRC Press.
- Pagani, M., Lombardi, F., Guzzetti, S., Rimoldi, O., Furlan, R., Pizzinelli, P. et al. (1986). Power spectral analysis of heart rate and arterial pressure variabilities as



a marker of symphatho-vagal interaction in man and conscious dog. *Circulation Research*, 59(2), 178–193.

- Pagani, M., & Malliani, A. (2000). Interpreting oscillations of muscle sympathetic nerve activity and heart rate variability. *Journal of Hypertension*, 18(12), 1709–1719.
- Pagani, M., Malfatto, G., Pierini, S., Casati, R., Masu, A. M., Poli, M. et al. (1988). Spectral analysis of heart rate variability in the assessment of autonomic diabetic neuropathy. *Journal of the Autonomic Nervous System*, 23(2), 143–153.
- Petrek, J. A., Pressman, P. I., & Smith, R. A. (2000). Lymphedema: Current issues in research and management. CA: A Cancer Journal for Clinicians, 50(5), 292–307.
- Petrek, J. A., & Heelan, M. C. (1998). Incidence of breast carcinoma-related lymphedema. *Cancer*, 83, 2776–2781.
- Pomeranz, B., Macaulay, R. J., Caudill, M. A., Kutz, I., Adam, D., Gordon, D. et al. (1985). Assessment of autonomic function in humans by heart rate spectral analysis. *The American Journal of Physiology*, 248(1 Pt 2), H151–H153.
- Rechlin, T., Weis, M., Spitzer, A., & Kaschka, W. P. (1994). Are affective disorders associated with alterations of heart rate variability? *Journal of Affective Disorders*, 32(4), 271–275.
- Schnoz, M., Laubli, T., & Krueger, H. (2000). Co-activation of the trapezius and upper arm muscle with finger tapping at different rates and trunk postures. *European Journal of Applied Physiology*, 83(2–3), 207–214.
- Schumacher, A. (2004). Linear and nonlinear approaches to the analysis of R-R interval variability. *Biological Research for Nursing*, 5(3), 2115–221.
- Smeltzer, D. M., Stickler, G. B., & Schirger, A. (1985). Primary lymphedema in children and adolescents: A follow-up study and review. *Pediatrics*, 76(2), 206–218.
- Stein, P. K., Bosner, M. S., Kleiger, R. E., & Conger, B. M. (1994). Heart rate variability: A measure of cardiac autonomic tone. *American Heart Journal*, 127(5), 1376–1381.
- Selye, H. (1978). The Stress of Life. New York: McGraw-Hill.
- Sztajzel, J. (2004). Heart rate variability: A noninvasive electrocardiographic method to measure the autonomic nervous system. *Swiss Medical Weekly*, 134(35–36), 514–522.
- Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability: standards of measurement, physiological interpretation, and clinical use. *Circulation*, 93, 1043–1065.
- Wittlinger, H., & Wittlinger, G. (1998). *Textbook of Dr. Vodder's Manual Lymph Drainage*. Heidelberg: Haug.
- Wall, P. D., & Melzack, R. (1994). Textbook of Pain. Edinburgh: Churchill Livingstone.
- Weissleder, H., & Schuchhardt, C. (2001). Lymphedeam: Diagnosis and Therapy. Koln: Viavital Verlag GmbH.
- Zefferino, R., L'Abbate, N., Facciorusso, A., Potenza, A., Lasalvia, M., Nuzzaco, A. et al. (2003). Assessment of heart rate variability (HRV) as a stress index in

RIGHTSLINK()

an emergency team of urban police. *Giornale italiano di medicina del lavoro ed ergonomia*, 25, 167–169.

Zhong, Y., Jan, K. M., Ju, K. H., & Chon, K. H. (2006). Quantifying cardiac sympathetic and parasympathetic nervous activities using principal dynamic modes analysis of heart rate variability. *American Journal of Physiology. Heart and Circulatory Physiology*, 291(3), H1475–H1483.

RIGHTSLINK