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Original contribution

Cranial rhythmic impulse related to the Traube-Hering-Mayer oscillation: comparing laser-Doppler flowmetry and palpation

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The primary respiratory mechanism (PRM) as manifested by the cranial rhythmic impulse (CRI), a fundamental concept to cranial osteopathy, and the Traube-Hering-Mayer (THM) oscillation bear a striking resemblance to one another. Because of this, the authors developed a protocol to simultaneously measure both phenomena. Statistical comparisons demonstrated that the CRI is palpably concomitant with the low-frequency fluctuations of the THM oscillation as measured with the Transonic Systems BLF 21 Perfusion Monitor laser-Doppler flowmeter. This opens new potential explanations for the basic theoretical concepts of the physiologic mechanism of the PRM/CRI and cranial therapy. Comparison of the PRM/CRI with current understanding of the physiology of the THM oscillation is therefore warranted. Additionally, the recognition that these phenomena can be simultaneously monitored and recorded creates a new opportunity for further research into what is distinctive about the science and practice of osteopathic medicine.

(Key words: osteopathic medicine, cranial osteopathy, primary respiratory mechanism, cranial rhythmic impulse, cardiovascular system, lymphatic system, laser-Doppler flowmetry, Traube-Hering-Mayer oscillation)

In 1865, Traube[1] reported the measurement of a fluctuation in pulse pressure with the frequency of respiration that persisted after respiration had been arrested. These findings were corroborated by Hering [2] in 1869. Independently, Mayer[3] identified similar oscillations with a slower rate in 1876. These phenomena, now known collectively as the Traube-Hering-Mayer (THM) oscillation,[4] have been measured in association with blood pressure,[1,4-10] heart rate, [4,10,11] cardiac contractility,[12] pulmonary blood flow,[13] cerebral blood flow and movement of the cerebrospinal fluid,[14,15] and peripheral blood flow, including venous volume and thermal regulation.[4,8,10,16] This whole-body phenomenon, which exhibits a rate typically slightly less than and independent of respiration, bears a striking resemblance to the primary respiratory mechanism (PRM).

The PRM was first described by Sutherland[17] in 1939. In 1961, Woods[18] coined the term *cranial rhythmic impulse* (CRI) to describe the palpable sensation of the PRM. The PRM is considered to be integrally linked to cellular metabolism. Magoun[19] stated: "This cycle manifests as the cranial rhythmic impulse and represents a dynamic metabolic interchange in every cell, with each phase of action." The PRM affects all regions of the body.[19-24] The CRI, as a manifestation of the PRM, is most evident to palpation in the head. It is, however, palpable in every part of the body.[24] It is described in terms of amplitude and rate, with most authors identifying the normal rate as 10 to 14 cycles per minute.[18,19,21,23,24] The PRM/CRI, though frequently synchronous with respiration, has a periodicity independent of the cardiac and respiratory cycles.[18,20,24-26]

The PRM/CRI is a decidedly controversial aspect of osteopathic medicine. It is a subtle enough phenomenon to be easily overlooked by untrained clinicians. This has led many to doubt its existence and incorrectly state that it has not been measured. Many authors,[18,20,26-30] however, have reported measuring the CRI.

Fryman,[20] Upledger and Vredevoogd,[22] Geiger,[31] and MacPartland and Mein[31a] have commented on the similarity between the THM and the CRI. Fernandez and Lecine[28] attempted to measure both phenomena simultaneously but did not demonstrate a statistically significant relationship.

We developed a protocol to measure the THM and the PRM/CRI simultaneously.

Methods

Subjects

Twelve healthy subjects over 18 years of age (6 males; 6 females, none pregnant) were recruited from the faculty and students of the Chicago College of Osteopathic Medicine (Midwestern University, Downers Grove, Ill). All signed IRB-approved informed consent forms.

Conditions and protocol

Cranial examinations were performed by one of the authors (N.S.) in sequence in 1 day in a quiet (essential for instrumentation) small office, with low-level illumination provided by incandescent lighting and an ambient temperature of approximately 20°C. Prior to each examination, an adhesive Doppler probe was placed onto the left earlobe of each sub-

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ject. Following this, each subject lay quietly on the examination table for approximately 5 minutes prior to the onset of data acquisition. It was essential that the probe and leads were free of tension so that earlobe blood flow was not compromised. Laser-Doppler flowmetry provided measurement of the relative velocities of blood (hemoglobin) while simultaneously, the examiner, at the head of the table, palpated the CRI using light touch with the hands in a biparietal-hold position. Both examiner and subject were blinded to activities at the computer screen and keyboard.

A 2-minute-long equilibration-period record was recorded. Following the equilibration period, the examiner would, using a subdued voice, enunciate "f" at the instant a cranial flexion event began, and a second investigator, operating the computer console in the examination room, would depress a key entering the first event mark into the digitized flowmetry record. Thereafter, serial extension ("e") and flexion ("f") events were marked into the record. This method of recording could introduce a 150-millisecond reaction-time delay into the CRI record, 300 milliseconds if the reaction times of both the examiner and the recording investigator are considered.[32] Continuous, unbroken records of approximately 5 minutes' duration were recorded for each subject.

Laser-Doppler flowmetry

The perfusion monitor (Transonic Systems Inc, Ithaca, NY) determines the Doppler velocity change of the erythrocyte (hemoglobin) in circulating blood, and that information is digitized for subsequent data reduction. The device uses an optic fiber probe that rests on the skin surface, which causes no discomfort to the subject. The probe (type R) has two optic fibers: one sends laser light into the tissue, the other transfers the light reflected from those tissues to a photo detector for subsequent electronic processing (WinDaq Data Acquisition and Playback Software, also from Transonic Systems).

Data reduction and statistics

The acquisition software was able to export both the time-domain and frequency-domain data in database format, permitting further data processing as required. Palpatory and flowmetry time-domain data were compared using the paired samples *t*-test statistic (2-tailed significance),[33] with statistical power determined by the method of Dupont and Plummer.[34] Comparisons of mean THM frequencies among subjects were evaluated using the one-way analysis of variance in conjunction with the Bonferroni range test for pair-wise comparisons (significance, $P < .05$).[33]

Results

Combined laser-Doppler flowmetry and CRI records: qualitative overview

Figure 1 presents two different subject records that display all of the features observed in the 12 records of this study. The oscillating waveforms are records of the flowmetry relative blood velocity changes detected at the earlobe (recorded as an output voltage); the vertical event marks denote the flexion and extension events detected through palpation (entered by technician). In the 7-minute period presented in *Figure 1*, the high-frequency oscillations of the Doppler record, such as those arising from the heartbeat, are compressed and appear as spectral noise so that the low-frequency oscillations may be observed easily. The earliest event mark (extreme left mark) of each recorded segment denotes a flexion event. Thereafter, the event marks denote alternate extension and flexion throughout the sequence.

In *Figure 2* (top), a portion of subject 2's record is expanded along the time axis to reveal the fine structure of the cardiac cycle and the relationship of this cycle to the THM oscillation. Three THM cycles are displayed. The amplitudes of the THM waves are nearly double those of cardiac cycle waves. The THM cycle corresponds to large relative changes in blood velocity and, therefore, is of a magnitude that ought to be readily palpable through osteopathic techniques, particularly if these blood velocity changes are proportional to blood volume changes (compare with Burch and colleagues[35]).

Laser-Doppler detection produces blood velocity records with considerably more detail than could be obtained with earlier technologies, permitting the observation of signal fine-structure and more precise signal quantification. At the time scale presented in *Figure 1*, several features are apparent: (1) The record oscillates at a regular frequency of approximately 0.1 cycles/sec (6 cycles/min). (2) The amplitude of the signal is not constant but instead oscillates between relatively higher and lower values, giving the overall record the appearance of "waves upon waves." (3) The waveform exhibits nodes where the signal is suppressed or greatly distorted. These nodes represent beat notes caused by the destructive interference of multiple, overlapping component signals. Constructive interference gives rise to signals of high amplitude. (4) Because of interference effects, the phase of the THM signal changes with time. Thus, it is not possible to relate, for example, a flexion event as always being associated with a flowmetry maximum. After a signal inversion, flexion would correspond to a Doppler minimum. What can be determined and correlated with the palpatory findings is the periodic rate, defined by the positions in time of the maxima and minima. Wave apices (maxima/minima) correspond to time points where the change in blood velocity passes through zero (*Figure 2*). (5) The frequency of the oscillation is not constant but, as will be demonstrated later, varies with time in a regular fashion. [The peak-to-peak variation in time is apparent in the record of subject 9 (*Figure 1*).] Signal variation with time indicates frequency modulation among interacting signals[36] and implies the presence of a capacitance in the physiologic mechanism responsible for blood velocity oscillation.[8,16]

The event-mark sequences of *Figures 1 and 2* exhibit the following features: (1) The sequence mimics the slow-wave [0.1 cycles/sec (6 cycles/min)] Traube-Hering component of the Doppler record. For virtually all of the recorded events, the event marks correspond in time to either maxima or minima in the Doppler record. (2) Event marks do not appear for each maximum or minimum; rather, the event marks appear to correspond to each full

oscillatory cycle of a maximum plus a minimum. That is, event marks denote adjacent maxima or minima. (3) Although event marks generally mark maxima or minima, there are instances when the mark falls between these. Thus, in all of the records examined, the correspondence of marks and signal apices exhibits some irregularity. (4) At nodes in the Doppler record, the CRI still can be palpated, with the regularity of the event record usually being maintained and with no palpable change in CRI amplitude. (5) As with the Doppler record, the frequency of event marks varies with time in a regular fashion, and these variations correspond with those of the Doppler record.

Laser-Doppler power spectrum: qualitative overview

Figure 3 presents the power spectrum of a 13-minute flowmetry record segment using an input-averaging value of 10. The power spectrum is computed from time-domain data (energy vs. time), such as presented in *Figures 1 and 2*, through a Fourier transformation to generate a frequency-domain spectrum (energy vs. frequency). The spectrum of *Figure 3* reveals the component signals that are combined in the original time-domain Doppler record. The frequencies of individual signals are indicated on the abscissa, while the relative signal amplitudes (areas) are related to the contribution each signal makes to the original time-domain record. Signals at frequencies greater than approximately 0.5 cycles/sec (30 cycles/min) are attributable to the pulse (cardiac cycle) and will not be considered further here. These signals give rise to the rapid beats seen in *Figures 1 and 2*.

The low-frequency components that give rise to the THM oscillation in the Doppler record lie in the frequency range of 0 to 0.5 cycles/sec (30 cycles/min). For reference, the signal at 0.32 cycles/sec (19 cycles/min) has been assigned to the respiration rate,[4,37-41] while the signal at 0.02 cycles/sec (1.2 cycles/min) has been attributed by various authors to the thermal regulatory component.[8,16,42,43] Of interest to this study is the high-amplitude signal at 0.10 cycles/sec (6 cycles/min) whose origins are not fully understood,[1-4,44] but

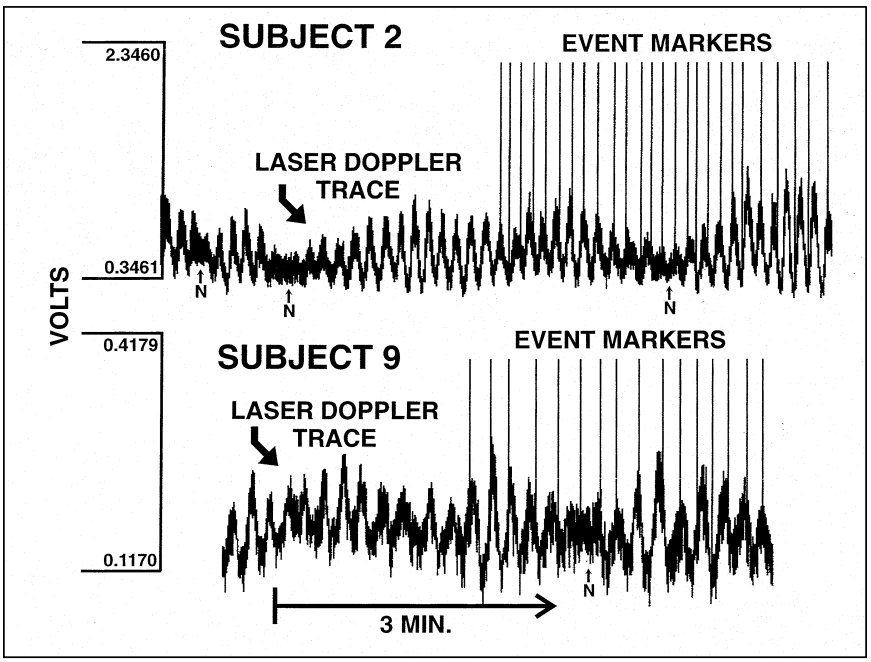

Figure 1. *Compressed laser-Doppler flowmeter relative blood velocity (waveform) and flexion-extension records (vertical event marks) from two subjects (N, interference nodes).*

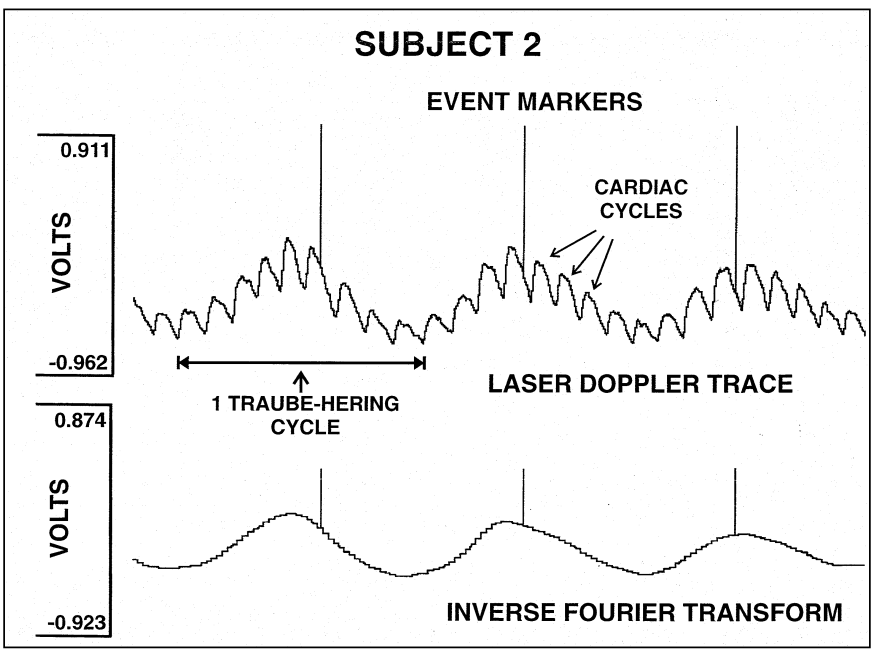

Figure 2. *Expanded laser-Doppler flowmeter relative blood velocity record of subject 2.* Top—*Flowmeter trace, revealing fine structure of the cardiac cycle. The double-headed arrow indicates the wavelength of one Traube-Hering-Mayer cycle.* Bottom—*Traube-Hering-Mayer wave form component only of the top trace. The bottom waveform was created from the top waveform by filtering (removing) the high-frequency cardiac component, leaving only the low-frequency baro and respiratory components. Inverse Fourier transformation of this filtered data generates the bottom trace. Both traces are in register with respect to time, and the event markers indicate the positions of the palpatory findings.*

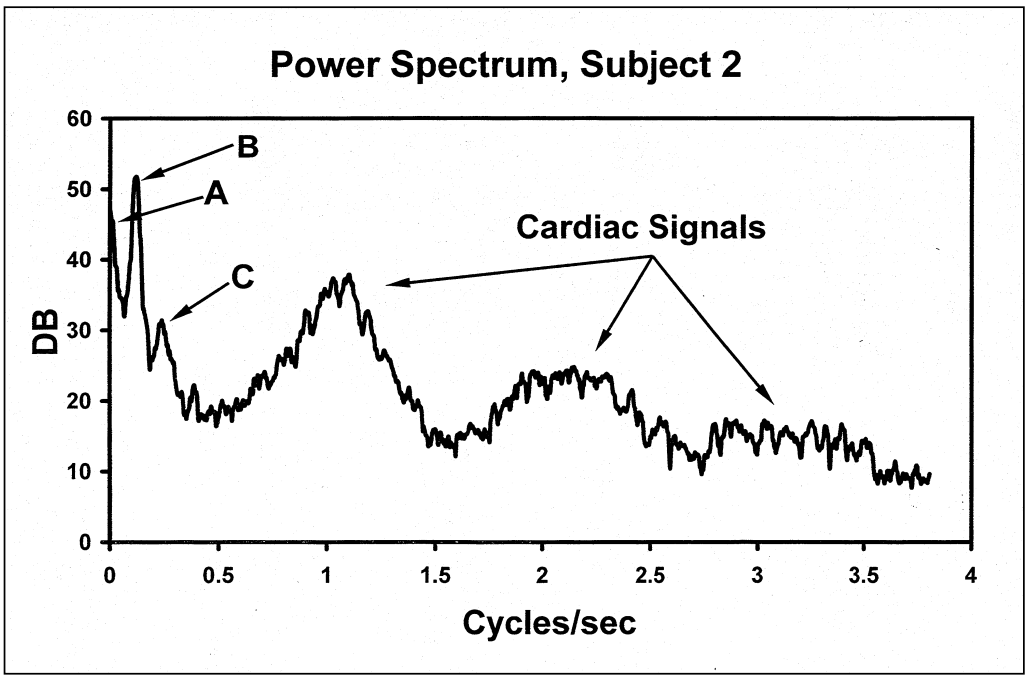

Figure 3. *Power spectrum of the laser-Doppler flowmeter record presented in* Figure 1 *subject 2.* **A**, *thermal signal.* **B,** *baro signal.* **C,** *respiration.*

which has been assigned to baroreceptor function.[45] The signal amplitudes shown in *Figure 3* indicate that these three signals comprise the principal components of the slow-wave oscillations observed in the original flowmetry time-domain record. This is illustrated in *Figure 4* (bottom), which is an inverse Fourier transform spectrum of the data of *Figure 3* after high-frequency filtering. This filtering process removed those components of the original record that were above 0.5 cycles/sec (30 cycles/min).

Minor signals also are observed in the power spectra that have not been assigned. Of particular note is a signal at 0.39 cycles/sec (23.4 cycles/min). Oscillations in this frequency range have been attributed to lymphatic vasomotion.[46-52] Some of the minor signals undoubtedly represent harmonics of the major signals; others may represent fundamental physiologic functions. Each, however, will contribute added complexity to the time-domain record.

Combined record: quantification of time-domain data

Descriptive statistics. Twelve subjects participated in the study. Of these, 11 provided high-quality data for analysis. For subject 12, the signal-to-noise ratio observed in the laser-Doppler (time-domain) output was too low for precise quantitative measurement. However, the Fourier transform (frequency-domain) record of subject 12 included all of the features observed for the other 11 subjects.

Recorded frequencies for maxima and minima were distributed uniformly among the 11 test records (n = 613; mean, 56; range, 39 to 77). There were 166 flexion events and 162 extensions (n = 328) associated equally between maxima (n = 164) and minima (n = 164), with no correlation between the occurrence of a maximum or minimum and the palpation of a flexion or extension event (Pearson's R value, −0.085; approx. sig., 0.123).

Laser-Doppler flowmetry compared with palpation. The time at which a maximum or minimum occurred in the flowmetry record was compared with the time recorded for the nearest flexion or extension event. For almost all flexion or extension events, association of the event with the nearest maximum or minimum presented no difficulty (*Figure 2*). For 2% of events, signal assignment was problematic because the flexion/extension event mark fell either approximately midway between maxima or minima or because the signal-to-noise ratio at that point was insufficient for precise maximum or minimum location. It was possible, however, to resolve all assignment ambiguities through the use of spectral filtering and expansion routines.

Considering all flexion or extension events recorded for the 11 subjects with high-quality data, and their corresponding flowmetry maxima or minima, the two groups are the same. By the paired *t*-test there was no statistical difference between the time values recorded for palpated flexion or extension events and the corresponding flowmetry maxima or minima (N = 328; mean difference between pairs, flowmetry time-palpation time, −0.078; SD, 1.361; 2-tailed sig., 0.303).

As may be anticipated from *Figures 1 and 2*, both groups of time values were highly correlated (N = 328 data pairs; correlation, 1.000; sig., 0.000). The statistical comparison was repeated after introducing a 150-millisecond reaction time delay for the technician into the flexion/extension record. This computation caused a slight convergence of the two means with a change in sign of the mean difference (mean difference between pairs, 0.072; SD, 1.361; 2-tailed sig., 0.336). The computed statistical power[34] was 0.527 (alpha, 0.336; difference, 0.0724; sigma, 1.3614; N = 328). The addition of a second 150-millisecond reaction time delay for the examiner (300 millisecond total introduced delay), however, caused

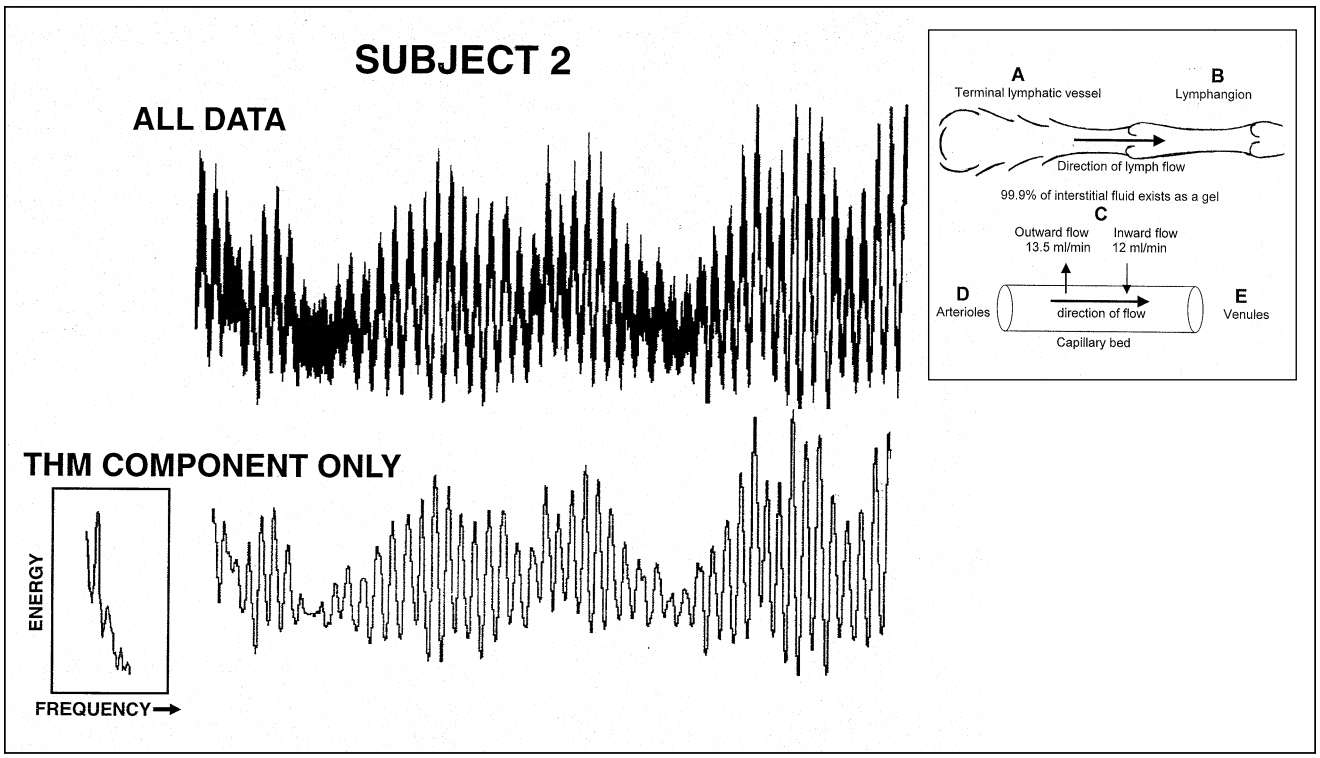

Figure 4. *Inverse Fourier transform time-domain spectra from subject 2; top spectrum, all frequency domain data used in the inverse computation; bottom spectrum, only the frequency component lying below 0.5 cycles/sec (30 cycles/min) used. The bottom spectrum is the THM oscillation. The insert box shows that portion of the Fourier transform spectrum used to compute the inverse THM spectrum (see* Figure 3*).*

Table 1
Descriptive Statistics (11 Subjects) for the Relative Blood Velocity Oscillation Determined by Laser-Doppler Flowmetry

Subject No.	No. of cycles	Mean cycle period (sec/cycle)	Standard deviation	Standard error	95% Confidence interval (mean)		Minimum	Maximum
					Lower bound	Upper bound		
1	57	10.20	1.89	0.25	9.69	10.70	4.80	14.88
2	55	8.59	0.93	0.12	8.34	8.85	6.36	12.00
3	45	10.87	2.19	0.32	10.21	11.53	7.20	14.40
5	55	9.97	2.22	0.29	9.37	10.57	5.08	16.00
6	46	9.94	2.05	0.30	9.32	10.55	7.70	16.80
7	45	13.47	1.38	0.20	13.06	13.89	11.52	16.64
8	66	7.59	1.04	0.12	7.33	7.85	5.60	10.18
9	53	12.03	1.74	0.23	11.55	12.51	7.04	17.80
10	37	11.75	2.05	0.33	11.06	12.43	8.00	16.20
11	75	9.63	1.84	0.21	9.21	10.06	6.24	13.44
12	57	11.67	2.46	0.32	11.01	12.32	5.12	17.92
Total	591	10.35	2.42	0.099	10.15	10.54	4.80	17.92

Table 2
Multiple Comparisons of the Traube-Hering-Mayer Component of the Relative Blood Velocity Oscillation Laser-Doppler Record (sec/cycle; One-way Analysis of Variance, Range Test: Bonferroni)

(I) Subject No.	(J) Subject No.	Mean difference (I-J)	Standard error	Significance	95% Confidence interval	
					Lower bound	Upper bound
1	2	1.60*	0.349	0.000	0.44	2.76
	3	-0.67	0.368	1.000	-1.89	0.55
	5	0.22	0.349	1.000	-0.93	1.39
	6	0.26	0.366	1.000	-0.95	1.48
	7	-3.27*	0.368	0.000	-4.50	-2.05
	8	2.60*	0.334	0.000	1.49	3.71
	9	-1.83*	0.352	0.000	-3.01	-0.66
	10	-1.55*	0.390	0.004	-2.85	-0.25
	11	0.56	0.324	1.000	-0.51	1.64
	12	-1.47*	0.346	0.001	-2.62	-0.32
2	3	-2.27*	0.371	0.000	-3.51	-1.03
	5	-1.37*	0.352	0.006	-2.54	-0.20
	6	-1.34*	0.369	0.016	-2.57	-0.11
	7	-4.88*	0.371	0.000	-6.11	-3.64
	8	1.00	0.337	0.168	-0.12	2.12
	9	-3.44*	0.355	0.000	-4.62	-2.25
	10	-3.15*	0.392	0.000	-4.46	-1.84
	11	-1.03	0.328	0.088	-2.13	0.053
	12	-3.07*	0.349	0.000	-4.23	-1.91
3	5	0.89	0.371	0.858	-0.33	2.13
	6	0.93	0.387	0.895	-0.35	2.22
	7	-2.60*	0.389	0.000	-3.90	-1.30
	8	3.27*	0.357	0.000	2.08	4.46
	9	-1.16	0.374	0.105	-2.41	0.081
	10	-0.88	0.410	1.000	-2.24	0.48
	11	1.23*	0.348	0.023	0.076	2.39
	12	-0.80	0.368	1.000	-2.02	0.42
5	6	0.032	0.369	1.000	-1.19	1.26
	7	-3.50*	0.371	0.000	-4.74	-2.26
	8	2.37*	0.337	0.000	1.25	3.50
	9	-2.06*	0.355	0.000	-3.25	-0.88
	10	-1.78*	0.392	0.000	-3.08	-0.47
	11	0.33	0.328	1.000	-0.76	1.42
	12	-1.70*	0.349	0.000	-2.86	-0.53

*Mean difference is significant at the .05 level.

Table 2 (continued)
Multiple Comparisons of the Traube-Hering-Mayer Component of the Relative Blood Velocity Oscillation Laser-Doppler Record (sec/cycle; One-way Analysis of Variance, Range Test: Bonferroni)

(I) Subject No.	(J) Subject No.	Mean difference (I-J)	Standard error	Significance	95% Confidence interval	
					Lower bound	Upper bound
6	7	-3.53*	0.387	0.000	-4.82	-2.24
	8	2.34*	0.354	0.000	1.16	3.52
	9	-2.09*	0.372	0.000	-3.33	-0.85
	10	-1.81*	0.408	0.001	-3.17	-0.45
	11	0.30	0.346	1.000	-0.84	1.45
	12	-1.73*	0.366	0.000	-2.95	-0.51
7	8	5.88*	0.357	0.000	4.69	7.07
	9	1.44*	0.374	0.007	0.19	2.68
	10	1.72*	0.410	0.002	0.36	3.09
	11	3.84*	0.348	0.000	2.68	5.00
	12	1.80*	0.368	0.000	0.57	3.03
8	9	-4.44*	0.340	0.000	-5.58	-3.30
	10	-4.15*	0.379	0.000	-5.42	-2.89
	11	-2.04*	0.311	0.000	-3.07	-1.00
	12	-4.07*	0.334	0.000	-5.19	-2.96
9	10	0.28	0.395	1.000	-1.03	1.60
	11	2.40*	0.331	0.000	1.29	3.50
	12	0.36	0.352	1.000	-0.80	1.53
10	11	2.11*	0.371	0.000	0.88	3.35
	12	0.079	0.390	1.000	-1.21	1.37
11	12	-2.03*	0.324	0.000	-3.11	-0.95

*Mean difference is significant at the .05 level.

the two means to diverge significantly (mean difference between pairs, 0.222; SD, 1.361; 2-tailed sig., 0.003).

Mean frequency variance among subjects. The mean oscillatory frequencies determined by both laser-Doppler flowmetry and palpation (these are equivalent) differed among subjects even though the experimental circumstances (supine, awake, and at rest) were essentially identical for all (*Table 1*). Pair-wise comparisons of computed means (*Table 2*) also were tested for significance by the one-way analysis of variance ($P < .05$). Of the 55 possible combinations of study subject pairs, 38 (69%) differed significantly.

Frequency variability. As mentioned previously, examination of either the flowmetry or the palpation records of all subjects revealed the presence of both higher-frequency and lower-frequency segments that alternated in a periodic manner, suggesting the presence of a frequency-modulated component.[53] Moreover, these frequency changes were not related in any obvious way to analogous amplitude changes that also occurred in all records. To document the periodic frequency changes and to assess their magnitudes within and among individual subject records, two contiguous groups of approximately 10 complete cycles each, the shortest group and the longest group, were selected from each subject record. All selected sequences were separated within each subject record by at least 2 minutes. Frequencies for each cycle (in cycles/min) were computed from the time data, and pairs of short and long segments within

each subject record were compared by *t*-test for equality of means. Two examples, one from a higher-frequency (subject 8) and one from a lower-frequency (subject 11), are presented in *Table 3*. The mean differences between higher- and lower-frequency sets within each subject record are statistically significant ($P < .01$), with the high-frequency (subject 8) exhibiting a frequency difference of 1.51 cycles/min and the low-frequency (subject 11) exhibiting a difference of 1.73 cycles/min.

Comments

The statistical comparisons demonstrate that the CRI is concomitant with low-frequency fluctuations in blood velocity as measured with the Transonic Systems BLF 21 Perfusion Monitor laser-Doppler flowmeter. Moreover, the observed concomitance is sustained at all points in the record even though blood-velocity-change frequency may vary, with periodicity, as much as 20% (*Table 3*). These findings imply that the PRM/CRI and the THM oscillation are simultaneous, if not the same phenomenon. This opens new possible explanations for the basic theoretical concepts of the physiologic mechanism of the PRM/CRI and cranial therapy. Comparison of the PRM/CRI with current understanding of the physiologic mechanism of the THM oscillation is, therefore, warranted.

Primary respiratory mechanism. The PRM is described as the driving force associated with the activity of cellular metabolism.[17,19,21,24] The THM oscillations are intimately involved in the regulation of peripheral blood flow and, consequently, tissue perfusion.[4,8,10,16] Circulatory and body core temperature homeostasis are considered to be a result of the THM oscillation.[8] A hypothetical explanation for the PRM can be devised by employing our understanding of the THM oscillation.

The THM oscillation affects all tissues of the body through its impact on the entire circulatory system. Blood pumped from the heart, the rhythm of which fluctuates under the influence of the THM oscillation,[11,12] arrives in all of the capillary beds in the body via arteries and arterioles whose walls are contracting synchronously at the THM frequency.[8,10,16] Blood pressure and capillary blood velocity are consequently oscillating at the THM frequency.

Cellular metabolic exchange occurs within the interstitial space. As much as 99.9% of interstitial fluid exists in a gel-like state. Fluid cannot move as freely through this gel as it can in a purely liquid medium. The interstitial gel, however, demonstrates elasticity.[54] Something must facilitate fluid movement in the extracellular space.

The THM blood flow and pressure oscillation in the thicker-walled arterial system are less constrained as the blood passes through the capillaries and enters the venous system. The capacitance of these thin-walled veins allows for significantly greater volume fluctuations with proportionate displacement of adjacent structures. In the capillary beds, local contractile responses to distention have been demonstrated in some tissues.[5(p127)]

It has been suggested that the arteriolar and venular vasomotion and blood pressure fluctuation that result from the THM oscillation aid in the distribution and mixing of all of the extravascular fluids and may mechanically facilitate the passage of fluid through capillary and lymphatic walls.[55] Thus, local (direct) and neural (indirect) control mechanisms act synergistically to satisfy the metabolic demands of the peripheral tissues. Locally, the activity of the musculature of the vascular bed is modified and integrated by changes in the composition of the extracellular fluid. Neural control is exercised via specialized sensory endings of peripheral afferent cells within the integrative centers of the central nervous system. Response occurs to varying levels of oxygen, carbon dioxide, and hydrogen ion concentration, as well as temperature of the blood and extracellular fluid.[56] Or, as Magoun[19] proposed of the PRM, the THM oscillation facilitates "dynamic metabolic interchange in every cell, with each phase of action."

Whole-body phenomenon. The PRM/CRI and the THM oscillation share the quality of being demonstrable throughout the body. Multiple authors describe the PRM/CRI as palpable in all areas of the body.[19-24] The THM oscillations are recognized to occur throughout the body.[57] They have been measured simultaneously in the right index finger, right second toe, and pinna of the right ear.[35]

Rate of the PRM/CRI. The PRM/CRI has a generally agreed-upon rate of 0.17 to 0.23 cycles/sec (10 to 14 cycles/min).[17,18,21,23,24] Many researchers have measured the CRI, using various methods, on human subjects[18,20,25-29] and animals,[30,58] identifying rates between 0.083 and 0.23 cycles/sec (5 and 14 cycles/min).

Further, Becker[59] describes two components to the PRM/CRI. They are the "fast tide," at a rate of 0.13 to 0.20 cycles/sec (8 to 12 cycles/min), and the "slow tide," at a rate of 0.01 cycles/sec (0.6 cycles/min). Fryman[20] attempted to determine what relation might exist between peripheral volumetric changes and the rhythmic cycles of the cranium by using a pressure transducer applied to the head to monitor the CRI, in conjunction with plethysmography applied to the arm or finger. In so doing, she noted that cranial motion recordings coincided with appendicular volume changes. Plethysmography also demonstrated "long slow cycles from 50 to 60 seconds" duration, which were thought not to be related to cranial changes.[20] Upledger and Karni,[29] using plethysmography to monitor the CRI, identified a "frequency of (0.15 to 0.18 cycles/sec) 9 to 11 cpm" and an "even slower frequency of (0.02 to 0.03 cycles/sec) 1 to 2 cpm."

Within the THM oscillation, two distinct frequencies exist that are significant to this discussion. The Traube-Hering component of the THM oscillation demonstrates a frequency range of 0.09 to 0.17 cycles/sec,[8-10,16,35,60] or 5 to 10 cycles/min. The Mayer component demonstrates a frequency range from 0.01 to 0.09 cycles/sec,[9,10] or 0.6 to 5.4 cycles/min. It could be argued that a single frequency of oscillation shared between the PRM/CRI and the THM oscillation is coincidental; however, the occurrence of two distinct frequencies shared between the PRM/CRI and the THM oscillation renders coincidence a highly improbable explanation.

Table 3
Comparisons of Shorter and Longer Oscillations Within Individual Subject Records (*t*-test for Equality of Means)

Subject No.	Short or long cycles	N	Mean (sec/cycle)	Standard deviation	Mean difference	Significance (2-tailed)
8	Short	9	6.49	0.63	1.51	0.001
	Long	9	8.00	0.93		
11	Short	11	8.88	0.72	1.73	0.006
	Long	10	10.61	1.68		

Relationship between the PRM/CRI and pulmonary respiration. Pulmonary respiration has always been recognized as closely associated with, yet independent of, the PRM/CRI. The respiratory cooperation of the patient is often employed in association with cranial treatment.[17,59] Cranial manipulation has been said to affect respiration,[61] and spontaneous deep sighing respiration has been reported coincidental with the therapeutic end point.[23] As shown by the Fourier analysis (*Figures 3 and 4*), the component parts of the total THM oscillation, that is, Mayer (0.01 to 0.09 cycles/sec), Traube-Hering (0.09 to 0.17 cycles/sec), and respiration (0.2 to 0.3 cycles/sec), are distinctly separate.[4,9,10,53,62] The components at 0.01 to 0.09 cycles/sec and 0.09 to 0.17 cycles/sec, however, are closely linked to, and may be modulated by, the pulmonary respiration component at 0.2 to 0.3 cycles/ sec.[39,40]

Fluctuation of the cerebrospinal fluid and motion of the central nervous system. The PRM/CRI is described as consisting of five distinct component parts.[17,19,21-24] Of these, the fluctuation of the cerebrospinal fluid (CSF) and the inherent mobility of the central nervous system (CNS) are of particular interest in the context of the THM oscillation. Motion of the brain[63] and motion of the CSF in synchrony with the cardiac cycle has been demonstrated using magnetic resonance velocity imaging.[63,64] For a brief period during systole, there is a net inflow of blood into the brain, causing it to expand in volume. This causes the central portion of the brain and the brainstem to be displaced caudally. An amount of CSF approximating the volume change of the brain is displaced from the ventricles and intracranial subarachnoid space.

During systole, the CSF moves medially from the lateral ventricles of the cerebral cortex into the third ventricle and in a craniocaudad direction from the third ventricle into the fourth ventricle and in the subarachnoid space surrounding the spinal cord. During diastole, with the return of the caudal displacement of the brain, CSF motion reverses direction. The fourth ventricle acts as a "mixing chamber" to allow for CSF oscillation. The system must demonstrate capacitance. The spinal dural sac acts as the required capacitor.

Oscillations of cortical metabolism (9.58 cycles/min in an awake, nonanesthetized cat) with associated fluctuations in blood volume have been demonstrated by reflectance spectrophotometry of the cortical cytochrome oxidase redox state. The data[65] suggest that the cyclic increases in cortical oxidative metabolism represent the primary oscillatory process, followed by reflex hemodynamic changes that affect intracranial blood volume. Volume oscillations representing presumed THM waves have been measured in the brains of conscious healthy humans using ultrasound.[66]

As the intracranial blood volume increases, CSF is displaced from the interior of the brain case into the extracranial subarachnoid space, increasing the amount of CSF in the spinal dural sac capacitor. As intracranial blood volume decreases, the tension of the spinal dural sac facilitates the return of CSF into the skull. Because of this synchronicity with and relation to THM oscillation, the CSF could be described as "ebbing and flowing."

Entrainment and manipulation of the PRM/CRI. Manipulative treatment directed at affecting the PRM/CRI is often used to modulate the rate (frequency), amplitude, and direction of the wave.[19(p339)] Respiratory cooperation of the patient can be used in association with cranial treatment.[17,59] Spontaneous deep sighing respiration has been reported coincidental with the therapeutic end point.[23]

Entrainment of frequency occurs when two nonlinear oscillatory systems are coupled and operating at close but different frequencies.[8,16,36,45] The coupling causes the two oscillators to lock into a common frequency. The THM oscillation has been entrained using rhythmic alteration of body position,[45] exposure to fluctuating temperature,[16] and respiratory activity.[4,38,39,45] Entrainment of THM has been accomplished using baroreceptors and vasomotor reflexes; the lower limit of the entrainment bandwidth is 0.0841 (SD, 0.0030) cycles/sec, and the upper limit is 0.1176 (SD, 0.0013) cycles/sec.[49] Entrainment of the THM by the respiratory rate specifically occurs over a similar frequency range of 5 breaths/min (0.083 cycles/sec) to 7 breaths/min (0.12 cycles/sec).[40] Although cranial manipulation involves more complexity of intervention than merely modulating the PRM/CRI, the concept of oscillatory entrainment offers an interesting explanation for this one aspect of treatment, as has been proposed by MacPartland and Mein.[31a]

Conclusion

The results of this study indicate that the PRM/CRI and the THM oscillation occur

simultaneously, though they may or may not represent the exact same phenomenon. Like blood pressure, serum glucose, and all other diagnostically significant parameters, the PRM/CRI and the THM are unique among individuals (*Table 2*). While the clinical norms for the PRM/CRI have been measured, the significance of deviations from normative values currently exists only in anecdotal form. Similarly, the normative range of the THM oscillation has been measured, but little information exists as to its relevance regarding pathologic conditions. The use of laser-Doppler flowmetry provides a quantifiable, noninvasive, instrumental method for documenting normative blood velocity values and their clinical relevance. As such, it may also provide a method to study the PRM/CRI, thereby expanding our knowledge of the THM oscillation and our basic understanding of the complexity of the PRM. Laser-Doppler flowmetry also may present new opportunities to study the dynamics of cranial manipulation.

The THM oscillation may well represent one aspect of the complex clinical arena of Sutherland's discovery. It may explain the rate and rhythm of the CRI and offer insight into the physiologic mechanism of the PRM. It may represent a component of the inherent mobility of the CNS. It does not yet explain the complex patterns of motion observed when palpating the CRI, and it offers no explanation of membranous strain dysfunctions. Nonetheless, the recognition that the CRI, one aspect of the PRM, may be monitored and recorded through the observation of the THM oscillation provides an opportunity for further research into that which is distinctive in osteopathic medicine.

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References

1. Traube L. Uber periodische Tatigkeitsanderungen des Vasomotorischen und Hemmungs-Nervenzentrums. *Cbl Med Wiss* 1865;56:881-885.

2. Hering E. Uber Athembewegungen des Gefasssystems. *Sitzungb d k Akad d W math naturw* 1869;60:829-856.

3. Mayer S. Uber spontane Blutdruckschwankungen. *Sitzungb d k Akad d W math naturw* 1876;67: 281-305.

4. Penaz J. Mayer waves: history and methodology. *Automedica* 1978;2:135-141.

5. *Best and Taylor's Physiologic Basis of Medical Practice*. 12th ed. Baltimore, Md: Williams & Wilkins, 1990.

6. Bykov KM. *Textbook of Physiology*. Moscow: Foreign Languages Publishing House, 1958; 142.

7. Guyton AC. *Textbook of Physiology*. 3rd ed. Philadelphia, Pa: WB Saunders Co., 1966; 293.

8. Hyndman BW. The role of rhythms in homeostasis. *Kybernetik* 1974;15:227-236.

9. Akselrod S, Gordon D, Ubel FA, Shannon DC, Barger AC, Cohen RJ. Power spectral analysis of heart rate fluctuation: a quantitative probe of beat-to-beat cardiovascular control. *Science* 1981;213:220-221.

10. Akselrod S, Gordon D, Madwed JB, Snidman NC, Shannon DC, Cohen RJ. Hemodynamic regulation: investigation by spectral analysis. *Am J Physiol* 1985;249:H867-H875.

11. Fuller BF. The effects of stress-anxiety and coping styles on heart rate variability. *Int J Psychophysiol* 1992;12:81-86.

12. Negoescu R, Filcescu V, Boanta F, Dinca-Panaitescu S, Popovici C. Hypobaric hypoxia: dual sympathetic control in the light of RR and QT spectra. *Rom J Physiol* 1994;31:47-53.

13. Szidon JP, Cherniack NS, Fishman AP. Traube-Hering waves in the pulmonary circulation of the dog. *Science* 1969;164:75-76.

14. White DN. The early development of neurosonology: III. Pulsatile echoencephalography and Doppler techniques. *Ultrasound Med Biol* 1992;18:323-376.

15. Clarke MJ, Lin JC. Microwave sensing of increased intracranial water content. *Invest Radiol* 1983;18:245-248.

16. Kitney RI. An analysis of the nonlinear behavior of the human thermal vasomotor control system. *J Theor Biol* 1975;52:231-248.

17. Sutherland WG. *The Cranial Bowl*. Mankato, Minn: Free Press Co; 1939.

18. Woods JM, Woods RH. A physical finding relating to psychiatric disorders. *JAOA* 1961;60: 988-993.

19. Magoun HI. *Osteopathy in the Cranial Field*. 2nd ed. Kirksville, Mo: The Journal Printing Company, 1966.

20. Frymann VM. A study of the rhythmic motions of the living cranium. *JAOA* 1971;70:928-945.

21. Becker RE. Craniosacral trauma in the adult. *Osteopathic Annals* 1976;4:43-59.

22. Upledger JE, Vredevoogd JD. *Craniosacral Therapy*. Chicago, Ill: Eastland Press, 1983.

23. Sergueef N. *Le B.A.BA du crânien. Paris, France: SPEK, 1986.*

24. Lay E. Cranial field. In: Ward RC, ed. *Foundations for Osteopathic Medicine*. Baltimore, Md: Williams & Wilkins; 1997:901-913.

25. Lay EM, Cicora RA, Tettambel M. Recording of the cranial rhythmic impulse. *JAOA* 1978;78:149.

26. Wirth-Pattullo V, Hayes KW. Interrater reliability of craniosacral rate measurements and their relationship with subjects' and examiners' heart and respiratory rate measurements. *Phys Ther* 1994; 74:908-916.

27. Zanakis MF, Cebelenski RM, Dowling D, Lewandoski MA, Lauder CT, Kirchner KT, et al. The cranial kinetogram: objective quantification of cranial mobility in man. *JAOA* 1994;93:759.

28. Fernandez D, Lecine A. L'enregistrement de l'onde de Traube-Herring et de la palpation cranienne simultanee. *Kinesitherapie Scientifique* 1990; 292:33-40.

29. Upledger JE, Karni Z. Strain plethysmography and the cranial rhythm. *Proc XII Internat Conf Med Biol Eng*, Jerusalem, Israel, Aug 19-24, 1979, Part IV, p 69.

30. Michael DK, Retzlaff EW. A preliminary study of cranial bone movements in the squirrel monkey. *JAOA* 1975;74:866-869.

31. Geiger AJ. Generating dialogue concerning cranial plate motion [letter]. *JAOA* 1992;92:1088-1093.

31a. MacPartland J, Mein EA. Entrainment of the cranial rhythmic impulse. *Altern Ther Health Med* 1997;3:40-45.

32. Takagi M, Watanabe S. Two different components of contingent negative variation (CNV) and their relation to reaction time under hypobaric hypoxic conditions. *Aviat Space Environ Med* 1999;70:30-34.

33. Nie NH, Hayadlai Hull C, Jenkins JG, Steinbrenner K, Bent DH. *SPSS: Statistical Package for the Social Sciences*. 2nd ed. New York, NY: McGraw-Hill; 1975:181-202.

34. Dupont WD, Plummer Jr WD. Power and sample size calculations. A review and computer program. *Controlled Clin Trials* 1990;11:116-128.

35. Burch GE, Cohn AE, Neumann C. A study by quantitative methods of the spontaneous variations in volume of the finger tip, toe tip, and posterior-superior portion of the pinna of resting normal white adults. *Am J Physiol* 1942;136:433-447.

36. Hyndman BW, Kitney RI, Sayers BM. Spontaneous rhythms in physiological control systems. *Nature* 1971;233:339-341.

37. Dornhorst AC, Howard P, Leathart GL. Respiratory variations in blood pressure. *Circulation* 1952;6:553-558.

38. Sayers BM. Analysis of heart rate variability. *Ergonomics* 1973;16:17-32.

39. Barman SM, Gebber GL. Basis for synchronization of sympathetic and phrenic nerve discharges. *Am J Physiol* 1976;231:1601-1607.

40. Ahmed AK, Harness JB, Mearns AJ. Respiratory control of heart rate. *Eur J Appl Physiol* 1982;50:95-104.

41. Bachoo M, Polosa C. Properties of the inspiration-related activity of sympathetic preganglionic neurones of the cervical trunk in the cat. *J Physiol (London)* 1987;385:545-564.

42. Burton AC, Taylor RM. A study of the adjustment of peripheral vascular tone to the requirements of the regulation of body temperature. *Am J Physiol* 1940;129:565-577.

43. Bornmyr S, Svensson H, Lilja B, Sundkvist G. Skin temperature changes and changes in skin blood flow monitored with laser Doppler flowmetry and imaging: a methodological study in normal humans. *Clin Physiol* 1997;17:71-81.

44. Lewis T. Studies of the relationship between respiration and blood-pressure. Part II. Facts bearing on the relationship of different factors in the production of respiratory curves of blood-pressure. *J Physiol (London)* 1908;37:233-255.

45. Nasimi SG, Harness JB, Marjanovic DZ, Knight T, Mearns AJ. Periodic posture stimulation of the baroreceptors and the local vasomotor reflexes. *J Biomed Eng* 1992;11:307-312.

46. McHale NG, Roddie IC. The effect of transmural pressure on pumping activity in isolated bovine lymphatic vessels. *J Physiol* 1976;261:255-269.

47. Olszewski WL, Engeset A. Lymphatic contractions. *N Engl J Med* 1979;300:316.

48. Ohhashi T, Azuma T, Sakaguchi M. Active and passive mechanical characteristics of bovine mesenteric lymphatics. *Am J Physiol* 1980;239: H88-H95.

49. McHale NG, Roddie IC, Thornbury KD. Nervous modulation of spontaneous contractions in bovine mesenteric lymphatics. *J Physiol* 1980; 309:461-472.

50. Olszewski WL, Engeset A. Intrinsic contractility of prenodal lymph vessels and lymph flow in human leg. *Am J Physiol* 1980;239:H775-H783.

51. Mislin H. The lymphangion. In: Foldi M, Casley-Smith JR, eds. *Lymphangiology*. Stutgart, NY: F.K. Schatauer-Verlag, 1983;165-175.

52. Johnston MG, Kanalec A, Gordon JL. Effects of arachidonic acid and its cyclo-oxygenase and lipoxygenase products on lymphatic vessel contractility in vitro. *Prostaglandins* 1983;25:85-98.

53. Kobayashi M, Musha T. 1/f Fluctuation of heartbeat period. *IEEE Trans Biomed Eng* 1982;BME-29:456-457.

54. Johnston MG, ed. *Experimental Biology of the Lymphatic Circulation*. Amsterdam, The Netherlands: Elsevier; 1985.

55. Parsons RK, McMaster PD. The effect of the pulse upon the formation and flow of lymph. *J Exp Med* 1938;68:353-376.

56. Laborit H. *Stress and Cellular Function*. Philadelphia, Pa: JB Lippincott Co. 1959;19-21.

57. Burton AC, Taylor RM. A study of the adjustment of peripheral vascular tone to the regulation of body temperature. *Am J Physiol* 1940;129:565-577.

58. Retzlaff EW, Michael DK, Roppel RM. Cranial bone mobility. *JAOA* 1975;74:869-873.

59. Becker RE. *Life in Motion*. Portland, Ore: Rudra Press; 1997.

60. Chess GF, Tam RMK, Calaresu FR. Influence of cardiac neural inputs on rhythmic variations of heart period in the cat. *Am J Physiol* 1975;228:775-780.

61. Younozai R, Fryman VM, Nardell BE, Pryor MJ, Senicki M. Effects of temporal manipulation on respiration. *JAOA* 1981;80:751.

62. Barron DH. Vasomotor regulation. In: Ruch TC, Fulton JF, ed. *Medical Physiology and Biophysics*. 18th ed. Philadelphia, Pa: WB Saunders Co, 1960:691-707.

63. Enzmann DR, Pelc NJ. Normal flow patterns of intracranial and spinal cerebrospinal fluid defined with phase-contrast cine MR imaging. *Radiology* 1991;178:467-474.

64. Feinberg DA, Mark AS. Human brain motion and cerebrospinal fluid circulation demonstrated with MR velocity imaging. *Radiology* 1987;163:793-799.

65. Vern BA, Schuette WH, Leheta B, Juel VC, Radulovacki M. Low-frequency oscillations of cortical oxidative metabolism in waking and sleep. *J Cereb Blood Flow Metab* 1988;8:215-226.

66. Jenkins CO, Campbell JK, White DN. Modulation resembling Traube-Hering waves recorded in the human brain. *Eur Neurol* 1971;5:1-6.