General Internal Medicine and Clinical Innovations

Editorial

The human heart rate variability; Neurobiology of psychophysiological well being and planetary resonance

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Since the dawn of human life, the innate perception of heart beat, being the source of life and the shine of the spirit that will depart only if the heart beating cease. The study of heart beat all over the time line of medicine from Galen and before to Ibn al-Nafis, and Ibn Sina passing to William Gilbert and his primitive attempts to record heart stimuli ending to Willem Einthoven who invented the modern ECG, was always concerned with the heart contraction. There was no clear perception of the importance of variation between heart beats or frequency bands associated with it. The understanding of Heart rate variability, defined as the change in the time intervals between adjacent heartbeats, is an emergent property of interdependent regulatory systems that operates on different time scales to adapt to environmental and psychophysiological Challenges. The importance of HRV as an index of the functional status of physiological control systems was noted only in 1965 when it was found that fetal distress is preceded by reductions in HRV before any changes occur in HR itself. The introduction of signal processing technologies that can acquire continuous time series data from physiological processes such as heart rate (HR), blood pressure (BP), and nerve activity, make it abundantly apparent that biological processes vary in complex and nonlinear ways, even during so called "steady-state" conditions [1]. This in our perspective is complementary but not competitive to the great physiologist Walter Bradford Cannon who expand Claude Bernard concept of homeostasis and the steady state condition that body cells, tissues and organs are strived to maintain. Healthy, optimal function as we perceive it in 2017 is the result of continuous, dynamic, bi-directional interactions among multiple neural, hormonal, and mechanical control systems at both local and central levels.

The average 24-hour HR in healthy people is approximately 73 bpm. Higher HRs are independent markers of mortality in a wide spectrum of conditions [2]. As HR increases there is less time between heartbeats for variability to occur, thus HRV decreases. At lower HRs there is more time between heartbeats and variability naturally increases. This is called cycle length dependence. Elderly patients with ischemic heart disease or other pathologies develop less variability at increasingly lower HRs and ultimately lose the relationship between HR and variability, to the point that variability does not increase with reductions in HR [3]. Even in healthy subjects, the effects of cycle length dependence should be taken into account when assessing HRV.

HRV analysis is, beyond the scope of this editorial, but can be done with various analytical approaches, although the most commonly used are frequency domain (power spectral density) analysis and time domain analysis. During the time line of HRV science developments, major breakthrough in understanding of the complex interacting mechanisms that operate HRV came after understanding HRV frequencies. The European Society of Cardiology and the North American Society of Pacing and Electrophysiology Task Force Report on HRV divided heart rhythm oscillations into 4 primary frequency bands; high-frequency (HF): from 0.15 Hz to 0.4 Hz, low-frequency (LF): from 0.04 Hz to 0.15 Hz, very-low-frequency (VLF): from 0.0033 to0.04 Hz, and ultra-low-frequency (ULF): below 0.0033 Hz [4].

The high frequency band(HF) reflects parasympathetic or vagal activity and is frequently called the respiratory band because it corresponds to the HR variations related to the respiratory cycle known as respiratory sinus arrhythmia. The mechanisms linking the variability of HR to respiration are complex and involve both central and reflex interactions [5]. In terms of psychological regulation, reduced vagally mediated HRV has been linked to reduced self regulatory capacity and cognitive functions that involve the executive centers of the prefrontal cortex. This is consistent with the finding that lower HF power is associated with stress, panic, anxiety, or worry [1].

The low frequency band(LF) was previously called the "baroreceptor range" or "mid-frequency band" by many researchers, since it primarily reflects baroreceptor activity while at rest [6]. Baroreceptors are stretch-sensitive mechanoreceptors located in the chambers of the heart and vena cavae, carotid sinuses (which contain the most sensitive mechanoreceptors), and the aortic arch. The vagus nerves are a major conduit though which afferent (ascending) neurological signals from the heart are relayed to the brain, including baroreflex signals. Baroreflex gain is commonly calculated as the beat-to-beat change in HR per unit of change in systolic BP [7]. The cardiovascular system resonance frequency is a distinctive high-amplitude peak in the HRV power spectrum around 0.1 Hz [1].

The very low frequency band (VLF) seems to be drastically under investigated in the medical literature. Although all 24-hour clinical measures of HRV reflecting low HRV are linked with increased risk of adverse outcomes, the VLF band has stronger associations with all-cause mortality than the LF and HF bands [8-10] .Low VLF power has been shown to be associated with arrhythmic death [11] and posttraumatic stress disorder(PTSD) [12]. Additionally, low power in this band has been associated with high inflammation [13,14] and has

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Received: April 14, 2017; **Accepted:** April 25, 2017; **Published:** April 28, 2017

been correlated with low levels of testosterone. the VLF rhythm appears to be produced by the heart itself and may be an intrinsic rhythm that is fundamental to health and wellbeing. This cardiac origin of the VLF rhythm is also supported by studies showing that sympathetic blockade does not affect VLF power. Furthermore, VLF activity remains in quadriplegics, whose sympathetic innervation of the heart and lungs is disrupted [15].

The ultra low frequency band(ULF) originates primarily from circadian oscillation in HR although other very slow-acting regulatory processes, such as core body temperature regulation, metabolism, and the renin-angiotensin system likely add to the power in this band [16]4.

Increasing evidence from clinical, physiological, and anatomical research has identified cortical, subcortical and medulla oblongata structures involved in self-regulation. Oppenheimer and Hopkins mapped a detailed hierarchy of cardiac control structures among the cortex, amygdala and other subcortical structures, all of which can modify cardiovascular related neurons in the lower levels of the neuraxis [16]. They suggest that the amygdala is involved with refined integration of emotional content in higher centers to produce cardiovascular responses that are appropriate for the emotional aspects of the current circumstances.

The insular cortex and other centers such as the orbitofrontal cortex and cingulate gyrus can overcome emotionally entrained responses by inhibiting or enhancing them. Imbalances between the neurons in the insula, amygdala and hypothalamus may initiate cardiac rhythm disturbances and arrhythmias. The data suggest that the insular and medial prefrontal cortexes are key sites involved in modulating the heart's rhythm, particularly during emotionally charged circumstances [1].

Thayer and Lane have also described the same set of neural structures which they call the central autonomic network (CAN). The CAN is involved in cognitive, affective, and autonomic regulation. The CAN is related to HRV and linked to cognitive performance. They have shown that vagally mediated HRV is correlated with prefrontal cortical performance and the ability to inhibit unwanted memories and intrusive thoughts.

Significant and crucial findings sometimes revolutionize basic philosophies and fundamentals. This is very conspicuous in the case of the 10th cranial nerve (the vagus nerve) which is believed in scientific communities to be efferent visceral control nerve. In fact: the majority (80–90%) of vagal nerve fibers are afferent, communicating the state of the body to the brain (Berthoud and Neuhuber 2000). Since the vagal efferent dominate cardiac control (Levy 1990), and the vagus contains a high percentage of afferent fibers(Porges 1995), it is theorized that both the input and output of the Central Autonomic Network (CAN) in the brain are directly linked to HRV [17].

Furthermore, more vagal fibers are related to cardiovascular pathways than other organs [18]. Complex patterns of cardiovascular afferent nerve activity occur across time scales from milliseconds to minutes [19]. The intrinsic cardiac nervous system has both short-term and long-term memory functions, which can influence HRV and afferent activity related to BP, rhythm, rate, and hormonal factors [19-21]. The intrinsic cardiac neurons (sensory, interconnecting, afferent, and motor) can operate independently of central neuronal command,and their network is sufficiently extensive to be characterized as its own "little brain" in the heart [22,23].

The afferent nerves play a critical role in physiological regulation and affect the heart's rhythm and HRV. Efferent sympathetic and parasympathetic activity is integrated in the heart's intrinsic nervous system, with the signals arising from the mechanosensory and chemosensory neurons in the heart. The neural output of the intrinsic cardiac nervous system then travel to the brain via afferent pathways in the spinal column and vagus nerve. Intrinsic cardiac afferent neurons project to nodose and dorsal root ganglia,the spinal cord, brainstem, hypothalamus, thalamus or amygdala and then to the cerebral cortex [24-26].

John and Beatrice Lacey were the first to suggest a causal role of the heart in modulating cognitive functions such as sensory-motor and perceptual performance [27-29]. They suggested that cortical functions are modulated via afferent input from pressure sensitive neurons in the heart, carotid arteries, and aortic arch. Their research focused on activity occurring within a single cardiac cycle, and they confirmed that cardiovascular activity influences perception and cognitive performance.

Research by Velden and Wolk later demonstrated that cognitive performance fluctuated at a rhythm around 10 Hz and showed that the modulation of cortical function via the heart's influence was due to afferent inputs on the neurons in the thalamus, which globally synchronizes cortical activity [30,31]. An important aspect of their work was the finding that it is the "pattern and stability" (of the rhythm) of the heart's afferent inputs, rather than the number of neural bursts within the cardiac cycle, that are important in modulating thalamic activity, which in turn has global effects on brain function.

There has since been a growing body of research indicating that afferent information processed by the intrinsic cardiac nervous system can influence activity in the frontocortical areas and motor cortex, affecting psychological factors such as attention level, motivation, perceptual sensitivity, and emotional processing [24,32-37].

Hyeong-Dong Park (Nature Neuroscience) documented neural events locked to heartbeats before stimulus onset predict the detection of a faint visual grating in the posterior right inferior parietal lobule and the ventral anterior cingulate cortex, two regions that have multiple functional correlates and that belong to the same resting-state network [38].

McCraty and colleagues introduced the term physiological coherence to describe the degree of order, harmony, and stability in the various rhythmic activities within living systems over any given time period [39]. This harmonious order signifies a coherent system that has an efficient or optimal function directly related to the ease and flow in life processes. By contrast, an erratic, discordant pattern of activity denotes an incoherent system whose function reflects stress and inefficient utilization of energy in life processes. Specifically, heart coherence (also referred to as cardiac coherence or resonance) can be measured by HRV analysis wherein a person's heart rhythm pattern becomes more ordered and sine-wave like at a frequency of around 0.1 Hz (10 seconds). A coherent heart rhythm is defined as a relatively harmonic, sine wave– like, signal with a very narrow, high-amplitude peak in the LF region of the HRV power spectrum with no major peaks in the VLF or HF regions [1].

Heartbeat evoked potentials (HEPs) can be used to identify the specific pathways and influence of afferent input from the heart to the brain. HEPs are segments of electroencephalogram (EEG) that are synchronized to the heartbeat. The ECG R-wave is used as a timing source for signal averaging, resulting in waveforms known as HEPs.

Changes in these evoked potentials associated with the heart's afferent neurological input to the brain are detectable between 50 and 550 ms after each heartbeat [40]. MacKinnon et al. (2013) reported that HRV influences the amplitude of heartbeat evoked potentials (HEP N250s). In this specific context, self-induction of either negative or positive emotion conditions by recalling past events reduced HRV and N250 amplitude. In contrast, resonance frequency breathing (breathing at a rate that maximizes HRV amplitude) increased HRV and HRV coherence (auto-coherence and sinusoidal pattern) above baseline and increased N250 amplitude [17].

A ground breaking scientific fact is the discovery of the intimate relationship between the human heart represented by HRV and extremes of creations as large as the Solar Geomagnetic activity and as small as subcellular pathways. At subcellular level blood platelet mtDNA methylation levels were found to be higher in heart patients compared with healthy persons. Blood mtDNA methylation levels were negatively associated with PM2.5 exposure and modified the adverse relationships between PM2.5 exposure and heart rate variability outcomes [41].

Different HRV patterns documented to different emotions. The heart generates a series of electromagnetic pulses in which the time interval between each beat varies in a complex manner. These pulsing waves of electromagnetic energy give rise to interference patterns when they interact with magnetically polarizable tissues and substances [42]. We believe HRV and the pulsating heart electromagnetic field is the carrier of non verbal communication in biology.

Alexander Chizhevskywork in 1920 followed by Franz Halberg (1897-2013) and our team in the HeartMath Institute (HMI) and the Global Coherence Initiative(GCI) established the scientific prove of earth magnetic field correlation to biology in earth.

Plethora of medical literature document the statistical correlation between solar storms and myocardial infarctions, arrhythmias, depression epilepsy, and road traffic accidents.

The longest investigation of synchronized data recorded from human HRV and solar activity was done by our group (Alabdulgader, McCraty and colleagues) in the Saudi HRV study. A total of 960 24-hour HRV recordings were obtained from a group of 16 women. Overall, the study strongly confirms that daily autonomic nervous system activity, as reflected by HRV measures, is affected generally by changes in solar and geomagnetic activity, rather than only periods of magnetic disturbances. All of the HRV measures, except inter beat intervals, were negatively correlated with solar wind speed, and the LF and HF power were negatively correlated with the magnetic field mean data from the Saudi Arabia sensor site, but not the California sensor site, suggesting that local measurements are important. The most likely mechanism for explaining how solar and geomagnetic influences affect human health and behavior are a coupling between the human nervous tissue in brain and heart and resonating geomagnetic frequencies, called Schumann resonances, which occur in the earth-ionosphere resonant cavity and Alfvn waves [43].

Associations between the human heart and circulation and the environment near and far at the level of local and global populations in both space and time is philosophical practical medicine which was implanted in our way of thinking by the great Franx Halber, founder of chronobiology in modern science [44]. Incorporation of this huge spectrum between genes and galaxies in medical practice to diagnose and treat diseases was achieved by us and others. The great success of the HeartMath Institute in treating stress and many psychophysiological diseases using their model of HRV modulation namely the heart coherence and associated techniques is well known but beyond the scope of this editorial. Modulating HRV to treat systemic hypertension was found to be a novel non pharmacological modality for lowering blood pressure in hypertensive patients [45,46]. We found chronologically interpreted ambulatory blood pressure monitoring an intelligent concept to utilize cardiovascular continuous variations in synchronization with cosmos to diagnose vascular variability disorders in promising new modality to stratify risk factors for heart disease in human [47].

This documentation of orchestration between earth magnetic field and HRV is highly creative.Luc Montagnier, and co-researchers discovered that epigenetic information related to DNA could be detected as electromagnetic signals radiated from solutions containing DNA. They demonstrated that this information could be transferred to and instruct the re-creation of DNA in a remote test tube of water containing the appropriate basic constituents of DNA by electromagnetic frequency fields of 7.8 hertz, which is, the first Schumann resonance frequency [48].

Riganello F and colleagues reported Heart rate variability analyses appear to be applicable to assess residual or emerging (higher level) function in brain-injured patients with disordered consciousness and to predict outcome [49]. The secrets of relations between consciousness as we perceive it in our current life and HRV and human emotions is mysterious direction in science that should evolve to raise our global consciousness.

The Global Coherence Initiative (GCI) program of HeartMath Institute encourage humans to keep their hearts coherent with HRV peaks at 0.1 Hz. It is postulated that as increasing numbers of people add coherent energy to the global field, it helps strengthen and stabilize mutually beneficial feedback loops among human beings and with the earth itself to be in planetary resonance.

Hear Rate Variability science and innovations stands in a critical intersection between cardiac sciences, psychology, cosmology, quantum physics and consciousness research. This variation in human heart beats seems to be in a highly sophisticated and delicate connections to the global commander from genes to galaxies and to carry the secrets of life and beyond.

References

1. Rollin McCraty, Fred Shaffer (2015) Heart Rate Variability: New Perspectives on Physiological Mechanisms, Assessment of Self-regulatory Capacity, and Health Risk. *Glob Adv Health Med* 4: 46-61. [Crossref]

2. Palatini P1 (1999) Elevated heart rate as a predictor of increased cardiovascular morbidity. *J Hypertens Suppl* 17: S3-10. [Crossref]

3. Umetani K, Duda CL, Singer DH (1996) Aging effects on cycle length dependence of heart rate variability. Proceedings of the 1996 Fifteenth Southern Biomedical Engineering Conference: Dayton, Ohio.

4. Heart rate variability standards of measurement, physiological interpretation, and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *Circulation* 93: 1043-1065. [Crossref]

5. Hopkins D, Ellenberger H (1994) Cardiorespiratory neurons in the medulla oblongata: Input and output relationships. In Neurocardiology. New York: Oxford University Press 219-244.

6. Malliani A (1995) Association of heart rate variability components with physiological regulatory mechanisms. In Heart Rate Variability. Armonk NY: Futura Publishing Company, Inc 173-188.

7. Bowers EJ, Murray A (2004) Effects on baroreflex sensitivity measurements when

different protocols are used to induce regular changes in beat-to-beat intervals and systolic pressure. *Physiol Meas* 25: 523-538.

8. Tsuji H, Venditti FJ Jr, Manders ES, Evans JC, Larson MG, et al. (1994) Reduced heart rate variability and mortality risk in an elderly cohort. The Framingham Heart Study. *Circulation* 90: 878-883. [Crossref]

9. Schmidt H, Müller-Werdan U, Hoffmann T, Francis DP, Piepoli MF, et al. (2005) Autonomic dysfunction predicts mortality in patients with multiple organ dysfunction syndrome of different age groups. *Crit Care Med* 33: 1994-2002. [Crossref]

10. Tsuji H, Larson MG, Venditti FJ Jr, Manders ES, Evans JC, et al. (1996) Impact of reduced heart rate variability on risk for cardiac events. The Framingham Heart Study. *Circulation* 94: 2850-2855. [Crossref]

11. Bigger JT Jr, Fleiss JL, Steinman RC, Rolnitzky LM, Kleiger RE, et al. (1992) Frequency domain measures of heart period variability and mortality after myocardial infarction. *Circulation* 85: 164-171. [Crossref]

12. Shah AJ, Lampert R, Goldberg J, Veledar E, Bremner JD, et al. (2013) Posttraumatic stress disorder and impaired autonomic modulation in male twins. *Biol Psychiatry* 73: 1103-1110. [Crossref]

13. Lampert R, Bremner JD, Su S, Miller A, Lee F, et al. (2008) Decreased heart rate variability is associated with higher levels of inflammation in middle-aged men. *Am Heart J* 156: 759. [Crossref]

14. Carney RM, Freedland KE, Stein PK, Miller GE, Steinmeyer B, et al. (2007) Heart rate variability and markers of inflammation and coagulation in depressed patients with coronary heart disease. *J Psychosom Res* 62: 463-467. [Crossref]

15. Berntson GG, Bigger JT Jr, Eckberg DL, Grossman P, Kaufmann PG, et al. (1997) Heart rate variability: origins, methods, and interpretive caveats. *Psychophysiology* 34: 623-648. [Crossref]

16. Oppenheimer S, Hopkins D (1994) Suprabulbar neuronal regulation of the heart. In Neurocardiology. New York: Oxford University Press 309-341.

17. Starr MacKinnon, Richard Gevirtz, Rollin McCraty, Milton Brown (2013) Utilizing Heartbeat Evoked Potentials to Identify Cardiac Regulation of Vagal Afferents During Emotion and Resonant Breathing. *Appl Psychophysiol Biofeedback* 38: 241-255. [Crossref]

18. Cameron OG (2002) Visceral sensory neuroscience: interception. New York: Oxford University Press.

19. Armour JA, Kember GC (2004) Cardiac sensory neurons. In Basic and clinical neurocardiology. New York: Oxford University Press 79-117.

20. Kleiger RE, Stein PK, Bigger JT Jr (2005) Heart rate variability: measurement and clinical utility. *Ann Noninvasive Electrocardiol* 10: 88-101. [Crossref]

21. Ardell JL, Cardinal R, Vermeulen M, Armour JA (2009) Dorsal spinal cord stimulation obtunds the capacity of intrathoracic extracardiac neurons to transduce myocardial ischemia. *Am J Physiol Regul Integr Comp Physiol* 297: R470-477. [Crossref]

22. Armour JA (1991) Anatomy and function of the intrathoracic neurons regulating the mammalian heart. In Reflex control of the circulation. Boca Raton, FL: CRC Press 1-37.

23. Armour JA (2008) Potential clinical relevance of the 'little brain' on the mammalian heart. *Exp Physiol* 93: 165-176. [Crossref]

24. McCraty R, Atkinson M, Tomasino D, Bradley RT (2009) The coherent heart: heartbrain interactions, psychophysiological coherence, and the emergence of system-wide order. Boulder Creek, CA: Institute of Heartmath.

25. Armour JA (1994) Peripheral autonomic neuronal interactions in cardiac regulation. In Neurocardiology New York: Oxford University Press 219-244.

26. Kukanova B, Mravec B (2006) Complex intracardiac nervous system. *Bratisl Lek Listy* 107: 45-51. [Crossref]

27. Lacey BC, Lacey JI (1974) Studies of heart rate and other bodily processes in sensorimotor behavior. In: Obrist Cardiovascular psychophysiology: current issues in response mechanisms, biofeedback, and methodology. Chicago: Aldine 538-564.

28. Lacey JI (1967) Somatic response patterning and stress: Some revisions of activation theory, In: Psychological stress: issues in research. New York: Appleton-Century-Crofts 14-42.

29. Lacey JI, Lacey BC (1970) Some autonomic-central nervous system interrelationships. In: Physiological correlates of emotion. New York: Academic Press 205-227.

30. Wolk C, Velden M (1987) Detection variability within the cardiac cycle: toward a revision of the "baroreceptor hypothesis." *J Psychophysiol* 1: 61-65.

31. Wolk C, Velden M (1989) Revision of the baroreceptor hypothesis on the basis of the new cardiac cycle effect. In Psychobiology: issues and applications. Amsterdam: Elsevier Science Publishers B.V. 371-379.

32. Lane RD, Reiman EM, Ahem GL, Thayer JF (2001) Activity in medial prefrontal cortex correlates with vagal component of heart rate variability during emotion. *Brain Cognit* 47: 97-100.

33. McCraty R, Atkinson M, Bradley RT (2004) Electrophysiological evidence of intuition: Part 2. A system-wide process? *J Altern Complement Med* 10: 325-336. [Crossref]

34. Svensson TH, Thoren P (1979) Brain noradrenergic neurons in the locus coeruleus: inhibition by blood volume load through vagal afferents. *Brain Res* 172: 174-178. [Crossref]

35. Schandry R, Montoya P (1996) Event-related brain potentials and the processing of cardiac activity. *Biol Psychol* 42: 75-85. [Crossref]

36. Montoya P, Schandry R, Müller A (1993) Heartbeat evoked potentials (HEP): topography and influence of cardiac awareness and focus of attention. *Electroencephalogr Clin Neurophysiol* 88: 163-172. [Crossref]

37. Zhang JX, Harper RM, Frysinger RC (1986) Respiratory modulation of neuronal discharge in the central nucleus of the amygdala during sleep and waking states. *Exp Neurol* 91: 193-207. [Crossref]

38. Hyeong-Dong Park, Stéphanie Correia, Antoine Ducorps, Catherine Tallon-Baudry (2014) Spontaneous fluctuations in neural responses to heartbeats predict visual detection. *Nature Neuroscience* 17: 612-618.

39. Tiller WA1, McCraty R, Atkinson M (1996) Cardiac coherence: a new, noninvasive measure of autonomic nervous system order. *Altern Ther Health Med* 2: 52-65. [Crossref]

40. Schandry R, Montoya P (1996) Event-related brain potentials and the processing of cardiac activity. *Biol. Psychol* 42: 72-85. [Crossref]

41. Byun HM, Colicino E, Trevisi L, Fan T, Christiani DC, et al. (2016) Effects of Air Pollution and Blood Mitochondrial DNA Methylation on Markers of Heart Rate Variability. *J Am Heart Assoc* 5. [Crossref]

42. Rollin McCraty, Annette Deyhle. The Science of Interconnectivity: Exploring the human earth connection.

43. Abdullah Alabdulgade, Rollin McCraty, Mike Atkinson, Alfonsas Vainoras, Kristina Berškiene, et al. (2015) Human Heart Rhythm Sensitivity to Earth Local Magnetic Field Fluctuations. *Journal of Vibroengineering*:17.

44. Franz Halberg, Germaine Cornélissen, Rollin McCraty, Jerzy Czaplicki, Abdullah A. Al-Abdulgader (2011) Time Structures (Chronomes) of the Blood Circulation, Populations' Health, Human Affairs and Space Weather. *World Heart Journal* 3.

45. Alabdulgader AA (2012) Coherence: a novel nonpharmacological modality for lowering blood pressure in hypertensive patients. *Global Adv Health Med* 1: 56-64. [Crossref]

46. Abdullah Alabdulgader (2016) Modulation of heart rate variability: A novel non-pharmacological modality for lowering blood pressure in in hypertensive patients. *J Clin Exp Cardiolog.*

47. Abdullah A. Al-Abdulgader, Germaine Cornélissen Guillaume, Franz Halberg (2011) Vascular Variability Disorders in the Middle East:Case Reports. *World Heart Journal* 2.

48. Montagnier L, Aissa J, Del Guidice E, Lavallee C, Tedeschi A, et al. (2011) DNA waves and water. *Journal of Physics* 306: 1-10.

49. Riganello F, Dolce G, Sannita WG (2012) Heart rate variability and the central autonomic network in the severe disorder of consciousness. *J Rehabil Med* 44: 495-501. [Crossref]