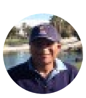
Physiology of long pranayamic breathing: Neural respiratory elements may provide a mechanism that explains how slow deep breathing shifts the autonomic nervous system

ARTICLE *in* MEDICAL HYPOTHESES · FEBRUARY 2006

Impact Factor: 1.07 · DOI: 10.1016/j.mehy.2006.02.042 · Source: PubMed

4 AUTHORS, INCLUDING:



Ravinder Jerath
Augusta Women's Center
14 PUBLICATIONS **113** CITATIONS

Vernon A Barnes
Georgia Regents University
52 PUBLICATIONS **933** CITATIONS

Medical Hypotheses (2006) x, xxx–xxx

medical hypotheses

http://intl.elsevierhealth.com/journals/mehy

Physiology of long pranayamic breathing: Neural respiratory elements may provide a mechanism that explains how slow deep breathing shifts the autonomic nervous system

Ravinder Jerath *, John W. Edry, Vernon A. Barnes, Vandna Jerath

Augusta Women's Center, 2100 Central Avenue, Suite 6 & 7, Augusta, GA 30904, United States

Received 20 January 2006; accepted 23 February 2006

Summary Pranayamic breathing, defined as a manipulation of breath movement, has been shown to contribute to a physiologic response characterized by the presence of decreased oxygen consumption, decreased heart rate, and decreased blood pressure, as well as increased theta wave amplitude in EEG recordings, increased parasympathetic activity accompanied by the experience of alertness and reinvigoration. The mechanism of how pranayamic breathing interacts with the nervous system affecting metabolism and autonomic functions remains to be clearly understood. It is our hypothesis that voluntary slow deep breathing functionally resets the autonomic nervous system through stretch-induced inhibitory signals and hyperpolarization currents propagated through both neural and non-neural tissue which synchronizes neural elements in the heart, lungs, limbic system and cortex. During inspiration, stretching of lung tissue produces inhibitory signals by action of slowly adapting stretch receptors (SARs) and hyperpolarization current by action of fibroblasts. Both inhibitory impulses and hyperpolarization current are known to synchronize neural elements leading to the modulation of the nervous system and decreased metabolic activity indicative of the parasympathetic state. In this paper we propose pranayama's physiologic mechanism through a cellular and systems level perspective, involving both neural and non-neural elements. This theoretical description describes a common physiological mechanism underlying pranayama and elucidate the role of the respiratory and cardiovascular system on modulating the autonomic nervous system. Along with facilitating the design of clinical breathing techniques for the treatment of autonomic nervous system and other disorders, this model will also validate pranayama as a topic requiring more research.

Introduction

Pranayama: a brief review

''Pranayama'' (the practice of voluntary breath control, consisting of conscious inhalation, reten-

* Corresponding author. Tel.: +1 706 736 5378; fax: +1 706 738 9922.
E-mail address: RJ605R@aol.com (R. Jerath).

0306-9877/$ - see front matter © 2006 Published by Elsevier Ltd.
doi:10.1016/j.mehy.2006.02.042

tion and exhalation) is often practiced in conjunction with ''dhyana'' (meditation), and ''asanas'' (physical posture) [1]. Versions of pranayama vary from single nostril breathing to belly breathing. Pranayama consists of three phases: ''puraka'' (inhalation); ''kumbhaka'' (retention) and ''rechaka'' (exhalation) that can be either fast or slow [2]. Although all pranayama has three phases, different forms of pranayama evoke dissimilar and sometimes opposite responses in the subject depending on variables such as which nostril is used or the speed of the respiration. Pranayama has been researched mostly for its beneficial applications in treatment of cardiovascular diseases such as hypertension [2—4], pulmonary disease such as asthma [5—7], autonomic nervous system imbalances [8], and psychologic or stress related disorders [4,9].

Pranayama is known to improve pulmonary function [10] and cardiovascular profile [2—4]. A Buteyko breathing device, which mimics pranayama, was shown to improve symptoms and reduce bronchodilator use in asthma patients [5,7]. Pranayama has also been shown, over time, to reduce oxygen consumption per unit work [11]. ''Kapalabhati'', a fast breathing pranayamic technique, has been shown to promote decarboxylation and oxidation mechanisms in the lungs which is believed to ''quiet'' the respiratory centers [12]. Alteration in information processing at the primary thalamocortical level inducing modification in neural mechanisms regulating the respiratory system [13] may contribute to pranayama's beneficial pulmonary effects. In studies that examined pranayama as a form of exercise, nostril breathing was shown to increase hand grip strength of both hands [14]. Pranayama, by reducing risk factors associated with cardiovascular disease [15], has shown that it is not only theraputic but also preventative. Reduction in oxidative stress levels with increased superoxide dismutase and decreased number of free radicals may explain in part the beneficial long-term impact pranayama has on the cardiopulmonary system [16].

Pranayama is known to increase neural plasticity and to alter information processing making it a possible treatment for psychological and stress disorders or improving one's psychological profile [4,9]. Higher improvement in IQ and social adaptation parameters were noticed in mentally retarded children after yogic training including pranayama [17]. Sudarshan Kriya Yoga, which includes pranayama, has been used as a public health intervention for treatment of post traumatic stress disorder, depression, stress related medical illnesses, substance abuse, and rehabilitation of criminal offenders for its ability to enhance well being, mood, attention, mental focus, and stress tolerance [9]. In conjunction with other yogic techniques, pranayama has been shown to decrease symptoms of irritable bowel syndrome by enhancing parasympathetic activity of gastrointestinal tract and by reducing effects of stress [18]. It has been mentioned as a possible treatment for symptoms of epilepsy [1] and has been shown to increase plasticity of motor control indicating that it might have applications in rehabilitation programs [19].

Different forms of pranayama activate different branches of the autonomic nervous system effecting oxygen consumption, metabolism and skin resistance. Pranayamic breathing, characterized by brief breath retention, caused significant increases in oxygen consumption and metabolic rate while pranayamic breathing, characterized by long breath retention, caused lowering of oxygen consumption and metabolic rate [20]. This demonstrates that slow breathing enhances parasympathetic activation. In another study using breathing exercises mimicking pranayama, slow breathing over a period of three months was shown to improve autonomic function while fast breathing did not have an effect on the autonomic nervous system [8]. Slow breathing pranayamic exercises show a strong tendency of improving or balancing the autonomic nervous system through enhanced activation of parasympathetic nervous system. In contrast to slow pranayamic breathing, nostril breathing, both through right nostril, left nostril, and both nostrils, has been shown to increase baseline oxygen consumption indicative of sympathetic discharge of the adrenal medulla [21]. Contradictorily, left nostril breathing has been shown to increase volar galvanic skin resistance interpreted as a reduction in sympathetic nervous activity [21]. Although nostril breathing and short pranayamic breathing practices are capable of altering the autonomic nervous system, more research is required to fully understand their clinical benefits [21].

Pranayama may also affect the immune system. Inhibition of the sympathetic nervous system has been shown to enhance function of the immune system in several forms of meditation including mindfulness meditation, Qigong, and Transcendental meditation [22—25]. Since pranayama has been shown to shift the autonomic nervous system away from sympathetic dominance [8,26] it is probable that pranayama may have beneficial immune effects similar to meditation. More studies are needed to elucidate pranayama's direct effect on immune function.

Although many studies show pranayama is a beneficial technique, there have been studies that indicate possible risks especially associated with fast breathing versions. If done improperly, fast breathing pranayama can cause hyperventilation and may hyperactivate the sympathetic nervous system [27] which may stress the body. Pneumothorax has been attributed to fast breathing ''Kapabhati'' pranayama in one case study [28]. Some studies indicate that deep breathing similar to slow breathing pranayama may agitate symptoms of bronchial hyperactivity. Deep breathing induced parasympathetic activity is correlated with bronchial hyperactivity in asthmatics [29]. It is possible that pranayamic parasympathetic activity may elicit bronchial hyperactivity in asthmatics as well.

Pranayamic breathing has been shown to be a beneficial clinical application in the treatment of psychological disorders as well as physiological diseases. Research has revealed pranayamic breathing to be a low risk, cost effective adjunct treatment that can be potentially applied to improve symptoms associated with cardiovascular disorders, autonomic disorders, and psychological disorders including those involving stress [9]. Although slow pranayamic breathing is said to be one of the most practical relaxation techniques [2] and holds a great deal of potential in the treatment of autonomic and psychological disorders, two problems exist in present research that prevent full application and understanding of this practice. The first is that there is no coherent model for the mechanism underlying slow pranayamic breathing. A physiological description of the pranayamic mechanism would provide insight into the cellular physiology of deep breathing and the dynamic connection between the nervous system and respiration. Secondly, many studies report only the effects of pranayama, yogic postures, and meditation collectively. In future research, pranayama needs to be studied exclusively without meditation or postures in order to fully understand the pranayamic mechanism. Within the research conducted on the many different types of pranayama, slow rhythmic pranayamic breathing seems to be the most practical and hold the most physiological benefit.

Slow pranayama, a treatment for autonomic disorders

Slow pranayamic breathing, characterized as regular slow frequency respiration with long periods of breath retention has been known to cause short-term and long-term changes in physiology. One long-term effect of pranayamic breathing is the improvement in autonomic function [3]; specifically, with slow breathing pranayama there is a noted increase in parasympathetic activity and a decrease in sympathetic dominance [8]. It has been suggested that the cardio-respiratory system can be normalized through rhythmic breathing exercises [30,31] such as slow pranayama.

Short-term effects of slow pranayamic breathing include increased galvanic skin resistance (a non-neural response) [21], decreased oxygen consumption [20], decreased heart rate, decreased blood pressure [3], and increased amplitude of theta waves [32]. Increase theta amplitude and delta waves during breath retention and slow breathing is indicative of a parasympathetic state while alpha and beta waves signify activity. Both the short-term and long-term effects of pranayamic breathing indicate a dynamic alteration of the autonomic system.

There are several chemical and non-chemical mechanisms that may account for some of the physiologic phenomena experienced by pranayama practitioners. No significant changes in arterial blood gases were noted after pranayama practice indicating a neural mechanism for pranayama's effect [33]. Increased melatonin production after a regimen of slow breathing pranayamic exercises has been attributed to pranayama's tendency to create a sense of relaxation and well being in the subject [4]. Breath holding, an essential part of pranayama, is shown to induce theta waves [32]. A decrease in breathing frequency can increase synchronization of brain waves eliciting delta wave activity [34] indicating parasympathetic dominance. Although these mechanisms provide some clues to pranayama's mechanism, the neural mechanism that causes this body-wide autonomic shift is largely unknown [3]. It has been proposed that certain voluntary breathing exercises can modulate the parasympathetic and sympathetic nervous system bringing their levels of activation into a normal range [35]. Some have proposed that pranayama alters autonomic responses to breath holding perhaps by increasing vagal tone and decreasing sympathetic discharges [26]. It has been suggested that pranayama ''balances'' the autonomic nervous system through stretch-induced inhibitory signals of abdominal muscles (specifically the diaphragm) and even nerve endings in the nose [32]. It is abundantly evident that respiration and the parasympathetic response are intricately connected. What is not clear, however, is the cellular mechanism that integrates respiration and the parasympathetic response.

Hypothesis

The general cellular mechanism of pranayama

It is our hypothesis that voluntary, slow, deep breathing functionally resets the autonomic nervous system through stretch-induced inhibitory signals and hyperpolarization currents propagated through both neural and non-neural tissue which synchronizes neural elements in the heart, lungs, limbic system, and cortex.

It is suspected that deep pranayamic breathing, by voluntary control, dynamically modulates the autonomic nervous system by heightening generation of two physiologic signals: (1) Pranayama increases frequency and duration of inhibitory neural impulses by activating stretch receptors of the lungs during above tidal volume inhalation (as seen in the Hering Breuer's reflex). (2) Pranayama heightens generation of hyperpolarization current by stretch of connective tissue (fibroblasts) localized around the lungs (see Fig. 1). It is recognized that inhibitory impulses, produced by slowly adapting receptors (SARs) in the lungs during inflation [36], play a role in controlling typically autonomic functions such as breathing pattern, airway smooth muscle tone, systemic vascular resistance, and heart rate [37]. Stretch of connective tissue fibroblasts are capable of effecting the membrane potential of nervous tissue [38]. Both hyperpolarization and inhibitory impulses generated by stretch of neural and non-neural tissue of the lungs are the likely agents of autonomic shift during pranayamic breathing.

Inhibitory current synchronizes rhythmic cellular activity between the cardiopulmonary center [39] and the central nervous system [40]. Inhibitory current regulates excitability of nervous tissues [41] and is known to elicit synchronization of neural elements which typically is indicative of a state of relaxation [42]. Synchronization within the hypothalamus and the brainstem [43] is likely responsible for inducing the parasympathetic response [44] during breathing exercises. The strongest cardioventilatory coupling, a parasympathetic-type phenomenon, occurs when there is decreased

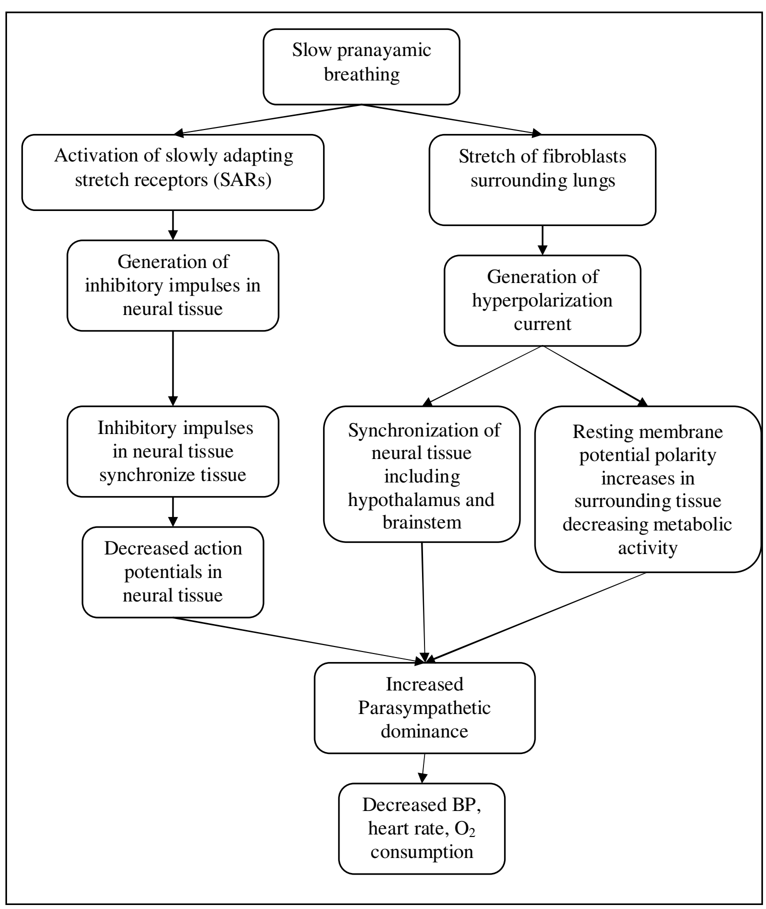


Figure 1 Diagram of the series of events that occur during the autonomic shift present in pranayamic slow breathing.

breathing frequency [45] similar to that found in slow pranayama. Cardioventilatory coupling, found during deep breathing exercises, indicates a synchronization mechanism that coordinates neural and non-neural activity. It is likely that inhibitory synchronized activity between the lungs and brain elicits parasympathetic-like states.

Hyperpolarization affects the autonomic nervous system by modulating neuronal excitability [46], resting membrane potential [39], and generating rhythmic brain activity [40]. It is well documented that hyperpolarization of tissues manifests itself in parasympathetic-like changes [47]. Hyperpolarization is generated during stretch of fibroblasts in tissue surrounding the lungs [38]. Similarly, in some neurons, hyperpolarization current inhibits unsynchronized neuronal input [46] thereby increasing the dominance of synchronized input. Stretch of lung fibroblasts likely contributes to the generation of the slower wave brain activity and the parasympathetic autonomic shift present during slow pranayamic breathing exercises.

There are several ways to test the hypothesis that voluntary slow pranayama functionally resets the autonomic nervous system through stretch-induced inhibitory signals and hyperpolarization currents propagated through both neural and non-neural tissue. Simultaneous, intracellular, in vivo recordings of fibroblasts and endothelium in the lungs and heart during slow pranayamic breathing would show that hyperpolarizing currents, originating in the lungs, are being propagated long distances affecting the cellular metabolism as well as nervous system excitability. Blockade of inhibitory signals during activation of lung stretch receptors would likely show a decrease in the parasympathetic effect of slow pranayamic breathing consistent with our model. A recording measuring autonomic indicators such as respiratory sinus arrhythmia (RSA), the frequency of heart rate variability, and EEG during the practice of pranayama would also suggest that this deep breathing technique resets the autonomic nervous system through parasympathetic shift and causes increased synchronization of neural elements with the heart, lungs, and cortex.

This hypothesis presents a common physiological mechanism underlying several forms of breathing exercises and elucidates the role of the respiratory and cardiovascular system on modulating the autonomic nervous system. Revealing the cellular mechanisms that shifts the autonomic nervous system towards parasympathetic dominance is important for the general understanding of yogic breathing practices and breathing physiology. The cooperative action of pulmonary slowly adapting stretch receptors, heart and lung fibroblasts, vascular endothelium, nervous system glia and neurons during voluntary deep breathing needs to be further investigated at the cellular level.

Conclusion

Slow pranayamic breathing generates inhibitory signals and hyperpolarizing current within neural and non-neural tissue by mechanically stretching tissues during breath inhalation and retention. It is likely that inhibitory impulses in cooperation with hyperpolarization current initiates the synchronization of neural elements in the central nervous system, peripheral nervous system, and surrounding tissues ultimately causing shifts in the autonomic balance towards parasympathetic dominance. Further experimental research of the cooperative cellular mechanisms of pranayama is needed to confirm this theory.

References

[1] Yardi N. Yoga for control of epilepsy. Seizure 2001;10(1): 7—12.
[2] Chodzinski J. The effect of rhythmic breathing on blood pressure in hypertensive adults. J Undergrad Res 2000;1(6).
[3] Singh S et al. Role of yoga in modifying certain cardiovascular functions in type 2 diabetic patients. J Assoc Physician India 2004;52:203—6.
[4] Harinath K et al. Effects of Hatha yoga and Omkar meditation on cardiorespiratory performance, psychologic profile, and melatonin secretion. J Altern Complement Med 2004;10(2):261—8.
[5] Cooper S et al. Effect of two breathing exercises (Buteyko and pranayama) in asthma: a randomised controlled trial. Thorax 2003;58(8):674—9.
[6] Singh V, Chowdhary R, Chowdhary N. The role of cough and hyperventilation in perpetuating airway inflammation in asthma. J Assoc Physician India 2000;48(3):343—5.
[7] Vedanthan P et al. Clinical study of yoga techniques in university students with asthma: a controlled study. Allergy Asthma Proc 1998;19(1):3—9.
[8] Pal G, Velkumary S, Madanmohan. Effect of short-term practice of breathing exercises on autonomic functions in normal human volunteers. Indian J Med Res 2004;120(2): 115—21.
[9] Brown R, Gerbarg P. Sudarshan Kriya Yogic breathing in the treatment of stress, anxiety, and depression: part II—clinical applications and guidelines. J Altern Complement Med 2005;11(4):711—7.
[10] Malhotra V et al. Study of yoga asanas in assessment of pulmonary function in NIDDM patients. Indian J Physiol Pharmacol 2002;46(3):313—20.
[11] Raju PS et al. Comparison of effects of yoga & physical exercise in athletes. Indian J Med Res 1994(100):81—6.
[12] Desai B, Gharote M. Effect of Kapalabhati on blood urea, creatinine and tyrosine. Act Nerv Super (Praha) 1990;32(2): 95—8.

[13] Telles S et al. Alterations of auditory middle latency evoked potentials during yogic consciously regulated breathing and attentive state of mind. Int J Psychophysiol 1993;14(3):189—98.

[14] Raghuraj P et al. Pranayama increases grip strength without lateralized effects. Indian J Physiol Pharmacol 1997;41(2):129—33.

[15] Bijlani R et al. A brief but comprehensive lifestyle education program based on yoga reduces risk factors for cardiovascular disease and diabetes mellitus. J Altern Complement Med 2005;11(2):267—74.

[16] Bhattacharya S, Pandey U, Verma N. Improvement in oxidative status with yogic breathing in young healthy males. Indian J Physiol Pharmacol 2002;46(3):349—54.

[17] Uma K et al. The integrated approach of yoga: a therapeutic tool for mentally retarded children: a one-year controlled study. J Ment Defic Res 1989;33(5):415—21.

[18] Taneja I et al. Yogic versus conventional treatment in diarrhea-predominant irritable bowel syndrome: a randomized control study. Appl Psychophysiol Biofeedback 2004;29(1):19—33.

[19] Telles S et al. Plasticity of motor control systems demonstrated by yoga training. Indian J Physiol Pharmacol 1994;38(2):143—4.

[20] Telles S, Desiraju T. Oxygen consumption during pranayamic type of very slow-rate breathing. Indian J Med Res 1991;94:357—63.

[21] Telles S. Breathing through a particular nostril can alter metabolism and autonomic activities. Indian J Physiol Pharmacol 1994;38(2):133—7.

[22] Collins M, Dunn L. The effects of meditation and visual imagery on an immune system disorder: dermatomyositis. J Altern Complement Med 2005;11(2):275—84.

[23] Davidson R et al. Alterations in brain and immune function produced by mindfulness meditation. Psychosom Med 2003;65(4):564—70.

[24] Lee M et al. Effects of Qigong on immune cells. Am J Chin Med 2003;31(2):327—35.

[25] Takahashi T et al. Changes in EEG and autonomic nervous activity during meditation and their association with personality traits. Int J Psychophysiol 2005;55(2):199—207.

[26] Bhargava R, Gogate M, Mascarenhas J. Autonomic responses to breath holding and its variations following pranayama. Indian J Physiol Pharmacol 1988;32(4):257—64.

[27] Telles S, Nagarathna R, Nagendra H. Physiological measures of right nostril breathing. J Altern Complement Med 1996;2(4):479—84.

[28] Johnson D, Tierney M, Sadighi P. Kapalabhati pranayama: breath of fire or cause of pneumothorax? a case report. Chest 2004;125(5):1951—2.

[29] Kallenbach J et al. Reflex heart rate control in asthma. Evidence of parasympathetic overactivity. Chest 1985;87(5):644—8.

[30] Gopal K et al. The cardiorespiratory adjustments in 'pranayama', with and without 'bandhas', in 'vajrasana'. Indian J Med Sci 1973;27(9):686—92.

[31] Iyengar BKS. Light on pranayama: the yogic art of breathing. New York: Crossroad; 1998.

[32] Austin JH. Zen and the brain. Cambridge (MA): MIT Press; 1998.

[33] Pratap V, Berrettini W, Smith C. Arterial blood gases in pranayama practice. Percept Mot Skill 1978;46(1):171—4.

[34] Busek P, Kemlink D. The influence of the respiratory cycle on the EEG. Physiol Res 2005;54:327—33.

[35] Sydorchuk L, Tryniak M. Effect of voluntary regulation of the respiration on the functional state of the autonomic nervous system. Lik Sprava 2005(1—2):65—8.

[36] Matsumoto S et al. Inhibitory mechanism of slowly adapting pulmonary stretch receptors after release from hyperinflation in anesthetized rabbits. Life Sci 2000;67(12):1423—33.

[37] Schelegle E, GreenSchelegle J. An overview of the anatomy and physiology of slowly adapting pulmonary stretch receptors. Respir Physiol 2001;125(1—2):17—31.

[38] Kamkin A et al. Electrical interaction of mechanosensitive fibroblasts and myocytes in the heart. Basic Res Cardiol 2005;100(4):337—45.

[39] Siegelbaum R, Robinson S. Hyperpolarization activated cation current: from molecules to physiological function. Annu Rev Physiol 2003:65.

[40] Roberts L, Greene J. Hyperpolarization — activated current (Ih): a characterization of subicular neurons in brain slices from socially and individually housed rats. Brain Res 2005;1040(1—2):1—13.

[41] Cuttle MF et al. Modulation of a presynaptic hyperpolarization — activated cationic current (Ih) at an excitatory synaptic terminal in the rat auditory brainstem. J Physiol 2001;534(3):733—44.

[42] Westbrook GL. In: Kandel ER, Schwartz JH, Jessell TM, editors. Principles of neuroscience. New York: McGraw-Hill; 2000.

[43] Newberg A, Iversen J. The neural basis of the complex mental task of meditation: neurotransmitter and neurochemical considerations. Med Hypotheses 2003;61(2):282—91.

[44] Lutz A et al. Long-term meditators self-induce high-amplitude gamma synchrony during mental practice. Proc Natl Acad Sci USA 2004;101(46):16369—73.

[45] Tzeng Y, Larsen P, Galletly D. Cardioventilatory coupling in resting human subjects. Exp Physiol 2003;88(6):775—82.

[46] Migliore M, Messineo L, Ferrante M. Dendritic Ih selectively blocks temporal summation of unsynchronized sistal inputs in CA1 pyramidal neurons. J Comput Neurosci 2004;16(1):5—13.

[47] Benson H. The relaxation response: the therapeutic effect. Psychiatry 1974;37:1694—5.