Yogic Breathing and Meditation: When the Thalamus Quiets the Cortex and Rouses the Limbic System

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This paper presents a neurophysiological model for the effects of yogic breathing techniques, called Pranayama (including Ujjayi, Bhastrika, and Sudarshan Kriya), chanting, and meditation as taught in Art of Living (AOL) courses. Methodologic challenges in future studies of yogic breathing include choice of control groups, blinding and randomization. Functional MRI (magnetic resonance imaging) is the best current modality for studying the brain during Pranayama, chanting and meditation. Directions for future clinical research in neuropsychiatric conditions will be discussed.

Sudarshan Kriya yoga has been most studied by Dr. Janakiramaiah and colleagues at NIMHANS (National Institute of Mental Health and Neurosciences), particularly for the treatment of dysthymia and depression.[1,2] They describe three sequential breathing cycles separated by 30 second periods of normal breathing: Ujjayi - slow breathing 3 cycles per minute; Bhastrika - rapid exhalation at 20-30 cycles per minute; and Sudarshan Kriya - rhythmic, cyclical breathing of slow, medium and fast cycles. Between Bhastrika and the Sudarshan Kriya there is brief chanting. These variations of rhythmic breathing are practiced while sitting with eyes closed and awareness focused on the breath. A state of relaxed sleepiness comes by the end of the cyclical breathing and the process ends with a ten-minute rest in a tranquil supine position. This can be followed by a 20-minute meditation. The breathing practice enables meditation to proceed with much less effort than otherwise. We will correlate data on each method of breathing with related research on hyperventilation, slow breathing, vagal nerve stimulation, and meditation to further understand their effects on the brain.

Ujjayi Breathing

Ujjayi breathing is a strained, somewhat forceful inspiration and expiration against airway resistance using a slow breathing cycle of 3-4 breaths per minute. Fokkema reviewed the literature in animals and humans on strained breathing and its cardiovascular and behavioral affects[3]. Active behavior is inhibited in animals during strained breathing. It occurs naturally under stressful circumstances. Muscle tension and laryngeal reflexes cause a strong reduction of airflow in the glottis resulting in prolonged expiration/inspiration, and an elevated intrathoracic pressure. The resulting increases in blood pressure and carbon dioxide (CO2) level further stimulate the strained breathing pattern. This causes increased brain perfusion and prevents hyperventilation. Renal sodium excretion is decreased. Ujjayi induces oscillations of blood pressure and an exaggeration of the normal respiratory sinus arrhythmia. It also stimulates vagal nerve afferents to the brain, ultimately increasing attention and vigilance. In nature, Ujjayi most often occurs in an animal that has been defeated in battle by a dominant animal or when it is confronted with a dominant animal by whom it had previously been defeated. The original stimulus for the breathing pattern derives from the hypothalamic vigilance area, which is separate from the hypothalamic defense area. In other words, the animal, if unable to escape and use fight or flight reflexes, becomes extremely vigilant in order to protect itself. Stimulation of the vagal efferent neurons induces a parasympathetic reduction in heart rate and most likely a withdrawal of sympathetic input to the heart. This restores energy in preparation for activity. A strong respiratory rhythm in the vagus nerve is set up, such that enhanced respiratory sinus arrhythmias occur; these have been noted in anxious patients awaiting surgery, in rats during submissive behavior, and in advanced yogis during meditation[4]. An increase in the tidal volume during Ujjayi breathing probably prevents a tendency toward hypoxia/hypercapnia (decreased O_2/increased CO_2), which might otherwise occur during hypoventilation. Further research would be valuable to investigate concurrent blood gas changes. Similar breathing in animals is associated with arousal, attentiveness, tension, and submission. Enhanced respiratory sinus arrhythmias, particularly with low heart rate, occur in rats and humans during perceptual tasks requiring enhanced attention, especially when an immediate response is not needed.

Strained breathing can be overdone. It may occur as a component of cardiovascular disease, obstructive sleep apnea, and high altitude mountain sickness. Aside from these pathological conditions, it probably functions to inhibit an activated physiology and restore energy reserves. This has survival value in circumstances in which enhanced attention is required and/or during defensive behavior when prolonged mental activation cannot immediately lead to physical activity. It also enhances cerebral perfusion necessary for very active mental processing.

Bhastrika

A series of studies reported that Pranayama exercises (including Bhastrika, Kapalabhati, Nauli, and Agnisara) led to central nervous system (CNS) excitation. Roldan and Dostalek described three major excitatory patterns on the brain EEG including a peculiar arc or wicket rhythm of 12 to 17 Hz with maximal amplitude over the parietocentral region of the non-dominant hemisphere, a 26 to 33 Hz sinusoidal rhythm, and a high amplitude paroxysmal activity similar to

recordings made during orgasm in humans and animals[5 6]. The subjective experience is one of stimulation followed immediately by relaxation. The mechanism probably involves stimulation of visceral (including vagal) afferents. Kapalabhati increased the low mid frequency component of heart rate variability (an indirect measure of sympathetic nervous system function) while alternate nostril breathing had complex effects, apparently increasing both sympathetic and parasympathetic tone (measured by the high and low frequency component of heart rate variability)[7].

The highly active rhythms observed during Bhastrika and other excitatory forms of pranayama are similar to the gamma-frequency band of neuroactivity described in other situations. The gamma band is hypothesized to reflect synchrony of neural assemblies involved in integration of perceptions of various features of an object within a sensory modality, across modalities and over time, i.e. a form of accumulated complex knowledge. For further discussion, see Kwon[8].

Summary of Breathing Practices

To review at this point, the practice begins with *Ujjayi* breathing. People's subjective experience is one of being calmed. The proposed neurophysiology based on the current data involves a shift to parasympathetic dominance through vagal stimulation. Although I could find no EEG studies during this breathing state, we might expect that there would be an alpha rhythm and that brain imaging would probably show a quieting of executive pathways in the frontal cortex, pathways involved in planning, anticipation, worry, and the carrying out of plans of action.

Bhastrika seems to be excitatory with activation of temporo-parietal cortical areas. The subjective experience is one of excitation during the breathing followed by emotional calming with mental activation and alertness.

Sudarshan Kriya and Hyperventilation: Potential Mechanisms Underlying Clinical Effects

Although there are no studies recording EEG effects or brain imaging during Sudarshan Kriya, there is a large body of data on voluntary hyperventilation in humans, mechanical ventilation in brain injured patients, animal models of hyperventilation under anesthesia, and in vitro preparations of hippocampal slices. Clinically hyperventilation has been used either to activate the EEG in order to identify epileptic patients or to reduce pressure in the brain after traumatic brain injury.

An excellent review by Patel and Maulsby found *no substantive evidence to support the theory* (which is still quoted in literature from old research) *that EEG slowing with hyperventilation is due to cortical hypoxia secondary to cerebral vasoconstriction and/or secondary to hypocapnia* (decreased level of CO_2).[9] In fact, the intact thalamus is necessary for the hyperventilation response and hypocapnia produces decreased activity in the mesencephalic reticular formation. They propose that these two phenomena are associated with the slow wave states found during EEG activation by hyperventilation, which are similar to drowsiness (practitioners of Sudarshan Kriya commonly experience drowsiness during or at the end of the hyperventilation cycle followed by an "edge of sleep" state or indeed sleep, at least in the beginning of training). They suggest that the vasoconstriction observed during mechanical hyperventilation may reduce cerebral blood flow by about 50% due to a central neurogenic response to hypocapnia at a brain stem level. This depressant effect on the mesencephalic reticular formation is associated with depression of cortical activity and slow waves.

The most common EEG response to hyperventilation in a normal individual consists of diffuse slowing; in adults this response is most prominent at the vertex of the brain. Slowing will disappear within a minute after stopping hyperventilation. In patients prone to epilepsy, focal slow wave activity is enhanced, particularly in the temporal region and 3 Hz spike and wave activity may be detected only during hyperventilation.

Balzamo and colleagues, reviewing prior data in addition to their own research, show that the diffuse slowing response to hyperventilation can be abolished by lesions of the non-specific thalamic projection system (NSTPS) and the thalamus or by destruction of both vagal nerves.[10] Peripheral vagal afferents from the respiratory system play a major role in EEG changes caused by artificial hyperventilation and they probably input to the NSTPS.

Tomita-Gotoh and Hayashida recorded direct shifts in electrical potential from scalp electrodes when they induced hypocapnia and hypercapnia in human subjects. Hyperventilation increased cortical excitability with hypocapnia (decreased CO_2)[11]. Seyal, et al. showed widespread increased excitability as measured by motor evoked potentials with hyperventilation in six human subjects. They also noted that hyperventilation improved visual function in patients with multiple sclerosis[12]. Kukumberg and associates found increased cortical excitability in ten human subjects[13]. Hyperventilation also increases the excitability of skin and motor nerves. In experimental animals, hyperventilation increases excitability of hippocampal neurons. The hippocampus is responsible for attention and orientation. Carbon and associates most recently have shown that fifteen minutes of hyperventilation increased sensorimotor cortex excitability measured by direct current magnetoencephalography[14].

The peripheral physical effects of voluntary hyperventilation include modest hypocapnia and normal PO2 leading to increased cardiac output, increased renal blood flow, increased lithium and sodium excretion, and a minor increase in renal sympathetic activity.[15] Djarova and colleagues showed that three minutes of hyperventilation produced a brief increase in cortisol and human growth hormone in 11 men[16].

A major concern in using Sudarshan Kriya in large groups of patients with possible medical problems and/or advanced age derives from a controversy in the treatment of traumatic brain injury. The issue is about whether the cerebrovascular constriction and consequent hypoxia might induce significant ischemia that would be counterproductive in brain injury patients. However, in a PET scan study, brief moderate hyperventilation did not decrease cerebral metabolism in patients with traumatic brain injury[17]. Hyperventilation used in this study was more intense than what would be experienced during Sudarshan Kriya.

In a rat hippocampal slice model, hyperventilation with hypocapnia led to strong stabilization of gamma oscillations in the rat hippocampus, probably due to increased effectiveness of GABA (gamma-amino butyric acid) inhibitory networks in the cortex[18]. We have noted gamma rhythms previously in relationship to Bhastrika and it is possible that Sudarshan Kriya may produce gamma rhythms after initial activation and appearance of delta and theta rhythms. Gamma rhythms are necessary in sensory integration and higher cognitive function.

Posse, et al. showed that hyperventilation quieted the frontal and parietoccipital cortex but not subcortical areas on functional MRI imaging[19]. Voluntary hyperventilation did not decrease cerebral oxygen use in normal subjects[20]. Although the form of hyperventilation used in their study was much more intense than during Sudarshan Kriya, it is relevant.

Using electroencephalographic measures of activity from somatosensory and auditory cortex, Huttunen and colleagues showed that voluntary hyperventilation suppressed long-latency evoked responses from the cortex while early short-latency responses were less reduced. They suggested that the increased cortical excitability caused by hyperventilation-induced hypocapnia led to spontaneous firing of cortical neurons. They also noted studies showing enhancement of excitatory glutamate synaptic transmission with concurrent suppression of GABA receptor-mediated postsynaptic inhibition.[21]

The underlying basis of this cortical excitability, particularly in the sensori-motor cortex is probably due to thalamic activation (see figure 1). Using PET scans, Prevett and associates showed that hyperventilation increased blood flow to the thalamus in patients with generalized epilepsy in whom typical absence seizures were induced by voluntary hyperventilation[22].

Marrosu and co-workers showed that SPECT measures of cerebral blood flow correlated with hyperventilation-enhanced interictal lateralized discharges in epileptic patients[23]. They suggested that hyperventilation-induced slow activity on EEG is probably the result of synchronous activity in non-specific thalamocortical projecting systems (see Patel and Maulsby). However, in epileptics other mechanisms also affect transition from slow waves to spike-wave discharges.

Acute cognitive effects of hyperventilation have been studied for many years. Two recent reviews show that hyperventilation briefly decreases attention and cognitive function[24, 25]. Reviewing studies of Pavlovian and operant control of emotion and cognition through modification of breathing behavior, Ley describes the complex interrelationship between breathing and cognition and emotion. Two examples of cognitions and emotions associated with specific breathing patterns are: surrender with Ujjayi breathing and excitement with hyperventilation. However, the process can also work in reverse: specific breathing patterns such as the unique rhythmic cycles of Sudarshan Kriya can induce particular cognitions and emotions.

Slow Breathing and Asanas

Spicuzza and colleagues compared the response of ten experienced yoga practitioners (with an average of eight years of practice) with twelve healthy controls who had never practiced yoga[26]. Slow yoga breathing decreased chemoreflex (changes in breathing rate in response to changes in the pressure of O_2 and CO_2 in the blood) responses to hypoxia and hypercapnia in both groups. Their data also suggest that long term practice of yoga breathing independently reduces chemoreflex sensitivity as opposed to just slow breathing. They hypothesized adaptation of peripheral/central chemoreceptors to chronic CO_2 retention or adaptation of pulmonary stretch receptors to a habit of deep slow respiration which would reduce vagal afferent discharge to bulbopontine brainstem centers that send projections to the thalamus and limbic systems. These effects are calming and reduce the risk of cardiovascular disease.

In healthy elderly people the effects on the baroreflex of yoga postures (Asanas) and breathing (not clearly specified) were compared to aerobic exercise training. Six weeks of training of three hours a week in yoga led to a decreased heart rate, increased vagal tone, and an increase in aerobic capacity of approximately 11 percent. The yoga improved baroreflex sensitivity whereas aerobic training did not.[27] Khanam showed that yoga training (Asanas and Pranayama) for one week decreased sympathetic reactivity in asthma patients and improved pulmonary ventilation[28].

Related to this are observations of unusual autonomic control by yogi adepts. When asked to describe how mastery of autonomic function was accomplished, one yogic master said that one must start by developing a slow even breathing at the rate of one or two breaths per minute and that regulation of the respiratory cycle is the most important step in controlling afferent vagal nerve fibers to attain control of the autonomic nervous system. Even in untrained subjects, slow breathing at eight cycles per minute versus fifteen and thirty per minute prevents a suppressive effect on parasympathetic input to the heart under the threat of an electric shock[4]. The consequent enhancement of vagal tone reduces the effects of stress on the heart.

Benson and colleagues correlated slow breathing observed during meditation in eight Chi Kung and four Kundalini yoga meditators with dramatic heart rate oscillations. The amplitude of oscillations was greater than in the premeditation state. It was similar to but greater than that found in elite athletes and healthy adults during sleep. They suggest that this "autonomic exercise" may benefit the heart. We will return to this point in the discussion of chanting.[29] Slow breathing improves baroreflex sensitivity which tends to become abnormal with aging, cardiac disease, and hypertension.

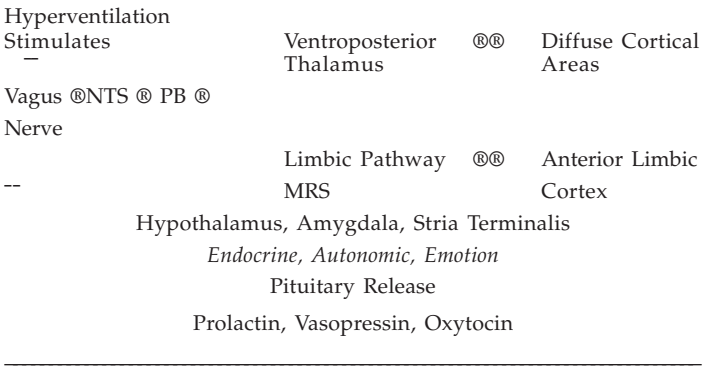
Vagal Nerve Stimulation

Unilateral vagal nerve stimulation (VNS) is an effective treatment in many cases of epilepsy and a promising experimental treatment for depression. Data on VNS is relevant to hyperventilation because both stimulate vagal afferents to the thalamus, exerting powerful effects on excitability of the cortex as well as producing an edge of sleep state (For a review of VNS effects in epilepsy see Schachter and Saper).[30] Electrical stimulation is only applied to the left vagus nerve because stimulation of the right vagus nerve would cause undesirable cardiac effects. Vagus nerve afferents synapse in the nucleus

tractus solitarius (NTS) (see figure 1). The NTS ascends to the parabrachial nucleus which diverges into two pathways. One inputs to the hypothalamus, amygdala, stria terminalis, and limbic cortex, affecting autonomic, endocrine, and emotional control. The other pathway from the parabrachial nucleus goes to several thalamic nuclei and thence to the cortex. Thalamic intralaminar and mid-line nuclei project diffusely to the cerebral cortex and probably influence cortical synchronization or desynchronization. In animals slower stimulation (1 to 17 Hz) causes EEG synchronization while high frequency stimulation (greater than 30 Hz) results in EEG desynchronization. Two PET studies of VNS on activation of the human brain reported thalamic activation and inconsistent results in areas of the cortex and subcortical structures. Rhoden's studies suggested that VNS raised the seizure threshold by releasing GABA and glycine in wide spread brain structures[5].

The use of biofeedback to produce what has been called the ***SMR (sensori-motor rhythm)*** pattern has been shown to decrease seizure frequency (For a review and discussion of SMR in treating seizures see Sterman)[31]. Sterman and colleagues found an EEG pattern of 12 to 20 cycles per second localized to the sensory motor cortex which they labeled SMR for "sensori-motor rhythm." Behavior associated with SMR included suppressed physical activity and increased vigilance. Inhibition of movement was necessary but not sufficient for production of the SMR rhythm which is preceded by a six-second period of muscle decreased tone. SMR was found to originate in the somatosensory relay nucleus of the thalamus (the ventrobasal nucleus). Furthermore, SMR was associated with suppression of muscle tone, suppression of further somatosensory information through the thalamus to the cortex, and stretch reflex excitability.

Figure 1. Vagus Nerve Stimulation



NTS = nucleus tractus solitarius; PB = parabrachial nucleus; MRS = mesolimbic reward system

The ventrobasal nuclei relay information not only to the cortex, but also to related cells in the adjacent thalamic nucleus called the nucleus reticularis. Oscillations of activity between these two nuclei set up the SMR recorded on the cortex. Producing SMR in humans requires a decrease in body and eye movement. Training mammals, including humans, to produce this rhythm, resulted in a more focused state, resistance to seizures, and improved memory, vigilance, attention, and sleep. A different EEG pattern (4 to 12 Hz rhythm in the parietal cortex) was found to be associated with reward and food consumption. This theta, slow wave pattern was named ***"post-reinforcement synchronization"*** or ***PRS***. Similar EEG activity occurs during drinking, grooming, and drowsiness. PRS is also related to a thalamocortical gating mechanism involving lateral geniculate, posterior dorsal and other thalamic reticular nuclei. This can occur prior to sleep onset and during certain stages of sleep. PRS may also be seen with chemical interference with GABA inhibition on thalamic relay neurons resulting in seizures.

Sterman's group found that when an animal became satiated and sat quietly, alternating SMR and PRS frequencies could be seen in the sensory motor cortex and the ventrobasal thalamus. They hypothesized that the alternation of SMR and PRS frequencies in mammals reflected excitation-inhibition sequences in thalamocortical circuits that efficiently code and integrate information and then reset the system for the next challenge. I would further suggest that the SMR pattern represents vigilance and a search for reward. The PRS sequence is associated with satiety and pleasure. In humans, PRS was associated with a dreamy "edge of sleep" state, appearance of normally suppressed images and emotions, and reduced cortical excitability. Training humans to produce SMR resulted in improved sleep patterns, improved memory functions, better attentional and organizational skills in patients with attention deficit disorder, and enhanced seizure control in epileptics. It was also useful in the treatment of substance abuse.

Sterman notes that the "edge of sleep" state is therapeutically useful in allowing suppressed emotional content to emerge. He suggests that withdrawal of brain stem and normal thalamocortical regulatory influences leads to relative cortical disinhibition. This allows suppressed emotion, cognitions, and trauma memories to be evoked while providing a state of calmness and relaxation that reduces the risk of retraumatization.

A pilot study by Bhatia and colleagues compared 19 Art of Living teachers who regularly practice Sudarshan Kriya with 15 control subjects. EEGs of AOL teachers showed increased beta activity (similar to SMR) in the left parieto-occipital region indicating cortical activation by the underlying ***thalamic generator*** (set of neurons in the thalamus that sets up a clear rhythm in a related cortical area). This occurred with increased alpha activity suggesting increased calm and relaxation combined with increased vigilance and attention[32].

For a more complete understanding of the relationship of cortical EEG activity to underlying subcortical activity, neurotransmitters and subjective states, see Lubar[33]. Lubar discusses three kinds of cortical resonance loops (see figure 2). ***Local*** resonance occurs between one narrow cortical column and the next and produces high frequency EEG patterns above 30 Hz such as gamma. ***Regional*** resonances develop between macrocolumns (groups of narrow cortical columns) that are near each other and produce intermediate frequencies such as alpha and some beta. ***Global*** resonances can develop between widely separated regions such as the frontal-parietal or frontal-occipital. These produce resonances in the delta and theta range. All three types of resonance can occur spontaneously or be stimulated by thalamic generators. ***Coherence*** is a measure of the degree to which any two areas

in the cortex are functionally connected. Low coherence means that two areas are functionally disconnected. High coherence implies that they are related in function, but not differentiating the functions adequately. In general, the posterior cortex is more involved in local functions dealing with visual processing and image formation.

The left hemisphere is involved in analytical and sequential processes. The right hemisphere is more involved in intuitive synthetic processes. EEG activity in the delta/theta sleep spindle mode is associated with a low frequency burst mode through the non-specific thalamocortical projection system to layer 1 of the cortex (see figure 2).

Figure 2. Neocortical Resonance Modes

EEG: Delta - - Sleep Spindle - - Alpha - - Mu - - Beta - - Gamma	
Hypercoupled States - - - Optimal Coupling - - - Hypocoupled States	
Low frequency burst	Single action potential mode
Non-specific thalamocortical	Specific thalamocortical
Neocortical layer 1	Neocortical layer 4
Brainstem Neurotransmitters	
Serotonin	
Ach	Nor Epinephrine Dopamine
Global Resonances:	Local, Regional Resonances:
Visual Imagery	**Attention, Orgasm, Bliss**

Adapted with permission. Lubar, 1997 [33]

These may be conceptualized as hypercoupled states and often involve acetylcholine, serotonin, and norepinephrine. Faster frequencies, such as beta and gamma, operate through specific thalamocortical projections, particularly to layer 4 of the neocortex. Weak associations (hypocoupled) between different areas of the cortex having predominantly local resonances are more reliant upon dopamine.

Chanting

Breathing practices are followed by a brief chanting of "Om." Our model hypothesizes that the seemingly simple Om chant has complex effects on the brain. The verbal stimulation and the vibrational component of the chant probably contribute to activation of Wernecke's area and the thalamus. However, other complex physiologic effects are contributory. Telles, et al. studied seven experienced yogic meditators during a control period and then while mentally chanting Om. They showed decreased metabolism, decreased heart rate, and increased peripheral vascular resistance. These findings were interpreted as a sign of increased mental alertness in the context of physiological relaxation.[34]

Significantly, Bernardi and co-workers showed that a simple yogic chant (as well as the traditional Latin version of the Hail Mary) decreased the breathing rate to approximately six times per minute, synchronizing it with the inherent six times per minute natural fluctuation in blood pressure known as Mayer's waves.[35] In 1876 Mayer described this ten-second cycle which is thought to be related to both vagal and sympathetic activity generated by a central nervous system oscillator in the medulla or possibly by the interaction of the baroreflex sympathetic response and the vagal response to respiratory changes in blood pressure. They showed marked effects on synchronization and increased variability in respiratory signals, cardiovascular rhythms, and cerebral blood flow velocity. This meant that sympathetic and parasympathetic (vagal) outflow were synchronized, also resulting in rhythmic fluctuations of heart rate and cerebral blood flow with improved heart rate variability and baroreflex sensitivity. (Changes in the opposite direction are poor prognostic factors in heart disease.)

Meditation and Pranayama

Many studies of meditation find that less experienced practitioners often fall asleep (lose consciousness). However, yogis are able to remain conscious despite the appearance the sleep spindles on EEG that are characteristic of sleep.[4] It can be hypothesized that those who train in meditation can attain theta- alpha states close to the edge of sleep while maintaining awakeness and alertness. Alternation or simultaneous activation of the thalamocortical generators responsible for the SMR and PRS patterns may be involved.

There are more studies on the physiology of transcendental meditation (TM) than any other meditation form, although there are some studies of yogic, Zen and Chi Kung. A 1992 review of the physiology of meditation by Jevning et al focusing on TM, found that meditation was associated with increased central nervous system activity, probably increased cerebral blood flow and increased cardiac output, yet a generally hypometabolic state.[36] Data suggesting increased plasma renin and prolactin were noted. Janakiramaiah described increased prolactin after only one Sudarshan Kriya and Pranayama session[1]. Jevning and colleagues found apparent cessation of CO2 generation by muscle, decreased meditator sensitivity to increased PCO2 and a dramatic 5-fold elevation of plasma vasopressin.[41] Subjectively deep states of meditation were accompanied by high amplitude theta and/or fast frequency beta bursts consistent with the alternation of SMR with PRS states. Other authors reviewed by them propose a role for the reticular activating system and specific thalamic nuclei in generating alpha activity in the frontal cortex with the hypothalamus causing the hypometabolic state observed during meditation. They postulate that meditation activates non-specific thalamic nuclei while blocking external sensory inputs. These authors did not know at that time about the data on SMR and PRS training. It should be emphasized that ideally meditation is a state of increased brain alertness. Relaxation training may increase cardiac parasympathetic tone [37], but not have other effects associated with meditation.

Two randomized controlled studies found that yogic meditation enhanced seizure control in epileptics.[38] Some theta frequencies may be associated with a tendency to seizure activity. Panjwani and colleagues showed that six months practice of yoga and meditation shifted EEG frequencies in epileptics from the theta range to beta/alpha with decreased seizure frequency.[39] Notably, this is similar to the EEG changes in the biparietal areas observed with SMR treatment with biofeedback, and with valproate (anticonvulsant) administration.

Travis and colleagues presented data showing that 11 experienced TM practitioners showed greater theta/alpha power during stages three and four sleep compared to nine short-term practitioners and 11 non-practitioners. They also had increased rapid eye movement density during REM periods.[40] The subjective experience of "transcending" during meditation was marked by low breath frequency, higher respiratory sinus arrhythmia amplitudes, higher EEG alpha amplitude, and alpha coherence.[41] A somewhat different analysis of the data might have also shown increased theta activity in certain circuits as was found by Aftanas and Golocheikine in 16 long-term and 1 short-term meditators using the Sahaja yoga meditation.[42] Their study, using more complex spectral analysis of the EEG showed that blissful states during meditation correlated with left anterior frontal theta coherence in experienced meditators as well as coherence between anterior mid-line frontal areas (like the sensory motor cortex) and posterior association areas. These theta waves were different from the theta waves observed during normal sleep. Travis and Wallace proposed a two-phase model of TM, which is adequate to explain both findings.[43] They suggested that the first phase of meditation involves shutting down orbital and basal frontal cortex activity, leading to a quieting of mind and body. The second phase maintains the quieter levels of function while the emergence of thalamocortical generators results in enhanced attention in a state of consciousness not associated with processing specific perceptual and cognitive content. In what they called their attention/intention model, alpha synchrony (I would also say theta coherence during bliss or transcendence) indicates a state of intention of the mind toward itself (the mind turning its attention to itself). Travis and Wallace propose that the prefrontal cortex initially activates the nucleus reticularis of the thalamus to inhibit specific and non-specific activity in thalamocortical circuits. To maintain and deepen the meditative state, basal ganglia-thalamocortical circuits feed forward to the globus pallidus and then inhibit the mesencephalic reticular formation and the medial dorsal nuclei of the thalamus. This is relevant to the effect of Sudarshan Kriya on enhancing the ease of meditation because hyperventilation shuts off the mesencephalic reticular formation and stimulates thalamocortical circuits, which enhance meditation and wakefulness.

Illuminating the EEG findings is one PET study of meditation during a relaxation state (Yoga Nidra) versus normal consciousness in 9 experienced yoga teachers showed an activation of the hippocampus and a shutting off of cingulate and frontal cortex normally used in planning, anticipation, and other intellectual activity. This pattern is remarkably similar to the activation pattern during REM sleep except that in REM the anterior cingulate and hippocampus are active, whereas during yoga meditation the anterior cingulate is shut down. The meditative state was characterized by increased activity in the hippocampus responsible for orientation and the posterior sensory and associative cortical areas known to be activated by imagery whereas normal consciousness requires activity in the executive attentional system of the forebrain and the cerebellum.[44] Travis and Pearson postulate that the transcendent experience can be seen in the junction points between waking, dreaming, and sleeping and that this transcendent experience can be integrated with waking, dreaming, and sleeping with long experience in meditation.[45]

Although there have been no direct observations of yoga masters during blissful states of ecstasy, there is one EEG study of 7 famous Chi Kung masters in China that showed an EEG alpha activity predominantly in the anterior cortical areas with the peak frequency being slower than the normal resting state. This was suggested to be a state of increased excitation and is consistent with other findings during yoga meditation and TM.[46] Therefore, I would suggest that continued practice in meditation develops the ability to increase activity in the activated bands (alpha, beta, gamma) of the EEG and to increase dopamine stimulation (figure 2) through the mesolimbic reward systems (figure 1), which results in the experience of bliss. The seasoned practitioner can experience joy despite any sensory input and in any state of consciousness.

Endocrine Changes with Meditation and Pranayama

Endocrine changes have been associated with meditation and Sudarshan Kriya. A study of three months practice of Sudarshan Kriya resulted in significant reduction in cortisol (one measure of stress response system activation in the brain) and correlated significantly with decreased depression scores over a three-week period.[47] Another study of 12 highly trained yoga meditators (average 6-1/2 years experience) compared with 11 elite runners showed an increase in CRH (corticotrophin-releasing hormone) but not endorphins after meditation. The 11 elite runners showed the same magnitude CRH release after running as the meditators, but the runners also released a beta-endorphin not found in the meditators.[48] The hypothalamic pituitary adrenal (HPA) axis is essential in the stress response and survival of mammalian species. It is abnormally overactivated during biological depression and is alternately overactivated and depleted in patients with post-traumatic stress disorder.

There is data suggesting that meditation and probably hyperventilation cause release of pituitary hormones (figure1), probably through hypothalamic output mediated through vagal afferents. The 5-fold increase in vasopressin associated with meditation is significant[41]. There is older evidence that vasopressin is decreased in depression.[49] Furthermore, vasopressin and oxytocin receptors are generally found together in the brain. Oxytocin has been described as the "cuddle" hormone. It is very important for affiliative and monogamous social bonding in voles. It is known that stimulation of oxytocin in voles will result in increased bonding to the next vole with which that animal comes in contact with [50]. In humans, there is evidence that oxytocin is necessary for uterine contractions in childbirth, generates milk production in new mothers, influences bonding and affection of the mother for her baby [51], and is a primary sexual arousal hormone. There is some evidence of low oxytocin levels in major depression and of SSRIs (selective serotonin reuptake inhibitors) facilitating oxytocin secretion [52]. The antidepressant effect of SSRIs may be partially mediated by oxytocin. Further studies of oxytocin release during Sudarshan Kriya in depressed patients would elucidate the importance of this mechanism. I hypothesize that hyperventilation in Sudarshan

Kriya releases oxytocin as well as vasopressin. Thus oxytocin may turn out to be the "I belong to you" hormone.

Sudarshan Kriya and Depression

How might Sudarshan Kriya yoga treat depression? A considerable body of data suggests that biological depression is associated with excessive stress response system activation. Almost all effective antidepressants calm this system down. Preliminary evidence suggests that Sudarshan Kriya also quiets the stress response system. Furthermore, there is some evidence that the relationships between the two cerebral hemispheres, between the anterior and posterior parts of the brain, and between the top of the cortex and the subcortical regions are disturbed in depression. (For a review, see Kinsbourne.[53]) Pranayama practices probably help balance the activity between the cortical and subcortical regions. Sudarshan Kriya may work like electronic vagal nerve stimulation, which has been shown to be effective for depression. What role the increased parasympathetic and sympathetic activity induced by various Pranayama have in improving the function of the stress response remains speculative but is likely to be extremely important. Activation of forebrain reward systems may also play a role. Changes in acid base balance in the brain are less likely to be a significant part of the effects of the breathing.[9] Another unexplored phenomenon is that intense breathing (or indeed regular breathing) causes the expiration of oxidant chemicals from body metabolism called TBARS (thiobarbituric reactive acid substances). Whether detoxification of oxidants by exhalation through the lungs is beneficial for disorders involving excess oxidation damage (such cardiovascular and neurodegenerative disease) and whether this would enhance the effect of antioxidants in delaying aging, is purely hypothetical. Also hyperventilation increases renal output; whether this enhances excretion of other harmful substances is unknown.

Methodological Problems

What methodological problems face us in designing studies of Pranayama? The same criticisms applied to the study of yoga and epilepsy also confound the study of Pranayama in depression. [38] In most studies the number of subjects is small. The design is often an open series without adequate controls, without randomization or adequate double blinding. Some reports are based on single case studies. It is difficult to achieve a perfectly blind condition in yoga studies, particularly since trained yoga instructors must monitor patients in the learning and practice phases. However, outside raters could come in and evaluate patients blindly, i.e. without knowledge of their condition. Studies of imaging during Pranayama would benefit from using several modalities to enhance validity such as computerized EEG mapping in addition to functional MRI. At the current time, Functional MRI is better than PET scanning because of its increased resolution and the possibility of obtaining multiple serial measures.[19]

Another complication is that the Sudarshan Kriya and related Pranayama are taught in a basic Art of Living course which also involves aspects of group therapy and attitudinal changes such as how to live life and deal with stress. This introduces non-specific factors that may be curative in the treatment of depression, as opposed to a purely specific factor of breathing techniques. Nevertheless, my clinical impression is that these aspects are so beneficial that I would not advise depriving patients of these non-specific factors.

Even if Pranayama induces a short-term remission of depression, what is the long-term compliance rate? Are these practices too complicated and time consuming for most patients? That does not appear to be the case from studies in India by Janakiramaiah in which 80% of the subjects do continue to practice and benefit. A cross-cultural multi-center study would address the question of differences in compliance rates between India and America. In numerous studies of antidepressant medication the three-month dropout rate is around 50%.

Perhaps the biggest challenge is to design an adequate placebo or control for breathing. Should there be a chemical, inert placebo? Should there be a sham method of breathing such as the relaxed slow abdominal breathing often used in relaxation courses? Another significant variable in the effects of these practices on the brain involves comparison of very experienced practitioners such as instructors with those recently practicing and those who have never practiced the techniques using brain imaging and other measures.

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Healing Experience

Myself, Swadesh Kumar, working as a Forest Officer. I was suffering from high blood pressure and cold and taking medicines for it regularly. I did basic course of Art of Living in October 2001. I am doing Sudarshan Kriya daily. Now my blood pressure is normal and I am not taking any medicine.

Swadesh Kumar Dhiman,
D.F.O. Nahan,
Dist Sirmour, Himachal Pradesh.
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I was a patient of Chronic Allergic Bronchitis, this disease always keeps me depressed. The term "Chronic" indicated that I had to suffer all my life. Of course apart from this, my worried parents were always miserable seeing me in misery. I was on steroid inhalers and faced 100s of problems. I joined the Art of Living course because my friend asked me to do so as it was a good course and I just joined the basic course.

There was a total change in me. I have not touched inhalers. I am grateful to my lord Poojya Sri Sri Ravi Shankar Ji (Guruji)

As Guruji says world is beautiful I happen to see it now the world is really beautiful.

Jai Gurudev,

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