The Role of Autonomic Nervous System in Rapid Breathing Practices

Dr K.K. Deepak, *MD, PhD*

Additional Professor
Department of Physiology
All India Institute of Medical
Sciences, New Delhi

The respiration is a unique physiologic function, which is automatic. It is normally maintained without volition but could be modified to great extent by volition. It has an ability to modulate several visceral functions. The relationship in not one way, there are several physiological states like pain, emotion, and cardiovascular challenges that modify the respiration in a patterned manner. For this reason the respiration has emerged is an important subject matter for psychosomatic studies both as dependent parameter and also a modifier of psychophysiological states.

1. The effect of respiration on autonomic rhythms

In the autonomic nervous system various rhythms are present, these include very low frequencies (0.001 to 0.04 Hz, relates to thermoregulatory cycles), low frequencies (0.04 to .15 Hz, relates to sympathetic rhythm) and high frequencies (0.15 to 0.4 Hz, relates to parasympathetic rhythm). The relationship between the respiration and parasympathetic is manifested as sinus arrhythmia , the heightened form of which can be seen during deep breathing. Even at rest this relationship exists and is mainly due to central spill over of respiratory drive to cardiac centers in the brain, besides some peripheral factors. In our autonomic function lab, we are able to quantify these autonomic rhythms (Deepak et al 1999. Deepak et al 2000, Deepak et al 1996). Raghuraj and colleagues (1998) demonstrated that a type of rapid breathing (Kapalbhati) enhances the sympathetic drive to myocardium (Low frequency power of HRV), enhanced the sympathovagal balance towards sympathetic side. This was also accompanied by decreases in vagal tone (high frequency power of HRV). While alternating nostril breathing at slow pace did not change thease autonomic drives significantly.

Badra et al (2001) studied the influence of three types of breathing [spontaneous, frequency controlled at 0.25 Hz (i.e. 15 per minute), and hyperventilation with 100% oxygen] and apnea on R-R interval, arterial pressure, and sympathetic rhythms (measured by inserting microelectrodes in the muscle, called muscle sympathetic nerve activity or MSNA) in healthy young adults. They quantified autonomic rhythms for low (0.05-0.15 Hz) and high (0.15-0.3 Hz, this frequency falls in respiratory range) frequencies; estimated vagal baroreceptor-cardiac reflex gain at low frequencies and muscle sympathetic nerve spectra. Coherence among signals varied as functions of both frequency and time. Removing the frequencies of respiration did influence the high frequencies i.e. the parasympathetic activity. The mode of breathing did not influence low-frequency oscillations (sympathetic ones), and they persisted during apnea. This study proves the independence of low-frequency rhythms (sympathetic) from respiratory activity and suggests that the close correlations that may exist among arterial pressures, R-R intervals, and muscle sympathetic nerve activity at respiratory frequencies result from the influence of respiration on these measures. These are mediated through vagus. These results suggested that the correlation between respiration and autonomic rhythms is limited to parasympathetic rhythms.

Chemically mediated cardiovascular responses induce both hyperventilation and sympathetic activation. These responses are likely to have different effects on variability in cardiovascular system. The effect of hyperventilation on the interactions between changes in R-R interval (RR) and muscle sympathetic nerve activity (MSNA) and changes in neurocirculatory variability has been examined by Van De Borneet et al (2001) in four conditions: 1) controlled breathing, 2) maximal end-expiratory apnea, 3) isocapnic voluntary hyperventilation, and 4) hypercapnia-induced hyperventilation. The sympathetic drive to periphery (as measured by MSNA) increased by about 70% during apnea. This did not change during isocapnic hyperventilation but showed more than double increase during hypercapnic hyperventilation. The total parasympathetic drive to myocardium (as measure by HRV of R-R intervals) decreased during both isocapnic and hypercapnic hyperventilation. There was intense tachycardia during hypercapnic hyperventilation. This study categorically shows that respiration can induce oscillation in various autonomic drives, which lose their concordance (Van De Borneet et al 2001) under extremes of physiological conditions. Such changes in physiological limits may be forming potent stimuli to induce long-term changes (adaptations?) in the autonomic set-up.

1.1 Hemodynamic responses

Hyperventilation is a powerful physiological stimulus and has been used in epileptic patients to induce provocation to precipitate incipient seizures. Hyperventilation is also well known to affect the myocardial electrical activity in subjects without heart disease. It can produce spasm in patients with angina. The autonomic nervous system appears to play a mediator's role in these responses. A Greek study subjected 369 healthy volunteers (200 men, 169 women) to prolonged hyperventilation (30 breaths/minute for 5 min and 10 min recovery) under continuous ECG monitoring and to exercise testing (Alexopoulos and colleagues, 1995). Hyperventilation resulted in an immediate (within the first min), significant increase in heart rate, a further small increase at 2nd minute of

hyperventilation, and a subsequent small decrease in heart rate at later portion of hyperventilation period. A sudden reduction of about 20% in heart rate was observed on stoppage of hyperventilation. Males showed slightly higher increase in heart rate then females. The blood pressure showed rises with hyperventilation, with their highest value towards the end. The double product (heart rate-pressure product) increased by 43.6% with hyperventilation, a change that was only 19.1% of the respective rate-pressure product observed with exercise. This study proves that hyperventilation induces tremendous sympathetic stimulation thus, producing elaborate and dramatic hemodynamic response. The detailed analysis suggested that the smoking blunts hyperventilation induced hemodynamic response.

1.2 Local vascular changes

Hyperventilation induced chemoreflex not only induces powerful generalized autonomic changes but also induces local circulatory changes. In one study local blood flow in remote organs (the anterior tibial muscle and the skin of the calf) were measured during 3 minute prolonged hyperventilation by Steurer et al 1995. During hyperventilation, mean muscle blood flow increased in normal subjects as well as patients of hyperventilation syndrome. The changes of skin blood flux during hyperventilation were not significant. The blood flow response did not differ significantly in controls and patients. This is paradoxical response when we analyze this in light of generalized circulatory responses. During generalized responses, the blood pressure shoots up and there is intense sympathetic discharge (Alexopoulos et al, 1995, Van De Borne et al 2001). Possibly the release of vasoactive substances and/or a stimulation of the autonomic nervous system could have mediated the response observed by Steurer et al 1995. However it appears that local responses are compensatory adjustment to generalized changes induces by integrated autonomic reflexes.

1.3 Gastrointestinal effects

The gastrointestinal system is one system which is consistently responsive to the stress. Our lab has shown the altered autonomic functions in the patients of irritable bowel syndrome (Punyabati et al 2002), non-ulcer dyspepsia (Shelke 2001) and duodenal ulcers (Hoq et al 1998). As stated above the hyperventilation-induced hypocapnia affects hemodynamic Function. It also affects other organs namely the colon. Symptoms attributable to hyperventilation are common among patients with the irritable bowel syndrome(IBS). It has been suggested that hyperventilation may exacerbate the alimentary symptoms of IBS. Hyperventilation is a one of the regular experiment performed in the physiology classes in undergraduate teaching currently. Typical colonic sensation is common experience with the one who has performed hyperventilation. Probably the reduced carbon dioxide level increases the colonic motility. In an excellent study, Bharucha et al 1996 has tried to prove the relationship between humoral mediators, splanchnic blood volume and hypocapnia. In first part of study, colonic tone, sensation, plasma levels of cortisol, beta-endorphin, selected gut neuropeptides, norepinephrine, epinephrine, and splanchnic blood volume were measured during two

sequences of hypocapnic hyperventilation. In second part of the study colonic tone and sensation were assessed during isocapnic hyperventilation and abdominal compression. Hypocapnic hyperventilation, but not eucapnic hyperventilation or abdominal compression, significantly increased colonic tone and sensitivity to balloon distention without altering humoral mediators or splanchnic blood volume. Plasma norepinephrine level increased and splanchnic blood volume decreased during 5 minutes after hyperventilation, consistent with homeostatic responses (Bharucha et al 1996). In another study Ford et al (1995) have shown that Hypocapnic but not isocapnic hyperventilation produced an increase in colonic tone and phasic contractility in the transverse and sigmoid regions and an increase in pulse rate and pulse interval variability. The findings are consistent with inhibition of sympathetic innervations to the colon or direct effects of hypocapnia on colonic smooth muscle, or both. These physiological gut responses suggest that some of the changes in colonic function are caused by altered autonomic control mechanisms. It is likely that these effects of hyperventilation may be mediated from direct effect of CO_2 washout on colonic muscle. However, reflex changes from central autonomic control of colonic smooth muscle may not be ruled out.

2. Respiratory responses to autonomic challenges

In physiological systems, the two variables can influence each other depending on time of occurrence. As we know that the respiration influences the autonomic rhythms, it is also true the autonomic challenges are able to modify the respiration. Naschitz et al (2000) studied this equation. They compared the ventilatory responses to autonomic challenge evoked by upright tilting. The upright tilting is best-known stimulus to increase the sympathetic activity. In our lab we have demonstrated the effect of head-up tilting on the parasympathetic reactivity (Jahan et al 1996). Patients of chronic fatigue syndrome were tilted on a head-up tilt table after giving adequate rest in lying posture. During and after tilt the blood pressure (BP), heart rate (HR), respiratory rate (RR), and end-tidal pressure of carbon dioxide were measured The blood pressure, heart rate, respiratory rate, and during end-tidal pressure of carbon dioxide during lying posture were similar in both patients and controls. During tilt, patients with chronic fatigue syndrome developed significantly lower systolic blood pressure, diastolic blood pressure, and, and an end-tidal pressure of carbon dioxide. There were significant increases in heart rate and respiratory rate. The patients of chronic fatigue syndrome showed intense hyperventilation and postural tachycardia in response to postural stress. Thus, altered respiratory and cardiovascular responses may form good objective tests to assess chronic fatigue syndrome which otherwise is often missed for long period or patient receives unnecessary diagnostic test drills. This finding also indicates towards modifiability of respiratory function as a consequence of sympathetic challenge.

3. The emotion-autonomic responses-respiration relationship

A large body of literature is available to suggest the relationship between emotional exteriorization and respiratory changes.

Hyperventilation has been largely considered in theories of panic disorder. Wilhelm et al (2001) performed a peculiar study. They examined the predictive value of psychological and physiological parameters pertaining to voluntary hyperventilation. Physiological responses were recorded in 14 patients of panic disorder, 24 patients of social phobia, and 24 controls. The subjects/patients were made to exercise six cycles of 1-minute of fast breathing alternating with 1 minute of recovery, followed by 3 minutes of fast breathing and 10 minutes of recovery. Speed of fast breathing was kept at 18 cycles/minute by using audio-pacing, and depth aimed at achieving an end-tidal pCO2 of 20 mmHg by feedback. These values were achieved equally by all groups. During fast breathing, both groups of patients reported more anxiety than controls, and their feelings of difficulty in breathing. Skin conductance declined more slowly in panic disorder patients over the six 1-minute fast breathing periods. At the end of the final 10-minute recovery, panic disorder patients reported more awareness of breathing, dyspnea, and fear of being short of breath, and their pCO$_2$s, heart rates, and skin conductance levels had returned less toward normal levels than in other groups. Their lower pCO$_2$s were associated with a higher frequency of sigh breaths. The patients of panic disorder and social phobia showed more distress than controls to equal amounts of hypocapnia. The patients of panic disorder had delayed recovery of physiological parameters and symptoms than social phobia patients or controls. (Wilhelm et al 2001). Thus, the relationship between respiration and autonomic goes haywire in patients of panic disorder and social phobia, the same can be utilized for settling the diagnosis along with clinical symptoms. In another in-flight study on phobiacs, the respiratory rate and minute ventilation, indicators of hyperventilation, did not differ between phobiacs and controls, however, the phobiacs paused more during inspiration than did controls. Phobiacs also showed more skin conductance fluctuations and less respiratory sinus arrhythmia (Wilhelm and Roth 1988). These results certainly indicate that monitoring of multiple physiological parameters in the real environment is informative.

In an entirely diagonal connotation, enhanced breathing (hyperventilation) forms a part of a fight-or-flight response, and it gets stimulated when the person is extremely agitated. It is also seen when there are intense negative emotions. A study using emotional imagery found hyperventilation responses during imagery of high-arousal scenes. These emotional imagery scripts bring about autonomic changes. These autonomic changes may modulate the hyperventilatory responses. A research group for stress, health, and well-being at department of Psychology, University of Leuven has carried out a research (Van Diest et al 2001) to delineate the relationship between emotional changes and respiratory changes. They used four emotional scripts—depicting relaxing, fearful, depressive, and pleasant situations— without suggestions of autonomic or respiratory responses. After each imagery trial, participants rated their imagery. The results showed significant drop in end tidal carbon dioxide during the fearful and pleasant scripts. In the subjects who did not imagine concomitant autonomic changes (as suggested), this effect was much smaller. Subjects imagining script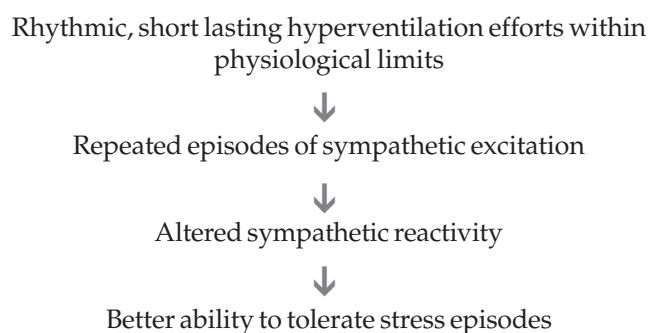s without autonomic response information found it harder to imagine the scripts vividly and reported lower levels of subjective arousal. This study suggests two important things firstly, there is a definite relationship among emotion, autonomic responses and respiration; second, the autonomic responses are important mediator emotion-induced respiratory changes irrespective of the type of emotions.

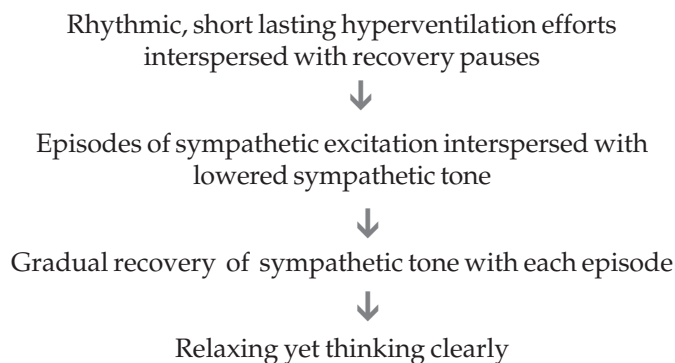## 4. Mechanisms of integration of respiratory induced psychosomatic changes and Clinical application

A Russian research investigated the autonomic instability (vegetocirculatory dystonia) and hyperventilation syndrome, who did not have any detectable organic lesion of nervous system. The scientists used a complex treatment method, which included the breathing exercises with feedback mechanisms, the correction of psychosomatic and vascular disturbances, the massage of neck region and a head, psychotherapy, angioprotective, vegeto- and psychotropic drug therapy. The disappearance of acute vascular attacks and episodes of migraine, the normalization of all breathing parameters were observed in all 35 patients. The study suggests that breathing practices can form a part of management strategies for illness.

We our selves have carried out a large study to document the autonomic changes during Sudarshan Kriya (a well defined sequence of rhythmic breathing with paced breathing at varying rates and depths interspersed with definite pauses, described elsewhere in this book). The results will be published soon. Several beneficial effects have been reported consequent to rhythmic rapid breathing accompanied by adequate pauses at varying degrees of breathing rate. This book elsewhere describes these positive changes. In pursuance to understanding of pieces of information cited above the following hypothesis is put forward to explain the beneficial effects of Sudarshan Kriya. Our physiological systems have immense capacity to adapt any intense physiological stimuli if it falls within the tolerance limits. The body system over a period attempts to blunt the response. This theory is largely evidenced by physiological system when they are exposed to altered environmental adverse situations like high altitude, chronic hypoxia, chronic thermal changes in the environment. How the Sudarshan Kriya can reduce stress levels over a period is an enigma to be answered? It is hypothesized that repeated episodes of rapid breathing results in hypocapnic internal milieu. This results in sequences of sympathoexicitation in a session or may have a repeated sequence over several sessions (Fig 1).

Fig 1

Rhythmic, short lasting hyperventilation efforts within physiological limits

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Repeated episodes of sympathetic excitation

↓

Altered sympathetic reactivity

↓

Better ability to tolerate stress episodes

Fig. 2

Rhythmic, short lasting hyperventilation efforts
interspersed with recovery pauses

↓

Episodes of sympathetic excitation interspersed with
lowered sympathetic tone

↓

Gradual recovery of sympathetic tone with each episode

↓

Relaxing yet thinking clearly

Thus, the physiological systems are going to oppose this stimulus and this may result in blunting of sympathetic response. The repeated episodes of this blunting may extend to other physiologic situation where sympathetic excitation plays important role in shaping the manifestation namely, the stress related sympathoexcitation. Thus, in a controlled situation, the Sudarshan Kriya may provide antidote to stress by physiologically counteracting the sympathetic effects. In a normal situation (in absence of stress) the practice of rapid breathing interspersed with adequate pauses of slow breathing, may provide tool for relaxation and vivid imagery (Fig. 2). How much the suggestibility contributes to these psychosomatic responses, is a matter of future study. Whatever the case may be the respiratory rhythm is a powerful physiological stimulus, which has a capacity to alter several psychosomatic functions.

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