Physiological and Biochemical Rationale of Yogasana

Vimal Singh Gusain[1], Anant Narayan Sinha[1], Joshil Kumar Behera[2]

[1]Associate Professor, [2]Assistant Professor, Department of Physiology, Veer Chandra Singh Garhwali Government Institute of Medical Science and Research, Srinagar, Uttarakhand (India)

Abstract

Yogic exercises have been known to increase mental and physical control of the body. Earlier practices of yogasan and pranayam have revealed physical and mental well being. Yoga has great therapeutic potential in management of related diseases stress. It improves the psyche of the individual because training causing decrease in psychic stimuli to vasomotor and respiratory centre hence there is less increase in sympathetic activity and less decrease in parasympathetic activity with an optimal blood flow distribution. Greater amount of fat is utilized for providing energy sparing glycogen.

Keywords: *Pranayama, Yogasana, Meditation.*

Introduction

With the fast expanding knowledge in various fields man has to toil not physically but mentally. The need for exercise both physical and mental, for total well being of an individual is no longer unknown to a common man. Physical exercise need to be included as a routine in our day-to-day life, as majority of us lead a sedentary life. Stress leads to generation of free radicals in animal muscle as evidenced by direct measurements of free radicals with the electron paramagnetic resonance technique and by indirect determination of product of free radical reactions. Antioxidant enzymes act directly or indirectly to remove reactive oxygen species and thus elevation of these enzymes with training suggests an increased demand for protection against free radicals. Such a practice leads to an increase in resting tidal volume, decrease in respiratory rate, increase in vital capacity and breath holding time.

Corresponding Author:
Dr. Anant Narayan Sinha
Assosiate Professor, Department of Physiology, Veer Chandra Singh Garhwali Government Institute of Medical Science and Research, Srinagar, Uttarakhand (India)
e-mail: ansinha1973@yahoo.in

Yogic exercise

Yoga can be divided into four main categories.

Raja yoga	- The mystical yoga
Karma yoga	- The path of selfless service
Bhakti yoga	- The path of devotion
Jnana yoga	- The yoga of knowledge

Raja yoga is said to be the king of yoga because it is directly concerned with the mind. A very important component in the Raja yoga practice is the pranayama. Pranayama is restraint of Prana or breath having three components.

Puraka	- inhalation of breath
Kumbhaka	- retention of breath
Rechaka	- exhalation of breath

The time taken for Inhalation/breath retention/ exhalation is kept at 1:4:2 in pranayama.

Various forms of pranayama

Pranayama	- inhalation and exhalation
Bhastrika	- hyperventilation for 10 seconds and then a deep breath

Kapal bhati	- same as in bhastrika but with forced expiration
Bvjjayi	- inhalation/retention, exhalation with glottis partially closed
Sitkari	- breathing through folded tongue
Sitakari	- breathing with a hissing sound
Suryabheda	- inhalation through left nostril
Bandhatraya	- Mule bandha controlling anus during inhalation
Jalandra Bandha	- press chin against chin in kumbhaka
Diana Banda	- draws up abdomen during exhalation
Kevala Kumbhaka	- constitutes an advanced form of pranayama

Pulmonary changes during exercise Udupa et al[1] showed reduction in body weight, improved lung function, decrease respiratory rate, increased vital capacity and breath holding time with yogic exercises. Similar results were also reported by Nayar et al[2]. Udupa et al[3] assessed biochemical parameters in 10 young adults after six months of training and found decrease in catecholamines, cholinesterase and blood sugar level. Increase in mono amine oxidase (MAO), diamine oxidase (DAO) plasma cortisol, serum protein levels has also been reported. Alexander [4] reported 11% increase in PEFR in patients given only relaxation therapy for bronchial asthma.

Sahay [5] found an increase in creatine phosphokinase (CPK) and decrease in pyruvate lactate rates, which was suggestive of increased muscular activity. Kulpati[6] followed 75 patients of COPD in three different groups. The first group received conventional treatment; second group did breathing exercises alone, while the third group did yogic exercises. Author reported that the group undertaking yogic exercises best maintained their lung function. Murlidhara [7] found a significant improvement in cardiac recovery index after 10 weeks of training. They inferred this to be due to para sympathetic predominance. Makwana et al [8] showed effect of short-term yogic practice on ventilatory function. An increased vital capacity, FEV and decreased respiratory rate was observed after 10 weeks of training.

Relationship of free radical generation with exercise: Exercise has a unique relationship with the free radical theory as during exercises when Vo_2 is elevated 10 to 15 fold above rest, it is very likely that free radicals are produced to a greater extent compared with rest. Considering that thousands of radicals produced in each resting cell every day, it is tempting to speculate on the number of free radicals that may be produced as a result of elevated metabolism. Furthermore, damage to active tissue is likely to occur and oxidative stress reactions are known to increase during exercise.

Oxidative stress is due to excess free radical generation in our body. A free radical is defined as a species molecule or atom capable of independent existence with unpaired electron (s) in its outermost orbital. A dot designates the presence of one or more unpaired electron eg. $O_2^{\cdot -}$. A radical might donate its unpaired electron to another molecule or accept an electron from another molecule in order to pair. The living beings are continually exposed to reactive oxygen species (ROS). Such a challenge comes from external noxious sources such as ionizing radiations, toxic drugs, chemicals, and environmental pollutants. The living cell is also capable of generating reactive oxygen species by itself and some cell types are ever specialized to do so. [9]

Potential mechanisms of free radical generation during strenuous exercise

- Mitochondrial electron chain
- Anoxia- reoxygenation
- Mechanical damage to the muscles
- Increased inhalation of environmental pollutants containing free radicals and/or initiators of free radical generating reactions in the body
- Oxidation of catecholamines

Despite exercise induced free radical changes there is a positive side to oxidative stress associated with regular exercise. An elaborate defense system providing varying degrees of cell protection against free radicals has evolved in all species. Select components of this defense system have been reported to increase in trained tissues following regular exercise. [10]

Potential mechanism of exercise mediated free radical production

There are several mechanisms that could potentially lead to the generation of free radicals during exercise. During oxidative phosphorylation in the mitochondria, oxygen is reduced by the mitochondrial electron transport system to generate ATP and water. However, during this process some of the molecular oxygen (~2%) of the oxygen consumed in the mitochondria can bind to single electron, which leak from electron carriers in the respiratory chain, resulting in the formation of superoxide (O_2^-) radical. [11, 12]

Furthermore, regular strenuous exercise has been found to lead to increases in both the number and size of mitochondria. [13] Thus, increased flow and metabolism of oxygen in the exercising muscles can enhance the production of O_2^- in the mitochondria. The latter may lead to enhanced generation of H_2O_2 and highly reactive hydroxyl radicals.

Strenuous exercise is known to stimulate catecholamine secretion in circulation. They enhance the cardiac performance needed to increase the blood flow to the exercising muscles. Furthermore, they promote glycogenolysis in the liver to supply glucose to muscles and stimulate mobilization of fatty acids. Both these processes are needed to meet the increased requirement of energy for the exercising muscle. [14] There is evidence that catecholamines could potentially generate free radicals in the body either through auto-oxidation or through metal ion or superoxide catalyzed oxidation.[11,15]

The superoxide radicals thus generated are considered for the formation of H_2O_2 and highly reactive hydroxyl OH* radicals in the presence of copper and iron. [16] There are various defence substances which act as major biological antioxidant compounds. [17]

Super Oxide Dismutase: Super oxide dismutase is classified into three distinct classes depending on the metal ion content: Cu/Zn SOD, Mn SOD and Fe SOD. Any reduction in the level of SOD invariably leads to an impaired protection against the toxic effects of O_2* and this might lead to severe cellular damage. [18] The result of the reaction by SOD is the H_2O_2. This substance by itself can produce damage. It can be neutralised by either of the two mechanisms by catalase or by glutathione enzyme.

Catalase: Catalase is a tetrahemin enzyme with each monomer having tightly bound NADPH molecule. Catalase reduces hydrogen peroxide and thus serves a protective role. The increased H_2O_2 concentration and lipid peroxide levels are often associated with a decresed catalase activity. It has been observed that catalase prevents free radical induced aldehyde formation, lipid peroxidation and DNA scissions caused by H_2O_2.[19]

Glutathione Peroxidase ($GSHP_X$): It can also neutralize H_2O_2. It occurs in two forms: selenium dependent $GSHP_X$ (catalyses the reduction of all H_2O_2) and selenium independent $GSHP_X$ (catalyses the reduction of only organic H_2O_2). H_2O_2 which escapes the scavenging enzymes viz; SOD, catalase and glutathione peroxidase has a great propensity to form a highly damaging hydroxyl radical (OH^*). These are neutralized by the various compounds of the primary defense system i.e. vitamins A, C, E, peroxides, and Uric acid.

Vitamin E is one of the most widely distributed anti-oxidant and major free radical chain terminator. [20] In contrast to vitamin E, Vitamin C is hydrophilic and functions better in an aqueous environment. it directly reacts with O_{2*} and OH* and various hydroperoxides as a reducing and anti-oxidant agent. Vitamin C offers the most effective protection against plasma lipid peroxidation. [21] Moreover, Vitamin C serves both as anti oxidant and pro oxidant. [22] Carotenoids protect lipids against peroxidation by quenching free radicals and other ROS, notably singlet molecular oxygen. [23] Uric acid may act by preserving plasma ascorbate. [24] The OH^* radical which goes un neutralized by the scavenging compounds like vitamin E, C, carotene, can directly cause great amount of damage to lipids, protein, DNA, carbohydrates.

Lipid peroxidation: Lipids within the cell membrane of higher organisms contain large number of polyunsaturated fatty acid side chains. Such fatty acids are prone to undergo lipid peroxidation, involves the generation of carbon radicals followed by production of peroxide radicals. [25] Lipid peroxidation has been identified as a basic deteriorative reaction in a variety of pathological conditions. Biomembranes and sub cellular organelles are the major sites of lipid peroxidation. [26] Its initiation can be due to any species which is capable of abstracting one hydrogen atom. Since hydrogen atom has only one electron, this leaves behind an unpaired electron on the carbon atom. The carbon radical in a polyunsaturated fatty acid tends to be stabilized by a molecular rearrangement to produce a conjughated

diene. This diene reacts with O_2 to give hydroperoxy radical. Lipid peroxidation (malonaldehyde formation) was increased by an acute bout of exercise in hepatic mitrochondria of untrained rats. The author suggested that antioxidant enzymes in liver and skeletal muscle are capable of adapting to exercise to minimize oxidative injury caused by free radicals. [27] Physical training and fasting erythrocyte activities of free radical scavenging enzyme systems was tested in sedentary men. It showed increased catalase and glutathione reductase in erythrocytes. [28] Although antioxidant enzyme activities are related to skeletal muscle oxidative capacity, the effects of exercise training on anti-oxidant enzymes in skeletal muscle cannot be predicted by measured changes in oxidative capacity. [29] A significant uphill was noticed in glutathione-S-transferase, super oxide dismutase and xanthine oxidase activities with the increase in exercise period. Lipid peroxidation in terms of MDA expression was also elevated with exercise. Ji LL [30] concluded that aging is accompanied with an elevation of antioxidant enzymes activities and lipid peroxidation in skeletal muscle probably due to the increased oxygen free radical production and reaction. Bicycle racers performing aerobic exercise showed increases erythrocyte activity of super oxide dismutase, catalase and glutathione peroxidise.[31]

Ji[32] concluded that exhaustive exercise can impose a severe oxidation stress on skeletal muscle and that peroxides, systems as well as antioxidant enzymes are important in coping with free radical mediated injury. Sardessai advised that the best approach for healthy individuals is to regularly consume adequate amounts of antioxidant rich foods e.g. fruits and vegetables.

Facts about Yogasana:

- The yogic kriya brings about cleaning of inner tracts and desensitization of the nerve endings. It has been documented that inflammatory mediators such as air pollution activate sensory nerve endings in the airways causing cough, chest tightness and brochoconstriction.[33]
- Practice of yoga reduces the emotional disturbances there by modifying the airway resistance in easy breathing and well being of the patients.[34]
- Relaxation exercise probably influence the hypothalamus through continuous feedback of slow rhyrhmic proprioceptive and interoceptive impulses and tend to set it at a lower level. [35]
- It has been hypothesized that meditation stimulates neocortex in such a way that these areas produce inhibitory neurotransmitter GABA. This ultimately inhibits caudal sympathetic area hypothalamus while leaving para sympathetic unaffected decrease in firing results in parasympathetic dominance. [36]

Conclusion

A practitioner of yogasana tries to keep his attention on the act of breathing, leading to concentration. This act of concentration removes his attention from worldly worries and 'de-stresses' him. This stress free individual is able to adapt better to the daily emotional, physical and mental stresses.

Source of Funding: None

Conflict of Interest: None

Acknowledgement: None

Ethical Clearance: Not required as it is a review of articles.

References

1. Udupa KN, Singh RH. The scientific basis of yoga. JAMA1972; 220 (10):1365.
2. Nayar HS, Mathur RM, Kumar RS Effects of yogic exercises on human physical efficiency. Indian J Med Res 1975; 63:1369–76.
3. Udupa KN, Singh RH, Sattiwar RM. Physiological and biochemical studies on the effect of yogic and certain other exercises. Ind J Med Res 1975; 63(4):620-24.
4. Alaxander AB. The immediate effects of systemic relaxation training on peak expiratory flow rates in asthmatic children. Psychosom Med 1974; 34: 388.
5. Sahay BK . Biochemical parameters in normal volunteers before and after yogic practices. Ind J Med Res 1982; 76 :144-48.
6. Kulpati DDS. The influence of physical conditioning by yogasanas and breathing exercises in patients of chronic obstructive lung disease. J Asso Phy India 1982; 30;12, 865-68.
7. Muralidhara DV, Ranganathan KV. Effects of yoga practice on cardiac recovery index. Ind. J. Physiol, Pharmacol 1982; 26(4): 279-83.
8. Makwana K, Khirwad Kar, Gupta N. Effect of short-term yoga practice on ventilatory function

tests. Ind J Physiol Pharmacol 1988; 32, 3,202-08.

9. Jesberger JA. Richardson JS. Oxygen free radicals and brain dysfunction. Int Neuroscience 1991; 57:1-17.
10. Alessio HM, Goldfarb A. Lipid peroxidation and scavenger enzymes during exercise. Adaptive response to training. J Appl Physiol 1988; 64: 1333- 36.
11. Freeman BA and Crapo JD. Biology of disease: free radicals and tissue injury, Lab. Invest 1982; 47:412-26.
12. Boveries, A, Chance B. The mitochondrial generation of hydrogen peroxide: General properties and effects of hyperbaric oxygen. Biochem J 1973; 134:707-16.
13. Holloszy JO, Booth FW. Biochemical adaptations to endurance exercise in muscle. Annu Rev Physiol 1976; 38:273-91.
14. Terjung RL. Endocrine systems. In: Sports Medicine and Physiology (Strauss, R. H., ed.), pp. 1979; 147-165, W. B. Saunders, Philadelphia, PA.
15. Jewett SL, Eddy LJ, Hochstein P. Is the antioxidation of catecholamines involved in ischemia-reperfusion injury? Free Radical Biol Med1989; 6: 185-88.
16. Halliwell. Oxidants and human disease: Some new concepts. FASEB J 1987; 1:358-64.
17. Mahdi AA, Singh R, Singh RK. Role of reactive oxygen species and antioxidants in human diseases: An overview. Persp Biol Sci 1996; 55-70.
18. Tayarami I, Cloez I, Clement M & Bourro J.M . Anitoxidant enzymes and related trace elements in aging brain capillaries and choroids plexus. J Neurochem 1989; 53: 817-24.
19. Sinha BK, Patterson MA. Free radical metabolism of hydralazine binding and degradation of nucleic acids. Biochem Pharmacol 1983; 32:3279-84.
20. Burton GW, Joyce A, Ingold KU. First proof that vitamin E is major lipid soluble chain breaking antioxidant in human blood plasma. Lancet 1982; 2(8293):327.
21. Frei B Ascorbic acid protects lipid in human plasma and LDL, against oxidative damage. Am J. Clin Nutr 1991; 54 Suppl. 1113s-18s.
22. Bendich A, Machilin LJ, Scandurra O. The antioxidant role of vitamin C. Adv Free Radical Biol Med 1986; 2:419-44.
23. Foote CS, Denny RW. Chemistry of singlet oxygen VIII. Quenching by B-carotene. J Am Chem Soc 1988; 90:263-65.
24. Sevanian A, Davies KJA, Hochtein P. Conservation of vitamin C by uric acid in blood. J Free Radical Biol Med 1985; 1:117-24.
25. Sohal RS. Relationship between metabolic rate, lipofuscin accumulation and lysosomal enzyme activity during aging in the adult housefly, Musca domestica. Exp Gerontol 1981; 16(4):347-55.
26. Tappel AL . Lipid peroxidation to cell components. Fed Proc 1970; 29:239.
27. Ji LL, Stratman FW, Lardy HA. Enzymatic down regulation with exercise in rat skeletal muscle. Arch Biochem Biophys 1988; 263:137-49.
28. Ohno H, Yahata T, Sato Y, Yamamura K, Taniguchi N. Physical training and fasting erythrocyte activities of free radical scavenging enzyme systems in sedentary men. Eur J Appl Physiol 1988; 57(2): 173-76.
29. Laughlin MH, Simpson T, Sexton WL, Brown OR, Smith JK, Korthuis RJ. Skeletal muscle oxidative capacity, antioxidant enzymes, and exercise training. J Appl Physiol.1990; 68(6): 2337-43.
30. Ji LL, Dillon D, Wu E. Alteration of antioxidant enzymes with aging in rat skeletal muscle and liver. Am J Physiol 1990; 258(4. 2): R918-23.
31. Mena P, Maynar M, Gutierrez JM, Maynar J, Timon J, Campillo JE. Erythrocyte free radical seavenger enzymes in bicycle professional racers. Adaptation to training. Int J Sports Med 1991; 12(6): 563-66
32. Ji LL, Fu R. Responses of glutathione system and antioxidant enzymes to exhaustive exercise and hydroperoxide. J Appl Physiol 1992; 72(2):549-54.
33. Barnes PJ. Neural control of human airways in health & disease. Am Rev Resp Dis 1986; 134: 1289-1314.
34. Gore MM. Effect of yogic treatment on some pulmonary function asthmatics. Yoga Mimansa 1982; 120: 51-54.
35. Datey KK, Deshmukh SN, Dalvi CP, Vinekar SL "Shavasan": A yogic exercise in the management of hypertension. Angiology. 1969; 20(6):325-33.
36. Bagga OP, Gandhi A. A comparative study of the effect of Transcendental Meditation (T.M.) and Shavasana practice on cardiovascular system. Indian Heart J 1983; 35(1):39-45.