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EFFECT OF YOGA AND PRANAYAMA ON LIFE STYLE DISEASE

Physiology

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ABSTRACT

The need for exercise both physical and mental, for the total well being of an individual, is no longer unknown to a common man. Physical exercise needs to be included as a routine in our day-to-day life, as the majority of us lead a sedentary life. Stress leads to the 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. Yoga practice is useful in the management of various lifestyle diseases type 2 diabetes, obesity, hypertension, and dyslipidaemia. In type 2 diabetic subjects increased brain glucose levels has been observed that alter the brain metabolites and may impair cognitive function. In study by Rajani et al. HbA1c levels were less in test group than in the controls, because yogasanas and pranayama normalized the increased plasma glucose levels by increasing the peripheral uptake of glucose or by increasing the pancreatic insulin secretion. This study clearly emphasize that yogasanas and pranayama has a positive effect on overall cognitive performance in type 2 diabetes subjects. The various postures during yoga practice help to improve the sensitivity of β-cells to glucose, thereby improving insulin secretion, and increase the blood supply to the muscle and muscle relaxation, thereby improving glucose uptake. Improvements in hormonal homeostasis also improve glycaemic control in people with diabetes mellitus . Yoga therapy also results in immunomodulation by reducing proinflammatory responses and improving immune function. (Rajni et al., 2017)

Ttryambake (2015) had showed that the mean blood pressure on day 1 and day 10 shows the difference in systolic blood pressure was 21.33 mmHg and diastolic blood pressure was 11.54 mmHg after doing pranayam. Pranayama increases frequency and duration of inhibitory neural impulses by activating pulmonary stretch receptors during above tidal volume inhalation as in HeringBruer reflex, which bring about withdrawal of sympathetic tone in the skeletal muscle blood vessels, leading to widespread vasodilatation, thus causing decrease in peripheral resistance and thus decreasing the diastolic blood pressure. This mechanism is supported by the fact that afterhyoscine-N-butylbromide, the parasympathetic blocker, it was observed that blood pressure was not decreased significantly as a result of pranayama, as it was observed when no drug was administered. (Ttryambake., 2015)

KEYWORDS

Blood pressure, Hypertension, Pranayama, Yogasana

BACKGROUND

Today we are living in an era, where science has made so much progress that we can get any information from any corner of the globe merely by the click of a mouse. With the fast-expanding knowledge in various fields, one has to toil not physically but mentally. The need for exercise both physical and mental, for the total well being of an individual, is no longer unknown to a common man. Physical exercise needs to be included as a routine in our day-to-day life, as the majority of us lead a sedentary life. Stress leads to the 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. Exercise has a positive effect on respiratory functions. Such a practice leads to an increase in resting tidal volume, a decrease in respiratory rate, an increase in vital capacity, and breath-holding time.

Yogic exercise

Yogic exercises have been known to increase mental and physical control of the body. Earlier practices of yogasana and pranayam have revealed physical and mental well being. Yoga has great therapeutic potential in the management of related disease stress. It improves the psyche of the individual because training causing a 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. A greater amount of fatis utilized for providing energy sparing glycogen.

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

Of these practices, 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 a restraint of Prana or breath having three components.

- Puraka- i.e. inhalation of breath
- Kumbhaka- i.e. retention of breath
- Rechaka- i.e. 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
- KapalBhati - same as in bhastrika but with forced expiration
- Bvjjayi - inhalation/ retention, exhalation with the glottis partially closed
- Sitkari - breathing through the folded tongue
- Sitakari - breathing with a hissing sound
- Suryabheda- inhalation through the left nostril
- Bandhatraya- Mule bandha controlling anus during inhalation
- JalandraBandha- press chin against chin in kumbhaka

- Diana Banda- draws up abdomen during exhalation
- KevalaKumbhaka- constitutes an advanced form of pranayama

Pulmonary changes during exercise

Water R Miles of yoga university USA 1964, was the first person to study the respiratory changes during pranayama. He observed that regular practice of pranayama could reduce oxygen consumption, increase work efficiency and such a practice of yoga would be beneficial in much hypoxic condition.

Udupa et al (1972) showed a 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. Udupa et al (1975) assessed biochemical parameters in 10 young adults after six months of training and found a decrease in catecholamines, cholinesterase, and blood sugar level. Increase in monoamine oxidase (MAO), diamine oxidase (DAO) plasma cortisol, serum protein levels has also been reported. Alexander reported an 11% increase in PEFR in patients given only relaxation therapy for bronchial asthma.

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

Relationship of a 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, free radicals are likely 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 bodies. A free radical is defined as a species molecule or atom capable of independent existence with an unpaired electron(s) in its outermost orbital.A dot designates the presence of one or more unpaired electron eg. O_2^-. A radical might donate its unpaired electron to another molecule or accept an electron from another molecule 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 (Jasbergerand Richardson 1991).

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 (Alessio and Goldfarb 1988).

Potential mechanism of exercise mediated free radical production

Several mechanisms 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 a single electron, which leaks from electron carriers in the respiratory chain, resulting in the formation of superoxide (O_2^-) radical (Freeman and Crapo., 1982, Boverisand Chance., 1973).

Furthermore, regular strenuous exercise has been found to lead to increases in both the number and size of mitochondria (Holloszyand Booth.,1976). 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 the mobilization of fatty acids. Both these processes are needed to meet the increased requirement of energy for the exercising muscle (Terjung.,1979). 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 (Freeman and Crapo.,1982; Jewett., 1989).

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 (Halliwell et al 1987).Various defense substances act as major biological antioxidant compounds (Mahdi etal, 1996).

Super Oxide Dismutase

Superoxide 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 impaired protection against the toxic effects of O_2* and this might lead to severe cellular damage (Tayarami et al, 1989). The result of the reaction by SOD is the H_2O_2 This substance by itself can produce damage. It can be neutralized by either of the two mechanisms by catalase or by glutathione enzyme.

Catalase

Catalase is a tetrahemin enzyme with each monomer having a 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 decreased catalase activity. It has been observed that catalase prevents free radical-induced aldehyde formation, lipid peroxidation, and DNA scissions caused by H_2O_2 (Sinha and Patterson, 1983).

Glutathione Peroxidase

Glutathione peroxidase ($GSHP_X$) can also neutralize H_2O_2. It occurs in two forms viz; selenium-dependent $GSHP_X$ (catalyzes the reduction of all H_2O_2) and selenium independent $GSHP_X$ (catalyzes the reduction of only organic H_2O_2).Hydrogen peroxide 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 (Burton et al 1982). 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 hydroperoxidesas a reducing and anti-oxidant agent. Vitamin C offers the most effective protection against plasma lipid peroxidation (Frei 1991). Moreover, Vitamin C serves both as an antioxidant and pro-oxidant (Bendich et al., 1986).Carotenoids protect lipids against peroxidation by quenching free radicals and other ROS, notably singlet molecular oxygen (Foote and Denny, 1988). Uric acid may act by preserving plasma ascorbate (Sevanian et al., 1985).The OH^* radical which goes unneutralized by the scavenging compounds like vitamin E, C, carotene, can directly cause a great amount of damage to lipids, protein, DNA, carbohydrates.

Lipid peroxidation

Lipids within the membrane of cells from higher organisms contain a large number of polyunsaturated fatty acid side chains. Such fatty acids are prone to undergo a process known as lipid peroxidation, which involves the generation of carbon radicals followed by the production of peroxide radicals (Sohal., 1981). Lipid peroxidation has been identified as a basic deteriorative reaction in a variety of pathological conditions. Biomembranes and subcellular organelles are the major sites of lipid peroxidation (Tappel., 1970). 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 conjugated diene. This diene reacts with O_2 to give hydroperoxy radicals. Lipid peroxidation (malonaldehyde formation) was increased by an acute bout of exercise in hepatic mitochondria of untrained rats. The author suggested that antioxidant enzymes in the liver and skeletal muscle are capable of adapting to exercise to minimize oxidative injury caused by free radicals (Ji., 1988). Physical training and fasting erythrocyte activities of free radical scavenging enzyme systems were tested in sedentary men. It showed increased catalase and glutathione reductase in erythrocytes (Ohno., 1988). Although antioxidant enzyme activities are related to skeletal muscle oxidative capacity, the effects of exercise training on antioxidant enzymes in skeletal muscle cannot be predicted by measured changes in oxidative capacity (Laughlin et al.,1990). A significant uphill was noticed in glutathione-S-transferase, superoxide 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(1990) concluded that aging is accompanied by 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 superoxide dismutase, catalase, and glutathione peroxidase (Mena et al., 1991).

Ji (1992) concluded that exhaustive exercise can impose 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 to be known about Yogasana

- The yogic kriya brings about the 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 the cough, chest tightness, and bronchoconstriction (Barnes., 1986).
- It is believed that most of the diseases are caused due to accumulation of toxins and morbid matter in the body. the practice of yoga reduces the emotional disturbances thereby modifying the airway resistance in easy breathing and well being of the patients (Gore, 1982)
- Relaxation exercise probably influences the hypothalamus through continuous feedback of slow rhythmic proprioceptive and interoceptive impulses and tend to set it at a lower level (DateyKK., 1969).
- The practice of medication can help by decreasing the deep-rooted stresses of day-to-day life. It has been hypothesized that meditation stimulates neocortex in such a way that these areas produce inhibitory neurotransmitter GABA. This ultimately inhibits the caudal sympathetic area hypothalamus while leaving para sympathetic unaffected decrease in firing results in parasympathetic dominance (Bagga and Gandhi, 1983).

CONCLUSION

Yogasana is an art of the control of breathing. A practitioner of yogasana not only tries to breathe but at the same time 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 can adapt better to daily emotional, physical, and mental stresses. In essence, if we bring down the stress level of an individual, the free radical production also goes down.

REFERENCES

1. Alaxander AB (1974). The immediate effects of systemic relaxation training on peak expiratory flow rates in asthmatic children. Psychosom Med. 34: 388.
2. AlessioHM, Goldfarb A (1988). Lipid peroxidation and scavenger enzymes during exercise. Adaptive response to training.J Appl Physiol. 64: 1333- 36.
3. Barnes PJ (1986). Neural control of human airways in health & disease. Am Rev Resp Dis. 134: 1289-1314.
4. Bagga OP, Gandhi A (1983). A comparative study of the effect of Transcendental Meditation (T.M.) and Shavasana practice on cardiovascular system. Indian Heart J. 35(1):39-45.
5. Bendich A, Machilin LJ, Scandurra O(1986). The antioxidant role of vitamin C. Adv Free Radical Biol Med. 2:419-44.
6. Boveries, A, Chance B. (1973). The mitochondrial generation of hydrogen peroxide: General properties and effects of hyperbaric oxygen. Biochem J 134:707-16.
7. Burton GW, Joyce A, Ingold KU (1982). First proof that vitamin E is major lipid soluble chain breaking antioxidant in human blood plasma. Lancet. 2(8293):327.
8. Datey KK, Deshmukh SN, Dalvi CP, Vinekar SL (1969)"Shavasan": A yogic exercise in the management of hypertension. Angiology. 20(6):325-33.
9. Foote CS, Denny RW(1988). Chemistry of singlet oxygen VIII. Quenching by B-carotene. J Am Chem Soc. 90:263-65.
10. Freeman BA and Crapo JD (1982). Biology of disease: free radicals and tissue injury, Lab. Invest.47:412-26.
11. Frei B (1991) Ascorbic acid protects lipid in human plasma and LDL, against oxidative damage. Am J. ClinNutr. 54 Suppl. 1113s-18s.
12. Gore MM (1982). Effect of yogic treatment on some pulmonary function asthmatics. Yoga Mimansa. 120: 51-54.
13. Halliwell (1987). Oxidants and human disease: Some new concepts. FASEB J. 1:358-64.
14. Holloszy JO, Booth FW (1976). Biochemical adaptations to endurance exercise in muscle. Annu Rev Physiol. 38:273-91.
15. Jesberger JA (1991). Richardson JS. Oxygen free radicals and brain dysfunction. Int Neuroscience57:1-17.
16. Jewett SL, Eddy LJ, Hochstein P (1989). Is the antioxidation of catecholamines involved in ischemia-reperfusion injury? Free Radical Biol Med. 6: 185-88.
17. Ji LL, Stratman FW, Lardy HA (1988). Enzymatic down regulation with exercise in rat skeletal muscle. Arch BiochemBiophys 263:137-49.
18. Ji LL, Dillon D, Wu E (1990). Alteration of antioxidant enzymes with aging in rat skeletal muscle and liver.Am J Physiol 258(4. 2): R918-23.
19. Ji LL, Fu R (1992). Responses of glutathione system and antioxidant enzymes to exhaustive exercise and hydroperoxide. J Appl Physiol. 72(2):549-54.
20. Mahdi AA, Singh R, Singh RK (1996). Role of reactive oxygen species and antioxidants in human diseases: An overview. PerspBiol Sci. 55-70.
21. Kulpati DDS (1982). The influence of physical conditioning by yogasanas and breathing exercises in patients of chronic obstructive lung disease. J AssoPhy India. 30;12, 865-68.
22. Laughlin MH, Simpson T, Sexton WL, Brown OR, Smith JK, Korthuis RJ (1990). Skeletal muscle oxidative capacity, antioxidant enzymes, and exercise training. J Appl Physiol. 68(6): 2337-43.
23. Makwana K, KhirwadKar, Gupta N (1988). Effect of short-term yoga practice on ventilatory function tests. Ind J PhysiolPharmacol. 32, 3,202-08.
24. Mena P, Maynar M, Gutierrez JM, Maynar J, Timon J, Campillo JE (1991). Erythrocyte free radical seavenger enzymes in bicycle professional racers. Adaptation to training. Int J Sports Med.; 12(6): 563-66
25. Muralidhara DV, Ranganathan KV (1982). Effects of yoga practice on cardiac recovery index. Ind. J. Physiol, Pharmacol, 26(4): 279-83.
26. Nayar HS, Mathur RM, Kumar RS (1975). Effects of yogic exercises on human physical efficiency. Indian J Med Res.63:1369–76.
27. Ohno H, Yahata T, Sato Y, Yamamura K, Taniguchi N (1988). Physical training and fasting erythrocyte activities of free radical scavenging enzyme systems in sedentary men. Eur J ApplPhysiol ; 57(2): 173-76.
28. Rajani, S., Archana, R., Indla, Y. R., & Rajesh, P. (2017). Beneficial effects of yogasanas and pranayama in limiting the cognitive decline in Type 2 diabetes. National journal of physiology, pharmacy and pharmacology, 7(3), 232.
29. Sahay BK (1982). Biochemical parameters in normal volunteers before and after yogic practices. Ind J Med Res. 76 :144-48.
30. Sevanian A, Davies KJA, Hochtein P (1985). Conservation of vitamin C by uric acid in blood. J Free Radical Biol Med. 1:117-24.
31. Sinha BK, Patterson MA(1983). Free radical metabolism of hydralazine binding and degradation of nucleic acids. BiochemPharmacol. 32:3279-84.
32. Sohal RS (1981).Relationship between metabolic rate, lipofuscin accumulation and lysosomal enzyme activity during aging in the adult housefly, Muscadomestica. ExpGerontol.16(4):347-55.
33. Tappel AL(1970). Lipid peroxidation to cell components. Fed Proc. 29:239.
34. Tayarami I, Cloez I, Clement M &Bourro J.M (1989). Anitoxidant enzymes and related trace elements in aging brain capillaries and choroids plexus. J Neurochem. 53: 817-24.
35. Terjung RL (1979) Endocrine systems. In: Sports Medicine and Physiology (Strauss, R. H., ed.), pp. 147-165, W. B. Saunders, Philadelphia, PA.
36. Ttryambake RG. (2013)The Effectiveness of Pranayama on Blood Pressure of Hypertensive Patients. International Journal of Science and Research (IJSR).4(8); 561-564.
37. Udupa KN, Singh RH (1972). The scientific basis of yoga. JAMA;220 (10):1365.
38. Udupa KN, Singh RH, Sattiwar RM (1975). Physiological and biochemical studies on the effect of yogic and certain other exercises. Ind J Med Res. 63(4):620-24.