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EFFECT OF YOGA ON CARDIAC OUTPUT

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Abstract: *It is already established by improving arterial–cardiac baroreflex sensitivity and the dynamic Starling mechanism, high-intensity exercise training enhanced integrated cardiovascular control. While yoga has been shown to have favorable physiological effects in individuals with increase in cardiac output is unknown. The researcher reviewed the published papers in PubMed from 1995 to 2025 and tried to elicited mechanism behind effect of yoga on cardiac output. Yoga may be an option for people to increase cardiac output which may be sue to development of blood flow autoregulatory system and the sympathetic nervous system and also vasoactive circulating hormones. Not only that the Frank–Starling mechanism, little changes in atrial pressure result in big changes in the heart's contraction strength. little changes in atrial pressure are caused by changes in venous return which yield increase in cardiac output.*

Index Terms **– Yoga, Cardiac, Output, Effect.**

I. INTRODUCTION

The amount of blood that flows from the left ventricle into the aorta is known as cardiac output, and the system that regulates it consists of numerous parts in addition to the heart. The mechanisms controlling venous return are largely in charge of controlling the heart's output since the heart's output rate cannot be greater than the rate of venous return to it. The pressure gradient and flow resistance across the vascular system have an impact on venous return. The volume of blood passing through the system, the circulatory system's unstressed vascular volume, its capacitance, mean systemic pressure, and right atrial pressure are some of the variables that affect the pressure gradient for venous return. The overall vascular resistance from the aortic valve to the right atrium is known as resistance to venous return. The body's organs and tissues receive cardiac output, which is typically allocated based on metabolic needs. The primary factor influencing a tissue's blood flow resistance is its metabolic requirement for oxygen. By changing the resistance of the tiny arteries and arterioles supplying its capillary network, each gram of tissue may regulate the flow of blood through its microcirculation. Although it is a significant and intriguing field, the physiology of the microcirculation is outside the purview of this talk. The subject is covered in detail in another volume of this series. It is adequate for our purposes to say that the oxygen content of the local extracellular fluid has the biggest long-term impact on microcirculatory resistance, even if a number of factors can influence it. A local feedback control system governs the flow of blood via tissues. An increase in metabolic activity causes a patch of tissue to use more oxygen, which raises the rate at which oxygen is removed from the local extracellular fluid below the rate at which it is delivered from the blood in the capillaries. As a result, the extracellular fluid's oxygen content decreases. The arterioles supplying the capillaries in that area of the tissue dilate as a result of local mechanisms that are responsive to the oxygen concentration in the extracellular fluid. The rate of oxygen diffusion into the extracellular fluid can grow as a result of the ensuing decrease in resistance, which also improves blood flow to the capillaries and oxygen delivery. The process keeps on until the oxygen entry rate brings the extracellular fluid's oxygen concentration back to a level that is close to the target or set point concentration. In response to shifting metabolic demands, this negative feedback regulation mechanism acts in a matter of seconds to keep oxygen concentration close to the set point level by regulating tissue blood flow throughout the body. The short-term tissue autoregulatory mechanism is the name given to the control system. Additionally, a related long-term system functions over days and weeks; mechanisms of the long-term autoregulatory system induce the creation of new capillaries and other microcirculatory channels in response to protracted decreases in tissue oxygen concentration. Long-term increases in oxygen transport to the tissue are made possible by the increasing capillary density. In order to achieve a state of mastery over the modifications of the mind (Chitta Vritti Nirodhah, the sage Patanjali's definition of yoga), the integrated yoga intervention comprised easy and safe physical, mental, emotional, and intellectual practices. This was achieved through effortless blissful inner awareness during all practices. Both comprehensive cardiac rehabilitation and exercise-only rehabilitation programs are successful in lowering cardiac mortality, according to a systematic evaluation of cardiac rehabilitation research involving 7683 individuals with coronary heart disease from previous meta-analyses. With exercise-based cardiac rehabilitation, cardiac mortality decreased by 31%, and with a comprehensive cardiac rehabilitation program, it decreased by 26%. The effectiveness of intensive lifestyle modification programs in lowering risk factors in patients following coronary artery bypass grafting was not well-established until more recent randomized prospective studies, despite the fact that such studies had demonstrated the role of such programs in risk-factor modification and extending survival among post-myocardial infarction patients prior to 2000. But no such review was found on effect of yoga on cardiac out. From this point of view the researcher intends to investigate whether there is any positive effect of yoga on cardiac output or not.

2. Methodology: The researcher reviewed the published papers in PubMed from 1995 to 2025 and tried to elicited mechanism behind effect of yoga on cardiac output. After removing duplicates, the search yielded 946 references. 19 entries from 73 papers were

obtained for full-text review after records were eliminated based on title and abstract. Following full-text examination, 11 studies were eliminated because they did not satisfy the review criteria. A total of 9 records were suitable for the review.

3. Discussion: Heart rate (HR) times stroke volume (SV) equals cardiac output, according to the basic concept of cardiac output (CO = HR x SV). The quantity of blood the heart pumps out with each beat is known as the stroke volume, whereas the heart rate is the number of heart-beats per minute. A number of factors impact stroke volume and, consequently, cardiac output, including contractility (the capacity of the heart muscle to contract), afterload (resistance to ejection), and preload (venous return). Signals from the sinoatrial node, which depolarizes automatically at an intrinsic rate of 60 to 100 times per minute, are used to calculate heart rate. The other primary driver of cardiac output, SV, is likewise influenced by a number of variables. Preload, contractility, and afterload all influence how much blood is expelled with each beat. All of the elements that lead to passive muscular tension in the muscles at rest are represented by preload. The amount of blood in the ventricles just prior to systole, or the end-diastolic ventricular volume, determines preload. Increased end-diastolic blood volume returned to the heart causes the cardiac muscles to passively stretch more. The Frank-Starling law of the heart states that this causes the ventricles to contract more forcefully. The force of myocyte contraction, also known as inotropy, is referred to as contractility. The heart can pump more blood out of the heart as the contraction force increases, which raises the stroke volume. Afterload is the last factor that determines stroke volume. All of the elements that contribute to overall tension during isotonic contraction are represented by afterload. Therefore, the amount of systemic resistance the ventricles must overcome in order to expel blood into the vasculature might be linked to afterload. Unlike preload and contractility, afterload is inversely correlated with stroke volume and proportional to systemic blood pressures. Numerous signaling pathways, such as enhanced sympathetic tone, catecholamine production, and thyroid hormone circulation, can raise cardiac output. Through the beneficial effects of chronotropy (timing), dromotropic (conduction speed), and lusitropy (myocardial relaxation rate), these mechanisms raise HR. By increasing venous return through receptor-mediated vasoconstriction, these factors also raise preload.

4. Conclusion: It may be concluded that alterations in lifestyle or the implementation of yoga intervention decreased routine psychological stressors and repeated practice of breathing and relaxation techniques which are known to lower sympathetic activity by downregulating the cortical-hypothalamic-pituitary-adrenal axis, could be the potential mechanism. Lower peripheral vascular resistance and lower blood pressure to decreased sympathetic activity.

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