HEART RATE VARIABILITY RELATED WITH THYROID FUNCTION

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ABSTRACT: Heart rate is the number of times the heart contracts or relax per minute. The normal heart rate is 72 beats per minute. The cardiovascular signs and symptoms of thyroid disease are some of the most profound and clinically relevant findings that accompany both hyperthyroidism and hypothyroidism. Autonomic dysfunction may contribute to cardiovascular morbidity in subclinical hypothyroid patients. It is controversial whether the abnormality exists in sympathetic or the parasympathetic function. It is also not known whether the severity of autonomic dysfunction is related to the degree of thyroid deficiency. Altered thyroid functions are associated with variation in autonomic regulation of cardiovascular activity. The thyroid hormone plays a major role in the heart rate variability of hyperthyroid and hypothyroid patients. Several treatment measures are available for maintaining the normal heart rate in both hyperthyroid and hypothyroid patients.

KEYWORDS: Heart rate, hyperthyroidism, hypothyroidism, thyroid hormone, tachyarrhythmias.

INTRODUCTION:

Heart rate is the number of times the heart contracts or relax per minute . The normal heart rate is 72 beats per minute. The heart rate normally varies with physical exercise and other pathological conditions. After exercise the heart rate increases rapidly. The cardiovascular manifestations of thyroid hormone excess ,including tachycardia, a widened pulse pressure, a brisk carotid and peripheral arterial pulsation, a hyperkinetic cardiac apex, and loud first heart sound have long been recognised and are a cornerstone for clinical diagnosis[1,2]. Several hormones play a major role in maintaining the heart rate. Among the several hormones thyroid hormone plays an important role. The heart rate variability may be classified or noticed less than two categories hyperthyroidism and hypothyroidism. The hyperthyroidism may be as a result from the combined effects of thyroid hormone on certain molecular pathways on the heart and vasculature, at both the genomic and nongenomic level [3]. In spontaneous human hyperthyroidism, the high cardiac output state is sustained prevalently by changes in peripheral hemodynamic (vascular hypothesis), or by changes in myocardial contractility (myocardial hypothesis) [4, 5]. Thus in this article we are going to see the heart rate variability related with the thyroid function.

THYROID HORMONE AND HEART RATE:

Heart rate is an important mechanism for the regulation of cardiac output. Apart from determining the rate of cardiac ejection, it affects both systolic and diastolic function. A high heart rate also increases the rate of myocardial relaxation, thus improving early cardiac filling (lusitropic effect) [6]. Pacing induced increase in contraction frequency generally reduces preload and stroke volume, so that cardiac output remains constant. On the other hand, an increased heart rate reduces diastolic filling time and, thus, leads to greater dependence on atrial systole [7, 8]. Changes in thyroid status markedly influence cardiac contractile and electrical activity. The predominant route by which triiodothyronine (T3) affects cardiac action is by exerting a direct effect in cardiac myocytes through binding to thyroid hormone nuclear receptor isoforms. In addition, T3 modifies cardiac action by alterations in the vascular system and decreases after load of the left ventricle by subtle modification related to the sympathetic system. Increased or reduced action of thyroid hormone on certain molecular pathways in the heart and vasculature causes relevant cardiovascular derangements. It is well established that overt hyperthyroidism induces a hyper dynamic cardiovascular state (high cardiac output with low systemic vascular resistance), which is associated with a faster heart rate, enhanced left ventricular (LV) systolic and diastolic function, and increased prevalence of supra ventricular tachyarrhythmias - namely, atrial fibrillation - whereas overt hypothyroidism is characterized by the opposite changes[9].

HEART RATE VARIABILITY IN HYPERTHYROIDISM PATIENTS:

Preload is the hemodynamic force exerted on the ventricular wall during filling and, thus, corresponds to ventricular end-diastolic wall stress or tension *sensu strictu*. It contributes greatly to the determination of ventricular end-diastolic volume and modulates myocardial performance significantly. Total blood volume and atrial contraction may also significantly contribute to regulate the cardiac preload [10]. In fact, increased indices of early transmitral peak flow velocity and shortened LV isovolumic relaxation time in hyperthyroid patients may reflect greater venous return, which leads to an increased proto-diastolic transmitral pressure gradient (due to increased atrial pressure) and earlier mitral valve opening[11,12]. Alternatively, the shorter isovolumic relaxation time may be due to improved diastolic function , which, in turn, would allow the increased venous return to be

accommodated without relevant changes in filling pressure [13]. This interpretation is supported by the observation of comparable Values of LV end-diastolic volume and pressure in hyperthyroid patients and normal subjects [14]. After load is the hemodynamic force exerted on the ventricular wall during ejection, corresponding, therefore, to end-systolic wall stress or tension . It contributes to the determination of ventricular end-systolic volume and modulates myocardial performance significantly. Alternatively, given the correlation between systolic arterial pressure and end-systolic ventricular pressure, end-systolic wall stress estimated from cuff sphygmomanometer measurements is a reliable measure of ventricular after load [15]. The increase in heart rate and preload could play a major role in augmenting LV performance in human hyperthyroidism, thus reinforcing the notion that the hyperkinetic cardiovascular state in human hyperthyroidism is an adaptive response to the increase in the peripheral metabolic demand promoted by thyroid hormone [16, 17].

HEART RATE VARIABILITY IN HYPOTHYROIDISM PATIENTS:

The thyroid hormone influences the autonomic nervous system. Thyroid hormone deficiency is associated with increased sympathetic influence on the cardiovascular autonomic system [18]. The lower HF component of the HRV reflected a decrease in vagal tone. Power spectral analysis was more sensitive than other tests. Some of the traditional tests have found autonomic dysfunction in both subclinical and hypothyroid patients but no major intergroup differences [19]. Autonomic abnormalities can begin early, even in the subclinical stage of hypothyroidism and are comparable in severity to that seen in hypothyroid patients. Metabolic effects of decreased thyroid hormone, may also lead to increased protein deposition in extracellular space, resulting in water accumulation in myocardial wall, fibrosis in ventricular wall, all leading to increased regional in homogeneity of ventricular repolarisation. Autonomic dysfunction has been linked to prehypertension, to family history of hypertension and may lead to development of hypertension [20]. Sympathetic function abnormality was more prominent although selective parasympathetic dysfunction was also seen. Apparently there was no relationship between the autonomic function score and TSH or TPO levels.

EFFECT OF THYROID HORMONE ON HEART RATE:

Increased or reduced action of thyroid hormone on certain molecular pathways in the heart and vasculature causes relevant cardiovascular derangements. It is well established that overt hyperthyroidism induces a hyper dynamic cardiovascular state (high cardiac output with low systemic vascular resistance), which is associated with a faster heart rate, enhanced left ventricular (LV) systolic and diastolic function, and increased prevalence of supra ventricular tachyarrhythmias - namely, atrial fibrillation - whereas overt hypothyroidism is characterized by the opposite changes[21]. Extensive evidence indicates that the cardiovascular system responds to the minimal but persistent changes in circulating thyroid hormone levels, which are typical of individuals with subclinical thyroid dysfunction. Subclinical hyperthyroidism is associated with increased heart rate, atrial arrhythmias, increased LV mass, impaired ventricular relaxation, reduced exercise performance, and increased risk of cardiovascular mortality [22]. Administration of thyroid hormone or its analogue 3, 5-diiodothyropropionic acid greatly benefits these patients, highlighting the potential role of thyroid hormone treatment in patients with acute and chronic cardiovascular disease. Subclinical hypothyroidism is associated with impaired LV diastolic function and subtle systolic dysfunction and an enhanced risk for atherosclerosis and myocardial infarction [23].

CONCLUSION:

Heart rate variabilties are closely related with the hormones related to thyroid function in hyperthyroid state of patients and following treatment may return the patients' condition towards almost normal**.** The augmented cardiac performance accompanying human hyperthyroidism seems to be mostly an adaptive response to changes in peripheral hemodynamic rather than the result of a mandatory enhancement of myocardial contractility. The cardiac output reserve directly correlates with exercise capacity, and given the direct relationship between the absolute metabolic cost of workload and fatigue perception, it is not surprising that hyperthyroid patients often complain of low exercise capacity and tolerance. Therefore thyroid hormone plays a major role in the heart rate variability in conditions like hyperthyroidism and hypothyroidism. It is also involved in other heart functions.

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