Pulse Rate can be taken as a marker for serum levels of T3 and T4 in thyrotoxic patients.

Abas R. Al-Musawy
M.B.Ch.B ,C.A.B.M

Waleed M. Al-Mashhadani
PhD pharmacology

Waffaa N. Saieed
MSc pharmacology

Abstract:

Background: The study was designed to evaluate the correlation between clinical parameters:(pulse rate and pulse pressure) and serum levels of triiodothyronine (T3) and thyroxine (T4) in thyrotoxic patients.

Setting: The Specialized Centre for Endocrinology and Diabetes, Baghdad, Iraq.(2002 – 2003)

Design: Randomized biochemical screening study.

Outcome measures:-serum levels of T3 and T4, Resting pulse rate, and pulse pressure.

Results: statistically significant positive correlation between T3 and T4 levels and pulse rate ($r=0.428$ $p<0.01$, $r=0.328$ $p<0.05$ respectively). While no significant correlation was found between pulse pressure and neither T3 nor T4.

Conclusion: the study suggests using pulse rate as measured clinically as a marker for serum levels of T3 and T4 in thyrotoxic patients.

Key words: T3, T4, pulse rate, thyrotoxicosis.

Introduction:

For many years a relationship has been recognized between thyroid hormone, the heart, and the peripheral vascular system. In 1786, Parry [1] first described the clinical features of patients with thyrotoxicosis that include palpitation, irregular pulse, and dyspnea. Forty-nine years later, Graves [2] provided descriptions of diffuse toxic goiter The profound cardiac manifestations of thyrotoxicosis led early observers to wrongly conclude that the disease originated within the heart [3] . Eventually, researchers acknowledge that an overactive thyroid gland was the direct course of the heart disease [4,5]: and it is well established that overt hyper-thyroids includes a hyperdynamic cardiovascular state (high cardiac output with low systemic vascular resistance) which is associated with a faster heart rate , in hatred left ventricular systolic and diastolic function, and increased prevalence of supraventricular tachyarrhythmias [6,7,8,9] .Additionally, it has been found that the most common cardiovascular finding in thyrotoxic patients is tachycardia with 90% of patients having a resting heart rate that exceed 90 beat / mint,[10].

Change in cardiac parameters encountered in hyperthyroidism result from the activity of thyroid hormone on certain molecular pathways in the heart and vasculature which include genomic and nongenomic actions [11,12].

Because the heart is an organ sensitive to the action of thyroid hormone, and measurable changes in cardiac performance are detected with small variations in thyroid hormone serum concentrations[13], we aim in this study to find a possible correlation between resting pulse rate and serum level of T3 and T4(thyroxine) in thyrotoxic patients.

Patients & Methods:

A randomizes biochemical screening was performed on thirty six thyrotoxic patients (age mean=35.27±9.648 year), at the Specialized Centre for Endocrinology and Diabetes in Baghdad-Iraq.(2002–2003). The patients were newly diagnosed as thyrotoxic patients on basis of clinical examinations and Thyroid Function Test by Radioimmunoassay.

For each of the patients clinical measurements of pulse pressure and resting radial pulse rate was done, followed by estimation of serum levels of T3 and T4 by radioimmunoassay.

A statistical test of correlation was done between results of clinical measurements and biochemical results of T3 and T4 by using SPSS analytic system.

Results:

1-The mean ,Std, SME , Minimum , and Maximum value of age, pulse pressure, pulse rate, and serum levels of T3,and T4 are showed in table 1.

Table 1:The means of different parameters measured in this study

	AGE	P.P	PR	T3	T4
Mean	35.27	78.65	105.43	2.9432	14.44
Std. Deviation	9.648	25.621	11.901	1.25001	4.057
SEM	1.586	4.212	1.957	0.20550	0.667
Minimum	18	40	75	1.10	2
Maximum	60	140	146	7.00	23

2-A statistically significant positive correlation was found between T3 and T4 levels and pulse rate (r=0.428 ,p<0.01 & r=0.328 ,p<0.05 respectively) (figures 1&2), while no significant correlation was found between pulse pressure and neither T3 nor T4 (figures 3 & 4).

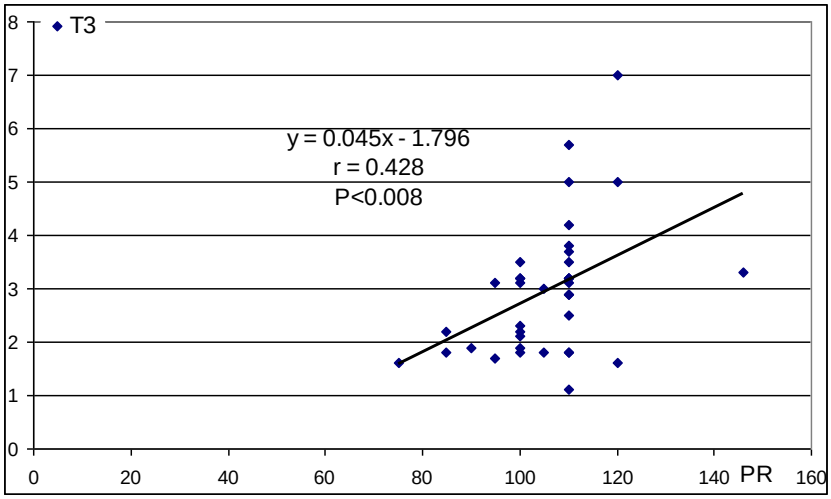

Fig.1 Correlation between T3 level and pulse rate.

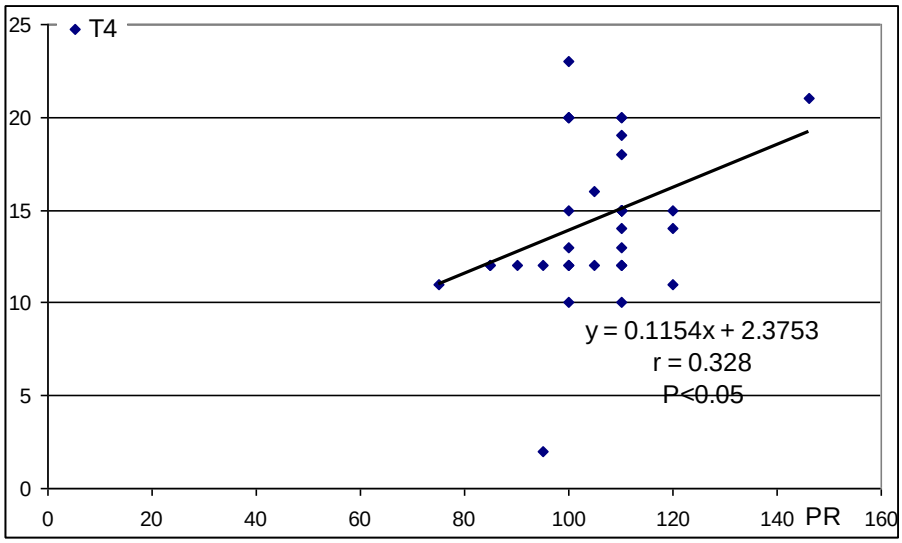

Fig.2 Correlation between T4 and pulse rate

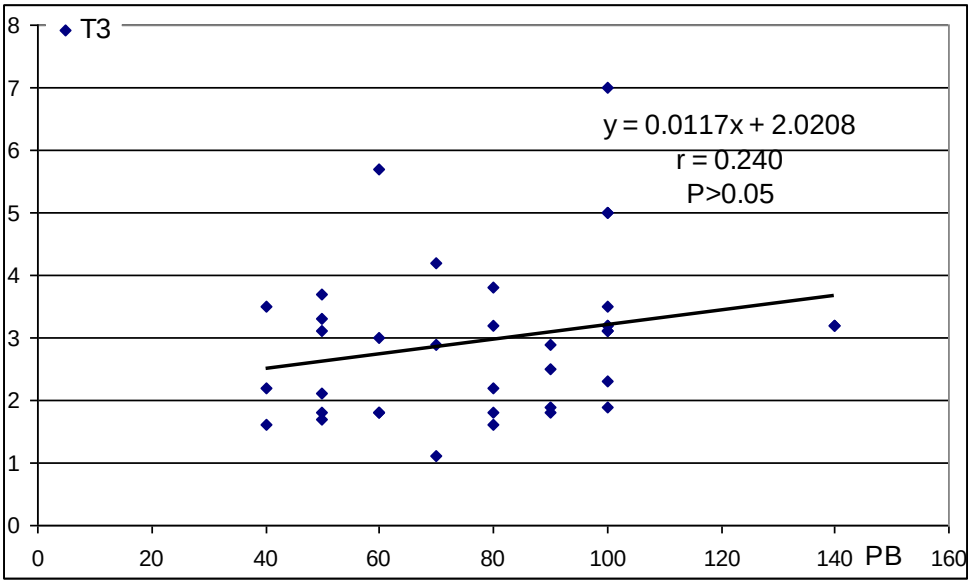

Fig.3: Correlation between Pulse Pressure and T3

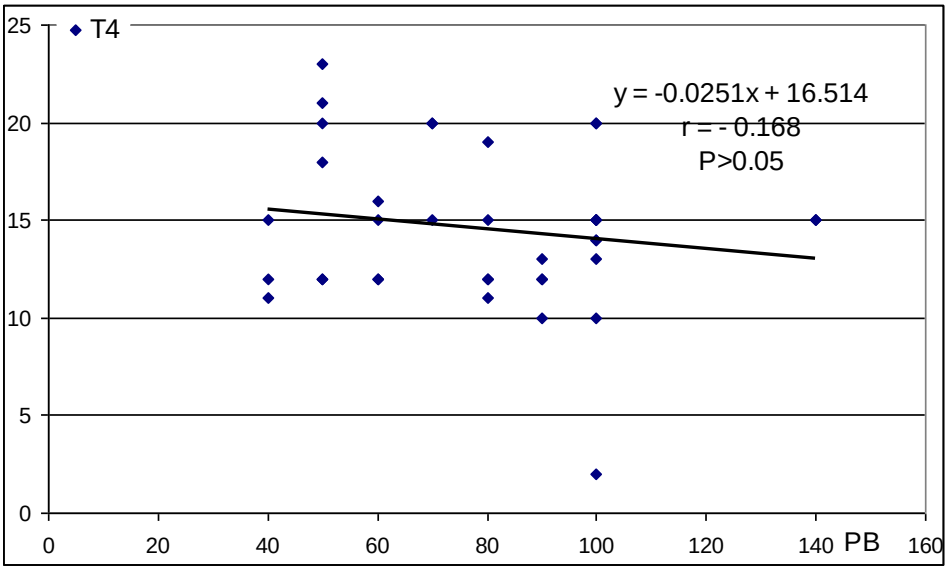

Fig.4 Correlation between Pulse Pressure and T4.

Discussion:

Patients with hyperthyroid heart disease frequently complain of symptoms related to chronotropic alteration, as they often experience palpitation as well as irregular and vigorous heart beat [7].Chronotropic effects of thyroid hormone have been assessed using 24 hour ECG recordings; hyperthyroid patients show an increase in heart rate through out sleeping and wake hours, whereas in hypothyroid patients a decrease in basal, average, and maximal heart rates was found even most of them were not having bradycardia at rest [14].Moreover, Studies using an isolated heart with experimental thyrotoxicosis show increased heart rates and shorter mean effective refractory periods than heart from euthyroid animals [15].

The actions of thyroid hormone occur mostly through triiodothyronine (T3) binding nuclear receptors that regulate expression of thyroid hormone responsive genes [16]. Changes in cardiac function are mediated by T3 regulation of cardiac specific genes [17], a number of which have been

recognized as targets for transcriptional activation by thyroid hormone like ; myocin heavy chain alpha (MHC-α), sarcoplasmic reticulum-ATPase (SERCA), Na-K-ATPase, β-adrenergic receptor, cardiac troponin I, and atrial natriuretic peptide [18,19,20,21,22,23]. Conversely, the transcription of other genes such as MHC-b is suppressed by thyroid hormone [24].Thyroid hormone can also increase β-adrenergic receptor expression and, consequently, β-adrenergic sensitivity, in addition it enhances expression of the stimulatory subunit of the guanosine triphosphate (GTP)-binding protein (Gs) [25].

Contrary to all those genomic actions of thyroid hormone, the nongenomic pathways on plasma membrane, thyroid hormone prolongs the inactivation of the Na-channels in cardiomyocytes[26],and enhances the intracellular uptake of Na and the secondary activation of the myocardial sarcolemmal Na – Ca exchange that may explain the acute inotropic activity of thyroid hormone[12]. T3 also exerts a direct effect on L – type calcium channel and enhances calcium entry into cardiomyocytes[27].

Most of above chronotropic effects of thyroid hormones may explain the results in present study which found a significant positive correlation between the resting pulse rate and serum levels of T3 and T4 in thyrotoxic patients. Therefore we would like to bring to mind using this clinical parameter as a marker or an indicator for serum levels of T3 and T4 in thyrotoxic patients

Conclusion:

The results in the present study may indicate that resting pulse rates may be used as a marker for the serum levels of T3 and T4 in thyrotoxic patients

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