

Assessment of variations in systemic arterial pressure and mean arterial pressure in hypothyroid patients

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Abstract:

Background: Thyroid hormone influences cardiac functions and vascular resistance. Any dysfunction in thyroid hormone synthesis will produce profound cardiovascular effects. Arterial hypertension is known to be frequently associated with hypothyroidism. Hypothyroidism mainly affects diastolic blood pressure (DBP), narrowing the pulse pressure (PP) which can alter the mean arterial pressure. This study intends to find the variations in systemic arterial pressure and also the association of TSH with it. **Aim:** To find the variations in systemic arterial pressure and mean arterial pressure in hypothyroidism and to find the association of TSH with it. **Methodology:** This cross-sectional study involved 30 clinically diagnosed hypothyroid patients recruited from Endocrinology OP, Tertiary care hospital, Chennai. Serum Thyroid-stimulating hormone levels were estimated in all the subjects. Arterial blood pressure was recorded in all the subjects in the sitting posture and the results obtained. The results were tabulated and analysed by applying Pearson correlation coefficient and student "t" test. **Results:** Our study showed that the diastolic pressure was elevated in study participants compared to systolic blood pressure and also there was narrowing of pulse pressure. Hence mean arterial pressure was also elevated. There was a statistically significant positive correlation between TSH and systemic arterial pressure and mean arterial pressure. **Conclusion:** Our study concludes that there is an association between serum TSH and systemic arterial pressure.

Keywords: diastolic blood pressure (DBP), hypothyroidism, mean arterial pressure (MAP), systolic blood pressure (SBP), thyroid-stimulating hormone (TSH)

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Introduction:

In India, hypothyroidism is more common endocrine disorder with prevalence of 11%.¹ Hypothyroidism is diagnosed when there is increased thyroid stimulating hormone and the thyroid hormone levels may be normal or decreased. Thyroid hormones influence the cardiovascular system by decreasing the systemic

vascular resistance and increasing the basal heart rate, myocardial contractility and blood volume.² Hence, coronary artery diseases and hypertension are known to be frequently associated with thyroid disorders.³ In hypothyroid patients, increased vascular resistance and decreased vascular compliance has led to an increase in diastolic

pressure.⁴ Thus, the pulse pressure is narrowed which can also alter the mean arterial pressure.

One of the major public health issues in both economically developed and developing nations is hypertension (HTN). It is a leading contributor to cardiac conditions like coronary artery disease and stroke, as well as chronic renal failure, heart failure, arrhythmia, dementia, and early mortality.⁵ In addition to the vast majority of patients with primary (essential) hypertension, 10% of people also have secondary hypertension. Thyroid disorders represent a small percentage as the underlying cause for secondary hypertension.⁶ Both overt and subclinical thyroid disorders may initially manifest as hypertension.⁷

Thus, this study intends to find the variations in systemic arterial pressure and also its association with TSH in hypothyroidism.

Materials & Methods:

This descriptive cross-sectional study was started after obtaining Institutional Ethics Committee clearance. A sample of 30 clinically diagnosed hypothyroid patients were recruited from Endocrinology OP of tertiary care hospital by simple random sampling method.

Inclusion Criteria:

1. Both genders
2. Age between 18 and 60 years.
3. Patients clinically diagnosed as hypothyroid with or without treatment

Exclusion Criteria:

1. Age less than 18 and more than 60 years
2. Hyperthyroid individuals
3. Known case of hypertension
4. Patients with any chronic neurological, cardiovascular, renal disorder and psychiatric illness.
5. Pregnancy and lactation

After obtaining written informed consent from the participants, a thorough clinical history was obtained. After adequate rest of 10 minutes, blood pressure (BP) was measured using sphygmomanometer and stethoscope. The study participants were made to sit comfortably on a chair with back support. The blood pressure cuff was tied around the left arm of the participant kept at their heart level. The cuff pressure was raised gradually until the disappearance of radial pulse by palpation. Now the pressure in the cuff was raised further by 30 mmHg and then, the cuff pressure was decreased slowly. The reappearance of radial pulse was recorded as SBP by palpatory method. This was done to avoid auscultatory gap. Then by auscultatory method the blood pressure was recorded by the following steps:

The stethoscope was placed lightly on the brachial artery in cubital fossa. The cuff pressure was increased to 30 mmHg above the SBP determined by palpatory method. While auscultating the brachial artery, the cuff pressure was decreased at a rate of 2 – 3 mmHg. The reading at which Korotkoff sound appears was noted as SBP and the disappearance of the sound was noted as DBP. Thus, both SBP and DBP were recorded by auscultatory method.

Initially BP was recorded in both arms. When there was disparity, the arm with higher value was used for BP recording. At least 3 BP readings were taken at 2 minutes interval. Then the average of the three BP recordings were taken.

Pulse pressure was calculated using the formula $SBP - DBP$, and Mean arterial pressure using the formula $DBP + \frac{1}{3} PP$. 2ml of fasting blood was collected for estimating the serum TSH level. Data was collected and analyzed using SPSS software. Pearson correlation coefficient and student 't' test was used to evaluate the association between serum TSH and blood pressure variables.

Results:

the 30 participants only one was male and remaining were female.

The age of participants in the study were between 18 and 60 years with mean age of 41.9 ± 10.4 . Of

Figure. I: Distribution of Systolic blood pressure amongst the hypothyroid⁸

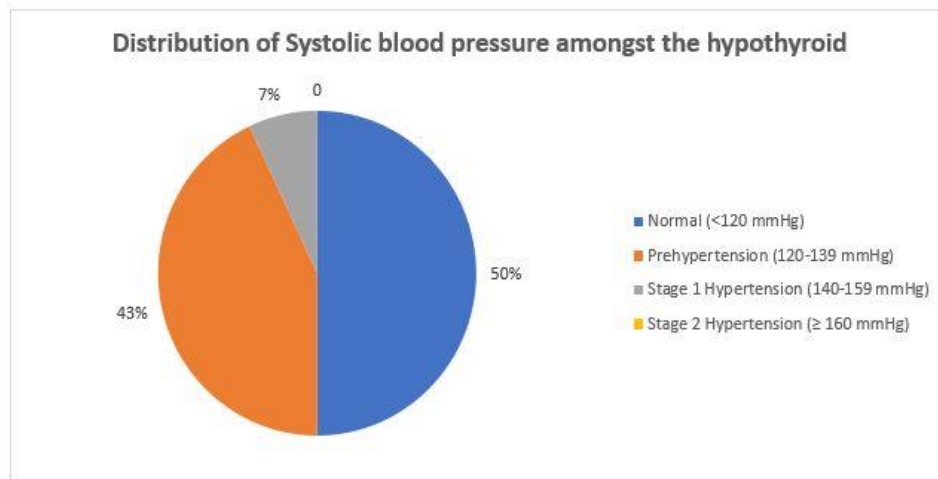


Figure. II: Distribution of Diastolic blood pressure amongst the hypothyroid⁸

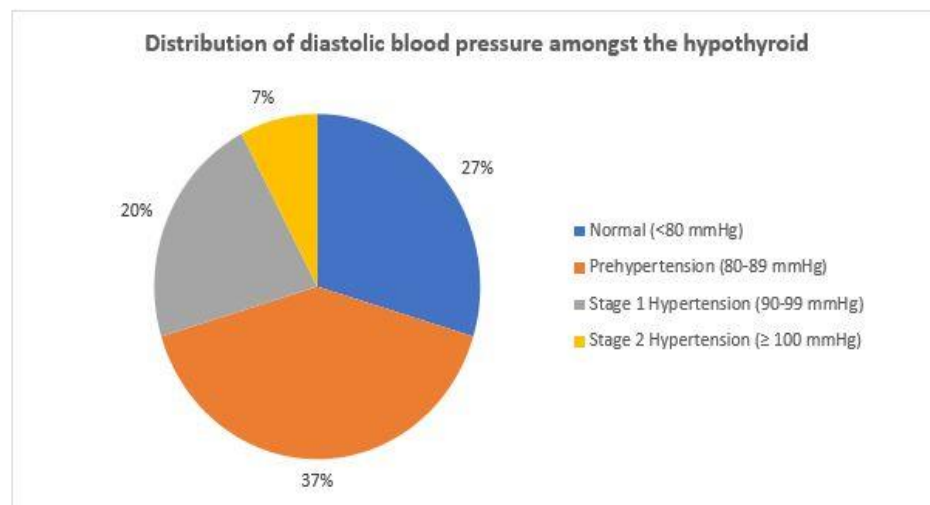


Figure. III: Distribution of mean arterial pressure amongst the hypothyroid⁸

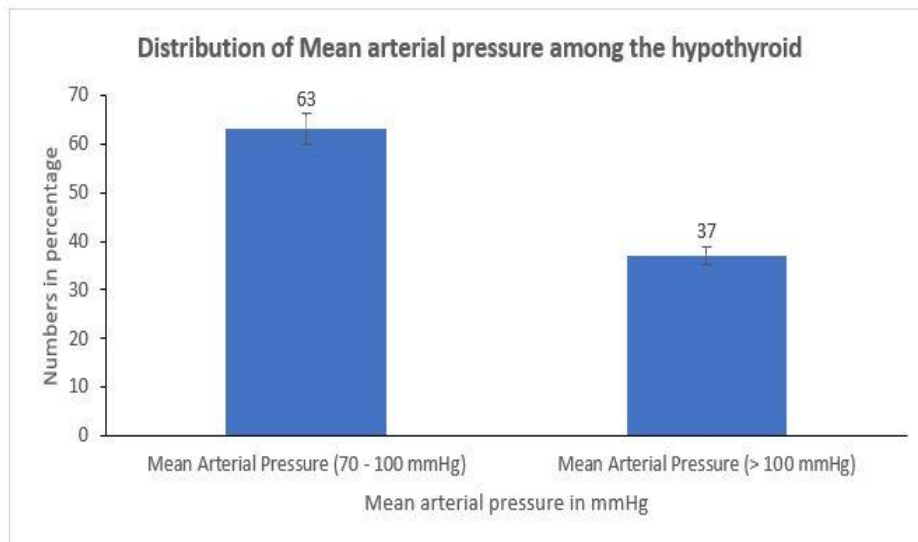


TABLE I: Association of TSH with systemic arterial pressure

PARAMETER	CORRELATION COEFFICIENT (r)	p VALUE
TSH vs SBP	0.05	< 0.001
TSH vs DBP	0.03	< 0.001
TSH vs MAP	0.04	< 0.001
TSH vs PP	0.04	0.12

The mean systolic blood pressure and diastolic blood pressure were 115.7 ± 16.2 and 84.8 ± 11.1 respectively. From the above values the pulse pressure was calculated by systolic pressure-diastolic pressure and found to be 30.9 ± 10.4 and the mean arterial pressure was measured by the formula diastolic pressure + 1/3 of pulse pressure and value was 95.1 ± 12.0 among hypothyroid.

Diastolic pressure was elevated in 64% of study participants compared to systolic blood pressure which was 50% of the study participants. Pulse pressure was narrowed in 80% of study participants. The mean arterial pressure was also elevated in 37% of the study participants.

The distribution of systolic blood pressure amongst the hypothyroid was shown in Figure. I.⁸

The distribution of diastolic blood pressure amongst the hypothyroid was shown in Figure. II.⁸

The distribution of mean arterial pressure amongst the hypothyroid was shown in Figure. III.⁸

The mean serum TSH value among the study participants were 20.8 ± 2.7 . There was a statistically significant positive correlation between TSH and systolic blood pressure, diastolic blood pressure and mean arterial pressure. There was a positive correlation between TSH and pulse pressure which was not statistically significant as shown in Table I.

Discussion:

Our study showed that the diastolic pressure was elevated in study participants compared to systolic blood pressure and also there was narrowing of pulse pressure. Hence mean arterial pressure was

also elevated. There was a statistically significant positive correlation between TSH and systemic arterial pressure and mean arterial pressure. These findings were similar to the following studies. Ittermann et al. in his study involving 10,000 children and adolescents, found a positive correlation between elevated serum TSH levels and both systolic and diastolic blood pressure.⁹ Our study also showed a positive correlation between TSH level and both systemic and diastolic blood pressure.

In a study by Udovcic et al in hypothyroid patients, as DBP increases the pulse pressure narrows.¹⁰ In our study DBP was elevated in 64 % of hypothyroid with narrowing of pulse pressure which was consistent with the above study.

A study by Berta et al. showed that elevated DBP was present in ~30% of patients with overt hypothyroidism.¹¹ In our study, 64% of participants had elevated DBP. Among them 37% of participants were in prehypertension stage, 20% of participants were in stage 1 hypertension, 7% of the participants in stage 2 hypertension.

Thyroid hormones reduces the systemic vascular resistance by downregulating angiotensin II type 1 receptor (AT1R) in vascular smooth muscle cell.¹² It also increases the enzyme involved in converting adenosine monophosphate (AMP) to adenosine which is a potent vasodilator.¹³ It also stimulates Nitric oxide production via activation of the phosphatidyl inositol 3-kinase/protein kinase B pathway in vascular myocytes.¹⁴ By all the mechanisms normal levels of thyroid hormones produce vasodilatation and thus helps in decreasing the blood pressure. In hypothyroidism due to decreased thyroid hormone synthesis systemic vascular resistance increases leading to hypertension. Thus, in our hypothyroid participants, we observed systemic hypertension due to increased systemic vascular resistance.

On contrary to our study, A. Amouzegar et al. in his study showed that there was no significant correlation between serum TSH level and mean arterial pressure.¹⁵ Further studies conducted by Walsh et.al and Liu et.al also found no correlation between continuous measurements of systolic and diastolic blood pressure and serum TSH levels.^{16,17} But our study shows a positive correlation between TSH and mean arterial pressure.

Conclusion:

Our study showed that there is a statistically significant association between serum TSH and SBP, DBP and MAP. Particularly higher percentage of hypothyroid patients had elevated DBP and MAP. There was no statistically significant association between TSH and pulse pressure. This study insists the importance of early detection, prevention and treatment of hypothyroidism. Thyroid profile should also be done in hypertensive individuals so that overt and subclinical hypothyroidism cannot be missed out. Regular BP follow up for all hypothyroid patients should be done.

Limitations:

An elaborate study with larger sample size would be helpful to further strengthen our findings. The study can also be extended with values of T₃ and T₄.

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Conflict of interest: Nil

References:

1. Gottwald-Hostalek, U.; Schulte, B. Low Awareness and Under-Diagnosis of Hypothyroidism. *Curr. Med. Res. Opin.* 2022, 38 (1), 59–64.

2. Mazza, A.; Beltramello, G.; Armigliato, M.; Montemurro, D.; Zorzan, S.; Zuin, M.; Rampin, L.; Marzola, M. C.; Grassetto, G.; Al-Nahas, A.; Rubello, D. Arterial Hypertension and Thyroid Disorders: What Is Important to Know in Clinical Practice? *Ann. Endocrinol.* 2011, 72 (4), 296–303.
3. Fommei, E.; Iervasi, G. The Role of Thyroid Hormone in Blood Pressure Homeostasis: Evidence from Short-Term Hypothyroidism in Humans. *J. Clin. Endocrinol. Metab.* 2002, 87 (5), 1996–2000.
4. Saito, I.; Ito, K.; Saruta, T. Hypothyroidism as a Cause of Hypertension. *Hypertension* 1983, 5 (1), 112–115.
5. Mills, K. T.; Stefanescu, A.; He, J. The Global Epidemiology of Hypertension. *Nat. Rev. Nephrol.* 2020, 16 (4), 223–237.
6. Charles, L.; Triscott, J.; Dobbs, B. Secondary Hypertension: Discovering the Underlying Cause. *Am. Fam. Physician* 2017, 96 (7), 453–461.
7. Young, W. F., Jr.; Calhoun, D. A.; Lenders, J. W. M.; Stowasser, M.; Textor, S. C. Screening for Endocrine Hypertension: An Endocrine Society Scientific Statement. *Endocr. Rev.* 2017, 38 (2), 103–122.
8. Harrison. T.R, Resnick. W. R.; Hauser. S. L., Longo. D. L.; Jameson. J. L., Loscalzo. J. Harrison's Principle of Internal Medicine, 20th edition.; Part 6: Disorders of the Cardiovascular System, Chapter 271: Hypertensive Vascular Disease; Vol. Volume 1.
9. Ittermann, T.; Thamm, M.; Wallaschofski, H.; Rettig, R.; Völzke, H. Serum Thyroid-Stimulating Hormone Levels Are Associated with Blood Pressure in Children and Adolescents. *J. Clin. Endocrinol. Metab.* 2012, 97 (3), 828–834.
10. Udovcic, M.; Pena, R. H.; Patham, B.; Tabatabai, L.; Kansara, A. Hypothyroidism and the Heart. *Methodist DeBakey Cardiovasc. J.* 2017, 13 (2), 55–59.
11. Berta, E.; Lengyel, I.; Halmi, S.; Zrínyi, M.; Erdei, A.; Harangi, M.; Páll, D.; Nagy, E. V.; Bodor, M. Hypertension in Thyroid Disorders. *Front. Endocrinol.* 2019, 10, 482.
12. Fukuyama, K.; Ichiki, T.; Takeda, K.; Tokunou, T.; Iino, N.; Masuda, S.; Ishibashi, M.; Egashira, K.; Shimokawa, H.; Hirano, K.; Kanaide, H.; Takeshita, A. Downregulation of Vascular Angiotensin II Type 1 Receptor by Thyroid Hormone. *Hypertension* 2003, 41 (3), 598–603.
13. Tamajusuku, A. S. K.; Carrillo-Sepúlveda, M. A.; Braganhol, E.; Wink, M. R.; Sarkis, J. J. F.; Barreto-Chaves, M. L. M.; Battastini, A. M. O. Activity and Expression of Ecto-5'-Nucleotidase/CD73 Are Increased by Thyroid Hormones in Vascular Smooth Muscle Cells. *Mol. Cell. Biochem.* 2006, 289 (1), 65–72.
14. Carrillo-Sepúlveda, M. A.; Ceravolo, G. S.; Fortes, Z. B.; Carvalho, M. H.; Tostes, R. C.; Laurindo, F. R.; Webb, R. C.; Barreto-Chaves, M. L. M. Thyroid Hormone Stimulates NO Production via Activation of the PI3K/Akt Pathway in Vascular Myocytes. *Cardiovasc. Res.* 2010, 85 (3), 560–570.
15. Abdi, H.; Gharibzadeh, S.; Tasdighi, E.; Amouzegar, A.; Mehran, L.; Tohidi, M.; Azizi, F. Associations Between Thyroid and Blood Pressure in Euthyroid Adults: A 9-Year Longitudinal Study. *Horm. Metab. Res.* 2018, 50.

16. Walsh, J. P.; Bremner, A. P.; Bulsara, M. K.; O'Leary, P.; Leedman, P. J.; Feddema, P.; Michelangeli, V. Subclinical Thyroid Dysfunction and Blood Pressure: A Community-Based Study. *Clin. Endocrinol. (Oxf.)*2006, 65 (4), 486–491.
- Xue, H.; Li, N.; Yu, J.; Shi, L.; Bai, X.; Hou, X.; Zhu, L.; Lu, L.; Wang, S.; Xing, Q.; Teng, W. A Cross-Sectional Survey of Relationship between Serum TSH Level and Blood Pressure. *J. Hum. Hypertens.*2010, 24 (2), 134–138.
17. Liu, D.; Jiang, F.; Shan, Z.; Wang, B.; Wang, J.; Lai, Y.; Chen, Y.; Li, M.; Liu, H.; Li, C.;