

# Assessment of left atrial mechanical functions in thyroid dysfunction

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## KEY WORDS

diastolic dysfunction, echocardiography, hypothyroidism, left atrial mechanical function

## ABSTRACT

**INTRODUCTION** Thyroid hormone deficiency can lead to the impairment of cardiac function.

**OBJECTIVES** The aim of the study was to determine the left atrial (LA) mechanical function in patients with subclinical hypothyroidism (SHT) and overt hypothyroidism (OHT) and investigate associations of LA mechanical function with diastolic function.

**PATIENTS AND METHODS** Twenty-six patients with newly diagnosed SHT (mean age,  $42.2 \pm 12.5$  years), 21 patients with OHT ( $40.2 \pm 8.5$  years) and 28 healthy volunteers ( $42.4 \pm 11.2$  years) were enrolled in this study. Patients were evaluated by standard M-mode echocardiographic measurements, mitral Doppler flow analysis, and tissue Doppler parameters at the lateral, septal, and right ventricular annuli. LA volumes were measured using the disc method, and the parameters of LA mechanical function were calculated.

**RESULTS** The active emptying volume (AEV) and active emptying fraction (AEF) were significantly higher in the OHT and SHT groups compared with controls. The passive emptying volume and passive emptying fraction were lower in the OHT and SHT groups compared with controls, but the differences were not significant. The conduit volume and the E/A ratio were significantly lower in the OHT and SHT groups compared with controls. The lateral and septal E/Em were significantly higher in the OHT and SHT groups than in the control group, but the septal Em/Am was significantly lower. Diastolic function parameters showed significant associations with AEV and AEF.

**CONCLUSIONS** LA mechanical function is impaired in patients with thyroid dysfunction. Our findings suggest that this impairment is secondary to that of the left ventricular diastolic function.

**INTRODUCTION** It is well known that the cardiovascular system is one of the most important targets of thyroid hormones. These hormones play a critical role in cardiac homeostasis because they can affect cardiac contractility, heart rate, diastolic function, and systemic vascular resistance through genomic and nongenomic mechanisms in both physiological and pathological conditions.<sup>1-3</sup> A decrease in thyroid function has been associated with various heart diseases such as myocardial infarction,<sup>4</sup> coronary atherosclerosis,<sup>5</sup> and congestive heart failure.<sup>6</sup> Previous studies have shown that the restoration of normal thyroid function can reverse abnormal cardiovascular hemodynamics.<sup>1-3</sup>

The left atrium (LA) acts as a reservoir during systole, conduit during early diastole, and active contractile chamber in late diastole.<sup>7</sup> The LA function mechanically facilitates the transition between flow through the pulmonary venous circulation and the intermittent filling of the left ventricle (LV).<sup>8</sup> Approximately 30% of the LV output comes from the LA. During diastole, the LA is directly exposed to LV pressure, which increases when LV diastolic function is impaired.<sup>9</sup> LA mechanical function has been studied in relation to a number of diseases including dilated cardiomyopathy, nonorgan-specific autoimmune disorders, particularly scleroderma, rheumatoid arthritis, and primary Sjogren's syndrome, which

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are closely related to autoimmune thyroid disease, the most frequent cause of goiter in countries without iodine deficiency.<sup>10–14</sup>

The objective of this study was to investigate LA mechanical function and its association with LV diastolic function in patients with subclinical hypothyroidism (SHT) and overt hypothyroidism (OHT). To our knowledge, this is the first study to investigate LA mechanical function in hypothyroid patients.

**PATIENTS AND METHODS** A total of 47 patients who were treated at our endocrinology and metabolism outpatient clinics and whose laboratory tests revealed decreased thyroid function were enrolled in this study. Patients were referred to our outpatient clinics because they had symptoms that could possibly be linked to impairment of thyroid function. The normal reference ranges for thyroid function tests in our biochemical laboratory are as follows: thyroid-stimulating hormone (TSH), 0.4–4 mU/ml; free triiodothyronine (FT<sub>3</sub>), 1.8–4.7 pg/ml; and free thyroxine (FT<sub>4</sub>), 0.8–1.9 ng/dl. The diagnostic criteria for SHT included elevated serum TSH and normal FT<sub>3</sub> and FT<sub>4</sub> levels, while the criteria for OHT included elevated TSH (>12 mU/ml) and decreased FT<sub>3</sub> and FT<sub>4</sub> levels. Based on those criteria, we enrolled 26 patients with SHT (20 women, 6 men; mean age, 42.2 ± 12.5 years) and 21 patients with OHT (17 women, 4 men; mean age, 40.2 ± 8.5 years). The control group included 28 subjects (21 women, 7 men; mean age, 42.4 ± 11.2 years) with no abnormalities on a physical examination, normal results of cardiac imaging tests (electrocardiogram, echocardiogram, exercise stress test), and no history of medication use.

Patients were excluded from the study if they had hypertension, diabetes mellitus, arrhythmia, or if they took medication that could affect heart rate or rhythm and serum thyroid hormone levels. Patients with angina pectoris, a history or suspicion of coronary artery disease, bundle branch block on electrocardiography, or history of chronic obstructive pulmonary disease were also excluded from the study. Moreover, we excluded patients with valvular heart disease, pericardial effusion, or a history of pericarditis on an echocardiogram. Those enrolled in the study were instructed not to smoke or consume caffeinated drinks on the day of the echocardiographic examination. On the day of the echocardiographic examination, 5 patients in the SHT group, 2 patients in the OHT group, and 2 patients in the control group had blood pressure slightly above 140/90 mmHg. Nonetheless, each study participant underwent a thorough examination. The weight and height of each participant was measured and recorded. The body surface area (BSA) was measured with the following formula:  $BSA (m^2) = 0.20247 \times \text{height} (m)^{0.725} \times \text{weight} (kg)^{0.425}$ .<sup>15</sup> Systolic and diastolic blood pressures and heart rate of each participant were recorded, and all patients had electrocardiography done. Thyroid function tests, lipid

panels, and fasting glucose levels were measured using venous blood obtained after 8 hours of fasting. An informed consent was obtained from each participant. The study was approved by the local ethics committee.

**Echocardiographic examination** Standard echocardiography was performed using a Vingmed Vivid System 5 device (General Electric, Horten, Norway). A 2.5 MHz probe was used for Doppler measurements and a 2.5–3.5 MHz probe was used for tissue Doppler measurements. All measurements represented an average taken from 3 cardiac cycles. Two-dimensional echocardiographic measurements (LV dimensions, wall thickness, and mitral inflow velocities) were performed according to the standards published by the American Society of Echocardiography.<sup>16</sup> The LV ejection fraction was calculated according to the Simpson method.<sup>17</sup>

In order to obtain the best signal-to-noise ratio, LV pulsed-tissue Doppler imaging (TDI) was performed in the apical 4-chamber view using a 5-mm pulsed Doppler sample volume with as little optimal gain as possible. Care was taken to align the echo image so that the annular motion was parallel to the TDI cursor. Spectral pulsed-wave Doppler signal filters were adjusted until the Nyquist limit was 15–20 cm/s. The sample volume was placed at the septal and lateral annulus of the mitral valve of the LV and also at the tricuspid annulus of the right ventricle. The peak velocities of the systolic phase (Sm), early diastolic phase (Em), and late diastolic phase (Am) were measured. All echocardiographic measurements were performed by a single operator who was blinded to the clinical and laboratory data of the participants. The Em/Am and E/Em ratios for septal and lateral annular segments were measured.

**Left atrial mechanical function** LA volumes were measured by the disc method in the apical 4-chamber view at end systole ( $V_{\max}$ ), end diastole ( $V_{\min}$ ), and at the onset of atrial systole ( $V_p$ ). All volumes were indexed according to the BSA and expressed as ml/m<sup>2</sup>. The stroke volume was calculated by subtracting the LV end-systolic volume (LVESV) from the LV end-diastolic volume (LVEDV). LVEDV and LVESV were calculated using the biplane method of discs (modified Simpson's rule) in the apical 4-chamber views at end diastole and end systole, respectively, as recommended by the American Society of Echocardiography.<sup>16</sup> The following LA emptying function parameters were also calculated: LA passive emptying volume (PAV) =  $V_{\max} - V_p$ ; LA passive emptying fraction (PEF) =  $(V_{\max} - V_p)/V_{\max}$ ; LA active emptying volume (AEV) =  $V_p - V_{\min}$ ; LA active emptying fraction (AEF) =  $(V_p - V_{\min})/V_p$ ; conduit volume = (LV stroke volume – [ $V_{\max} - V_{\min}$ ]); and LA total emptying volume (TEV) =  $V_{\max} - V_{\min}$ .<sup>18</sup>

**TABLE 1** Clinical and demographic data

|                         | OHT, n = 21 | SHT, n = 26 | Control, n = 28 | P value |
|-------------------------|-------------|-------------|-----------------|---------|
| age, y                  | 40.2 ± 8.5  | 0.5 ± 0.2   | 42.4 ± 11.2     | 0.25    |
| sex, F/M                | 17/3        | 20/6        | 21/7            | 0.69    |
| BMI, kg/m <sup>2</sup>  | 29.8 ± 5.0  | 26.6 ± 3.9  | 27.4 ± 4.2      | 0.40    |
| SBP, mmHg               | 134 ± 10    | 131 ± 10    | 128 ± 11        | 0.39    |
| DBP, mmHg               | 83 ± 6      | 82 ± 8      | 76 ± 8          | 0.53    |
| heart rate, beat/min    | 75 ± 19     | 77 ± 9      | 77 ± 8          | 0.95    |
| FBG, g/dl               | 91 ± 6      | 94 ± 10     | 90 ± 9          | 0.27    |
| TC, mmol/l              | 11.2 ± 2.5  | 11.5 ± 2.4  | 8.8 ± 1.2       | <0.001  |
| TG, mmol/l              | 7.6 ± 1.8   | 8.7 ± 3.8   | 6.8 ± 2.0       | 0.10    |
| HDL, mmol/l             | 2.7 ± 0.8   | 2.8 ± 1.0   | 2.5 ± 0.6       | 0.41    |
| LDL, mmol/l             | 6.9 ± 2.4   | 6.6 ± 2.6   | 5.3 ± 0.9       | 0.02    |
| TSH, mU/ml              | 55.1 ± 43.2 | 10.5 ± 1.3  | 2.1 ± 1.1       | <0.001  |
| FT <sub>3</sub> , pg/ml | 2.3 ± 1.1   | 3.1 ± 0.8   | 2.9 ± 0.7       | 0.01    |
| FT <sub>4</sub> , ng/ml | 0.5 ± 0.2   | 1.1 ± 0.2   | 1.2 ± 0.2       | <0.001  |

Abbreviations: BMI – body mass index, DBP – diastolic blood pressure, FBG – fasting blood glucose, FT<sub>3</sub> – free triiodothyronine, FT<sub>4</sub> – free thyroxine, HDL – high-density lipoprotein, LDL – low-density lipoprotein, SBP – systolic blood pressure, TC – total cholesterol, TG – triglyceride, TSH – thyroid-stimulating hormone

**Statistical analysis** Data analysis was performed using SPSS for Windows version 18.0 (Chicago, Illinois, United States). Quantitative variables were presented as mean and standard deviation. The Shapiro–Wilk test was used to assess the parametric test assumptions of the variables. A one-way analysis of variance was used to compare the 3 groups that met the parametric test assumptions. The Kruskal–Wallis analysis of variance was used in cases where the parametric test assumptions were not met. To determine if there was a significant difference between 2 groups, a 2-sample unpaired *t* test was used when parametric test assumptions were met, while the Mann–Whitney *U* test was used when parametric test assumptions were not met. A *P* value of less than 0.05 was considered statistically significant.

**Reproducibility** The coefficient of variation between the measurements was used to assess the reproducibility of the LA mechanical function. The coefficient of variation was expressed as a percentage and was calculated by dividing the standard deviation of the differences between the repeated measurements by the averages of the repeated measurements. Intraobserver variability was assessed in 15 randomly selected subjects (5 from each of the 3 groups) by repeating the measurements under the same baseline conditions. The intraobserver variability was 7.4%, 5.7%, and 7% for  $V_{\max}$ ,  $V_{\min}$ , and  $V_p$ , respectively.

**RESULTS** There were no differences in demographic, clinical, or laboratory characteristics between the groups (TABLE 1). The M-mode measurements (LVESD and posterior wall) were significantly higher in the OHT group compared with the other groups (*P* = 0.049, *P* = 0.04). There were

no significant differences between any of the other parameters.

**Diastolic function** Based on conventional parameters, the mitral valve Doppler analysis revealed that the A wave was higher, while the E/A ratio was significantly lower in the OHT and SHT groups compared with those in the control group (TABLE 2). The isovolumic relaxation time and isovolumic contraction time were significantly longer in the OHT and SHT groups than in the control group (101 ± 17 ms, 88 ± 26 ms, and 81 ± 18 ms; *P* = 0.02; and 80 ± 62 ms, 62 ± 24 ms, 47 ± 17 ms; *P* = 0.008; respectively). The ejection time was significantly shorter in the OHT and SHT groups compared with the control group (TABLE 2). Conventional diastolic parameters obtained using the mitral valve Doppler analysis were similar between the OHT and SHT groups.

**Tissue Doppler parameters** Tissue Doppler imaging revealed that the Em measured at the lateral and septal annuli was significantly lower in the OHT and SHT groups than in the control group (TABLE 3). However, the E/Em ratio measured at the lateral and septal annuli was significantly higher in the OHT and SHT groups than in the control group (TABLE 3). The Em/Am ratio measured at the septal annulus was significantly lower in the OHT and SHT groups compared with the control group (TABLE 3). There were significant differences between the OHT and SHT groups with respect to the Em/Am septal and Em lateral velocities measured at the lateral annulus (*P* < 0.001 and *P* < 0.001, respectively).

**Left atrial mechanical functions** AEV and AEF were significantly higher in the OHT and SHT groups compared with the control group (9.4 ± 2.8 ml/m<sup>2</sup>, 8.6 ± 3.8 ml/m<sup>2</sup>, and 5.9 ± 3.0 ml/m<sup>2</sup>; *P* = 0.001;

**TABLE 2** Standard echocardiographic and conventional Doppler data

|           | OHT, n = 21 | SHT, n = 26 | Control, n = 28 | P value |
|-----------|-------------|-------------|-----------------|---------|
| LVEDD, mm | 45.8 ± 4.2  | 45.2 ± 4.2  | 46.4 ± 3.4      | 0.55    |
| LVESD, mm | 29.7 ± 4.4  | 26.9 ± 4.2  | 27.4 ± 2.9      | 0.04    |
| IVS, mm   | 9.2 ± 1.2   | 8.8 ± 1.4   | 8.3 ± 1.6       | 0.08    |
| PW, mm    | 9.7 ± 1.2   | 9.0 ± 1.3   | 8.9 ± 0.9       | 0.04    |
| LAD, mm   | 34.2 ± 2.7  | 34.4 ± 3.4  | 34.6 ± 2.3      | 0.88    |
| AoD, mm   | 26.8 ± 2.8  | 26.6 ± 2.5  | 27.6 ± 3.3      | 0.15    |
| E, m/s    | 0.72 ± 0.12 | 0.73 ± 0.16 | 0.78 ± 0.12     | 0.23    |
| A, m/s    | 0.75 ± 0.13 | 0.74 ± 0.14 | 0.60 ± 0.11     | <0.001  |
| E/A       | 0.97 ± 0.20 | 1.0 ± 0.30  | 1.3 ± 0.36      | <0.001  |
| EDT, ms   | 188 ± 61    | 194 ± 49    | 194 ± 49        | 0.49    |
| IVRT, ms  | 101 ± 17    | 88 ± 26     | 81 ± 18         | 0.02    |
| ET, ms    | 242 ± 50    | 257 ± 19    | 274 ± 33        | 0.03    |
| ICT, ms   | 80 ± 62     | 62 ± 24     | 47 ± 17         | 0.008   |
| EF, %     | 66.2 ± 3.3  | 65.8 ± 3.3  | 66.5 ± 2.3      | 0.68    |

Abbreviations: A – mitral inflow late diastolic wave, AoD – aortic diameter, E – mitral inflow early diastolic wave, EDT – E-wave deceleration time, EF – ejection fraction, ET – ejection time, IVS – interventricular septum, IVRT – isovolumic relaxation time, ICT – isovolumic contraction time, LAD – left atrial diameter, LVEDD – left ventricular end-diastolic diameter, LVESD – left ventricular end-systolic diameter, PW – posterior wall

and 61% ± 11%, 58% ± 16%, 39% ± 12%;  $P < 0.001$ ; respectively). The conduit volume was significantly lower in the OHT and SHT groups compared with controls (TABLE 4).

A post-hoc test showed that there were no differences in the LA mechanical function between the SHT and OHT groups. AEF and AEF were significantly higher, while the conduit volume was significantly lower in the SHT and OHT groups compared with controls. A correlation analysis revealed that the E/Em ratio measured at the septal annulus was positively correlated with AEF and AEF ( $r = 0.24$ ,  $P = 0.04$ ;  $r = 0.34$ ,  $P = 0.003$ ; respectively). There was a weak correlation between the septal Em/Am ratio and AEF ( $r = -0.26$ ;  $P = 0.03$ ).

**DISCUSSION** The results of this study suggest that hypothyroidism is associated with decreased LA mechanical function. Conventional methods such as mitral Doppler flow analysis and tissue Doppler imaging revealed that diastolic function in patients with thyroid dysfunction is also impaired. Impairment of the LA mechanical function was associated with impairment of diastolic function.

The cardiovascular system is one of the most important targets of the thyroid hormone, which causes cardiovascular effects both through genomic and nongenomic mechanisms. These effects include altered cardiac contractility, heart rate, diastolic function, and systemic vascular resistance.<sup>19</sup> Thyroid hormone deficiency can alter cardiac muscle function both by decreasing various enzymes that are involved in the regulation of myocyte calcium flow<sup>20</sup> and by decreasing the expression of various contractile proteins.<sup>21</sup> A study on rats reported that functional changes in the cardiac muscle, such as alterations in calcium uptake and release, may jointly lead to

depressed inotropism.<sup>22</sup> Brenta et al.<sup>23</sup> reported that SHT altered collagen, myocardial fiber orientation, tissue water content, and capillary blood flow distribution. Deterioration of cardiac myocyte functions caused by thyroid hormone deficiency may play a role in impaired diastolic function and LA mechanical function. Several diseases (e.g., Hashimoto's disease) may affect cardiac function by autoimmune processes, causing abnormalities such as fibrosis of the heart. We did not investigate pathological changes in cardiac myocytes in patients with hypothyroidism because this was outside the scope of this study.

Previous studies reported impairment of the LV diastolic function as the primary cardiac anomaly in patients with SHT and OHT. It is characterized by prolonged myocardial relaxation and impaired early filling. However, narrowed pulse pressure, systemic hypertension, and subnormal systolic function are observed in OHT, which is characterized by a slight reduction in the ejection fraction and stroke volume.<sup>24</sup> In particular, mechanical LA adaptations compensate for an increase in ventricular filling impairment, in which the reservoir/pump complex is activated to the limit of the preload reserve of the cavity.<sup>25</sup> To ensure sufficient filling, impaired LV filling increases the LA volume, which increases the LA wall stress and leads to the enlargement of the LA.<sup>26</sup> In our study, AEF and AEF were increased in patients with hypothyroidism when compared with controls, and we determined that these parameters were related to late atrial diastolic filling. This indicates that LA mechanical functions may be impaired in patients with early-stage hypothyroidism. The correlation between AEF and AEF and E/A, E/Em, and Em/Am ratios indicates that they are related to diastolic function. Lack of any significant differences in the values of PEV and PEF between patients with hypothyroidism and controls is



**TABLE 3** Tissue Doppler parameters

|                      | OHT, n = 21 | SHT, n = 26 | Control, n = 28 | P value |
|----------------------|-------------|-------------|-----------------|---------|
| Sm (lateral), cm/s   | 8.1 ± 2.4   | 9.4 ± 2.5   | 9.3 ± 2.1       | 0.09    |
| Em (lateral), cm/s   | 9.1 ± 2.2   | 10.0 ± 2.4  | 12.3 ± 2.8      | <0.001  |
| Am (lateral), cm/s   | 7.7 ± 1.8   | 9.2 ± 2.9   | 8.8 ± 2.2       | 0.10    |
| Sm (septal), cm/s    | 7.6 ± 1.6   | 7.6 ± 1.4   | 8.3 ± 1.7       | 0.19    |
| Em (septal), cm/s    | 7.5 ± 2.2   | 7.3 ± 1.9   | 9.8 ± 2.2       | <0.001  |
| Am (septal), cm/s    | 8.0 ± 2.1   | 9.1 ± 1.8   | 8.2 ± 1.8       | 0.14    |
| Sm (tricuspid), cm/s | 12.3 ± 2.0  | 12.4 ± 2.1  | 12.6 ± 2.0      | 0.85    |
| Em (tricuspid), cm/s | 12.5 ± 4.4  | 11.0 ± 2.9  | 12.3 ± 2.3      | 0.32    |
| Am (tricuspid), cm/s | 11.6 ± 4.4  | 12.6 ± 3.1  | 10.8 ± 3.8      | 0.07    |
| Em/Am (lateral)      | 1.20 ± 0.27 | 1.13 ± 0.59 | 1.52 ± 0.65     | 0.09    |
| Em/Am septal         | 0.96 ± 0.25 | 0.83 ± 0.29 | 1.25 ± 0.44     | <0.001  |
| E/Em (lateral)       | 0.08 ± 0.02 | 0.07 ± 0.02 | 0.06 ± 0.01     | 0.015   |
| E/Em (septal)        | 0.10 ± 0.03 | 0.10 ± 0.02 | 0.08 ± 0.02     | 0.006   |

Abbreviations: Am – late diastolic myocardial velocity, Em – early diastolic myocardial velocity, Sm – peak systolic myocardial velocity

**TABLE 4** Left atrial mechanical function

|                                      | OHT, n = 21 | SHT, n = 26 | Control, n = 28 | P value |
|--------------------------------------|-------------|-------------|-----------------|---------|
| V <sub>max</sub> , ml/m <sup>2</sup> | 24.2 ± 6.4  | 25.7 ± 7.0  | 25.0 ± 6.2      | 0.72    |
| V <sub>min</sub> , ml/m <sup>2</sup> | 6.3 ± 2.9   | 6.7 ± 4.5   | 9.2 ± 3.8       | 0.01    |
| V <sub>p</sub> , ml/m <sup>2</sup>   | 15.7 ± 4.4  | 15.2 ± 7.4  | 15.1 ± 5.1      | 0.92    |
| PEV, ml/m <sup>2</sup>               | 8.4 ± 3.8   | 10.4 ± 3.8  | 9.9 ± 2.8       | 0.98    |
| PEF, %                               | 35 ± 10     | 42 ± 16     | 40 ± 10         | 0.07    |
| AEV, ml/m <sup>2</sup>               | 9.4 ± 2.8   | 8.6 ± 3.8   | 5.9 ± 3.0       | <0.001  |
| AEF, %                               | 61 ± 11     | 58 ± 16     | 39 ± 12         | <0.001  |
| CV, ml/m <sup>2</sup>                | 20.7 ± 4.7  | 22.5 ± 7.0  | 28.8 ± 6.0      | <0.001  |
| SV, ml/m <sup>2</sup>                | 38.5 ± 5.4  | 41.6 ± 7.7  | 44.6 ± 6.4      | 0.02    |
| TEV, ml/m <sup>2</sup>               | 17.8 ± 4.3  | 19.0 ± 4.1  | 15.8 ± 3.8      | 0.01    |

Abbreviations: CV – conduit volume, AEF – active emptying fraction, AEV – active emptying volume, PEF – passive emptying fraction, PEV – passive emptying volume, TEV – total emptying volume, V<sub>max</sub> – maximum volume at end-systole, V<sub>min</sub> – minimal volume at end-diastole, V<sub>p</sub> – volume at the beginning of atrial systole

attributed to the fact that patients with a disease that can deteriorate diastolic function, such as hypertension, were excluded from the study. The most consistent cardiac abnormality recognized in SHT patients is LV diastolic dysfunction, which is characterized by reduced myocardial relaxation. There have been a large number of studies using a conventional mitral flow Doppler analysis<sup>27–30</sup> and tissue Doppler analysis<sup>31–33</sup> to investigate LV function in patients with OHT and SHT.

In our study, diastolic function was impaired in both groups as compared with the control group, which is consistent with previous studies. Although there were no significant differences in LA diameters and ejection fraction, we found that isovolumic relaxation time, which is an indicator of diastolic dysfunction, and isovolumic contraction time, which is one of the indicators of systolic function, were prolonged in all hypothyroid patients. A mitral Doppler flow analysis revealed diastolic dysfunction in 25 patients (54%; 23 patients with grade 1, 2 patients with grade 2) in the hypothyroid groups. A low rate of diastolic

dysfunction may be due to the fact that participants were newly diagnosed with hypothyroidism, and that patients with hypertension, which is one of the most common causes of diastolic dysfunction, were excluded from the study. However, diastolic function was assessed using tissue Doppler imaging in addition to conventional methods (mitral Doppler flow analysis). The septal Em/Am ratio was impaired, which is an indication of diastolic dysfunction.

Pearce et al.<sup>34</sup> reported that TSH levels were not associated with LA diameter in the general population in the Framingham Heart Study.<sup>34</sup> In our study, we found no difference in LA diameter between the groups. Although we observed impaired diastolic function in patients with hypothyroidism, LA diameters were similar between the groups. TSH levels were not associated with the LA diameter. During diastole, the LA is directly exposed to LV pressure, which increases with LV diastolic dysfunction. Consequently, LA pressure increases to maintain adequate LV filling. We believe that impairment of the LA mechanical function is caused by the compensation of diastolic dysfunction by the active phase.<sup>12,35</sup> Thyroid hormone deficiency may also have an effect on atrial and ventricular myocytes. However, further large-scale studies are needed to clarify this issue.

In conclusion, PEV and PEF of the LA are impaired in patients with thyroid dysfunction. During the early stages of hypothyroidism, impaired LA mechanical function may be observed simultaneously with LV diastolic dysfunction. We hypothesize that elevated end-diastolic LV pressure and an increase in the AEV of the LA are associated with a compensatory mechanism in LA contraction in the early phase of hypothyroidism.

**Study limitations** The major limitations of this study were the cross-sectional design and limited number of participants in each group, which was primarily owing to our extensive exclusion criteria. In particular, the criteria of hypertension

and suspicion of coronary artery disease (PEV and PEF) led to exclusion of a large number of patients. Another limitation was our lack of possibility to perform coronary angiography, which is the most accurate method to exclude coronary artery disease. LA mechanical function was assessed using the disc method. LA volumes could have been confirmed by the elliptical method. However, the elliptical method also requires M-mode measurements and has several disadvantages associated with M-mode border detection. Although this study provides a novel insight into the effects of thyroid hormones on LA function, it had an exploratory design with a small sample size. A large-scale study is needed to investigate the relationship between thyroid hormones and LA function in the general population. Another limitation of our study is that LA volume was measured using only a 4-chamber view. LA volume is asymmetrical, and usually both 2- and 4-chamber views are required to estimate LA volume.<sup>36</sup>

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# Ocena czynności mechanicznej lewego przedsionka w zaburzeniach czynności tarczycy

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## SŁOWA KLUCZOWE

czynność mechaniczna lewego przedsionka, dysfunkcja rozkurczowa, echokardiografia, niedoczynność tarczycy

## STRESZCZENIE

**WPROWADZENIE** Niedobór hormonów tarczycy może prowadzić do upośledzenia czynności serca.

**CELE** Celem badania była ocena czynności mechanicznej lewego przedsionka (*left atrium* – LA) u chorych z subkliniczną niedoczynnością tarczycy (*subclinical hyperthyroidism* – SHT) i jawną niedoczynnością tarczycy (*overt hyperthyroidism* – OHT) oraz związku czynności mechanicznej LA z czynnością rozkurczową.

**PACJENCI I METODY** Do badania zakwalifikowano 26 chorych z nowo rozpoznaną SHT (średnia wieku:  $42,2 \pm 12,5$  roku), 21 chorych z OHT ( $40,2 \pm 8,5$  roku) i 28 zdrowych ochotników ( $42,4 \pm 11,2$  roku). U pacjentów wykonywano standardowe pomiary echokardiograficzne w trybie M-mode, oceniano przepływ przez zastawkę mitralną metodą doplerowską oraz parametry doplera tkankowego na poziomie pierścieni bocznych, przegrodowego i prawokomorowego. Objętość lewego przedsionka mierzono metodą dysków, a parametry czynności rozkurczowej LA wyliczano.

**WYNIKI** Objętość aktywnego opróżniania (*active emptying volume* – AEV) i frakcja aktywnego opróżniania (*active emptying fraction* – AEF) były znacząco większe w grupach OHT i SHT, w porównaniu z grupą kontrolną. Objętość i frakcja biernego opróżniania były mniejsze w grupach OHT i SHT w porównaniu z grupą kontrolną, ale te różnice nie były znaczące. Objętość przewodzenia oraz wskaźnik E/A były znacząco mniejsze w grupach OHT i SHT w porównaniu z grupą kontrolną. Boczne i przegrodowe E/Em były znacząco większe w grupach OHT i SHT w porównaniu z grupą kontrolną, natomiast przegrodowe Em/Am było znacząco mniejsze. Parametry czynności rozkurczowej wykazywały znaczący związek z AEV i AEF.

**WNIOSKI** U chorych z dysfunkcją tarczycy czynność mechaniczna LA jest upośledzona. Uzyskane wyniki sugerują, że upośledzenie tej czynności jest wtórne do upośledzenia czynności rozkurczowej lewej komory.

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