Impact of subclinical hypothyroidism treatment in systolic and diastolic cardiac function: errata

Ricardo Mendes Martins, Regina Helena Alves Fonseca, Marta Maria Turano Duarte, Vaneska Spinelli Reuters, Márcia Martins Ferreira, Cloyra Almeida, Alexandru Buescu, Patrícia de Fátima dos Santos Teixeira, Mario Vaisman

Correção do Artigo

Impact of subclinical hypothyroidism treatment in systolic and diastolic cardiac function

Arq Bras Endocrinol Metab. 2011;55(7):460-7

Na página 463, na table 1, onde se lê:

Table 1. Baseline characteristics of patients initially randomized to the intervention groups and of patients that concluded the study, according to the intervention group (placebo or L-T4 use)

	Patients initially randomized*		Patients that concluded the study*	
	L-T4 (n = 17)	Placebo (n = 16)	L-T4 (n = 9)	Placebo (n = 13)
Age (years)	49.9 ± 9.3 (49)	44.6 ± 8.1 (45)	51.7 ± 10.2 (49)	44.4 ± 8.9 (44)
TSH (µUI/mL)	7.6 ± 3.6 (7.2)	7.9 ± 3.2 (6.8)	8.6 ± 3.1 (8.4)	8.2 ± 3.4 (7.8)
BMI (kg/m^2)	27.3 ± 3.2	24.9 ± 3.8	27.2 ± 3.8	23.9 ± 3.0
SAP (mmHg)	116.4 ± 15.4	114.6 ± 9.7	116.0 ± 14.6	115.0 ± 8.5
DAP (mmHg)	72.1 ± 12.4	73.1 ± 8.5	69.7 ± 14.7	73.0 ± 6.7
HR (bpm)	64.7 ± 11.8	69.3 ± 7.4	61.3 ± 11.1	67.9 ± 7.0
IRT	96.1 ± 18.8	93.5 ± 13.1	94.6 ± 7.3	91.8 ± 14.5
EW	0.78 ± 0.12	0.72 ± 0.27	0.78 ± 0.1	0.71 ± 0.3
AW	0.63 ± 0.14	0.64 ± 0.06	0.59 ± 0.2	0.62 ± 0.1
E/A	1.3 ± 0.4	1.4 ± 0.3	1.42 ± 0.4	1.36 ± 0.3
DT	243.8 ± 40.1	231.8 ± 52.4	229.8 ± 39.9	228.0 ± 57.2
CO	3.8 ± 1.0	4.1 ± 0.8	4.1 ± 0.8	4.2 ± 0.8
Diastolic dysfunction (%)**	81.2	75.0	66.7	76.9
CI	2.6 ± 0.3	2.7 ± 0.5	2.6 ± 0.2	2.7 ± 0.4
EF	0.73 ± 0.06	0.75 ± 0.05	0.75 ± 0.06	0.75 ± 0.05
ICT	52.5 ± 9.5	50.9 ± 5.9	51.8 ± 10.4	52.0 ± 5.8
LVFT	317.4 ± 32.2	306.2 ± 21.9	323.5 ± 25.6	305.7 ± 20.9
LDV	4.4 ± 0.2	4.5 ± 0.4	4.5 ± 0.3	4.5 ± 0.4
LA	3.4 ± 0.3	3.4 ± 0.3	3.5 ± 0.4	3.4 ± 0.2
AO	2.8 ± 0.3	2.7 ± 0.3	2.9 ± 0.3	2.7 ± 0.3
DLVV	92.1 ± 8.9	99.5 ± 24.5	97.5 ± 15.7	96.3 ± 20.7
SLVV	26.6 ± 11.3	23.1 ± 7.1	28.6 ± 14.8	23.0 ± 6.6
LVM	107.7 ± 28.6	108.8 ± 21.8	130 ± 19.9	105 ± 29.8
Tei RV	0.34 ± 0.15	0.34 ± 0.14	0.36 ± 0.16	0.31 ± 0.15
Tei RV > 0.4 (%)	31.3	33.3	33.3	33.3
Tei LV	0.35 ± 0.13	0.37 ± 0.09	0.40 ± 0.1	0.37 ± 0.1
Rei LV > 0.4 (%)	30.0	40.0	44.4	41.7
Tei LV	0.35 ± 0.13	0.37 ± 0.09	0.40 ± 0.1	0.37 ± 0.1

p-values obtained in the comparison of each variable at baseline in the L-T4 and placebo group were > 0.100. * E/A ≤ 1.0 or IRT ≥ 100 ms or DT ≥ 220 ms. ** Frequency of patients with at least one of the next abnormal parameters of diastolic function: E/A ≤ 1.0, IRT ≥ 100 ms or DT ≥ 220 ms.

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Diastolic dysfunction (%)**	81.2	75.0	66.7	76.9
CI (L/min/m^2)	2.6 ± 0.3	2.7 ± 0.5	2.6 ± 0.2	2.7 ± 0.4
EF (%)	0.73 ± 0.06	0.75 ± 0.05	0.75 ± 0.06	0.75 ± 0.05
ICT (ms)	52.5 ± 9.5	50.9 ± 5.9	51.8 ± 10.4	52.0 ± 5.8
LVFT (ms)	317.4 ± 32.2	306.2 ± 21.9	323.5 ± 25.6	305.7 ± 20.9
LDV (ml)	4.4 ± 0.2	4.5 ± 0.4	4.5 ± 0.3	4.5 ± 0.4
LA (cm)	3.4 ± 0.3	3.4 ± 0.3	3.5 ± 0.4	3.4 ± 0.2
AO (cm)	2.8 ± 0.3	2.7 ± 0.3	2.9 ± 0.3	2.7 ± 0.3
DLVV (ml)	92.1 ± 8.9	99.5 ± 24.5	97.5 ± 15.7	96.3 ± 20.7
SLVV (ml)	26.6 ± 11.3	23.1 ± 7.1	28.6 ± 14.8	23.0 ± 6.6
LVM (g)	107.7 ± 28.6	108.8 ± 21.8	130 ± 19.9	105 ± 29.8
Tei RV	0.34 ± 0.15	0.34 ± 0.14	0.36 ± 0.16	0.31 ± 0.15
Tei RV > 0.4 (%)	31.3	33.3	33.3	33.3
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p-values obtained in the comparison of each variable at baseline in the L-T4 and placebo group were > 0.100. * E/A ≤ 1.0 or IRT ≥ 100 ms or DT ≥ 220 ms. ** Frequency of patients with at least one of the next abnormal parameters of diastolic function: E/A ≤ 1.0, IRT ≥ 100 ms or DT ≥ 220 ms.

Na página 464, na table 2, onde se lê:

Table 2. Variation in specific parameters of diastolic function, left ventricle mass and aortic root diameter

	Variation (mean ± SD)				
	L-T4	p-value*	Placebo	p-value†	p-value‡
ΔDT	-9.33 ± 38.83 (-30.0)	0.231	-18.77 ± 60.17 (-30.0)	0.437	0.684
ΔIRT	-5.78 ± 13.36 (-8.0)	0.634	-5.15 ± 23.08 (+2.0)	0.196	0.943
ΔEW	-0.02 ± 0.13 (-0.01)	0.892	0.13 ± 0.33 (+0.03)	0.470	0.222
ΔAW	-0.01 ± 0.14 (-0.01)	0.817	-0.02 ± 0.12 (-0.04)	0.389	0.759
ΔE/A	0.03 ± 0.42 (+0.04)	0.491	0.05 ± 0.23 (+0.04)	0.283	0.867
ΔLVM	-1.327 ± 45.7 (-17.0)	0.499	+9.5 ± 32.14 (+5.85)	0.305	0.351
ΔAO	**-0.12 ± 0.2**	**0.102**	**+0.12 ± 0.3**	**0.184**	**0.043**

*: Comparison between before and after means of the L-T4 group (paired test); †: Comparison between before and after means in the placebo group (paired test); ‡: Comparison between mean variations that occurred in the L-T4 and placebo groups.

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ΔE/A	0.03 ± 0.42 (+0.04)	0.491	0.05 ± 0.23 (+0.04)	0.283	0.867
ΔLVM (g)	-1.327 ± 45.7 (-17.0)	0.499	+9.5 ± 32.14 (+5.85)	0.305	0.351
ΔAO (cm)	**-0.12 ± 0.2**	**0.102**	**+0.12 ± 0.3**	**0.184**	**0.043**

*: Comparison between before and after means of the L-T4 group (paired test); †: Comparison between before and after means in the placebo group (paired test); ‡: Comparison between mean variations that occurred in the L-T4 and placebo groups.

Hypertension-related factors in patients with active and inactive acromegaly: errata

Daniela Fedrizzi, Ticiana Costa Rodrigues, Fabíola Costenaro, Rosana Scalco, Mauro Antônio Czepielewski

Correção do Artigo

Hypertension-related factors in patients with active and inactive acromegaly

Arq Bras Endocrinol Metab. 2011;55(7):468-74

No Abstract onde se lê:

Conclusions: Our findings suggest that blood pressure levels in patients with active acromegaly are very similar, and depend on excess GH. However, once the disease becomes controlled and IGF-1 levels decrease, their blood pressure levels will depend on the other cardiovascular risk factors.

Leia-se:

Conclusions: Blood pressure (BP) levels in patients with active acromegaly dependent of the GH excess. However, once the disease becomes controlled and IGF-1 levels decrease, their blood pressure levels are depend on the other cardiovascular risk factors.

A rational approach to the diagnosis of polycystic ovarian syndrome during adolescence: errata

Paulina M. Merino, Ethel Codner, Fernando Cassorla

Correção do Artigo

A rational approach to the diagnosis of polycystic ovarian syndrome during adolescence

Arq Bras Endocrinol Metab. 2011;55(8):590-8

Na página 595, primeira coluna, segundo parágrafo onde se lê:

Villarroel and cols. (40), from our group, reported that girls with PCOM have higher AMH levels than girls without PCOM (72.5 ± 6.1 *vs.* 33.4 ± 2.6 pmol/L; P < 0.0001), and lower FSH levels (5.4 ± 0.3; 6.2 ± 0.2 mUI/ml; P < 0.036).

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Na página 595, segunda coluna, último parágrafo onde se lê:

In 160 obese adolescents girls, the subgroup with the metabolic syndrome (48 girls) demonstrated significantly higher testosterone and DHEA-S concentrations compared with the 112 girls without MS.

Leia-se:

In 160 obese adolescents girls, the subgroup with the metabolic syndrome **(MS)** (48 girls) demonstrated significantly higher testosterone and DHEA-S concentrations compared with the 112 girls without MS.

Na página 595, na legenda da figura, onde se lê:

Figure 1.

Leia-se:

Figure 1. Longitudinal study of polycystic ovarian morphology in individual girls at baseline and follow-up. The ultrasonographic status of the 18 girls who participated in the three evaluations is shown at the right; the numbers for the two girls who withdrew from the study are shown on the left. Adapted from "Codner E, et al. Polycystic ovarian morphology in postmenarchal adolescents. Fertil Steril. 2011;95:702-6".

Na página 596, Table 2, onde se lê:

Table 2. Proposed diagnostic criteria for polycystic ovarian syndrome in adolescence. Features: + Present, - Absent, +/- Controversial. The presence of a "√" indicates that this classification agrees that the diagnosis of PCOS phenotype. The diagnosis of oligo-anovulation and hyperandrogenism differ between adults and adolescents (see text). Adapted from "Merino P, Schulin-Zeuthen C, Codner E. Current diagnosis of polycystic ovary syndrome: expanding the phenotype but generating new questions. Rev Med Chil. 2009;137:1071-80" and "Azziz R, Carmina E, Dewailly D, et al. Positions statement: criteria for defining polycystic ovary syndrome as a predominantly hyperandrogenic syndrome: an Androgen Excess Society guideline. J Clin Endocrinol Metab. 2006;91:4237-45".

	Potential phenotypes															
Diagnostic criteria	**A**	**B**	**C**	**D**	**E**	**F**	**G**	**H**	**I**	**J**	**K**	**L**	**M**	**N**	**O**	**P**
Biochemical hyperandrogenism	+	+	+	+	-	-	+	-	+	-	+	-	-	-	+	-
Hirsutism	+	+	-	-	+	+	+	+	-	-	+	-	-	+	-	-
Oligo-anovulation	+	+	+	+	+	+	-	-	-	+	-	-	+	-	-	-
PCOM	+	-	+	-	+	-	+	+	+	+	-	+	-	-	-	-
PCOS in adults																
NIH 1990	√	√	√	√	√	√										
Rotterdam 2003	√	√	√	√	√	√	√	√	√	√						
AES 2006	√	√	√	√	√	√	√	√	√	√						
PCOS in adolescence	√	√	√	√	√	√	√	√	√	√						
Our proposal	√	√	+/-	√	√	√	+/-									
Long-term metabolic risk	√	√	√	√	√	√	+/-	+/-	+/-	-						

Leia-se:

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Biochemical Hyperandrogenism	+	+	+	+	-	-	+	-	+	-	+	-	-	-	+	-
Hirsutism	+	+	-	-	+	+	+	+	-	-	+	-	-	+	-	-
Oligo-anovulation	+	+	+	+	+	+	-	-	-	+	-	-	+	-	-	-
PCOM	+	-	+	-	+	-	+	+	+	+	-	+	-	-	-	-
PCOS in adults																
NIH 1990	√	√	√	√	√	√										
Rotterdam 2003	√	√	√	√	√	√	√	√	√	√						
AES 2006	√	√	√	√	√	√	√	√	√							
PCOS in Adolescence																
Our proposal	√	√	+/-		√	√	+/-									
Metabolic risk long-term	√	√	√	√	√	√	+/-	+/-	+/-	-						

Na página 596 onde se lê:

Disclosure: no potential conflict of interest relevant to this article was reported.

Leia-se:

Disclosure: this work was partially supported by Fondecyt Grants 1050452 and 1100123 to Ethel Codner and 1095118 to Fernando Cassorla.

Hormônio de crescimento em crianças e adolescentes com fibrose cística: errata

Pollyana Garcia Amorim, Thaís de Barros Mendes, Lílian Santiago Pinho de Oliveira, Gil Guerra-Júnior, José Dirceu Ribeiro

Correção do Artigo

Impact of subclinical hypothyroidism treatment in systolic and diastolic cardiac function

Hormônio de crescimento em crianças e adolescentes com fibrose cística

Arq Bras Endocrinol Metab. 2011;55(9):671-6

Na página 671 onde se lê:

A fibrose cística (FC) é a doença genética com herança autossômica recessiva mais comum na população de origem caucasiana, afetando cerca de 1 a 2.500 nascidos vivos.

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A fibrose cística (FC) é a doença genética com herança autossômica recessiva mais comum na população de origem caucasiana, afetando cerca de 1 **em** 2.500 nascidos vivos.