# Evaluation of selected clinical and diagnostic parameters in girls with *anorexia nervosa* (I)

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Submitted: 2008-06-23 Accepted: 2008-07-05 Published online: 2008-08-30

*Key words:* anorexia nervosa; body weight loss; routine laboratory tests; hormonal tests.

Neuroendocrinol Lett 2008; 29(4):421-427 PMID: 18766154 NEL290408R01 © 2008 Neuroendocrinology Letters • www.nel.edu

**OBJECTIVES:** Body weight loss in patients with *anorexia nervosa* (AN) is accompanied by a number of hormonal and metabolic disorders. The scope and intensity of these disorders may have a considerable influence on the prognosis in this disease. The goal of the study was an evaluation of selected diagnostic examinations in comparison with clinical data of female patients with AN.

**PATIENTS & METHODS:** Retrospective studies involved eighty-seven (87) patients with AN. On therapy commencement, routine laboratory tests (full blood cell count, serum concentrations of sodium, potassium, glucose, cholesterol, triglycerides, total calcium, phosphates, total protein and the urea) and hormonal tests (TSH, TSH, FT<sub>4</sub>, FT<sub>3</sub>, E<sub>2</sub>, T, cortisol measured at 8<sup>00</sup>, 17<sup>00</sup> and 24<sup>00</sup>, LH and FSH in stimulation test with GnRH) were performed in each patient. Correlations were determined between clinical data and the measured hormone concentrations.

**RESULTS:** In the studied girls, the mean values of routine laboratory tests, performed at the beginning of the therapy, were within the normal ranges (except hypernatremia). The mean concentrations of LH, FSH and  $FT_4$  were below reference values; the mean concentration of cortisol considerably exceeded the standard range. Statistically significant relations were demonstrated between BMI values and the concentrations of LH,  $E_2$  and cortisol.

**CONCLUSIONS**: Body weigh loss is not significantly reflected by abnormal results of routinely performed laboratory tests. Hypogonadotropic hypogonadism and hypercortisolemia are the most characteristic hormonal symptoms in girls with AN.

#### **INTRODUCTION**

Abstract

Anorexia nervosa (AN) is a pathological syndrome, in which food intake is consciously limited to obtain slim body shape by successively increasing body weight loss. The etiology of this disease has still been unknown, while researchers have commonly discarded any attempts to identify one causative factor. Instead, biological, psychological, family, as well as social aspects are taken into account. A distinct increase of AN morbidity is actually observed [7]. The incidence of this disease, assessed in the population of girls in puberty period and of young women, is within the range from 0.22% up to 0.85% [5].

Body weight deficit, sometimes substantial, is a dominating syndrome in AN. Body weight loss is accompanied in patients with AN by numerous metabolic and hormonal abnormalities. The scope and severity of these disorders may, from the clinical point of view, significantly affect the prognosis and the therapeutic process in these girls.

The goal of the study was an evaluation of the results of selected hormonal tests and routine laboratory tests, the latter routinely performed in patients with AN, and a correlation of obtained laboratory results with the clinical status of the patients.

### PATIENTS AND METHODS

Retrospective studies included eighty-seven (87) patients with AN, hospitalised at the Department of Endocrinology and Metabolic Diseases of the Medical University in Lodz, during the years 1995–2003. The diagnosis of AN was made, following the criteria of the American Psychiatric Society and the DSM-IV-R classification of mental diseases [2]. It was the first hospitalisation because of feeding disorders for all the patients, included in the reported study. In all the patients, secondary amenorrhoea was found. The age of the studied patients was 13.8 to 21.1 years (the mean age:  $16.1\pm1.5$ ; x±SD).

At the therapy onset clinical data were measured: height, body weight at the therapy onset (BW-O), body mass index (BMI), body weight loss (BWL), the percentage of body weight loss (BWL%), body weight loss with respect to disease duration (BWL/time), the percentage of body weight loss with respect to disease duration (BWL%/time).

In each patients, on the beginning of the therapy, routine laboratory tests were ordered, such as blood cell count, including the red blood cell count (RBC) haemoglobin (Hb) concentration, the hematocrit (Ht) index, the mean percentual concentration of hemoglobin in erythrocyte (MCHC), the white blood cell count (WBC), and the number of thrombocytes (PLT), as well as concentrations of: sodium (Na), potassium (K), total calcium (Ca), phosphates (P), glucose (Glu), cholesterol (Ch), triglycerides (TG), total protein (Prot) and Urea (Ure). Also, serum concentrations of thyrotropin (TSH), free thyroxine (FT<sub>4</sub>), free triiodothyronine (FT<sub>3</sub>), estradiol (E<sub>2</sub>), testosterone (T) and cortisol at 8<sup>00</sup> (Cort8), at 17<sup>00</sup> (Cort17) and at 24<sup>00</sup> (Cort24) were measured. A stimulation test with gonadoliberin (GnRH) was also performed to assess luteotropin (LH) and follitropin (FSH) release before, after 30 (LH30, FSH30) and after 60 minutes (LH60, FSH60) the administration of 100 µg GnRH.

All the hormonal measurements were performed by the electrochemiluminescence assay (ECLIA).

Correlations was determined between the clinical data and the measured hormone concentrations.

Statistical analysis was performed, based on typical location (the mean value, the median value) and dispersion (standard deviation) measures. Parameters in nominal scales were compared by the typical Chi<sup>2</sup> test. The ANOVA analysis of variance or typical variants of Student's t test was used to compare differences between the mean values. The typical correlation coefficients (r-Spearman's and Pearson's) were also calculated.

The level of significance at p<0.05 was accepted for all the performed comparisons and calculated statistics.

## RESULTS

Clinical data of the studied group were collected during subjective examination of the patients and in the history, obtained from their parents, and are presented in Table 1.

Table 2 presents results of routine laboratory tests, performed in the examined patients at the start of their therapy.

Having analysed the obtained results, we found out that in the studied group of girls with AN, only the mean concentration of sodium (147.75±3.76 mmol/l) was above the range of the reference values (136–145 mmol/l). The mean values of other tests were within

Гable	1. Clinical	data of	examined	patient with	AN at the	onset of therapy.
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	The mean value±SD	The median value	Minimal value	Maximal value
Age (years)	16.1 ± 1.5	15.9	13.8	21.1
Height (meters)	$1.62 \pm 0.07$	1.63	1.46	1.78
BW-O	38.11 ± 5.27	38.00	24.70	50.40
BMI value	14.40 ± 1.52	14.47	11.13	17.26
Disease duration (months)	11.1 ± 5.8	9.0	3.0	28.0
BWL (kg)	15.95 ± 6.17	15.00	8.00	44.50
BWL% (%)	29.27 ± 7.03	28.84	15.69	48.00
BWL/time (kg/month)	1.82 ± 1.11	1.56	0.39	7.42
BWL%/time (%/month)	3.34 ± 1.71	2.97	0.77	8.06

	The mean value ±SD	The median value	Minimal value	Maximal value
RBC (M/µl)	4.36 ± 4.69	4.41	2.55	5.32
Hb (g/dl)	12.81 ± 1.13	12.90	8.30	16.00
Ht (%)	39.63 ± 3.67	40.00	25.50	50.00
MCHC (g/dl)	33.12 ± 1.00	33.10	30.60	35.30
WBC (K/µl)	4.58 ± 1.11	4.30	2.26	7.74
PLT (K/µl)	193.38 ± 45.20	192.00	99.00	320.00
Na (mmol/l)	147.75 ± 3.76	149.00	137.00	156.00
K (mmol/l)	4.30 ± 0.31	4.30	3.40	4.90
Ca (mmol/l)	$2.26 \pm 0.08$	2.27	2.00	2.51
P (mmol/l)	1.22 ± 0.09	1.20	0.90	1.43
Glu (mg/dl)	70.93 ± 6.86	70.00	51.00	91.00
Ch (mg/dl)	187.69 ± 32.71	187.00	111.00	268.00
TG (mg/dl)	75.24 ± 18.07	73.00	44.00	164.00
Prot (mg/dl)	$6.84 \pm 0.64$	6.80	5.30	7.70
Ure (mg/dl)	32.48 ± 7.78	32.00	17.00	49.00

**Table 2.** Results of routine laboratory tests in the examined girls with AN at the onset of therapy.

**Table 3.** Concentrations of hormones in blood serum of the examined girls with AN at the onset of therapy.

	The mean value $\pm$ SD	The median value	Minimal value	Maximal value
TSH (mIU/l)	1.37 ± 0.91	1.10	0.20	4.80
FT <sub>4</sub> (ng/dl)	0.89 ± 0.21	0.90	0.70	1.48
FT <sub>3</sub> (pg/ml)	1.63 ± 0.42	1.70	0.80	3.73
LH (IU/I)	$0.42 \pm 0.76$	0.10	0.10	5.70
LH30 (IU/I)	2.45 ± 2.54	2.10	0.10	13.00
LH60 (IU/I)	2.12 ± 2.30	1.70	0.10	11.90
FSH (IU/I)	2.21 ± 2.32	1.20	0.10	8.50
FSH30 (IU/I)	6.15 ± 4.14	5.60	0.20	20.79
FSH60 (IU/I)	7.89 ± 4.96	7.30	0.50	25.82
E <sub>2</sub> (pg/ml)	15.18 ± 9.30	12.00	5.00	43.00
T (ng/ml)	0.33 ± 0.13	0.30	0.04	0.70
Cort8 (µg/dl)	$25.80 \pm 6.72$	25.61	12.58	44.70
Cort17 (µg/dl)	15.27 ± 4.25	14.79	5.70	27.41
Cort24 (µg/dl)	6.27 ± 3.79	5.34	1.60	21.30

normal ranges, although the mean concentration of glucose (70.93±6.86 mg/dl) only slightly exceeded the lower range of the reference value (70–105 mg/dl).

In some patients, the results of particular routine laboratory tests were beyond their normal ranges. Decreased concentrations of the following substances were observed: Glu – in 40 patients (45.97%), Hb – in 12 patients (13.79%), Ca – in 11 examined girls (12.64%), Prot – in 5 patients (5.74%), K – in 2 girls (2.29%). The WBC was decreased in 25 (28.73%), and the RBC – in 9 patients (10.34%). The Ht was decreased in 6 girls (6.69%), and the MCHC value – in 10 patients

(11.49%). Elevated concentrations of the following substances were found: Ch – in 22 patients (25.28%), Ure – in 3 patients (3.44%), Hb – in 3 girls (3.44%) and elevated Ht – in 4 patients (4.59%).

Table 3 presents the results of hormonal tests, performed in the treated patients. We found that the mean concentrations of FT<sub>4</sub> (0.89±0.21 ng/dl), LH (0.42±0.76 IU/l) and FSH (2.21±2.32 IU/l) were below the reference values in the studied group of anorectic girls (reference values, respectively: 0.93–1.7 ng/dl and 2.4–12.6 IU/l)). The mean concentrations of Cort8 (25.80±6.72 µg/dl), Cort17 (15.27±4.25 µg/dl) and Cort24 (6.27±3.79 µg/

Table 4. Results of correlation studies between hormonal concentrations and clinical data at the therapy onset in the
examined girls with AN; r – correlation coefficient, p – the level of significance. Statistically variable correlations

		LH	LH30	LH60	FSH	FSH30	FSH60
Height	r	-0.101	-0.080	-0.047	-0.037	0.008	-0.013
	р	0.353	0.462	0.666	0.732	0.945	0.905
BW-O	r	0.159	0.260	0.229	0.161	0.174	0.105
	р	0.140	0.015	0.033	0.136	0.107	0.332
BMI	r	0.298	0.417	0.351	0.248	0.232	0.159
	р	0.005	0.000	0.001	0.021	0.031	0.141
Disease duration	r	0.015	0.144	0.183	0.151	0.165	0.197
	р	0.889	0.182	0.089	0.164	0.126	0.068
BWL	r	-0.094	-0.124	-0.074	-0.033	-0.100	-0.079
	р	0.388	0.251	0.497	0.759	0.357	0.469
BWL%	r	-0.159	-0.231	-0.171	-0.092	-0.137	-0.089
	р	0,141	0.032	0.114	0.397	0.204	0.415
BWL/ time	r	-0.030	-0.202	-0.193	-0.122	-0.194	-0.210
	р	0.784	0.061	0.073	0.262	0.072	0.051
BWL%/ time	r	-0.045	-0.255	-0.248	-0.156	-0.213	-0.212
	р	0.680	0.017	0.020	0.150	0.048	0.048

**Table 5.** Results of correlation studies between hormonal concentrations and clinical data at the onset of therapy in the examined girls with AN.

 Statistically variable correlations

		E <sub>2</sub>	т	Cort8	Cort17	Cort24	FT <sub>3</sub>	FT <sub>4</sub>	TSH
Height	r	0.130	0.087	-0.118	-0.063	0.041	0.065	0.067	0.024
	р	0.231	0.425	0.276	0.564	0.708	0.550	0.536	0.823
BW-O	r	0.282	0.128	-0.274	-0.263	-0.259	0.224	0.228	-0.015
	р	0.008	0.239	0.010	0.014	0.015	0.037	0.034	0.890
BMI	r	0.286	0.092	-0.277	-0.309	-0.383	0.258	0.253	-0.021
	р	0.007	0.397	0.009	0.004	0.000	0.016	0.018	0.848
Disease duration	r	0.011	0.093	-0.190	-0.035	0.071	0.244	0.316	0.008
	р	0.922	0.394	0.078	0.749	0.513	0.023	0.003	0.940
BWL	r	-0.069	0.080	0.230	0.295	0.352	-0.211	-0.139	-0.082
	р	0.524	0.462	0.032	0.006	0.001	0.050	0.199	0.449
BWL%	r	-0.199	0.024	0.365	0.404	0.468	-0.303	-0.259	-0.095
	р	0.064	0.829	0.001	0.000	0.000	0.004	0.015	0.384
BWL/ time	r	-0.095	-0.038	0.281	0.198	0.235	-0.357	-0.296	-0.000
	r	0.383	0.724	0.008	0.066	0.029	0.001	0.005	0.997
BWL%/ time	r	-0.139	-0.073	0.321	0.184	0.210	-0.424	-0.367	0.029
	р	0.199	0.503	0.002	0.089	0.051	0.000	0.000	0.787

d/l) significantly exceeded their normal range (reference values, respectively: 6.2-19.4, 2.3-12.3, below  $3.6 \mu g/dl$ ).

and  $FT_3$  – in 2 patients (2.29%). Also, TSH concentration exceeded the reference values in 2 patients.

In some patients, the values of obtained hormonal test results, were beyond their reference ranges. For example, concentrations of the following substances were below their normal range:  $E_2$  – in 39 patients (44.82%)

Table 4 and Table 5 present the correlation status between studied parameters. It appears from the data in the table that in the group of examined patients, BW-O positively correlated with LH30 and LH60, as well as with  $E_{2}$ ,  $FT_{3}$  and  $FT_{4}$  concentrations, while it was reversibly proportional to Cort8, Cort17 and Cort24 concentrations. BMI values on admission positively correlated with LH30 and LH60, with FSH and FSH30 concentrations also with  $E_2$ , FT<sub>3</sub> and FT<sub>4</sub> concentrations, while being reversibly proportional to Cort8, Cort17 and Cort24 concentrations. Disease duration in the studied group positively correlated only with FT<sub>3</sub> and FT<sub>4</sub> concentrations.

The most pronounced positive correlation was observed between BWL% and Cort24, while the strongest negative correlation was found between BWL%/time and  $FT_3$  concentration. No statistically significant correlations were observed between the studied clinical parameters and T and TSH concentrations.

#### DISCUSSION

So far, it has not been possible to establish either an unequivocal clinical picture or the picture of metabolic disorders, typical for anorexia nervosa (AN); most probably, it may not be possible to in the near future either. Attempts are undertaken to determine particular auxiliary studies, which would, on one hand, be characteristics for AN, while, on the other, they would be useful in differentiating this disease with other organic pathologies, also leading to cachexia of the organism [25]. It is very important in the light of growing morbidity and mortality in result of this disease.

The opinions, regarding the value of routine laboratory tests in patients with AN, are not synonymous, thus preventing from any more general conclusions to be drawn, regarding their prognostic significance [13, 25, 28].

In our studies, the mean values of the results of routine laboratory tests, performed at the onset of therapy, were within reference value ranges, except the mean serum concentration of sodium, which slightly exceeded the normal range. However, while analysing the results of tests, performed in particular patients, we found out disorders of water-electrolyte balance, lipid metabolism, carbohydrate metabolism, as well as abnormal blood cell counts.

Dehydration has a considerable impact on the functioning of the whole organism of a patient with AN, causing serious disturbances, such as water-electrolyte balance disorders, especially hypokalemia. This pathology is interpreted as resulting from secondary hyperaldosteronism, induced by chronic hypovolaemia. In turn, potassium deficit enhances adynamia, dependent on muscular atrophy from malnutrition [6, 10]. In our studies, hypokalemia was observed in single patients (2.29%), while hypernatremia was identified in the majority in the patients, reflected by elevated mean sodium concentration in blood serum.

Hypoglycemia is rather sporadic in AN but if appears, it may lead to extremely dramatic developments. Glucose concentrations in the range, approximating 70 mg/ dl, are fairly common, however, the above mentioned biochemical deviation in patients with AN reveals an entirely asymptomatic course. Mattingly et Bhanji [13] are of the opinion that moderately decreased glucose concentration in AN may activate neurotransmitters in the hypothalamus, which stimulate corticotrophin (ACTH) and growth hormone (GH) secretion. The same authors underline also the effect of reduced glucose metabolism, occurring in the brains of girls with AN – because of low carbohydrate content (including monosaccharides) in diet – on the disease development [13]. The mean concentrations of glucose in the studied group only slightly exceeded the lower range of the reference values, what is in compliance with the observations of Mattingly and Bhanji [13]. Hypoglycemia was observed in 45.97% of the studied patients.

Disturbances in fat metabolism are manifested by hypertriglyceridemia and hypercholesterolemia. Zopii *et al.* have found total cholesterol concentrations to be significantly elevated in fasting condition of patients with AN [29]. This fact may, on one hand, be interpreted as resulting from improper nutrition, while on the other, it may be seen as a consequence of decreased thyrometabolic activity, especially that of FT<sub>3</sub>. It is also assumed that high levels of cholesterol, triglycerides and apolipoproteins B, with long maintained low body weight, are the risk factors of coronary disease [20, 29]. Scarce cases of hypercholesterolemia were observed, namely in 25.28% of all the studied patients.

Elevated concentrations of urea and decreased concentrations of creatinine have also been described in girls with AN [10, 15]. Our studies indicated normal urea concentrations in the majority of examined patients, its elevated values being observed but only in 3.44% of the patients.

A number of authors have provided evidence for insufficient calcium intake in the diet of anorectic patients [11, 12, 25]. Reduced calcium absorption in the alimentary tract and its enhanced excretion with urine is an additional factor, impairing the calcium-phosphate metabolism in patients with disturbed eating habits [18]. In spite of that, some authors have not observed either hypocalcemia or hypocalcemia-related, increased concentration of parathormone (PTH) in girls with AN [5]. Other researchers do, however, confirm the possibility of decreased concentration of total calcium in blood serum [19, 16]. Śmiech et al. [19], having examined forty-seven (47) patients with AN, found that hypocalcaemia and hypercalcuria occurred significantly more frequently in the study group than in twenty-seven (27) healthy women, although the mean values of calcium concentration in serum were within the normal range in either group. In our observation, hypocalcaemia was noted in 12.64% of the anorectic girls.

Decreased concentration of phosphates in blood serum is rather rarely observed. According to Harris [10], it occurs late and should be regarded as poor prognosis, since it is associated with rapid decompensation – myolysis of transversely-striated muscles, abnormal function of erythrocytes, secondary to adenosinotriphosphate (ATP) and 2–3 DPG, what leads to congestive heart failure and death [10]. In our patients with AN, normal concentrations of phosphates were found.

The reports, concerning changes of blood cell counts in anorectic girls are rather scarce. Zerbe [27] reports that leucopenia and anaemia, the latter in moderate degree, may occur in patients with eating disorders. Abella *et al.* [1] have found, on the basis of forty-four (44) cases of AN that degenerative changes in bone marrow correlate with the degree of body weight loss, while they have not noticed any distinct changes, regarding the number and structure of peripheral blood cells. In our studies, leucopenia was observed in 28.73% of the patients, aneamia in 10.34% of the studied patients.

The mechanism of menstruation disorders in anorectic girls has not yet been completely analysed, being still an object of interest among endocrinologists. It has been found that in patients with AN the response of gonadotropins to GnRH is disturbed, resembling the prepubescent period [7, 13]. Moreover, fasting impairs the release of hypothalamic GnRH [12, 23].

Samuels *et al.* have found that already short-term fasting (56 hours) decreases LH concentration and the amplitude of its pulses by 30%, while leaving LH pulse frequency unchanged. FSH concentrations drop by 13% only and without amplitude changing [17].

Our studies unequivocally confirm the observations of other authors, regarding hypogonadotropic hypogonadism to be found in anorectic girls with gonadotropin response to GnRH stimulation being typical for the prepubertal period.

Disorders in the hypothalamo-pituitary system lead to hypoestrogenism [7, 9, 14, 24]. Decreased concentration of oestrogens in girls with AN causes many secondary disturbances, both hormonal and metabolic. It is assumed that hypooestrogenism is one the causes of osteoporosis, observed in patients with AN [3, 11, 12, 14].

Beside the low secretion rate of  $E_2$  by the ovary, normal concentration of circulating testosterone has been described [16] and also confirmed in the present study. Yen [26] describes disturbed peripheral metabolism of both oestradiol and testosterone. The author claims that, in fasting girls, a shift of  $E_2$  metabolism takes place from 16 $\alpha$ -hydroxylation pathway onto 2-hydroxylation pathway, resulting in reduced synthesis of estriol, while the synthesis of catecholoestrogens (2-hydoxyoestrone) is unproportionally elevated. These are endogenous antioestrogens, binding with the receptors of oestrogens, however, not manifesting their biological activity. It is of particular importance in the brain [26]. In the present study, we found hypooestrogenism in 44.82% of anorectic patients.

Hypercortisolemia in AN is induced by excessive stimulation of the hypothalamo-pituitary-adrenal axis. Disorders of this axis are very similar to those, observed in 30–50% of patients with depression [16].

Gold *et al.* [8] have found high concentrations of cortisol in serum and urine of anorectic patients. It has been confirmed by an increased incidence of cortisol secretory episodes during the day, as well as by high concentration of corticoliberin (CRH) in the cerebrospinal fluid [8, 21]. High concentrations of cortisol may suppress hypothalamic release of GnRH in malnutrition.

Rubin and Kaye [16] have observed ACTH concentration in serum to be usually normal, being, however, decreased in the cerebro-spinal fluid of anorectic patients, while CRH concentration – similarly as that of vasopressin (ADH) - is elevated. These authors claim also that suppression test, performed with small doses of dexametasone, may bring about normal outcome in 50-90% of AN cases and that ACTH response to CRH administration is delayed. On the other hand, they have observed elevated cortisol concentrations after ACTH administration, what suggests increased secretory ability of the adrenal cortex [16]. Since dexametazone acts primarily on the pituitary, then the lack of suppression of ACTH and cortisol suggests increased suprapituitary (hypothalamic) stimulation of corticotropic cells by CRH and ADH [16].

Anorectic patients do not manifest cushingoidal symptoms, despite their excessive volumes of glycocorticosteroids. It is associated with the lack of sufficient volume of substrates for lipogenesis, deposition of fats and gluconeogenesis. The reduced sensitivity of receptors to glycocorticosteroids is also considered [8].

The mean concentrations of cortisol at 8<sup>00</sup>, 17<sup>00</sup> and 24<sup>00</sup> were much above the reference range in our studied group of girls with AN. Cortisol concentrations were the highest in the morning and the lowest during the night, thus no disturbances were found in the circadian rhythm of cortisolaemia.

Already short fasting period in healthy subjects contributes to slight inhibition of the hypothalamus-pituitary-thyroid axis. Samuels and Kramer have found that 56-hour fasting contributes to suppression of mean TSH levels and lower amplitude of TSH pulses, however, with unchanged pulse frequency. Nocturnal amplitudes of the pulses decrease by 60%, with abolition of the normal TSH wave [17].

In patients with AN, the most important changes occur in the conversion of thyroxine  $(T_4)$  into 3,5,3'triiodothyronine  $(T_3)$ , in result of the reduced activity of 5'-deiodinase with simultaneously increased concentration of 3,5,3'triiodothyronine  $(rT_3)$  [4]. Also Bentdal *et al.* [4] have observed decreased concentrations of  $T_3$  and  $T_4$  in serum, indicating a significantly negative correlation between  $T_3$  concentration and body weight loss. In turn, no such decreased concentration of  $T_3$  in the course of AN was observed by Palla and Litt [15].  $FT_4$  concentrations, obserevd in AN, are either normal [15] or – more often – decreased [4, 16, 6]. TSH concentrations may be normal or decreased and the response of TSH to thyroliberin (TRH) is, sometimes, delayed [22]. In the present study, the mean concentration of  $FT_4$  in the examined girls with AN was below the range of reference values with normal mean concentrations of  $FT_3$  and TSH. Only in two (2) patients (2.29%),  $FT_3$  concentration was below the normal range, while slightly elevated levels of TSH were observed in other two (2) patients (2.29%).

The statistically significant correlation, found in our study between BMI values and the concentrations of LH (p=0.005), LH30 (p<0.001), LH60 (p=0.001) and E<sub>2</sub> (p=0.007), indicates the key importance of body weight normalisation in stabilising the hypothalamo-pituitary-gonadal axis and restoring the normal menstrual cycle in patients with AN.

The negative correlations between BMI values and Cort8 (p=0.009), Cort17 (p=0.004) and Cort24 (p<0.001) provide firm foundations to claim that body weight increase is the key condition for hypercortisolaemia reduction in girls with AN. Our observations comply with the opinions of other authors, who claim that hormonal disorders in AN are the result of organism cachexy and the effect of the organism adaptation to caloric deficit, [28, 26].

The obtained results allow stating that body weigh loss is not significantly reflected by abnormal results of routinely performed laboratory tests, however, it is unquestionably the main cause of hormonal disorders in girls with AN – such as hypogonadotropic hypogonadism and hypercortisolemia.

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