

# Effect of intravenous dopamine infusion on pituitary and thyroid function and on nephroprotection

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## KEY WORDS

adrenocorticotrophic hormone, dopamine, heart failure, nephroprotection, thyroid-stimulating hormone

## ABSTRACT

**INTRODUCTION** Catecholamines, including dopamine, are used in cardiac intensive care.

**OBJECTIVES** The aim of the study was to assess the effect of intravenous dopamine infusion on the function of pituitary gland in patients with acute cardiac failure. We analyzed changes in the serum levels of thyroid-stimulating hormone (TSH) and adrenocorticotrophic hormone (ACTH), as well as potential nephroprotection.

**PATIENTS AND METHODS** The study involved 29 patients with chronic decompensated heart failure (New York Heart Association class III/IV; mean age  $77.4 \pm 13.3$  years). Dopamine was administered intravenously in doses varying from 1 to 5  $\mu\text{g/kg/min}$ . Measurements of TSH, free triiodothyronine ( $\text{FT}_3$ ), free thyroxine ( $\text{FT}_4$ ), and ACTH were taken directly before dopamine infusion, after 12 hours of continuous infusion, and 12 hours after the 72-hour infusion was completed.

**RESULTS** Serum  $\text{FT}_3$  levels were significantly higher before dopamine infusion than at 12 hours post infusion ( $5.12 \pm 1.16$  vs.  $4.27 \pm 0.89$  pmol/l,  $P < 0.005$ ). Serum  $\text{FT}_4$  levels before the infusion were significantly higher than after 12 hours of continuous infusion as well as after 12 hours post infusion ( $18.79 \pm 5.33$  vs.  $17.06 \pm 4.61$  pmol/l,  $P < 0.05$ ;  $18.79 \pm 5.33$  vs.  $16.26 \pm 4.53$  pmol/l,  $P < 0.05$ , respectively). There were no statistically significant differences between serum TSH and ACTH levels or in creatinine clearance before, during, and 12 hours post infusion.

**CONCLUSIONS** Intravenous infusion of dopamine may downregulate endocrine thyroid function; however, it has no significant effect on the pituitary gland-derived TSH and ACTH. There was no significant nephroprotective effect of low-dose dopamine infusion in patients with chronic decompensated chronic heart failure.

**INTRODUCTION** Catecholamines are commonly used in intensive care. They help maintain vital body functions by increasing peripheral blood flow. Synthetic epinephrine, norepinephrine, dopamine, and dobutamine are the most common catecholamines in the clinical setting. The population of patients diagnosed with advanced heart failure and episodes of decompensation is on the rise, as is the use of catecholamines in cardiology practice.

Dopaminergic neurons located in the central nervous system (CNS) are the main source of dopamine. Depending on the type of receptors, dopamine can exert either stimulating or inhibiting effect. By binding to receptors from the  $\text{D}_1$ -like family, (receptors

$\text{D}_1$  and  $\text{D}_5$ ), dopamine activates subunit Gs of protein G and indirectly stimulates adenylate cyclase.<sup>1</sup> On the other hand, activation of receptors belonging to the  $\text{D}_2$ -like family ( $\text{D}_2$ ,  $\text{D}_3$ , and  $\text{D}_4$ ) leads to the inhibition of protein G and adenylate cyclase and triggers sodium channels.<sup>1,2</sup> Due to the large numbers of dopamine receptors distributed throughout the CNS, endogenous dopamine has the ability to regulate a broad range of physiological functions such as locomotion and cognition. Dopamine also plays an important role in neurohormonal regulation, modulates renal function, and the function of gastrointestinal and cardiovascular systems.<sup>3</sup> Generally, dopamine does not cross the blood-brain barrier;

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**TABLE 1** Characteristics of the patient population (n = 29)

Parameter	Percentage, %
male sex	41
ischemic heart disease	65
valvular heart disease	76
diabetes	45
hypertension	79
atrial fibrillation	76
chronic obstructive pulmonary disease	21
treatment	
β-blockers	76
angiotensin-converting enzyme inhibitors or AT1-receptor antagonists	72
diuretics	76
aldosterone receptor antagonists	72
statins	83
anticoagulants	69
antiplatelet drugs	62

nevertheless, some regions within the hypothalamus and pituitary gland lack such a barrier, allowing for the exchange of active components to occur and exogenous dopamine to enter the CNS and modulate its function.<sup>4,5</sup>

Acting in the periphery, dopamine can either inhibit (via D2-like receptor family) or stimulate (via D1-like receptor family) the release of catecholamines. Interestingly, low doses of dopamine downregulate, while high doses stimulate, the release of norepinephrine.<sup>6,7</sup>

Dopamine receptors are also present in the kidneys. It was shown that intravenous infusion of dopamine increases glomerular filtration, diuresis, and natriuresis.<sup>8</sup> The mechanism underlying the increase in diuresis and natriuresis has not yet been elucidated and several theories have been put forward, including the increase in cardiac output due to the stimulation of  $\beta_1$  receptors, D1- and  $\alpha$  receptor-mediated increase in renal flow and perfusion, and finally, downregulation of the release of aldosterone and inhibition of sodium-potassium adenosinetriphosphatase in renal tubular epithelium.<sup>9</sup>

In current medical practice, dopamine treatment is commonly used as a mean to achieve nephroprotection. Nevertheless, it has to be emphasized that very few studies unequivocally supported the use of dopamine as a beneficial treatment in acute renal failure.<sup>10</sup> Moreover, treatment with dopamine may lead to severe adverse effects such as cardiac ischemia, arrhythmia, hypoventilation, increase in pulmonary shunt, and disturbance of gastrointestinal motility and blood flow.<sup>11</sup> Indeed, the majority of reports involving patients with sepsis or early renal dysfunction failed to confirm the postulated nephroprotective action of dopamine.<sup>12-14</sup>

Dysfunction of hypothalamus–pituitary–thyroid axis, commonly occurring in critically ill patients, may be further amplified by drugs affecting

neurohormonal balance, as it is in the case of dopamine.<sup>4</sup> Intravenous administration of dopamine in patients with multiorgan injuries resulted in decreased thyroid-stimulating hormone (TSH), triiodothyronine ( $T_3$ ), and thyroxine ( $T_4$ ) release, while discontinuation of the infusion led to a 3-fold increase in TSH levels as well as increase of serum  $T_3$ . These fluctuations were usually disappearing after 24 hours post infusion.<sup>15</sup> Inhibition of several other hormones was reported following intravenous dopamine infusion, including growth hormone (GH), prolactin (PRL), and luteinizing hormone.<sup>15-18</sup> Dopamine infusions administered in low birth weight infants caused transient reduction of  $T_4$ , TSH, and PRL levels with a subsequent increase after discontinuing the treatment, while GH levels remained unchanged.<sup>19</sup>

Our current study assesses the effect of intravenous dopamine infusion on pituitary function including changes in serum TSH and ACTH levels, thyroid function, and potential nephroprotective effect in patients with acute heart failure

**PATIENTS AND METHODS** The study involved 29 patients of the cardiology department admitted in 2009–2010 with acute heart failure diagnosed as chronic decompensated heart failure (New York Heart Association class III/IV; mean age  $77.4 \pm 13.3$  years). There were 17 women (mean age  $81.9 \pm 8.6$  years) and 12 men (mean age  $71.1 \pm 14.4$  years). Diagnosis of chronic heart failure was established at least 6 months prior to the admission. Characteristics of the study population are presented in **TABLE 1**.

Patients with stage 4/5 kidney damage, liver failure, thyroid disease, neoplastic disease, acute inflammatory disorders, individuals undergoing immunosuppressive or glucocorticoid therapy, as well as those with glycemia above 400 mg/dl ( $22.2 \text{ mmol/l}$ ) were excluded from the study.

Intravenous dopamine infusion was administered in doses varying from 1 to 5  $\mu\text{g/kg/min}$ . Blood pressure was carefully monitored during the procedure, as recommended in the guidelines of the European Society of Cardiology and the Polish Cardiac Society on the management of chronic decompensated heart failure. Dopamine was administered only in patients who failed to respond to previous optimized pharmacological treatment and continued to display the symptoms of decompensated chronic heart failure. Patients who required increase in dopamine dose above the initially established limit were excluded from the study. Each infusion lasted 72 hours. The measurement of TSH, free triiodothyronine ( $FT_3$ ), free thyroxine ( $FT_4$ ), and ACTH levels was taken directly before the infusion (timepoint 0), at 12 hours (timepoint 1), and 12 hours after the infusion was completed (timepoint 2).

Laboratory measurements were performed using the Wizard2470 automatic gamma counter. The serum levels of  $FT_3$  and  $FT_4$  were measured using radioimmunoassay kits RIAZENco $FT_3$  and RIAZENco $FT_4$ , respectively (ZenTech, Angleur, Belgium). The normal values were 3.4–7.2 pmol/l for  $FT_3$  and 10.3–25.7 pmol/l for  $FT_4$ . The serum

**TABLE 2** Changes in hormone levels during the infusion of dopamine; timepoint 0 – directly before infusion; timepoint 1 – 12 hours into the infusion procedure; timepoint 2 – 12 hours after the infusion

Hormone	Timepoint	Mean value	Lowest value	Highest value	SD
FT <sub>3</sub> , pmol/l	0	5.12	3.00	8.07	1.16
	1	4.79	2.90	7.49	1.10
	2	4.27	2.38	5.86	0.89
FT <sub>4</sub> , pmol/l	0	18.79	4.82	35.01	5.33
	1	17.06	4.48	32.13	4.61
	2	16.26	3.33	25.92	4.53
TSH, $\mu$ IU/ml	0	2.01	0.07	12.17	2.16
	1	1.52	0.08	6.53	1.26
	2	1.91	0.09	8.53	2.00
ACTH, ng/l	0	36.88	4.47	310.11	57.29
	1	24.22	4.55	86.54	22.60
	2	23.15	4.69	47.18	14.38

Abbreviations: ACTH – adrenocorticotrophic hormone, FT<sub>3</sub> – free triiodothyronine, FT<sub>4</sub> – free thyroxine, SD – standard deviation, TSH – thyroid-stimulating hormone

levels of TSH were measured using the radioimmunoassay kit (DIAsource ImmunoAssays S.A., Belgium). The normal values for TSH were 0.2–3.2  $\mu$ IU/ml. The serum levels of human adrenocorticotrophic hormone (ACTH) were measured in EDTA plasma using the radioimmunoassay kit (DIAsource ImmunoAssays S.A.). The normal values were 9.6–49.7 pg/ml.

The glomerular filtration rate (GFR) was calculated according to the Modification of Diet in Renal Disease formula (MDRD):

$$\text{GFR} = 170 \times (\text{SCr})^{-0.999} \times (\text{age})^{-0.176} \times (\text{BUN})^{-0.17} \times (\text{alb})^{-0.318} \times C$$

where: C value – 1 if male, 0.762 if female, 1.18 if African American; BUN – blood urea nitrogen; alb – albumin level, SCr – serum creatinine level.

For the purpose of this study, simplified MDRD formula was used:

$$\text{eGFR} = 186 \times (\text{SCr})^{-1.154} \times (\text{age})^{-0.203} \times (0.742 \text{ if female})$$

**TABLE 3** Hemodynamic parameters and renal function after dopamine infusion

Parameter	Values	
	mean	SD
PR at timepoint 0, min	78.9	24.29
PR at timepoint 1, min	82.97	19.23
SBP at timepoint 0, mmHg	99.24	19.12
SBP at timepoint 1, mmHg	111.86	14.16
DBP at timepoint 0, mmHg	56.83	11.03
DBP at timepoint 1, mmHg	62.1	12.74
GFR at timepoint 0, ml/min	40.86	18.5
GFR at timepoint 1, ml/min	46.83	21.86
GFR at timepoint 2, ml/min	50.24	24.69

For timepoints see [TABLE 2](#)

Abbreviations: DBP – diastolic blood pressure, SBP – systolic blood pressure, GFR – glomerular filtration rate, PR – pulse rate, others – see [TABLE 2](#)

where: eGFR – estimated GFR (ml/min/1.73 m<sup>2</sup>).

Our study was performed in agreement with the guidelines of the Helsinki Declaration and was approved by the local ethics committee.

**Statistical analysis** The results obtained in our study were subsequently analyzed statistically. First, repeated measures analysis of variance (ANOVA) test with 3 measurements for each variable was performed. Next, the post hoc Turkey test for the group of variables with statistical differences was performed in order to establish the statistical significance of the differences. Additionally, changes in pulse rate and blood pressure measured prior to and after 12 hours of dopamine infusion and GFR values measured at timepoints 0, 1, and 2 were analyzed using repeated measures ANOVA test. Finally, the Pearson's correlation coefficient (*r*) was calculated, with the level of statistical significance of *P* < 0.05.

**RESULTS** The ejection fraction (EF) was assessed by echocardiography and was 40.0%  $\pm$  15.4%. In 65% of the patients, ischemia was the underlying cause of heart failure; valvular heart disease and other cardiovascular diseases were also observed (independently or coexistent with ischemia; [TABLE 1](#)).

The results are summarized in [TABLES 2–5](#) and [FIGURES 1–4](#).

Serum FT<sub>3</sub> levels 12 hours after dopamine infusion were significantly lower than before the infusion (*P* < 0.005). Serum FT<sub>4</sub> levels at 12 hours of continuous infusion as well as 12 hours after the infusion were significantly lower than before the infusion (*P* < 0.05 and *P* < 0.005, respectively).

There was no statistically significant difference between consecutive measurements of serum TSH and ACTH during and after the infusion. At timepoint 1 (12 hours into the infusion procedure), a borderline correlation between the levels

**TABLE 4** Pearson's correlation coefficient and statistical significance

	ACTH at timepoint 0	ACTH at timepoint 1	ACTH at timepoint 2
TSH at timepoint 0	$r = 0.04$ $P = 0.89$	$r = 0.36$ $P = 0.25$	$r = -0.17$ $P = 0.6$
TSH at timepoint 1	$r = -0.0015$ $P = 0.99$	$r = 0.56$ $P = 0.056$	$r = 0.15$ $P = 0.65$
TSH at timepoint 2	$r = 0.45$ $P = 0.14$	$r = 0.06$ $P = 0.85$	$r = -0.003$ $P = 0.99$

For time points see [TABLE 2](#); at  $P < 0.05$ , there is no correlation between TSH and ACTH

Abbreviations: see [TABLE 1](#)

of TSH and ACTH was demonstrated ( $r = 0.563$ ,  $P = 0.56$ ).

While the GFR calculated using the MDRD formula increased from 40.86 ml/min at timepoint 0 to 50.24 ml/min at timepoint 2, the observed increase was not statistically significant.

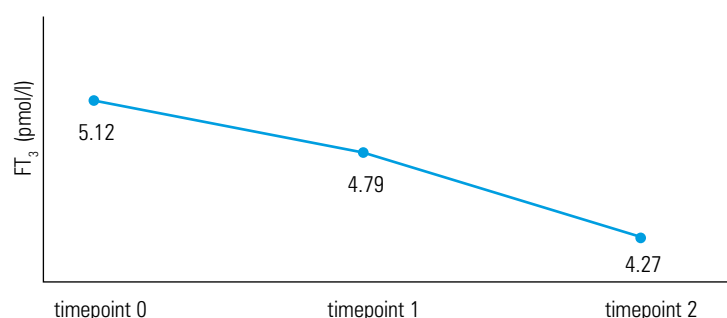
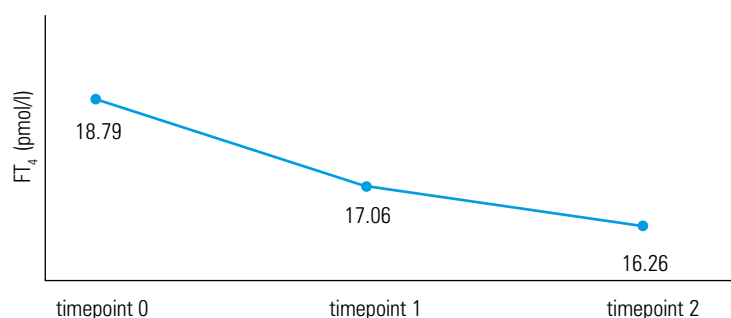
When pulse rate and systolic and diastolic blood pressure were analyzed at various timepoints, no statistically significant differences were noted.

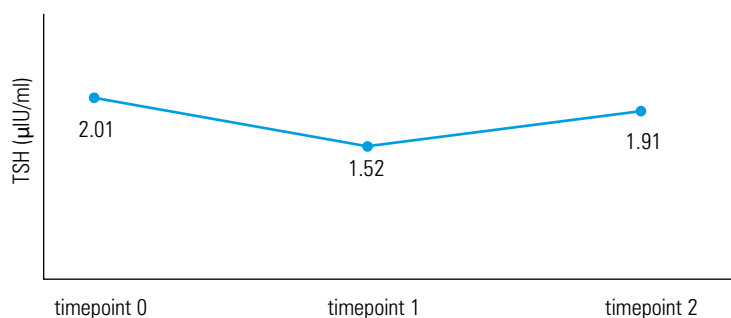
**DISCUSSION** Episodes of decompensation affecting the study population were caused by various pathophysiological mechanisms, making it particularly difficult to identify and characterize a single factor responsible for their occurrence. Additionally, such multifactorial etiology complicates the process of assessing a possible effect of dopamine on the function of hypothalamus–pituitary–thyroid axis in critically ill patients. According to our findings, the effect of dopamine infusion on neurohormonal function

of the anterior pituitary seems to depend on the administered dose. We did not observe a significant effect of small doses of dopamine on serum TSH and ACTH levels. Nevertheless, thyroid function was significantly suppressed as suggested by the decreased levels of serum FT<sub>3</sub> and FT<sub>4</sub> after dopamine infusion.

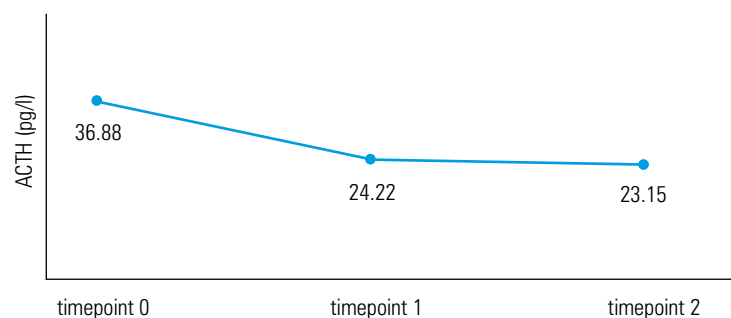
While analyzing our data from the “cause-effect” point of view, it is important to mention the previously published studies reporting a decrease of thyroid hormone levels in severely ill patients regardless of a treatment modality. Euthyroid sick syndrome diagnosed in these patients is characterized by low levels of FT<sub>3</sub> and FT<sub>4</sub> due to various extrathyroidal factors, such as systemic diseases, surgery or injuries, rather than to thyroid dysfunction.<sup>20,21</sup> On the other hand, the pituitary circadian rhythmic release of ACTH is stimulated by hypothalamus-derived corticotropin-releasing hormone. The highest ACTH levels are typically released in the morning and decrease continuously throughout the day to reach the lowest values in the evening. Several stress factors such as pain, hypoxia, hypoglycemia, surgery, etc., may deregulate this pattern of release.<sup>22</sup> It was shown that disturbance in circadian activation of the hypothalamus–pituitary axis in critically ill patients was related to the severity of the disease.<sup>23</sup> Importantly, such disturbances may be additionally enhanced iatrogenically, e.g., by administration of dopamine.<sup>4</sup>

The decision to administer dopamine is often supported by the need for so called nephroprotection, which allows to preserve diuretic and endocrine kidney function, but most importantly, to maintain efficient plasma filtration rate as measured by creatinine clearance. The mechanisms that promote a dopamine-dependent increase in diuresis and natriuresis have not yet been fully characterized. Moreover, the majority of reports published up to date have not confirmed the potentially beneficial effect of dopamine infusion on kidney function. The administration of 3 µg/kg/min for 2 hours in sepsis patients without concomitant oliguria resulted in increased diuresis but had no effect on creatinine clearance and did not lead to nephroprotection.<sup>12</sup> Combination therapy of 1 µg/kg/min of dopamine and furosemide proved to be beneficial compared with

**FIGURE 1** Mean values of serum free triiodothyronine (FT<sub>3</sub>); for time points see [TABLE 2](#)**FIGURE 2** Mean values of serum free thyroxine (FT<sub>4</sub>); for timepoints see [TABLE 2](#)



**FIGURE 3** Mean values of serum thyroid-stimulating hormone (TSH); for timepoints see TABLE 2



**FIGURE 4** Mean values of serum adrenocorticotrophic hormone (ACTH); for timepoints see TABLE 2

furosemide alone in patients with acute renal failure in the course of malaria, but only when serum creatinine levels were below 4.5 mg%.<sup>10</sup> A significant increase in the GFR was only observed when the dopamine dose reached 7 µg/kg/min. Renal function represented by creatinine clearance was proportionally related to the mean arterial blood pressure.<sup>10</sup>

Bellomo et al.<sup>13</sup> reported no statistically significant differences in the creatinine level, hospitalization time, as well as in the frequency of subsequent kidney replacement therapies and deaths between dopamine- and placebo-treated patients with early renal dysfunction. Therefore, the authors concluded that infusion of small doses of dopamine does not improve prognosis in these patients.

In our study, we did not observe significant changes in the GFR nor systemic blood pressure during intravenous dopamine infusion. We can therefore agree with other investigators that dopamine is not nephroprotective.<sup>14</sup> This issue is currently extensively investigated, and was recently discussed during the 2011 Conference of the European Society of Cardiology in Paris, during the session on intensive cardiologic care in patients with advanced heart failure. While Professor Kenneth Dickstein (University of Bergen, Norway) suggested that the practice of administering so called diuretic doses of dopamine should possibly be discontinued, Muriel Jessup (University of Pennsylvania, United States) presented a case report of a patient prior to the implantation of ventricular assist device whose intensive

care therapy included the administration of diuretic doses of dopamine. Moreover, such treatment has also been long employed in the United States in patients with end-stage heart failure. Certainly, this debate is far from over, and our report provides further data that may prove valuable in the ongoing discussion.

Due to the lack of support for dopamine as an agent able to provide isolated nephroprotection independently without concurrent circulatory improvement, its administration should be limited to patients with a specific hemodynamic profile.<sup>24,25</sup> Arbitrary use of diuretic doses of dopamine should be discouraged, while its administration as a vasopressor should require careful consideration and individual assessment of each particular case.<sup>26</sup>

The main limitation of our study is a small sample size, which is a common drawback of the studies conducted in intensive care unit patients. It stems from a dilemma of whether to select large groups of patients that are quite heterogeneous in terms of diagnoses and treatment modalities, or much smaller, but relatively homogenous. Our study would also further benefit from analyzing the serum levels of B-type natriuretic peptide (BNP)/N-terminal pro BNP.

Intravenous dopamine infusion has a potential to decrease thyroid function; however, it does not inhibit pituitary release of TSH and ACTH. The current study fails to support the concept that low dose dopamine infusion can improve kidney function in patients with chronic decompensated chronic heart failure.

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# Wpływ dożylniej infuzji dopaminy na czynność przysadki mózgowej, tarczycy oraz na nefroprotekcję

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## SŁOWA KLUCZOWE

dopamina, hormon  
adrenokortykotropowy,  
hormon tyreotropowy,  
nefroprotekcja,  
niewydolność serca

## STRESZCZENIE

**WPROWADZENIE** Aminy katecholowe, w tym dopamina, znajdują zastosowanie w intensywnej terapii kardiologicznej.

**CELE** Celem pracy była ocena wpływu dożylniej infuzji dopaminy na czynność przysadki u chorych z ostrą niewydolnością serca. Analizowano zmiany stężenia hormonu tyreotropowego (*thyroid-stimulating hormone* – TSH) i adrenokortykotropowego (*adrenocorticotrophic hormone* – ACTH) w surowicy oraz czynność tarczycy, a także potencjalną nefroprotekcję.

**PACJENCI I METODY** Do badania włączono 29 chorych ze zdekompensowaną przewlekłą niewydolnością serca (klasa czynnościowa New York Heart Association III/IV; średni wiek  $77,4 \pm 13,3$  roku). W badaniu stosowano dożylną infuzję dopaminy w dawkach od 1 do  $5 \mu\text{g/kg/min}$ . Oznaczano stężenia TSH, wolnej trijodotyroniny ( $\text{FT}_3$ ), wolnej tyroksyny ( $\text{FT}_4$ ) i ACTH bezpośrednio przed rozpoczęciem infuzji dopaminy, po 12 godzinach jej trwania oraz 12 godzin po zaprzestaniu 72-godzinnej terapii.

**WYNIKI** Stężenie  $\text{FT}_3$  w surowicy przed infuzją dopaminy było istotnie wyższe niż 12 godzin po zakończeniu leczenia ( $5,12 \pm 1,16$  vs  $4,27 \pm 0,89$  pmol/l;  $p < 0,005$ ). Stężenie  $\text{FT}_4$  w surowicy przed infuzją dopaminy było znamienne większe niż po 12 godzinach infuzji oraz 12 godzin po zakończeniu infuzji dopaminy ( $18,79 \pm 5,33$  vs  $17,06 \pm 4,61$  i  $16,26 \pm 4,53$  pmol/l; odpowiednio,  $p < 0,05$  i  $p < 0,005$ ). Nie wykazano istotnych statystycznie różnic między stężeniem TSH i ACTH w surowicy, a także między klirensiem kreatyniny przed rozpoczęciem infuzji dopaminy, w jej trakcie oraz 12 godzin po zakończeniu.

**WNIOSKI** Dożylna infuzja dopaminy jest jednym z potencjalnych czynników wpływających hamująco na hormonalną czynność tarczycy, jednak nie wpływa istotnie na czynność przysadki w zakresie uwalniania TSH oraz ACTH. Nie udokumentowano, aby stosowanie infuzji dożylniej dopaminy w niskich dawkach istotnie poprawiało czynność nerek u chorych ze zdekompensowaną przewlekłą niewydolnością serca.

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