ISSN 1607-419X
УДК 616.12-008.331

Circadian blood pressure profile in patients with chronic cardiorenal syndrome

V. A. Serov[1], A. M. Shutov[1], D. V. Serova[1], S. V. Shevchenko[2], E. U. Shmelkova[2]

[1] Ulyanovsk State University, Ulyanovsk, Russia
[2] Ulyanovsk Central City Hospital, Ulyanovsk, Russia

Corresponding author:
Valeriy A. Serov,
MD, PhD, Professor, Ulyanovsk State University, L. Tolstoy street, Ulyanovsk, 432027 Russia.
Phone: + 7(8422)55–27–08.
E-mail: Valery_serov@mail.ru

Received 5 October 2014;
accepted 27 November 2014.

Abstract

Objective. To determine the features of circadian blood pressure (BP) profile depending on renal function in patients with chronic heart failure (CHF). **Design and methods.** We examined 211 patients with CHF (126 men and 85 women), including 125 (59,2%) patients with cardiorenal syndrome and 86 (40,8%) patients without chronic kidney disease (CKD). Mean age was 58,1 ± 10,8 years. CKD was diagnosed according to the Scientific Society of Russian Nephrologists guidelines 2012. **Results.** An increase in time and area indices of arterial hypotension in patients with CHF, associated with CKD 3B stage was detected. Also, the nocturnal BP fall reduction was associated with increasing severity of CKD. **Conclusions.** 1. The increase in nocturnal BP fall is associated with increasing in daytime area and time indices of arterial hypotension in patients with cardiorenal syndrome. 2. Twenty-four-hour ambulatory BP monitoring allows to obtain additional information on hemodynamic parameters in patients with CHF and should be recommended for this group of patients.

Key words: chronic heart failure, chronic kidney disease, twenty-four-hour ambulatory blood pressure monitoring

For citation: Serov VA, Shutov AM, Serova DV, Shevchenko SV, Shmelkova EU. Features of the circadian blood pressure profile in patients with chronic cardiorenal syndrome. Arterial'naya Gipertenziya = Arterial Hypertension. 2014;20(6):538–545.

Особенности суточного профиля артериального давления у больных хроническим кардиоренальным синдромом

В. А. Серов[1], А. М. Шутов[1], Д. В. Серова[1], С. В. Шевченко[2], Е. Ю. Шмелькова[2]

[1] Федеральное государственное образовательное учреждение высшего профессионального образования «Ульяновский государственный университет», Ульяновск, Россия
[2] Государственное учреждение здравоохранения «Центральная городская клиническая больница г. Ульяновска», Ульяновск, Россия

Контактная информация:
Серов Валерий Анатольевич, Ульяновский государственный университет, ул. Льва Толстого, д.42, Ульяновск, Россия, 432027.
Тел.: + 7(8422)55–27–08.
E-mail: Valery_serov@mail.ru

Статья поступила в редакцию 05.10.14 и принята к печати 27.11.14.

Резюме

Цель исследования — изучение особенностей суточного профиля артериального давления (АД) у больных хронической сердечной недостаточностью (ХСН) в зависимости от функционального состояния почек. **Материалы и методы.** Обследовано 211 больных ХСН (126 мужчин и 85 женщин), из них 125 (59,2 %) пациентов с кардиоренальным синдромом и 86 (40,8 %) больных без хронической болезни почек (ХБП). Средний возраст составил 58,1 ± 10,8 года. ХБП диагностирована согласно Рекомендациям Национального общества нефрологов России 2012 года. **Результаты.** Выявлено увеличение индексов времени и площади артериальной гипотонии у больных ХСН, ассоциированной с ХБП 3Б стадии и уменьшение степени ночного снижения как систолического, так и диастолического АД по мере нарастания тяжести ХБП. **Выводы.** 1. Для больных хроническим кардиоренальным синдромом характерно уменьшение степени ночного снижения АД при увеличении индексов времени и площади артериальной гипотонии в дневные часы. 2. Суточное мониторирование АД позволяет получить дополнительную информацию о состоянии гемодинамики у больных ХСН и может быть рекомендовано для включения в обследование данной категории больных.

Ключевые слова: хроническая сердечная недостаточность, хроническая болезнь почек, суточное мониторирование артериального давления

Для цитирования: Серов В. А., Шутов А. М., Серова Д. В., Шевченко С. В., Шмелькова Е. Ю. Особенности суточного профиля артериального давления у больных с хроническим кардиоренальным синдромом. Артериальная гипертензия. 2014:20(6):538–545.

Introduction

Due to common mechanisms of development and progression of cardiovascular and renal diseases, cardiorenal interrelations have become a relevant issue of research [1]. Arterial hypertension (HTN) is one of these mechanisms. The Framingham study showed that there is more than 1.-5 fold increase in the risk of congestive heart failure (CHF) with an increase in systolic blood pressure (SBP) for 20 mm Hg and pulse pressure for 16 mmHg [2]. Systolic HTN and impaired daily blood pressure (BP) profile, namely lack of BP nocturnal decline, are common in patients with renal dysfunction. Chronic kidney disease

Table 1

CHARACTERISTICS OF THE PATIENTS WITH CONGESTIVE HEART FAILURE AND DIFFERENT DEGREE OF RENAL DYSFUNCTION

Parameter	eGFR ≥ 15 and < 30 ml/min/1,73 m² (n=14) — CKD stage 4	eGFR ≥ 30 and < 45 ml/min/1,73 m² (n = 34) — CKD stage 3b	eGFR ≥ 45 and < 60 ml/ min/1,73 m² (n = 73) — CKD stage 3a	eGFR ≥ 60 ml/min/1,73 m² (n = 86)
Age, years	61.9 ± 11.4*	63.8 ± 7.8***	59.9 ± 8.5***	53.4 ± 11.9
Female/male(n, %)	6/8 (42.9%)	16/18 (47.1%)	18/55 (24.7%)**	41/45 (47.7%)
Causes of CHF: - HTN, n (%) - CAD, n (%) - CAD + HTN, n (%) - other heart diseases, n (%)	2 (14.3%) 0 12 (85.7%) 0	3 (8.8%) 0 31 (91.2%)* 0	5 (6.8%)* 4 (5.5%) 63 (86.3%)* 1 (1.4%)	19 (22.1%) 5 (5.8%) 59 (68.6%) 3 (3.5%)
CHF NYHA functional class	2.6 ± 0.7*	2.6 ± 0.6**	2.5 ± 0.7**	2.1 ± 0.7
Left ventricular ejection fraction, %	54.8 ± 12.0	55.2 ± 8.0	55.6 ± 13,3	58.8 ± 10.3
Diabetes mellitus, n (%)	6 (42.9%)	10 (29.4%)	19 (26.0%)	22 (25.6%)
Anemia	6 (42.9%)***#^	4 (11.8%)	10 (13.7%)	4 (4.7%)

Note: CHF — congestive heart failure; eGFR — estimated glomerular filtration rate; CKD — chronic kidney disease; HTN — arterial hypertension; CAD — coronary artery disease; * — p < 0.05; ** — p < 0.01; *** — p < 0.001 as compared tp the group of patients with CHF and renal dysfunction eGFR ≥ 60 ml/min/1,73 m2; # — p < 0.05 compared to patients with CHF and CKD stage 3a; ^ — p < 0.05 compared to patients with CHF and CKD stage 3b.

(CKD) occurs in 9.2–71.2% CHF patients [3]. It is considered a type 2 chronic cardiorenal syndrome and is characterized by an increase in overall and cardiovascular mortality. However, excessive BP reduction also leads to an increased mortality in CHF patients [4].

Ambulatory blood pressure monitoring (ABPM) is widely used to clarify the diagnosis and selection of the optimal treatment strategy in HTN patients. However, circadian BP profile in patients with CHF and CKD currently is poorly understood.

The aim of our study was the assessment of the circadian BP rhythm in patients with chronic cardiorenal syndrome.

Design and methods

The study involved 211 patients with CHF I–IV NYHA (85 women and 126 men). The average age of patients was 58.1 ± 10.8 years. Diagnosis and treatment of CHF was conducted in accordance with the national guidelines for diagnosis and treatment of CHF [5]. The primary cause of CHF was a combination of coronary artery diseases (CAD) and HTN — 80.1% (n = 169). The mean left ventricular ejection fraction was 56.7 ± 11.3%.

24-hour ABPM was performed by the oscillometric method with

Table 2

OFFICE BLOOD PRESSURE AND AMBULATORY BLOOD PRESSURE MONITORING DATA IN PATIENTS WITH CONGESTIVE HEART FAILURE AND DIFFERENT DEGREE OF RENAL DYSFUNCTION

Parameter	eGFR ≥ 15 and < 30 ml/min/1,73 m² (n=14) — CKD stage 4	eGFR ≥ 30 and < 45 ml/min/1,73 m² (n = 34) — CKD stage 3b	eGFR ≥ 45 and < 60 ml/min/1,73 m² (n = 73) — CKD stage 3a	eGFR ≥ 60 ml/min/1,73 m² (n = 86)
Office SBP, mm Hg	140.8 ± 30.1	147.9 ± 30.5	141.0 ± 27.0**	154.9 ± 31.7
Office DBP, mm Hg	83.8 ± 13.9	91.1 ± 15.6	88.1 ± 14.4	92.7 ± 16.4
Office PBP, mm Hg	56.9 ± 21.8	56.8 ± 20.6	52.6 ± 11.9**	62.2 ± 20.3
Mean 24-h SBP, mm Hg	149.0 ± 27.2**##^^^	123.4 ± 14.2**	132.6 ± 14.2	133.3 ± 18.0
Mean 24-h DBP, mm Hg	83.7 ± 12.6^^	73.1 ± 11.1**##	79.8 ± 10.7	80.0 ± 11.1
Mean 24-h PBP, mm Hg	65.6 ± 23.2**##^^	56.8 ± 20.6	56.8 ± 20.6	52.9 ± 12.7
24-h SBP time index, %	71.9 ± 39.9*##^^^	50.5 ± 10.7	42.1 ± 32.0	45.7 ± 33.7
24-h SBP area index, %	498.6 ± 347.5***###^^^	113.5 ± 137.5*	176.4 ± 220.4	199.3 ± 225.3
24-h DBP time index, %	45.5 ± 31.4	24.4 ± 25.2*	35.9 ± 29.1	37.2 ± 28.3
24-h DBP area index, %	157.0 ± 151.4^	65.4 ± 90.4	101.1 ± 121.5	101.3 ± 120.9
Daytime SBP, mm Hg	148.5 ± 27.1*##^^^	124.6 ± 14.4**#	133.1 ± 16.4	136.0 ± 18.6
Daytime DBP, mm Hg	84.2 ± 12.3#	75.0 ± 11.6**##	81.2 ± 10.5	81.7 ± 11.0
Daytime SBP, mm Hg	64.5 ± 22.9*#^^	49.4 ± 10.1	53.1 ± 13.3	53.8 ± 11.9
Daytime PBP time index, %	69.2 ± 39.5*##^^^	26.4 ± 27.0**	37.9 ± 34.2	44.0 ± 34.1
Daytime SBP area index, %	283.8 ± 199.9*#^^^	71.8 ± 102.4*	133.7 ± 189.4	147.6 ± 196.0
Daytime DBP time index, %	38.8 ± 29.4^	19.6 ± 24.1*	30.5 ± 30.3	33.0 ± 30.3
Daytime DBP area index, %	81.3 ± 83.8^	41.8 ± 66.4	69.7 ± 95.2	71.8 ± 105.8
Nocturnal SBP, mm Hg	148.9 ± 29.9***###^^^	120.5 ± 17.9	127.0 ± 19.0	127.0 ± 18.7
Nocturnal DBP, mm Hg	81.5 ± 15.1*^^	68.7 ± 12.4*#	75.3 ± 11.9	73.8 ± 11.5
Nocturnal PBP, mm Hg	65.7 ± 23.3**##^^	51.8 ± 13.8	51.8 ± 13.2	52.5 ± 23.3
Nocturnal SBP time index, %	77.4 ± 41.3*#^^	40.3 ± 38.8	54.0 ± 38.4	50.6 ± 38.4
Nocturnal SBP area index, %	377.2 ± 367.9**#^	150.8 ± 238.5	174.5 ± 251.9	157.7 ± 222.4
Nocturnal DBP time index, %	59.1 ± 39.5	37.0 ± 34.1	51.3 ± 36.3	47.5 ± 33.1
Nocturnal DBP area index, %	188.4 ± 242.4*^	74.8 ± 108.3	104.7 ± 127.5	92.7 ± 145.4

Note: BP — blood pressure; CHF — congestive heart failure; eGFR — estimated glomerular filtration rate; CKD — chronic kidney disease; SBP — systolic BP; DBP — diastolic BP; PBP — pulse BP; * — $p < 0.05$; ** — $p < 0.01$; *** — $p < 0.001$ compared to patients with CHF and eGFR ≥ 60 ml/min/1,73 m2; # — $p < 0.05$; ## — $p < 0.01$; ### — $p < 0.001$ compared to patients with CHF and CKD stage 3a; ^ — $p < 0.05$; ^^ — $p < 0.01$; ^^^ — $p < 0.001$ compared to patients with CHF and CKD stage 3b.

an interval between measurements 15/30 minutes (at daytime/nighttime, respectively). Circadian BP profile was evaluated by the degree of nocturnal reduction of systolic (SBP) and diastolic blood pressure (DBP), based on conventional criteria for two-phase BP rhythm. Hypotension was diagnosed when office BP was ≤ 100/60 mm Hg, and when ABPM daytime and nighttime BP was ≤ 100/60 and ≤ 85/47 mm Hg, respectively [6].

Glomerular filtration rate was calculated using the EPI formula, was diagnosed with CKD was diagnosed according to the recommendations of the Scientific Society of Nephrology of Russian Federation (2012) [7]. The mean eGFR was 56.3 ± 18.0 ml/min/1.73 $m^{2.}$ Patients with stage 5 CKD (n=3) were excluded from the analysis due to the small sample and impossibility to determine statistically significant differences in ABPM indicators. Patients with CHF and eGFR ≥ 60 ml/min/1.73 m^2 showed neither proteinuria, nor kidney changes by ultrasound examination. Depending on the renal function, all patients were divided into 4 groups: group 1 had a GFR ≥ 60 ml/min/1.73 m^2, group 2 included included patients with CHF and eGFR ≥ 45 and < 60 ml/min/1 73 m^2 (CKD stage 3a), the third group included patients with eGFR ≥ 30 and < 45 ml/min/1.73 m^2 (CKD stage 3b), the fourth group included CHF patients with eGFR ≥ 15 and < 30 mL/min/1.73 m^2 (CKD stage 4). Characteristics of CHF patients depending on the CKD stage are presented in Table 1.

For statistical analysis a software "Statistica v. 6.0" was used, and mean values, and standard deviation were calculated. The significance of differences was determined either by Student t-test or Mann-Whitney test for independent samples, χ^2 (Yates adjustment) depending on the variable distribution type. Indicators are represented as M ± SD. Differences were considered significant at $p < 0.05$.

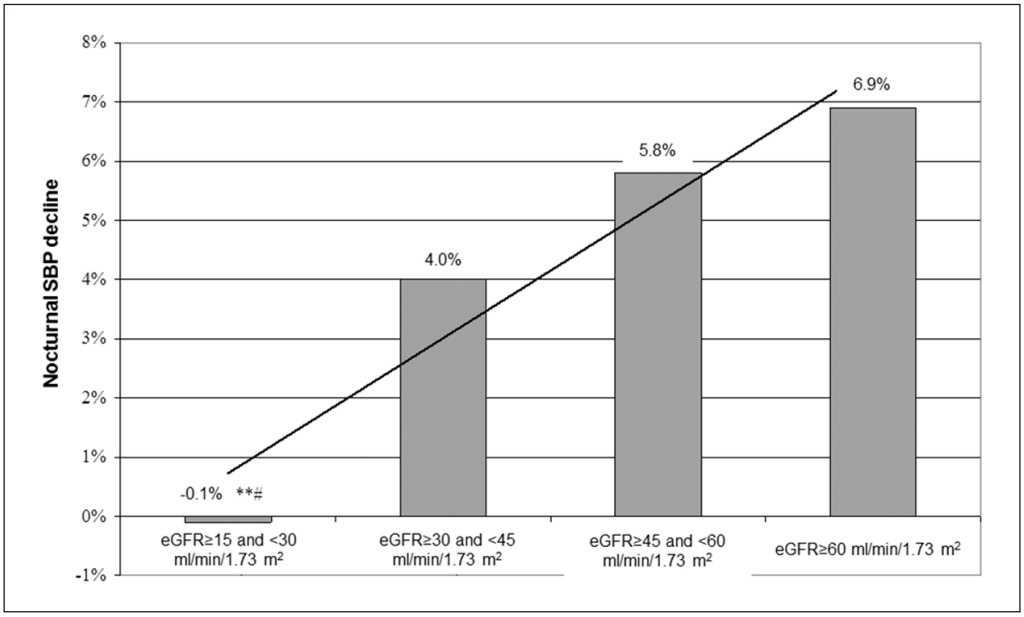
Results

eGFR was inversely related with age and CHF NYHA functional class in CHF patients. Men were significantly more prevalent among patients with CKD stage 3a, but there were no gender differences when patients with other CKD stages were compared. In CHF patients with CKD stage 4, anemia was rather common.

Office SBP, DBP, and PP between groups did not differ significantly, except for the reduction of office SBP and PP in patients with CHF associated with CKD stage 3a compared to patients with CHF with GFR ≥ 60 ml/min/1.73 m^2 ($p = 0.01$ and $p = 0.009$, respectively).

ABPM showed a decrease in most SBP and DBP values with the decline in eGFR in patients with CKD stage 3, the differences

Figure 1. The association between nocturnal systolic blood pressure decline and renal function in patients with chronic heart failure



Note: SBP — systolic blood pressure; eGFR — estimated glomerular filtration rate.

Table 3

TIME AND AREA INDEX OF HYPOTENSION IN PATIENTS WITH CONGESTIVE HEART FAILURE

Parameter	eGFR ≥ 15 and < 30 ml/min/1,73 m² (n=14) — CKD stage 4	eGFR ≥ 30 and < 45 ml/min/1,73 m² (n = 34) — CKD stage 3b	eGFR ≥ 45 and < 60 ml/min/1,73 m² (n = 73) — CKD stage 3a	eGFR ≥ 60 ml/min/1,73 m² (n = 86)
24-hour time index of systolic hypotension, %	8.8 ± 21.8	9.6 ± 16.2**##	3.7 ± 7.7	4.2 ± 8.3
24-hour area index of systolic hypotension, %	20.4 ± 46.8*#	19.9 ± 46.6*#	5.5 ± 12.8	6.5 ± 15.1
24-hour time index of diastolic hypotension, %	6.0 ± 12.2	21.2 ± 23.8**###	8.1 ± 11.0	9.7 ± 13.8
24-hour area index of diastolic hypotension, %	8.7 ± 19.5^	41.3 ± 62.4**###	10.7 ± 21.0	15.7 ± 25.6
Daytime time index of systolic hypotension, %	12.8 ± 31.6	12.5 ± 21.5*#	4.8 ± 10.0	5.5 ± 12.0
Daytime area index of systolic hypotension, %	19.4 ± 48.7*#	21.2 ± 46.0**##	5.4 ± 12.9	6.3 ± 14.9
Daytime time index of diastolic hypotension, %	9.0 ± 18.1	23.4 ± 27.1**###	9.7 ± 14.2	10.2 ± 15.6
Daytime area index of diastolic hypotension, %	8.7 ± 18.8	42.1 ± 69.8***###	10.0 ± 21.0	12.8 ± 22.6
Nocturnal time index of systolic hypotension, %	2.2 ± 5.6	1.5 ± 7.4	1.4 ± 6.4	0.7 ± 2.8
Nocturnal area index of systolic hypotension, %	1.0 ± 3.1	0.4 ± 2.1	0.4 ± 2.0	1.1 ± 6.9
Nocturnal time index of diastolic hypotension, %	0.8 ± 3.1	14.5 ± 25.0##	5.2 ± 11.5	6.7 ± 17.0
Nocturnal area index of diastolic hypotension, %	0.2 ± 0.8	20.4 ± 43.4##	4.2 ± 14.7	10.8 ± 31.8

Note: BP — blood pressure; CHF — congestive heart failure; eGFR — estimated glomerular filtration rate; CKD — chronic kidney disease; * — $p < 0.05$; ** — $p < 0.01$; *** — $p < 0.001$ compared to patients with CHF and eGFR ≥ 60 ml/min/1.73 m2; # — $p < 0.05$; ## — $p < 0.01$; ### — $p < 0.001$ compared to patients with CHF and CKD stage 3a; ^ — $p < 0.05$; ^^ — $p<0.01$; ^^^ — $p < 0.001$ compared to patients with CHF and CKD stage 3b.

were significant in patients with stage 3B CKD. At the same time BP increased in patients with CHF associated with CKD stage 4 (Table 2). Noteworthy, there was a significant increase in the time indices and nocturnal systolic hypotension index of area in patients with CHF associated with CKD stages 3b (Table 3). Opposite changes of hypotension indices of time and area were observed in patients with CHF associated with CKD stage 4.

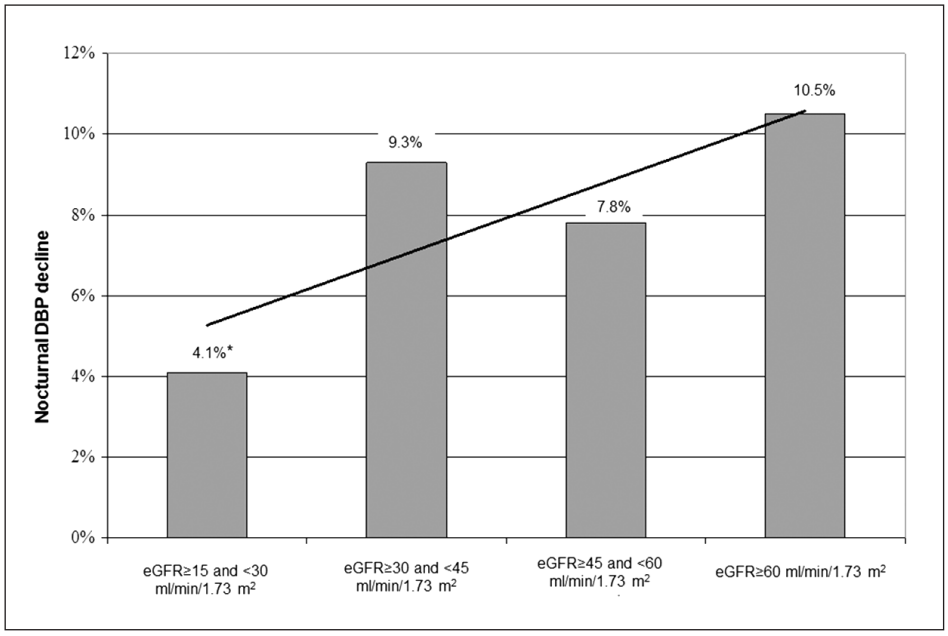
The degree of nocturnal SBP and DBP decline significantly decreased as the CKD severity increased (Fig. 1 and 2).

Discussion

Elevated BP is well-known to be associated with higher cardiovascular risk. In contrast to the general population, increased SBP is a favorable factor in CHF patients [8]. High SBP was suggested to characterize an earlier phase of CHF with a higher cardiac output. In addition, guidelines are more accurately followed in CHF by both physicians and patients, and many drugs have an antihypertensive effect. Based on a study of about 7500 patients, Lee D. S. et al. [9] found an U-shaped relation between mortality and both SBP and DBP in patients with CHF, with the lowest mortality in patients with SBP within 120–139 mm Hg. This is explained by the deleterious effects of both BP overload in HTN and tissue hypoperfusion in hypotension leading to target organ damage (blood vessels, heart, kidneys, and brain).

An impairment of circadian BP rhythm can affect target organs. While there is a 10–20% nocturnal BP decline in healthy people,

Figure 2. The association between nocturnal diastolic blood pressure decline and renal function in patients with chronic heart failure



Note: DBP — diastolic blood pressure; eGFR — estimated glomerular filtration rate.

patients with CKD, HTN, type 1 and 2 diabetes mellitus show a lack of nocturnal BP reduction (non-dipper), or even its increase (night-peaker), which contributes to the progression of target organ damage. For example, "non-dipper" type is associated with an increase in the relative thickness of the left ventricular wall, left ventricular mass index and serum levels of atrial and brain natriuretic peptides, even at a normal level BP [10]. Insufficient nocturnal SBP reduction is associated with a 2.21-fold increase in relative risk of CHF development in men [11]. In patients with preexisting CHF, abnormal circadian BP profile is an important predictor of re-hospitalization and mortality [12].

The main pathophysiological mechanisms of circadian BP profile impairment are an increased sympathetic activity, decreased vagal stimulation, impaired sodium excretion, physical inactivity, and smoking. Abnormal daily BP profile is commonly found in patients with diabetes mellitus, coronary artery disease, cerebrovascular disease, and CHF [13].

Renal function also plays a significant role. After unilateral nephrectomy, circadian BP changed to a "non-dipper" type without any significant BP elevation in kidney donors [14].

The results of our studies suggest that an abnormal circadian BP profile can be considered is one of the pathogenetic mechanisms of unfavorable prognosis in patients with CHF associated with CKD, along with increased activity of the renin-angiotensin-aldosterone system, anemia, atrial fibrillation and mitral regurgitation.

The results showed a significant association between BP and renal function in patients with chronic cardiorenal syndrome. A paradox was found: eGFR decline was accompanied by a reduction in both SBP and DBP in patients with CKD stage 3a, and an increase in BP in patients with CKD stage 4. It might be due to a more careful use of angiotensin converting enzyme inhibitors and angiotensin II receptor antagonists in patients with severe decrease in eGFR. However, despite the increase in the mean SBP and DBP in patients with CHF associated with CKD stage 4, time and area daytime indices of systolic hypotension remained elevated. We assume that it reflects a more profound BP dysregulation in progressive CKD, which requires careful drug choice and strict BP control, and office BP alone is insufficient.

Conclusions

Renal dysfunction in CHF patients is associated with insufficient nocturnal BP decline and an increase in the daytime hypotension indices of time and area.

ABPM provides additional data on hemodynamics in CHF patients and should be recommended as a part of clinical examination in CHF.

Conflict of interest

The authors declare no conflict of interest.

References

1. Smirnov AV, Dobronravov VA, Kayukov IG. Chronic kidney disease nowadays. Arterial'naya Gipertenziya = Arterial Hypertension. 2006;12(3):185–193. In Russian.

2. Haider AW, Larson MG, Franklin SS et al. Systolic blood pressure, diastolic blood pressure, and pulse pressure as predictors of risk for congestive heart failure in the Framingham Heart Study. Ann Intern Med. 2003;138(1): 10–16.

3. Serov VA, Shutov AM, Menzorov MV et al. Epidemiology of chronic kidney disease in patients with chronic heart failure. Nephrology. 2010;1:50–55. In Russian.

4. Grigorian-Shamagian L, Gonzalez-JuAnatey JR, Vazquez R, Cinca J, Bayes-Genis A, Pascual D et al. Association of blood pressure and its evolving changes with the survival of patients with heart failure. J Card Fail. 2008:14(7):561–568.

5. National guidelines of RSSC and SSHF for diagnosis and treatment of CHF (third revision). Heart Failure. 2010;57(1):3–62. In Russian.

6. Ratova LG, Dmitriev VV, Tolpygina SN, Chazova IE. 24-hour ambulatory blood pressure monitoring in clinical practice. Arterial'naya Gipertenziya = Arterial Hypertension. 2001;7(2):3–14. In Russian.

7. Chronic kidney disease: basics, definition, diagnostics, screening, approaches to prevention and treatment. National guidelines. Clinical Nephrology. 2012;4:4–26. In Russian.

8. Raphael CE, Whinnett ZI, Davies JE, Fontana M, Ferenczi EA, Manisty CH et al. Quantifying the paradoxical effect of higher systolic blood pressure on mortality in chronic heart failure. Heart. 2009;95(1):56–62.

9. Lee DS, Ghosh N, Floras JS, Newton GE, Austin PC, Wang X et al. Association of blood pressure at hospital discharge with mortality in patients diagnosed with heart failure. Circ Heart Fail. 2009;2(6):616–623.

10. Hoshide S, Kario K, Hoshide Y, Umeda Y, Hashimoto T, Kunii O et al. Associations between nondipping of nocturnal blood pressure decrease and cardiovascular target organ damage in strictly selected community-dwelling normotensives. Am J Hypertens. 2003;16(6):434–438.

11. Ingelsson E, Björklund-Bodegard K, Lind L, Arnlöv J, Sundström J. Diurnal blood pressure pattern and risk of congestive heart failure. J Am Med Assoc. 2006:295(24);2859–2866.

12. Shin J, Kline S, Moore M, Gong Y, Bhanderi V, Schmalfuss CM. Association of diurnal blood pressure pattern with risk of hospitalization or death in men with heart failure. J Card Fail. 2007:13(8);656–662.

13. de la Sierra A, Redon J, Banegas JR, Segura J, Parati G, Gorostidi M et al. Prevalence and factors associated with circadian blood pressure patterns in hypertensive patients. Hypertension. 2009;53(3):466–472.

14. Goto N, Uchida K, Morozumi K, Ueki T, Matsuoka S, Katayama A et al. Circadian blood pressure rhythm is disturbed by nephrectomy. Hypertens Res. 2005;28(4):301–306.

Author information:

Valeriy A. Serov, MD, PhD, DSc, Professor, Department of Internal and Professional Diseases, Medical Faculty, Ulyanovsk State University;

Aleksandr M. Shutov, MD, PhD, DSc, Professor, Head, Department of Internal and Professional Diseases, Medical Faculty, Ulyanovsk State University;

Diana V. Serova, MD, PhD student, Department of Internal and Professional Diseases, Medical Faculty, Ulyanovsk State University;

Svetlana V. Shevchenko, MD, Head, Cardiology Department, Ulyanovsk Central City Hospital;

Elena Yu. Shmelkova, MD, Deputy Chief Physician, Ulyanovsk Central City Hospital.