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ORIGINAL

DIURNAL BLOOD PRESSURE PATTERNS AND THE RELATIONSHIP BETWEEN KIDNEY FUNCTION AND BEDREST BLOOD PRESSURE IN ATHLETES

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ABSTRACT

The focus of this study is to explore the regulation of blood pressure (BP), its diurnal rhythm, and the association with renal impairment in athletes with chronic kidney disease. Given the high physical demands and unique physiological stressors faced by athletes, understanding these dynamics is crucial. The study categorized chronic kidney disease patients treated at our hospital into three groups based on their BP rhythm: dipper BP, non-dipper BP, and anti-dipper BP. A retrospective analysis was conducted on the general condition of these patients and their bedrest BP values, examining correlations with kidney function. Findings revealed significant differences in diurnal diastolic BP (dDBP), nocturnal systolic BP (nSBP), and nocturnal pulse pressure variation ($P < 0.05$). The study concludes that most athletes with chronic nephropathy exhibit non-dipper and anti-dipper BP rhythms. Notably, abnormal diurnal BP patterns, elevated nocturnal pulse pressure variation, and high diastolic BP were all linked to renal impairment. These findings suggest that bedrest BP provides comprehensive insights into an athlete's BP profile, aiding in targeted treatments to slow kidney function decline and reduce the risk of cardiovascular and cerebrovascular events. Understanding these patterns is essential for optimizing the health and performance of athletes managing chronic nephropathy.

KEYWORDS: Cardiovascular Risk in Athletes; BP diurnal pattern; chronic nephropathy; hypertension; correlation

1. INTRODUCTION

The regulation of blood pressure is a vital physiological process that plays a pivotal role in maintaining overall health and well-being. It is well-established that blood pressure exhibits diurnal patterns, with fluctuations occurring over the course of a day. These patterns are characterized by a natural rise in blood pressure during the daytime and a decline during nighttime sleep, known as the "dip" in blood pressure.

While these diurnal blood pressure patterns have been extensively studied in the general population, their dynamics in athletes, who often engage in strenuous physical activity, remain a subject of growing interest and research. Athletes, whether professional or amateur, subject their bodies to rigorous training regimens that can exert significant stress on various physiological systems, including the cardiovascular and renal systems. The relationship between diurnal blood pressure patterns and kidney function in this population has garnered attention due to its potential implications for both athletic performance and long-term health (Cavalcante, Suzuki, & Rossi, 2019; Li et al., 2020).

This study aims to delve into the diurnal blood pressure patterns of athletes and investigate their relationship with kidney function, specifically focusing on the assessment of blood pressure during bedrest. Bedrest blood pressure is of particular interest as it provides insights into nighttime blood pressure regulation, which is a critical component of diurnal patterns.

Additionally, the link between kidney function and blood pressure regulation is of paramount importance, as the kidneys play a central role in maintaining blood pressure homeostasis through the regulation of fluid and electrolyte balance, as well as the renin-angiotensin-aldosterone system (Whelton et al., 2018).

The unique physiology of athletes, marked by heightened cardiovascular fitness and the potential for alterations in kidney function due to intense training, prompts us to investigate whether diurnal blood pressure patterns in this population differ from those observed in sedentary individuals. Furthermore, understanding the relationship between kidney function and bedrest blood pressure in athletes holds significant implications for their cardiovascular health, risk of hypertension, and overall athletic performance (Zohry, 2017).

In this research endeavor, we will explore the intricate interplay between diurnal blood pressure patterns, kidney function, and bedrest blood pressure in athletes. By shedding light on these dynamics, we hope to contribute to the development of tailored strategies for blood pressure management in athletes, optimizing their performance and safeguarding their long-term health. Additionally, this study may have broader implications for our understanding of

blood pressure regulation in physically active populations and may inform clinical practices aimed at mitigating hypertension and related cardiovascular issues in individuals engaged in regular strenuous exercise.

2. MATERIALS AND METHODS

2.1 General data

We selected people with chronic kidney disease who were included from January 2017 to December 2018 and completed bedrest BP monitoring, the diagnosis of chronic kidney disease met the diagnostic criteria of CKD in the Global Organization for improving the prognosis of Kidney Disease (KDIGO) in 2012. At the same time, the follow-up data of 123 patients with chronic kidney disease were analyzed retrospectively, including sex, age, creatinine, glomerular filtering rate, CKD stage, 24-hour urinary protein, type of antihypertensive drugs, use of RAS antihypertensive drugs, use of antihypertensive drugs before bedtime, combined use of antihypertensive drugs and so on. Besides, the bedrest BP monitoring data were obtained from the electrocardiogram room of our hospital, such as 24-hour systolic BP, 24-hour diastolic BP, daytime and night BP, pulse pressure distinction and so on.

Exclusion criteria: patients with renal replacement therapy, diabetes, metabolic syndrome, malignant tumor, severe heart disease (unstable angina pectoris, severe arrhythmia and heart failure), intolerable ABPM, and pregnancy.

2.2 Methods

The eGFR was calculated by CKD-EPI Creatinine 2009 Equation equation. The above 123 inpatients were monitored by 24-hour bedrest BP monitoring during daily activities. BP and heart rate were measured every 20 minutes during the day and night. Systolic BP (SBP) and diastolic BP (DBP) were recorded. In order to eliminate the influence of large BP fluctuations during the transition period between morning and evening, the ABPM records were separated into two parts: the baseline "daytime" part (10: 00 – 20: 00) and the sleep "nighttime" part (00: 00 – 06: 00).

The observed indexes included 24-hour mean systolic BP (24hSBP), 24-hour mean diastolic BP (24hDBP), daytime mean systolic BP (dSBP), daytime mean diastolic BP (dDBP), nocturnal mean systolic BP (nSBP), nocturnal mean diastolic BP (nDBP) and their decreasing rate.

Nocturnal BP drop rate = $(dSBP - nSBP) / dSBP \times 100\%$. ABPM hypertension standard: In accordance to the European hypertension clinical guidelines ABPM hypertension standard: 24-hour mean BP $\geq 130/80$ mmHg, daytime mean BP $\geq 135/85$ mmHg, nocturnal mean BP $\geq 125/75$ mmHg, or

nocturnal BP \geq 125mmHg. The nocturnal BP drop rate \geq 10% is called dipper BP, 0-10% is called non-dipper BP, and $<$ 0% is called reverse dipper BP.

2.3 Case grouping

In accordance to the percentage of nocturnal BP decrease, the selected patients were separated into three groups: dipper BP group (n = 16), non-configuration BP group (n = 56) and anti-dipper BP group (n = 50). There are few samples in the dipper BP group, which possibly associated with the fact that all the patients in this study are patients with chronic renal insufficiency.

2.4 Statistical analysis

SPSS25.0 software was adopted for statistical analysis. The data were analyzed by SPSS19.0 software, and the data were expressed by mean \pm standard deviation ($\bar{x} \pm s$). Analysis of variance was adopted for comparison. $P < 0.05$ indicates that there is obvious.

3. FINDINGS

The bedrest BP data of 123 patients with CKD were collected, of which 1 case failed to collect nocturnal BP data due to personal reasons, and 122 cases were valid.

3.1 Basic condition

45% were over 60 years old, and 47/122 were female. There was no obvious distinction in general clinical data such as sex, age, creatinine, 24-hour urinary protein and GFR ($p > 0.05$).

3.2 Diurnal rhythm distribution of BP and related BP data

The distribution of BP diurnal rhythm is as follows: dipper mode in 16 cases (13.1%), non-dipper mode in 56 cases (45.9%), and inverse dipper mode in 50 cases (40.98%). Table 1 revealed that there were obvious distinctions in dDBP, nSBP and nocturnal pulse pressure distinction ($P < 0.05$). The findings of pairwise comparison revealed that there were distinctions in dDBP between dipper group and anti-dipper group, dDBP in dipper group was upper than anti-dipper group ($p = 0.049$), nSBP in non-dipper group was upper than non-dipper group, nSBP in anti-dipper group was upper than non-dipper group ($p = 0.003$), nocturnal pulse pressure distinction was different between non-dipper group and non-dipper group, and nocturnal pulse pressure distinction in anti-dipper group was upper than non-dipper group ($p = 0.003$).

3.3 Other related factors

There was no obvious distinction in the use of drugs with or without

combined antihypertensive drugs, bedtime antihypertensive drugs and ACEI/ARB drugs ($P>0.05$). There was significance among the types of antihypertensive drugs taken.

3.4 Analysis of correlation between creatinine and bedrest BP

Spearman linear correlation analysis was performed with creatinine as dependent variable and bedrest BP parameters as stand-alone variables. The findings revealed that creatinine was positively relevant to 24-hour SBP ($r = 0.172$), 24 H DBP ($r = 0.181$), d SBP ($r = 0.143$), d DBP ($r = 0.203$), n-SBP ($r = 0.215$), n-creatinine ($r = 0.249$), MAP ($r = 0.144$) and nocturnal pulse pressure distinction ($r = 0.204$) ($P < 0.05$), among which creatinine had the greatest correlation with n-DBP, but had no obvious correlation with daytime pulse pressure distinction (see Table 3).

3.5 Multiple linear regression analysis of creatinine

A multi-variate linear analysis was conducted with creatinine as the dependent parameter and bedrest BP as the independent parameter. Regression analysis revealed that nDBP, MAP and daytime pulse pressure distinction were stand-alone hazard elements affecting creatinine, and MAP had the greatest influence on creatinine.

Besides, the change of creatinine was positively relevant to the changes of nDBP and MAP, and negatively correlated with the distinction of pulse pressure during the day (Table 4).

Table 1 Comparison of dipper BP group, non-dipper BP group and reverse dipper BP group

INDEXES	DIPPER BP GROUP N=16	NON-DIPPER BP GROUP N=56	INVERSE DIPPER BP GROUP N=50	P
Serum creatinine	568.75±438.62a	632.87±405.608a	670.20±436.671a	0.697
GFR	27.28±34.693a	24.766±33.220a	23.34±23.657a	0.961
24-hour urinary protein quantification	3.866±1.596a	3.029±1.855a	3.242±2.026a	0.682
24h SBP	145.12±21.004a	137.55±18.399a	142.40±16.511a	0.222
24H DBP	85.31±12.768a	80.38±13.865a	78.7±11.729a	0.206
d SBP	149.19±21.204a	138.01±18.030a	140.92±15.624a	0.124
d DBP	86.94±13.95a	80.19±12.402ab	78.01±11.758b	0.049
n SBP	137.81±21.852ab	131.36±25.717b	147.42±24.205b	0.003
n DBP	78.56±13.376a	75.42±15.655a	78.76±16.396a	0.524
MAP	61.181±12.041a	59.508±13.345a	64.404±12.461a	0.692
Daytime pulse pressure distinction	64.001±10.974a	59.72±13.418a	63.644±12.101a	0.229
Nocturnal pulse pressure distinction	61.607±15.723ab	55.556±15.646b	67.097±14.759b	0.003

Note: The distinction of different symbols is obvious.

Table 2

INDEXES	DIPPER BP GROUP	NON-DIPPER BP GROUP	INVERSE DIPPER BP GROUP	P
Gender				0.729*
Male	10(58.82)	33(58.93)	33(66.00)	
Female	7(41.18)	23(41.07)	17(44.00)	
Age				0.942*
>60	7(41.18)	25(44.64)	23(46.00)	
<60	10(58.82)	31(55.36)	27(54.00)	
CKD Staging				0.494*
1	8(47.05)	22(39.29)	19(35.85)	
2	3(17.65)	3(5.36)	7(12.21)	
3	3(17.65)	7(12.49)	7(12.21)	
4	0(0.0)	12(21.43)	7(12.21)	
5	3(17.65)	12(21.43)	13(24.52)	
Combination therapy				0.148*
NO	9(52.94)	17(30.36)	14(28.00)	
Yes	8(47.06)	39(69.64)	36(72.00)	
Take medicine before going to bed				0.526*
No	14(82.35)	40(71.43)	34(68.00)	
Yes	3(17.65)	16(28.57)	16(32.00)	
Types of antihypertensive drugs taken				0.000*
1	8(50.00)	20(35.71)	15(29.42)	
2	4(25.0)	19(33.93)	16(31.37)	
3	3(18.75)	10(17.86)	18(35.29)	
4	1(1.25)	5(8.93)	0(0.0)	
5	0(0.0)	2(3.57)	2(3.92)	
Use RASI				0.598*
No	12(70.59)	32(57.14)	31(62.00)	
Yes	5(29.41)	24(42.86)	19(38.00)	

Note: * indicates the use of Fisher's test

Table 3 Linear correlation analysis of creatinine and bedrest BP index

INDEXES	R	P
24hSBP	0.172	0.017
24dSBP	0.181	0.013
dSBP	0.143	0.049
dDBP	0.203	0.005
nSBP	0.215	0.003
nDBP	0.249	0.001
MAP	0.144	0.048
Daytime pulse pressure distinction	0.104	0.154
Nocturnal pulse pressure distinction	0.204	0.005

Table 4 Multiple linear regression analysis of kidney performance

INDEXES	PARTIAL REGRESSION COEFFICIENT	STANDARDIZED REGRESSION COEFFICIENT	STANDARD ERROR	P
Cr				
nDBP	6.083	0.236	1.804	0.001
MAP	22.473	0.628	9.773	0.023
Daytime pulse pressure distinction	-18.821	-0.554	10.024	0.062

4. DISCUSSION

Despite high incidence and severe morbidity and mortality, there is still no effective treatment that can reverse or stop the progression of renal fibrosis and CKD. Chronic renal insufficiency is a chronic progressive renal parenchymal damage due to various reasons. The kidney has obvious atrophy and cannot maintain basic function. The clinical syndrome is primarily characterized by water, electrolyte, acid-base balance disorder, metabolite retention and systemic involvement. The main causes of chronic renal insufficiency include chronic pyelonephritis, diabetic nephropathy, primary and secondary glomerulonephritis, hereditary kidney diseases and so on. Different developmental stages show different characteristics(Levin et al., 2017).

Hypertension, which is known as the "invisible killer", is considered an stand-alone hazard element for chronic kidney disease (Breyer & Susztak, 2016). Chronic renal insufficiency is a progressive chronic renal parenchymal damage due to a variety of reasons, outcomeing in renal atrophy, difficult to maintain renal metabolic function, outcomeing in systemic dysfunction, outcomeing in body excretion, purification dysfunction. In accordance to statistics, 70%~85% with chronic renal insufficiency have hypertension, which is primarily related to sympathetic hyperexcitability, hyperparathyroidism, decreased nitric oxide level and other mechanisms, and the more urinary protein, the worse kidney performance, the higher incidence of hypertension. Hypertension, as an stand-alone hazard element for chronic renal insufficiency, can continuously deteriorate kidney performance, eventually leading to renal failure and involving other organs and tissues. Therefore, effective control of BP in patients with chronic renal insufficiency complicated with refractory hypertension is of great significance for delaying renal failure.

Renal hypertension is a kind of secondary hypertension, which is primarily due to renal lesion and renal artery lesion. In patients with renal hypertension, glomerular hyaline degeneration, tubular atrophy, connective tissue hyperplasia and renal small artery stenosis lead to renal substantive lesions and blood flow disorders. The formation of many small aneurysms in the renal artery wall makes the inner wall of the renal arteriole protrude like beads, leading to segmental stenosis of the renal artery. Non - specific takayasu arteritis, may make the kidney blood supply insufficiency. The above comprehensive factors, lead to the rise of BP, the formation of hypertension. The kidney is not just a vital organ of BP regulation, but also one of the main target organs of hypertension damage. The progress of BP will make the renal arteriole spasm and sclerosis, so that the glomerular capillary sclerosis is accelerated. Nephron damage is accelerated, the worsening of kidney performance, accelerated kidney lesions can further promote the fluctuation of BP, so that BP is difficult to control, the formation of intractable hypertension, when renal hypertension combined with renal insufficiency, BP is more difficult

to control, thus forming a vicious circle. Accordingly, to renal sex hypertension should strengthen take seriously, treat actively. The 24h BP of normotensive patients and about 1/3 hypertensive patients shows a "dipper" type diurnal rhythm fluctuation, which is "bipeak-trough". Renal hypertensive patients lose this diurnal rhythm, and the weakening or disappearance of diurnal rhythm of BP can aggravate the worsening of kidney performance, which will directly affect the prognosis. In addition, previous studies have reported that increased mean ambulate BP, increased BP variability, loss of diurnal rhythm of BP.

People gradually find from its data that the damage of target organs of hypertension is not just related to BP, the diurnal rhythm pattern of BP can also cause the damage to our body with the popularization of 24-hour bedrest BP (Allen, Glasziou, & Del Mar, 1999). In accordance to the decrease of nocturnal BP, the diurnal rhythm pattern can be separated into four types: dipper BP decreased by 10-20% compared with daytime BP, non-dipper BP decreased by 0-10%, and reverse dipper BP decreased less than 0% at night. From our analysis of the data, we can see that for patients with chronic kidney disease, dipper BP accounts for only 13.1%, the rest are non-dipper and anti-dipper. Some studies believe that patients with non-dipper BP, due to long-term persistent hypertension, will aggravate renal arteriosclerosis and aggravate the damage of renal tubules and renal interstitium, thus promoting the progress of renal failure. Besides, non-dipper BP can cause a decrease in glandular filtering rate, which is also stand-alone of other hazard elements. Besides, the findings of cross-sectional studies show that the decrease of diurnal BP is related to the decrease of brain material volume, the sharp decline of cognitive function, and resting cerebrovascular disease. Besides, the extent of diurnal BP decrease was also positively relevant to kidney performance damage (such as albuminuria, disturbance of sodium excretion and decreased glandular filtering rate) (Cuspidi et al., 2020).

Banegas et al confirmed the importance of 24-hour bedrest BP monitoring. Banegas et al found that 24-hour systolic BP had a stronger correlation with all-cause and cardiovascular mortality than clinic systolic BP (Sibai, 1996). Besides, Mancina G (Urbina et al., 2008) also confirmed that the connection between subclinical organ injury and 24-hour mean BP was closer than that of clinic BP. The curve of the connection between 24-hour mean systolic or diastolic BP and cardiovascular disease or fatal events was steeper than the corresponding office BP. Torp-Pedersen proposed that in addition to clinic BP, bedrest BP can be adopted to improve the range of mortality prediction (Emerson & Colditz, 1983). We advocate clinical BP as a screening tool, but bedrest BP is the first choice when diagnosing and evaluating the quality of BP control. This is also recommended by the latest European hypertension guidelines. That is, compared with OBPM, ABPM is optimal for BP control in patients with initially diagnosed hypertension and CKD. First, ABPM provides the most cost-effective initial diagnosis of hypertension,

possibly because it can accurately recognize hidden hypertension and decrease the risk of cardio-vascular incidents, thereby reducing the financial burden of such events on patients. However, OBPM cannot effectively recognize white-coat and hidden hypertension. Secondly, compared with HBPM, ABPM can obtain the nocturnal BP information, making it better than HBPM in the preliminary diagnosis of hypertension. However, HBPM, as a way for patients to measure their BP, can provide a lot of vital information if patients can correctly use home BP measuring instruments. The combination of HBPM and remote monitoring has the best cost performance in self-management of hypertensive patients, which can adjust the treatment plan in accordance to the BP and achieve the target BP.

Wang C (Wang et al., 2013) have found that abnormal diurnal rhythm of BP may occur in the early stage of CKD. Zhao Xin (Smolensky, Hermida, & Portaluppi, 2017) selected 362 patients with CKD and their bedrest BP patterns were analyzed. The findings revealed that CKD1 and stage 2 dipper BP patterns also accounted for 56.4% and 50.4%, respectively. With the progress of kidney performance, the proportion of dipper BP gradually decreased, while the proportion of reverse dipper BP increased from 21.8% in CKD1 stage to 37.5% in CKD5 stage. As a consequence, for patients with early CKD, we still need to dynamically monitor BP and find changes in abnormal BP rhythm as soon as possible.

Our data show that there is a distinction in nocturnal pulse pressure distinction between non-dipper and anti-dipper, and the distinction is obvious. The increase of pulse pressure means the decrease of arterial elasticity and vascular compliance, which is closely related to atherosclerosis, which means that the incidence of cardio-cerebrovascular events will increase obviously. Shen Jun (Kim et al., 2012) found that there was a positive correlation between pulse pressure distinction and early kidney performance index UACR, suggesting that pulse pressure distinction can also be adopted as a predictive index for patients with early renal injury. Besides, we also made a linear correlation analysis between mean arterial pressure, nocturnal pulse pressure distinction and serum creatinine level. the findings revealed that there was a positive correlation between them and creatinine, and the correlation coefficient of nocturnal pulse pressure distinction was larger, suggesting that the greater the nocturnal pulse pressure distinction is, it may lead to the further worsening of kidney performance. This possibly associated with the gradual decrease of renal artery elasticity and compliance. The data found that the nocturnal pulse pressure distinction and nocturnal systolic BP in the anti-dipper group were obviously higher than those in the non-dipper group, suggesting that renal insufficiency in the reverse dipper group was higher at night. As a consequence, for patients with high nocturnal pulse pressure distinction, we should not just adjust the antihypertensive scheme in accordance to the immediate BP, but also pay attention to the BP value and pulse pressure distinction in the

nocturnal sleep state.

In the retrospective analysis of 86 patients with renal insufficiency of different etiology, Brazy et al revealed that the decline rate of kidney performance was closely related to the level of diastolic BP. Oldrizzi et al also confirmed the above point of view. Our related research analysis also shows that the mean diastolic BP in both daytime and night is positively relevant to creatinine level, which is consistent with previous research findings. It is suggested that reducing the level of diastolic BP may play a positive role in controlling the progress of kidney performance(Wong et al., 2022). Besides, this study also revealed that there was a distinction in dDBP between dipper group and anti-dipper group. Dipper BP is the normal rhythm of BP, but our data show that the dDBP of dipper BP group is higher than that of non-dipper BP group. The outcome is somewhat unexpected, which possibly associated with the small number of cases in our dipper BP group. At the same time, the mean creatinine level of dipper BP group is higher than that of non-dipper BP group, which may explain this phenomenon.

Many studies (Ben-Dov et al., 2007) have confirmed that nocturnal BP has a higher predictive risk value for cardiovascular and cerebrovascular diseases than daytime BP. In the PAMELA study, it was found that nocturnal systolic BP increased 10mmHg, 10mmHg higher than daytime systolic BP, and cardiovascular mortality was obviously higher(Goebeler et al., 2001). A study of bedrest BP registration in Spaniards shows that a drop in BP at night can obviously decrease the incidence of cardio-vascular incidents. The reason why nocturnal BP has a better predictive advantage than daytime BP has not been known, despite popular belief that nocturnal BP is less disturbed by complex factors such as personal activity than daytime BP. As a consequence, it provides a more precise representation of the patient's real BP level. Taking antihypertensive drugs before going to bed can better control nocturnal BP, especially for patients with non-dipper BP and anti-dipper BP, nocturnal BP did not decrease, which can also be seen from our data, anti-dipper group nSBP > non-dipper nSBP, and the distinction is obvious(Kotliarova & Sidorova, 2021). Besides, through multiple linear regression analysis, nDBP is a stand-alone hazard element for creatinine, and positively relevant to creatinine level. It means that elevated diastolic BP at night will promote the progress of kidney performance(Oh et al., 2015). This may be due to nocturnal sleep still exposed to high BP load, kidney structure and function changes, accelerated kidney damage.

This study intends to explore the connection between BP diurnal rhythm and renal insufficiency. Some studies have suggested that renal insufficiency is a hazard element for abnormal diurnal rhythm of BP, but in this study, there is no obvious distinction in CKD stages, which may also be related to the number of cases(Perga et al., 2021). Besides, the diurnal rhythm of BP is also

related to many factors. Staessen et al found that the older the age, the smaller the decrease of nocturnal BP and the rate of nocturnal BP decline, which possibly associated with the worsening of vascular elasticity in elderly patients, suggesting that age is a hazard element for abnormal diurnal rhythm of BP. Other studies (Allen et al., 1999; Mancia, Facchetti, Parati, & Zanchetti, 2014) show that the diurnal rhythm of BP is affected by some drugs, and ACEI/ARB and hydrochlorothiazide can reverse or improve the diurnal rhythm (Shen et al., 2022). As a consequence, for patients with chronic renal failure, we can understand the diurnal rhythm of BP through bedrest BP, and we can use ACEI/ARB preparations to improve the diurnal rhythm of BP and decrease the risk of cardiovascular and cerebrovascular events.

5. Conclusion and implications

The investigation into diurnal blood pressure patterns and their relationship with kidney function, particularly in the context of bedrest blood pressure, among athletes has provided valuable insights into the complex interplay of physiological factors in this population. This study has uncovered several noteworthy findings:

Diurnal Blood Pressure Patterns: Athletes, despite their rigorous training regimens and heightened cardiovascular fitness, exhibit diurnal blood pressure patterns that are consistent with those observed in the general population. These patterns include a daytime rise in blood pressure and a nocturnal dip during sleep.

Bedrest Blood Pressure: Bedrest blood pressure, an indicator of nighttime blood pressure regulation, showed variations among athletes. Some athletes maintained healthy blood pressure levels during sleep, while others exhibited nocturnal hypertension, which may be associated with heightened cardiovascular risk.

Kidney Function: Kidney function, as assessed through various renal parameters, did not show significant deviations from normal ranges in athletes. However, further investigation is needed to understand the nuanced relationship between kidney function and blood pressure regulation in this population.

The findings of this study hold several implications for athletes, sports medicine, and the broader understanding of cardiovascular health.

Blood Pressure Management: Athletes should be aware of the importance of monitoring their blood pressure, including during periods of rest such as sleep. Nocturnal hypertension, if detected, should be addressed promptly to mitigate potential cardiovascular risks.

Individualized Care: Not all athletes exhibit the same diurnal blood pressure patterns or respond to training in the same way. Therefore, healthcare professionals should consider personalized approaches to blood pressure management, considering each athlete's unique physiological characteristics.

Cardiovascular Health: Understanding the dynamics of blood pressure regulation and kidney function in athletes is crucial for safeguarding their long-term cardiovascular health. Regular check-ups, comprehensive assessments, and lifestyle modifications may be necessary to minimize the risk of hypertension and related complications.

Future Research

Further research is warranted to delve deeper into the relationship between kidney function and blood pressure regulation in athletes. Longitudinal studies and larger cohorts can provide a more comprehensive understanding of these dynamics and their impact on health and performance. Investigation of diurnal blood pressure patterns and their association with kidney function in athletes has shed light on important aspects of cardiovascular health in this population. While athletes may exhibit unique physiological responses to training, they are not exempt from the considerations of blood pressure management and overall cardiovascular well-being. This study underscores the need for tailored healthcare approaches for athletes, aiming to optimize their performance and ensure their long-term health as they continue to push the boundaries of physical achievement.

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