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HEMODYNAMIC CHARACTERISTICS IN PATIENTS WITH PERMANENT ATRIAL FIBRILLATION ACROSS DIFFERENT HEART RATE RANGES AND LEFT VENTRICULAR EJECTION FRACTION LEVELS USING CONTINUOUS NON-INVASIVE BLOOD PRESSURE MONITORING

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Aim. Permanent atrial fibrillation (AF) requires rate control. However, the optimal heart rate (HR) remains a matter of debate. Hemodynamic parameters such as mean arterial pressure (MAP) and the proportion of hemodynamically ineffective beats (HIB), derived from pulse pressure (PP), may provide insight into the hemodynamic characteristics of AF at different HRs and left ventricular ejection fractions (LVEF).

Methods. The study included 135 patients with permanent AF (aged 37-90 years, symptom class 1-2A according to the mEHRA scale). For each patient, beat-to-beat systolic BP, diastolic BP, and PP were measured using the "volume clamp" method over a 15-minute period with the "Cardiotechnika-SAKR" system (Incart, Saint Petersburg). On each cardiac cycle, MAP and its variability were calculated using two independent metrics: Average Real Variability (ARV) and Root Mean Square of Successive Differences (RMSSD).

Results. The percentage of HIBs, defined per patient as deviations from the mean PP, considered as 1 («mild» <0.75, «moderate» <0.5, «severe» <0.25) - significantly increased with higher HR. Across all HR ranges (60-89 and >110 bpm), except 90-110 bpm, HIBs were more frequent in patients with reduced LVEF (<50%) than in those with preserved LVEF (≥50%) ($p<0.05$). Significant inter-individual differences in HIBs were observed among patients with similar average HRs within both the 60-89 and 90-110 bpm groups, in both preserved and reduced LVEF subgroups. MAP decreased with increasing HR; at HR >110 bpm, MAP was 80.4 ± 12.3 mmHg in the reduced LVEF group versus 94.1 ± 14.1 mmHg in the preserved LVEF group ($p<0.05$). With rising HR, MAP did not change significantly in either preserved or reduced LVEF groups, and no between-group differences were found across HR ranges ($p>0.05$).

Conclusion. Heart rate control in permanent AF should be individualized, hemodynamically guided, and account for LVEF. Beat-to-beat monitoring enables identification of the optimal HR that minimizes HIBs and MAP variability while maintaining MAP within accepted reference values, particularly in patients with impaired contractile function.

Keywords: permanent atrial fibrillation, beat-to-beat blood pressure, «volume clamp» method, hemodynamics, hemodynamically ineffective beats, mean arterial pressure, blood pressure variability, heart rate, RMSSD, ARV

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Atrial fibrillation (AF) is the most common form of cardiac arrhythmia, characterised by chaotic electrical activity in the atria, resulting in ineffective atrial contractions. The condition has a significant impact on patients' quality of life and is associated with an increased risk of stroke, heart failure, and mortality. With increasing life expectancy and the growing prevalence of risk factors such as hypertension, obesity, and diabetes mellitus, the incidence of AF continues to rise steadily [1-4].

The permanent form of this arrhythmia implies a single available therapeutic approach-heart rate (HR) control. Determination of the optimal HR in patients with AF has been the subject of numerous studies, among which RACE II, AFFIRM, and AF-CHF are considered foundational in this context [5-7]. These studies formed the basis for principles well known to every practising cardiologist and reflected in current Russian clinical guidelines and European consensus documents: achievement of an HR not exceeding 110 beats per minute in asymptomatic patients and below 80 beats per minute in patients with pronounced symptoms. These recommendations apply to patients without heart failure (HF), whereas in patients with HF, maintenance of an HR below 100 beats per minute is considered preferable.

Despite ongoing debate and published post hoc analyses highlighting limitations of existing approaches,

these HR ranges remain target values for clinical practice, albeit with less than the highest class of recommendation [1, 2, 8-10]. A potential solution for determining optimal HR may lie in the assessment of haemodynamic characteristics of AF using a device that records arterial blood pressure (BP) on a beat-to-beat basis. Although primarily known as a tool for autonomic function testing, this method has also proven reliable for accurate BP measurement in AF, with peripheral values calibrated against synchronous brachial measurements obtained on the contralateral arm using the Korotkoff method [11, 12].

Quantification of so-called haemodynamically ineffective cardiac contractions (HICCs), which essentially represent an instrumental objectification of the well-known physical examination finding of pulse deficit, together with assessment of mean haemodynamic arterial pressure (MHAP), reflecting the degree of continuous peripheral perfusion, may provide insight into the haemodynamic features of AF at different HR levels.

Thus, the aim of this study was to analyse haemodynamic changes in patients with permanent atrial fibrillation at different heart rates and left ventricular ejection fraction values.

MATHERIAL AND METHODS

The study included 135 patients aged 37 to 90 years (84 men and 51 women) with permanent non-valvular AF, without evidence of intraventricular conduction disturbances (QRS duration <120 ms on electrocardiography (ECG)), and with a symptom severity class according to the modified EHRA (mEHRA) scale corresponding to class 1 (asymptomatic) or class 2A (mild symptoms not interfering with daily activities). The clinical study was conducted in accordance with Good Clinical Practice standards and the principles of the Declaration of Helsinki and was approved by the local ethics committee of the medical centre. Written informed consent for participation in the study was obtained from all patients. The clinical characteristics of the study population are presented in Table 1.

The study had an observational, hypothesis-generating design. All patients meeting the inclusion criteria who presented to the clinic between September 2023 and December 2024 were consecutively enrolled. No a priori sample size calculation was performed.

Determination of haemodynamic parameters

Each patient underwent continuous beat-to-beat recording of systolic, diastolic, and pulse arterial blood pressure (PBP) for 15 minutes using the unloaded artery method, with simultaneous 12-lead ECG recording. Measurements were performed using the Cardiotechnika-SAKR system (Inkart JSC, Saint Petersburg, Russia; patents RU 2694737 C1 and RU 2698447 C1, V.V. Pivovarov et al.) [13-15].

Mean haemodynamic arterial pressure

MHAP reflects a stable level of tissue perfusion in the systemic circulation and averages approximately 100 mmHg (normal range 70-100 mmHg) [16]. Since arterial pressure changes represent a complex periodic function, MHAP is not equal to the half-sum of maximal (systolic) and minimal (diastolic) pressures, but rather corresponds to the mean of infinitesimal pressure changes from maximum to minimum over a single cardiac cycle [17]. Therefore, for automated calculation of MHAP for each cardiac cycle, the following formula was used:

$$p_{\text{cp}} = \frac{1}{\Delta t}\int_{t_1}^{t_2} p(t)\ dt$$

where p_{cp} - MHAP over the time interval; $p(t)$ - instantaneous pressure at time t; t_1, t_2 - start and end times of the cardiac cycle; $\Delta t = t_2 - t_1$ - duration of the cardiac cycle.

Variability of mean haemodynamic arterial pressure

Variability of MHAP in this study was assessed using two independent indices: ARV and RMSSD. Both indices were calculated based on MHAP data obtained for each patient for every cardiac cycle. The ARV index reflects the mean absolute change in MHAP between successive heartbeats and is sensitive to short-term pressure instability, independently of its absolute value [18]. Formally, it was calculated as the arithmetic mean of the absolute differences between adjacent measurements:

$$\text{ARV} = \frac{1}{N-1}\sum_{i=1}^{N-1}|MAP_{i+1} - MAP_i|$$

where ARV - average real variability of MHAP, reflecting the mean absolute difference between successive measurements; N - total number of MHAP (MAP) measurements over the analysed period; MAP_i - MHAP value at the i-th measurement; $|MAP_{i+1} - MAP_i|$ - absolute difference between adjacent MHAP measurements.

In turn, RMSSD represents the square root of the mean of squared differences between successive MHAP values; it is more sensitive to abrupt fluctuations and is widely used in variability analysis of autonomic regulation [19]:

$$\text{RMSSD} = \sqrt{\frac{1}{N-1}\sum_{i=1}^{N-1}(MAP_{i+1} - MAP_i)^2}$$

where RMSSD - root mean square of successive differences, an index of short-term MHAP variability; N - total number of MHAP (MAP) measurements over the analysed period; MAP_i - MHAP value at the i-th measurement; $(MAP_{i+1} - MAP_i)^2$ - squared difference between adjacent MHAP measurements.

Before intergroup comparisons, statistical assumptions were tested: normality of distribution was assessed using the Shapiro-Wilk test, and homogeneity of variances using Levene's test. In cases of normal distribution and variance homogeneity, one-way analysis of variance (ANOVA) was applied (for ≥3 groups) or Student's t-test. When normality assumptions were violated, non-parametric equivalents were used, including the Kruskal-Wallis test or the Mann-Whitney U test. A p value < 0.05 was considered statistically significant.

RESULTS

Based on the results of 15-minute beat-to-beat blood pressure monitoring, mean values of systolic, diastolic, and pulse blood pressure (PBP) were obtained. The mean PBP was accepted as an individual reference value for each

patient (set as "1"). Deviations from this value were categorised as moderate, marked, and severe. Thus, following completion of the recording, the software automatically calculated, for each patient, the percentage of cardiac contractions corresponding to values below 0.75, below 0.5, and below 0.25 of the mean PBP taken as 1. In this manner, HICCs of varying severity were identified. Alternatively, this approach may be described as determining the proportion of cardiac contractions that are 25%, 50%, and 75% less effective than the individual mean PBP taken as 100%.

Haemodynamically ineffective cardiac contractions

Among the 135 patients, 33 had a reduced left ventricular ejection fraction (LVEF <50%), while 102 patients had preserved LVEF (≥50%). The absolute LVEF values in these groups were 43.67 ± 4.74% and 68.29 ± 11.05%, respectively. Patients were stratified according to mean HR ranges (60-89, 90-110, and >110 beats per minute), which allowed assessment of the effect of HR on the proportion of HICCs. This stratification reflects both physiological patterns of ventricular filling and ejection and clinically relevant HR control thresholds recommended in Russian and international guidelines for the management of AF. Groups with preserved and reduced LVEF were compared across the above HR ranges.

The proportion of HICCs increased significantly with increasing HR in both patient groups ($p < 0.001$) (Table 2). At the same time, patients with reduced LVEF demonstrated a significantly higher overall proportion of HICCs compared with patients with preserved LVEF in the HR ranges of 60-89 and >110 beats per minute. In the HR range of 60-89 beats per minute, the proportion of moderate HICCs was 17.8 ± 8.5% in the reduced LVEF group compared with 10.5 ± 5.9% in the preserved LVEF group ($p < 0.001$). The proportion of marked HICCs was also higher in patients with reduced LVEF, with mean values of 7.3 ± 6.0% versus 3.6 ± 4.0%, respectively ($p = 0.004$). Differences in the proportion of severe deviations did not reach statistical significance (3.8 ± 4.9% vs 2.2 ± 3.0%; $p = 0.09$).

At an HR of 90-110 beats per minute, no statistically significant differences between groups were observed for any of the parameters ($p > 0.5$). Pronounced differences in HICCs were again observed at HR > 110 beats per minute: the proportion of moderate HICCs was 48.2 ± 4.7% in the reduced LVEF group compared with 38.5 ± 7.0% in the preserved LVEF group ($p = 0.006$); the proportion of marked HICCs was 42.07 ± 6.52% versus 28.29 ± 9.56% ($p = 0.006$); and the proportion of severe deviations reached 34.4 ± 8.6% and 20.2 ± 9.7%, respectively ($p = 0.005$).

Given the presence of samples with non-normal distribution, statistical comparisons were additionally performed using non-parametric tests (Kruskal-Wallis and Mann-Whitney tests). No substantial discrepancies from the previously obtained results were identified.

Mean haemodynamic arterial pressure

When analysing mean haemodynamic arterial pressure (MHAP) in relation to HR in both patients with preserved and reduced LVEF, no statistically significant differences were identified (Table 2). In both subgroups, data distribution deviated from normality and variance homogeneity was violated; therefore, non-parametric methods were applied. According to the Kruskal-Wallis test, MHAP values did not change significantly with increasing HR either in patients with preserved LVEF ($p = 0.087$) or in those with reduced LVEF ($p = 0.081$). Comparison of MHAP values between the two LVEF groups across all HR ranges also revealed no statistically significant differences (Mann-Whitney test, $p = 0.075$-0.65).

The maximum individual MHAP values in the preserved LVEF group reached 126.5 mmHg at an HR of 60-89 beats per minute and 123.0 mmHg at an HR of 90-110 beats per minute. In the reduced LVEF group, maximum MHAP values also exceeded 120 mmHg, but only at HRs up to 110 beats per minute; at HR > 110 beats per minute, MHAP did not exceed 100 mmHg in any patient. Moreover, hypoperfusion profiles (MHAP − SD <70 mmHg) were observed specifically in this subgroup.

Variability of mean haemodynamic arterial pressure

Under sinus rhythm, variability of mean haemodynamic arterial pressure (MHAP) remains relatively stable and minimal. However, in AF, pronounced irregularity of cardiac cycles results in a substantial increase in MHAP variability. This phenomenon is likely of even greater clinical relevance than a simple increase or decrease in mean MHAP level. It is precisely the abrupt, albeit short-term, episodes of hypoperfusion characteristic of AF that may adversely affect perfusion of vital organs.

The use of two complementary variability indices (ARV and RMSSD) allows for a more comprehensive characterisation of short-term haemodynamic fluctuations. In the present study, these indices were calculated for all patients and additionally stratified by mean HR ranges (60-89, 90-110, and >110 beats per minute), enabling an

Table 1.
Clinical characteristics and pharmacological therapy of patients

Parameter	Value
Arterial hypertension, n (%)	131 (97.0)
Coronary artery disease, n (%)	27 (20.0)
History of myocardial infarction, n (%)	9 (6.7)
History of stroke, n (%)	8 (5.9)
Type 2 diabetes mellitus, n (%)	22 (16.3)
Pharmacological therapy	
ACE inhibitors / ARBs / ARNIs, %	112 (83.0)
Beta-blockers, %	130 (96.3)
MRAs, %	33 (24.4)
SGLT2 inhibitors, %	51 (37.8)
Statins, %	90 (66.7)
Metformin, %	22 (16.3)
NOACs, %	131 (97.0)

Note: ACE inhibitors - angiotensin-converting enzyme inhibitors; ARBs - angiotensin II receptor blockers; ARNIs - angiotensin receptor-neprilysin inhibitors; MRAs - mineralocorticoid receptor antagonists; SGLT2 - sodium-glucose cotransporter 2; NOACs - novel oral anticoagulants.

integrated analysis of dynamic blood pressure stability depending on myocardial functional status.

The analysis demonstrated that ARV and RMSSD differed significantly according to HR, but only in patients with preserved LVEF (Table 3). In this subgroup, a statistically significant increase in both ARV ($p < 0.001$) and RMSSD ($p < 0.001$) was observed when transitioning from an HR range of 60-89 beats per minute to the ranges of 90-110 and >110 beats per minute. The highest variability values were recorded during tachycardia (>110 beats per minute): ARV reached 6.0 ± 2.3 mmHg, and RMSSD reached 7.3 ± 2.8 mmHg.

In contrast, no such dependence was observed in patients with reduced LVEF: neither ARV ($p = 0.85$) nor RMSSD ($p = 0.98$) demonstrated statistically significant changes across HR ranges. When comparing patients with preserved and reduced LVEF within each of the three HR ranges, no statistically significant differences were identified (all $p > 0.05$), except for higher MHAP variability in patients with reduced LVEF at an HR of 60-89 beats per minute (ARV: $p = 0.004$; RMSSD: $p = 0.01$).

Sensitivity analysis using non-parametric tests fully reproduced the identified differences: all key comparisons retained statistical significance at level of 0.05, confirming the robustness and reliability of the obtained results.

DISCUSSION

In the present study, haemodynamic characteristics continuously determined on a beat-to-beat basis in patients with permanent AF were, for the first time, compared not only with HR but also with left ventricular function. The obtained data help to clarify why the "universal" target HR ranges proposed in large controlled trials (AFFIRM, RACE II, AF-CHF) often prove to be clinically inadequate in everyday practice.

Haemodynamically ineffective cardiac contractions

From a physiological perspective PBP, albeit indirectly, reflects stroke volume and vascular tone; therefore, its dynamics in AF may serve as an integral marker of cardiac cycle efficiency, reflecting both the quality of ventricular mechanical activation and the degree of peripheral resistance. Although the thresholds for HICCs were initially selected empirically, they successfully capture the patterns of haemodynamic response variability in AF, allowing quantitative assessment of the degree of reduction in contraction efficiency depending on heart rate and myocardial contractile function.

The number of HICCs increased proportionally with HR; however, the magnitude of this phenomenon was significantly greater in patients with reduced LVEF. Differences were already evident in the HR range of 60-89 beats per minute and became maximal during tachycardia >110 beats per minute (for example, severe HICCs: 34.4 ± 8.6% vs 20.2 ± 9.7%; $p = 0.005$). These findings indicate an additional contribution of impaired pump function to AF haemodynamics: rhythm irregularity combined with reduced contractility markedly increases the proportion of contractions with low stroke volume and, consequently, the risk of peripheral hypoperfusion.

Despite the presence of general trends, substantial individual deviations from the apparent overall patterns were identified. Thus, among patients with preserved LVEF in the HR subgroup of 60-89 beats per minute, despite similar mean HR values (~83 beats per minute according to 15-minute monitoring), pronounced differences in the proportion of HICCs were observed. In one patient, the proportions reached 30.9% moderate, 20.2% marked, and 13.3% severe contractions, whereas in another patient they were only 9.8%, 1.6%, and 0.1%, respectively. A similar pattern was observed in the HR range of 90-110 beats per minute: at comparable mean HR values (~100 beats per minute over 15 minutes), one patient exhibited 26.2% moderate, 22.9% marked, and 20.3% severe HICCs, while another showed only 10.0%, 5.0%, and 3.1%, respectively.

Comparable interindividual differences in HICC parameters were also observed in patients with reduced LVEF within the same HR ranges. However, at HR values exceeding 110 beats per minute, such pronounced interindividual variability in HICCs was no longer observed in either the preserved or reduced LVEF groups. This phenomenon likely underlies the difficulties encountered in determining whether a "lenient" or "strict" HR control strategy is optimal. Apparently, the individual structure of AF rhythm exerts a significant influence on haemodynamics at comparable HR values, which is particularly important to consider at HRs below the threshold beyond which rhythm control becomes ineffective and the proportion of HICCs increases sharply regardless of rhythm organisation.

Table 2.
Parameters of haemodynamically ineffective cardiac contractions in patients with preserved and reduced left ventricular ejection fraction across different heart rate ranges (n = 135)

LVEF, %	HR, bpm	n	HICCs, %			MHAP, mmHg	SD BP, mmHg	p*
			Moderate	Marked	Severe			
≥50	60-89	60	10.5±5.9	3.6±4.0	2.1±2.9	101±15	5.9±1.6	<0.001
	90-110	28	21.1±6.9	13.1±7.2	8.8±6.4	96±13	6.2±1.6	
	>110	14	38.5±7.0	28.3±9.9	20.2±9.7	94±14	6.3±1.8	
<50	60-89	16	17.8±8.5	7.3±6.0	3.7±4.6	95±14	6.1±2.6	<0.001
	90-110	11	22.2±9.0	12.6±9.2	7.4±8.2	98±12	5.7±0.7	
	>110	6	48.2±4.7	42.1±7.1	34.4±8.6	80±12	6.3±1.5	

Note: HICCs - haemodynamically ineffective cardiac contractions; LVEF - left ventricular ejection fraction; HR - heart rate; MHAP - mean haemodynamic arterial pressure; SD - standard deviation; p - one-way ANOVA.

It is precisely in the HR range below 110 beats per minute that individual differences in rhythm structure make the greatest contribution to haemodynamic response variability, whereas above this threshold the impact of irregularity is attenuated due to overall destabilisation of the cardiac cycle and a reduction in stroke volume of virtually every contraction.

It should be emphasised that the obtained data are not proposed as "target" values for HICCs. Rather, they should be regarded as individual characteristics suitable for dynamic monitoring. Establishment of universal therapeutic targets requires large prospective studies linking these metrics to clinical outcome.

Mean haemodynamic arterial pressure

Unlike systolic and diastolic blood pressure, which may exhibit substantial fluctuations, MHAP is characterised by relative stability. I.P. Pavlov considered this parameter to be one of the homeostatic constants of the organism. MHAP reflects a stable level of tissue perfusion without accounting for pressure pulsations.

In AF, particularly in its chronic form, activation of the sympathoadrenal system is typical and serves as a compensatory mechanism in the setting of reduced cardiac output. This leads to peripheral vasoconstriction, an increase in total peripheral vascular resistance, and elevation of diastolic blood pressure. MHAP depends primarily on diastolic pressure; therefore, a significant increase in diastolic blood pressure results in a corresponding rise in MHAP. This effect is especially pronounced at high HR, when shortening of diastole limits the time available for arterial emptying, thereby contributing to the maintenance of elevated pressure during the diastolic phase. Thus, under AF conditions, MHAP may remain high or even increase despite a reduction in stroke volume, reflecting a mismatch between central haemodynamics and peripheral vascular resistance.

The obtained data confirm the relative stability of this parameter regardless of HR level and myocardial contractile function. In patients with preserved LVEF, a tendency toward elevated MHAP was observed in some cases, which may reflect enhanced sympathetic activation, increased vascular tone, and preserved adaptive reserves. Achievement of MHAP values exceeding 120 mmHg in individual patients may have pathophysiological consequences, including increased afterload and subsequent vascular wall remodelling.

In patients with reduced LVEF, MHAP tended to decrease with increasing HR; however, this change did not reach statistical significance ($p = 0.075$). At HR values exceeding 110 beats per minute, none of the patients demonstrated MHAP levels above 100 mmHg, which may indicate critical vulnerability of this population to tachycardia in the setting of AF.

Variability of MHAP

Analysis of ARV and RMSSD demonstrated that in patients with preserved LVEF, MHAP variability increased with rising HR ($p < 0.001$), whereas no comparable changes were observed in the reduced LVEF group. A plausible explanation is that under conditions of impaired pump function, pressure variability is limited by a "ceiling" imposed by low cardiac output, whereas in patients with preserved LVEF, fluctuations in preload and afterload become more pronounced at higher HR. Another potential explanation for the absence of increasing MHAP variability with rising HR in the reduced LVEF group is the presence of angiosclerosis, increased arterial wall stiffness, and reduced arterial compliance, which limit the amplitude of pressure oscillations in response to changes in stroke volume.

Thus, pharmacological HR reduction aimed at decreasing MHAP variability in patients with reduced LVEF is likely to be of less benefit than in patients with preserved LVEF. Nevertheless, monitoring of absolute MHAP values remains clinically relevant.

Study limitations

A limitation of the study is the relatively small number of patients with reduced LVEF, particularly in the subgroup with HR >110 beats per minute. In addition, participants received different combinations of pharmacological therapies, which may have heterogeneous effects on peripheral vasoregulation and blood pressure variability. Despite consecutive enrolment, the cohort was drawn from a single specialised centre and did not include patients with pronounced AF-related symptoms (mEHRA ≥2B) or severe heart failure (NYHA class IV). Therefore, extrapolation of the results to populations with a higher symptomatic burden and/or decompensated heart failure should be undertaken with caution and requires confirmation in future studies.

No separate statistical power calculation was performed; consequently, results obtained in small subgroups (n <10) should be considered descriptive and interpreted cautiously. Stratification by type and dose of medications was not performed, which may introduce systematic bias. The use of the domestic Cardiotechnika-SAKR system, with its specific algorithmic approach for calibrating peripheral blood pressure values against simultaneous contralateral brachial measurements, does not allow full extrapolation of the findings to other systems employing the unloaded artery method [20]. At the same time, this reflects the uniqueness of the system used, which, among currently known analogous devices, uniquely enables accurate blood pressure measurement in AF owing to its built-in mathematical algorithms referencing Korotkoff sound.

CONCLUSION

In Western clinical practice, the physical examination finding of pulse deficit appears to have long been abandoned, based on the notion of its limited clinical util-

Table 3.

Indices of mean arterial pressure variability in patients

LVEF, %	HR, bpm	n	ARV±SD, mmHg	RMSSD±SD, mmHg
≥50%	60-89	60	4.0±1.2	5.3±1.6
	90-110	28	4.8±1.5	6.4±1.9
	>110	14	6.0±2.3	7.3±2.8
<50%	60-89	16	5.3±2.3	6.8±2.9
	90-110	11	5.5±1.7	6.9±1.9
	>110	6	5.9±1.6	7.1±1.8

Note: ARV - average real variability; SD - standard deviation; RMSSD - root mean square of successive differences.

ity [21]. This view is partly justified, as pulse deficit assessment by physical examination is inherently subjective and depends on individual tactile sensitivity. However, the advent of non-invasive beat-to-beat blood pressure monitoring enables objective quantification of this parameter. Continuous blood pressure monitoring at each cardiac contraction allows determination of an individually optimal heart rate, adjusted for the proportion of haemodynamically ineffective cardiac contractions, mean haemodynamic arterial pressure, and MHAP variability.

The obtained findings support the rationale for an individualised, haemodynamically oriented approach to heart rate control in AF, particularly in patients with impaired cardiac pump function, and provide a justification for incorporating beat-to-beat blood pressure monitoring into clinical practice to optimise therapeutic strategies.

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