Masked Hypertension, Circadian Dysregulation and Early Target-Organ Damage in Young Servicemen: a Multistage Detection Algorithm and Its Six-Month Effect in a Prospective Cohort of 300 Men

Buriyev U.A.[1*], **Nuritdinov N.A.**[1]

[1] *Military Medical Institute, University of Military Security and Defence of the Republic of Uzbekistan, Tashkent, Uzbekistan*

[*] *Corresponding author: Buriyev U.A., Military Medical Institute, Tashkent, Uzbekistan*

Running title: *Multistage detection of hypertension in young servicemen*

Abstract

Objective. To characterise the structure of blood-pressure (BP) phenotypes, circadian dysregulation and early target-organ damage (TOD) in young servicemen and to evaluate a multistage algorithm of early detection and personalised prevention of arterial hypertension. **Materials and methods.** A prospective single-centre cohort study with an organisational intervention included 300 male servicemen aged 18–30 years (2023–2024). Office BP, 7-day home BP monitoring (HBPM), 24-h ambulatory BP monitoring (ABPM), echocardiography, carotid ultrasound and renal markers were assessed. Servicemen with high-normal BP, masked or manifest hypertension (n=196) were followed for 6 months in a main group (algorithm with personalised prevention, n=97) and a comparison group (standard surveillance, n=99). **Results.** Only 32.0 % of servicemen were normotensive; high-normal BP was found in 26.0 %, masked hypertension in 23.0 % and manifest hypertension in 19.0 %. Exercise-related BP surges were recorded in 37.3 % (up to 53.7 % in special-purpose units). The dipper profile fell from 60.4 % in normotensives to 29.0 % in masked and 19.3 % in manifest hypertension, while night-peakers rose from 4.2 to 17.4 and 24.6 %. Left-ventricular mass index increased from 84±12 to 96±15 g/m², carotid intima–media thickness from 0.58±0.06 to 0.72±0.09 mm and microalbuminuria from 3.1 to 14.0 % (all $p<0.01$), with changes already present at the high-normal and masked stages. Nocturnal systolic BP correlated with LVMI ($\rho=0.41$), IMT ($\rho=0.38$) and ACR ($\rho=0.39$). After 6 months the algorithm reduced office SBP/DBP by 5.8/5.3 %, nocturnal SBP by 6 mm Hg, morning surge from 31±7 to 25±6 mm Hg, exercise-related episodes from 2.1±0.9 to 1.0±0.6 per day (−52.4 %), increased the dipper share from 29.9 to 47.4 %, and produced regression of LVMI (−5.4 %), IMT (−0.04 mm) and microalbuminuria (10.3→5.2 %); the comparison group did not change (between-group $p\leq0.047$). **Conclusions.** Office measurement identifies only part of hypertensive burden in young servicemen; masked forms and disturbed circadian regulation are associated with early cardiac, vascular and renal involvement. A multistage sequence — office BP → HBPM → ABPM → target-organ assessment — combined with correction of sleep, service load and behavioural factors normalises circadian BP regulation and partly reverses early TOD.

Keywords: *arterial hypertension; masked hypertension; servicemen; ambulatory blood pressure monitoring; non-dipper; night-peaker; target-organ damage; intima–media thickness; microalbuminuria; prevention.*

Introduction

Arterial hypertension remains the leading modifiable risk factor for ischaemic heart disease, stroke and heart failure; the number of adults with hypertension worldwide has doubled since 1990 to about 1.28 billion, and control remains inadequate in most of them [1, 2]. Blood pressure elevated from young adulthood is associated with earlier target-organ damage and a higher lifetime risk of cardiovascular events, and even values in the high-normal range in people under 40 predict events later in life [3, 4]. Current guidelines therefore recommend out-of-office measurement — home and 24-h ambulatory monitoring — to identify masked and borderline hypertension and to start prevention in working-age individuals [5–7]. Masked hypertension carries a cardiovascular risk comparable to sustained hypertension [8, 9], and nocturnal blood pressure, insufficient nocturnal dipping and an exaggerated morning surge are independent predictors of outcomes and of subclinical organ damage [10–13].

Young servicemen form a distinct population in which occupational stress, irregular sleep, night duties, high physical loads and nicotine use converge; elevated blood pressure is recorded in military personnel already at a young age, and the standard office measurement performed at periodic examinations underestimates the real haemodynamic load [14, 15]. Studies of hypertension in Uzbekistan and neighbouring countries have addressed mainly the general middle-aged

population, comorbidity and adherence; studies applying complex out-of-office monitoring to early detection in young military contingents are fragmentary and have not produced a unified algorithm for unit medical services. The present study was designed to fill this gap.

The aim of the study was to improve the effectiveness of early detection of arterial hypertension in young servicemen by optimising the system of blood-pressure monitoring and introducing personalised preventive measures to reduce cardiovascular complications.

Materials and methods

Design and participants. An open prospective single-centre cohort study with an organisational intervention was carried out in 2023–2024 at the clinical bases of the Military Medical Academy of the Armed Forces and the Central Military Hospital of the Ministry of Defence of the Republic of Uzbekistan. The cohort comprised 300 male servicemen aged 18–30 years (mean 23.6±3.1 years; median 23 [21; 26]) serving by conscription (60.3 %) or contract (39.7 %) in motorised infantry (44.7 %), air-assault (17.3 %), special-purpose (13.7 %) and signal, engineering and rear units (24.3 %); median service duration was 2.1 [1.0; 3.4] years, mean BMI 24.4±2.1 kg/m². Examinations were performed during periodic medical examinations in a period of relative well-being, after the standard period of adaptation to service. Secondary hypertension, previous vascular events and significant rhythm or conduction disorders were exclusion criteria. All participants gave written informed consent.

Blood-pressure phenotypes. Office BP was measured with validated Omron M6 Comfort devices after 5 min of rest, on both arms, twice, with the higher value recorded. Home BP monitoring (Omron M2 Basic) was performed for 7 days with two morning and two evening readings in a standardised diary; 24-h ABPM (Schiller BR-102 Plus; 20-min daytime and 40-min night-time intervals) was recorded under usual service conditions. Four phenotypes were defined according to the 2018 ESC/ESH criteria [5], consistent with the 2023 ESH guidelines [23]: normal BP (office <130/80 mm Hg, normal ABPM); high-normal BP (office 130–139/80–89 mm Hg with a normal 24-h profile); masked hypertension (office <140/90 mm Hg with 24-h mean ≥130/80 or daytime mean ≥135/85 mm Hg); manifest hypertension (office ≥140/90 mm Hg confirmed by ABPM). Exercise-related BP surges were defined as HBPM or daytime ABPM readings ≥135/85 mm Hg coinciding with service activity. Circadian profiles were classified by nocturnal SBP fall as dipper (10–20 %), non-dipper (<10 %), over-dipper (>20 %) and night-peaker (nocturnal rise) [6]; morning surge and daytime SBP variability (SD) were calculated from ABPM.

Target organs and laboratory tests. Echocardiography (GE Vivid 3) provided left-ventricular mass index (LVMI, indexed to body surface area), relative wall thickness (RWT) and ejection fraction; LV hypertrophy was defined as LVMI >115 g/m² and concentric remodelling as RWT >0.42 with normal LVMI [16]. Carotid duplex scanning measured intima–media thickness (IMT) of the common carotid artery 10 mm proximal to the bifurcation; IMT >0.9 mm, local thickening >50 % of adjacent segments and non-stenotic atherosclerosis were recorded [17]. Fasting lipids, glucose, HbA1c, uric acid and creatinine were measured on a Cobas c311 analyser in an ISO 15189-accredited laboratory; eGFR was calculated by CKD-EPI [18]; microalbuminuria was defined as 30–300 mg/day or albumin-to-creatinine ratio (ACR) >30 mg/g [19]. Physical activity and service load were assessed with a questionnaire adapted from IPAQ [20], together with sleep duration and the number of night duties.

Intervention. Servicemen with high-normal BP, masked or manifest hypertension (n=204) entered the 6-month phase; after exclusion of incomplete ABPM (n=5) and organisational drop-out (n=3), 196 were analysed: the main group (n=97) passed all stages of the algorithm — HBPM training, repeated ABPM, circadian profiling, target-organ assessment and individual prevention (normalisation of sleep ≥7 h and reduction of non-mandatory night duties; a temporarily lighter

physical-training regimen for servicemen with ≥3 exercise-related episodes per day; 20–30-min morning recovery periods for night-peakers; ≥10-min recovery after intense loads in air-assault and special-purpose units; salt restriction and smoking counselling) — while the comparison group (n=99) received standard surveillance. The groups were comparable in age (23.5±3.2 vs 23.7±3.1 years), service duration, branch composition and baseline office and ambulatory BP (all p>0.05). ABPM was repeated at 3 and 6 months.

Statistics. Data were processed in Statistica 10.0 and SPSS 26. Distributions were tested by Kolmogorov–Smirnov; data are given as M±SD, Me [Q1; Q3] or n (%). Groups were compared by the Mann–Whitney test, Pearson's χ^2 test and the χ^2 test for trend across phenotypes; associations were assessed by Spearman's ρ. $p<0.05$ was considered significant.

Results

Structure of BP phenotypes. Only 96 of 300 servicemen (32.0 %) were normotensive; 78 (26.0 %) had high-normal BP, 69 (23.0 %) masked hypertension and 57 (19.0 %) manifest hypertension, so that almost half of the cohort (49.0 %) was in the zone of disturbed BP regulation not identifiable by office measurement alone (Table 1, Fig. 1). The share of manifest hypertension rose from 10.4 % at 18–20 years to 40.9 % at 28–30 years (3.9-fold), whereas masked hypertension remained stable at 20–25 % across age bands. Exercise-related BP surges were recorded in 112 servicemen (37.3 %), increasing with age from 31.0 to 45.5 % and with service intensity — 35.8 % in motorised infantry, 50.0 % in air-assault and 53.7 % in special-purpose units versus 21.9 % in signal, engineering and rear units; masked hypertension was most frequent in special-purpose units (29.3 %) and least frequent in rear units (15.1 %).

Table 1. Distribution of blood-pressure phenotypes and exercise-related BP surges by age band and service branch (n=300)

Stratum	n	BP phenotype, n (%)				Exercise-related BP surges, n (%)
		normal BP	high-normal BP	masked HTN	manifest HTN	
Age 18–20 years	58	22 (37.9)	18 (31.0)	12 (20.7)	6 (10.4)	18 (31.0)
Age 21–24 years	129	44 (34.1)	36 (27.9)	32 (24.8)	17 (13.2)	44 (34.1)
Age 25–27 years	69	20 (29.0)	17 (24.6)	16 (23.2)	16 (23.2)	30 (43.5)
Age 28–30 years	44	10 (22.7)	7 (15.9)	9 (20.5)	18 (40.9)	20 (45.5)
Motorised infantry	134	46 (34.3)	33 (24.6)	32 (23.9)	23 (17.2)	48 (35.8)
Air-assault units	52	15 (28.8)	12 (23.1)	14 (26.9)	11 (21.2)	26 (50.0)
Special-purpose units	41	10 (24.4)	11 (26.8)	12 (29.3)	8 (19.5)	22 (53.7)
Signal, engineering and rear units	73	25 (34.2)	22 (30.1)	11 (15.1)	15 (20.5)	16 (21.9)
Whole cohort	**300**	**96 (32.0)**	**78 (26.0)**	**69 (23.0)**	**57 (19.0)**	**112 (37.3)**

Note. Exercise-related surges — at least one HBPM/daytime ABPM reading ≥135/85 mm Hg coinciding with service activity. Red — maximum, green — minimum values of the unfavourable indicators.

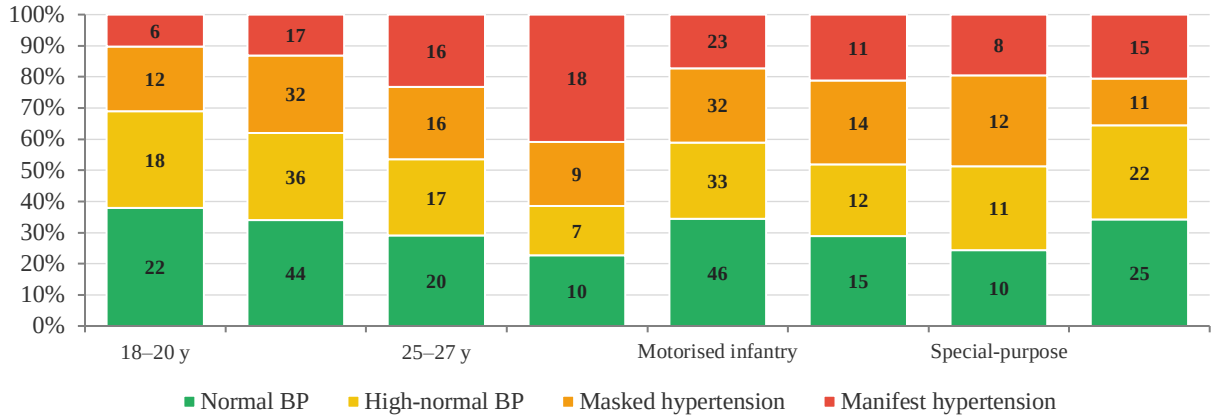


Fig. 1. Structure of BP phenotypes by age band and service branch (numbers in bars — n).

Clinical, behavioural and metabolic gradient. The frequency of complaints increased from normotension to manifest hypertension: headache from 15.6 to 43.9 %, dizziness from 7.3 to 24.6 %, sleep disturbance from 12.5 to 31.6 %, reduced exercise tolerance from 10.4 to 35.1 % and palpitations from 9.4 to 29.8 % (all p<0.05 for trend). Anthropometric, behavioural and metabolic indicators shifted consistently towards an unfavourable profile (Table 2): BMI ≥25 kg/m² was present in 18.8 % of normotensives and 42.1 % of manifest hypertensives, current smoking in 18.8 vs 36.8 %, sleep <6 h at least three times a week in 22.9 vs 42.1 %, ≥4 night duties per month in 25.0 vs 43.9 % and a family history of early cardiovascular disease in 22.9 vs 45.6 %. Total and LDL cholesterol, triglycerides, fasting glucose and HbA1c rose stepwise while HDL cholesterol fell; already between high-normal BP and masked hypertension LDL cholesterol differed by 0.20 mmol/l (p=0.04). Hyperuricaemia increased from 4.2 to 15.8 % (p=0.01 for trend).

Table 2. Anthropometric, behavioural and metabolic characteristics of servicemen by BP phenotype (n=300)

Indicator	Normal BP (n=96)	High-normal BP (n=78)	Masked HTN (n=69)	Manifest HTN (n=57)	p
Body-mass index, kg/m², M±SD	23.8±1.7	24.6±1.9	24.9±1.9	25.3±2.0	<0.05*
Waist circumference, cm, M±SD	82.5±5.0	84.3±5.5	85.1±5.6	86.4±5.8	<0.05*
BMI ≥25 kg/m², %	18.8	—	—	42.1	<0.05
Current smoking, n (%)	18 (18.8)	20 (25.6)	22 (31.9)	21 (36.8)	<0.05 (trend)
Sleep <6 h ≥3 times/week, n (%)	22 (22.9)	25 (32.1)	26 (37.7)	24 (42.1)	<0.05 (trend)
Night duties ≥4 per month, n (%)	24 (25.0)	28 (35.9)	28 (40.6)	25 (43.9)	<0.05 (trend)
Family history of early CVD, n (%)	22 (22.9)	24 (30.8)	25 (36.2)	26 (45.6)	<0.05†
Total cholesterol, mmol/l, M±SD	4.34±0.64	4.55±0.66	4.76±0.71	4.92±0.75	0.001†
LDL cholesterol, mmol/l, M±SD	2.55±0.54	2.78±0.58	2.98±0.62	3.12±0.66	<0.001†
HDL cholesterol, mmol/l, M±SD	1.27±0.24	1.22±0.22	1.18±0.22	1.15±0.21	0.02†
Triglycerides, mmol/l, M±SD	1.15±0.36	1.27±0.41	1.34±0.43	1.42±0.46	0.01†
HbA1c, %, M±SD	5.1±0.3	5.2±0.3	5.3±0.3	5.4±0.3	0.02†
Hyperuricaemia (>420 µmol/l), n (%)	4 (4.2)	6 (7.7)	7 (10.1)	9 (15.8)	0.01 (trend)

*Note. * — Mann–Whitney test versus normal BP, significant for masked and manifest hypertension; † — comparison of extreme groups (normal BP vs manifest hypertension); trend — χ² test for trend across phenotypes. Green — most favourable, red — least favourable values; "—" — not reported for intermediate groups.*

Circadian regulation and ambulatory parameters. The distribution of circadian profiles changed markedly across phenotypes (Fig. 2): the physiological dipper pattern was present in 60.4 % of normotensives, 43.6 % with high-normal BP, 29.0 % with masked and only 19.3 % with manifest hypertension, whereas non-dippers increased from 27.1 to 49.1 % and night-peakers from 4.2 to 24.6 % (χ², p<0.001). ABPM parameters formed a steep gradient (Table 3): 24-h SBP rose from 117±6 to 134±9 mm Hg and nocturnal SBP from 103±6 to 121±9 mm Hg (+17.5 %); nocturnal SBP fall decreased more than threefold (13.8±2.4 → 4.2±1.7 %), the morning surge rose

from 21±5 to 36±8 mm Hg and daytime SBP variability from 10.2±2.0 to 14.8±2.7 mm Hg (all p<0.001). A morning surge >35 mm Hg was found in 3.1 % of normotensives and 24.6 % of manifest hypertensives.

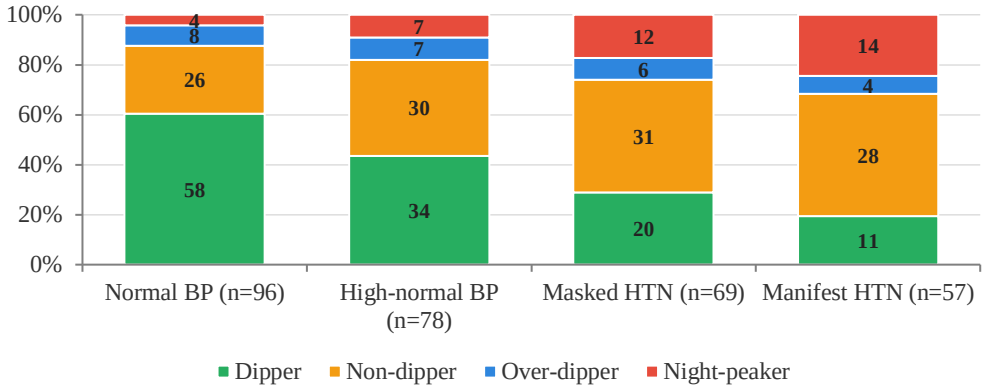


Fig. 2. Circadian BP profiles by phenotype (numbers in bars — n): loss of the dipper pattern and accumulation of night-peakers as the phenotype worsens.

Early target-organ damage. Structural and functional changes were detectable from the high-normal stage (Table 3, Fig. 3). LVMI increased from 84±12 g/m² in normotensives to 88±13, 92±14 and 96±15 g/m² in high-normal, masked and manifest hypertension (p<0.05 vs normal for every group), RWT from 0.37±0.05 to 0.42±0.07; concentric remodelling was found in 7.3, 14.1, 20.3 and 24.6 % and LV hypertrophy in 2.1, 5.1, 8.7 and 15.8 % with preserved ejection fraction. Carotid IMT rose by 8.6, 15.5 and 24.1 % relative to normotensives, and the prevalence of IMT >0.9 mm increased 3.6-, 6.9- and 10.9-fold (2.1 → 7.7 → 14.5 → 22.8 %; p<0.001); non-stenotic atherosclerosis was ten times more frequent in masked hypertension than in normotension (10.1 vs 1.0 %). Microalbuminuria increased from 3.1 to 6.4, 10.1 and 14.0 % (p=0.01) and eGFR <90 ml/min/1.73 m² from 6.3 to 19.3 %. For masked hypertension, differences from normotension were significant for LVMI, IMT and microalbuminuria (p=0.04), confirming preclinical organ involvement in a form of hypertension invisible to office measurement.

Table 3. Ambulatory BP parameters and markers of target-organ damage by BP phenotype (M±SD or n (%); n=300)

Parameter	Normal BP (n=96)	High-normal BP (n=78)	Masked HTN (n=69)	Manifest HTN (n=57)	p
24-h SBP, mm Hg	117±6	123±7	128±8	134±9	<0.001
Nocturnal SBP, mm Hg	103±6	109±7	114±8	121±9	<0.001
Nocturnal SBP fall, %	13.8±2.4	10.1±2.1	6.9±1.9	4.2±1.7	<0.001
Morning SBP surge, mm Hg	21±5	25±6	31±7	36±8	<0.001
Daytime SBP variability (SD), mm Hg	10.2±2.0	11.4±2.3	13.0±2.5	14.8±2.7	<0.001
LVMI, g/m²	84±12	88±13	92±14	96±15	<0.001
Relative wall thickness	0.37±0.05	0.39±0.06	0.41±0.06	0.42±0.07	0.01
Concentric remodelling, n (%)	7 (7.3)	11 (14.1)	14 (20.3)	14 (24.6)	0.02
LV hypertrophy (LVMI >115 g/m²), n (%)	2 (2.1)	4 (5.1)	6 (8.7)	9 (15.8)	0.03
Carotid IMT, mm	0.58±0.06	0.63±0.07	0.67±0.08	0.72±0.09	<0.001
IMT >0.9 mm, n (%)	2 (2.1)	6 (7.7)	10 (14.5)	13 (22.8)	<0.001
Non-stenotic atherosclerosis, n (%)	1 (1.0)	4 (5.1)	7 (10.1)	9 (15.8)	0.002
Microalbuminuria ≥30 mg/day, n (%)	3 (3.1)	5 (6.4)	7 (10.1)	8 (14.0)	0.01
eGFR <90 ml/min/1.73 m², n (%)	6 (6.3)	8 (10.3)	9 (13.0)	11 (19.3)	0.01

Note. p — between-group comparison (Mann–Whitney for quantitative variables, χ^2 for proportions); LVMI — left-ventricular mass index; IMT — intima–media thickness; eGFR — estimated glomerular filtration rate (CKD-EPI).

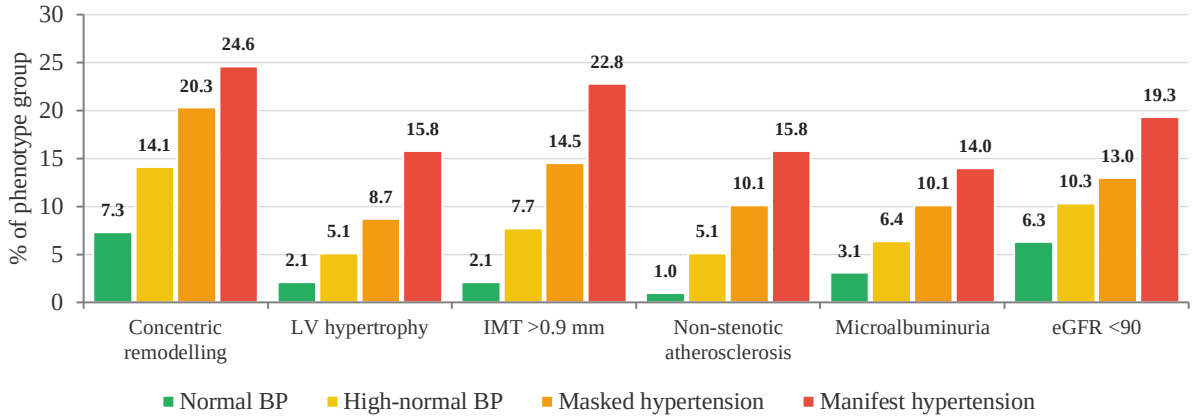


Fig. 3. Prevalence of early cardiac, vascular and renal damage by BP phenotype: a monotonic gradient starting at the high-normal stage.

Circadian profile and the kidney. Renal markers depended on the type of nocturnal profile: microalbuminuria was present in 4.9 % of dippers, 12.2 % of non-dippers, 12.0 % of over-dippers and 21.6 % of night-peakers (p=0.004), ACR rose from 9.8 to 18.6 mg/g (+90 %; p<0.001), eGFR fell from 112±9 to 101±12 ml/min/1.73 m² and hyperuricaemia increased from 2.4 to 16.2 % (p=0.003). Nocturnal SBP was the parameter most closely associated with organ damage (Fig. 4): ρ=0.41 with LVMI, 0.38 with IMT, 0.39 with ACR and −0.33 with eGFR (all p<0.001); the morning surge correlated with LVMI (ρ=0.35) and IMT (ρ=0.36), and SBP variability with ACR (ρ=0.32). Within phenotypes the strength of the nocturnal SBP–LVMI association increased from ρ=0.22 in normotensives to ρ=0.41 in manifest hypertension. Service branch modified the picture: night-peakers were twice as frequent in special-purpose (22.0 %) and air-assault (17.3 %) units as in motorised infantry (9.7 %; p=0.018), and the same units showed the highest IMT (0.70±0.08 mm), IMT >0.9 mm (17.1 %), concentric remodelling (24.4 %) and microalbuminuria (17.1 %) versus 4.1–6.8 % in signal, engineering and rear units (p≤0.032).

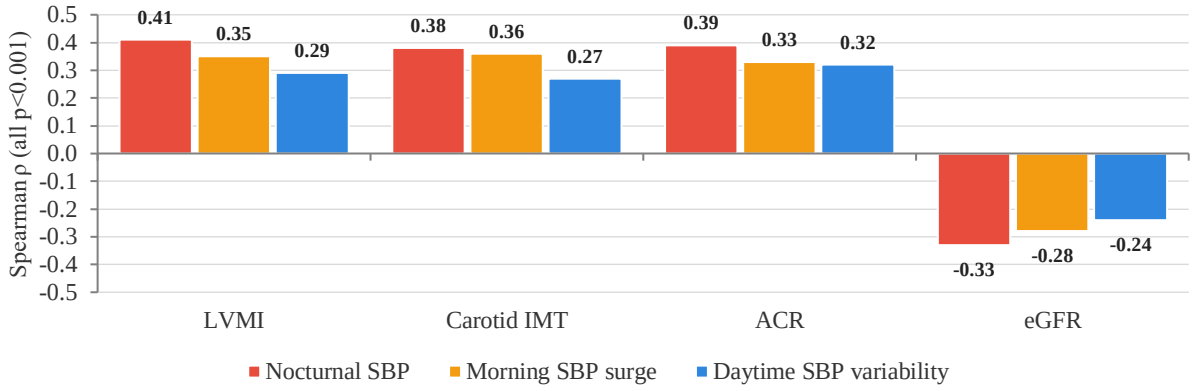


Fig. 4. Correlations of ambulatory BP parameters with markers of target-organ damage (n=300).

The multistage algorithm. On the basis of these patterns a four-level scheme was formulated: (1) office measurement with phenotypic stratification; (2) 7-day HBPM to capture exercise-related reactivity (≥135/85 mm Hg episodes); (3) 24-h ABPM with classification of the circadian profile, morning surge (≥30 mm Hg) and variability; (4) target-organ assessment — echocardiography, carotid ultrasound, microalbuminuria and eGFR — for high-normal BP, masked hypertension and unfavourable profiles. Integral stratification (phenotype + load reactivity + circadian profile + organ markers) defines a high-risk group (masked hypertension with non-dipper/night-peaker profile and organ changes) for intensive correction — sleep normalisation, reduction of night duties, restriction of high-intensity loads, weekly HBPM, repeated ABPM after 3 months and dynamic control of

organ markers — and a low-to-moderate-risk group for lifestyle measures with quarterly office control.

Six-month effect of the algorithm. All measured domains improved only in the main group (Table 4, Fig. 5). Office SBP and DBP decreased by 5.8 and 5.3 % ($p<0.001$) and the share of servicemen with office BP ≥140/90 mm Hg halved (29.9 → 14.4 %; $p=0.004$), whereas the comparison group changed by −1.3 and −0.8 %. On ABPM, 24-h and nocturnal SBP fell by 6 mm Hg, nocturnal dipping recovered from 6.9±1.9 to 9.8±2.3 %, the morning surge decreased from 31±7 to 25±6 mm Hg and SBP variability by 1.6 mm Hg (all $p<0.001$ between groups). The dipper share rose from 29.9 to 47.4 %, non-dippers fell from 48.5 to 34.0 % and night-peakers from 21.6 to 12.4 % ($p\leq0.004$), with an unchanged profile structure in the comparison group. Exercise-related episodes decreased from 2.1±0.9 to 1.0±0.6 per day (−52.4 %), the share of servicemen with ≥3 episodes per day from 28.9 to 12.4 %, maximal SBP/DBP during loads by 12/6 mm Hg, episodes after night duty from 21.6 to 9.3 % and during physical training from 44.3 to 22.7 %. Target organs responded: LVMI decreased from 92±14 to 87±13 g/m² (−5.4 %; $p=0.003$), RWT from 0.41 to 0.38, concentric remodelling from 14.4 to 7.2 % ($p=0.018$); IMT from 0.67±0.08 to 0.63±0.07 mm ($p<0.001$) and IMT >0.9 mm from 10.3 to 6.2 % ($p=0.047$); microalbuminuria from 10.3 to 5.2 % ($p=0.041$), ACR by 19 % and eGFR increased by 3 ml/min/1.73 m² ($p=0.03$). LV hypertrophy halved (6.2 → 3.1 %) without reaching significance ($p=0.09$).

Table 4. Six-month dynamics of blood pressure, circadian regulation, load reactivity and target-organ markers in the main and comparison groups

Indicator	Main group (n=97)			Comparison group (n=99)			p
	baseline	6 months	Δ	baseline	6 months	Δ	
Office BP							
Office SBP, mm Hg	134.6±7.8	126.8±6.9	−5.8 %	133.9±7.6	132.1±7.4	−1.3 %	<0.001
Office BP ≥140/90 mm Hg, n (%)	29 (29.9)	14 (14.4)	−15.5 pp	30 (30.3)	27 (27.3)	−3.0 pp	0.004
24-h ABPM							
24-h SBP, mm Hg	128±8	122±7	−6	127±8	126±8	−1	<0.001
Nocturnal SBP, mm Hg	114±8	108±7	−6	113±8	112±8	−1	<0.001
Nocturnal SBP fall, %	6.9±1.9	9.8±2.3	+2.9	7.1±1.8	7.5±1.9	+0.4	<0.001
Morning SBP surge, mm Hg	31±7	25±6	−6	30±7	29±7	−1	<0.001
Circadian profile							
Dipper, n (%)	29 (29.9)	46 (47.4)	+17.5 pp	30 (30.3)	32 (32.3)	+2.0 pp	<0.001
Night-peaker, n (%)	21 (21.6)	12 (12.4)	−9.2 pp	21 (21.2)	20 (20.2)	−1.0 pp	0.004
Exercise-related BP reactivity (HBPM)							
Episodes ≥135/85 mm Hg per day, M±SD	2.1±0.9	1.0±0.6	−52.4 %	2.0±0.8	1.8±0.8	−10.0 %	<0.001
Maximal SBP during load, mm Hg	164±10	152±9	−12	163±11	161±10	−2	<0.001
Episodes during physical training, n (%)	43 (44.3)	22 (22.7)	−21.6 pp	41 (41.4)	37 (37.4)	−4.0 pp	<0.001
Target organs							
LVMI, g/m²	92±14	87±13	−5.4 %	91±13	90±13	−1.1 %	0.003
Concentric remodelling, n (%)	14 (14.4)	7 (7.2)	−7.2 pp	13 (13.1)	12 (12.1)	−1.0 pp	0.018
Carotid IMT, mm	0.67±0.08	0.63±0.07	−0.04	0.66±0.08	0.66±0.08	0	<0.001
IMT >0.9 mm, n (%)	10 (10.3)	6 (6.2)	−4.1 pp	9 (9.1)	9 (9.1)	0	0.047
Microalbuminuria ≥30 mg/day, n (%)	10 (10.3)	5 (5.2)	−5.1 pp	9 (9.1)	9 (9.1)	0	0.041
ACR, mg/g, Me [Q1; Q3]	14.6 [10.1; 19.3]	11.8 [8.7; 16.4]	−19 %	14.2 [9.9; 18.6]	14.0 [10.1; 18.1]	−1 %	<0.001
eGFR, ml/min/1.73 m²	106±10	109±9	+3	107±10	107±10	0	0.03

Note. Δ — absolute change (mm Hg, ml/min/1.73 m², mm), relative change (%) or change in percentage points (pp); p — between-group comparison of the change. Baseline values did not differ between groups (p>0.05).

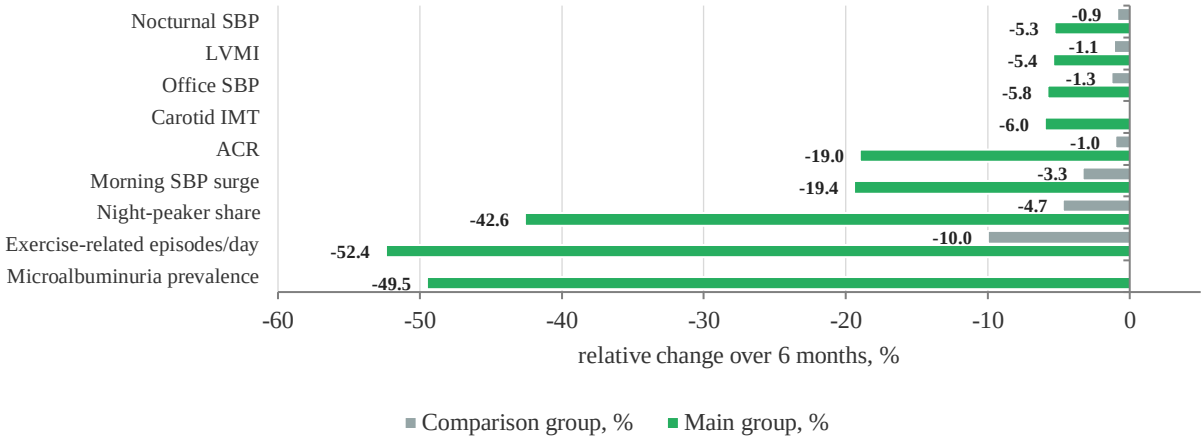


Fig. 5. Relative change of key indicators after 6 months: consistent improvement in the main group versus minimal drift in the comparison group.

Discussion

The principal finding is that in men aged 18–30 years serving in the armed forces the true hypertensive burden is concealed: only one in three is normotensive, and almost half occupy the high-normal and masked zone in which office measurement is uninformative. The 23.0 % prevalence of masked hypertension exceeds the 10–15 % reported in general adult populations [6, 8] and is consistent with the high rates of newly diagnosed hypertension in active-duty military cohorts, where deployment-related stress and physical load are the drivers [14, 15]. The specific contribution of the service environment is demonstrated by exercise-related BP surges, present in more than half of air-assault and special-purpose personnel, and by the concentration of night-peakers and organ changes in the same units at a comparable mean age.

The second finding is that early target-organ damage in this population is determined less by the absolute level of BP than by the failure of its circadian regulation. Nocturnal SBP, the morning surge and daytime variability were the parameters most closely related to LVMI, carotid IMT and albuminuria, and the night-peaker profile was accompanied by a 4.4-fold higher prevalence of microalbuminuria than the dipper profile. This reproduces, in a young cohort, the prognostic hierarchy established for nocturnal BP and blunted dipping in the IDACO and ABC-H analyses [10–12] and the association of the morning surge with vascular damage [13]. Importantly, the changes were present already at the high-normal and masked stages — LVMI 88–92 g/m², concentric remodelling 14–20 %, IMT 0.63–0.67 mm, microalbuminuria up to 10.1 % — which supports meta-analytic evidence that untreated masked hypertension is associated with subclinical cardiac damage and with a cardiovascular risk similar to sustained hypertension [8, 9, 24]. The behavioural and metabolic gradient (smoking, short sleep, night duties, dyslipidaemia, hyperuricaemia) accompanying the phenotypes agrees with the known effects of sleep deprivation and shift work on BP [21, 22].

The third finding is that the process is reversible at this stage. The intervention did not include drug therapy; it consisted of self-monitoring training, correction of sleep and recovery, redistribution of service load and behavioural counselling, applied to the mechanisms identified in the cross-sectional part — nocturnal BP, morning surge, variability and load reactivity. Within six months it restored the dipper profile in nearly half of the main group, halved night-peakers and exercise-related episodes and produced measurable regression of LVMI, carotid IMT and albuminuria, whereas the comparison group drifted by 0–1 mm Hg. The size of the effect on organ markers — a 5 % reduction of LVMI and a 0.04-mm reduction of IMT — is modest in absolute terms, but it is obtained in men without established hypertension and reflects the early, functional

stage of remodelling. The absence of comparable changes in the comparison group indicates that the improvement is not regression to the mean.

Practical implications. The proposed sequence — office BP → 7-day HBPM → 24-h ABPM → target-organ assessment — requires no equipment beyond that available to unit medical services and can be integrated into periodic medical examinations. Servicemen with high-normal office BP (130–139/80–89 mm Hg) should be considered a mandatory group for HBPM and ABPM irrespective of complaints, because 38.5 % of them are non-dippers and early organ changes are already detectable; those with non-dipper or night-peaker profiles, or with ≥3 exercise-related episodes per day or BP rises after night duty, constitute the priority group for correction of sleep, night-duty schedules and physical training, with re-evaluation after 2–4 weeks and repeated ABPM after 3 months.

Limitations. The study was single-centre and included only men; the intervention was allocated at unit level without randomisation or blinding, and the observation period was six months, so the durability of the effect and its translation into hard outcomes remain to be established. Circadian classification was based on a single baseline ABPM; reproducibility of dipping status is known to be moderate. Physical activity and sleep were self-reported. These limitations do not affect the direction of the associations, which were consistent across cardiac, vascular and renal markers.

Conclusions

1. In servicemen aged 18–30 years the structure of BP is dominated by borderline and hidden forms: normal BP is found in 32.0 %, high-normal BP in 26.0 %, masked hypertension in 23.0 % and manifest hypertension in 19.0 %; exercise-related BP surges occur in 37.3 %, with maximal frequency at 25–30 years and in units with high physical and stress load.

2. Worsening of the phenotype is accompanied by progressive circadian dysregulation — the dipper share falls from 60.4 to 19.3 % and night-peakers rise from 4.2 to 24.6 % — and nocturnal SBP, morning surge and variability are the parameters most strongly associated with LVMI, carotid IMT and albuminuria (ρ up to 0.41).

3. Early cardiac, vascular and renal changes are already present at the high-normal and masked stages (LVMI 88–92 g/m², concentric remodelling 14–20 %, IMT 0.63–0.67 mm, microalbuminuria up to 10.1 %), which justifies mandatory out-of-office monitoring and target-organ assessment in these groups.

4. The multistage algorithm with personalised non-pharmacological prevention normalises circadian regulation (dipper share 29.9 → 47.4 %, night-peakers 21.6 → 12.4 %), halves exercise-related BP episodes and partly reverses early organ damage (LVMI −5.4 %, IMT −0.04 mm, microalbuminuria 10.3 → 5.2 %) within six months, and is recommended as the basis of departmental standards for early detection of hypertension in military units.

Conflict of interest. The authors declare no conflict of interest.

Funding. The study was carried out within the research plan of the Military Medical Academy of the Armed Forces of the Republic of Uzbekistan without external funding.

Ethics. The protocol was approved by the institutional ethics committee; all participants gave written informed consent.

Author contributions. U.A.B. — concept, examination of servicemen, data analysis, manuscript; N.A.N. — supervision, study design, critical revision.

References

1. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in hypertension prevalence and progress in treatment and control from 1990 to 2019: a pooled analysis of 1201 population-representative studies with 104 million participants. Lancet. 2021;398(10304):957–980. doi:10.1016/S0140-6736(21)01330-1

2. Mills KT, Stefanescu A, He J. The global epidemiology of hypertension. Nat Rev Nephrol. 2020;16(4):223–237. doi:10.1038/s41581-019-0244-2

3. Lewington S, Clarke R, Qizilbash N, Peto R, Collins R; Prospective Studies Collaboration. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. Lancet. 2002;360(9349):1903–1913. doi:10.1016/s0140-6736(02)11911-8
4. Yano Y, Reis JP, Colangelo LA, et al. Association of blood pressure classification in young adults using the 2017 American College of Cardiology/American Heart Association blood pressure guideline with cardiovascular events later in life. JAMA. 2018;320(17):1774–1782. doi:10.1001/jama.2018.13551
5. Williams B, Mancia G, Spiering W, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. Eur Heart J. 2018;39(33):3021–3104. doi:10.1093/eurheartj/ehy339
6. Parati G, Stergiou G, O'Brien E, et al. European Society of Hypertension practice guidelines for ambulatory blood pressure monitoring. J Hypertens. 2014;32(7):1359–1366. doi:10.1097/HJH.0000000000000221
7. Stergiou GS, Palatini P, Parati G, et al. 2021 European Society of Hypertension practice guidelines for office and out-of-office blood pressure measurement. J Hypertens. 2021;39(7):1293–1302. doi:10.1097/HJH.0000000000002843
8. Fagard RH, Cornelissen VA. Incidence of cardiovascular events in white-coat, masked and sustained hypertension versus true normotension: a meta-analysis. J Hypertens. 2007;25(11):2193–2198. doi:10.1097/HJH.0b013e3282ef6185
9. Banegas JR, Ruilope LM, de la Sierra A, et al. Relationship between clinic and ambulatory blood-pressure measurements and mortality. N Engl J Med. 2018;378(16):1509–1520. doi:10.1056/NEJMoa1712231
10. Boggia J, Li Y, Thijs L, et al. Prognostic accuracy of day versus night ambulatory blood pressure: a cohort study. Lancet. 2007;370(9594):1219–1229. doi:10.1016/S0140-6736(07)61538-4
11. Hansen TW, Li Y, Boggia J, Thijs L, Richart T, Staessen JA. Predictive role of the nighttime blood pressure. Hypertension. 2011;57(1):3–10. doi:10.1161/HYPERTENSIONAHA.109.133900
12. Salles GF, Reboldi G, Fagard RH, et al. Prognostic effect of the nocturnal blood pressure fall in hypertensive patients: the Ambulatory Blood Pressure Collaboration in Patients With Hypertension (ABC-H) meta-analysis. Hypertension. 2016;67(4):693–700. doi:10.1161/HYPERTENSIONAHA.115.06981
13. Kario K, Pickering TG, Umeda Y, et al. Morning surge in blood pressure as a predictor of silent and clinical cerebrovascular disease in elderly hypertensives: a prospective study. Circulation. 2003;107(10):1401–1406. doi:10.1161/01.cir.0000056521.67546.aa
14. Smoley BA, Smith NL, Runkle GP. Hypertension in a population of active duty service members. J Am Board Fam Med. 2008;21(6):504–511. doi:10.3122/jabfm.2008.06.070182
15. Granado NS, Smith TC, Swanson GM, et al. Newly reported hypertension after military combat deployment in a large population-based study. Hypertension. 2009;54(5):966–973. doi:10.1161/HYPERTENSIONAHA.109.132555
16. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28(1):1–39.e14. doi:10.1016/j.echo.2014.10.003
17. Lorenz MW, Markus HS, Bots ML, Rosvall M, Sitzer M. Prediction of clinical cardiovascular events with carotid intima-media thickness: a systematic review and meta-analysis. Circulation. 2007;115(4):459–467. doi:10.1161/CIRCULATIONAHA.106.628875
18. Levey AS, Stevens LA, Schmid CH, et al. A new equation to estimate glomerular filtration rate. Ann Intern Med. 2009;150(9):604–612. doi:10.7326/0003-4819-150-9-200905050-00006
19. Gerstein HC, Mann JF, Yi Q, et al. Albuminuria and risk of cardiovascular events, death, and heart failure in diabetic and nondiabetic individuals. JAMA. 2001;286(4):421–426. doi:10.1001/jama.286.4.421
20. Craig CL, Marshall AL, Sjöström M, et al. International physical activity questionnaire: 12-country reliability and validity. Med Sci Sports Exerc. 2003;35(8):1381–1395. doi:10.1249/01.MSS.0000078924.61453.FB
21. Gangwisch JE, Heymsfield SB, Boden-Albala B, et al. Short sleep duration as a risk factor for hypertension: analyses of the first National Health and Nutrition Examination Survey. Hypertension. 2006;47(5):833–839. doi:10.1161/01.HYP.0000217362.34748.e0
22. Manohar S, Thongprayoon C, Cheungpasitporn W, Mao MA, Herrmann SM. Associations of rotational shift work and night shift status with hypertension: a systematic review and meta-analysis. J Hypertens. 2017;35(10):1929–1937. doi:10.1097/HJH.0000000000001442
23. Mancia G, Kreutz R, Brunström M, et al. 2023 ESH Guidelines for the management of arterial hypertension. J Hypertens. 2023;41(12):1874–2071. doi:10.1097/HJH.0000000000003480
24. Cuspidi C, Sala C, Tadic M, Rescaldani M, Grassi G, Mancia G. Untreated masked hypertension and subclinical cardiac damage: a systematic review and meta-analysis. Am J Hypertens. 2015;28(6):806–813. doi:10.1093/ajh/hpu231