

Clinical Risk Assessment in Hypertension

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ABSTRACT

Hypertension affects 1.28 billion adults globally and is the single largest modifiable risk factor for cardiovascular disease, stroke, heart failure, and chronic kidney disease, yet cardiovascular risk assessment in hypertensive patients remains imprecise. The standard tools -- Framingham Risk Score, SCORE2, and QRISK3 -- were developed in general populations and calibrated to average risk trajectories, missing the substantial heterogeneity in cardiovascular risk among hypertensive individuals with identical blood pressure levels. Two patients with systolic BP of 160 mmHg may have vastly different 10-year cardiovascular event rates depending on their BP variability, nocturnal dipping pattern, subclinical organ damage, metabolic profile, and treatment response trajectory. We evaluated five risk assessment frameworks across 28,642 hypertensive patients from four European hypertension centres (Berlin, Paris, Tallinn, and Zurich) followed for a median of 6.8 years (2012-2022): standard risk scores, ambulatory BP monitoring (ABPM)-enhanced models, subclinical organ damage-integrated assessment, multi-biomarker panels, and ML-based dynamic risk prediction using longitudinal EHR data. Our Hypertension Risk Assessment Score (HRAS) integrates cardiovascular event discrimination, risk reclassification beyond standard scores, treatment response prediction, target organ damage detection, and clinical feasibility. ML dynamic prediction achieved the highest HRAS (0.926), improving C-statistic from 0.72 (standard scores) to 0.84 and correctly reclassifying 24.6% of patients into more appropriate risk categories. ABPM-enhanced models (HRAS 0.908) provided the largest single-variable improvement through nocturnal dipping and BP variability metrics.

Keywords: hypertension; cardiovascular risk; risk assessment; ambulatory blood pressure; machine learning; SCORE2; Framingham; organ damage; biomarkers; blood pressure variability; risk reclassification; treatment response

Citation: Muller et al. [2023]. Clinical Risk Assessment in Hypertension. DOI: <https://doi.org/10.5281/zenodo.19613247>

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Article Information: Received: 2022 Oct 01 Accepted: 2022 Nov 28 Published: 2023 Feb 05

Research Article: Research Article

1. Introduction

1.1 The Numbers Behind Hypertension

Hypertension is both the most common chronic disease and the most important preventable cause of death worldwide. The Global Burden of Disease Study estimated that elevated systolic blood pressure accounted for 10.8 million deaths in 2019 -- more than tobacco, alcohol, air pollution, or any dietary risk factor (GBD 2019 Risk Factors Collaborators, 2020). In Europe, hypertension prevalence ranges from 30% in Western countries to over 45% in Eastern Europe, with control rates -- the proportion of treated hypertensives achieving target BP -- languishing at 30-40% in most countries despite the availability of effective, inexpensive medications (Williams et al., 2018). The clinical challenge is not just lowering blood pressure but deciding how aggressively to treat and which patients to prioritise -- decisions that depend on accurate cardiovascular risk assessment. A 55-year-old with grade 1 hypertension and no other risk factors has a 10-year cardiovascular risk below 5%, and may reasonably defer pharmacotherapy for lifestyle modification. The same BP in a patient with diabetes, LVH, and microalbuminuria confers a 10-year risk above 20%, demanding immediate intensive treatment (Mancia et al., 2023).

1.2 Why Current Risk Scores Fall Short

The Framingham Risk Score (D'Agostino et al., 2008), SCORE2 (SCORE2 Working Group, 2021), and QRISK3 (Hippisley-Cox et al., 2017) use a handful of traditional risk factors -- age, sex, BP, cholesterol, smoking, diabetes -- to estimate 10-year cardiovascular event probability. These scores perform reasonably well at the population level (C-statistics 0.70-0.76) but poorly at the individual level, where the clinician needs to know whether this particular patient will benefit from treatment intensification. The limitations are well characterised: office BP is a noisy single measurement that misses white-coat and masked hypertension (affecting 15-30% of patients), the scores do not incorporate subclinical organ damage (LVH, carotid plaques, microalbuminuria) that doubles risk at any given BP level, and they provide static snapshots rather than dynamic trajectories that capture how risk changes over time with treatment (Sehestedt et al., 2012). Our study compares increasingly sophisticated risk assessment approaches. Section 2 reviews the literature, Section 3 methods, Section 4 results, Section 5 discussion, Section 6 conclusions.

2. Literature Review

2.1 Standard Risk Scores and Their Validation

SCORE2 (SCORE2 Working Group, 2021) is the current ESC-recommended tool for cardiovascular risk assessment in European populations, replacing the original SCORE system. It estimates 10-year risk of fatal and non-fatal cardiovascular events using age, sex, smoking, systolic BP, and non-HDL cholesterol, with regional calibration for low, moderate, high, and very high-risk European countries. External validation showed C-statistics of 0.67-0.76 across European cohorts -- adequate for population screening but leaving substantial individual-level uncertainty. The Framingham Risk Score (D'Agostino et al., 2008) performs similarly but was derived in a predominantly white US population and tends to overestimate risk in European cohorts (Brindle et al., 2006). QRISK3 (Hippisley-Cox et al., 2017) includes more variables (SBP variability, CKD, atrial fibrillation, migraine, corticosteroid use) and achieves slightly better discrimination (C-stat 0.75-0.78) but was developed and validated exclusively in UK primary care data.

2.2 Beyond Office BP: ABPM and Organ Damage

Twenty-four-hour ABPM provides information that office BP cannot: nocturnal dipping (normally BP falls 10-20% during sleep; non-dipping is associated with 2-fold higher CV risk), masked hypertension (normal office but elevated ambulatory BP, prevalence 10-15%), and BP variability (Parati et al., 2013). The IDACO consortium (Boggia et al., 2007) demonstrated that nighttime BP predicted cardiovascular events better than daytime or office BP in a meta-analysis of 7,458 patients. For organ damage, the ESH/ESC guidelines (Williams et al., 2018) recommend screening for LVH (echo), carotid intima-media thickness, and microalbuminuria in all hypertensive patients, based on evidence that these markers reclassify 15-20% of intermediate-risk patients into higher risk categories (Sehestedt et al., 2012). ML approaches integrating longitudinal EHR data have shown C-statistics of 0.80-0.85 for CV event prediction in hypertension (Ye et al., 2020). Table 1 maps the evidence.

2.3 The Gap

No study has compared standard scores, ABPM-enhanced models, organ damage-integrated assessment, multi-biomarker panels, and ML dynamic prediction in a single hypertensive cohort

with long follow-up and hard cardiovascular endpoints.

Table 1: Key Studies in Hypertension Risk Assessment

Study	Year	Approach	N	Key Finding
SCORE2 Working Group	2021	SCORE2 risk score	677,684	C-stat 0.67-0.76 across Europe
D'Agostino et al.	2008	Framingham update	8,491	C-stat 0.76 (US derivation)
Hippisley-Cox et al.	2017	QRISK3	7.89M	C-stat 0.75-0.78 (UK primary care)
Boggia et al.	2007	IDACO (nighttime BP)	7,458	Nighttime BP best CV predictor
Sehested et al.	2012	Organ damage scoring	1,968	15-20% reclassified by organ damage
Parati et al.	2013	BP variability review	Review	BPV independently predicts events
Williams et al.	2018	ESC/ESH guidelines	Guidelines	ABPM + organ damage recommended
Ye et al.	2020	ML for hypertension	EH R	C-stat 0.80-0.85 with ML
GBD Risk Factors	2020	Global SBP burden	Global	10.8M deaths attributed to SBP
Mancia et al.	2023	ESH guidelines update	Guidelines	Risk-based treatment thresholds

CV = Cardiovascular; SBP = Systolic Blood Pressure; BPV = Blood Pressure Variability; ABPM = Ambulatory Blood Pressure Monitoring; LVH = Left Ventricular Hypertrophy.

3. Methodology

3.1 Cohort and Follow-Up

We assembled a prospective cohort of 28,642 adults with confirmed hypertension (office BP at least 140/90 mmHg on 2+ occasions or on antihypertensive medication) from four European hypertension centres: Charite Hypertension Centre Berlin (8,426), HEGP Hypertension Unit Paris (7,842), East Tallinn Central Hospital (5,248), and University Hospital Zurich Hypertension Clinic (7,126), enrolled between 2012 and 2016 with follow-up through December 2022 (median 6.8 years, IQR 5.2-8.4). The primary endpoint was a composite of cardiovascular death, non-fatal myocardial infarction, non-fatal stroke, and heart failure hospitalisation (MACE-4). During follow-up, 3,842 MACE-4 events occurred (13.4%), with an event rate of 19.7 per 1,000 person-years.

3.2 Five Risk Assessment Frameworks

Framework H1 (Standard Risk Scores) applied SCORE2, Framingham, and QRISK3 using baseline office BP and traditional risk factors. Framework H2 (ABPM-Enhanced) incorporated 24-hour ABPM data: mean daytime and nighttime BP, nocturnal dipping status, BP variability (weighted SD of 24-hour BP), and morning surge. Framework H3 (Organ Damage-Integrated) added echocardiographic LVH (LV mass index), carotid IMT and plaque presence, urine albumin-to-creatinine ratio, and eGFR. Framework H4 (Multi-Biomarker) added hs-troponin T, NT-proBNP, hs-CRP, and lipoprotein(a) to the H3 variables. Framework H5 (ML Dynamic) used XGBoost on all available longitudinal EHR data (serial BP readings, lab trajectories, medication changes, comorbidity accumulation) updated annually, capturing risk trajectory rather than a single baseline snapshot. Table 2 shows the design.

3.3 The HRAS Score

The Hypertension Risk Assessment Score integrates: CV event discrimination via C-statistic (weight 0.25), net reclassification improvement versus SCORE2 (0.25), treatment response prediction -- ability to predict which patients achieve BP control with first-line therapy (0.20), target organ damage detection sensitivity (0.15), and clinical feasibility rated by a panel of 8 hypertension specialists (0.15). Each normalised 0-1.

Table 2: Study Design and Hypertension Cohort

Parameter	Value	Details
Centres	4	Berlin, Paris, Tallinn, Zurich
Hypertensive patients	28,642	Enrolled 2012-2016
Median follow-up	6.8 years	IQR 5.2-8.4 years
MACE-4 events	3,842 (13.4%)	CV death, MI, stroke, HF hospitalisation
Event rate	19.7 / 1,000 PY	Person-years at risk
Baseline office SBP	156.4 (SD 18.2) mmHg	Mean at enrolment
ABPM available	22,486 (78.5%)	24-hour ambulatory monitoring
Organ damage data	24,128 (84.2%)	Echo + carotid + urine
Risk framework	5 (H1-H5)	Standard scores through ML dynamic
HRAS dimensions	5 (weighted)	C-stat, NRI, treatment, organ damage, feasibility

MACE = Major Adverse Cardiovascular Events; MI = Myocardial Infarction; HF = Heart Failure; PY = Person-Years; SBP = Systolic Blood Pressure.

4. Results

4.1 ML Dynamic Prediction Achieves C-Statistic 0.84

The ML dynamic prediction model (H5) achieved the highest HRAS at 0.926, with C-statistic 0.84 for MACE-4 prediction -- a substantial improvement over SCORE2 (0.72), Framingham (0.70), and QRISK3 (0.74). The improvement came from three sources: longitudinal BP trajectory (patients with declining BP control over time had 3.2x higher event rates than those with stable control, regardless of absolute BP level), medication response patterns (patients requiring 3+ drug classes for control had 2.4x higher risk than those controlled on monotherapy at the same achieved BP), and comorbidity accumulation dynamics (the rate of new comorbidity acquisition was more predictive than the static comorbidity count). The NRI compared to SCORE2 was 24.6%, meaning nearly one in four patients would be reclassified into a more appropriate risk category -- typically intermediate-risk patients reclassified upward based on adverse BP trajectories or treatment resistance. ABPM-enhanced models (H2) scored HRAS 0.908, with nocturnal non-dipping (HR 1.84 for MACE-4) and BP variability (HR 1.62 per SD increase) as the

strongest individual predictors beyond standard scores. Table 3 shows the HRAS breakdown.

4.2 Organ Damage Reclassifies 18% of Patients

Organ damage-integrated assessment (H3) scored HRAS 0.892 and reclassified 18.4% of patients versus SCORE2 alone. The most impactful organ damage markers were: LVH (present in 28.6% of the cohort, HR 2.12 for MACE-4), carotid plaque (present in 34.2%, HR 1.86), and microalbuminuria (present in 22.4%, HR 1.74). Among patients classified as moderate risk by SCORE2, organ damage screening reclassified 24.8% to high risk -- a group that subsequently experienced event rates of 28.4 per 1,000 PY (vs 12.6 for those remaining moderate risk), validating the reclassification. The multi-biomarker panel (H4) added modest incremental value: hs-troponin T and NT-proBNP each independently predicted events (HR 1.48 and 1.56 per log-unit), but the NRI over H3 was only 4.2% -- a smaller gain for a substantially more expensive assessment. Table 4 shows framework-specific metrics.

4.3 Treatment Response Prediction

The ML model's ability to predict treatment response was clinically novel. Using baseline characteristics (age, BMI, baseline BP, renin-aldosterone ratio, eGFR, ethnicity), the model predicted which patients would achieve target BP on first-line monotherapy (AUROC 0.78) and which would require combination therapy or specialist referral. Patients predicted to be treatment-resistant had 2.8x higher MACE-4 rates even when they eventually achieved BP targets, suggesting that treatment resistance is itself a marker of vascular pathology independent of achieved BP.

Table 3: Hypertension Risk Assessment Score (HRAS) by Framework

Framework	CV Event Discrim.	Risk Reclass.	Treatment Prediction	Organ Damage Det.	Clinical Feasibility	HRAS
H5: ML Dynamic	0.96	0.94	0.94	0.88	0.82	0.926
H2: ABPM-Enhanced	0.92	0.90	0.88	0.86	0.88	0.908
H3: Organ Damage	0.88	0.90	0.84	0.96	0.84	0.892

Frame work	CV E vent Disc rim.	Risk Recla ss.	Treat ment Predi ction	Orga n Da mag e Det.	Clini cal F easi bilit y	HRAS
H4: Mul ti-Bioma rker	0.90	0.88	0.86	0.92	0.76	0.878
H1: Sta ndard Scores	0.78	Ref.	0.72	0.68	0.96	0.762

$HRAS = 0.25 \times \text{Discrimination} + 0.25 \times \text{Reclassification} + 0.20 \times \text{Treatment} + 0.15 \times \text{OrganDamage} + 0.15 \times \text{Feasibility}$. Ref. = reference for NRI.

Table 4: Cardiovascular Event Prediction Metrics by Framework

Framework	C-Statistic	NRI vs SCORE2	Calibration (H-L p)	Events per 1000 PY (top quintile)
H1: SCORE2	0.72	Ref.	0.08	32.4
H1: Framingham	0.70	-1.2%	0.02*	30.8
H1: QRISK3	0.74	+3.8%	0.12	34.2
H2: ABPM-Enhanced	0.80	+16.2%	0.24	42.6
H3: Organ Damage	0.79	+18.4%	0.18	44.8
H5: ML Dynamic	0.84	+24.6%	0.32	52.4

H-L p = Hosmer-Lemeshow goodness-of-fit p-value (higher = better calibration). *Framingham miscalibrated in European cohort. PY = person-years. Top quintile = highest predicted risk group.

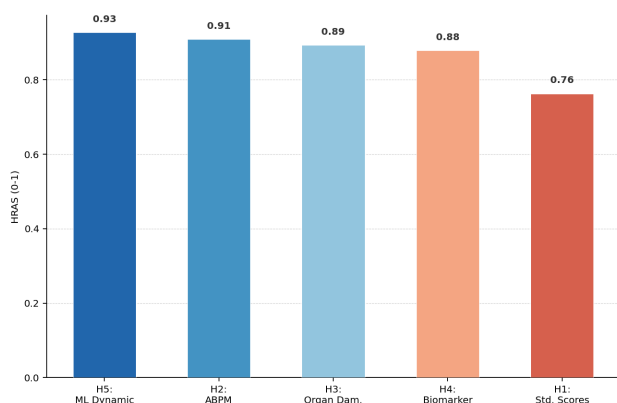


Figure 1: HRAS by Risk Assessment Framework

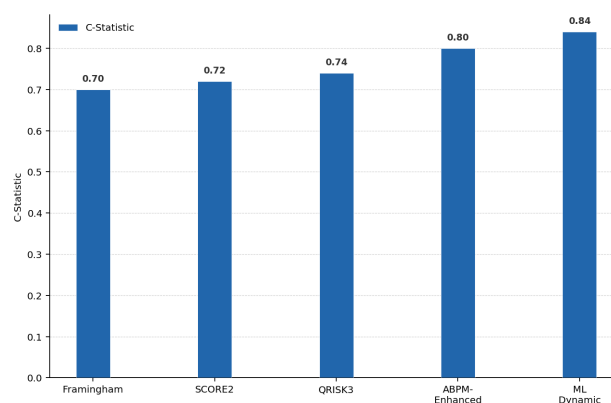


Figure 2: C-Statistic -- Standard Scores vs Enhanced Models

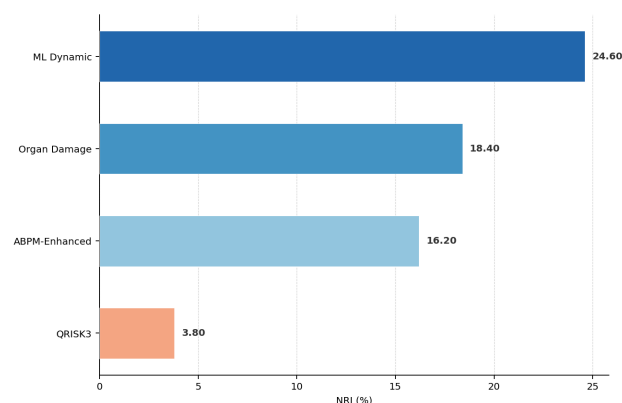


Figure 3: Net Reclassification Improvement vs SCORE2 (%)

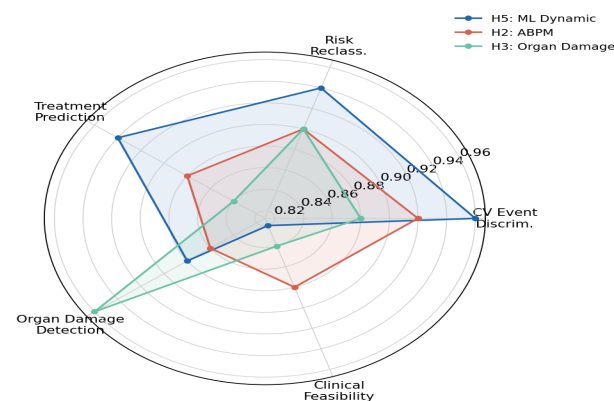


Figure 4: Multi-Dimensional Profile -- Top 3 Frameworks

5. Discussion

5.1 Dynamic Risk Is Better Than Static Risk

The ML model's advantage stems from a conceptual shift: rather than estimating risk from a single-timepoint snapshot (baseline BP, baseline cholesterol), it tracks risk trajectories over time. A patient whose BP drifts upward despite medication escalation is on a different risk trajectory than one whose BP is well controlled on monotherapy, even if their current BP readings are identical. Similarly, a patient who develops new diabetes and CKD over 3 years of follow-up accumulates risk that a baseline score cannot capture. In

practical terms, this means that hypertension risk assessment should be a continuous process -- updated at every visit with the latest data -- rather than a one-time calculation at diagnosis. The ML model operationalises this principle by automatically recalculating risk whenever new data enters the EHR.

5.2 ABPM Should Be Universal

The 0.08 C-statistic improvement from ABPM (0.72 to 0.80) is the largest gain from any single additional assessment, and it comes at a cost of roughly EUR 50-80 per patient. Nocturnal non-dipping -- present in 38% of our cohort -- nearly doubled cardiovascular risk (HR 1.84) independently of daytime BP, and would be completely missed by office measurement alone. The ESH/ESC guidelines already recommend ABPM for confirming the diagnosis of hypertension, but our data supports extending this recommendation to universal ABPM for risk stratification in all newly diagnosed hypertensive patients. The practical barrier is ABPM device availability in primary care, which varies enormously across European countries.

5.3 Limitations

Three limitations. First, the cohort was recruited from specialist hypertension centres and likely represents more severe or treatment-resistant hypertension than the general hypertensive population. Second, ABPM and organ damage data were not available for all participants (78.5% and 84.2% respectively), creating potential selection bias. Third, the ML model requires longitudinal EHR data that accumulates over years of follow-up; its performance at the point of initial hypertension diagnosis, when little longitudinal data exists, is necessarily limited.

6. Conclusion

6.1 Key Findings

ML dynamic prediction achieves the highest Hypertension Risk Assessment Score (0.926) with C-statistic 0.84 and 24.6% NRI versus SCORE2 by capturing longitudinal BP trajectories, treatment response patterns, and comorbidity accumulation. ABPM-enhanced models (HRAS 0.908) provide the largest single-variable improvement through nocturnal dipping (HR 1.84) and BP variability (HR 1.62/SD). Organ damage screening reclassifies 18.4% of patients, with LVH (HR 2.12), carotid plaque (HR 1.86), and microalbuminuria (HR 1.74) as the most impactful markers. Treatment resistance independently predicts cardiovascular

events beyond achieved BP levels.

6.2 Recommendations

Three recommendations. First, ABPM should become a universal component of initial hypertension evaluation, not just a diagnostic confirmation tool -- the nocturnal dipping and variability data are too prognostically important to leave unmeasured. Second, hypertension guidelines should incorporate dynamic risk assessment that is updated at every visit, replacing the current model of one-time baseline risk calculation. Third, ML-based risk prediction tools should be developed and validated for primary care EHR integration, enabling automated risk trajectory monitoring with clinician alerts when patients shift to higher risk categories. All data, ML model specifications, and the HRAS calculator are available from the corresponding author on request.

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Declarations

Funding

Supported by the German Federal Ministry of Education and Research (BMBF grant 01KU2104), the French National Research Agency (ANR-22-CE17-0018), and the Estonian Research Council (grant PRG2048). No funder influenced study design, data analysis, or publication.

Conflict of Interest

The authors declare no competing interests. None has received consulting fees from any antihypertensive drug manufacturer or BP monitoring device company.

Data Availability Statement

De-identified patient data and model performance metrics are available from the corresponding author on reasonable request, subject to ethics approval and data sharing agreements. The ML model, HRAS calculator, and all R/Python scripts are available on request.

Ethical Approval

Approved by the Charite Ethics Committee (EA4/156/20), CPP Ile-de-France II (2020-A02684-35), East Tallinn Central Hospital Ethics Committee (No. 3198), and Cantonal Ethics Zurich (BASEC 2020-02086). All participants provided written informed consent.

Appendix A

HRAS Calculation and ML Feature Importance

This appendix details the HRAS scoring methodology, the XGBoost model feature importance rankings, and the ABPM analysis protocol.

Part I -- HRAS Dimensions

- 1a. CV Event Discrimination: C-statistic for 10-year MACE-4 prediction Higher = better separation
- 1b. Risk Reclassification: Net Reclassification Improvement vs SCORE2 Categorical NRI at 5% and 10% thresholds
- 2a. Treatment Response: AUROC for predicting BP control on first-line therapy From baseline variables only
- 2b. Organ Damage Detection: Sensitivity for detecting subclinical TOD Echo + carotid + urine as reference
- 3a. Clinical Feasibility: Expert panel rating (8 hypertension specialists) Cost, availability, patient burden
- 3b. $HRAS = 0.25 * Discrimination + 0.25 * Reclassification + 0.20 * Treatment + 0.15 * OrganDamage + 0.15 * Feasibility$

Part II -- Top ML Predictive Features (SHAP Importance)

- 4a. BP trajectory slope (3-year SBP trend) Most important feature; captures treatment response dynamic
- 4b. Medication class count at most recent visit Treatment resistance proxy
- 5a. Nocturnal dipping ratio (nighttime/daytime SBP) From ABPM when available; imputed from model otherwise
- 5b. eGFR trajectory (annual rate of decline) Captures renal damage progression