

Hypertension & Atrial Fibrillation

PREVENT • DETECT • PROTECT

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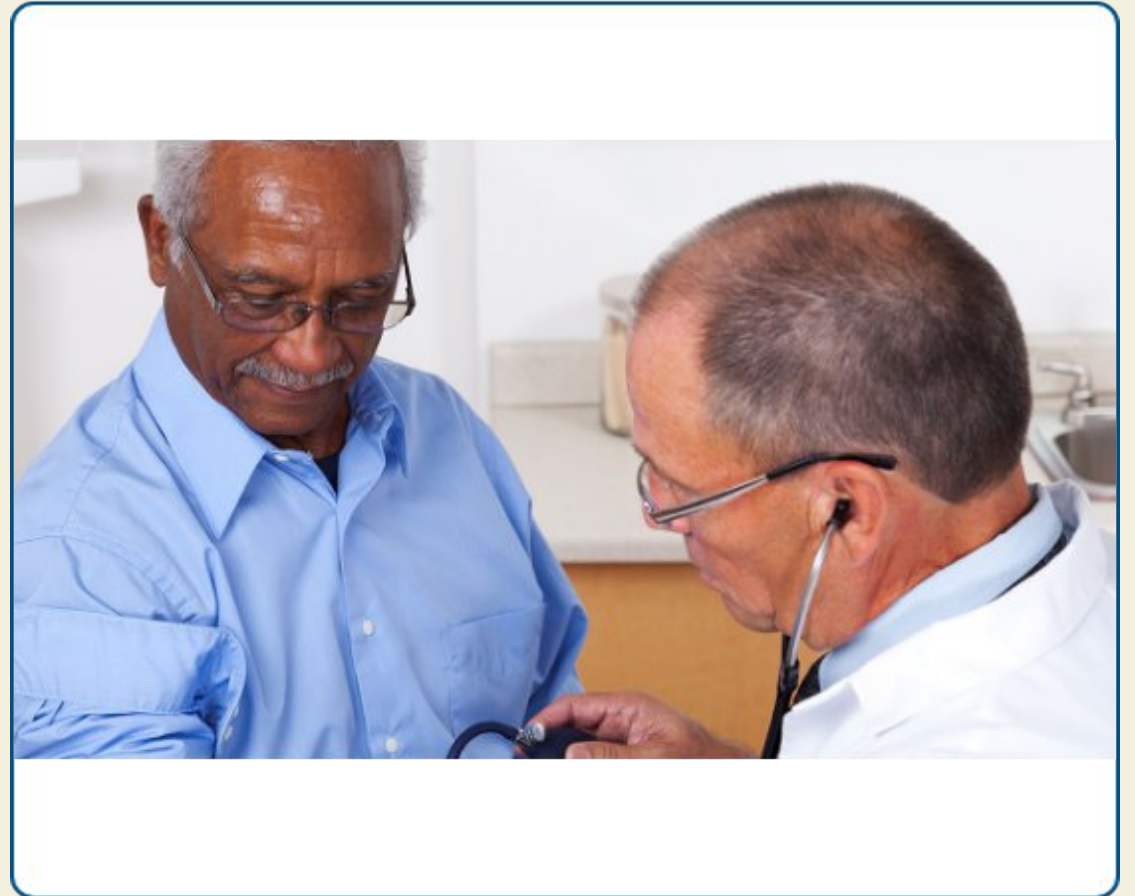
A Familiar Hypertension Consultation

Patient Profile:

- 68-year-old male with a history of essential hypertension.
- Asymptomatic; absolutely no complaints of palpitations or chest pain.
- Office Blood Pressure: **156/92 mmHg**.
- Clinical finding: **Irregularly irregular pulse** noted on physical exam.

The Critical Question:

What diagnostic and therapeutic milestones must happen before this patient leaves the clinic today?



Hypertension Remodels the Atrium



Pressure Load

Chronic systemic hypertension increases LV afterload, leading to diastolic dysfunction. This translates directly to elevated left atrial pressures and mechanical stretch.



Atrial Disease

Sustained mechanical stress induces profound structural changes. Fibroblast proliferation causes extensive atrial fibrosis, electrical uncoupling, and atrial enlargement.



Atrial Fibrillation

The highly fibrotic, dilated atrium creates the perfect substrate for macro-reentrant circuits. Triggers (often pulmonary vein ectopy) can now easily initiate and sustain AF.

SPRINT: Intensive BP Control Lowers Incident AF



*Intensive blood pressure management (SBP <120) yields a staggering **26% lower** hazard of new-onset atrial fibrillation.
(95% CI 0.56–0.98)*

BP Targets Depend on Good Measurement

ESC 2024 Guidelines

The aggressive treatment target for systolic blood pressure is now established at **120–129 mmHg** for most patients on treatment, provided it is well tolerated.

Achieving this narrow therapeutic window requires absolute precision. Estimations or casual readings will result in either inadequate protection or iatrogenic hypotension.

Clinical Validation

Single, hurried office readings are no longer acceptable for guiding intensive therapy.

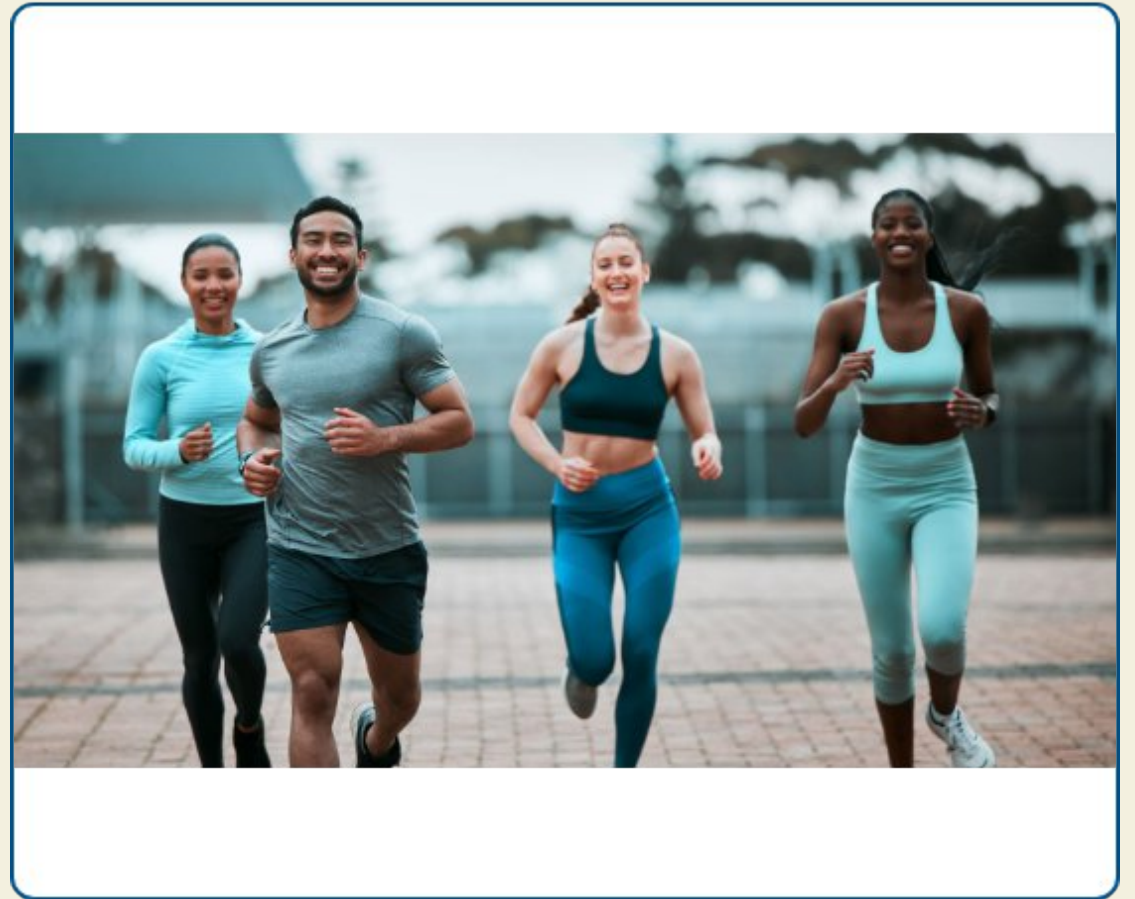
Diagnostic accuracy relies entirely on **repeated standardized office readings** combined with mandatory out-of-office confirmation (Ambulatory or Home BP Monitoring).

The Wider AF Substrate: Beyond BP

Optimizing the substrate improves outcomes (ARREST-AF 2025):

- **Weight Management:** Obesity induces pericardial adiposity and systemic inflammation, directly promoting atrial myopathy.
- **Physical Fitness:** Structured cardiorespiratory fitness programs improve endothelial function and autonomic balance.
- **Sleep Apnoea:** Obstructive sleep apnea causes intermittent hypoxia and negative intrathoracic pressure swings, mechanically stretching the atrium.

Aggressive risk factor modification significantly increases **freedom from AF at 12 months** following catheter ablation.



Detection Begins During Routine Care



For all patients aged ≥ 65 years, rhythm assessment should be a mandatory component of every clinical contact, regardless of the presenting complaint.



Simple pulse palpation is highly sensitive. Any detection of an irregular pulse or patient-reported symptoms (palpitations, unexplained fatigue) demands immediate investigation.



An irregular pulse is a clinical sign, not a diagnosis. A standard 12-lead ECG is legally and medically required to confirm the presence of Atrial Fibrillation.



In patients with high-risk profiles (e.g., severe hypertension, structural heart disease) but a normal ECG, consider extended monitoring (Holter, patch devices) to capture paroxysmal episodes.

Screening Alerts Need ECG Confirmation

100%

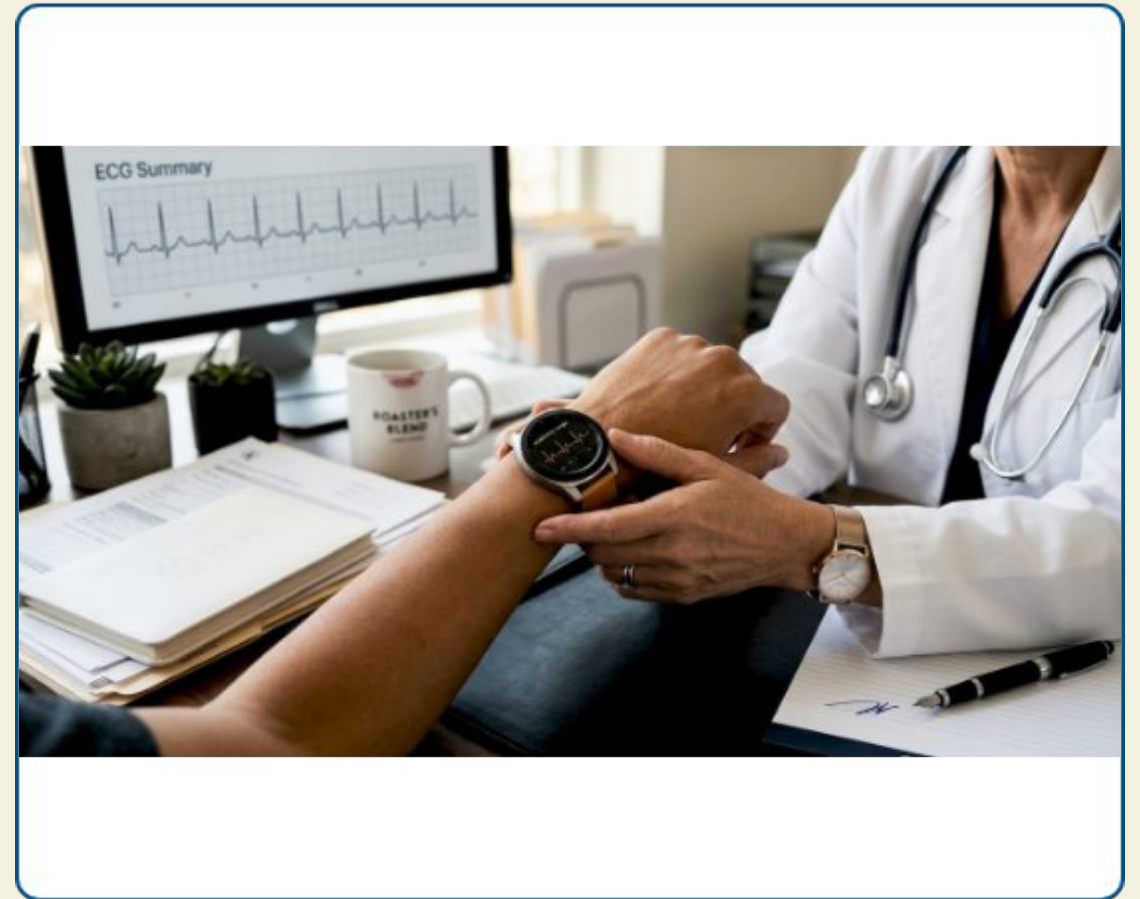
Require Clinical Review

A smart-watch notification or an automated blood pressure cuff warning is a screening alert, **not a clinical diagnosis**.

The Pathway:

Device Alert → Physician ECG Review → Formal AF Diagnosis

Prematurely initiating powerful anticoagulants based solely on a consumer device alert exposes the patient to severe bleeding risks without confirmed benefit.



Clinical AF: ESC CHA₂DS₂-VA Thresholds

Score	Recommendation	Clinical Rationale
0	Reassess as risk evolves	The stroke risk is currently too low to justify the inherent bleeding risk of OAC therapy. Monitor annually.
1	Consider anticoagulation	A nuanced discussion is required. Weigh patient preference, specific risk factors (e.g., age 65-74 vs hypertension), and bleeding hazards.
≥ 2	Anticoagulation Recommended	The stroke risk clearly outweighs bleeding risks. OAC therapy is indicated to prevent severe ischemic events.

Note: In the newly revised CHA₂DS₂-VA score, gender has been removed as an independent risk factor.

Anticoagulant Choice and Safety

- **First-Line Therapy:** Direct Oral Anticoagulants (DOACs) are standard of care for eligible patients due to superior safety profiles (less intracranial hemorrhage).
- **VKA Exceptions:** Vitamin K Antagonists (Warfarin) remain mandatory for mechanical prosthetic valves or moderate-to-severe mitral stenosis.
- **Prescribing Safety:** Ensure the correct dose based on age, weight, and a recent renal review (CrCl). Confirm reliable patient access to the medication.
- **Minimizing Bleeding:** Safer anticoagulation requires strict BP control (avoiding SBP >160), reviewing concomitant drugs (NSAIDs, antiplatelets), and regular follow-up.

ARTESiA: Subclinical AF (Risk vs. Reward)

Comparing Apixaban vs Aspirin per 1,000 patient-years

The Benefit

4.6

Fewer Strokes / Emboli

Anticoagulation successfully reduces the incidence of ischemic events in subclinical AF.

The Harm

7.7

More Major Bleeds

However, it causes more major bleeding events than it prevents strokes.

Conclusion: Brief, device-detected AF needs meticulous patient selection based on AF burden, baseline stroke risk, and bleeding risk.

EAST-AFNET 4: Early Rhythm Control



*Implementing early rhythm control (AF diagnosed ≤ 1 year) in patients with cardiovascular conditions yields a **Hazard Ratio of 0.79** for CV death, stroke, HF, or ACS hospitalisation.*

Our Patient Returns



Diagnosis Confirmed

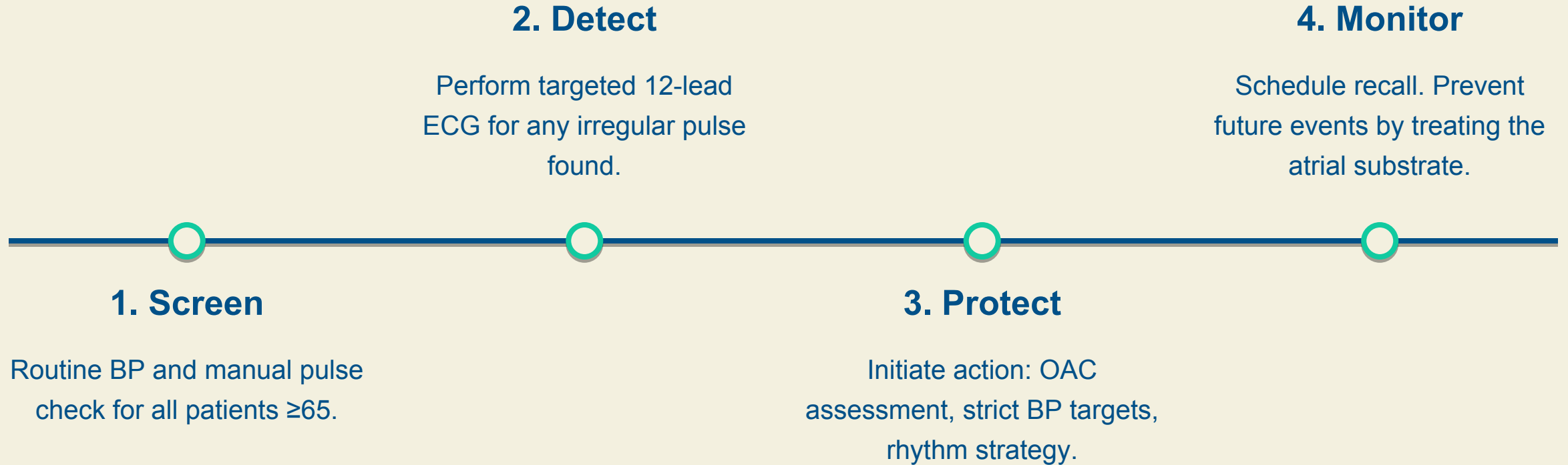
The 12-lead ECG confirmed clinical Atrial Fibrillation.

Age 68 + Hypertension = CHA₂DS₂-VA Score 2

Immediate Action Plan For Today:

- **Anticoagulation assessment:** Discuss DOAC initiation.
- **BP Optimisation:** Target SBP 120-129 mmHg to protect the atrium and reduce intracranial bleeding risk.
- **Early Rhythm Review:** Refer for evaluation of rhythm control strategy (EAST-AFNET 4).

The Hypertension Clinic Pathway



Discussion: Antihypertensive Selection

-  Standard therapy should be an ACE inhibitor or ARB combined with a Calcium Channel Blocker (CCB) or a thiazide-like diuretic. These offer excellent baseline control and vascular protection.
-  Beta-blockers should NOT be used as primary antihypertensives unless there is an absolute, additional compelling indication (e.g., Heart Failure with reduced Ejection Fraction, or recent Myocardial Infarction).
-  When choosing a drug for AF rate control, you must absolutely account for Left Ventricular Ejection Fraction (LVEF). Non-dihydropyridine CCBs (Verapamil, Diltiazem) are strictly contraindicated in patients with HFrEF.

Discussion: US Device-Episode Thresholds

Guidance for subclinical, device-detected AF episodes:

Episode Duration	Risk (CHADS VASc)	Recommendation
≥ 24 Hours	≥ 2	Oral Anticoagulation is reasonable . The risk profile mirrors clinical AF.
5 Minutes – < 24 Hours	≥ 3	Oral Anticoagulation may be reasonable , requiring shared decision making and weighing bleeding risks.
< 5 Minutes	Any Score	No OAC indicated for the episode alone. Monitor for progression to longer episodes.

Discussion: Keeping the Evidence Separate



SPRINT

Incident AF Prevention

Applies to patients without AF.
Demonstrates that intensive BP control stops the disease process before it starts.



ARREST-AF

Ablation Recurrence

Applies to patients undergoing invasive rhythm control. Proves that modifying the substrate protects the ablation investment.



ARTESiA

Subclinical AF & Stroke

Applies only to stroke-detected, asymptomatic cohorts. Highlights the delicate balance of preventing stroke vs causing major bleeds.

Thank You

Questions?

Pakistan Hypertension League 2026