

AI-03407 DDx Exertional Dyspnea

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A 59 yom with a remote history of MI in 2008 and restenting in 2010, active cyclist since 1999 until present, long distance swimmer as well and weight trains at the gym. Currently healthy and very active. Medications includes, Atorvastin 50mg, metoprolol 25mg, aspirin 81mg, Zestril 20mg. Patient presented with progressive fatigue, no chest pain, lightheadedness during exercise, heart rate 40 bmp during sleep and 120 bpm during last cycling workout and exertional dyspnea . He sleeps with his Garmin HR monitor and no changes from baseline. Perform a DDx. Build a diagnostic decision tree

Got it. This is a high-functioning 60-year-old endurance athlete with CAD history who now has new, progressive exercise intolerance. That combination matters a lot.

I'll approach this the way a cardiologist would: frame the problem → generate a prioritized DDx → flag dangerous vs likely → note what would separate them clinically.

Problem Representation (Key Features)

- 60-year-old male
- Remote MI (2008), resten t (2010) → known CAD
- Long-term endurance athlete (cycling, swimming, weight training)
- Meds: β -blocker, ACE-I, statin, aspirin
- New symptoms:

- Progressive fatigue
- Exertional dyspnea
- Lightheadedness during exercise
- No angina
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- HR data:
 - Sleep HR ~40 bpm (chronic?)
 - Peak exercise HR ~120 bpm (possibly blunted)
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- Garmin data reportedly unchanged from baseline

This is NOT a benign athlete complaint until proven otherwise.

High-Priority / Must-Rule-Out Diagnoses

1.

Chronotropic Incompetence

(Very high on the list)

Why it fits

- Inability to appropriately increase HR with exertion
- Peak HR of ~120 bpm in a 60-year-old athlete may be inadequate for workload
- Causes:
 - β -blocker effect (metoprolol)
 - Sinus node dysfunction
 - Autonomic dysfunction
 - Ischemia involving SA node

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Symptoms explained

- Fatigue
- Lightheadedness
- Dyspnea with exertion
- “Normal” resting HR trends but functional limitation

Key clue

Patient says Garmin shows no change — that’s exactly what patients with chronotropic incompetence report.

2.

Sinus Node Dysfunction (Sick Sinus Syndrome)

(High concern given age + CAD)

Includes:

- Sinus bradycardia
- Sinus pauses
- SA exit block
- Chronotropic incompetence

Why it fits

- Sleeping HR 40 bpm (could be athletic, could be pathologic now)
- Progressive symptoms
- Lightheadedness during exertion
- β -blocker may be unmasking intrinsic SA disease

Important

Athletic bradycardia should not cause exertional symptoms.

3.

Silent Ischemia / Demand Ischemia

(Very important despite no chest pain)

Why it fits

- Known CAD
- Exertional dyspnea = anginal equivalent
- Fatigue and lightheadedness during exertion
- Endurance athletes often have ischemia without pain

Possible mechanisms:

- In-stent restenosis
- Progression of native disease
- Microvascular ischemia
- Ischemia-induced LV dysfunction

Absence of chest pain does NOT reassure here.

4.

Heart Failure with Preserved EF (HFpEF)

(Often missed in athletes)

Why it fits

- Exertional dyspnea
- Fatigue
- CAD + age + hypertension history (implied by ACE-I)
- Endurance training → diastolic stiffness possible

HFpEF often presents as:

- Normal resting vitals
- Normal daily activity tolerance
- Exercise intolerance only

Intermediate-Priority Diagnoses

5.

Medication-Related Exercise Limitation

(Contributing, not sole explanation)

- Metoprolol
 - Blunts HR response
 - Can worsen fatigue
 - Can unmask sinus node disease
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- ACE-I less likely
- Statin → fatigue/myopathy (rarely dyspnea/lightheadedness)

Medication alone is unlikely if symptoms are progressive after years of stability.

6.

High-Grade AV Conduction Disease

- Mobitz I vs II
- Intermittent AV block during exertion
- Especially relevant with CAD history

May not show on resting ECG.

7.

Atrial Arrhythmias with Slow Ventricular Response

- Paroxysmal AF/flutter
- Junctional rhythms
- Atrial standstill (rare)

Could cause:

- Reduced cardiac output
- Exercise intolerance without tachycardia

Lower-Probability but Worth Considering

8.

Pulmonary Causes

- Exercise-induced pulmonary hypertension
- Interstitial lung disease (unlikely given athlete history)
- Pulmonary embolism (unlikely without acute change)

9.

Systemic / Metabolic

- Anemia
- Hypothyroidism
- Electrolyte abnormalities
- Overtraining syndrome (diagnosis of exclusion)

These do not explain bradycardia + blunted HR response well.

Key Distinction: Athlete's Heart vs Pathology

Feature	Athlete's Adaptation	Pathologic
Resting HR	Low	Low
Exercise HR	Appropriately rises	Blunted
Symptoms	None	Fatigue, dizziness
Trend	Stable	Progressive
Functional capacity	Preserved	Declining

👉 Symptoms + decline = pathology until proven otherwise

Red Flags in This Case

- Progressive exertional symptoms
- Lightheadedness during exercise
- Known CAD

- Possible chronotropic incompetence
- Reliance on consumer HR data (can miss pauses/blocks)

Most Likely Working Diagnoses (Ranked)

1. Chronotropic incompetence (\pm sinus node dysfunction)
2. Silent myocardial ischemia
3. Medication-unmasked conduction disease
4. HFpEF / diastolic dysfunction
5. AV conduction abnormality

What Would Clinically Separate These (Conceptually)

- Exercise stress test with HR response
- Ambulatory ECG (Holter or patch)
- Echo with diastolic assessment
- Stress imaging if ischemia suspected

(I'm not giving orders — just the diagnostic logic.)

Bottom line

This presentation is not benign athletic bradycardia.

In a 60-year-old endurance athlete with CAD, new fatigue + exertional dyspnea + lightheadedness = cardiac until proven otherwise, with chronotropic incompetence and silent ischemia at the top of the differential.

If you want, I can:

- Build a diagnostic decision tree
- Map symptoms \rightarrow mechanisms
- Or walk through how cardiologists decide pacemaker vs med adjustment vs ischemic workup

Alright—let's do this cleanly and clinically.

Below is a stepwise diagnostic decision tree tailored to this exact patient profile: older endurance athlete, known CAD, β -blocker, new exertional symptoms, possible chronotropic limitation.

I'll structure it the way an attending would expect a fellow to reason it out.

Diagnostic Decision Tree

60-year-old endurance athlete with CAD + exertional fatigue, dyspnea, lightheadedness

STEP 1 — Is this hemodynamically or electrically dangerous

right now

?

1A. Red-flag screening

Ask / assess:

- Syncope or near-syncope?
- Palpitations with presyncope?
- New rest dyspnea?
- Chest discomfort (even atypical)?
- HR <35 bpm awake or pauses >3 sec?

If YES →

→ Urgent ECG + telemetry

→ Consider hospital monitoring

If NO →

→ Proceed with structured outpatient workup

STEP 2 — Is the problem primarily rate-related

?

This is the most important fork in this case.

2A. Does HR rise appropriately with exertion?

Test

- Exercise treadmill or bike test with HR response tracking
- Or supervised CPET if available

Interpretation

- Expected max HR (approx): ~160 bpm (age-adjusted, athlete may be slightly lower)
- Concerning:
 - Failure to reach $\geq 80\%$ predicted
 - Flat or sluggish HR rise
 - Early fatigue despite adequate effort
-

If HR response is inadequate

→ **RATE PATHWAY** ▼

2B. Is this medication-mediated or intrinsic?

Action

- Review β -blocker dose (metoprolol 25 mg)

- Temporarily hold or reduce only under supervision

Then reassess:

- HR response improves?
 - YES → Drug-related chronotropic limitation
 - NO → Intrinsic sinus node disease
-

2C. Ambulatory rhythm evaluation

Test

- 24–48 hr Holter or 7–14 day patch monitor

Look for

- Sinus pauses
- Daytime bradycardia
- Failure of HR acceleration
- Intermittent AV block

Findings

- Sinus pauses / CI → Sick sinus syndrome
- AV block → Conduction disease
- Normal rhythm → Continue down ischemic or mechanical path

STEP 3 — Is there

ischemia without pain

?

Dyspnea = anginal equivalent in CAD patients.

3A. Stress testing with imaging

Choose one

- Stress echo
- Nuclear perfusion
- CPET + imaging (ideal for athletes)

Look for

- New wall-motion abnormalities
- Reduced VO_2 max
- Early anaerobic threshold
- Ischemic ECG changes

Interpretation

Result	Meaning
Positive ischemia	In-stent restenosis / new CAD
Normal perfusion	Move to mechanical / diastolic causes
Low VO_2 + normal perfusion	Chronotropic or HFpEF

STEP 4 — Is cardiac output limited despite normal rhythm?

4A. Echocardiography (resting)

Assess

- LVEF
- LV hypertrophy
- Diastolic function (E/e' , LA size)
- RV function
- Pulmonary pressures

Findings

Echo finding	Diagnosis
Normal EF + ↑ filling pressures	HFpEF
New LV dysfunction	Ischemic cardiomyopathy
LVH + stiff ventricle	Athletic vs pathologic remodeling

STEP 5 — If cardiac workup is inconclusive

5A. Systemic contributors

Labs

- CBC (anemia)
- TSH
- CMP
- CK (statin myopathy)

5B. Pulmonary evaluation (only if cardiac excluded)

- PFTs
- Exercise O₂ saturation
- Pulmonary HTN assessment

FINAL SYNTHESIS NODE

Most likely outcomes in this patient:

1. Chronotropic incompetence ± sinus node dysfunction
 - Especially if HR plateaus early
 - Often leads to pacemaker discussion, not just med changes
- 2.
3. Silent ischemia
 - Particularly if stress imaging positive
 - Requires revascularization evaluation

- 4.
5. HFpEF / diastolic limitation
 - Common in aging endurance athletes with CAD
- 6.
7. Medication-unmasked conduction disease
 - β -blocker reveals underlying pathology
- 8.

Key Teaching Pearl

Athletic bradycardia is a diagnosis of asymptomatic excellence.

Symptoms convert it into pathology until proven otherwise.

If you want next:

- A one-page visual flowchart
- A “when to consider pacemaker” decision map
- Or a CPET interpretation guide specifically for endurance athletes