

AI-03513 Heart Murmurs

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What are types of heart murmurs. Explain the pathophysiology. Provide a description

Heart murmurs are **audible vibrations caused by turbulent blood flow** within the heart or great vessels. Turbulence occurs when blood moves at high velocity, passes through a narrowed opening, leaks backward, or flows abnormally between chambers.

They are classified by **timing, cause, and quality**.

I. Classification by Timing in the Cardiac Cycle

1. Systolic Murmurs

Occur between **S1 and S2** (ventricular contraction).

A. Ejection (Crescendo–Decrescendo) Murmurs

Caused by blood flowing through a narrowed outflow tract.

- **Aortic Stenosis**
- **Pulmonic Stenosis**

Pathophysiology:

Ventricular pressure rises → blood forced through narrowed semilunar valve → high-velocity jet → turbulence in ascending aorta or pulmonary artery.

Results in pressure overload → concentric hypertrophy.

Description:

Harsh, crescendo-decrescendo; often radiates (AS → carotids).

B. Holosystolic (Pansystolic) Murmurs

Uniform intensity throughout systole.

- **Mitral Regurgitation**
- **Tricuspid Regurgitation**
- **Ventricular Septal Defect**

Pathophysiology:

Ventricular pressure exceeds atrial pressure immediately after S1 → blood regurgitates continuously during systole → constant turbulence.

- MR → LV → LA backflow → volume overload → eccentric hypertrophy.
- VSD → LV → RV shunt (if high pressure gradient).

Description:

Blowing, plateau-shaped.

C. Late Systolic Murmur

- **Mitral Valve Prolapse**

Pathophysiology:

Redundant mitral leaflets bulge into LA mid-systole → chordae tension → regurgitant flow.

Description:

Mid-systolic click followed by murmur.

2. Diastolic Murmurs

Occur between **S2 and S1** (ventricular relaxation).

A. Early Diastolic (Decrescendo)

- **Aortic Regurgitation**
- **Pulmonic Regurgitation**

Pathophysiology:

High arterial pressure → blood flows back into ventricle during diastole → volume overload → LV dilation (AR).

Description:

Blowing, high-pitched, decrescendo.

B. Mid-to-Late Diastolic (Rumbling)

- **Mitral Stenosis**
- **Tricuspid Stenosis**

Pathophysiology:

Atrial pressure exceeds ventricular pressure → turbulent filling across narrowed AV valve.

Chronic pressure overload → atrial enlargement → atrial fibrillation risk.

Description:

Low-pitched rumble; opening snap (MS).

3. Continuous Murmurs

Occur throughout systole and diastole.

- **Patent Ductus Arteriosus**

Pathophysiology:

Persistent connection between aorta and pulmonary artery → continuous pressure gradient → continuous shunt flow.

Description:

“Machinery-like” murmur.

II. Functional (Physiologic) Murmurs

Innocent / Flow Murmurs

Seen in:

- Fever
- Anemia
- Pregnancy
- Hyperthyroidism

Pathophysiology:

Increased cardiac output → higher flow velocity through normal valves → turbulence without structural disease.

Soft, systolic, no radiation.

III. Pathophysiologic Mechanisms Behind Murmurs

All murmurs arise from one of four mechanisms:

1. Increased Flow Across Normal Valve

Example: anemia → reduced viscosity → higher velocity.

2. Forward Flow Through Narrowed Valve (Stenosis)

Pressure overload → hypertrophy.

3. Backward Flow Through Incompetent Valve (Regurgitation)

Volume overload → dilation.

4. Shunt Between Chambers

Pressure gradient → abnormal directional flow (VSD, PDA).

Hemodynamic Consequences

Lesion Type	Primary Load	Structural Change
Stenosis	Pressure overload	Concentric hypertrophy
Regurgitation	Volume overload	Eccentric hypertrophy
Shunt	Volume ± pressure	Chamber enlargement

Acoustic Characteristics Used in Description

Clinicians describe murmurs by:

1. Timing (systolic/diastolic/continuous)
2. Shape (crescendo, decrescendo, plateau)
3. Location (aortic, pulmonic, tricuspid, mitral areas)
4. Radiation (carotids, axilla)
5. Pitch (high vs low)
6. Intensity (Grade I–VI)

Physiologic Summary

Normal flow is **laminar** (Reynolds number low).

Murmurs occur when:

- Velocity increases
- Viscosity decreases
- Orifice narrows
- Pressure gradient rises

These increase Reynolds number → turbulence → vibration → audible murmur.

If you'd like, I can next:

- Map murmurs to pressure–volume loops
- Create a diagnostic flowchart
- Compare murmurs in athletes vs elderly
- Or explain murmurs from a cardiology board-review perspective

DDx hearth murmurs. What are early signs without a stethoscope? Fatigue or angina or dyspnea

Differential Diagnosis (DDx) of Heart Murmurs

A murmur is a **sign**, not a diagnosis. The DDx is organized by mechanism and clinical context.

I. Structural Valve Disease

Stenotic Lesions (pressure overload)

- **Aortic Stenosis**
- **Pulmonic Stenosis**
- **Mitral Stenosis**
- **Tricuspid Stenosis**

Mechanism: Obstructed forward flow → pressure gradient → hypertrophy → reduced cardiac output.

Regurgitant Lesions (volume overload)

- **Aortic Regurgitation**
- **Mitral Regurgitation**
- **Tricuspid Regurgitation**
- **Pulmonic Regurgitation**

Mechanism: Backflow → chamber dilation → progressive heart failure.

II. Congenital Shunts

- Ventricular Septal Defect
- Atrial Septal Defect
- Patent Ductus Arteriosus

Mechanism: Abnormal communication → turbulent shunting → volume overload.

III. Dynamic / Functional Outflow Obstruction

- Hypertrophic Cardiomyopathy

Mechanism: Thickened septum → dynamic LV outflow obstruction → ischemia, syncope.

IV. High-Output / Physiologic Murmurs

- Anemia
- Fever
- Pregnancy
- Hyperthyroidism

Mechanism: Increased velocity across normal valves → turbulence.

V. Secondary / Structural Non-Valvular Causes

- Papillary muscle rupture (post-MI)
- Infective endocarditis
- Rheumatic heart disease
- Aortic root dilation

Early Signs WITHOUT a Stethoscope

Before a murmur is heard, **hemodynamic consequences** often appear first. These are more important clinically than the sound itself.

1. Dyspnea (Most Common Early Symptom)

Mechanism:

- LV dysfunction → ↑ left atrial pressure
- Pulmonary venous congestion → interstitial edema
- Reduced lung compliance

Clinical Clues:

- Shortness of breath on exertion
- Orthopnea (needs more pillows)
- Paroxysmal nocturnal dyspnea

Common in:

- Mitral regurgitation
 - Mitral stenosis
 - Aortic regurgitation (late)
-

2. Fatigue / Exercise Intolerance

Mechanism:

- Reduced forward cardiac output
- Poor skeletal muscle perfusion
- Early anaerobic metabolism

Common in:

- Aortic stenosis

- Advanced regurgitation
- Cardiomyopathy

Often subtle and progressive.

3. Angina (Chest Pain Without CAD)

Mechanism:

- Increased myocardial oxygen demand (pressure overload)
- Reduced coronary perfusion pressure
- LV hypertrophy compresses microvasculature

Classically seen in:

- **Aortic Stenosis**
- **Hypertrophic Cardiomyopathy**

Important: Angina may occur even with normal coronary arteries.

4. Syncope / Presyncope

Mechanism:

- Fixed cardiac output
- Inability to increase flow during exertion
- Cerebral hypoperfusion

Red flag for:

- Severe aortic stenosis
 - Hypertrophic cardiomyopathy
-

5. Palpitations

Mechanism:

- Atrial enlargement → atrial fibrillation

- Ventricular dilation → ectopy

Seen in:

- Mitral stenosis
- Mitral regurgitation

6. Peripheral Signs (You Can See These)

Volume Overload (Regurgitation)

- Bounding pulse (wide pulse pressure in AR)
- Visible carotid pulsations
- Warm extremities

Pressure Overload (Stenosis)

- Slow-rising weak pulse (AS)
- Cool extremities
- Reduced exercise capacity

Symptom Patterns by Lesion

Symptom	Likely Mechanism	Common Lesions
Dyspnea	Pulmonary congestion	MR, MS
Angina	Pressure overload ischemia	AS, HCM
Syncope	Fixed output	AS, HCM
Fatigue	Low forward flow	Any severe lesion
Palpitations	Atrial dilation	MS, MR

What Appears FIRST?

Most common early order:

1. Exercise intolerance
2. Dyspnea on exertion
3. Fatigue
4. Then angina or syncope (more severe disease)

Many patients are **asymptomatic for years**, especially in regurgitant lesions due to compensatory dilation.

Red Flags (Urgent Evaluation Needed)

- New exertional syncope
 - Angina with known murmur
 - Rapid onset dyspnea
 - Leg swelling + orthopnea
 - Fever + new murmur (possible endocarditis)
-

Clinical Insight

The murmur is just the sound of turbulence.

The danger is:

- Pressure overload → hypertrophy → ischemia → arrhythmia
- Volume overload → dilation → systolic failure
- Shunt → pulmonary hypertension → right heart failure

Symptoms reflect **hemodynamic stress**, not the sound.

If you'd like, I can next:

- Create a practical screening checklist for primary care
- Provide a cardiology-style board exam differential framework
- Explain murmur progression over decades

- Or map symptoms to echo findings and pressure–volume loops

Murmur vs BP, any relation physiologically?

Yes — **heart murmurs and blood pressure (BP) are physiologically related**, but they reflect **different aspects of cardiovascular function**:

- **Murmur = flow turbulence**
- **Blood pressure = vascular pressure & cardiac output**

They often influence each other through **hemodynamics**.

Core Physiology Linking Murmur & Blood Pressure

Blood flow through the heart depends on:

Flow = Pressure gradient ÷ Resistance

Murmurs occur when:

- flow velocity increases
- pressure gradients rise
- orifices narrow
- viscosity changes

Blood pressure affects all of these.

How Blood Pressure Influences Murmurs

1. High Blood Pressure (Hypertension)

Physiologic Effects

- ↑ systemic vascular resistance (afterload)
- LV must generate higher pressure to eject blood
- promotes LV hypertrophy & valve degeneration

Murmur Effects

- may **accentuate regurgitant murmurs**
- may worsen **Aortic Regurgitation** due to higher aortic pressure pushing blood backward
- contributes to degenerative **Aortic Stenosis** over time

Clinical insight

Hypertension increases stress on the aortic valve and root → structural changes
→ murmurs later.

2. Low Blood Pressure (Hypotension)

Physiologic Effects

- reduced pressure gradient
- decreased forward flow

Murmur Effects

- murmurs may become **softer** due to lower flow
- severe valve disease may appear deceptively mild

Seen in:

- cardiogenic shock
 - severe heart failure
-

How Valve Disease Alters Blood Pressure

1. Aortic Stenosis

Mechanism:

Narrow valve → obstructed outflow → reduced stroke volume

BP Pattern:

- narrow pulse pressure
- low systolic pressure
- weak, delayed pulse

This leads to:

- dizziness
 - syncope
 - poor exercise tolerance
-

2. Aortic Regurgitation

Mechanism:

Blood leaks back into LV during diastole.

BP Pattern:

- high systolic pressure (large stroke volume)
- very low diastolic pressure (backflow)
- **wide pulse pressure**

Physical signs:

- bounding pulse
 - head bobbing (de Musset sign)
 - visible pulsations
-

3. Mitral Regurgitation

Mechanism:

Blood regurgitates into LA → reduced forward output.

BP Pattern:

- normal or low systemic BP
 - reduced effective perfusion despite normal readings
-

4. Mitral Stenosis

Mechanism:

Obstructed LV filling → reduced cardiac output.

BP Pattern:

- often normal
- may trend low with advanced disease

Pulse Pressure Patterns & Murmurs

Pulse pressure = systolic – diastolic

Pattern	Associated Murmur
Wide pulse pressure	Aortic regurgitation
Narrow pulse pressure	Aortic stenosis
Normal pulse pressure	Mitral stenosis
Hyperdynamic pulse	High-output states

Murmurs & Hypertension: Long-Term Relationship

Chronic hypertension can lead to:

- LV hypertrophy
- aortic root dilation
- valve degeneration
- diastolic dysfunction

These changes increase risk of:

- aortic stenosis
- aortic regurgitation

- functional mitral regurgitation
-

High-Output States: Low BP Resistance, Loud Flow

Murmurs may appear when flow velocity rises despite normal valves:

- anemia
- pregnancy
- fever
- hyperthyroidism

Lower viscosity → turbulence → soft systolic murmur.

Why Severe Murmurs Don't Always Mean High BP

A loud murmur depends on **flow velocity**, not pressure alone.

Examples:

- Severe stenosis + low cardiac output → quiet murmur
- Mild stenosis + high flow → loud murmur

Clinicians always interpret murmurs with hemodynamics.

Quick Physiologic Summary

BP influences murmurs by:

- ✓ altering pressure gradients
- ✓ changing flow velocity
- ✓ modifying afterload
- ✓ promoting structural valve disease

Valve disease influences BP by:

- ✓ altering stroke volume
 - ✓ changing pulse pressure
 - ✓ affecting systemic perfusion
-

Practical Takeaway

If you observe:

- **wide pulse pressure + bounding pulse** → think aortic regurgitation
- **weak, delayed pulse + dizziness** → think aortic stenosis
- **normal BP but severe fatigue** → forward flow may be reduced

Blood pressure alone doesn't rule out serious valve disease.

If you want, I can next:

- explain pulse patterns you can feel at the wrist
- give a self-screen checklist for early valve disease
- compare murmurs in athletes vs hypertensive patients
- show how cycling endurance training affects murmurs & BP

For diagnostic, how to use BP to determine heart issues

Blood pressure (BP) is not a diagnosis by itself, but **patterns in BP and pulse characteristics** provide powerful clues about cardiac structure, valve disease, cardiac output, and vascular health.

Think of BP as a **hemodynamic fingerprint**.

How BP Helps Diagnose Heart Problems

BP reflects:

- **Cardiac output (CO)**
- **Systemic vascular resistance (SVR)**

- **Arterial elasticity**
- **Valve integrity**
- **Circulatory volume**

$$\text{BP} = \text{CO} \times \text{SVR}$$

Changes in these variables reveal underlying cardiac pathology.

1. Pulse Pressure: One of the Most Useful Clues

Pulse Pressure (PP) = Systolic – Diastolic

Normal: ~40 mmHg

Wide Pulse Pressure (>60)

Suggests:

- **Aortic Regurgitation**
- arterial stiffness (aging)
- hyperthyroidism
- anemia
- high-output states

Physiology:

- ↑ stroke volume raises systolic
- regurgitation lowers diastolic

Clues:

- bounding pulse
 - visible pulsations
 - head throbbing sensation
-

Narrow Pulse Pressure (<25)

Suggests:

- **Aortic Stenosis**
- heart failure
- cardiogenic shock
- severe dehydration

Physiology:

- reduced stroke volume
- poor forward flow

Clues:

- weak pulse
 - dizziness
 - exercise intolerance
-

2. Systolic BP Patterns

Low Systolic BP

Suggests:

- reduced cardiac output
- advanced heart failure
- severe valve disease
- shock states

Seen in:

- severe aortic stenosis

- cardiomyopathy
-

High Systolic BP

Suggests:

- arterial stiffness
- increased stroke volume
- chronic hypertension

May lead to:

- LV hypertrophy
 - valve degeneration
 - diastolic dysfunction
-

3. Diastolic BP Patterns

Low Diastolic Pressure

Suggests:

- **Aortic Regurgitation**
- vasodilation
- sepsis (if acute)

Why:

Blood leaks back into LV → diastolic pressure drops.

High Diastolic Pressure

Suggests:

- increased vascular resistance
- chronic hypertension
- endothelial dysfunction

Consequences:

- coronary perfusion impairment
 - LV hypertrophy
-

4. Orthostatic BP Changes

Measure BP lying, sitting, standing.

Drop ≥ 20 systolic or ≥ 10 diastolic

Suggests:

- autonomic dysfunction
- dehydration
- heart failure
- medication effect

Symptoms:

- dizziness when standing
 - faintness
 - blurred vision
-

5. BP Response to Exercise

Exercise BP reveals cardiac performance.

Normal:

- systolic rises
- diastolic stable/slight drop

Abnormal Patterns:

Drop in systolic BP with exertion

Red flag for:

- severe **Aortic Stenosis**
- cardiomyopathy
- ischemia

Exaggerated rise (>210 mmHg men)

Suggests:

- future hypertension
 - vascular stiffness
-

6. Pulse Quality (Felt at Wrist)

Even without equipment:

Bounding pulse

→ wide pulse pressure → AR, high output

Weak, thready pulse

→ low cardiac output

Slow-rising pulse (pulsus parvus et tardus)

→ aortic stenosis

Irregular pulse

→ atrial fibrillation

7. BP Symmetry Between Arms

Normal difference: <10 mmHg

Difference >15–20 mmHg

Consider:

- subclavian artery stenosis
 - aortic dissection (acute)
 - vascular disease
-

8. BP & Heart Failure

As heart failure progresses:

Early:

- normal BP

Later:

- narrow pulse pressure
- low systolic pressure
- poor perfusion signs

Late-stage hypotension = poor prognosis.

Putting It Together: Pattern Recognition

Pattern 1:

160/50 + bounding pulse

→ think **Aortic Regurgitation**

Pattern 2:

95/80 + weak pulse + dizziness

→ think low stroke volume, severe stenosis, HF

Pattern 3:

Normal BP but severe fatigue

→ forward flow impairment (MR, cardiomyopathy)

Pattern 4:

BP drops with exercise

→ severe outflow obstruction

Important Limitation

Normal BP does **NOT** rule out:

- valve disease
- cardiomyopathy
- coronary disease
- arrhythmia

BP is one piece of the diagnostic puzzle.

When BP Findings Suggest Urgent Evaluation

Seek care if BP patterns accompany:

- syncope
- chest pain
- shortness of breath

- new exercise intolerance
 - irregular heartbeat
 - sudden BP asymmetry
-

Clinical Insight

BP tells you:

- ✓ how hard the heart pumps
- ✓ how well blood flows forward
- ✓ whether blood leaks backward
- ✓ vascular health and stiffness

A murmur tells you turbulence.

BP tells you **hemodynamic consequence**.

If you want, I can next:

- teach how to check pulse pressure & pulse quality yourself
- create a self-screening checklist for cyclists/endurance athletes
- explain BP patterns during long climbs & fatigue
- show differences between athletic heart vs pathological findings

Explain more "hemodynamic fingerprint"

What clinicians mean by a "hemodynamic fingerprint"

Every heartbeat generates a unique pattern of **pressures, flows, and vascular responses**. When you measure blood pressure, feel the pulse, or observe circulation signs, you are sensing the **net result of cardiac pumping, valve function, vascular tone, blood volume, and arterial elasticity**.

That composite pattern acts like a **fingerprint** — it reflects how the cardiovascular system is functioning at that moment.

Instead of looking at BP as a single number, clinicians interpret:

- systolic pressure
- diastolic pressure
- pulse pressure
- pulse contour & strength
- changes with position or exercise
- peripheral perfusion signs

Together, these reveal underlying physiology.

The Physiology Behind the “Fingerprint”

1. Cardiac Output (Pump Performance)

Determined by:

- stroke volume
- heart rate
- contractility
- preload

Low output → narrow pulse pressure, weak pulses

High output → bounding pulses, widened pulse pressure

2. Valve Integrity & Flow Direction

Valves ensure one-way flow. If they narrow or leak, pressure patterns change.

Example:

- **Aortic Stenosis** → reduced forward flow → low systolic & narrow pulse pressure
 - **Aortic Regurgitation** → backflow → low diastolic & wide pulse pressure
-

3. Systemic Vascular Resistance (SVR)

Arterioles regulate resistance.

High resistance → elevated diastolic pressure

Low resistance → low diastolic pressure & warm extremities

Seen in:

- hypertension (high SVR)
 - sepsis or heat (low SVR)
-

4. Arterial Elasticity

Healthy arteries cushion pressure waves.

Stiff arteries:

- raise systolic pressure
- widen pulse pressure
- increase cardiac workload

Common with aging & chronic hypertension.

5. Circulating Blood Volume

Low volume → low preload → weak pulse & hypotension

High volume → increased venous return & pressure

Seen in:

- dehydration
 - hemorrhage
 - fluid overload states
-

Why Patterns Matter More Than Numbers

Two patients can have **the same BP** but very different physiology.

BP	Possible Physiology
120/80	healthy cardiovascular system
120/80	severe mitral regurgitation with reduced forward flow
120/80	compensated heart failure
120/80	endurance athlete with high stroke volume

The **pattern & context** define the fingerprint.

Classic Hemodynamic Fingerprints

1. Wide Pulse Pressure + Bounding Pulse

Example: 160/50

Suggests:

- **Aortic Regurgitation**
- high-output state
- arterial stiffness

Physiology:

Large stroke volume ↑ systolic

Diastolic falls due to runoff or backflow

2. Narrow Pulse Pressure + Weak Pulse

Example: 95/80

Suggests:

- low stroke volume
- heart failure
- severe **Aortic Stenosis**

Physiology:

Obstructed or weak pump → reduced forward flow

3. High Systolic + Normal Diastolic

Example: 160/80

Suggests:

- arterial stiffness
 - chronic hypertension
 - increased LV workload
-

4. Normal BP + Poor Perfusion Signs

Normal reading but:

- fatigue
- cold hands
- exercise intolerance

Suggests:

Reduced effective forward flow (e.g., **Mitral Regurgitation**)

5. BP Drop When Standing (Orthostatic Pattern)

Suggests:

- volume depletion
 - autonomic dysfunction
 - heart failure
-

Pulse Wave Characteristics (Part of the Fingerprint)

Clinicians feel the arterial waveform:

Bounding pulse

→ high stroke volume or wide pulse pressure

Weak, thready pulse

→ low cardiac output

Slow-rising pulse

→ aortic outflow obstruction

Irregular pulse

→ arrhythmia

How Exercise Changes the Fingerprint

Normal Response

- systolic rises
- pulse strengthens
- perfusion improves

Abnormal Patterns

Drop in systolic pressure:

→ severe outflow obstruction or cardiomyopathy

Failure of pulse to strengthen:

→ impaired cardiac output

Hemodynamic Compensation: Why Symptoms Appear Late

The body compensates via:

- ventricular hypertrophy
- chamber dilation
- neurohormonal activation
- increased heart rate
- vascular constriction

Because of compensation, abnormal fingerprints can exist **years before symptoms**.

Practical Analogy

Think of circulation like a plumbing system:

- Pump = heart
- Valves = one-way gates
- Pipes = arteries
- Pressure = driving force
- Flow pattern = system health

A hemodynamic fingerprint shows:

- ✓ pump strength
 - ✓ valve leaks or narrowing
 - ✓ pipe stiffness
 - ✓ fluid volume
 - ✓ flow efficiency
-

Why This Matters Clinically

Recognizing patterns allows early detection of:

- valve disease
- heart failure
- vascular stiffness
- shock states
- autonomic dysfunction

Often **before imaging** is done.

Key Takeaway

A single BP reading is a number.

A **hemodynamic fingerprint** is the pattern created by:

- ✓ pressure
- ✓ flow
- ✓ pulse quality
- ✓ vascular tone
- ✓ physiologic response

Interpreting the pattern reveals the cardiovascular story.

If you want, I can next:

- teach how to read your pulse like a clinician
- explain how endurance training alters your hemodynamic fingerprint
- show early warning fingerprints for cyclists & athletes
- or provide a self-check routine for cardiovascular health

Is it safe to say that if I can climb 3000 ft total in cycling and the overall duration of 5 hours, that my heart valves are in good shape? Age 59, AMI at 42, LAD occlusion, now no dyspnea, angina and dizziness since post MI.

Short answer: **your ability to complete a 5-hour ride with ~3000 ft of climbing without dyspnea, angina, or dizziness is a strong functional sign that your heart is performing well under stress**, but it does **not guarantee that your heart valves are structurally normal**.

Given your history of prior myocardial infarction (MI) with LAD occlusion, what you're describing is encouraging — but functional capacity and valve integrity are related yet different.

What Your Cycling Performance Suggests

Climbing 3000 ft over 5 hours requires:

- ✓ sustained cardiac output
- ✓ adequate coronary perfusion
- ✓ effective oxygen delivery
- ✓ good stroke volume
- ✓ absence of flow-limiting outflow obstruction

If you had **severe valve disease**, you would typically experience:

- exertional breathlessness
- chest pressure
- dizziness or near syncope
- early fatigue

Your lack of these symptoms suggests:

- ✓ good forward flow
 - ✓ adequate cardiac reserve
 - ✓ stable coronary perfusion
 - ✓ no severe hemodynamic obstruction
-

Why Exercise Capacity ≠ Valve Normality

Valve disease exists on a spectrum.

Mild or moderate disease may produce **no symptoms** and still allow high performance.

Conditions that may remain silent for years:

- early **Aortic Stenosis**
- mild **Mitral Regurgitation**
- mild **Aortic Regurgitation**

Athletes sometimes compensate via:

- increased stroke volume
- ventricular remodeling
- enhanced oxygen extraction

So performance alone cannot rule out early structural abnormalities.

What Your Performance DOES Strongly Rule Out

Based on your symptom-free climbs, it is unlikely you have:

- ✗ severe aortic stenosis
- ✗ advanced mitral stenosis
- ✗ severe regurgitation causing pulmonary congestion
- ✗ critical cardiac output limitation
- ✗ exercise-induced hypotension

These conditions typically cause **clear exertional symptoms**.

Important Context: Prior MI & LAD Occlusion

After an LAD event, long-term concerns include:

1. Left ventricular remodeling

May affect valve function indirectly.

2. Functional mitral regurgitation

LV dilation can alter valve geometry.

3. Reduced ejection fraction

May limit exercise capacity.

Your current performance suggests:

- ✓ good myocardial adaptation
 - ✓ preserved functional capacity
 - ✓ adequate coronary perfusion during exertion
-

Signs During Cycling That Would Suggest Valve or Cardiac Issues

Watch for:

During climbs:

- chest tightness or pressure
- unusual breathlessness
- dizziness or “tunnel vision”
- sudden drop in power output

After rides:

- prolonged recovery fatigue
- nighttime breathlessness
- leg swelling

If absent → reassuring.

When Valve Disease Typically Becomes Symptomatic

Aortic Stenosis

- exertional dizziness

- chest pressure
- reduced power on climbs

Mitral Regurgitation

- breathlessness
- fatigue
- decreased endurance

Aortic Regurgitation

- pounding pulse
 - breathlessness later
 - declining endurance
-

What Cardiologists Use to Confirm Valve Health

Even in high-performing athletes:

- echocardiogram (valve structure & flow)
- ejection fraction
- chamber size
- pressure gradients
- stress testing if indicated

For someone with prior MI, periodic echo surveillance is common.

How to Interpret Your Current Status

Based on what you described:

Very reassuring signs:

- ✓ strong endurance
- ✓ no dyspnea

- ✓ no angina
- ✓ no exertional dizziness
- ✓ ability to sustain climbs

This suggests **good functional cardiac performance**.

But not a guarantee of structurally perfect valves.

Practical Takeaway

It is reasonable to say:

- 👉 Your heart is functioning well under load.
- 👉 Severe valve disease is unlikely.
- 👉 Your cardiovascular fitness is protective.

It is not accurate to conclude:

- ✗ "My valves are definitely normal."
-

Smart Monitoring for a 59-Year-Old Endurance Cyclist with Prior MI

Consider periodic:

- echocardiogram (every few years or per cardiologist)
 - lipid & metabolic control
 - blood pressure tracking
 - exercise tolerance monitoring
 - heart rate recovery tracking
-

When to Seek Evaluation Promptly

If you develop:

- new breathlessness on climbs
- chest discomfort

- dizziness or near fainting
- sudden drop in endurance
- irregular heartbeat

These may indicate structural or ischemic changes.

If you want, I can next:

- explain how to distinguish cardiac vs fitness fatigue on climbs
- describe “normal aging heart” vs pathological changes in cyclists
- outline optimal training intensity post-MI
- or provide a heart health checklist for endurance athletes over 50

About the heart sound in the stethoscope, is there a difference between A P T M (S1, S2 normal) or the pattern is different? What is the sound of S3 and S4

Heart Sounds: The Full Picture

S1 and S2 — The Baseline "Lub-Dub"

The classic **lub-dub** represents one cardiac cycle:

- **Lub** = S1 (start of systole)
 - **Dub** = S2 (start of diastole)
-

The Valves Within S1 and S2

S1 and S2 are each made up of **two components**, corresponding to the closure of specific valves:

S1 (Lub) — nearly simultaneous, heard as one sound:

- **M** (Mitral) closes *just before* **T** (Tricuspid)
- M1 → T1, but so close together (~0.02s) it sounds like a single "lub"

S2 (Dub) — also two components, but slightly more separated:

- **A** (Aortic) closes *just before* **P** (Pulmonic)

- A2 → P2

The classic mnemonic order across the cycle: **M → T → A → P**
(or remembered as "**Many Tiny Animals Play**")

Physiological splitting of S2 occurs on *inspiration* because increased venous return delays pulmonic closure slightly, making A2 and P2 audible as separate sounds — "*lub, da-dub*". This is normal.

S3 — "Kentucky Gallop" (Lub-Dub-ta)

lub – dub – ta
or phonetically: "**Ken-tuc-ky**"

- Occurs **early diastole**, just after S2
 - Caused by rapid ventricular filling hitting a non-compliant or volume-overloaded ventricle
 - Low-pitched, best heard at the apex with the bell of the stethoscope
 - **Normal in children and young adults / athletes**
 - **Pathological in adults** → suggests HF, dilated cardiomyopathy, MR, AR
-

S4 — "Tennessee Gallop" (taLub-Dub)

ta – lub – dub
or phonetically: "**Ten-nes-see**"

- Occurs **late diastole**, just *before* S1
 - Caused by atrial contraction forcing blood into a stiff, non-compliant ventricle ("atrial kick")
 - Low-pitched, best heard at the apex with the bell
 - **Almost always pathological** → suggests LVH, hypertension, aortic stenosis, hypertrophic cardiomyopathy
 - Cannot occur in atrial fibrillation (no organized atrial contraction)
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Summary Table

Sound	Timing	Phonetic	Cause	Normal?
S1 (Lub)	Start of systole	LUB	M + T closure	✓
S2 (Dub)	Start of diastole	DUB	A + P closure	✓
S3	Early diastole	lub-dub- ta / "Ken-tuc-ky"	Rapid filling / volume overload	In young/athletes only
S4	Late diastole	ta -lub-dub / "Ten-nes-see"	Atrial kick into stiff ventricle	✗ Almost always pathological