DYNAMIC AUSCULTATION AND CLINICAL APPLICATIONS

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I THE PHYSIOLOGICAL BASIS

- **Core Concept:** Dynamic auscultation systematically alters circulatory hemodynamics using physiological bedside maneuvers or vasoactive pharmacological agents to modify the **intensity, duration, or timing** of heart sounds and cardiac murmurs, enabling accurate bedside differentiation of valvular, congenital, and myocardial lesions.
- **Four Primary Hemodynamic Determinants:**
  - **Venous Return and Ventricular End-Diastolic Volume (Preload):** Determines ventricular cavity geometry and forward stroke volume.
  - **Systemic Vascular Resistance (Afterload):** Determines the impedance to left ventricular ejection vs. retrograde/shunt regurgitant flow.
  - **Myocardial Contractility (Inotropic State):** Modifies ejection velocity and dynamic outflow tract gradients.
  - **Diastolic Cycle Length (R-R Interval):** Modifies ventricular filling time and post-extrasystolic potentiation.

II COMMON BEDSIDE MANEUVERS

A Respiration (Inspiration vs. Expiration)

- **Physiology:** Normal inspiration generates negative intrathoracic pressure (drawing systemic venous return into the right atrium and right ventricle) while increasing pulmonary vascular capacitance (transiently pooling blood in the lungs and reducing left ventricular preload by 5 to 10 percent); expiration reverses these changes.
- **Clinical Application:**
  - **Right-Sided Murmurs and Sounds:** Almost all right-sided murmurs and sounds—**Tricuspid Regurgitation (Carvallo Sign)**, **Tricuspid Stenosis**, **Pulmonary Regurgitation**, **Valvular Pulmonary Stenosis murmur**, and right-sided **S3/S4**—**increase in intensity during inspiration**.
  - **Crucial Exception — Pulmonary Ejection Click:** The **valvular Pulmonary Stenosis Ejection Click** is the **only right-sided acoustic event that decreases (softens or disappears) during inspiration**, because augmented inspiratory venous return into a stiff right ventricle elevates RV end-diastolic pressure above pulmonary artery diastolic pressure, prematurely floating the stenotic pulmonary valve leaflets partially open in late diastole before ventricular systole begins.
  - **Left-Sided Murmurs: Increase or become clearer during held expiration** as pooled pulmonary venous blood returns to the left heart and lung tissue recedes from the chest wall.

B The Valsalva Maneuver (Four Phases; Exam Focus on Phase II Strain and Phase III/IV Release)

- **Physiology:** Forced expiration against a closed glottis (or maintaining **40 mmHg for 15 to 20 seconds** on a manometer/syringe). During **Phase II (Strain Phase)**, sustained positive intrathoracic pressure compresses the venae cavae, **sharply decreasing venous return (preload), right and left ventricular end-diastolic volumes, stroke volume, and arterial pulse pressure**, accompanied by reflex sinus tachycardia.
- **Clinical Application:**
  - **Decrease in Intensity:** Virtually all forward flow and regurgitant murmurs (**Aortic Stenosis, Pulmonary Stenosis, Mitral Regurgitation, Aortic Regurgitation, Tricuspid Regurgitation, Mitral Stenosis, and VSD**) **soften** during Phase II strain because stroke volume falls.
  - **Paradoxical Increase (The Two Exceptions):** Only two conditions show an increase or lengthening during Phase II strain: (1) the dynamic outflow murmur of **Hypertrophic Obstructive Cardiomyopathy (HOCM) increases in intensity** because reduced LV cavity volume brings the anterior mitral leaflet closer to the hypertrophied interventricular septum, worsening Systolic Anterior Motion (**SAM**) and LVOT obstruction; and (2) in **Mitral Valve Prolapse (MVP)**, reduced LV volume causes the redundant leaflet to prolapse **earlier in systole**, moving the **mid-systolic click closer to S1** and **lengthening** the late-systolic murmur.
  - **Release Phase (Phase III/IV):** Upon release of strain, right-sided murmurs return to baseline intensity **immediately (within 1 to 2 beats)**, whereas left-sided murmurs return to baseline only after **4 to 8 beats** (the time required for blood to traverse the pulmonary circulation).

C Positional Changes (Sudden Standing vs. Squatting / Passive Leg Raising)

- **Sudden Standing from Squatting (Mimics Valsalva Phase II):** Pooling of 300 to 800 mL of blood in the lower extremities **acutely reduces venous return (preload) and LV cavity size**. **Increases** the murmur of **HOCM** and moves the **MVP click earlier** (lengthening the MVP murmur), while **decreasing** the murmurs of **Aortic Stenosis, Pulmonary Stenosis, MR, AR, and VSD**.
- **Rapid Squatting or Passive Leg Raising:** Simultaneously **increases venous return (preload)** by compressing lower-extremity veins and **increases systemic vascular resistance (afterload)** by kinking the femoral and iliac arteries. **Increases** the murmurs of **Aortic Stenosis, Pulmonary Stenosis, Mitral Regurgitation, Aortic Regurgitation, and VSD**; conversely, it **markedly softens the murmur of HOCM** (by distending the LV cavity and increasing aortic root pressure, which pushes the anterior mitral leaflet away from the septum) and **delays the MVP click toward S2** (shortening the MVP murmur).

D Sustained Isometric Handgrip Exercise

- **Physiology:** Clenching a handgrip dynamometer (or a rolled blood pressure cuff) at 30 to 50 percent maximal effort for 20 to 30 seconds while breathing normally (avoiding accidental Valsalva) **markedly increases Systemic Vascular Resistance (Afterload)** and arterial blood pressure.
- **Clinical Application:**
  - **Increases Left-Sided Regurgitant and Shunt Murmurs: Mitral Regurgitation (MR)**, **Aortic Regurgitation (AR)**, and **Ventricular Septal Defect (VSD)** murmurs **increase significantly** because elevated aortic impedance drives more blood backward across the incompetent valve or left-to-right across the VSD, and also increases **Mitral Stenosis** (secondary to reflex tachycardia) and **LV S3/S4 gallops**.
  - **Decreases HOCM and Valvular Aortic Stenosis: Markedly softens HOCM** (elevated aortic pressure splints open the LVOT and reduces SAM) and **softens or leaves unchanged the murmur of Valvular Aortic Stenosis** (because elevated aortic pressure reduces the peak-to-peak transvalvular velocity gradient across the fixed orifice).

E Post-Extrasystolic Pause (Post-PVC Beat) or Long R-R Cycle in Atrial Fibrillation

- **Physiology:** The compensatory pause following a premature ventricular contraction (PVC) or a long R-R cycle in atrial fibrillation increases **LV diastolic filling time (preload)** and enhances **sarcoplasmic reticulum calcium release (post-extrasystolic potentiation / contractility)**, while aortic diastolic pressure falls during the long pause (reducing **afterload**).
- **Clinical Application (Differentiating AS from MR at the Apex):**
  - **Aortic Stenosis (and HOCM):** The ejection murmur becomes **markedly louder** in the beat immediately following a PVC/long pause (in HOCM, heightened contractility + lower aortic diastolic afterload worsens SAM, creating the classic **Brockenbrough-Braunwald-Morrow Sign** on catheterization where LV systolic pressure rises while aortic pulse pressure paradoxically narrows).
  - **Holosystolic Mitral Regurgitation:** The intensity of a rheumatic or ischemic **Mitral Regurgitation murmur remains virtually unchanged** after a long pause because there is very little isovolumic contraction phase (blood decompresses immediately into the low-pressure left atrium throughout systole) and lower aortic afterload favors forward aortic ejection.

Cardiac Lesion	Inspiration	Valsalva Phase II / Sudden Standing	Rapid Squatting / Leg Raise	Isometric Handgrip	Amyl Nitrite Inhalation	Post-PVC Beat (Long Pause)
Valvular Aortic Stenosis (AS)	Decreases / No change	**Decreases**	**Increases**	**Decreases** (or unchanged)	**Increases**	**Markedly Increases**
HOCM (Dynamic LVOTO)	No change	**Increases**	**Decreases**	**Decreases**	**Markedly Increases**	**Markedly Increases**
Mitral Valve Prolapse (MVP)	No change	Click **earlier**; murmur **longer**	Click **later**; murmur **shorter**	Click later; murmur variable	Click earlier; murmur variable	Click earlier
Mitral Regurgitation (MR) / VSD	Decreases / No change	**Decreases**	**Increases**	**Markedly Increases**	**Markedly Decreases**	**Unchanged**
Aortic Regurgitation (AR)	No change	**Decreases**	**Increases**	**Markedly Increases**	**Decreases**	Increases
Mitral Stenosis (MS)	Decreases	**Decreases**	**Increases**	**Increases** (via HR rise)	**Increases** (via HR/CO rise)	Longer murmur in long cycle
TR / Valvular PS Murmur	**Increases (Carvallo)**	**Decreases** (immediate return on release)	**Increases**	No change	**Increases** (via venous return)	Increases

III PHARMACOLOGICAL DYNAMIC AUSCULTATION

- **Amyl Nitrite Inhalation:** Potent rapid-acting systemic venodilator and arterial vasodilator (lasting 30 to 60 seconds) that **acutely drops SVR (afterload) and blood pressure**, triggering a reflex **sympathetic surge (tachycardia and increased contractility)** and a secondary increase in venous return.
  - **Murmurs that Increase:** All forward ejection and inflow stenotic murmurs—**Valvular Aortic Stenosis**, **HOCM**, **Mitral Stenosis** (thereby differentiating the **Austin Flint murmur of severe AR** [which **softens** with amyl nitrite] from true organic **Rheumatic Mitral Stenosis** [which **intensifies** with amyl nitrite]), **Pulmonary Stenosis**, and **Tricuspid Regurgitation**.
  - **Murmurs that Decrease:** Left-sided regurgitant and left-to-right shunt murmurs—**Mitral Regurgitation, Aortic Regurgitation, VSD, PDA**, and the **Austin Flint murmur**—plus the systolic murmur of **Tetralogy of Fallot** (unlike isolated Valvular PS, dropping SVR in TOF increases R-to-L shunting across the VSD and **decreases** RVOT flow and murmur intensity).
- **Phenylephrine (Alpha-1 Agonist):** Acutely **elevates SVR (afterload)** without direct inotropic stimulation (mimicking isometric handgrip). **Increases** the murmurs of **MR, AR, VSD, PDA, and Tetralogy of Fallot** (by forcing blood across the RVOT into the lungs), while **decreasing the murmur of HOCM**.

IV PRACTICAL CONSIDERATIONS IN THE INDIAN CLINICAL CONTEXT

- **Multivalvular Rheumatic Heart Disease:** In Indian clinics where combined **Rheumatic Mitral and Aortic Valve Disease** is common, bedside maneuvers—specifically **post-PVC/long-cycle auscultation in atrial fibrillation** and **isometric handgrip**—rapidly distinguish an **apical holosystolic murmur of Rheumatic MR** from **apical radiation of Calcific/Rheumatic Aortic Stenosis (Gallavardin phenomenon)**.
- **Austin Flint vs. Organic Rheumatic Mitral Stenosis:** In severe chronic Aortic Regurgitation with an apical mid-diastolic rumble, **isometric handgrip** or **vasodilators** help distinguish functional **Austin Flint rumble** (which increases with handgrip and decreases with vasodilators) from concomitant **Rheumatic Mitral Stenosis** (which has a loud S1 and Opening Snap).
- **Standardizing the Bedside Valsalva Maneuver:** Because patients often fail to maintain an adequate closed-glottis strain on verbal command alone, having the patient **blow forcefully into a 10 or 20 mL syringe barrel to displace the plunger** for 15 seconds ensures a reproducible **Phase II Valsalva strain** in the outpatient clinic.

KEY REFERENCES

- **Lembo NJ, Dell'Italia LJ, Crawford MH, O'Rourke RA:** Bedside diagnosis of systolic murmurs, *New England Journal of Medicine*, 1988.
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- **O'Gara PT, Loscalzo J:** Physical Examination of the Cardiovascular System, in *Harrison's Principles of Internal Medicine*, 21st Ed., McGraw Hill, 2022.