A nursing home patient with a PMH of CAD, HTN, DM, and PAD presents to the ED with confusion and lethargy. He is found to be hypotensive with a blood pressure of 82/49. His LVEF is 40%. What is the next treatment to identify the cause of this patient’s hypotension?

A. Administer a fluid bolus
B. Screen his urine
C. Order an ECG
D. A & B
E. B & C

Hemodynamic homeostasis depends on four things:

- Preload
- Afterload
- Contractility
- Heart Rate

When treating open-heart surgery patients and patients in the CV ICU, these are the variables we treat to change the patient’s

- Cardiac Output
- Oxygen Delivery
- Blood Pressure
- Organ perfusion
Cardiac Output

- Cardiac Output is the measure of the volume of blood, and thereby how much oxygen, can be delivered.
- Cardiac Output = Stroke Volume x Heart Rate
- How much blood the heart pumps per minute = (the amount it pumps per contraction) x HR
- The amount it pumps per contraction is called STROKE VOLUME

Cardiac Output = SV x HR

- What three factors determine SV?
  1. Preload
     - How much fluid is in the pump?
  2. Afterload
     - How much resistance is there to pumping?
  3. Contractility
     - How strong is the pump?
**Preload**

- What fills the ventricle prior to its contraction
- a.k.a. LVEDV, LVEDP
- The greater you fill the pump, the more comes out of the pump.
- Frank-Starling
Pressure volume loops

Overview

- Time

LV Pressure
LV Volume

MV opens
MV closes

Overview: Diastole - relax. To send apart

- Start with diastole

LV Pressure
LV Volume

AV closes
AV opens
MV opens
MV closes
Fill the Ventricle - Diastole

This curve lengthens and shortens due to LV preload or LVEDP. Diuresis, nitrates and blood loss to the left, volume to the right.

Frank-Starling says increased preload leads to improved sarcomere length and therefore increased LV contraction.
Fill the Ventricle - Diastole

Increased preload then leads to increased stroke volume, regardless of what happens next in the curve.

Lucitropy & Diastolic Function

A compliant LV will accept volume. A non-compliant LV will accept a limited volume before pressure rises. The heart's ability to relax and accept volume is called LUCITROPY. Poor lucitropy leads to Diastolic Dysfunction. Compliance = ΔV/ΔP

Empty the Ventricle – Systole- contract to send together

AV opens when LV pressure exceeds Aortic Pressure
MV opens
MV closes when LV pressure is exceeded by Aortic Pressure
AV closes
AV closes when LV pressure is exceeded by Aortic Pressure

MV opens

MV closes

QRS Complex?

AV opens when LV pressure exceeds Aortic Pressure

LV Pressure

LV Volume

AV closes

P-wave

QRS Complex

MV opens

MV closes

AV opens

http://www.lumen.luc.edu/lumen/meded/dlearning/fac_train

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Instructional Designer
Isovolumic Contraction

- Anything that changes this gradient therefore changes the pressure at which the AV opens.
- Decreased Afterload
- Increased Afterload

Afterload

- Technically “impedance to ejection.”
- Could be in the heart - so not “after.”
- HOCM, AS
- Measured by Wall Tension - a top 3 cardiac oxygen consumer.

LaPlace : Wall Tension = \( \frac{P_r}{2t} \)

- \( P_r \) : Pressure in the myocardium in systole
- \( r \) : radius in LV chamber
- \( t \) : Ventricular thickness

Ejection: Effect of Afterload on SV

- Decreasing Afterload increase SV
- Increasing Afterload decreases SV
Ejection: Effect of Afterload on SV

Decreasing Afterload increases SV
Increasing Afterload decreases SV

Contractility
Slope of this line represents contractility
Acutely changing afterload decreases SV, not contractility.

Isovolumic relaxation

Heart sounds
A 70 y/o man with HTN, Obesity, and DM presents with confusion after being prescribed a diuretic. The contractility is known to be normal and hypertension is treated with an afterload reducer. Predict the pressure volume loop.
A 50 y/o man with HTN, Obesity, and DM presents with confusion after being prescribed a diuretic. The contractility is known to be normal and hypertension is treated with an afterload reducer. Predict the pressure volume loop.

PL, AL, Contractility.

What has changed?

A 70 y/o man with HTN, Obesity, and DM presents with confusion after being prescribed a diuretic. The contractility is known to be normal and hypertension is treated with an afterload reducer. Predict the pressure volume loop.

PL, AL, Contractility.

What has changed?

- PL is diminished
- AL is unchanged
- C is unchanged
What's the Stroke Volume?
What's the Ejection Fraction?

LVEDV
LVESV

Left Ventricular
End (Diastolic = Preload)
(Systolic)
Volume
What’s the Stroke Volume?

What’s the Ejection Fraction?

Full - Empty = SV

LVEDV - LVESV = SV

LVEDV x 100 = EF

Normal EF = ~55%

150 - 75 = 75ml Stroke Volume

x 100 = 50% Ejection Fraction
Stroke Volume
- If C.O. is 5 L/m, and HR is 100, SV is?
- EF is?

Stroke Volume
- If C.O. is 5 L/m, and HR is 100, SV is 50ml
- EF is?

Recap
- When managing patients, when considering hemodynamics, what matters?
- 4 things increase CO:
  1. Increasing HR
     - Also increases contractility
  2. Increasing Preload
  3. Decreasing Afterload (SVR)
  4. Increasing Contractility
Blood Pressure - organ perfusion

- Mean Arterial Pressure
- MAP = 2/3 DBP + 1/3 SBP
- Ohm’s Law.
- V = IR
- MAP = CO x SVR
- What contributes to the SVR?

Vascular Resistance

Resistance is proportional to:

Viscosity x Length / Radius to 4th power

SVR

- SVR = MAP - RAP / CO (X 80)
- SVR is calculated
SVR vs. Afterload

- Afterload is impedance to ejection and is (not easily) measured by Wall Tension. It is a measure of what the heart works against.
- SVR is a calculation of vascular resistance. It is after the heart.
- Increasing SVR increases afterload. Increasing afterload does not necessarily increase SVR.
- Worsening Aortic Stenosis
  - SVR = MAP - RAP / CO
  - SVR is CALCULATED. MAP is MEASURED.

Starling Forces

\[ P_l = \text{interstitium}; \ P_c = \text{capillary} \]
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Thank you.