Patient Presentation

- 28 yo Male with a history of Chronic Fatigue Syndrome (CFS) secondary to Postural Orthostatic Tachycardia Syndrome (POTS)
- Monitors attached to the patient, stable vitals (HR 76, BP 130/72)
- Upon standing up, he immediately becomes tachycardic (HR 111)
- and is noted to have distinct pulse oximeter waveform morphology, both of which resolve within two minutes of lying down on the scanner
- Blood pressure remains stable during positioning and tachycardia

Anatomy of the Dicrotic Notch

- Biphasic waveform is a function of reflected pulse waves from the periphery
- Point of reflection is based on impedance and elasticity of the vascular tree, which narrows distally (and stiffens with age)
- Reducing the human vascular tree into an asymmetric T-tube simplifies the model
Timing of closure of the aortic valve fixed (i.e., duration of systole does not change with SVR)

In the finger, second wave reflected from the lower extremities

Timing of the reflected wave is a function of impedance; as SVR decreases, the wave reflects at more distal portions of the vascular tree, and arrives later

Anatomy of the Dicrotic Notch: Aortic Valve, SVR, and Shape

HYPERadrenergic POTS is caused by a norepinephrine transporter mutation, causing impaired reuptake

Upon standing, physiologic orthostatic vasoconstriction of the lower extremities occurs

Decreased catecholamine uptake at sympathetic nerve endings leads to systemic catecholamine spillover and tachycardia

Blood pressure usually normal but can be high or paradoxically low

Tx: May include clonidine, beta blockade

HYPOadrenergic POTS is caused by selective denervation of extremities, leading to impaired vasoconstriction

Upon standing, impaired vasoconstriction allows blood pooling in dependent extremities

Venous pooling causes decreased venous return, leading to intrathoracic hypovolemia and compensatory reflex tachycardia

Blood pressure usually normal but can be low

Tx: Vasoconstrictors, mineralocorticoid analogs

Two Unrelated Mechanisms

Back to Our Patient…

Dx: Denervation POTS!

This is our patient's waveform upon standing:

The prominent, deep dicrotic notch suggests inappropriately low SVR and an inability to compensate for orthostasis with lower extremity vasoconstriction.

Can we test our hypothesis?
Upon crossing his legs, vascular impedance changes and so does the waveform (and heart rate).

Standing from a squat provokes increased tachycardia secondary to amplified lower extremity vasodilation.

**Take Home Message**: A basic understanding of pulse oximeter waveform interpretation may grant the anesthesiologist additional insight into patient (patho)physiology.

References: