Describe the physiology of a vasovagal syncope.

A “vasovagal” syncope is a loss of consciousness from excessive autonomic reflex activity.

Vasovagal syncope is typically triggered by seeing blood, pain, emotional stress, or prolonged standing. Time varying magnetic field (i.e. transcranial magnetic stimulation)

Underlying mechanism involves the nervous system slowing the heart rate and dilating blood vessels resulting in low blood pressure and therefore not enough blood flow to the brain.

Recovery happens without specific treatment. Prevention involves avoiding the triggers. Drinking sufficient fluids, salt, and exercise may also be useful.

Mechanism:
Regardless of the trigger, the mechanism of syncope is similar

- The nucleus tractus solitarii of the brainstem is activated directly or indirectly by the triggering stimulus
- Results in simultaneous enhancement of parasympathetic nervous system (vagal) tone and withdrawal of sympathetic nervous system tone.

This results in a spectrum of hemodynamic responses:

- On one end of the spectrum is the cardioinhibitory response
  - ↓HR, ↓contractility → ↓CO → LOC
  - This response results primarily from enhancement in parasympathetic tone.
- On the other end of the spectrum is the vasodepressor response
  - ↓BP without change in HR.
  - This occurs due to dilation of the blood vessels, probably as a result of withdrawal of sympathetic nervous system tone.

The majority of people with vasovagal syncope have a mixed response somewhere between these two ends of the spectrum. One account for these physiological responses is the Bezold-Jarisch reflex.

CVS Challenges
- ↓ in MAP: = due to ↓ CO
- Hydrostatic effects on CPP:
  - ↓MAP at level of brain
  - effect = immediate.
  - NB: ↓ MAP at brain level is offset by a similar:
    - ↑CVP venous side (brain circulation is like an inverted U-tube) as well as on the ↓CSF pressure.
    - CPP is further augmented by an increase in VR from the brain to the heart in the erect position.
- Summary: the main challenge to the CVS (and the brain circulation) is ↓ MAP

The CVS response
- baroreceptor reflex mechanism:
  - ↓ MAP ⇒ sensed by carotid (mainly) and aortic baroreceptors ⇒ ↓ traffic up to NTS ⇒ via medullary control centre ⇒ ↑ SNS outflow and ↓ PNS outflow.
  - The ↑ SNS outflow causes: [ remember: MAP (minus RAP) = CO x SVR ]
    - [↑preload] peripheral venoC ⇒ ↑ VR ⇒ ↑ CO ⇒ ↑ MAP
- [↑afterload] peripheral vasoC ⇒ ↑ SVR ⇒ ↑ MAP (slight ↓ in SV due to afterload increase, but net effect = ↑ MAP)
- ↑ cardiac contractility ⇒ ↑ CO ⇒ ↑ MAP
- ↑ Heart rate ⇒ ↑ CO ⇒ ↑ MAP

**NB:** Baroreflex ⇒ vasoconstriction = more effective than venoconstriction to restore MAP!
(not to be confused with the vascular function curves where venoconstriction shifts the curve more up than what vasoconstriction rotates it downwards)

- Cerebral pressure autoregulation: a.k.a. the myogenic mechanism:
  - effective at maintaining constant cerebral blood flow in a MAP range of 50–150 mmHg
  - It effects this by changing the CVR.
  - Onset is not immediate though.

  \[ \text{CBF} = \frac{\text{MAP} - (\text{CVP or ICP})}{\text{CVR}} \]

  ← ← arterial baroreflex

  ← ← pressure autoregulation

- Activity: Mm pump further augments VR
  - in conjunction with the one-way valves in the veins to prevents further venous pooling

**Examiner Comments:**

41% of candidates passed this question.

Generally, there was a lack of knowledge about this topic with many candidates confusing vasovagal syncope with a Valsalva or orthostatic hypotension. A “vasovagal” is from excessive autonomic reflex activity in contrast to orthostatic hypotension which is a failure of the autonomic reflex response.

A good place to start was with a description of vasovagal syncope, also known as neurocardiogenic syncope. It is benign, self-limiting and caused by an abnormal or exaggerated autonomic response to various stimuli (which should have been listed). The mechanism should have been described including the various receptors involved.