CARDIOVASCULAR MODULE PHYSIOLOGY

SYNCOPE

GRADE: FIRST YEAR – ASHUR MEDICAL COLLEGE

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MODULE: SYSTEMIC (PHYSIOLOGY)

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OBJECTIVES

- 1. UNDERSTAND THE DEFINITION OF BARORECEPTORS.
- 2. UNDERSTAND THE DEFINITION OF SYNCOPE.
- 2. KNOW THE BASIC ETIOLOGIES OF SYNCOPE.
- 3. LIST THE TYPES OF SYNCOPE :

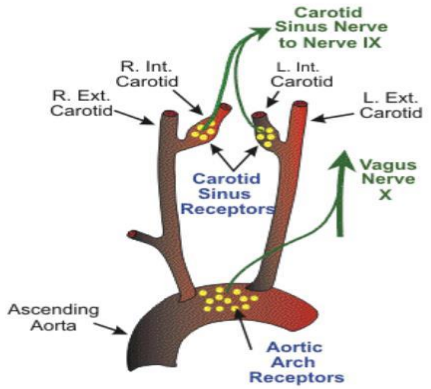
1. Reflex syncope (neurally mediated):
1.1.Vasovagal
1.2 Situational
1.3. carotid sinus syncope.
2. Orthostatic
3. Cardiogenic

INTRODUCTION : BARORECEPTORS

Baroreceptors are a type of mechanoreceptors that allow for relaying information derived from blood pressure within the autonomic nervous system. Information is then passed in rapid sequence to alter the total peripheral resistance and cardiac output, maintaining blood pressure within a preset, normalized range.

Location of baroreceptors

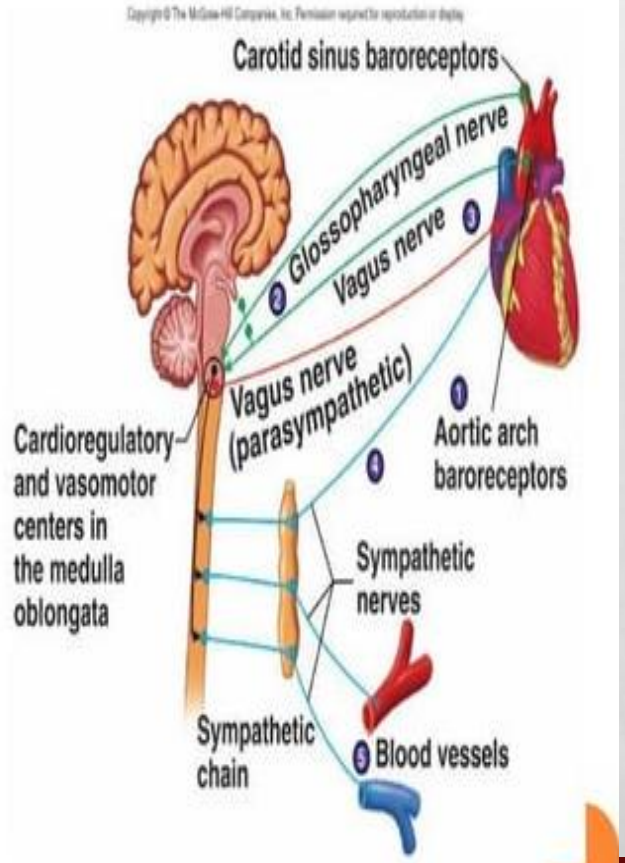
- Baroreceptors sense stretch and rate of stretch by generating action potentials (voltage spikes)
- Located in highly distensible regions of the circulation to maximise sensitivity

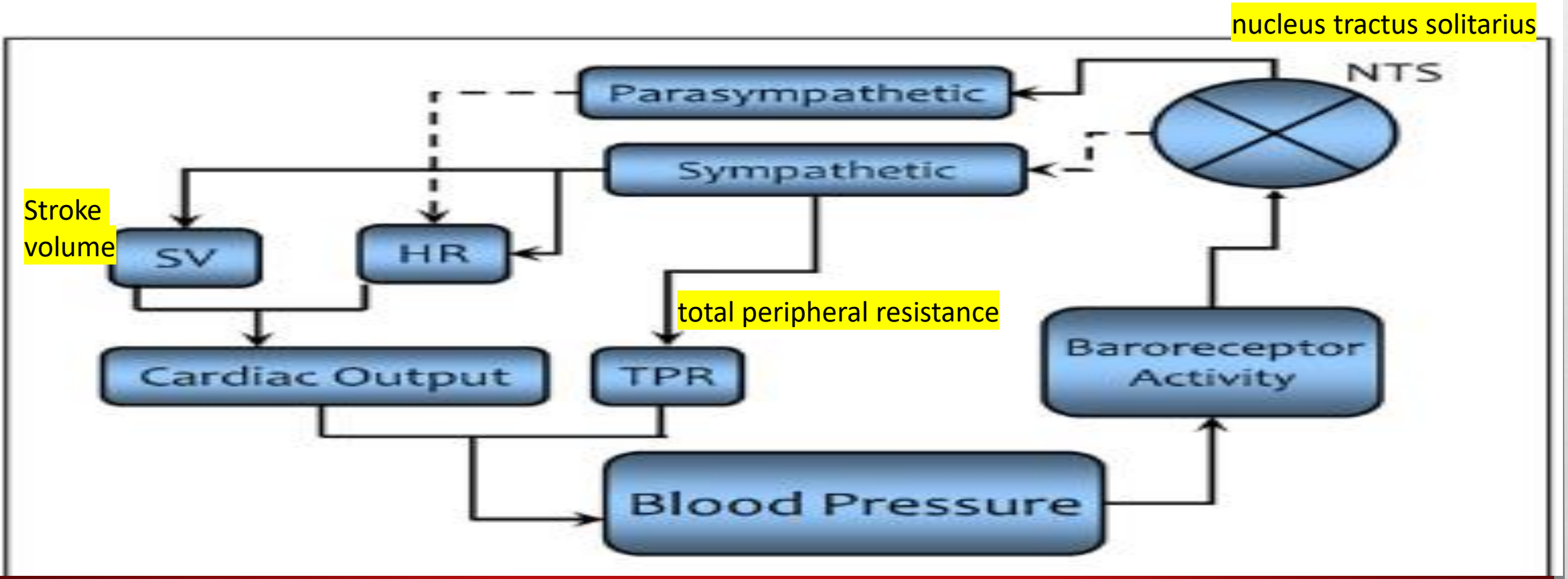


http://www.cvphysiology.com/Blood Pressure/bp012 baroreceptor anat.gif

High Pressure Baroreceptors
Intact baroreceptors are extremely effective in preventing rapid changes in blood pressure from moment to moment or hour to hour.
Stretching of the baroreceptors as a result of increased blood pressure causes an increase in the activity of the vagal nerve through the brain stem . It also causes inhibition of the sympathetic outflow and ultimately leads to decreased heart rate and blood pressure. VICE VERSA.

1. Baroreceptors in the carotid sinus and aortic arch monitor blood pressure
2. Action potentials are conducted by the sensory nerves to the cardio regulatory and vasomotor centers in the medulla oblongata .
3. Increased parasympathetic stimulation of the heart decreases the heart rate.
4. Increased sympathetic stimulation of the heart increased the heart rate and stroke volume
5. Increased sympathetic stimulation of blood vessels increases vasoconstrictor.





nucleus tractus solitarius: **in the medulla oblongata of the brainstem.**

DEFINITION

temporary (transient)

Syncope is defined as a transient, self-limited loss of consciousness with an inability to maintain a postural tone that is followed by spontaneous recovery.

Caused by : a sudden drop in blood flow to the brain.

The term syncope excludes seizures, coma, shock, transient ischemic attacks or other states of altered consciousness.

PATHOPHYSIOLOGY OF SYNCOPE:

- DECREASED CEREBRAL PERFUSION IS COMMON TO ALL CAUSES OF SYNCOPE
- DECREASED CEREBRAL PERFUSION FOR AS LITTLE AS 3-5 SECONDS CAN RESULT IN SYNCOPE

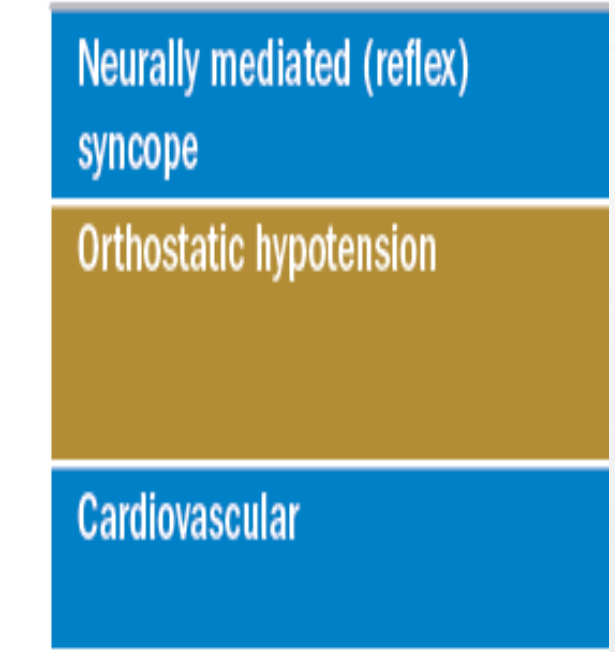
The underlying mechanism is global hypoperfusion of both the cerebral cortices .

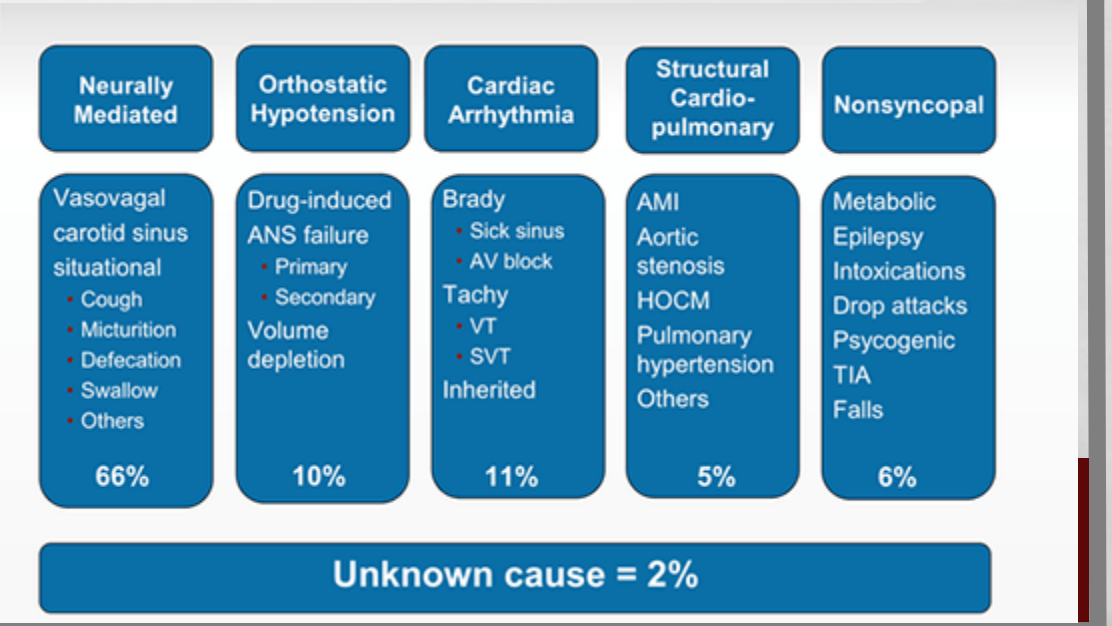
Decreased cerebral perfusion may occur as a result of decreased cardiac output and decreased systemic vascular resistance (venous pooling). Further pathophysiology is discussed according to type.



Types of syncope

1. Reflex syncope (neurally mediated)
1.1.Vasovagal
1.2 Situational
1.3. carotid sinus syncope.
2. Orthostatic
3. Cardiogenic





1. REFLEX SYNCOPE (NEURALLY MEDIATED)

PATHOPHYSIOLOGY

Enhancement of parasympathetic nervous system (vagal) tone and withdrawal of sympathetic nervous system tone. It occurs when there is a sudden drop in blood pressure and heart rate due to a reflex response of the autonomic nervous system.

1-

– Increased Parasympathetic activity
• Cardioinhibitory effect
Chronotropic (heart rate) effect

2 -

– Decreased Sympathetic activity
• Vasodilation
– Pooling of blood in the periphery, decreased venous return
- Drop in blood pressure during the fainting episode
– Negative Ionotropic (heart contraction)

3 – These contribute to » Decreased Cardiac Output the subsequent fainting (syncope).

The pathophysiology of cough syncope involves several mechanisms: **Increased intrathoracic pressure, Valsalva-like maneuver**, **Reflex-mediated changes**

Orthostatic: Baroreceptors may be involved initially no vagal

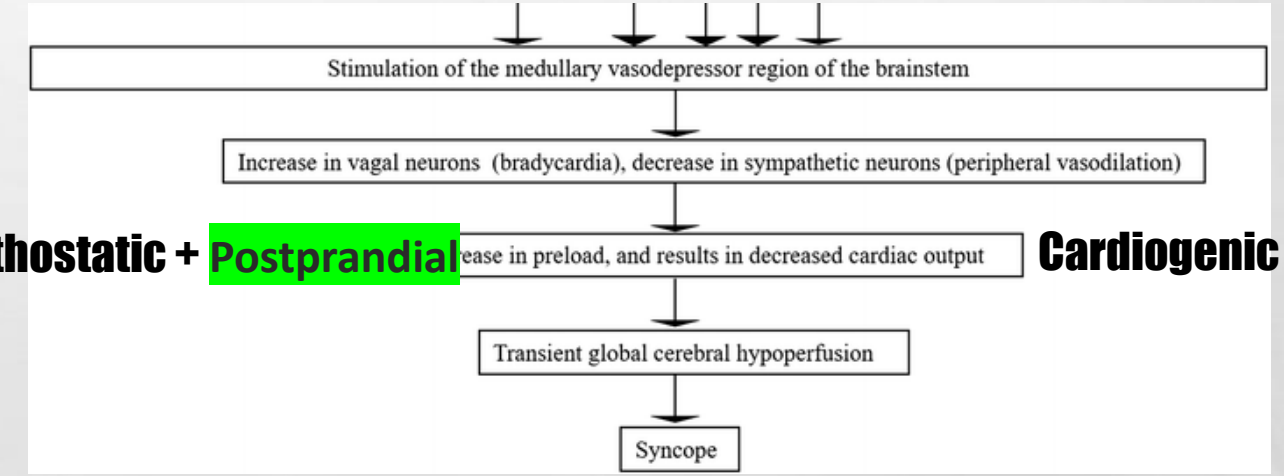
Vasovagal

Initial increased sympathetic tone

Baroreceptors+ carotid sinus

2-Orthostatic + Postprandial

Situational+ orthostatic → Cardiopulmonary receptors(low-pressure volume receptors)



1- Orthostatic + Postprandial

Cardiogenic

1.1. VASOVAGAL SYNCOPE (NEUROCARDIOGENIC SYNCOPE)

Is one of the most common causes of syncope ~ 66%.
The majority of patients with vasovagal syncope are females between 15 and 45 years old.

The pathophysiology of cough syncope involves several mechanisms: **Increased intrathoracic pressure, Valsalva-like maneuver, Reflex-mediated changes**
Orthostatic: Baroreceptors may be involved initially no vagal

Vasovagal
Initial increased sympathetic tone **Baroreceptors+ carotid sinus** **2-Orthostatic + Postprandial**

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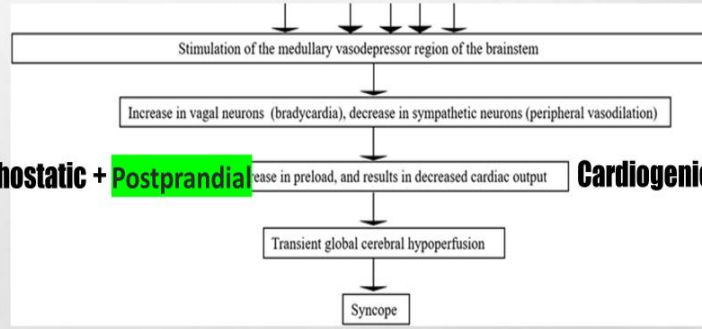


The sequence of events

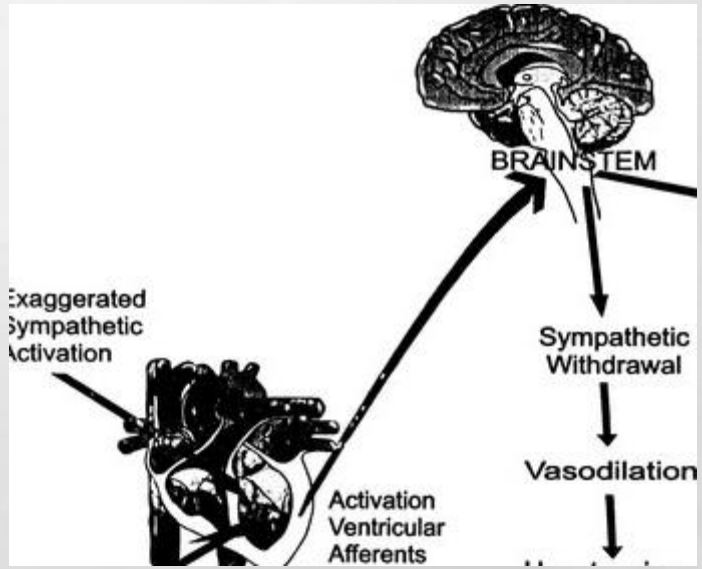
1.Trigger: An external trigger activates the autonomic nervous system and may involve increased sympathetic tone

2- Autonomic Response: The autonomic nervous system responds by causing blood vessels to dilate (vasodilation) and the heart rate to decrease (bradycardia). This results in a sudden drop in blood pressure and decreased blood flow to the brain.

3-Loss of Consciousness

4- Recovery

The vasovagal reaction results in an initial exaggerated sympathetic activation, followed by an abrupt decrease in sympathetic tone.



Cardiovagal function refers to the parasympathetic control of heart rate.The vagal response is a neurocardiogenic response that involves the central nervous system, peripheral nervous system, and cardiovascular system. The vagal response causes blood pressure and heart rate to drop suddenly.

In other cases, it is caused by

CAUSES OF VASOVAGAL SYNCOPE



Common external triggers **that initiate vagal response :**

- **Emotional stress; fear**
- **Having blood drawn; the sight of blood**
- Exposure to high temperatures
- **Pain and Trauma**
- prolonged periods of upright posture :

This results in an increased amount of peripheral venous pooling → the venous return to the heart falls → ventricular inotropy.

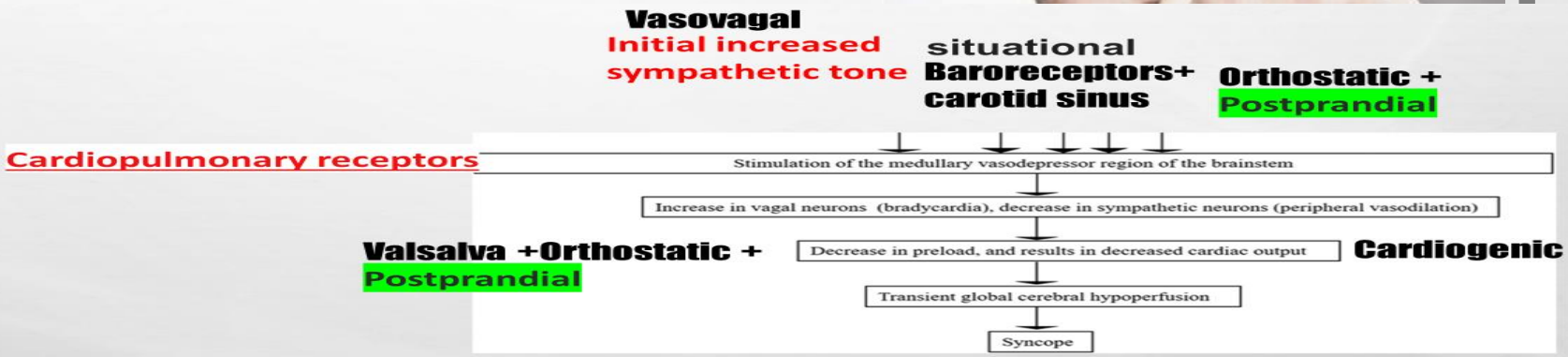
This hypercontractile state is believed to activate mechanoreceptors that would normally fire only during stretch.

The sudden increase in neural traffic to the medulla is thought to mimic the conditions seen in hypertension causing an apparent “paradoxical” decline in sympathetic activity that results in hypotension, bradycardia, and ultimately syncope.

1.2. SITUATIONAL

Valsalva maneuver





Situational syncope describes Abnormal autonomic nervous system responses to activities such as micturition, swallowing, exercise-induced or during Valsalva maneuver.
These causes tend to be more benign and do not predict poor outcomes.
Cough and lough syncope are types of situational syncope.

Cardiopulmonary receptors, also known as low-pressure volume receptors, are **located within the atria, ventricles, and pulmonary vasculature**. They are **sensitive to stretching of the vessel wall resulting from increased pressure from within**. Activation of these receptors can lead to the modulation of sympathetic nervous system activity, resulting in **vasodilation** and a decrease in **blood pressure**.

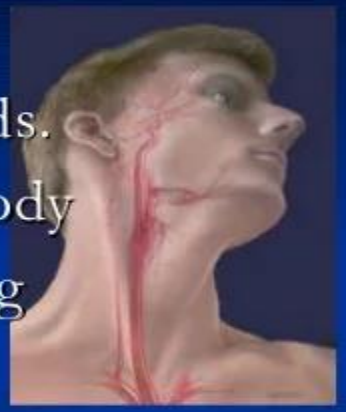
Situational Syncope

- Situations…
  - Cough
  - Micturition
  - Defecation
  - Swallow
  - Diving
  - Tussive or laughter syncope
    - More common in obese men over 40, smokers with chronic cough and COPD, children with asthma
  - More on micturition syncope
    - Older men, early morning, exacerbated by medications

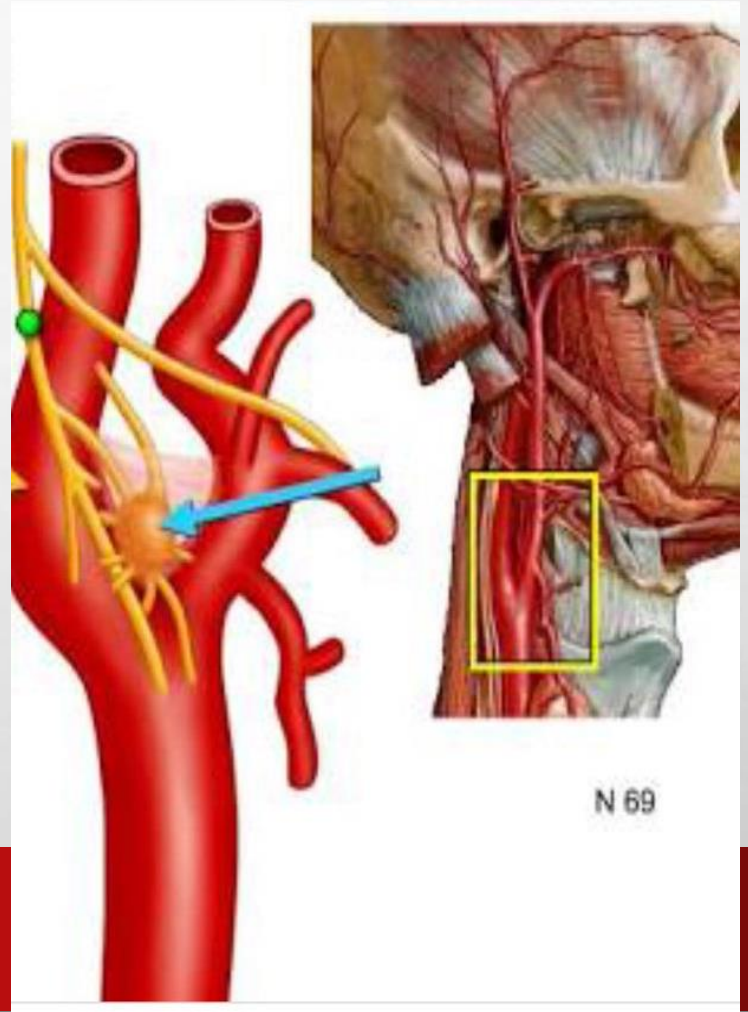
Triggering Event →
Increased Intrathoracic/Intra-abdominal Pressure →
Decreased Venous Return and Cardiac Output → activation of low-pressure volume baroreceptors →
Withdrawal sympathetic **Nervous System Response → vasodilation** and a decrease in **blood pressure →**
Transient reduced Cerebral Perfusion

1.3. CAROTID SINUS SYNCOPE

Carotid Sinus Syndrome



- Common in elderly/atherosclerotic ds.
- Sensitive baro receptors at carotid body
- Any pressure i.e. sudden neck turning or tight collar /shaving
- slowing of heart rate and fall in blood pressure
- Poor brain perfusion → Syncope



2. ORTHOSTATIC SYNCOPE

Orthostatic hypotension is defined as a sustained decline of ≥ 20 mmHg in systolic or ≥ 10 mmHg in diastolic blood pressure upon standing.

Orthostatic hypotension occurs as a result of impaired autonomic reflexes towards immediate gravitational pooling of about 500 to 1000ml of blood to the lower extremities. Reduced Venous Return to Heart → Activation of Baroreceptors → Reflexive Decrease in Heart Rate ar Blood Pressure → Syncope

Exacerbating factors:

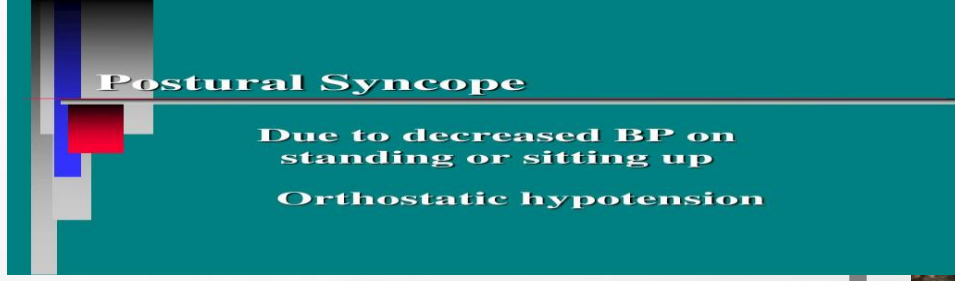
1-Alcohol & drugs such such as vasodilators and antidepressants block orthostatic reflexes.

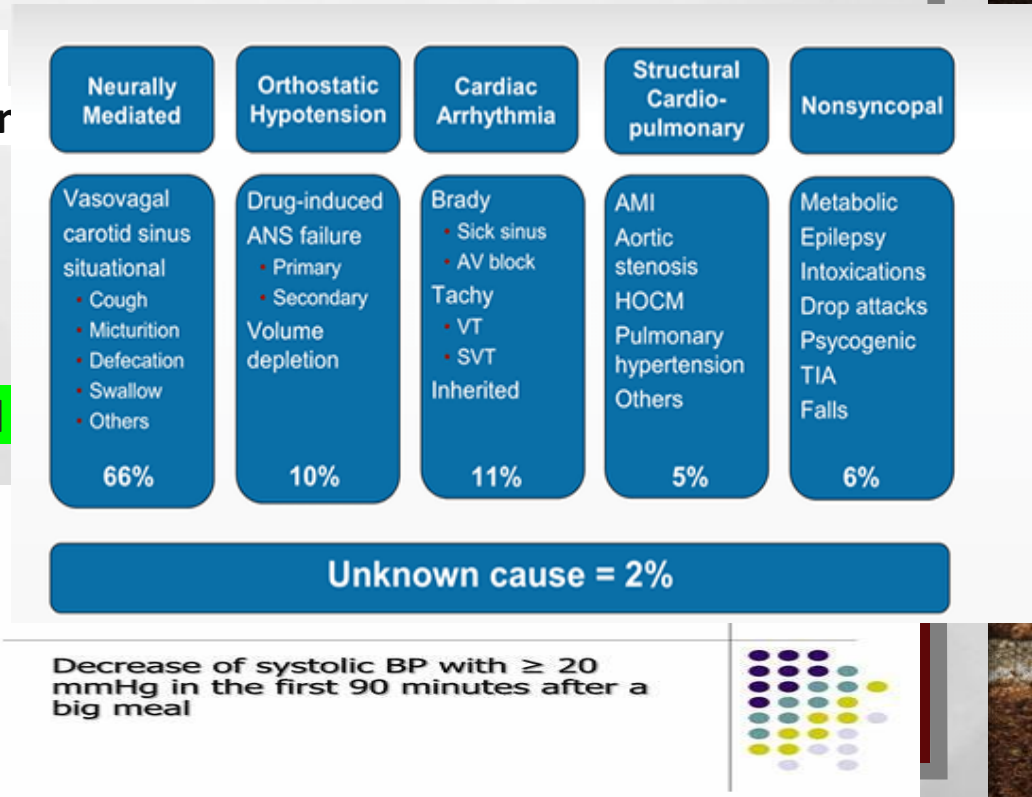
2- Volume depletion due decreased intravascular volume , e.g blood loss and dehydration (vomiting, diarrhea, poor oral intake, and diuretics).

3- Secondary autonomic insufficiency can be due to diabetes, uremia, or spinal injury.

4- Postprandial syncope.

5- Pure autonomic failure (PAF) is an uncommon, sporadic neurodegenerative condition marked by a steadily declining autonomic regulation





Orthostatic stress

↓

Decreased venous return to the heart

↓

Decreased cardiac output

↓

Activation of baroreceptors

↓

Increased sympathetic activity

↓

Increased heart rate and
force of ventricular contraction
in a partially empty ventricle

↓

Stimulation of mechanoreceptors
in the heart

↓

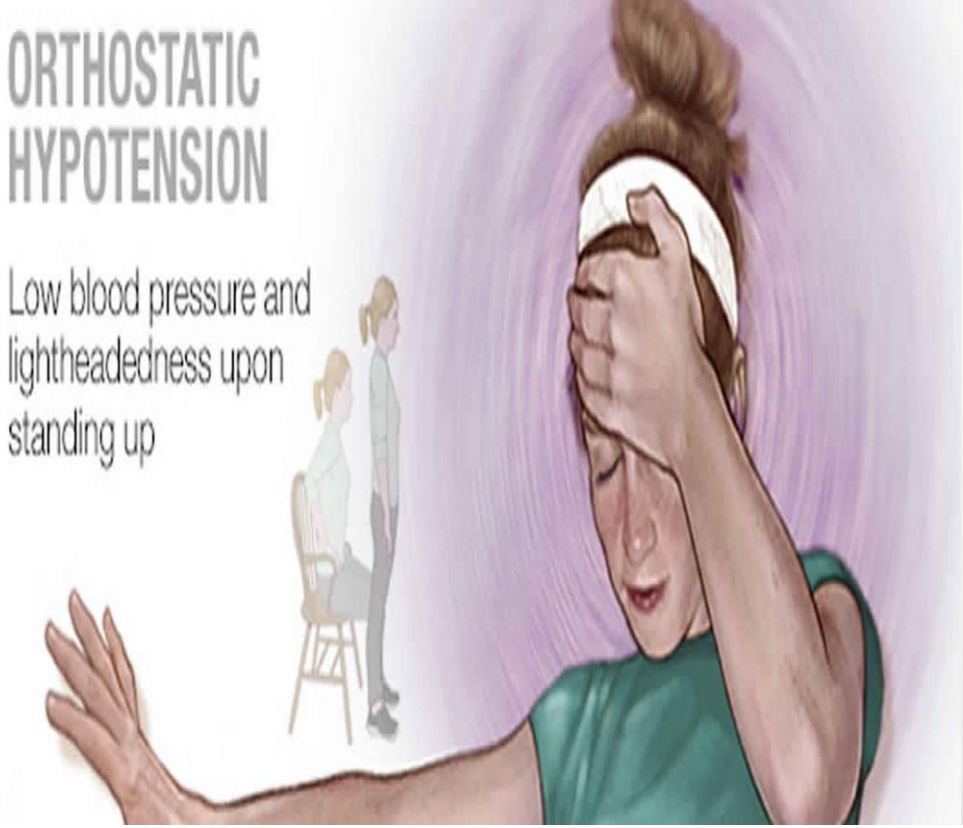
Brainstem

↓ ↓

Sympathetic withdrawal | Increased parasympathetic output

↓ ↓

Syncope



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There is no initial vagal hyperactivity associated with this venous pooling and this distinguishes orthostatic syncope from neurocardiogenic syncope.