Blood Pressure Management in the Elderly

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Disclosure Information

* **I have no financial relationships to disclose**

Objectives

* To define the terms orthostatic hypotension, postprandial hypotension, and autonomic failure syndrome.
* To be able to diagnose orthostatic hypotension, postprandial hypotension, and autonomic failure syndrome.
* To be able to treat orthostatic hypotension, postprandial hypotension, and autonomic failure syndrome.
* To understand complications associated with orthostatic hypotension, postprandial hypotension, and autonomic failure syndrome.

Hypotension unawareness

* Parallels hypoglycemia in terms of symptoms and severity
* Blood pressure drops without usual associated symptoms
* Inability to sense symptoms from either deficit in neural network or impaired afferent signaling or impairment
* Patients harbor this disorder for an extended period before developing insensitivity
* Absence of warning signs presents a **serious risk** to the patient

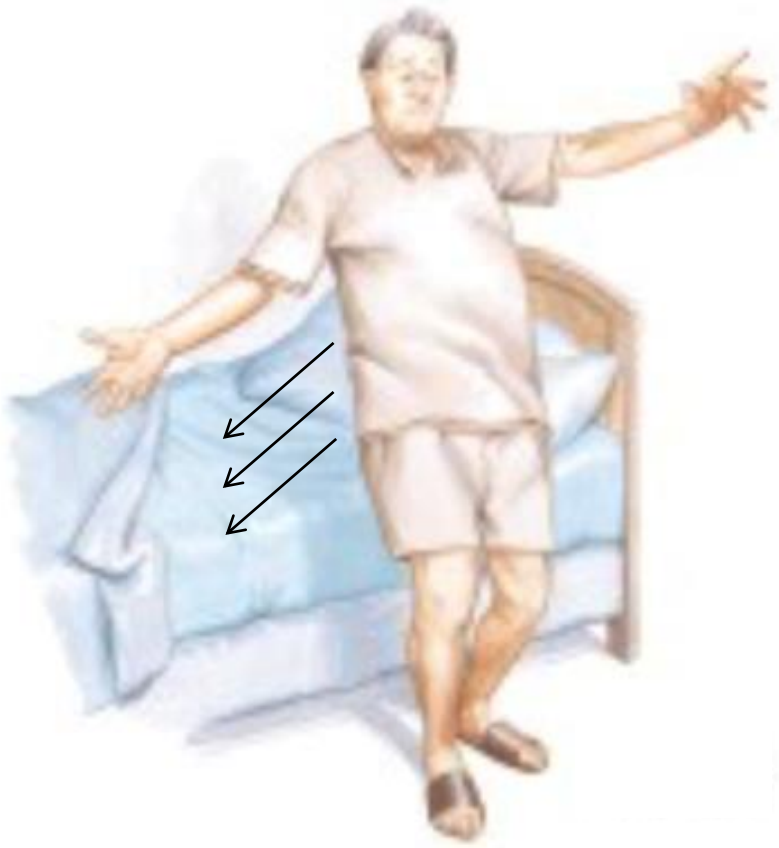
Orthostatic Hypotension: Definition

Within three minutes of standing when compared to seated or supine:

Decrease of systolic blood pressure of at least 20 mm Hg

or

Decrease of diastolic blood pressure of at least 10 mm Hg



Orthostatic Hypotension: Causes

* Medications
* Volume depletion
* Fluid depletion or food ingestion
* Increased temperature
* Physical deconditioning
* Autonomic neuropathy
* Systolic hypertension

Orthostatic Hypotension

Physiologic mechanisms:

Normal hemodynamic response to changes in posture:

* Baroreceptor reflex
* Increase in sympathetic outflow
* Increase peripheral vascular resistance
* Increase venous return
* Increase cardiac output

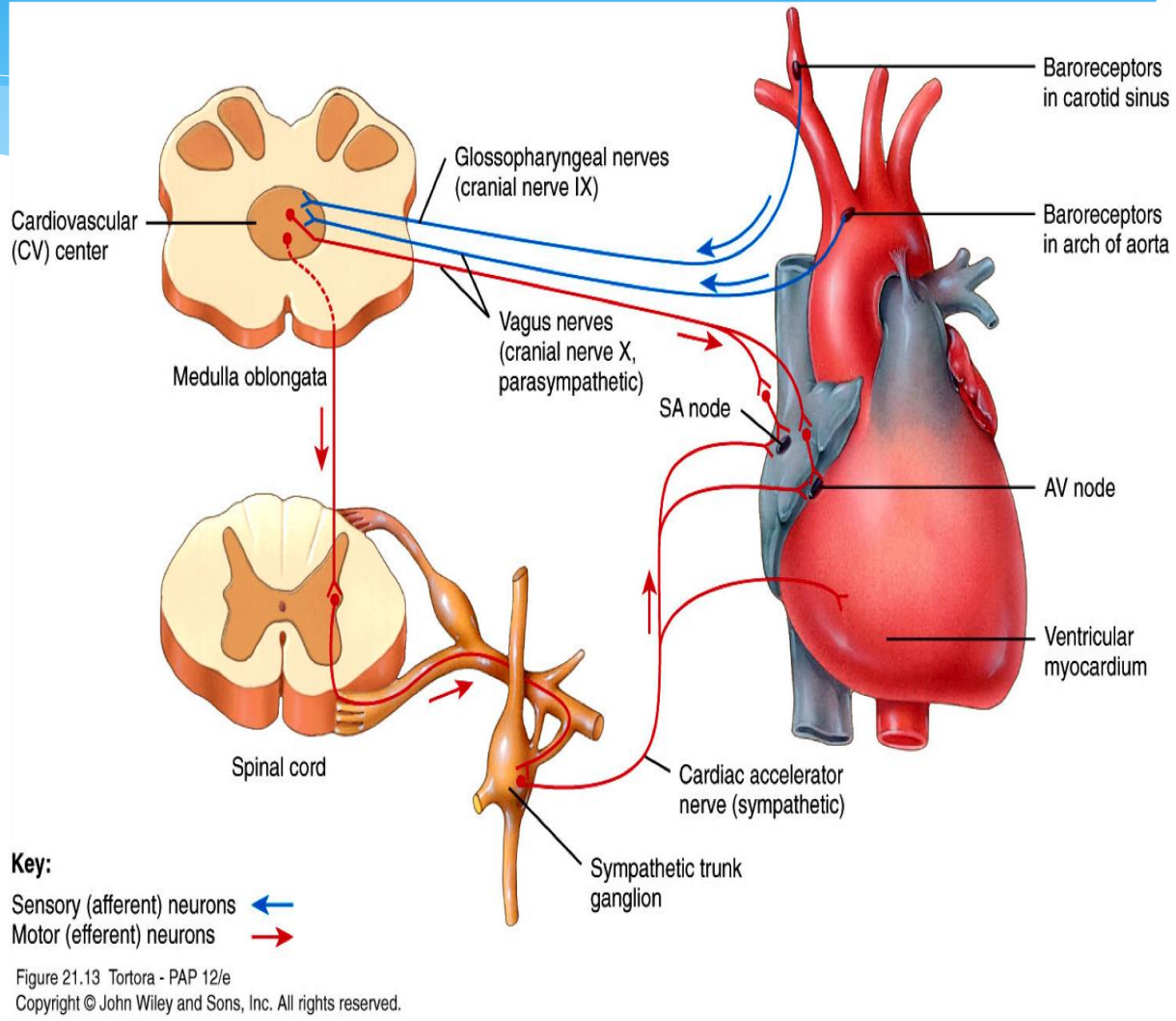


Figure 21.13 Tortora - PAP 12/e

Aging Physiology

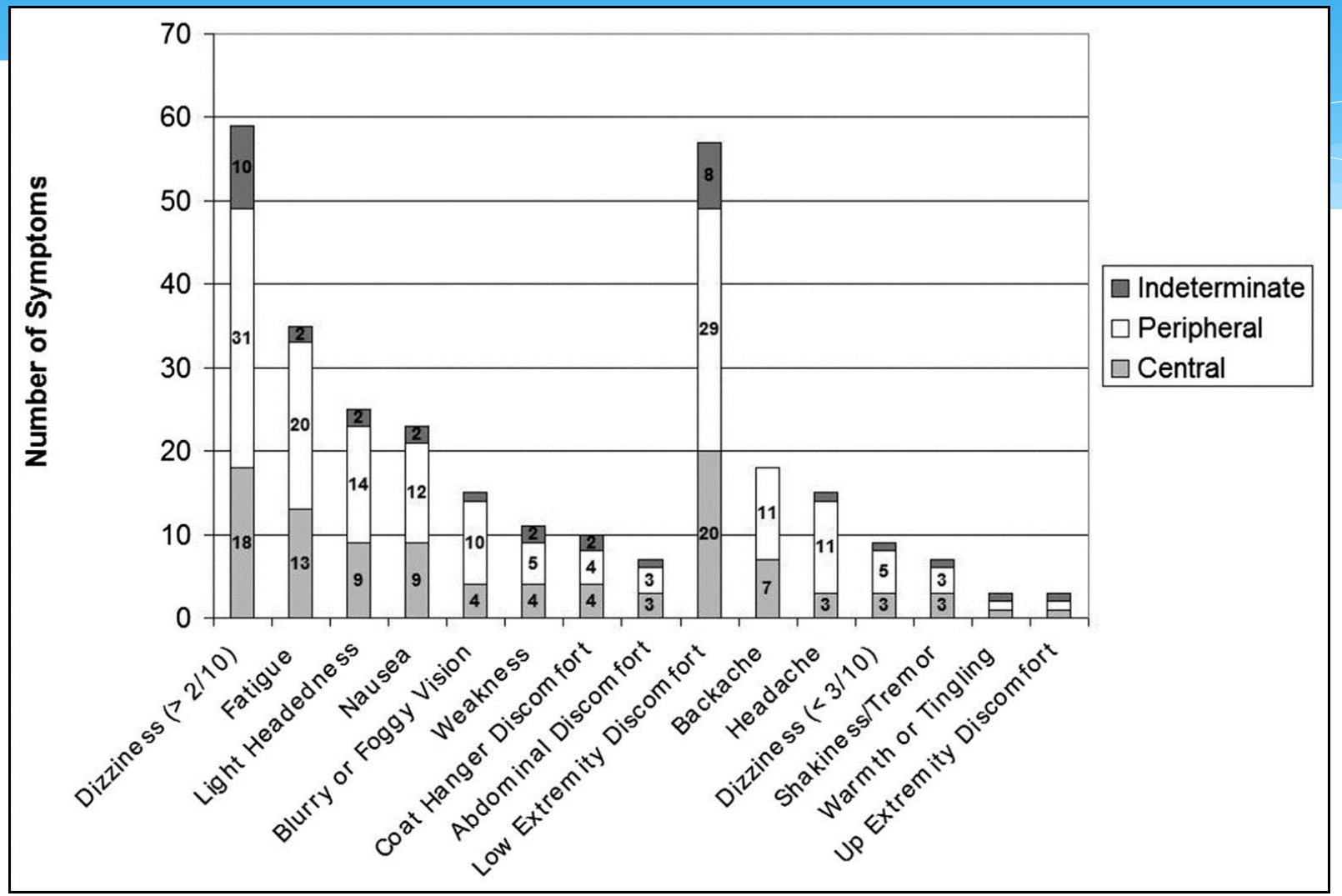
As we age, the following changes hemodynamic occur:

* Increased thickness of the intima/media
* Increased vascular stiffness
* Increased blood pressure variability
* Impaired blood pressure homeostasis
* Decreased vascular compliance
* Decreased baroreceptor sensitivity

Orthostatic Hypotension

* Occurs in 20% to 50 % of the elderly
* Often seen in conjunction with **systolic hypertension**
  * Further blunting of the baroreceptor reflex, decrease vasculature and ventricular compliance
  * Chronic hypertension increases upper and lower limits of cerebral blood flow regulation; small drops in blood pressure can lead to **severe** debilitation (stroke, MI, increased mortality, and falls)
* Can produce **syncope**
* Marker for mortality: Long-term effects → **SEVERE** debilitation

Orthostatic Hypotension: Symptoms



Reference: Arbogast, S., et. al., American Journal of Medicine, Vol 122, No 6, June 2009, page 576

Orthostatic Hypotension: Symptoms

Common symptoms:

* Lightheadedness
* Visual blurring
* Dizziness
* Generalized weakness
* Fatigue
* Palpitations
* Nausea
* Abdominal discomfort
* Poor memory
* Asymptomatic
* Coat hanger headache

Diagnosis

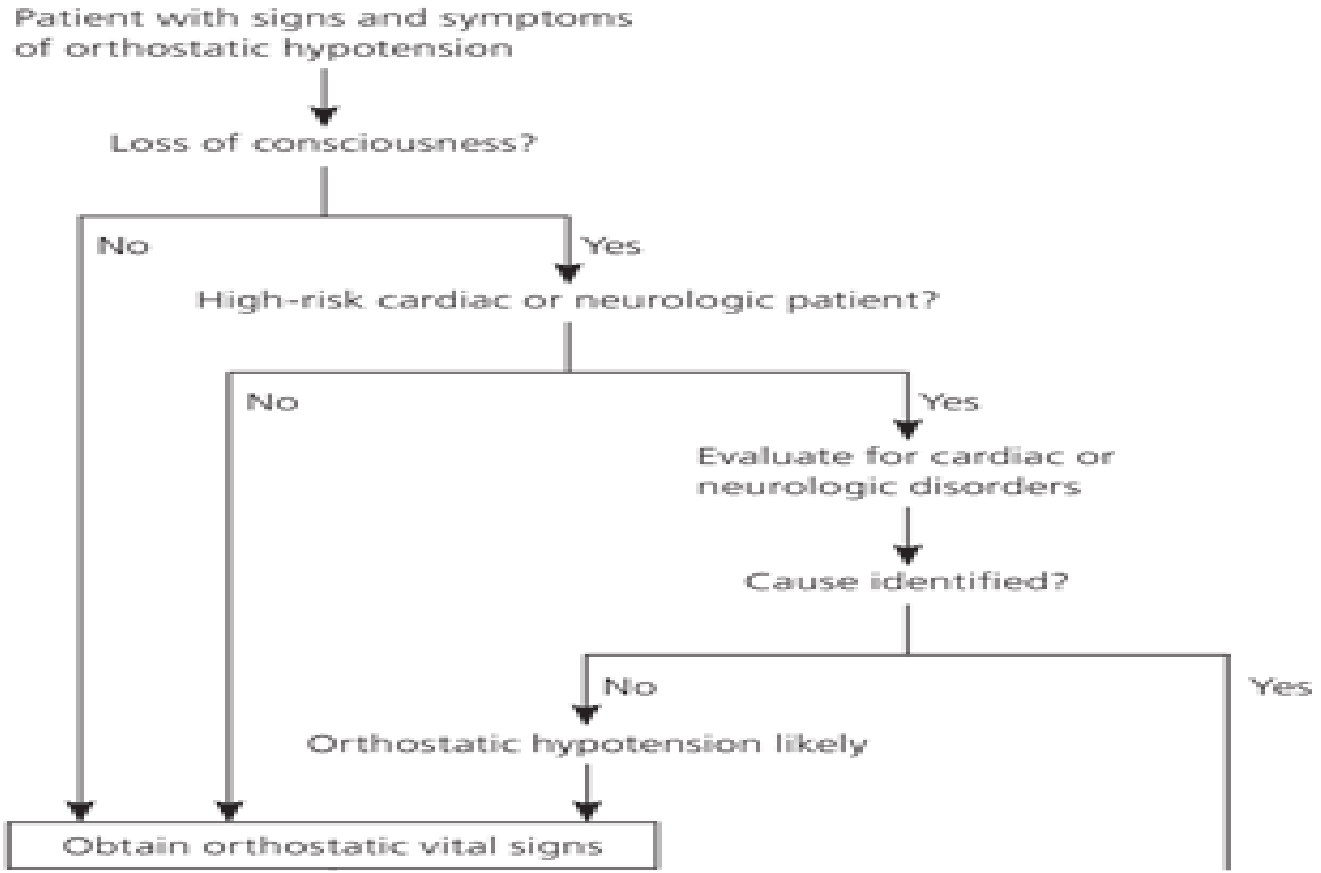
* History, history, history!

* Head-up tilt table test:

  Consider in:

  * Parkinsonian syndrome (Multiple system atrophy or Parkinson disease)
  * Peripheral neuropathy (diabetes, amyloidosis, small fiber neuropathy, pure autonomic failure)

Diagnostic flow chart in the acute care setting:



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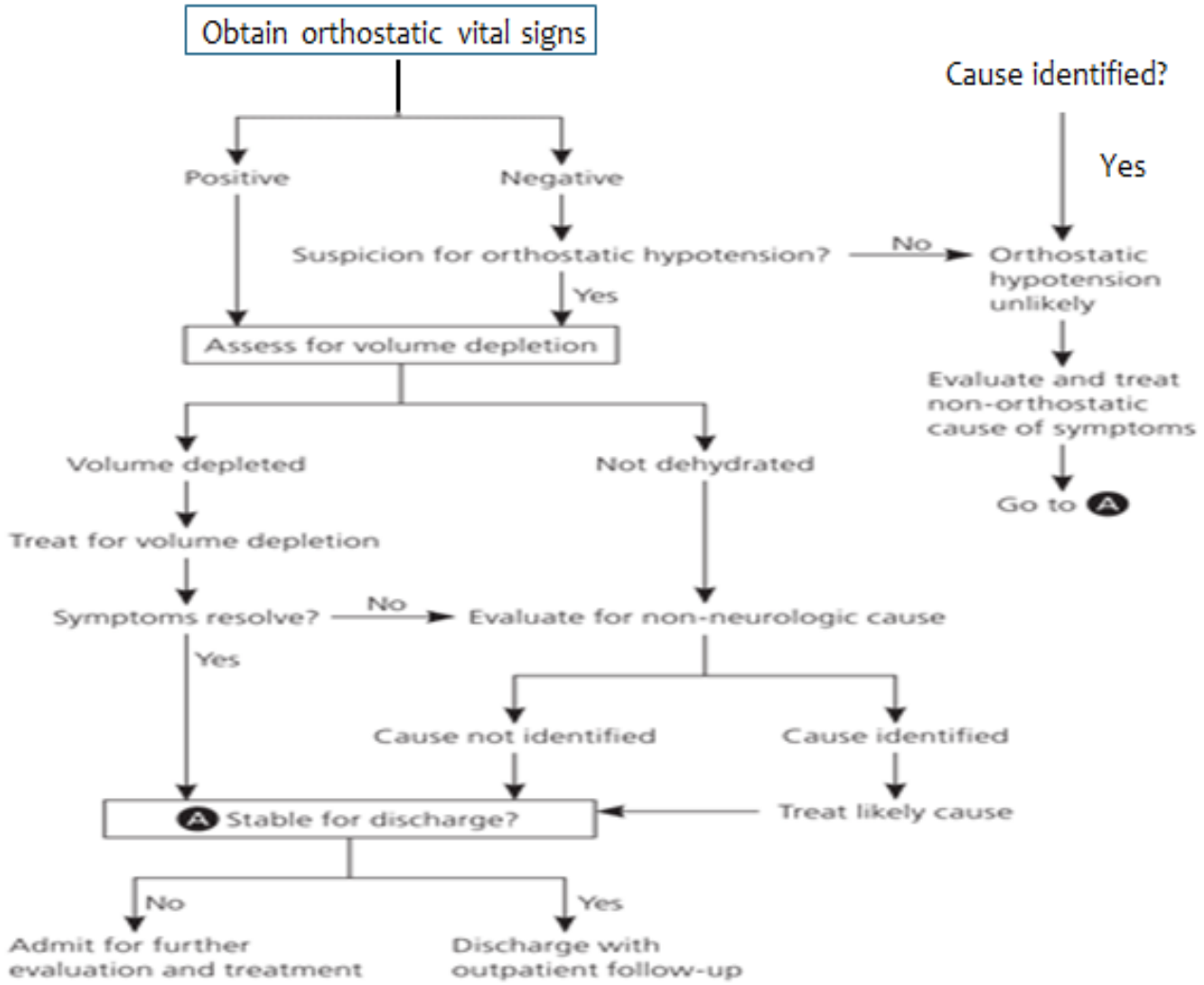


Head-up tilt-table testing

* Quiet room with a temperature of 68°F to 75°F.
* Patient should be supine and rest for 5 minutes before testing is started.
* Continuously monitor heart rate. Measure blood pressure at regular intervals.
* The table should be slowly elevated to between 60° to 80° for 3 minutes.
* Considered **positive** if systolic blood pressure falls 20 mm Hg below baseline **or** if diastolic blood pressure falls 10 mm Hg below baseline.
* If symptoms occur, the patient should be returned to the supine position immediately.
* **Various procedures exist for performing, but key findings are the same consistently.**

Orthostatic Hypotension: Treatments

Non-pharmacologic treatments:

* Stop or decrease medications (beta blockers, CCBs, ACEIs/ARBs)
* Avoid warm events and straining
* Stretch legs and stand up slowly
* **Increase** sodium intake
* Waist-high compression stockings
* Elevate head of bed 5-20°

Orthostatic Hypotension: Treatments

Pharmacologic treatments:

* Fludrocortisone – volume expansion
* Midodrine – alpha agonist – increases vascular tone
* NSAIDs – inhibit prostaglandin synthesis – increases systemic vascular resistance (caution: GI bleeds)

Postprandial Hypotension

* **Diagnosis/Definition:** Decrease in systolic blood pressure of ≥ 20 mm Hg or a decrease in systolic blood pressure below 90 mm Hg from a pressure of ≥ 100mm Hg within 2 hours after a meal.

Postprandial Hypotension: Epidemiology

* Associated with syncope, falls, coronary events, and stroke.
* First described in 1977 in a patient with severe Parkinson disease
* Common among **institutionalized** geriatric patients
* Prevalence in institutionalized elders is approximately 25%-38%
* This is an under-recognized cause of syncope

Postprandial Hypotension

* **Mechanism:** poorly understood
* Limited evidence available
* Blood in the splanchnic circulation pools following a meal combined with inadequate sympathetic outputs to maintain cardiac output and systemic vascular resistance
* Other possible mechanisms include insulin-induced vasodilation and release of vasodilatory gastrointestinal peptides

Postprandial Hypotension: Risk Factors

Table 1 Risk Factors for Postprandial Hypotension

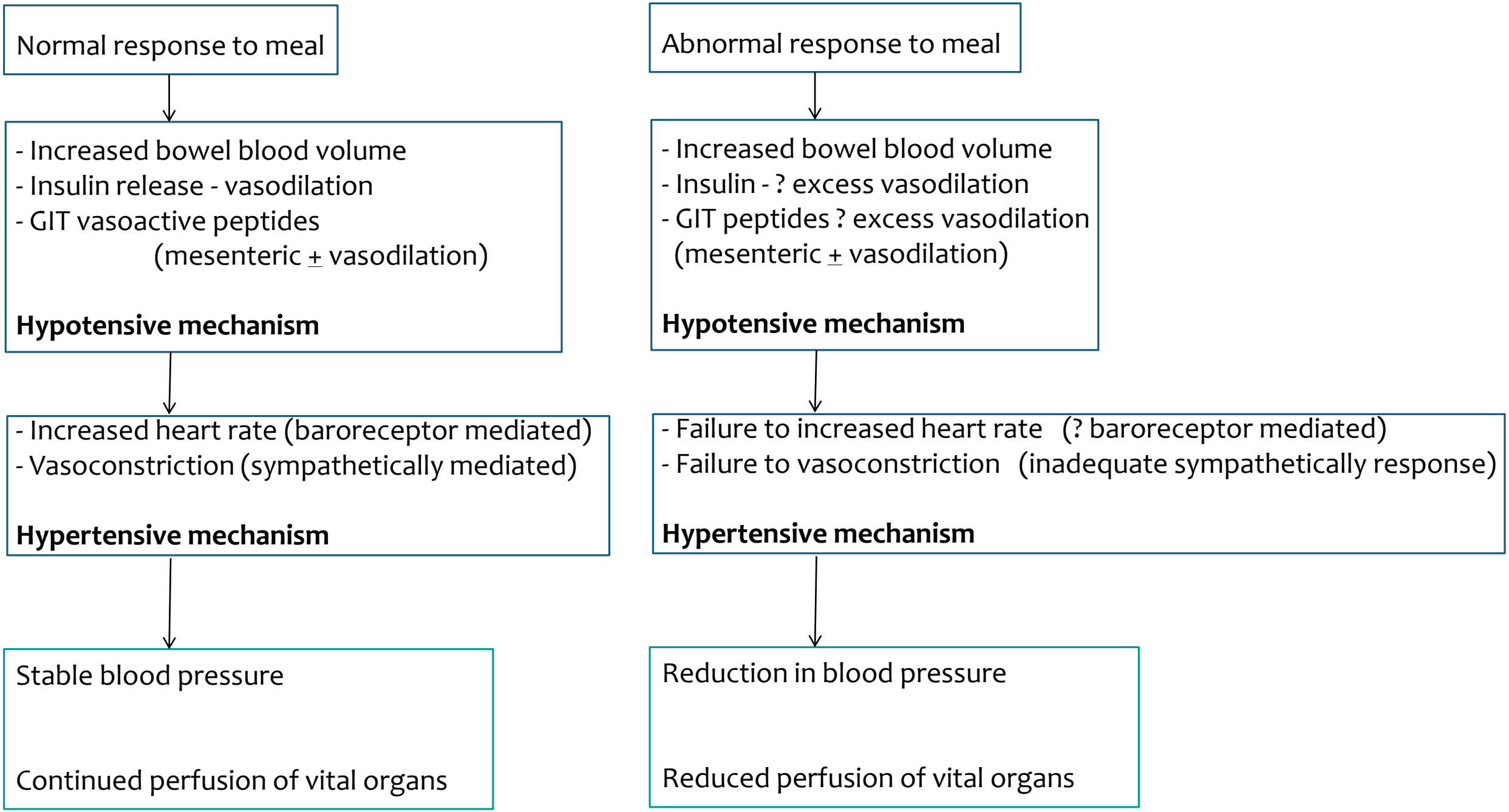
Medications	Polypharmacy (>3 medications)
	Diuretics
Meals	Carbohydrate-rich meals
	Breakfast
	Hot meals
Comorbid conditions	Diabetes mellitus
	Autonomic dysfunction
	Parkinson disease
	Hypertension
	End-stage renal disease on hemodialysis
	Fragile X mutation

Postprandial Hypotension: Treatment Options

Table 2 Treatment Options for Postprandial Hypotension

Nonpharmacologic Modifications	Pharmacotherapy		
	Drug	Dose	Common Side Effects
Drink water before meals	Caffeine	60-200 mgs	Restlessness, palpitations, insomnia
Decrease carbohydrate intake	Alpha-glucosidase inhibitors		Diarrhea, flatulence
Eat frequent, smaller meals	Acarbose	100 mg	
Assume a recumbent or sitting position after a meal	Voglibose	200 μg	
	Guar gum	4 gm	Diarrhea, flatulence, abdominal pain
	Octreotide	50 μg	Arrhythmia, abdominal and injection site pain

Optimal therapy has not been defined.


Normal response to meal
- Increased bowel blood volume
- Insulin release - vasodilation
- GIT vasoactive peptides
(mesenteric ± vasodilation)
Hypotensive mechanism
- Increased heart rate (baroreceptor mediated)
- Vasoconstriction (sympathetically mediated)
Hypertensive mechanism
Stable blood pressure
Continued perfusion of vital organs
Abnormal response to meal
- Increased bowel blood volume
- Insulin - ? excess vasodilation
- GIT peptides ? excess vasodilation
(mesenteric ± vasodilation)
Hypotensive mechanism
- Failure to increased heart rate (? baroreceptor mediated)
- Failure to vasoconstriction (inadequate sympathetically response)
Hypertensive mechanism
Reduction in blood pressure
Reduced perfusion of vital organs

Postprandial Hypotension: Treatment Options

- Caffeine acts as an adenosine receptor antagonist.
- Acarbose is an α-glucosidase inhibitor which has been known to attenuate postprandial hypotension in a small trial of patients with autonomic failure.
- Controversial due to conflicting studies.

Postprandial Hypotension: Treatment Options

- Guar gum reduces the magnitude of the fall in blood pressure after oral glucose reducing gastric emptying and glucose absorption; may represent a novel approach to the treatment of postprandial hypotension.

- Octreotide – somatostatin analogue that increases splanchnic vascular resistance preventing pooling of blood in the gut; must be given SQ 30 minutes before each meal; expensive; often leads to diarrhea and pain at the injection site; reserved for severely symptomatic patients.

Autonomic Failure Syndrome

- **Definition**: progressive neurodegenerative disease causing parkinsonism, cerebellar, pyramidal, autonomic, and urological dysfunction in any combination characterized by autonomic failure with orthostatic hypotension and movement disorders

- Lots of other names: Shy-Drager syndrome, multi-system degeneration, multi-system atrophy, idiopathic autonomic failure, and idiopathic orthostatic hypotension

Autonomic Failure Syndrome

- **Classification:** primarily by predominant motor defect

  Parkinsonian form: MSA-P (also called striatonigral degeneration)

  Cerebellar form: MSA-C (also called olivopontinecerebellar degeneration)
  Mixed/Multiple form: MSA-M (has features of MSA-P/MSA-C)

- **Mechanism:** lack of baroreflex buffering of acute changes in blood pressure; patient is unable to elevate plasma norepinephrine in response to standing; supine hypertension

Autonomic Failure Syndrome: Symptoms

* Urinary retention
* Erectile dysfunction
* Syncope
* Fecal incontinence
* Constipation
* Truncal Ataxia

Autonomic Failure Syndrome: Diagnosis

* Autopsy results: Neuropathologic association with neurodegenerative changes in striatonigral or olivopontocerebellar structures

Autonomic Failure Syndrome: Treatment for Orthostatic hypotension

- Non-pharmacologic therapy:
  - Water intake may improve systolic pressure (cohort study, 28 persons with autonomic failure showed a mean increase in 33 mm Hg after drinking 480 mL water)
- Pharmacologic therapy:
  - Midodrine 10 mg BID-TID (randomized trial without intention to treat analysis, 171 pts, 40 with MSA, standing blood pressure improved at all time points, $p < 0.001$ – Ref: JAMA 1997 Apr 2;277(13):1046)
  - Droxidopa – approved by the FDA in 2014 with orphan product designation to treat neurogenic orthostatic hypotension; boxed warning risk of supine hypotension

Autonomic Failure Syndrome: Supine Hypertension

- Caused by residual sympathetic tone acting on hypersensitive adrenoreceptors and unopposed by loss of baroreflex buffering capacity

- Proposed mechanism:
  - Increase in intravascular volume
  - Increase in cardiac output
  - Increase in vascular tone
  - Combination of the above

Autonomic Failure Syndrome: Supine Hypertension

- Treatment:
  - Avoid supine position by resting in reclining chair if tired
  - Remove support stockings when supine
  - Sleep in head-up tilt position (head of bed elevated 6-9 inches) to reduce nocturnal sodium loss and diurnal orthostatic hypotension
  - Take last dose of medication ≥ 4 hours before bedtime
  - Occupational therapy

References

- 1. Arbogast, S., et. al., American Journal of Medicine, Vol 122, No 6, June 2009, page 576
- 2. Sarasin FP, Louis-Simonet M, Carballo D, et al. Prospective evaluation of patients with syncope: a population-based study. *Am J Med.* 2001;111(3):177–184.
- 3. Lanier JB, Mote MB, Clay EC, et. Al. Evaluation and Management of Orthostatic Hypotension. *Am Fam Physician.* 2011 Sep 1;84(5):527-536.

Question 1

Mrs. Smith is an 82-year-old female with a history of Parkinson disease. She presents following a hospital admission for an unwitnessed fall during which she was diagnosed and treated for congestive heart failure. Her current medications include aspirin 81 mg daily, carbidopa-levodopa QID, carvedilol 6.25 mg BID, iron sulfate 325 mg daily, fludrocortisone 0.1 mg daily, lisinopril 5 mg daily, KCl 20 mEq daily, allopurinol 300 mg daily, midodrine 5 mg TID, pravastatin 80 mg QHS, and tamsulosin 0.4 mg daily.

What is the next best step in the management of this patient?

A. 2D echo to determine ejection fraction
B. Bilateral lower extremity venous dopplers
C. Bilateral carotid artery ultrasound
D. Measurement of orthostatic blood pressures
E. Add lasix 40 mg daily to optimize diuresis in the setting of congestive heart failure

Question 1 – part 2

Mrs. Smith's orthostatic blood pressures are determined to be: 150/72 supine, 140/88 sitting, and 128/64 standing, with a reading of 158/88 upon return to the supine position.

Which of the following is not associated with this condition?

A. CVA
B. MI
C. Decreased mortality
D. Falls
E. Syncope

Question 2

Mr. Thomas is a 90-year-old man with a past medical history of diabetes and Parkinson disease who lives in a skilled nursing facility. You are seeing him today for monthly follow up. While discussing his care with the nursing staff, you learn that three days ago, after eating a large dinner, he was noted to have become flaccid and difficult to arouse 30 minutes after dinner. A review of his medications notes that he is on levemir 45 units at bedtime, metformin 1000 mg BID, sinemet 25/100 mg QID, valsartan/HCTZ 320/25 mg daily, norvasc 5 mg daily, and novolog 10 units QAC TID. Which of the following is true regarding this patient's symptoms?

A. Octreotide is the definitive treatment for his symptoms.

B. The degree of postprandial hypotension is inversely coordinated with the number of cardiovascular and psychotropic medications a patient is prescribed.

C. Blood in the splanchnic circulation pools during times of fasting combined with inadequate sympathetic output to maintain cardiac output and systemic vascular resistance lead to symptoms described above.

D. Insulin induces vasoconstriction, decreasing the blood supply in the splanchnic circulation and ultimately decreasing cardiac output and systemic vascular resistance

E. Age related changes increases in blood pressure that are associated with less compliant vasculature may be partly responsible for the presence of postprandial hypotension in health elderly