

APTA Academy of Cardiovascular & Pulmonary Physical Therapy^{*}

ADULT VITAL SIGN INTERPRETATION IN ACUTE CARE GUIDE 2021

Joint Task Force of APTA Acute Care and the Academy of Cardiovascular & Pulmonary Physical Therapy of the American Physical Therapy Association

Approved by the Board of Directors of the APTA Academy of Cardiovascular & Pulmonary Physical Therapy March 2021

Approved by APTA Acute Care Physical Therapy Board of Directors April 2021



Disclaimer: This document is intended to provide education and guidance on vital sign interpretation for adult populations but does not replace sound clinical judgment. While vital signs are the primary determinants to assess patient readiness for mobility and response to activity, these values need to be corroborated with other patient findings. Although recommendations are provided for various conditions, individual patient vital signs and co-morbidities should guide clinical decision making. Any questions of medical stability should be discussed with the healthcare team. It is recommended to read the introduction prior to going to any other section of this document for important contextual information.



APTA Acute Care and the Academy of Cardiovascular & Pulmonary Physical Therapy Task Force Committee Leaders

Kimberly Levenhagen, PT, DPT

Wound Care Certified Certified Lymphedema Therapist Fellow, National Academies of Practice

Traci Norris, PT, DPT

Board-Certified Clinical Specialist in Geriatric Physical Therapy

Ann Fick, PT, MS, DPT

Board-Certified Clinical Specialist in Cardiovascular and Pulmonary Physical Therapy

Angela Abeyta Campbell, PT, DPT

Board-Certified Clinical Specialist in Cardiovascular and Pulmonary Physical Therapy

Ethel Frese, PT, DPT

Board-Certified Clinical Specialist in Cardiovascular and Pulmonary Physical Therapy Catherine Worthingham Fellow of the American Physical Therapy Association

Morgan Lopker, PT, DPT

Ashley Poole, PT, DPT

Board-Certified Clinical Specialist in Cardiovascular and Pulmonary Physical Therapy

Task Force Committee Members

Leonard Arguelles, PT, DPT

Board-Certified Clinical Specialist in Cardiovascular and Pulmonary Physical Therapy

Ma Rodelyn Berdin, PT, DPT

Fellow of the Texas Physical Therapy Association

Kathryn Brito, PT, DPT

Katharine Coombes, PT, DPT

Jamie Dyson PT, DPT

Amandeep Gill PT, DPT

Christiane Perme, PT

Board-Certified Clinical Specialist in Cardiovascular and Pulmonary Physical Therapy Fellow of the American College of Critical Care Medicine

Komal Shah PT, DPT

Board-Certified Clinical Specialist in Neurologic Physical Therapy

Kathy Swanick PT, DPT

Board-Certified Clinical Specialist in Orthopedic Physical Therapy



TABLE OF CONTENTS

| INTRODUCTION ⁵ |
|--|
| GENERAL VITAL SIGN INTERPRETATION ADULT POPULATION6 |
| VITAL SIGN INTERPRETATION IN THE INTENSIVE CARE UNIT |
| ICU Support Devices and Effects on Vital Signs 12 |
| Sepsis |
| SPECIAL POPULATION CONSIDERATIONS ¹⁵ |
| Acute Coronary Syndrome/Myocardial Infarction |
| Heart Failure |
| Peripheral Arterial Disease •••••••••••••••••••••••••••••••••• |
| Aortic Aneurysm |
| Venous Thromboembolic Disease 17 |
| Lung Disease •••••••••••••••••••••••••••••••••• |
| Diabetes Mellitus |
| Oncologic Conditions |
| Neurologic Conditions |
| PHARMACOLOGY 24 |
| REFERENCES |



Introduction

The purpose of obtaining vital signs (VS) is to detect and monitor physiologic states and assess activity responses to aid in exercise prescription. VS determine patient risk for adverse events, such as cardiovascular episodes and syncope. Compared to outpatient settings, hospitalized patients present more often with abnormal or labile VS and are at a higher risk of immediate events requiring acute care physical therapists to assess and monitor VS with greater frequency. Assessing pulse rate (PR), respiratory rate (RR), temperature, and blood pressure (BP) are essential components of a systems review in a physical therapy (PT) examination.¹ Additionally, tissue oxygenation, measured by pulse oximetry, is necessary to assess hypoxemia. Physical therapists should correlate current VS values with other data points such as symptoms, baseline VS, medication schedule, lab values, and comorbidities when making decisions about patient care.

Normal Values:

- Values presented are for adults
- · Baseline health and fitness influence VS, particularly PR and BP

Frequency of VS Monitoring:

- · VS at rest help determine readiness for PT intervention, in conjunction with other findings
- VS during PT interventions and recovery assess hemodynamic and oxygenation responses and stability
- PT providers (physical therapists and physical therapist assistants) should follow the institution policy and procedures regarding activity
- PT providers should monitor VS for adverse reactions, especially during medication adjustments, transfusions, or other procedures
- · Critical Care: (refer to Vital Sign Interpretation in the Intensive Care Unit for more details)
 - Intensive care units (ICU) involve more specialized monitoring and invasive treatments that cannot be handled safely in the general wards/floors
 - VS should be assessed continually during PT interventions in the ICU

Accuracy of Measurement:

- Accuracy of VS measurement and documentation is of extreme importance, but education on techniques is beyond this guideline's scope
- Poor technique can cause inaccurate BP measurement²
- The Academy of Cardiovascular and Pulmonary Physical Therapy offer several videos
- (#VitalsAreVital) to guide PT providers and students to perform VS accurately
- VS are dynamic measurements that can be influenced by many factors
 - Caffeine, smoking, stress, agitation/delirium, and other factors can contribute to an elevated PR, RR, and BP
 - Medications and administration timing can influence VS
 - BP is exceptionally dynamic; interpretation in the context of trends is best
 - Documentation should include the patient position, extremity, or activity during VS measurement (i.e. sitting, supine, rest, mobility)

Vital Sign Interpretation for General Adult Population

| Blood Pressure (BP) | | | | | |
|--|-----------|-----|---------|--|--|
| Blood pressure (BP) = Cardiac Output (CO) x Total Peripheral Resistance (TPR) CO = Stroke Volume (SV) x Heart Rate (HR) | | | | | |
| Categories ^{3*} Systolic (SBP) (mmHg) Diastolic (DBP) (mmHg) | | | | | |
| Normal | < 120 | and | < 80 | | |
| High-normal/Elevated/ Pre-hypertensive | 120 - 129 | and | < 80 | | |
| Stage 1 Hypertension | 130 - 139 | or | 80 - 89 | | |
| Stage 2 Hypertension | ≥ 140 | or | ≥ 90 | | |
| Hypertensive Crisis ≥ 180 and/or > 120 | | | | | |

*Other organizations (International Society of Hypertension, American College of Physicians, American Academy of Family Physicians, 8th Joint National Committee) not listed in this table have guidelines regarding hypertension categories. Refer to these references for additional evidence.¹⁵⁰⁻¹⁵²

- **Hypotension**: < 80 mmHg SBP; < 60 mmHg DBP^{4,5}
- **Mean Arterial Pressure (MAP)**: Average pressure of the blood in the arteries during a cardiac cycle; can serve as an indicator of perfusion to vital organs
 - MAP = [SBP + (2 x DBP)]/3⁶
 - Normal MAP: 70 110 mmHg⁷⁻⁹
 - MAP < 60 mmHg can result in \downarrow perfusion of vital organs
 - Consult with the medical team if MAP < 65 mmHg to determine appropriateness of activity
 - Low values can be a sign of stroke, internal bleeding, sepsis, etc.
 - High values can be a sign of kidney failure, heart failure, etc.
- Pulse Pressure (PP) = SBP DBP
 - Normal PP range: 40 60 mmHg¹⁰
 - PP outside of the normal range is a significant factor in the development of heart disease
 - Low or "narrowed" (< 25% SBP) can be a sign of heart failure (HF) (low SV), aortic valve stenosis, blood loss, etc.¹⁰
 - Chronic elevation (> 59 mmHg) can be a sign of arterial resistance, HF, + SBP, aging, etc.^{7,1}

BP - Clinical Considerations

Assess for BP trends as normal fluctuations occur (e.g. nocturnal or postprandial dipping)¹²
 SBP ↑ with hypervolemia and ↓ with hypovolemia

Normal Exercise Response

- SBP + in a linear fashion, 10 mmHg per Metabolic Equivalent (MET) until physiologic maximum (dampened response in patients on beta blockers)¹³ (Refer to Pharmacology Section for more details)
- Monitor BP post PT intervention until returns to baseline
- Hypertension (HTN)
 - HTN is generally asymptomatic, so symptoms should not drive the need for VS assessment
 - Monitor for the following symptoms: headaches; visual impairments; confusion; pounding in chest, neck, or ears
 - In most cases, there is no known cause
 - Potential causes: hypercalcemia; thyroid diseases; full bladder; sympathetic stimulation; stress/ anxiety; white coat HTN; hypervolemia^{11,14}
 - Cardiovascular risk \downarrow with \downarrow BP, but dosage amounts of antihypertensive medications may be associated with \uparrow adverse effects, including \uparrow fall risk¹⁵

APTA Acute Care. O APTA Academy of Cardiovascular & Pulmonary Physical Therapy

Hypotension

- Potential causes:
 - Parasympathetic stimulation, hyperkalemia, hypokalemia, hypocalcemia, anoxia, acidosis, hypovolemia, bedrest
 - Cardiac dysrhythmia¹⁶ (Refer to Heart Rate/Pulse Rate Section for more details)
 - Medications (Refer to Pharmacology Section for more details)
 - Adrenal insufficiency
 - Valsalva:
 - To prevent, ask the patient to breathe rhythmically, count, or talk during PT intervention
 - Monitor for the following symptoms: lightheadedness/dizziness; nausea; breathlessness
- Orthostatic (postural) hypotension is a \downarrow SBP > 20 mmHg or \downarrow DBP > 10 mmHg on standing within three minutes
 - Monitor for the following symptoms: lightheadedness; diaphoresis; dizziness; confusion; blurred vision

Older Adults: Cognition and Blood Pressure

- Treatment of HTN to ↓ SBP to < 140 mmHg can ↓ the development of cognitive impairment^{17,18}
- Hypotension (< 120/75 mmHg) is associated with ↓ cognitive function in older adults¹⁹
- Orthostatic hypotension is more prevalent in people with dementia. Cerebral hypoperfusion is associated with cognitive impairment in a study of adults ≥ 50 years old. People with orthostatic hypotension demonstrated + scores on tests of global cognitive function and memory vs. those without orthostatic hypotension, especially in women.²⁰
- The average orthostatic SBP response is found to be significantly ↓ in older adults with dementia, so a larger drop in SBP from sitting to standing ↑ the odds of a dementia diagnosis²¹

Heart Rate (HR) and Pulse Rate (PR)

- Normal resting rate: 60 100 beats/min
- Tachycardia: > 100 beats/min
 - Relative tachycardia: ↑ resting PR > 20 beats/min from usual/baseline
- Bradycardia: < 60 beats/min
 - Relative bradycardia: resting PR > 20 beats/min from usual/baseline
- Heart Rate (HR): measured by ECG (ventricular rate)
 - resting HR is associated with risk of all-cause and cardiovascular mortality. Mortality + as resting HR +, but there is significant + risk of cardiovascular mortality with resting HR > 90 beats/min.²²
 Specifically, this is recognized in older vs. younger adults.²³
- Pulse Rate (PR): pulses palpated at an artery or measured by pulse oximetry

| Pulse Grade | Description ²⁴ |
|---------------|---|
| Absent (0) | No perceptible pulse |
| Thread (1+) | Barely perceptible, easily obliterated with slight pressure |
| Weak (2+) | Difficult to palpate, slightly stronger than thread, can be obliterated with light pressure |
| Normal (3+) | Easy to palpate, requires moderate pressure to obliterate |
| Bounding (4+) | Very strong, hyperactive |

HR and PR - Clinical Considerations

- Normal response with exercise: ↑ 10 beats/min per MET then returns to pre-exercise level in 3-5 minutes¹³
 - Consider using Borg Rating of Perceived Exertion (RPE) Scale and Breathlessness Scale as additional measurement tools
- It is important to consider the clinical significance of the dysrhythmia both at rest and in response to PT intervention¹⁶
 - Electrolyte imbalances can ↑ risk of dysrhythmias



- Most abnormal rhythms have a negative impact on CO that can lead to symptoms such as hypotension, weakness, fatigue, dizziness, syncope, diaphoresis, and mental confusion, and thus must be considered clinically or hemodynamically significant
- If patient's pulse is irregularly irregular or regularly irregular, the clinician should auscultate apical HR for at least 60 seconds²⁵
- Must determine if the resting dysrhythmia is clinically/hemodynamically significant (+ CO) to decide if PT intervention is appropriate
 - If PT is determined to be appropriate, then it is important to analyze the impact of the intervention on the patient's dysrhythmia If the dysrhythmia is worsening and/or symptoms of compromised CO are occurring or \uparrow , then the appropriate decision would likely be to \downarrow the exercise workload or stop the intervention depending on the magnitude of the change.
- Postural orthostatic (autonomic) tachycardia syndrome (POTS): PR of > 120 beats/min or an + of > 30 beats/min from supine to standing with no ↓ in BP
 - POTS is multifactorial with contributions from impaired sympathetically mediated vasoconstriction, excessive sympathetic drive, volume dysregulation, impaired carotid baroreceptor control, baroreceptor failure, and deconditioning^{26,27}
 - Monitor symptoms e.g. fatigue, light-headedness, exercise intolerance, cognitive impairment

Respiratory Rate (RR)²⁴

- Normal resting rate (Eupnea): 12 18 breaths/min with equal rate and depth
- Bradypnea: < 10 breaths/min
- Potential causes: opioids; hypothyroidism; brain disorders
- Tachypnea: > 24 breaths/min (usually shallow)
 - Potential causes: pain; emotion; fever; metabolic disorders; + elasticity of lungs (emphysema); resistance to air passages (asthma); hypoxemia; hypercapnia; ↓ tidal volume; an abnormally low blood pH (acidosis)

RR - Clinical Considerations

- Drug and alcohol-related depression of RR can cause respiratory arrest
- Sitting and standing have \star work of breathing compared to supine
- Use Borg RPE Scale for monitoring (not to exceed 4 5/10 during activity) or the talk test ("just barely can respond in conversation" during activity)^{28,29}
- For individual's RR = 45 breaths/min use caution; if RR = 50 breaths/min no exercise³⁰

Blood Oxygen Saturation (SpO2)³¹

- Normal: > 95%
- Below average for population: 91 94%
- Collaborate with team: < 90%

Blood Oxygen Saturation - Clinical Considerations³²

- SpO₂ = peripherally measured O₂ saturation via pulse oximetry. If peripheral blood flow is adequate, SpO_2^2 is a good approximation of SaO_2 SpO_2^2 is an index of partial pressure of oxygen (O_2) and may \downarrow if O_2 diffusion \downarrow
- If SpO₂ is low, there is \downarrow O₂ delivery to the peripheral tissue
- Document if patient is on room air or the amount of supplemental O2, the O2 delivery device, and conditions under which measurement is taken
- Monitor for the following signs and symptoms of hypoxemia e.g. confusion, wheezing, changes in HR, diaphoresis, clubbing, changes in nail bed color
- Inaccurate readings may occur with movement, damage to nail bed or nail polish, blisters, or poor perfusion (cold fingers)
 - Ear or forehead monitor may be required
 - Check PR against pulse oximeter HR to verify accuracy of pulse oximeter reading
 - Fingertip monitors have the least accuracy compared to other monitors
- Inaccurate readings often occur with dark-skinned individuals^{33,34}



- Black patients more likely than white patients to have falsely high readings, overestimating arterial oxygenation during hypoxia
- Individuals with type 2 diabetes with glycated hemoglobin (HbA1c) > 7% may result in a falsely high reading
- Persons with advanced, chronic obstructive pulmonary disorders may retain carbon dioxide (CO₂), so supplemental O₂ should be used judiciously (Refer to Lung Disease Section for more details)
 Check physician orders for titration of O₂

Temperature^{24,35}

- Normal core body temperature: 35.5 37.5°C (95.9 99.5°F)
 - Oral: 37°C (98.6°F)
 - Axillary: 36.45°C (97.6°F)
 - Rectal: 0.27°- 0.38°C (0.5° 0.7°F) > oral temperature
 - Lowest point in body temperature usually occurs at ~4 a.m. and the peak occurs at ~6 p.m.
- Hypothermia: Rectal temperature of < 35°C (95°F)
 - Early stage (35° 32.8°C; 95° 91°F) signs and symptoms e.g. shivering ↑ BP and PR, vasoconstriction, and diuresis
 - Intermediate stage (32.2° 24°C; 90° 75°F) signs and symptoms e.g. ↓ in metabolism, ↓ PR, ↓ BP, ↓
 RR, muscular rigidity, tremor, respiratory and metabolic acidosis
 - Third stage (when all attempts at compensation by the temperature regulatory center fail) signs and symptoms e.g. ↑ HR, ↓ BP, ↑ RR, leukocytosis, acidosis, ↑ pulmonary wedge pressure, and right atrial pressure
 - Potential causes: dermal disease (i.e. burns, psoriasis); drug-induced; exposure to cold weather conditions or cold water; metabolic disorders; and neuromuscular inefficiency³⁶
 - Hypothermia caused by infection with bacteremia is associated with
 + systemic vascular resistance and
 + cardiac index (CI) than patients who are non-bacteremic with hypothermia
- Hyperthermia (febrile): Oral: > 37.5°C (99.5°F); Rectal: > 38°C (100.5°F)
 - Signs and symptoms e.g. headache, change in gait speed, speech, or mental status
 - Potential causes: exercise with environmental temperatures > 32.2°C (90°F) and humidity > 90%; cancer and related treatments; or infectious conditions

Temperature - Clinical Considerations

- Exercise and heavy exertion may + the core temperature 1 1.5°C (2 3°F), but usually returns to normal within 30 minutes of cessation of exertion³⁷
- Any change of temperature high or low can be a sign of sepsis
 - For every 1°C ↑ in temperature, there is a 13% ↑ in metabolic rate (7% per degree F)
 - • concern for elderly and individuals with restrictive lung disease and cardiovascular disease
 - In the elderly, confusion may be the first sign of an infection instead of temperature change³⁸

Vital Sign Interpretation in the Intensive Care Unit (ICU)

ICU – Background

This section includes information to assist PT providers to understand important concepts related to O₂ transport/utilization and clinical applications when monitoring VS in the ICU. Multiple factors influence VS, including medical diagnosis, medical stability, laboratory values, blood chemistry, and pharmacologic interventions. Tissue oxygenation and perfusion of the brain and vital organs are paramount in the VS hierarchy. Mean arterial pressure (MAP), tissue oxygenation, pH, and serum chemistry are primary VS determinants of patient stability. These are major predictors of ICU mortality along with RR, PR, and body temperature utilized in scales like the **APACHE II**.³⁹

The key for effective and safe PT management of patients in ICU is to have patient-centered goals and strong interdisciplinary collaboration. Patients with critical illness usually have multiple medical problems and require special considerations. It is imperative that specific discussions about VS with the ICU team and appropriate orders addressing VS ranges are specified.

APTA Acute Care. (CARA Academy of Cardiovascular & Pulmonary Physical Therapy

The PT provider is expected to communicate to the ICU team any deviation from the prescribed ranges during the PT intervention. Due to the high medical complexity of these patients, documentation should indicate a close and continuous monitoring of VS throughout the PT intervention. Additional information which may help with clinical decision making include:

- The recent trends in VS should be reviewed for a better understanding of the patient's clinical condition
- The VS may fluctuate during some medical interventions, such as ventilator weaning, medication titration, adjustments in circulatory support flow rates, and many others.
- A significant change in VS in the absence of a known cause can potentially be a poor prognostic sign
- It is expected for VS to fluctuate frequently and sometimes significantly during PT interventions in ICU **Minimum PT documentation for VS in the ICU is**:
- Specific amount of supplemental O₂ and/or circulatory support (e.g. pharmacologic or mechanical)
- VS at rest, with position change, peak exercise/activity, and cool-down/recovery values

Perfusion - Indicates the delivery of blood to vital organs/tissues. It is measured indirectly (Liters/min - L/min) based on the factors described below

| Cardiac Output (CO) | CO = HR x SV (Stroke Volume) Normal values vary by gender and body size Normal adult resting CO : 4.0 - 8.0 L/min | | |
|---------------------|---|--|--|
| Cardiac Index (CI) | Factors in body habitus for CO to allow same normal value levels to be used across patients of varying body sizes Normal CI: 2.5 - 4.0 L/min/m² | | |
| Blood Pressure (BP) | BP is primarily determined by CO and systemic vascular resistance (SVR). It includes values for the systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) MAP is average pressure during a single cycle MAP is largely based on DBP because most of the cardiac cycle is spent in diastole MAP = [SBP + (2 × DBP)]/3 Normal MAP: 70 - 110 mmHg MAP < 60 mmHg can result in + perfusion of vital organs Consult with the medical team if MAP < 65 mmHg to determine appropriateness of activity BP can be assessed non-invasively using a cuff/ sphygmomanometer or an electronic cuff. The manual readings are more accurate; however, the electronic cuffs are used in the ICU because they can be cycled/ recorded automatically at set time intervals. BP can be assessed invasively using an arterial line and, in this case, the arterial line transducer must remain at the level of the right atrium for accurate reading If the transducer is below right atrium will have a false, high reading If the transducer is above right atrium will have a false, low reading A BP cuff reading should be used if the PT provider is unable to ensure that the transducer is correctly leveled with the right atrium such as during ambulation | | |
| Heart Rate (HR) | Normal HR: 60 - 100 beats/min. PT providers must monitor patients in the ICU continuously for sudden changes in heart rate or rhythm It is important for physical therapists to understand the reasons for abnormal HR prior to making clinical decisions, including, but not limited to hypovolemia, anxiety, medications, and pain. Higher HRs shorten the diastolic time period which is the portion of the cardiac cycle when coronary arteries and ventricles fill | | |

APTA Acute Care. (CAPTA Academy of Cardiovascular & Pulmonary Physical Therapy

| Pulmonary Artery Pressure (PAP) | Normal values include: Pulmonary artery systolic pressure (PASP): 15 - 25 mmHg Pulmonary artery diastolic pressure (PADP): 8 - 15 mmHg Mean pulmonary artery pressure (MPAP): 10 - 20 mmHg MPAP = [PASP + (2 × PADP)] Pulmonary artery wedge pressure (PAWP) or pulmonary artery occlusion pressure (PAOP): 6 - 12 mmHg |
|---|---|
| Cerebral Perfusion Pressure (CPP) | CPP = MAP - ICP Normal CPP: 60 - 80 mmHg |
| Intracranial Pressure (ICP) | Normal ICP: 5 - 15 mmHg (acceptable levels as high as 22 mmHg; discuss with medical team)^{40,41} + ICP is indicator of excessive compression on the brain, which can lead to cerebral ischemia and herniation ICP is used as proxy for CPP + ICP may trigger an + in arterial pressure to overcome ICP and maintain CPP resulting in relative hypertension, bradycardia, and bradypnea referred to as Cushing's triad, Cushing reflex or vasopressor response The ICP transducer must be leveled with the tragus of the patient's ear to provide an accurate reading Prior to any activity during PT: The PT provider should coordinate with nursing to verify that the drain is clamped to prevent unintentional excessive cerebrospinal fluid draining⁴² Discuss with the ICU team the target ICP prior to PT intervention Recognize that ICP will change with any positional changes of the patient During PT intervention: ICP must be monitored closely and target range strictly followed ICP transducer must be releveled/recalibrated every time a patient changes position to avoid inaccurate ICP reading Monitoring of neurological status is imperative throughout PT interventions to assess for changes in pupil reactivity, reflexes, mentation, and arousal which could indicate an + ICP and + CPP |
| Oxygenation and Ventilation | |
| Oxygen Saturation • SpO ₂ • SaO ₂ • SvO ₂ | SpO₂ = peripherally measured O₂ saturation via pulse oximetry. If peripheral blood flow is adequate, SpO₂ is a good approximation of SaO₂. SaO₂ = oxyhemoglobin saturation measured via arterial blood gas (ABG) Normal SpO₂ and SaO₂: 95 - 100% Need to document how much supplemental O₂ and/or ventilatory support is being used to attain these values SvO₂ = mixed venous O₂ saturation and indirect measure of peripheral O₂ extraction Normal SvO₂: 65 - 75% If SvO₂ is too high (in presence of hypoxemia), the body is having trouble using (extracting) the circulating O₂ Potential causes: sepsis; poisoning; or other conditions causing abnormal circulatory shunting If SvO₂ is too low, tissues are extracting an excessive amount of O₂. Potential causes: + O₂ delivery; + Hgb; + SaO₂ (hypoxemia); + CO (any form of shock, arrhythmia); or + O₂ demand (hyperthermia, shivering, pain, seizures) |
| Γ a tial pressure of O_2 (Pd O_2) | Normal PaO₂: 80 - 100 mmHg |

APTA Acute Care. (CAPTA Academy of Cardiovascular & Pulmonary Physical Therapy

| PaO ₂ /FiO ₂ ratio (aka P/F Ratio) | Ratio of PaO₂ via ABG to fraction of inspired O₂ (FiO₂) Widely used indicator of hypoxemia Normal PaO₂/FiO₂ (P/F) ratio: ~ 400 - 500 mmHg (~55 - 65 kPa) P/F ratio < 200 - 300 mmHg indicates possible need for mechanical ventilation P/F ratio < 150 indicates severe hypoxemia; medical team should consider prone positioning in ARDS and /or possible ECMO Improvement is indicated by the patient's ability to maintain normal PaO₂ on less supplemental O₂ (less FiO₂). Therefore, an endurance goal in the ICU may be to have a better P/F ratio versus isolated improvement in SpO₂. For example, a patient who ambulates in the ICU with an SpO₂ of 90% on room air is oxygenating better than a patient who ambulates with an SpO₂ of 93% but requires 50% face mask to maintain SpO₂ |
|---|--|
| Additional Factors that Affec | t Oxygen Binding/Transport and Oxygen Consumption |
| Body Temperature Lactate pH Hemoglobin | Normal core body temperature: 35.5 - 37.5°C (95.9 - 99.5°F) Disruption of thermoregulation is common in the critically ill Severe hyperthermia could lead to multiorgan failure Hyperthermia also + oxygen-hemoglobin association resulting in + SpO₂ Anemia, acidosis, and + lactate levels can all impair O₂ delivery to tissues pH < 7.35 (acidosis) + oxygen-hemoglobin association resulting in + SpO₂ and impaired O₂ delivery + blood lactate levels are common in patients in the ICU. Excess lactate accumulation + pH (metabolic acidosis) which + oxygen-hemoglobin association resulting in + SpO₂ and impaired O₂ delivery Hyperlactatemia on ICU admission is associated with worse prognosis Anemia can impair tissue oxygenation despite a lack of change in SpO₂/SaO₂ levels (tachycardia may result) |

ICU Support Devices and Effects on Vital Signs

Continuous Renal Replacement Therapy (CRRT) - Clinical Considerations

- Monitor for hypotension⁴³
- PT providers should work within MAP parameters as prolonged duration of MAP < 73 mmHg can accelerate the progression of acute kidney injury⁴⁴
- Patients on CRRT usually have multiple medical problems or comorbidities and may also require vasopressor support. PT providers are encouraged to discuss the patient's current medical status with ICU medical team and establish a target MAP range for the PT intervention.
- Central venous pressure (CVP) may be a preferred target in hemodynamic therapy during CRRT as it reflects relative changes in blood volume. Maintaining CVP < 8 mmHg during the early phase of septic shock can prevent further damage to renal function and improve the survival rate of critically ill patients.⁴⁵

Extracorporeal Membrane Oxygenation (ECMO) - Clinical Considerations

General

- Different types of ECMO
 - VA = veno-arterial ECMO
 - VV = veno-venous ECMO
- All changes to ECMO support required during the PT intervention should be performed by a qualified ECMO specialist
- Due to the high medical complexity of patients on ECMO support, the PT provider's documentation should indicate close monitoring of VS during the PT intervention

Blood Pressure

- MAP goal is typically > 65mmHg but < 90 mmHg to limit afterload and to promote forward flow⁴⁶ The a-line waveform can look dampened or even flat; only displaying the MAP. This flattened waveform does not mean that the a-line is inaccurate.47
- Hypotension could be due to inadequate flow or inadequate systemic vascular resistance (SVR) Pulsatility

Pulsatility = Pulse Pressure/Mean Arterial Pressure (PP/MAP)⁴⁸

- Right after implantation, most VA ECMO patients have poor or no heart pulsatility⁴⁷
- Lack of pulsatility on the arterial waveform could be due to poor myocardial function, excessive ECMO support, inadequate preload, or right ventricular failure; may result in thrombus, myocardial ischemia, or pulmonary edema⁴⁶

Heart Rate and Rhythm

- Monitor ECG for dysrhythmias⁴⁶
- Loss of pulses or cyanosis/coolness of a limb could lead to distal limb ischemia⁴⁶

Oxygenation/Ventilation

- Should be continuously monitored via the ECMO circuit⁴⁹
- ↓ SvO, and
 ↑ lactate suggest inadequate O, delivery; excessive O, consumption can lead to fever or shivering46
- For VV ECMO patients, the saturation target is rarely 100%. A saturation in the low 90s is sufficient.⁴⁷
- Gas exchange monitor for inadequate partial pressure of oxygen (PaO₂) or excessive CO₂ elimination46
- Patients may require additional mechanical ventilation or ECMO support to perform PT intervention due to + CO, O₂ consumption and CO₂ production during the physical activity
- Patients with minimal native lung function (tidal volumes < 200 mL, or no change in PaO, with an + in fraction of inspired oxygen (FiO₂) to 1.0 for 30 minutes to assess contribution of lungs to gas exchange) may require + ECMO blood flow during the rehabilitation activity
- Patients with recovering lung function (tidal volumes > 200 mL and incremental change in PaO, following \uparrow in FiO₂ to 1.0) may require \uparrow FiO₂ via the ventilator and/or \uparrow ECMO blood flow during the rehabilitation activity. Fluid boluses may be required to avoid complications related to changes to ECMO flow settings

Temperature

- ECMO should not affect temperature.⁴⁶ If patient has an + temperature, it could be a sign of infection. Flow Rate (liters/minute of blood flow that the ECMO circuitry is generating)
- Should be continuously monitored via the ECMO circuit⁴⁹

Intra-Aortic Balloon Pump (IABP) - Clinical Considerations

General

- IABP locations can be femoral or axillary. Patients with femoral IABPs are typically on bedrest with limitations in hip flexion and head of bed elevation. Patients with axillary IABPs can mobilize out of bed and/or ambulate. Protocols may vary depending on institution.
- Patients should be monitored continuously and any change in hemodynamics should be reported to the medical team immediately^{50,51}
- When auscultating heart and breath sounds, the IABP will need to be placed on standby. Any changes to IABP settings (including standby mode) should only be performed by trained personnel.⁵²

Blood Pressure

- The aortic BP measured from the tip of the IABP on the balloon pump console is likely to be the most accurate or the truest reflection of the patient's central BP; therefore, the BP reading from the IABP is generally considered the gold standard⁵³
- As a pulse moves through the arterial system away from the aorta, SBP ↑ and DBP may ↓, because of reflection of the pressure wave. Peripheral SBP i.e., from the radial artery is usually > the aortic SBP, but the MAP measured peripherally may be ↓^{50,53}

Pulses

- Monitor pedal and radial pulses on the affected side. If the balloon moves up/down it can occlude perfusion to the limbs.51,52
- Monitor for signs of limb ischemia (color, sensation, temperature, movement, and capillary refill)^{51,52}



Positive Pressure Ventilation (Invasive and Non-Invasive) - Clinical Considerations

- Continuous VS monitoring for patients on mechanical ventilation is imperative during PT intervention to ensure patient safety
- Target VS parameters should be determined based on current medical status after discussion with ICU team
- BP trends ↓ and HR trends ↑ due to ↑ intrathoracic pressures
- Positive pressure ventilation may ↑ intra-thoracic pressures resulting in ↓ CO and ↓ BP when right ventricular preload is affected. Positive end expiratory pressure (PEEP) further ↑ airway pressures and may ↓ venous return.
- Endotracheal suctioning may precipitate a vasovagal reaction resulting in profound bradycardia and hypotension. The PT provider might consider pre-hyperoxygenation prior to suctioning to avoid O₂ desaturation.

Ventricular Assist Device (VAD) - Clinical Considerations

Blood Pressure

- MAP should be maintained between 70 90 mmHg during exercise⁵⁸
- If unable to detect BP with a cuff, an a-line is the most accurate method. Doppler ultrasound is an alternative in patients without an a-line.⁵⁴
- Patients with pulsatile devices should have a BP goal of SBP of < 130 mmHg and a DBP of < 85 mmHg 55
- MAP > 90 mmHg is associated with stroke and pump thrombosis⁵⁴

Heart Rate and Rhythm

• There is either no palpable pulse or a weak pulse with a continuous or non-pulsatile device. Gold standard for measuring HR and rhythm is ECG. Device does not affect ECG.⁵⁶

Oxygen Saturation

- Measuring SpO, with pulse oximetry is unreliable with continuous or non-pulsatile devices
- · Arterial blood gas measurement is the gold standard but only gives one snapshot in time
- Monitor for signs and symptoms of hypoxia: changes in skin color; confusion; cough;
 + HR;
 + RR;
 shortness of breath; or sweating⁵⁷

Flow Rate

- VAD flow rate = CO
- LVAD flow rates should remain > 3 4 liters/minute⁵⁸

Parameters regarding safe and effective exercise include.58

- Borg RPE Scale of no > 13/20 without onset of signs and symptoms of angina
- Electrocardiogram (ECG) changes including ST shifts no > 1 mm and/or no + ventricular arrhythmias
- Dyspnea no > 5/10
- MAP maintained between 70 90 mmHg
- LVAD flow remains above 3 L/min

Sepsis - Pathophysiology/Background⁵⁹

- · Defined as a life-threatening organ dysfunction due to a dysregulated host response to an infection
- Risk factors: age, frailty, multiple comorbidities, indwelling lines or catheters, invasive procedures,
- breach in skin integrity, and immunosuppression
- Prompt recognition and treatment of sepsis improves survival
 - Quick Sequential (sepsis-related) Organ Failure Assessment (qSOFA) is a quick screening tool⁶⁰ • An individual with at least two of the following indicates strong consideration for ICU
 - admission due to organ dysfunction
 - RR > 22 breaths/minute
 - Change in mental status
 - SBP < 100 mmHg

Sepsis - Vital Sign Recommendations⁵⁹

- MAP is the primary clinical target during medical treatment for sepsis
 MAP Goal: > 65 mmHg
- ECG, temperature, oxygenation (PaO₂/ FiO₂ ratio), electrolytes, glucose, and arterial blood gas (ABGs) are closely monitored
- Older adults as well as individuals undergoing cancer treatments are less likely to develop an
 temperature with sepsis

Sepsis - Clinical Considerations

- Early activity and minimization of sedatives can help prevent critical Illness acquired weakness/ myopathy and critical illness polyneuropathy⁶¹
- Warrants close monitoring of all VS with special attention to MAP. ECG, ABG, and lab values (electrolytes, glucose, CBC) should also be followed.

Special Population Considerations

Acute Coronary Syndrome/Myocardial Infarction - Background/Physiology

Acute Coronary Syndrome (ACS):62,63

- Unstable angina (UA) absence of cardiac myocyte death
- Myocardial infarction (MI) (myocardial cell necrosis):
 - STEMI: ST-elevated MI resulted from total occlusion thrombus
 - NSTEMI: non-ST elevated MI resulted from partial occlusion with/without collateral circulation

Physiology:

- Dilation of ventricular chamber (infarcted wall thins) = + systolic function
 - Blood pressure: ↓ CO may result in hypotension with exercise
 - ↑ Vasoconstriction + ↑ Afterload = ↑ Ischemia
- · Reduction of angina with nitroglycerin which acts as a vasodilator
- Tachycardia: vasoconstriction and ↓ CO, HR may ↑ at an abnormal rate in response to exercise
- ECG⁶⁴
 - STEMI: ST elevation ≥ 1mm
 - NSTEMI or UA : transient ST changes > 0.5 mm with possible;
 - Bundle Branch Block (BBB)
 - Sustained Ventricular Tachycardia (SVT)
- Cardiac Markers⁶⁴
 - Elevation of troponin I (Tnl), troponin T (TnT), or creatine kinase-MB (CK-MB) indicate necrosis

| Acute Coronary Syndrome/Myocardial Infarction - Vital Sign Recommendations ^{13,65-67} | | | | |
|--|--|--|--|--|
| Stable angina, stable and/or down trending Reasons to stop PT intervention | | | | |
| troponins, stable ECG; Initiation of PT intervention is | Unable to comfortably speak | | | |
| likely to be appropriate: | RR > 40 breaths/min | | | |
| RR < 30 breaths/min; able to speak comfortably | Onset of S3 heart sound | | | |
| Resting HR < 120 beats/min | HR ↓ > 10 beats/min | | | |
| • CI ≥ 2.0 L/min/m | SBP ↓ > 10 mmHg | | | |
| • CVP < 12 mmHg | MAP ↑ > 10 mmHg | | | |
| MAP of a minimum of 60 mmHg | CVP ↑ or ↓ > 6 mmHg | | | |
| • SpO ₂ > 90% | • SpO ₂ < 90% or a $\downarrow \ge 4\%$ | | | |
| • SBP ⁻ < 110 mmHg | New onset or worsening of cardiac | | | |
| | dysrhythmia | | | |
| | Return of pre-MI angina like pain | | | |

APTA Acute Care. C Academy of Cardiovascular & Pulmonary Physical Therapy

| Heart Failure - Background/Physiology | | | | |
|---|--|--|--|--|
| Heart Failure with Preserved Ejection Fraction (HFpEF): diastolic dysfunction with EF between 55% and 75% Heart Failure with Reduced Ejection Fraction (HFrEF): systolic dysfunction with EF < 40% Physiology: SV is ↓ in both HFpEF and HFrEF therefore tachycardia (HR ≥ 100 beats/min) commonly occurs to help maintain or lessen the reduction of CO BP = CO X Total Peripheral Resistance (TPR) Given the ↓ CO that frequently occurs, hypotension at rest (< 90/60) and a ↓ SBP with exercise are common (↓ SBP > 10 mmHg) RR may be ↑ especially in HFrEF due to pulmonary edema Dyspnea may be a limiting factor during exercise | | | | |
| Heart Failure - Vital Sign Recommendations68-70 | | | | |
| Stable HF: Initiation of PT intervention is appropriate:Reasons to stop PT intervention:. RR < 30 breaths/min; able to speak comfortably | | | | |
| Heart Failure - Clinical Considerations | | | | |
| Important to monitor signs and symptoms of HF during every visit especially in patients with low EFs Worsening of signs and symptoms suggests decompensation and a medication adjustment may be needed (Refer to Pharmacology Section for more details) Important to monitor for signs and symptoms of hypotension Monitor for jugular vein distention and peripheral edema with right heart failure Monitor RR, PR, and rhythm (ECG analysis e.g. atrial fibrillation, PVCs) Auscultate for S3, pulmonary crackles before and after exercise and rib level where heard, and + cough Document level of Borg RPE Scale or a Breathlessness Scale with vitals | | | | |

Peripheral Arterial Disease - Background/Physiology

- Atherosclerotic disease impeding blood flow in arteries of extremities (> lower extremity)
- If O₂ demand exceeds O₂ supply to the periphery, claudication may occur
- As disease progresses, more severe symptoms e.g. resting pain and skin changes
- Complete obstruction to blood flow leads to limb loss⁷¹
- Ankle-brachial index (ABI) (ankle SBP/arm SBP) of \leq 0.90 suggests PAD
 - Normal is 0.9 to 1.3 except in patients with diabetes
 - The lower this value, the patient may experience more pain⁷²

Peripheral Arterial Disease - Vital Sign Recommendations

- Prevalence of other Cardiovascular Disease (CVD) such as coronary and carotid atherosclerosis is found in ≥ 50% in PAD patients⁷³
 - With more severe disease, steep \uparrow in BP may occur due to atherosclerosis and a diminished vascular bed
- VS assessment imperative at rest and with activity such as: HR/ECG, RR, Borg RPE Scale, signs or symptoms of cardiac compromise (chest discomfort, dizziness)
- BP: If upper extremity PAD (e.g. subclavian arterial stenosis), SBP values can be ≥ 15-20 mmHg ↓ in that arm⁷⁴

- Use BP in the arm with the higher value
- Pulse oximetry: take in lesser involved extremity
- Assess for signs and symptoms of intermittent claudication

Peripheral Arterial Disease - Clinical Considerations

- Obstruction of blood flow leads to ↓ pulses, ↓ endurance, impaired sensation, and muscle atrophy in extremities
 - Assess for fall risk and integumentary changes
- Screen for other signs and symptoms of CVD such as angina or stroke
 - Structured supervised exercise training improves exercise tolerance and walking distance75-78
 - Patient to walk through some pain to moderate pain, rest and repeat as tolerated with appropriate VS response

Aortic Aneurysm - Background/Physiology

Atherosclerosis and systolic HTN are common causes of the two main types of aortic aneurysms; thoracic and abdominal. An aortic aneurysm is a pathologic dilation of the aorta that is \geq 50% than normal or \geq 3 cm in size. Surgery is considered once the diameter of the abdominal aortic aneurysm (AAA) is \geq 5.5 cm to avoid rupture of the aneurysm.⁷⁹ In patients with a thoracic aortic aneurysm (TAA), acute dissection, a tear of the inner lining of the thoracic aorta, is more common than rupture. In those with TAA, once symptoms occur, surgery is indicated.⁸⁰

Aortic Aneurysm - Vital Sign Recommendations

- BP: Important to assess during activity to avoid excessive stress on the weakened area. Often SBP of \leq 140 mmHg is suggested for those with AAA⁸¹
- Tachycardia and \downarrow BP especially with patient complaints of sudden abdominal pain could be a sign of a ruptured aneurysm⁸²

Aortic Aneurysm - Clinical Considerations

- Research suggests moderate activity such as treadmill and stair climbing in patients with small AAAs does not + the risk of rupture. Therefore, moderate exercise should be promoted if the PT provider monitors VS, especially BP.⁸³
 - Signs and symptoms of an aortic aneurysm are often vague if there are any symptoms⁷⁹
 - tow back/abdominal pain or non-tender abdominal pulsating mass near the umbilical area may
 indicate an AAA
 - These are more prevalent in men, smokers and individuals > 60 years old
 - Aortic murmur and chest/upper back pain (complaints of ripping/tearing) may be seen in individuals with a thoracic aneurysm

Venous Thromboembolic (VTE) Disease - Background/Physiology

- Venous thrombosis is a clot obstructing venous blood flow
- Deep venous thrombosis (DVT) and pulmonary embolism (PE) are both manifestations of this disease
 - Distal DVTs: below the knee; more common; and less likely to become a PE than proximal DVT
 - PE: clot reaching the lungs⁸⁴

Venous Thromboembolic Disease - Vital Sign Recommendation

• All vital signs, including RR, should be assessed before and with activity in any patient at risk for or diagnosed with venous thrombosis

Venous Thromboembolic Disease - Clinical Considerations

- Many patients in the acute care setting are at risk for VTE disease (DVT and PE)
- Mechanical compression, early activity and anticoagulation are necessary for prevention⁸⁵
- Use Padua Prediction Score to assess global risk of VTE in hospitalized patients (medical team uses score to assess need for thromboprophylaxis)⁸⁶
- DVT signs and symptoms: pain, ipsilateral swelling, warmth or redness

APTA Acute Care. (CAPTA Academy of Cardiovascular & Pulmonary Physical Therapy

- Primary risk factors for hospitalized patients are age > 75 years-old, active cancer, reduced activity, previous VTE, major surgery including total hip or knee arthroplasty^{87,88}
 - Use the Wells Clinical Decision Tool to assess for lower extremity DVT risk⁸⁹
 - If results indicate a possible DVT, immediately inform the patient's physician
 - See American Physical Therapy Association Clinical Practice Guidelines for the Management of Patients at Risk for or Diagnosed with DVT⁹⁰
- PE signs and symptoms may be vague
 - Unexplained shortness of breath (most common), + in SpO₂, anxiety, pleuritic chest pain, cough, and tachycardia⁸⁴
 - Use the Padua prediction tool for global VTE risk assessment⁸⁶
 - Use the Geneva prediction tool to assess for PE likelihood⁹¹
 - Use Khorana VTE risk assessment for those with history of cancer or cancer treatments⁹²

| Lung Disease ^{93,94} | | | | |
|--|---|--|--|--|
| Restrictive dysfunction and obstructive lung diseases (below) are both marked by progressive dyspnea, hypoxia and decline in FEV1 (forced expiratory volume in one second) In advanced stages, both restrictive dysfunction and obstructive diseases are predispositions for cor pulmonale (right ventricular failure with pulmonary hypertension and right ventricular strain) Potential signs and symptoms of heart failure include: jugular venous distention; peripheral edema; ascites and rapid decline in SpO₂ with activity | | | | |
| Restrictive Lung Disease and Dysfunction - Background/Physiology93-95Obstructive Lung Disease - Background/ Physiology93,94,96 | | | | |
| Defined as impaired ability to get air in, with a FEV1/FVC ratio > 85% as a diagnostic threshold Restrictive lung dysfunction can be primary (lung pathology) or secondary (extrapulmonary pathology); and can be acute or chronic Both primary and secondary pathologies typically have low lung volumes and ↓ diffusing capacity for carbon monoxide Secondary restrictive dysfunction includes any condition that impairs thoracic expansion: advanced pregnancy; neuromuscular weakness; thoracic integumentary restriction (e.g. major burn injury); thoracic musculoskeletal restriction (e.g. severe kyphoscoliosis, multiple rib fractures); major surgery; obesity; etc. Paired with immobility, can lead to ↑ risk of pneumonia and/or atelectasis Primary (pulmonary) restrictive lung diseases include pneumonia, interstitial lung diseases and to acute respiratory distress syndrome (ARDS) Respiratory infection risk ↑ (e.g. colds, flu, pneumonia) Malnutrition: individuals appear emaciated due to ↑ work of breathing and inability to take in appropriate amounts of nutrition | Defined as impaired ability to get air out, with FEV1/FVC ratio < 70% as a diagnostic threshold Chronic obstructive pulmonary disease (COPD), which includes chronic bronchitis, emphysema, bronchiectasis, asthma, and cystic fibrosis Hyperinflation and + residual volume are common with moderate to severe disease with a flat and weak diaphragm especially seen in emphysema and cystic fibrosis Inspiratory to expiratory ratios approach 1:3 to 1:5 Moderate to severe obstructed expiratory flow can predispose CO₂ retention Headaches, especially upon waking in the morning, flush skin, disorientation, and dizziness are potential signs and symptoms of hypercapnia CO₂ can be assessed by end-tidal CO₂ or arterial blood gas With chronic CO₂ retention, patient may depend on hypoxia to drive breathing thus the clinician needs to carefully titrate the supplemental O₂ and assess for signs and symptoms of hypercapnia. For patients with CO₂ retention, the target SpO₂ range might be below 90%. | | | |

APTA Acute Care. (CAPTA Academy of Cardiovascular & Pulmonary Physical Therapy

| Chronic, Restrictive Lung Diseases - Vital Sign Recommendations ^{93,94,97} | Chronic, Obstructive Lung Diseases - Vital Sign Recommendations ⁹⁸⁻¹⁰⁰ |
|--|---|
| Often need + amounts of supplemental O₂ during activity SpO₂ may drop precipitously with advanced disease; O₂ delivery dose and method/ device may need to be adapted Rapid, shallow breathing volumes with possible ↓ residual volume Minute ventilation (RR x tidal volume) driven proportionally more by RR than tidal volume Inspiratory to expiratory ratios approach 1:1 (normal 1:2) Note: secondary (extrapulmonary) lung dysfunction, such as severe kyphoscoliosis or obesity, may present with tachypnea and/or tachycardia and + work of breathing; though O₂ desaturations are uncommon unless accompanied by a pulmonary infection, e.g. aspiration pneumonia²⁵ Acceptable Parameters for Initiation of PT Intervention: RR < 40 breaths/min; able to speak comfortably HR: 60 - 120 beats/min (caution if > 120) Pulse oximetry: > 90% Need for supplemental O₂: generally, keep SpO₂ > 90% Reasons to Stop or Modify PT Intervention: Unable to comfortably speak SpO₂ < 85%, especially if titration of supplemental O₂ is ineffective HR > 10 beats/min | ↑ resting HR may occur due to an + work of breathing as well as chronic, frequent bronchodilator use Therefore, the use of breathlessness scales and the talk test along with HR response to activity is needed Supplemental O₂ use is often needed with moderate to advanced disease, but use should be judicious. Over-oxygenation may ↓ the hypoxic drive. O₂ titration with PT intervention (with MD order) is generally beneficial SpO₂ target with activity in the acute setting is generally 88-92% Except for those who retain CO₂, where their goal may be upper to mid 80's based on documented blood gas (consult with healthcare providers) Acceptable Parameters for Initiation of PT Intervention: RR < 30 breaths/min; able to speak comfortably Pulse oximetry: > 90% at rest (+/-supplemental O₂) HR: 60 - 120 beats/min (caution if > 120) Reasons to Stop or Modify PT Intervention: Unable to comfortably speak \$SpO₂ < 85% especially if titration of supplemental O₂ is ineffective HR > 10 beats/min; |
| Chronic, Restrictive Lung Diseases - Clinical Considerations:93,94,97 | Chronic, Obstructive Lung Diseases - Clinical Considerations ^{93,94,97-100} |
| Difficulty pacing breath and ↑ accessory muscle use is common Patients may need ↑ RR to get sufficient ventilation, so slow deep breaths are not encouraged In early phases of restrictive lung disease: may only present with tachypnea with activity: will progress to tachypnea at rest Hypoxemia will occur sooner in disease process and be more pronounced than obstructive dysfunction Document level of Borg RPE Scale Inspiratory muscle training can be helpful early in the disease process | Two central drivers of respiratory drive: Patients with COPD can be chronically hypercarbic and have limited O₂ drive (hypoxemia) Supplemental O₂ should be titrated to meet the SpO₂ orders from the medical provider Be sure to return the supplemental O₂ back to the pre-intervention levels after PT intervention. If patient is unable to tolerate weaning to pre-intervention levels, notify the medical team. Document level of dyspnea and/or Borg RPE Scale Goal 3 - 5 on 0 - 10 modified Borg RPE Scale Goal 11 - 13 on 6 - 20 Borg RPE Scale (Borg RPE Scale of 12 - 13 generally corresponds with 60% peak VO₂) |

Diabetes Mellitus - Physiology/Background

Diabetes Mellitus (DM) is a prime risk factor for cardiovascular disease and can lead to multi-system impairments. A common complication of DM is autonomic neuropathy which can lead to orthostatic hypotension, silent ischemia, and impaired HR and BP responses during PT intervention.

Diabetes Mellitus - Vital Sign Recommendation

Heart Rate

- Monitor for dysrhythmias, especially atrial fibrillation.101
- · Monitor for tachycardia or bradycardia, especially during position changes
- Monitor HR recovery response (HR should \downarrow > 12 beats within one-minute post-PT intervention)¹⁰²

Blood Pressure

Monitor for orthostatic hypotension (\downarrow SBP > 20 mmHg or \downarrow DBP > 10 mmHg on standing within three minutes)^{103,104}

Respiratory Rate

- Monitor level of dyspnea using Breathlessness Scale
- Pulse Oximetry
 - Pulse oximetry may overestimate blood O₂ saturation in those patients with poorly controlled type 2 diabetes who have \uparrow HbA1c levels, suggesting that arterial blood gas analysis may be needed to determine accurate blood O₂ saturation levels¹⁰⁵

Diabetes Mellitus - Clinical Considerations

- Monitor for symptoms of silent ischemia that may include fatigue, nausea/vomiting, sweating, arrhythmia, and dyspnea¹⁰⁶
 - Use Borg RPE or a Breathlessness Scale with vitals
 - It is important to know your patient's blood glucose levels as well as vital signs^{107,108}
 - Pre-exercise blood glucose should be between 90 250 mg/dL
 - For patients with a pre-exercise blood glucose < 90 mg/dL, they should ingest 15 30 grams of carbohydrates prior to activity
 - If glucose is 250 350 mg/dL test for ketones, collaborate with the healthcare team. Exercise should be avoided if moderate-to-large amounts of ketones are present. If ketones are negative or low mild-to-moderate intensity exercise may be done.
 - If glucose is ≥ 350 mg/dL no exercise should be done if moderate-to-large amounts of ketones are present. If ketones are negative or low insulin may be adjusted prior to beginning mild-to-moderated exercise.
- If the patient is receiving insulin, it is important to know the timing of the administration of insulin and timing of the last meal
- Autonomic neuropathy can lead to + resting HR in early stages of the disease, but in the later stages, HR may become fixed and unresponsive with exercise^{106,109}
- Patients may exhibit slow HR recovery post-PT intervention¹⁰²

Oncologic Conditions - Physiology/Background

Due to the variations in cancers and cancer-related treatments (surgery, radiation, chemotherapy, and hormone), adaptations to exercise may be required with changes in lab values (anemia). Some side-effects may be acute such as radiation to the trunk, which can cause inflammation of the lung tissue and impair oxygen diffusion. Radiation also can lead to delayed-onset lung tissue scarring that can occur months to years post-radiation. Some chemotherapeutic agents can cause pulmonary fibrosis, cardiomyopathy, and/or accelerated atherosclerosis. Cancer and the related side effects from treatment warrant careful monitoring of VS and symptoms during interventions. Active cancer and chemotherapy increase risk of VTE events (Refer to Venous Thromboembolic Disease Section for more details)

APTA Acute Care. (CAPTA Academy of Cardiovascular & Pulmonary Physical Therapy

Oncologic Conditions - Vital Sign Recommendations

- Exercise should be terminated for the following: \downarrow HR and BP with \uparrow workload; new onset of dysrhythmia; SpO₂ <88%; RR <5 breaths/minute or > 40 breaths/minute^{110,111}
- Monitor for dizziness, nausea, chest pain, pallor, diaphoresis, or shortness of breath during activity¹¹¹
- Should avoid exercise if body temperature > 38.3°C (101°F)¹¹²

Oncologic Conditions - Clinical Considerations

- Physical therapists should collaborate with the medical team to determine safe exercise limits based on the individual's trends (lab values and VS) to avoid serious adverse events
- Radiation to the neck can result in increased HR and variability in BP due to dysfunction of the arterial baroreceptors¹¹³
- Since oncologic treatments may affect HR, the physical therapist should consider using a Borg Breathlessness Scale, Borg, or Modified Borg RPE Scale to monitor appropriate progression of intervention¹¹¹
- The American College of Sports Medicine Roundtable Consensus Statement recommended the need to adapt exercise programs for the individual per health status, adjuvant treatments, and anticipated disease trajectory¹¹⁴
- Individuals undergoing cancer treatments are at a higher risk of injurious falls. Multifactorial causes of falls may include but are not limited to anemia, hypovolemia, hypotension, altered cognition, pain medication, and altered pedal sensation¹¹⁵

Neurologic Conditions - Physiology/Background

Acute neurologic episodes such as stroke (cerebral vascular accident), traumatic brain injury (TBI), and spinal cord injuries (SCI) can cause abnormalities in VS that warrant careful monitoring. Assessment and interpretation should continue across continuum of care as abnormalities may persist beyond hospital discharge. For example, approximately 40% of patients with a stroke remain hypertensive beyond one week after initial event.^{116,117}

In patients with chronic or progressive conditions such as Parkinson's disease (PD), multiple sclerosis (MS), Guillain-Barre syndrome (GBS), amyotrophic lateral sclerosis (ALS), and myasthenia gravis, VS should be closely monitored. These patients are at ↑ risk for orthostatic hypotension, ↓ exercise tolerance, impaired HR and rhythm responses, and respiratory insufficiency.⁶⁵

Acute Stroke - Vital Sign Recommendations

Blood Pressure

Blood pressure needs to be managed carefully after an acute stroke. Persistent \uparrow SBP can \uparrow the risk of hemorrhagic transformation of ischemic stroke and lead to \uparrow ed bleeding area in hemorrhagic stroke. Aggressive \downarrow of BP can induce cerebral hypoperfusion.¹¹⁸

Acute Ischemic Stroke BP parameters per 2019 AHA Guidelines¹¹⁶

- Thrombolytic agent (i.e., tPA) administered: Permissive hypertension up to 180/105 mmHg
- Thrombolytic agent not administered: In absence of comorbid conditions requiring rapid +, permissive hypertension up to 220/120 mmHg

If > 220/210 mmHg, \downarrow by 15% in initial 24 hours post-stroke may be indicated

Mechanical thrombectomy performed: Moderate BP control of < 180/105 mmHg is currently

recommended. Some support for \star target ranges based on degree of recanalization

Intracerebral Hemorrhage¹¹⁹

Maintain SBP < 140 mmHg

Subarachnoid Hemorrhage (Aneurysmal)

- Target SBP < 160 mmHg may ↓ risk of rebleeding
 - Exception: induced hypertension indicated in the presence of vasospasm or delayed cerebral ischemia. Verify target BP for individual patient^{120,121}

Heart Rate and Rhythm

• Continuous ECG monitoring for at least 24 - 48 hours post-stroke is common. Risk for cardiac complications is ↑ed and abnormal findings may contribute to future neurologic events.^{116,119}

Oxygen Saturation

- Per the AHA Guidelines, titrate O₂ to keep SpO₂ ≥ 94% for patients in the acute phase of stroke recovery. ↓ SpO₂ levels within the initial hours post-stroke have been reported to be associated with ↑ mortality. Consult with medical team to provide supplemental O₂ and follow state practice act guidelines to provide and titrate supplemental O₂.^{116,122}
- In patients with acute stroke, respiratory muscle weakness, aspiration risk, pneumonia, and sleep apnea may all contribute to hypoxia¹²³

Traumatic Brain Injury (TBI) - Vital Sign Recommendations

Recommendations for patients with moderate to severe TBI defined by modified Glasgow Coma Scale (GCS) score < 12

 When feasible, SBP, CPP, and ICP should be continuously monitored. Even brief periods of hypotension, hypoxemia, and cerebral HTN have been associated with worse outcomes.¹²⁴

Blood Pressure

- SBP recommendations associated with \downarrow mortality and improved outcomes:¹²⁵
- Patients 50 69 years old: SBP ≥ 100 mmHg
- Patients 15 49 years old or > 70 years old: SBP ≥ 110 mmHg
- Without capacity for invasive monitoring target SBP of 120 mmHg is recommended to prevent secondary injury¹²⁴

Heart Rate

- HR > 100 bpm without obvious stimulus may suggest paroxysmal sympathetic hyperactivity and warrant review of BP, HR, and temperature¹²⁶
- Intracranial pressure (ICP) and Cerebral perfusion pressure (CPP)
- Maintain ICP < 22 mmHg and CPP between 60 70 mmHg¹²⁵

Spinal Cord Injury (SCI) - Vitals Sign Recommendations

Blood Pressure

 Collaborate with medical team to address hypotension (SBP < 90 mmHg) prior to PT intervention. MAP goal of 85 - 90 mmHg is recommended for at least 1 week following injury for adequate spinal cord perfusion.¹²⁷

Oxygen Saturation

Patients with spinal cord injuries typically present with impaired pulmonary function secondary to a
restrictive dysfunction (Refer to Lung Disease Section for more details) depending on type and level of
injury. Clinicians should monitor SpO₂ levels within normal range, especially if these individuals have
ineffective cough or excessive mucus retention.¹²⁸

Neurologic Conditions - Clinical Considerations

The initiation of early mobilization (8 - 24 hours post-event) in patients s/p ischemic stroke, including those who receive pharmacologic or surgical interventions, is considered safe and associated with improved outcomes if vital signs and neurologic stability are monitored.¹²⁹⁻¹³¹

The management of patients with acute and subacute TBI focuses on preventing systemic and intracranial secondary injuries cascading from the original insult. Best practice includes multimodal monitoring to prevent common VS alterations (e.g.: ↑ICP, ↓CPP, ↓CBF, ↓BP, ↓SpO2), associated ↑ mortality, and inferior neurologic outcomes.¹²⁵

- Interventions including decompressive craniectomy or drain placement may be indicated to maintain threshold values
- Paroxysmal sympathetic hyperactivity (aka neurological storming): Abnormal response to afferent stimulation including movement resulting in transient + in sympathetic and motor activity including + of BP, PR, RR, temperature, sweating, muscle tone, and posturing. May not manifest until after patients are weaned from sedation.¹²⁶



BP should be monitored in individuals with SCI due to possible disruption of the sympathetic nervous system. This disruption can result in \star vascular tone and/or orthostatic hypotension. Autonomic dysreflexia (AD) also may occur, causing an acute, unregulated elevation of SBP, which is often associated with baroreflex-mediated bradycardia¹³²

- Patients exhibiting AD with HTN should be positioned upright for postural reduction of SBP. PT providers should seek to immediately identify and remove noxious stimulus.
- ↓ sympathetic stimulation may blunt cardiovascular response to exercise.

Patients meeting one or more of the following criteria should be screened for orthostatic hypotension, and blood pressure should be monitored during and after exercise. Exercise may exacerbate neurogenic orthostatic hypotension¹³³

- Neurodegenerative disorder associated with autonomic dysfunction (PD, MS, Multiple System Atrophy, Dementia with Lewy Bodies)
 - Over 1/3 of patients with PD met criteria for orthostatic hypotension and may be asymptomatic¹³⁴
- Unexplained fall or syncopal event
- Peripheral neuropathies are associated with autonomic dysfunction (DM, Amyloidosis, HIV)
- > 70 years of age and meets frailty criteria or takes multiple medications
- Postural dizziness or non-specific symptoms when standing

In patients with neuromuscular or neurodegenerative diseases that impact the brainstem, it is critical to monitor pulmonary status and VS¹³⁵⁻¹³⁷

- Patients with MS-associated brainstem demyelination have ↑ risk of dysrhythmia, requiring close ECG and vital sign monitoring¹³⁸⁻¹⁴⁰
- Patients with MS may have blunted vascular response and ↓ ability to maintain BP, resulting in ↑ prevalence of postural orthostatic tachycardia syndrome (POTS), orthostatic hypotension, orthostatic intolerance, and postural dizziness¹⁴¹
- Patients with GBS-associated disturbance of baroreceptor regulation may have ↑ BP lability and require close monitoring¹⁴²
- Patients with some neurodegenerative conditions may exhibit blunted or variable chronotropic response to exercise. Use Borg RPE Scale in addition to PR to monitor exercise intensity and tolerance⁶⁵

APTA Acute Care. Academy of Cardiovascular & Pulmonary Physical Therapy

Pharmacologic Considerations for Vital Signs^{13,144-146}

In this section, we highlight certain classes of medications and their associated VS implications. This is not an all-inclusive list, and specific drugs listed under each drug medication class are examples. Additional resources should be consulted for increased depth of information regarding other aspects about the medications and other medication classes not listed here. While it is not in a physical therapist's scope to prescribe medications, it is within a physical therapist's scope to consider the impact on health, function, movement, and disability. Per APTA, "it is within the physical therapist's professional scope of practice to administer and store medication to facilitate outcomes of physical therapist patient and client management."¹⁴⁷

The influences of other factors such as timing, dosing, patient population, absorption, distribution, metabolism, elimination, pharmacodynamics, and polypharmacy are not included in this table. Adverse reactions are more likely to occur with the first dose or when a dose is increased. Medications given as IV drips generally have a short half-life and are dosed in mcg/kg/min. Dose titration up or down can result in physiologic fluctuations that necessitate closer monitoring of VS as well as other signs and symptoms. The first table contains medications that primarily treat cardiovascular and/ or pulmonary dysfunctions and can have a direct effect on VS. The second table contains additional medications that also require close monitoring due to their effects on VS.

| Medication Class | Common Indications | Effects on VS | Common and/or Dangerous Adverse Reactions | Clinical Considerations |
|---|--|---|---|---|
| Angiotensin Converting Enzymes (ACE) Inhibitors • Lisinopril (Zestril, Prinivil) • Ramipril (Altace) | HTN Heart failure Coronary artery disease Renal disease | → BP (SBP > DBP) at rest and with exercise ↑ Exercise tolerance in patients with HF | Dry, hacking cough Angioedema Hypotension Dizziness | Closely monitor BP If patient has dry, hacking cough, consider consult to prescribing practitioner; many patients can switch to angiotensin II receptor blocker (same clinical outcome with less frequency of cough) |
| Adrenergic Agonists (Combined Alpha/ Beta) • Levophed (norepinephrine bitartrate) | Shock (septic, cardiogenic, anaphylactic) Heart failure | ↑ BP via ↑ in peripheral vascular resistance ↑ HR ↑ MAP | Peripheral tissue necrosis Extravasation Dysrhythmias Myocardial ischemia Hyperglycemia | Classified as vasopressors and often referred to as "pressors" Closely monitor BP Monitor for impairment in peripheral tissue perfusion Extremely short acting (½-life ~ 3 minutes) necessitates continuous intravenous drip |

APTA Acute Care. 🔅 APTA Academy of Cardiovascular & Pulmonary Physical Therapy

| Medication Class | Common Indications | Effects on VS | Common and/or Dangerous Adverse Reactions | Clinical Considerations |
|--|--|---|--|--|
| Alpha Blockers Alfuzosin (Uroxatral) Silodosin (Rapaflo) Tamsulosin (Flomax) Doxazosin (Cardura) Terazosin (Hytrin) Prazosin (Minipress) | HTN Benign prostatic hyperplasia | • ↓BP | Dizziness Headache Hypotension (exacerbated by postural changes | Hypotension, dizziness, and syncope are most likely to occur with first doses and when + dosage or switching between forms Assess orthostatic VS regularly If positive for orthostatic hypotension: discuss with medical team; consider modifying treatment plan, e.g. perform activities in sitting |
| ACE Inhibitor/ARB Sacubitril and Valsartan (Entresto) | Heart failure with ↓ ejection fraction | • ↓BP | Hypotension Fatigue Hyperkalemia Renal failure Dizziness Cough | Monitor BP, HR, and ECG |
| Angiotensin II receptor blocker (ARB) Losartan (Cozaar) Valsartan (Diovan) | HTN Heart failure Renal disease/ failure | • ↓BP | Dizziness Hyperkalemia Hypotension Dry cough Angioedema Diarrhea Weight loss | ARBs may be preferred if patients are unable to tolerate the side effects of ACE Inhibitors, such as coughing |
| Anticoagulants Apixaban (Eliquis) Warfarin (Coumadin) | Prevention and treatment of VTE and other blood clots, including atrial fibrillation and artificial heart valves | No change, unless major bleeding event | Bleeding (risk higher for anticoagulants) Spinal/Epidural Hematoma | Watch for signs of bleeding that could include dizziness, as well tachycardia and hypotension |
| Antiplatelet Agents Clopidogrel (Plavix) Ticagrelor (Brilinta) | Secondary prevention of MI or ischemic stroke Peripheral artery disease | No change, unless major bleeding event Bradyarrhyth- mias and dyspnea (15-20% of patients on Brilinta) | Bleeding Dyspnea Bradycardia/ Bradyarrhyth- mias (Brilinta) | Watch for signs of bleeding that could include dizziness, as well tachycardia and hypotension. Monitor HR and ECG |



| Medication Class | Common Indications | Effects on VS | Common and/or Dangerous Adverse Reactions | Clinical Considerations |
|---|---|---|---|--|
| Beta Blockers Metoprolol (Toprol XL, Lopressor) Atenolol (Tenormin) Carvedilol (Coreg) | Acute MI HTN Heart failure with systolic dysfunction Dysrhythmia Migraine prophylaxis | ↓ BP (SBP > DBP) ↓ resting HR ↓ HR with exercise | Bronchospasm Heart block Masks signs and symptoms of hypoglycemia | Use RPE scale in addition to HR to monitor exercise intensity ↑ risk of developing or worsening heart block; monitor ECG Auscultate to monitor for bronchospasm |
| Calcium Channel Blockers • Diltiazem (Cardizem) • Verapamil (Calan) | HTN Dysrhythmias Ischemic heart disease Vasospastic angina Peripheral vasospasm (e.g. Raynaud's, Buerger's) | • ↓ BP • ↓ HR | Bradycardia Orthostatic hypotension Syncope Dizziness Lightheaded- ness Headache | Hypotension, dizziness and syncope are most likely to occur with first doses and when ↑ dosage or switching between forms May contribute to ↑ fall risk ↑ risk of developing or worsening heart block; monitor ECG Assess orthostatic VS regularly. If positive for orthostatic hypotension: discuss with medical team; consider modifying treatment plan, e.g. perform activities in sitting |

APTA Acute Care. C Academy of Cardiovascular & Pulmonary Physical Therapy

| Medication Class | Common Indications | Effects on VS | Common and/or Dangerous Adverse Reactions | Clinical Considerations |
|--|--|--|--|---|
| Potassium (K+) Sparing Diuretics/ Aldosterone Antagonists • Amiloride (Midamor) • Spironolactone (Aldactone) | Heart failure Hypokalemia HTN | • ↓BP | Fluid/electrolyte imbalance Dizziness Headache Dysrhythmia Hyperuricemia/ gout flare | Monitor for signs and symptoms of fluid-electrolyte imbalance e.g. hyperkalemia, fatigue, confusion, nausea, and al- tered heart rate; discuss abnormal findings with the medical team |
| Loop and Thiazide Diuretics Furosemide (Lasix) Hydrochlorothi- azide (HydroDiuril) | Certain renal disorders HTN ↓ edema resulting from: Heart failure Cirrhosis Meniere's | ↓ BP (SBP > DBP) | Hypotension (exacerbated by postural changes) Dehydration Ventricular dysrhythmias (due to potassium imbalances) Hyperglycemia in patients with DM Hyperuricemia/ gout flare | Assess orthostatic VS regularly If positive for orthostatic hypotension: discuss with medical team; consider modifying treatment plan, e.g. perform activities in sitting; consid- er discussing fluid status with healthcare team Monitor ECG |
| Anticholinergics (Inhaled) • Ipratropium (Atrovent) • Tiotropium (Spiriva) | Bronchocon- striction/ bronchospasm Asthma COPD | ↑ temperature ↑ ECG changes | Bronchitis Headache | Monitor for CNS effects such as dizziness and blurred vision Monitor for signs and symptoms of anticholinergic poisoning i.e. tachycardia, flushing, anhi- drosis (inability to perspire), hyperther- mia, mydriasis (dilated pupils), agitated delirium, and diminished bowel sounds; discuss abnormal findings with the medical team |

APTA Acute Care

| Medication Class | Common Indications | Effects on VS | Common and/or Dangerous Adverse Reactions | Clinical Considerations |
|---|---|--|--|--|
| Beta, Agonists (Inhaled) · Albuterol (Ventolin; Proventil) | Bronchocon- striction/ bronchospasm Asthma COPD | Acute ↑ HR with administration | Tremor Nervousness Bronchospasm Tachycardia | Monitor for immediate hypersensitivity reactions i.e. urticaria, angioedema, rash, bronchospasm, oropharyngeal edema, including ana- phylaxis; discuss abnormal findings with the medical team |
| Beta, Agonists (Positive Inotrope) • Dopamine (Alpha and Beta, agonist activity) | Shock (cardiogenic, hypovolemic, septic, anaphylactic, etc.) Heart failure | ↑ BP via ↑ in SV and vasoconstriction resulting in ↑CO + Chronotropic effects ↑ HR | Dysrhythmias (↑ incidence of tachydysrhyth- mias in patients with shock) | Use RPE scale in addition to HR to monitor exercise intensity Monitor ECG |
| Cardiac Glycosides (Positive Inotrope) • Primacor (Milrinone) | Acute decompensat- ed heart failure Dysrhythmia | ↑ BP via ↑ in SV ↓ Peripheral vascular resistance | Dysrhythmias (tachycardia, ventricular, supraventricu- lar) Hypotension Headache Bronchospasm | Administered intravenously Short half-life (~2 hrs) Use RPE scale in addition to HR to monitor exercise intensity Monitor ECG |
| Other Positive Inotropes ∙ Dobutamine | Shock (cardiogenic, hypovolemic, septic, anaphylactic, etc.) | ↑ BP via ↑in SV and significant vasoconstriction (pressor effect) | Headache Anxiety Dyspnea Severe HTN Asthma exacerbation Dysrhythmias (bradycardia, irregular) | Closely monitor BP Use RPE scale in addition to HR to monitor exercise intensity |
| Nitrates and Nitrites • Nitroglycerin (Rectiv, Nitrolingual, Nitrostat) • Isosorbide dinitrate (Isordil) • Amyl nitrite - inhaled | Angina pectoris Hypertensive crisis Hypertensive pulmonary edema Heart failure Low CO syndromes Acute MI | ↓ BP via systemic vasodilation ↑ HR at rest and with exercise | Headache Dizziness Orthostatic hypotension Nausea | Closely monitor BP Nitroglycerin requires special handling and storage to avoid skin contact with the medication |

APTA Acute Care. C Academy of Cardiovascular & Pulmonary Physical Therapy

| Additional Non- Cardiovascular and Pulmonary Medication Classes | Common and/or Dangerous Adverse Reactions Affecting VS and PT Intervention | Clinical Considerations | |
|---|--|--|--|
| Neuromuscular Blocking Agents Nimbex (Cisatracurium) Rocuronium (Zemuron) | Dysrhythmia Hypotension ICU-acquired muscle weakness | Closely monitor BP, PR, and ECG Monitor for impairment of muscle weakness including diaphragm Requires mechanical ventilation | |
| Sedative-Hypnotics Benzodiazepines Alprazolam (Xanax) Diazepam (Valium) Lorazepam (Ativan) Midazolam (Versed) Nonbenzodiazepines Eszopiclone (Lunesta) Zaleplon (Sonata) Zolpidem (Ambien) | CNS depression Drowsiness Amnesia Muscle incoordination Impaired cognition/delirium | Monitor for fall risk (especially in older adults) Monitor for neuromusculo- skeletal impairments Monitor for changes in mental status/cognition Any abrupt or overly rapid reduction in benzodiazepine dose among chronic users can produce withdrawal Monitor for withdrawal symptoms i.e. tremor, anxiety, perceptual disturbances, dysphoria, psychosis, and seizures; discuss symptoms with medical team | |
| General Anesthetics Propofol (Diprivan) Ketamine (Ketalar) | Delirium Dysrhythmia (bradycardia or tachycardia) Hypotension or hypertension Apnea/Respiratory depression Diplopia Hypertonia Hyperlipidemia (Propofol) CO (Propofol) cardiac contractility and vasoconstriction (Ketamine) | Long term use can lead to dependency so monitor for withdrawal symptoms i.e. tremor, anxiety, perceptual disturbances, dysphoria, psychosis, and seizures; discuss abnormal findings with medical team Monitor for changes in mental status/cognition Patients with obesity may take longer to recover from Propofol sedation Closely monitor RR, SpO₂, BP, PR, and ECG | |
| Opioids • Oxycodone (OxyContin) | Hypotension Respiratory depression/apnea CNS depression ↑ ICP Dizziness Confusion | Monitor closely for respiratory depression, especially during initiation or dose escalation. CO₂ retention from opioid - induced respiratory depression can exacerbate the sedating effects of opioids. Monitor for fall risk (especially in older adults) Monitor for changes in mental status/cognition Long term use can lead to dependency so monitor for withdrawal symptoms, i.e. tremor, anxiety, perceptual disturbances, dysphoria, psychosis, and seizures; discuss symptoms with medical team | |



| Additional Non- Cardiovascular and Pulmonary Medication Classes | Common and/or Dangerous Adverse Reactions Affecting VS and PT Intervention | Clinical Considerations | |
|---|---|---|--|
| Skeletal Muscle Relaxants Baclofen-oral (Lioresal) Cyclobenzaprine (Flexeril) Metaxalone (Skelaxin) | Hypotension Bradycardia CNS depression | Monitor for signs of toxicity including dysrhythmias Closely monitor BP for orthostatic hypotension Monitor for fall risk (especially in older adults) Monitor for neuromusculo- skeletal impairments Monitor for changes in mental status/cognition | |
| Agents for Bipolar Disorder Lithium (Lithobid) Tegretol (Carbamazepine) Depakote (Divalproex) | Tardive dyskinesia Potential for cardiovascular instability as a sign of toxicity i.e. syncope, bradycardia, AV block, other atrial/ventricular dysrhythmias | Any acute changes in cognition, neuromuscular system, or VS should warrant an urgent discussion with the medical team, due to possibility of toxicity | |
| Tricyclic Antidepressants Amitriptyline (Amitriptyline) Clomipramine (Anafranil) Imipramine (Tofranil) | Orthostatic hypotension Dysrhythmia CNS depression Headache Syncope Tardive dyskinesia | Monitor for suicidal thinking and behavior in children, adolescents, and young adults (18 - 24 years of age) with major depressive disorder and other psychiatric disorders; discuss abnormal findings with medical team Closely monitor BP for orthostatic hypotension Monitor for fall risk due to ataxia and impaired coordination Monitor for neuromusculo- skeletal impairments Monitor for changes in mental status/cognition Monitor for anticholinergic effects i.e. constipation, xerostomia, blurred vision, urinary retention | |
| Anti-Parkinsonian agents Carbidopa/Levodopa (Sinemet, Duopa) Dopamine agonist (Requip, Mirapex) | Dysrhythmia Hypotension (exacerbated by postural changes) Dizziness Syncope Neuroleptic malignant syndrome (tachycardia, fever, confusion, BP instability) is a rare but potentially fatal adverse reaction associated with abrupt withdrawal | Monitor for signs of toxicity including dysrhythmias i.e. tachycardia Closely monitor BP for orthostatic hypotension Monitor for fall risk Monitor for neuromusculo- skeletal impairments Medications may be stopped due to medical procedures; discuss with medical team Closely monitor VS especially BP in advanced stages of Parkinson's Disease due to autonomic instability¹⁴⁸ Monitor for neuroleptic malignant syndrome and discuss abnormal findings with medical team | |



| Additional Non- Cardiovascular and Pulmonary Medication Classes | Common and/or Dangerous Adverse Reactions Affecting VS and PT Intervention | Clinical Considerations |
|--|---|---|
| Corticosteroids (oral) Prednisone (Rayos) Dexamethasone (Decadron) | Impairs tissue healing and immune response (can inhibit ability to generate fever) High-dose or prolonged use effects: Adrenal atrophy/ crisis Cushing's syndrome Hyperglycemia Dyslipidemia HTN Hypokalemia Osteopenia/ osteoporosis Proximal myopathy Peptic ulcers/ GI bleeds Mood changes to psychosis | Monitor for other signs and symptoms of fever Monitor BP, HR, and ECG Monitor for fall risk (especially in older adult) Monitor for neuromusculo- skeletal impairments |
| Angiogenesis InhibitorsAvastin (Bevacizumab) | • HTN | Monitor VS frequently especially BP |
| Insulin ¹⁴⁹ | Hypoglycemia leads to ↑ BP and ↑ HR | Monitor VS closely Monitor for signs and symptoms of hypoglycemia |



References

- 1. Guide to Physical Therapist Practice 3.0. In: Alexandria, VA: American Physical Therapy Association; 2014: http://guidetoptpractice.apta.org/. Accessed January 21, 2021.
- 2. Frese EM, Fick A, Sadowsky HS. Blood pressure measurement guidelines for physical therapists. Cardiopulm Phys Ther J. 2011;22(2):5-12.
- Whelton PK, Carey L, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Hypertension. 2018;71(6):1269-1324.
- 4. Cheshire W. Autonomic Disorders and Their Managment In: L G, AI S, eds. Goldman-Cecil Medicine. 25th ed. St. Louis, MO: Elsevier Saunders; 2016.
- 5. Calkins H, Zipes D. Hypotension and Syncope. In: Zipes D, Libby P, Bonow D, Mann G, Tomaselli G, Braunwald E, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine 11th ed. St. Louis, MO: Elsevier; 2019.
- 6. DeMers D, Wachs D. Physiology, Mean Arterial Pressure. StatPearls. StatPearls Web site. https:// www.ncbi.nlm.nih.gov/books/NBK538226/. Published 2020. Updated August 22, 2020. Accessed January 21, 2021.
- 7. Avanzini F, Alli C, Boccanelli A, et al. High pulse pressure and low mean arterial pressure: two predictors of death after a myocardial infarction. Journal of hypertension. 2006;24(12):2377-2385.
- 8. Vedel AG, Holmgaard F, Rasmussen LS, et al. Perfusion Pressure Cerebral Infarct (PPCI) trial the importance of mean arterial pressure during cardiopulmonary bypass to prevent cerebral complications after cardiac surgery: study protocol for a randomised controlled trial. Trials. 2016;17(1):247.
- 9. Walsh M, Devereaux PJ, Garg AX, et al. Relationship between Intraoperative Mean Arterial Pressure and Clinical Outcomes after Noncardiac Surgery: Toward an Empirical Definition of Hypotension. Anesthesiology. 2013;119(3):507-515.
- Homan T, Bordes S, Cichowski E. Physiology, Pulse Pressure. StatPearls Publishing. StatPearls Web site. https://www.ncbi.nlm.nih.gov/books/NBK482408/. Published 2020. Updated June 7, 2020. Accessed January 21, 2021.
- 11. Pickering T, Hall JE, Appel LJ, et al. Recommendations for Blood Pressure Measurement in Humans and Experimental Animals. Circulation. 2005;111(5):697-716.
- 12. Bloomfield D, Park A. Night time blood pressure dip. (1949-8462 (Print)).
- 13. ACSM. Guidelines for Exercise Testing and Prescription. 10th ed. Philadelphia, PA: Wolters Kluwer; 2018.
- O'Brien E, Asmar R, Beilin L, et al. European Society of Hypertension recommendations for conventional, ambulatory and home blood pressure measurement. Journal of Hypertension. 2003;21(5):821-848.
- 15. Qaseem A, Wilt TJ, Rich R, Humphrey L, Frost J, Forciea MA. TREATMENT OF HYPERTENSION IN ADULTS OVER AGE 60 TO HIGHER VS LOWER TARGETS: A CLINICAL PRACTICE GUIDELINE FROM THE AMERICAN COLLEGE OF PHYSICIANS AND THE AMERICAN ACADEMY OF FAMILY PHYSICIANS. Ann Fam Med. 2017;15(2):185-186.
- 16. Watchie J. Cardiovascular and Pulmonary Physical Therapy: A Clinical Manual. 2nd ed. St. Louis, MO: Elsevier Saunders; 2010.
- 17. Group TSMIftSR. Effect of Intensive vs Standard Blood Pressure Control on Probable Dementia: A Randomized Clinical Trial. JAMA. 2019;321(6):553-561.
- Hughes D, Judge C, Murphy R, et al. Association of Blood Pressure Lowering With Incident Dementia or Cognitive Impairment: A Systematic Review and Meta-analysis. Jama. 2020;323(19):1934-1944.
- 19. Momtaz YA, Hamid TA, Haron SA, Bagat MF, Mohammadi F. Prevalence of hypotension and its association with cognitive function among older adults. Aging Ment Health. 2018;22(4):447-452.

APTA Acute Care. Academy of Cardiovascular & Pulmonary Physical Therapy:

- 20. Frewen J, Savva GM, Boyle G, Finucane C, Kenny RA. Cognitive performance in orthostatic hypotension: findings from a nationally representative sample. J Am Geriatr Soc. 2014;62(1):117-122.
- 21. O'Hare C, Kenny R-A, Aizenstein H, et al. Cognitive Status, Gray Matter Atrophy, and Lower Orthostatic Blood Pressure in Older Adults. Journal of Alzheimer's Disease. 2017;57:1239-1250.
- 22. Zhang D, Shen X, Qi X. Resting heart rate and all-cause and cardiovascular mortality in the general population: a meta-analysis. Canadian Medical Association Journal. 2016;188:E53-E63.
- 23. Li K, Yao C, Yang X, Dong L. Effect of Resting Heart Rate on All-Cause Mortality and Cardiovascular Events According to Age. Journal of the American Geriatrics Society. 2017;65(5):989-994.
- 24. O'Sullivan S, Schmitz T, Fulk G. Physical Rehabilitation. 7th ed. Philadelphia, PA: F.A. Davis; 2019.
- 25. Paz J, West M, Panasci K, Greenwood K. Acute Care Handbook for Physical Therapists. 5th ed. St. Louis, MO: Elsevier; 2019.
- 26. Fu Q, Levine BD. Exercise and non-pharmacological treatment of POTS. Auton Neurosci. 2018;215:20-27.
- 27. Chan E, Anderson CS, Wang X, et al. Early Blood Pressure Lowering Does Not Reduce Growth of Intraventricular Hemorrhage following Acute Intracerebral Hemorrhage: Results of the INTERACT Studies. Cerebrovascular Diseases Extra. 2016;6(3):71-75.
- 28. Persinger R, Foster C, Gibson M, Fater DC, Porcari JP. Consistency of the talk test for exercise prescription. Med Sci Sports Exerc. 2004;36(9):1632-1636.
- 29. Borg G. Perceived exertion as an indicator of somatic stress. Scand J Rehabil Med. 1970;2(2): 92-98.
- 30. Respiratory Responses to Exercise. PT Direct. https://www.ptdirect.com/training-design/ anatomy-and-physiology/acute-respiratory-responses. Published 2020. Accessed January 21, 2021.
- 31. ATS/ACCP Statement on cardiopulmonary exercise testing. Am J Respir Crit Care Med. 2003;167(2):211-277.
- 32. Hafen BB, Sharma S. Oxygen Saturation. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2020.
- 33. Sjoding MW, Dickson RP, Iwashyna TJ, Gay SE, Valley TS. Racial Bias in Pulse Oximetry Measurement. N Engl J Med. 2020;383(25):2477-2478.
- 34. Bickler PE, Feiner JR, Severinghaus JW. Effects of skin pigmentation on pulse oximeter accuracy at low saturation. Anesthesiology. 2005;102(4):715-719.
- 35. Jefferies S, Weatherall M, Young P, Beasley R. A systematic review of the accuracy of peripheral thermometry in estimating core temperatures among febrile critically ill patients. Crit Care Resusc. 2011;13(3):194-199.
- 36. McCullough L, Arora S. Diagnosis and Treatment of Hypothermia. Am Fam Physician. 2004;70:2352-2332.
- 37. Gleeson M. Temperature regulation during exercise. Int J Sports Med. 1998;19 Suppl 2:S96-99.
- 38. Goodman C, Fuller K. Pathology. 5th ed. St. Louis, MO: Elsevier; 2020.
- 39. Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. Crit Care Med. 1985;13(10):818-829.
- 40. Koenig MA. Cerebral Edema and Elevated Intracranial Pressure. Continuum (Minneap Minn). 2018;24(6):1588-1602.
- 41. Ragland J, Lee K. Critical Care Management and Monitoring of Intracranial Pressure. J Neurocrit Care. 2016;9(2):105-112.
- 42. Le Roux P. Frontiers in Neuroscience Intracranial Pressure Monitoring and Management. In: Laskowitz D, Grant G, eds. Translational Research in Traumatic Brain Injury. Boca Raton, FL: CRC Press/Taylor and Francis Group; 2016.
- 43. Mayer KP, Joseph-Isang E, Robinson LE, Parry SM, Morris PE, Neyra JA. Safety and Feasibility of Physical Rehabilitation and Active Mobilization in Patients Requiring Continuous Renal Replacement Therapy: A Systematic Review. Crit Care Med. 2020;48(11):e1112-e1120.
- 44. Poukkanen M, Wilkman E, Vaara ST, et al. Hemodynamic variables and progression of acute kidney injury in critically ill patients with severe sepsis: data from the prospective observational FINNAKI study. Crit Care. 2013;17(6):R295-R295.

APTA Acute Care. Academy of Cardiovascular & Pulmonary Physical Therapy

- 45. Wang X-T, Wang C, Zhang H-M, Liu D-W. Clarifications on Continuous Renal Replacement Therapy and Hemodynamics. Chinese Medical Journal. 2017;130(10):1244-1248.
- 46. Chung M, Shiloh AL, Carlese A. Monitoring of the Adult Patient on Venoarterial Extracorporeal Membrane Oxygenation. The Scientific World Journal. 2014;2014:393258.
- 47. Mossadegh C. Monitoring the ECMO. In: Mossadegh C, Combes A, eds. Nursing Care and ECMO. Cham: Springer International Publishing; 2017:45-70.
- 48.Jankowski P, Kawecka-Jaszcz K, Czarnecka D, et al. Pulsatile but not steady component of blood pressure predicts cardiovascular events in coronary patients. Hypertension. 2008;51(4):848-855.
- 49. Rickelmann C, Knoblauch DJ. Incorporating Safe Patient-Handling Techniques to Mobilize Our Most Complex Patients on Extra Corporeal Membrane Oxygenation. Critical Care Nursing Quarterly. 2018;41(3):272-281.
- 50. Estep JD, Trachtenberg BH, Loza LP, Bruckner BA. Continuous flow left ventricular assist devices: shared care goals of monitoring and treating patients. Methodist Debakey Cardiovasc J. 2015;11(1):33-44.
- 51. Khan T, Siddiqui A. Intra-Aortic Balloon Pump. StatPearls. https://www.ncbi.nlm.nih.gov/books/ NBK542233/. Published 2020. Updated May 5, 2020. Accessed January 21, 2021.
- 52. AACN, Wiegand D. AACN Procedure Manual for High Acuity, Progressive, and Critical Care. 7th ed. St. Louis, MO: Elsevier Evolve; 2017.
- 53. Knippa S. Blood Pressure Monitoring During Intra-Aortic Balloon Pumping. Crit Care Nurse. 2019;39(2):99-101.
- 54. Castagna F, Stöhr EJ, Pinsino A, et al. The Unique Blood Pressures and Pulsatility of LVAD Patients: Current Challenges and Future Opportunities. Current Hypertension Reports. 2017;19(10):85.
- 55. Feldman D, Pamboukian SV, Teuteberg JJ, et al. The 2013 International Society for Heart and Lung Transplantation Guidelines for mechanical circulatory support: Executive summary. The Journal of Heart and Lung Transplantation. 2013;32(2):157-187.
- 56. EMS Guide Complete Document. In: Clinicians ICoCA, ed.2019: https://www.mylvad.com/sites/ default/files/EMS%20Field%20Guides/2019%20Field%20Guides/Total%20Document/2019%20 EMS%20Field%20Guide%20Complete%20Document.pdf. Accessed January 21, 2021.
- 57. Aldrich TK, Gupta P, Stoy SP, Carlese A, Goldstein DJ. Pulseless Oximetry: A Preliminary Evaluation. Chest. 2015;148(6):1484-1488.
- 58. Scheiderer R, Belden C, Schwab D, Haney C, Paz J. Exercise guidelines for inpatients following ventricular assist device placement: a systematic review of the literature. Cardiopulm Phys Ther J. 2013;24(2):35-42.
- 59. Singer M, Deutschman CS, Seymour CW, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315(8):801-810.
- 60. Marik PE, Taeb AM. SIRS, qSOFA and new sepsis definition. Journal of thoracic disease. 2017;9(4):943-945.
- 61. Kayambu G, Boots R, Paratz J. Early physical rehabilitation in intensive care patients with sepsis syndromes: a pilot randomised controlled trial. Intensive Care Med. 2015;41(5):865-874.
- 62. Amsterdam EA, Wenger NK, Brindis RG, et al. 2014 AHA/ACC Guideline for the Management of Patients With Non–ST-Elevation Acute Coronary Syndromes. Circulation. 2014;130(25):e344-e426.
- 63. O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Journal of the American College of Cardiology. 2013;61(4):e78-e140.
- 64. Kumar A, Cannon CP. Acute coronary syndromes: diagnosis and management, part I. Mayo Clinic proceedings. 2009;84(10):917-938.
- 65. ACSM's Exercise Management for Persons with Chronic Diseases and Disabilities. Champaign, IL: Human Kinetics; 2016.

APTA Acute Care. C Academy of Cardiovascular & Pulmonary Physical Therapy

- 66. Achttien RJ, Staal JB, van der Voort S, et al. Exercise-based cardiac rehabilitation in patients with coronary heart disease: a practice guideline. Neth Heart J. 2013;21(10):429-438.
- 67. Sanderson BK, Southard D, Oldridge N. AACVPR consensus statement. Outcomes evaluation in cardiac rehabilitation/secondary prevention programs: improving patient care and program effectiveness. J Cardiopulm Rehabil. 2004;24(2):68-79.
- 68. AACVPR. Guidelines for Cardiac Rehabilitation and Secondary Prevention Programs. Champaign, IL: Human Kintetics; 2004.
- 69. Omar W, Pandey A, Haykowsky MJ, Berry JD, Lavie CJ. The Evolving Role of Cardiorespiratory Fitness and Exercise in Prevention and Management of Heart Failure. Curr Heart Fail Rep. 2018;15(2):75-80.
- 70. Shoemaker MJ, Dias KJ, Lefebvre KM, Heick JD, Collins SM. Physical Therapist Clinical Practice Guideline for the Management of Individuals With Heart Failure. Physical Therapy. 2020;100(1):14-43.
- 71. Bonaca M, Creager MA. Peripheral artery disease. In: Zipes DP, Libby P, Bonow RO, Mann DL, Tomaselli G, Braunwald E, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 11th ed. Philadelphia, PA: Elsevier; 2019.
- 72. Wang JC, Criqui MH, Denenberg JO, McDermott MM, Golomb BA, Fronek A. Exertional Leg Pain in Patients With and Without Peripheral Arterial Disease. Circulation. 2005;112(22): 3501-3508.
- 73. Hirsch AT, Criqui MH, Treat-Jacobson D, et al. Peripheral Arterial Disease Detection, Awareness, and Treatment in Primary Care. JAMA. 2001;286(11):1317-1324.
- 74. Gerhard-Herman MD, Gornik HL, Barrett C, et al. 2016 AHA/ACC Guideline on the Management of Patients With Lower Extremity Peripheral Artery Disease: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2017;135(12):e686-e725.
- 75. Fakhry F, Rouwet EV, den Hoed PT, Hunink MG, Spronk S. Long-term clinical effectiveness of supervised exercise therapy versus endovascular revascularization for intermittent claudication from a randomized clinical trial. Br J Surg. 2013;100(9):1164-1171.
- 76. Fokkenrood HJP, Bendermacher BLW, Lauret GJ, Willigendael EM, Prins MH, Teijink JAW. Supervised exercise therapy versus non-supervised exercise therapy for intermittent claudication. Cochrane Database of Systematic Reviews. 2013(8).
- 77. Lane R, Ellis B, Watson L, Leng GC. Exercise for intermittent claudication. Cochrane Database Syst Rev. 2014(7):Cd000990.
- 78. Murphy TP, Cutlip DE, Regensteiner JG, et al. Supervised Exercise, Stent Revascularization, or Medical Therapy for Claudication Due to Aortoiliac Peripheral Artery Disease: The CLEVER Study. Journal of the American College of Cardiology. 2015;65(10):999-1009.
- 79. Braverman A, Thompson R, Sanchez L. Diseases of the aorta. In: Bonow RO, Mann D, Zipes D, Libby P, eds. Braunwald's heart disease. 9th ed. Philadelphia: Elsevier; 2011:1309.
- 80. Hiratzka LF, Creager MA, Isselbacher EM, et al. Surgery for Aortic Dilatation in Patients With Bicuspid Aortic Valves. Circulation. 2016;133(7):680-686.
- 81. Aronow WS. Prevention/detection/management of abdominal aortic aneurysm. AME Medical Journal. 2016;1(3).
- 82. Jim J, Thompson R. Clinical features and diagnosis of abdominal aortic aneurysm. UptoDate. https://www-uptodate-com/contents/clinical-features-and-diagnosis-of-abdominal-aorticaneurysm Published 2019. Accessed December 1, 2019.
- 83. Myers J, McElrath M, Jaffe A, et al. A Randomized Trial of Exercise Training in Abdominal Aortic Aneurysm Disease. Medicine & Science in Sports & Exercise. 2014;46(1):2-9.
- 84.Goldhaber S. Pulmonary embolism. In: Zipes DP, Libby P, Bonow D, Mann D, Tomaselli G, Braunwald E, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine 11th ed. Philadelphia, PA: Elsevier; 2019.

APTA Acute Care. (CAPTA Academy of Cardiovascular & Pulmonary Physical Therapy:

- 85. Pai M, Douketis J. Prevention of venous thromboembolic disease in acutely ill hospitalized medical adults. UptoDate. http://www.uptodate.com/prevention-of-venous-thromboembol-ic-disease-in-acutely-ill-hospitalized-medical-adults. Published 2019. Accessed September 21, 2020.
- 86. Barbar S, Noventa F, Rossetto V, et al. A risk assessment model for the identification of hospitalized medical patients at risk for venous thromboembolism: the Padua Prediction Score. J Thromb Haemost. 2010;8(11):2450-2457.
- 87. Bates SM, Jaeschke R, Stevens SM, et al. Diagnosis of DVT: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest. 2012;141(2 Suppl):e351S-e418S.
- 88. Alikhan R, Cohen AT, Combe S, et al. Risk factors for venous thromboembolism in hospitalized patients with acute medical illness: analysis of the MEDENOX Study. Arch Intern Med. 2004;164(9):963-968.
- 89. Wells PS, Anderson DR, Rodger M, et al. Evaluation of D-Dimer in the Diagnosis of Suspected Deep-Vein Thrombosis. New England Journal of Medicine. 2003;349(13):1227-1235.
- 90. Hillegass E, Puthoff M, Frese EM, Thigpen M, Sobush DC, Auten B. Role of Physical Therapists in the Management of Individuals at Risk for or Diagnosed With Venous Thromboembolism: Evidence-Based Clinical Practice Guideline. Phys Ther. 2016;96(2):143-166.
- 91. Le Gal G, Righini M, Roy PM, et al. Prediction of pulmonary embolism in the emergency department: the revised Geneva score. Ann Intern Med. 2006;144(3):165-171.
- 92. Dutia M, White RH, Wun T. Risk assessment models for cancer-associated venous thromboembolism. Cancer. 2012;118(14):3468-3476.
- 93. DeTurk W, Cahalin L. Cardiovascular and Pulmonary Physical Therapy. 3rd ed. New York City, NY: McGraw Hill; 2017.
- 94. Hillegass E. Essentials of Cardiopulmonary Physical Therapy. 4th ed. St. Louis, MO: Elsevier; 2017.
- 95. King T. Approach to the adult with interstitial lung disease: Diagnostic testing. UptoDate. https://www.uptodate.com/contents/approach-to-the-adult-with-interstitial-lung-diseasediagnostic-testing" \I "H1. Published 2020. Accessed September 20, 2020.
- 96. Vogelmeier CF, Criner GJ, Martinez FJ, et al. Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Lung Disease 2017 Report. GOLD Executive Summary. Am J Respir Crit Care Med. 2017;195(5):557-582.
- 97. Raghu G, Collard HR, Egan JJ, et al. An Official ATS/ERS/JRS/ALAT Statement: Idiopathic Pulmonary Fibrosis: Evidence-based Guidelines for Diagnosis and Management. American Journal of Respiratory and Critical Care Medicine. 2011;183(6):788-824.
- 98. Driscoll BR, Howard LS, Earis J, Mak V. British Thoracic Society Guideline for oxygen use in adults in healthcare and emergency settings. BMJ Open Respiratory Research. 2017;4(1):e000170.
- 99. Beasley R, Chien J, Douglas J, et al. Thoracic Society of Australia and New Zealand oxygen guidelines for acute oxygen use in adults: 'Swimming between the flags'. Respirology. 2015;20(8):1182-1191.
- 100. Chronic obstructive pulmonary disease in over 16s: diagnosis and management. In: Guideline N, ed. Vol 115. London: National Institute for Health and Care Excellence; 2019: https://www.ncbi.nlm.nih.gov/books/NBK542426/. Accessed January 24, 2021.
- 101. De Sensi F, De Potter T, Cresti A, Severi S, Breithardt G. Atrial fibrillation in patients with diabetes: molecular mechanisms and therapeutic perspectives. Cardiovascular Diagnosis and Therapy. 2015;5(5):364-373.
- 102. Sydó N, Sydó T, Merkely B, et al. Impaired Heart Rate Response to Exercise in Diabetes and Its Long-term Significance. Mayo Clinic Proceedings. 2016;91(2):157-165.
- 103. Bokhari SRA, Akhtar F, Abid Q-U-A, et al. Managing Postural Hypotension in Diabetic Auto nomic Dysfunction When Adrenergic Drugs are Contraindicated: Case Report and Review of Literature. Cureus. 2018;10(1):e2039-e2039.

APTA Acute Care. C Academy of Cardiovascular & Pulmonary Physical Therapy:

- 104. Sutter JR, Matson AW. Orthostatic and Exertional Hypotension: Review and Implications for Physical Therapy. Journal of Acute Care Physical Therapy. 2020;11(1).
- 105. Pu LJ, Shen Y, Lu L, Zhang RY, Zhang Q, Shen WF. Increased blood glycohemoglobin A1c levels lead to overestimation of arterial oxygen saturation by pulse oximetry in patients with type 2 diabetes. Cardiovascular Diabetology. 2012;11(1):110.
- 106. Karayannis G, Giamouzis G, Cokkinos DV, Skoularigis J, Triposkiadis F. Diabetic cardiovas cular autonomic neuropathy: clinical implications. Expert Review of Cardiovascular Therapy. 2012;10(6):747-765.
- 107. American Diabetes Association Standards of Medical Care in Diabetes. In: Diabetes, ed.: American Diabetes Association; 2020: https://care.diabetesjournals.org/content/43/ Supplement_1. Accessed September 2020.
- 108. Colberg SR, Sigal RJ, Yardley JE, et al. Physical Activity/Exercise and Diabetes: A Position State ment of the American Diabetes Association. Diabetes Care. 2016;39:2065-2079.
- 109. Vinik AI, Erbas T. Chapter 22 Diabetic autonomic neuropathy. In: Buijs RM, Swaab DF, eds. Handbook of Clinical Neurology. Vol 117. Elsevier; 2013:279-294.
- 110.Schweickert WD, Pohlman MC, Pohlman AS, et al. Early physical and occupational therapy in mechanically ventilated, critically ill patients: a randomised controlled trial. Lancet. 2009;373(9678):1874-1882.
- 111. Williamson P. Exercise for Special Populations. 11th ed. Philadelphia: Lippincott-Raven; 2011.
- 112. Young-McCaughan S. Exercise in the rehabilitation from cancer. Medsurg Nurs. 2006;15(6): 384-387.
- 113. Timmers HJLM, Wieling W, Karemaker JM, Lenders JWM. Cardiovascular Responses to Stress after Carotid Baroreceptor Denervation in Humans. Annals of the New York Academy of Sciences. 2004;1018(1):515-519.
- 114. Campbell KL, Winters-Stone KM, Wiskemann J, et al. Exercise Guidelines for Cancer Survivors: Consensus Statement from International Multidisciplinary Roundtable. Med Sci Sports Exerc. 2019;51(11):2375-2390.
- 115. Campbell G, Wolfe RA, Klem ML. Risk Factors for Falls in Adult Cancer Survivors: An Integrative Review. Rehabil Nurs. 2018;43(4):201-213.
- 116. Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the Early Management of Patients With Acute Ischemic Stroke: 2019 Update to the 2018 Guidelines for the Early Management of Acute Ischemic Stroke: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke. 2019;50(12):e344-e418.
- 117. Willmot M, Leonardi-Bee J, Bath PM. High Blood Pressure in Acute Stroke and Subsequent Outcome. Hypertension. 2004;43(1):18-24.
- 118. Kang J, Ko Y, Park JH, et al. Effect of blood pressure on 3-month functional outcome in the subacute stage of ischemic stroke. Neurology. 2012;79(20):2018-2024.
- 119. Hemphill JC, 3rd, Greenberg SM, Anderson CS, et al. Guidelines for the Management of Spontaneous Intracerebral Hemorrhage: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke. 2015;46(7):2032-2060.
- 120. Connolly ES, Jr., Rabinstein AA, Carhuapoma JR, et al. Guidelines for the management of an eurysmal subarachnoid hemorrhage: a guideline for healthcare professionals from the American Heart Association/american Stroke Association. Stroke. 2012;43(6):1711-1737.
- 121. Tang C, Zhang TS, Zhou LF. Risk factors for rebleeding of aneurysmal subarachnoid hemorrhage: a meta-analysis. PLoS One. 2014;9(6):e99536.
- 122. Akca O, Nichols J, Stewart B, Elliott C, Remmel K, Lenhardt R. Association of Early Oxygenation Levels with Mortality in Acute Ischemic Stroke - A Retrospective Cohort Study. J Stroke Cerebrovasc Dis. 2020;29(2):104556.
- 123. Ferdinand P, Roffe C. Hypoxia after stroke: a review of experimental and clinical evidence. Exp Transl Stroke Med. 2016;8:9-9.

APTA Acute Care. Academy of Cardiovascular & Pulmonary Physical Therapy:

- 124. Brenner M, Stein DM, Hu PF, Aarabi B, Sheth K, Scalea TM. Traditional systolic blood pressure targets underestimate hypotension-induced secondary brain injury. Journal of Trauma and Acute Care Surgery. 2012;72(5):1456-1457.
- 125. Carney N, Totten AM, O'Reilly C, et al. Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition. Neurosurgery. 2016;80(1):6-15.
- 126. Meyfroidt G, Baguley IJ, Menon DK. Paroxysmal sympathetic hyperactivity: the storm after acute brain injury. Lancet Neurol. 2017;16(9):721-729.
- 127. Ahuja CS, Wilson JR, Nori S, et al. Traumatic spinal cord injury. Nat Rev Dis Primers. 2017;3:17018.
- 128. Schilero GJ, Bauman WA, Radulovic M. Traumatic Spinal Cord Injury: Pulmonary Physiologic Principles and Management. Clin Chest Med. 2018;39(2):411-425.
- 129. Arnold SM, Dinkins M, Mooney LH, et al. Very Early Mobilization in Stroke Patients Treated with Intravenous Recombinant Tissue Plasminogen Activator. Journal of Stroke and Cerebrovascular Diseases. 2015;24(6):1168-1173.
- 130. Bernhardt J, Churilov L, Ellery F, et al. Prespecified dose-response analysis for A Very Early Re habilitation Trial (AVERT). Neurology. 2016;86:2138-2145.
- 131. Silver B, Hamid T, Khan M, et al. 12 versus 24 h bed rest after acute ischemic stroke thromboly sis: a preliminary experience. J Neurol Sci. 2020;409:116618.
- 132. Eldahan KC, Rabchevsky AG. Autonomic dysreflexia after spinal cord injury: Systemic patho physiology and methods of management. Autonomic Neuroscience. 2018;209:59-70.
- 133. Eschlböck S, Wenning G, Fanciulli A. Evidence-based treatment of neurogenic orthostatic hy potension and related symptoms. Journal of Neural Transmission. 2017;124(12):1567-1605.
- 134. Merola A, Romagnolo A, Rosso M, et al. Orthostatic hypotension in Parkinson's disease: Does it matter if asymptomatic? Parkinsonism Relat Disord. 2016;33:65-71.
- 135. Sciacca S, Lynch J, Davagnanam I, Barker R. Midbrain, Pons, and Medulla: Anatomy and Syndromes. Radiographics. 2019;39(4):1110-1125.
- 136. Valencia-Sanchez C, Goodman BP, Carter JL, Wingerchuk DM. The spectrum of acute cardio pulmonary events associated with multiple sclerosis exacerbations. Mult Scler. 2019;25(6): 758-765.
- 137. Findling O, Hauer L, Pezawas T, Rommer PS, Struhal W, Sellner J. Cardiac Autonomic Dysfunc tion in Multiple Sclerosis: A Systematic Review of Current Knowledge and Impact of Immunotherapies. J Clin Med. 2020;9(2):335.
- 138. Racosta JM, Sposato LA, Morrow SA, Cipriano L, Kimpinski K, Kremenchutzky M. Cardio vascular autonomic dysfunction in multiple sclerosis: a meta-analysis. Mult Scler Relat Disord. 2015;4(2):104-111.
- 139. Kaplan TB, Berkowitz AL, Samuels MA. Cardiovascular Dysfunction in Multiple Sclerosis. Neu rologist. 2015;20(6):108-114.
- 140. Zanotto T, Hernandez ME, Medrano CN, Wilund KR, Sosnoff JJ. Cardiovascular Autonomic Dysfunction and Falls in People With Multiple Sclerosis: Is There a Link? An Opinion Article. Front Neurosci. 2020;14:610917-610917.
- 141. Huang M, Allen DR, Keller DM, Fadel PJ, Frohman EM, Davis SL. Impaired carotid baroreflex control of arterial blood pressure in multiple sclerosis. J Neurophysiol. 2016;116(1):81-87.
- 142. Mukerji S, Aloka F, Farooq MU, Kassab MY, Abela GS. Cardiovascular complications of the Guil lain-Barré syndrome. Am J Cardiol. 2009;104(10):1452-1455.
- 143. Bello-Haas VD. Physical therapy for individuals with amyotrophic lateral sclerosis: current in sights. Degener Neurol Neuromuscul Dis. 2018;8:45-54.
- 144. Ciccone C. Pharmacology in Rehabilitation. 5th ed. Philadelphia, PA: FA Davis; 2015.
- 145. epocrates. https://online.epocrates.com/drugs. Published 2021. Accessed December 2020.
- 146. Lexi-Drugs Monographs Online. UpToDate, Inc; 2021. Accessed March 2021.
- 147. APTA House of Delegates Position, P06-18-34-39 (2018).
- 148. Pfeiffer RF. Autonomic Dysfunction in Parkinson's Disease. Neurotherapeutics. 2020.
- 149. Association AD. Pharmacologic Approaches to Glycemic Treatment: Standards of Medical Care in Diabetes-2020. Diabetes Care. 2020;43(Suppl. 1 (Electronic)):S98-S110.

APTA Acute Care. (CARTA Academy of Cardiovascular & Pulmonary Physical Therapy

- 150. James PA, Oparil S, Carter BL, et al. 2014 Evidence-Based Guideline for the Management of High Blood Pressure in Adults: Report From the Panel Members Appointed to the Eighth Joint National Committee (JNC 8). JAMA. 2014;311(5):507-520.
- 151. Qaseem A, Wilt TJ, Rich R, Humphrey LL, Frost J, Forciea MA. Pharmacologic Treatment of Hypertension in Adults Aged 60 Years or Older to Higher Versus Lower Blood Pressure Targets: A Clinical Practice Guideline From the American College of Physicians and the American Academy of Family Physicians. Annals of Internal Medicine. 2017;166(6):430-437.
- 152. Unger T, Borghi C, Charchar F, et al. 2020 International Society of Hypertension Global Hyper tension Practice Guidelines. Hypertension. 2020;75(6):1334-1357.