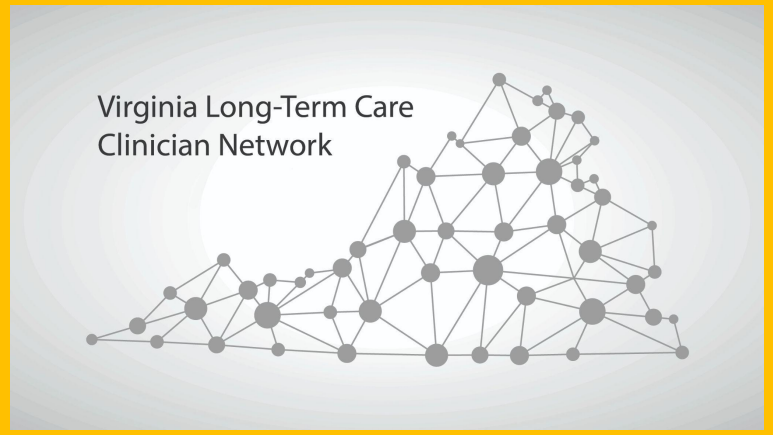
Virginia Long-Term Care Clinician Network

Monthly Forum
April 15, 2026



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Disclosure of Commercial Support:

We acknowledge that no commercial or in-kind support was provided for this activity.

The Virginia Long-Term Care Clinician Network is managed by VCU's Division of Geriatric Medicine, Virginia Center on Aging, and Department of Gerontology.

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Welcome!

As you join, please turn on cameras and mic or unmute your phone and say hello to your Virginia colleagues.

Any updates in the state or with you or your work?

Previous newsletters and other resources for you are at https://ltccn.vcu.edu/resources/

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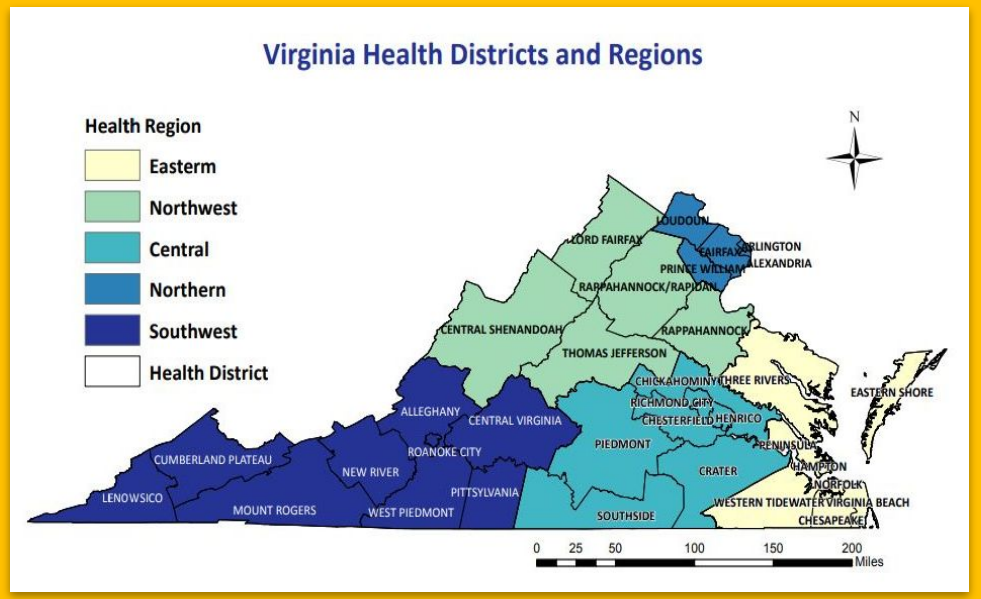
Welcome new members!

- **Michele Greep - Central Region**
- **Sarah Worz - Northern Region**



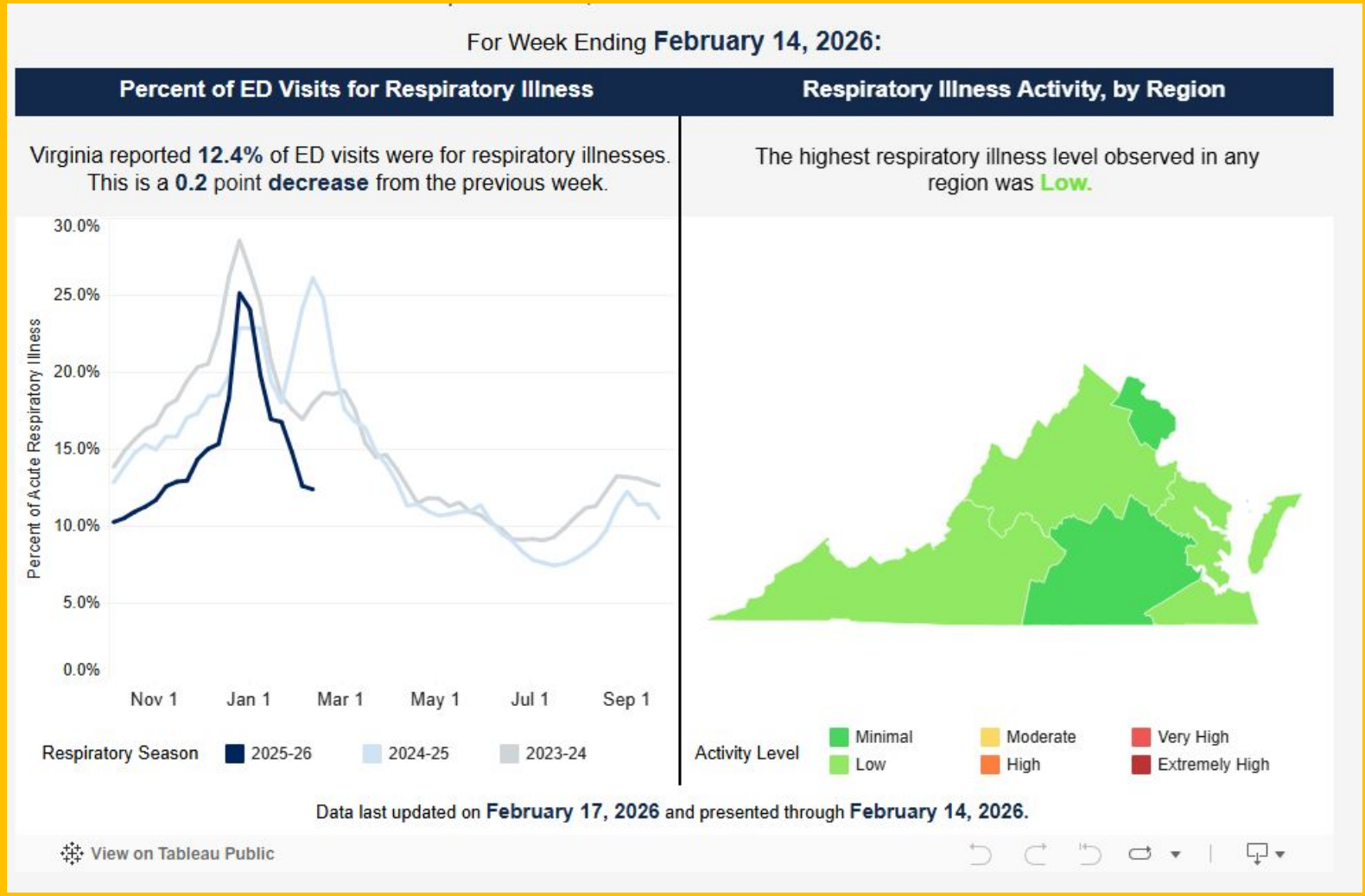
There are approximately 287 nursing homes and 580 assisted living facilities operating in Virginia. Within these, there are over 500 clinicians providing care. **We have 330 network members.** The Network provides ongoing learning and communication.

Remind your work colleagues to attend so they can get education, support and CME!

Waterfall Poll

Are you seeing?

1) COVID
2) Flu
3) RSV
4) All 3



https://www.vdh.virginia.gov/epidemiology/respiratory-diseases-in-virginia/data/#Illness-Trends

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Pulmonary Hypertension

Christian Bergman, MD, CMD

Associate Professor, Division of Geriatric Medicine, VCU

Case Presentation

61 yo M with CKD4, HFpEF (EF 65% 12/2025), severe pre-capillary pulmonary hypertension (most recent RHC 1/9/26), chronic hyponatremia, DMT2, HTN, and prior pericardial effusion requiring window via sternotomy who was initially admitted 12/28 for dry gangrene of the LEFT foot. Her hospital course has been c/b HFPEF exacerbation with diagnosis if severe pulmonary hypertension (pre-capillary) after RHC (1/9/25) and hyperkalemia. Nephrology was consulted for management of worsening renal function, hyperkalemia, and hyponatremia. She is improved and will be discahrged to SNF placement 1/21. You are now her SNF provider.

Case Presentation

1. WHO Group 1 Pulmonary Arterial Hypertension RHC 1/9-> Severe pre-capillary pulmonary hypertension (PCWP 10 mmhg, PVR 15 WU and ow cardiac output by Fick and Thermodilution. Currently undergoing testing while inpatient. Etiology possibly autoimmune related but further testing needed.
    a. -V/Q scan 1/12->The findings as above are felt to be consistent with a low probability for PE.
    b. -PFTs: Fev/Fvc:80, Fev1 50. Lungs revealed moderate restriction w/ air trapping.
    c. -Labs: ANA (neg), RF (neg), anti-centromere (neg), SCL-70 (neg), HIV (neg), kappa light changes 135/lambda light chains 165/kappa to lambda light chain ration 0.81, SPEP (Hypergammaglobulinemia and Hypoalbuminemia)
    d. -Follow up pending labs: systemic sclerosis panel (1/13), anti neutrop cytoplasmic AB (1/13)
    e. -Pulmonary consulted, Daily proBNPs to trend
    f. Sildenafil 20mg g8h (start 1/15)
    g. Ambrisentan 5mg daily (start 1/16)
    h. Discussed adding another medication in the future post discharge

Case Presentation

#HFPEF exacerbation, Initial concern for heart failure exacerbation. Underwent aggressive diuresis. RHC revealing for severe pulmonary hypertension. question as whether patient truly has heart failure or if symptoms/presentations that have been attributed to heart failure were actually related to uncontrol pulmonary hypertension

#Hold GDMT in the setting of AKI, see below: - Beta-blocker: DO NOT USE BETA BLOCKERS given pulmHTN - ACE/ARB/ARNI: hold given renal function Losartan 12.5 mg qd - SGLT2i: hold given renal function Empagliflozin 12.5 mg daily - MRA: none - CHF consult recs - PYP scan cannot be done at the VA, plan for community care - Outpatient follow-up scheduled

Case Presentation

Admission Med List

- lasix 40 daily
- ambrisentan 5 mg daily
- sildenafil 20 mg every 8 hours
- asa 81 daily
- Lipitor 40 daily
- duloxetine 30 nightly
- Synthroid 25 mcg daily

Case Presentation

Admission Labs

BMP - Na 132, K 4.9, Cl 101, CO2 18, BUN 43, Cr 2.6, glu 119, Ca 8.2

CBC - WBC 7.6, Hgb 9, PLT 319

SNF Rehab Course

- Endorsed worsening shortness of breath
- Treated as CHF exacerbation
- CXR with mild pulm vascular congestion
- beta blocker restarted, diuretics increased (Attempting to optimize CHF mgmt, diurese)
- BP stable 120-135
- AKI worsened, shortness of breath worsened
- Sent back to hospital - volume overloaded, IV diuretics in hospital; echo with severe HTN, hospital had to hold sildenafil due to hypotension.

What should we have done differently?

Pulmonary Hypertension

2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension

Developed by the task force for the diagnosis and treatment of pulmonary hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS).

Endorsed by the International Society for Heart and Lung Transplantation (ISHLT) and the European Reference Network on rare respiratory diseases (ERN-LUNG).

Table 5 Haemodynamic definitions of pulmonary hypertension

Definition	Haemodynamic characteristics
PH	mPAP >20 mmHg
Pre-capillary PH	mPAP >20 mmHg PAWP ≤15 mmHg PVR >2 WU
IpcPH	mPAP >20 mmHg PAWP >15 mmHg PVR ≤2 WU
CpcPH	mPAP >20 mmHg PAWP >15 mmHg PVR >2 WU
Exercise PH	mPAP/CO slope between rest and exercise >3 mmHg/L/min

CO, cardiac output; CpcPH, combined post- and pre-capillary pulmonary hypertension; IpcPH, isolated post-capillary pulmonary hypertension; mPAP, mean pulmonary arterial pressure; PAWP, pulmonary arterial wedge pressure; PH, pulmonary hypertension; PVR, pulmonary vascular resistance; WU, Wood units.
Some patients present with elevated mPAP (>20 mmHg) but low PVR (≤2 WU) and low PAWP (≤15 mmHg); this haemodynamic condition may be described by the term 'unclassified PH' (see text for further details).

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Table 6 Clinical classification of pulmonary hypertension

GROUP 1 Pulmonary arterial hypertension (PAH)
- 1.1 Idiopathic
  - 1.1.1 Non-responders at vasoreactivity testing
  - 1.1.2 Acute responders at vasoreactivity testing
- 1.2 Heritable[a]
- 1.3 Associated with drugs and toxins[a]
- 1.4 Associated with:
  - 1.4.1 Connective tissue disease
  - 1.4.2 HIV infection
  - 1.4.3 Portal hypertension
  - 1.4.4 Congenital heart disease
  - 1.4.5 Schistosomiasis
- 1.5 PAH with features of venous/capillary (PVOD/PCH) involvement
- 1.6 Persistent PH of the newborn

GROUP 2 PH associated with left heart disease
- 2.1 Heart failure:
  - 2.1.1 with preserved ejection fraction
  - 2.1.2 with reduced or mildly reduced ejection fraction[b]
- 2.2 Valvular heart disease
- 2.3 Congenital/acquired cardiovascular conditions leading to post-capillary PH

GROUP 3 PH associated with lung diseases and/or hypoxia
- 3.1 Obstructive lung disease or emphysema
- 3.2 Restrictive lung disease
- 3.3 Lung disease with mixed restrictive/obstructive pattern
- 3.4 Hypoventilation syndromes
- 3.5 Hypoxia without lung disease (e.g. high altitude)
- 3.6 Developmental lung disorders

GROUP 4 PH associated with pulmonary artery obstructions
- 4.1 Chronic thrombo-embolic PH
- 4.2 Other pulmonary artery obstructions[c]

GROUP 5 PH with unclear and/or multifactorial mechanisms
- 5.1 Haematological disorders[d]
- 5.2 Systemic disorders[e]
- 5.3 Metabolic disorders[f]
- 5.4 Chronic renal failure with or without haemodialysis
- 5.5 Pulmonary tumour thrombotic microangiopathy
- 5.6 Fibrosing mediastinitis

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PULMONARY HYPERTENSION

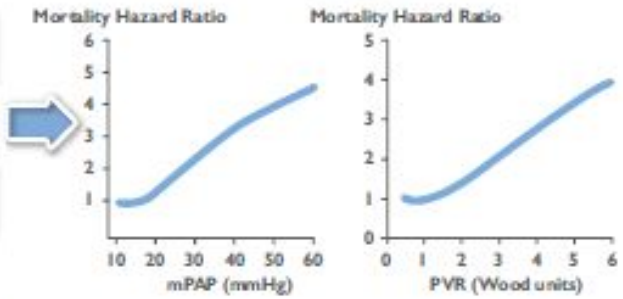
Prevalence

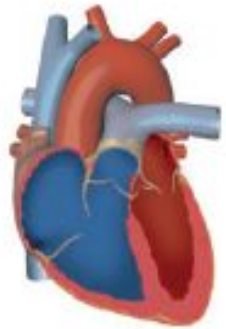
1%

Global population

Pulmonary congestion in post-capillary PH

Pulmonary vascular disease / obstruction in pre-capillary PH

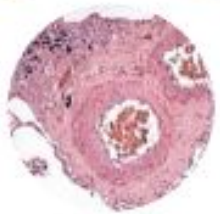




Right heart failure

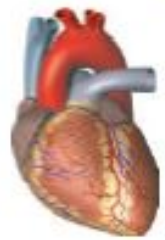
CLINICAL CLASSIFICATION

Pulmonary arterial hypertension (PAH)



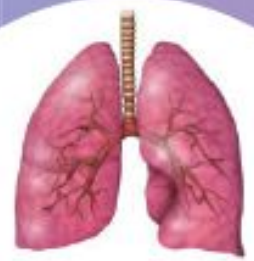
- Idiopathic/heritable
- Associated conditions

PH associated with left heart disease



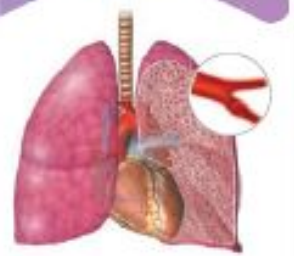
- IpcPH
- CpcPH

PH associated with lung disease



- Non-severe PH
- Severe PH

PH associated with pulmonary artery obstructions



- CTEPH
- Other pulmonary obstructions

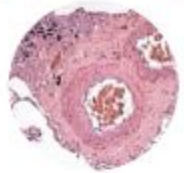
PH with unclear and/or multifactorial mechanisms

?

- Haematological disorders
- Systemic disorders

CLINICAL CLASSIFICATION

Pulmonary arterial hypertension (PAH)



- Idiopathic/heritable
- Associated conditions

PH associated with left heart disease

- IpcPH
- CpcPH

PH associated with lung disease

- Non-severe PH
- Severe PH

PH associated with pulmonary artery obstructions

- CTEPH
- Other pulmonary obstructions

PH with unclear and/or multifactorial mechanisms

- Haematological disorders
- Systemic disorders

PREVALENCE

Rare	Very common	Common	Rare	Rare
				

THERAPEUTIC STRATEGIES

Medical therapy
- PAH drugs
- CCB in responders

Lung transplantation

IpcPH:
- Treatment of LHD[a]

CpcPH:
- Treatment of LHD[a]
- Potentially: PAH drugs (trials)

PH-lung disease:
- Optimized care of underlying lung disease

Severe PH:
- Potentially: PAH drugs (trials)

Surgical therapy:
- PEA

Interventional:
- BPA

Medical therapy:
- PH drugs

Optimized treatment of underlying disease
- Potentially: PAH drugs (trials)

ESC ERS

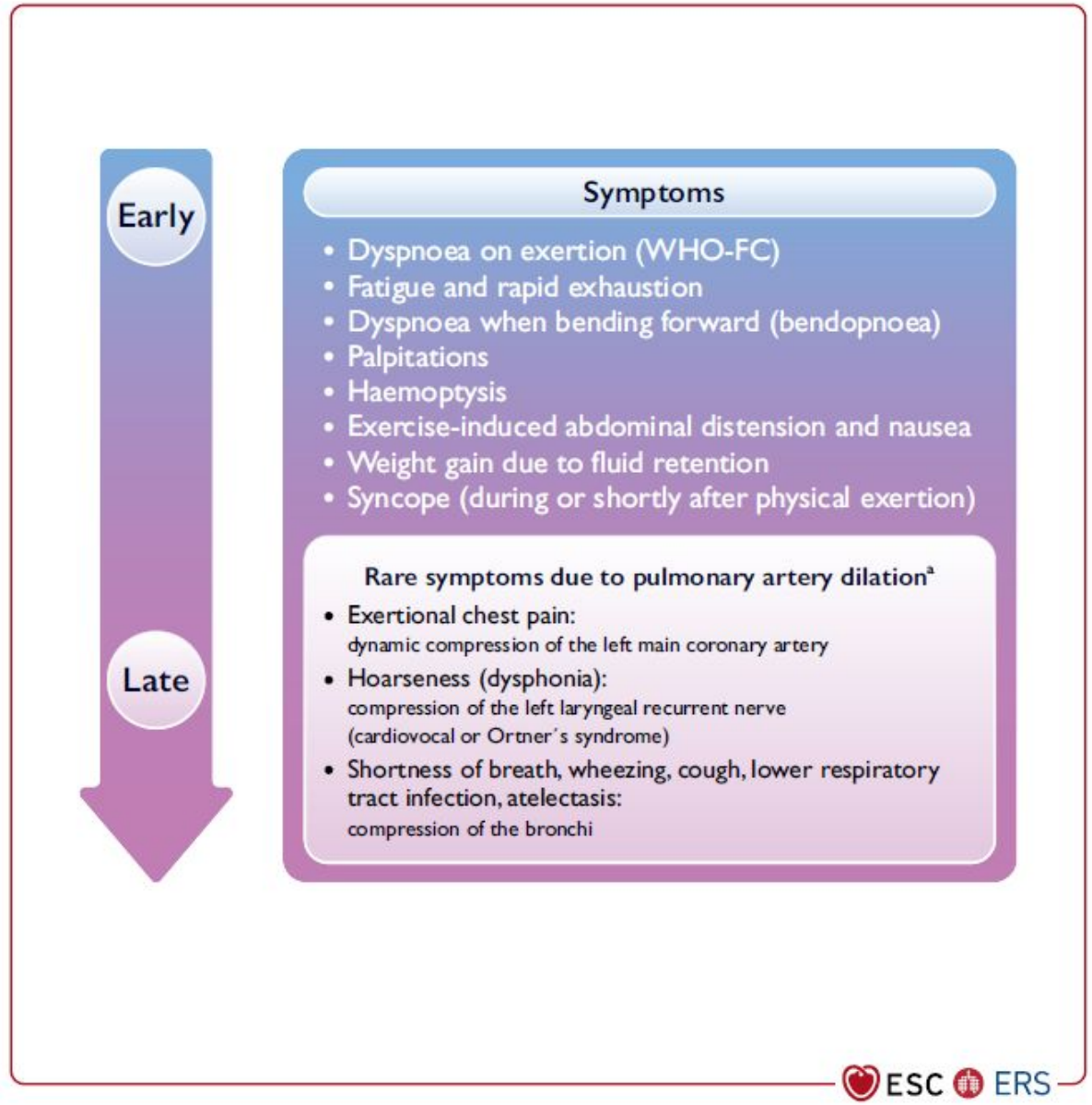


Figure 2 Symptoms in patients with pulmonary hypertension. WHO-FC, World Health Organization functional class. [a]Thoracic compression syndromes are found in a minority of patients with PAH with pronounced dilation of the pulmonary artery, and may occur at any disease stage and even in patients with otherwise mild functional impairment.

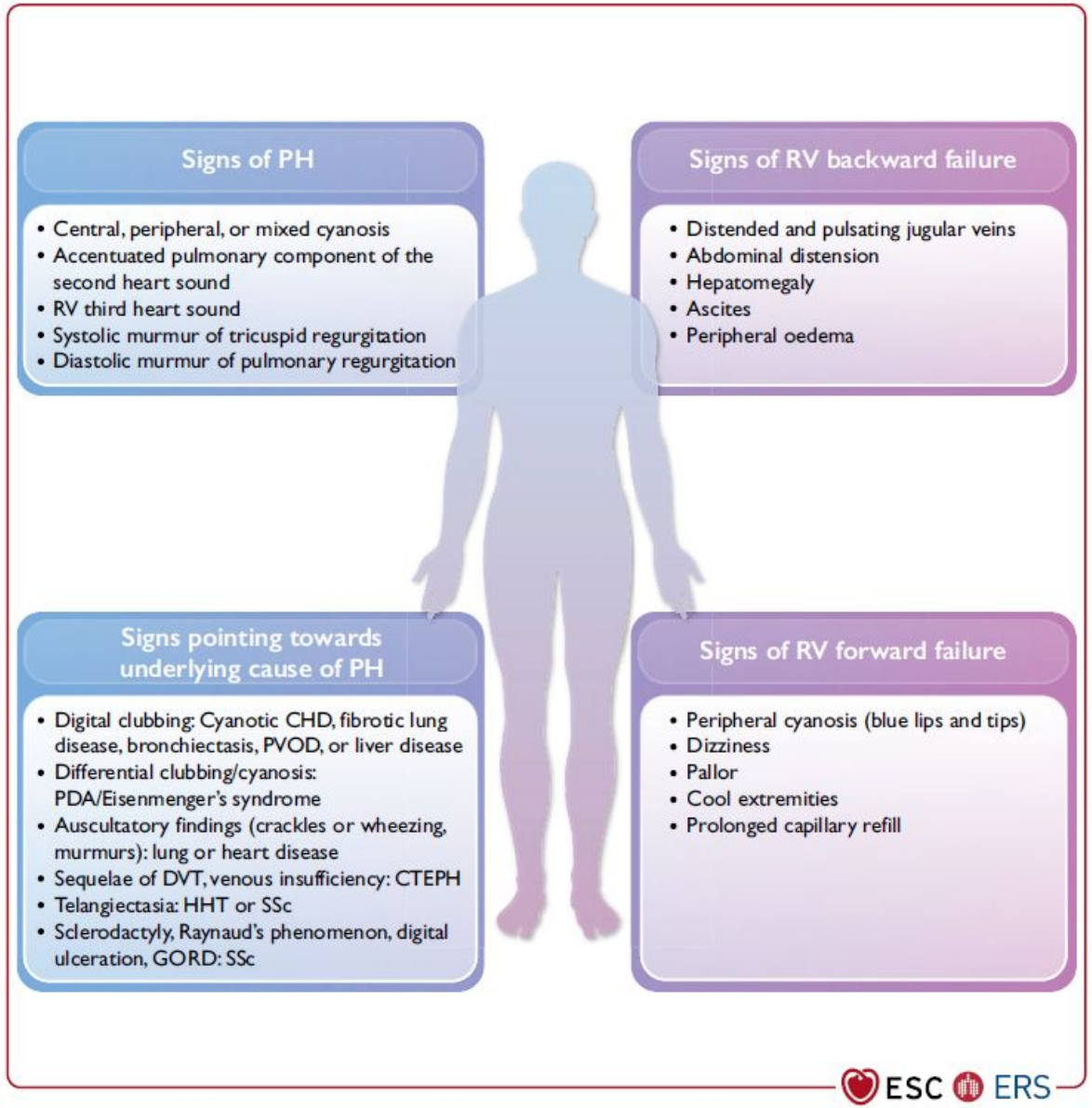
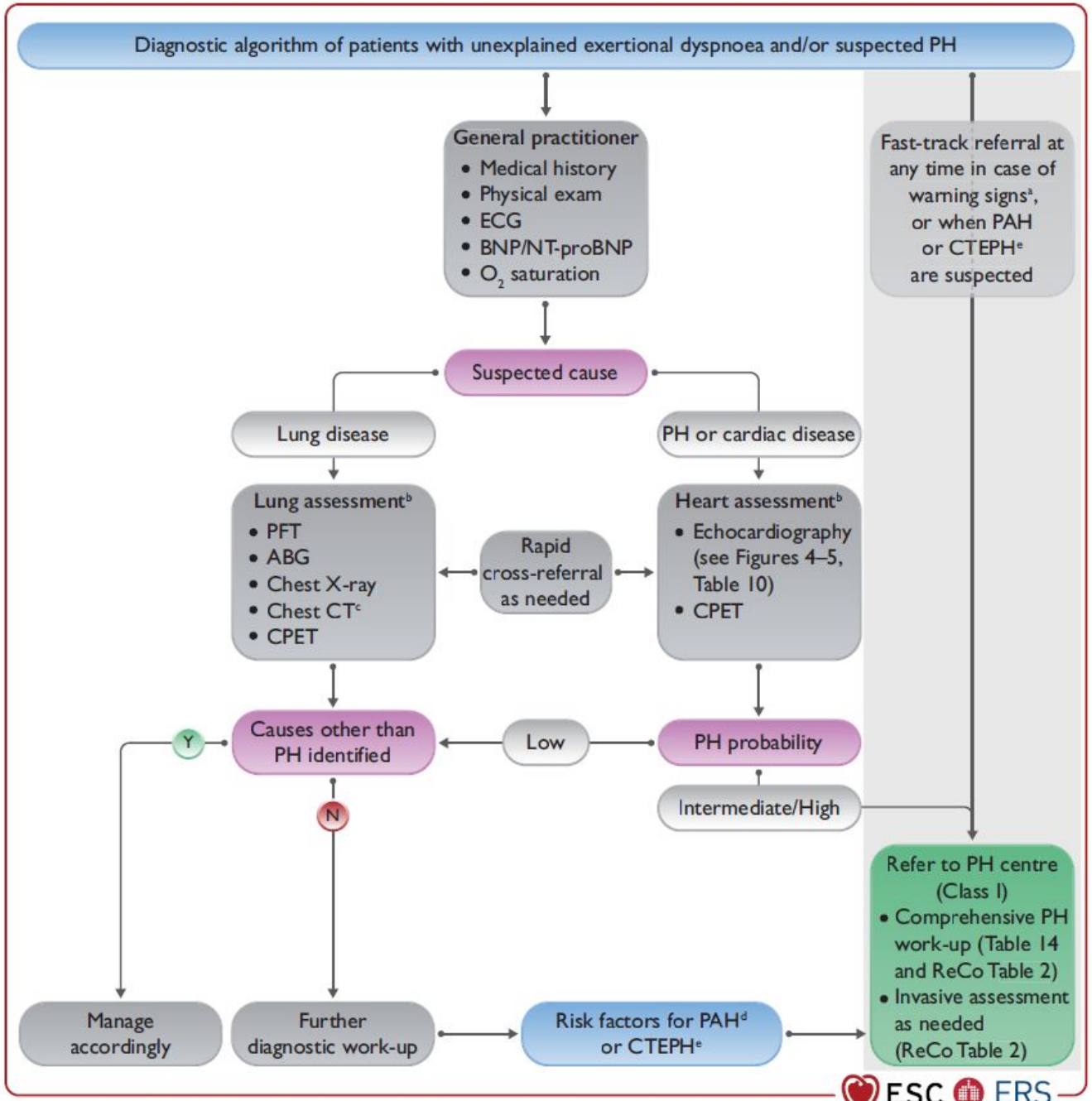


Figure 3 Clinical signs in patients with pulmonary hypertension. CHD, congenital heart disease; CTEPH, chronic thrombo-embolic pulmonary hypertension; DVT, deep venous thrombosis; GORD, gastro-oesophageal reflux disease; HHT, hereditary haemorrhagic telangiectasia; PDA, patent ductus arteriosus; PH, pulmonary hypertension; PVOD, pulmonary veno-occlusive disease; RV, right ventricle; SSc, systemic sclerosis.


Diagnostic algorithm of patients with unexplained exertional dyspnoea and/or suspected PH
General practitioner
• Medical history
• Physical exam
• ECG
• BNP/NT-proBNP
• O_2 saturation
Fast-track referral at any time in case of warning signs[a], or when PAH or CTEPH[e] are suspected
Suspected cause
Lung disease
PH or cardiac disease
Lung assessment[b]
• PFT
• ABG
• Chest X-ray
• Chest CT[c]
• CPET
Rapid cross-referral as needed
Heart assessment[b]
• Echocardiography (see Figures 4–5, Table 10)
• CPET
Y
Causes other than PH identified
Low
PH probability
N
Intermediate/High
Refer to PH centre (Class I)
• Comprehensive PH work-up (Table 14 and ReCo Table 2)
• Invasive assessment as needed (ReCo Table 2)
Manage accordingly
Further diagnostic work-up
Risk factors for PAH[d] or CTEPH[e]
ESC ERS

WHO Classification vs. Clinical Classification 5 Groups

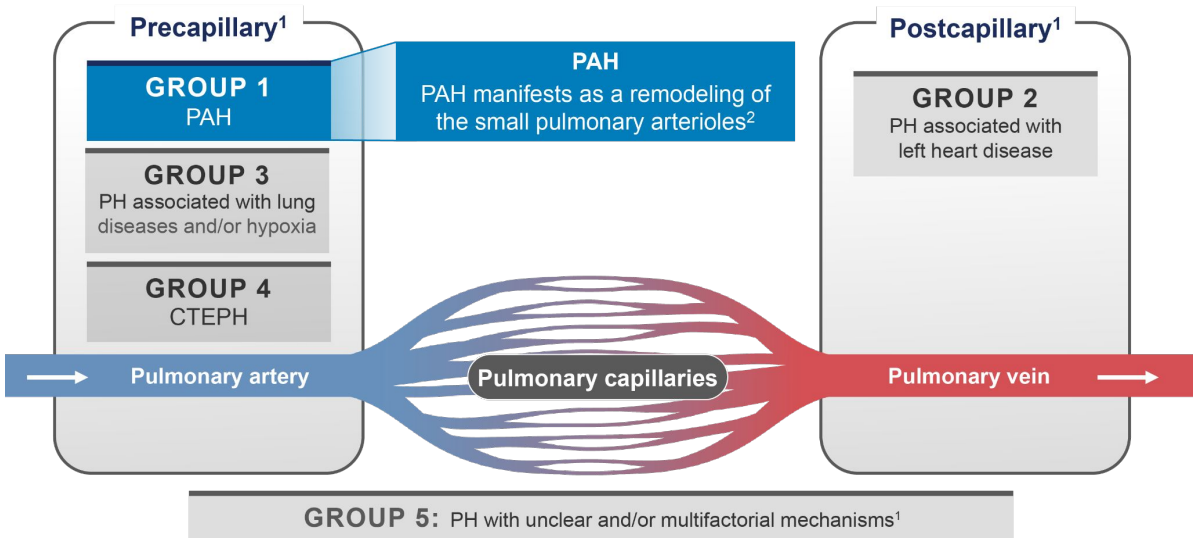


Table 15 World Health Organization classification of functional status of patients with pulmonary hypertension

Class	Description[a]
WHO-FC I	Patients with PH but without resulting limitation of physical activity. Ordinary physical activity does not cause undue dyspnoea or fatigue, chest pain, or near syncope
WHO-FC II	Patients with PH resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity causes undue dyspnoea or fatigue, chest pain, or near syncope
WHO-FC III	Patients with PH resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary activity causes undue dyspnoea or fatigue, chest pain, or near syncope
WHO-FC IV	Patients with PH with an inability to carry out any physical activity without symptoms. These patients manifest signs of right HF. Dyspnoea and/or fatigue may even be present at rest. Discomfort is increased by any physical activity

PH, pulmonary hypertension; WHO-FC, World Health Organization functional class.
[a]Functional classification of PH modified after the New York Heart Association functional classification according to the World Health Organization 1998.[147]

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Table 16 Comprehensive risk assessment in pulmonary arterial hypertension (three-strata model)

Determinants of prognosis (estimated 1-year mortality)	Low risk (<5%)	Intermediate risk (5–20%)	High risk (>20%)
Clinical observations and modifiable variables			
Signs of right HF	Absent	Absent	Present
Progression of symptoms and clinical manifestations	No	Slow	Rapid
Syncope	No	Occasional syncope[a]	Repeated syncope[b]
WHO-FC	I, II	III	IV
6MWD[c]	>440 m	165–440 m	<165 m
CPET	Peak VO_2 >15 mL/min/kg (>65% pred.) VE/VCO_2 slope <36	Peak VO_2 11–15 mL/min/kg (35–65% pred.) VE/VCO_2 slope 36–44	Peak VO_2 <11 mL/min/kg (<35% pred.) VE/VCO_2 slope >44
Biomarkers: BNP or NT-proBNP[d]	BNP <50 ng/L NT-proBNP <300 ng/L	BNP 50–800 ng/L NT-proBNP 300–1100 ng/L	BNP >800 ng/L NT-proBNP >1100 ng/L
Echocardiography	RA area <18 cm^2 TAPSE/sPAP >0.32 mm/mmHg No pericardial effusion	RA area 18–26 cm^2 TAPSE/sPAP 0.19–0.32 mm/mmHg Minimal pericardial effusion	RA area >26 cm^2 TAPSE/sPAP <0.19 mm/mmHg Moderate or large pericardial effusion
cMRI[e]	RVEF >54% SVI >40 mL/m^2 RVESVI <42 mL/m^2	RVEF 37–54% SVI 26–40 mL/m^2 RVESVI 42–54 mL/m^2	RVEF <37% SVI <26 mL/m^2 RVESVI >54 mL/m^2
Haemodynamics	RAP <8 mmHg CI ≥2.5 $L/min/m^2$ SVI >38 mL/m^2 SvO_2 >65%	RAP 8–14 mmHg CI 2.0–2.4 $L/min/m^2$ SVI 31–38 mL/m^2 SvO_2 60–65%	RAP >14 mmHg CI <2.0 $L/min/m^2$ SVI <31 mL/m^2 SvO_2 <60%

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Management

6.3.1.5. Cardiovascular drugs

No data from rigorous clinical trials are available on the usefulness and safety of drugs that are effective in systemic hypertension or left-sided HF, such as angiotensin-converting enzyme inhibitors, angiotensin receptor blockers (ARBs), angiotensin receptor–neprilysin inhibitors (ARNIs), sodium–glucose cotransporter-2 inhibitors (SGLT-2is), beta-blockers, or ivabradine in patients with PAH. In this group of patients, these drugs may lead to potentially dangerous drops in blood pressure, heart rate, or both. Likewise, the efficacy of digoxin/digitoxin has not been documented in PAH, although these drugs may be administered to slow ventricular rate in patients with PAH who develop atrial tachyarrhythmias.

Back to our case - What would you have done?

SNF Rehab Course

- Endorsed worsening shortness of breath
- Treated as CHF exacerbation
- CXR with mild pulm vascular congestion
- beta blocker restarted, diuretics increased (Attempting to optimize CHF mgmt, diurese)
- BP stable 120-135
- AKI worsened, shortness of breath worsened
- Sent back to hospital - volume overloaded, IV diuretics in hospital; echo with severe HTN, hospital had to hold sildenafil due to hypotension.

Thoughts?

A Value Based Purchasing webinar training series for nursing home staff

In partnership with the Virginia Department of Medical Assistance Services (DMAS) and its Nursing Facility Quality Improvement Program, VCU Gerontology is launching a 6-part webinar series, Pathways to Quality: Advancing Nursing Home Care for Better Outcomes, dedicated to elevating the quality of care for nursing home residents.

Next Up: Pressure Ulcers: Causes, Risk Factors and Care Paths

Landing Page: https://tictoolkit.vcu.edu/learning-center/pathways/
Registration: https://form.jotform.com/260075945620154

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Open Forum

Any questions or ideas from the talk?

Todays CE Code is

40435

Text this code to 804-625-4041

Thank you for joining us!

Updates and News - See News Updates via email and newsletter

Next Monthly Forum:

- **May 20 - Anticoagulation in PALTC - Dr. Christensen**

Your Calendar Link - In the Zoom Registration Confirmation email you received today, there's a calendar link to update your calendar for future meetings.

On your way out of our meeting today, kindly answer a brief feedback survey.

Invite your colleagues! They can register at ltccn.vcu.edu