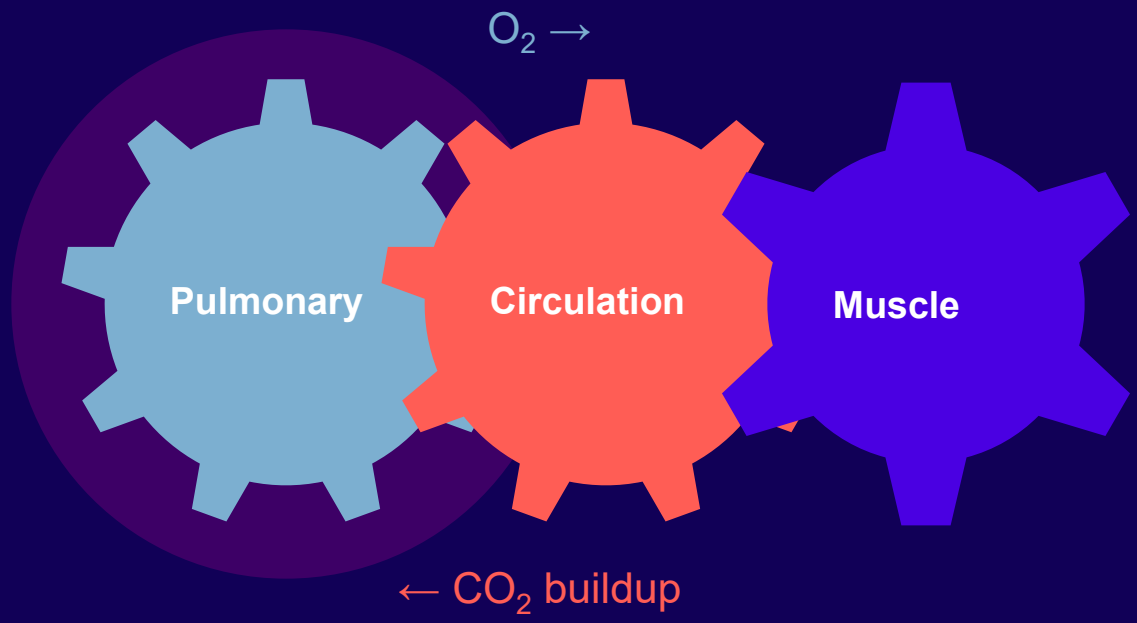
UTAH ACP 2026

March 12, 2026

Obesity-Related Respiratory Failure



adapted from Wasserman et al.

What it is, who has it, & why the discharge is so hard

Brian W Locke MD MSCI

Pulmonary & Critical Care Medicine, Intermountain Medical Center

COI: Mountain Biometrics (automated data harmonization) — unrelated to this work

Funding:

- ATS ASPIRE Fellowship
- NIH NHLBI T32 — University of Utah PCCM
- Intermountain PCCM Seed Grant: FUND-Hypercapnia
- Utah Population Database via HCI & HCI Cancer Center Support Grant P30 CA2014

Learning Objectives

01	Define obesity hypoventilation syndrome and explain why "obesity-related respiratory failure" may be a more useful clinical framework
02	Identify patients at risk for obesity-related respiratory failure — including common misdiagnoses
03	Explain why discharging patients on BPAP is so difficult — including the historical and regulatory origins of device categories, qualification pathways, and payment structure
04	Discuss the emerging role of GLP-1 receptor agonists in obesity-related respiratory failure

Obesity Hypoventilation Syndrome (OHS)

Textbook definition requires ALL of: (know this for exams)

- Obesity (BMI ≥ 30 kg/m²)
- Daytime hypercapnia ($PaCO_2$ ≥ 45 mmHg at sea level)
- Sleep-disordered breathing (OSA or sleep hypoventilation)
- Exclusion of other causes of hypoventilation

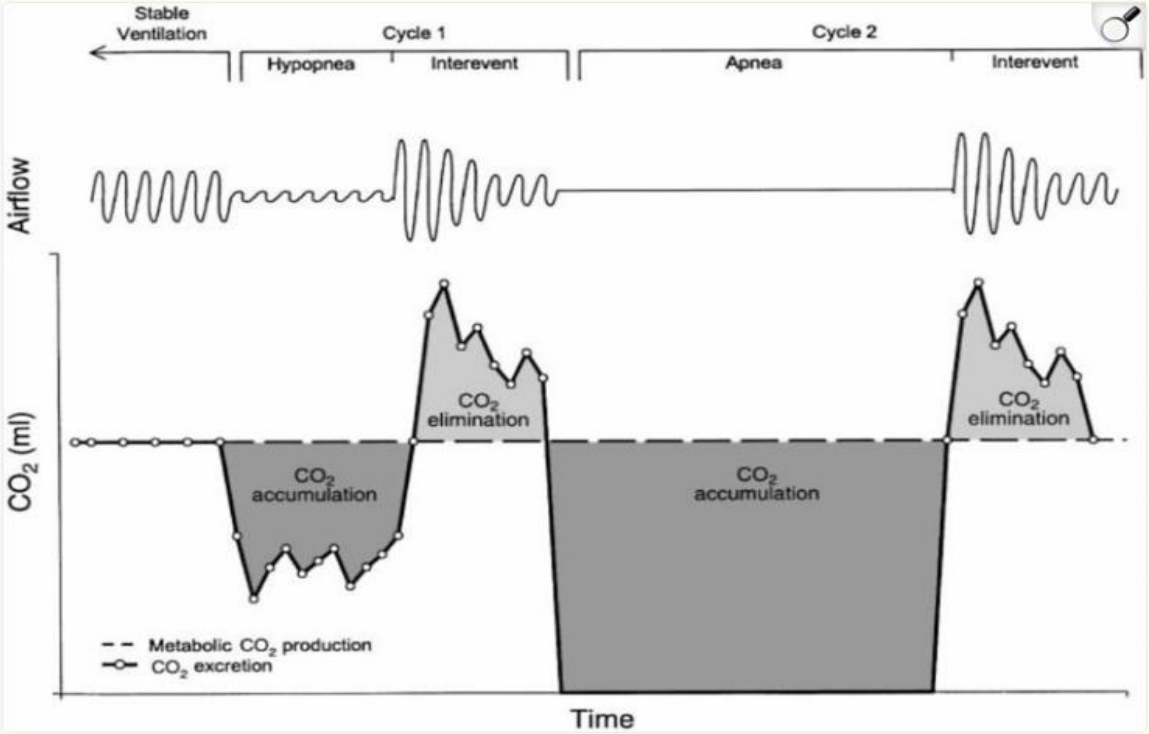
Those last two criteria are the problem in practice...

Requirement of Sleep Disordered Breathing?

- Either:
  - OSA (AHI over 5 /hr and symptoms, or 15/hr)
  - Sleep Hypoventilation (10 cmH_2O rise of nocturnal CO_2 over 55 mmHg)

Does it matter? No

If hypothetically, someone w/ BMI of 40 kg/m2 has an AHI of 4 and nocturnal CO_2 of 52 and daytime of 47… would they not have OHS?



How OSA Contributes to Hypercapnia
Berger et al, J Appl Physiol 88:257-264 (2000)

Exclusion of Other Causes Is Problematic

Person at Timepoint A:

BMI 40, AHI 15, ABG 7.45/50 (pCO_2)

Person at Timepoint B:

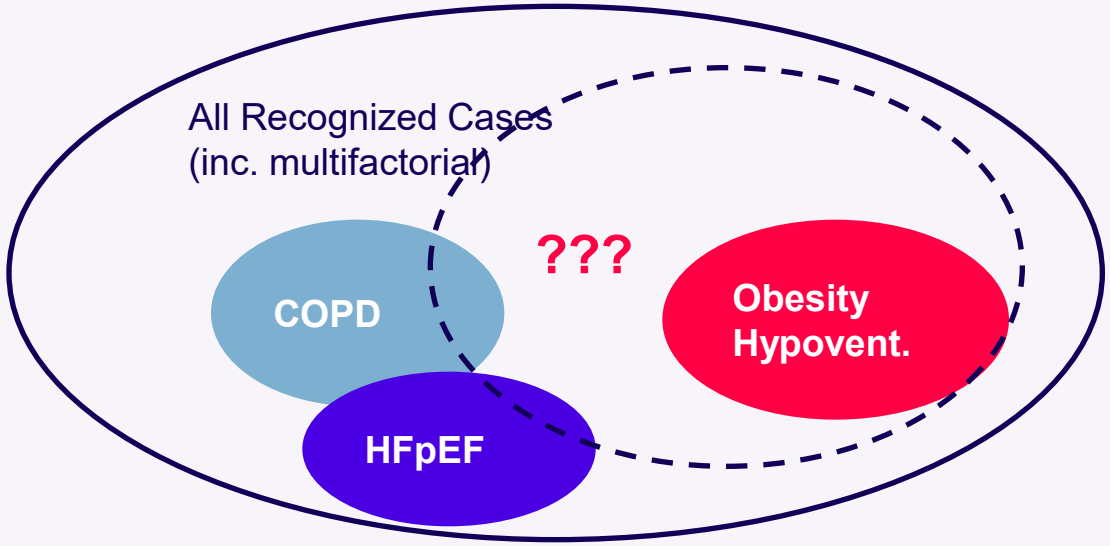
Same values + H_2FPEF score suggesting HFpEF

Did we just cure their OHS?

Obviously not.

Operationalize this as "Is obesity having a substantial causal contribution to this patient's hypercapnia?", not the exclusion of other causes.

Multi-Causal Model



Disease contributions overlap & vary



Key: Obesity is a causal contributor — not defined by exclusion of other causes

A more useful mental model:

"Obesity-Related Respiratory Failure"

Obesity is a primary driver of the respiratory failure, regardless of comorbidities

Better management implications

MOC Question

In a 65-year-old patient with **normally functioning lungs** (normal A–a gradient of 10 mmHg) breathing **room air** (FiO_2 0.21) at **Salt Lake City elevation** (~4,000 ft; P_b 656 mmHg), using the simplified alveolar gas equation (PH_2O 47 mmHg, RQ 0.8):

To what approximate $PaCO_2$ would it need to rise before SpO_2 drops below 90% (PaO_2 ~60 mmHg)?

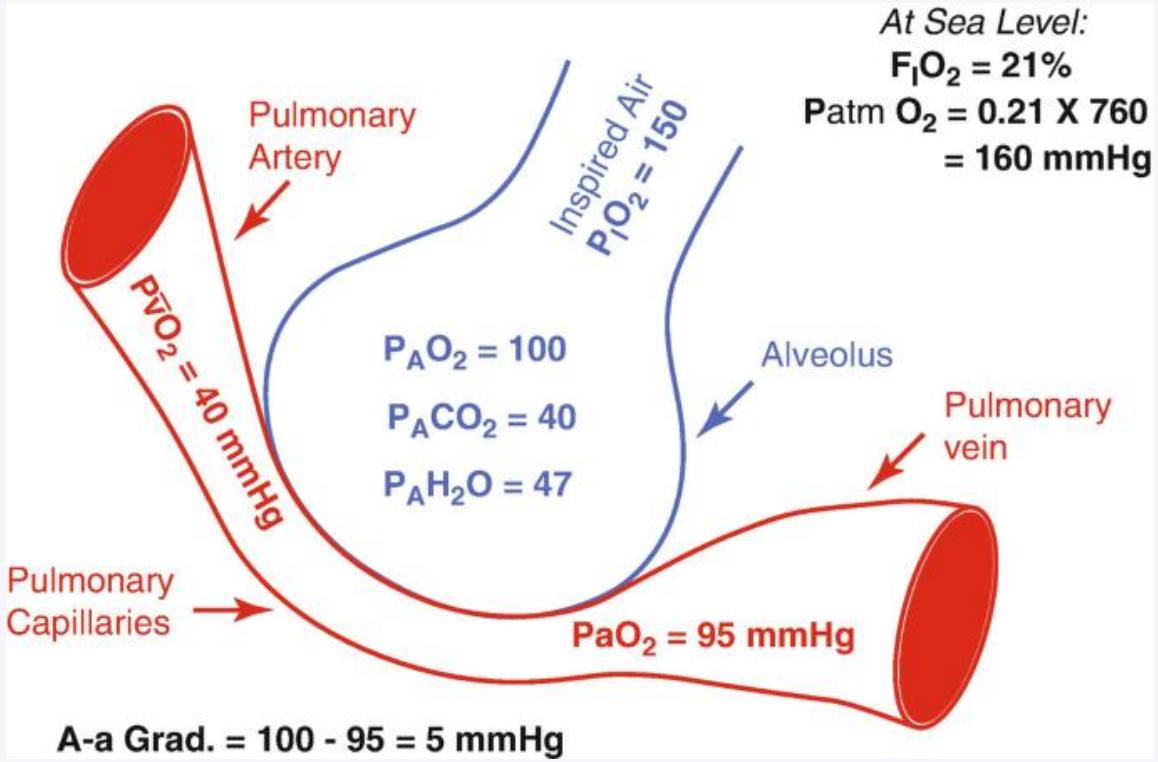
A. 45 mmHg

B. 55 mmHg

C. 65 mmHg

D. 75 mmHg

E. 85 mmHg



Recall: $P_AO_2 = FiO_2 \times (P_b - PH_2O) - (PaCO_2 / RQ)$ O_2 is **17.8 mmHg lower** at 4,500 ft

Hypercapnia in SLC

✓ **Correct: A. ~45 mmHg**

1. **$SpO_2 \rightarrow PaO_2$** SpO_2 ~90% ≈ PaO_2 ~60 mmHg
2. **PiO_2 at altitude** $PiO_2 = 0.21 \times (656 - 47) =$ **128 mmHg**
3. **Alveolar gas eq.** $P_AO_2 = 128 - 1.25 \times PaCO_2$
   Target: $PaO_2 = P_AO_2 - 10 = 60 \rightarrow$ **$P_AO_2 = 70$ mmHg**
4. **Solve for $PaCO_2$** $70 = 128 - (PaCO_2 / 0.8)$
   $PaCO_2 / 0.8 = 58 \rightarrow$ **$PaCO_2 \approx 46$ mmHg**

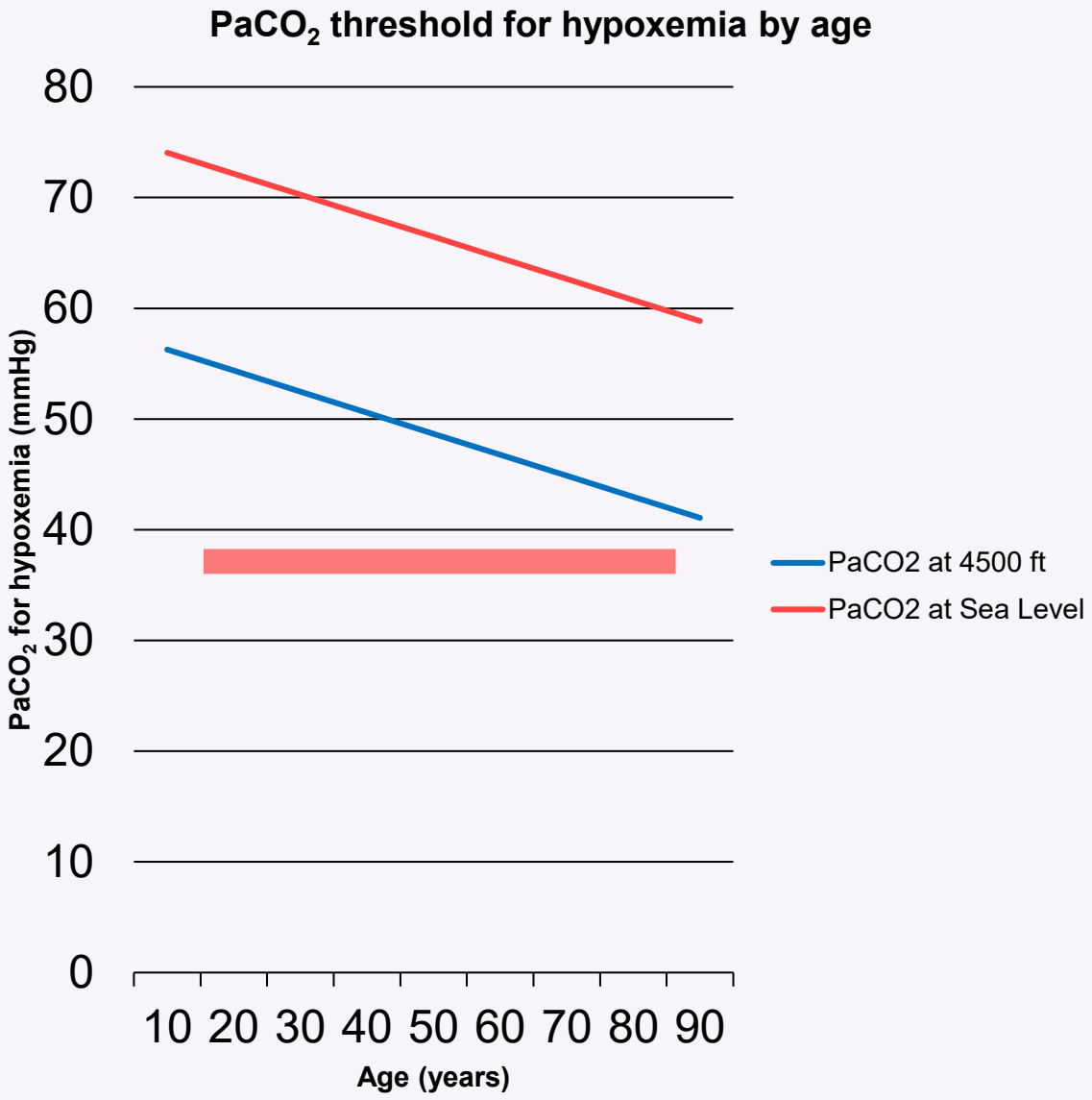
Clinical takeaway: At our elevation, even **mild elevations** in $PaCO_2$ lead to hypoxemia — regardless of additional lung pathology.
Normal $PaCO_2$ of 40 mmHg sits just 6 mmHg below the 46 mmHg threshold — minimal safety margin at altitude.

SpO_2 is routinely obtained; $PaCO_2$ (or $TcCO_2$) is not → SLC enables earlier identification

Ref: Hendrix JM, Burns B. Alveolar Gas Equation. StatPearls. 2024.

$P_AO_2 = FiO_2 (P_B - 47) - (PaCO_2/R)$ O_2 is **17.8 mmHg lower** at 4,500 ft

💡 **Why ~40 mmHg?** pH can be maintained at any $PaCO_2$ with enough HCO_3^-, so why not tolerate higher CO_2?
Likely trade-off: less O_2 buffer vs. work of breathing — still poorly understood.



What Is Hypercapnic Respiratory Failure?

Hypercapnia = $PaCO_2$ above expected range

- ≥ 45 mmHg at sea level
- ≥ 41–42 mmHg in Salt Lake City (elevation matters! Coders don't know this though)

Respiratory failure = failed homeostasis, not just a lab value

- Not explained by metabolic alkalosis (that's respiratory *compensation*, not failure)

Key clinical point:

At our elevation, hypercapnia often presents as **hypoxemia even with a normal A-a gradient**

Someone severely hypercapnic will not be on room air at our elevation

Hypercapnic Respiratory Failure is **Common**

American Journal of Respiratory and Critical Care Medicine

Home > American Journal of Respiratory and Critical Care Medicine > List of Issues > Volume 205, Issue 8

Population Prevalence of Hypercapnic Respiratory Failure from Any Cause

Yewon Chung [1,2,3]*, Frances L. Garden [1,3], Guy B. Marks [1,2,3], and Hima Vedam [1,2,3]
- Author Affiliations

[1] University of New South Wales
Sydney, New South Wales, Australia

Population-standardized period prevalence of $PaCO_2 \geq 45$ mmHg (excluding iatrogenic causes):

150* per 100,000 person/year

- Reference: Decompensated Cirrhosis 94.9 (US, 2017) per 100,000 person-year



Hypercapnic Respiratory Failure is **Prognostically Important**

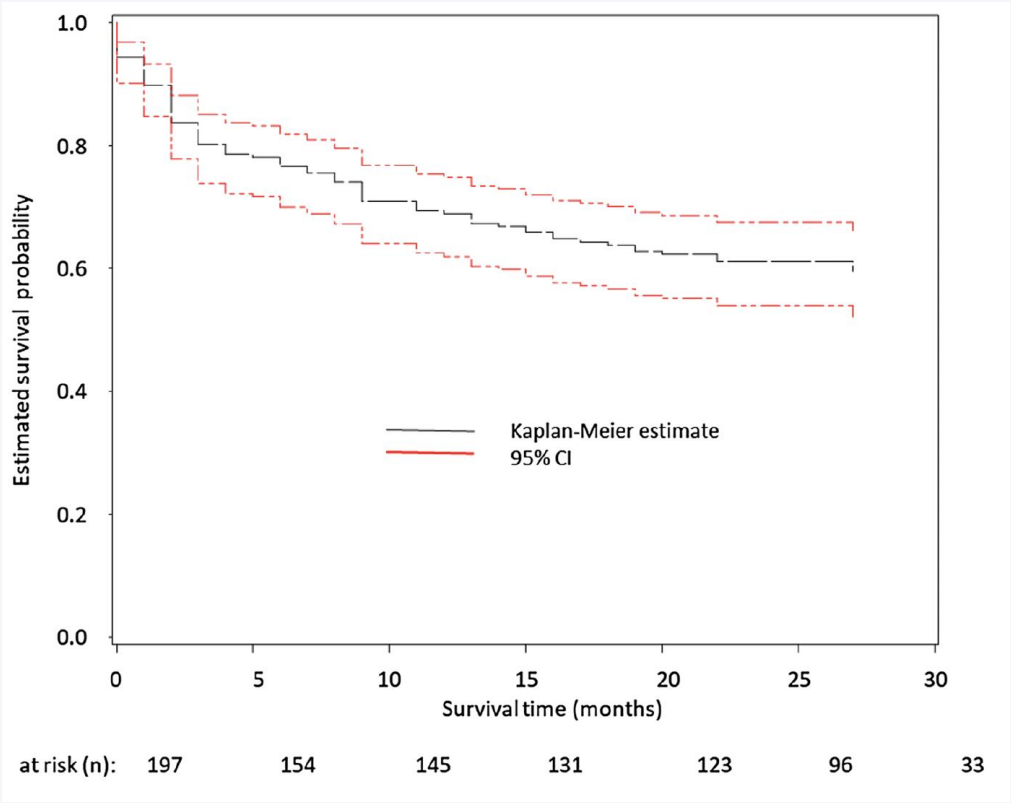
Lung (2020) 198:121–134
https://doi.org/10.1007/s00408-019-00300-w

COPD

Check for updates

Risk of Readmission and Mortality Following Hospitalization with Hypercapnic Respiratory Failure

Amber J. Meservey[1] · Michael C. Burton[1] · Jeffrey Priest[1] · Charlotte C. Teneback[1] · Anne E. Dixon[1,2]



Inpatient ICD code for Hypercapnic Respiratory Failure @ U of VT

- **23% 30d readmission rate**
  - Same as CHF, more than MI
  - Usually (66%) with recurrent hypercapnia

DIAGNOSIS

Don’t **Miss** It,
Don’t **Misattribute** It

1. Hypercapnia hides behind hypoxemia — especially at our elevation
2. “COPD” and “asthma” labels are often wrong — verify before you trust

How to Spot It: Clinical Screening Pearls

Red flags that should trigger workup

- Unexplained hypoxemia in an obese patient
- Elevated serum bicarbonate (HCO_3), ? compensation for chronic CO_2 retention
- Recurrent “COPD/Asthma/HF exacerbations” without a credible diagnostic support

Clinical approach

- Obtain a ABG or VBG confirm hypercapnia (*we’ll talk device qualification later)
- Skeptically evaluate for “explanatory” causes (true COPD, asthma, CHF)
- Many patients have **multiple contributing conditions:** obesity is often one of them

Using HCO_3^- and K^+ to Identify Hypercapnia

Interval Likelihood Ratios

HCO_3^- alone (any K^+):

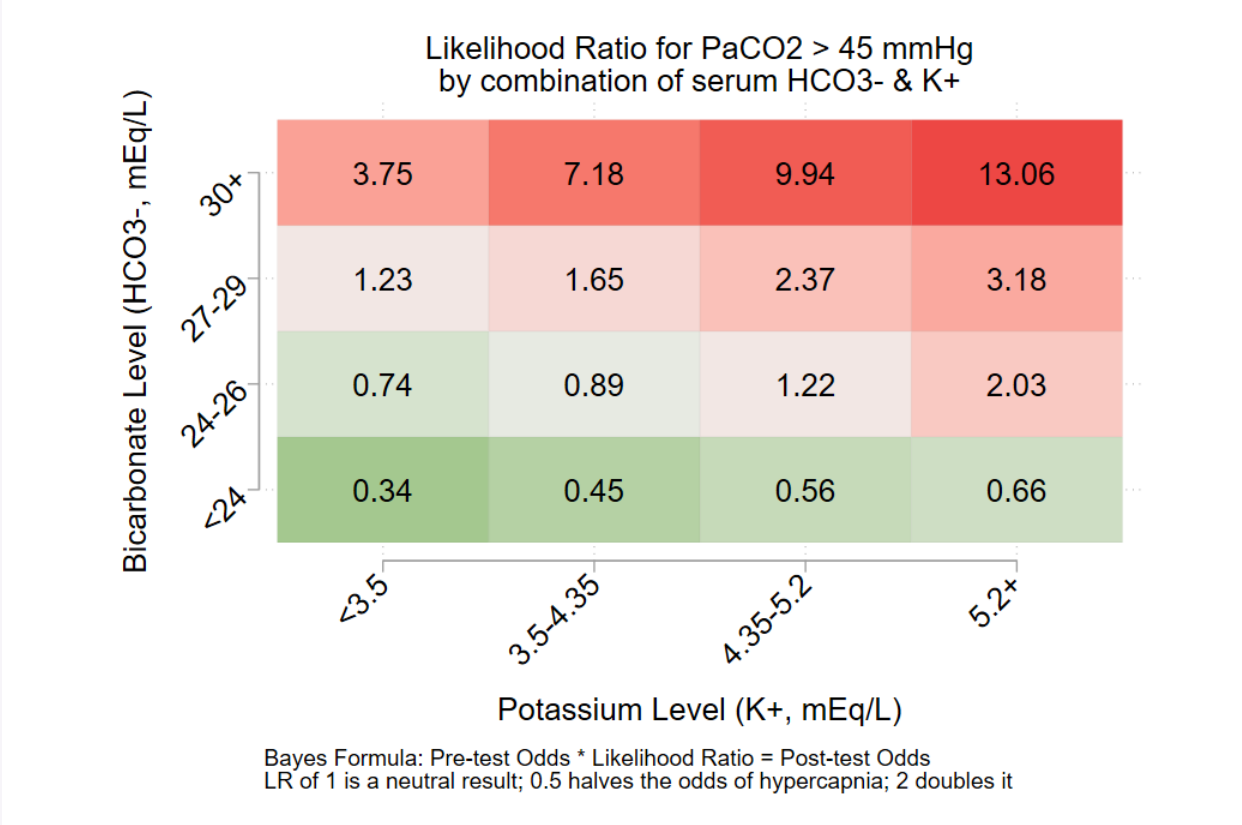
- <24: LR ~0.5 (halves odds)
- 24–26: LR ~1 (neutral)
- 27–29: LR ~2 (doubles odds)
- ≥30: LR ~9 (strong evidence)

K^+ shifts LR by 3–3.5×

- HypoK → less likely hypercapnia
- HyperK → more likely hypercapnia

Locke et al. N=870,019 in nationally representative EHR data (TriNetX, 75 U.S. health systems, 2022); 203,399 with paired ABGs & BMPs; 32% hypercapnic.

Figure 3: Joint Interval Likelihood Ratios



It's Often Not Just Hypercapnia

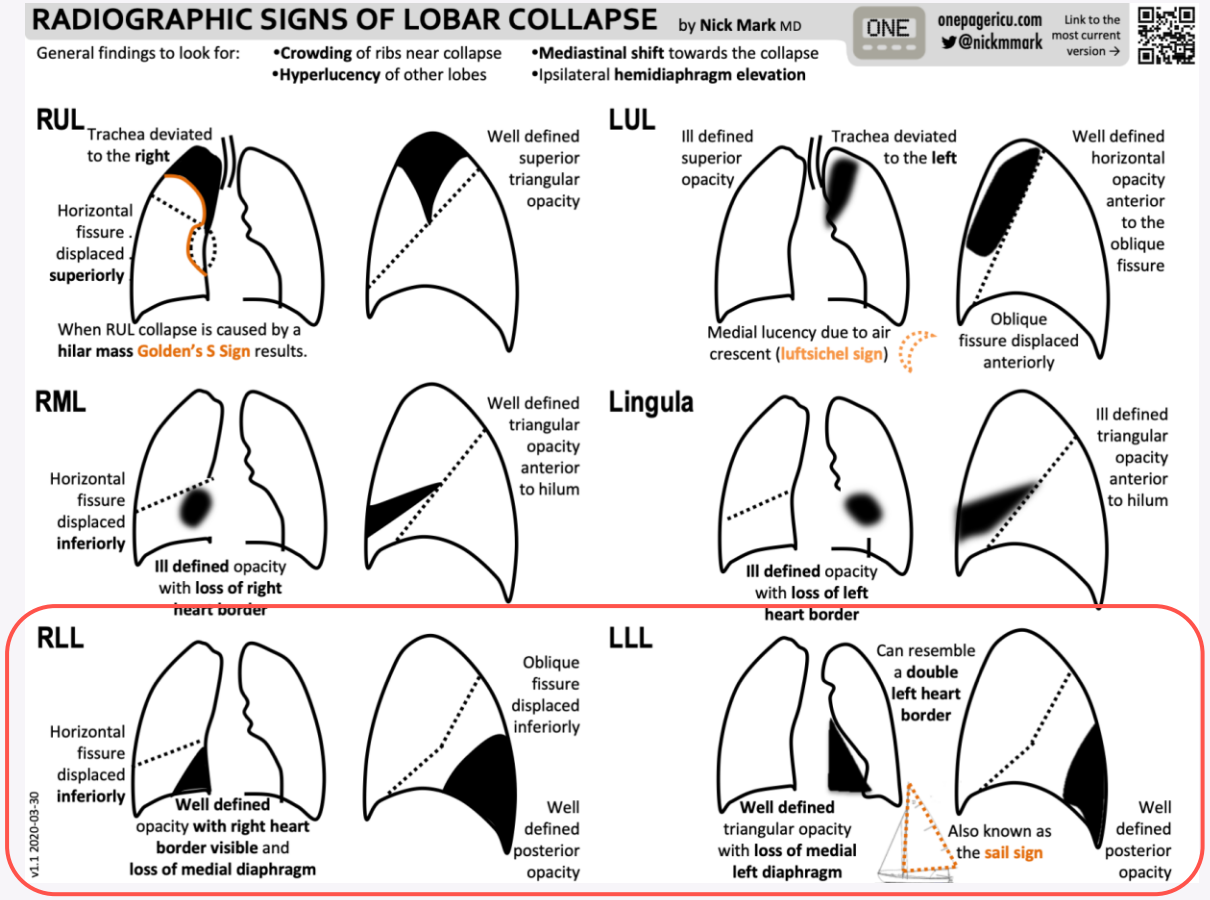
OHS itself causes:

- Hypercapnia
- Subsequent mild, correctable hypoxemia

But the obesity also causes:

- Atelectasis (esp. LLL & RLL)
- Observable on CXR (see patterns →)
- Shunts blood → **much more refractory hypoxemia**

Abdominal adiposity compresses the lower lobes — looks almost normal on CXR but the shunt fraction can be significant



Don’t **Misattribute** Hypercapnia

Missed diagnoses of “COPD” and “asthma” are common.
Most pre-existing diagnoses are inaccurate — only trust what you can **verify**.

✓ Goal 1: Don’t miss hypercapnia

Misdiagnosis Pitfalls in Obese Patients

"Asthma" in the obese patient

- Dyspnea + wheeze + obesity ≠ asthma; β-agonists / steroids prescribed empirically
  - Tricky — obesity IS a real risk factor for asthma, and asthma IS a hard diagnosis
- Do symptoms vary day-to-day? Triggered by exposures? Substantially resolved with ICS?

"COPD" in the obese patient

- If no PFTs, do they have a reason for COPD? (smoking?)
- Do they have emphysema?

Disjunctive Syllogism (Process of Elimination)

"When you have eliminated the impossible, whatever remains, however improbable, must be the truth."

— Sherlock Holmes, *The Sign of the Four*

In practice:

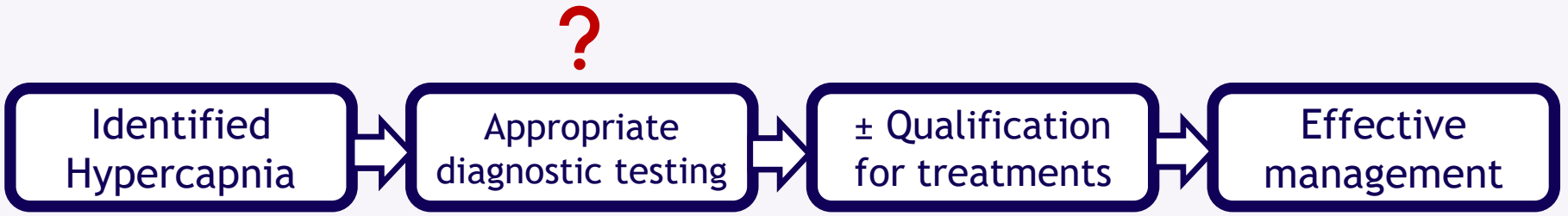
If hypercapnia is confirmed, something caused it. If workup fails to support COPD, asthma, CHF etc… the remaining suspect (OHS) becomes the working diagnosis.

Low diagnostic testing rates

Testing for most common causes of hypercapnia (e.g. spirometry, sleep testing) occurs **after hospitalization**.

Diagnostics require special resources, referrals, and high patient engagement; **barriers may reduce workup completion rates**.

Workup should, presumably, include tests for the commonest causes.

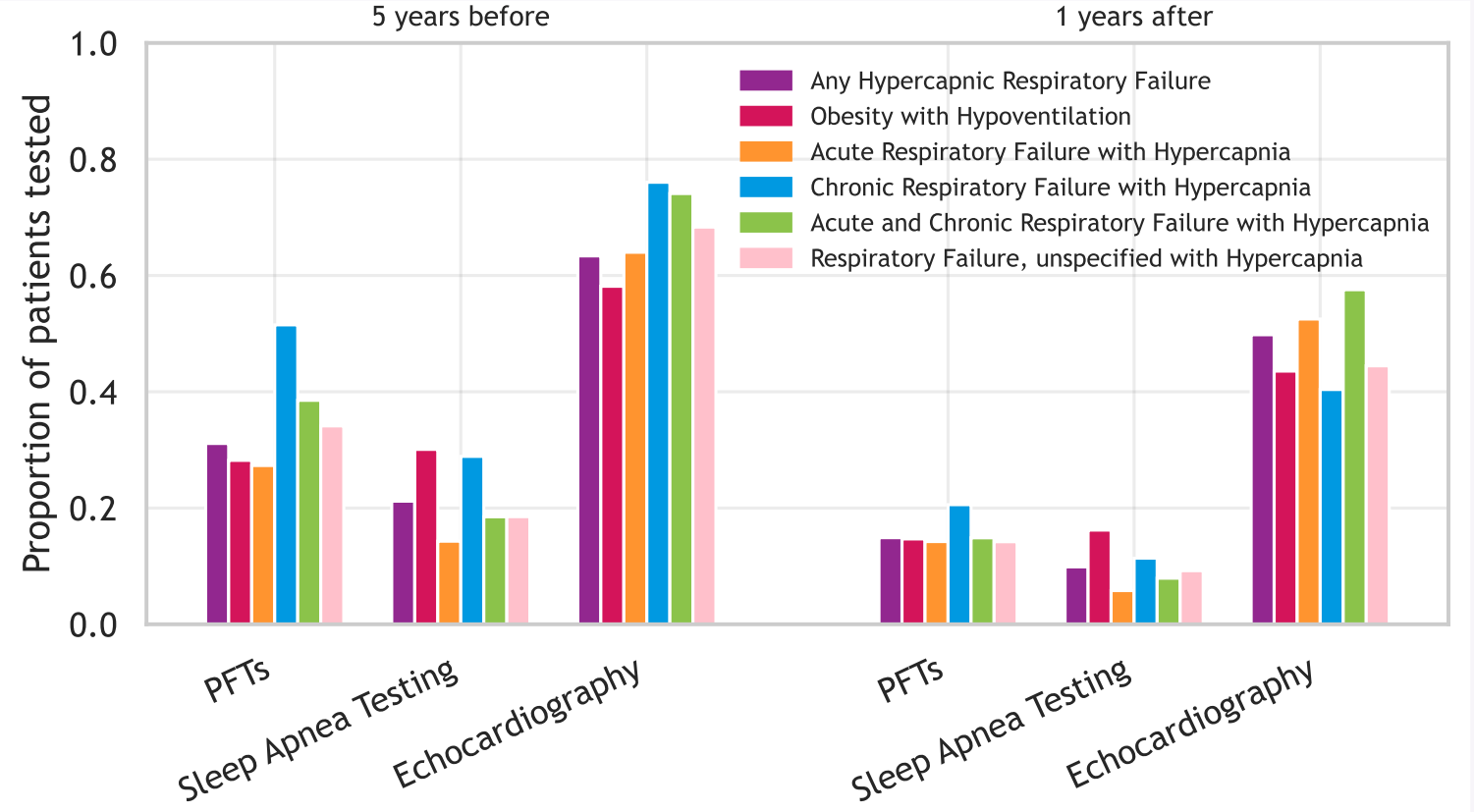


How Often Does Testing Actually Happen?

CO_2-PATH (Post-Acute Trajectories of Hypercapnia) Cohort

Utah All-Payer Claims Database: all hospitalizations statewide, 2015–2019

Identified patients with hypercapnic respiratory failure by ICD-10-CM codes



- ✓ Don’t **miss** hypercapnia
  At SLC elevation, even mild $PaCO_2$ elevation → hypoxemia. Atelectasis adds shunting on top.
- ✓ Don’t **misattribute** hypercapnia
  “COPD” and “asthma” labels in obese patients are often wrong. Skeptically evaluate, not empiric treatment.

Next: What can we do about it?

Why is the discharge management so hard?

Therapeutic options exist:

Evaluation and Management of Obesity Hypoventilation Syndrome
An Official American Thoracic Society Clinical Practice Guideline

Question 2: Should adults with OHS be treated with PAP—either CPAP or NIV—or not be treated with PAP?

Recommendation 2: For stable ambulatory patients diagnosed with OHS, we suggest treatment with PAP during sleep (*conditional recommendation, very low level of certainty in the evidence*).

Note: Patients with symptomatic OHS who have significant comorbidities and those with chronic respiratory failure after an episode of acute-on-chronic hypercapnic respiratory failure may particularly benefit from using PAP.

Respiratory Management of Patients With Neuromuscular Weakness
An American College of Chest Physicians Clinical Practice Guideline and Expert Panel Report

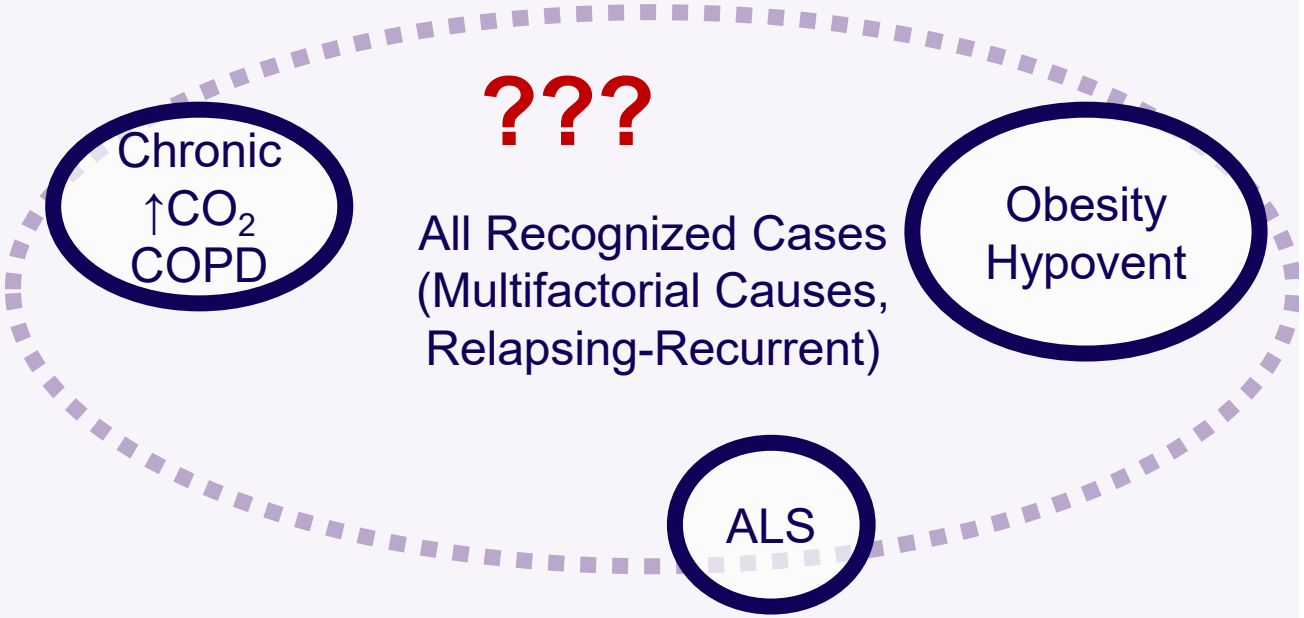
4. For patients with NMD and chronic respiratory failure, we recommend using NIV for treatment (Strong Recommendation, Very Low Certainty of Evidence).

Long-Term Noninvasive Ventilation in Chronic Stable Hypercapnic Chronic Obstructive Pulmonary Disease
An Official American Thoracic Society Clinical Practice Guideline

5. We **suggest** NIV with targeted normalization of Pa_{CO_2} in patients with hypercapnic COPD on long-term NIV (conditional recommendation, low certainty).

Two problems:

1. Patients in real life often have **multiple contributing causes** for their hypercapnia



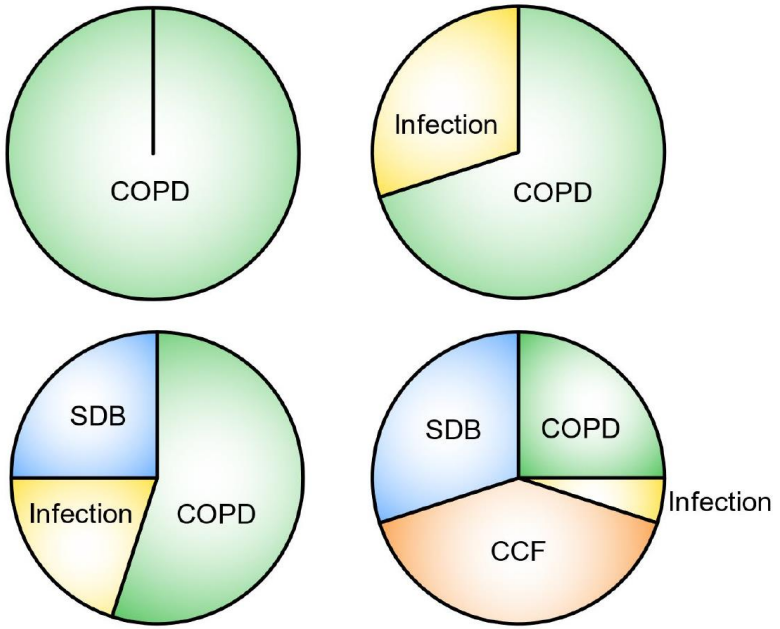
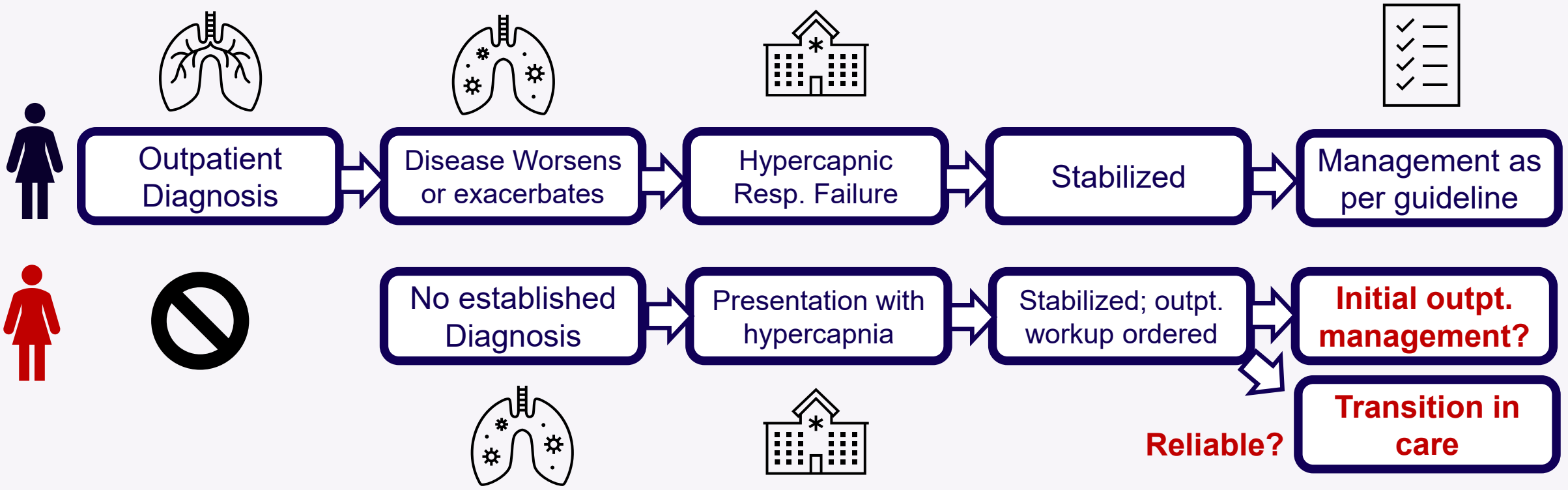


Figure 2.2. *Cause sets for hypercapnic respiratory failure. A single diagnosis of sufficient severity or multiple diagnoses working together may lead to hypercapnic respiratory failure. COPD Chronic obstructive pulmonary disease, SDB Sleep-disordered breathing, CCF Congestive cardiac failure.*

Two problems:

2. Hypercapnic respiratory failure often **precedes diagnosis** of the causative disease



The Discharge Paradox

ATS Guidelines (strong* recommendation *[the evidence isn't actually strong]***):**

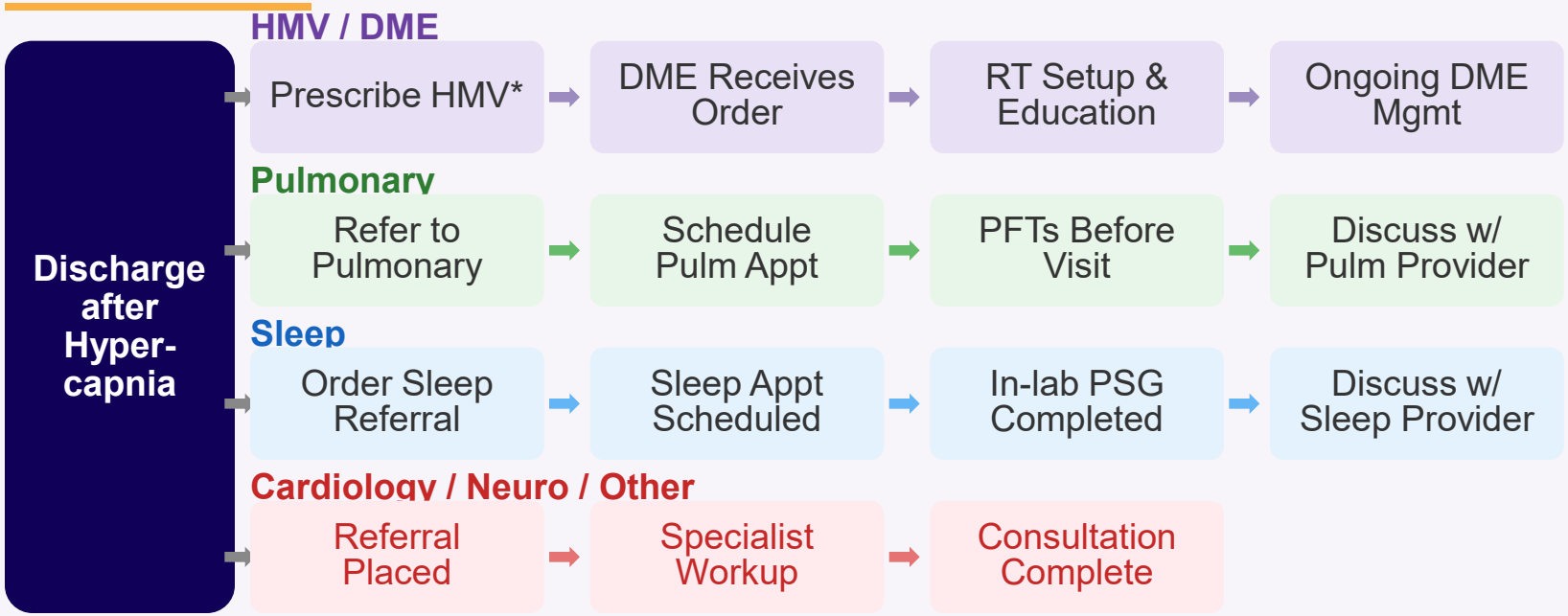
- Discharge with empiric NIV in hospitalized patients with OHS

Reality:

- No such thing as a "loaner" BPAP - **forbidden**
- Qualification requires testing that takes days to weeks, who owns
- Patient goes home on O_2 alone — or doesn't go home at all. Not

The evidence says "send them home with BPAP" — the system says "you can't."

Post-discharge diagnostic workflow: many opportunities to de-rail



⚠ Referral may not be placed

⚠ Special resources required

⚠ No single pathway owner

⚠ Lost to follow-up

So what can you do?

Call or message me
(or JB in U of U setting)

Consultative service
To clinic referral

Pulm disease navigator (future)
Owns the full pathway

How Did We Get Here?

1940s–50s

CO_2 retention physiology first described during polio epidemics. Coma was "cerebrelia" — thought to be viral encephalitis — actually hypercapnic respiratory failure.

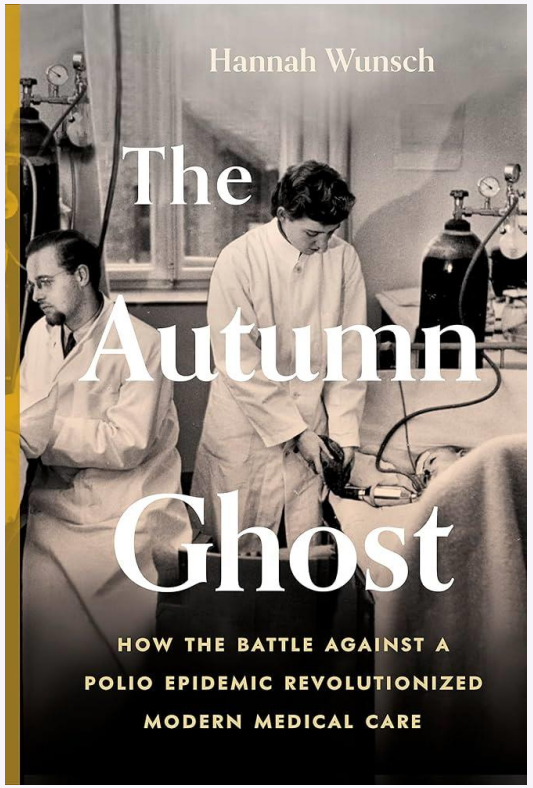
*Persistent severe hypercapnia is **only possible since** supplemental O_2*

1960s

Polio survivors need lifelong ventilation → March of Dimes respiratory centers (iron lungs). Centers close in 1960 (funding crisis).

1970s

Care transfers to CMS → trach + home mechanical ventilators become norm. National Coverage Determination written for their use (not anticipated to last).



1981–Present: CPAP → Convergence → Regulatory Split

1981 CPAP invented for OSA → later adapted as BPAP for other conditions

1990s HMVs, hypoventilation devices, and SDB devices converge technologically

1998 CMS creates "Respiratory Assist Device" (RAD) category to avoid tsunami of requests for home ventilators; NCD written

2025 Same 1998 NCD still governs BPAP qualification

The regulatory distinction today:

- **PAP devices =** treatment for sleep apnea; either constant or bilevel
- **RADs** = bilevel devices ± backup rate; evolved from CPAP
- **HMVs** = evolved from hospital ventilators; designed as life-support

Each got its own National Coverage Determination with distinct qualification pathways despite technological convergence

Home NIV Devices: RAD vs. PAP vs. HMV

	PAP for Sleep Breathing	RAD	HMV
What it is	CPAP / BPAP (for OSA / SDB)	Bilevel PAP ± backup rate (for hypoventilation)	Home mechanical ventilator (life-support)
HCPCS	E0601 (CPAP) E0470 / E0471 (BPAP)	E0470 / E0471	E0465 / E0466 / E0467
Examples	CPAP, AutoPAP, BPAP (AirSense, DreamStation)	BPAP-ST, VAPS (Aircurve ST, Luna G3)	Trilogy, Astral, VOCSN
Max pressure	~20–25 cmH_2O	~25–30 cmH_2O	~50–60 cmH_2O
Qualification	**Diagnostic Sleep Study (PSG or HSAT)**	**RAD NCD pathway: diagnosis + clinical criteria**	**Clinician attestation + qualifying diagnosis**
Payment	Rent-to-own (13 mo)	Rent-to-own (13 mo)	Monthly rental (indefinite)
Home RT	No	No	Yes — included in FSS
Key difference	Most common device; streamlined pathway	No home support after purchase	All-inclusive: device + supplies + RT visits

Where Does the Money Come From?

HMVs: Frequent and Substantial Servicing (FSS) payment

- Monthly rental — all-inclusive: device + supplies + maintenance + home RT services
- Payments continue indefinitely (no rent-to-own)

RADs and PAP devices: Rent-to-own (capped rental)

- 13 months of rental → ownership transfers to patient
- No home RT support, no ongoing supplies coverage

The scale:

- Medicare NIV charges reached $424.4 million in 2018
- OIG found CMS overpaid ~$86.6M on HMV (E0466) vs. non-Medicare rates (2016–18)

Current Medicare guidelines do not reflect modernization that other payers and countries have employed.

Prior National Coverage Determination: **HMVs**

Patient has a NMD, Thoracic Restrictive Disease, or Chronic Respiratory Failure from COPD

Where "the condition is life-threatening where interruption of respiratory support would quickly lead to serious harm or death."

- Unlike RADs, no definitions of medical conditions or what constitutes life-threatening were given

Old National Coverage Determination: **RADs**

I. Restrictive thoracic disorders

Documentation of a neuromuscular disease (e.g. amyotrophic lateral sclerosis) or a severe thoracic cage abnormality (e.g. post-thoracoplasty for tuberculosis [TB]).

One of the following:

- **Arterial blood gas (ABG)** $PaCO_2$, done while awake and breathing, the patient's prescribed Fi02 is ≥ 45 mm Hg.
- **Sleep oximetry** demonstrates oxygen saturation ≤ 88% for ≥ 5 minutes of nocturnal recording time (minimum recording time of 2 hours), done while breathing the patient's prescribed recommended FiO_2.
- **For neuromuscular disease only,** maximal inspiratory pressure is < 60 cm H_2O, **or** forced vital capacity (FVC) is < 50% predicted.

Chronic obstructive pulmonary disease (COPD) does not contribute significantly to patient's pulmonary limitation.

E0470 or E0471
Based on the treating practitioner's judgment

II. Severe COPD

ABG $PaCO_2$ is ≥ 52 mm Hg while patient is awake and breathing the prescribed FiO_2.

Sleep oximetry study demonstrates oxygen saturation ≤ 88% for ≥ 5 cumulative minutes of nocturnal recording time (minimum recording time of 2 hours), done while breathing oxygen at 2 liters per minute (LPM) or the patient's prescribed FiO_2 (whichever is higher).

Prior to initiating therapy, sleep apnea and treatment with CPAP has been considered and ruled out. (Note: Formal sleep testing is not required if there is sufficient information in the medical record to demonstrate that the patient does not suffer from some form of sleep apnea [obstructive sleep apnea (OSA), central sleep apnea (CSA) and/or complex sleep apnea (CompSA)] as the predominant cause of awake hypercapnia or nocturnal arterial oxygen desaturation).

E0470

IV. Hypoventilation

An initial ABG $PaCO_2$, done while awake and breathing the patient's prescribed FIO_2, is ≥ 45 mm Hg.

Spirometry shows an FEV1/FVC ≥ 70%.

- **An ABG** $PaCO_2$, done during sleep or immediately upon awakening, and while breathing the patient's prescribed FiO_2, shows the patient's $PaCO_2$ worsened ≥ 7mm Hg compared to the initial ABG result, **or**
- **A facility-based PSG or home sleep testing (HST)*** demonstrates oxygen saturation ≤ 88% for ≥ 5 minutes of nocturnal recording time (minimum recording time of 2 hours) that is not caused by obstructive upper airway events (i.e. AHI < 5).

E0470

The Qualification Maze

To qualify a patient for a RAD, the provider must:

- 1. Determine likely diagnosis: OSA, COPD, CSA, restrictive thoracic disease (NMD or thoracic cage abnormality), or hypoventilation
- 2. Proceed with focused testing for that specific diagnosis:
  - Polysomnography or home sleep apnea testing
  - Overnight oximetry
  - Spirometry
  - Arterial blood gas ($PaCO_2$)

The paradox:

- The COPD pathway requires nocturnal oximetry – not clinically relevant (30 years old!)
- The HMV NCD is so old and vague that it's often *paradoxically easier* to qualify for than a RAD **($$$)**
- These 1998 guidelines have not been updated to reflect the current evidence base, **yet**

What's Changing in the New Coverage Determinations for **RADs**?

NMD pathway: No change. | COPD / hypoventilation pathways: Updated below.

"COPD" — Outpatient	"COPD" — On Discharge
Chronic Respiratory Failure Dx ("COPD" includes all non-OSA hypercapnia) No PFTs required	Acute-on-Chronic Respiratory Failure Dx No PFTs required
$PaCO_2$ ≥ 52 mmHg 2+ weeks after hospitalization or without exacerbation of symptoms	Document full RAD settings within 24h of discharge
Sleep apnea is not the sole cause No PSG or overnight oximetry required	RAD needed to avoid symptoms or rapid $PaCO_2$ rise
Q6-month Continued Usage: confirm medically necessary, avg ≥5h/24hr usage, and improved CO_2, exacerbations, or symptoms	

Section Take Home Points: Devices

01 **Separate the mode from the purpose.** All three device categories (PAP, RAD, HMV) can deliver BPAP — what differs is the clinical intent: treat sleep-disordered breathing, treat hypoventilation, or provide life support.

02 **Prior sleep study? Use the PAP pathway.** If the patient already has a diagnostic sleep study, you can often qualify them for the device functionality they need through the streamlined sleep PAP pathway.

03 **Will they fail without a machine? Consider HMV.** A home mechanical ventilator can be used at least temporarily — qualification is straightforward, though cost is higher with ongoing rental.

4 **Everyone else? Navigate the RAD pathway.** For patients without a prior sleep study who aren't acutely failing, you'll need the RAD NCD pathway.

Phone a friend.

Emerging Role of GLP-1 Receptor Agonists

Weight Loss Improves OSA

- ~0.45 AHI events/hr reduction per 1% body weight lost
  - *Locke et al. Obes Rev. 2024; meta-analysis of 10 trials, n=854*

SURMOUNT-OSA Trial (n=469; two phase 3 RCTs)

- Tirzepatide: AHI −29.3 events/hr vs −5.5 placebo at 52 wk
- Also reduced body weight, hypoxic burden, hsCRP, systolic BP
  - *Malhotra et al. N Engl J Med. 2024;391(13):1193–1205*

GLP-1 RA Benefit Depends on Causal Role of OSA in Hypercapnia

- OSA found in **63–88%** of ICU hypercapnic patients when sleep testing is performed (~5× the rate identified by clinician assessment)
- Most patients with hypercapnic respiratory failure have an FDA indication for GLP1ra, but coverage challenging. (Unpublished work)
- The extent that OSA is causal vs. epiphenomenal in many of these cases is not established
  - *Locke, Brown, Sundar. Sleep Med Clin. 2024;19(2):339–356*

What We're Learning — And What's Changing

Benchmarking the problem statewide

- How often are patients with hypercapnic respiratory failure getting tested?
- What are the readmission, ED return, and mortality rates across Utah?
- Using Utah Population Database + All-Payer Claims (CO_2-PATH Cohort)

Moving toward risk-based (not diagnosis-based) referral

- Expedite workup and empiric management for high-risk patients
- Risk stratification via Select Health claims (AIM-HIGH project)

New NCD guidelines (2025) — finally modernizing RAD qualification

- No longer requires PFTs for non-OSA hypercapnia qualification
- Discharge pathway now has its own qualification track

Take Home Point

Obesity-related respiratory failure is **common**, **morbid**, **under-recognized**, and **misattributed**.

The diagnosis is hiding in plain sight — look for hypoxemia and elevated HCO_3 in obese patients with unexplained respiratory failure.

Regulatory and workflow barriers are real — but navigable.

Summary

01 OHS is a diagnosis of exclusion. **“Obesity-related respiratory failure”** is a more practical clinical framework.

02 It is **common, morbid, and under-recognized.** Use hypoxemia and elevated HCO_3 to trigger workup. Attribute the cause via skeptical process of elimination.

03 Discharging on BPAP is hard: **outdated NCDs, device categories, and payment structures** create a qualification maze — but these are changing.

04 **GLP-1 receptor agonists** are an emerging tool — weight loss may improve or resolve the underlying respiratory failure, but data are sparse.

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Scan for resources

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