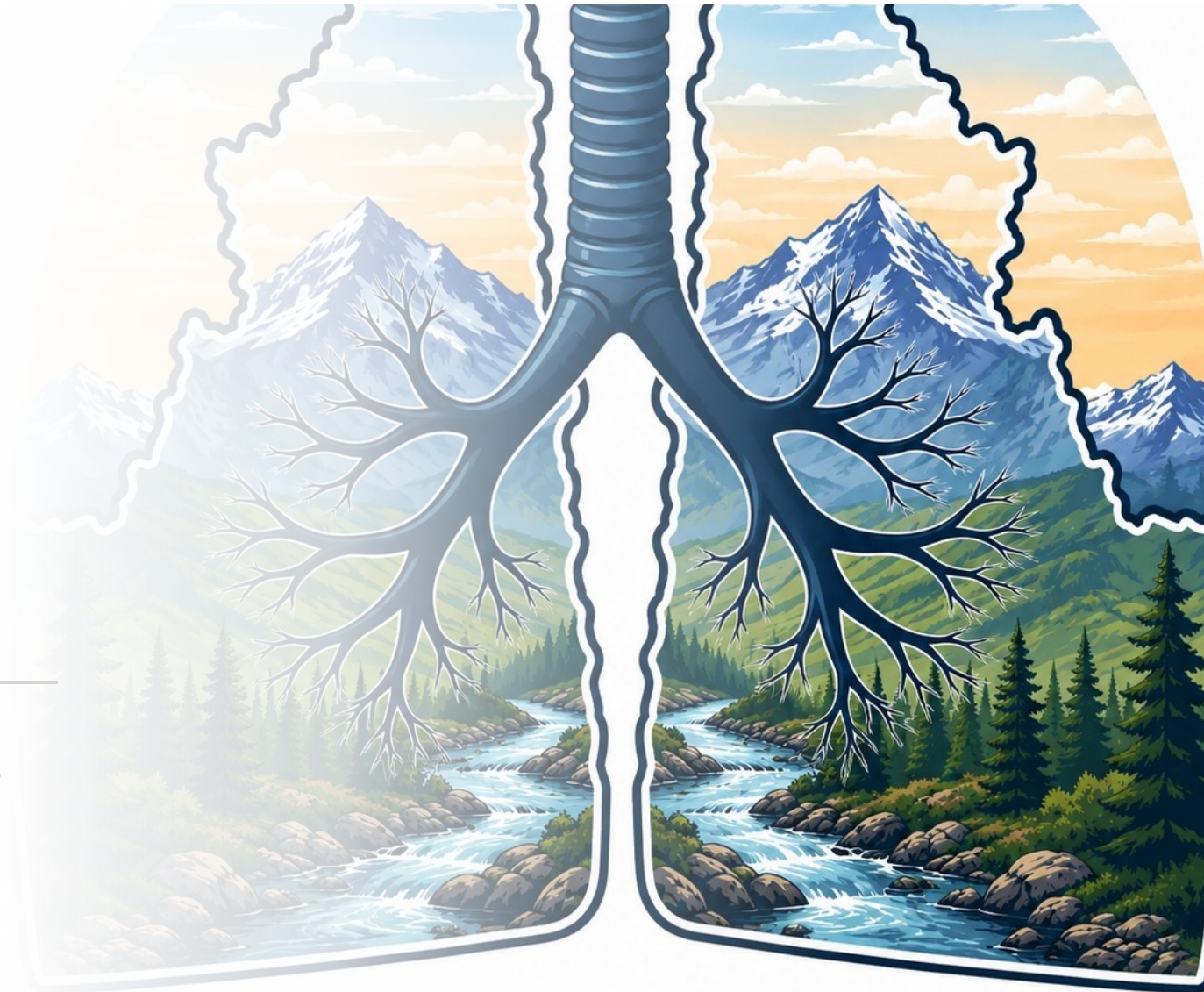


Idaho Society for Respiratory Care

Annual Conference
2026



verathon



Dräger

Aerogen®
Pioneering Aerosol Drug Delivery



Thank You
VENDORS



INOmax
Total Care®



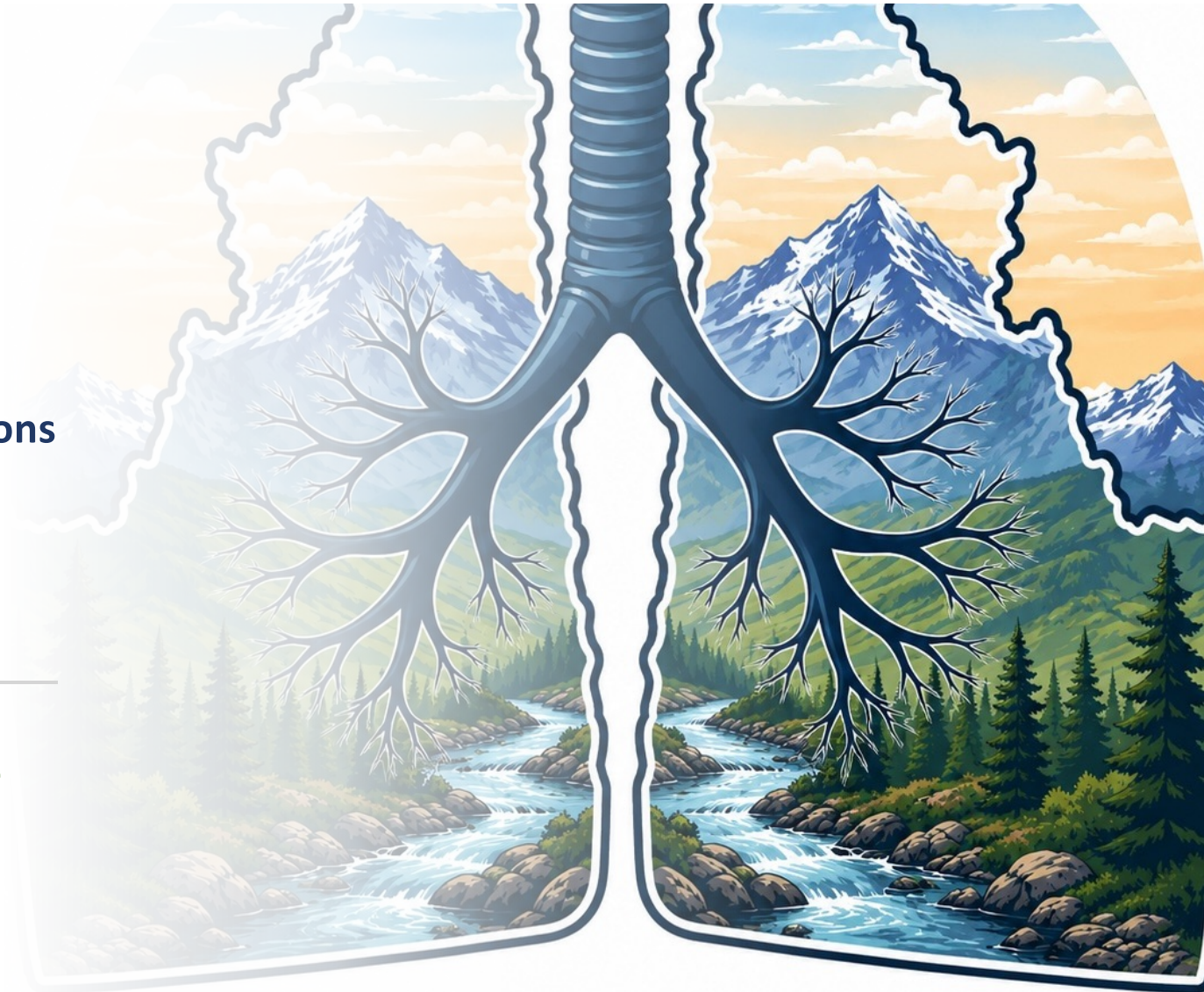
ZOLL®

GETINGE*

WELCOME

- Vendor Hall
- THANK YOU
- Reminder: CRCE Evaluations
- Breaks and Food
- Business Meeting @ 5pm

Annual Conference
2026



ISRC, 9/23/26

Insights into Healthcare's AI Evolution

Dr. Jason Blomquist

Disclosure

I have no conflicts of interest to disclose.

Any mention of a specific company or brand name is merely for illustration, and not to be seen as an endorsement.

Objectives

1. Participants will understand the differences between types of AI used in healthcare.
2. Participants will identify potential ethical issues with using AI in healthcare.
3. Participants will differentiate between viewing AI as a passive software tool versus an active "digital colleague".

AI-POWERED HEALTH PATHWAY

PART 1: CONVERSATIONAL HISTORY INTAKE

HEALTH DATA INTAKE

- **Patient Self-Report:** Fatigue for 3 weeks; Father has heart disease.
- **CHIEF COMPLAINT:** Fatigue
- **FAMILY HISTORY:** Heart disease (father)
- **LIFESTYLE:** Sedentary
- **SYMPTOM CONTEXT:** Fatigue ongoing for 3 weeks

COLLABORATIVE DECISION MAKING

AI PROCESSING CORE

DEEP LEARNING & NLP

PERSONALIZED RISK SCORE: 6.8/10
(Elevated)

TOP RISKS:
MYOCARDIAL INFARCTION
DIABETES

ACTIONABLE INSIGHTS:
COMMENCE STATIN,
DIETARY CHANGE,
150 MIN ACTIVITY/WEEK

SIMULATED OUTCOME:
Reduces 20-year event
risk by 60%

CLINICIAN DASHBOARD

PART 2: MULTI-PATH RISK SIMULATION & PREDICTION

FUTURE HEALTH TRAJECTORIES Simulate 20-year timeline

PATHWAY A: CURRENT LIFESTYLE (NO INTERVENTION)

20y
15
Increase weight High blood pressure
HIGH RISK:
CARDIOVASCULAR EVENT,
TYPE 2 DIABETES (70%)

PATHWAY B: MODEST INTERVENTION (DIET & EXERCISE)

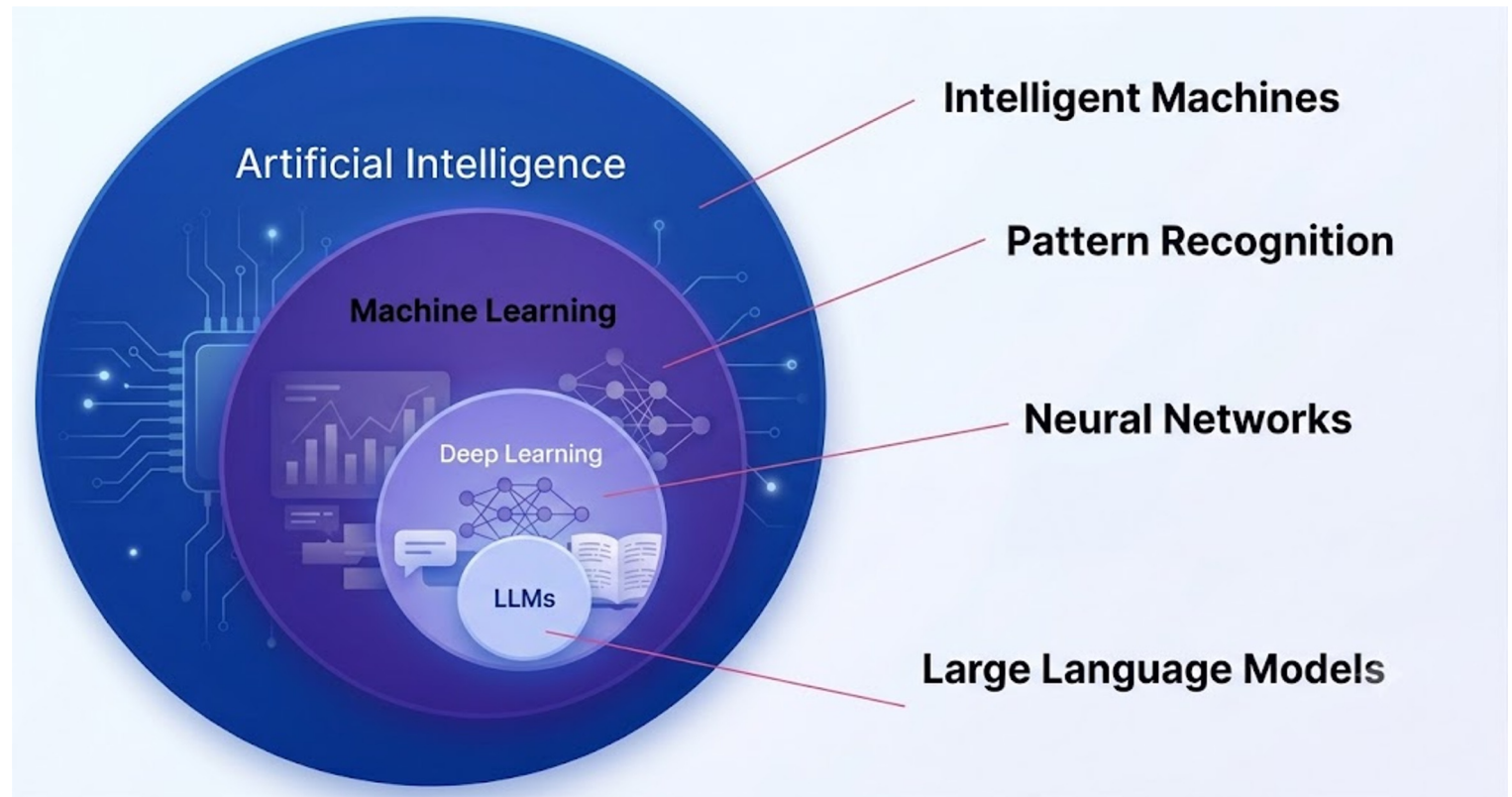
Improved metrics High metrics Type diabetes
MODERATE RISK:
CARDIOVASCULAR EVENT,
TYPE 2 DIABETES (35%)

PATHWAY C: EARLY INTERVENTION (PERSONALIZED PLAN + MEDICATION)

Optimal health Health health Type health
LOW RISK:
CARDIOVASCULAR EVENT,
TYPE 2 DIABETES (10%)

Task Focus:

- Predictive
- Generative
- Agentic

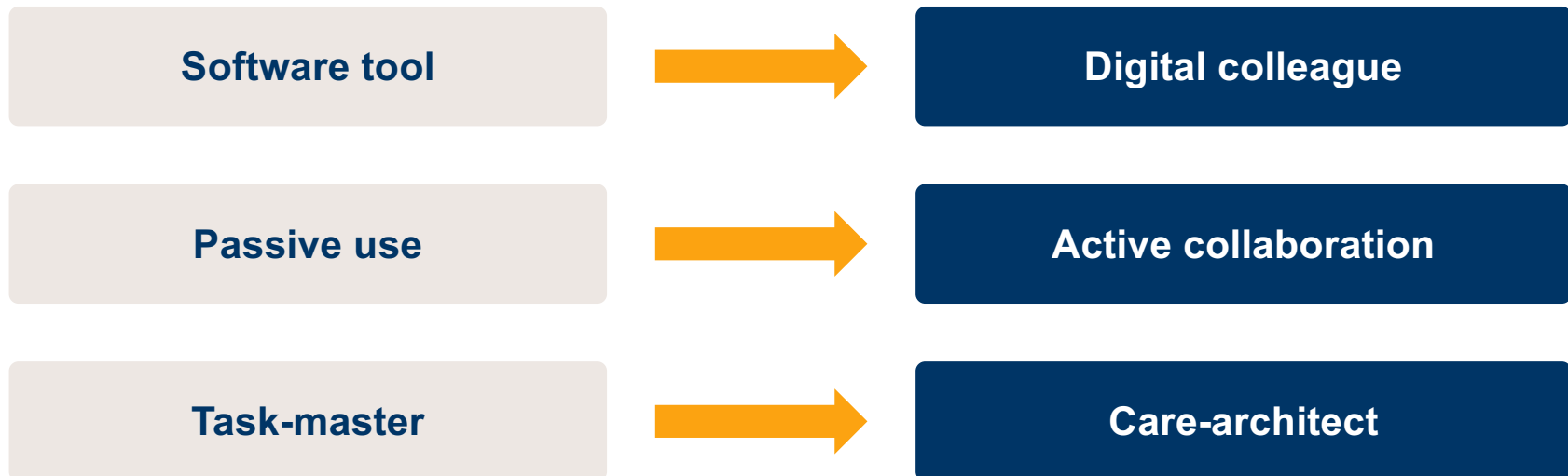


It's already in the chart

Art clinical documentation
Emmie patient-facing assistant
Penny revenue cycle
Cosmos & Curiosity
the data, and predictions from it
Ergo Visit
Guided outpatient visits
Agent Factory
organizations deploying agents

- Ambient documentation
- AI-drafted nursing assessments, end-of-shift care plans, etc.
- Inpatient Insights
- The assistant answering portal messages
- An AI agent phoning patients after discharge
- AI at the bedside TV
- Predictive and generative decision support

From tool to colleague



automation · augmentation · agency

Our responsibility

“Balancing the risks and benefits of technologies requires that nurses **stay informed** about developments, acknowledge the **potential benefits and harms**, maintain the **dignity** of the recipient of care, complement the **relational nature of nursing**, and ensure the **voice of nursing** is present when decisions are made in healthcare systems”

Code of Ethics for Nurses
Provision 7.5

Challenge: The documentation paradox

AI drafting their assessments and notes in real-time during a patient encounter

No longer "**can you document** an assessment?"

but "**can you identify what's missing** from the AI assessment and add human elements that matter?"

- **The patient's own words**
- **A family dynamic seen in the room**
- **A subtle change from yesterday**
- **The thing not said**

Challenge: The patient trust evolution

PATIENT

I asked the app about my new blood pressure medication and it said dizziness means the dose is too high, so I only took half this morning.

Caregiver

Dizziness is worth paying attention to, and the app was right that it can be a dose issue. It can also mean you stood up too fast or had not eaten, and the app could not see that you

validate

contextualize

educate

Challenge: The equity and justice imperative

Was this built on
patients like mine?

Training data is a convenience sample.

Challenge: The predictive alert dilemma

DETERIORATION RISK

LOW

Predicted by model · updated
0700

vs

Caregiver **ASSESSMENT**

*"She is not herself this
morning. Something has
changed."*

"I am concerned about this result."

The ETHICAL Framework

E – Exploration & Evaluation

T – Transparency

H – Human-Centered

I – Integrity (Professional & Academic)

C – Continuous Learning

A – Accessibility & Inclusivity

L – Legal & Regulatory

Three things

- 1 You know your professional role.
- 2 Supervise AI the way you would supervise a person.
- 3 Be in the room where it gets designed.

Accountability does not transfer. Neither does the responsibility to be in the room.

Jason Blomquist

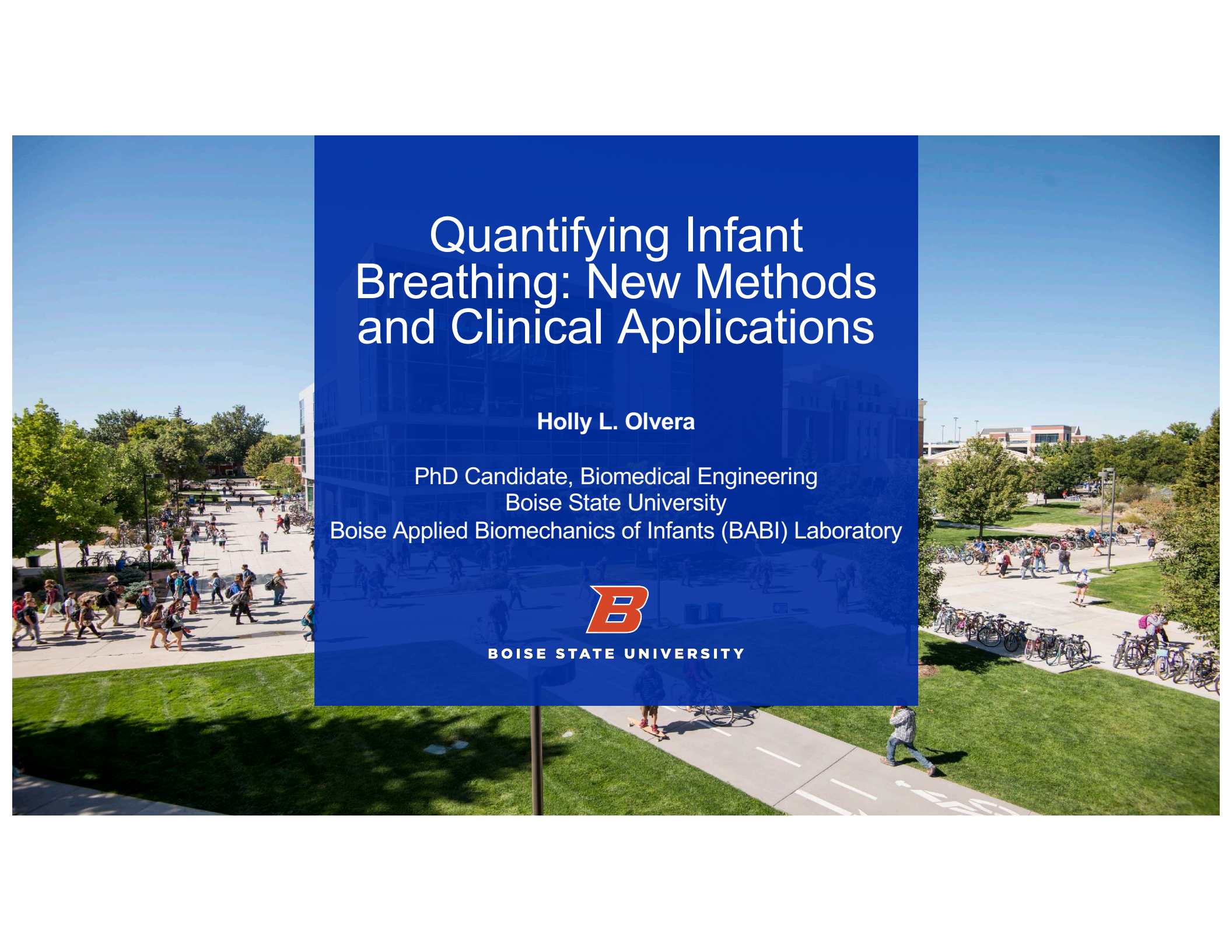
jasonblomquist@boisestate.
edu

Be Curious
Be Courageous
Be Tenacious



Contact information

Let's Collaborate!

A wide-angle photograph of a Boise State University campus. In the foreground, a paved walkway leads through a green lawn. To the left, a large group of students is walking. To the right, many bicycles are parked in racks. In the background, there are modern university buildings and more trees under a clear blue sky.

Quantifying Infant Breathing: New Methods and Clinical Applications

Holly L. Olvera

PhD Candidate, Biomedical Engineering
Boise State University
Boise Applied Biomechanics of Infants (BABI) Laboratory



BOISE STATE UNIVERSITY

Project Collaborators & Disclosures



BOISE STATE UNIVERSITY
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Holly Olvera
Shameka Kimmel



BOISE STATE UNIVERSITY
Computational Biosciences Lab

Clare Fitzpatrick, PhD Lab Director
Andrew Bossert



Keith Rhoades, Founder



PRODUCT SAFETY • DESIGN • PERFORMANCE

Aaron A. Cadotte, Founder



HOSPITALS • RESEARCH • FOUNDATION

John Carroll, MD
Brandi Whitaker, PhD



BOISE STATE UNIVERSITY
Department of Respiratory Care

Megan Koster, EdD, RRT, RRT-NPS
Camille Stover, MET, RRT, RRT-NPS

This research is supported by the NICHD, NIH Grant No. 1R01HD113921

Disclosure: EM owns Mannen Bio, LLC, an expert and engineering consulting company.

Learning Objectives

- Challenges associated with measuring respiratory biomechanics in infants
- How respiratory monitoring & biomechanical analysis come together
- Informing further research and future clinical practices

History of the BABI Lab – the People



History of the BABI Lab – the Why



~60% supine to supine
~40% supine to prone
Some before baby could roll
BIOMECHANICS-related

History of the BABI Lab – the Work



Supine: Evidence for easier rolling

- Increased trunk flexion
- More similar to a seat than lying flat



Prone: Infants are struggling

- 2x increase of abdominal activity
- Increased trunk movement

History of the



All Info
Be Rec

In the v
a new s
risks

By Rachel Rob
Last updated:



Sleeping
Not Sa

By

By ANNA WERI

Stud
Roc
dea

Public Law 117–126 117th Congress

An Act

May 16, 2022
[H.R. 3182]

Safe Sleep for
Babies Act of
2021.
15 USC 2051
note.

15 USC 2057d.
Deadline.

15 USC 2057e.
Deadline.

To provide that inclined sleepers for infants and crib bumpers shall be considered banned hazardous products under section 8 of the Consumer Product Safety Act, and for other purposes.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled,

SECTION 1. SHORT TITLE.

This Act may be cited as the “Safe Sleep for Babies Act of 2021”.

SEC. 2. BANNING OF INCLINED SLEEPERS FOR INFANTS.

(a) IN GENERAL.—Not later than 180 days after the date of enactment of this Act, inclined sleepers for infants, regardless of the date of manufacture, shall be considered a banned hazardous product under section 8 of the Consumer Product Safety Act (15 U.S.C. 2057).

(b) INCLINED SLEEPER FOR INFANTS DEFINED.—In this section, the term “inclined sleeper for infants” means a product with an inclined sleep surface greater than ten degrees that is intended, marketed, or designed to provide sleeping accommodations for an infant up to 1 year old.

SEC. 3. BANNING OF CRIB BUMPERS.

(a) IN GENERAL.—Not later than 180 days after the date of enactment of this Act, crib bumpers, regardless of the date of manufacture, shall be considered a banned hazardous product under section 8 of the Consumer Product Safety Act (15 U.S.C. 2057).

(b) CRIB BUMPER DEFINED.—In this section, the term “crib bumper”—

(1) means any material that is intended to cover the sides of a crib to prevent injury to any crib occupant from impacts against the side of a crib or to prevent partial or complete access to any openings in the sides of a crib to prevent a crib occupant from getting any part of the body entrapped in any opening;

(2) includes a padded crib bumper, a supported and unsupported vinyl bumper guard, and vertical crib slat covers; and

(3) does not include a non-padded mesh crib liner.

Approved May 16, 2022.

LEGISLATIVE HISTORY—H.R. 3182:

CONGRESSIONAL RECORD:

Vol. 167 (2021): June 22, considered in House.

June 23, prior proceedings vacated; considered and passed House.

Vol. 168 (2022): May 3, considered and passed Senate.



of Rock
pers

ns and safety
Commission

Learning Objectives

- Challenges associated with measuring respiratory biomechanics in infants
- How respiratory monitoring & biomechanical analysis come together
- Informing further research and future clinical practices

Is Infant Breathing as Simple as it Looks?

Chest wall motion?



Respiratory monitoring tells us important information about gas exchange and physiology, but it does not completely describe respiratory mechanics.

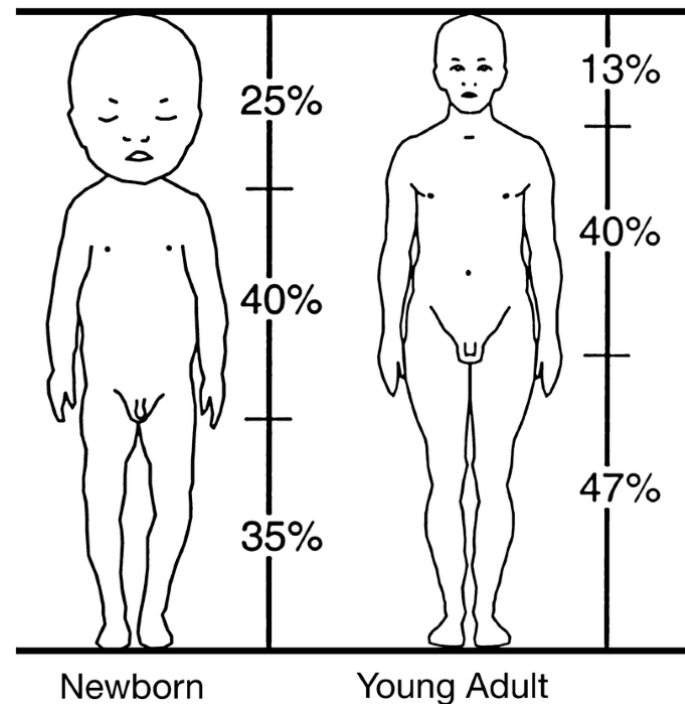


Abdominal motion?

An Infant is Not a Small Adult

Infant Breathing:

- Highly compliant chest wall
- Diaphragmatic breathing
- Smaller airways
- Rapid respiratory rates



How do we Quantify Breathing?

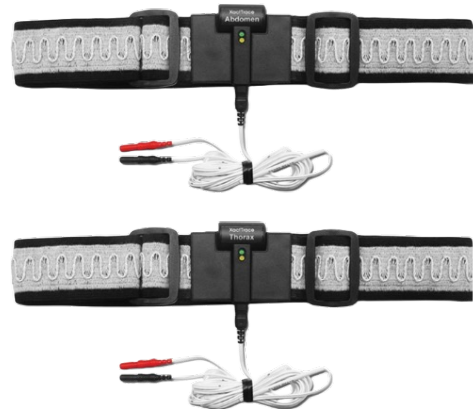
Clinical Monitoring Tools



Measurement Problem

How do we Quantify Breathing?

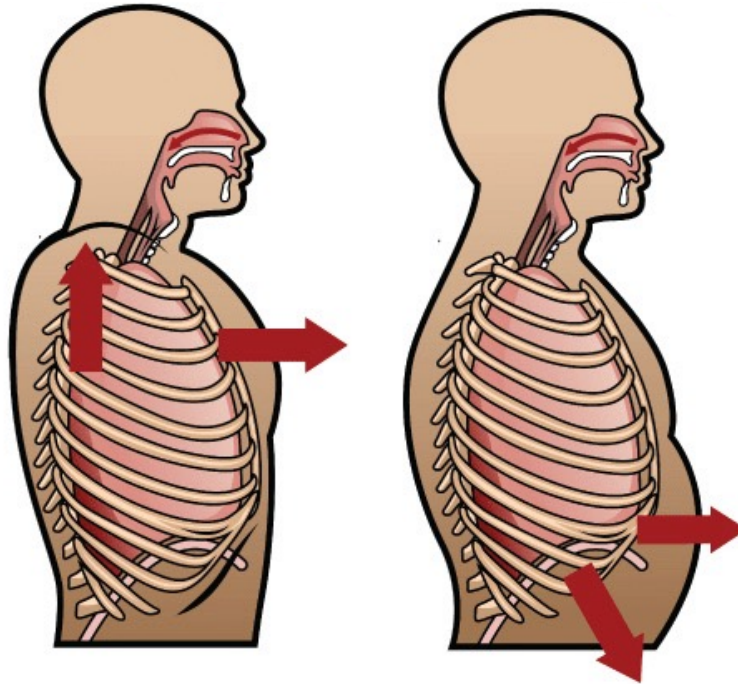
Traditional Pulmonary Measurement Tools



Measurement Problem

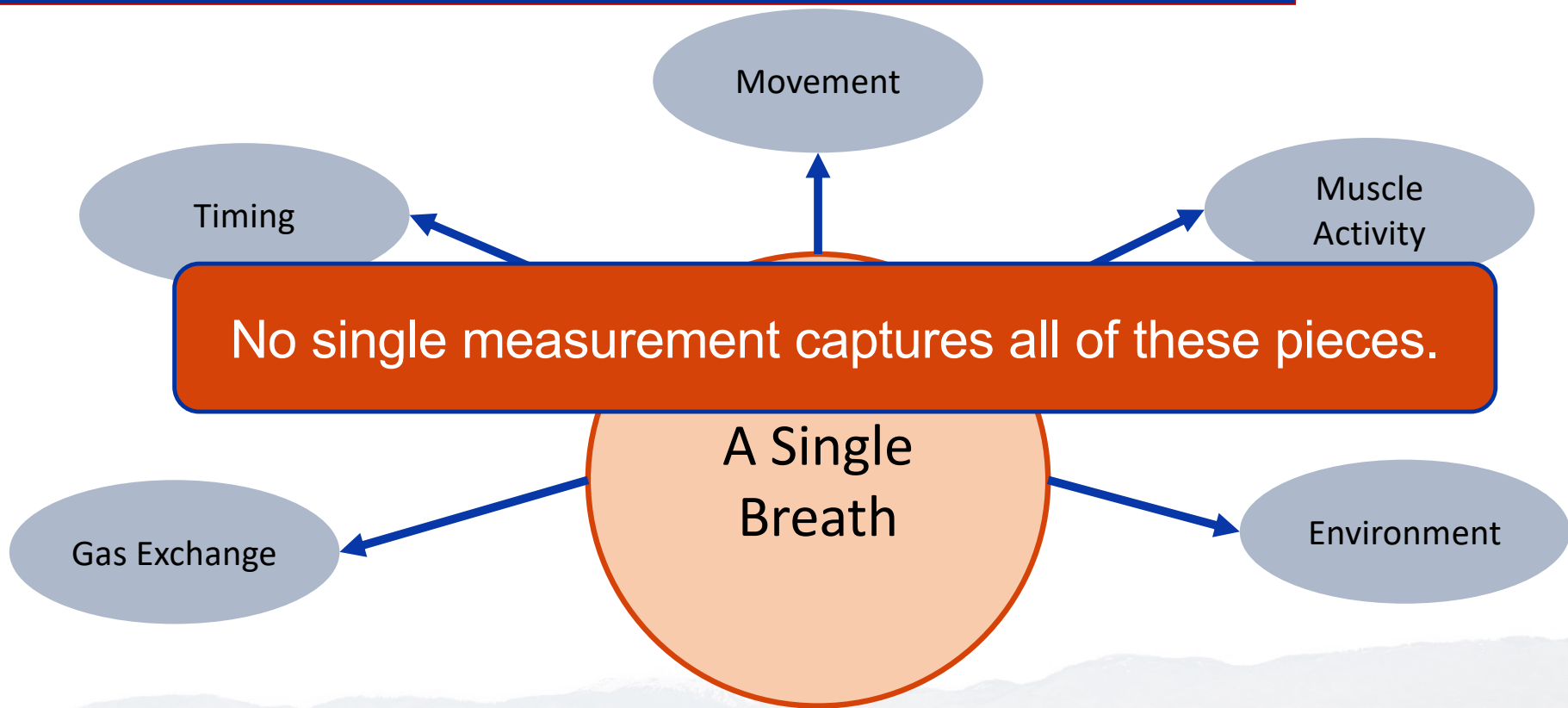
How do we Quantify Breathing?

Visual Assessment



Measurement Problem

Deconstructing a Single Breath



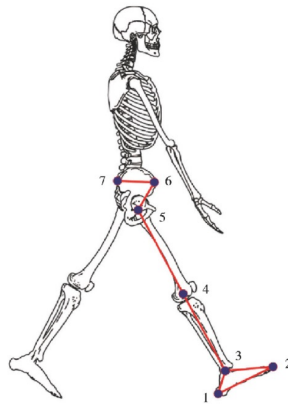
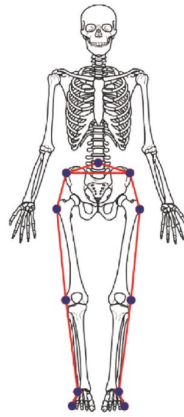
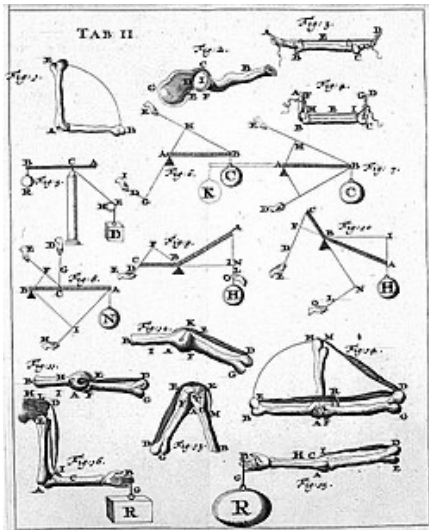
Building a Robust Measurement System

Three Layers:

1. What is happening physiologically?
2. What is happening mechanically?
3. What is the respiratory system doing to produce that breathing motion?

What is Biomechanics?

Mechanical Forces + Physics Principles → Living Organisms



Engineering Approach

Building a Robust Measurement System

Three Layers:

1. What is happening physiologically?
2. What is happening mechanically?
3. What is the respiratory system doing to produce that breathing motion?

Building a Robust Measurement System

1. What is happening physiologically?



Respiratory Rate
EtCO₂



Heart Rate
SpO₂

Building a Robust Measurement System

2. What is happening mechanically?



Engineering Approach

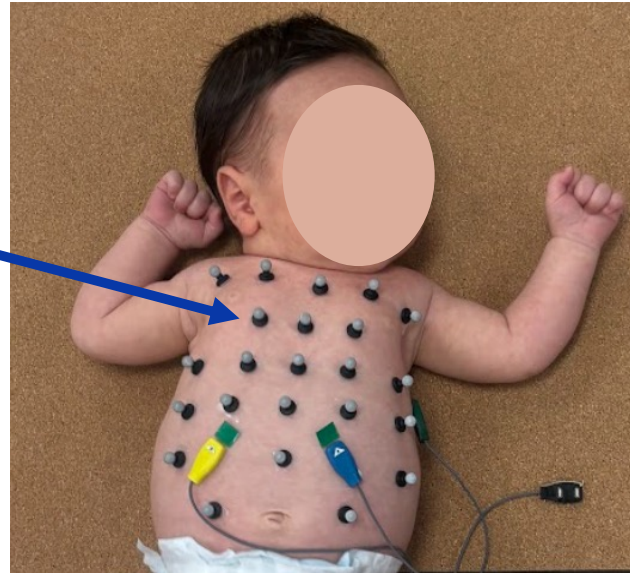


Building a Robust Measurement System

2. What is happening mechanically?

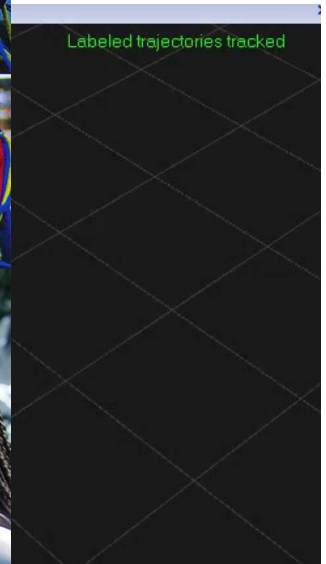
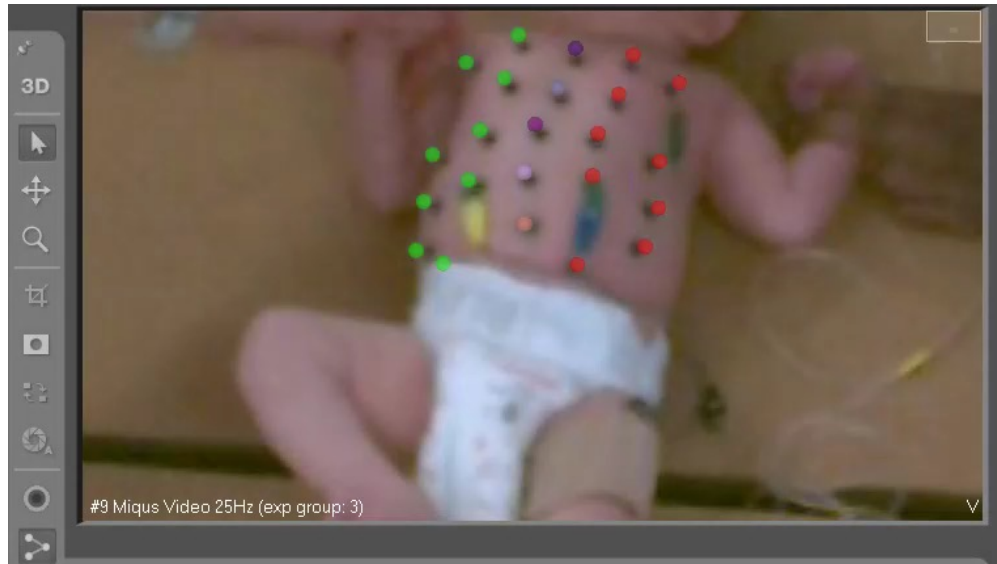


Positional Data



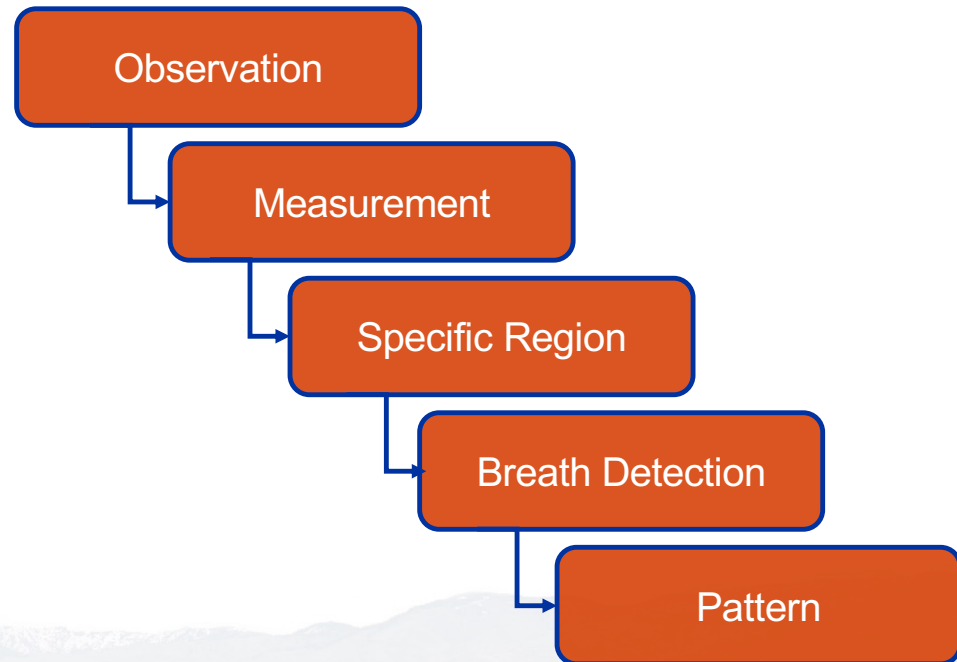
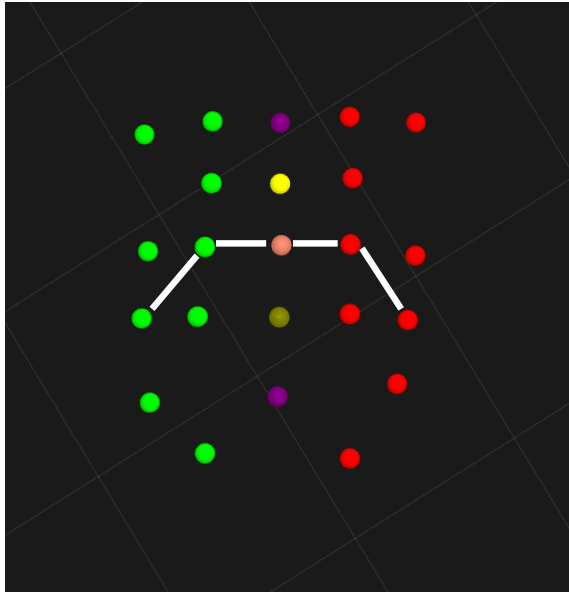
Building a Robust Measurement System

2. What is happening mechanically?

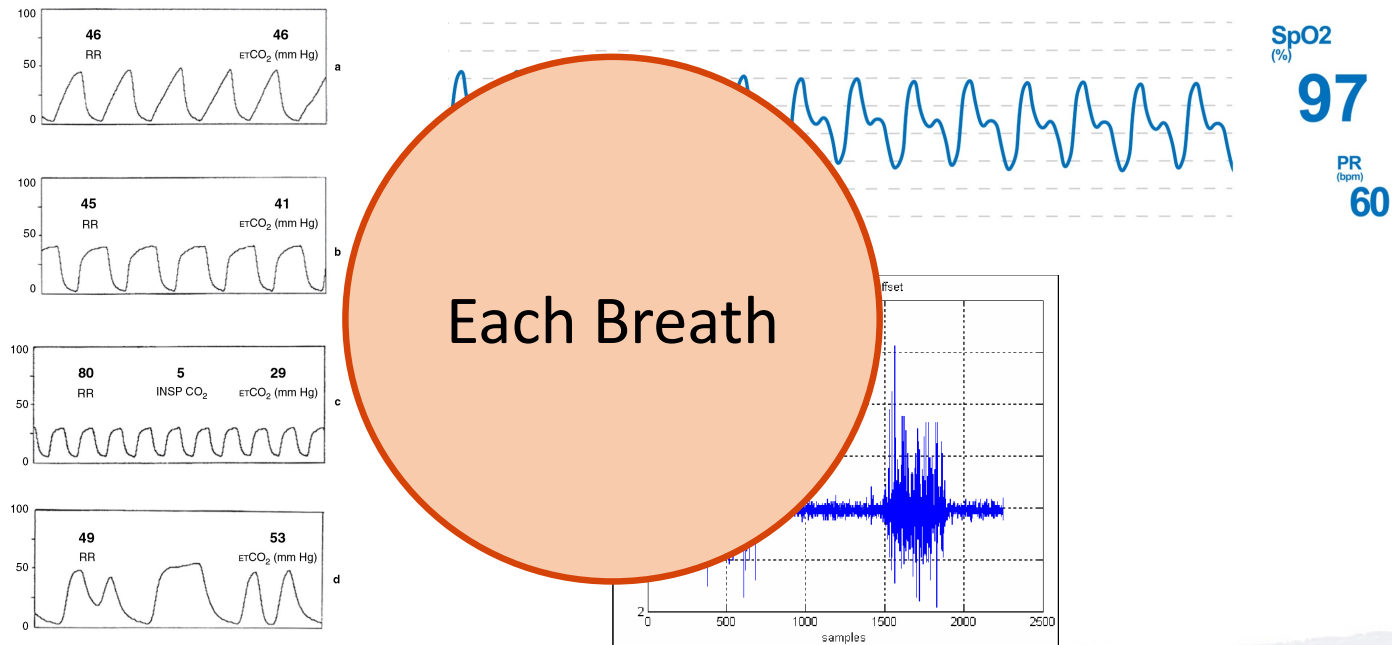


Building a Robust Measurement System

3. What is the respiratory system doing to produce that breathing motion?



Synchronizing the Signals



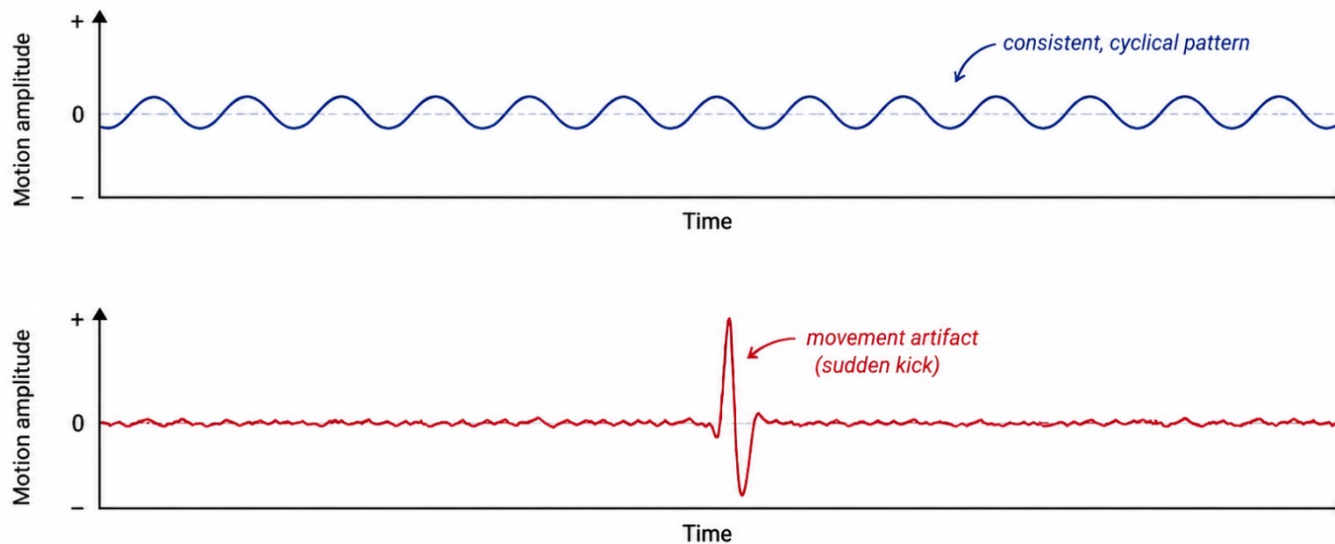
Why Methodology Matters



- Artifact identification
- Breath selection
- Signal quality
- Synchronization
- Repeatability
- Marker placement

Why Methodology Matters

The difference between movement and breathing



Why Methodology Matters

Infant Positioning



Validation Problem

Multimodal Respiratory Biomechanics

Positioning

Work of Breathing

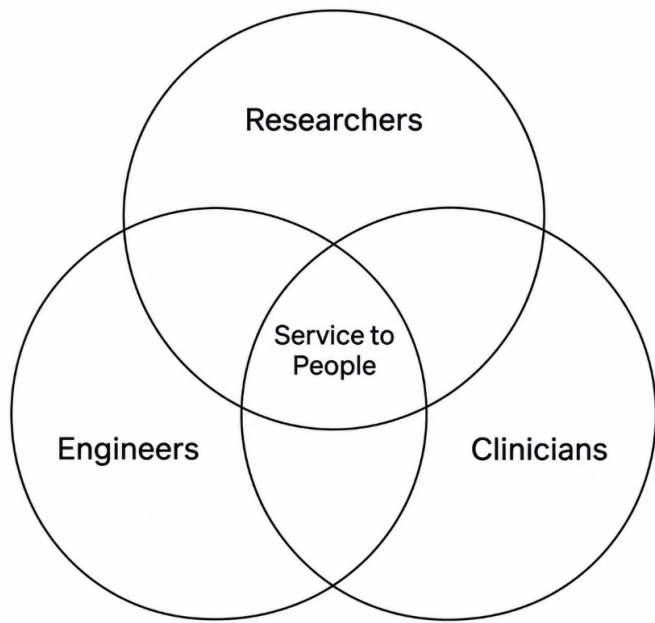
Development

Environment



Research Opportunity

The Greater Impact



Design



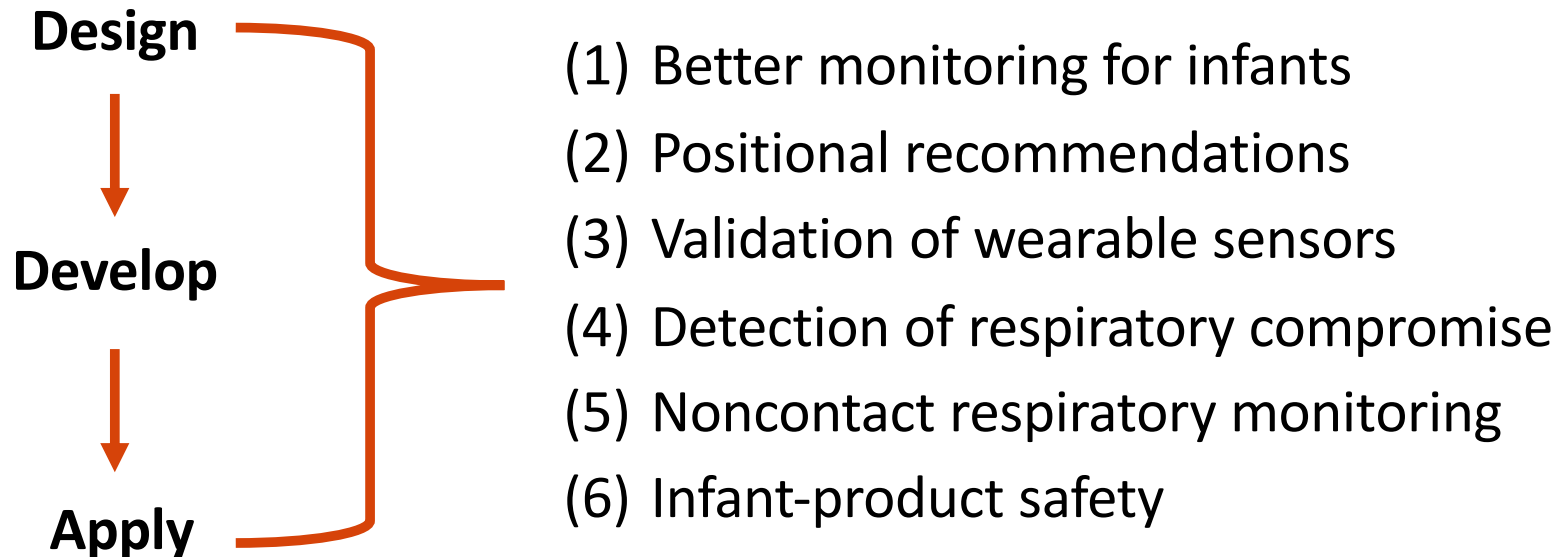
Develop



Apply

Clinical Translation

The Greater Impact



The Future

What would “translation” actually look like?

The Complete Picture



Take-Home



Thank you! Questions?

BabiLab@BoiseState.edu



BOISE STATE UNIVERSITY

Lung Cancer Screening 2026: An Evidence-Based Overview

**Paula Carvalho, MD, FCCP
Professor of Medicine
Division of Pulmonary, Critical Care, and Sleep Medicine
University of Washington
Academic Section Chief and Director, Intensive Care
Boise VA Medical Center**

Learning objectives: Our agenda

1. Review who should be offered annual lung cancer screening (LCS) with low-dose noncontrast chest CT (LDCT)
 2. Review the randomized-trial evidence for mortality benefit of LCS
 3. Review important screening complications (false positives and overdiagnosis)
 4. Compare US Preventive Services Task Force (USPSTF), American Cancer Society (ACS), and Medicare (CMS) eligibility criteria
 5. Apply a practical screening pathway using shared decision-making and smoking cessation
 6. A review of LCS radiology
-

LCS: Why screen for lung cancer?

- Lung cancer often produces very few or nonspecific symptoms until disease is advanced
- The goal of LCS is to detect cancer earlier, when curative treatment is more likely
- LDCT uses substantially less radiation than a standard diagnostic chest CT and is the only recommended screening test for LCS

USPSTF lung cancer screening recommendation; ACS Cancer Facts & Figures 2026

Low-dose chest CT: Is it safe?

Low-dose chest CT scans use minimal radiation and do not cause detectable damage to human DNA

Radiation levels in low-dose CT are $< 20\%$ that of standard chest CT or ≤ 1.0 millisievert (mSv)

1 mSV = ~ 10 chest X-rays or natural environmental radiation exposure over a few months

Sakane et al. Radiology 2020;295:439

Low-dose chest CT: Effects on DNA

- Human lymphocytes were tested before and after chest CT scans
- Standard-dose CT scans caused double-stranded DNA breakage and chromosome abnormalities
- Low-dose CT scans produced no DNA damage, and biological genetic harm was not detected



Radiology

ORIGINAL RESEARCH • THORACIC IMAGING

Biological Effects of Low-Dose Chest CT on Chromosomal DNA

Hiroaki Sakane, MD • Mari Ishida, MD, PhD • Lin Shi, PhD • Wataru Fukumoto, MD, PhD • Chiem Sakai, PhD • Yoshihiro Miyata, MD, PhD • Takafumi Ishida, MD, PhD • Tomoyuki Akita, PhD • Morihito Okada, MD, PhD • Kazuo Awai, MD, PhD • Satoshi Tashiro, MD, PhD

From the Department of Diagnostic Radiology, Graduate School of Biomedical Health Sciences, Hiroshima University, Hiroshima, Japan (H.S., W.F., K.A.); Department of Cardiovascular Physiology and Medicine, Graduate School of Biomedical and Health Sciences, Hiroshima University, Hiroshima, Japan (M.I., C.S.); Departments of Cellular Biology (L.S., S.T.) and Surgical Oncology (Y.M., M.O.), Research Institute for Radiation Biology and Medicine, Hiroshima University, 1-2-3 Kasumi, Minamiku, Hiroshima 734-8553, Japan; Department of Epidemiology, Infectious Disease Control and Prevention, Graduate School of Biomedical and Health Sciences, Hiroshima University, Hiroshima, Japan (T.A.); and Department of Cardiovascular Medicine, Fukushima Medical University, Fukushima, Japan (T.I.). Received February 20, 2019; revision requested August 1; revision received December 23; accepted January 3, 2020. Address correspondence to S.T. (e-mail: tashiro@hiroshima-u.ac.jp).

Supported by Japan Society for the Promotion of Science Kakenhi (grants JP18H03373, JP17K16445, JP16H01312, JP15H02821, JP26430114) and Tsuchiya Medical Foundation. Study supported in part by the Center of World Intelligence Project for Nuclear S&T and Human Resource Development by Ministry of Education (Development of a high-throughput radiation dose estimation system by using PNA-FISH method), Japan Science and Technology Agency Core Research for Evolutional Science and Technology (JPMJCR18S3), the International Atomic Energy Agency (IAEA CRP E35010), the Program of the Network-Type Joint Usage/Research Center for Radiation Disaster Medical Science of Hiroshima University, Nagasaki University and Fukushima Medical University.

Conflicts of interest are listed at the end of this article.

See also the editorial by Brenner in this issue.

Radiology 2020; 295:439–445 • <https://doi.org/10.1148/radiol.2020190389> • Content codes: **CT** **CH**

Why screen for lung cancer?

Lung cancer screening is not a substitute for smoking cessation!
Smoking cessation is still the most important prevention strategy



Lung Cancer Screening/US recommendations and evidence/September 2026

ACS Cancer Facts & Figures 2026

How do we use low-dose CT (LDCT) in lung cancer screening?

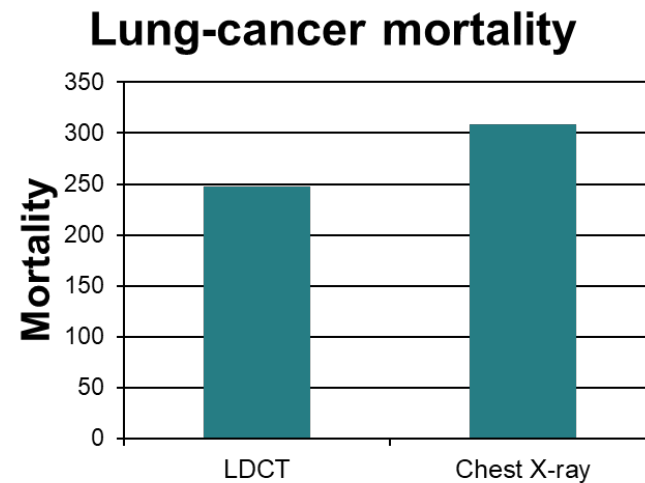


A screening scan is for people WITHOUT signs/symptoms of lung cancer
Abnormal findings are managed with standardized nodule assessment and follow-up rather than automatically proceeding to biopsy

Lung Cancer Screening/US recommendations and evidence/September 2026

Efficacy: National Lung Screening Trial (NLST):

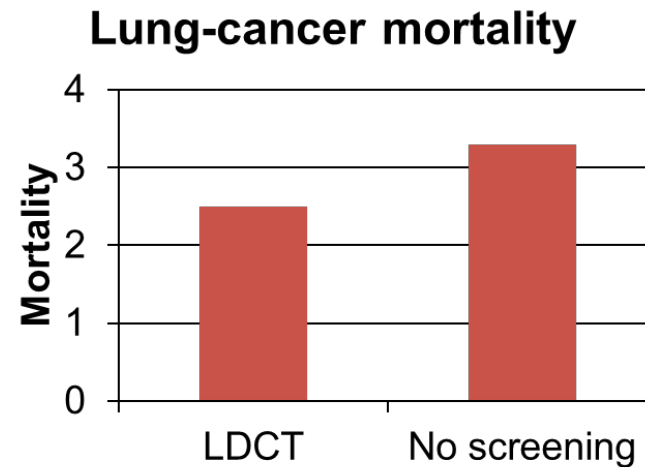
- 53,454 high-risk adults were randomized to 3 annual rounds of LDCT or chest radiography
- LDCT reduced lung-cancer mortality by 20% versus chest x-ray (95% CI, 6.8%–26.7%)
- All-cause mortality was also reduced by 6.7%
- LDCT detected more early-stage cancers, but positive screens were common and most were false positives



National Lung Cancer Screening Trial, NEJM 2011;365:395

Efficacy: NELSON randomized trial

- 15,792 participants were randomized to volume-based LDCT or no screening
- At 10 years, lung-cancer mortality was lower with screening : rate ratio 0.76 (95% CI, 0.61–0.94)
- Approximate relative reduction: 24%
- The trial supports a mortality benefit from LDCT beyond the NLST comparison with chest radiography



De Koning et al., NEJM 2020;382:503
USPSTF evidence review 2021

Overall evidence: Mortality benefit is consistent

USPSTF evidence review:

- NLST lung-cancer mortality incidence rate ratio (IRR) = 0.85 (15% mortality reduction in LCS group)
- NELSON IRR = 0.75 (25% mortality reduction in LCS group)
- Meta-analysis of randomized LDCT trials:
Lung-cancer mortality: Relative risk (RR) = 0.84 (95% CI, 0.75–0.93), with a reduced risk of death of 16% in LCS group
- Number needed to screen (NNS) depends on population and follow-up:
About 323 in NLST over 6.5 years and 130 in NELSON over 10 years
- Benefit is present only in people at high baseline risk

USPSTF updated evidence review 2021

Hoffman et al., J Gen Intern Med 2020;35:3015

DeKoning et al., NEJM 2020;382:503

Overall evidence: Mortality benefit is consistent

USPSTF evidence review:

- *Screening lower-risk people yielded fewer prevented deaths and had more false positives per death prevented!*

USPSTF updated evidence review 2021

Hoffman et al., J Gen Intern Med 2020;35:3015

DeKoning et al., NEJM 2020;382:503

LCS: Harms and limitations

False-positive findings can lead to:

- Repeat imaging
- PET/CT
- Invasive procedures: Bronchoscopy, biopsy, surgery

National Lung Screening Trial (*New England Journal of Medicine* 2011):

- 24.2% of LDCT screening results were positive across 3 rounds
- 96.4% of positive results were false positives under the trial's criteria

National Lung Cancer Screening Trial, NEJM 2011;365:395

USPSTF evidence review 2021

Current U.S. recommendations (2026):

Organization	Age	Smoking history	When to stop screening
USPSTF	50–80	≥20 pack-years; currently smoking or quit ≤15 years	Stop after 15 smoke-free years, or if major health limits curative surgery or life expectancy
ACS	50–80	≥20 pack-years; currently or formerly smoking	Annual LDCT for generally healthy people; ACS no longer uses a 15-year quit cutoff
CMS / Medicare	50–77	≥20 pack-years; currently smoking or quit ≤15 years	Asymptomatic; annual LDCT; counseling/shared decision-making before first scan

USPSTF (2021)

ACS lung cancer screening guideline/current 2026 materials

CMS NCD 210.14

LCS: A practical approach to screening

1. Confirm age and smoking exposure: Calculate pack-years

Pack-years = packs per day × years smoked

2. Determine smoking status and years since quitting
3. Confirm the patient is asymptomatic for lung cancer;
(symptomatic patients need diagnostic evaluation, not screening)
4. Assess health status and ability/willingness to undergo curative treatment
5. Use shared decision-making and arrange annual LDCT if eligible
6. For current smokers, pair screening with smoking-cessation treatment!



Clinical examples:

60-year-old • 1 pack/day × 25 years, currently smoking
USPSTF:

55-year-old • 2 packs/day × 10 years, quit 7 years ago
USPSTF:

68-year-old • 1 pack/day × 20 years, quit 18 years ago
USPSTF:
ACS:

48-year-old • 1 pack/day × 30 years, currently smoking
USPSTF:

Clinical examples:

60-year-old • 1 pack/day × 25 years, currently smoking
USPSTF: **Eligible**



55-year-old • 2 packs/day × 10 years, quit 7 years ago
USPSTF:

68-year-old • 1 pack/day × 20 years, quit 18 years ago
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Clinical examples:

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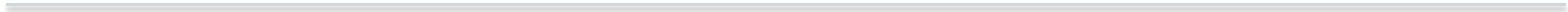


55-year-old • 2 packs/day × 10 years, quit 7 years ago
USPSTF: **Eligible**



68-year-old • 1 pack/day × 20 years, quit 18 years ago
USPSTF:
ACS:

48-year-old • 1 pack/day × 30 years, currently smoking
USPSTF:



Clinical examples:

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USPSTF:



55-year-old • 2 packs/day × 10 years, quit 7 years ago
USPSTF:



68-year-old • 1 pack/day × 20 years, quit 18 years ago
USPSTF: Not eligible
ACS:



48-year-old • 1 pack/day × 30 years, currently smoking
USPSTF:

Clinical examples:

60-year-old • 1 pack/day × 25 years, currently smoking

USPSTF:



55-year-old • 2 packs/day × 10 years, quit 7 years ago

USPSTF:



68-year-old • 1 pack/day × 20 years, quit 18 years ago

USPSTF: **Not eligible**

ACS: **Eligible**



48-year-old • 1 pack/day × 30 years, currently smoking

USPSTF:

Clinical examples:

60-year-old • 1 pack/day × 25 years, currently smoking
USPSTF:



55-year-old • 2 packs/day × 10 years, quit 7 years ago
USPSTF:



68-year-old • 1 pack/day × 20 years, quit 18 years ago
USPSTF:
ACS:



48-year-old • 1 pack/day × 30 years, currently smoking
USPSTF: **Not eligible yet due to age**



LCS: Take-away messages

- Annual LDCT reduces lung-cancer mortality in appropriately selected high-risk adults
 - **US Preventive Services Task Force:**
Age 50–80 + ≥ 20 pack-years + currently smoking or quit ≤ 15 years
 - **American Cancer Society:**
Age 50–80 + ≥ 20 pack-years, currently/formerly smoking, no quit-year cutoff
 - **Centers for Medicare/Medicaid Services :**
Age 50–77 + ≥ 20 pack-years + currently smoking or quit ≤ 15 years
- Screening works best as a program:
Shared decision-making, standardized nodule follow-up, annual adherence,
and smoking cessation



LCS: Useful references

1. Screening for Lung Cancer: US Preventive Services Task Force Recommendation Statement. JAMA 2021;325:962
 2. National Lung Screening Trial Research Team: Reduced lung-cancer mortality with low-dose CT screening. NEJM 2011;365:395
 4. De Koning et al. Reduced lung-cancer mortality with volume CT Screening in a randomized trial. NEJM 2020; 382: 503
 5. American Cancer Society Lung Cancer Screening Guidelines: Cancer facts and figures 2026
 6. Bandi et al. Lung cancer screening in the US. JAMA Intern Med 2022; 184: 882
 7. Silvestri et al. Outcomes from more than 1 million people screened for lung cancer with low-dose CT imaging. Chest 2023; 164:241
 8. Sadate et al. Systematic review and meta-analysis on the impact of lung cancer screening by low-dose computed tomography. Eur J Cancer 2020; 134:107
-

Boise VA Medical Center



Lung Cancer Screening A Team Approach

Dr. Michael Citrone

Low Dose CT Technique

- One breath-hold (thoracic motion is problematic);
- Thin image thicknesses (≤ 2.5 mm, ≤ 1.0 mm preferred); reconstruction of coronal and sagittal reformations as well as MIPS may be helpful and are encouraged.
- CTDIvol < 3.0 mGy for a standard sized patient with adjustments made for smaller and larger patients.
- Typically requires a 16 detector row (or greater) scanner to meet these requirements.
- No IV contrast.
- AI for nodule detection.

Lung Cancer Screening

 American College of Radiology™		Lung-RADS® v2022	Release Date: November 2022
Lung-RADS	Category Descriptor	Findings	Management
0	Incomplete Estimated Population Prevalence: ~ 1%	Prior chest CT examination being located for comparison (see note 9) Part or all lung(s) cannot be evaluated Findings suggestive of an inflammatory or infectious process (see note 10)	Comparison to prior chest CT; Additional lung cancer screening CT imaging needed; 1-3 month LDCT
1	Negative Estimated Population Prevalence: 39%	No lung nodules OR Nodule with benign features: - Complete, central, popcorn, or concentric ring calcifications OR - Fat-containing	12-month screening LDCT
2	Benign - Based on imaging features or indolent behavior Estimated Population Prevalence: 45%	Juxtapleural nodule: - < 10 mm (524 mm³) mean diameter at baseline or now AND - Solid; smooth margins; and oval, lentiform, or triangular shape Solid nodule: - < 6 mm (< 113 mm³) at baseline OR - Now < 4 mm (< 34 mm³) Part solid nodule: - < 6 mm total mean diameter (< 113 mm³) at baseline Non solid nodule (GGN): - < 30 mm (< 14,037 mm³) at baseline, now, or growing OR - > 30 mm (> 14,037 mm³) stable or slowly growing (see note 7) Airway nodule, subsegmental - at baseline, now, or stable (see note 11) Category 3 lesion that is stable or decreased in size at 6-month follow-up CT OR Category 4B lesion proven to be benign in etiology following appropriate diagnostic workup	
3	Probably Benign - Based on imaging features or behavior Estimated Population Prevalence: 9%	Solid nodule: - > 6 to < 8 mm (> 113 to < 268 mm³) at baseline OR - Now 4 mm to < 6 mm (34 to < 113 mm³) Part solid nodule: - > 6 mm total mean diameter (> 113 mm³) with solid component < 6 mm (< 113 mm³) at baseline OR - Now < 6 mm total mean diameter (< 113 mm³) Non solid nodule (GGN): - > 30 mm (> 14,037 mm³) at baseline or now Atypical pulmonary cyst: (see note 12) - Growing cystic component (mean diameter) of a thick-walled cyst Category 4A lesion that is stable or decreased in size at 3-month follow-up CT (excluding airway nodules)	
4A	Suspicious Estimated Population Prevalence: 4%	Solid nodule: - > 8 to < 15 mm (> 268 to < 1,767 mm³) at baseline OR - Growing < 8 mm (< 268 mm³) OR - Now 6 to < 8 mm (113 to < 268 mm³) Part solid nodule: - > 6 mm total mean diameter (> 113 mm³) with solid component > 6 mm to < 8 mm (> 113 to < 268 mm³) at baseline OR - Now or growing < 4 mm (< 34 mm³) solid component Airway nodule, segmental or more proximal - at baseline (see note 11) Atypical pulmonary cyst: (see note 12) - Thick-walled cyst OR - Multilocular cyst at baseline OR - Thin- or thick-walled cyst that becomes multilocular	
4B	Very Suspicious Estimated Population Prevalence: 2%	Airway nodule, segmental or more proximal - stable or growing (see note 11) Solid nodule: - > 15 mm (> 1,767 mm³) at baseline OR - Now or growing > 8 mm (> 268 mm³) Part solid nodule: - Solid component > 8 mm (> 268 mm³) at baseline OR - Now or growing > 4 mm (> 34 mm³) solid component Atypical pulmonary cyst: (see note 12) - Thick-walled cyst with growing wall thickness/nodularity OR - Growing multilocular cyst (mean diameter) OR - Multilocular cyst with increased loculation or now/increased opacity (nodular, ground glass, or consolidation) Slow growing solid or part solid nodule that demonstrates growth over multiple screening exams (see note 8)	Referral for further clinical evaluation Diagnostic chest CT with or without contrast; PET/CT may be considered if there is a > 8 mm (> 268 mm³) solid nodule or solid component; tissue sampling; and/or referral for further clinical evaluation Management depends on clinical evaluation, patient preference, and the probability of malignancy (see note 13)
4X	Estimated Population Prevalence: < 1%	Category 3 or 4 nodules with additional features or imaging findings that increase suspicion for lung cancer (see note 14)	
S	Significant or Potentially Significant Estimated Population Prevalence: 10%	Modifier: May add to category 0-4 for clinically significant or potentially clinically significant findings unrelated to lung cancer (see note 15)	As appropriate to the specific finding

Nodule Interpretation

- benign – calcified; fat; or $< 4\text{mm}$ micronodule
- abnormal - $> 10\text{mm}$, enlarging $> 7\text{mm}$
- indeterminate - $4 - 10\text{mm}$; enlarging $< 7\text{mm}$

Lung Cancer Screening: Interpretation

- **Negative screen**
No significant abnormalities
- **Negative screen, minor abnormalities not suspicious for lung cancer**
Benign nodules, micronodules, atelectasis/scar, coronary artery calcification (?)
- **Negative screen, significant abnormalities not suspicious for lung cancer**
Aortic aneurysm, mediastinal/thyroid mass, pericardial/pleural effusions, axillary adenopathy, chest wall lesion, spine lesion

Lung Cancer Screening: Interpretation

- Positive screen

Nodule 4-10mm or enlarging nodule

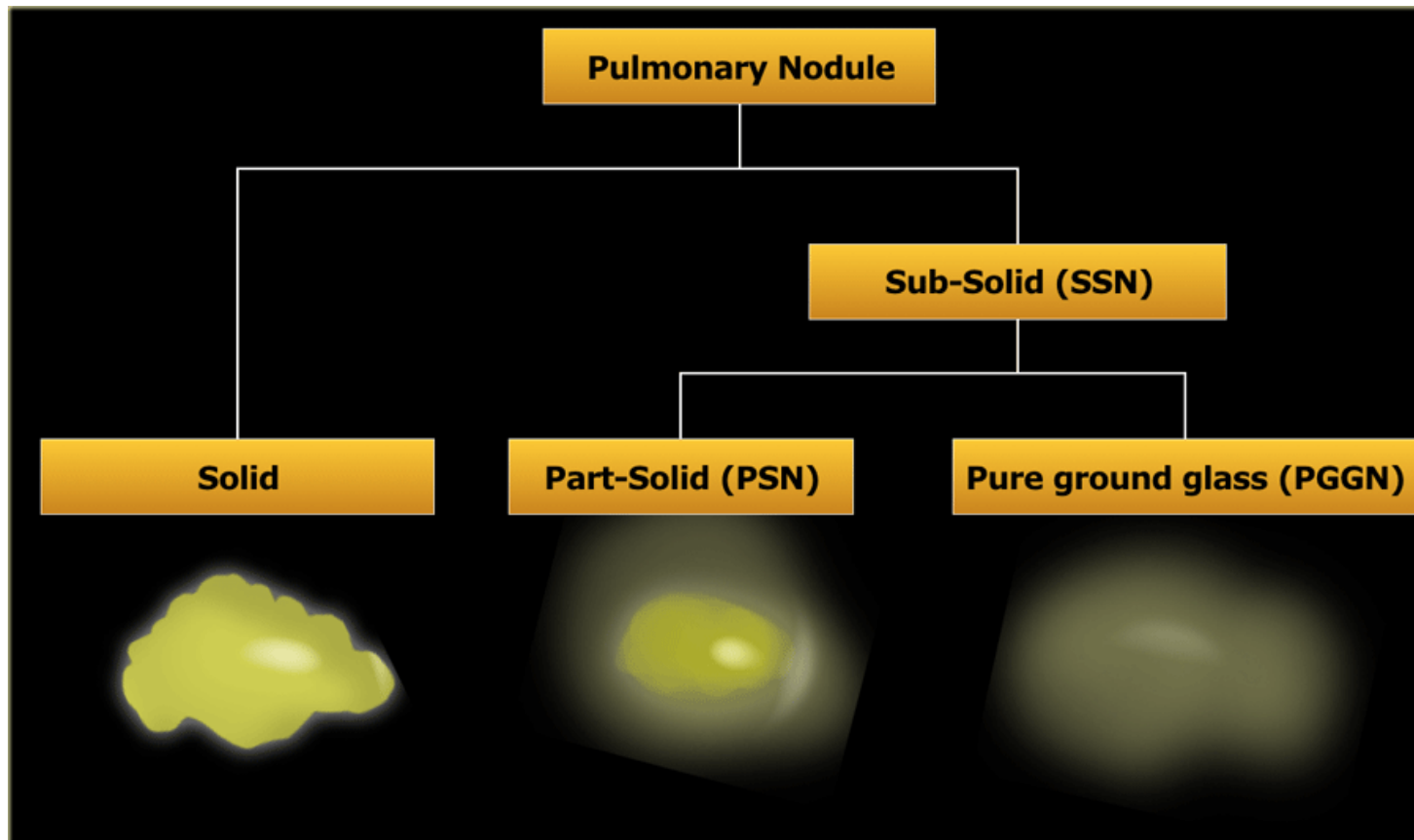
- Positive screen

Nodule >10mm, enlarging nodule ≥ 7 mm,
lung mass, other non-specific abnormality
suspicious for lung cancer

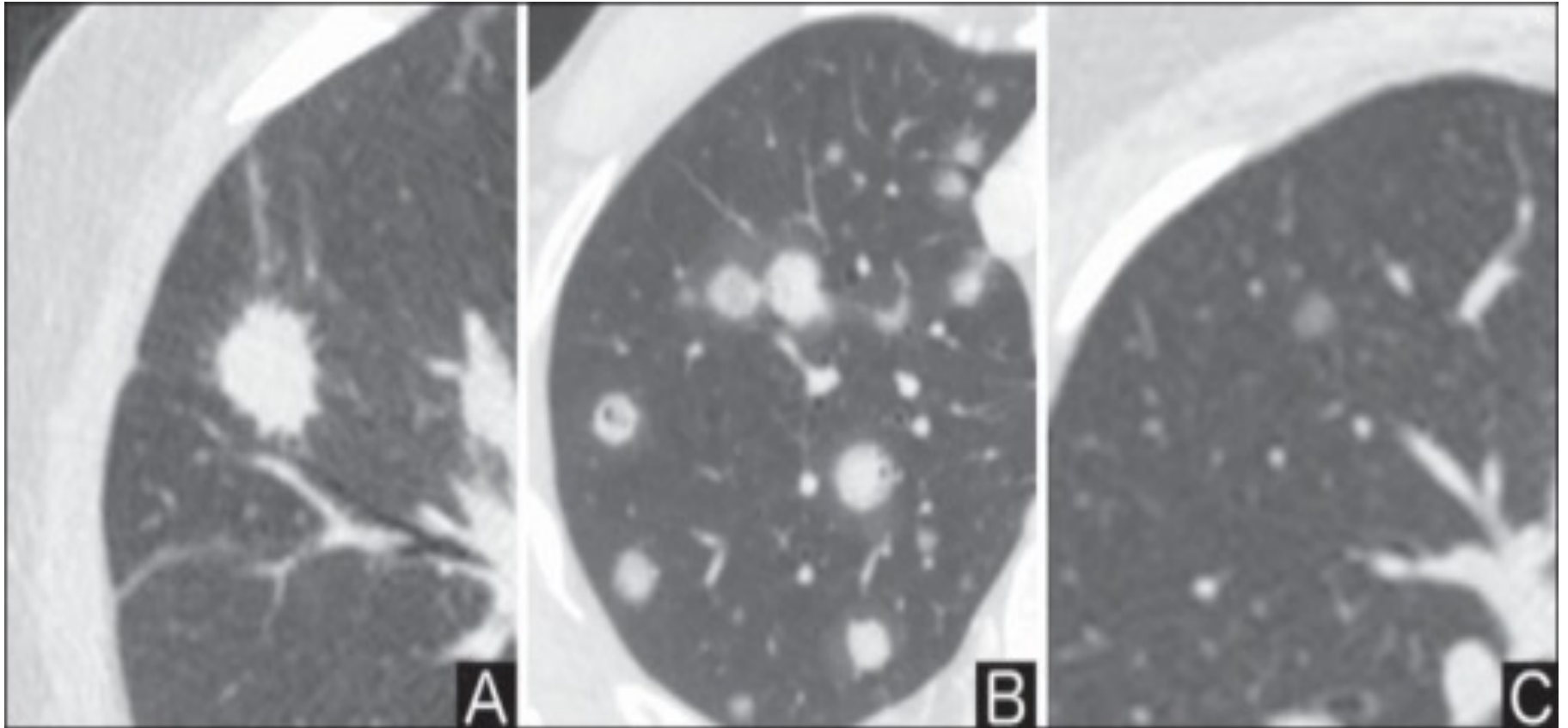
Lung Cancer Screening: Potential Recommendations

- No intervention – continue screening
- Comparison with historical
- Thin section CT: 3, 6, 12 months
- Diagnostic CT
- CT nodule densitometry
- PET
- Biopsy

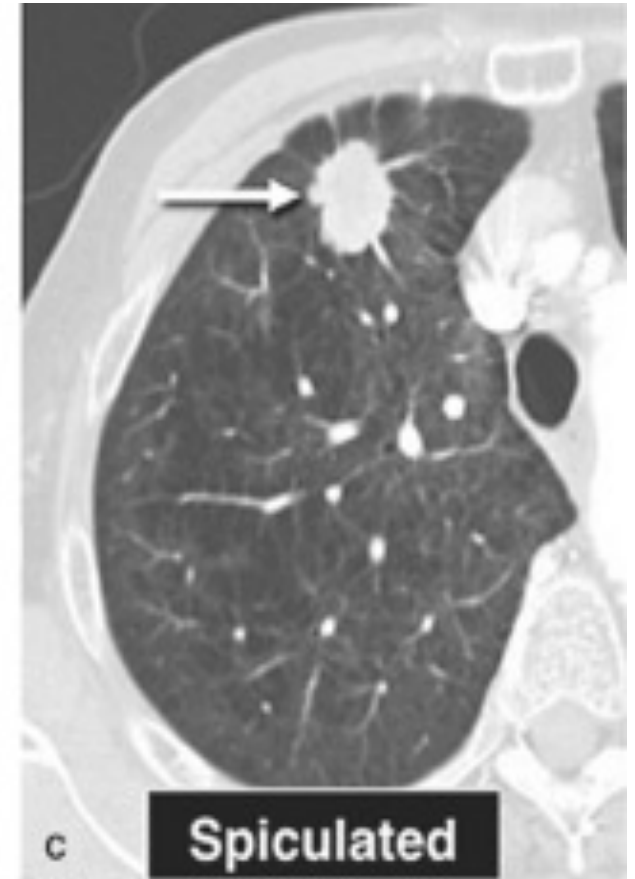
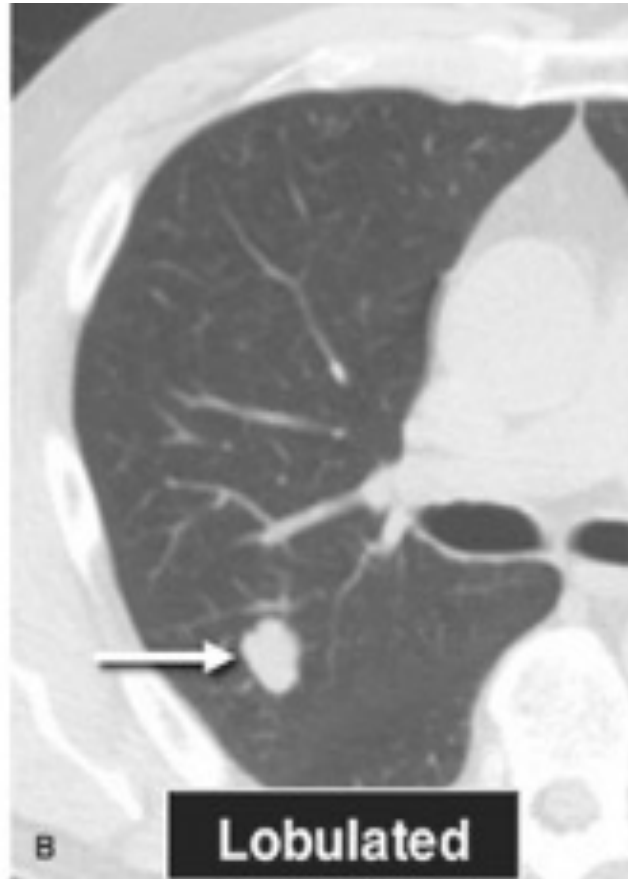
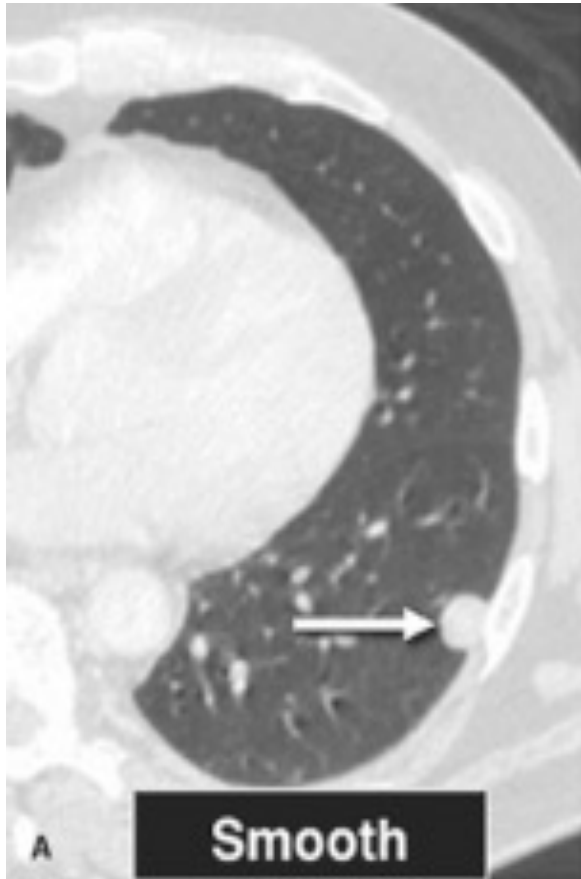
Types of Nodules



Types of Nodules

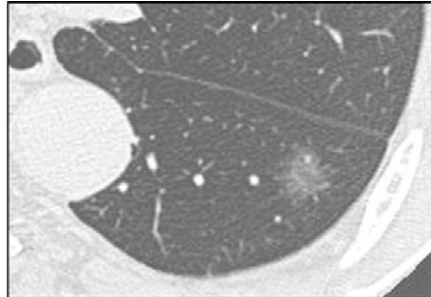


Types of Nodule Borders

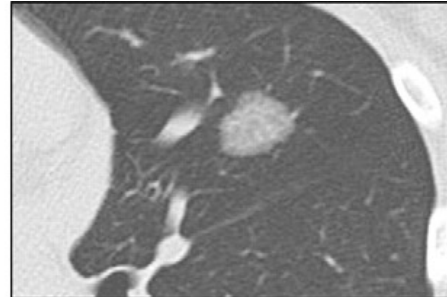


Types of Nodules

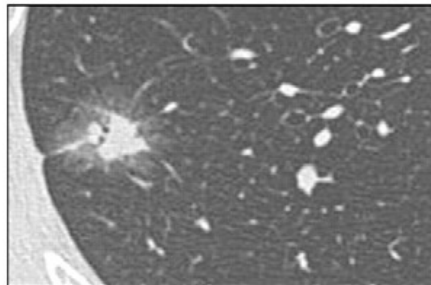
Type 1
Homogeneous
Pure GGO



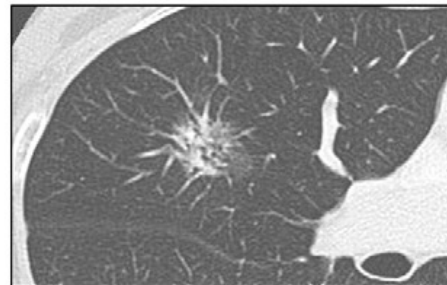
Type 2
Homogeneous
Semi-consolidation



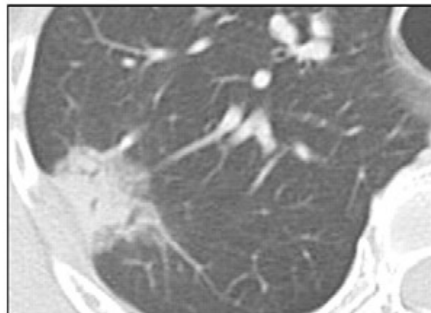
Type 3
Heterogeneous
Halo (>50% GGO)



Type 4
Heterogeneous
Mixed (>50% GGO)



Type 5
Solid / GGO
(<50% GGO)



Type 6
Pure Solid

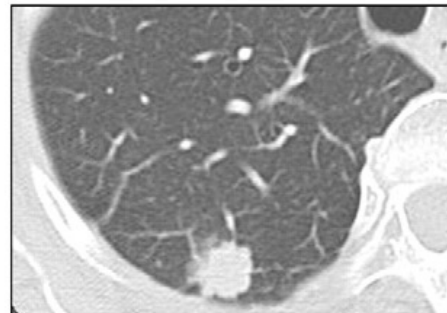
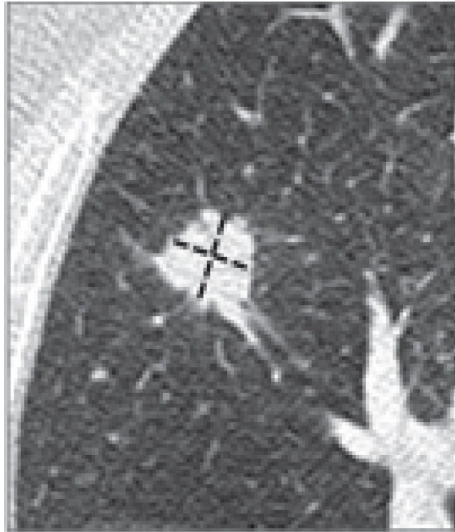


Fig. 1 Suzuki classification of GGOs. Radiographic classification of lung nodules according to the ground glass

Measurement of Nodules

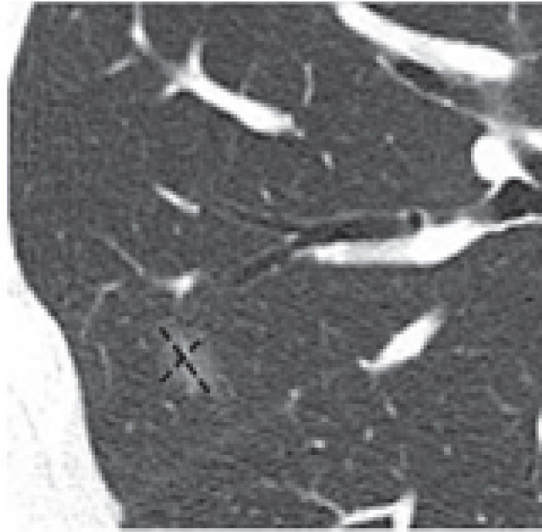
MANUAL MEASUREMENT OF THE DIFFERENT TYPES OF NODULES

SOLID



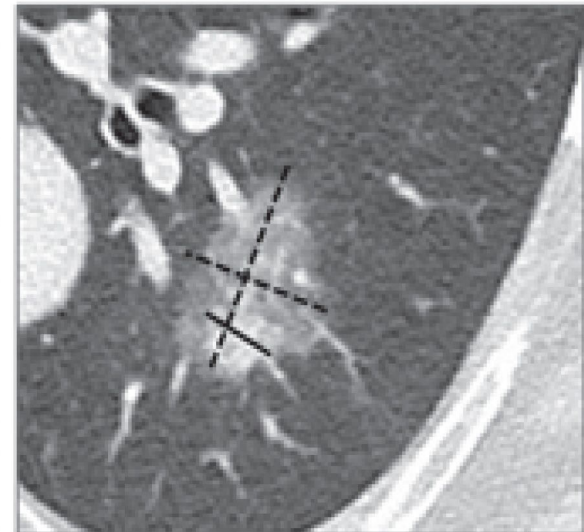
$8.6 + 8.5 = 17.1 / 2 = 8.5$
Nodule measuring 9 mm

NONSOLID



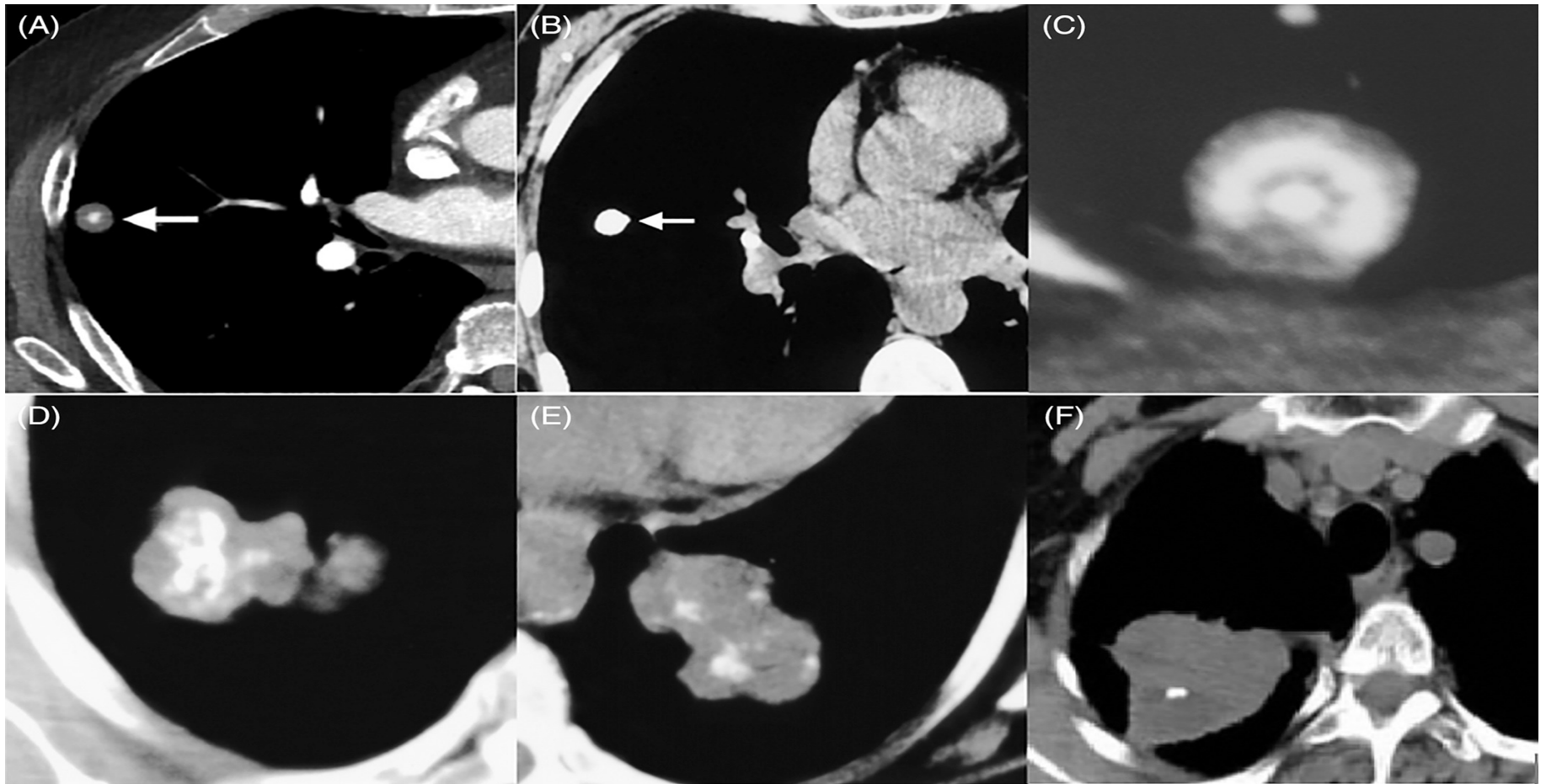
$8 + 6.6 = 14.6 / 2 = 7.3$
Nodule measuring 7 mm

PART-SOLID



Nodule measuring 26 x 18 mm
Solid component measuring 11 mm

Calcification within Nodules

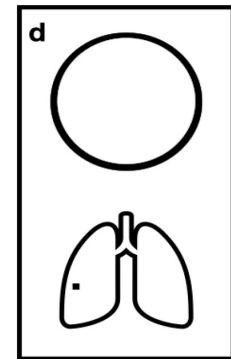
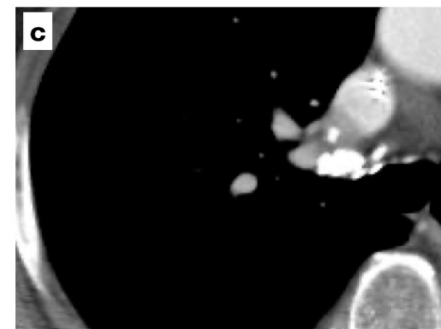
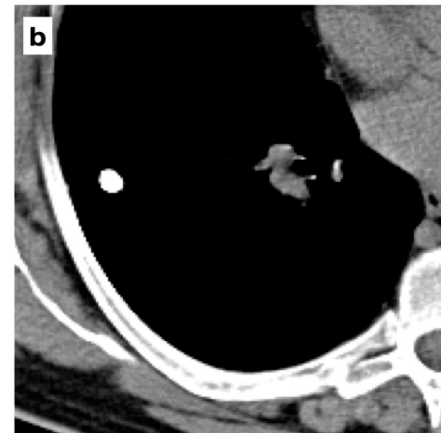


Lung RADS v2022

Lung-RADS	Category Descriptor	Findings	Management
1	Negative Estimated Population Prevalence: 39%	No lung nodules OR Nodule with benign features: • Complete, central, popcorn, or concentric ring calcifications OR • Fat-containing	12-month screening LDCT
2	Benign - Based on imaging features or indolent behavior Estimated Population Prevalence: 45%	Juxtapleural nodule: • < 10 mm (524 mm³) mean diameter at baseline or new AND • Solid; smooth margins; and oval, lentiform, or triangular shape	
		Solid nodule: • < 6 mm (< 113 mm³) at baseline OR • New < 4 mm (< 34 mm³)	
		Part solid nodule: • < 6 mm total mean diameter (< 113 mm³) at baseline	
		Non solid nodule (GGN): • < 30 mm (< 14,137 mm³) at baseline, new, or growing OR • ≥ 30 mm (≥ 14,137 mm³) stable or slowly growing (see note 7)	
		Airway nodule , subsegmental - at baseline, new, or stable (see note 11)	
		Category 3 lesion that is stable or decreased in size at 6-month follow-up CT OR Category 4B lesion proven to be benign in etiology following appropriate diagnostic workup	

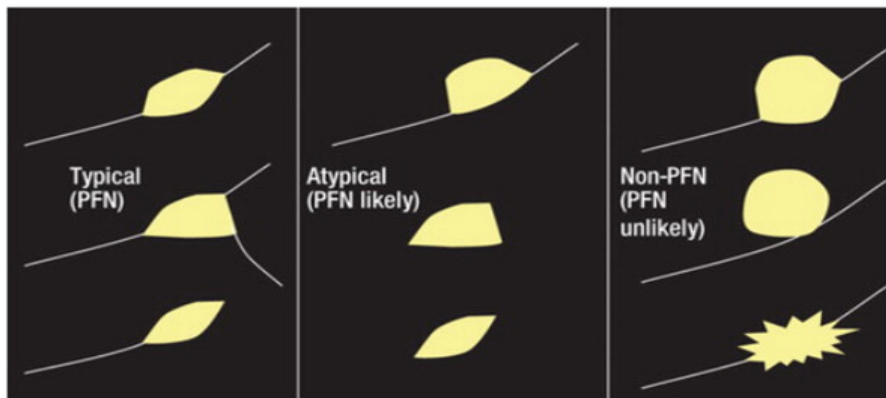
Lung RADS 1

- Calcified nodule (LR-1)



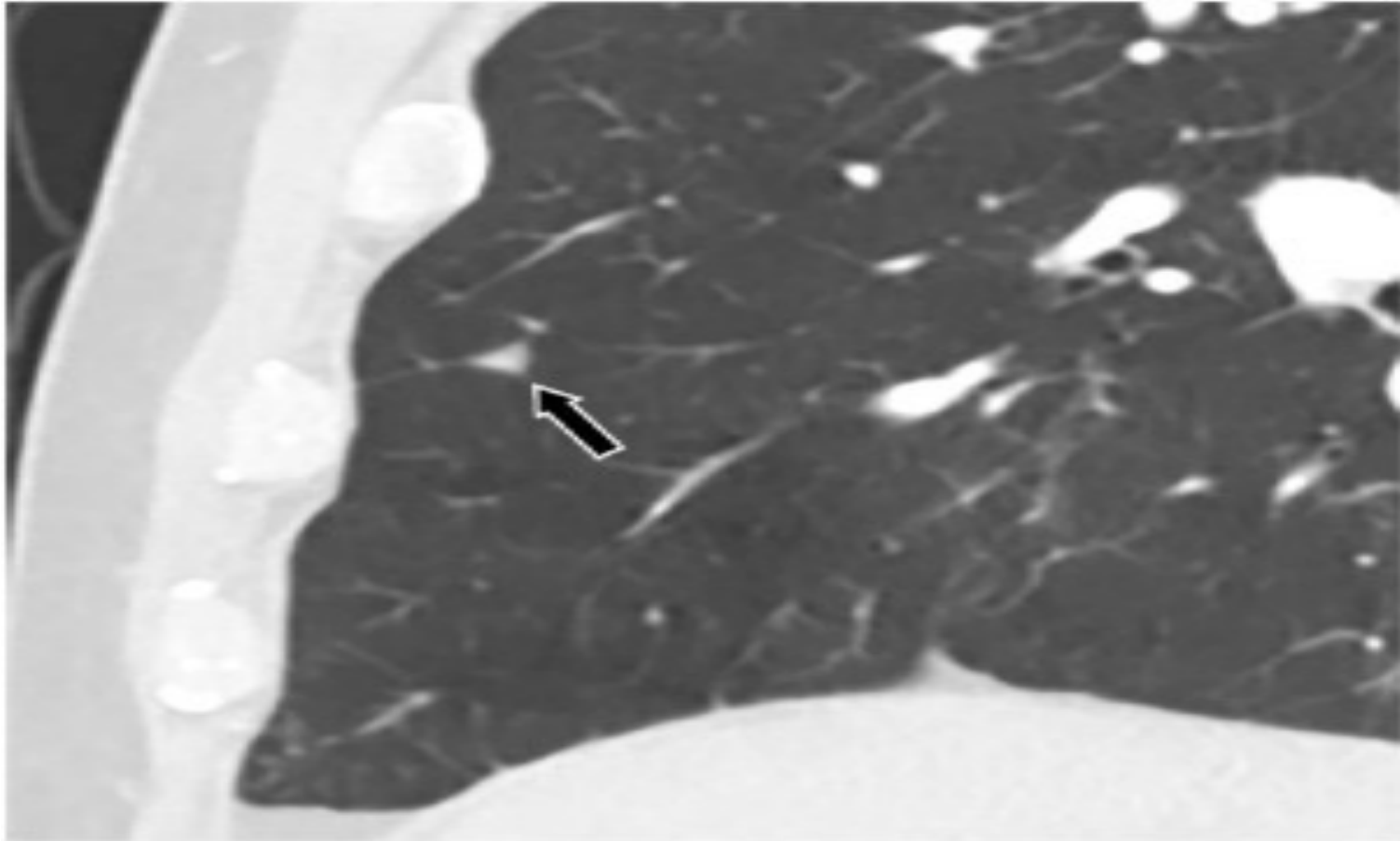
Lung RADS 2: Fissural Lymph Node

How I Do It: Management of Pleural-attached Pulmonary Nodules in Low-Dose CT Screening for Lung Cancer



- Minimizing workup for benign nodules is critical to the success of low-dose CT screening for lung cancer.
- Pulmonary nodules previously defined as “perifissural” less than 10 mm in average diameter with specific morphologic features are now recognized as having a high likelihood of being benign.

Lung RADS 2: Fissural Lymph Node

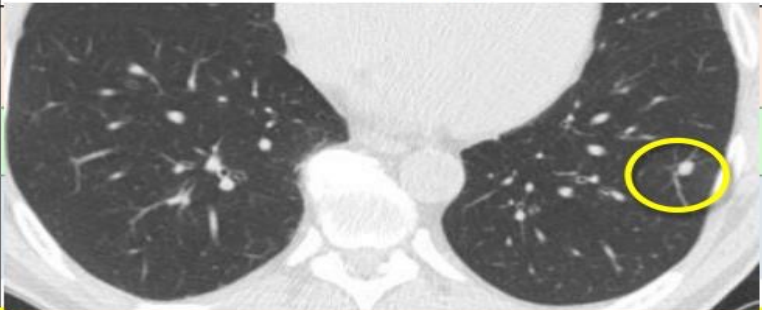


Lung RADS v2022

Lung-RADS	Category Descriptor	Findings	Management
3	Probably Benign - Based on imaging features or behavior Estimated Population Prevalence: 9%	Solid nodule: • ≥ 6 to < 8 mm (≥ 113 to < 268 mm³) at baseline OR • New 4 mm to < 6 mm (34 to < 113 mm³)	6-month LDCT
		Part solid nodule: • ≥ 6 mm total mean diameter (≥ 113 mm³) with solid component < 6 mm (< 113 mm³) at baseline OR • New < 6 mm total mean diameter (< 113 mm³)	
		Non solid nodule (GGN): • ≥ 30 mm ($\geq 14,137$ mm³) at baseline or new	
		Atypical pulmonary cyst: (see note 12) • Growing cystic component (mean diameter) of a thick-walled cyst	
		Category 4A lesion that is stable or decreased in size at 3-month follow-up CT (excluding airway nodules)	

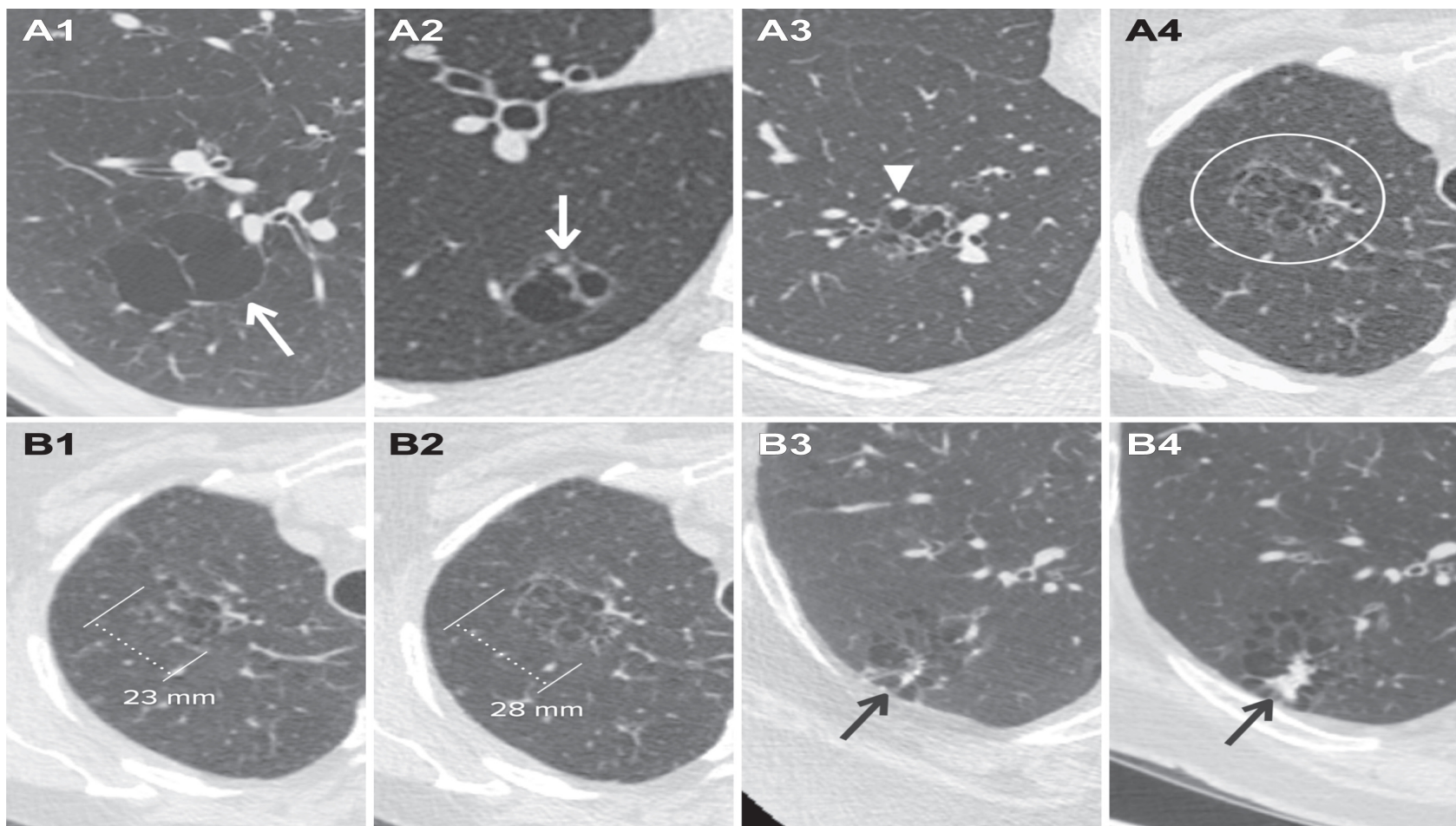
Lung RADS 3: Probably Benign

Assessment Categories: (+) CT screen

Category	Category Descriptor	Category	Findings
Benign Appearance or Behavior	Nodules with a very low likelihood of becoming a clinically active cancer due to size or lack of growth	2	
Probably Benign	Probably benign finding(s) - short term follow up suggested; includes nodules with a low likelihood of becoming a clinically active cancer	3	solid nodule(s): ≥ 6 to < 8 mm at baseline OR new 4 mm to < 6 mm part solid nodule(s) ≥ 6 mm total diameter with solid component < 6 mm OR new < 6 mm total diameter non solid nodule(s) (GGN) ≥ 20 mm on baseline CT or new



Atypical Pulmonary Cyst



Lung RADS v2022

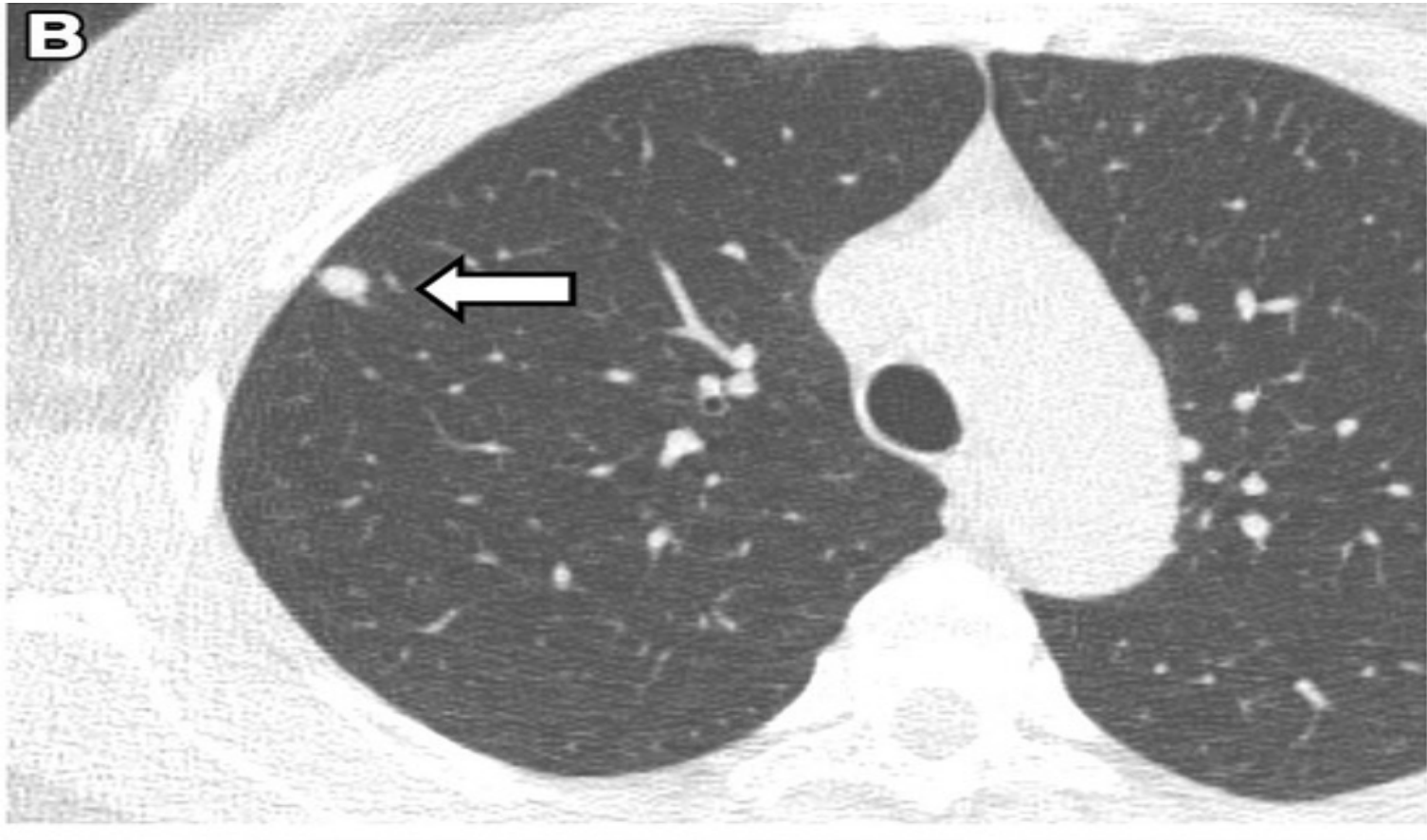
Lung-RADS	Category Descriptor	Findings	Management
4A	Suspicious Estimated Population Prevalence: 4%	Solid nodule: • ≥ 8 to < 15 mm (≥ 268 to $< 1,767$ mm ³) at baseline OR • Growing < 8 mm (< 268 mm ³) OR • New 6 to < 8 mm (113 to < 268 mm ³)	3-month LDCT; PET/CT may be considered if there is a ≥ 8 mm (≥ 268 mm ³) solid nodule or solid component
		Part solid nodule: • ≥ 6 mm total mean diameter (≥ 113 mm ³) with solid component ≥ 6 mm to < 8 mm (≥ 113 to < 268 mm ³) at baseline OR • New or growing < 4 mm (< 34 mm ³) solid component	
		Airway nodule , segmental or more proximal - at baseline (see note 11)	
		Atypical pulmonary cyst: (see note 12) • Thick-walled cyst OR • Multilocular cyst at baseline OR • Thin- or thick-walled cyst that becomes multilocular	
4B	Very Suspicious Estimated Population Prevalence: 2%	Airway nodule , segmental or more proximal - stable or growing (see note 11)	Referral for further clinical evaluation
		Solid nodule: • ≥ 15 mm ($\geq 1,767$ mm ³) at baseline OR • New or growing ≥ 8 mm (≥ 268 mm ³)	Diagnostic chest CT with or without contrast; PET/CT may be considered if there is a ≥ 8 mm (≥ 268 mm ³) solid nodule or solid component; tissue sampling; and/or referral for further clinical evaluation Management depends on clinical evaluation, patient preference, and the probability of malignancy (see note 13)
		Part solid nodule: • Solid component ≥ 8 mm (≥ 268 mm ³) at baseline OR • New or growing ≥ 4 mm (≥ 34 mm ³) solid component	
		Atypical pulmonary cyst: (see note 12) • Thick-walled cyst with growing wall thickness/nodularity OR • Growing multilocular cyst (mean diameter) OR • Multilocular cyst with increased loculation or new/increased opacity (nodular, ground glass, or consolidation)	
		Slow growing solid or part solid nodule that demonstrates growth over multiple screening exams (see note 8)	
4X	Estimated Population Prevalence: $< 1\%$	Category 3 or 4 nodules with additional features or imaging findings that increase suspicion for lung cancer (see note 14)	

Nodule and Other Characteristics for 4X

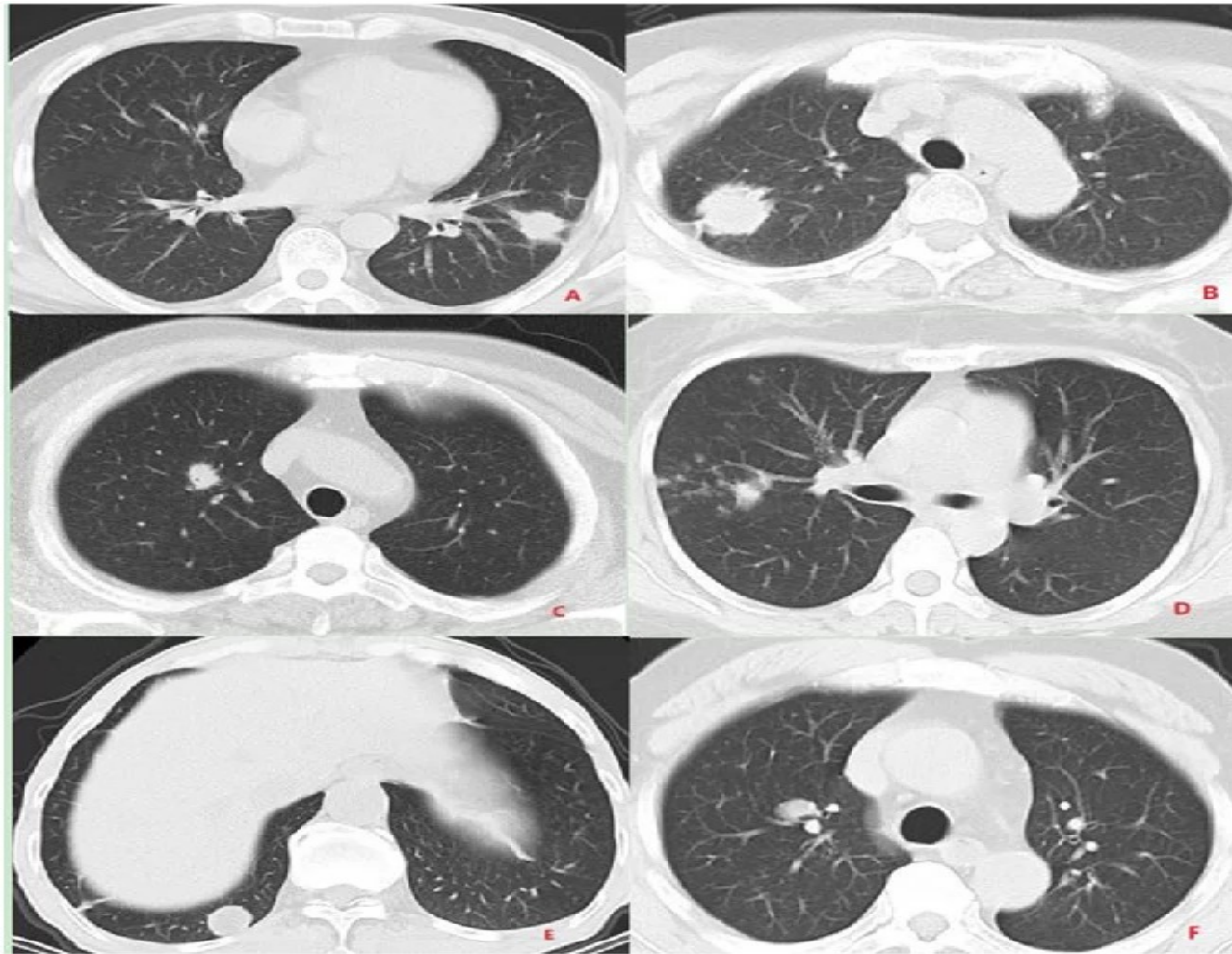
4X	Estimated Population Prevalence: < 1%	Category 3 or 4 nodules with additional features or imaging findings that increase suspicion for lung cancer (see note 14)
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14. Category 4X: Category 3 or 4 nodules with additional imaging findings that increase the suspicion of lung cancer, such as spiculation, lymphadenopathy, frank metastatic disease, a GGN that doubles in size in 1 year, etc. 4X is a distinct Lung-RADS category; X should not be used as a modifier.

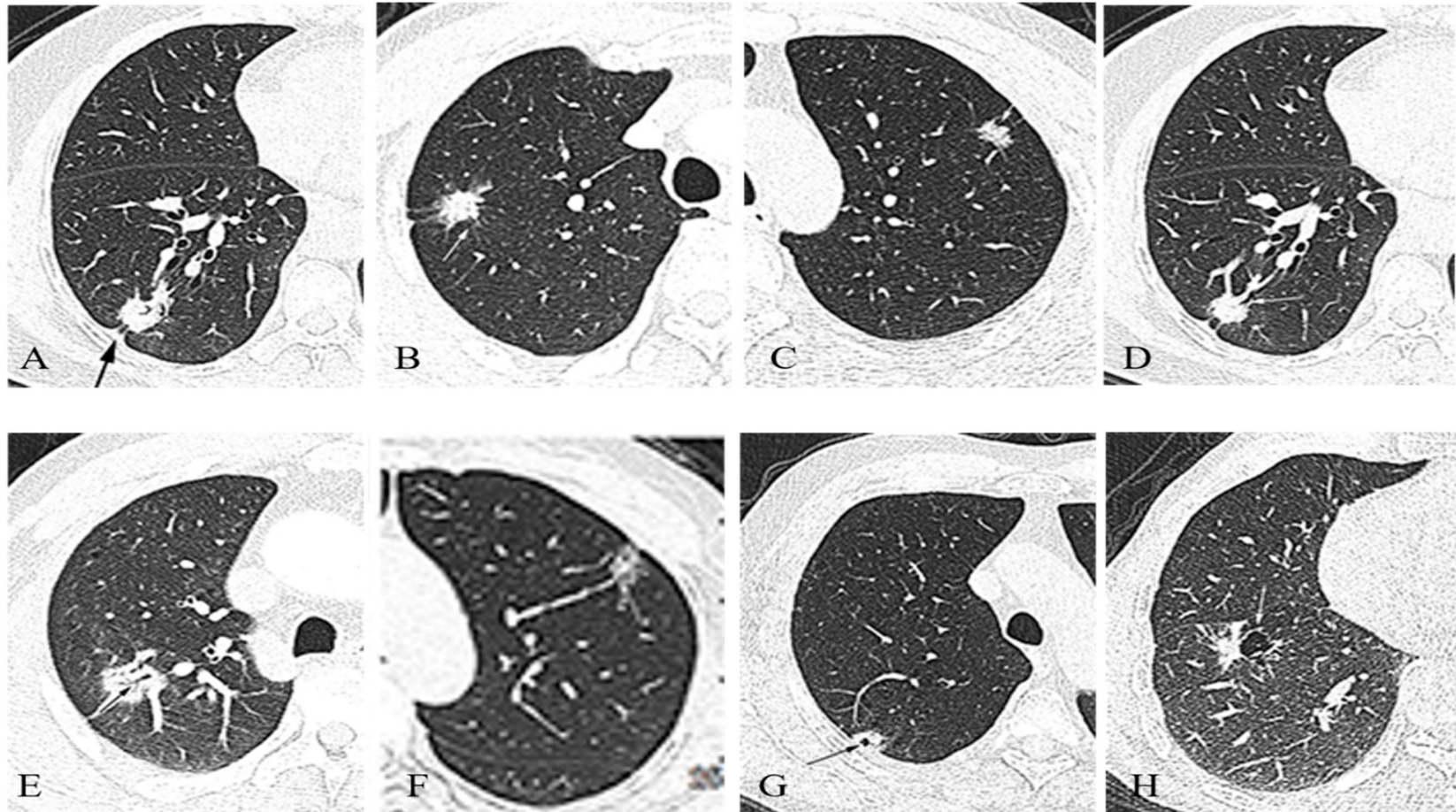
LR 4A Nodule



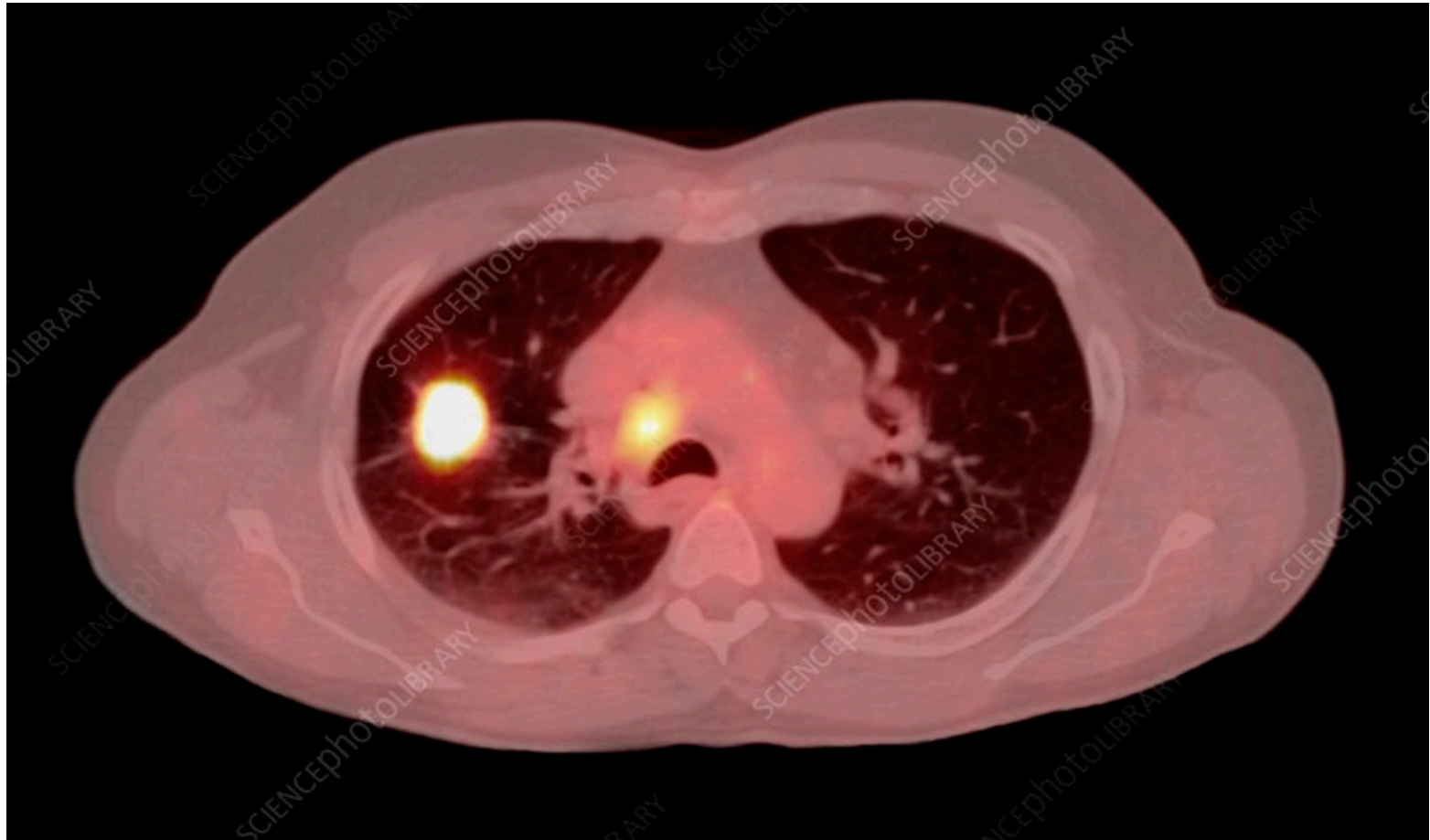
LR 4B and 4X Nodules



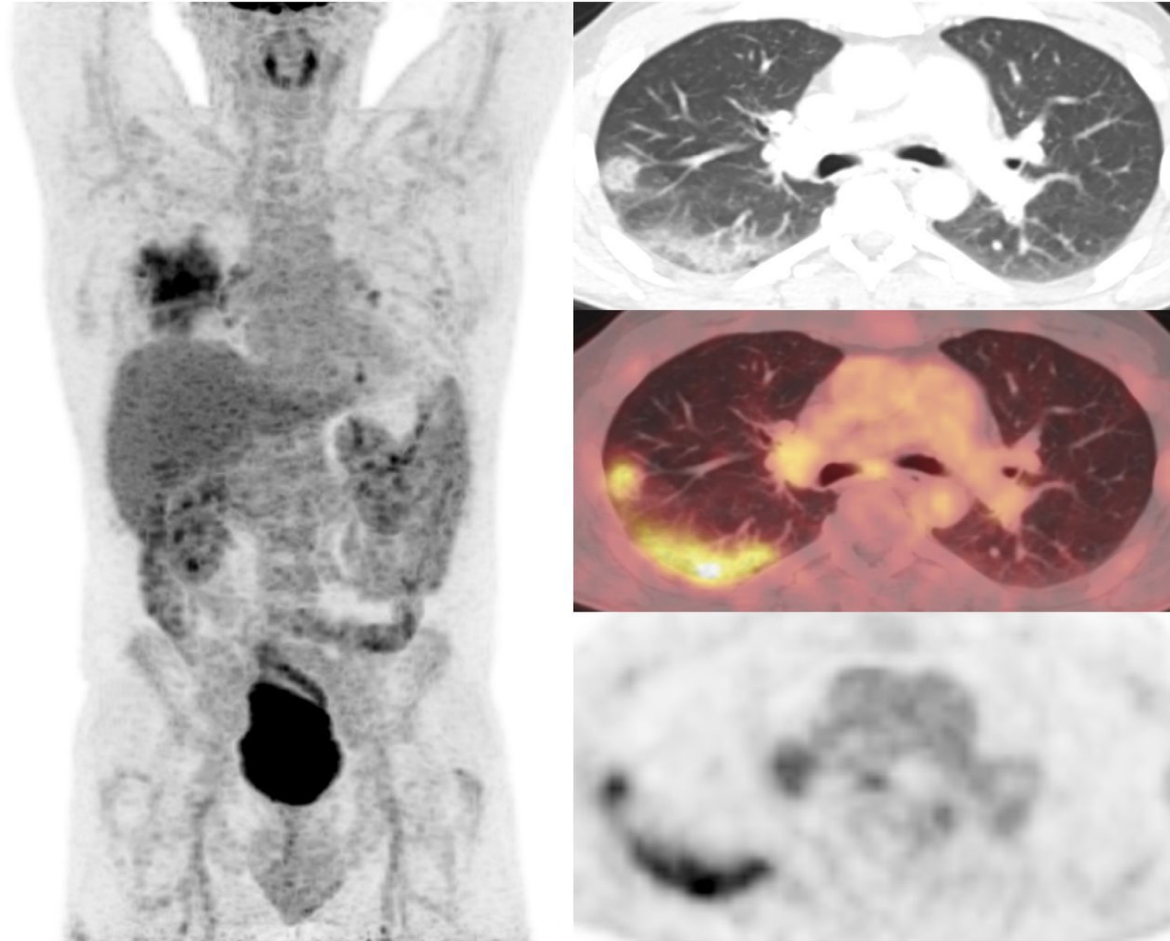
LR 4B and 4X Nodules



PET/CT of a LR 4X Nodule



Multifocal Lung Carcinoma on PET/CT



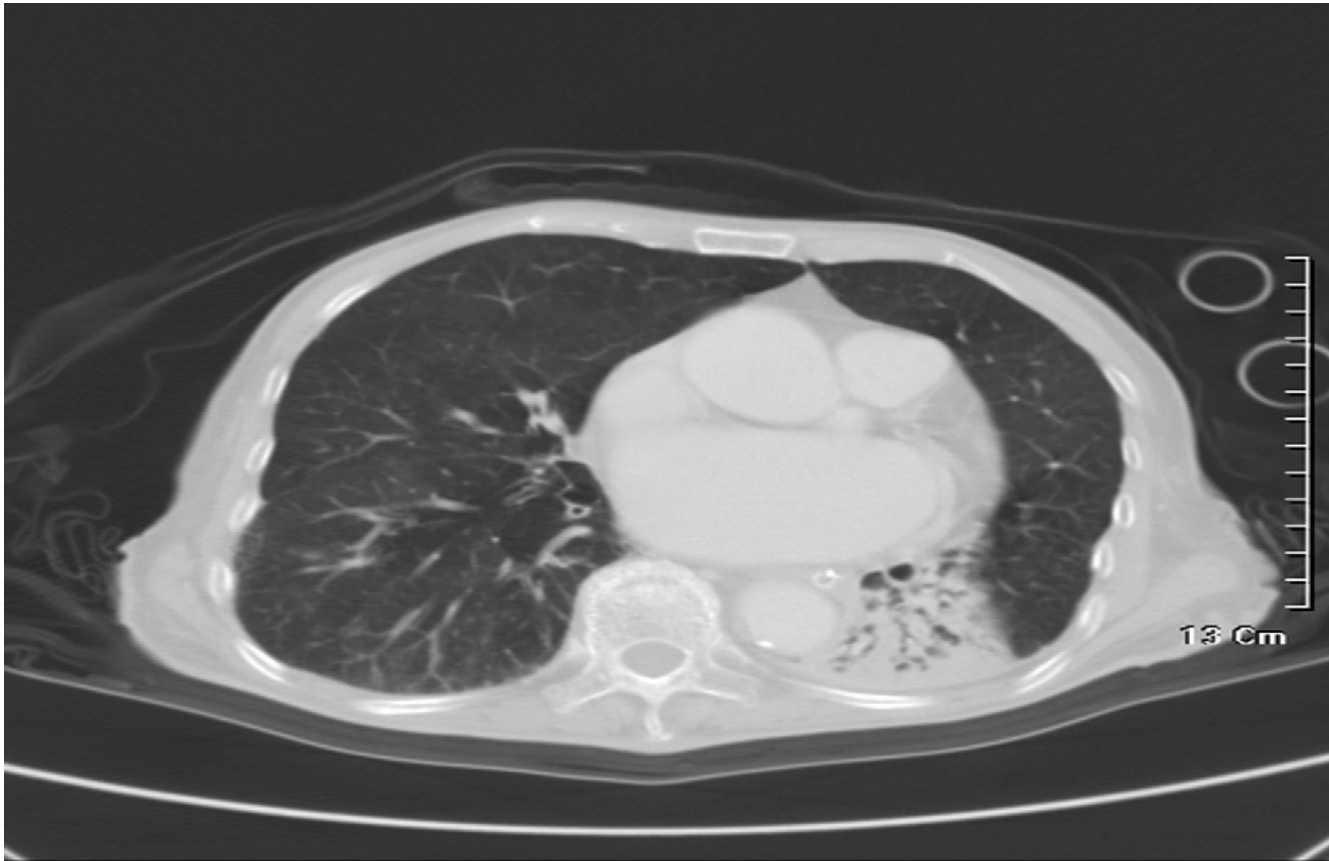
Lung-RADS v2022

Lung-RADS	Category Descriptor	Findings	Management
0	Incomplete Estimated Population Prevalence: ~ 1%	Prior chest CT examination being located for comparison (see note 9)	Comparison to prior chest CT;
		Part or all of lungs cannot be evaluated	Additional lung cancer screening CT imaging needed;
		Findings suggestive of an inflammatory or infectious process (see note 10)	1-3 month LDCT

10. Suspected Infectious or Inflammatory Findings:

- Lung-RADS 0 with 1-3 month follow-up LDCT may be recommended for pulmonary findings suggesting an indeterminate infectious or inflammatory process. Such findings may include segmental or lobar consolidation, multiple new nodules (more than six), large solid nodules (≥ 8 mm) appearing in a short interval, and new nodules in certain clinical contexts (e.g. immunocompromised patient). At 1-3 month follow-up, a new Lung-RADS classification and management recommendation should be provided based on the most suspicious nodule.
- New solid or part solid nodules with imaging features more concerning for malignancy than an infectious or inflammatory process meeting Lung-RADS 4B size criteria may be classified as such with appropriate diagnostic and/or clinical evaluation.
- Some findings indicative of an infectious or inflammatory process may not warrant short-term follow-up (e.g. tree-in-bud nodules or new < 3 cm ground glass nodules). These nodules may be evaluated using existing size criteria with a Lung-RADS classification and management recommendation based on the most suspicious finding.

Pneumonia to include Aspiration



Aspiration Pneumonitis with Tree-in-Bud Pattern

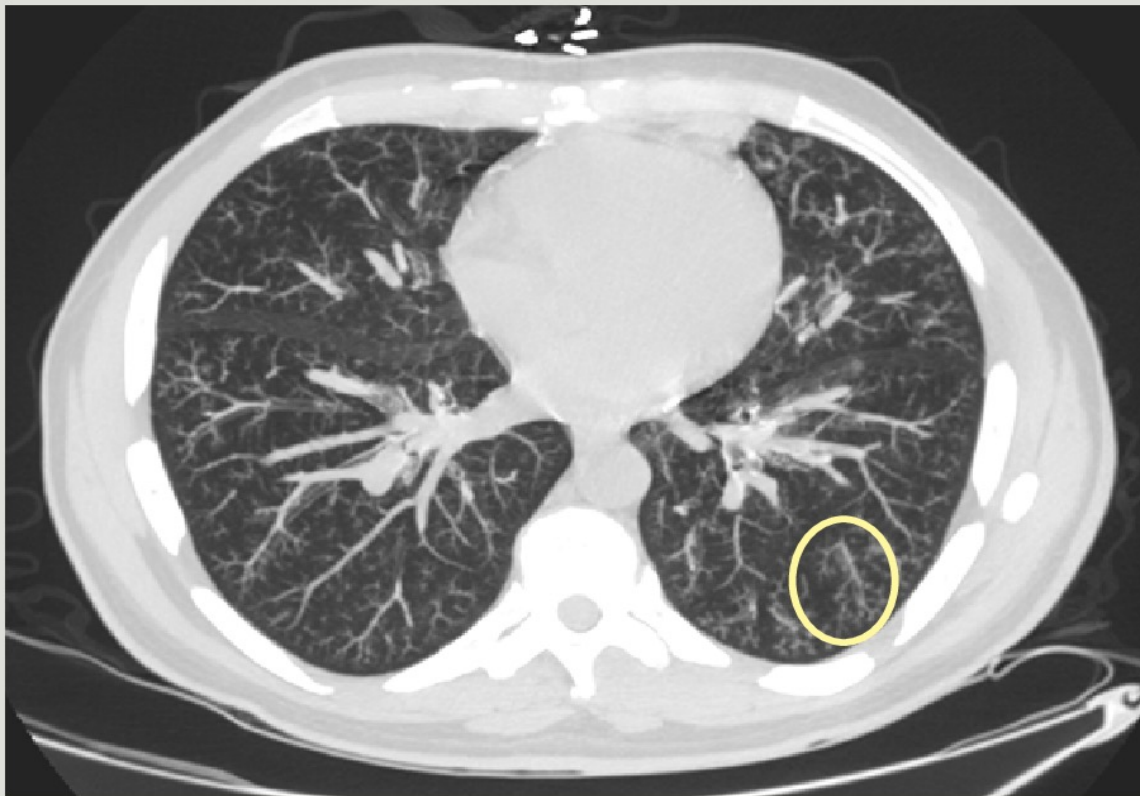


Figure 1: CT scan with tree-in-bud pattern

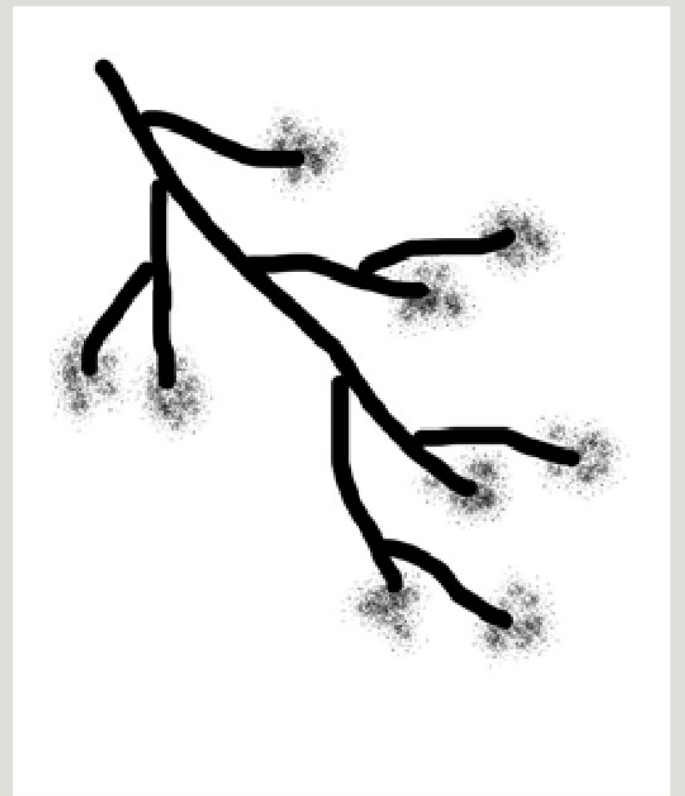


Figure 2: Drawing rendition of tree-in-bud

Hiatal Hernia

Types of hiatal hernias



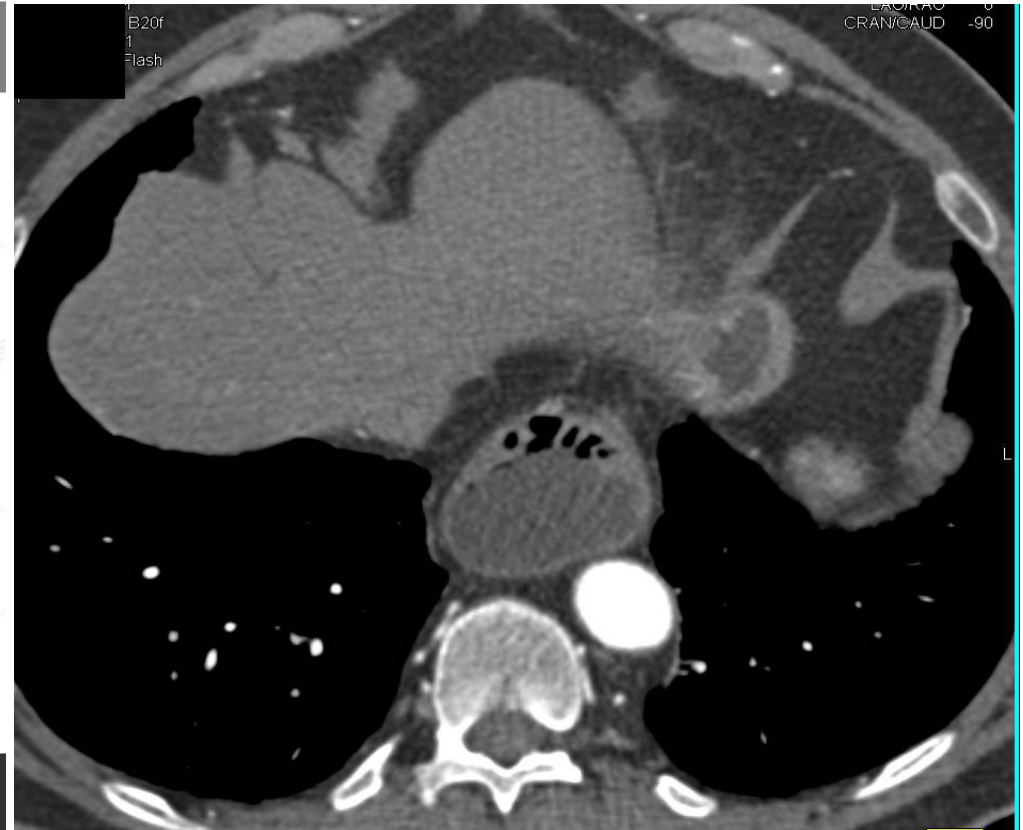
Normal Stomach



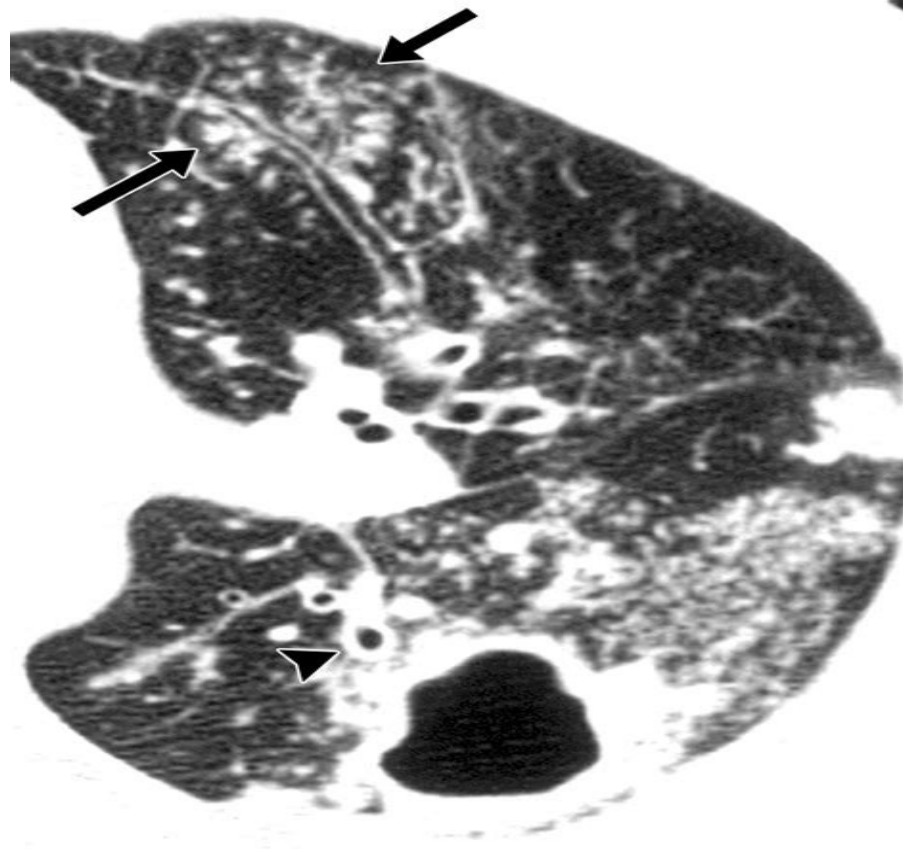
Sliding Hiatal Hernia



Paraesophageal
Hiatal Hernia



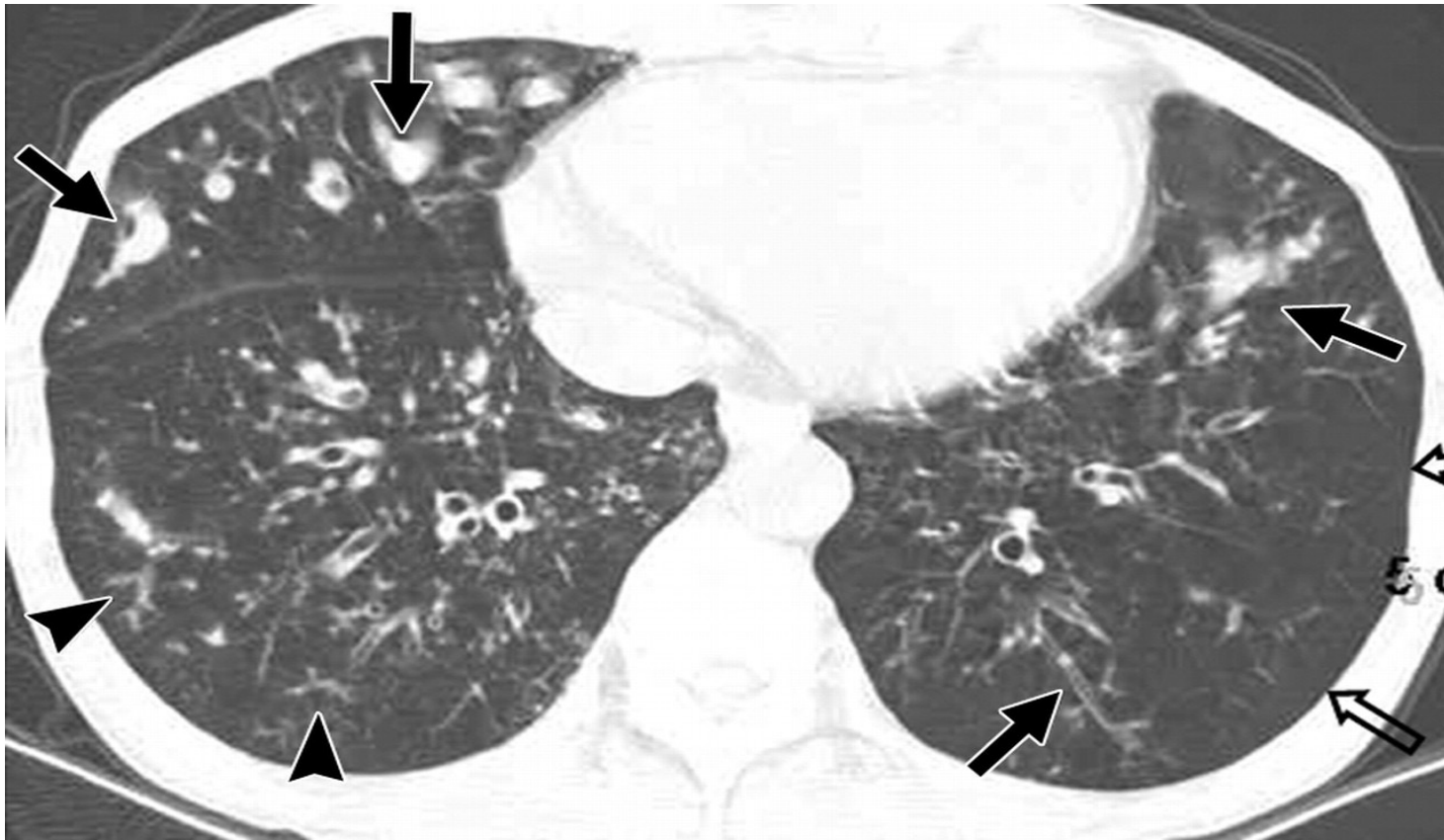
Severe Multifocal Aspiration or Fungal Pneumonia



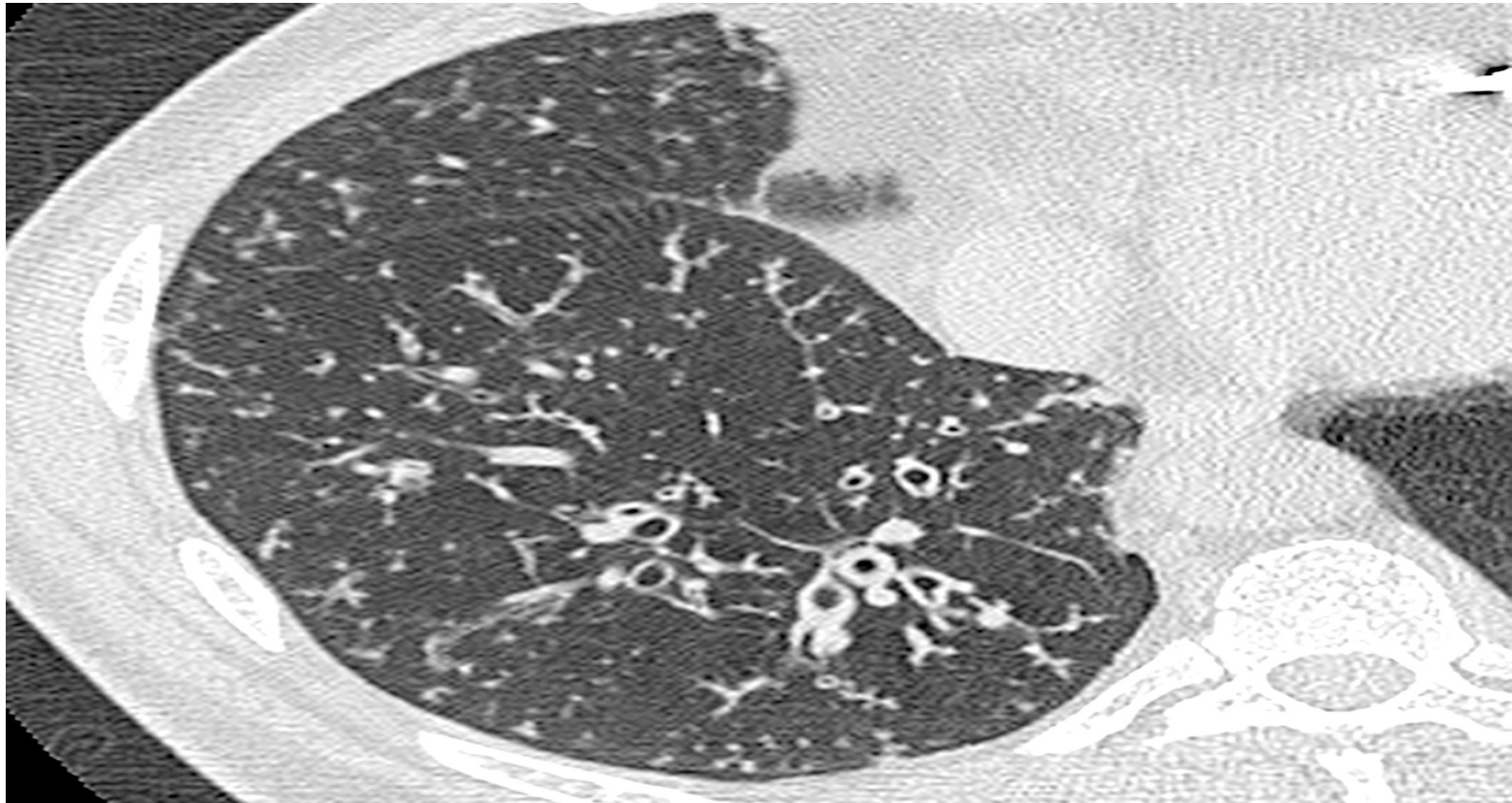
TB Fungal Infection



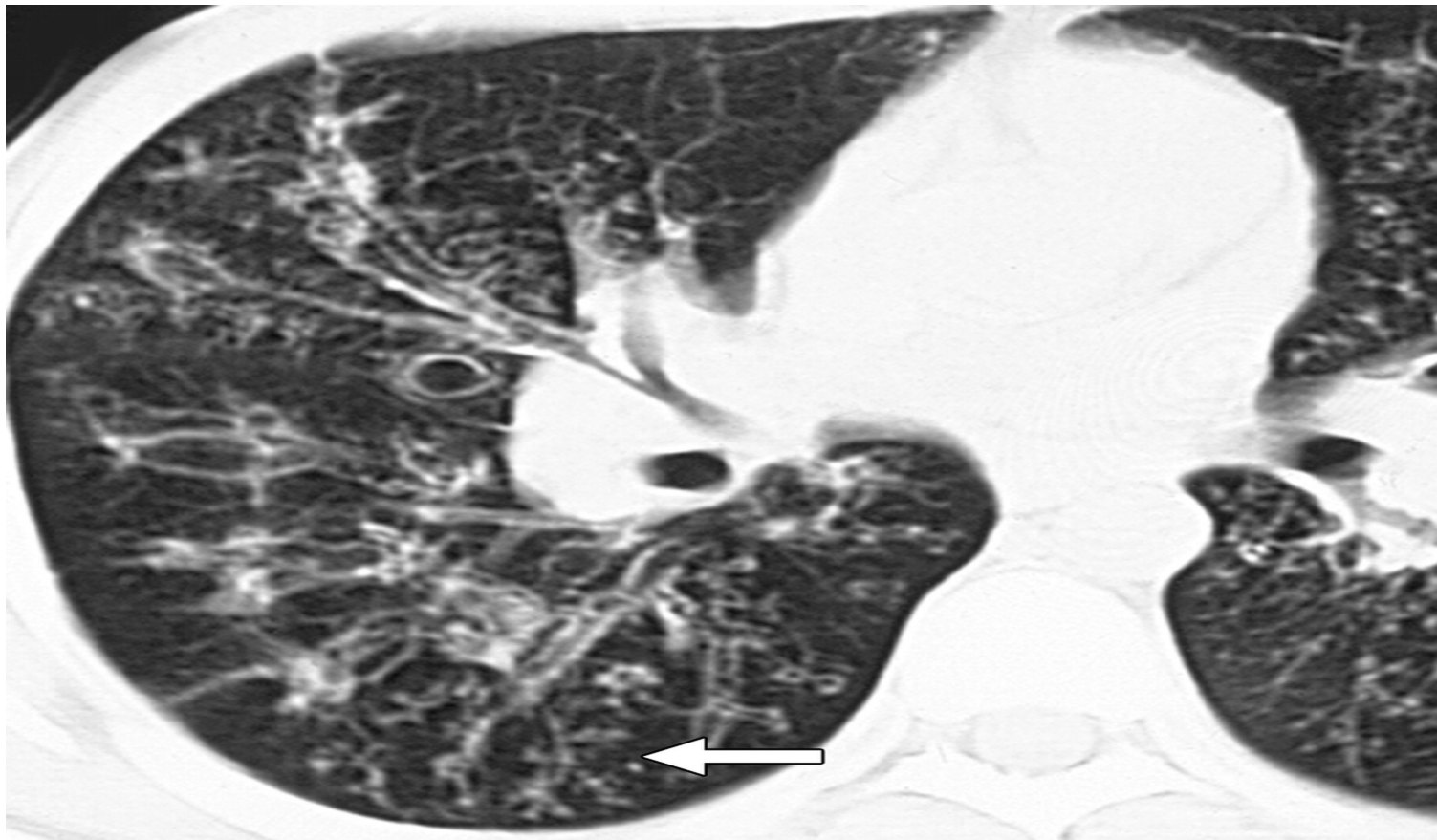
Bronchiectasis, Bronchitis, Mucous Plugging and Inflammatory Bronchiolitis



Bronchiectasis with Probable Inflammatory Bronchiolitis



Probable Both Aspiration and Inflammatory Bronchiolitis



Often Mixed Picture of a Suspicious Appearing Nodule and Multifocal Infection/Inflammation

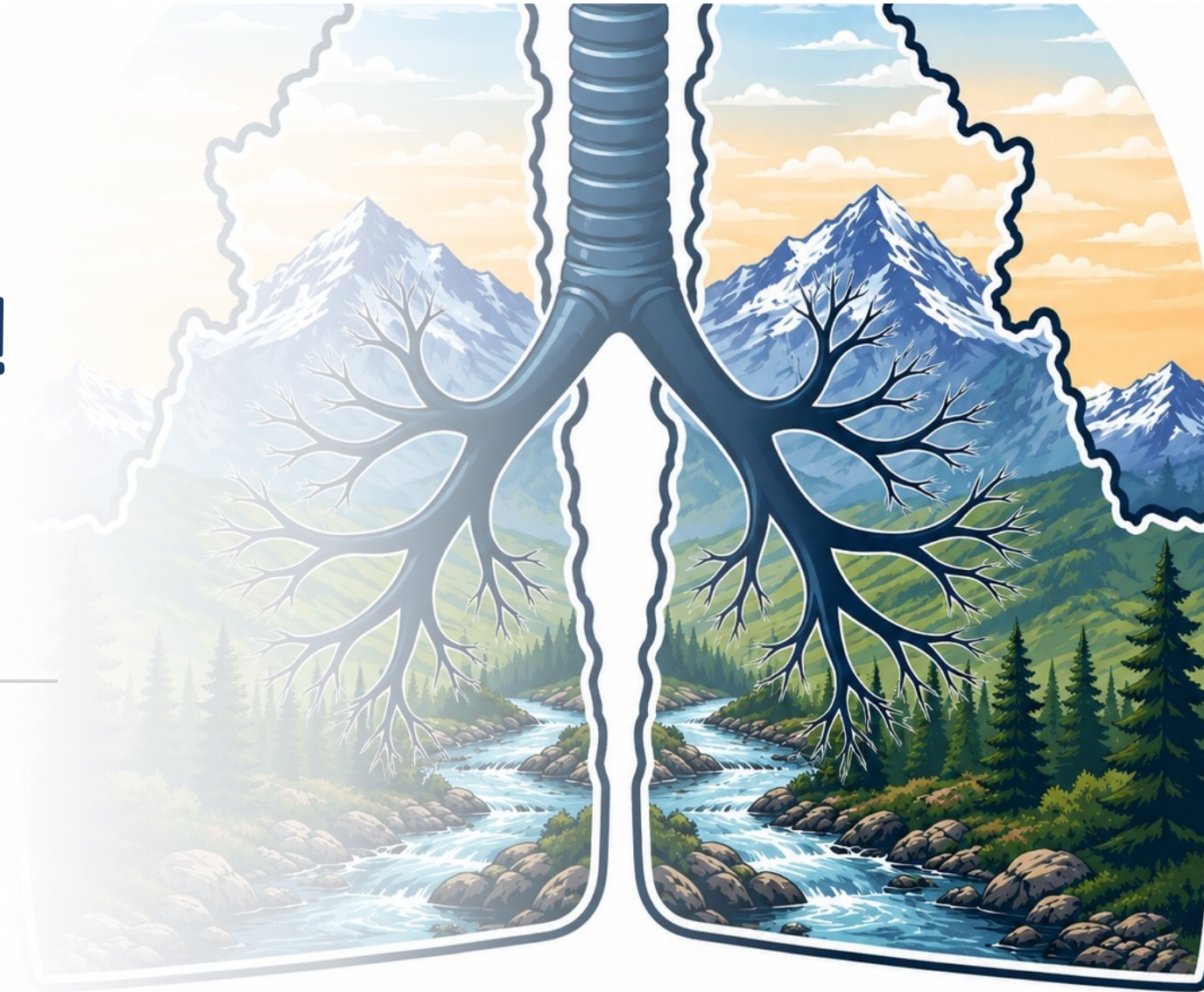
- At LCS Conference, group decided to treat with a Mucokinetic protocol +/- Antibiotics and get 6-8 week F/U LDCT.
- Also would involve Respiratory Therapists for aggressive Pulmonary Toilet.
- Many LR-3 and LR-4 Nodules reverted to LR-2 after F/U CT.
- Almost all also reported improvement in Respiratory symptoms.

S Modifier

- Reserved for new significant findings unrelated to Lung Cancer that require ASAP referral and treatment.
 - New or worsening CHF.
 - Aortic aneurysm.
 - Previously unknown severe coronary artery calcifications.
 - Solid or hollow organ masses on noncontrast LDCT requiring contrast enhanced CTs of the abdomen and pelvis.
 - New hydronephrosis or biliary dilatation.
 - Suspicious bone lesions or new mod-severe spinal compression fractures.
 - Thyroid nodules >1.5 cm.

Questions?

Break Time!
Visit the
Vendors!



Physiology in Critical Care

Paula Carvalho, MD, FCCP

Professor of Medicine

Division of Pulmonary, Critical Care, and Sleep Medicine

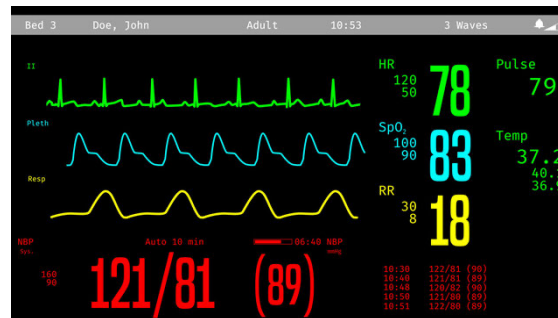
University of Washington

Academic Section Chief and Director, Intensive Care

Boise VA Medical Center

Learning objectives: Our agenda

- A case-based review of physiological principles in critical illness
- An approach to diagnosis, management, and re-evaluation in the care of critically ill patients



ICU patients: How do we think through a problem?

1. What failed?

2. What physiology explains it?

3. What can I measure now?

5. Did it work?



4. What intervention should change the physiology?

Case 1:

A 68-year-old man with COPD (FEV1 = 1.0 L)
was admitted to the ICU for a COPD exacerbation

His wife told us that he had taken oxycodone 5 mg
because he had awakened with a headache that morning

SpO2 = 91% on ambient air

Case 1 (cont'd):

Lab results:

ABG on ambient air:

- pH = 7.42
- PCO_2 = 68 mm Hg
- PO_2 = 58 mm Hg

Serum HCO_3^- =

- 32 mEq/L
-

Case 1: Question 1

What is the acid-base diagnosis?

ABG on ambient air:

- pH = 7.42
- PCO_2 = 68 mm Hg
- PO_2 = 58 mm Hg

- A. Respiratory acidosis
- B. Metabolic acidosis
- C. Metabolic alkalosis
- D. I don't have a clue

Serum HCO_3^- =

- 32 mEq/L
-

Case 1: Question 2

On the ABG, the pH was 7.42
Is this correct?

- A. YES, it is perfect!
 - B. A little bit off, OK for government work
 - C. Not even close
-

Acid-base balance: How do we maintain it?

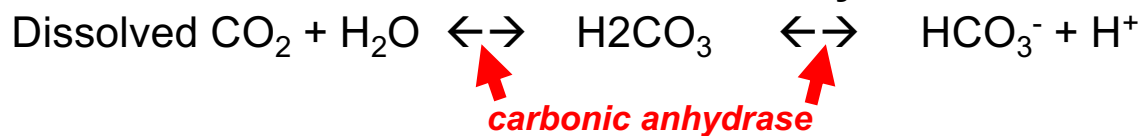
- Buffering systems: Continuous

Intracellular mechanisms:

*Phosphate buffers
Cytosolic proteins*

Extracellular mechanisms:

The bicarbonate-carbon dioxide buffer system:



Acid-base balance: How do we maintain it?

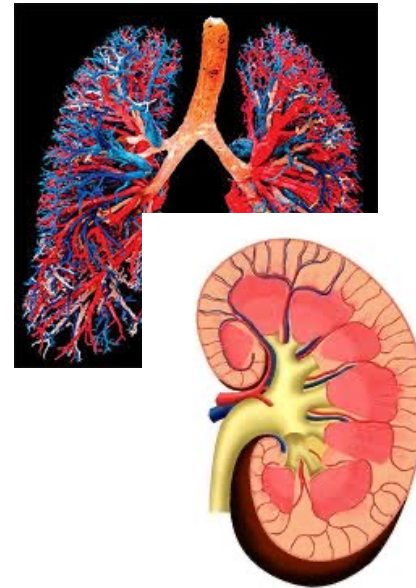
- **Buffering systems:** Continuous

- **The lungs: *FAST!***

(start immediately to adjust CO_2 ,
complete in 12-24 hours)

- **The kidneys: *SLOW...***

(at least 24-48 hours to adjust HCO_3)



The Labs:

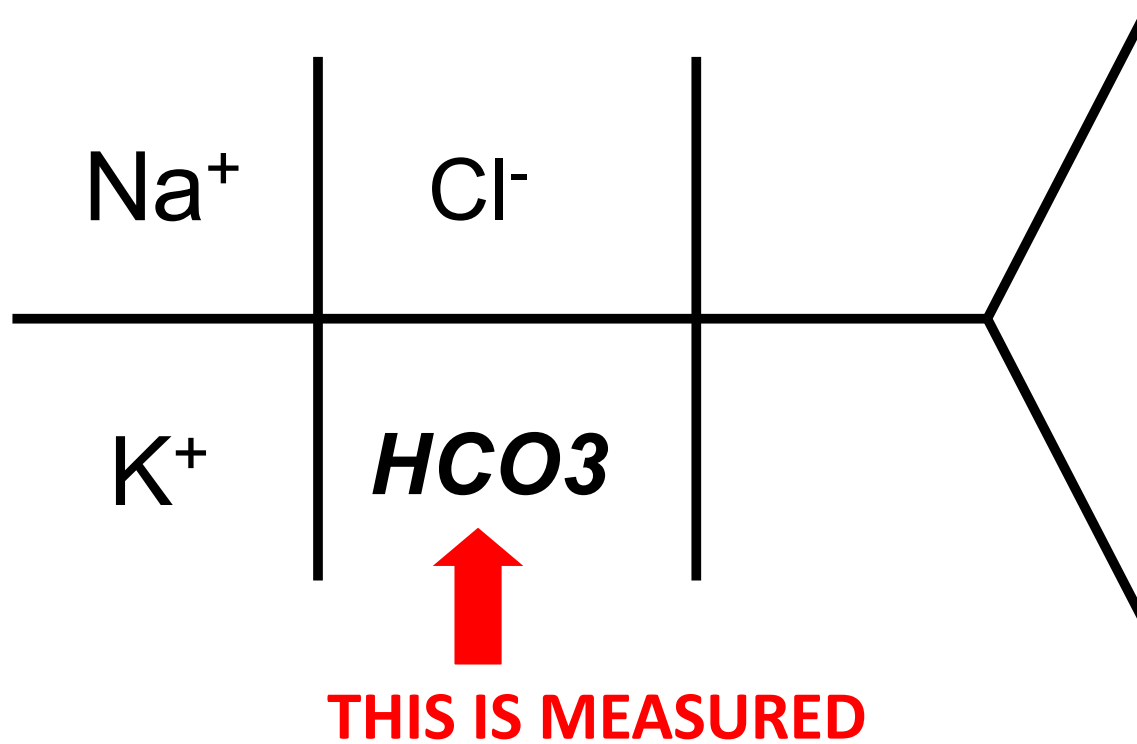
Arterial blood gases:

- ✓ The pH defines the ***overall*** acid-base status
- ✓ The PaCO₂ defines the ***respiratory*** acid-base status

Electrolyte/bicarbonate panel:

- ✓ The measured bicarbonate defines the ***metabolic*** acid-base status
-

The chemistry panel:



Arterial blood gases:

- pH 7.40 (7.35 – 7.45)
- PCO₂ 40 mm Hg (35-45)
- PO₂ 75-100 mm Hg
Depends on where you are.
- TCO₂ (base) 23-27 mmol/L



THIS IS CALCULATED

Internal consistency:

- The blood gas machine measures the pH, PaCO₂, and PaO₂ of the arterial blood
 - The level of base is calculated by the blood gas machine
 - The REAL measurement of base is the HCO₃⁻ measured by the chemistry machine
 - ***To do acid-base diagnosis, these values must be internally consistent!***
-

The Henderson-Hasselbalch equation:

$$\text{pH} = 6.1 + \log [\text{HCO}_3^- / \text{PaCO}_2 \times 0.03]$$

The Henderson-Hasselbalch equation:

$$\text{pH} = 6.1 + \log [\text{HCO}_3^- / \text{PaCO}_2 \times 0.03]$$

Too hard to use for calculation in your head!

So we use the modified Henderson-Hasselbalch equation instead!

The modified Henderson-Hasselbalch equation:

$$H^+ = 24 \times \frac{PaCO_2}{HCO_3}$$



The modified Henderson-Hasselbalch equation:

$$H^+ = 24 \times \frac{PaCO_2}{HCO_3}$$

$$H^+ = 24 \times \frac{\text{Lungs}}{\text{Kidney}}$$


Acid-base diagnosis:

***Step 1:* Determining internal consistency**

Acid-base diagnosis:

Step 1: Determining internal consistency

Calculate the H⁺ concentration:

$$[\text{H}^+] = 24 \times \text{PaCO}_2 / \text{HCO}_3^-$$

Acid-base diagnosis:

***Step 2:* Determining internal consistency**

Use the calculated $[H^+]$ to determine the pH

$$\text{pH} = -\log [H^+]$$

Estimating the pH from the H⁺ concentration:

If the [H ⁺] is...	Then the Calculated pH is...
100	7.00
79	7.10
63	7.20
50	7.30
40	7.40
32	7.50
25	7.60

Back to our patient: Question 1

What is the acid-base diagnosis?

ABG on ambient air:

- pH = 7.42
- PCO_2 = 68 mm Hg
- PO_2 = 58 mm Hg

- A. Respiratory acidosis
- B. Metabolic acidosis
- C. Metabolic alkalosis
- D. I don't have a clue

Serum HCO_3^- =

- 32 mEq/L

Back to our patient: What is the acid-base diagnosis?

ABG on ambient air:

- pH = 7.42
- PCO₂ = 68 mm Hg
- PO₂ = 58 mm Hg

- H⁺ = 24 x 68/ 32
- H⁺ = 51

Serum HCO₃⁻ =

- 32 mEq/L
-

Back to our patient:
What is the acid-base diagnosis?
Is the pH internally consistent?

- $H^+ = 24 \times 68 / 32$
- $H^+ = 51$
- If the H^+ is 51, the pH should be ~ 7.29 to 7.30
(not 7.42!)

H^+	pH
50	7.30
40	7.40

Back to our patient:

Question 1: What is the acid-base diagnosis?

$$\begin{aligned} H^+ &= 24 \times 68 / 32 \\ H^+ &= 51 \text{ (pH } \sim 7.30) \end{aligned}$$

- A. Respiratory acidosis
- B. Metabolic acidosis
- C. Metabolic alkalosis
- D. I don't have a clue

Question 2:

On the ABG, the pH was 7.42. Is this correct?

- A. YES!
 - B. A little bit off, OK for government work
 - C. Not even close
-

Back to our patient:

Question 1: What is the acid-base diagnosis?

$$\begin{aligned} H^+ &= 24 \times 68 / 32 \\ H^+ &= 51 \text{ (pH } \sim 7.30) \end{aligned}$$

- A. Respiratory acidosis
- B. Metabolic acidosis
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Back to our patient:

Question 1: What is the acid-base diagnosis?

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- A. Respiratory acidosis
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- C. Metabolic alkalosis
- D. I don't have a clue

Question 2:

On the ABG, the pH was 7.42. Is this correct?

- A. YES!
 - B. A little bit off, OK for government work
 - C. Not even close
-

Back to our patient:

- We cannot do acid-base diagnosis if the numbers are not internally consistent
 - The calculated pH using the modified Henderson-Hasselbalch equation should be close to the pH measured on the blood gas
 - If these criteria are not met, something is wrong and data cannot be used reliably
-

Follow-up: They believed the pH

- The patient was sent home the next morning with instructions to return in 2 weeks for outpatient follow-up
 - He received 2 albuterol treatments only
 - He was told to take ibuprofen for his headache and to continue his bronchodilators
-

Follow-up: They believed the pH

- The patient was sent home the next morning with instructions to return in 2 weeks for outpatient follow-up
 - He received 2 albuterol treatments only
 - He was told to take ibuprofen for his headache and to continue his bronchodilators
 - **He returned to the ER by ambulance 9 hours later after having a respiratory arrest at home**
-

Why do we sometimes see discrepancy between measured and calculated pH?

Most common: Pre-analytical sample delay

- If the ABG sample sits at room temperature before being placed into the machine, blood cells continue living and metabolizing
 - Blood cells consume glucose and increase PCO₂, artificially driving the pH down
-

Why do we sometimes see discrepancy between measured and calculated pH?

Lab analyzer interference:

- Central chemistry analyzers measure total CO₂ via enzymatic reactions
- Substances in a sick patient's blood (like high levels of LDH or lipids) can cross-react with central lab reagents, creating an artifactual shift in the central lab's chemistry values

Time between lab draws:

- If the ABG was drawn even 30 to 60 minutes apart from the peripheral blood draw for the chemistry panel, the patient's acute respiratory or metabolic state may have drastically shifted between the two draws
-

Why do we sometimes see discrepancy between measured and calculated pH?

Electrode drift and calibration error:

- The glass pH electrode or the PCO₂ Severinghaus electrode can drift due to protein coating or ineffective internal calibration solutions
 - If the sensor drifts, the electrical potential translated into pH is physically wrong, completely invalidating the relationship with PCO₂
-

What will we do differently with the next patient?

1. High index of suspicion for symptoms of hypercapnia
 2. Patients with baseline hypercapnia should avoid respiratory depressants as much as possible
 3. Interpret acid-base physiology correctly and determine internal consistency prior to attempting diagnosis
-

Case 2:

- A 23-year-old man presents to the Emergency Department for difficulty swallowing and sore throat which had worsened over the past 4 days
 - The patient sought medical evaluation because his voice was becoming increasingly hoarse, swallowing was very painful, and he was developing worsening dyspnea
-

Case 2 (cont'd):



Case 2 (cont'd):



Case 2 (cont'd):

The patient was admitted to the ICU and was started on antibiotics and methylprednisolone

The respiratory therapist caring for the patient recommended heliox as a temporizing measure to help with dyspnea

Heliox is made with helium to oxygen proportions of 80:20, 70:30, and 60:40.

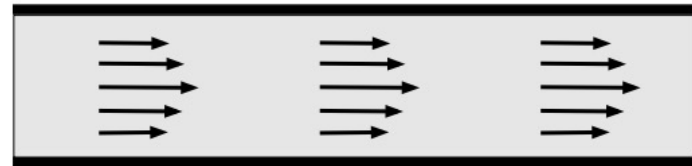
How does heliox help?

About heliox:

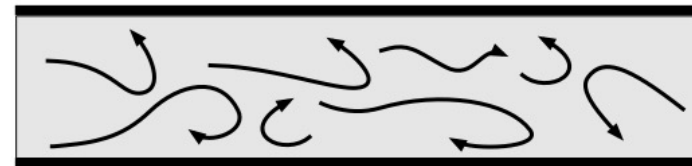
The flow of gas through a rigid tube can have one of two flow patterns

- Laminar flow is smooth
- Turbulent flow is disorganized

Laminar Flow



Turbulent Flow



The Reynolds number (Re):

- Whether laminar or turbulent flow prevails at any point in the airway is determined by the Reynolds number
- Laminar versus turbulent flow depends on the geometry of the airway, and the radius, velocity, density, and viscosity of gas
- This is represented mathematically as follows:

$$\text{Re} = \frac{\text{inertial force}}{\text{viscous force}}$$

$$= \frac{\text{density} \times \text{velocity} \times \text{diameter of tube}}{\text{viscosity}}$$

The Reynolds number (Re):

$$\text{Re} = \frac{\text{density} \times \text{velocity} \times \text{diameter}}{\text{viscosity}}$$

The boundaries between the types of flow are:

- Laminar flow when $\text{Re} < 2300$
 - Transient flow when $2300 < \text{Re} < 4000$
 - Turbulent flow when $\text{Re} > 4000$
-

The Reynolds number (Re):

- In the airways, the pressure gradient needed to drive airflow affects the work of breathing and varies on whether laminar or turbulent flow is present
- With laminar flow, the pressure gradient is directly proportional to the flow of gas:

$$\Delta \text{ Pressure} = \text{Flow} / \text{Resistance}$$

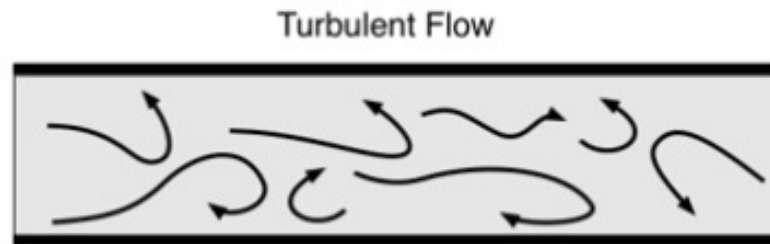
Laminar Flow



The Reynolds number (Re):

With turbulent flow, the pressure gradient is proportional to the square of the flow rate:

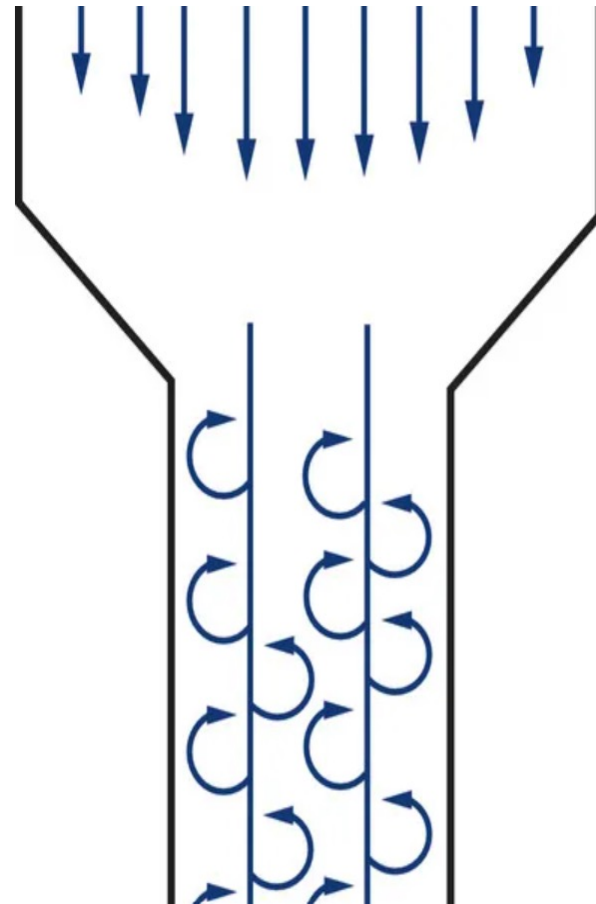
$$\Delta \text{ Pressure} = \text{Constant} \times (\text{Flow})^2$$



When turbulent flow prevails, a higher-pressure differential is needed to drive the same flow rate as compared with laminar flow conditions and work of breathing increases

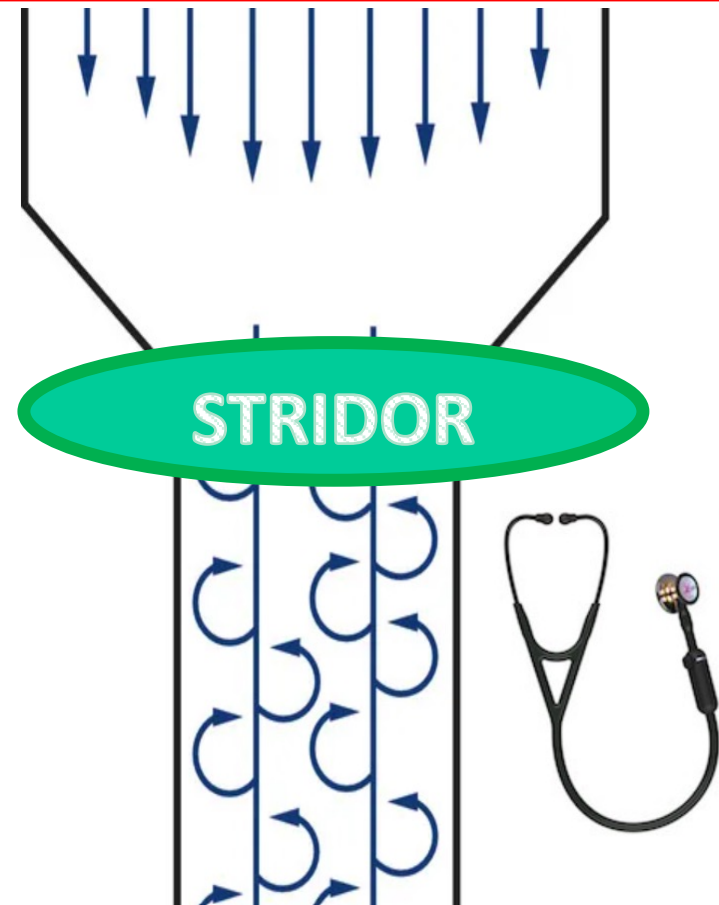
Airflow in upper airways:

- Gas velocity is high in the upper airway, which increases turbulent flow
- A focal constriction in this area alters the geometry of the airway and increases the amount of turbulent flow.
- This leads to increased work of breathing and impairs ventilation



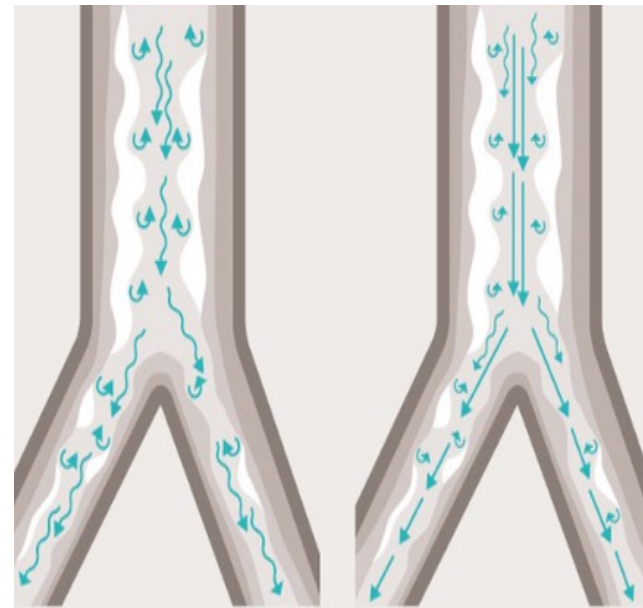
Airflow in upper airways:

- Gas velocity is high in the upper airway, which increases turbulent flow
- A focal constriction in this area alters the geometry of the airway and increases the amount of turbulent flow
- This leads to increased work of breathing and impairs ventilation



Airflow in upper airways:

- Because helium (MW ~ 4 g/mol) has a smaller molecular weight than nitrogen (MW ~ 28 g/mol), it has a much lower density than ambient air
- Breathing heliox decreases the Reynolds number, increases the tendency toward laminar flow and decreases the pressure differential the patient must generate to drive airflow
- As a result, the work of breathing decreases



Case 2: Question

Does heliox work in COPD exacerbations?

- A. Yes, it works great!
 - B. No, it doesn't work at all
 - C. I have no idea
-

Heliox in COPD: Does it work?

ORIGINAL ARTICLE

Heliox in the treatment of chronic obstructive pulmonary disease

R Andrews, M Lynch

Emerg Med J 2004;21:670-675. doi: 10.1136/emj.2003.011486

Objective: To determine if breathing helium oxygen mixtures in addition to conventional therapy in non-intubated adult chronic obstructive airways disease (COPD) patients reduces the arterial partial pressure of carbon dioxide (PaCO_2) more than conventional treatment alone, and confers an advantage in terms of the odds of intubation in the acute setting.

Design: Meta-analysis.

Setting: Diverse settings.

Participants: Adult patients with a diagnosis of either stable severe or acute COPD.

Main outcome measures: Decrease in partial pressure of arterial PaCO_2 and intubation rates.

Results: Combination of the results from trials measuring change in PaCO_2 in COPD subjects receiving conventional therapy but breathing helium-oxygen estimated a reduction of 0.78 KPa (1.44-0.13) beyond that produced by conventional therapy with air-oxygen breathing ($n=234$). Using quantitative and qualitative measures of validity it was found that most trials were unsatisfactory. Chief concerns were poor concealment of allocation and lack of blinding. Analysis excluding all papers with low methodological quality (Jadad <2) estimated the reduction in PaCO_2 conferred by use of Heliox breathing to be 0.22 KPa (+0.57 to -0.14). A non-significant reduction ($p=0.2$). When combined, the results from trials measuring the intubation rates of patients treated conventionally or with Heliox ($n=121$) the odds ratio of intubation was 0.096 (0.03-0.27).

Conclusion: Definitive evidence of a beneficial role of Heliox in treatment of severe COPD is lacking and therefore its wide scale use cannot be recommended based on this analysis. However, as a beneficial effect of Heliox breathing was reported in all trials, further investigation with a well conducted randomised controlled trial is warranted.

See end of article for authors' affiliations

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London SW18 2HS, UK;
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4 December 2003

Heliox in COPD: Does it work?

- Not so much
 - “Definitive evidence of a beneficial role of heliox in treatment of severe COPD is lacking and its wide scale use cannot be recommended”
 - Why not?
-

Heliox in COPD: Does it work?

Large proximal airways:

- Predominantly turbulent airflow
- Heliox decreases Reynolds number
- Decreased work of breathing

Small distal airways:

- COPD affects the more distal smaller airways
 - The cross-sectional area for airflow is large because there are so many distal airways, so flow is predominantly laminar
-

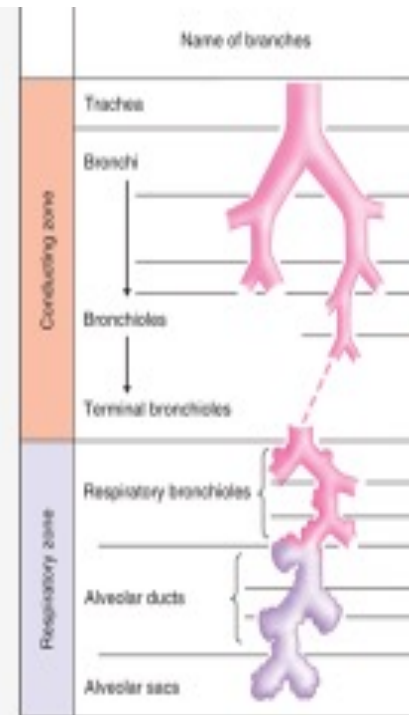
Heliox effect on proximal and distal airways:

Large proximal airways:

- Predominantly turbulent airflow
- Heliox decreases Reynolds number and decreases work of breathing

Small distal airways:

- COPD affects distal airways
- Large cross-sectional area for airflow is large due to many distal airways, so flow is predominantly laminar
- **Heliox has reduced effect**



Heliox

← +

← -

Source: Kim E. Barrett, Susan M. Berman, Scott Boitano, Heddwen L. Brooks: Ganong's Review of Medical Physiology, 25th Ed.
www.accessmedicine.com
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Case 3:

A 60-year-old woman is pulled unresponsive from the bedroom of her burning home

The fire was in the kitchen, but the bedroom filled with dense smoke

In the ER, the patient is unresponsive

Case 3 (cont'd):

- **Labs:**

Hct = 36%, Hgb = 13.6 g/dl

SaO₂ on RA = 96%

ABG on RA = pH 7.25 / PCO₂ 28 / PO₂ 93

What is the oxygen content?

Oxygen Content (CaO₂):

The total oxygen content =

Oxygen bound to hemoglobin + Oxygen dissolved in plasma

Normal value = 20 ml O₂/dl blood

$$\text{CaO}_2 = [1.34 \text{ (mlO}_2\text{/g Hgb)} \times \text{Hgb (g/dl)} \times \text{SaO}_2 \text{ (\%)}] + [\text{PaO}_2 \text{ (torr)} \times 0.003 \text{ (ml O}_2\text{/dl/torr)}] =$$

$$[1.34 \times 13.6 \times 0.96] + [93 \times 0.003] = \mathbf{17.8 \text{ ml O}_2\text{/dl}}$$

Case 3 (cont'd):

- **Labs:**

Hct = 36%, Hgb = 13.6 g/dl

SaO₂ on RA = 96%

ABG on RA = pH 7.25 / PCO₂ 28 / PO₂ 93

COHb = 25%

What is the oxygen content now?

Case 3 (cont'd):

What is the oxygen content with COHb = 25%?

- Hgb saturation = $100\% - 25\% = 75\%$
- $75\% \times 13.6 \text{ g/dl} = 10.2 \text{ g/dl}$ (effective Hgb)
- Her oxygen content is:
 $(1.34 \times 10.2 \times 0.96) + (93 \times 0.003) = 13.5 \text{ ml O}_2/\text{dl}$

Carbon monoxide results in a functional anemia as carboxyhemoglobin is not functional

Case 3 (cont'd):

The patient had persistent hypotension and suddenly had a full tonic-clonic seizure during intubation

One of the respiratory therapists detected “bitter almonds” in exhaled air following intubation

Case 3 (cont'd):

Labs:

Arterial blood gas:

pH 7.25 / PCO₂ 28 / PO₂ 93 / SpO₂ 96%

Venous blood:

pH 7.15 / PCO₂ 32 / PO₂ 85 / SpO₂ 92%

The abnormally high PvO₂ reflects
decreased peripheral oxygen extraction

Conditions with high SpO₂ > 75%:

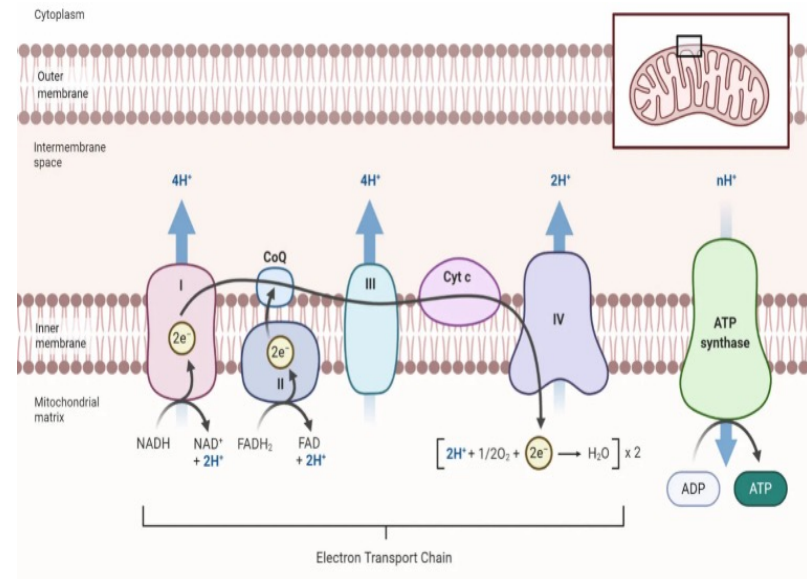
Condition	Mechanism
Severe sepsis	Multiple mechanisms impair O ₂ utilization
Cyanide intoxication	Cytochrome oxidase inhibition (low O ₂ utilization)
Upper extremity dialysis fistula	Arterialized blood returns to venous system

Diagnosis:

Cyanide toxicity

Cyanide: Failure of peripheral oxygen extraction

- The primary enzyme system at the cellular level that promotes oxygen in cell metabolism is the cytochrome oxidase system
- If this system is poisoned (cyanide), oxygen can no longer be extracted



Review:

- Acid base diagnosis and internal consistency are needed for diagnostic accuracy
 - Laminar and turbulent flow, the Reynolds number, and the effect of heliox on upper versus lower airways
 - Carbon monoxide causes decreased oxygen content due to functional anemia
 - Cyanide toxicity and decreased oxygen extraction with increased venous oxygen saturation
-

***Boise VA Medical Center
N.B. Charan, MD 2005***



Idaho Society for Respiratory Care State Meeting • CHRONO-NUTRITION SERIES

The Clock, the Fork, and the Lung

Why Timing Matters More Than You Think



Presented by

Dr. Amanda L. Leightner PhD, RRT, RRT-SDS, RPSGT, CCSH, FAARC, FNAP

Merrick Family Endowed Professor

Dr. Amy Y. Spurlock PhD, RN

Jody DeMeyer Endowed Chair of Nursing

LEARNING OBJECTIVES

By the end of this session, you will be able to



Describe

How circadian rhythms and meal timing influence respiratory physiology, sleep quality, and metabolic health.



Examine

Current evidence linking chrono-nutrition with inflammation, obesity, obstructive sleep apnea, and chronic respiratory disease.



Apply

Practical chrono-nutrition strategies to educate patients and support respiratory health across inpatient, outpatient, and sleep medicine settings.

ROADMAP

Tonight's schedule runs on a clock the body already keeps

01

The Circadian Foundation

Molecular clocks, respiratory physiology, sleep architecture, metabolic regulation, and occupational circadian risk.

02

The Chrono-Nutrition Evidence Base

Peer-reviewed data on meal timing, inflammation, obesity, cardiometabolic health, OSA, and chronic lung disease, including where evidence is still thin.

03

Bringing It to the Bedside

A patient-counseling framework and concrete strategies for inpatient, outpatient, and sleep medicine settings.

PART 1



The Circadian Foundation

How molecular clocks, lung physiology, sleep architecture, and metabolism are wired to the same 24-hour timekeeping system.

THE CIRCADIAN SYSTEM

One master clock, thousands of peripheral clocks

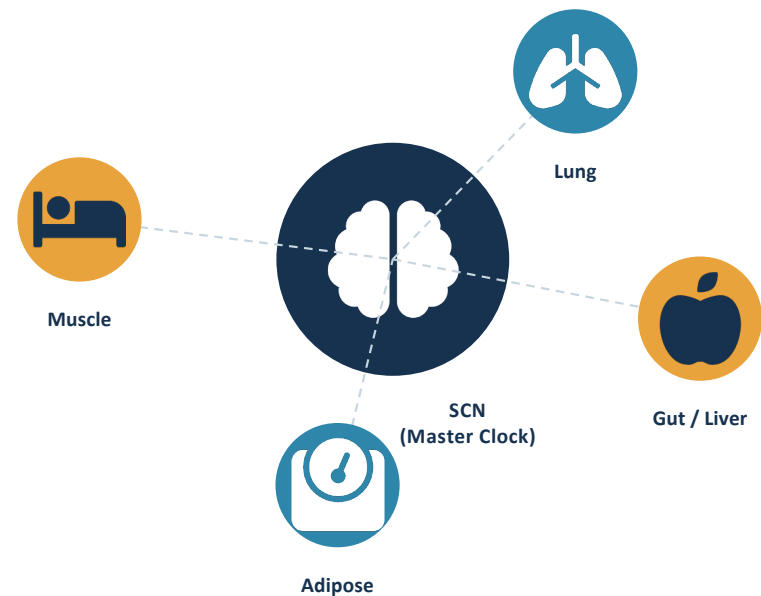
The suprachiasmatic nucleus (SCN)

sits in the hypothalamus and is entrained primarily by light. It synchronizes the timing of nearly every organ system in the body.

Peripheral clocks

in the lung, liver, gut, adipose tissue, and skeletal muscle run on the same clock-gene machinery but are entrained locally — largely by feeding time.

Clinical hook: meal timing is one of the strongest entraining cues for the lung's peripheral clock — exactly why chrono-nutrition matters to respiratory care.



The molecular gears that keep 24-hour time

A transcription–translation feedback loop — running in nearly every cell — completes one cycle approximately every 24 hours.

1

CLOCK : BMAL1 activate

This heterodimer binds E-box elements and drives transcription of Period (PER) and Cryptochrome (CRY) genes.

2

PER : CRY accumulate & repress

PER/CRY proteins build up in the cytoplasm, translocate to the nucleus, and inhibit their own activators, CLOCK:BMAL1.

3

The loop resets

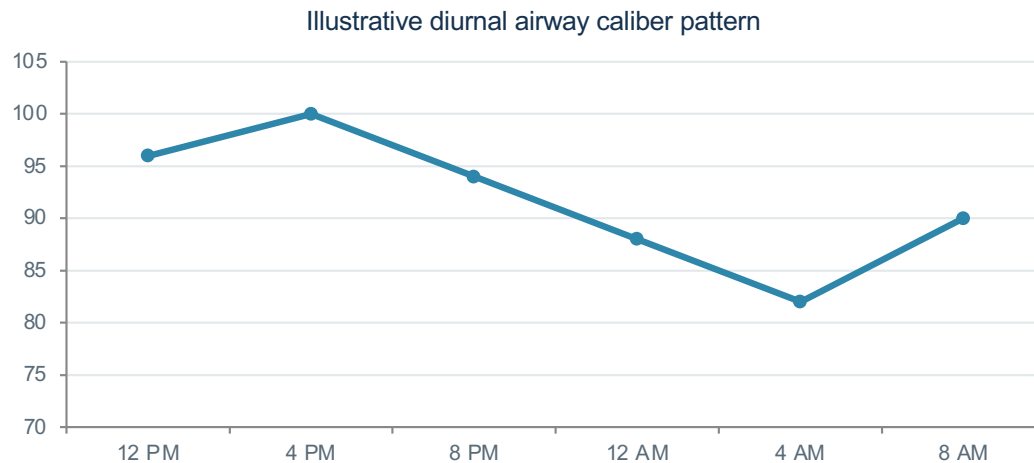
PER/CRY degrade, releasing CLOCK:BMAL1 to restart the cycle — the basis of every organ's local clock, including the lung.

Why feeding time matters: unlike the SCN (entrained by light), peripheral clocks in lung, gut, liver, and adipose tissue are entrained largely by the timing of nutrient intake.

RESPIRATORY PHYSIOLOGY

Lung function itself rises and falls on a 24-hour clock

FEV₁ and peak expiratory flow follow a reproducible diurnal rhythm — typically lowest in the early morning hours and highest in the afternoon — independent of sleep or activity.



- The endogenous circadian system worsens airway function at night — independent of sleep itself.
- Nocturnal asthma affects an estimated 30–70% of patients, with peak constriction near 4 AM.
- Roughly half of asthma-related deaths and respiratory arrests occur overnight.
- Normal circadian oscillation in airway caliber also contributes to nocturnal COPD exacerbations and worsening hypoxia.

ASTHMA MECHANISMS

A molecular link between clock disruption and airway inflammation

BMAL1 / FOXA2 signaling

In asthma patients, disrupted circadian clock gene expression — particularly low BMAL1 — correlates with elevated IL-6 and greater nocturnal symptom burden.

Mouse model confirmation

In an ovalbumin-induced asthma model, experimentally disrupting the light/dark cycle altered circadian clock gene expression and amplified airway inflammatory cytokine oscillation, mirroring the human pattern.



IL-6

significantly higher in the peripheral blood of asthma patients with nocturnal symptoms compared to those without.

Peak airway constriction in nocturnal asthma clusters around 4 AM, coinciding with this circadian inflammatory nadir.

COPD

COPD: current therapies don't correct the circadian decline



Rhythmic decline persists

Lung function in COPD shows the same rhythmic overnight decline seen physiologically — often with a larger amplitude and worse nocturnal hypoxia.



Standard drugs fall short

Corticosteroids and bronchodilators — the pharmacologic mainstays of COPD — fail to correct the rhythmic overnight decline in lung function or reduce mortality.



A chronotherapy opportunity

This gap is prompting research into circadian-informed dosing and timing strategies rather than new drug targets alone.

SLEEP QUALITY

Circadian timing shapes sleep architecture — and the airway



Sleep pressure & timing

Melatonin onset and core body temperature decline gate sleep onset; misaligned meal timing can delay both.



Upper airway tone

Airway dilator muscle activity and lung volumes fall overnight, compounding any anatomic predisposition to collapse.



Sleep ↔ metabolism loop

Short or poor-quality sleep increases appetite and caloric intake, feeding back into weight gain and airway risk.

Bottom line: sleep quality, airway patency, and metabolic signaling are not three separate problems — they share one circadian control system.

METABOLIC HEALTH

Hormones that govern hunger and energy also run on a clock

Cortisol

Peaks shortly after waking and declines through the day, priming morning glucose availability and alertness.

Melatonin

Rises in the evening and suppresses insulin secretion — late eating during high melatonin can worsen glucose tolerance.

Leptin & ghrelin

Satiety and hunger signaling both show diurnal rhythms; circadian misalignment blunts leptin and raises ghrelin, increasing intake.

Glucose tolerance

Identical meals produce a larger glycemic and insulin response late at night than in the morning.

OCCUPATIONAL RELEVANCE

Shift work: circadian misalignment hits staff, too

Rotating and night shifts — common for respiratory therapists and nurses — are a well-studied real-world model of circadian misalignment.



Metabolic syndrome risk

Significantly higher prevalence of metabolic syndrome among healthcare shift workers vs. day workers.



Obesity, T2D, hypertension

Shift work is associated with increased risk of overweight, obesity, type 2 diabetes, and elevated blood pressure.



Central ↔ peripheral desync

Simulated night-shift protocols induce measurable misalignment between the SCN and gut, liver, and pancreas rhythms.

Why raise this with clinical staff: the same chrono-nutrition principles you'll counsel patients on — consistent eating windows, avoiding late-night meals — apply directly to shift-working clinicians.

PART 2



The Chrono-Nutrition Evidence Base

What current peer-reviewed studies show and don't yet show about meal timing, inflammation, obesity, cardiometabolic health, OSA, and chronic lung disease.

MECHANISMS

The signaling molecules chrono-nutrition targets: mapped to the clock

Meal timing works because the hormones that govern hunger, satiety, and fuel storage each have a preferred time of day. Chrono-nutrition re-synchronizes these rhythms to the light/dark cycle.



Leptin & ghrelin

Adipose- and stomach-derived satiety/hunger signals oscillate across the day and are a primary target of meal-timing interventions.



Incretins (e.g., GLP-1)

Gut-derived incretin secretion varies with meal timing, shaping post-prandial glucose and insulin responses.



FGF-21 & GDF-15

Hepatokine / stress-response signals tied to fasting state, explored as re-synchronization targets.

GUT MICROBIOME

Meal timing sets the rhythm of the gut microbiome, too

The gut microbiota is intrinsically rhythmic

and its synchronization with the host's biological clock is essential for normal gut physiology and downstream metabolic and inflammatory signaling.

“Gut jet lag”

Irregular meal schedules, disordered eating times, and nocturnal light exposure desynchronize host and microbiota rhythms — a novel mechanism now linked to functional GI symptoms and broader metabolic dysfunction.

Relevance to respiratory care: the gut–lung axis means microbiota disruption from irregular eating may compound systemic inflammation relevant to asthma and COPD.

INFLAMMATION

Late-night eating is pro-inflammatory at the molecular level

Late-night eating

has been shown to drive physiological dysregulation and circadian misalignment, with accompanying gut microbial dysbiosis linked to lipid and inflammatory changes.

BMAL1 / FOXA2 signaling

The same clock-disruption pathway implicated in nocturnal asthma (Slide 8) provides a plausible mechanistic bridge between meal timing and airway inflammation.



Late-night eating triggers measurable circadian misalignment and microbial dysbiosis not just extra calories.

This positions eating-window timing , not only content , as a modifiable inflammatory exposure.

OBESITY

Time-restricted eating produces real, reproducible weight loss

~5%

body weight loss

8-hour TRE (12 PM–8 PM) vs. control, adults with obesity, 12 months

3–5%

clinically meaningful loss

Consistent range across meta-analyses and clinical evidence summaries of TRE

85–88%

adherence rate

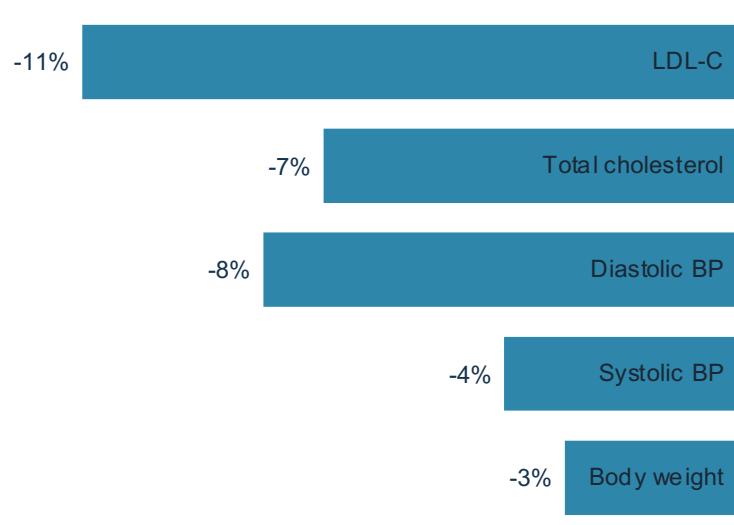
Reported across early-, late-, and self-selected TRE windows over 12 weeks

Clinical takeaway: TRE is at least as effective as conventional calorie restriction for weight loss and also improves fasting glucose and lipid profiles, with adherence rates that make it realistic for patients who struggle with traditional calorie counting.

CARDIOMETABOLIC HEALTH

Beyond the scale: 10-hour TRE moved the numbers that matter

A 12-week randomized trial of 10-hour time-restricted eating in adults with metabolic syndrome produced significant changes beyond weight alone — without any instruction to change diet quality or quantity.



- All changes statistically significant ($p < 0.05$) at 12 weeks.
- Reductions in blood pressure and lipids were not fully explained by weight loss alone.
- Eating window narrowed by ~4.4 hours on average, with no explicit calorie-counting instruction.

OBSTRUCTIVE SLEEP APNEA

What the trials show: weight loss, yes, direct sleep benefit, not yet proven



Randomized trials of time-restricted eating in adults with obesity/OSA have generally found significant weight loss but no significant change in sleep quality, insomnia severity, or OSA risk/severity scores versus controls.

12-month TRE vs. calorie restriction

- Both arms lost ~5% body weight
- No change in sleep quality, duration, insomnia severity, or OSA risk in either arm

12-week TRE in diagnosed OSA

- No significant change in body mass, body composition, or portable-monitor sleep parameters
- Small sample — likely underpowered

Early vs. late TRE (ChronoFast)

- Early time-restricted eating improved actigraphy-estimated sleep quality
- Effects on sleep may require $\geq 5\%$ weight loss to emerge

CHRONIC RESPIRATORY DISEASE

Chronotherapy spans the full range of chronic lung disease

A 2021 pharmaceuticals review maps circadian-informed treatment strategies across four major respiratory diseases, timing existing therapies rather than only developing new drugs.



Asthma

Circadian signature:

Nocturnal symptoms in 30–70% of patients; peak constriction ~4 AM.

Chronotherapy angle:

Timing bronchodilators/steroids to the pre-dawn circadian trough.



COPD

Circadian signature:

Rhythmic overnight decline in lung function and worsened hypoxia.

Chronotherapy angle:

Chronotherapy explored where standard timing fails to cut mortality.



Pulmonary fibrosis

Circadian signature:

Clock-gene disruption linked to fibrotic remodeling pathways.

Chronotherapy angle:

Circadian-informed antifibrotic dosing is an emerging research target.



Lung cancer

Circadian signature:

Tumor-suppressor and clock-gene crosstalk affects disease progression.

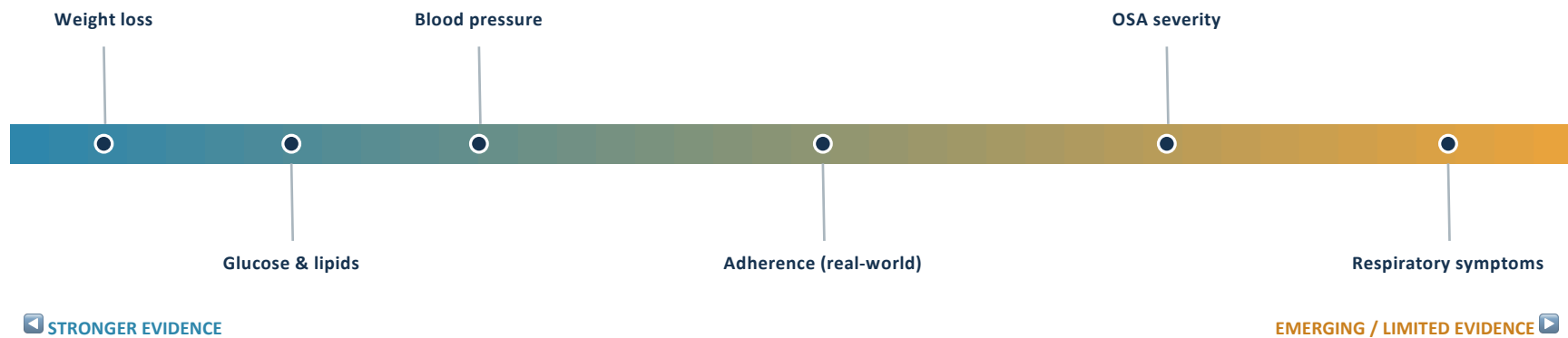
Chronotherapy angle:

Chronochemotherapy aims to reduce toxicity, preserve efficacy.

READING THE EVIDENCE CRITICALLY

Where the evidence is strong and where it is still thin

Not all chrono-nutrition claims rest on the same quality of evidence. Placing each outcome on a strength spectrum helps set honest expectations with patients and colleagues.



Counsel confidently

Weight loss, glucose, lipid, and blood-pressure benefits of TRE are supported by multiple RCTs.



Frame as promising, not proven

Direct sleep, OSA, and respiratory-symptom benefits from meal timing alone remain unproven in RCTs.

Source: Liu et al., *J Health Popul Nutr* 2026; O'Neal et al., *Frontiers in Nutrition* 2023.

PART 3



Bringing It to the Bedside

A practical counseling framework and concrete, low-cost chrono-nutrition strategies your teams can start using this week.



A five-step framework for chrono-nutrition counseling

1

1. Ask the eating-window question

When do you eat your first and last calories of the day? A 30-second question that reveals late-night eating patterns.

2

2. Assess chronotype and schedule

Shift work, caregiving duties, and evening chronotype all shape what's realistic tailor the plan, don't template it.

3

3. Set one collaborative goal

Real-world data show 16:8 is the most used TRE method start with a familiar, achievable structure.

4

4. Set expectations honestly

Be clear about what's well supported (weight, glucose, lipids) versus still emerging (sleep, OSA, respiratory symptoms).

5

5. Reassess and adjust

Qualitative research shows flexibility, not rigid rules, predicts sustained engagement; revisit the plan at follow-up.



Inpatient: protect the circadian window during admission

1

Align tray timing with daylight, not shift schedules

Advocate for consistent daytime meal delivery; avoid late-evening trays that push intake into the biological night.

2

Flag overnight labs and vitals for consolidation

Where clinically safe, cluster overnight interruptions to protect sleep opportunity and reduce circadian disruption.

3

Anticipate the 4 AM symptom window

Pre-position bronchodilators and monitoring for asthma/COPD patients ahead of the circadian FEV₁ dip.

4

Use light exposure as a low-cost adjunct

Encourage daytime light and dim evening light in patient rooms to support SCN entrainment during extended stays.



Outpatient: build chrono-nutrition into routine counseling

1

Start with an eating-window history

Make it a standard review-of-systems question, not a specialty referral trigger.

2

Recommend a consistent 10–12 hour eating window

Frame TRE as a structure, not a diet: favor consistency before narrowing the window further.

3

Set expectations honestly

Weight and metabolic benefits are well supported; direct sleep/OSA benefit from timing alone is not yet proven so say so.

4

Pair timing with disease-specific goals

For asthma/COPD patients, connect earlier dinners with reduced evening reflux and overnight symptom burden.

Sleep medicine: integrate timing into the sleep work-up



1

Add meal-timing questions to the sleep history

Late dinners and nighttime snacking are modifiable contributors worth documenting alongside PAP adherence.

2

Use weight loss as the primary chrono-nutrition lever

Position TRE as one option among several for achieving clinically meaningful weight loss to improve AHI — not a stand-alone sleep therapy.

3

Coordinate with dietitians on window, not just content

Nutrition referrals should address when patients eat, not only what — particularly for shift workers and delayed sleep phase.

4

Track outcomes that matter to the patient

Follow weight, AHI, and symptom scores together; do not assume timing changes alone will move sleep-study metrics.

TEAM-BASED CARE

Chrono-nutrition works best as a team effort



RT / Physician

Identify nocturnal symptom patterns; time bronchodilators and monitoring to circadian risk windows.



Nursing

Coordinate inpatient meal timing, protect sleep opportunity, and reinforce consistent eating-window messaging.



Dietitian

Design the specific eating-window plan and troubleshoot adherence barriers identified by the team.



Behavioral health

Support habit change for patients whose eating patterns are tied to shift work, stress, or mood.

A brief huddle question “what time is this patient actually eating?” surfaces most of the actionable information.

KEY TAKEAWAYS

What to carry back to your unit



Timing is physiology, not lifestyle trivia

Lung function, sleep architecture, and metabolic hormones all run on the same circadian clock.



Late eating tracks with inflammation

Circadian misalignment and late-night intake are linked to elevated IL-6 and nocturnal symptom burden.



TRE reliably improves weight & metabolic markers

Comparable to calorie restriction, with strong adherence and added blood-pressure/lipid benefits.



Be honest about the OSA/respiratory evidence gap

Weight loss helps OSA; direct sleep or symptom benefit from meal timing alone is not yet proven in RCTs.



Thank You

Questions & discussion

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Full references

See the two reference slides that follow for complete peer-reviewed citations supporting this presentation.

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Lunch

Sponsored by:



Why Isn't My Patient's Pneumonia Getting Better?

Idaho State Respiratory Care Conference
2026

Case

- 52y woman with SOB and cough 3-4 days
- ROS: + fatigue, myalgias, chest discomfort, ?chills
- Denies: fever, N/V
- PMHx: tobacco, chronic pain, anxiety, GERD
- Soc Hx: +tobacco, no travel, 1 week pta helped friend clean out a dirty shed

52yo with SOB and cough

- VS: 37.1, 90, 130/73, 80%RA, 100% 5L, 20
- Coarse BS bilaterally, no wheezes
- RRR
- Labs:
 - WBC 10.3, PMN 75%, Lymphs 17%, Eos 1.9%
 - Troponin 0.62, BNP 847
 - Procal 0.182
 - Respiratory PCR negative

CXR



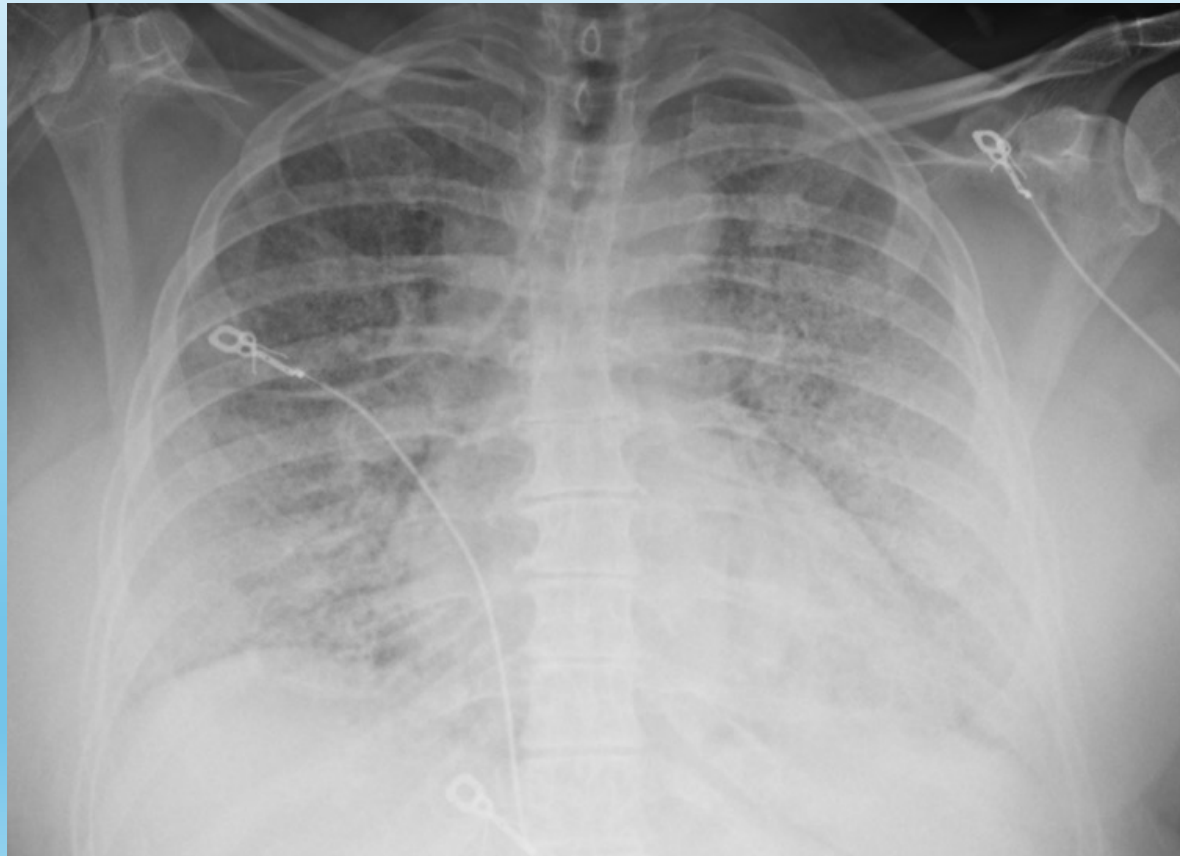
CTPA



Course

- Admitted
- Oxygen
- Ceftriaxone, Azithro
- RT
- Echo normal
- *Worsened with increased O2 needs*

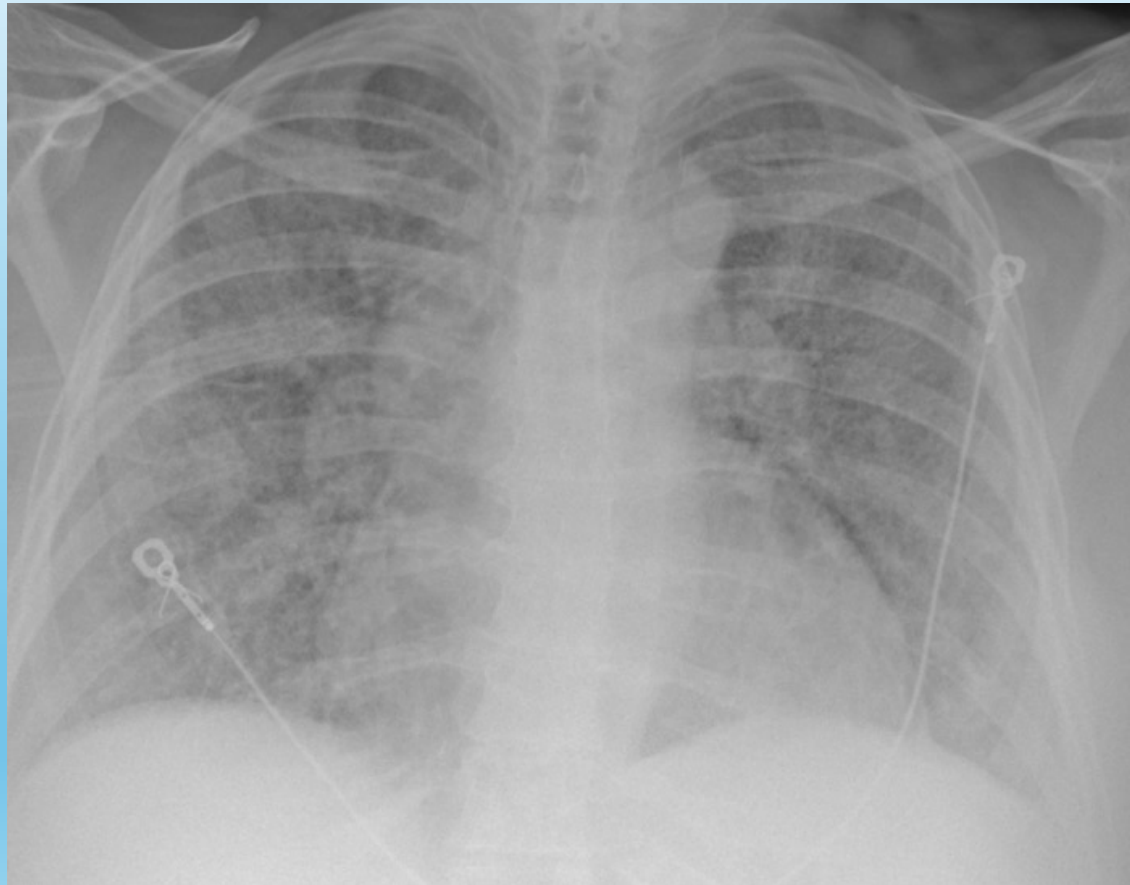
CXR HD #3



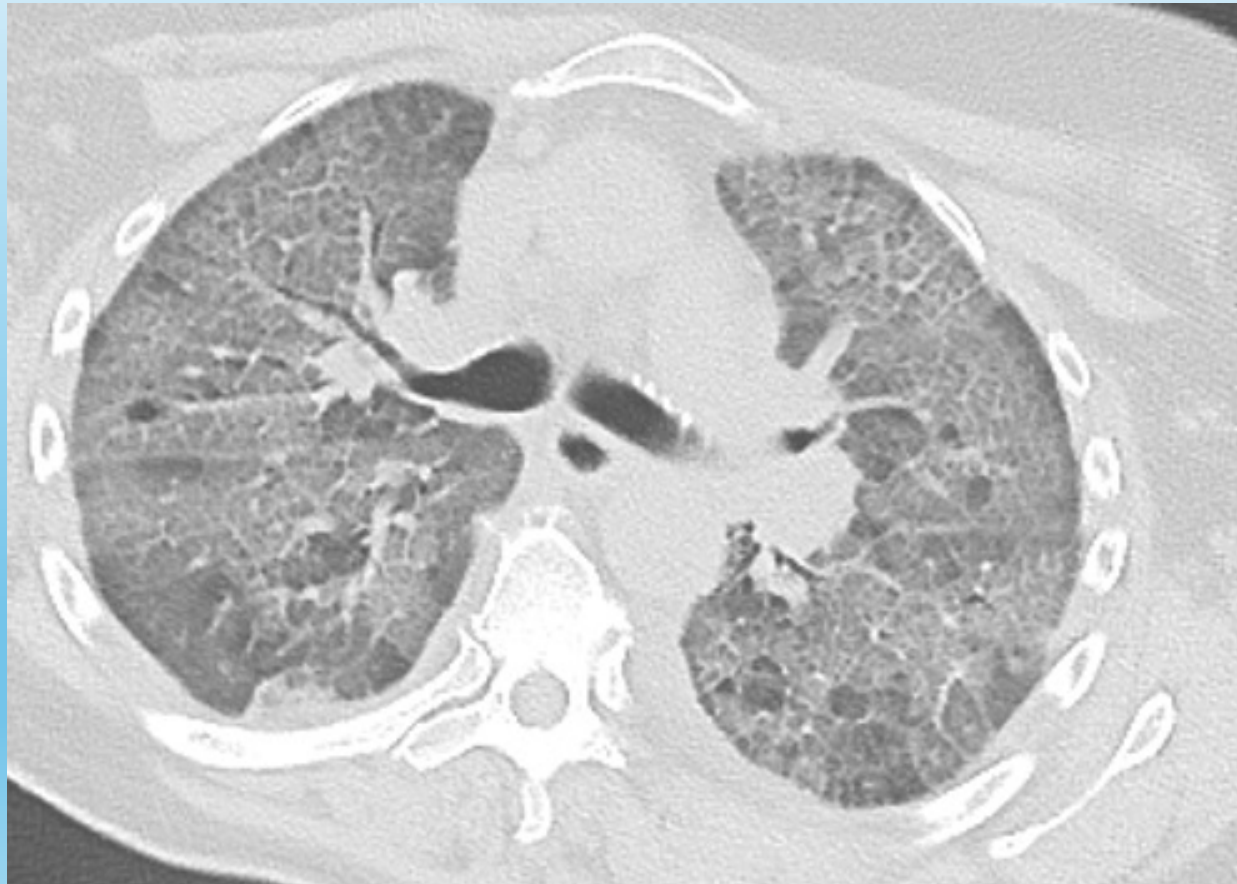
Course

- Additional hx of several episodes of LOC, possible aspiration
- Broadened antibiotic
- NIV – tolerated very well
- Presumptive dx of ALI/ARDS due to viral infection, possible aspiration
- *Stabilized, but no improvement*

CXR HD #6



Repeat Chest CT



What next?

- Stay the course?
- Should we intubate?
 - If yes, why?
- Bronchoscopy?
 - Will it help her?
 - Is it safe?
- Lung biopsy?
 - Same questions
- Steroids?

Bronchoscopy in ARF with pulmonary infiltrates

- When is it indicated?
- How may it help?
- What information can it tell us?

Indications for bronchoscopy in ARF

- Diagnosis uncertain
- Not responding to treatment
- Suspicion for specific treatable diagnosis
- Immunocompromise

What information can we learn from bronch?

- Airway inspection
 - Secretions
 - Bleeding, including diffuse alveolar hemorrhage
 - Foreign bodies
 - Trauma
 - Tracheobronchomalacia
- BAL
 - Generally well tolerated
 - Infection, including opportunistic infections
 - DAH
 - Cellularity
 - Cytology sometimes helpful

What information can we learn from bronch?

- Transbronchial lung biopsy
 - Higher risk (pneumothorax, bleeding), esp with PPV
 - Helpful for a few specific diagnoses – Sarcoid, HP, Lung tx rejection
 - Small sample size
- Endobronchial Ultrasound (EBUS)
 - Useful for sampling mediastinal/hilar lymph nodes often for dx/staging of malignancy
 - No role in ARF

Why not bronch everybody?

- Not everyone needs it
- Marginal yield
 - 30-60%
 - Varies depending on other risks (ie immunosuppression)
- Often requires intubation
- *Need to weigh the Risk vs Benefit of intubation and mechanical ventilation*

What helpful information might BAL reveal?

- Infection - unexpected, atypical or opportunistic
- Diffuse Alveolar Hemorrhage (DAH)
- Eosinophilic process – acute or chronic eosinophilic pneumonia
- Lymphocytic process – HP or sarcoid
- Rare causes
 - Pulmonary alveolar proteinosis
 - Lipoid pneumonia
- ***For many Dx, BAL is not diagnostic***

What about Corticosteroids?

Steroids beneficial

- Community Acquired Pneumonia
 - Probably yes
 - Greater benefit if shock or ARF
 - Covid
- ARDS
 - Probably yes
 - Severe – P/F <200
 - Early - < 14 days
- COPD/Asthma

Steroid beneficial – less common diagnoses

- Eosinophilic disease – AEP, CEP
- Cryptogenic Organizing Pneumonia (BOOP)
- Hypersensitivity Pneumonitis
- Sarcoidosis
- Pulmonary Vasculitis/DAH
- Connective Tissue Disease Associated (SLE, Scleroderma)
- Desquamative Interstitial Pneumonia, VALI
- Drug toxicity – (Chemo, Amio, Mtx, Dapto, nitrofurantoin)
- Radiation

Steroids maybe beneficial

- Flare of ILD/IPF
- Acute Interstitial Pneumonia
- Idiopathic Pneumonia Syndrome (BM tx pts)
- Pneumocystis – if used concurrently with antimicrobial rx

Steroids not beneficial or even harmful

- Pneumonia due to Influenza, fungus, TB
- Aspiration Pneumonia – limited data
- CHF
- IPF
- Lymphangitic spread of malignancy
- PAP
- Lipoid pneumonia

Why not just try steroids?

- Not beneficial and even harmful for some
- Hyperglycemia
- Fluid retention
- Immune suppression
- Weakness/muscle wasting
- Delirium
- *How much and for how long?*
- **Reality: we often do**

Back to our case...

- Opted to start steroids
- No better, no worse
- Case review
- Given hx of uncertain dx, tobacco use, unknown exposures, lack of improvement, and CT appearance, started to wonder about less common causes – DAH, AIP, PAP
- Decided to intubate and bronch/BAL
- *Sats dropped to 50s with BAL*

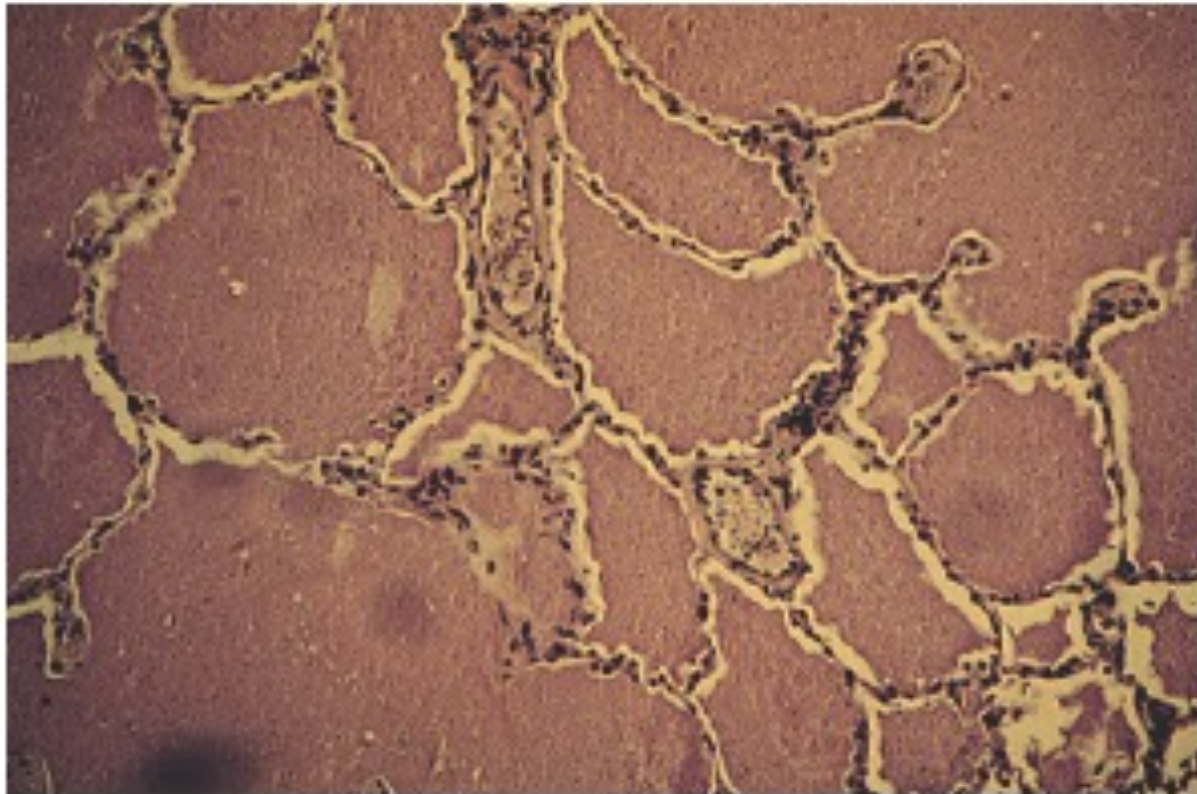
Diagnosis: **Pulmonary Alveolar Proteinosis**

- **Rare** – annual incidence of .2-.4 per million
- Typically gradual onset
- Progressive SOB, cough
- No specific lab indicators
- Autoimmune form may have + anti-GMCSF ab
- Chest CT – “*crazy-paving*”
- BAL “milky” fluid with abundant pink PAS + material
- Lung biopsy more definitive

Crazy-paving



PAP – with + PAS stain



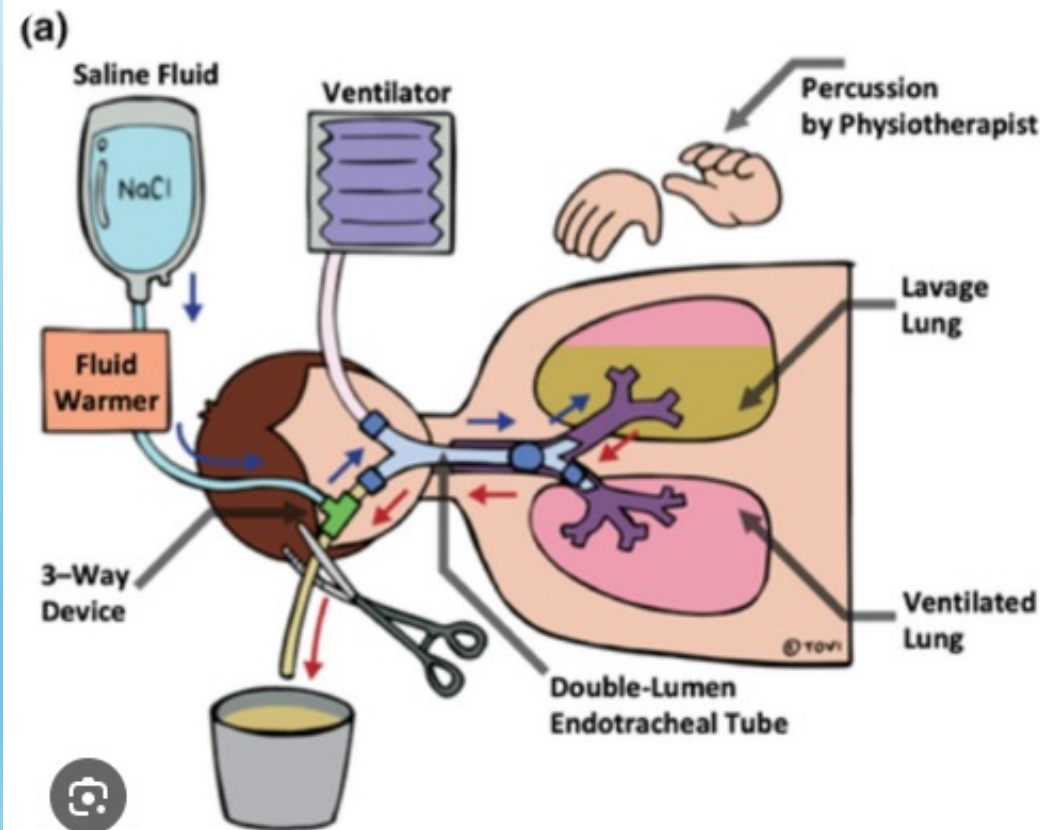
Pulmonary Alveolar Proteinosis

- Caused by abnormalities in GM-CSF, which affects production and **clearance of surfactant** by alveolar macrophages
- Accumulation of lipoproteinaceous material in alveoli
 - Minimal lung inflammation
 - Lung architecture preserved
- Usually autoimmune, less common with hematologic abnormalities

PAP - treatment

- Whole lung lavage
 - Severe cases
 - Gen anesthesia with DL ETT for single lung ventilation
 - 1L at time, up to 15L per lung
 - Initial lavage milky, progressively clears
- GM-CSF
 - Mild-moderate cases
 - Nebulized better than SQ

Whole Lung Lavage



Whole Lung Lavage

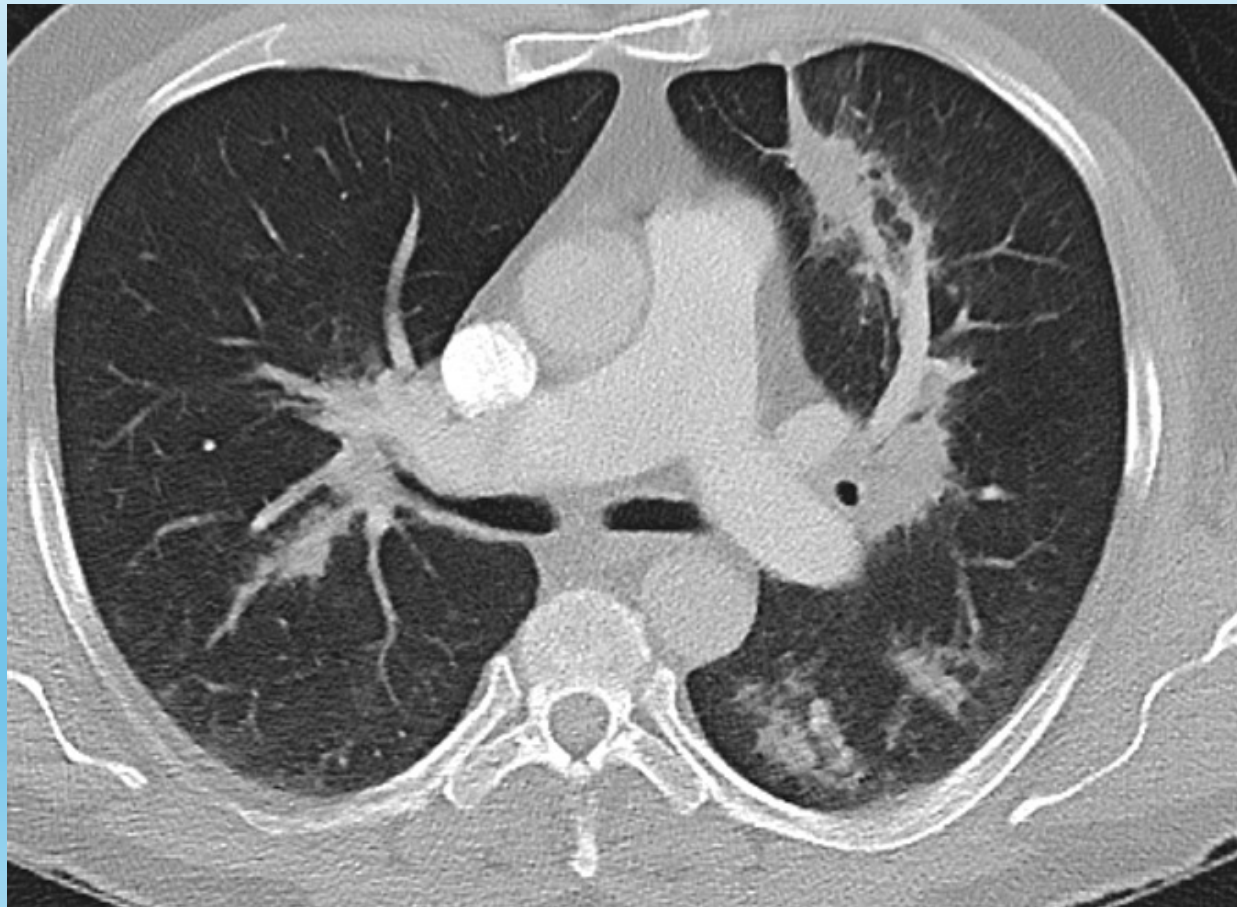


Disclaimer

63yo man with weakness, fatigue, cough

- SOB, occ fever
- WBC 9, 11% eos
- CRP 195 (<10)
- Procal mildly elevated
- Cultures negative
- Echo with valvular vegetations c/w endocarditis
- Treated with antibiotics
- Stabilized, d/c home

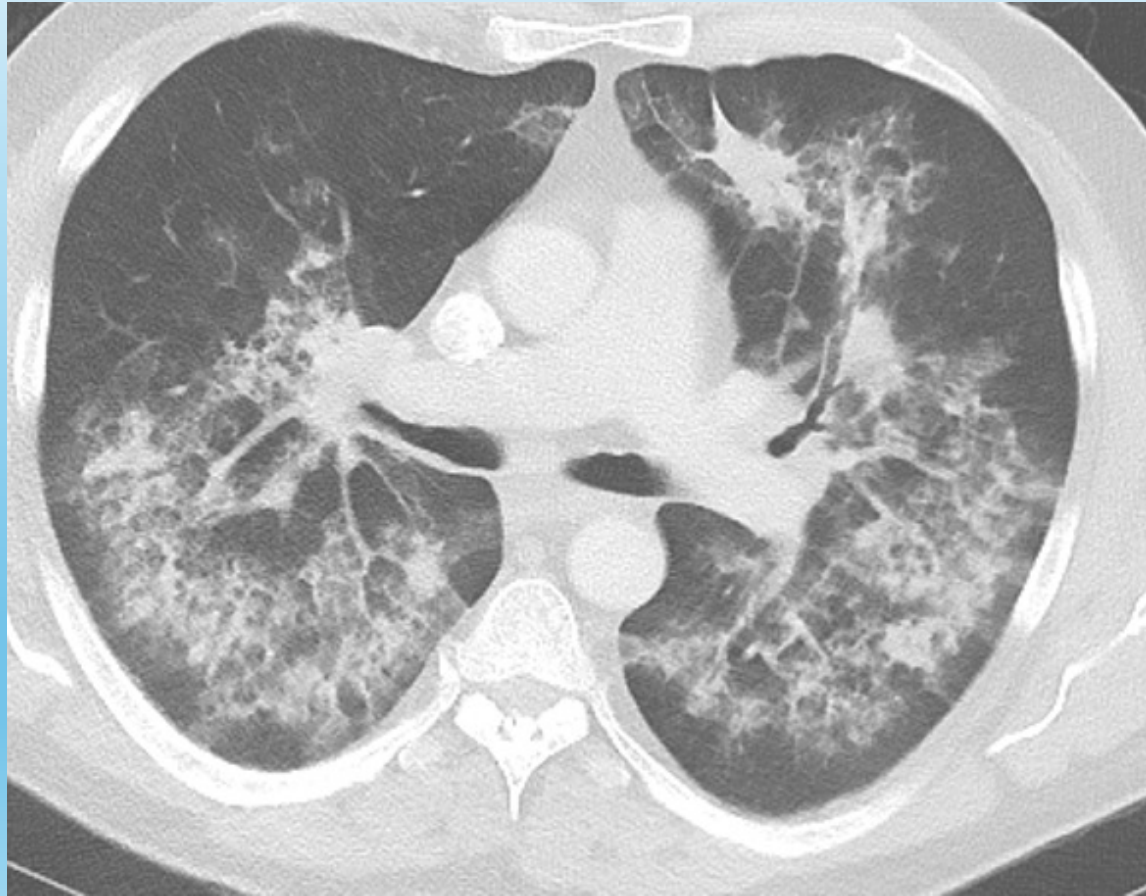
CTPA



1 week later

- Readmitted with SOB and hypoxemia
- Worsening cough, occ dark sputum, possible blood
- Afebrile, O2 sat 80s on RA
- WBC 13, 9% eos

Chest CT



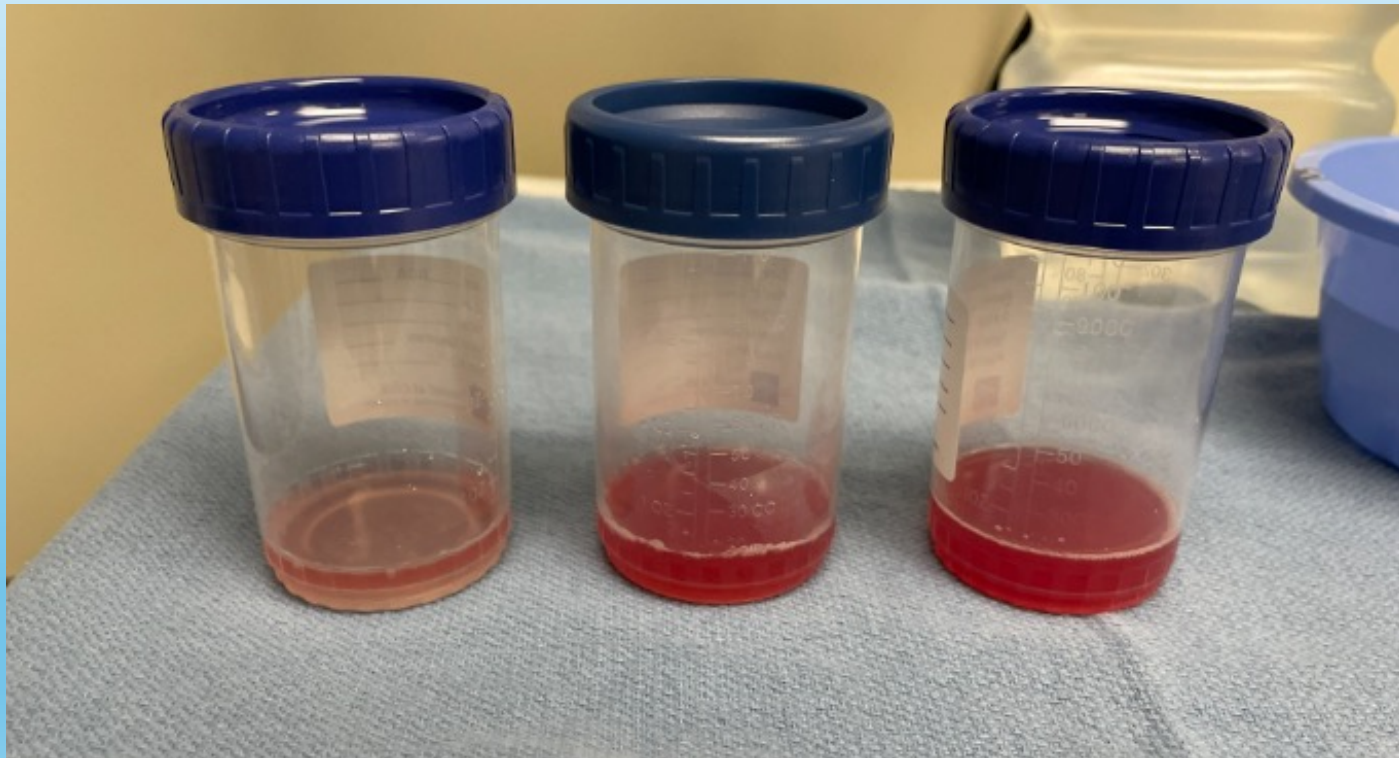
Bronchoscopy



Bronchoscopy



BAL fluid



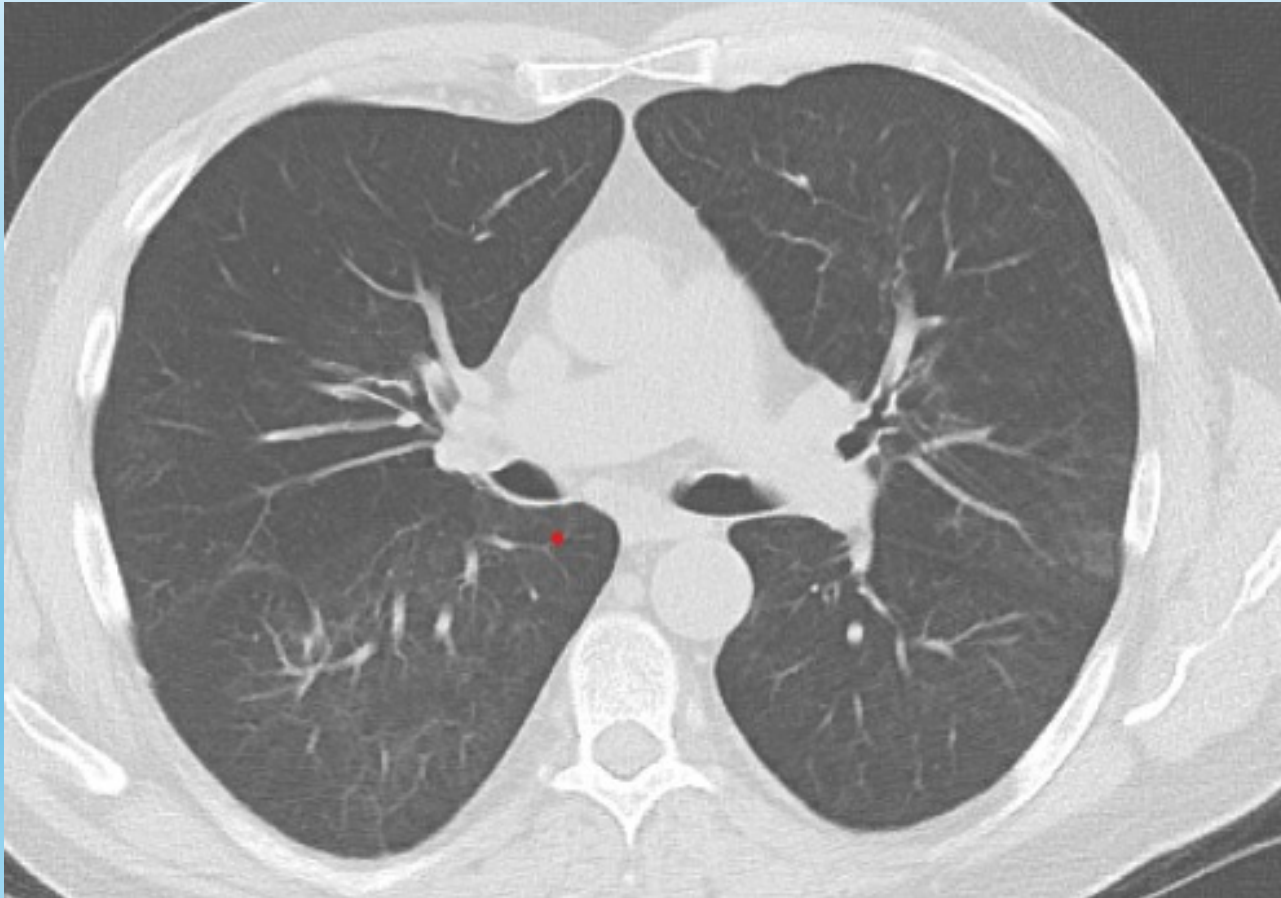
Dx: Diffuse Alveolar Hemorrhage (DAH)

- Pulmonary Vasculitis – ANCA, anti-GBM disease
- Lupus
- Antiphospholipid Antibody Syndrome (APS)
- Coagulopathy
- Congestive Heart Failure
- Crack cocaine
- ***Classic finding is progressively bloody return on BAL***

63yo with DAH

- ANCA +
- Dx: Eosinophilic granulomatosis with polyangiitis (EGPA) with multiorgan system involvement
- Improved with corticosteroids, Rituxan

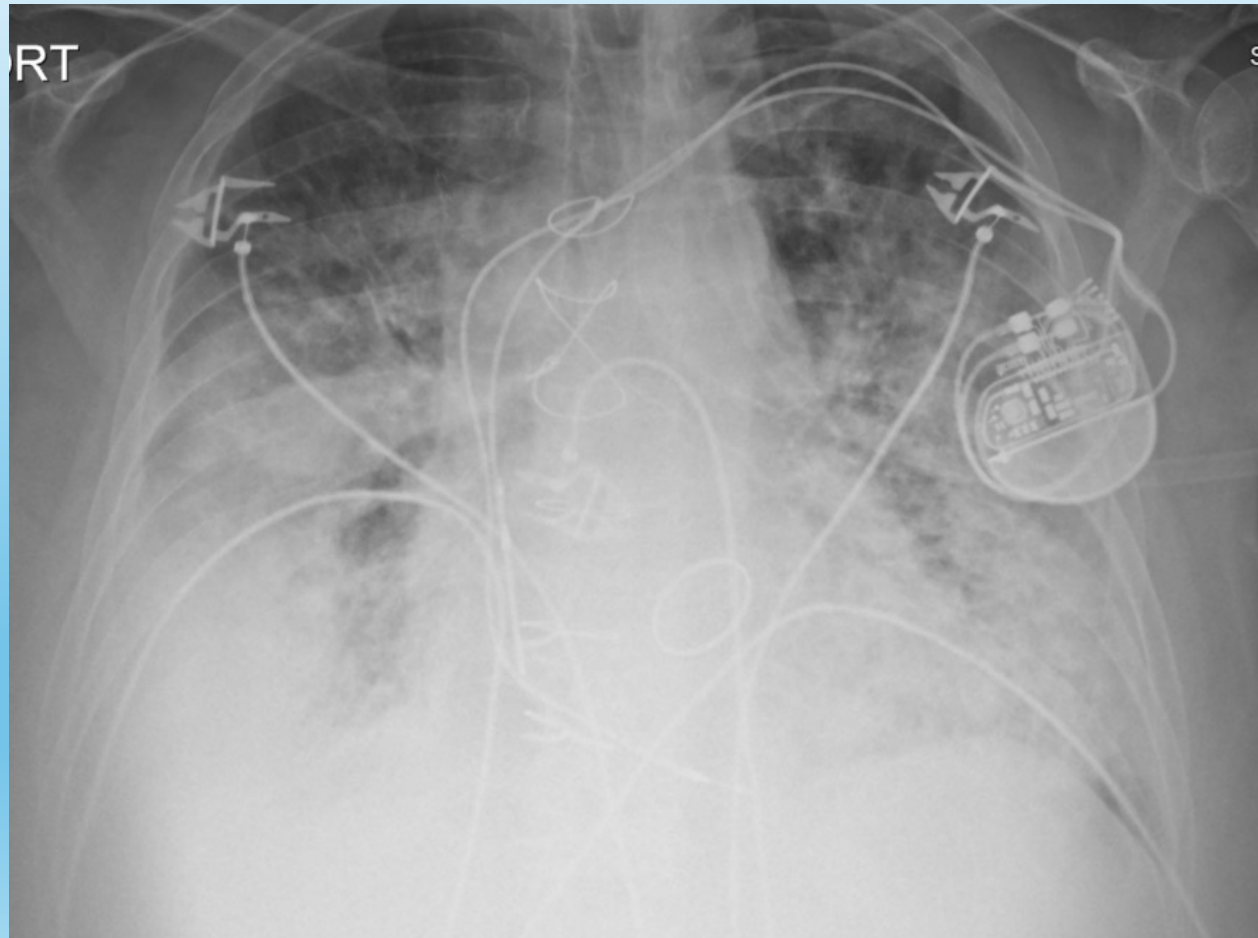
F/u CT 2 months later



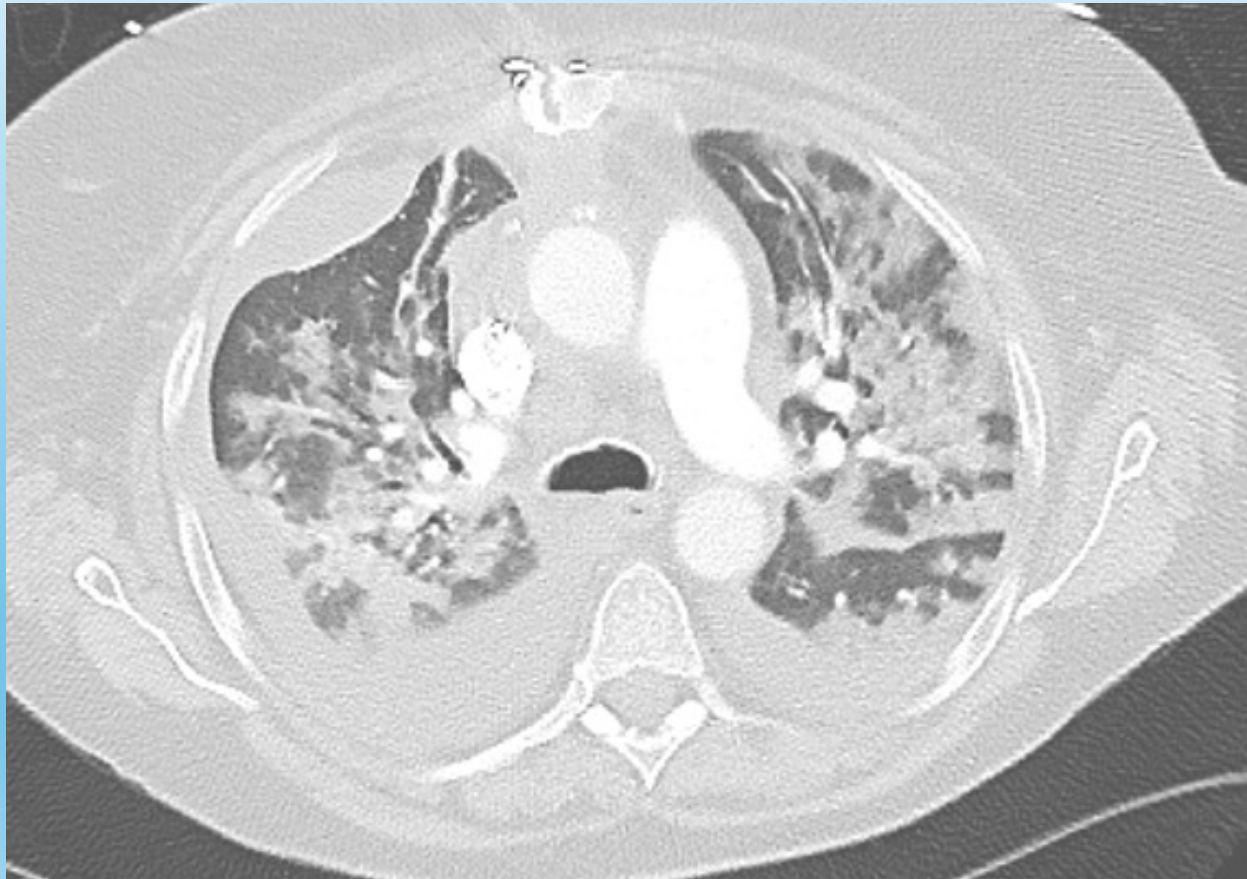
65yo woman with SOB

- h/o **Lupus**, on **MMF**, **APS**, on **warfarin**, afib s/p ablation, on **Amiodarone**, pacemaker
- Worsening SOB for 2 weeks, cough, swelling
- Afebrile, HR 130 – flutter
- WBC **17k**
- BNP **4,400**
- PCT **0.26**
- Resp PCR negative
- Started antibiotics
- Echo normal

CXR



Chest CT



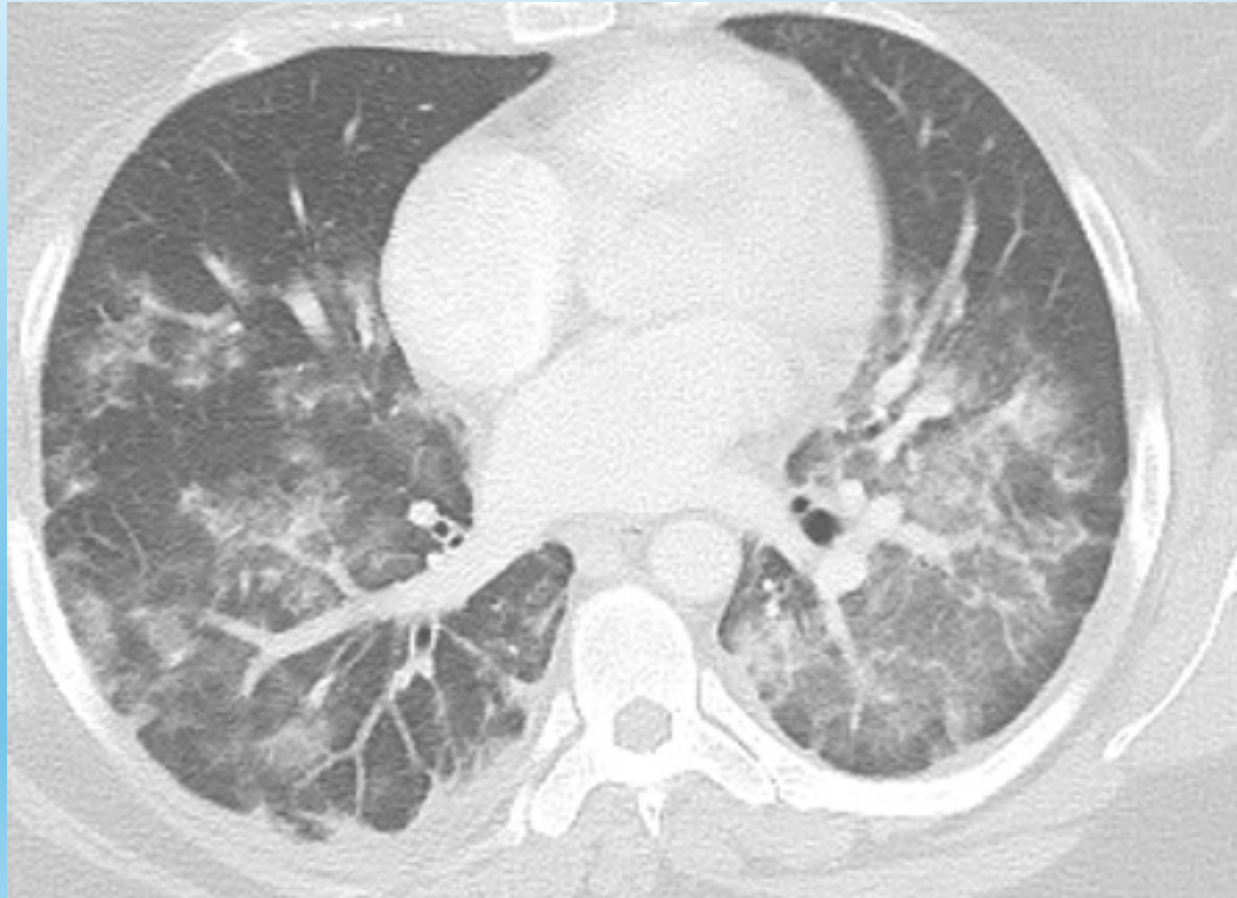
65yo with SOB

- Progressive hypoxemia, increased WOB
- Started NIV
- Started diuresis
- No improvement
- Intubated
- *Transferred to Boise*
- ***Bronchoscopy***

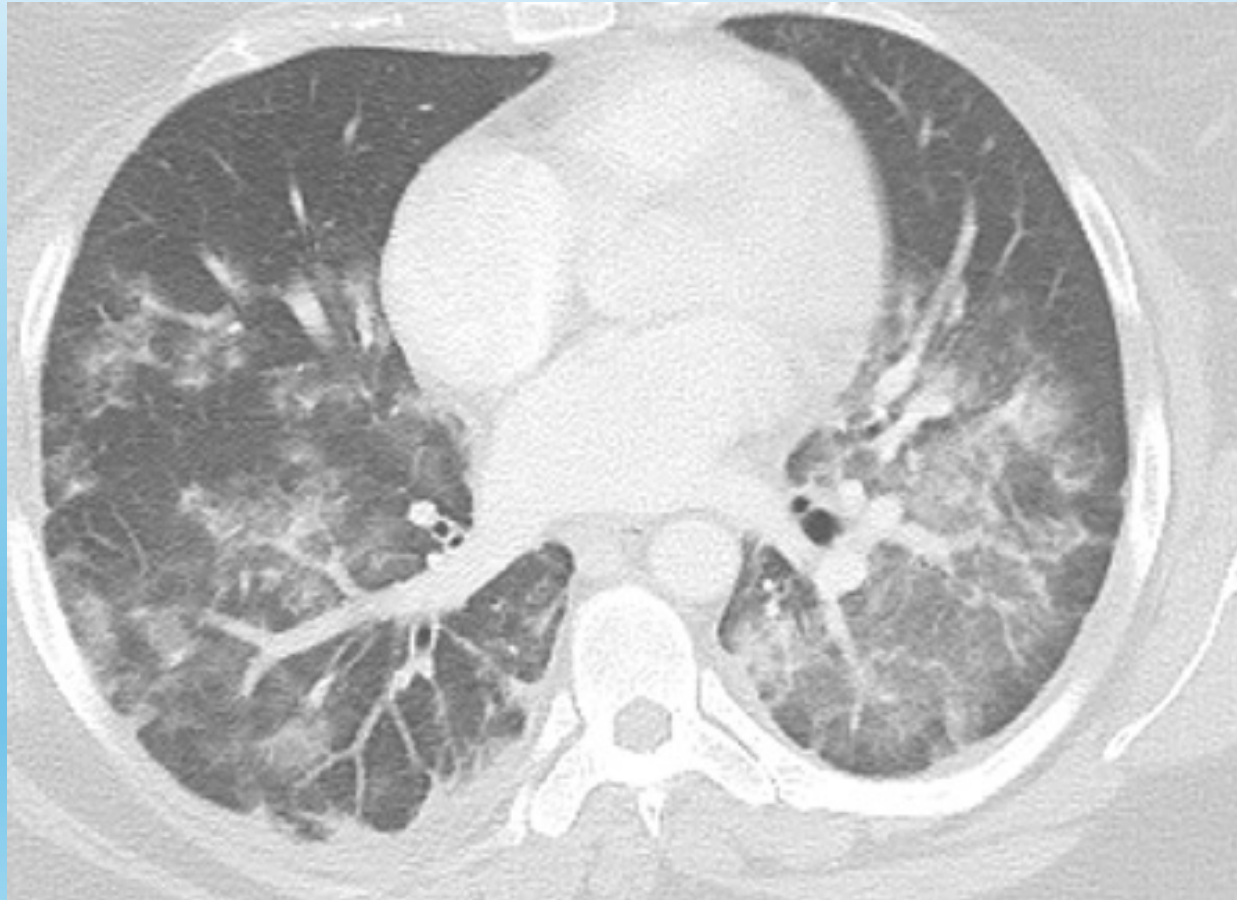
65yo with SOB

- BAL fluid
 - Clear
 - Bland
 - Micro negative
- Pacemaker interrogation showed HR 130 for 3 weeks due to Aflutter
- Improved with heart rate control, more diuresis
- Extubated
- Dx: **CHF/HFpEF, likely due to prolonged tachyarrhythmia**

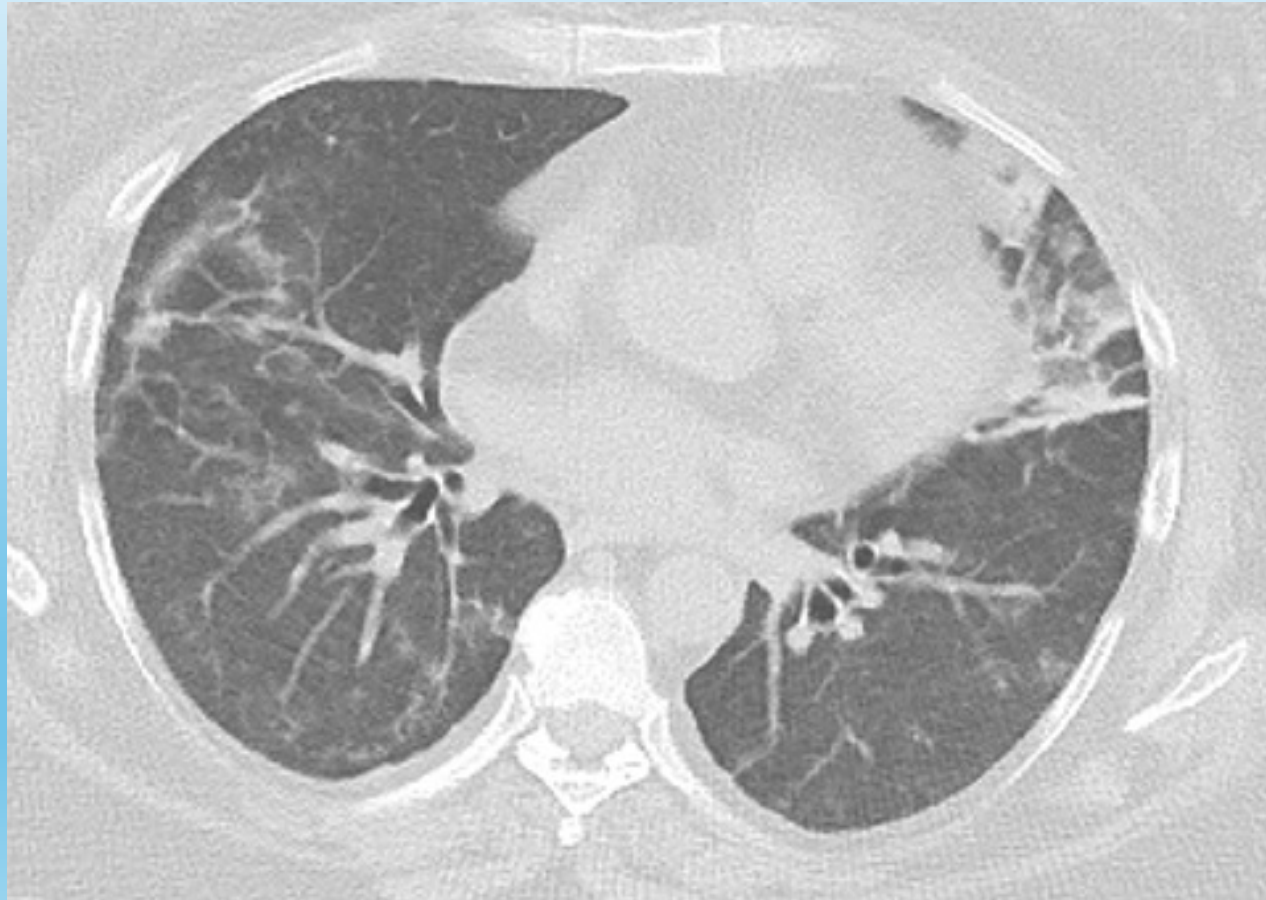
27yo with Lupus, MMF, APS, Warfarin



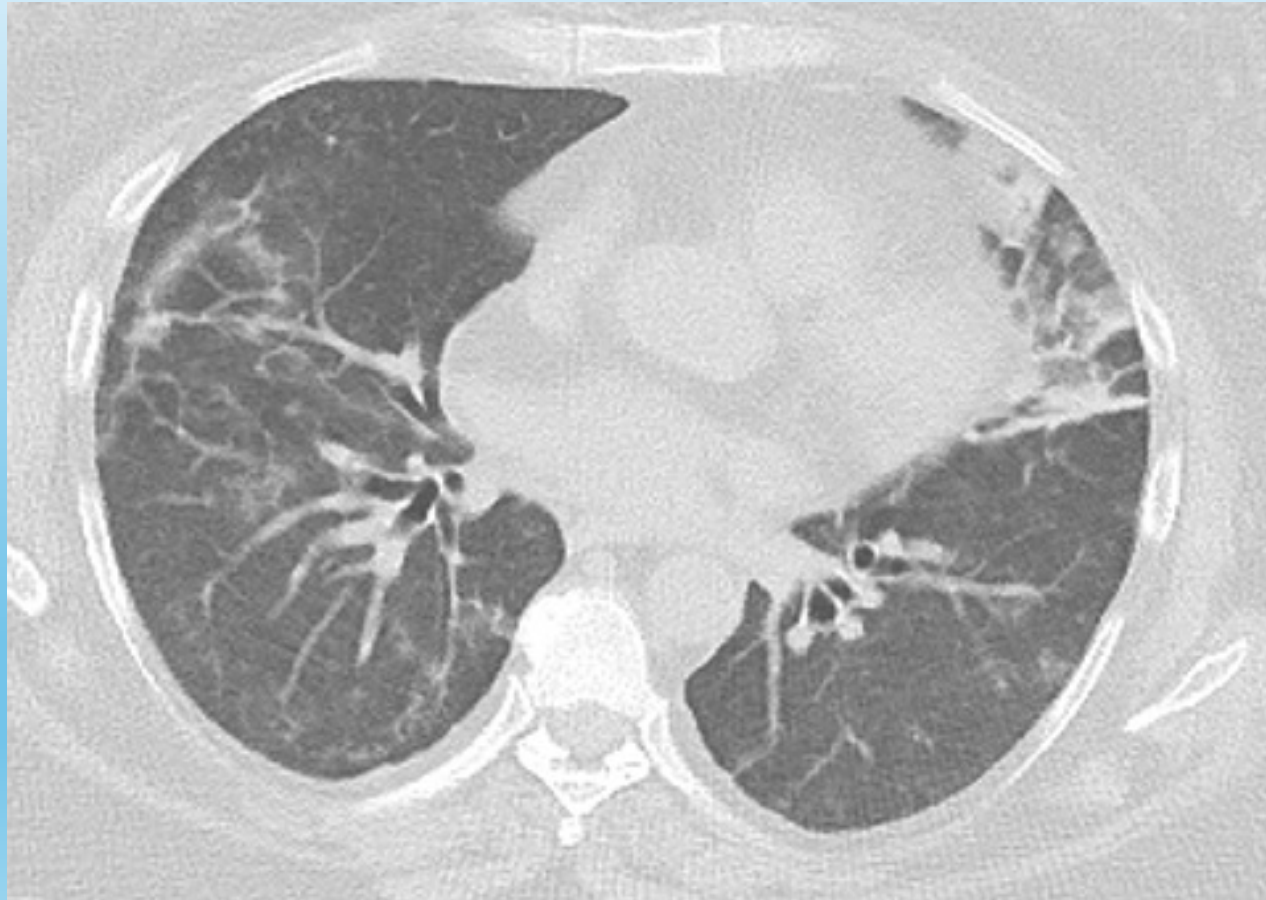
Dx: Lupus Pneumonitis



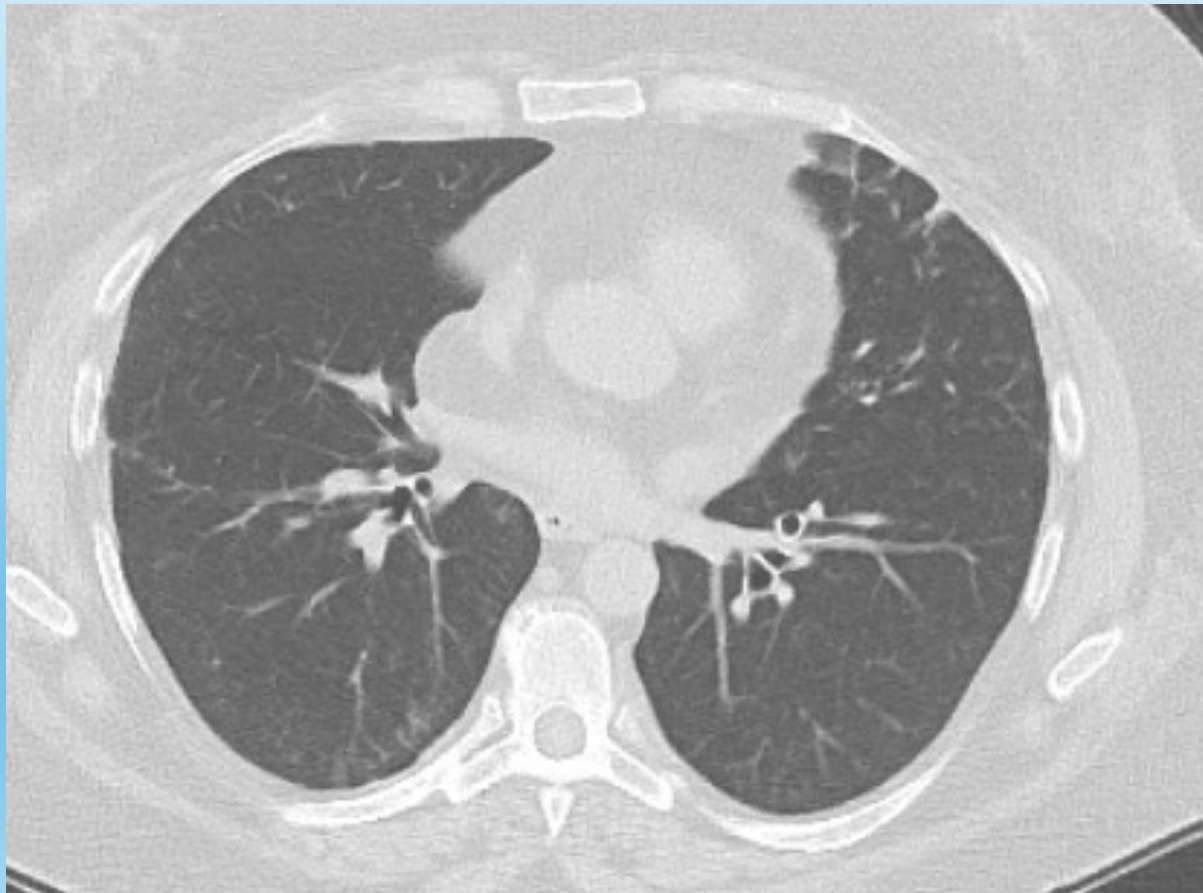
49yo with breast ca, on chemo, lymphopenia



Dx: Taxane toxicity



Taxane toxicity after Prednisone



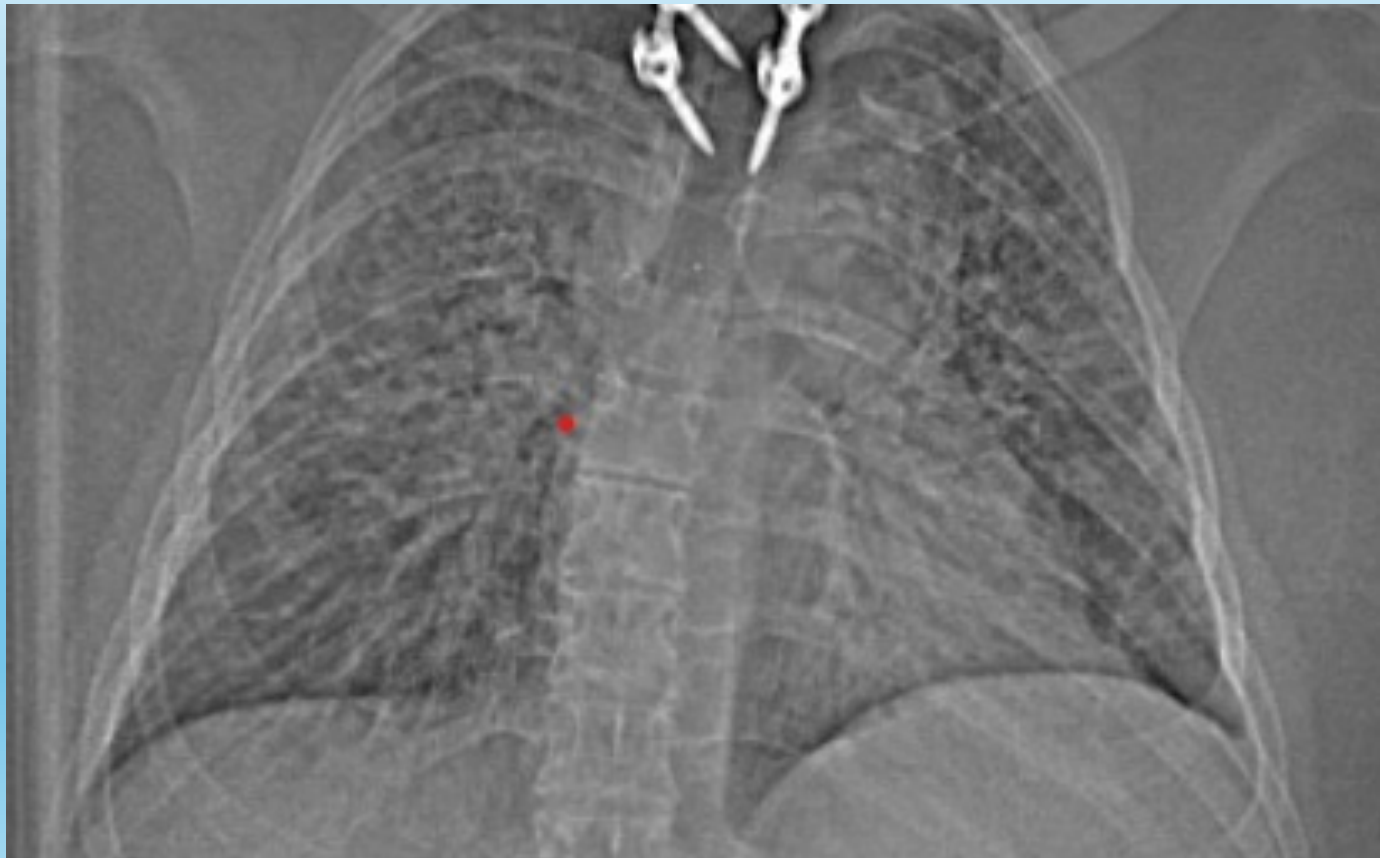
21yo with tib-fib fx, 2d later with fever, sob,
hypoxemia



Dx: Fat Embolism Syndrome



CT scout film



FES 6 days later



PFT BASICS:

What You Need To Know Outside The PFT Lab

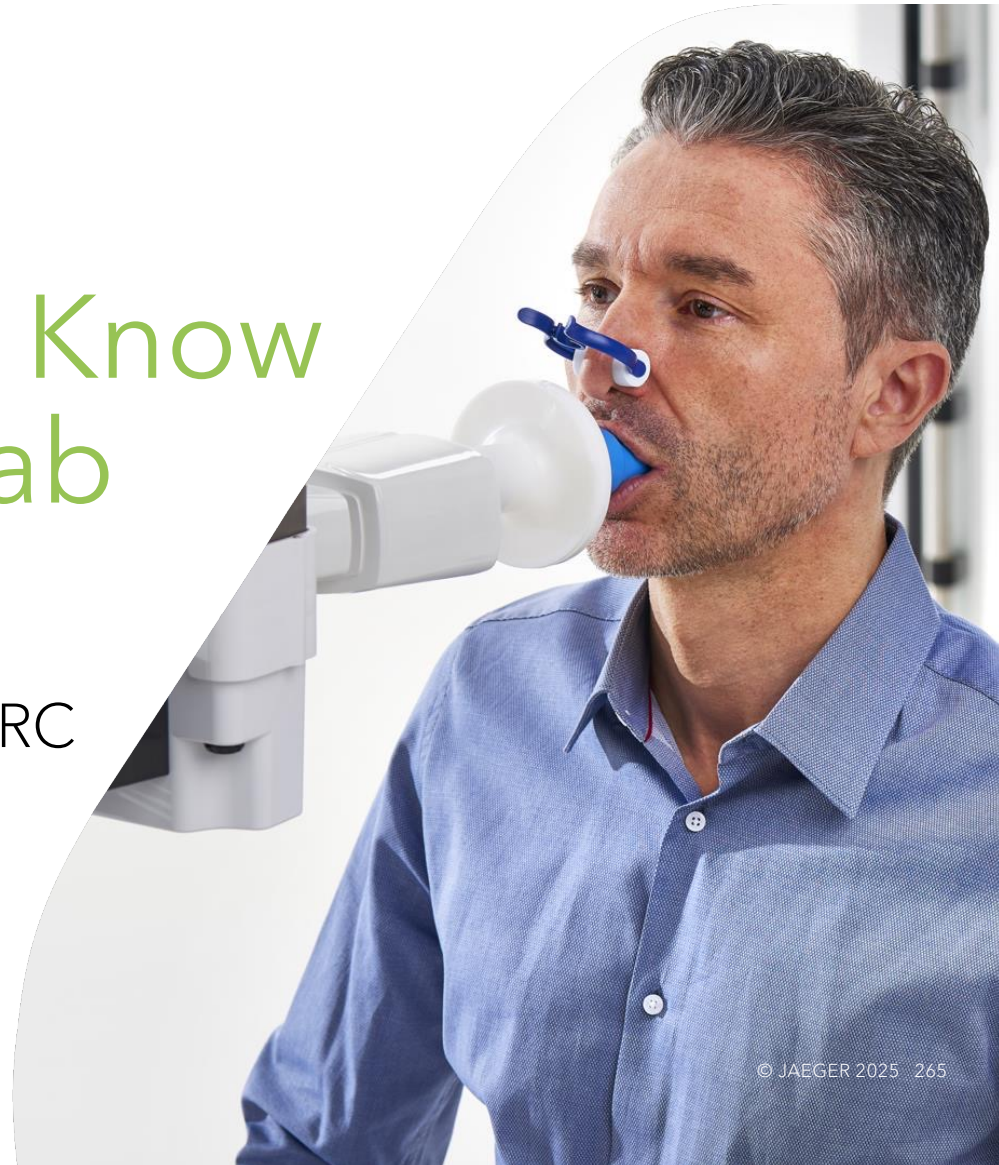
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Conflicts of Interest



I am a Sales Consultant for Jaeger Medical America.

I own Apex Pulmonary.

Any use of brand names is not in any way meant to be an endorsement of a specific product, but to merely illustrate a point of emphasis.

I am a PFT geek.



Objectives

1. Understand basic PFT measures and how they relate to lung function
2. Understand the basics of PFT interpretation
3. Understand some of the impacts of using race neutral predicted equations

Indications For Testing

Diagnostic

- **Evaluate symptoms, abnormal test**
- **Effect of disease on pulmonary function**
- **Screen at risk individuals**

Monitoring

- **Assess therapeutic intervention**
- **Describe course of diseases that affect lung function**
- **To monitor reactions to drugs known to affect pulmonary function**

Disability/impairment evaluations

- **Assess patients as part of a rehab program**
- **Assess individuals for legal reasons**

Public Health

- **Epidemiological surveys**

Contraindications For Testing

- Within one week of a M.I. **-ABSOLUTE-**
- When Pt. can't perform test optimally
 - Chest or abdominal Pain - fx ribs, pulled muscle
 - Oral/facial pain aggravated by the mouthpiece
 - Stress incontinence
 - Dementia or altered mental state
 - Persistent cough
- Unstable Hemodynamics
- Active Hemoptysis, Active TB
- Unstable Pneumothorax or Chest Tube in place
- Recent Chest, Abdominal, Face or Eye surgery

TIP- If a patient is on lifting restrictions, they shouldn't be doing testing

Common PFT Measures

Spirometry – how much and how fast

Lung Volume – total capacity of the chest

Diffusion Capacity – how well getting O₂
in and CO₂ out

Spirometry

Must know terms:

VC – Vital Capacity

FVC – Forced Vital Capacity

FEV1 – Force Expiratory Volume in one second

FEV1/FVC – Percentage of VC that can be forced out of the lungs in one second

Four distinct phases of an FVC

- 1) Maximal inspiration
- 2) Maximal "Blast" out
- 3) Continue blowing until all their air is out
- 4) Big, deep breath back in

Which is the most important?

Maximal Inspiration!

All of these are important, but without first taking a deep breath the rest are meaningless.

Spirometry

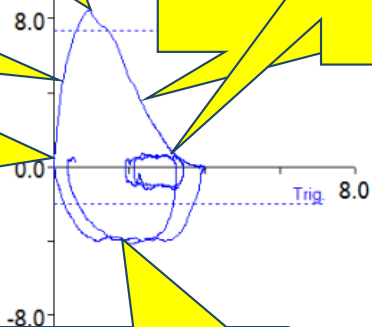
#3 Is there a good peak in flow?

#5 Is there a gradual decrease in flow

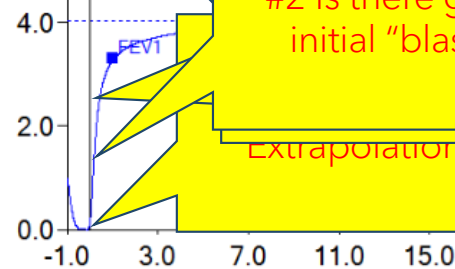
here appear plateau?

#2 Is there a good

#1 Is there Back Extrapolation?



#7 Is there a good, fast full inhalation?



#2 Is there good initial "blast"?

Extrapolation?

there a , fast full lation?

Common problems - Spirometry

Less than maximal breath in to start test

Hesitation at beginning of blowing

Poor effort

- Not exhaling completely
- Got lightheaded so will not try as hard next time
- Takes a small breath and continues to exhale
- Does not reach a plateau or zero flow
- Patient passes out
- Less than maximal effort

Cough

Tongue or Epiglottis in the way

Equipment issues

Spirometry

What Spirometry tells us

Condition of Airways

Intrathoracic

Extrathoracic

Airway Hyperresponsiveness

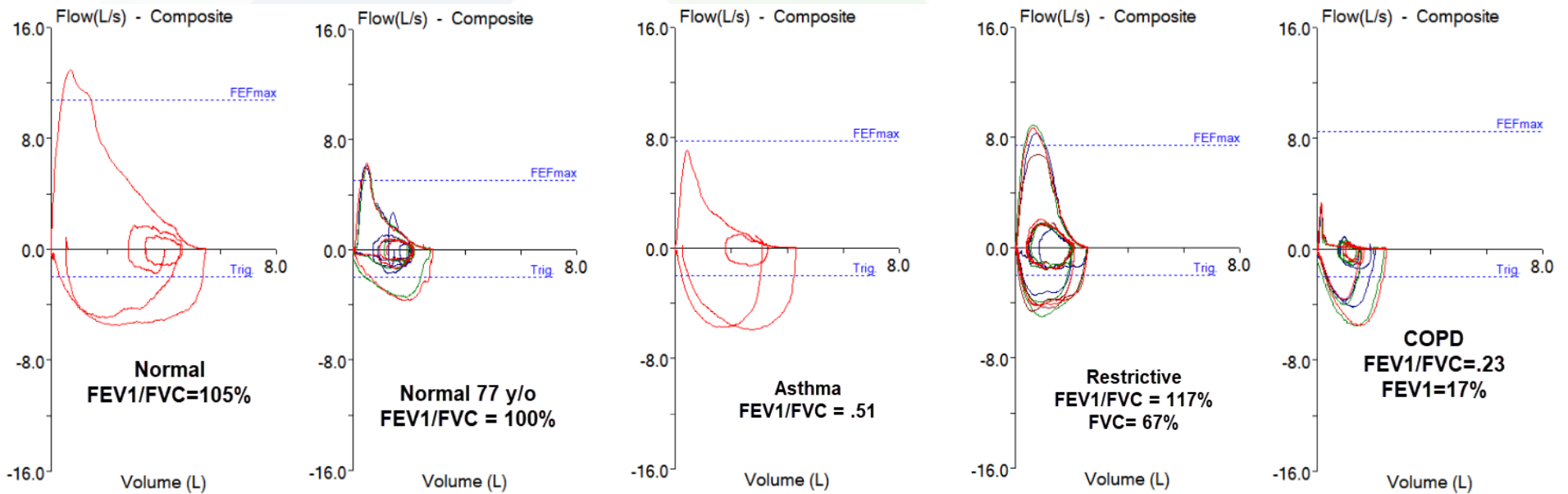
What Spirometry DOESN'T tell us

Restrictive disease

Emphysema

Cancer

Typical Flow Volume Loops



Lung Volume Testing

- 1) Body Plethysmography - Requires a body box
- 2) Nitrogen Washout - "Open" technique, uses demand valve
- 3) Helium Dilution - "Closed" technique requires desiccants
- 4) Imaging Techniques - Uses CT exam

Lung Volume Testing

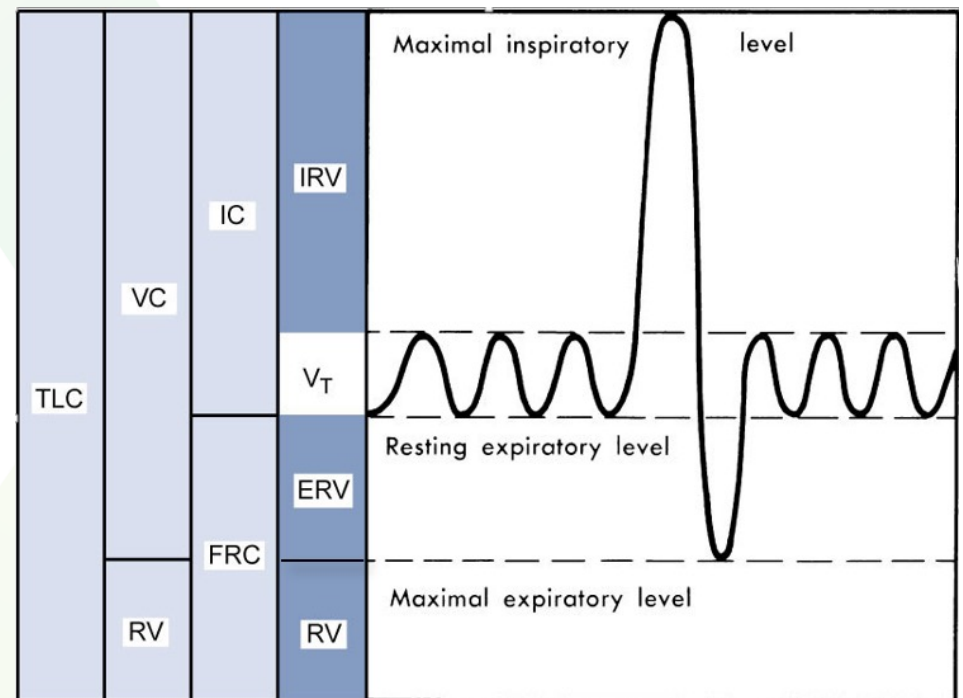
Must know terms:

FRC - Functional Residual Capacity

TLC - Total Lung Capacity

RV - Residual Volume

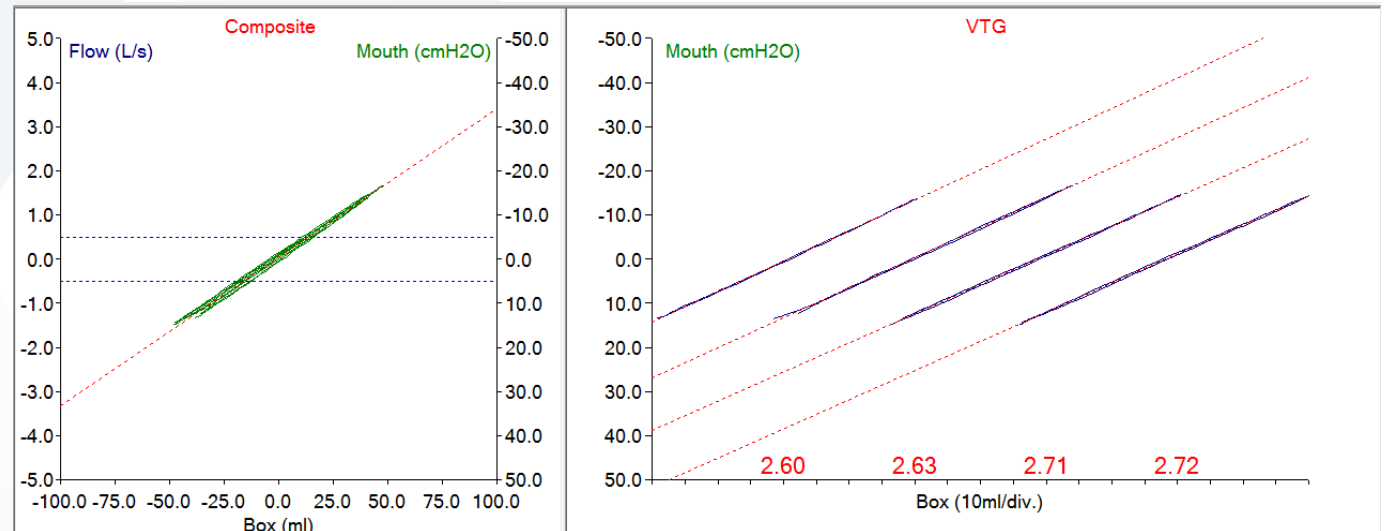
RV/TLC - Percentage of TLC that CANNOT be exhaled



Plethysmography

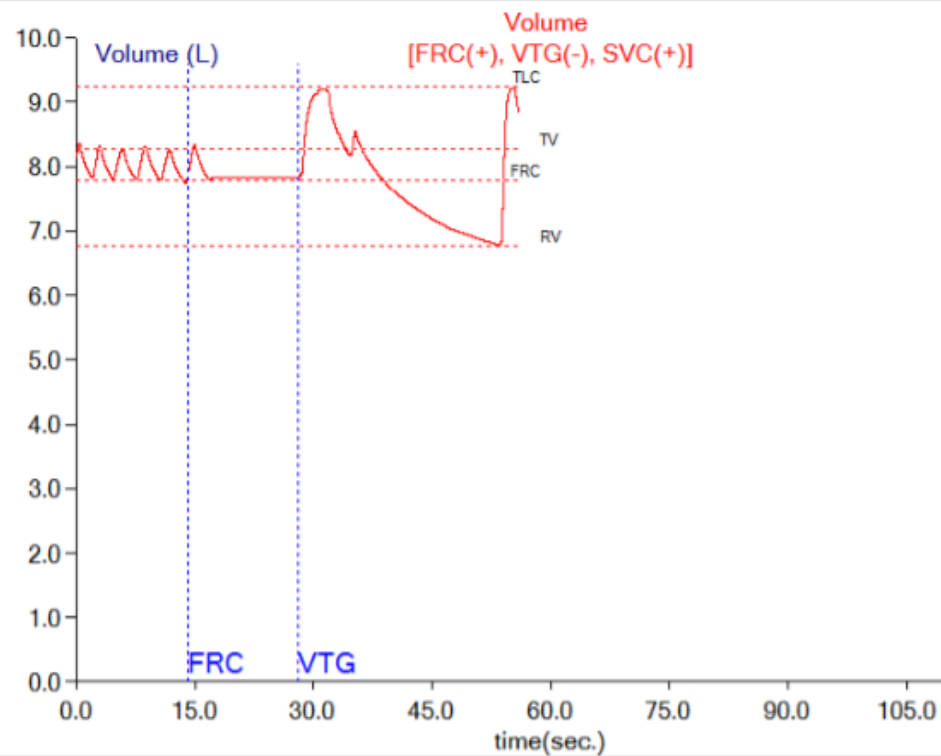
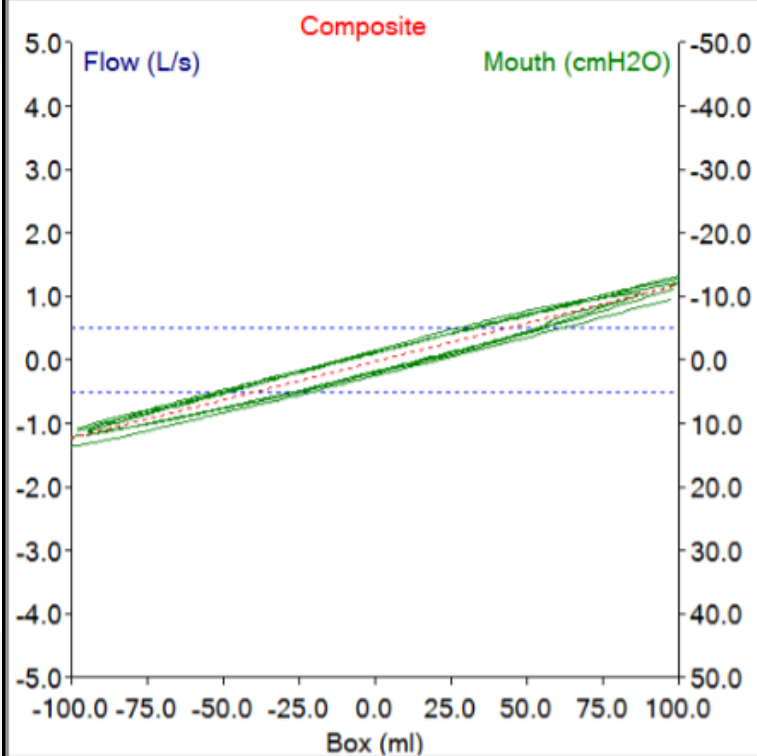


 J A E G E R



Uses Boyles Law – $P_1V_1=P_2V_2$

Start Test ▾ Choose ▾ X Delete Save Reported ▾ Undo Rx Drug RawBTPS(+) RawBTPS(-)



	Pred. [LLN]	%Pred.	Reported	%Comp	Current	Effort#: 1	* Effort#: 2					
FRC (L.btps)	3.77 [2.78]	206	7.75	101	+ 7.79	+ 7.71	+ 7.79					
VC (L.btps)	4.56 [3.60]	< 54	* 2.47	100	+ 2.47	- 2.28	+ 2.47					
TLC (L.btps)	7.25 [5.62]	126	9.12	101	+ 9.25	+ 9.00	+ 9.25					
RV (L.btps)	2.69 [2.02]	247	6.65	102	+ 6.77	+ 6.72	+ 6.77					
RV/TLC (%)	37.00	196	73	101	73	75	73					
IC (L.btps)	3.48	< 39	1.37	106	1.46	1.29	1.46					
TV (L.btps)			0.51	97	0.49	0.52	0.49					
ERV (L.btps)	1.07	102	1.10	92	1.01	0.99	1.01					
VTG (L.btps)			7.78	101	7.83	7.73	7.83					
Vtg (frequency) (/min)			32.78	91	29.72	35.85	29.72					
RAW (cmH2O/L/s)	1.18											



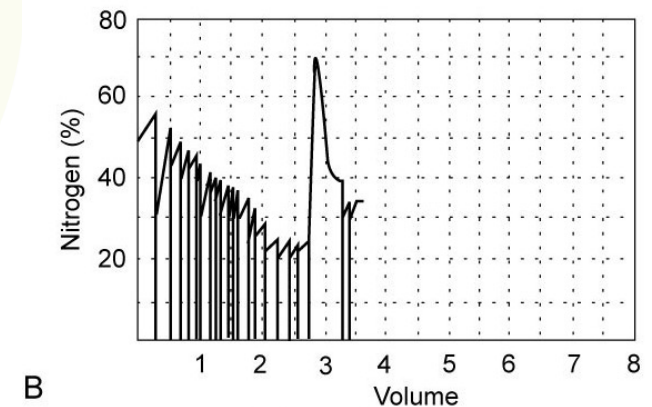
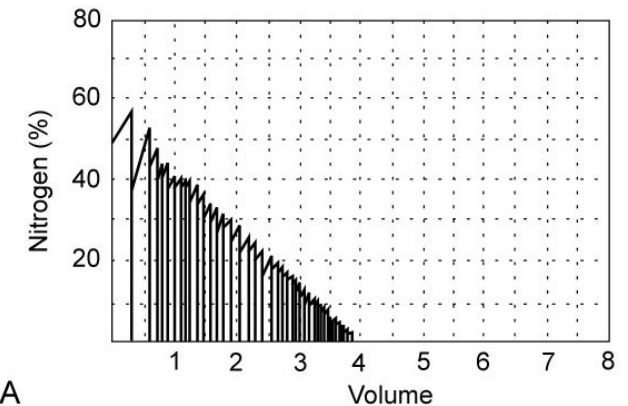
Nitrogen Washout

Top: Typical N₂ washout pattern

Notice gradual decline in percentage and plateau at the end.

Bottom: Typical graph when there is a leak in the system

Most common place for a leak? Mouthpiece!



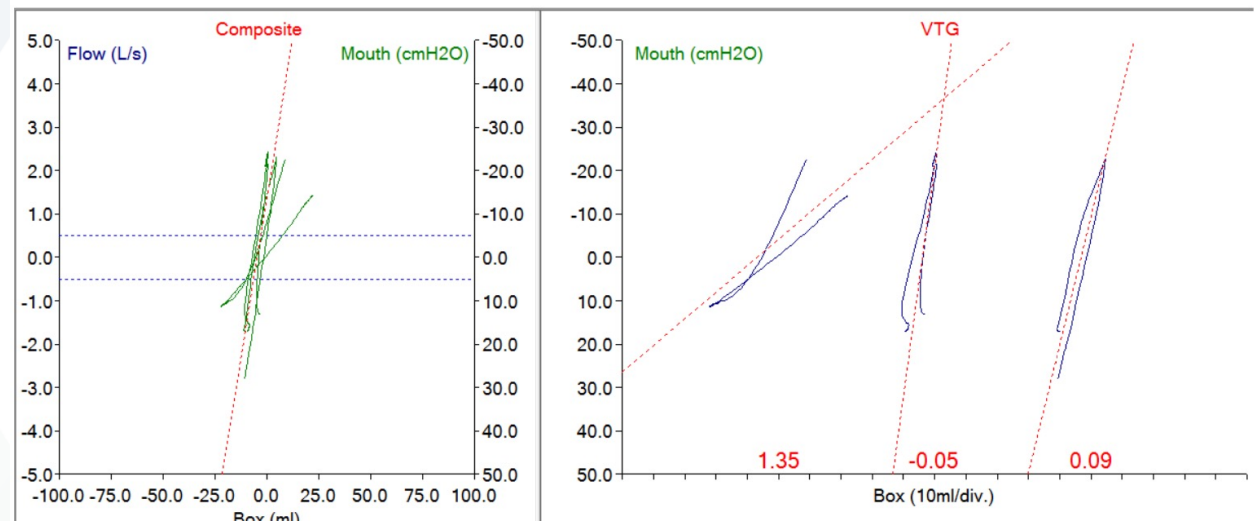
Common problems - Lung Volumes

Plethysmography

- Claustrophobia
- Size of the patient
- Panting speed
- Glottic closure

Nitrogen Washout

- Saliva
- Air leaks
- Jaw pain
- Anxiety
- Fatigue



Diffusion Capacity - DLCO

Must know terms:

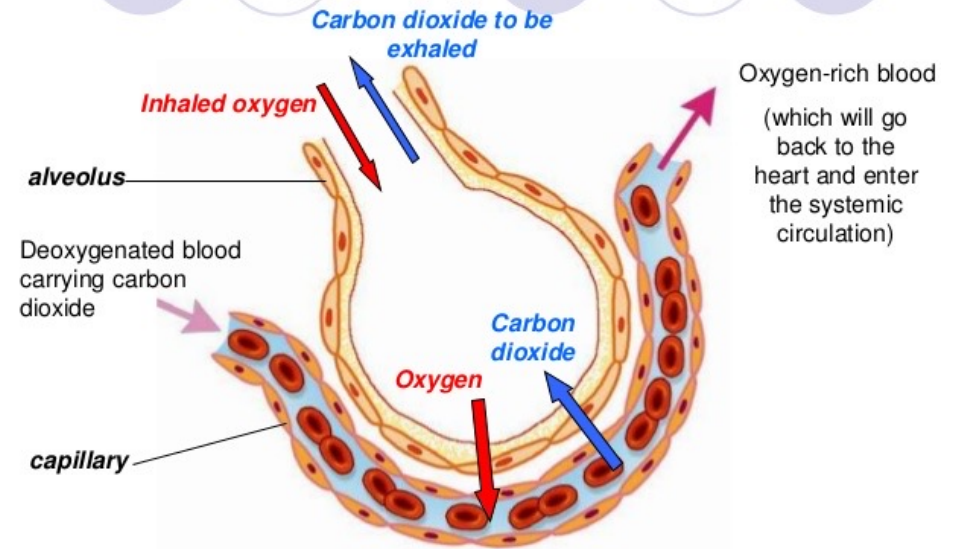
DLCO - Diffusion of Lung Carbon Monoxide

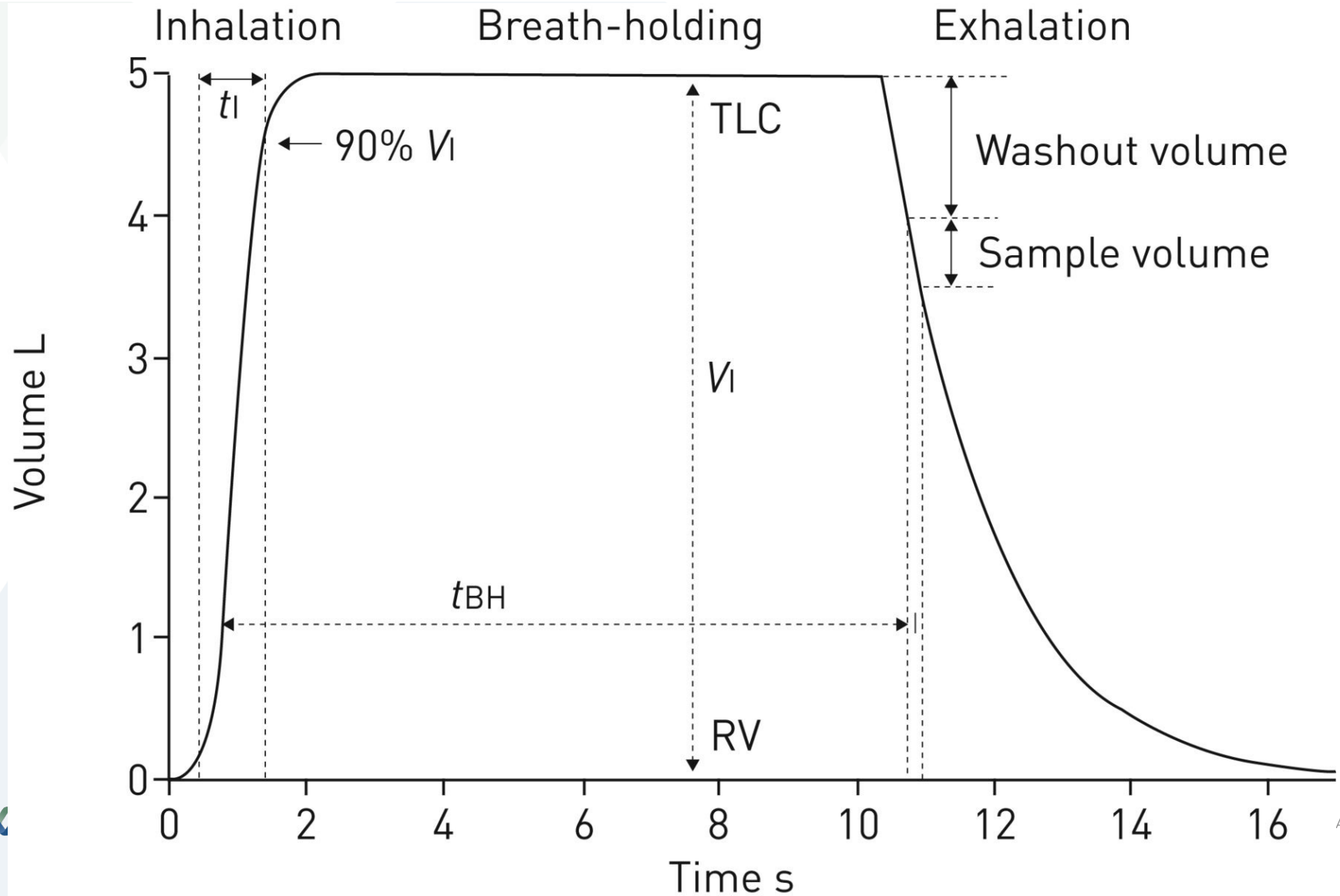
VA - Alveolar Volume

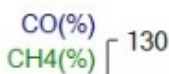
KCO - Carbon monoxide transfer coefficient

IVC - Inspiratory Vital Capacity

Gaseous Exchange in the Alveolus





[illegible]

Common problems – Diffusion Capacity

Not exhaling all the way to RV – both at beginning and at end of test

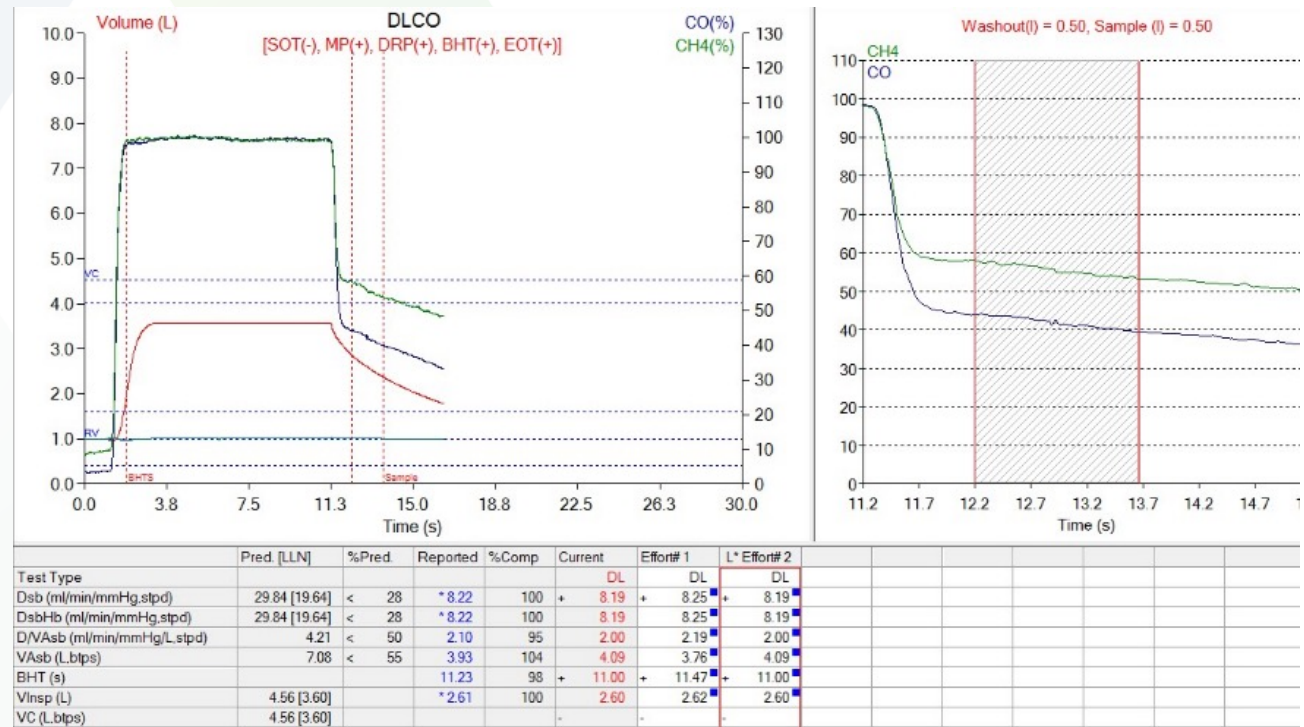
Not inhaling all the way to TLC

Cough

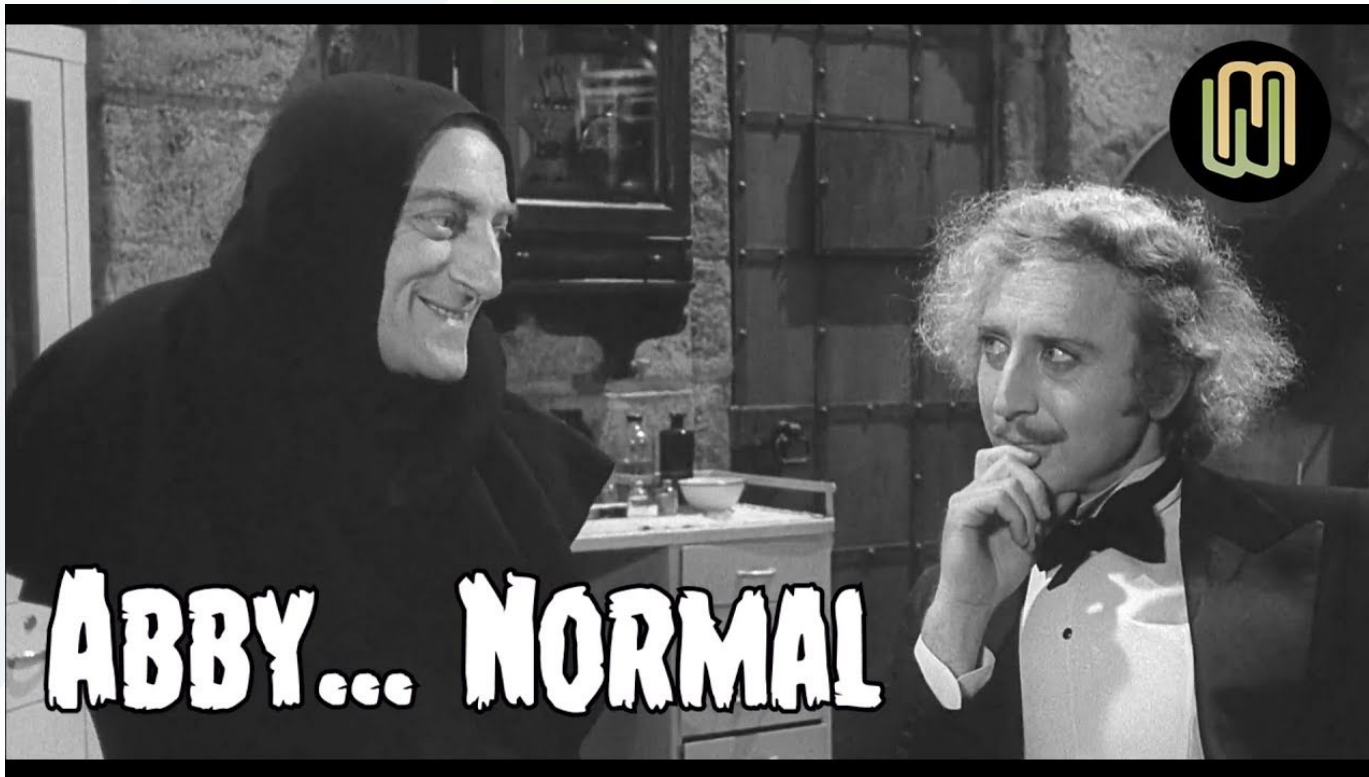
Smoking

Oxygen use

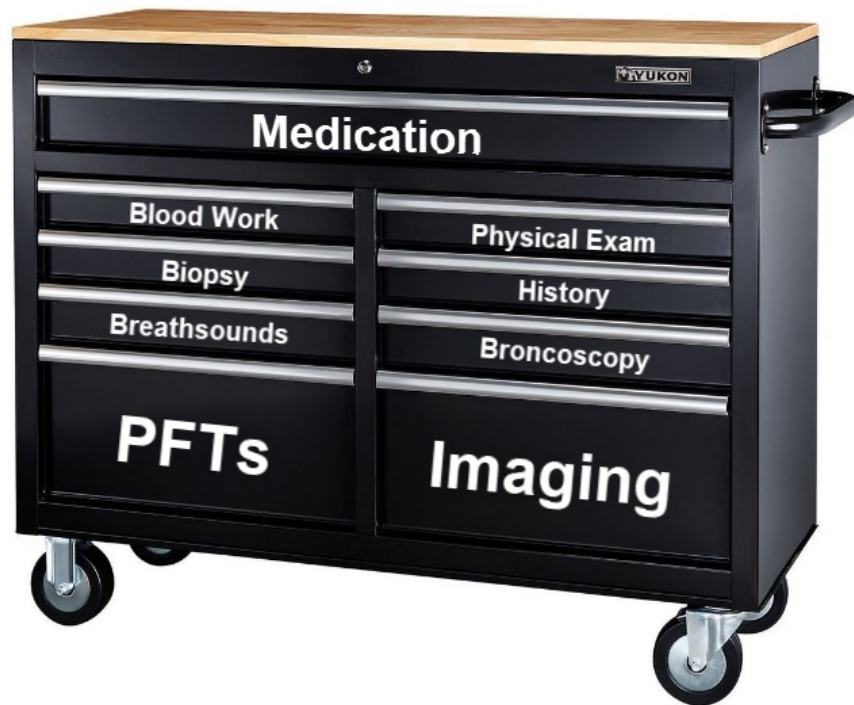
Wait times



Determining “normal vs abnormal”



Determining “normal vs abnormal”



PFTs characterize lung function

PFTs do not diagnose disease

99.99% Accuracy

"There is no pregnancy test for asthma"



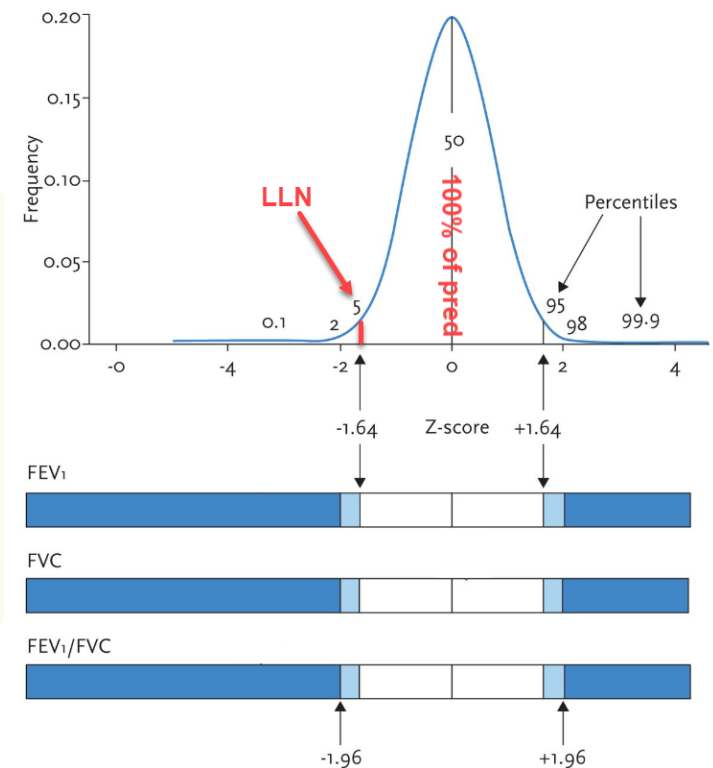
"Spirometry is not a pregnancy test for COPD"

Determining “normal vs abnormal”

The ERS/ATS Technical Standard for interpretation states:

The 5th and 95th percentile limits (-1.645 and +1.645 z-scores) of the healthy population can be used to identify individuals with unusually low or high results, respectively

Population defined z-scores or percentile values describe the chance the observed result falls within the distribution of values in healthy individuals (Figure 2). At the 5th percentile (corresponding to a z score of -1.645), there is a 5% chance that the results in a healthy individual would be at or below this level.



Determining “normal vs abnormal”

Fixed Cut-off

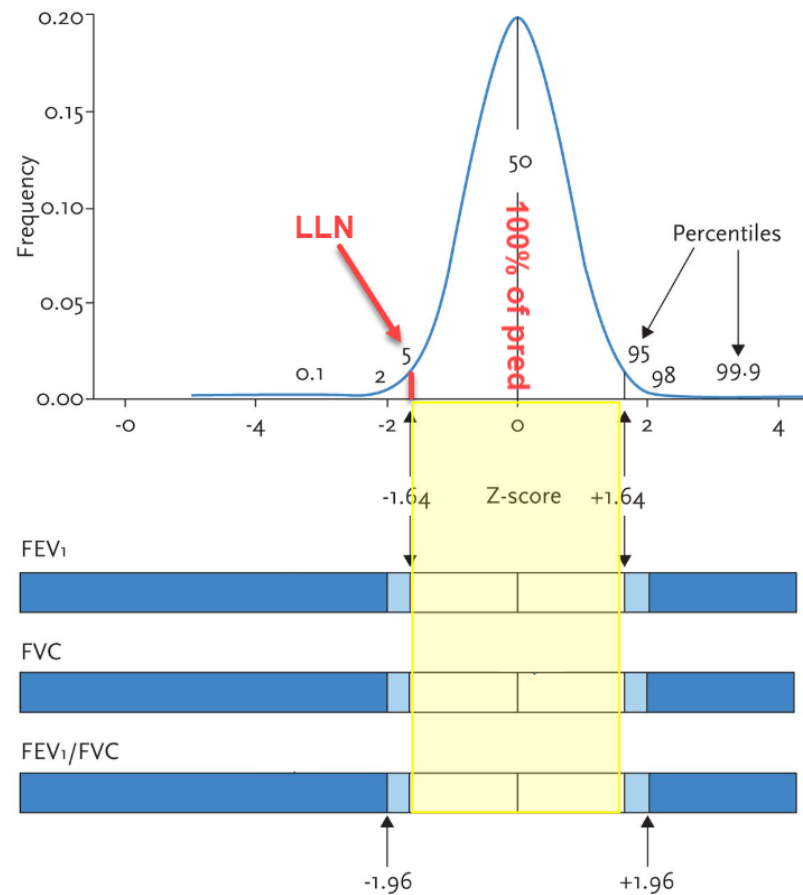
FVC	75-80%
FEV ₁	80%
FEV ₁ /FVC	70%
TLC	$\leq 80\%$ or $\geq 120\%$
FRC	75-80% or 120%
RV/TLC	≤ 120 -125%
DLCO	70-75%

vs

Statistical Limits

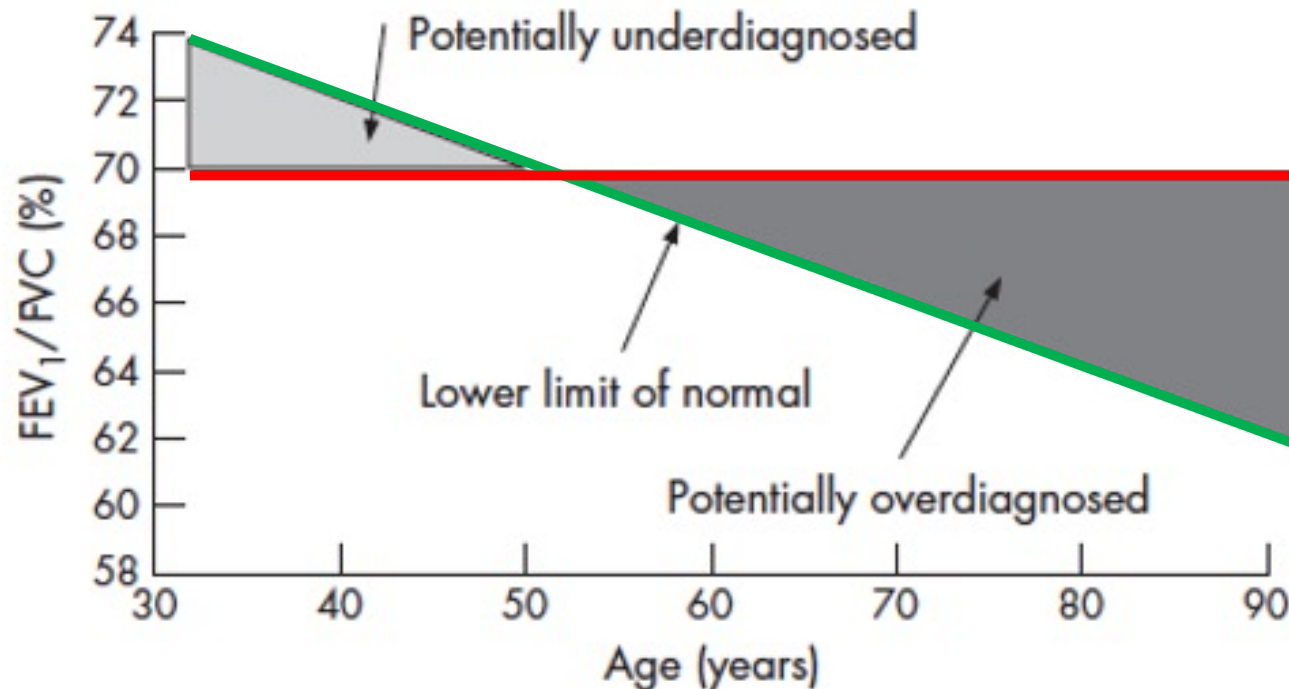
LLN/ <-1.64
LLN/ <-1.64
LLN/ <-1.64
LLN or ULN/ > 1.64
LLN or ULN/ > 1.64
ULN/ > 1.64
LLN/ <-1.64

Determining “normal vs abnormal”



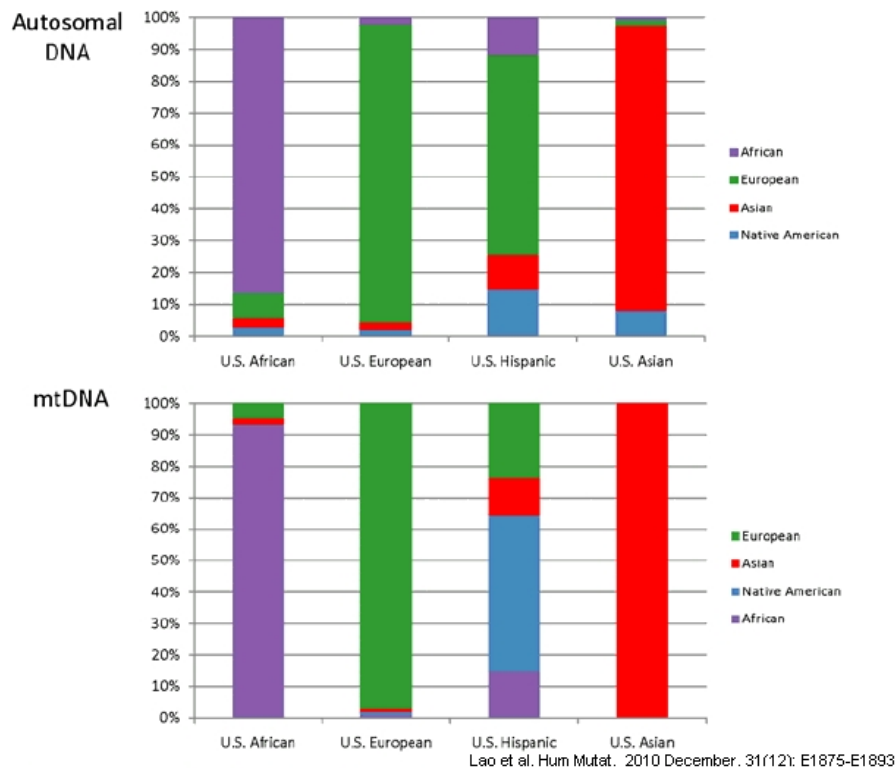
Determining “normal vs abnormal”

Fixed Cut-off vs Lower Limit of Normal(LLN)



Do we know who we are? We do!

Figure 2



Of 3,636 subjects of varying race/ethnicity, only 5 (0.14%) showed genetic cluster membership different from their self-identified race/ethnicity. Tang et al Am J Hum Genet. 2005 Feb;76(2):268-75. Epub 2004 Dec 29

Self-declaring race is accurate, less so for ethnicities Lao et al, Hum Mutat. 2010 December; 31(12): E1875-E1893

GLI Race Specific vs Race Neutral

ACHIEVING HEALTH EQUITY THROUGH RACE-NEUTRAL SPIROMETRY

Understanding the 2023 ATS position on Pulmonary Function Testing

Normalizing Lung Function for Spirometry



The idea that lung capacity of black persons is lower compared to whites is an example of scientific racism.¹

A race based correction factor is applied to spirometry, **reducing** the normal reference range of lung capacity by **10%-15%** for **Black** or **African-American patients**.²

Until recently, **social determinants of health** were largely **overlooked** and **technology has masked** how race-based algorithms are applied.^{2,3}

The Evolution of Race-neutral Spirometry

In 2023, **ATS** issued **new clinical recommendations** calling for the use of **race-neutral equations in spirometry**, a change representing an evolution in thought supported by recent evidence.⁴

A race-neutral reference equation provides better or equivalent relationships between spirometry and symptoms, airway structure, emphysema, and functional capacity.¹

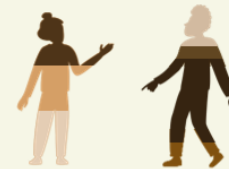


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RACE DOES NOT PREDICT GENETIC ANCESTRY

"Race is a social construct comprising broad, poorly defined categories that neither reliably predict genetic ancestry nor consistently unite people biologically."

- Collective statements from AAFP, ATS, AMA, AARPP



Classifying patients by race alone drives thinking away from the examination of other social, environmental, and genetic factors as drivers of disease.⁵ Genetic associations with pulmonary function do not provide evidence for the use of race or ethnicity in PFT interpretation.¹

Race-adjusted spirometry can be harmful because it

- May lead to the underdiagnosis and misdiagnosis of pulmonary disease in racial minority patients.^{2,7,8}
- Can reduce access to effective treatments.^{1,7,9,10}
- Impacts clinical risk of morbidity and mortality.^{1,11,12}

Misdiagnosis and Decreased Access to Care



ACT on Health Equity

EDUCATE leaders at your institution on how race-neutral spirometry could impact quality of care.

ADVOCATE for the implementation of ATS recommendations in lung function assessment and determine how race adjustment can be removed.

EVALUATE how you can best support patients whose diagnosis may be changed with race-neutral spirometry.

Abbreviations: AAFP: American Academy of Family Physicians; AAP: American Academy of Pediatrics; AMA: American Medical Association; ATS: American Thoracic Society; PFT: pulmonary function test.

1. Hersh CP, Hersh CP, Hersh CP, et al. Race and ethnicity in pulmonary function test interpretation: the official American Thoracic Society position. Am J Respir Crit Care Med. 2023;207(12):1558-1565. doi:10.1164/rccm.2023.02007. 2. Hersh CP, Hersh CP, Hersh CP, et al. Race and ethnicity in pulmonary function test interpretation: the official American Thoracic Society position. Am J Respir Crit Care Med. 2023;207(12):1558-1565. doi:10.1164/rccm.2023.02007. 3. Hersh CP, Hersh CP, Hersh CP, et al. Race and ethnicity in pulmonary function test interpretation: the official American Thoracic Society position. Am J Respir Crit Care Med. 2023;207(12):1558-1565. doi:10.1164/rccm.2023.02007. 4. Hersh CP, Hersh CP, Hersh CP, et al. Race and ethnicity in pulmonary function test interpretation: the official American Thoracic Society position. Am J Respir Crit Care Med. 2023;207(12):1558-1565. doi:10.1164/rccm.2023.02007. 5. Hersh CP, Hersh CP, Hersh CP, et al. Race and ethnicity in pulmonary function test interpretation: the official American Thoracic Society position. Am J Respir Crit Care Med. 2023;207(12):1558-1565. doi:10.1164/rccm.2023.02007. 6. Hersh CP, Hersh CP, Hersh CP, et al. Race and ethnicity in pulmonary function test interpretation: the official American Thoracic Society position. Am J Respir Crit Care Med. 2023;207(12):1558-1565. doi:10.1164/rccm.2023.02007. 7. Hersh CP, Hersh CP, Hersh CP, et al. Race and ethnicity in pulmonary function test interpretation: the official American Thoracic Society position. Am J Respir Crit Care Med. 2023;207(12):1558-1565. doi:10.1164/rccm.2023.02007. 8. Hersh CP, Hersh CP, Hersh CP, et al. Race and ethnicity in pulmonary function test interpretation: the official American Thoracic Society position. Am J Respir Crit Care Med. 2023;207(12):1558-1565. doi:10.1164/rccm.2023.02007. 9. Hersh CP, Hersh CP, Hersh CP, et al. Race and ethnicity in pulmonary function test interpretation: the official American Thoracic Society position. Am J Respir Crit Care Med. 2023;207(12):1558-1565. doi:10.1164/rccm.2023.02007. 10. Hersh CP, Hersh CP, Hersh CP, et al. Race and ethnicity in pulmonary function test interpretation: the official American Thoracic Society position. Am J Respir Crit Care Med. 2023;207(12):1558-1565. doi:10.1164/rccm.2023.02007. 11. Hersh CP, Hersh CP, Hersh CP, et al. Race and ethnicity in pulmonary function test interpretation: the official American Thoracic Society position. Am J Respir Crit Care Med. 2023;207(12):1558-1565. doi:10.1164/rccm.2023.02007. 12. Hersh CP, Hersh CP, Hersh CP, et al. Race and ethnicity in pulmonary function test interpretation: the official American Thoracic Society position. Am J Respir Crit Care Med. 2023;207(12):1558-1565. doi:10.1164/rccm.2023.02007.

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Who Decides Normal?

Global Lung Function Initiative (GLI)

Spirometry

74,187 results used to build predicted equations

3-95 years old

Lung Volumes

4,500-7,000 results used, all Caucasian

5-80 years old

DLCO/TLCO

12,660 results used, all Caucasian

5-85 years old

Age (yr)	Males				Females				Total
	Cauc.	Afr.Am.	N E Asia	S E Asia	Cauc.	Afr.Am.	N E Asia	S E Asia	
0-5	470			55	446			46	1017
5-10	8452	221		150	8307	227		153	17510
10-15	3924	318		271	3782	340		257	8892
15-20	1677	206	112	227	1557	235	115	292	4421
20-25	1090	137	145	251	1242	206	208	397	3676
25-30	1060	112	124	229	1203	149	217	343	3437
30-35	1106	99	114	248	1467	138	288	426	3886
35-40	1158	72	147	305	1495	132	513	485	4307
40-45	1296	68	121	274	1842	112	506	429	4648
45-50	1203	49	127	245	1782	59	389	404	4258
50-55	1077	58	126	211	1764	73	323	437	4069
55-60	912	40	126	188	1668	70	272	383	3659
60-65	760	44	120	164	1536	89	267	439	3419
65-70	605	34	63	141	1267	67	245	333	2755
70-75	519	34	66	94	1079	65	142	220	2219
75-80	317	18	14	26	727	50	71	91	1314
80-85	148	9	7	15	294	10	18	18	519
85-90	46	1	1	1	93	3	4	6	155
90-95	6		1		16			1	24
95	1				1				2
Total	25827	1520	1414	3095	31568	2025	3578	5160	74187

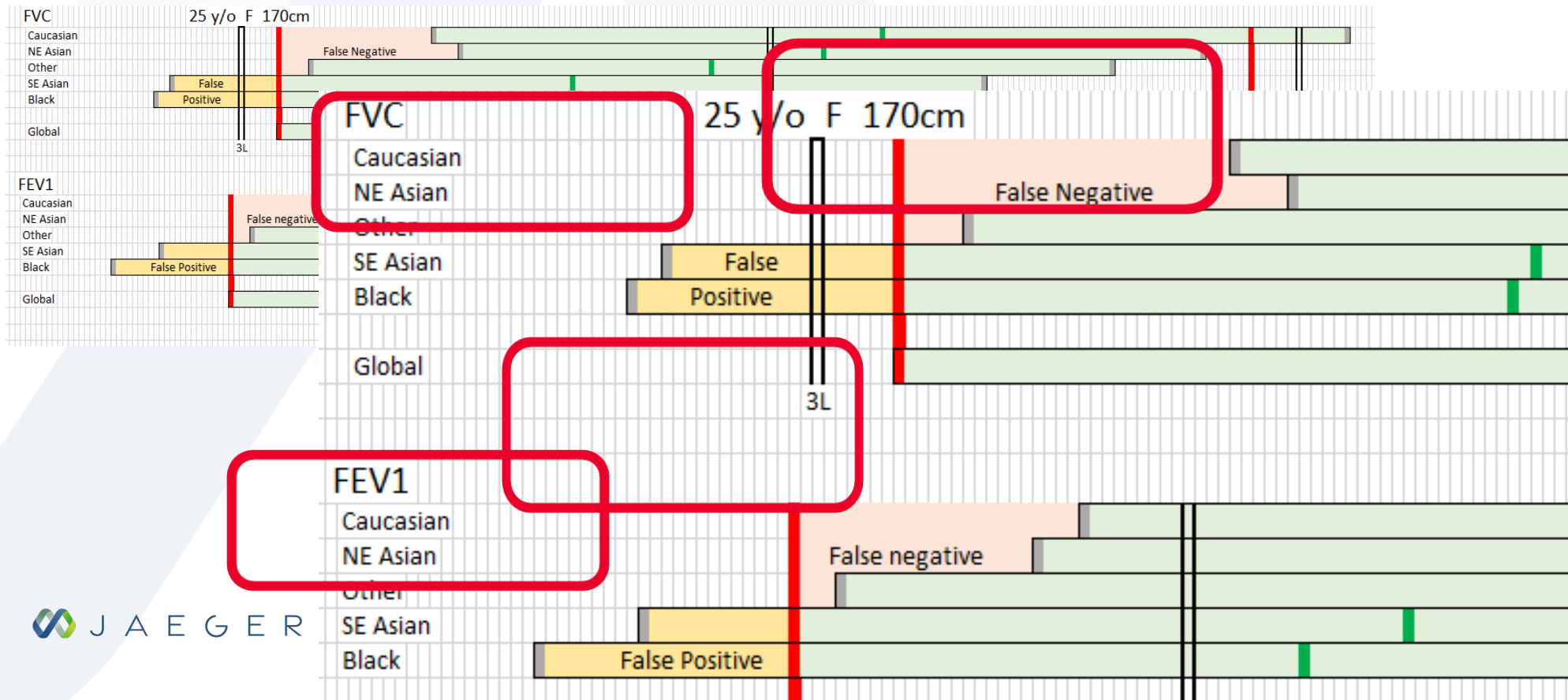
Cauc. = Caucasian; Afr.Am. = African American; S E Asia = South East Asian; N E Asia = North East Asian.

GLI Race Specific vs Race Neutral

Group	Females				Males			
	FEV ₁	FVC	FEV ₁ /FVC	FEF _{25-75%}	FEV ₁	FVC	FEV ₁ /FVC	FEF _{25-75%}
African American	-13.8	-14.4	0.6	-11.7	-14.7	-15.5	0.8	-12.9
North East Asia	-0.7	-2.1	1.1	-7.7	-2.7	-3.6	0.9	-3.2
South East Asia	-13.0	-15.7	2.9	-4.3	-9.7	-12.3	2.8	-0.9

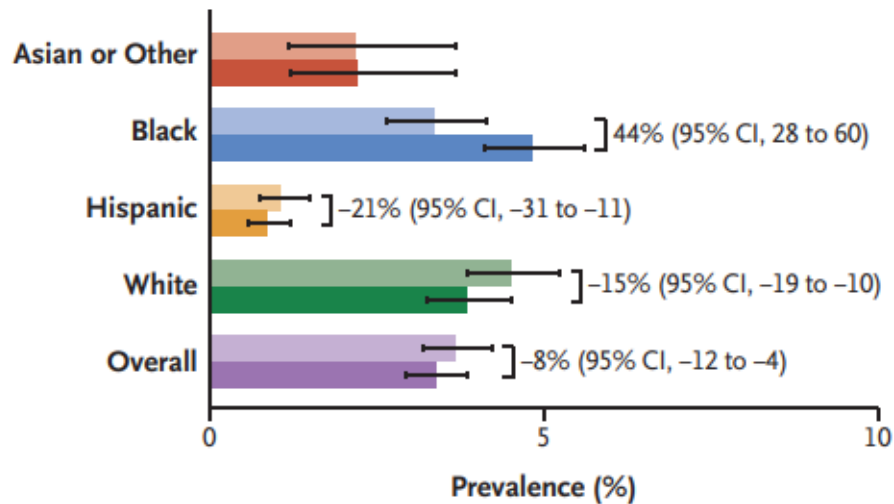
Group	Males		Females		Total
	N	Age (yr)	N	Age (yr)	
Caucasian	25,827	2.5-95	31,568	2.5-95	57,395
African American	1,520	6-85	2,025	6.1-87	3,545
North East Asia	1,414	16-91	3,578	16-88	4,992
South East Asia	3,095	3.3-86	5,160	3.2-92	8,255
Total	31,856		42,331		74,187

GLI Race Specific vs Race Neutral

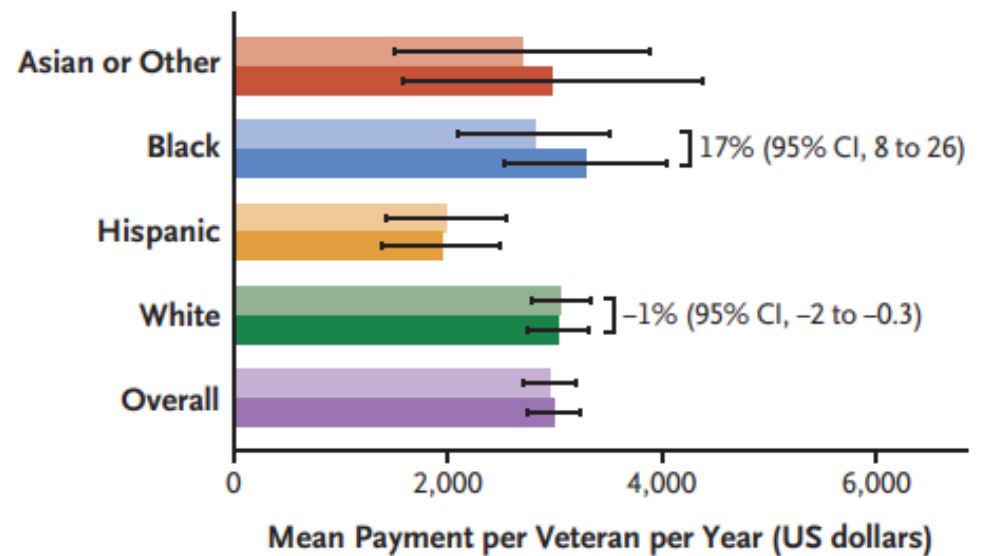


GLI Race Specific vs Race Neutral

C Moderate-to-Severe COPD (GOLD grade ≥ 2)

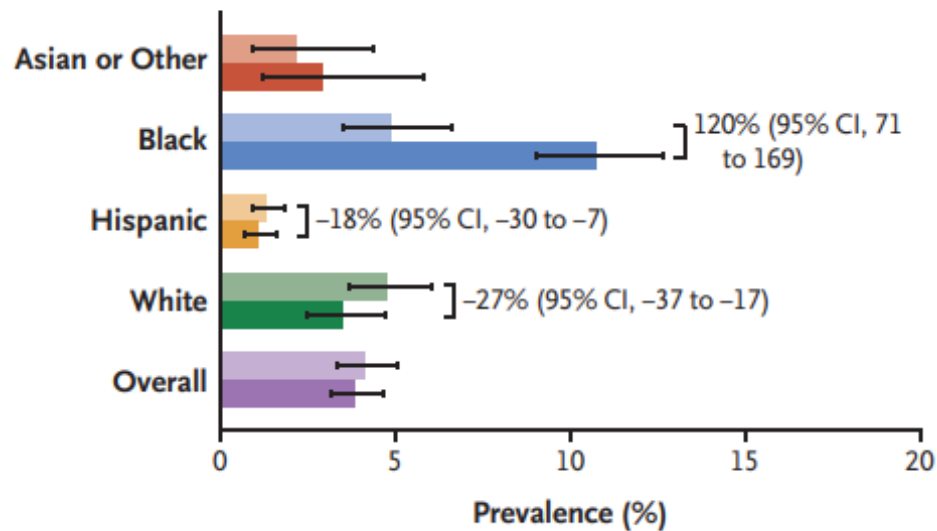


F Veterans Affairs Disability Payments

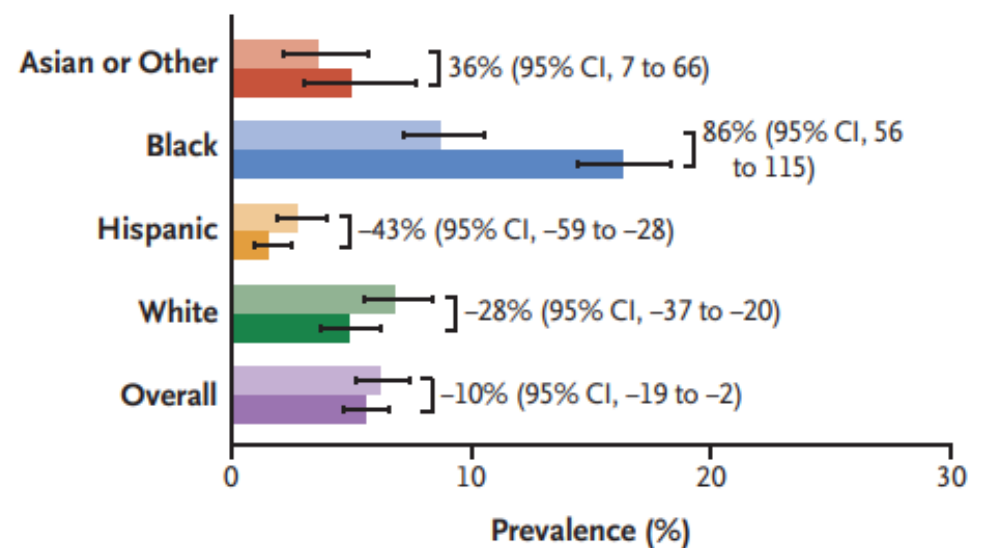


GLI Race Specific vs Race Neutral

E Moderate-to-Severe Impairment (AMA class ≥ 2)



D Occupational Disqualification (firefighting)



GLI Race Specific vs Race Neutral

Indisputable fact that people of different races have different lung volumes

Race does not equal ethnicity or citizenship

Present way of determining predicted values is not perfect but ignoring race is worse

The fact that we're different is not the problem, the problem is what we do with that information

Where does this end?



Objectives

1. Understand basic PFT measures and how they relate to lung function
2. Understand the basics of PFT interpretation
3. Understand some of the impacts of using race neutral predicted equations

PFT BASICS:

What You Need To Know Outside The PFT Lab

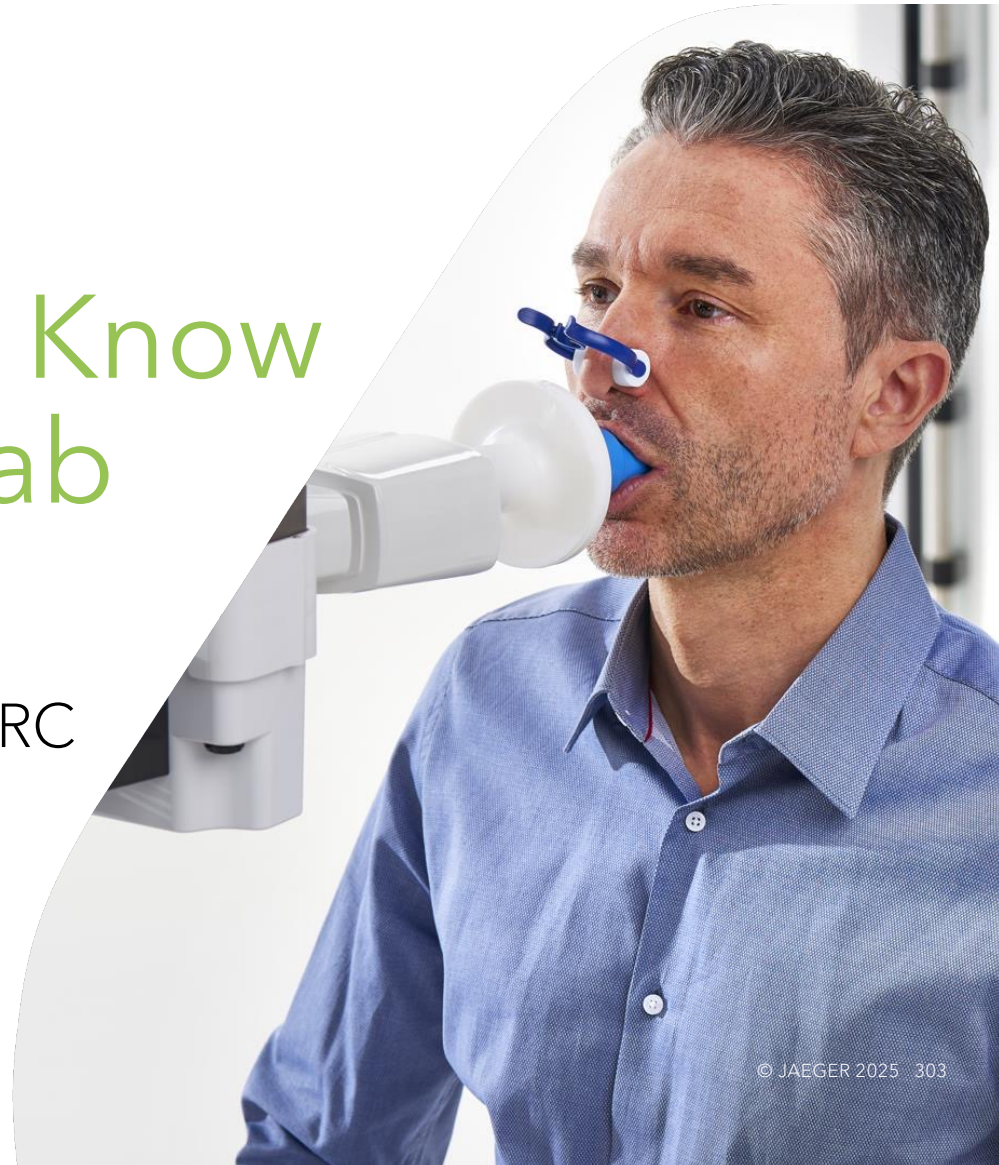
Ralph W Stumbo Jr RRT CPFT FAARC

Jaeger Medical America

Sales Consultant for WA, OR, ID, AK & NorCal

253-335-2704

Ralph.StumboJr@JaegerRDX.com





St. Luke's Children's

**SOLVING ANY MEDICAL EMERGENCY WHILE
IMPROVING TEAM PERFORMANCE**

**SUSAN N ROMERO, APRN, CPNP AC/PC
PEDIATRIC CRITICAL CARE NURSE PRACTITIONER
ST LUKE'S PEDIATRIC CRITICAL CARE UNIT
CRITA SIM LLC**

BASIC ASSUMPTION



**“WE BELIEVE ALL OF OUR PARTICIPANTS ARE INTELLIGENT, SKILLED, CARE
ABOUT DOING THEIR BEST, AND WANT TO IMPROVE.”**



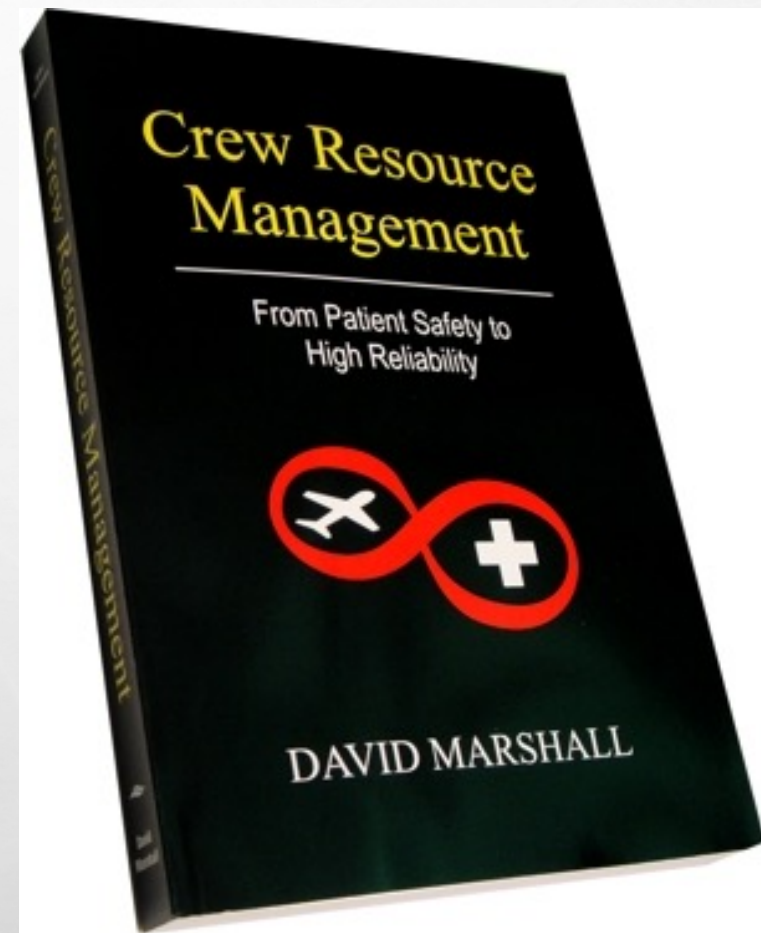
HISTORY OF SIMULATION EDUCATION

- **MOST OF OUR EXPERIENCE AND KNOWLEDGE IN SIMULATION HAS COME FROM OTHER INDUSTRIES LIKE...**
- **AVIATION**
- **NUCLEAR POWER**
- **MILITARY**
- **NASA**



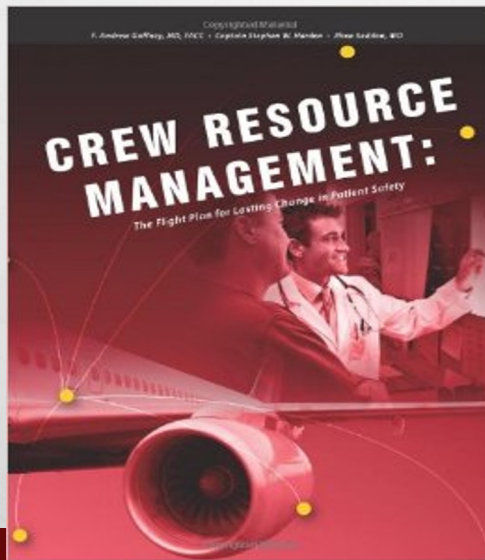
AVIATION: CREW RESOURCE MANAGEMENT (CRM)

- **IN TRAINING AND CRISIS MANAGEMENT, THE FOCUS IS ON:**
- **INTERPERSONAL COMMUNICATION**
- **LEADERSHIP**
- **DECISION MAKING**
- **TEAMWORK**
- **PROBLEM-SOLVING**
- **CREW RESOURCE MANAGEMENT OR COCKPIT RESOURCE MANAGEMENT (CRM) IS A SET OF TRAINING PROCEDURES FOR USE IN ENVIRONMENTS WHERE HUMAN ERROR CAN HAVE DEVASTATING EFFECTS. USED PRIMARILY FOR IMPROVING AIR SAFETY, CRM FOCUSES ON *INTERPERSONAL COMMUNICATION, LEADERSHIP, AND DECISION-MAKING* IN THE COCKPIT.**



COMPARING AVIATION TO HEALTHCARE

1990 CRM PRINCIPLES WERE ADAPTED TO MEDICINE IN ANESTHESIA REFERRED TO AS “**CRISIS RESOURCE MANAGEMENT**”



Crisis Resource Management

Ability, during an emergency, to translate knowledge of what needs to be done into effective real world activity

CRISIS RESOURCE MANAGEMENT (MADE EASY)

☐

Communication – closed loop

☐

Resources – human and equipment

☐

Management – global management with shared mental model

See a need, treat the need, re assess
Use a check list

☐

Check List??



Key to solving any emergency and imperative to work as a team



How to call for help

**911
Overhead page**



Who else calls for more help

**Initial responder
Administrative front desk
Security**



Posted numbers



Response algorithm

**CRISIS RESPONDS
=
COMMUNICATION**

COMMUNICATION IN A CRISIS

Closed loop

Voice control

Single leader speaks



RESOURCES

ROLES = HUMAN



Define roles

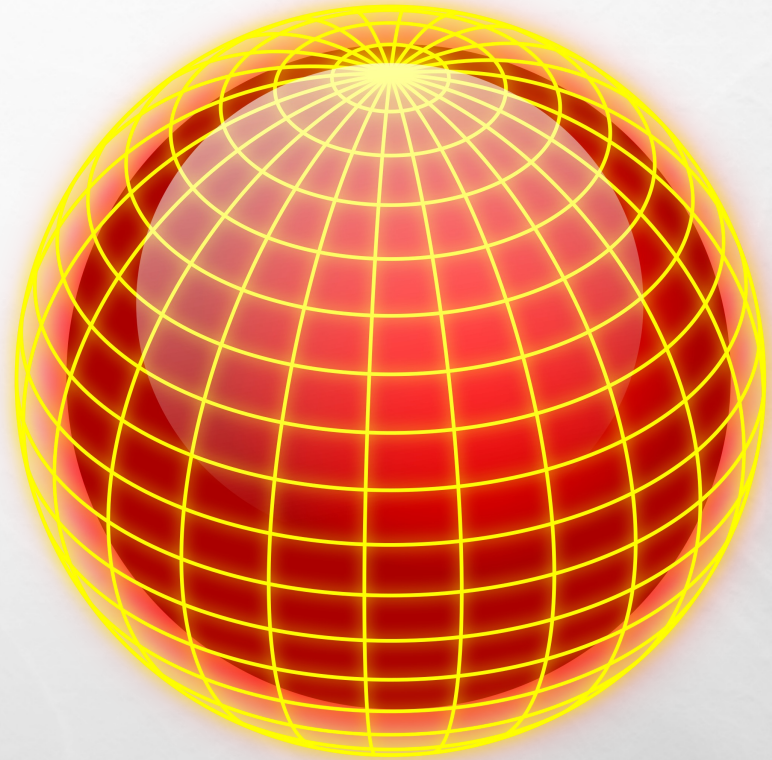


EQUIPMENT AND SUPPLIES



MANAGEMENT = GLOBAL ASSESSMENT

- **RECOGNIZE A PROBLEM**
- **IDENTIFY WHAT IS HAPPENING**
- **TREATMENT**
- **RE ASSESS THE SITUATION**



MANAGEMENT CHECKLIST, ANY TIME, ANYWHERE!

A = Airway

B = Breathing

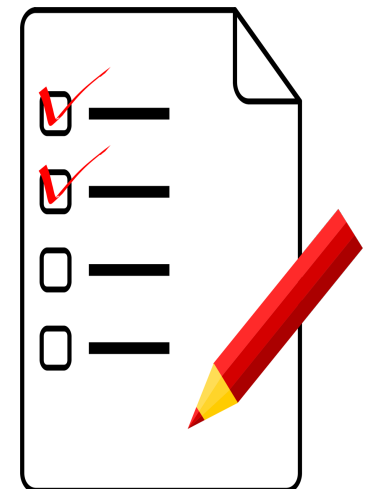
C = Circulation

D = Disability – WHAT????

E = Exposure

F = Family and Find a Friend

G = Glucose



SHARED MENTAL MODEL

The diagram illustrates a 'Shared Mental Model' process flow. It features a background of two stylized human heads in profile, facing each other. Inside the heads are various colored gears (blue, orange, pink, green) and dashed lines representing connections. The process flow is represented by a series of white boxes with black text, connected by lines. The flow starts with 'Walk through the ABCDEFG out loud', which branches into 'Recaps' and 'Summaries'. This is followed by 'Does anyone have any more ideas?', then 'Hand off to higher level of care', and finally 'Discussion with family'. A red bar is at the bottom of the slide.

**Walk through
the ABCDEFG
out loud**

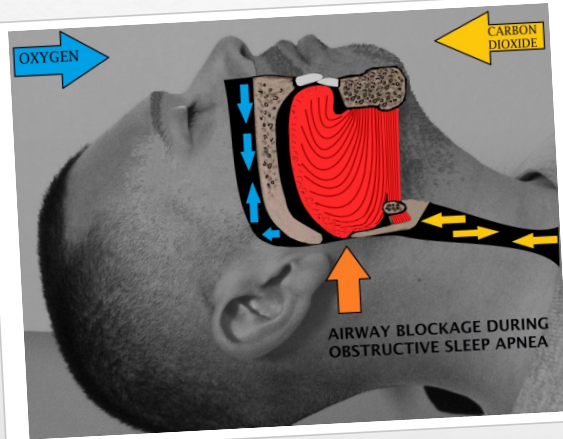
**“Does anyone
have any
more ideas”**

**Hand off to
higher level
of care**

**Discussion
with family**

Recaps

Summaries



OPEN IT



CLEAR IT



AIRWAY



Yes or NO

NO = do they have a pulse????

If + pulse – YOU ARE WINNING.

Breathe for them

- Adult 1 breath every 6 seconds
- Child 1 breath every 3 seconds

If breathing, then the evaluation starts

- Rate
- Quality
- Sounds



BREATHING





Disordered control of breathing



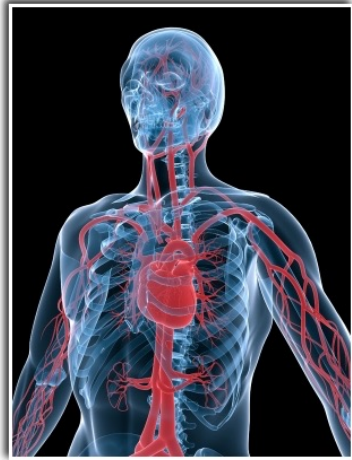
Extra Thoracic - Stridor



**Intrathoracic – Wheezes,
Crackles**

BREATHING...

GOOD OR BAD?



Pulse – yes or NO

- CPR rate for all 100- 120
- 30 compressions to 2 breaths
- Press hard and deep
- Allow for full recoil

+ pulse – winning again!

Rate

Quality – strong or weak

Cap Refill

Blood pressure

- Adult systolic > 90
- Kiddo's – check your charts!

CIRCULATION





The director of everything.



Do ABC right or else.....



**Start at the top (of the head)
and work down**

Check pupils



Not awake

What did you give?



Seizure????

Check sugar



Not awake

What did they take

**DISABILITY
= DA BRAIN**

DISABILITY



Awake or NOT

NOT – well now you got problems

Open up their eye lids

Tiny pupils tell you a lot

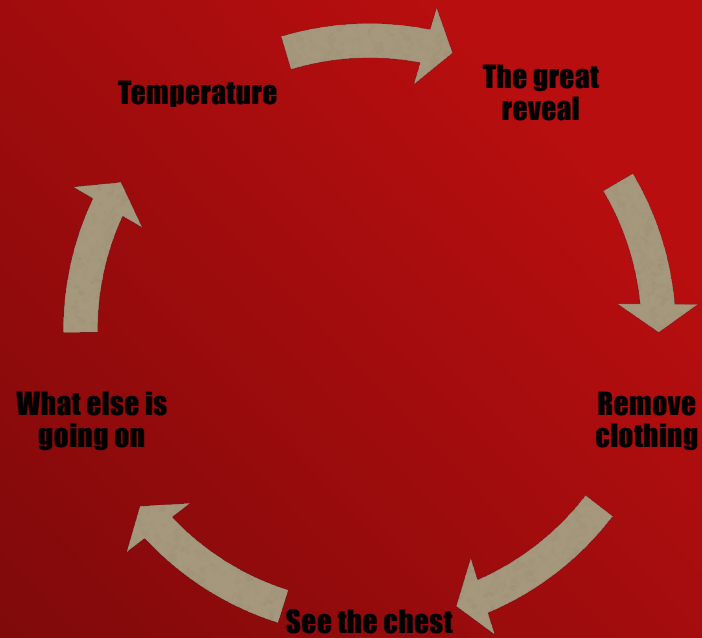
Pupils work – keep digging

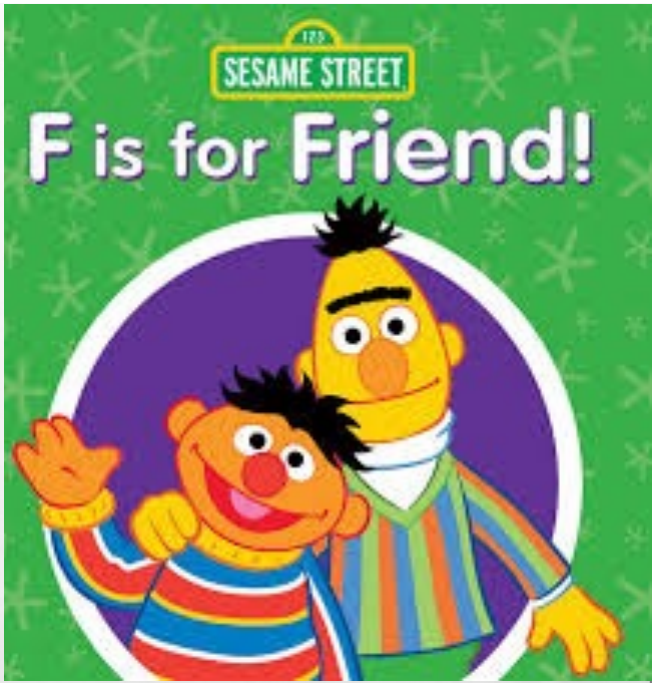
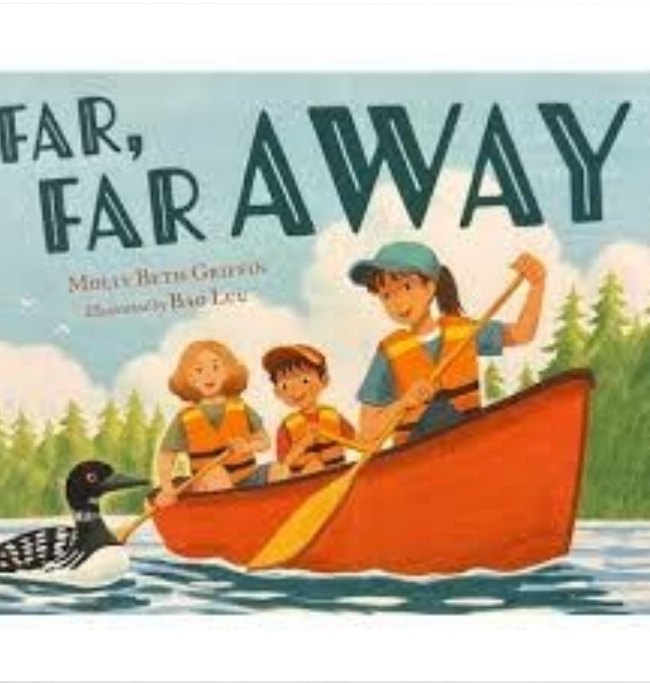
Disordered control of breathing – brain ain't working

Check glucose

- Carnitine palmitoyltransferase 1A (CPT1A) is an enzyme that helps the body break down fats for energy

EXPOSURE





**F IS
FOR.....**

SO MANY THINGS!

FIND YOUR FRIENDS AND THE FAMILY

Appoint

- **Appoint a staff member to attend the family**

Pretend

- **Pretend you are a sports caster**

Call

- **Call smarter or sillier people than you**

Notify

- **Notify their primary care doctor if bad stuff happened**
- **Who does this?**
- **Make PCP notification it part of the check list**

Follow up

- **Follow up with family**

Follow up

- **Follow up with your friends (coworkers)**

Find

- **Find EMS!**



Yep, this is often forgotten

Just a reminder to check it if you haven't, especially in:

Child you can pick up

Elderly

Anyone who fails the disability exam above

GLUCOSE





Symptoms of Hypoglycemia



Shaking or trembling.



Faster heart rate.



Extreme hunger.



Sweating.



Confusion/difficulty concentrating.



Dizziness.

Hs and Ts of ACLS - Reversible Causes of Cardiac Arrest

H Hypoxia

H Hypovolaemia

H Hypokalaemia/Hyperkalaemia

H Hypothermia

T Tension Pneumothorax

T Thrombosis

T Tamponade

T Toxins



IF YOU ARE A TOTAL NERD THERE ARE H'S AND T'S!



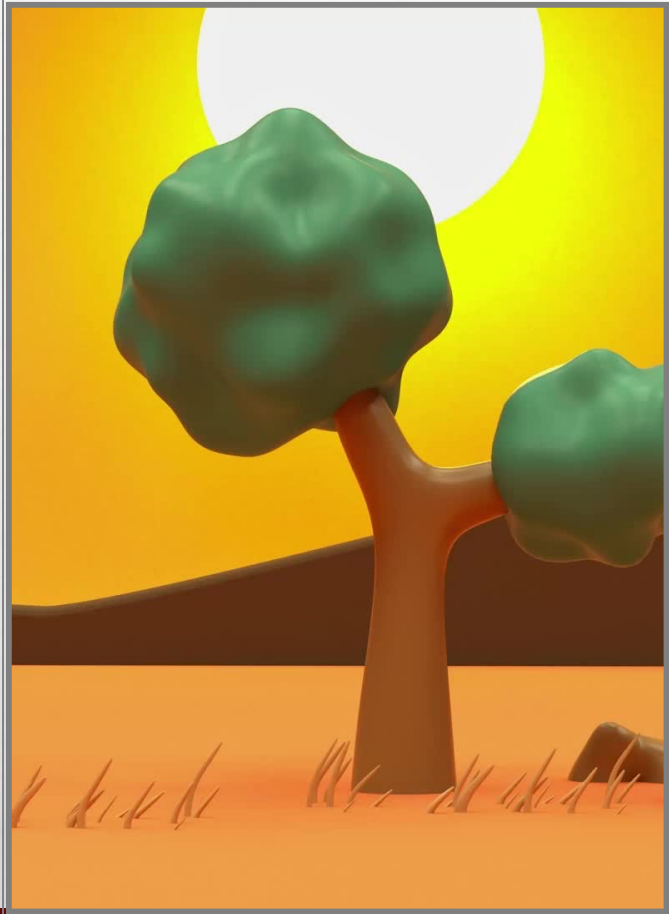


Practice together



SIMULATION

SO NOW WHAT?



WHY SIMULATION?

- **SIMULATION IS OFTEN IMMERSIVE IN NATURE:**
- **IMMERSIVE CONVEYS THE SENSE THAT PARTICIPANTS HAVE OF BEING IMMERSED IN A TASK OR SETTING AS THEY WOULD IF IN THE REAL WORLD”**
- **GABA, D. M. 2004**



SIMULATION EDUCATION

- INSTEAD OF *SEE ONE, DO ONE, TEACH ONE*, IT BECOMES:
- SEE ONE
- PRACTICE MANY (AS A TEAM)
- DO ONE (AS A TEAM)

METHODOLOGY OF SIMULATION

Immersed in a realistic scenario

Expected to think, talk, and DO

Challenged to go beyond their comfort level

Mistakes are ok.....*we learn from them*

Provided the opportunity to reflect on their performance in debriefings following scenario

Trained as a team

**SIMULATION
EDUCATION IS A
BRIDGE BETWEEN
CLASSROOM
LEARNING AND
REAL-LIFE
CLINICAL
EXPERIENCE**



A photograph of three healthcare professionals in a clinical setting, likely a simulation lab. They are wearing blue scrubs and white gloves, focused on a task. One person is holding a clear plastic bag, and another is using a stethoscope. In the background, there is a medical monitor displaying vital signs and a blood pressure cuff on a stand. The overall scene is dimly lit, with the text overlay being the primary source of brightness.

**OUR GOAL WITH SIMULATION IS
TO NOT HAVE A TEAM OF EXPERTS,
BUT CREATE EXPERT TEAMS**

A group of healthcare professionals, including nurses and doctors, are gathered in a clinical setting, possibly a hospital room or a training area. They are standing and looking towards the right side of the frame. A large red semi-transparent overlay covers the right half of the image, containing the text 'T – Together', 'E – Everyone', 'A – Achieves', and 'M - More' in bold black font, separated by horizontal lines. The background shows a window with a view of a parking lot and trees.

T – Together

E – Everyone

A – Achieves

M - More



TEAM

**GENERATES PERFORMANCE GREATER THAN THE
SUM OF THE PERFORMANCE OF ITS INDIVIDUAL
MEMBERS**

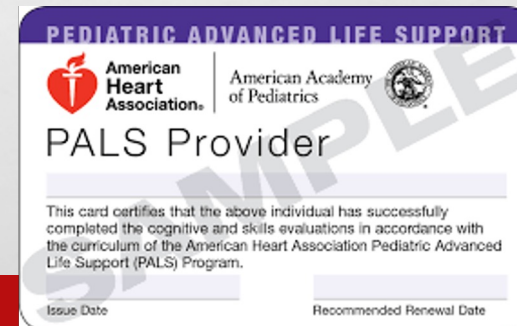
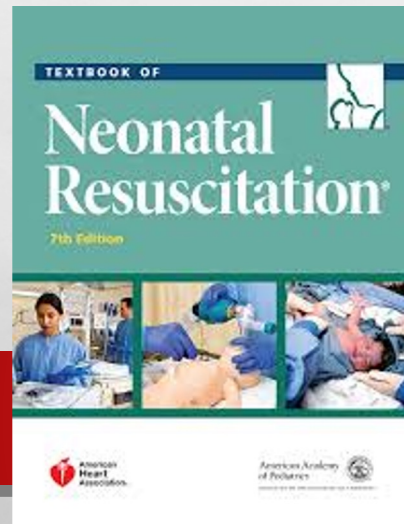
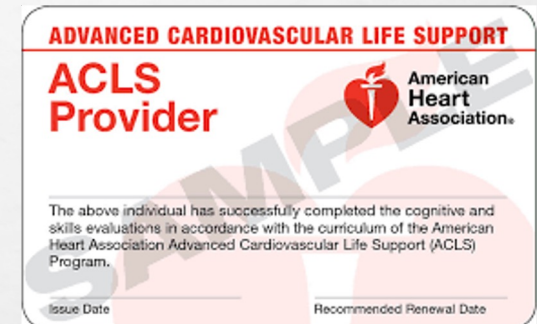
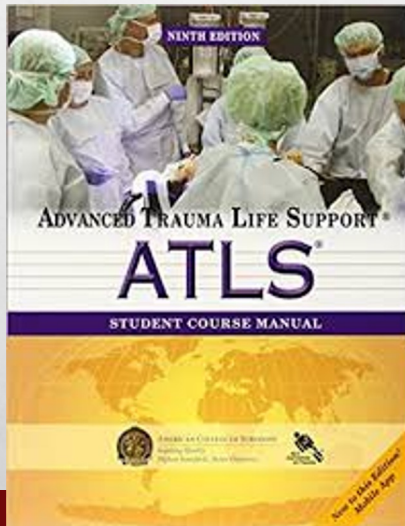
SIMULATION

- **HOW DO WE DO THIS?**
- **OR**
- **NOPE! NOT ME!**



THIS DOES NOT APPLY TO ME!

REALLY.....





SET THE STAGE

PRE BRIEF

BASIC ASSUMPTION



**“WE BELIEVE ALL OF OUR PARTICIPANTS ARE INTELLIGENT, SKILLED, CARE
ABOUT DOING THEIR BEST, AND WANT TO IMPROVE.”**

STEPS TO DEVELOP THE PREBRIEF

When – following introductions

Set the stage - identify the type of scenario

Review objectives – what is expected

How – orientation to the CRM

- **Communication – how to get help, closed-loop**
- **Resources – roles, resources and environment**
- **Management – Global assessments using ABCDEF**

Who else – embedded participant description

GROUND RULES



Vegas



Suspend disbelief



Fiction contract



Power of time out



Fancy Simulation

**Rapid cycle deliberate
practice**

Run through and repeat

what happens in
VEGASTM
stays in vegas



SIMULATION IS LIKE LAS VEGAS.....



SUSPENDING DISBELIEF

**The more real
you make it
the more
effective it
can be**

Just go with it

**Jump in an
make it real**



CRISIS RESOURCE MANAGEMENT (MADE EASY)

☐

Communication – closed loop

☐

Resources – human and equipment

☐

Management – global management with shared mental model

See a need, treat the need, re assess
Use a check list

☐

Check List??

SIMULATION FACILIATION



SCENARIO FACILITATION

Collaborates well within the facilitation teams

Provides clear orientations and briefings

Maintains scenario fidelity by minimizing intrusions and interruptions

Gives responsive and timely manipulations of the scenario

Recognizes salient actions and omissions while filtering out extraneous information

DEBRIEF

WHERE THE MAGIC
HAPPENS!





RAS – reactions, analysis, & summary



GAS – gather, analyze, & summarize

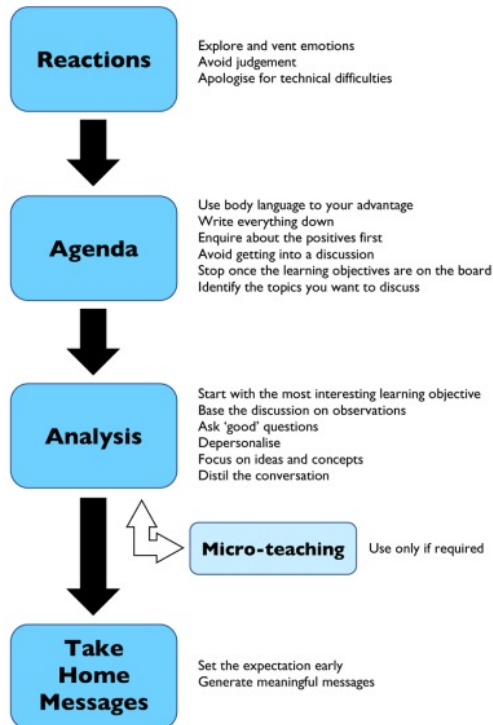


3D – defusing, discovery, & deepening



Diamond – description, analysis, & application

3-PHASE DEBRIEFING TECHNIQUE



*Adapted from the Scottish Centre for Simulation and Clinical Human Factors <https://scschf.org>

Figure 1. VAST's debriefing framework. VAST = Vital Anaesthesia Simulation Training.

DEBRIEFING MADE EASY



Initial reactions

**Tell me what happened
with this patient –
summary statement**



What went well



What would you change



One thing you would take away and teach someone else



Pro tip – say as little as possible

Sometimes



My greatest
accomplishment is just
keeping my mouth shut.



[HTTPS://YOUTU.BE/BGVEAXVOX5U?T=1](https://youtu.be/BGVEAXVOX5U?T=1)



ARE WE A TEAM OF EXPERTS, OR ARE WE AN EXPERT TEAM?



Crita Sim, LLC



Simulated Emergency Action Training

**Susan N Romero, MSN, CPNP AC/PC
Pediatric Critical Care Nurse Practitioner**

susan.romero@critasim.com

602-910-1348

THANK YOU

- **PLEASE FEEL FREE TO CONTACT ME AT ANY TIME.**
- **THANK YOU TO THIS FANTASTIC TEAM FOR INVITING ME HERE TODAY AND FOR ALL THE HELP EACH OF YOU PROVIDE YOUR TEAMS!**

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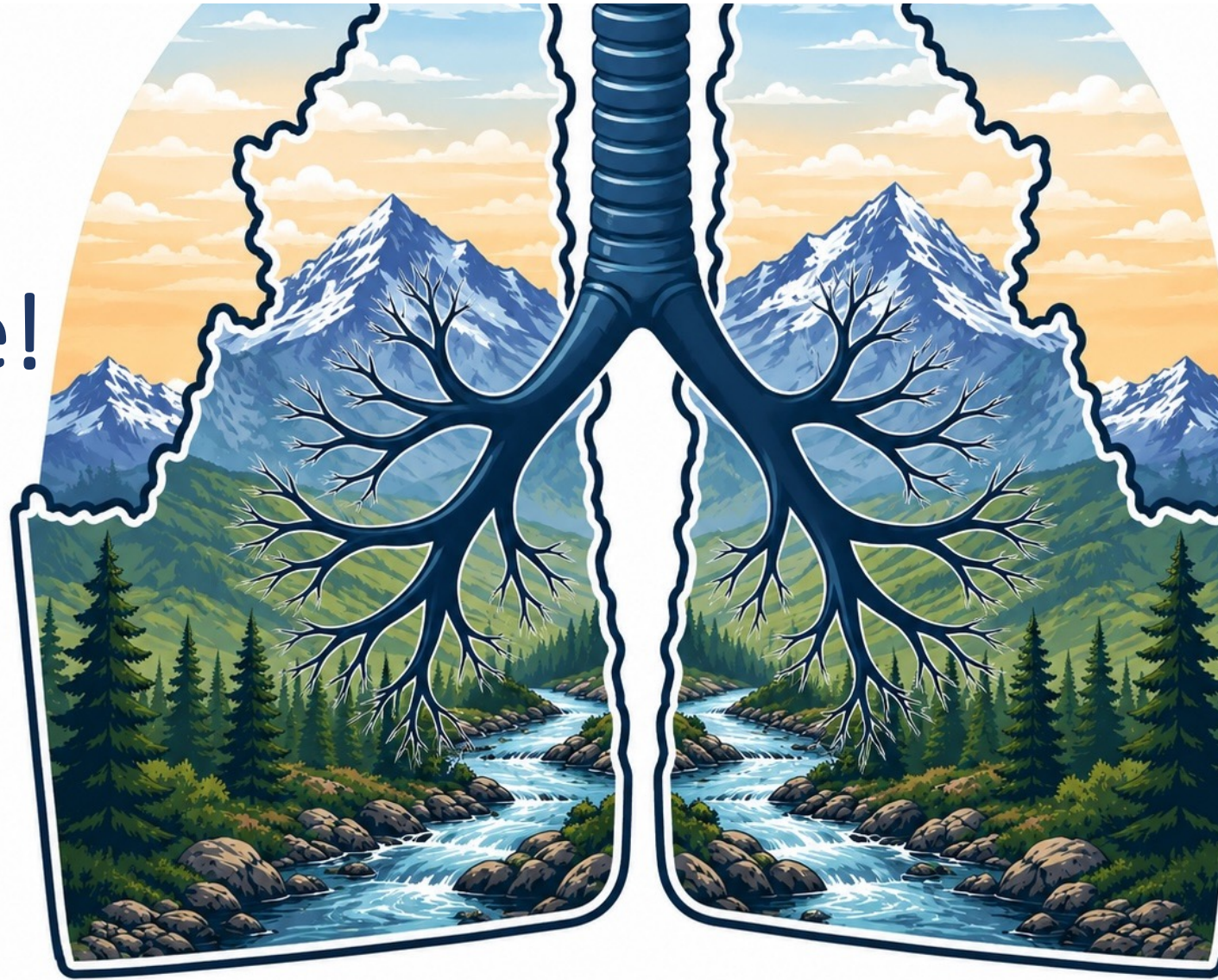
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THANK YOU!



Break Time!
Visit the
Vendors!



Idaho Society for Respiratory Care

Kratom, Cannabis, and the Sleeping Lung

Emerging Challenges in Respiratory and Sleep Medicine

Dr. Amanda L. Leightner PhD, RRT, RRT-SDS, RPSGT, CCSH, FAARC, FNAP
Merrick Family Endowed Professor
Boise State University

Disclosures & Format



Faculty Disclosure

No relevant financial relationships with ineligible companies related to the content of this activity.

This presentation reviews evidence on non-medical and unregulated substances. It does not constitute an endorsement of use and does not substitute for individualized clinical judgment.



Session Format



Interactive Lecture

Case-anchored, discussion-driven review of current evidence



Myth vs. Fact

Live polling moments to challenge common clinical assumptions



Visual Infographics

Mechanism and risk diagrams for point-of-care recall

Learning Objectives

By the end of this session, participants will be able to:

01

Review the Pharmacology

Describe the mechanisms of action of kratom (mitragynine, 7-hydroxymitragynine) and cannabis (THC, CBD) relevant to the respiratory and sleep systems.

02

Examine Physiologic Effects

Analyze how these substances alter sleep architecture, ventilatory drive, and pulmonary structure and function.

03

Integrate Evidence into Care

Apply current evidence to clinical decisions for patients using kratom and cannabis, with emphasis on polypharmacy and respiratory safety.

Roadmap for Today



Pharmacology

Kratom & cannabis mechanisms



Sleep & Lung

Architecture, ventilation, pulmonary health



Clinical Integration

Polypharmacy & respiratory safety

Look for the Myth vs. Fact prompts... we'll pause to test assumptions as a group.

Why This Matters Now

Two widely used, minimally regulated substances are increasingly showing up in respiratory and sleep practice.



~1.7–2 M+

past-year U.S. kratom users (NSDUH /
RADARS estimates)



6.1%

estimated lifetime kratom use
prevalence, U.S. adults



~2,800

EVALI hospitalizations/deaths reported
by CDC (through Feb 2020), majority
THC-vape linked



70%+

of chronic-opioid patients with co-
reported cannabis use showing altered
REM sleep in clinic cohorts

Both substances are frequently used alongside prescribed opioids, benzodiazepines, and other sedative-hypnotics often without clinician awareness.

Sources: SAMHSA NSDUH 2019–2021; RADARS System; Covvey et al., 2020; CDC EVALI Outbreak Report (2020); Farney et al., J Clin Sleep Med.

Kratom: What Are We Talking About?

- *Mitragyna speciosa* — a tropical tree in the coffee family (Rubiaceae), native to Southeast Asia
- Leaves contain >40 alkaloids; the two of greatest clinical relevance are mitragynine and 7-hydroxymitragynine (7-OH-MG)
- Sold in the U.S. as an unregulated botanical / dietary supplement — powders, capsules, teas, and concentrated extracts (“shots”)
- Dose-dependent, biphasic effects: stimulant-like at low doses, opioid-like sedation/analgesia at higher doses

DOSE-DEPENDENT EFFECT



LOW DOSE
Stimulant-like
(energy, alertness)

HIGH DOSE
Opioid-like
(sedation, analgesia)

~1–5 g

>5–15 g

Legal status: unscheduled federally; banned or restricted in several states and countries

Kratom Pharmacology: Receptor Mechanisms

- Mitragynine: partial agonist at μ -opioid receptors; antagonist at κ - and δ -opioid receptors
- Critically, mitragynine shows minimal recruitment of the β -arrestin-2 pathway, the signaling route most implicated in classical opioid respiratory depression
- 7-OH-mitragynine (a CYP3A-derived metabolite) is a more efficacious μ -agonist with a signaling profile closer to classical opioids
- Mitragynine also stimulates α 2-adrenergic receptors, contributing to sedative/analgesic effects through a non-opioid pathway

TWO ALKALOIDS, TWO PROFILES

Mitragynine

Partial μ -agonist • low β -arrestin-2 recruitment → limited respiratory depression in preclinical models

Rodent data: increased respiratory frequency (stimulant-like) at tested doses

7-Hydroxymitragynine

More potent μ -agonist, closer to classical opioids → significant, naloxone-reversible respiratory depression

Clinical concern: products enriched/marketed for high 7-OH-MG content

Source: Preclinical plethysmography studies of mitragynine and 7-OH-mitragynine (2025); Kruegel et al.; Obeng et al.

MYTH vs. FACT

Kratom & Opioid Overdose Risk

MYTH

“Kratom can't cause a fatal overdose because it's just a partial opioid agonist — it has a natural ceiling effect.”

FACT

Partial agonism at mitragynine's typical exposures does limit isolated respiratory depression in preclinical models. But 7-hydroxymitragynine is a much more potent, opioid-like agonist, product 7-OH-MG content is unregulated and variable, and the overwhelming majority of real-world kratom-associated deaths involve co-ingested opioids, benzodiazepines, or other depressants. “Natural ceiling” does not describe real-world use.

Sources: Bidirectional Effects on Breathing in Rats (2025); Olsen EO et al., MMWR 2019;68:326–327.

 Poll the room before revealing the fact panel.

PHARMACOLOGY CANNABIS

Cannabis: The Endocannabinoid System in Brief

- Δ^9 -THC: partial agonist at CB1 (predominantly CNS) and CB2 receptors drives psychoactivity, sedation, and analgesia
- CBD: minimal CB1/CB2 affinity; modulates serotonergic, adenosinergic, and GABAergic signaling largely non-intoxicating
- CB1 receptors are dense in brainstem respiratory and arousal centers, providing a plausible mechanism for ventilatory and sleep effects
- Legal cannabis products vary enormously in THC:CBD ratio and potency modern flower/concentrates often exceed 20–90% THC

TWO KEY CANNABINOIDS

THC

Psychoactive • CB1 partial agonist • sedating & REM-suppressing at higher doses

CBD

Non-intoxicating • low cannabinoid-receptor affinity • anxiolytic, CYP450 inhibitor

Sources: NIDA Cannabis Research Report; Hancox RJ, *Respirology* 2024;29:186–196.

Route of Administration Changes the Risk Profile

Onset, peak effect, and pulmonary exposure differ substantially by delivery method.

Smoked (Flower)

Onset: 1–5 min

Combustion products; strongest bronchitis association; deep inhalation + breath-hold pattern

Vaped (Oil/Concentrate)

Onset: 1–5 min

Perceived as “safer”; linked to EVALI outbreak via additives (vitamin E acetate)

Edible/Oral

Onset: 30–120 min

No pulmonary exposure; delayed onset raises overdose/re-dosing risk; first-pass yields more 11-OH-THC

Dabbing/Concentrate

Onset: <1 min

Very high THC potency (60–90%); highest acute intoxication and thermal-injury risk

Sources: Hancox RJ, Respiriology 2024; Blount BC et al., N Engl J Med 2020;382:697–705 (EVALI/vitamin E acetate).

MYTH vs. FACT

Vaping Cannabis vs. Smoking It

MYTH

“Vaping cannabis is the safe, lung-friendly alternative to smoking it.”

FACT

Vaping avoids combustion products but is not risk-free: it was the vehicle for the 2019–2020 EVALI epidemic, driven largely by vitamin E acetate used to dilute illicit THC cartridges. Legal, unadulterated vape products appear lower-risk than combusted flower for airway irritation, but 'vaping' is not a single risk category — product source and additives matter enormously.

Sources: Blount BC et al., *N Engl J Med* 2020;382:697–705; Layden JE et al., *N Engl J Med* 2020;382:903–916.

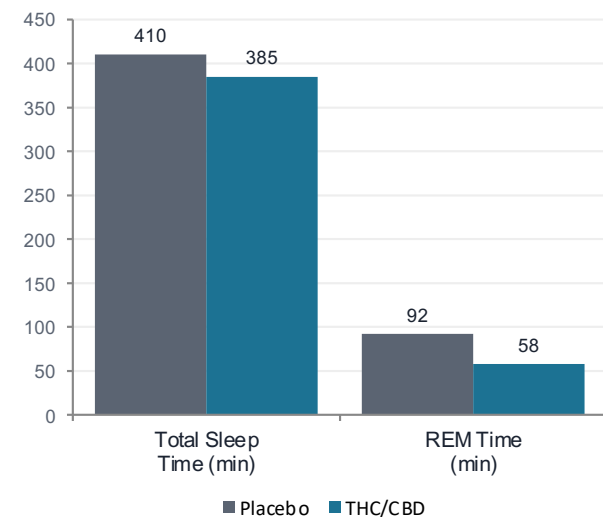
 Poll the room before revealing the fact panel.

SECTION 2 SLEEP & VENTILATION

Cannabis and Sleep Architecture

- Most consistent PSG finding: decreased sleep onset latency
- THC suppresses REM sleep in a dose-dependent way; effects on slow-wave (N3) sleep are inconsistent across studies
- A 2025 RCT: single-dose 10 mg THC/200 mg CBD reduced total sleep time (–24.5 min), cut REM time (–34 min), and delayed REM onset (+66 min) versus placebo
- Chronic/near-bedtime use is associated with increased N1 (light sleep) and sleep fragmentation the opposite of the subjective “better sleep” many patients report
- Withdrawal from heavy chronic use often produces vivid dreaming and disrupted sleep as a REM “rebound” phenomenon

PILOT RCT: THC/CBD vs. PLACEBO



Illustrative values from published pilot RCT data (n=20, insomnia)

Sources: Suraev et al., J Sleep Res 2026;e70124; Cannabis and sleep architecture: systematic review & meta-analysis, Sleep Med Rev 2025.

Kratom's Complicated Relationship with Sleep

Reported by Users

- Low doses often used earlier in the day for energy/mood can delay sleep onset if used too close to bedtime
- Higher, evening doses used by some for sedation and pain-related sleep disruption
- Chronic daily use can produce a withdrawal syndrome featuring insomnia, restlessness, and anxiety similar in character to opioid withdrawal

Objective (PSG/EEG) Evidence

- Controlled polysomnographic data in humans remain very limited most evidence is self-report/survey based
- Preclinical and pharmacologic data support a biphasic CNS effect (adrenergic stimulation at low dose, opioid-receptor sedation at high dose) that plausibly explains variable sleep effects
- No validated pharmacotherapy role for kratom in any sleep disorder evidence gap is a key discussion point for clinicians

Sources: Survey and case-series literature on kratom and sleep; Grundmann et al., Front Pharmacol 2024 (research needs).

SECTION 2 SLEEP & VENTILATION

Kratom and Ventilatory Control

- In controlled rodent studies, oral mitragynine alone showed no dose-related respiratory depression even at supra-typical doses
- By contrast, 7-OH-mitragynine produced respiratory depression comparable to morphine, fully reversible with naloxone
- This creates a critical product-safety issue: kratom preparations vary widely in mitragynine:7-OH-MG ratio, and some marketed extracts are deliberately enriched in 7-OH-MG
- Real-world overdose risk rises sharply with co-ingestion of other CNS/respiratory depressants pharmacology alone underestimates clinical risk

NALOXONE RESPONSIVENESS

Mitragynine

Minimal respiratory depression - naloxone not typically indicated for MG alone

7-OH-Mitragynine

Significant, naloxone-reversible respiratory depression - treat as opioid overdose

Adulterated / Polydrug Kratom

Unpredictable - assume opioid co-exposure until proven otherwise

Sources: Bidirectional Effects on Breathing in Rats (mitragynine/7-OH-MG), 2025; Behnood-Rod et al., Psychopharmacology 2022;239:1553–64.

SECTION 2 SLEEP & VENTILATION

Cannabis Smoking & Pulmonary Function

- Strong, consistent association with chronic bronchitis symptoms: cough, sputum production, wheeze
- Unlike tobacco, cannabis is not consistently linked to progressive FEV1 decline or COPD — evidence for airflow obstruction remains “unconvincing”
- Cannabis is associated with increased FVC and static lung volumes (TLC, RV) — a hyperinflation pattern possibly from deep-inhalation/breath-hold technique
- Lower specific airway conductance (sGaw) indicates large-airway effects despite preserved FEV1
- Bronchoscopy shows airway inflammation at far lower cumulative exposure than tobacco

CANNABIS vs. TOBACCO: LUNG FUNCTION PATTERN		
Finding	Cannabis	Tobacco
Chronic Bronchitis	Strong ↑	Strong ↑
FEV1 decline / COPD	Not established	Strong ↑
FVC / Lung Volumes	↑ (hyperinflation)	↓ over time
Airway Inflammation	Present, milder cumulative dose	Present, dose-dependent

Sources: Tan WC et al., Am J Respir Crit Care Med 2022;205:1067–74; Hancox RJ, Respirology 2024;29:186–196.

SECTION 2 — SLEEP & VENTILATION

Cannabis Vaping and EVALI

- E-cigarette/vaping product use-associated lung injury (EVALI): acute lung injury epidemic identified in 2019
- ~2,800 hospitalizations or deaths reported by CDC through February 2020; median patient age in the mid-20s
- Vitamin E acetate - a thickening/cutting agent in informally sourced THC vape cartridges is the additive most strongly linked to disease
- Presentation: dyspnea, cough, chest pain, GI symptoms, fever; imaging shows diffuse ground-glass opacities; bronchoalveolar lavage often shows lipid-laden macrophages
- 25–50% of hospitalized cases required mechanical ventilation across published cohorts

CLINICAL RED FLAGS

- Young patient, subacute dyspnea/cough + GI symptoms
- History of illicit or informally-sourced THC cartridge use
- Diffuse ground-glass opacities without infectious source identified
- Ask specifically about vaping patients may not volunteer THC vape use unprompted

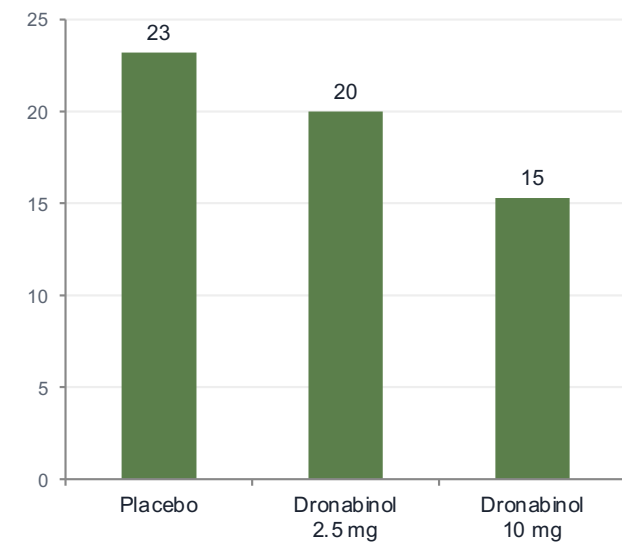
Sources: Blount BC et al., *N Engl J Med* 2020;382:697–705; CDC EVALI Outbreak Investigation (2020); Layden JE et al., *N Engl J Med* 2020;382:903–16.

SECTION 2 SLEEP & VENTILATION

Cannabis and Obstructive Sleep Apnea: The PACE Trial

- PACE (Pharmacotherapy of Apnea by Cannabimimetic Enhancement) the largest, longest RCT of a drug therapy for OSA to date
- 73 adults with moderate-severe OSA randomized to placebo, 2.5 mg, or 10 mg dronabinol (synthetic THC) nightly for 6 weeks
- 10 mg dronabinol reduced AHI and improved subjective sleepiness/treatment satisfaction vs. placebo; adherence was excellent (no CPAP-style drop-off)
- Mechanism: cannabinoid modulation of serotonergic and vagal afferent pathways stabilizing upper-airway/respiratory control during sleep
- Not FDA-approved for OSA and not a substitute for guideline-directed therapy — raised as an active investigational area, not a standard-of-care recommendation

AHI REDUCTION AT 6 WEEKS



Illustrative trend based on published PACE trial results (Carley et al., SLEEP 2018)

Sources: Carley DW et al., *Sleep* 2018;41(1):zxx184; Prasad B et al., *Front Psychiatry* 2013;4:1.

MYTH vs. FACT

Cannabis as a Universal Sleep Aid

MYTH

“If cannabis helps a patient fall asleep faster, it's improving their sleep.”

FACT

Reduced sleep-onset latency is real, but polysomnographic data show THC-predominant products commonly suppress REM sleep, increase REM latency, and with chronic or near-bedtime use, increase light-sleep (N1) fragmentation. Faster sleep onset does not equal restorative sleep architecture; patients need this distinction explained explicitly.

Sources: Suraev A et al., *J Sleep Res* 2026;e70124; Cannabis and sleep architecture: systematic review, *Sleep Med Rev* 2025.

 Poll the room before revealing the fact panel.

SECTION 3 CLINICAL INTEGRATION

Kratom, Overdose, and the Polydrug Reality

- CDC SUDORS (27 states, Jul 2016–Dec 2017): 152 of 27,338 overdose deaths tested positive for mitragynine; 91 were judged kratom-involved
- Only 7 of those decedents had no other substance detected and even those cases were not confirmed kratom-only causation
- FDA has attributed 44 deaths to kratom use, most involving polydrug exposure (including the Swedish “Krypton” deaths caused by an adulterant, O-desmethylnaloxone)
- A 2023 forensic analysis proposed a blood mitragynine level above ~1,000 ng/mL as associated with elevated overdose risk not a validated lethal threshold
- Bottom line: kratom-associated mortality is overwhelmingly a polypharmacy phenomenon, not a single-agent one

152 DEATHS, MITRAGYNINE-POSITIVE



Polydrug-involved deaths (145/152)

Most common co-detected substances

- Fentanyl / other opioids
- Benzodiazepines
- Alcohol
- Other prescription sedatives

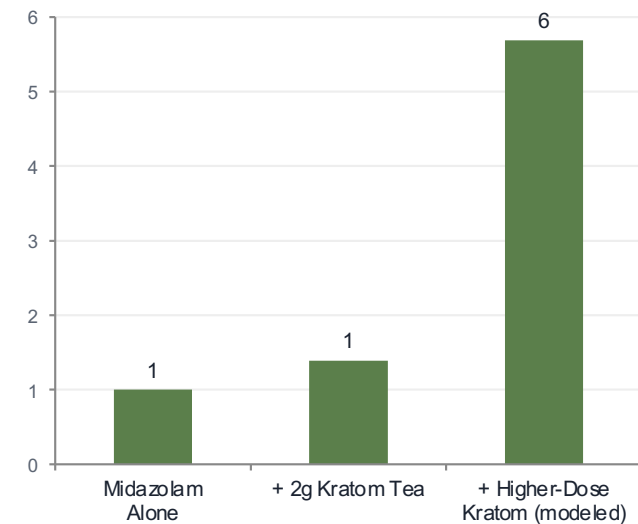
Sources: Olsen EO et al., MMWR 2019;68:326–327; Grundmann O et al., Front Pharmacol 2024; Papsun et al., 2023.

SECTION 3 CLINICAL INTEGRATION

Kratom Drug & Drug Interactions: A CYP450 Problem

- Mitragynine is a competitive and time-dependent inhibitor of CYP3A and a competitive inhibitor of CYP2D6
- Clinical PK study: a single low kratom dose (2 g tea) increased midazolam AUC by ~39% and C_{max} by ~50% primarily via intestinal CYP3A inhibition
- Modeling predicts substantially larger effects at higher, real-world kratom doses with potential for clinically significant increases in exposure to CYP3A/2D6 substrates
- Relevant substrates include many opioids, benzodiazepines, some antidepressants, and select cardiac medications

MIDAZOLAM EXPOSURE WITH KRATOM CO-DOSE



Fold-change in AUC (relative to 1.0 = midazolam alone)

Sources: Tanna RS et al., *Clin Pharmacol Ther* 2023;113:1–10; Tanna RS et al., *J Pharmacol Exp Ther* 2021;376:64–73.

SECTION 3 CLINICAL INTEGRATION

Stacking Sedatives: Cannabis + Opioids + Benzodiazepines

Three overlapping mechanisms compound respiratory risk when these agents are combined.



Pharmacodynamic

CB1, μ -opioid, and GABA-A signaling converge on brainstem respiratory centers additive-to-synergistic ventilatory depression



Pharmacokinetic

CBD inhibits CYP3A4/CYP2D6, slowing clearance of many opioids and benzodiazepines raising blood levels beyond the prescribed dose



Sedation / Arousal

Combined sedation blunts arousal responses to hypoxia/hypercapnia critical during sleep and in the post-operative period

Clinical takeaway: 56% of postoperative opioid-induced respiratory depression cases involved a concurrent sedative order patients with coexisting pulmonary disease or sleep-disordered breathing are at highest risk.

Sources: Boon M et al., *Ann Palliat Med* 2020;9:542–554; van Dorp et al. protocol data (THC–oxycodone ventilatory interaction).

SECTION 3 CLINICAL INTEGRATION

Perioperative & Anesthesia Considerations

- Kratom is not reliably detected on standard toxicology panels - relies on patient self-report
- CYP3A/2D6 inhibition may alter metabolism of induction agents, benzodiazepines, and opioids used perioperatively
- Abrupt discontinuation before surgery can precipitate withdrawal (anxiety, autonomic instability) that mimics or masks other perioperative complications
- Cannabis: chronic heavy users have shown tolerance requiring higher anesthetic/analgesic dosing in some case series; acute intoxication increases risk of hypotension, arrhythmia, and airway reactivity
- Both substances warrant explicit, non-judgmental preoperative screening — professional societies increasingly recommend structured disclosure questions

PREOP SCREENING PROMPTS

- “Do you use kratom, cannabis, or CBD products and how often?”
- “When was your last use before today?”
- “What form: smoked, vaped, edible, tea, capsule?”
- “Are you also taking opioids, benzodiazepines, or sleep aids?”

Sources: Narrative Review of Kratom Perianesthetic Implications, PubMed 2022; ASA statement on perioperative cannabis use.

Building a Substance-Inclusive Sleep & Respiratory History

Ask Directly, Without Judgment

- Normalize the question: “Many of my patients use cannabis or kratom ... do you?”
- Ask about frequency, dose/form, and timing relative to sleep or symptoms
- Clarify reason for use (sleep, pain, anxiety, energy, withdrawal management)

Map the Full Medication List

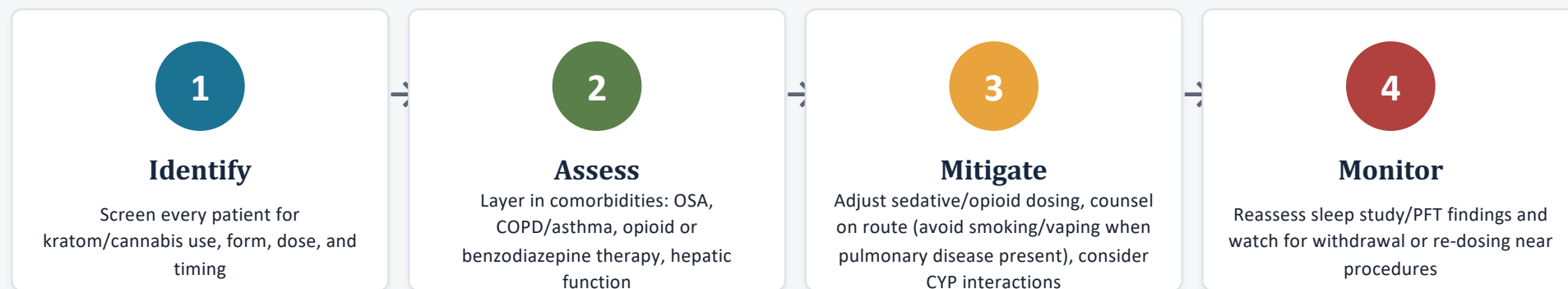
- Include OTC, herbal, and supplement use not just prescriptions
- Flag CYP3A/2D6 substrates (opioids, benzodiazepines, some antidepressants)
- Reassess at every visit use patterns and product potency change quickly

Connect to Respiratory/Sleep Findings

- Correlate reported use with AHI, oxygen desaturation, or daytime sleepiness trends
- Screen for bronchitis symptoms in any regular smoked/vaped cannabis user
- Document clearly this is a safety issue, not a moral one

A Practical Risk-Stratification Framework

Section 3 Clinical Integration



Higher-Risk Profile - Escalate Caution

- Concurrent opioid and/or benzodiazepine prescription + regular kratom or high-THC cannabis use
- Known OSA, COPD, or restrictive lung disease with any smoked/vaped substance use
- Perioperative period, especially within 24–48 hours of last use
- Products of unknown potency or purity (illicit vape cartridges, unregulated kratom extracts)

CASE VIGNETTE

Case 1 Kratom, an Opioid Prescription, and Undiagnosed OSA

Presentation

A 48-year-old man with chronic low back pain, prescribed oxycodone 10 mg BID, reports using kratom powder “a couple times a day” to “take the edge off” and help him sleep. He has loud snoring, witnessed apneas per his partner, and daytime sleepiness (Epworth 14). He has never had a sleep study and did not mention kratom use until directly asked.

Discussion Questions

- What respiratory risks does this combination raise, and why?
- How would you approach discontinuation vs. harm-reduction counseling?
- What work-up would you prioritize before any medication changes?

Clinical Pearl

Undiagnosed OSA plus opioid plus kratom (unpredictable 7-OH-MG content) is a respiratory-depression risk stack screen for sleep-disordered breathing before adjusting any sedating medication.

CASE VIGNETTE

Case 2 Cannabis Edibles, a Benzodiazepine, and Fragmented Sleep

Presentation

A 61-year-old woman on long-term alprazolam for anxiety reports taking a 10 mg THC edible most nights because “it knocks me right out.” She complains of feeling unrefreshed despite 8 hours in bed, with vivid, unpleasant dreams on the nights she skips the edible. She has mild COPD (FEV1 68% predicted).

Discussion Questions

- What does her sleep architecture likely look like on polysomnography?
- What CYP-mediated interaction should you consider with her alprazolam?
- How would you frame a conversation about tapering or route changes?

Clinical Pearl

Nightly THC use suppressing REM plus CBD/THC effects on benzodiazepine clearance is a pharmacokinetic and architecture problem — not just a preference issue. Address both together.

Clinical Practice Recommendations

Section 3 Clinical Integration

1 Screen routinely

Ask every respiratory and sleep-clinic patient about kratom and cannabis use as part of the standard medication reconciliation.

2 Clarify route & product

Route of administration and product potency change risk more than “use vs. no use” document specifics.

3 Reconcile for interactions

Check for CYP3A/2D6-mediated interactions with opioids, benzodiazepines, and other sedatives before adjusting doses.

4 Counsel on pulmonary risk

Advise against smoking/vaping in patients with COPD, asthma, or planned surgery; discuss non-inhaled alternatives.

5 Avoid reflexive alarm or dismissal

Evidence is nuanced — neither substance is uniformly benign nor uniformly dangerous; individualize the conversation.

6 Reassess longitudinally

Use patterns, product potency, and regulations are shifting quickly, revisit at each visit, not just intake.

Key Takeaways

Pharmacology isn't uniform

Mitragynine ≠ 7-OH-mitragynine; THC ≠ CBD. Which alkaloid/cannabinoid dominates changes the entire respiratory risk calculus.

Sleep effects cut both ways

Both substances can shorten sleep latency while degrading sleep architecture (REM suppression, fragmentation) subjective benefit ≠ objective quality.

Route of use matters as much as substance

Smoking and vaping carry distinct pulmonary risks (bronchitis, EVALI) that oral/tea forms do not share.

Polypharmacy is the dominant danger

Nearly all kratom-associated deaths, and the highest-risk cannabis scenarios, involve concurrent opioids, benzodiazepines, or other CNS depressants.

Selected References

Blount BC, et al. Vitamin E Acetate in Bronchoalveolar-Lavage Fluid Associated with EVALI. *N Engl J Med*. 2020;382:697–705.

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Tanna RS, et al. Clinical Assessment of the Drug Interaction Potential of Kratom. *Clin Pharmacol Ther*. 2023;113:1085–1095.

Tanna RS, et al. Refined Prediction of Pharmacokinetic Kratom-Drug Interactions: Time-Dependent Inhibition. *J Pharmacol Exp Ther*. 2021;376:64–73.

Thank You

Questions & Discussion

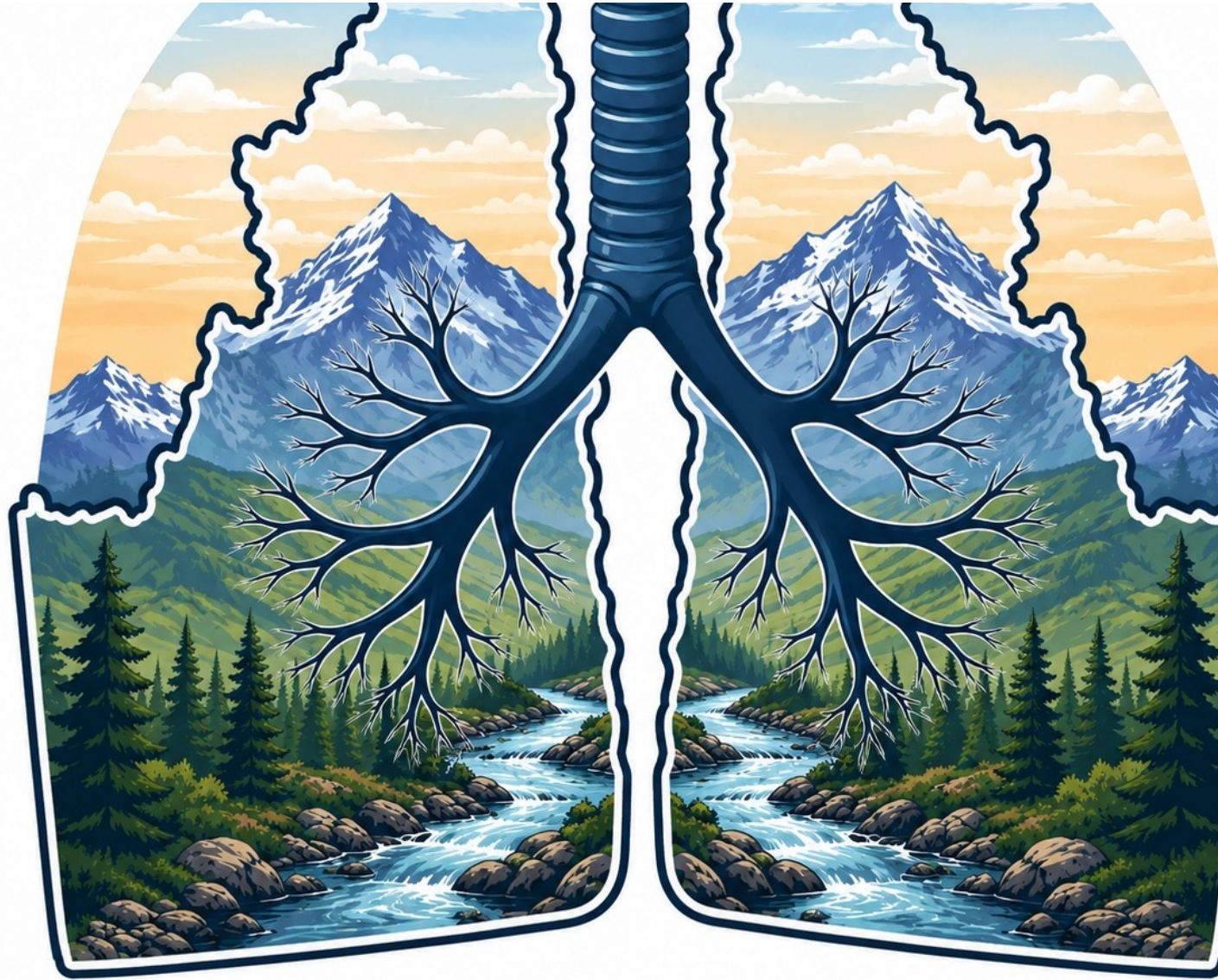
Dr. Amanda L. Leightner PhD, RRT, RRT-SDS, RPSGT, CCSH, FAARC, FNAP
Merrick Family Endowed Professor
Boise State University

Amandaleightner@boisestate.edu

ISRC

Annual Business Meeting

- AHAB Report
- Financial Forecast
- Changes to NBRC Testing
- HOD/Governmental Affairs Report
- Election Updates



Idaho Allied Health Advisory Board Legislative/Law Update 2026



Tim Seward, MBA, BS, RRT-NPS
Board Member

Disclosure & Disclaimers

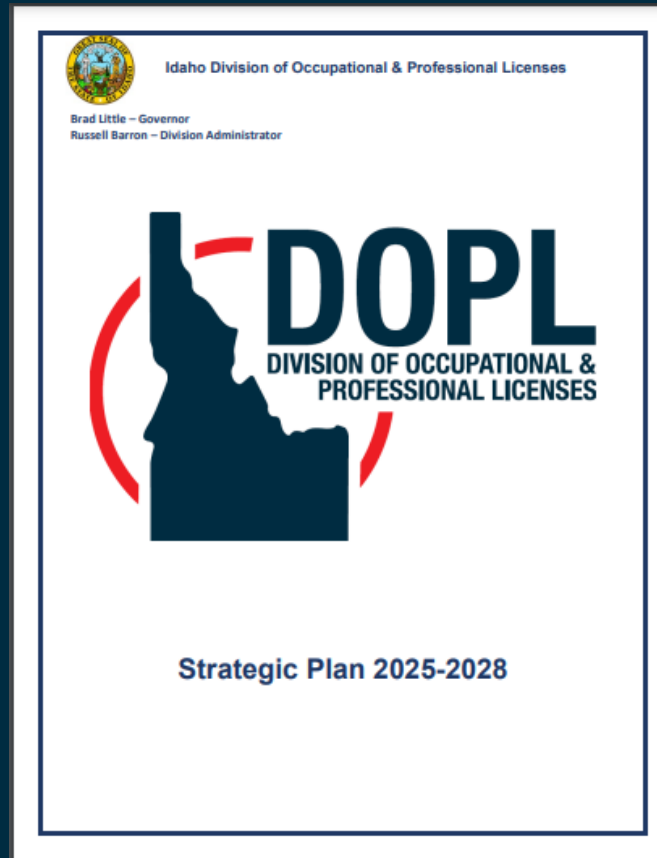
- No conflicts to disclose.
- Representing the Idaho Allied Health Advisory Board and the Division of Occupational and Professional Licenses (DOPL).



The Idaho Division of Occupational and Professional Licenses (DOPL) is providing this guidance as a public service. It is intended to provide general guidance and is non-binding. This does not constitute legal advice, nor may DOPL, its officers, employees, or agents be held liable for actions taken or omissions made in reliance on any information contained herein. The guidance does not have the full force or effect of law. If there is any discrepancy between this publication and the law, the provisions of the law will prevail.

Idaho Allied Health Advisory Board

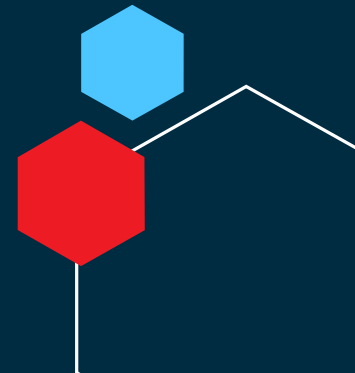
- Established through Executive Order 2020-10
- Brought together 48 Board and Commissions
- Divided into 4 Bureaus:
 - BCRE
 - OL
 - HP
 - Admin



Mission:
Consumer Protection and Public Safety

Vision:
Right Touch Regulation

Core Values:
Quality customer service, Balanced and fair regulation, Integrity, and Respect



Allied Health Advisory Board

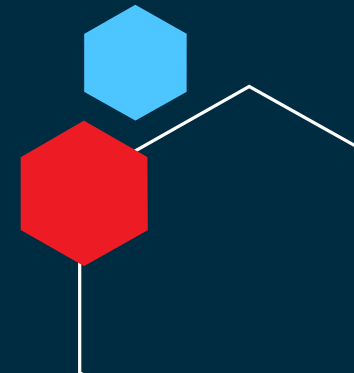


License Type	2025	2026
Athletic Trainers	315	380
Dietitians	934	1,345
Naturopathic Medical Doctors (NMD)	85	99
Respiratory Therapy	1,262	1,475
Total:	2,596	3,299

Respiratory Therapy	2025	2026
RT	108	125
PSG	1,153	1,350

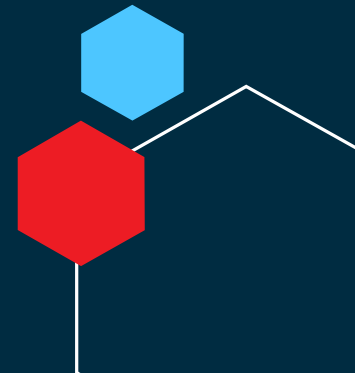


Same Day Licensure

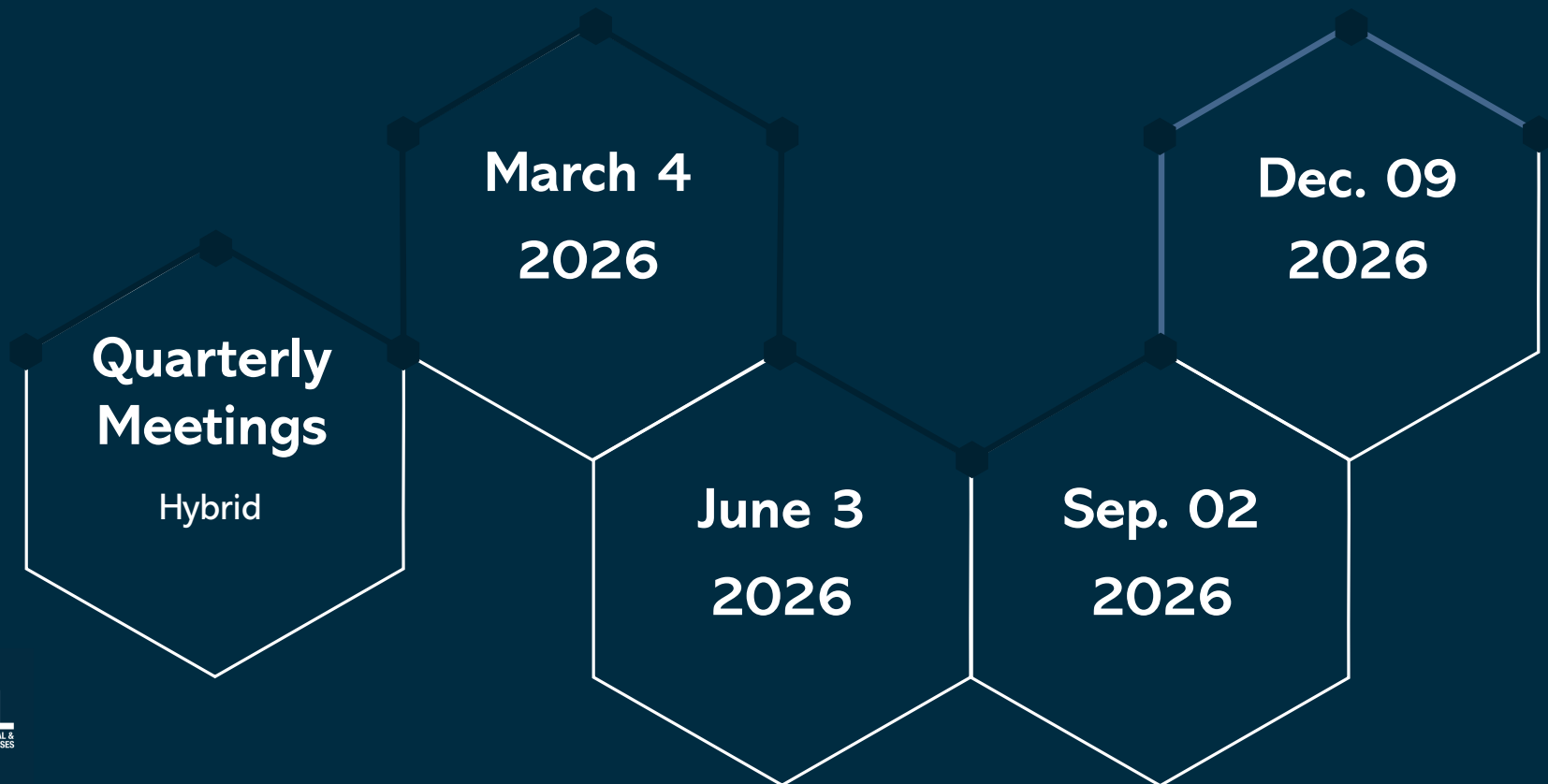


Allied Health Advisory Board Composition

Position	Name	Expiration
RT	Tim Seward	July 1, 2026
NMD	Cory Szybala	July 1, 2026
MD	Tara Erbele	July 1, 2027
DT	Kimberly Young	July 1, 2028
RT/PSG	Robb Hruska	July 1, 2028
AT	Dave Hammons	July 1, 2028
Public Member	Jessica Carman	May 22, 2030



Public Meetings

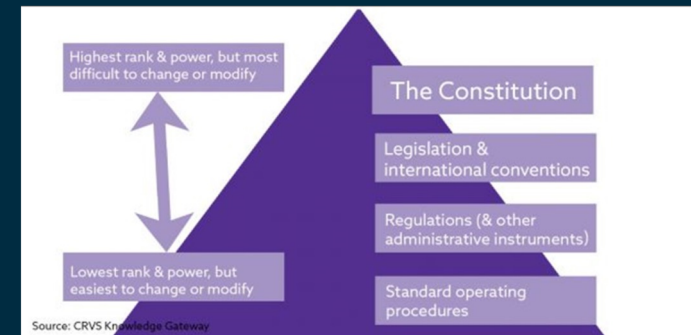


Statute vs Rule

- Statute - Idaho Code or State Law
 - Designated by Title and Chapter (54-17, 54-57, 67-26)
 - Set forth by the legislature, often in a constitution
 - Grants the Board their authority and the ability to promulgate rules
- Rule - Idaho Administrative Code (IDAPA)
 - IDAPA 24.36.01
 - May also be referred to as regulations
 - Derives authority from law and carries the same force
 - Fills in details beyond the statute
 - Approved by the legislature



Follow different paths through the legislature



Source: CRVS Knowledge Gateway

Zero Based Regulation (ZBR)

- All agency rule chapters shall be reviewed and be promulgated as specified in this Executive Order no later than sine die in 2026
- The agency review schedule shall be staggered
- Agencies start the new rulemaking from a zero-base and not seek to simply reauthorize their existing rule chapter.

Agencies must use the prospective analysis



Zero Based Regulation Impact

- Eliminate redundancies found in rule and statute
- Ensure practice standards are commensurate with surrounding states
- Remove unclear language and any potential conflicts with statute
- Hold licensees to high **Standards of Care**



2025 Position Statement

- Categorizes risk for respiratory equipment, supplies and medications and the potential of oversight needed.
 - High Risk:
 - Invasive mechanical ventilation
 - Moderate Risk:
 - Intermittent positive pressure devices, BiPAP rate >10.
 - Low Risk:
 - CPAP or BiPap with backup rate of less than 10



<https://dopl.idaho.gov/wp-content/uploads/2025/10/BOM-RT-Position-Statement.pdf>



BRAD LITTLE
Governor
RUSSELL BARRON
Administrator

State of Idaho
Division of Occupational and Professional Licenses
Board of Medicine

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dopl.idaho.gov

IDAHO STATE BOARD OF MEDICINE POSITION STATEMENT HOME DELIVERY AND SET UP OF RESPIRATORY EQUIPMENT, SUPPLIES, & MEDICATIONS

In Idaho, patients may have respiratory equipment, supplies, and medications delivered to their homes. The Board of Medicine recommends that a licensed respiratory care practitioner or a qualified healthcare provider educate the patient on usage and dosage at the time of prescribing. A healthcare provider should use professional judgement whether to provide services in person or through telehealth. For moderate and high risk respiratory devices, this provider should also perform the initial setup, assess the patient's tolerance, and conduct a clinical evaluation to ensure safe and effective use. The Board of Medicine believes that respiratory care services oversight may vary based on the level of risk associated with the intervention. Patients should consult with their medical team prior to making any changes to their treatment plan, including medication dosage, oxygen liter flow, equipment settings, or location.

High Risk: A respiratory therapist, pulmonologist, or other specialized healthcare provider trained in the management of respiratory disease should oversee the set-up, initiation, and patient training.

- Invasive mechanical ventilation

Moderate Risk: While any properly trained and licensed personnel can perform the initial setup, education, and training for the patient, it is recommended that a respiratory therapist or healthcare provider be involved in the process.

- Intermittent positive pressure breathing IPPB devices
- Intrapulmonary percussive ventilation devices
- Mechanical in-exsufflation devices
- BiPAP devices or other positive pressure devices with a backup rate greater than 10

Low Risk: A qualified healthcare professional should provide patient education and training. The initial setup can be performed by any individual qualified in the setup and delivery of the device or supplies.

- High frequency chest wall oscillation devices and supplies
- Nebulized equipment, supplies, and medications
- Oxygen equipment and/or supplies
- Positive pressure airway devices
- CPAP or BiPAP with a backup rate less than 10

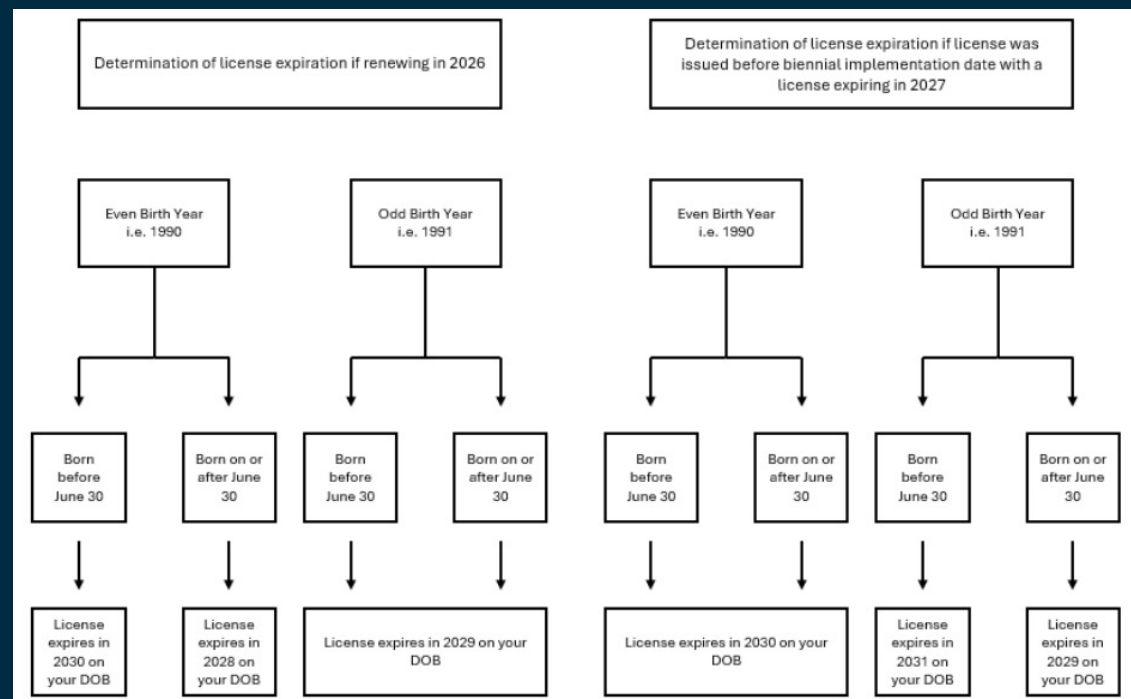
This position statement may not reflect public or private payer requirements. Individuals are encouraged to review payer criteria for Durable Medical Equipment (DME).

The Idaho Division of Occupational and Professional Licenses (DOPL) is providing this guidance document as a public service. It is intended to provide general guidance and is non-binding. This document does not constitute legal advice, nor may DOPL, its officers, employees, or agents be held liable for actions taken or omissions made in reliance on any information contained herein. Guidance documents do not have the full force or effect of law. If there is any discrepancy between this publication and the law, the provisions of the law will prevail.

Updated: 8/28/2025

Transition to Biennial Birthdate Licenses

- Ensure the Board has consistent predictable financials.
- Standardize Boards renewals to ensure licenses are issued as efficiently as possible.
- Fees reflect the elongated renewal period.
- Continuing education requirements remain the same.



<https://dopl.idaho.gov/pressrelease/big-changes-are-on-the-way-stay-informed-about-important-licensure-updates-that-may-impact-your-renewal-cycle-33/>



What questions do you have?

Orin.Duffin@dopl.idaho.gov

Tim.Seward@portmed.org

Treasury Report

2025 Final Revenue and Expenses

Revenue: \$21,056

Conference: \$ 17,384

AARC revenue share: \$3,672

Expenses: \$23,860

Conference: \$10,600

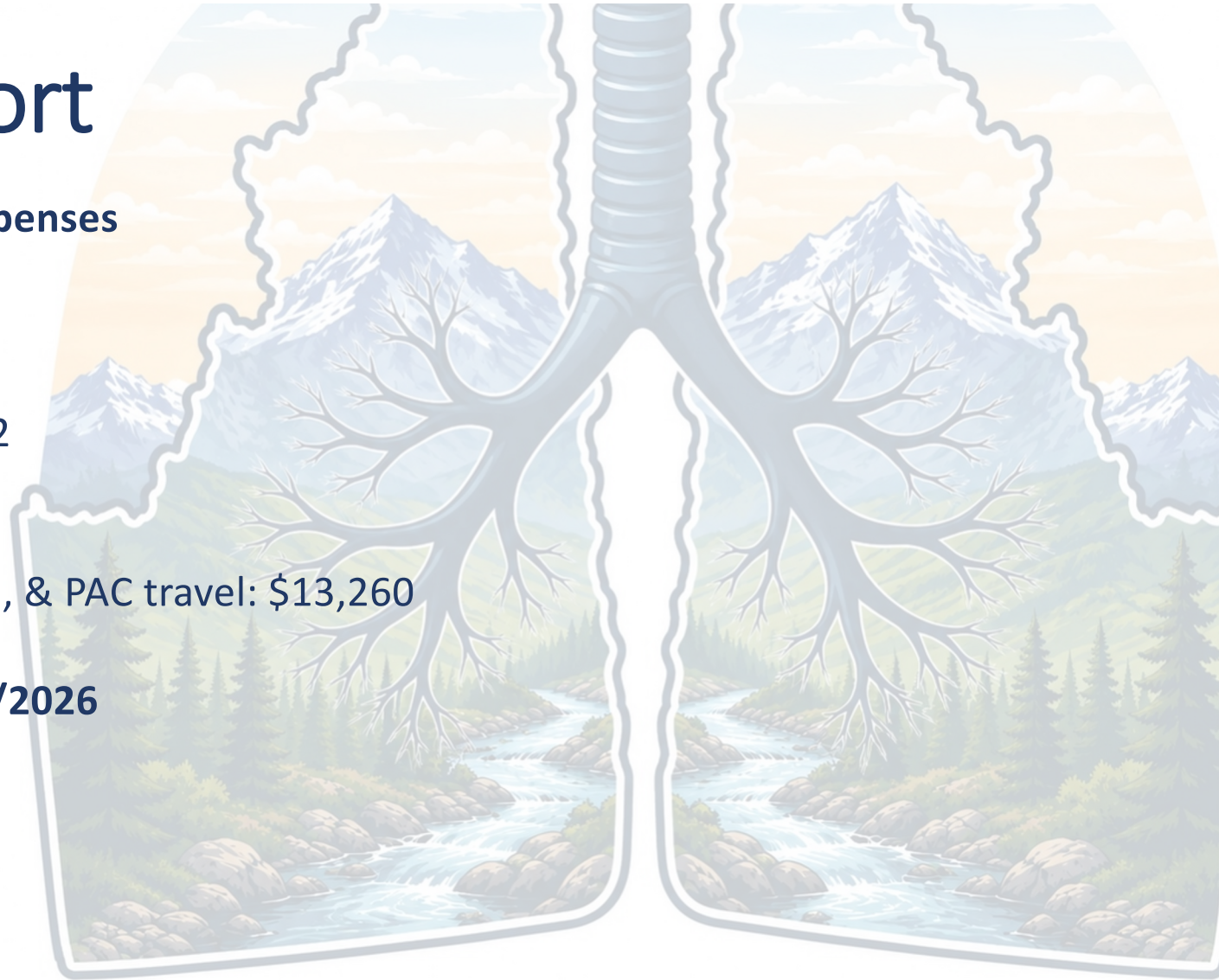
Advocacy, Board HOD travel, & PAC travel: \$13,260

Current Balance as of 9/18/2026

Checking: \$39,085

Savings: \$23,105

Total: \$62,190



Treasury Report- Position Change Proposals

Assistant Treasurer Position

- Increases transparency of treasurer role
- Allows for onboarding of position

Changing Treasurer to an Appointed Position

- In the event of a vacancy of the treasurer position, the assistant treasurer would be appointed to Treasurer

NBRC Changes

Respiratory Therapy Examination

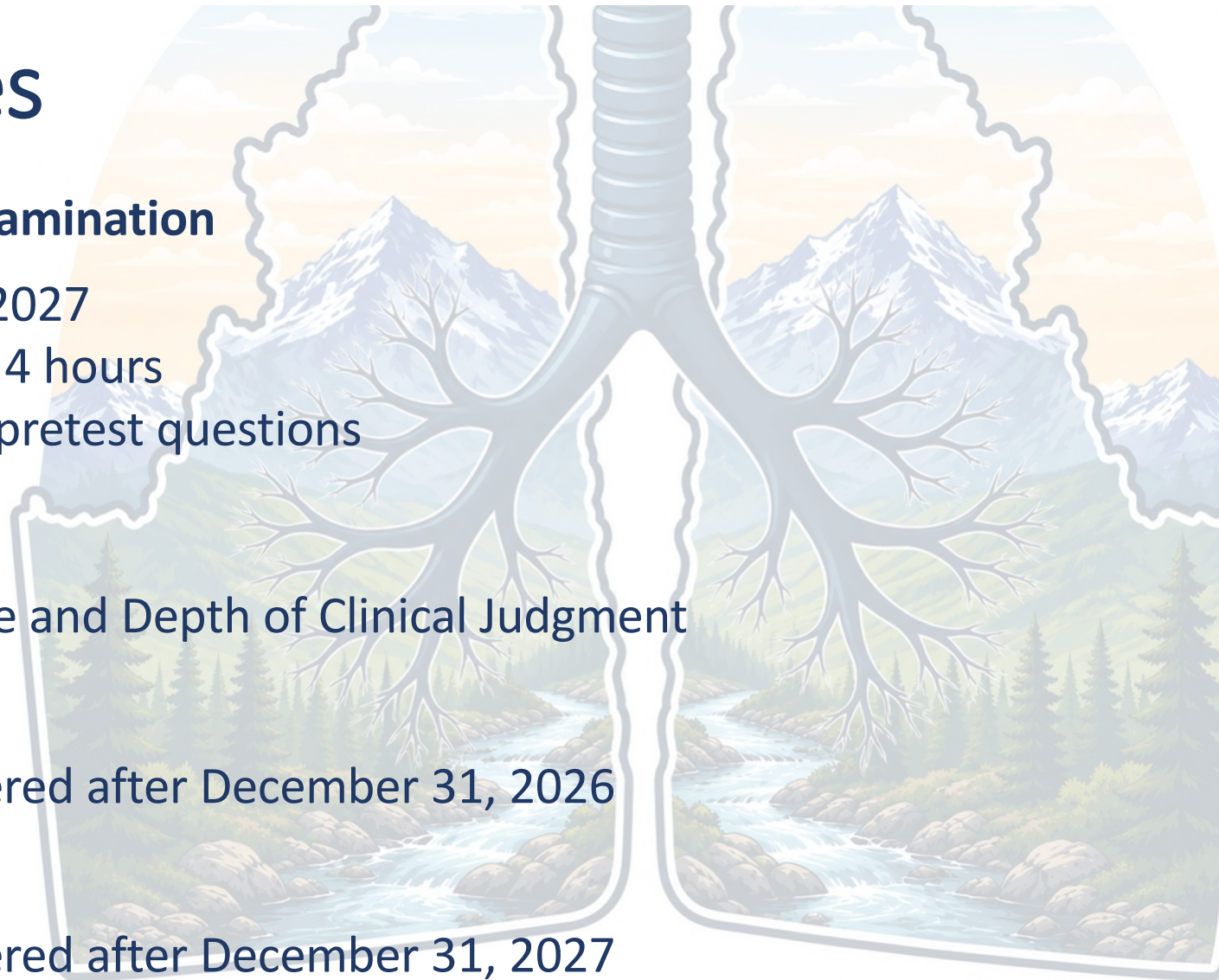
- Launches January 1, 2027
- 185 total questions – 4 hours
 - 160 questions, 25 pretest questions
- High Cut Score = RRT
- Low Cut Score = CRT
- Breadth of Knowledge and Depth of Clinical Judgment

TMC Exam

- Will no longer be offered after December 31, 2026

CSE Exam

- Will no longer be offered after December 31, 2027



HOD Report

PACT

- June 1-2, 2026
- 284 Meetings, 115 participants, 47 States + DC
- Met with ALL 4 Legislators
- Focus was on SOAR

Legislative Updates

- SOAR
- AIR
- SHARE
- RCIC
- Hill Day 2027

Summer Forum HOD

- Executive Director Dan Garrett is no longer with AARC
- Marketing and Faculty Grant Opportunities
- AARC Connect App is LIVE
- Focus has been on
 - Increasing ed opportunities/Member services
 - Increasing transparency
 - Supporting affiliates

We want to hear from YOU

Megankoster@boisestate.edu

Trevorlee@boisestate.edu

president@isrc.org



ISRC BOD Elections

Nominations **OPEN!** (09/23-10/14)

- President-Elect (3 yr. Term)
- Vice President (2 yr. Term)
- Director-at-Large (2 yr. Term)

Descriptions are in Form

Election Cycle to Follow

- 10/26-11/29

Terms Begin January 1, 2027

Nominate Here

