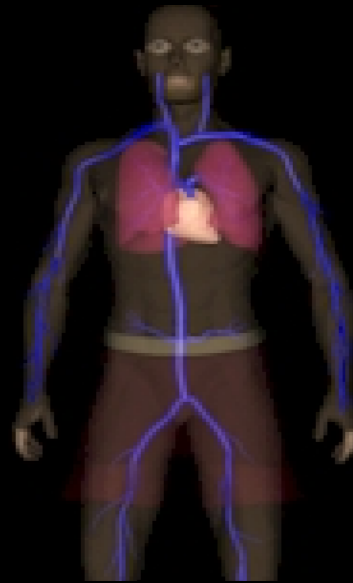


The Laboratory of Clinical Exercise Physiology

Lungs-Heart Interactions and Their Clinical Consequences



J. ALBERTO NEDER, MD, PhD, DSc, FRCPC, FERS

Professor of Respiratory Medicine and Physiology

Queen's University

Kingston, ON

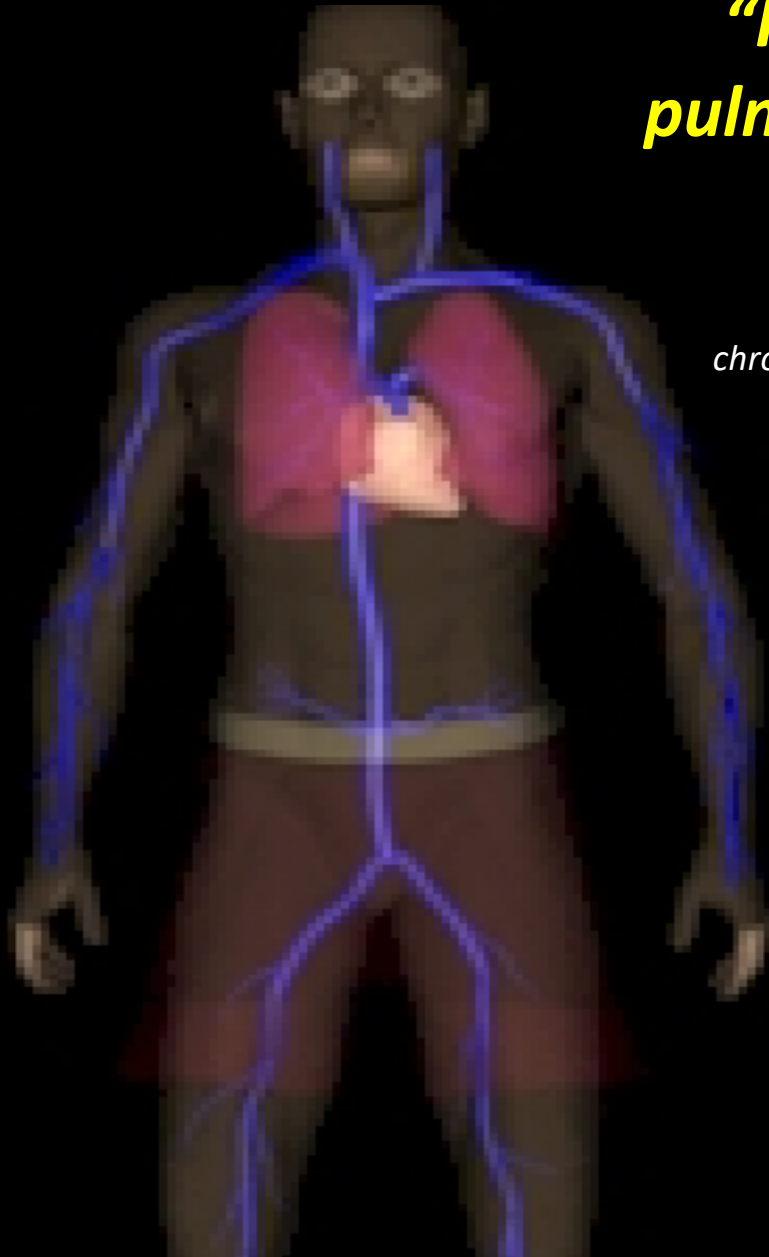
Canada



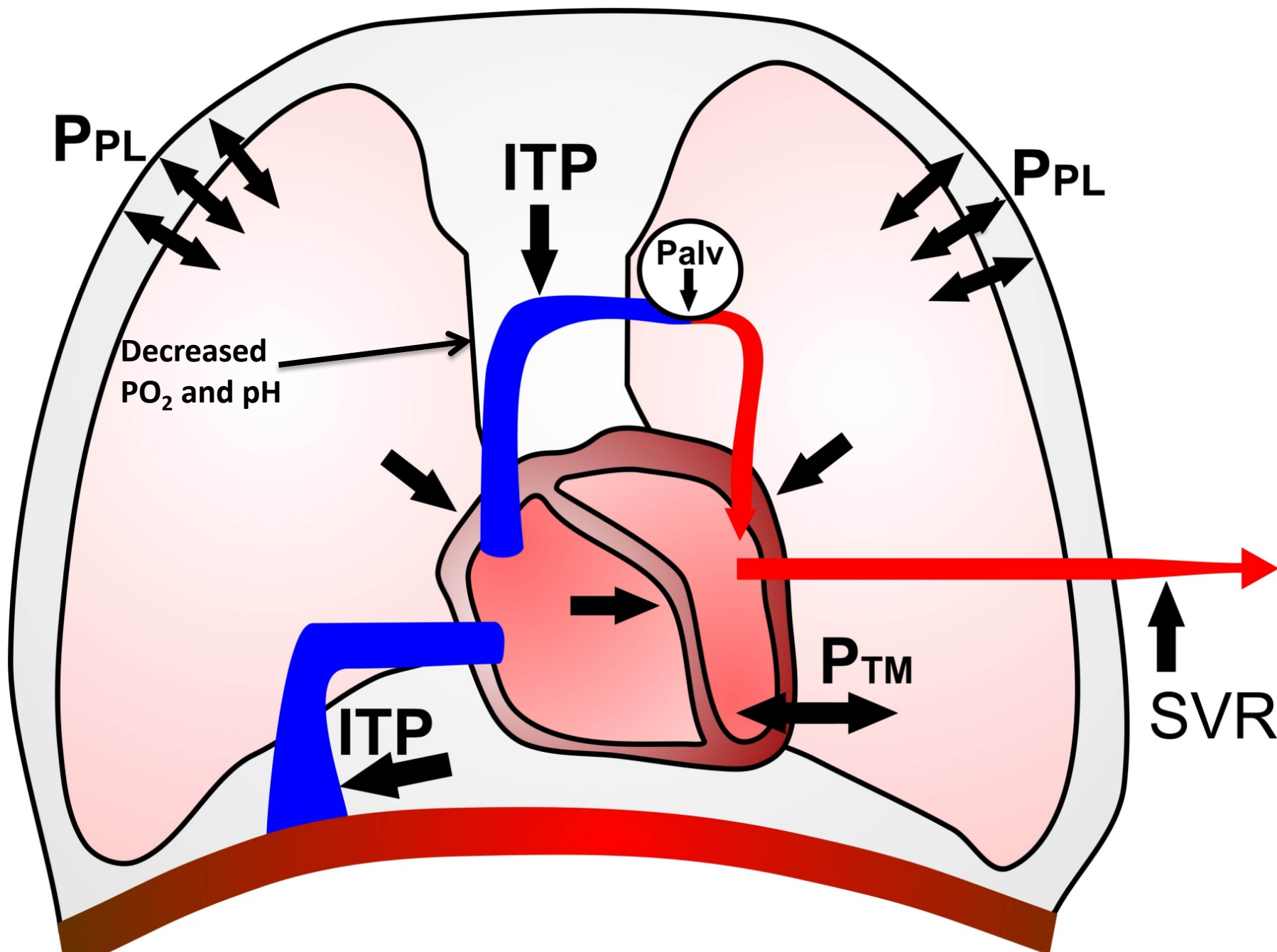
***“Functionally, it is obvious that the
pulmonary and circulatory apparatus
are one unit”***

Baldwin ED, Cournand A, Richards DW.

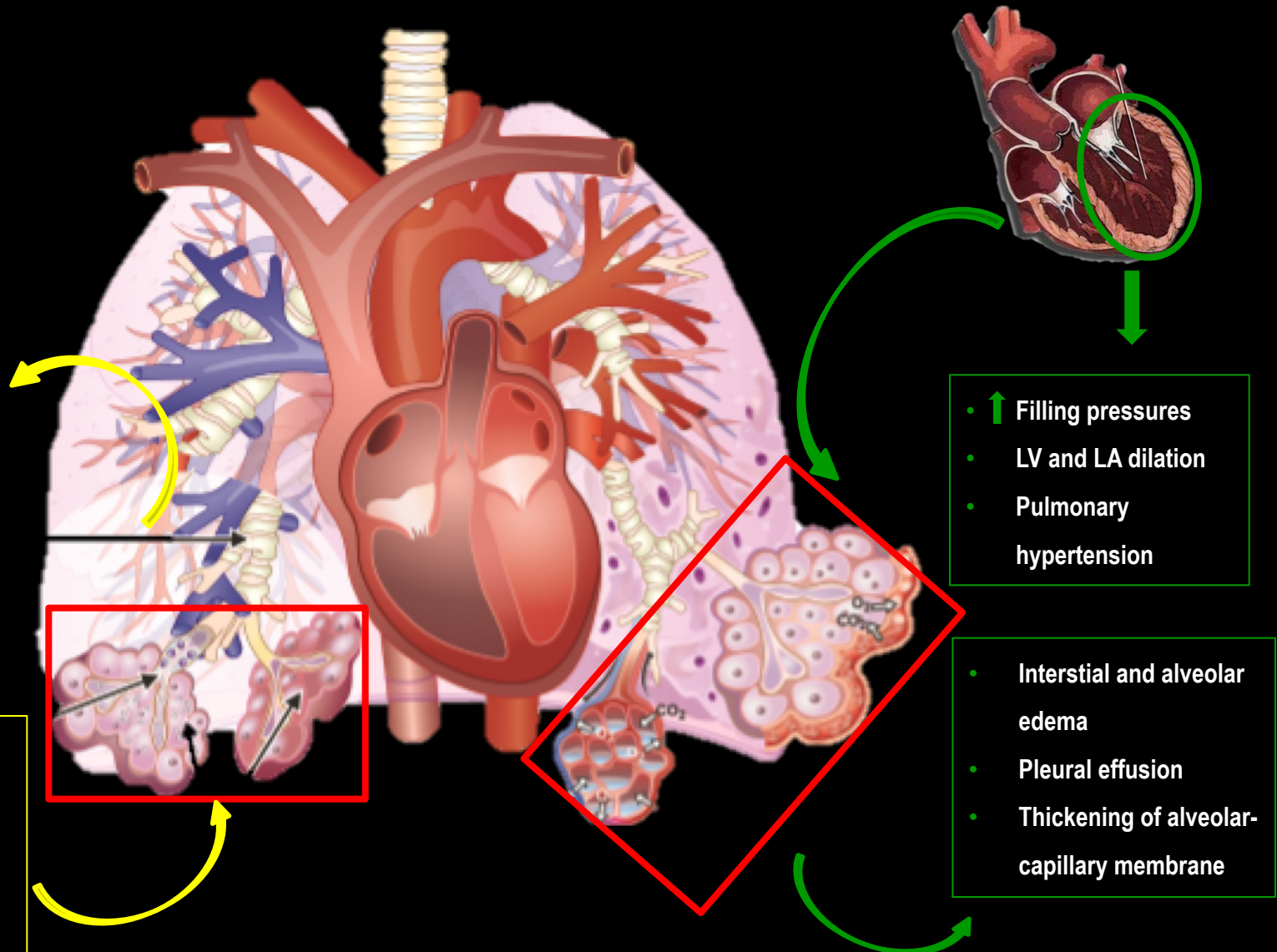
*Pulmonary insufficiency; a study of 122 cases of
chronic pulmonary emphysema. Medicine (Baltimore). 1949;28:201–37.*



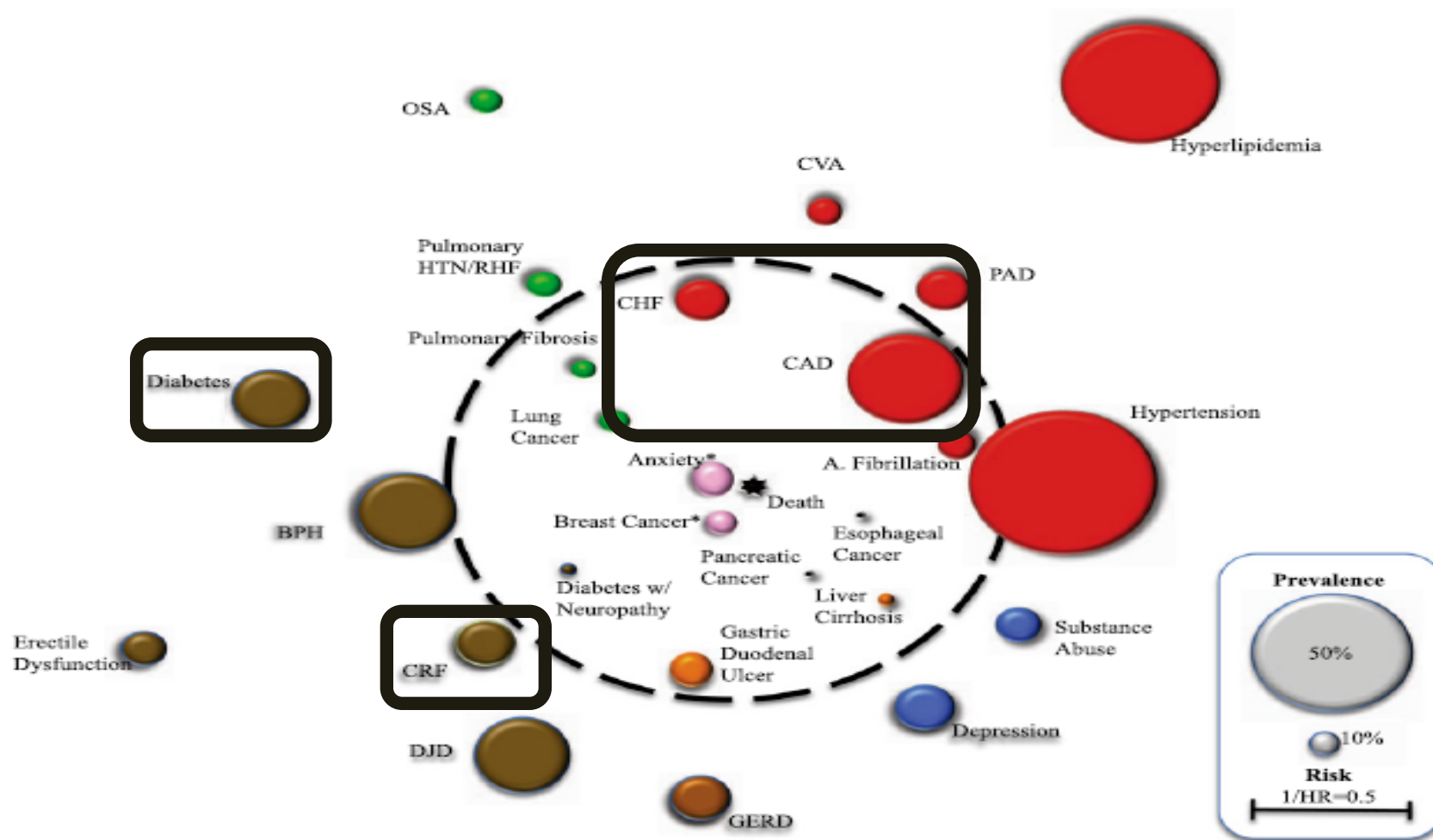
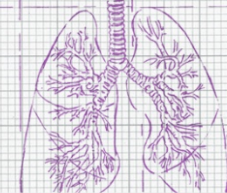
COPD effects *on* cardiac function



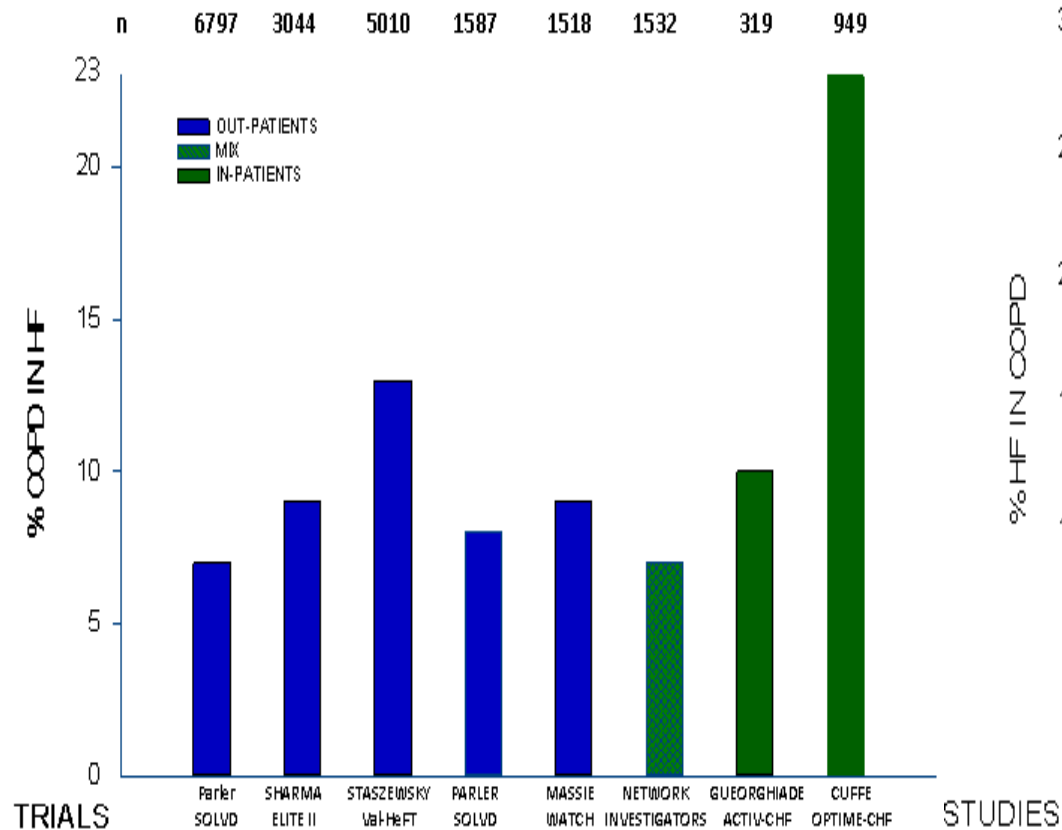
Pulmonary consequences of heart failure



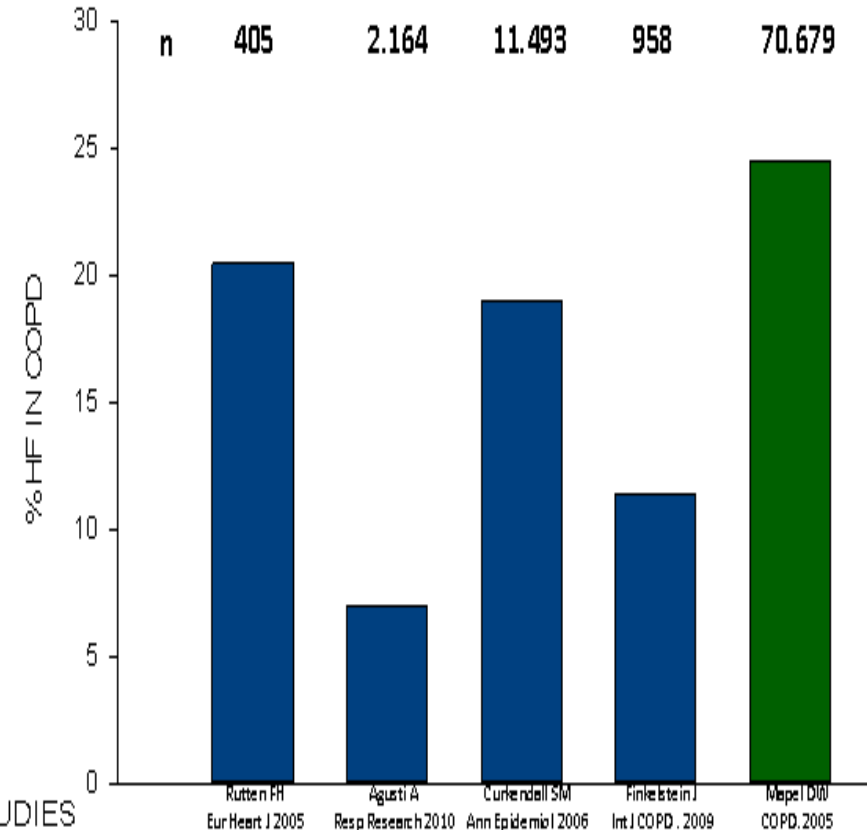
The “Co-Morbidoma” of COPD



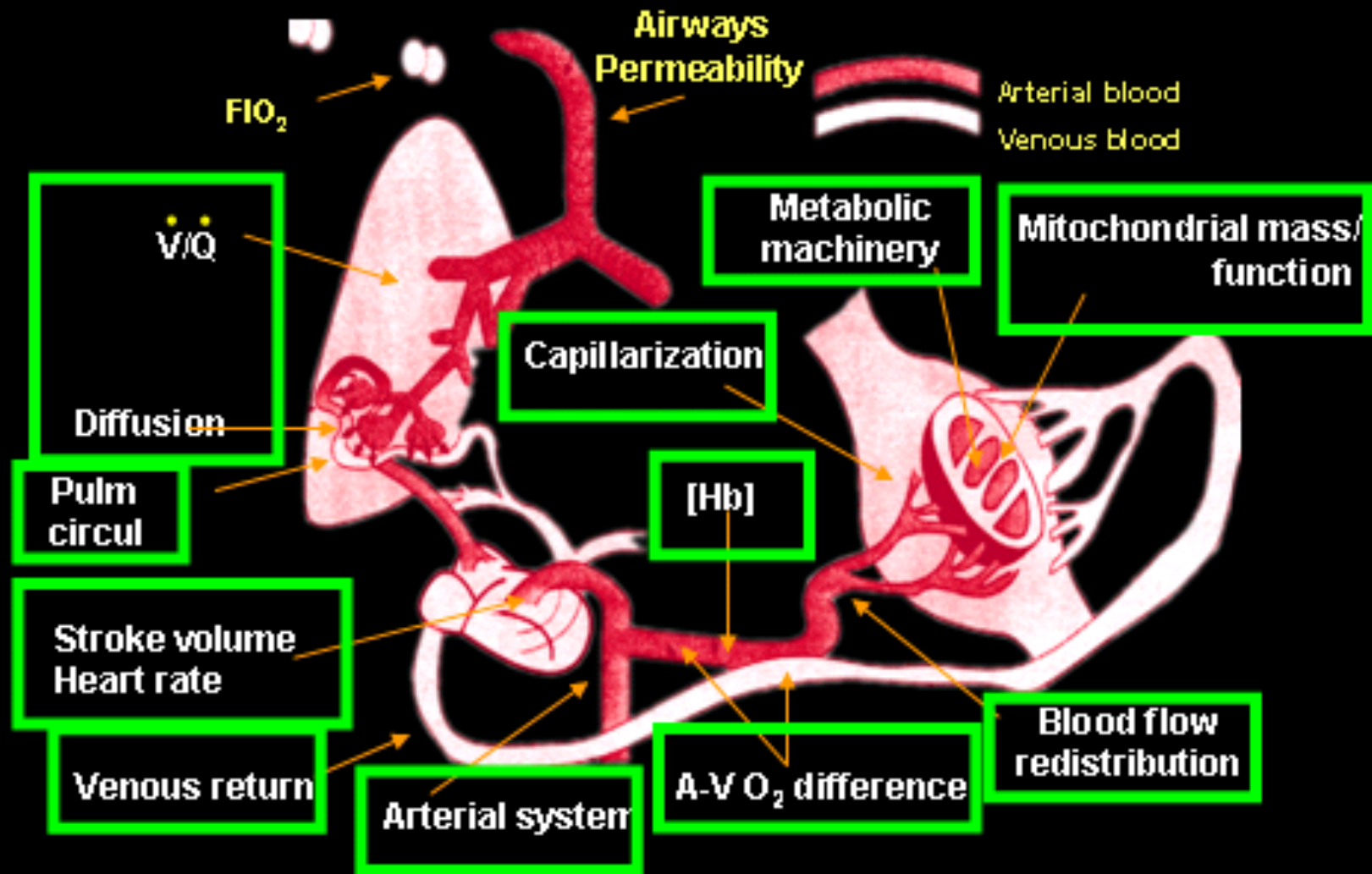
COPD in CHF



CHF in COPD



COPD-CHF Overlap: The “Perfect Storm”





Canada Foundation
for Innovation

Fondation canadienne
pour l'innovation



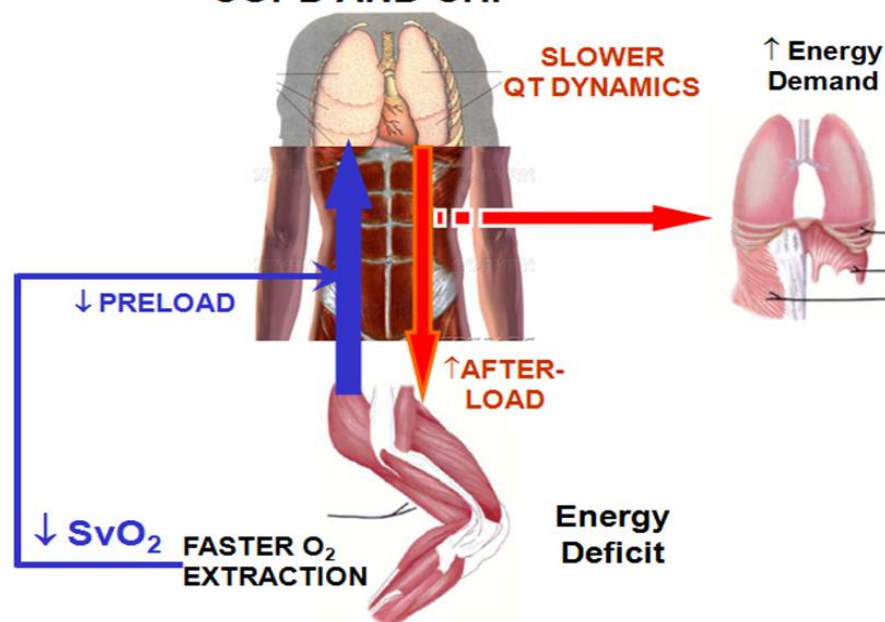
SEAMO
Southeastern Ontario Academic Medical Organization

- **Queen's** Laboratory of Clinical Exercise Physiology in Chronic Cardiopulmonary Diseases

Clinical,
Physiological and
Translational
Investigation in
COPD-CHF OVErlap
(CAPTIVE study)

THE PATHOPHYSIOLOGY OF EXERCISE INTOLERANCE IN COPD PATIENTS WITH CHRONIC HEART FAILURE

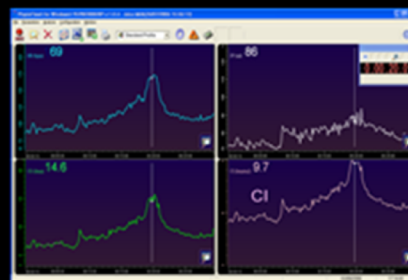
COPD AND CHF



EPOC
Arterialized
Blood



Cardioimpedance
Stroke Volume and
Cardiac Output



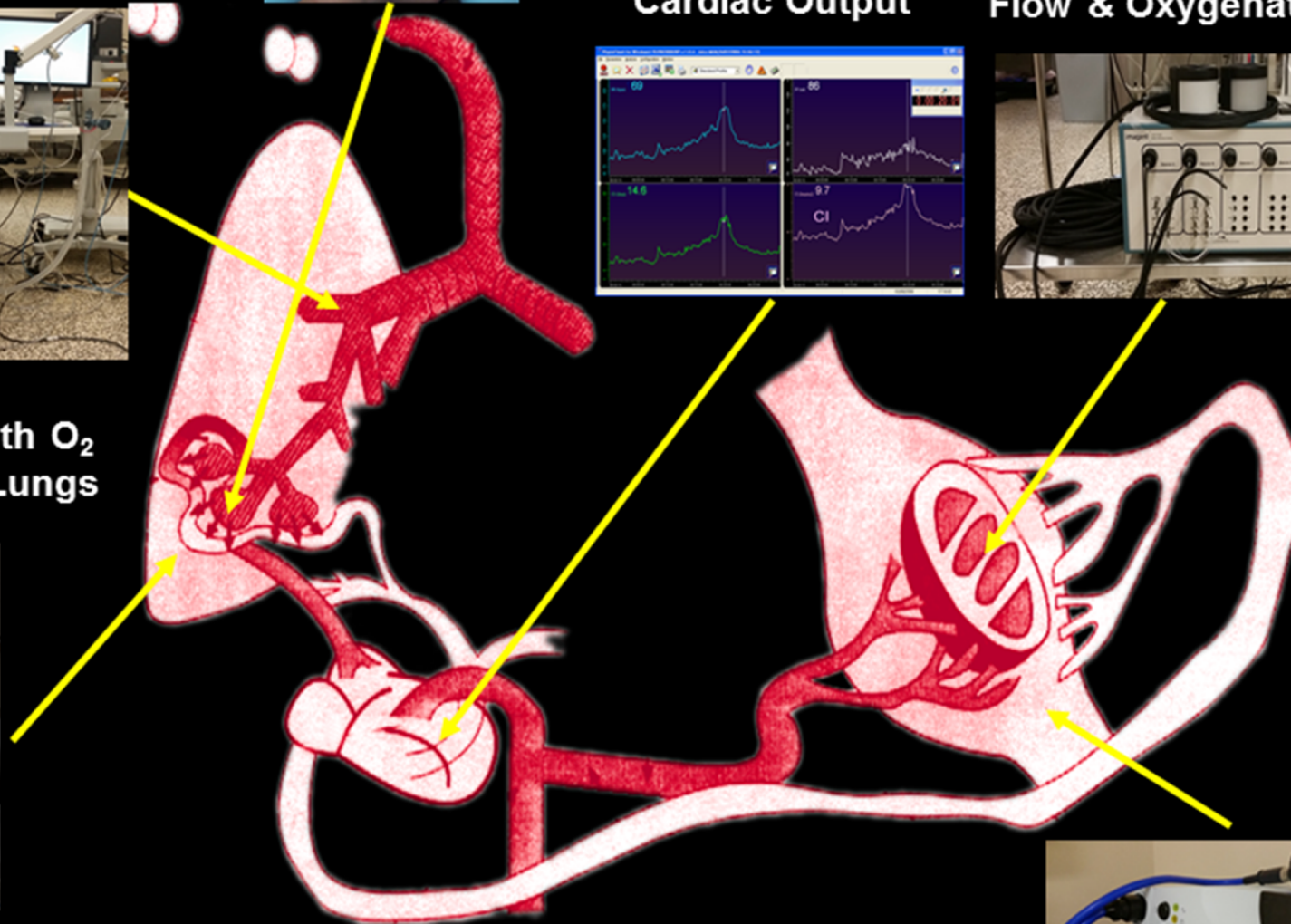
NIRS
Muscle blood
Flow & Oxygenation



CPET
Breath-by-Breath O_2
Uptake by the Lungs



Inert Gas Rebreathing
Pulmonary Blood
Flow



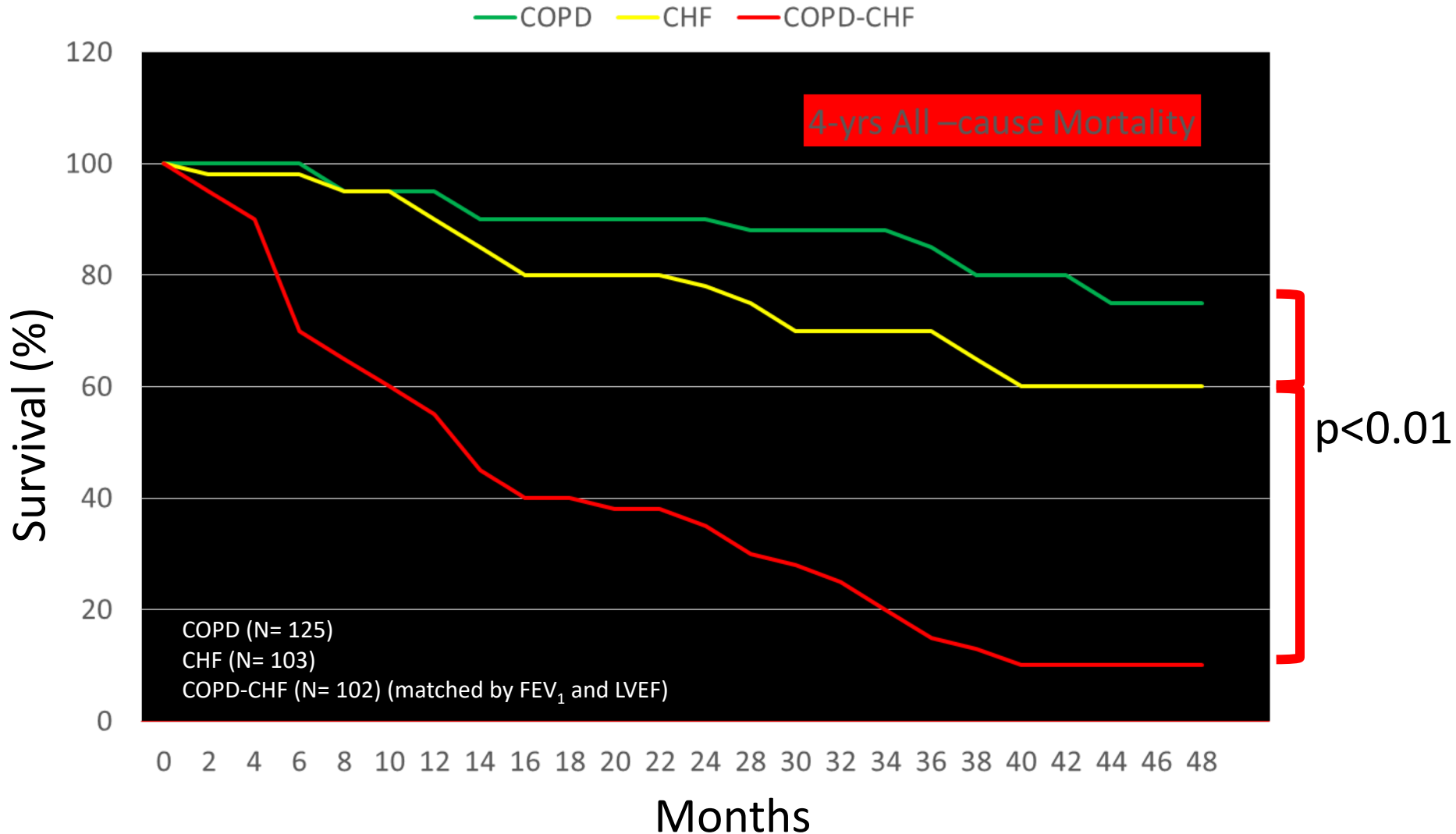
MAGSTIM
Muscle Fatigue



COPD-CHF OVERLAP: A DYSMAYING COMBINATION

The CAPTIVE Study: Main Results

4 yrs, prospective study, COPD-CHF Specialized Clinic



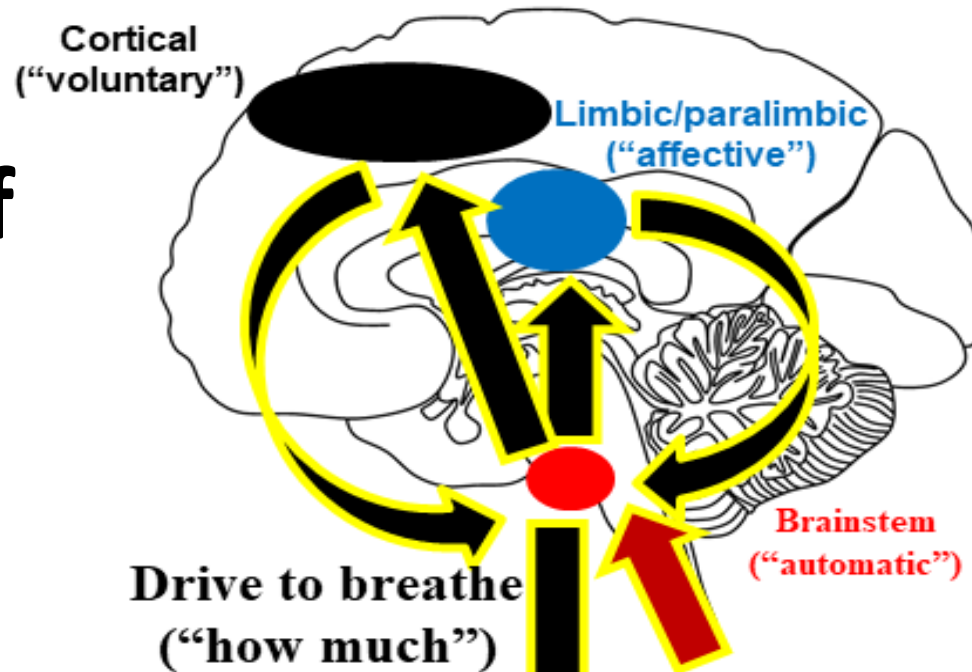
Overarching goals

- To develop a pathophysiological rationale to guide diagnosis and therapeutics in comorbid CHF-COPD
- Diagnosing CHF in COPD and vice-versa
- Treating CHF in COPD and vice-versa

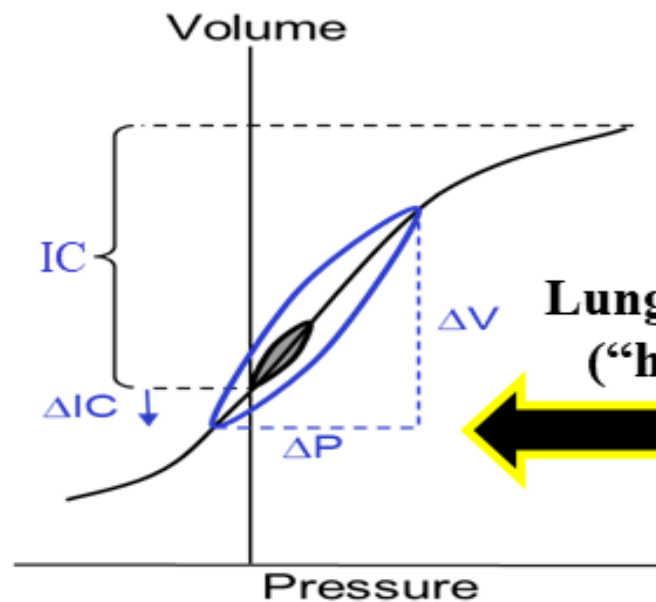
An example of direct translation to clinical care:

- **Why some patients with stable CHF-COPD are extremely short of breath on any exertion compared to others with same resting impairment in heart and lung function?**
- **How does the answer to this question impact on current clinical management ?**

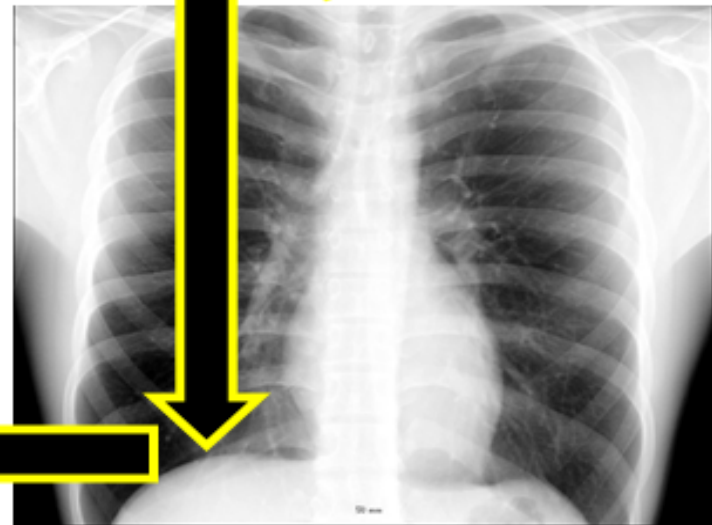
The origins of dyspnea in humans



Drive to breathe
("how much")



Lung mechanics
("how well")

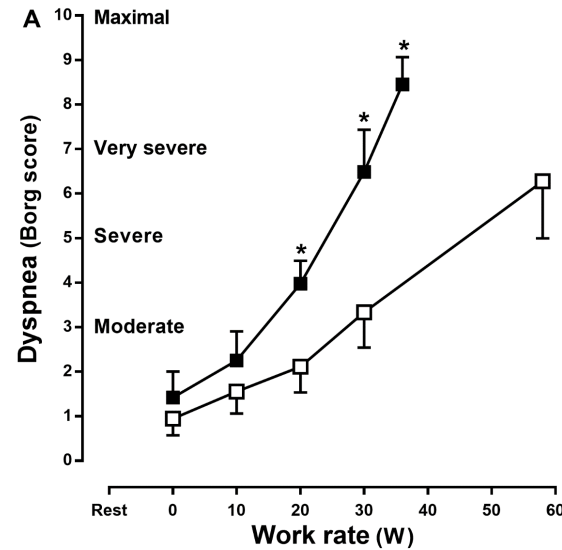


Excess Ventilation in Chronic Obstructive Pulmonary Disease-Heart Failure Overlap. Implications for Dyspnea and Exercise Intolerance.

Rocha A, Arbex FF,Neder JA. *Am J Respir Crit Care Med*. 2017 Nov 15;196(10):1264-1274

AMERICAN JOURNAL OF

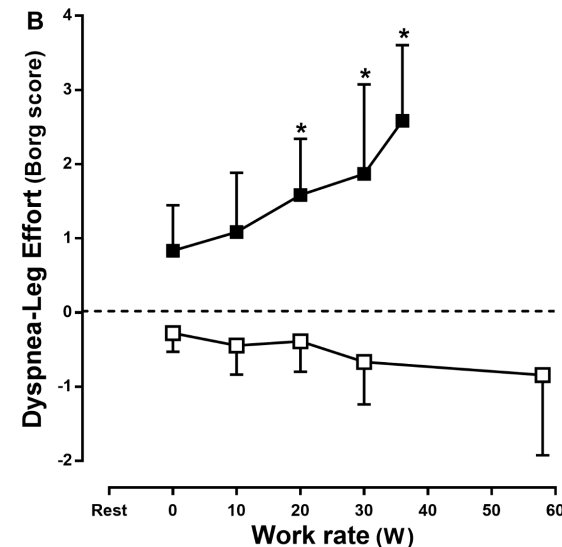
Respiratory and
Critical Care Medicine



Dyspnea burden

■ High

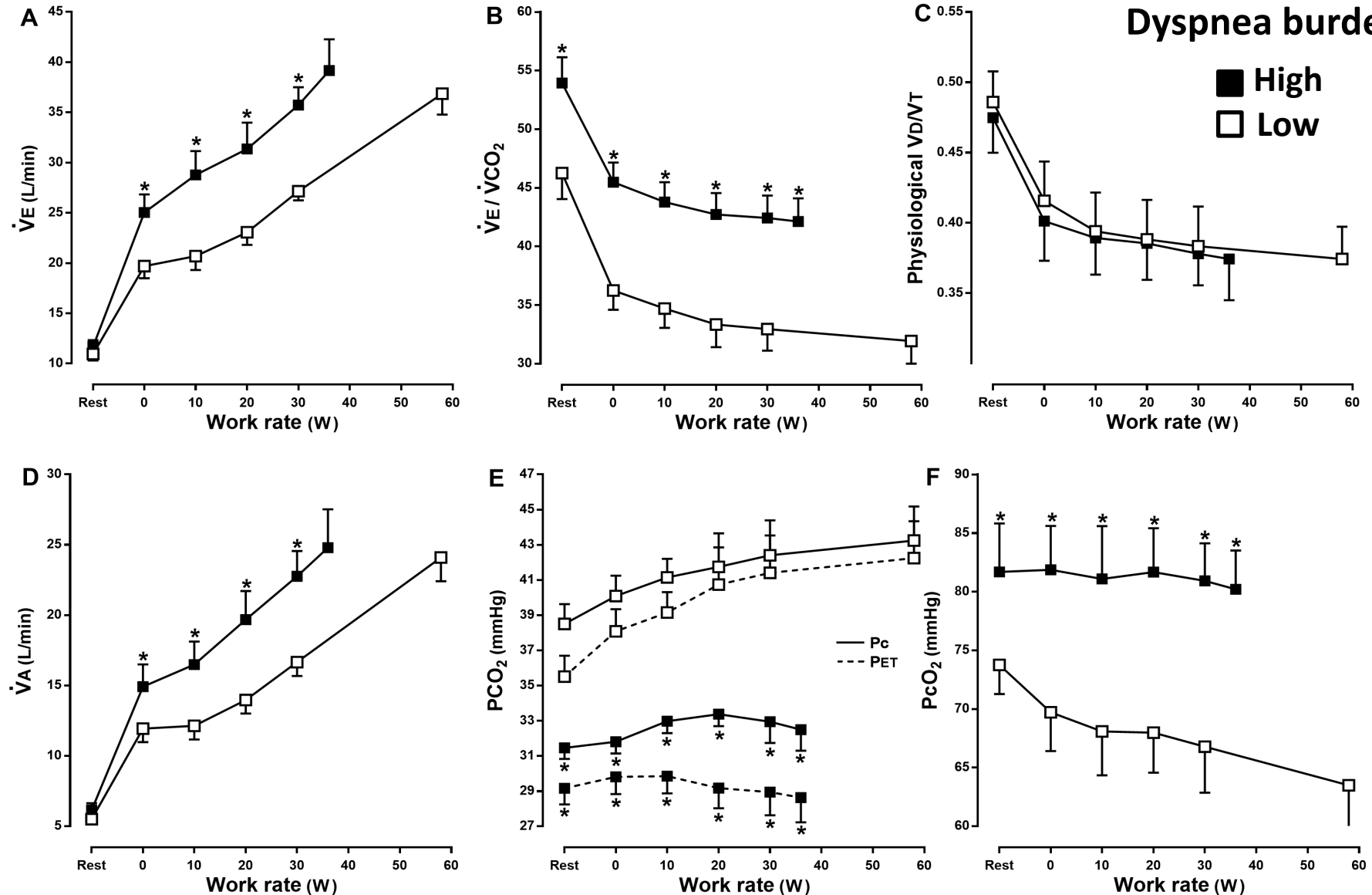
□ Low



Excess Ventilation in Chronic Obstructive Pulmonary Disease-Heart Failure Overlap. Implications for Dyspnea and Exercise Intolerance.

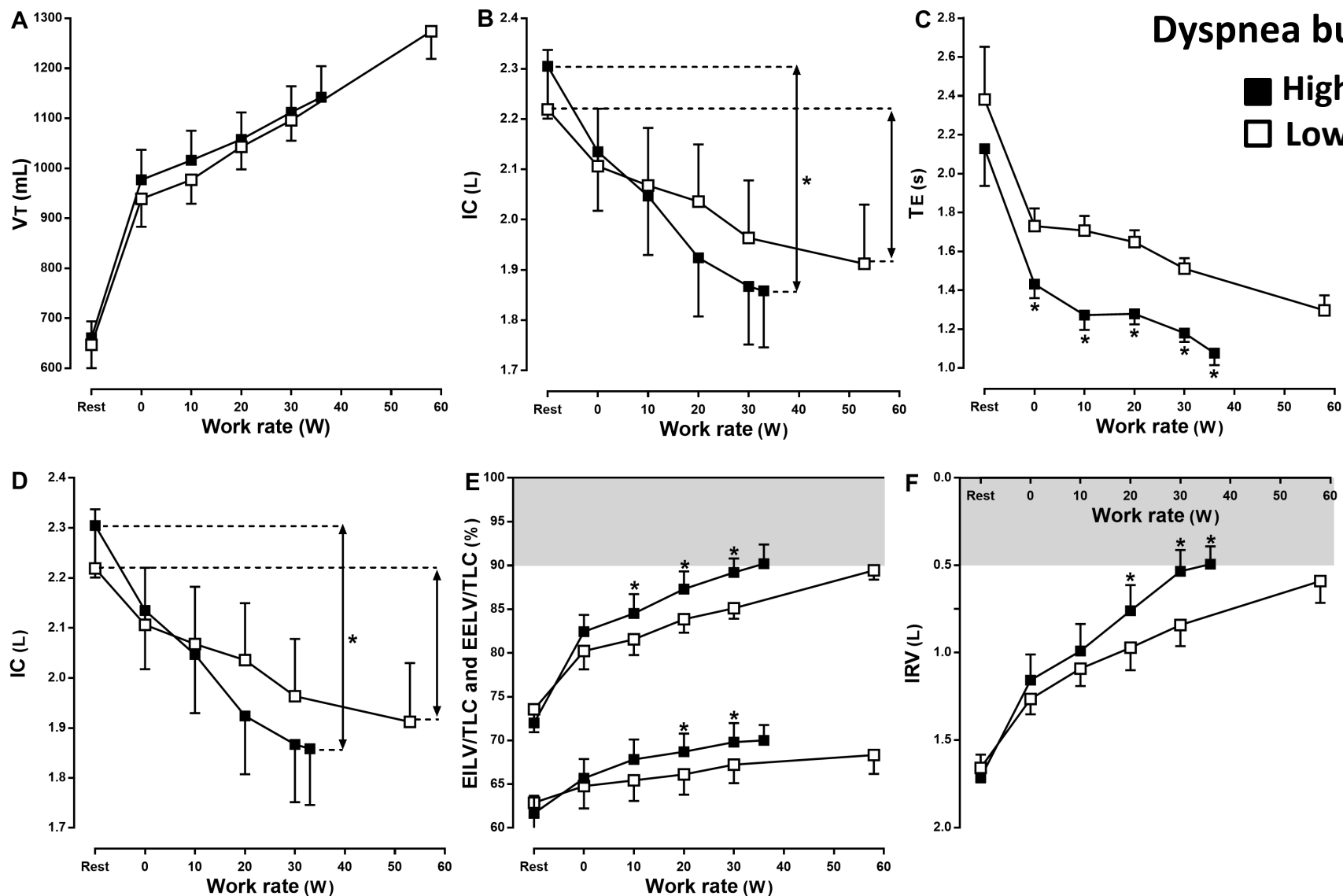
Rocha A, Arbex FF,Neder JA. *Am J Respir Crit Care Med*. 2017 Nov 15;196(10):1264-1274

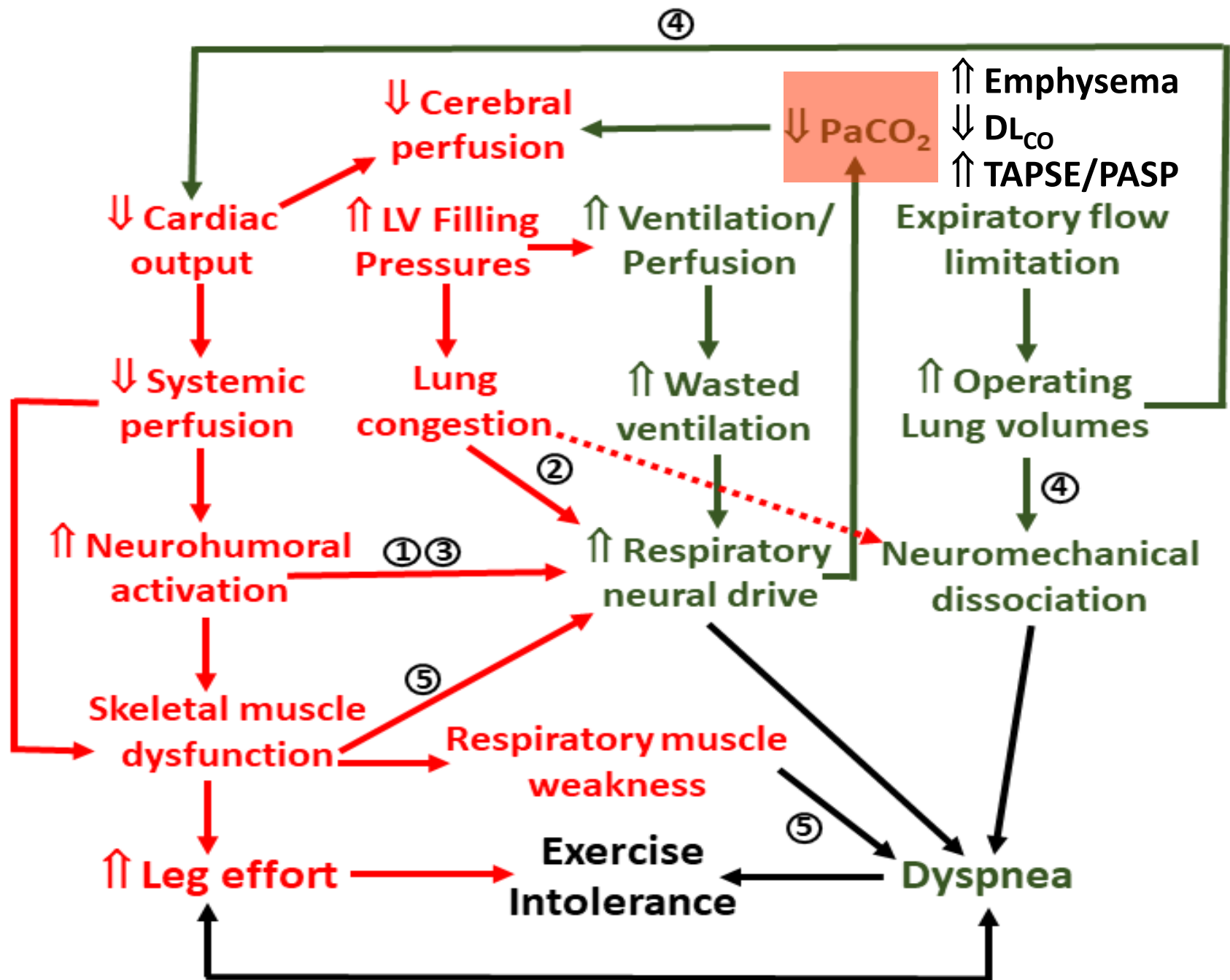
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Respiratory and
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Excess Ventilation in Chronic Obstructive Pulmonary Disease-Heart Failure Overlap. Implications for Dyspnea and Exercise Intolerance.

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An example of direct translation to clinical care:

- Why some patients with stable CHF-COPD are extremely short of breath on any exertion compared to others with same resting impairment in heart and lung function?

Because the ventilate more than required (fundamentally triggered by CHF) to overcome an increased dead space (due to both diseases) thereby lowering their PaCO₂ in a vicious circle that ends up worsening their mechanical problems (fundamentally Induced by COPD)

An example of direct translation to clinical care:

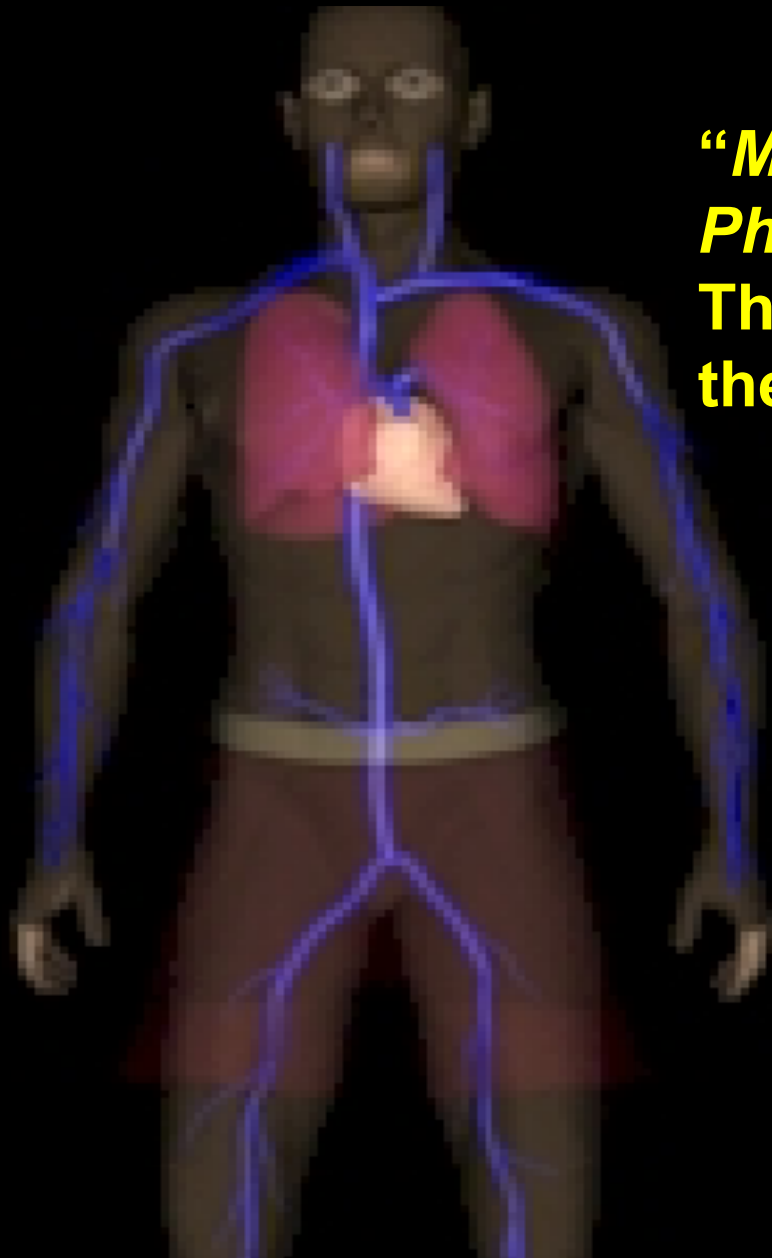
- Why some patients with stable CHF-COPD are extremely short of breath on any exertion compared to others with same resting impairment in heart and lung function?

Because the ventilate more than required (fundamentally triggered by CHF) to overcome an increased dead space (due to both diseases) thereby lowering their PaCO₂ in a vicious circle that ends up worsening their mechanical problems (fundamentally Induced by COPD)

- How does the answer to this question impact on current clinical management ?
 - a) We now know which imaging (emphysema burden), echocardiography (TAPSE/PASP) and lung function variables (PaCO₂, DL_{CO}) we must follow to identify those patients in whom CHF-COPD is more likely to be mechanistically linked to SOBOE
 - b) It is PARAMOUNT to decrease the sources of respiratory neural drive in these patients (either pharmacologically (central hemodynamics and sympathetic drive) and, importantly, non-pharmacologically – exercise training and early rehabilitation (ergorreceptors)

An Innovative Model of Care for Patients with Combined CHF-COPD

- **Multidisciplinary** (cardiology, respirology, geriatric medicine) and **multi-professional** (MDs, specialist nurses, physiotherapists, occupational therapists) health care teams devoted to the management of elderly patients with chronic-degenerative diseases (“**cardiopulmonary teams**”)
- Access to **combined cardiopulmonary rehabilitation** groups and **short-stay units** for optimal management of CHF and/or COPD acute decompensations
- **Web-based decision support tools** adapted to patients with combined cardiopulmonary disease
- **Self-management** kits for both diseases with patient education materials and key prescriptions
- **Training** sub-specialty residents (cardiology, respirology) on management of both diseases



***“Medicine is the science of sickness,
Physiology is the science of life;
Thus, Pathophysiology must be
the scientific basis of Medicine”***

**Claude Bernard
(1813-1878)**