Pulmonary Function Testing

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Normal structure/function

- Branching airway system (23 divisions)
  - 1st 5-6 generations cartilagenous, smooth muscle, mucus glands
  - More distal airways membranous

- 300,000,000 alveoli with surface area that of a tennis court

- Pulmonary capillary blood volume 100-200 ml at any given time

Lungs start off as single tubes and branch off 23 times!

Deeper in the lungs we lose the cartilage and the mucus. Airways become collapsible. They actually collapse in disease states (atelectasis).

Blood thickness in the lung is only one cell thick because we spread the blood out over the large surface area. This "thinness" is important for gaseous exchange.

The "choke point" is the part of the airway that most considerably resists airflow. At low volumes, the choke point (i.e. Point of Maximum Resistance - PMR) is in the small airways. They aren't full of air so they're collapsible. When the lung is full, the distal airways lose their collapsibility and the PMR is in the larger airways.
Normal structure/function

- Rest gas volume (functional residual capacity) is 30 ml/kg, **maximal gas volume (TLC) is 3 x this.**

- Ventilation is 5L/min at rest and goes to over 100L/min at max (**not the rate limiting step in exercise - CV system is**)

- Cardiac output matches ventilation nearly 1:1 \((V/Q = 1)\) up to CV max
Pulmonary Function Testing

- Goals of PFTs
- Normal values - interpretive principles
- Spirometry
- Lung volumes
- Diffusing capacity
Goals of PFTs

- **Characterize disease pathophysiology**
  - Airflow obstruction (COPD, asthma)
  - Lung restriction (pleural, parenchymal disorders)
  - Neuro-muscular dysfunction
  - Vascular disorders

- **Quantitate dysfunction**
  - disability assessment
  - risk evaluation

Categorize patients

Restriction makes the lungs stiffer

How bad is it? Describe impairment compared to normal values. Therapy is based on deviation from the norm. This is also important for analyzing risk. He gave surgery as an example. For any procedure that requires anesthesia, we have to analyze for risk since some drugs cause pulmonary toxicity. We can predict the patients who are susceptible and also follow them post-op to see how they're doing when we do start treatment.

Why PFT:
1. Put people in "physiological buckets" - Categorize according to disease states
2. Determine how ill our patients are and/or how likely they are to become ill (risk)
Normal values - interpretive principles

- PFT values predicted by age, sex, ht
- 95% confidence intervals for normals:
  - 80-120% predicted for spirometry
  - 70-130% predicted for others
- Grading severity:
  - mild if >80% predicted
  - moderate if 50-80% predicted
  - severe if 30-50% predicted
  - very severe if <30%

Question: When you do PFT with an athlete do you see anything special?
Answer: No. Training improves mostly muscles and cardiovascular function. A regular person is in the high 90s for oxygen saturation. Lung function doesn't have that much room for improvement with training.

What affects PFT:
Age: PFT values peak during the late teens
Sex: Different b/w men and women
Height: "Just because of physics..."
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NB: Race and ethnicity probably play a role but we don't know yet. Most of the info we have now is derived from caucasians.
Pulmonary Function Testing

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These 3 tests are 99% of work you do in PFT.
Spirometry

- Oldest clinical test still in use today - John Hutchinson in 1848 still has largest collection of normal values

- Patient inhales to total lung capacity and then completely exhales as rapidly as possible

- American Thoracic Society has comprehensive performance standards
The spirogram

In obstructive disease, FEV1 is reduced but FVC is usually normal. The FEV1/FVC ratio is low. FEV1 is low because obstruction is worse during expiration. This is the case because of the external pressure from our chest wall (During inspiration, we're expanding the chest and reducing the pressure). This is why we take spirometry readings during expiration. This curve is representative. Patients eventually get air out (parenchyma is normal) but it takes forever.

In restrictive disease, FVC is lower but the ratio of FEV1/FVC remains intact at >70%. It often increases.

Start here with deep breath (full lung)

Breath as rapidly as possible. Should go for 6 seconds. If the patient doesn't go for at least 6 seconds, we might need to repeat.

The ratio FEV1/FVC is always an important value to have.

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Most mammals can get 70% of vital capacity out in the first second. Irrespective of height, weight, etc.
## Patterns of pathophysiology

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* during exacerbations/methacholine

FEV1/FVC changes are episodic in asthma

For vascular disease, neither FEV1 nor FVC changes
The spirogram

- After medications
  - bronchodilators
  - methacholine

- Plotted as a flow-volume curve ("loop")

Information from the spirogram can be manipulated by taking medications and/or plotting a flow/volume curve.

Used in patients with normal FEV1/FVC ratio in whom we suspect asthma. This drug attempts to initiate an asthma attack.
The spirogram - post medications

- **Post bronchodilator (4 puffs beta agonist)**
  - increase by 15% considered significant
  - ?does it change clinical decisions

- **Post methacholine (up to 8mg/ml)**
  - diagnostic of asthma when FEV1 falls >20%
  - Dose response defines severity

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Controversy about how much this should affect decision making. Should you not give someone albuterol for emergency relief because they aren't improving by 15%?

Patient comes in with history that fits asthma... but their spirogram is normal. You can provoke an asthma attack using methacoline!

Interestingly, methacholine only works in people with asthma. It has no effect in people who don't have asthma.

**Question:** Why don't normal people feel the responses?

**Answer:** If you could answer it I'd give you a fellowship in pulmonology (I.e. We don't know)
Methacholine challenge

This happens for most of us (Hardly any effect)

This is what happens for people who have asthma that doesn't manifest itself with tests ("Closet asthmatics"). FEV1 reduces with increasing doses of methcoline
The spirogram

- After medications
  - bronchodilators
  - methacholine

- Plotted as a flow-volume curve ("loop")
The spirogram - converting to a flow volume curve

Normal Curve: Flow Volume against Time

Converted Curve: Flow Rate against Volume

Key:
A: Normal
B: Obstructive Lung Disease
C: Restrictive Lung Disease

The Flow rate vs. Volume graph helps us distinguish between small and large airway abnormalities. It's particularly useful at low lung volumes where small airways are PMR. Small airway disease is often a precursor to large airway disease and loss of lung volume. Finding this early allows us to catch the process before total compromise of lung function.

Manipulate the information from spirometry measurements to get flow rate data.
Flow-volume curve

- Allows better assessment of airway characteristics at low lung volumes
  - As lung empties, “choke point” for flow moves distally from large cartilagenous airways to small membranous airways
  - These small airways may be earliest site for airway diseases
Flow-volume curve

- Can be assessed:
  - visually - appears “concave” downward with airway disease and often below tidal loop
  - FEF 25-75 is mean flow during mid exhalation

- In setting of normal FEV1/FVC, abnl flow volume curve suggests early airway disease

The flow rate vs volume curves give very "noisy" numbers. The range of normal is very wide and it's difficult to interpret. He doesn't use it.

I.e. in early stages of disease, FEV1/FVC might be normal. We can catch the small airway disease early using the flow rate vs volume curves.
Graph for sequence of deep breath followed by rapid exhalation. This person has terrible lung function with a low initial flow and very dramatic disappearance of flow.

Breathing at Rest (Tidally)

When the patient is breathing tidally, there's a certain flow rate. When the patient takes the deep breath and breathes forcefully, flow actually ends up being slower than what it was during tidal breathing. This is very abnormal. Characteristic of collapsing airways. You push on them and they slam shut.
Upper Airway Evaluation

For each of these flow volume loops, the part above the x axis represents exhalation. The part below represents inhalation.

Exhalation is near normal but inhalation is impaired.

Variable Obstruction: Exhalation is impaired but inhalation is normal (during exhalation, pressure collapses airways)

Fixed obstruction: Both exhalation and inhalation are impaired (E.g. Upper airway narrowing at the anastomotic site of lung transplant)

Easy point on your pulmonary boards: Upper airway obstruction (laryngeal obstruction, goiter) constricts the trachea and flow volume loop becomes almost a square. The third one is most common. Upper airway narrowing is now very common because of lung transplants.
Pulmonary Function Testing

- Goals of PFTs
- Normal values - interpretive principles
- Spirometry
- Lung volumes
- Diffusing capacity

**Question:** Where's the midpoint for switching the chokepoint from large to small airways?

**Answer:** It's right around the midpoint of inspiration.
The spirogram measures the maximal amount of gas a subject can voluntarily move.

Lung volume testing is primarily aimed at measuring the remaining gas in the lung after full exhalation (residual volume).

Much more complicated than the spirogram, costs $40,000! Not performed as often.
Lung volumes

Lung gas volume at the “rest point” or functional residual capacity is measured by one of several techniques:

- plethysmography
- inert gas dilution
- nitrogen washout

Residual volume is then calculated by having patients fully exhale and subtracting this volume.
Body Plethysmography

Measurement of Static Lung Volumes
(Vtg: Volume of Thoracic Gas)
Static Lung Volumes

Clinical Application Notes

Normal N2 Wash-out Pattern

Flow Volume Loop Pulmonary Function Testing

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Patterns of pathophysiology

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With obstruction, air can't leave well so residual volume increases (remember expiration is the worse part of obstructive disease as the increased pressure collapses airways)

With restriction, you don't get enough air into the lungs. RV decreases

With neuromuscular pulmonopathy, you just can't push out enough air. RV increases.

No change in RV with vascular pulmonopathy
Pulmonary Function Testing

- Goals of PFTs
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- Diffusing capacity
Diffusing capacity

- CO uptake dependent on:
  - Vc: capillary blood volume (incl Hb)
  - Dm: alv-cap membrane properties

- CO uptake measured by
  - inhaling small concentrations CO
  - holding breath 10 sec
  - measuring exhaled CO
Pathologic changes that affect Dm, Vc and DLCO

A. Normal
B. Emphysema
C. IPF
D. Lobectomy
E. Vasculitis
F. CHF

- Loss of diffusion
- Fibrosis
- Destroy vessels
- Diffusion actually goes up in acute situation with left ventricular failure because capillary beds are engorged. Long standing failure however, damages vessels.

3 ways to increase diffusion without touching you:
- Increase HR (exercise)
- Lay you flat. Gravity affects much less
- Molar manoeuvre. Close glottis and inspire (opposite of valsalva)
The single breath DLCO

\[ DLCO = \ln \left( \frac{CO_i}{Cot} \right) \times VA \times \frac{1}{t} \]
Real time analysis allows adjustments to $V_d$ and $V_s$.
Measuring DLCO (TLCO)
What adjustments are needed for proper interpretation?

Factors that can impact the measurement:

- Hemoglobin
  - Men:
    - DLCOpredicted for Hb = DLCOpredicted $\times \frac{1.7 \cdot Hb}{(10.22 + Hb)}$
  - Women:
    - DLCOpredicted for Hb = DLCOpredicted $\times \frac{1.7 \cdot Hb}{(9.38 + Hb)}$

- PiO2
- COHb
- Ventilation distribution
- Lung volume

The factors on this page can skew your measurements for DLCO.

Equations to appropriately adjust DLCO to match standards (Didn't emphasize these equations so I wouldn't cram them...)

Most important
Measuring DLCO (TLCO)
What adjustments are needed for proper interpretation?

- Factors that can impact the measurement:
  - Hemoglobin
  - PiO2
  - COHb
  - Ventilation distribution
  - Lung volume

DLCO predicted for altitude = DLCO predicted / (1.0 + 0.0035[PAO2 - 120])

Patient with a lot of O2 will outcompete CO

More correction equations
Measuring DLCO (TLCO)
What adjustments are needed for proper interpretation?

- Factors that can impact the measurement:
  - Hemoglobin
  - PiO2
  - COHb

\[ \text{DLCO}_{\text{predicted for COHb}} = \text{DLCO}_{\text{predicted}} \times (102\% - \text{COHb}\%) \]

- Ventilation distribution
- Lung volume

Cigarettes have CO which affects the measurement since it's CO based test

CO correction equation
Measuring DLCO (TLCO)

What adjustments are needed for proper interpretation?

- Factors that can impact the measurement:
  - Hemoglobin
  - PiO2
  - COHb
  - Ventilation distribution
  - Lung volume

Top of the lung has a bit of dead space (more ventilation than perfusion). Bottom of the lung is a bit of a shunt (more perfusion than diffusion). This distribution varies in different individuals. Can skew results in one direction or the other.
Effects of poorly ventilated regions

- DLCO measures CO uptake from regions into which it is inhaled.
- In severe OAD, many regions cannot get measurable CO into them during a single breath and thus global DLCO appears reduced.
- Suspect this when the tracer gas dilution $V_a$ is very low - if the tracer gas cannot distribute, neither can the CO.
Measuring DLCO (TLCO)
What adjustments are needed for proper interpretation?

- Factors that can impact the measurement:
  - Hemoglobin
  - PiO2
  - COHb
  - Ventilation distribution
  - Lung volume

Lung volume will affect rate of gas diffusion
Lung volume effects - low Vi

- Less than maximal Vi
  - lower Vc and Dm (dark)
- Lobectomy/pneumonectomy
  - lower Dm, VC recruited (light)
- Simple DI/Va does NOT “correct” (ie not 1:1)

With a lobectomy, you'd suspect that there's decreased diffusion because of the decreased volume. This is true but not to the extent that you would imagine. The perfusion in the remaining lobes increases to provide compensation for the lost diffusion capacity.
If DL reduced proportionally to VA, suggests equal loss of lung and vascular tissue – not “normal”

If DL reduced less than VA (high DL/VA ratio), suggests poor effort, chest wall restriction, weakness

If DL reduced more than VA (low DL/VA), suggests vascular disease
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**Measure**: Measures gaseous exchange as opposed to mechanical behavior

**Differentiating test for emphysema**: If there's a problem with diffusion (in addition to the rest of the FEV1, FVC, FV profile), problem is emphysema since this problem reduces absorptive ability. If there's no effect on diffusion then the bronchi are affected.

**Question**: Seeing as RV test is least important but most expensive, does anyone do it?
**Answer**: 12,000 tests at duke - all have spiro, 50% have DLCO, 25% have RV

**You see this as an isolated finding (only DLCO is abnormal)** and you know its a vascular issue
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