# 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 airwars.

# Normal structure/function

Rest point of the lung = Volume of gas in the lung when you take the lungs out of a person (i.e. eliminate the contribution from chest muscles)

- 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)

Lungs will not make you stop exercising. The heart will. Doesn't make much sense to "train your lungs"

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

Altogether the lungs match ventilation (air flow) and perfusion (blood flow) very well. The top of the lung has more ventilation than perfusion and the bottom has more perfusion than ventilation.... but on average matching is almost perfect.

# Pulmonary Function Testing

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

Important for understanding the value of PFT

# 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%

I.e Normal is where 95% of the population exists. Note that 5% of normal people will be "abnormal." Just the way it works.

Usually, 95% confidence intervals are sufficiently represented in this rage of 80% to 120% for spirometry

These grading rules are arbitrary (convenient mathematically)

**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

- Goals of PFTs
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- Lung volumes
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These 3 tests are 99% of work you do in PFT.

# Spirometry

This guy put a pair of buckets in each other, had people blow into tubes (after taking a deep breath) and decided on vital capacity

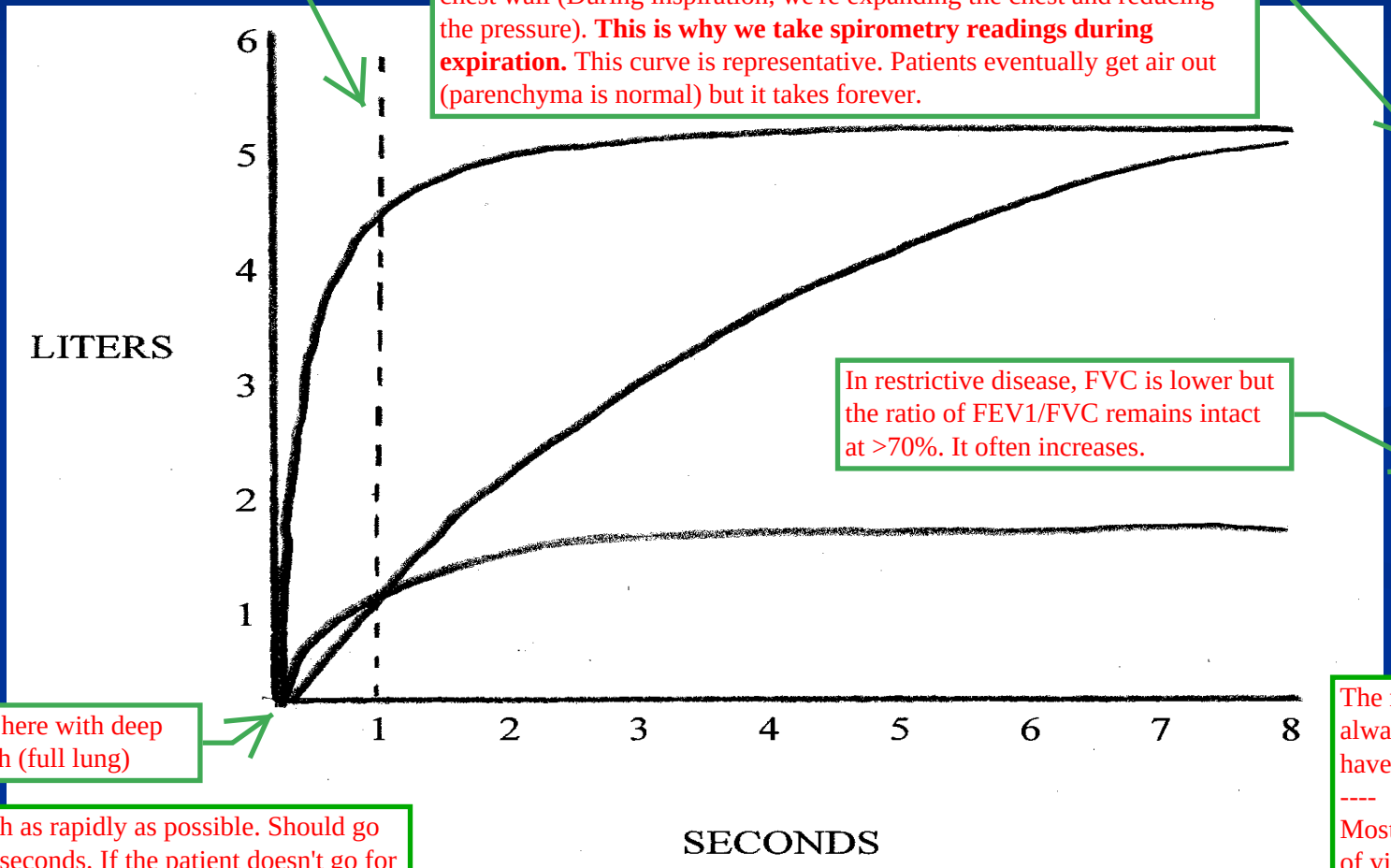
- Oldest clinical test still in use today - John Hutchinson in 1848 still has largest collection of normal values
- Patient inhales to total lung capacity and then completely exhales as rapidly as possible
- American Thoracic Society has comprehensive performance standards

# The spirogram

... don't use buckets anymore. Now use little flow censors.

Volume of curve at this line is the volume exhaled after 1s (FEV1)

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.




Normal
Obstruct

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

Restrict

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

| CATEGORY | FEV1/FVC | FVC | RV | DLCO |
|---|---|---|---|---|
| **Obstructed** | | | | |
| Asthma | nl/dec* | nl | | |
| Bronchitis | dec | nl | | |
| Emphysema | dec | nl | | |
| **Restricted** | nl | dec | | |
| **Neuromuscular** | dec | dec | | |
| **Vascular** | nl | nl | | |

FEV1/FVC changes are episodic in asthma

For vascular disease, neither FEV1 nor FVC changes

* during exacerbations/methacholine

# The spirogram

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

- After medications
  - bronchodilators
  - methacholine

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

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

# The spirogram - post medications

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

With albuterol for example

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%?

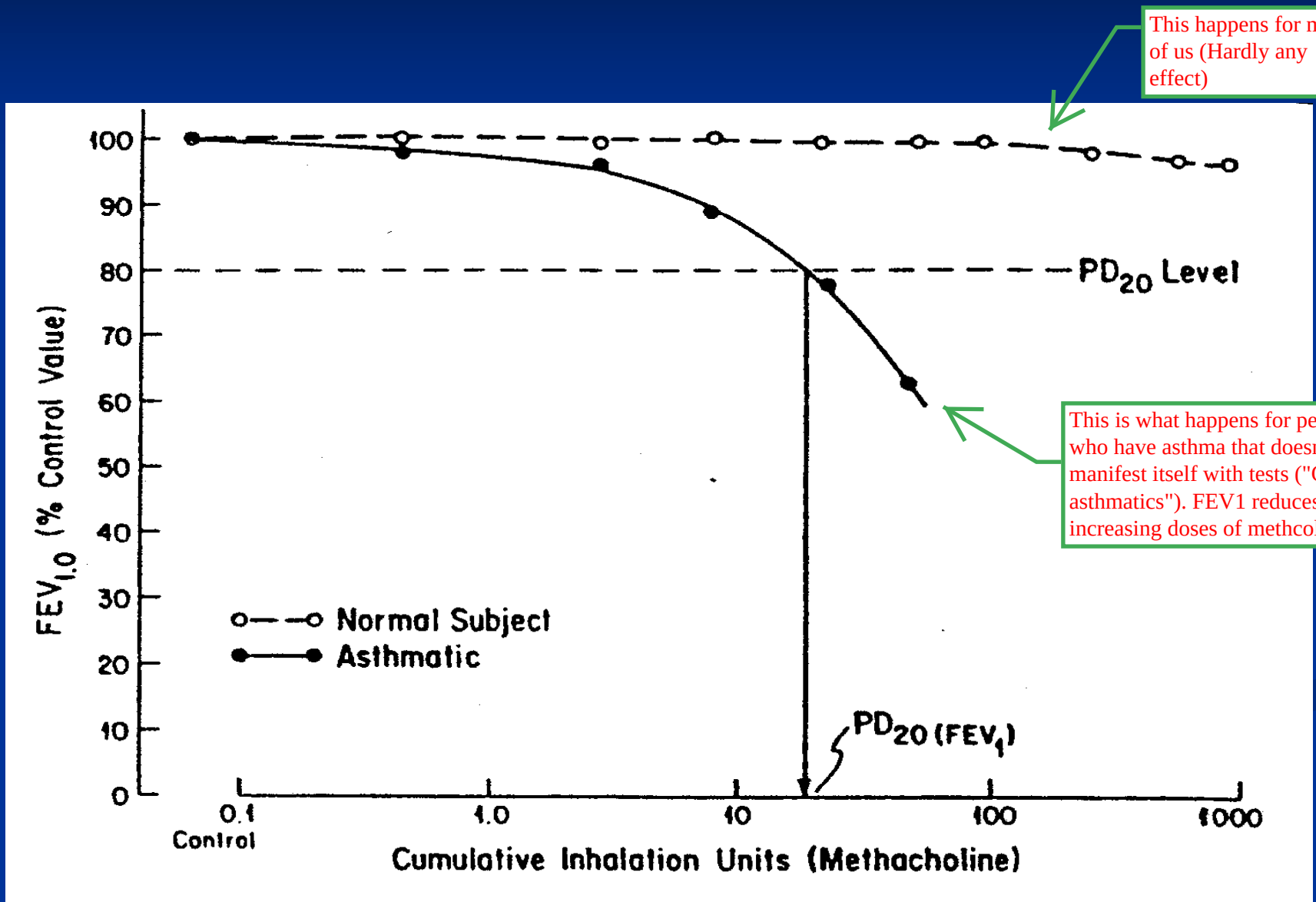
- Post methacholine (up to 8mg/ml)
  - diagnostic of asthma when FEV1 falls >20%
  - Dose response defines severity

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



# The spirogram

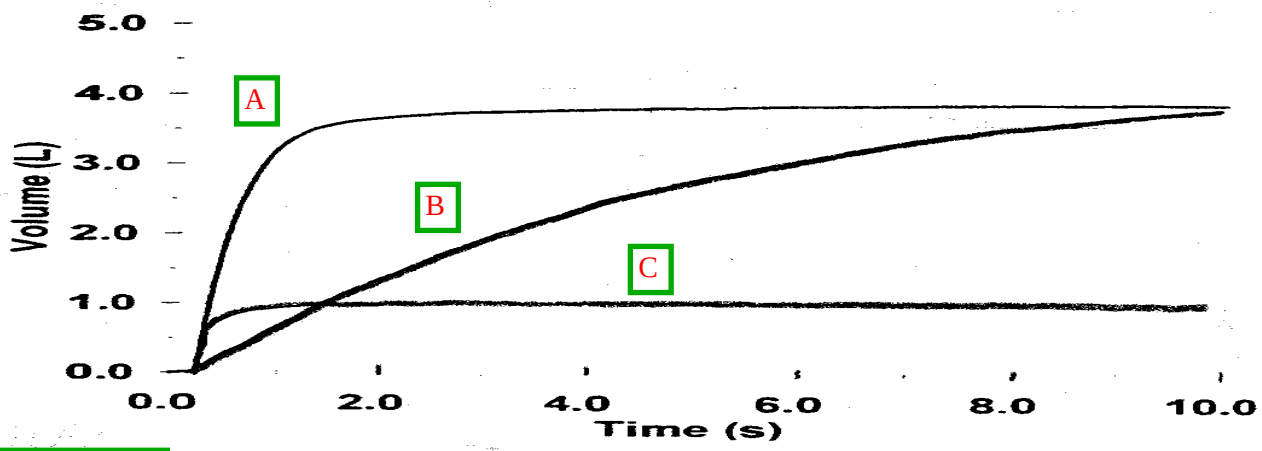
- After medications
  - bronchodilators
  - methacholine
- Plotted as a flow-volume curve ("loop")

Plot graph as flow rate against volume (compare volume against time curve from normal spirometry)

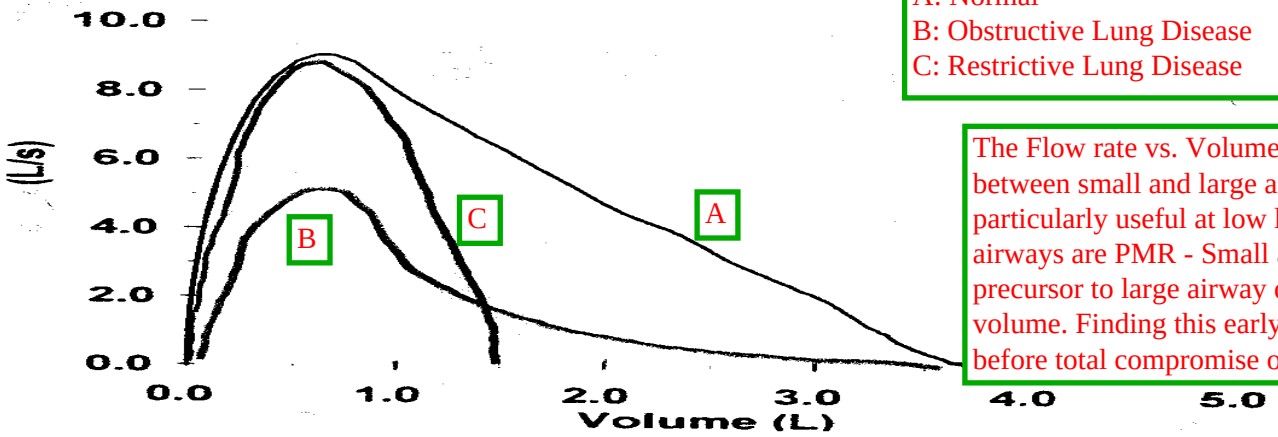
# The spirogram - converting to a flow volume curve

Manipulate the information from spirometry measurements to get flow rate data

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 catch the process before total compromise of lung function.

# Flow-volume curve

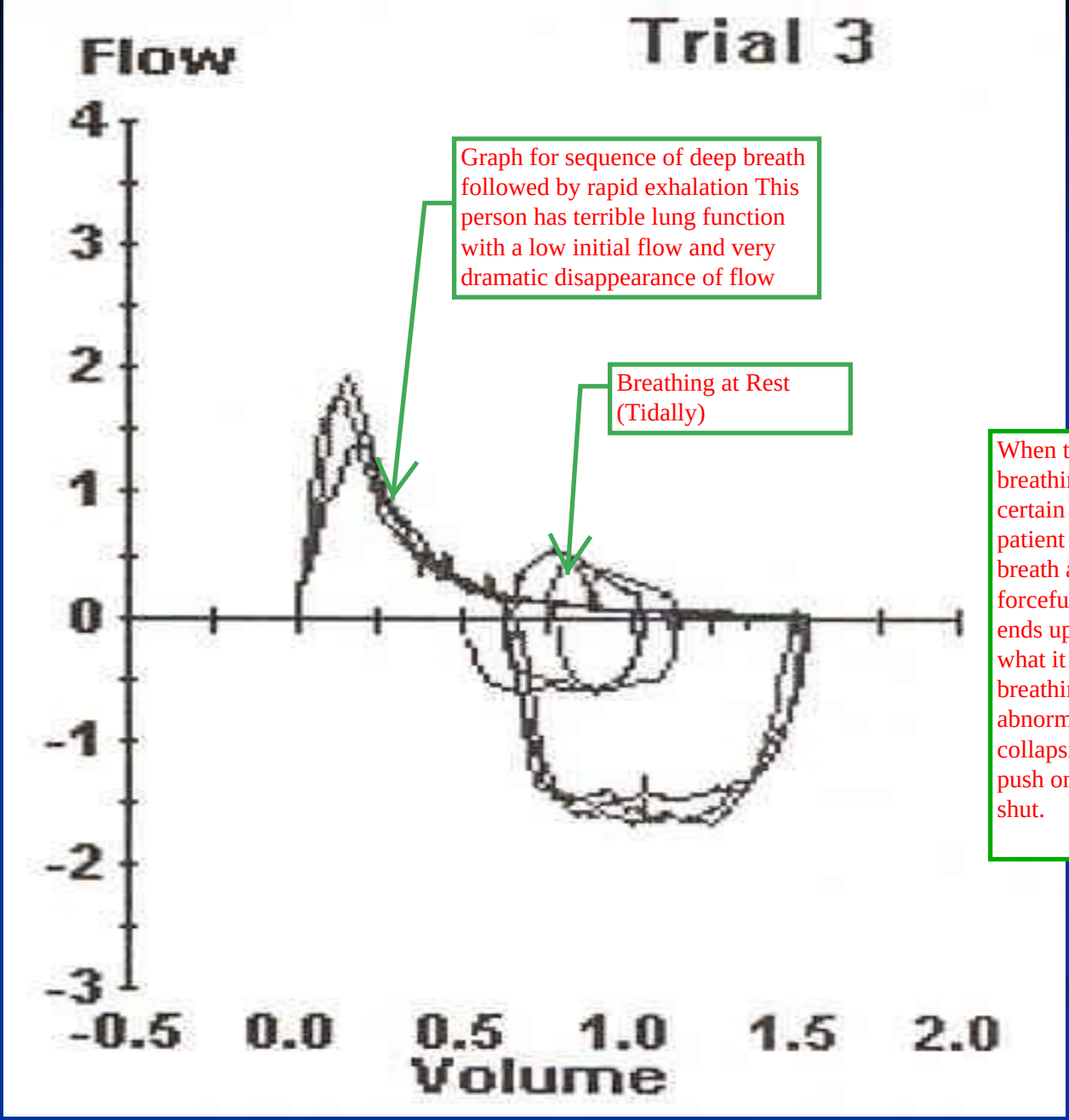
- Allows better assessment of airway characteristics at low lung volumes
  - as lung empties, "choke point" for flow moves distally from large cartilagenous airways to small membranous airways
  - these small airways may be earliest site for airway diseases

# 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

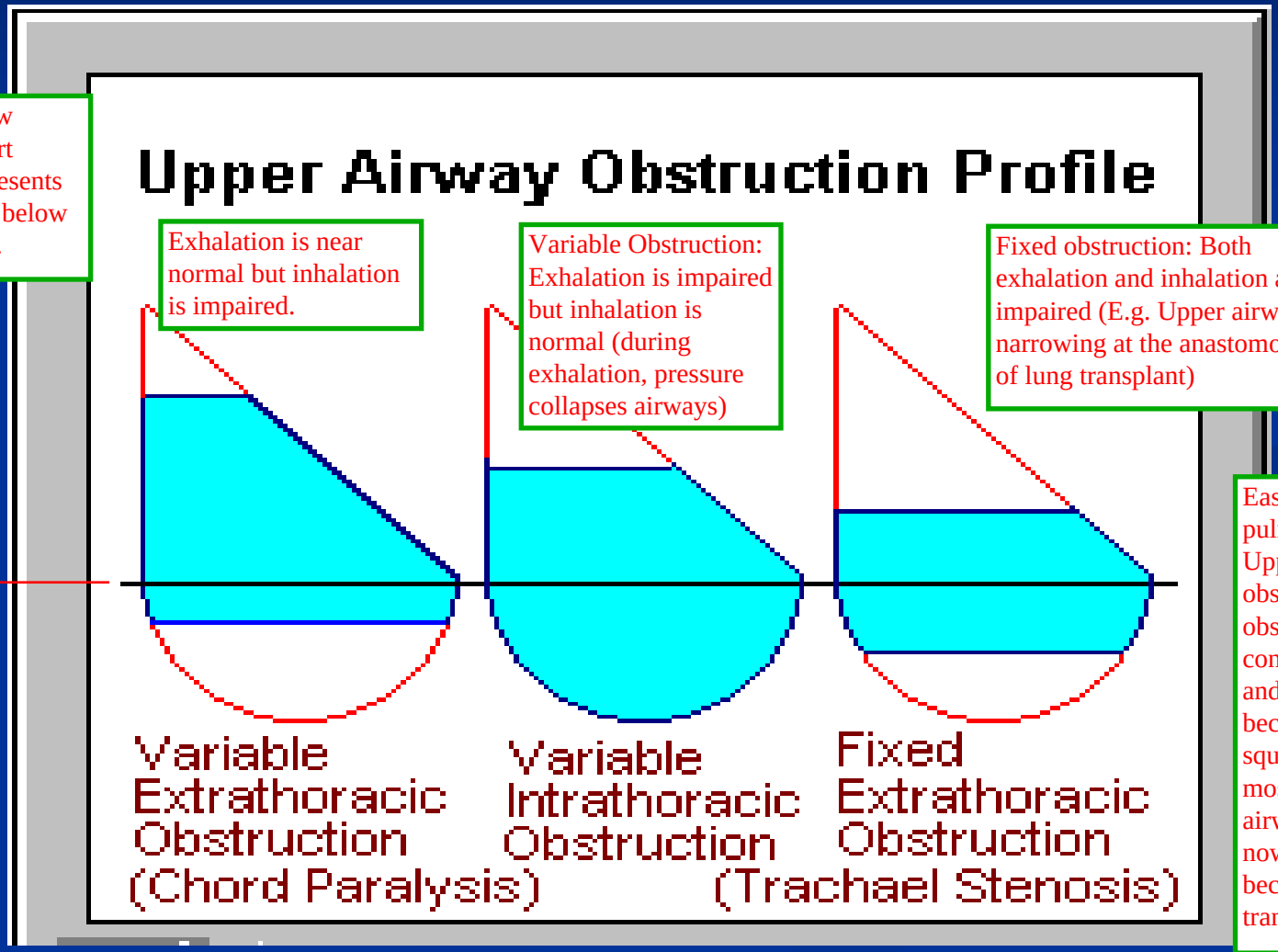
The flow rate vs volume curves give very "noisy" numbers. The range of normal is very wide and it's difficult to interpret. He doesn't use it.

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.


Flow
Trial 3
4
3
2
1
0
-1
-2
-3
-0.5
0.0
0.5
1.0
1.5
2.0
Volume
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.




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
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- Lung volumes
- Diffusing capacity

Most common, cheap but requires patient cooperation

Least important. Most expensive. Doesn't require too much patient cooperation

**Question:** Where's the midpoint for switching the chokepoint from large to small airways?
**Answer:** It's right around the midpoint of inspiration.

# Lung volumes

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

- 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)

Here, replace all the gas in the lung with inert gas, often helium (along with a known concentration of O2 so people keep breathing). This is done by diluting the lung gas with the inert gas over a period of time (4 - 7 minutes). To get TLC, measure the amount of inert gas that comes out when patient starts to breathe normal air (accounting for O2 part of "inert gas")

# Lung volumes

"We don't have time to go through this"

- 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

Put patient in a box. Patient exhales through a mouth piece. Mouth piece is closed to "seal" the box. Patient then inhales. Since it's a closed system, decrease in lung pressure with inhalation leads to changes in box pressure. Boyle's law is used to find volumes.

Patient exhales entire vital capacity (leaving only residual volume) and then inhales nitrogen gas. This nitrogen mixes with nitrogen in residual volume. Since most of the air in residual volume is nitrogen, total volume of nitrogen gives TLC (make assumptions about CO2, O2, etc left in the residual gas)

Knowing that you started with 80% you can measure volume.

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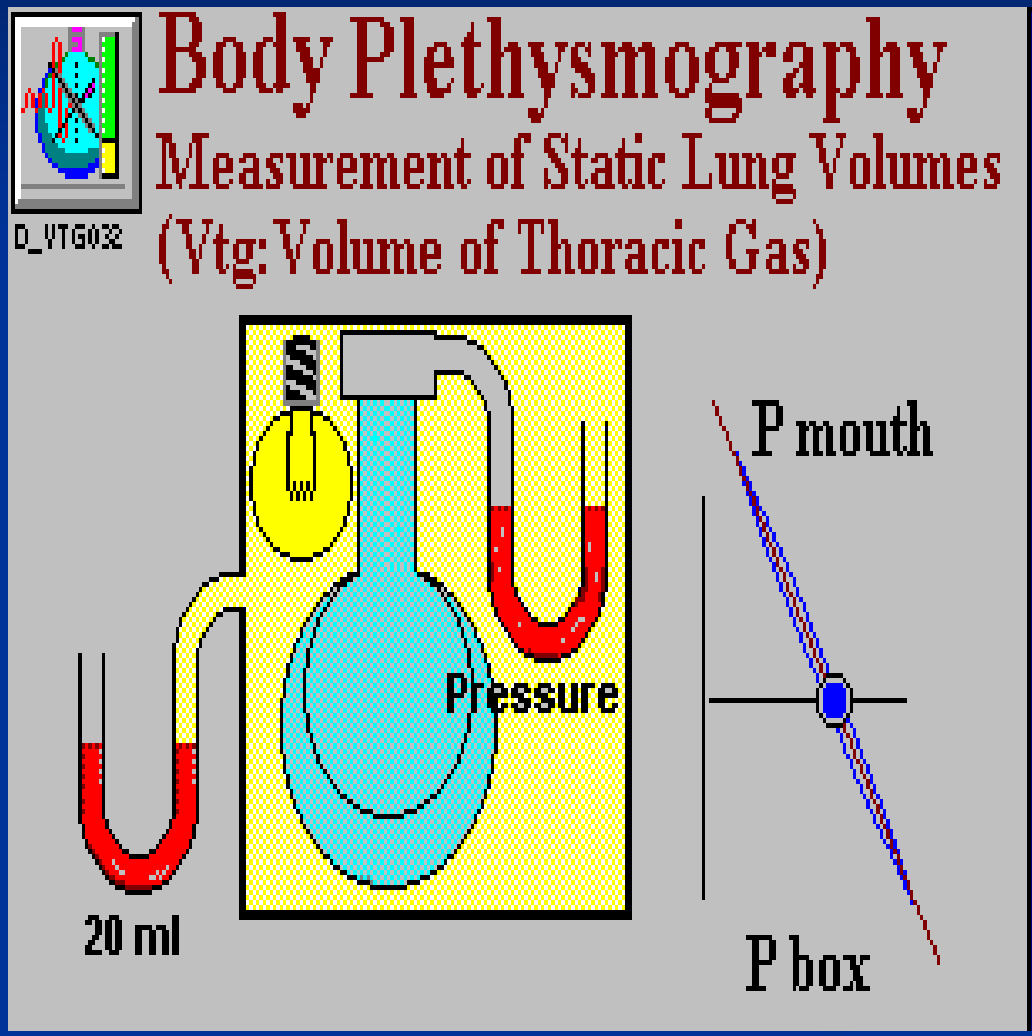
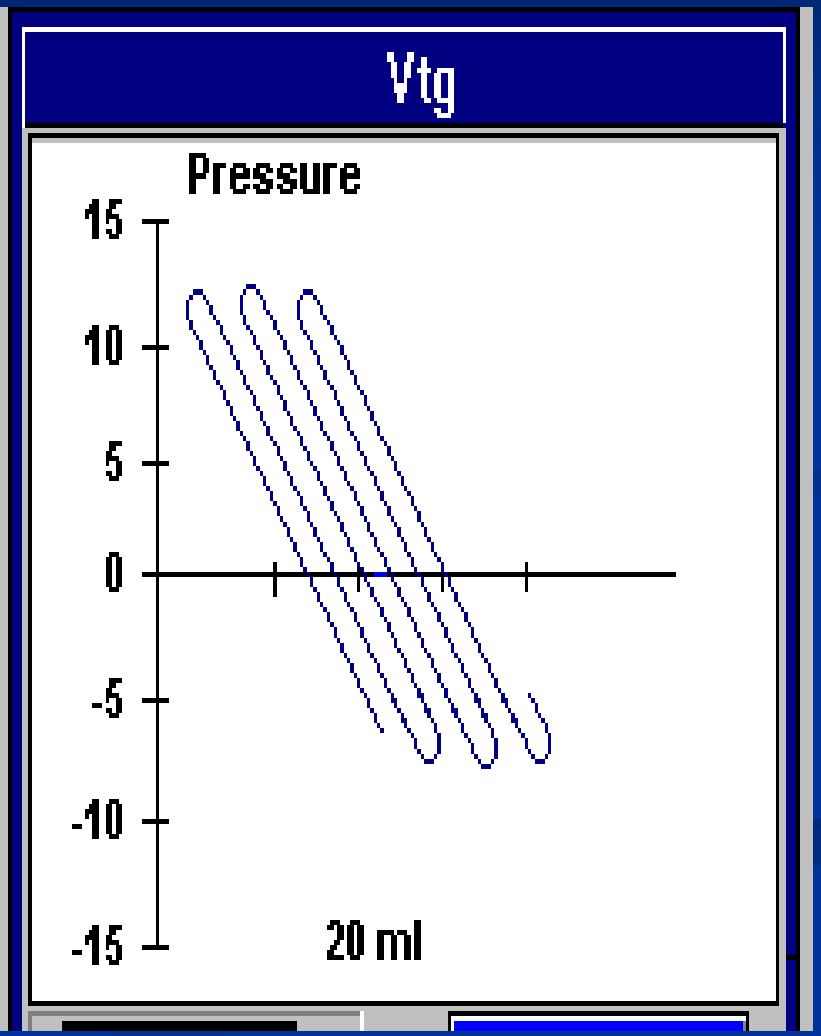
# Body Plethysmography



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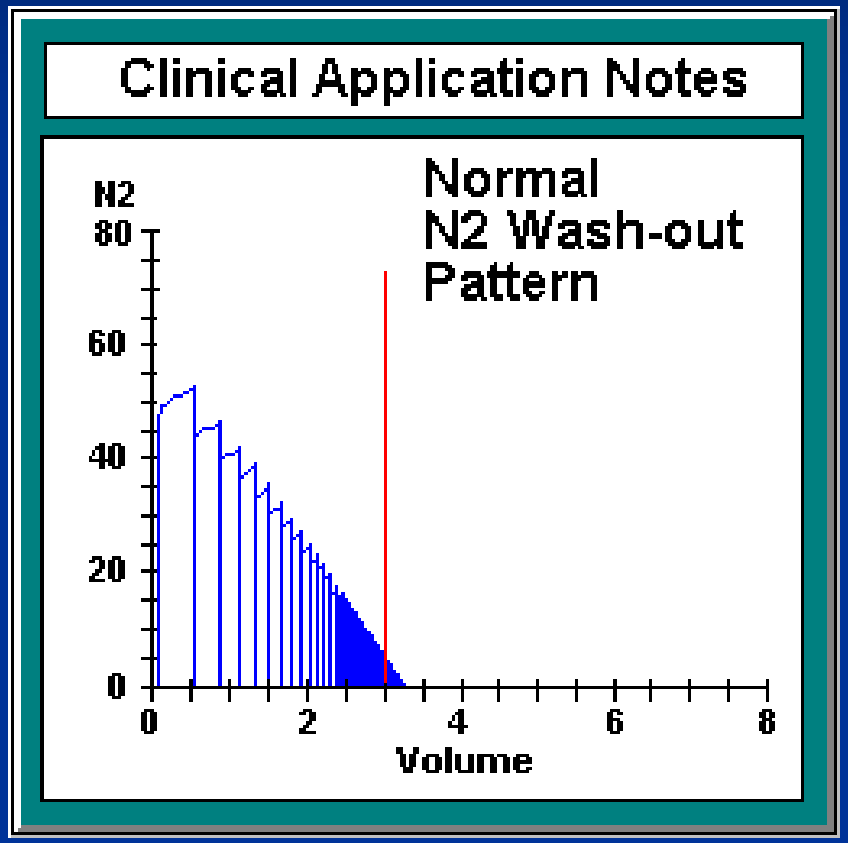
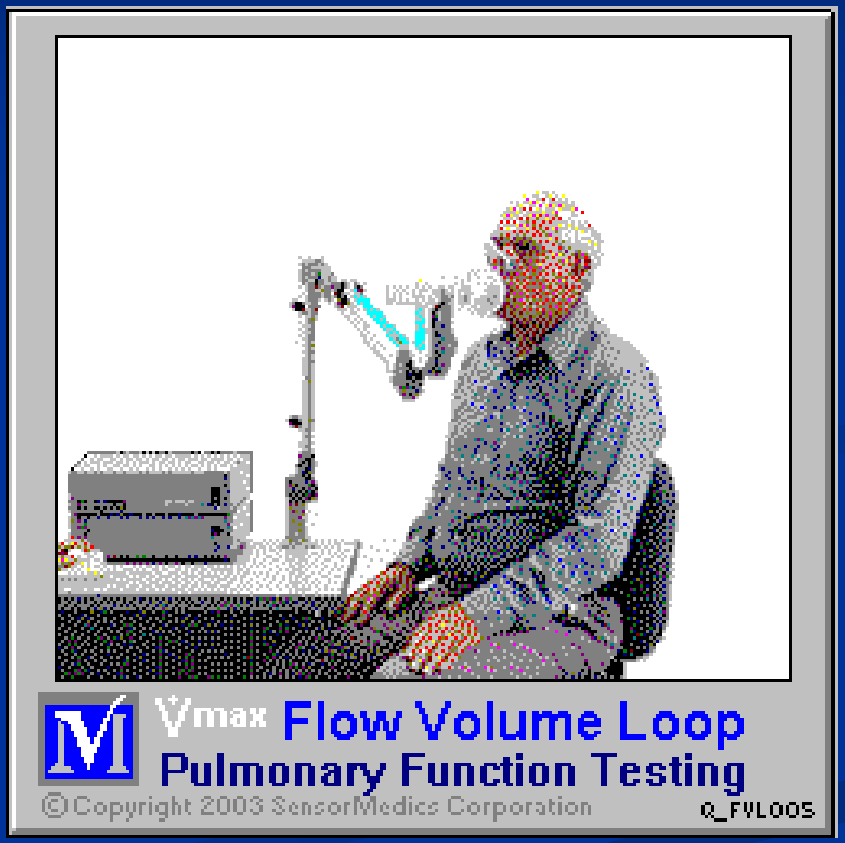
# Static Lung Volumes



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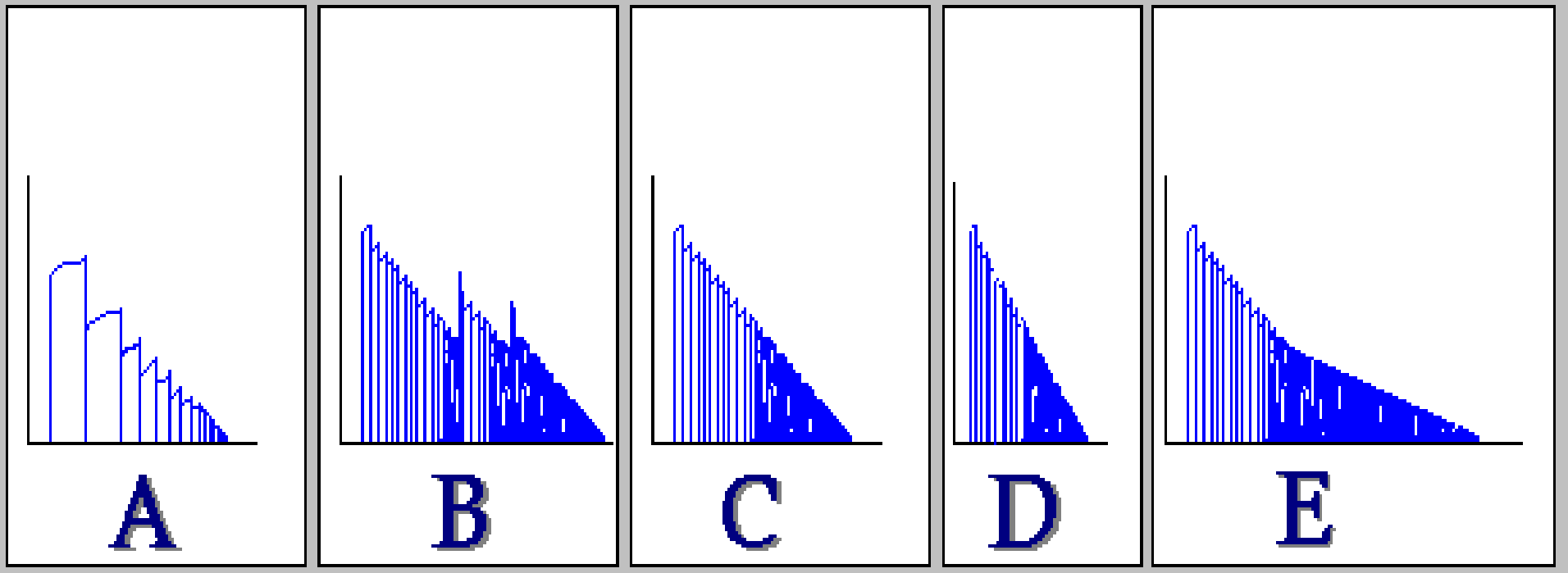




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# Patterns of pathophysiology

| CATEGORY | FEV1/FVC | FVC | RV | DLCO |
|---|---|---|---|---|
| **Obstructed** | | | | |
| Asthma | nl/dec* | nl | inc* | |
| Bronchitis | dec | nl | inc | |
| Emphysema | dec | nl | inc | |
| **Restricted** | nl | dec | dec | |
| **Neuromuscular** | dec | dec | inc | |
| **Vascular** | nl | nl | nl | |

* during exacerbations/methacholine

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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- Lung volumes
- 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

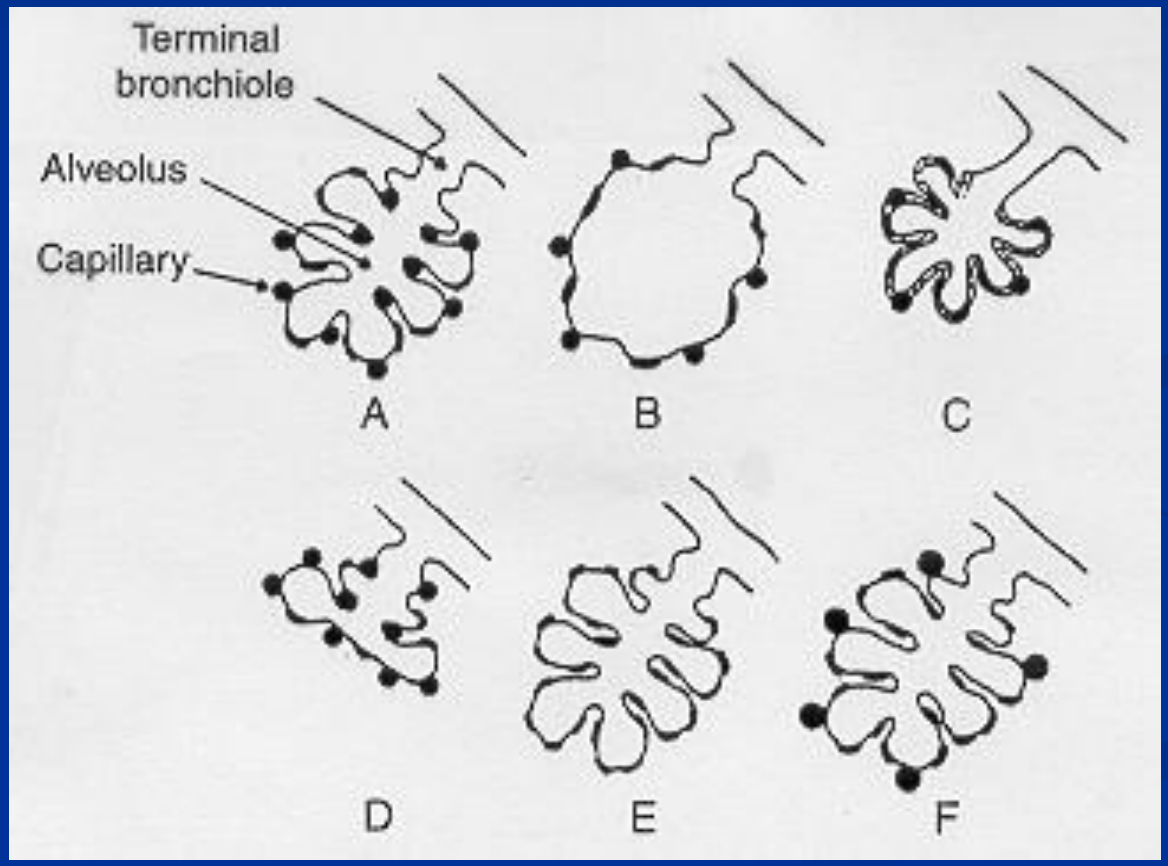
Measure of the amount of blood reaching alveolar capillary membranes

How much gas diffuses between the capillaries and the alveoli

Go through these steps to measure how well gas is diffusing into the capillaries (Compare with standards)

# Pathologic changes that affect Dm, Vc and DLCO

Think of diffusion test as a measure the amount of blood that is there to pick up CO




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 manoeuver. Close glottis and inspire (opposite of valsalvar)

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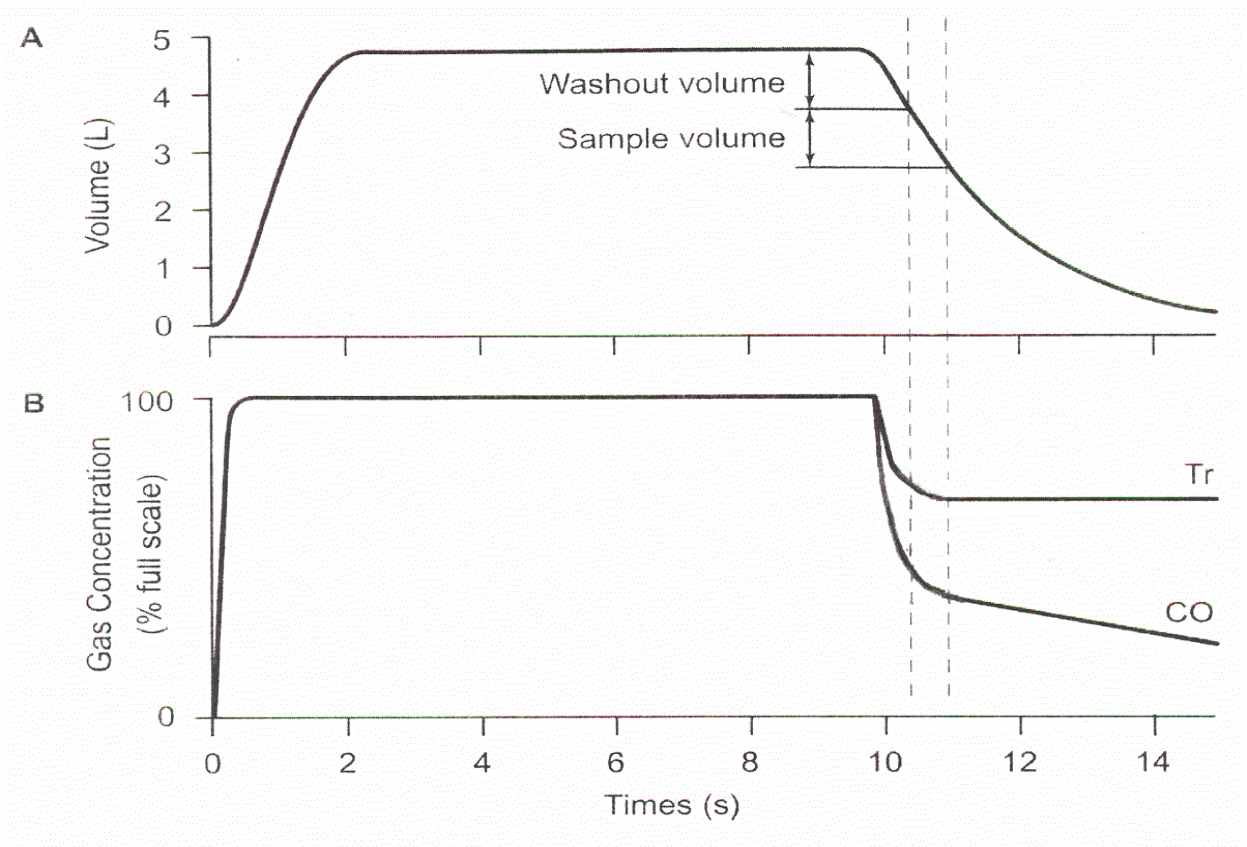
# The single breath DLCO




DLCO = ln (CO/i/Cot) x VA x 1/t

# Real time analysis allows adjustments to Vd and Vs

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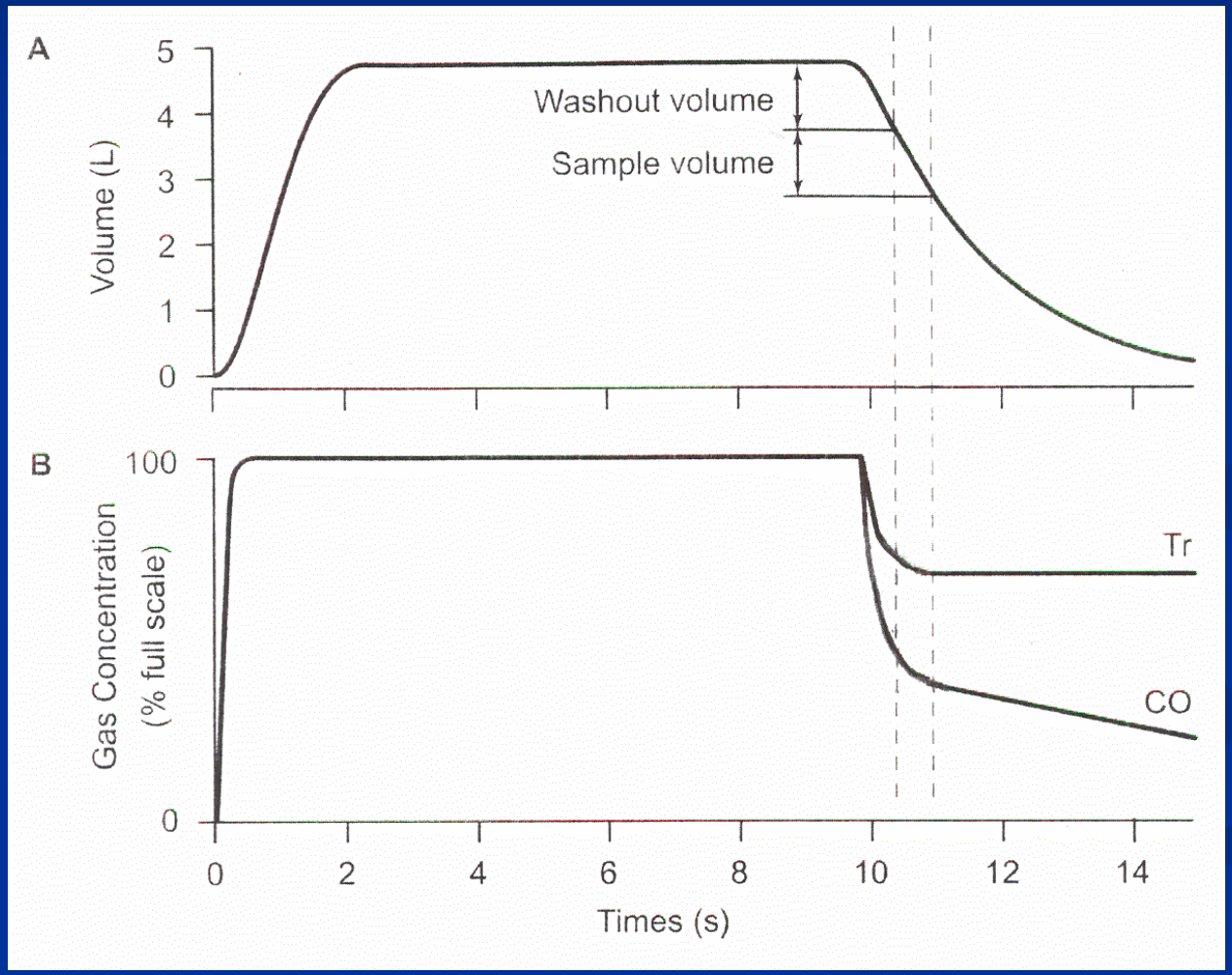

# Measuring DLCO (TLCO)

## What adjustments are needed for proper interpretation?

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

- Factors that can impact the measurement:
  - Hemoglobin

Most important

- Men:
- DLCOpredicted for Hb = DLCOpredicted 1.7 Hb / (10.22 + Hb)
- Women:
- DLCOpredicted for Hb = DLCOpredicted 1.7 Hb / (9.38 + Hb)

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

  - PiO2
  - COHb
  - Ventilation distribution
  - Lung volume

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

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

Patient with a lot of O2 will outcompete CO

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

More correction equations

  - COHb
  - Ventilation distribution
  - Lung volume

# Measuring DLCO (TLCO)

## What adjustments are needed for proper interpretation?

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

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

DLCOpredicted for COHb = DLCOpredicted (102% - COHb%)

CO correction equation

  - Ventilation distribution
  - Lung volume

# 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

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# Effects of poorly ventilated regions

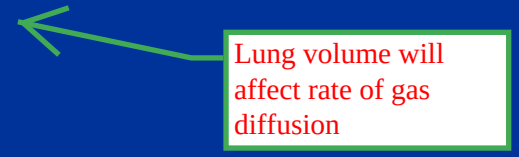
... skipped. Read the slide.

- 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 Va is very low - if the tracer gas cannot distribute, neither can the CO

Tracer gas is put into the lungs and its distribution is observed. Provides a rough measure of lung ventilation.

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

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



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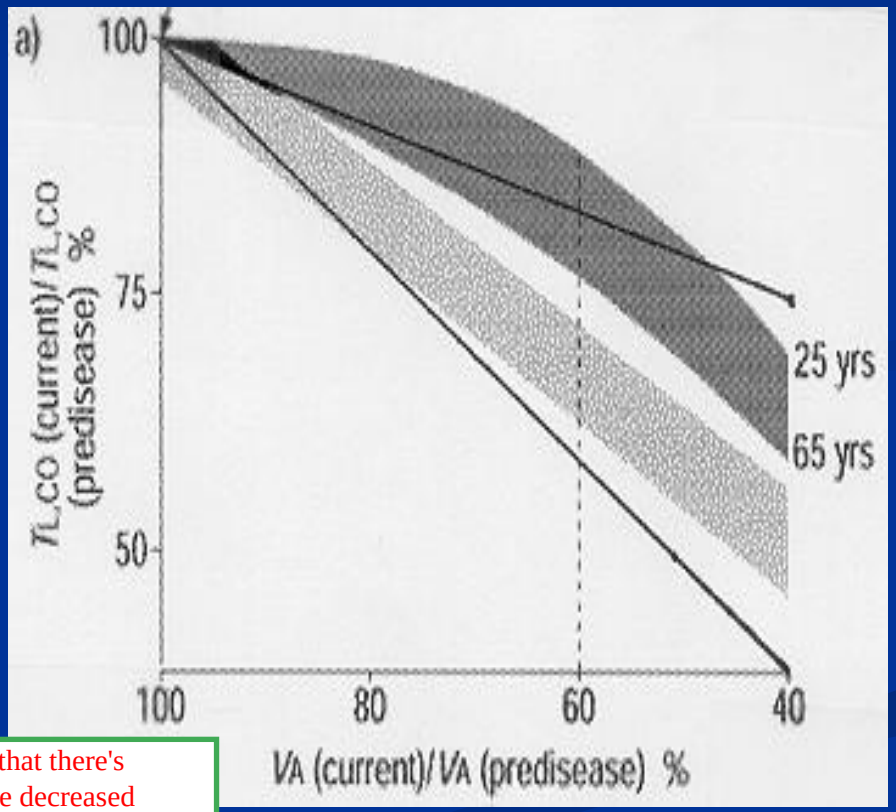
# Lung volume effects - low Vi

Dm = Membrane resistance
VC = Vital Capacity

- Less than maximal Vi
  - lower Vc and Dm (dark)
- Lobectomy/pneumonectomy
  - lower Dm, VC recruited (light)
- Simple Dl/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.

Ratio of Diffusing Capacity of Lung (DL) to Alveolar Volume (VA)

# DL/VA

... skipped. Read the slide

- 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

Gas diffuses into the blood okay. To do this, we have to get the gas to the alveoli. Here we're just not doing that well enough. (I.e. we're not getting enough of gas in contact with blood vessels)

- If DL reduced more than VA (low DL/VA), suggests vascular disease

Gas is getting into the alveoli but it's not diffusing into the blood.

# Patterns of pathophysiology

Measures gaseous exchange as opposed to mechanical behavior

| CATEGORY | FEV1/FVC | FVC | RV | DLCO |
|---|---|---|---|---|
| **Obstructed** | | | | |
| Asthma | nl/dec* | nl | nl/inc* | nl |
| Bronchitis | dec | nl | inc | nl |
| Emphysema | dec | nl | inc | dec |
| **Restricted** | nl | dec | dec | nl/dec |
| **Neuromuscular** | dec | dec | inc | nl |
| **Vascular** | nl | nl | nl | dec |

* during exacerbations/methacholine

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.

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

**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

# Pulmonary Function Testing

Recap of the things we talked about

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