The endurance athlete’s lung:

(everything you need to know in 40 minutes (or less))

Or...(The athlete’s lung: Everything we need to know in 40 minutes that has taken us 50 years to understand.)

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3 “Cs” of learning!

• Confusion.

• Contemplation.

• Comprehension.

• Today... I am responsible for the first... and you are responsible for the second and third. I challenge you to make it through to the other side.
A little history:

• “If we have seen a little further, it is by standing on the shoulders of Giants.”
  — Isaac Newton
A giant: Bruce David Dill

- Ph.D. degree in Chemistry from Stanford U 1925.
- Harvard University as a National Research Council Fellow to work with L. J. Henderson.
- In 1927 he became one of the founding faculty of the Harvard Fatigue Laboratory in the School of Public Health
- During World War II he served first with the U.S. Army Air Corps and later with the Quartermaster Corps, which awarded him the Legion of Merit.

In 1947, with the discontinuance of the Fatigue Laboratory appointment as scientific director of the Medical Division of the Army Chemical Center in Edgewood, Maryland. After his retirement from this position in 1961 he held research professorships at Indiana University with Dr Sid Robinson from 1960 – 1966 and then founded the Desert Research Institute at U Nevada. Widely considered one of the founding fathers of Exercise Physiology in the USA.
In the mid 1960’s ...

• Dill suggested “athletes suffer to an undo degree at altitude”

Hint* Mexico Olympic Games 1968
Athletes at altitude

• Athletes are much more susceptible to high altitude problems.... AMS, HAPE etc
• The higher their VO2 max the greater % decline with altitude
• Performance of the elite is much more affected that the sub-elite.
• Reductions in performance begin as low as 1-1500 mtrs in altitude.

Early observations
The great equalizer:

- Sea level
- Altitude

- VO2max

Endurance athlete

Sub elite athlete
Almost 50 years later....

• The evidence suggests that the lungs of elite highly trained endurance athletes ...

  – Are unable to adapt to the additional stress of altitude ( or other compromises EIB,EIA)

  – and... are already operating at or beyond their maximal capability at sea level.
In the mid 1970’s

- Researchers began to report the ‘incomplete saturation of arterial gases’ during heavy exercise in highly trained athletes.

- This had been reported before (late 1950’s) but no one paid much attention and thought the prevalence was low.
Exercise Induced Arterial Hypoxemia or (EIAH)

• Definition: Arterial saturation (SaO₂) below resting values (96-97%)

• Generally accepted as SaO₂ values below 92%.
  – (mild, moderate, or severe)

• For every 1% decline in SaO₂ below 92% there is a proportional decline in VO₂max (this is at sea level!)

• The more talented the cohort.. The greater the prevalence
Hb-O2 sat (SaO$_2$)
Why the incomplete saturation?

Or rather...

What is causing the Exercise Induced arterial hypoxemia?

And so what?
Causes of *all* Arterial Hypoxemia

- 1. Hypoventilation
- 2. Diffusion limitation
- 3. Shunt
- 4. Ventilation/perfusion mismatch
What does the lung do?

3 Terms we need to agree upon:

• **Ventilation** (diaphragm and accessory)
  – Convection or ‘bulk flow’ of air
  – “in comes the good air, out goes the bad”

• **Gas Exchange** (lung alveoli)
  – Between air and blood ($O_2$ & $CO_2$)

• **Respiration** (tissue)
  – Consumption or production of $O_2$ and $CO_2$
Ventilation

• Bulk flow of air in and out.
  – Diaphragm
  – Secondary accessory muscles

• Volumes (I and E)
  – Tidal volume and Fb

• Energetic cost
  1 ml/min to > 500 ml/min
Why is ventilation important (to performance)?

- $\text{VO}_2 = \text{VE} \ ((\text{FIO}_2) - (1-\text{FIO}_2/1-\text{FEO}_2-\text{FECO}_2 \times \text{FEO}_2))$

- Or ... another way of saying this is...

- $\text{VO}_2 = \text{Ventilation} \times (\text{differences in respiratory gases})$

- If VO2 max is important... ventilation is important.
Ventilation above the ventilatory threshold should increase disproportionately to metabolic rate.

- Alveolar PO2 should approach 150 mmHg or ambient air.
- Alveolar CO2 should approach ambient air (less than 35 mmHg).
- A-v gradients should expand.
• But in these highly trained athletes... neither PAO2 nor PACO2 changes appear adequate. (PAO2 IS ALVEOLAR O2 AND PCO2 ALVEOLAR CO2)

• And that is a big problem.

Cycling... which is a bigger ventilatory stimulant than running results in less EIAH.
Alveolar Gas Equations

- $V_A = V_E - V_{DS}$
  - where A is alveolar,
  - E is expired and
  - DS is dead space

- $PAO_2 = PIO_2 - RT\ VO_2/VA$
  - Where P is pressure, PIO2 inspired O2
  - RT gas constants VO2 is aerobic respiration
And ...

- \( \text{PACO}_2 = \frac{\text{VCO}_2}{\text{VA}} \)
  - \( \text{VCO}_2 \) is carbon dioxide production, \( \text{VA} \) is alveolar ventilation and \( \text{PACO}_2 \) is Alveolar CO2

So... in both cases, O2 and CO2, alveolar gases are related to the ratio of metabolic rate and alveolar ventilation.
The point is this:

- If VA is ‘inadequate’ then Alveolar O2 will be too low and Alveolar CO2 will be too high.

- This referred to as Inadequate Exercise Induced Hyperventilation

- And if this is so...
Then it is also true that:

• This ‘relative’ hypoventilation will cause a secondary diffusion limitation affecting gas exchange.

• In other words... gas exchange in the lung is dependent upon the appropriate alveolar gases... and alveolar gases are dependent upon ventilation.
And so... once again... why?

• What is (are) the mechanisms behind the inadequate exercise induced hyperventilation?

• Flow/volume limitation (possibly but not always... as FL>NFL VE

• Low ventilatory responsiveness
Flow volume limitation:

The overlap of the tidal envelope onto the maximal envelope defines F/V limitation.
From flow volume loops

• It appears that more than half the well trained elite athletes are “flow limited” ... though we might better label them as “flow maximized”.

• Flow limited athletes actually breath more than their non-flow limited counterparts.

• The consequence, however... is they have no ventilatory reserve. Their VE at VO2 max is equivalent to their ventilatory capacity. They are unable to respond to challenges.
• Ventilatory stimulants (caffeine for example) have only limited effects
Hypoxic and Hypercapneic ventilatory responsiveness
Elite endurance athletes

- Have a low ventilatory responsiveness
- As a result they are referred to as “non defenders” (of blood gases).
- In particular, they allow PaCO2 to go up with little increase in VE and allow PaO2 to drop with little ventilatory response.
- Oddly, then, endurance athletes do not make good mountaineers.
The time line here added to the confusion...

• We did not realize that athletes could be ‘flow limited’ when we were studying ventilatory responsiveness.

• Thus... the influence of HVR and HCVR as far as driving ventilation during exercise was masked by the effects of flow limitation.

• This may have been due to the acceptance of “ventilatory reserve” as a viable phenomena ....

• MVV vs Vcap.
MVV vs Vcap

• MVV is a 10 to 12 second maneuver of maximum breathing... but it is like predicting marathon performance from a 100 m run.

• Vcap is based upon the Flow/vol maximum loop.

• MVV is>>> Vcap and Vcap similar to VE @ max.
Cost of breathing

• And... if the athletes are not exercising at max... who cares? More over... if they are not exercising at VEmax... who cares?

• And they maybe able to compensate by high cardiac output (Q)... but...

• \((\text{VO}_{2\text{max}} = Q_{\text{max}} (a-\text{vo}_{2 \text{ diff}})_{\text{max}}))\)
Symmorphosis:

- A hypothesis, proposed by E Weibel and C R Taylor in 1981, postulating that biological systems adhere to an ‘economy of design’ giving a close match between their various structural and functional parameters. Hence, no single parameter in the system has unnecessary excess capacity, beyond the requirements of the system.

You can’t fool mother nature!
Central governor theory

• It isn’t random ‘failure’ it is all regulated by the CNS.

• And of course… metabo-reflex. There is a blood flow hierarchy that preserves brain, heart, and ventilatory function. This has been well known to exist for thermoregulatory function vs sk muscle blood flow.
Yes, it is complicated.

• But, any given athlete can be assessed as a means to determine the potential ‘causes’ of performance failure.

• From this, recommendations can be made as to the most appropriate strategies for success.
Causes of all Arterial Hypoxemia

• 1. Hypoventilation

• 2. Diffusion limitation

• 3. Shunt

• 4. Ventilation/perfusion mismatch
Gas Exchange

- Diffusion: movement of a gas along its concentration gradient - from high to low

- \( V_{\text{gas}} = \frac{A}{T} D_{\text{gas}} (P_1 - P_2) \)

- \( A = \text{area} \)
- \( T = \text{thickness} \)
- \( P = \text{partial pressure of the gas} \)
- \( D_{\text{gas}} = \frac{\text{sol}}{\text{sq rt MW}} \)
Simplified:

- $V_{\text{gas}} = K (P_1 - P_2)$

- The rate of diffusion across the lung is related to a series of constants but proportional to the gradient between the alveolar gases ($P_1$) and the mixed venous gases ($P_2$).

- Hypoventilation reduces diffusion by reducing this $P_1$-$P_2$ gradient (in particular alveolar $O_2$).
Lung transit time & cardiac output

Normal ‘available transit time is about .75 seconds. This leaves a reserve of nearly 0.5 seconds.

During exercise as cardiac output increases, available transit times shortens until it is less than required (about 0.25 sec)

Because transit times are less than required equilibrium times.. SaO2 falls.
• Endurance athletes have high VO2 max values because they have large cardiac outputs...

• 1) they have high stroke volumes

  – But... the high cardiac outputs cause short transit times... lowering saturation... compromising saturation. Chicken or egg?

• But do they have a choice? It is either bulk flow of blood as a compensation for the lower SaO2 or higher SaO2 at a lower cardiac output.
Respiration

• This is the process that takes place within the cells. Intermediary Metabolism

• $\text{C}_6\text{H}_{12}\text{O}_6 \rightarrow \text{H}_2\text{O}, \text{ATP}, \text{heat and CO}_2$
VO₂ max: Aerobic Capacity

• Definition: the highest volume of O₂ consumed during any one minute interval.

• Tested using a progressive, continuous, incremental exercise challenge. Mode dependent.

• Values are reported in liters of O₂ per minute or ml/kg/minute. 25 to 85 ml/kg/min
Theoretical maximum VO$_2$max

- $\text{VO}_2 = Q_{\text{max}} \ (a_{O_2} - v_{O_2 \text{ diff}}_{\text{max}})$

- Where $Q_{\text{max}} = \text{HR}_{\text{max}} \times \text{Stroke Volume}_{\text{max}}$
- $a_{O_2} =$ arterial O2 content
- $v_{O_2} =$ mixed venous content
- HR = heart rate and Stroke Volume
• \( \text{VO}_{2\max} = Q_{\max} (a_{O_2} - v_{o_2} \text{ diff}_{\max}) \)

• Where \( a_{O_2} = \text{gHb x O}_2/\text{gHb x SaO}_2 \)

• Put it all together then....
As SaO2 falls .... Due to relative hypoventilation, tissue and mixed venous values fall until they can not decrease any further. At this point a-vO2 difference is limited.

And If this is limited then, because

\[ \text{VO2max} = \text{Q max (a-vo2 diff)} \]

VO2 max is lowered unless ... there is some compensatory mechanism.
Causes of all Arterial Hypoxemia, in particular EIAH, seem to be multiple, and potentially different:

• 1. Hypoventilation

• 2. Diffusion limitation
And as we have seen before:

• The outcome may be the same symptom...
  EIAH

  – The cause may be one, two or three ...
    two of three or all three.

Athletes solve or *resolve* limitations as individuals.
After 40 years... we know that

• Elite endurance athletes tend to “under breath” during exercise relative to their metabolic rate. This isn't really hypoventilation... It is inadequate hyperventilation. But this leads to secondary diffusion limitations.

• Flow limitations & low ventilatory responsiveness are likely the cause of limited pulmonary performances at even moderate altitudes. But.... EIA and EIB need to be explored.

• High cardiac outputs contribute to EIAH and cause primary diffusion limitations in the lung. Again severe consequences at even modest altitude.

• May face limited ability to train when challenged by EIB EIA, URTI, and modest altitude.
Bottom line:

- Elite endurance athletes are limited in their ability to withstand “pulmonary” challenges.

- As Dill suggested: “elite endurance athletes suffer to an undue degree at altitude.”

- The non-flow limited athlete might be advantaged at altitude due to “standard altitude time qualification adjustments.”

- But you need to know the athlete’s susceptibility as this might determine, for example the adaptation to altitude training... or worse... the maladaptation to altitude competition and or training.
James E “Doc” Counsilman

• Used to caution ... “when training athletes in a team setting, treat all of them as equals, but train all of them as individuals.”

• We are reaching the day, through scientific discovery and modern technology, that this is truly possible.
Confused? Thank you!

Harms, Dempsey, Dill, Levin, Chapman, Weil, Tucker, Gavin, Derchak, Amann, Powers, Babb, West, Wagner, Faulkner, Robinson, Tanner, Stickford, McKenzie, Chapman and many, many others
Facts and fiction about the pulmonary system:

• Athletes have bigger lungs (no)
• Exercise causes lungs to grow (no)
• Athletes have more efficient lungs (no)
• Athletes breath more efficiently (no)
• Ventilatory apparatus adapt to exercise & training (maybe)
• Ventilatory apparatus can fatigue and limit performance (yes)
• The pulmonary system has unlimited reserve (no)