

Mallory: a story and a Mystery

 June 8, 1924, George Mallory and Andrew Irvine







hypoxia, 1862

Albuquerque, ~ 1500 meters



Ambient Pressure and oxygen

- (PO2) = %O2 x Pb water vapor
 - Sea level
 - PO2 = 760-47 x .2093 = 149 mmHg
- Albuquerque (5200 ft)
 PO2 = 630-47 x .2093 =122 mmHg
- Pikes Peak (14,300 ft)
 - PO2 = 430-47 x .2093 = 80 mmHg
- Everest (29,028 ft)
 - PO2 = 250-47 x .2093 = 43 mmHg

meters	Altitude	Pressure (Torr)	P ₁ O ₂ (Torr)	PA O2 (Torr)	So
5250-	17500-	392-	70 1	50 -	80
-	1 0000	431-	80 -	55 -	82-
4500-	15000-			60 -	84-
3750		473-	90 -	65 -	86 -
100			100 -	70 -	88-
3000		520-	110-	75 -	90-
2250-		572-		80 -	92-
			120-	85 -	
1508-	5000	629	130 -	90 -	94-
750 -	2500-	401-	140 -	95 -	96-
100	2000	071-		100 -	98 -
0	0	760	150		100-

Acute Pulmonary Responses

- Hyperventilation (at 4300 m, Ve increases 30%)
 caused by hypoxia (arterial chemoreceptors)
- $CO_2 + H_2O \rightarrow H_2CO_2 \rightarrow HCO_3^- + H^+$
 - decreases PACO2
 - decreases HCO3⁻
 - respiratory alkalosis
 increases PAO2
 - shifts Hb dissociation curve left
 - pulmonary hypertension
 - Cheyne-Stokes breathing



Acute responses: cardiovascular

- Decreased a-vO₂diff (decreases a)
- Increased resting and submax HR
- Decreased SV

 Hypoxia, ↑ TPR, ↓ PV, ↑ HR
- increase in submax Q
- max Q decreases slightly or remains the same

Acute Responses: body fluids

- · Increased fluid loss
 - lower water vapor, hyperventilation, vasoconstriction, diuresis
- Reduced plasma and blood volume
- · Increased hct and viscosity

Hypoxia-inducible factor

- Present in most cells and inactivated by the presence of O_{2}
- Hypoxia, HIF-1 is formed, moves to the cell nucleus, binds to a gene promoter
- Gene causes the transcription of mRNA for EPO
- · Also transcribes mRNA for VEGF
 - vascular endothelial factor causes growth of new blood vessels





Acclimatization: Body fluids

- Increased Epo from kidney (PO₂)
- · polycythemia with no increase in BV
- Increased 2,3-disphosphoglycerate
 - Shifts hb dissociation curve back to the right
 - compensation for alkalosis
- Excretion of HCO₃⁻
 - Restores acid-base balance

Acclimatization: cardiopulmonary

- Ve, further increases
 Increased sensitivity of arterial baroreceptors
- PAO₂, further increase
- submax HR remains elevated
- submax Q falls, SV lowers
- max Q lowers
- some restoration of VO_{2max}
 endurance trained athletes who live at altitude for years never regain their sea level VO_{2max}

Acclimatization: muscle

- Increased muscle capillarity
- reduced muscle fiber size
- increased mitochondria
- increased aerobic enzymes?
- increased reliance on carbs
- increased muscle myoglobin
- Body composition

loss of LBM and weight

 increased BMR, extra 340 kcal/d

Native responses

- Oxygen-carrying capacity of HA Peruvians is 28% > sea level residents
 - smaller size with a larger chest (barrel)
 - increased heart size
 - larger lungs, more capillaries
- Monge's disease (Chronic Altitude Sickness):
 - persons who live at altitude
 - symptoms similar to altitude sickness
 - hct 80, blue lips, clubbed fingers
 - sludging of RBC
 - more common in men

High Intensity Exercise

- · for 10s max cycling, no effect
- sprint activities less than 1 min are not impaired at moderate altitude
- More prolonged intense exercise
 - decreased max lactate
 - increased acidosis
 - due to reduced HCO₃⁻ and buffering capacity?

VO_{2max}

- Decrease VO_{2max}

 Proportional to reduction in P_b
- Decreased VO_{2max} is due to
 - reduced PaO₂
 - impaired O2 extraction from muscles
 - decreased Q_{max}
 - due to decreased $\mathrm{HR}_{\mathrm{max}}$ and $\mathrm{SV}_{\mathrm{max}}$



Above 1500m, VO_{2max} decreases by 9.2% each 1000m > fitness > effect



- 1978, Messner and Habeler were the first

Cardiorespiratory Endurance

- Decrease in VO_{2max} and increase in blood lactate independently decrease tolerance to prolonged exercise
 - time trials at 1-3 miles at 2300m were 2-13% slower

Cardiovascular Responses to Submaximal Exercise

- Greater increase Ve
- Increased VO₂ (work of breathing)
- Increased HR
- Decreased SV
- Increased Q (lower a-vO₂diff)
- No change muscle bf (increased hct)
- Increased blood lactate

Metabolic Response to Exercise

- Higher lactate during submax exercise, but < lactate at max
- No change in LT at given %VO_{2max}
- · Greater reliance on carbs

Lactate Paradox

- decreased <u>maximal</u> lactate after chronic altitude exposure
 - due to increased lactate uptake by active and inactive skeletal muscle, the heart, kidney, and liver
 - reduced ability of CNS to support exercise, lower maximal work intensities
 - reduced ability to mobilize glucose and thus form lactate (McArdle, pg 452)

Mexico City Olympics

- 1968 Olympics in Mexico City

 altitude of 2300 m, Pb 569 mmHg
- Beneficial effects

 jumping, throwing, sprinting
- Negative effects

 running distances > 1mile
- Sparked interest in best ways to train

Benefits of moderate altitude acclimatization

- Natives to moderate altitude (2000m) experience fewer problems with exposure to higher altitude (4300m)
 - less mountain sickness
 - 1/2 decrement in VO_{2max}
 - larger maximal Ve

Time for acclimation

- 2 wks to adapt to 2300m
- thereafter for each 610m increase in altitude, 1 additional wk up to 4572m

Altitude Training Questions?

- Can altitude living improve altitude performance?
- Can altitude living improve sea level performance?

Altitude living and altitude performance

- No doubt, altitude exposure improves altitude performance
 - $-\operatorname{increases}$ hct and hb concentrations
 - increases VO_{2max} 5-10%

Altitude training to improve sea level performance?

- Mixed results
 - 2300 to 3300m training for 2 wks improved 1500m and 1 mile race times at sea level
 - 3100 to 4000m training for 20-63d produced slower sea level times and decreased $\rm VO_{2max}$
- To obtain benefits, training must be done at low or moderate altitudes
- At higher altitudes athletes can't train well and times will be reduced

Live high and Train Low

- Train at lower altitude to optimize work
 outs
- Athletes who lived at 2500m but trained at 1250m had greater increases in 5000m run than
 - athletes who lived and trained at 2500m
 - athletes who lived and trained at sea level

Sea level altitude training

- Normbaric hypoxia
 - increase inspired nitrogen during training
 - hypoxic sleeping tent
- Hypobaric chambers



Altitude Illnesses

- Ravenhill Br. physician 1913
 - first categorized types of altitude illness in the Andes
- AMS, Acute mountain sickness
- High altitude pulmonary edema (HAPE)
- High altitude cerebral edema (HACE)
- Each vary with the rate of ascent and individual susceptibility



Acute Mountain Sickness

- Symptoms
 - headache, nausea, vomiting, dyspnea, insomnia
 - begins 6 to 96 hrs at altitudes > 3000m
 - -0.1 to 53% at altitudes from 2400 to 5500 m
 - 6.5% men, 22.2% women at 2400-3400m
 - 80% at 4200m

HAPE

- Rapidly ascend > 2700m
- 2% of people in 12 to 96 hrs
- fluid accumulation in lungs interferes with gas exchange, from pulm htn
- · cough, pink frothy sputum, rales
- shortness of breathe, extreme fatigue
- cyanosis, confusion, loss of consciousness
- more often in children and young adults
- give oxygen and DESCEND

HACE

- · Fluid accumulation in the cranial cavity
- hypoxia causes vasodilation of cerebral blood vessels
- 1% of people > 2700m
- · mental confusion, coma, death
- most cases at >4300m
- give oxygen and DESCEND

Prevention of Altitude Illnesses

- Gradual ascent

 no more than 300m/d above 3000m
- · Climb high, sleep low
- Drugs
 - acetazolamide (Diamox)
 - diuretic, increases $\text{HCO}_{3^{\text{-}}}$ excretion
 - dexamethasone
 - synthetic glucocorticoid (anti-inflammatory)
- High Carbohydrate diet (>70% cal.)

