Heart rate variability and altitude: implications for training

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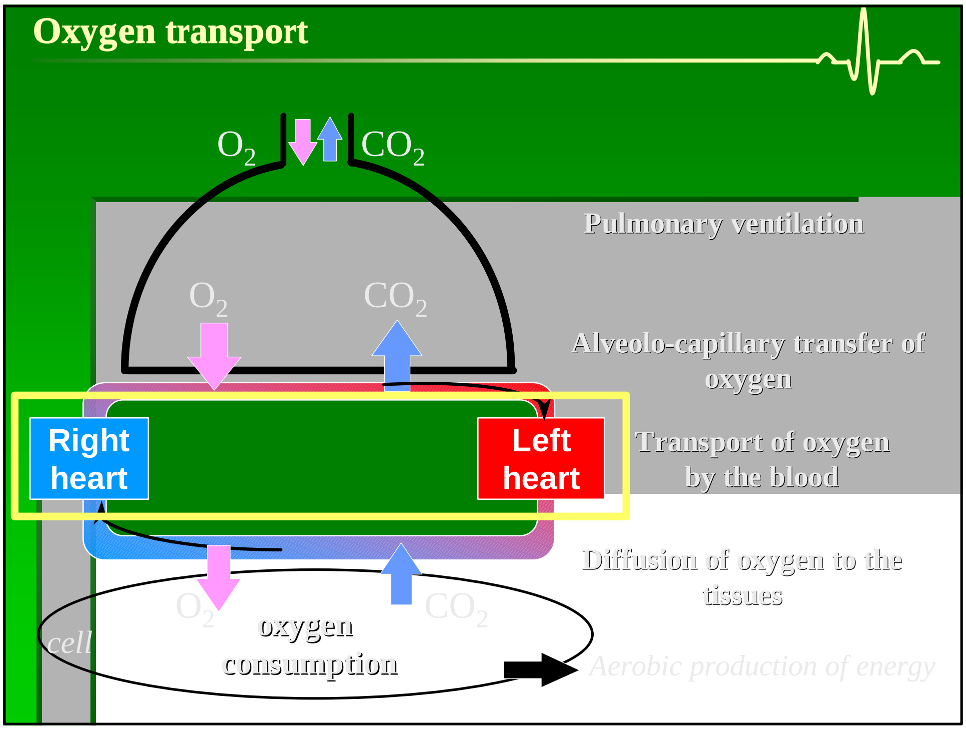
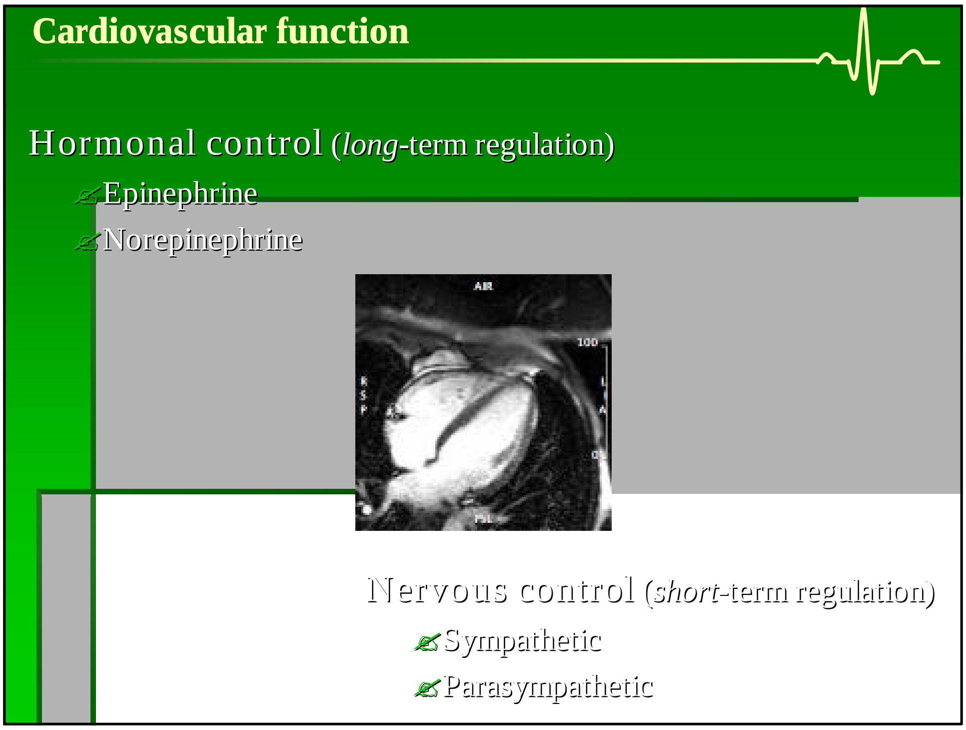
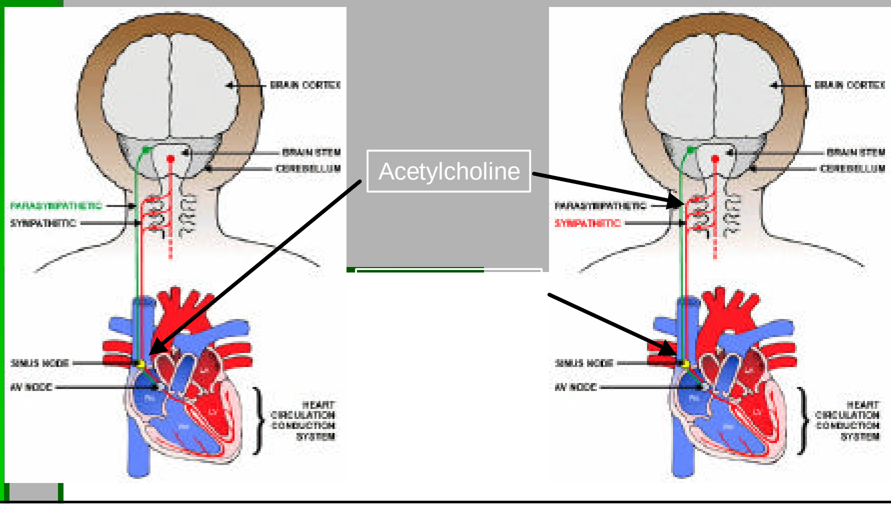

Oxygen transport
O2
CO2
Pulmonary ventilation
O2
CO2
Alveolo-capillary transfer of oxygen
Right heart
Left heart
Transport of oxygen by the blood
Diffusion of oxygen to the tissues
O2
CO2
cell
oxygen consumption
Aerobic production of energy


Cardiovascular function
Hormonal control (long-term regulation)
Epinephrine
Norepinephrine
Nervous control (short-term regulation)
Sympathetic
Parasympathetic

Autonomic nervous system

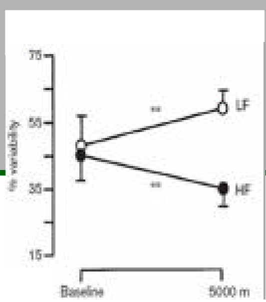
- Parasympathetic (ParaS)
- Sympathetic (S)

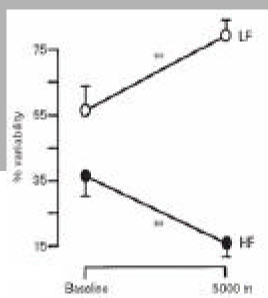


HRV - Origin

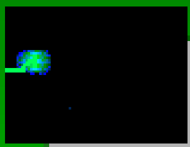
- Blood pressure variations during the respiratory cycle induces variability into HR
  - ? *respiratory sinus arrhythmia*

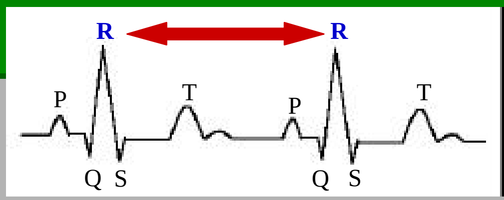
Mean R-R intervall variability

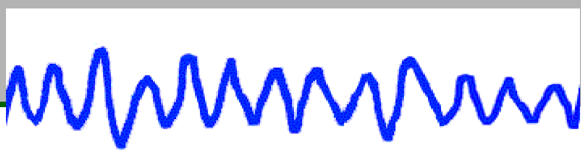




What is HRV?







The duration between 2 heart beats (RR interval in msec) is not constant

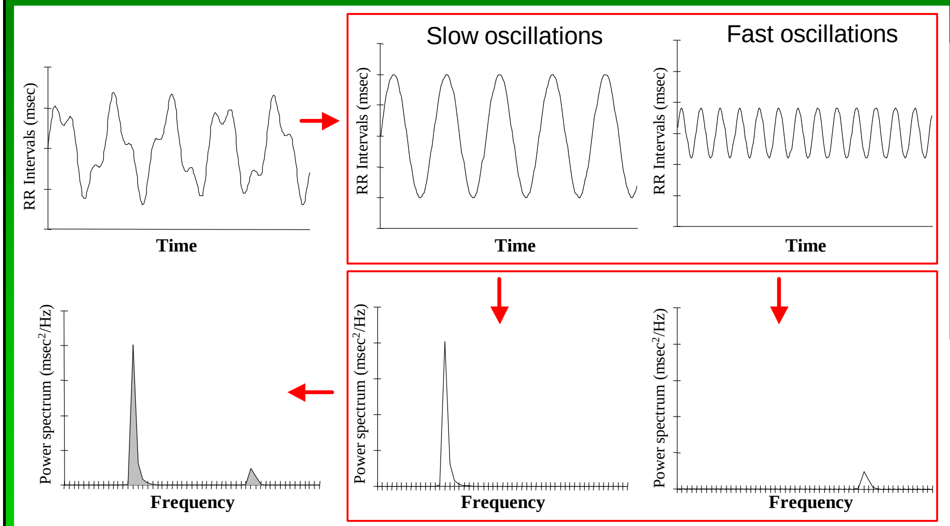
HRV - Analysis

- Temporal analysis
  - ? Quantitative description of the paraS modulation
  - ? Long-term monitoring
- Spectral analysis
  - ? *Qualitative*

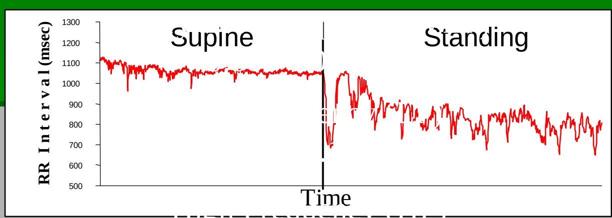
(ar

exercise *orthostatism*

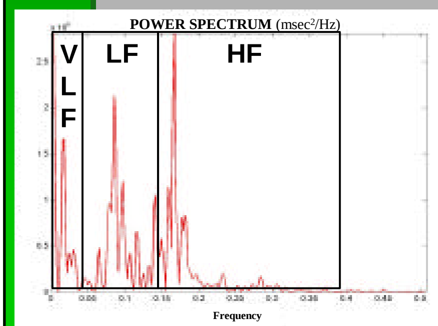
Spectral analysis



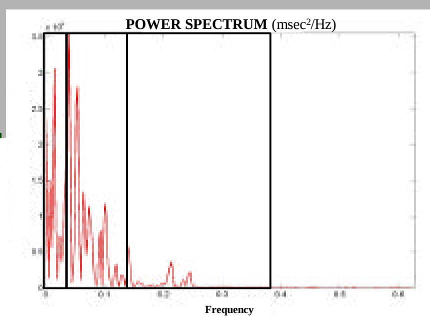
Spectral analysis



0.15 – 0.40 Hz



Supine



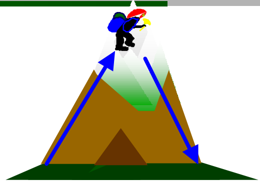
Standing position

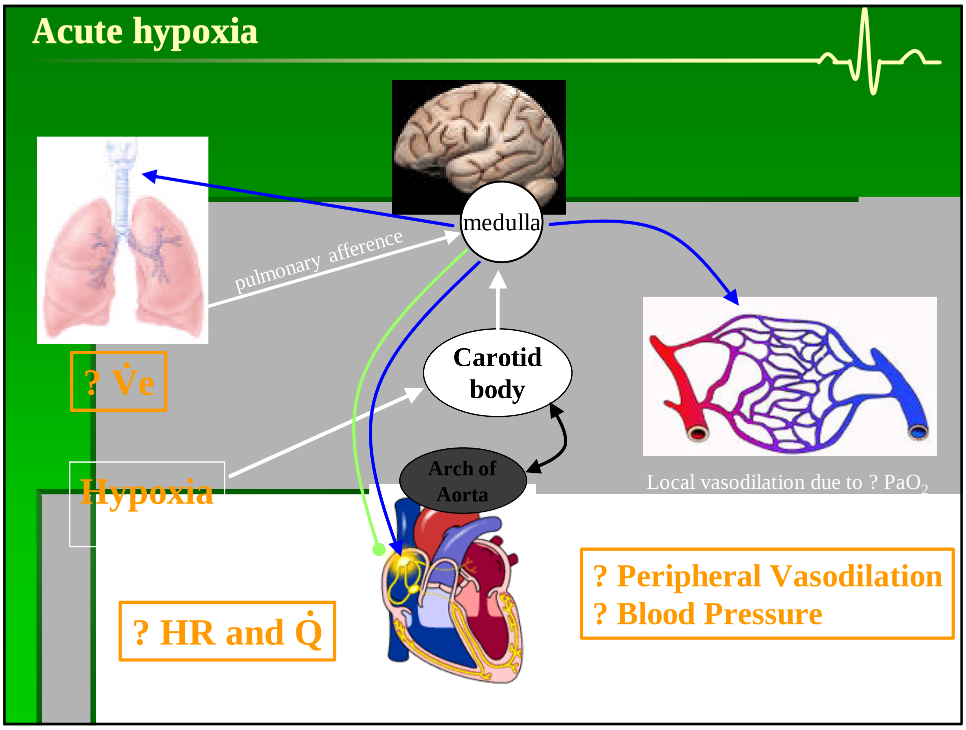
Physiological significance

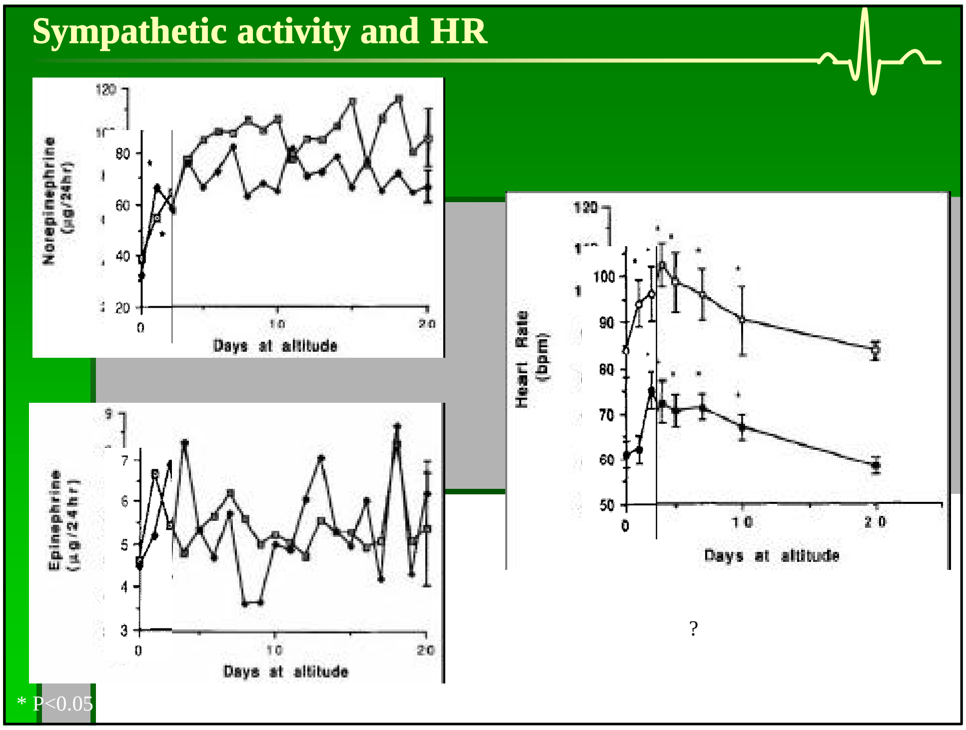
- Very Low Frequency
  - ? long term mechanisms of regulation
- Low Frequency
  - ? its physiological interpretation remains controversial
  - ? reflects both sympathetic and paraS modulations
- High Frequency
  - ? parasympathetic influence associated with breathing frequency (respiratory sinus arrhythmia)
- LF / HF ratio
  - ?

(or

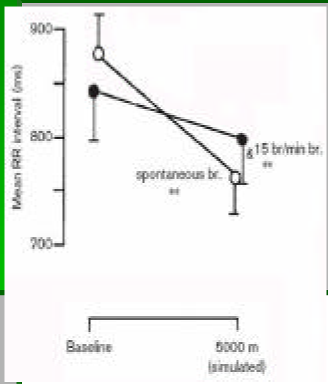
Acute hypoxia

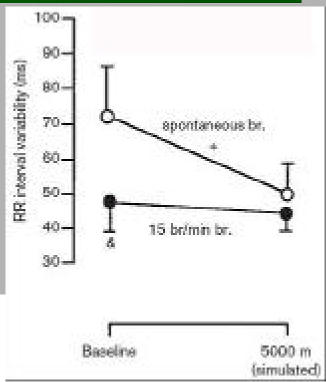



Acute hypoxia
medulla
pulmonary afference
Carotid body
Arch of Aorta
? V̇e
Hypoxia
? HR and Q̇
Local vasodilation due to ? PaO_2
? Peripheral Vasodilation
? Blood Pressure


Sympathetic activity and HR
Norepinephrine (µg/24hr)
Days at altitude
Epinephrine (µg/24hr)
Days at altitude
Heart Rate (bpm)
Days at altitude
?
* P<0.05

Effect on HRV

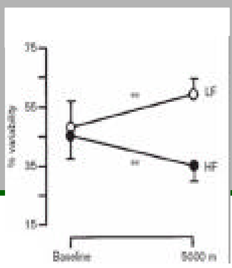


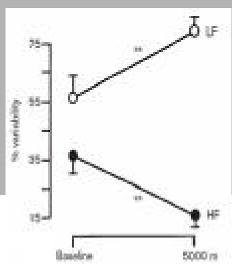


& P<0.05
* P<0.05

Effect on HRV

- Decrease in HF
- Increase in LF (related to respiratory frequency)
  - LF/HF ratio is usually increased, so is the sympathetic modulation

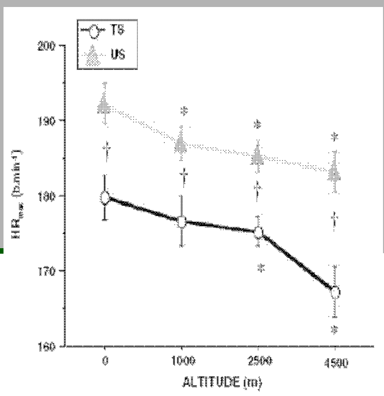




- Decrease in total spectrum power of HRV

Exercise

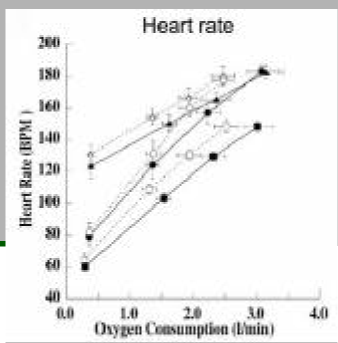
- HRmax is decreased
- Submaximal HR is increased



* P<0.05
† P<0.05

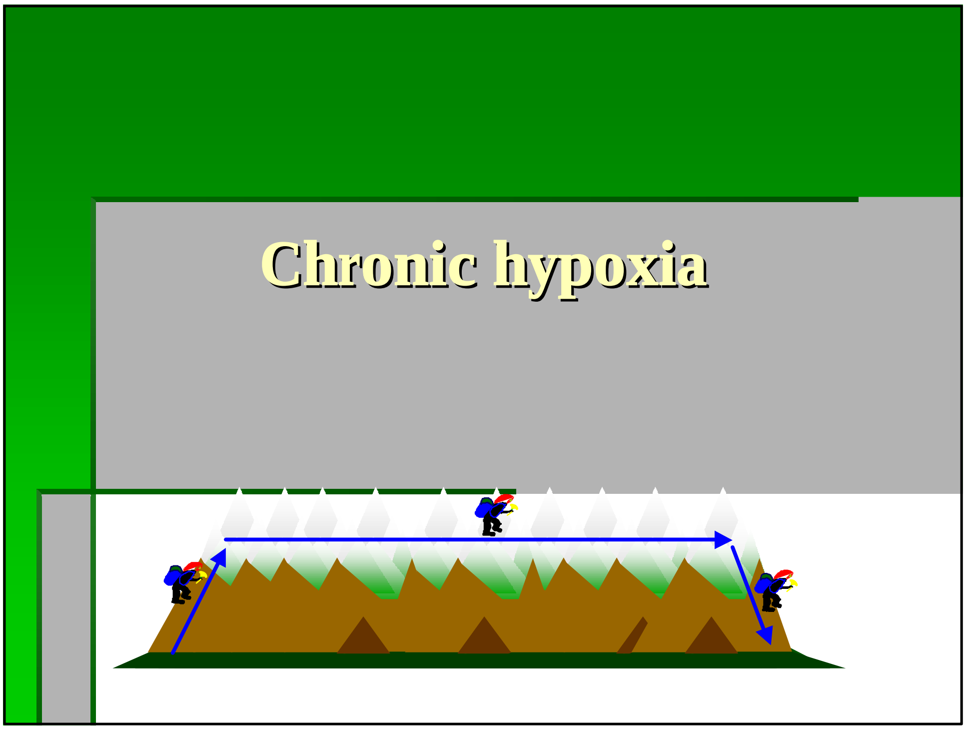
Exercise

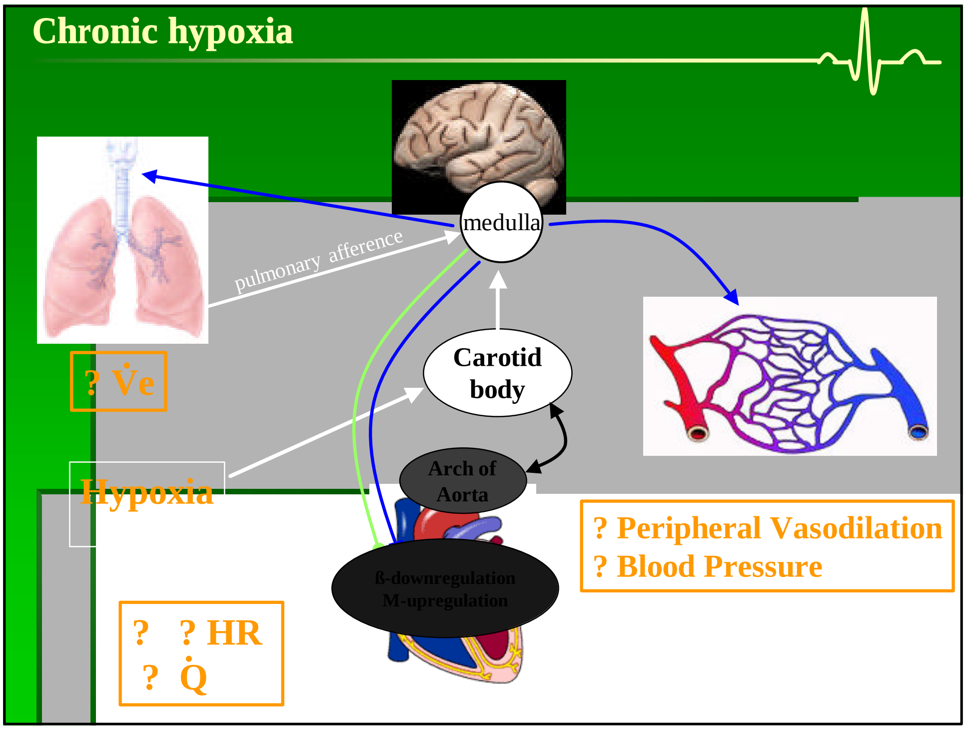
- Increase in HR is not limited by ß-sympathetic or parasympathetic blockade
  - Potential role of the a $_1$-adrenergic receptor stimulated by NAd



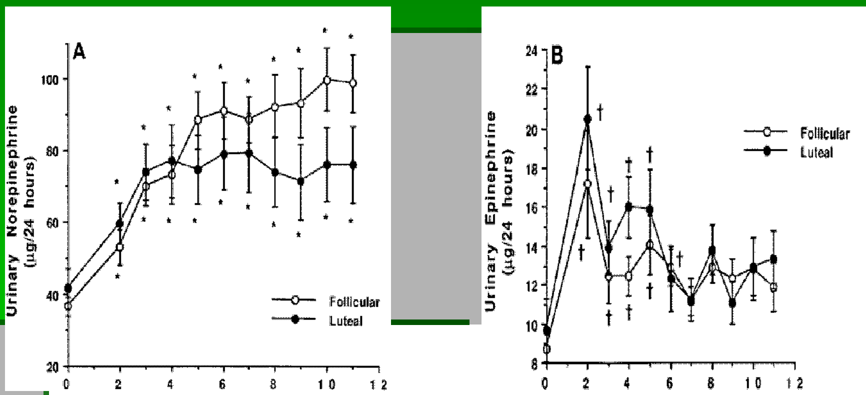
ß-symp
ß-symp
? control
? control

?


Chronic hypoxia


Chronic hypoxia
medulla
pulmonary afference
Carotid body
Arch of Aorta
ß-downregulation
M-upregulation
? V̇e
Hypoxia
? ? HR
? Q̇
? Peripheral Vasodilation
? Blood Pressure

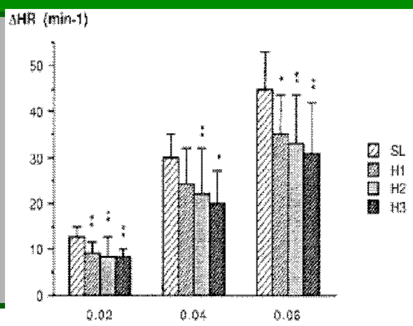
Sympathetic activity



Days in altitude

* P<0.05
† P<0.05

Sympathetic activity

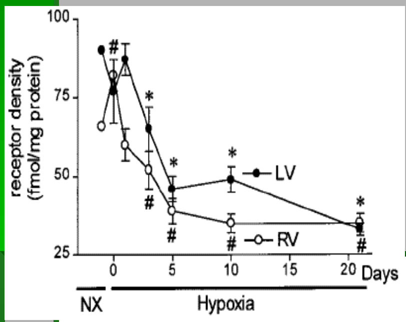


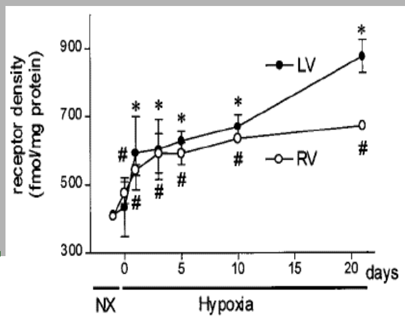
H1: 2 day
H2: 13 da
H3: 21 da

* P<0.05

Cardiac receptors

- ß-adrenergic receptors
- Muscarinic receptors

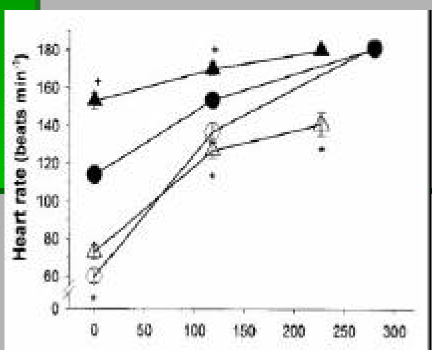




* P<0.05
# P<0.05

Parasympathetic activity

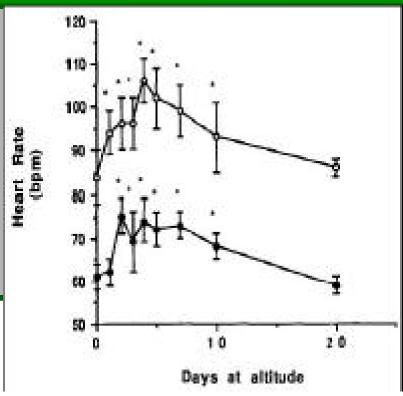
- Relative increase due to paraS blockade is greater at altitude vs. seal level (109% vs. 86%, resp.)
  - Enhanced parasympathetic neural activity accounts for the lowering of HR



Power output (Watts)

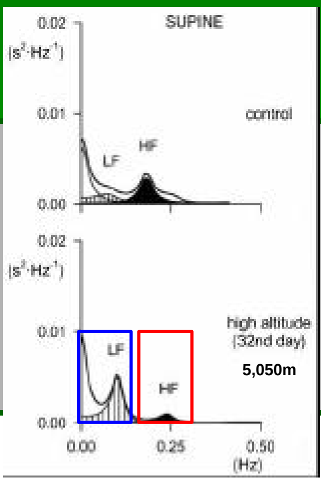
? Sea level Control
? Sea level Glycopyrrolate
? 5,260m Control
? 5,260m Glycopyrrolate

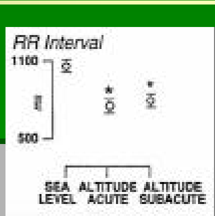
Heart Rate



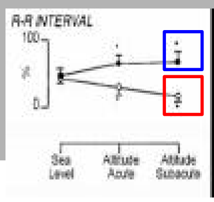
* P<0.05

Effect on HRV





1 day, 1 wk at 4,970m



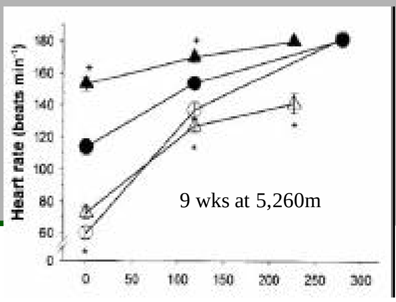
?

✍Total spectrum power is increased

Perini et al

Exercise

- HRmax is decreased for altitudes above 3,500m, due to:
  - decreased response to catecholaminergic stimulation (ß-adrenergic receptors)
  - increased parasympathetic tone

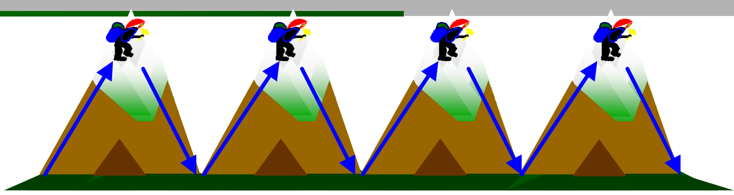


Power output (Watts)

* P<0.05
+ P<0.05

? ?

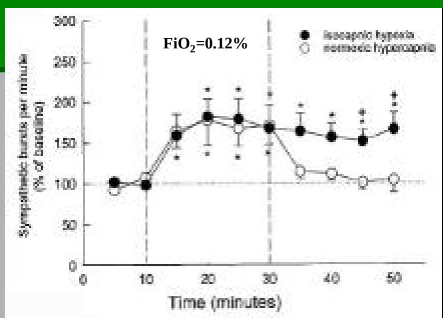
Intermittent hypoxia



Intermittent hypoxia

- Models of intermittent hypoxic exposure are multiple:
  - sleep apnea
  - altitude workers (Peru for example)
  - intermittent hypoxic training
- Intermittent hypoxic exposure is different from repeated acute exposures to hypoxia or discontinuous chronic hypoxia

Remnant effect of hypoxia



* P<0.05 vs. baseline
+ P<0.05 hypoxia vs. hypercapnia

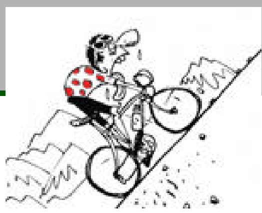
	HR (beats/min)
SL	62.8 ± 7.7
5,000 m	79.1 ± 11.0†‡
7,000 m	90.4 ± 13.7†‡
8,000 m	89.0 ± 11.3†‡
RSL	63.5 ± 10.0

† P<0.05 vs. SL
‡ P<0.05 vs. RSL

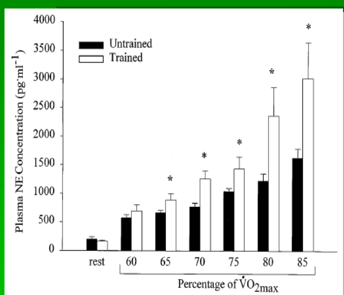
- Remnant effects last longer after continuous than discontinuous hypoxia

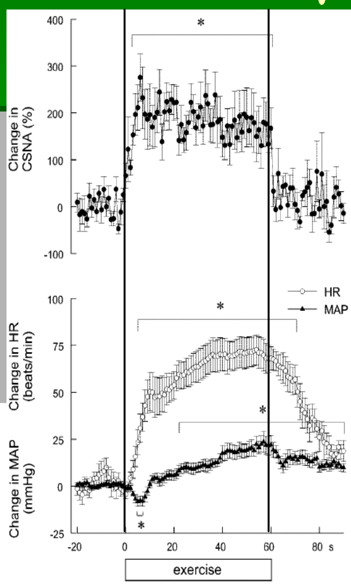
from Bous

“Acute” Exercise



Mechanisms





*

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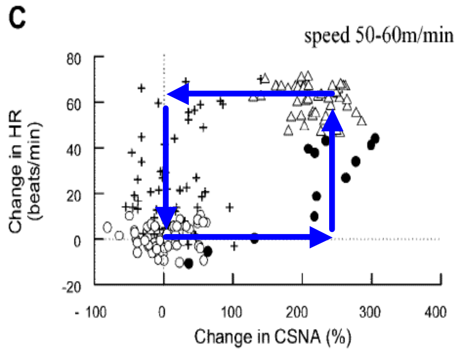
Kinetic

+ *after*:
Rapid drop in CSNA precedes HR decrease

+ *after*:
Gradual decrease in HR with no change in CSNA

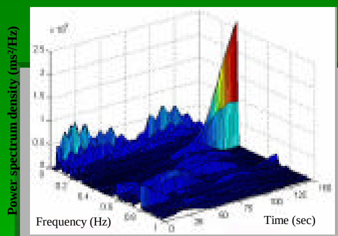


?

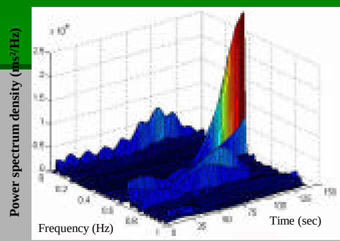
? *mid to late*:
Both CSNA and HR are elevated

HRV: methodological consideration

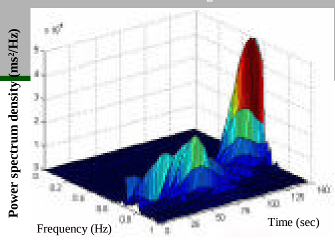
60% of PVO_2max



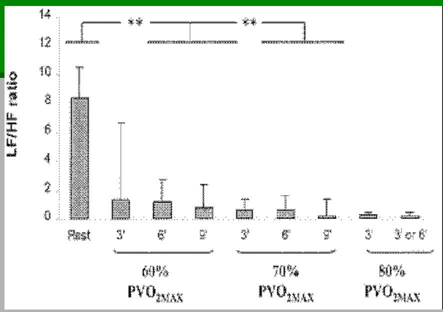
70% of PVO_2max



80% of PVO_2max



HRV: methodological consideration

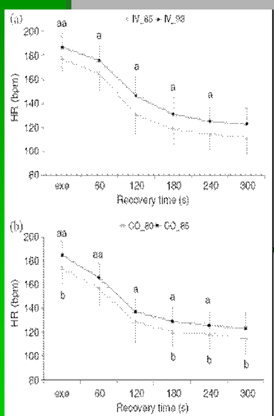


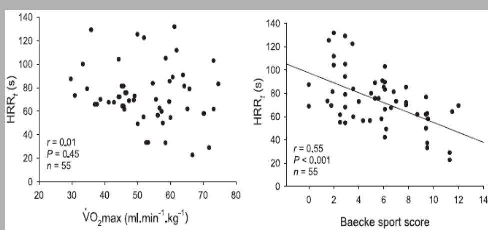
The standa rate d

** P<0.

Time course of recovery from exercise

- HR (time constant) recovery from an acute exercise is faster in subjects with moderate training load, whatever their fitness level



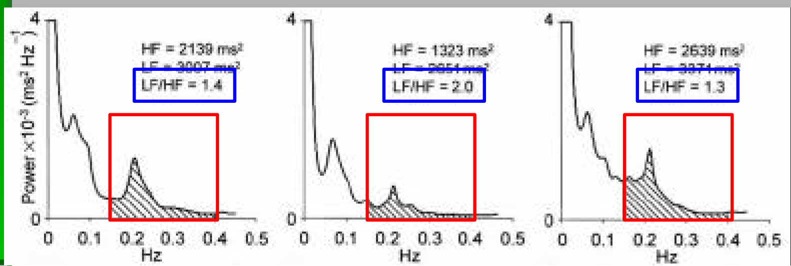


- Early recovery depends on the intensity

Kaikkone

Remnant effect of exercise

- The elevated resting HR post-exercise, associated with a high sympathetic modulation, can last for up to 24h



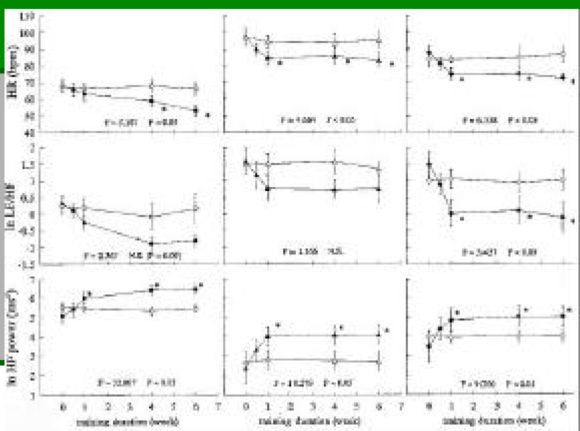
before the race

1 night after the race
75km X-country ski

2 nights after the race
75km X-country ski

- A p
pos

Remnant effect of exercise - trained vs. untrained



at rest

10min
after exercise

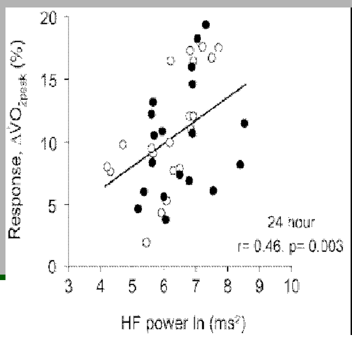
20min
after exercise

? Control group
? Training groups

* P<0.05

Remnant effect of exercise - trained vs. untrained

- Correlation between baseline HF component and response to 8 wk of aerobic training



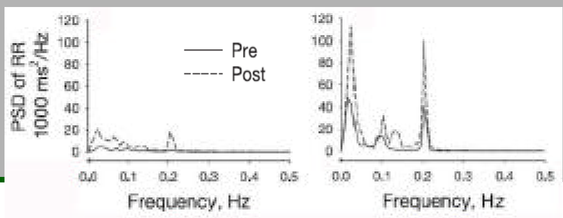
? Moderate-volume training group
? High-volume training group

inc

Endurance Training

Moderate intensity - total power of HRV

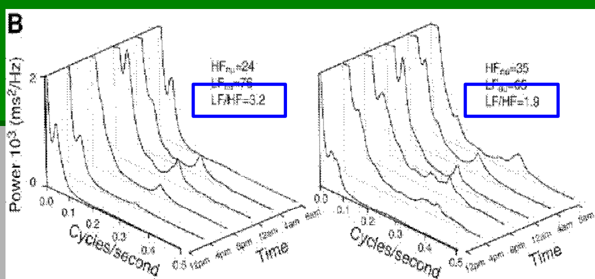
- Neurovegetative activity, as represented by power spectrum (PSD), is greater in athletes than in sedentary individuals



Sedentary senior
12mo of training

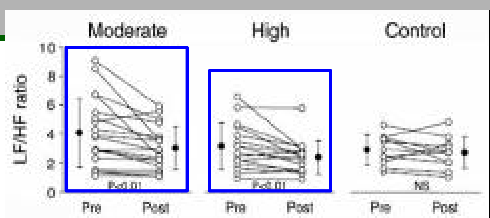
Young individual
6mo of training

Moderate intensity – sympathetic activity

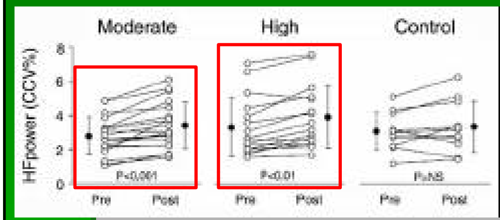


- Sympathetic component is decreased after training

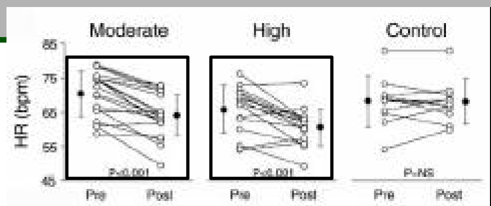
8wk
6 sessi
70-80%
30 or 6



Moderate intensity – paraS activity and HR

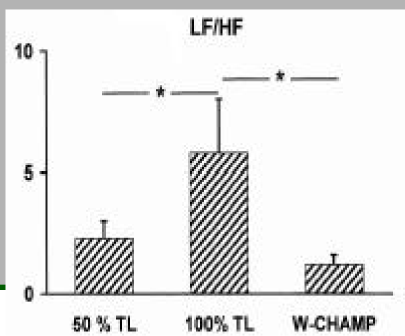


- Parasympathetic component is increased after training



High intensity – sympathetic activity

- Increase in the sympathetic component with high intensity training, rapidly reversible

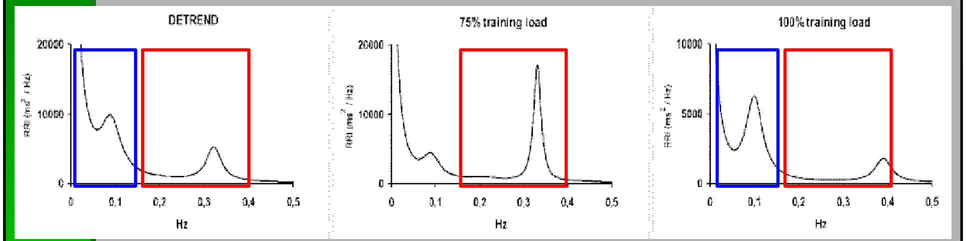


50% TL:
100% TL
W-CHAM

* P<0.05

High intensity – parasympathetic activity

- Decrease in the parasympathetic component with high intensity training



detrained | After ~3mo of training | ~20d before WC

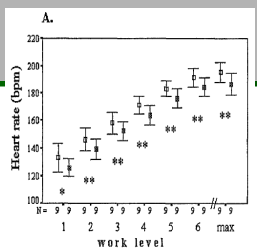
- Inc

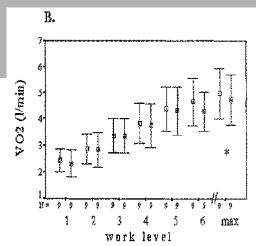
"Over-reaching"

- **Can be** characterized by:
  - a decreased total power of HRV with a LF predominance
  - an increased supine HR
  - a slow cardiac recovery from exercise
- But has also been described as inducing paraS modulation
- Can be considered as normal fatigue and not necessary associated with a drop in performance
- Can be observed during conditioning period of training when "non-pathological"

"Over-training"

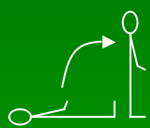
- **Can be** characterized by:
  - a large increase in total power of HRV with a high HF activity (endurance sports)
  - a decreased supine HR
  - a fast cardiac recovery from exercise
- Is always associated with a decrease in performance even in the absence of autonomic symptoms





* P<0.05
** P<0.0

Orthostatic hypotension



- Athletes have a lower tolerance to Tilt test vs. sedentary (proportional to their training status)
- This might be due to:
  - structural training adaptations: ? arterial and veinous compliance, ? cardiac compliance and hypertrophy, ? blood volume
  - decrease in baroreflex sensitivity
  - decrease in autonomic nervous system control on baroreflex, related to the parasymathetic modulation induced by training
- Orthostatic hypotension can be associated with overtraining

HRV and Training Follow-up Normoxia

Pre- and post-season differences

	Females (n=9)		Males (n=8)		Entire group
	Test 1	Test 2	Test 1	Test 2	
VO_{2max} (l/min)	3.65 (3.50–3.95)	3.75 (3.46–4.16)*	5.22 (4.60–6.43)	5.24 (4.64–6.39)	
HR_{max} (bpm)	196 (190–207)	196 (185–206)	198 (192–206)	196 (188–208)	
HR_{submax} (bpm)	188 (176–198)	184 (174–196)*	183 (173–196)	178 (168–187)*	**↓
Run time (RunT) (min)	17.9 (15.0–21.0)	19.0 (16.6–21.0)	19.1 (17.0–21.0)	19.8 (18.0–22.5)	*↑
RERmax	1.11 (1.04–1.18)	1.05 (0.97–1.12)*	1.10 (1.04–1.16)	1.08 (1.02–1.19)	**↓

✍ Performance is increased, but resting HR do not change

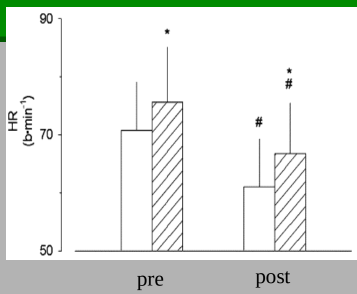
	Females (n=11)		Males (n=8)		Factor effects[2]	
	Test 1	Test 2	Test 1	Test 2	Test	Gender
Supine[1]						
Log HF power (mHz^2)	3.79 (3.06–4.48)	3.91 (3.32–4.59)	3.38 (2.87–3.88)	3.43 (2.26–3.91)		*
Log LF power (mHz^2)	3.06 (2.20–3.76)	2.93 (2.36–3.33)	2.78 (2.16–3.59)	2.97 (2.13–3.48)		
Log total power (mHz^2)	3.94 (3.25–4.51)	4.03 (3.43–4.60)	3.56 (2.96–4.04)	3.71 (3.08–4.01)	*	*

✍ Gender differences seem to exist ? Parasympathetic activity is greater in females than in males

* $P<0.05$
** $P<0.01$

Pre- and post-season differences

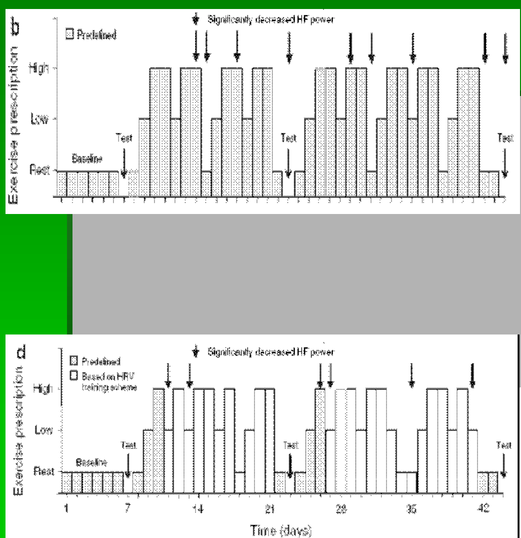
- resting HR is decreased and performance is increased
- No alteration in sympathetic or paraS markers
- Indirect marker of HR vagal reflex control could have been improved



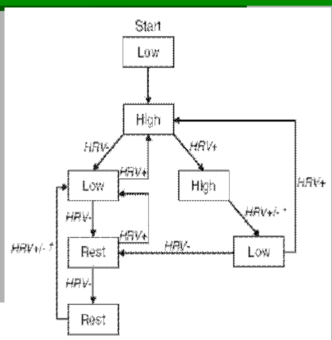
* P<0.05 between body position
\# P<0.05 pre vs. post

- Specific training adaptation (? in blood volume and cardiac dimension is less in swimmers than in runners) may explain these divergence
- These discrepancies may reflect methodological limitation in the use of HRV for long-term variations

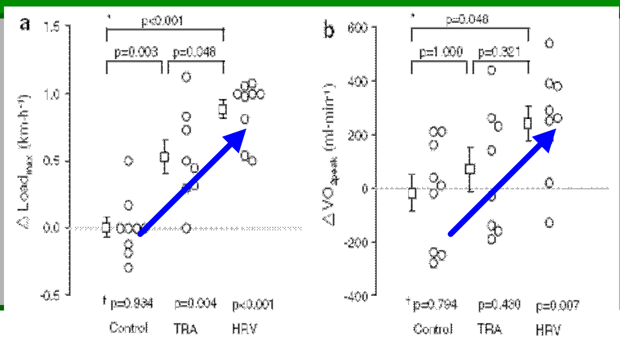
Training individualization



Low: 65% of HRmax, 40min
High: 85% of HRmax, 30min
+ warm-up/cool-down at 65%, 5min



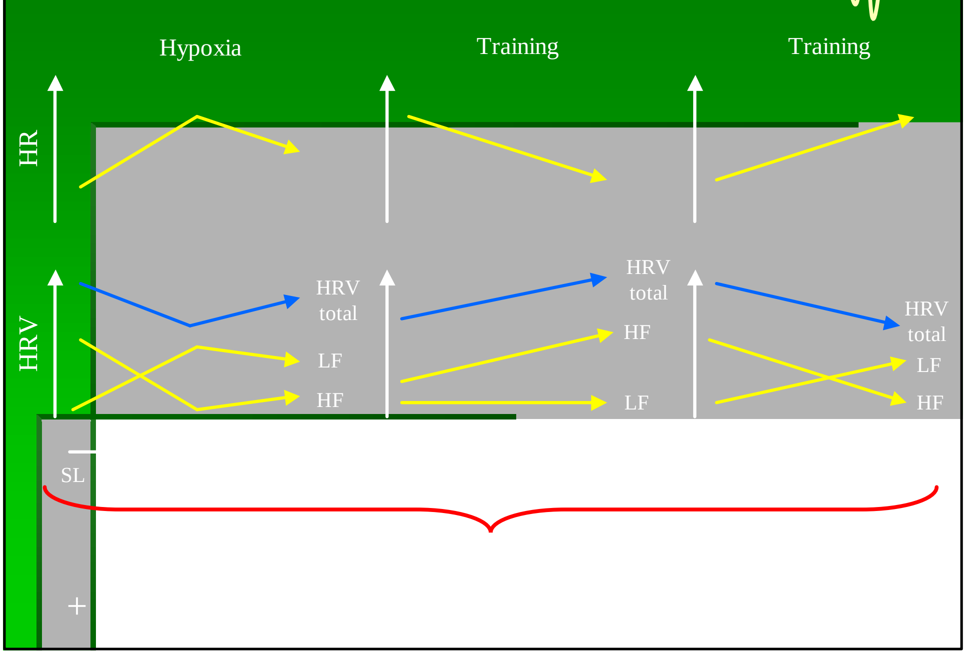
Training individualization



* P<0.05
† P<0.05

HRV and Training Follow-up Hypoxia

Summary – Hypoxia vs. Training

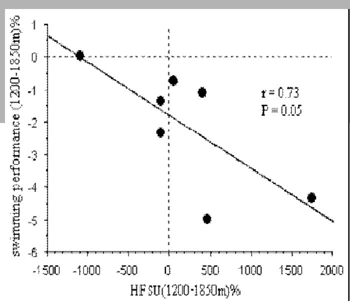


Altitude training - mild vs. moderate hypoxia

	Condition	Pre-test	Test 2	Test 3	Post-test
LF_{SU}	1200m	1037 ± 442	1267 ± 475*	941 ± 277	1550 ± 698
	1850m	1050 ± 373	913 ± 273#	841 ± 258	857 ± 358#
HF_{SU}	1200m	502 ± 225	1443 ± 1610	2182 ± 2803	2447 ± 2490*
	1850m	1004 ± 1214	577 ± 385	1099 ± 669	1393 ± 1384
LF_{SU}/HF_{SU}	1200m	2.41 ± 1.32	2.33 ± 1.74	2.12 ± 2.88	1.72 ± 1.87
	1850m	2.49 ± 2.38	4.34 ± 6.06	1.22 ± 0.84*	1.29 ± 1.29
Time 2000 m (s)	1200m	1471 ± 35			1432 ± 45*
	1850m	1457 ± 38			1450 ± 45

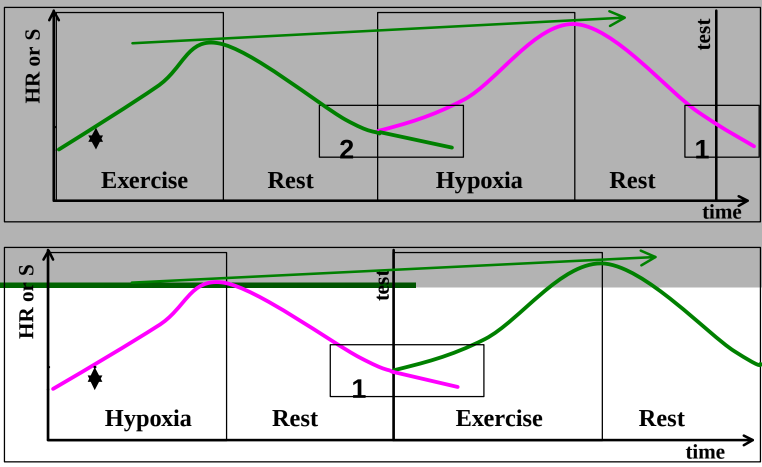
* $P<0.05$ vs. pre-test; # $P<0.05$ 1,200m vs. 1,850m

- Same relative training loads induce positive HRV adaptations (? HF_{SU} and LF_{ST}) at 1,200m and improve performance, but not at 1,850m
- HF sensitivity may help predict performance alteration

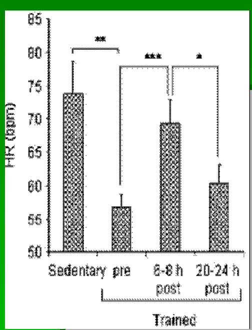


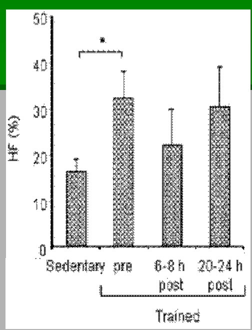
Intermittent hypoxia - *LHTL*

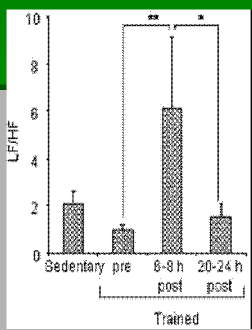
- What are the influences of the remaining effects of exercise and/or hypoxia?



LHTL – Remnant effects of exercise



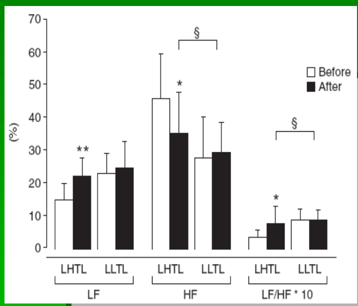


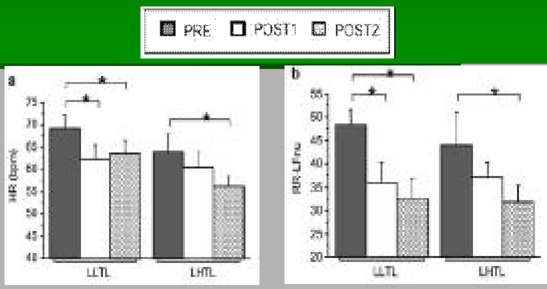


- Altitude does not affect intense-exercising remnant effects on HRV in a well-adapted population
- Living in altitude does not impair the autonomic response to training

* P<0.05

LHTL – Remnant effects of hypoxia





* P<0.05 before vs. after; ** P<0.01
§ P<0.05 LHTL vs. LLTL after

* P<0.05 vs. PRE

- Altitude exposure counteracts aerobic training induced alterations in autonomic control, but does not prevent further enhancement at the end of the exposure

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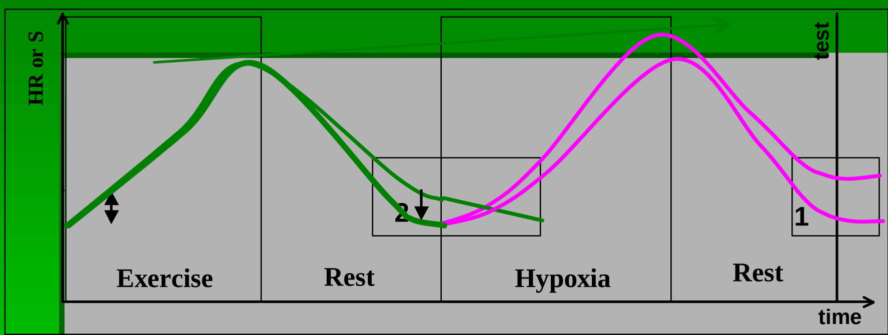
Intermittent hypoxia - *LHTL*



- LHTL can not be considered as repeated acute exposures to hypoxia
- This needs to be taken into account to controlling training with HRV

Take Home Message

- HRV is more useful on a day to day basis than for long-term comparison
- Overtraining being hard to diagnose, controlling training using HRV becomes very useful
- Training follow up over altitude training is ev
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