« Vivre en haut et s'entraîner en bas » et performance aérobie.

Altitude réelle (hypobarique) et altitude simulée (normobarique).

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Normobaric Hypoxia (NH)
vs.
Hypobaric Hypoxia (HH)

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Normobaric Hypoxia (NH) vs. Hypobaric Hypoxia (HH)

Combinations of barometric pressure (PB) and inspired fraction of oxygen (F_IO_2) that result in any inspired pressure of oxygen (P_IO_2) less than a normoxic value of 150 mmHg is by definition hypoxic (*Conkin and Wessel 2008*).

Altitude (m)	Pb (mm/Hg)	PO_2 (mm/Hg)	PAO_2 (mm/Hg)	$FO_{2\ normalisée}$ (%)
2 000	596	125	115	16,44
2 100	589	123	114	16,24
2 200	582	122	112	16,04
2 300	575	120	111	15,84
2 400	567	119	109	15,64
2 500	560	117	108	15,45
2 600	553	116	106	15,25
2 700	546	114	105	15,06
2 800	540	113	103	14,87
2 900	533	112	102	14,69
3 000	526 →	110	100	← 14,50
3 100	519	109	99	14,32
3 200	513	107	98	14,14
3 300	506	106	96	13,96
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

(Millet & Schmitt, 2011).

PO_2 can be perfectly matched between NH and HH

Inducing similar acute responses ? / long-term adaptations ?

Normobaric Hypoxia (NH) vs. Hypobaric Hypoxia (HH)

Recent evidences suggest that the physiological responses to normobaric and hypobaric hypoxia are different.

1. Ventilatory responses
2. Fluids
3. Pre-acclimatization and AMS severity
4. Performance
5. NO metabolism
6. Oxidative stress
7. Postural stability

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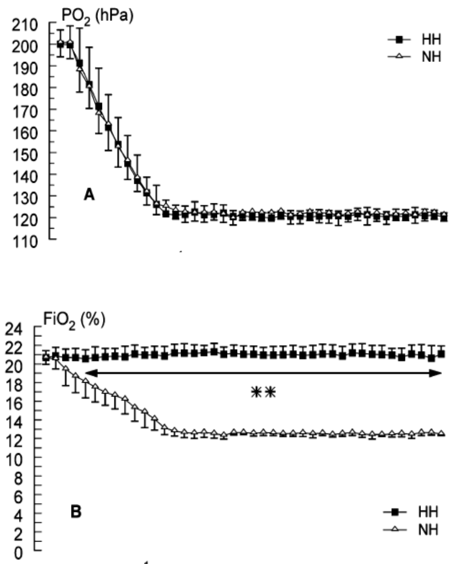
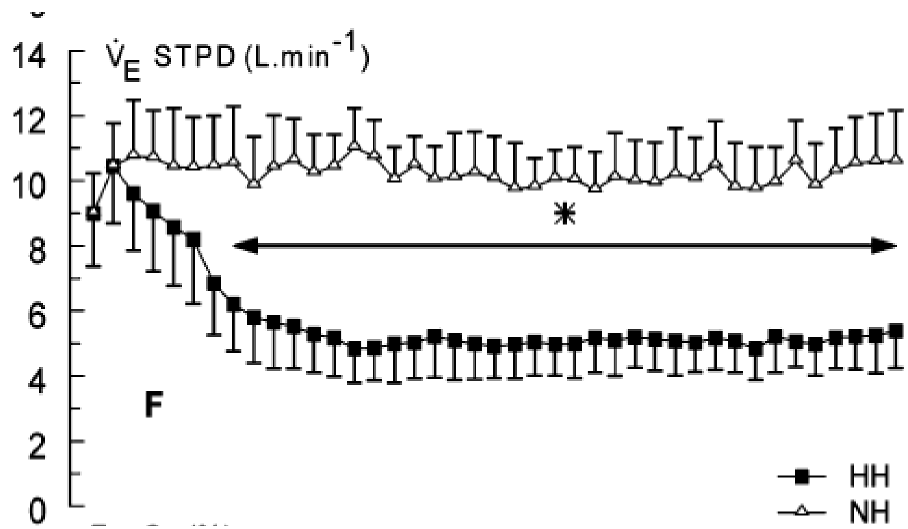
Normobaric Hypoxia (NH) vs. Hypobaric Hypoxia (HH)

1. Ventilatory responses

- Ventilation seems lower in HH than NH (*Loeppky et al. 1997; Savourey et al. 2003*)
- P_AO_2 is slightly higher in NH than HH, especially with hyperventilation.
- Specific adaptation of a lower tidal volume and a higher respiratory frequency.
- Trend to lower $P_{ET}O_2$ and $P_{ET}CO_2$ values in HH.
- Overall, this might come from a alveolar physiological dead space higher in HH.





Rest 40-min HH or NH ; PO2 = 120 hPa (4500 m).
(*Savourey et al. 2003*)

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Normobaric Hypoxia (NH) vs. Hypobaric Hypoxia (HH)

2. Fluids

- PB can modify the fluid circulation (e.g., pulmonary lymph) and the trans- alveoli-capillary membrane flux (*Levine et al. 1988*).
- This might induce a larger pulmonary vasoconstriction in HH and modify the O_2 diffusion by decreasing the pressure gradient.
- PB might influence also the N_2 and O_2 concentration in the cerebrospinal fluid and therefore change partly the central regulation of ventilation (*Conkin and Wessel 2008*).

Normobaric Hypoxia (NH) vs. Hypobaric Hypoxia (HH)

3. Pre-acclimatization and AMS severity

Pre- Acclimatization in HH

3-week of IAE (intermittent altitude exposures : 4 h.day, 5 d.wk, 4300 m).

30 h exposure to 4300 m altitude-equivalent (barometric pressure=446 mmHg)

- The severity of AMS decreased
- Resting $P_{ET}CO_2$ (mmHg) decreased (i.e. increase in ventilation)
- In conclusion… an effective alternative to chronic altitude residence for increasing resting ventilation and reducing the incidence and severity of AMS.

(*Beidleman et al. 2003*)

Pre- Acclimatization in NH

7.5 h each night for 7 nights in NH (F_IO_2: from 16.2 to 14.4%; 3100 m) or « sham »

5 days at HH (4,300 m)

- Only sleep $Sa(O_2)$ was higher and only AMS upon awakening was lower in the NH than sham
- …had no impact on AMS or exercise performance for the remainder of each day.

(Fulco et al. 2011)

Normobaric Hypoxia (NH) vs. Hypobaric Hypoxia (HH)

4. Performance

Meta-analysis show that LHTL in HH (e.g. Levine et al.) induces larger increase in sea-level power output than LHTL in NH (e.g. AIS)

Table II. Meta-analysis of effects on sea-level mean power output following adaptation to hypoxia experienced in studies with various protocols of natural and artificial altitude. Effects of mean and enhanced protocols are those predicted for controlled trials and maximal tests. Effects in parentheses are unclear (>5% chance of enhancement and >5% chance of impairment); otherwise **bold** indicates ≥50% chance of enhancement, *italic* indicates ≥50% chance of impairment, and plain font indicates ≥50% chance of trivial effect. These probabilistic outcomes are computed with reference to a smallest important change of 1%

Effect	Natural altitude protocols		Artificial altitude protocols			
	live-high train-high	live-high train-low	live-high 8–18 h/d, continuous, train-low	live-high 1.5–5 h/d, continuous, train-low	live-high <1.5 h/d, intermittent, train-low	live-low train-high 0.5–2 h/d
Effect of mean protocol[a] (%); ±90% CL[b]						
Elite	(1.6; ±2.7)	**4.0; ±3.7**	(0.6; ±2.0)		(0.2; ±1.8)	
Subelite	(0.9; ±3.4)	**4.2; ±2.9**	**1.4; ±2.0**	(0.7; ±2.5)	**2.6; ±1.2**	(0.9; ±2.4)
Effect of enhanced protocol[c] (%); ±90% CL						
Elite	**5.2; ±4.1**	**4.3; ±4.1**	(4.0; ±5.5)		(1.2; ±2.5)	
Subelite	**4.5; ±4.1**	**4.6; ±3.3**	**4.8; ±5.3**	**3.5; ±3.5**	**3.6; ±2.1**	**6.8; ±4.9**
Study characteristics changed by +1 SD or −1 SD for enhanced protocol	+ Altitude − Days exposure + Test day	− Altitude − Test day	+ Altitude + Hours hypoxia − Days exposure	− Altitude − Test day	+ Exposure ratio − Test day	− Altitude − Train intensity + Days exposure + Test day

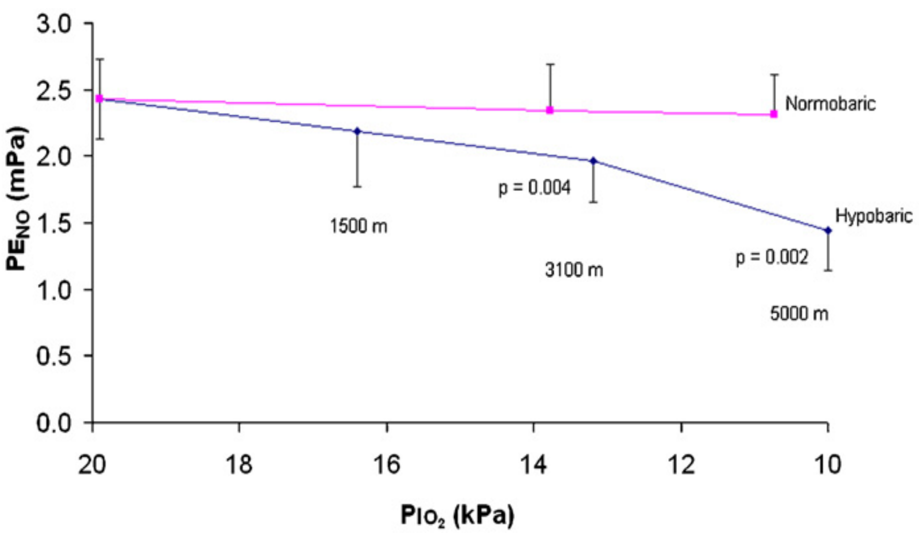
(Bonetti and Hopkins 2009)

Normobaric Hypoxia (NH) vs. Hypobaric Hypoxia (HH)

5. NO metabolism

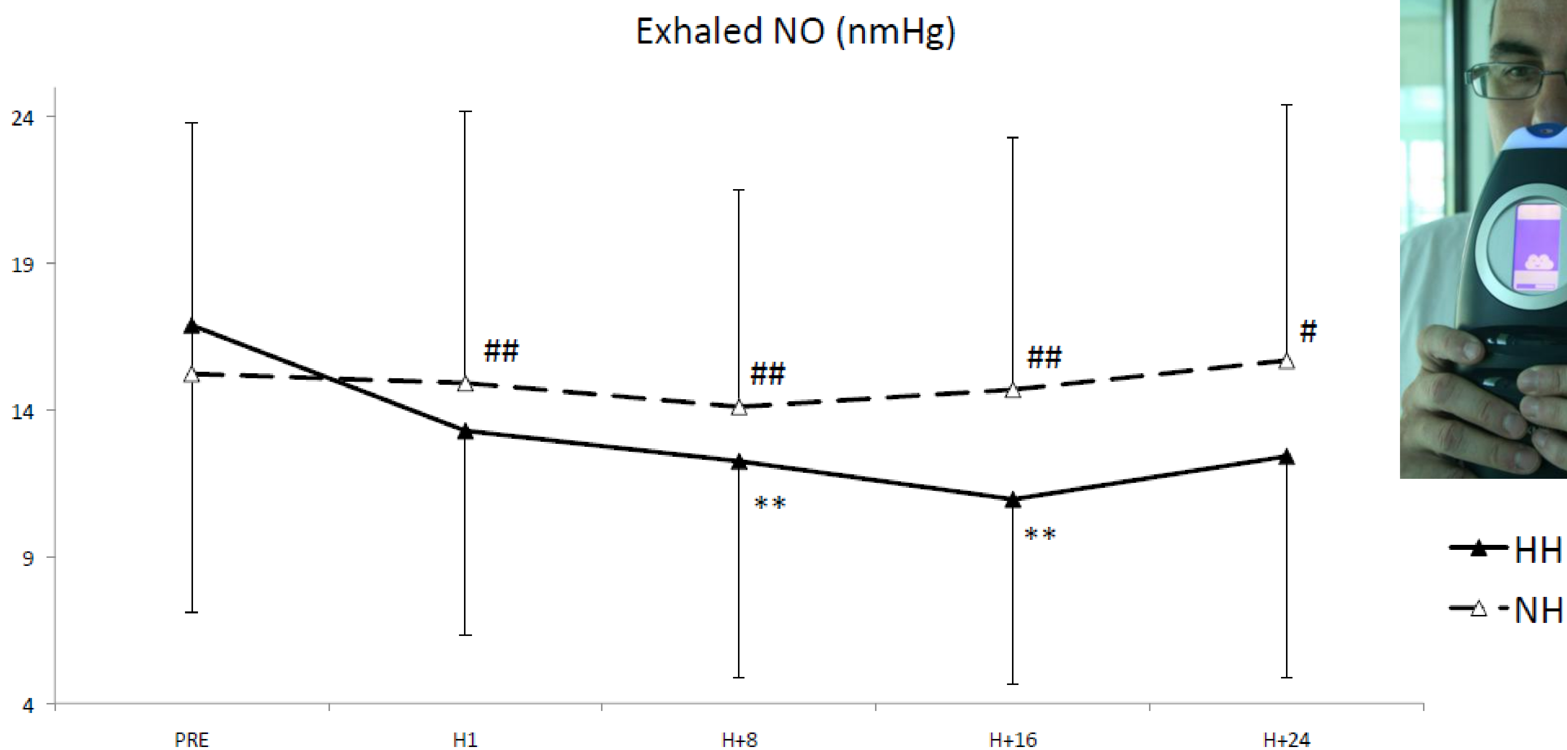
- NO exhaled is lower in HH than in NH (*Hemmingsson and Linnarsson 2009*).
- This would come from a higher diffusion from the upper tracts to the alveoli then to hemoglobin in HH.
- The level of NO recapture by the blood compartment would be higher in HH.
- It is unclear how and if this mechanism is related to the higher benefits to train in « real altitude » than in « simulated altitude » (*Kayser 2009*).

- Relationships between decrease in NO, pulmonary arterial pressure and the pulmonary oedema risk (*Busch et al. 2001; Duplain et al. 2000*).
- The decrease in exhaled NO comes from the epithelial cells of the respiratory tractus.



(Hemmingsson & Linnarsson, 2009)

Hypoxie Normobarique vs. Hypoxie Hypobarique



** p<0.01 for difference with PRE
p<0.01 for difference with HH
# p<0.05 for difference with HH

Different changes in oxidative stress and NO biodisponibility during 24 hours in hypobaric vs normobaric hypoxia

Faiss, R

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6. Oxidative stress

- In our preliminary study (*Faiss et al. 2011*), we show that some markers of the oxidative stress increased more in HH and in NH.
- AOPP increased more ($p<0.05$) in HH (+120%, +116% and +260% after 1h, 16h and 24h) than in NH (+13%, +23% and +88%, after 1h, 16h and 24h).
- SOD increased in HH (+42% and +26% after 16h and 24h) but remained stable in NH

24-h HH or NH (3000 m).

(*Faiss et al. 2011*)

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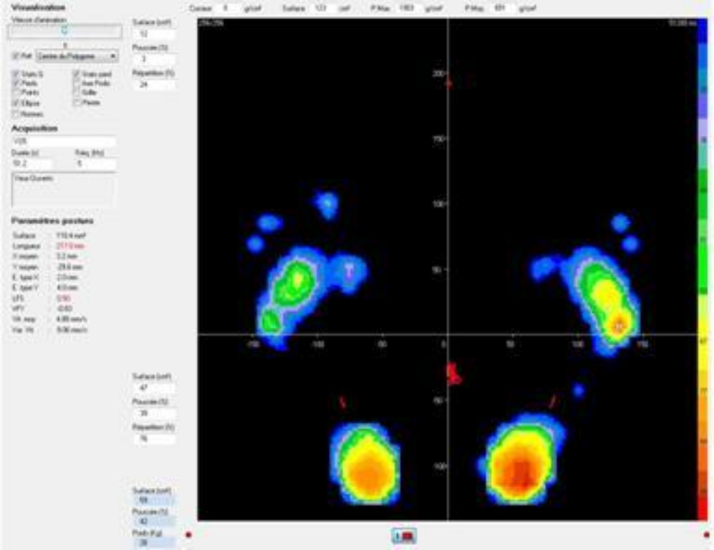
Normobaric Hypoxia (NH) vs. Hypobaric Hypoxia (HH)

7. Postural stability

- HH deteriorated postural stability in the antero-posterior plane, for the variance of speed and the surface of CoP, whereas no difference was observed between NH and NN. **..**
- **…suggest that hypobaria instead of hypoxia *per se* plays an important contribution to the altered balance classically reported in altitude**



NN or NH or HH (1700 and 3000 m).

(*Degache et al. High Alt Med Biol: in press*)

Hypoxie Normobarique vs. Hypoxie Hypobarique

L'amélioration des performances serait plus importante suite une préparation de type LHTL avec des expositions en altitude réelle qu'en altitude simulée *(Bonetti and Hopkins 2009)*

Les différences proviennent essentiellement de différences d'ordre hémodynamiques (systémique et pulmonaire) impliquant le monoxyde d'azote et régulé par le stress oxidatif

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Normobaric Hypoxia (NH) vs. Hypobaric Hypoxia (HH)

Current investigation

LHTL (18-d ; 2500 – 1200)

HH (Leukerbad, Switzerland)

NH (Prémanon, France)

Présentation des outils et méthodes expérimentales

1. **Déroulement de l'étude:**

Entrainement de type LHTL sur une période de 18 jours

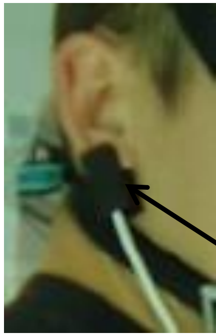
- En hypoxie normobarique (Prémanon, LH 2500m, TL 1200m)
- En hypoxie hypobarique (plusieurs possibilités en suisse)

Plan expérimental en cross-over

Tests effectués en conditions Pre-, Post + 7 jours et post + 21 jours

2. Tests effectués:

- Performance et échanges gazeux
- Paramètres hématologique du métabolisme de l'O_2 et du fer
- Oxygénation musculaire à l'exercice
- Stress oxydatif
- Métabolisme du NO
- Sensibilité du baroréflexe
- Variabilité cardiaque

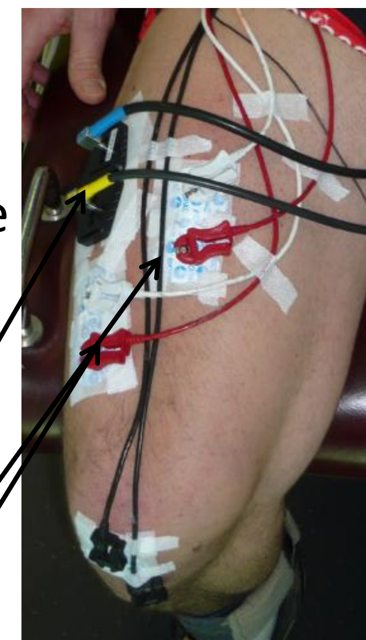


Measurements

- Power output (W)
- Heart rate (bpm)
- Pulse oxygen saturation (SpO_2)

- Oxygen uptake (VO_2)
- Ventilation (VE)
- CO_2 production (VCO_2)
- Expiratory end-tidal O_2-partial pressure ($PetO_2$)
- Expiratory end-tidal CO_2-partial pressure ($PetCO_2$)
- Breathing frequency (BF)

- Blood lactate
- Muscle activity EMG (RMS for VL, RF)
- Muscle oxygenation (NIRS)



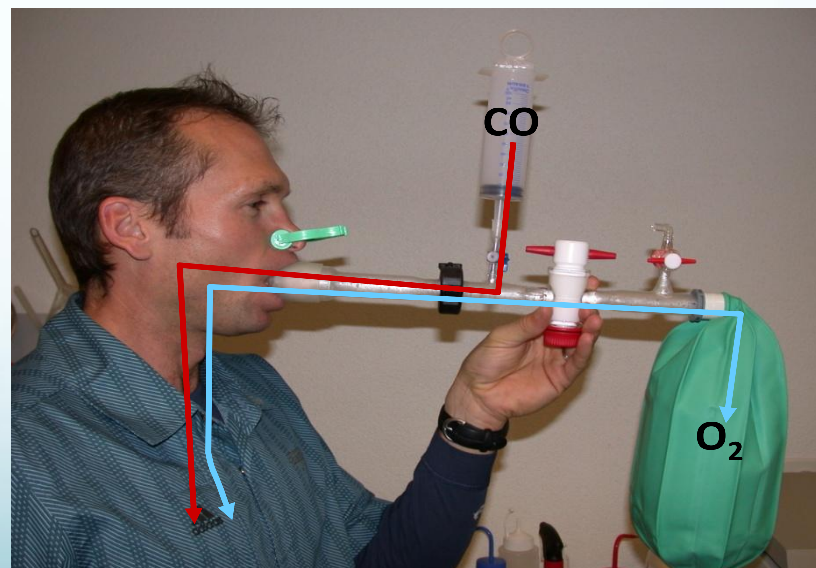
3. Certains outils et méthodes:

- **Le CO rebreathing method**: méthode permettant de calculer la masse totale en hémoglobine.
  - Fogh-Andersen et al. (1990) and Thomsen et al. (1991)
  - “Optimized method” by Schmidt and Prommer (2005)
  - with minor modifications (Prommer & Schmidt 2007)
  - Steiner and Werhlin (2011, comparability of methods)

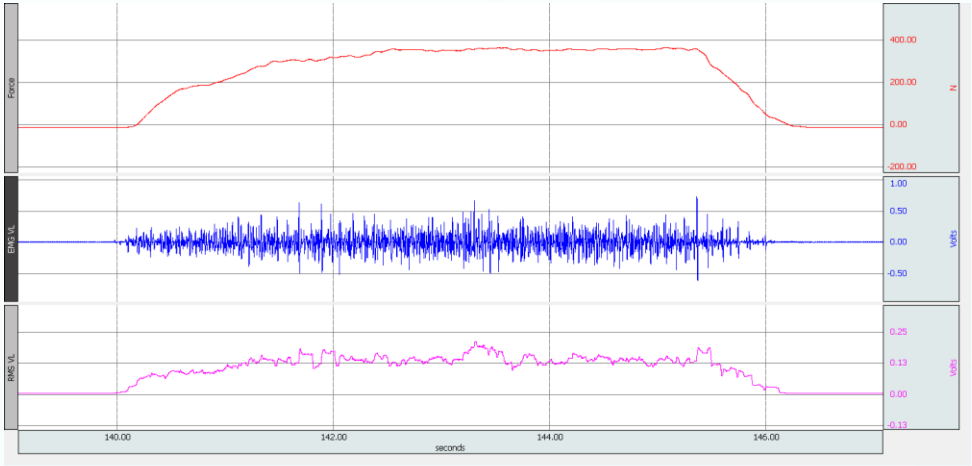
méthode originale (>10 minutes) vs. nouvelle méthode (2 minutes)

Principe de base:

1. Analyse des valeurs de HbCO et sO_2 sanguin
2. Analyse de [CO] endothéliale
3. Inhalation d'un mélange CO et O_2 (respectivement 100 ml et 3.5 l)
4. Respiration en circuit fermé pendant 1'50
5. H +2' nouvelle analyse de [CO] endothélial
6. H +6 et H +8 nouvelles analyse de HbCO et sO_2 sanguin
7. Calcul de la masse totale en hémoglobine

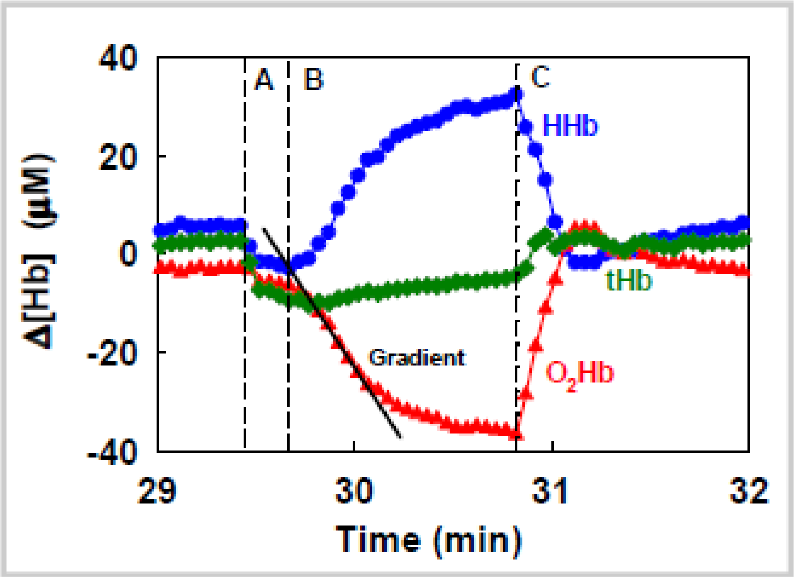


- **L’électromyographie (EMG)**: permet de mesurer l’activité électrique accompagnant l’activation des muscles.



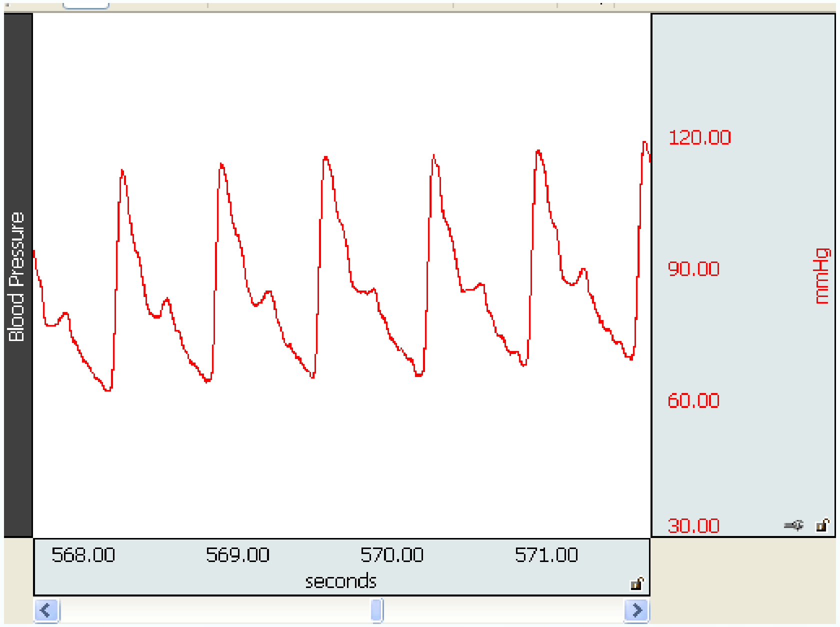
Principe de base: mesure d’une différence de potentiel électrique entre deux points à l’aide d’électrodes positionnées en surface du muscle.

- **La NIRS (Near Infrared Spectroscopy)**: mesure de l'oxygénation des muscles par spectroscopie électromagnétique.



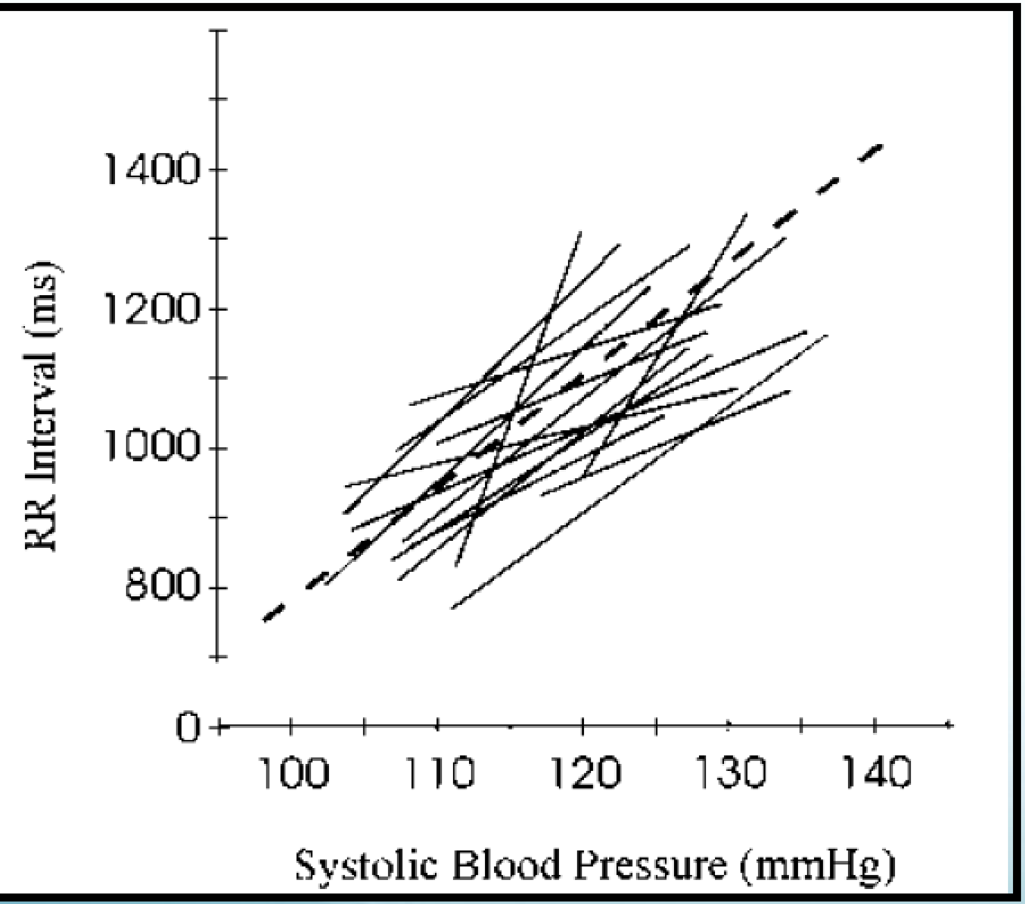
Example of measurement on muscle, where an exercise period (A) is followed by an occlusion (B-C) from which the local oxygen consumption (mVO_2) is determined from the gradient of the oxyhemoglobin (O_2Hb) signal.

- **Baroréflexe**



Sensibilité du baroréflexe

Méthode séquentielle



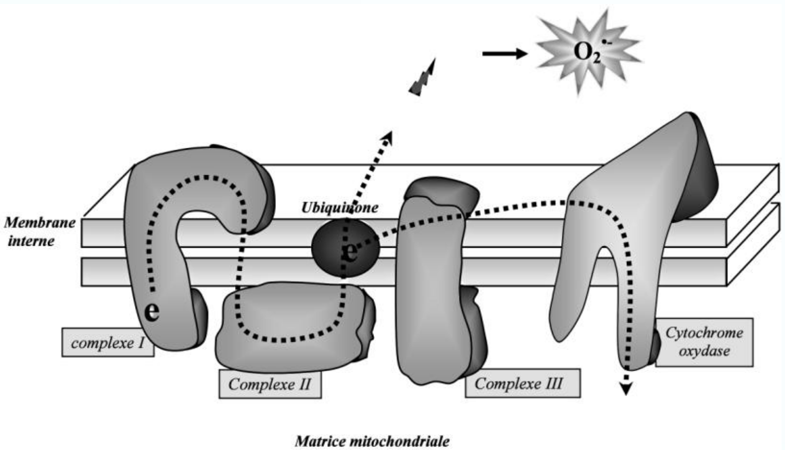
RR et SBP augmentent ou diminuent concomitamment pendant au minimum 3 bpm.

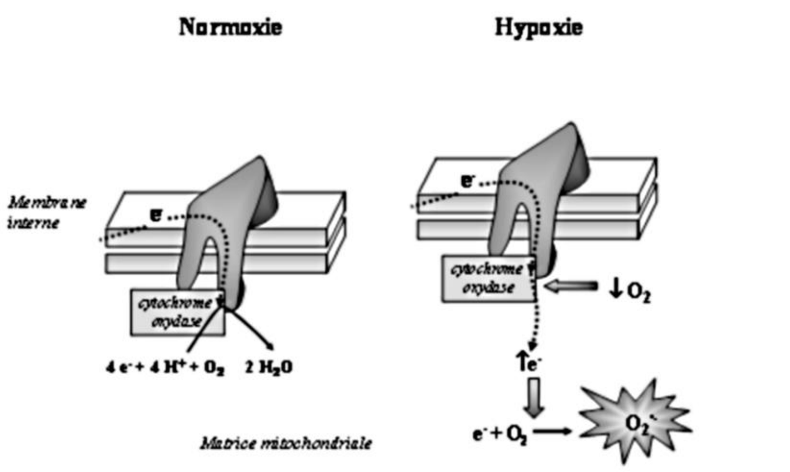
Changement > 1 mm Hg pour SBP et 4 ms pour RR.

Corrélation linéaire entre RR et SBP pour chaque séquence; si r>=0.80, calcul de la régression linéaire = BRS (ms/mm Hg).

Circulation. 1998;97:1362-1367

- **Stress oxydatif**





	Antioxydant	Action
Enzymes	SOD	Dismute $O_2^{\bullet-}$ en H_2O_2
	Catalase	Transforme H_2O_2 en H_2O et O_2
	GPX	Réduit H_2O_2 et ROOH
	Glutathion réductase	Recycle GSSG en GSH
Non-enzymatiques	Acide urique	Réduit $OH^{\bullet}$
	Glutathion	Réduit H_2O_2 et ROOH Recycle la vitamine C
	Vitamine C	Réduit $O_2^{\bullet-}$ et $OH^{\bullet}$ Recycle la vitamine E
	Vitamine E	Neutralise $ROO^{\bullet}$ et $RO^{\bullet}$ Limite l'action de la phospholypase A2
	Flavonoïde	Identique à la vitamine E
	β-carotène	Piège $^1O_2$ et peut réduire $ROO^{\bullet}$
	Lycopène	Piège $^1O_2$ et peut réduire $ROO^{\bullet}$
	Co-enzyme Q10	Réduit Fe^{3+} - $O2^{\bullet-}$

Les divers lieux

Gemmi (2400m)

Prémanon

Torrent (2350m)