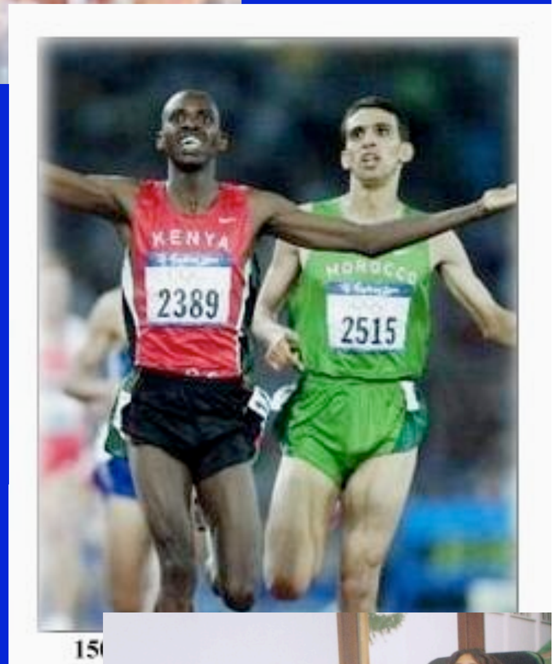
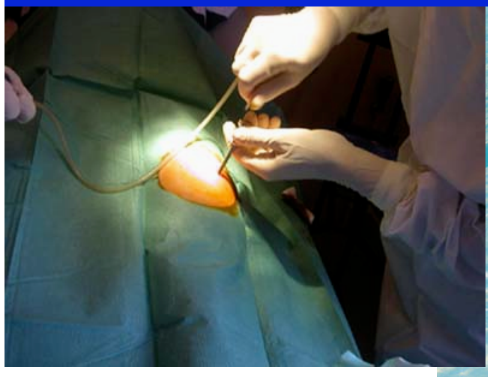
Altitude training: new perspectives



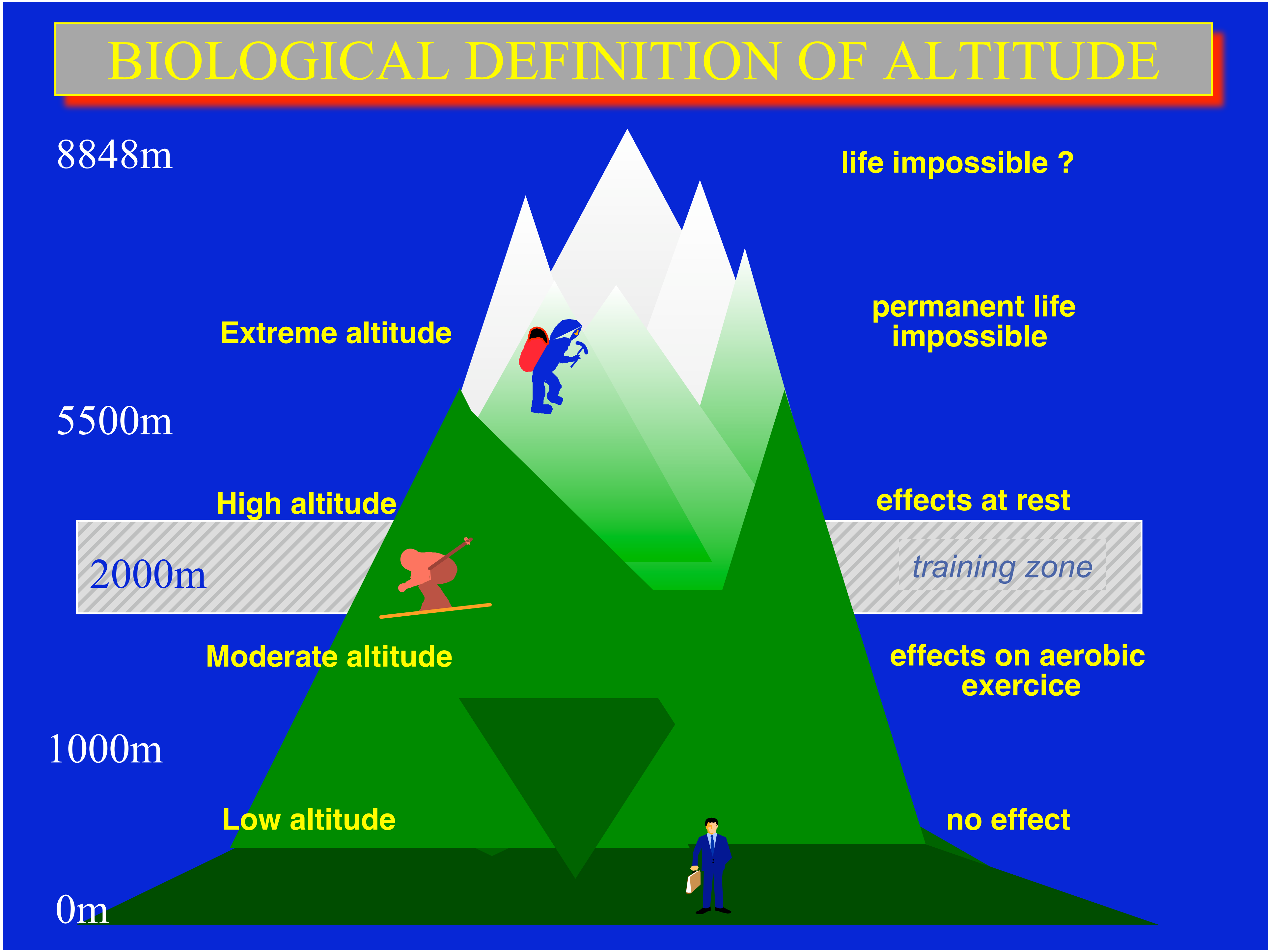
Prof. Jean-Paul Richalet

Laboratoire “Réponses cellulaires et fonctionnelles à l’hypoxie”



DUOBA, August 2009

Université Paris 13 - FRANCE



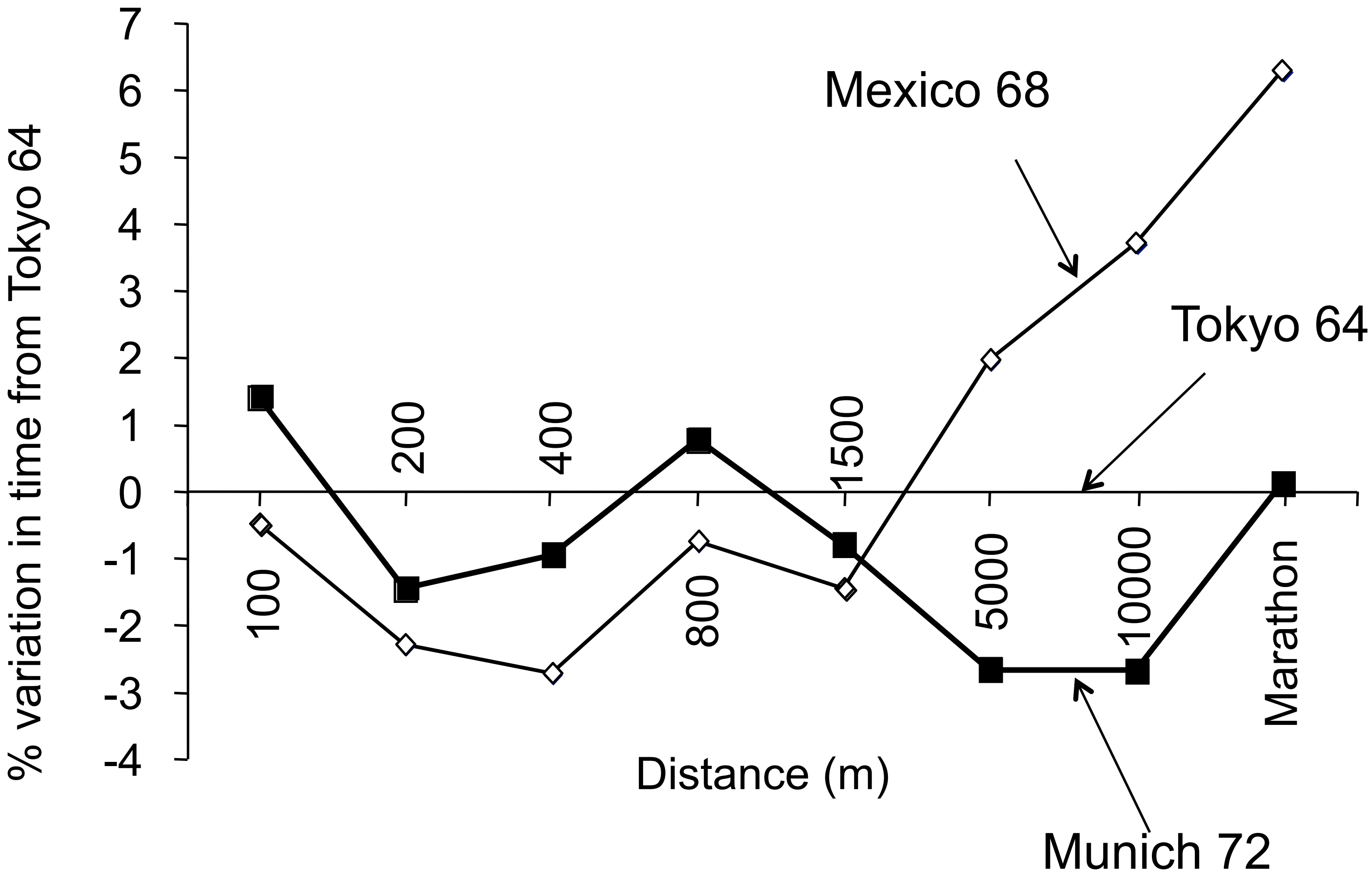
BIOLOGICAL DEFINITION OF ALTITUDE
8848m
life impossible ?
Extreme altitude
permanent life impossible
5500m
High altitude
effects at rest
2000m
training zone
Moderate altitude
effects on aerobic exercice
1000m
Low altitude
no effect
0m

Olympic games and high altitude: MEXICO, 1968

Bob Beamon, long jump: 8.90 m,
world record for 22 years

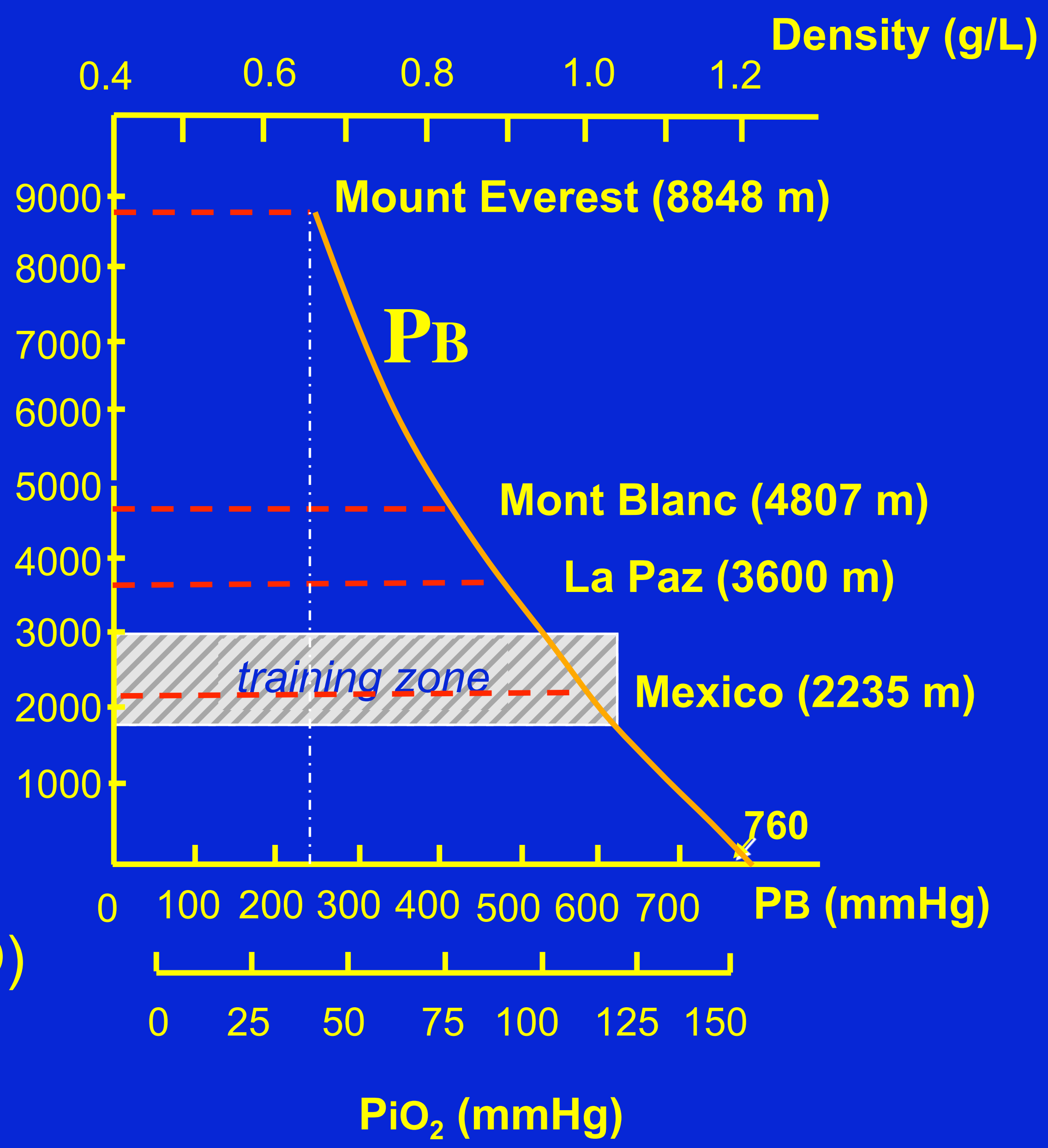
Ron Clarke, 10000m:
exhaustion at the end
of the race

Athletic results in Olympic games ‘64, ‘68 and ‘72



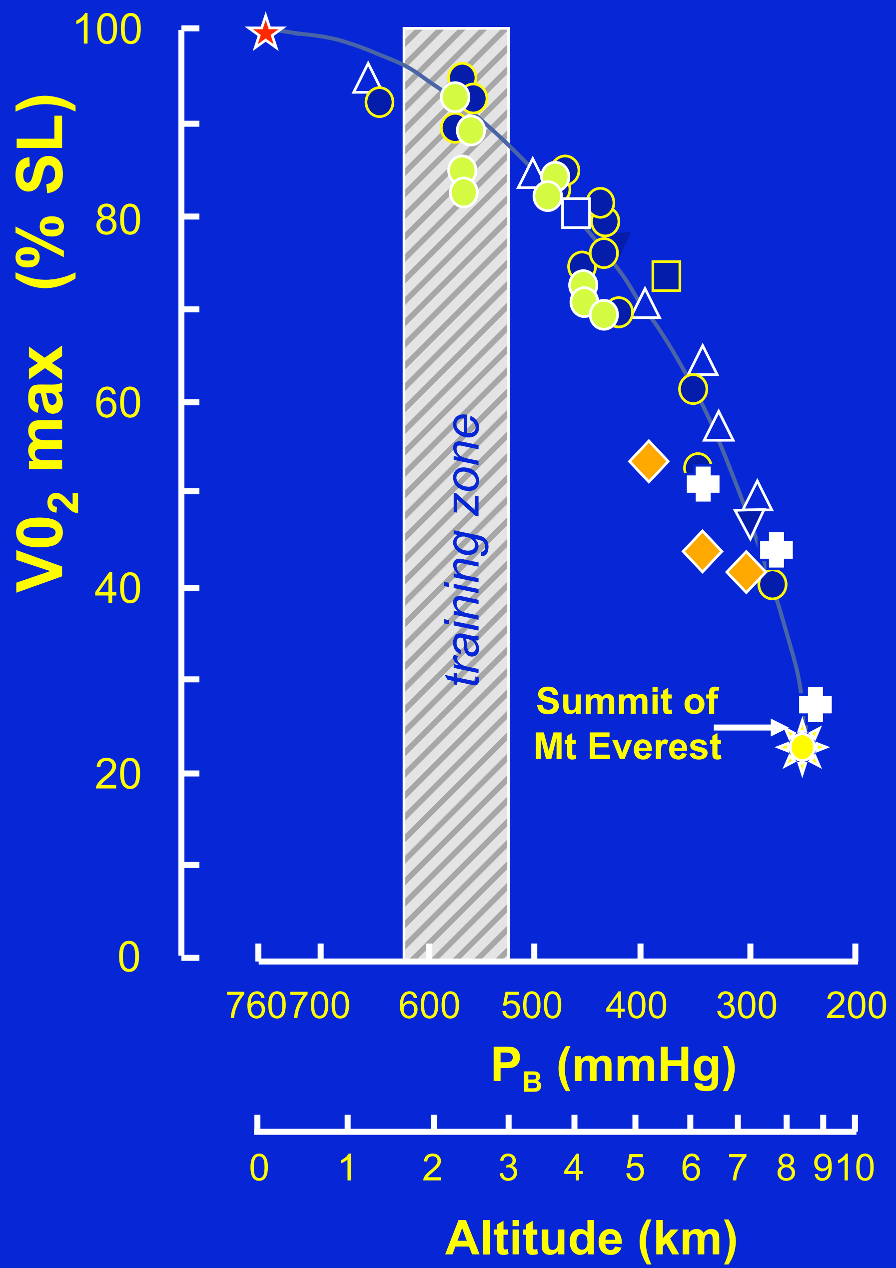
High altitude marathon - Cerro de Pasco - Peru: 4300 m !!

Decrease in barometric pressure and air density with altitude



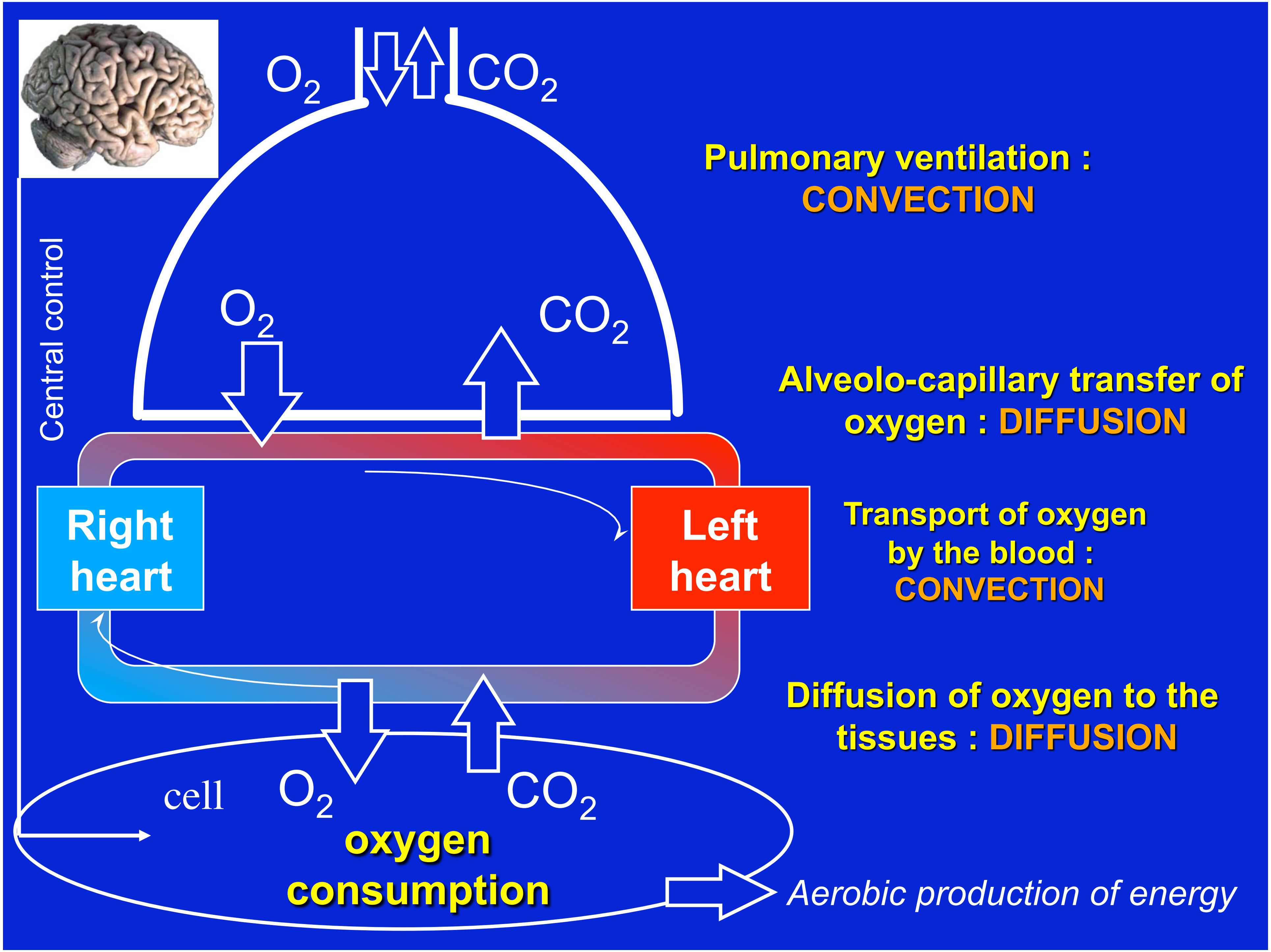
HYPOXIA =
lack of oxygen

$PiO_2 = FiO_2 \times (P_B - PH_2O)$



Maximal aerobic power (VO_2max) decreases with altitude

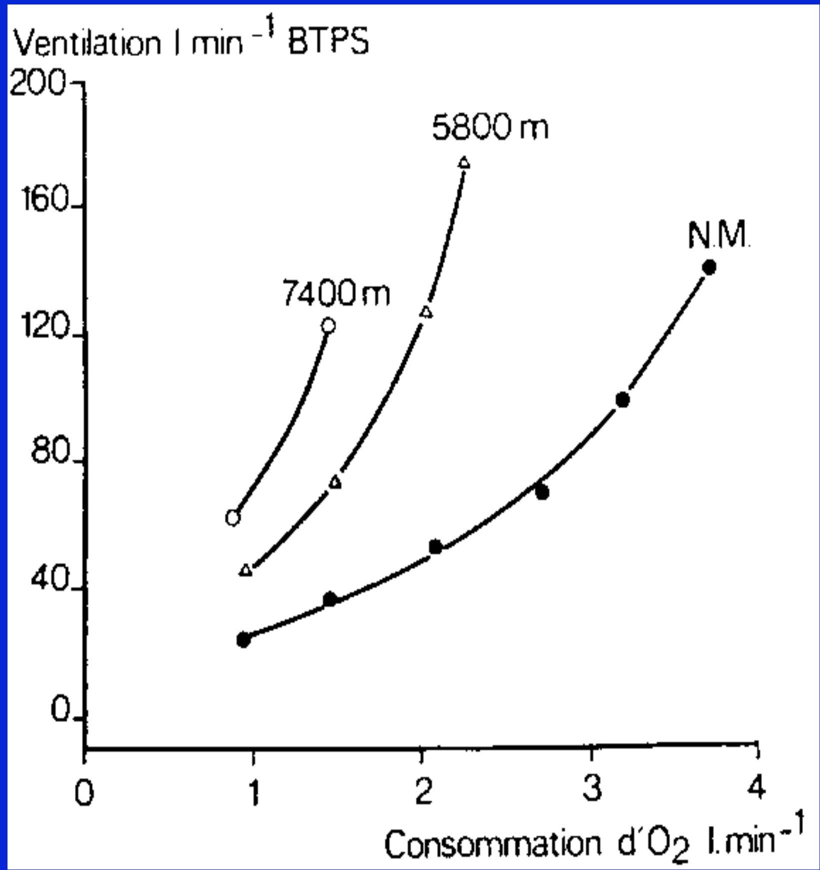
What are the limiting factors of performance ?



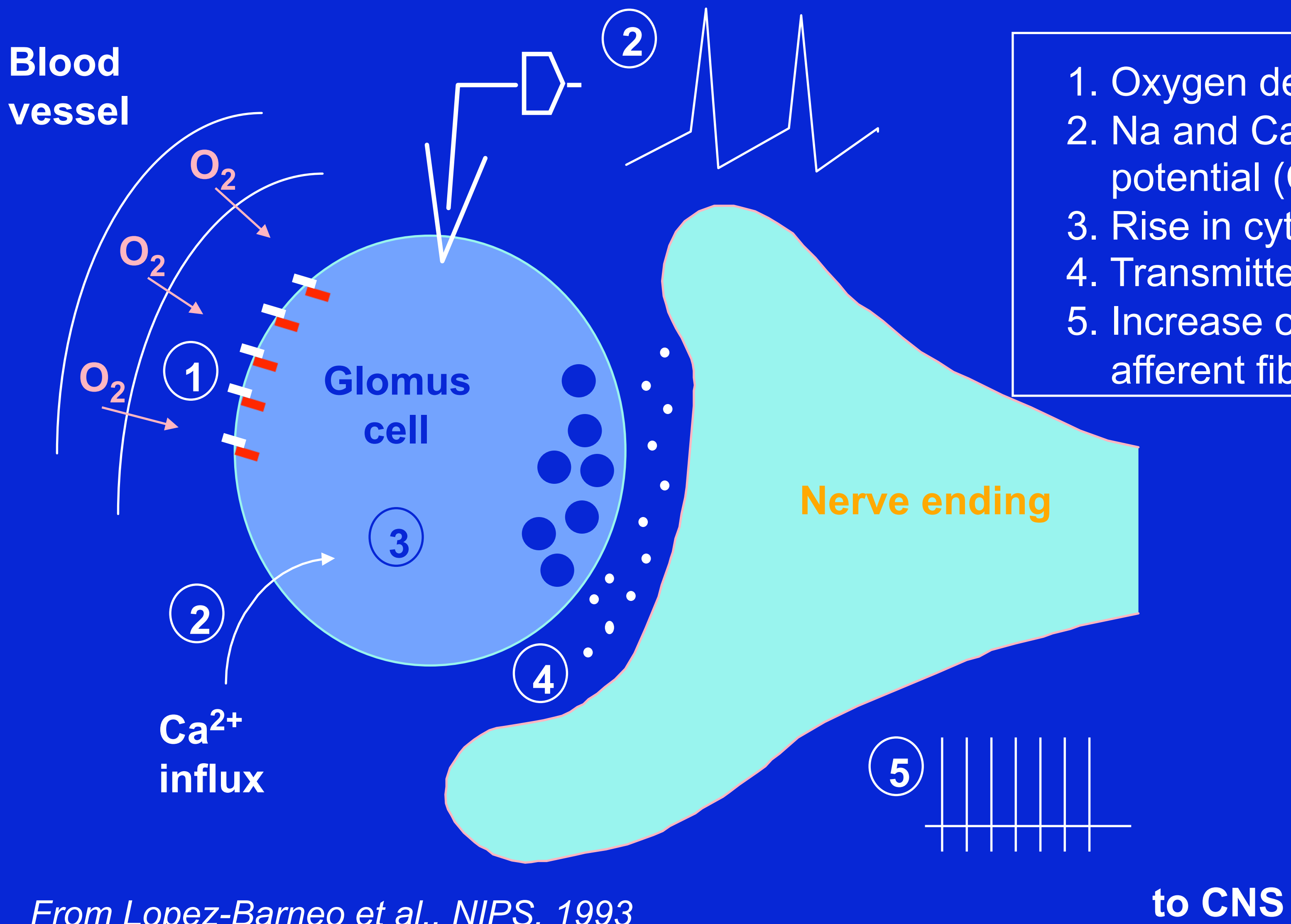
Central control
O_2
CO_2
Pulmonary ventilation :
CONVECTION
O_2
CO_2
Alveolo-capillary transfer of
oxygen : DIFFUSION
Right
heart
Left
heart
Transport of oxygen
by the blood :
CONVECTION
Diffusion of oxygen to the
tissues : DIFFUSION
cell
O_2
CO_2
oxygen
consumption
Aerobic production of energy

Chemoreceptors and acclimatization:

Ventilation increases
at rest and
at each level
of exercise



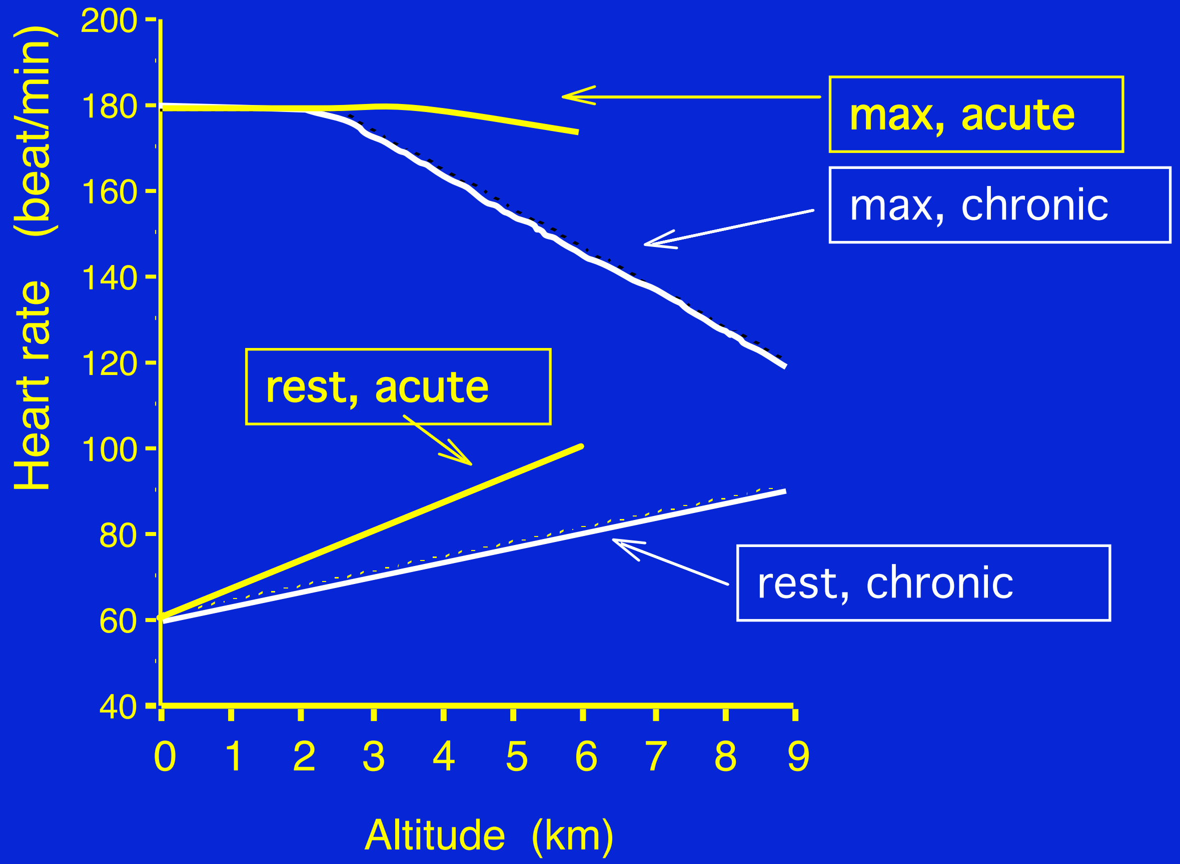
Carotid chemoreceptors : hypoxic sensors

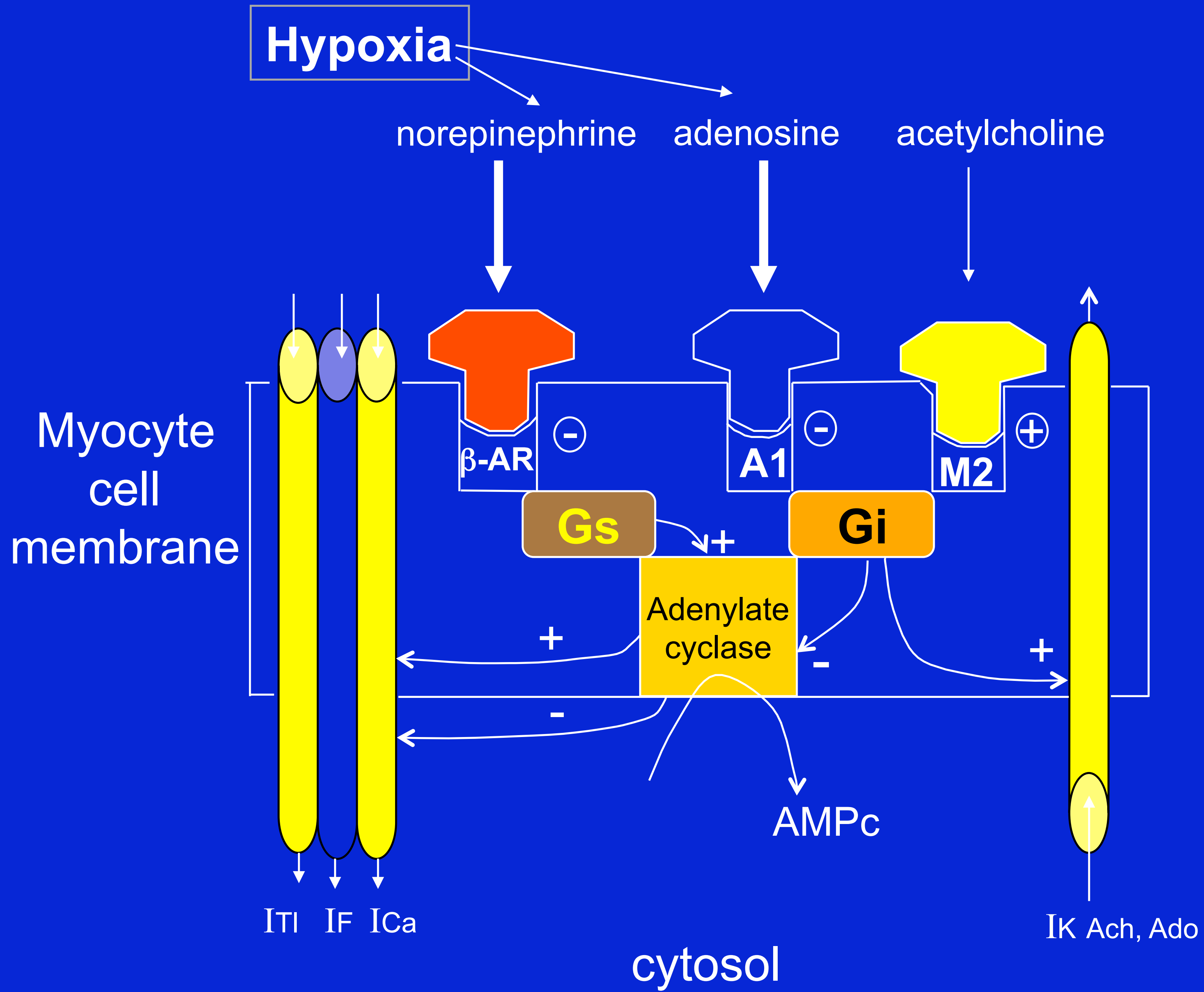


1. Oxygen detection
2. Na and Ca action potential (Ca influx)
3. Rise in cytosolic Ca
4. Transmitter release
5. Increase of firing in afferent fibers

From Lopez-Barneo et al., NIPS, 1993

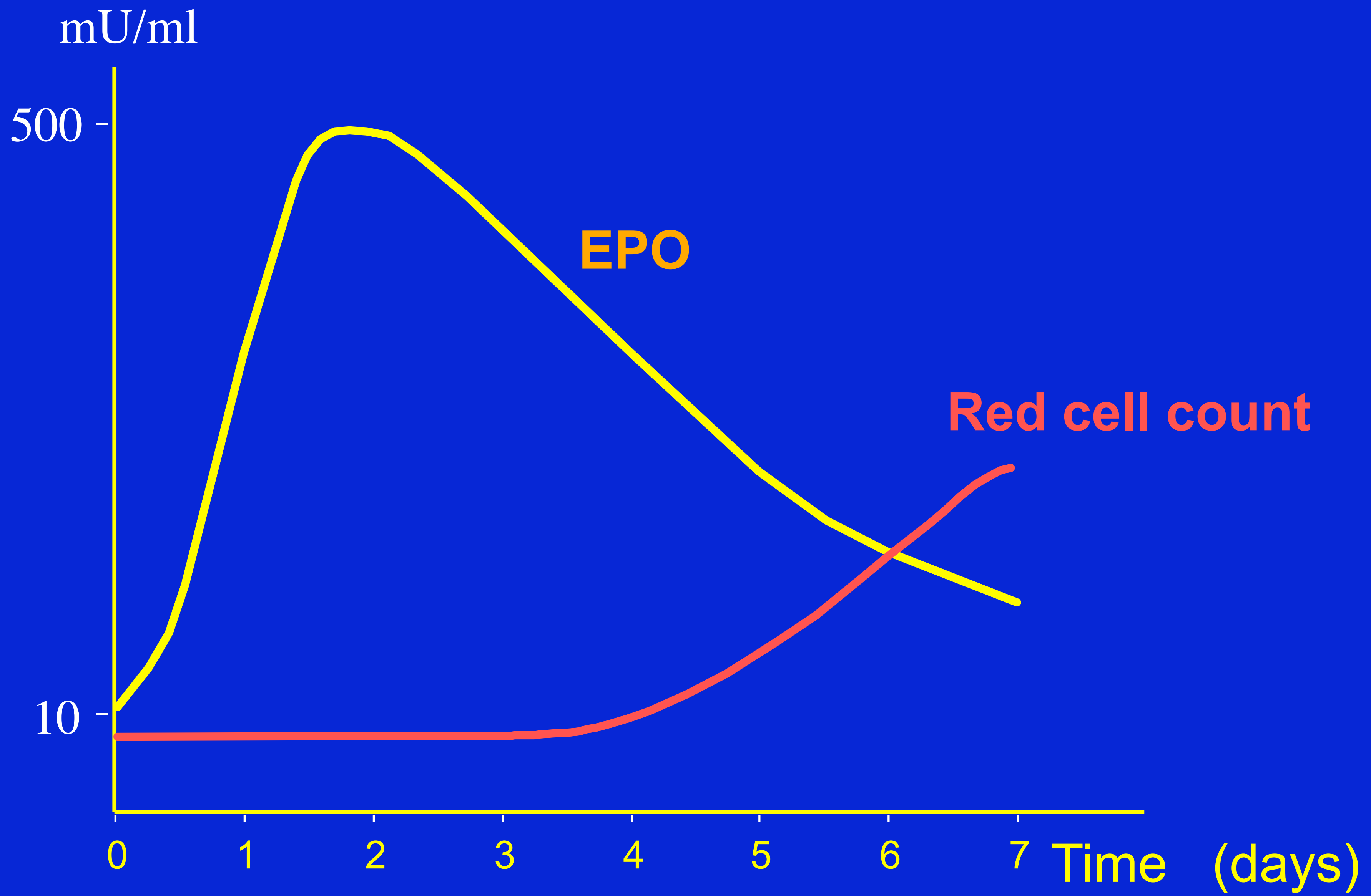
Adaptation of heart rate in acute and chronic hypoxia

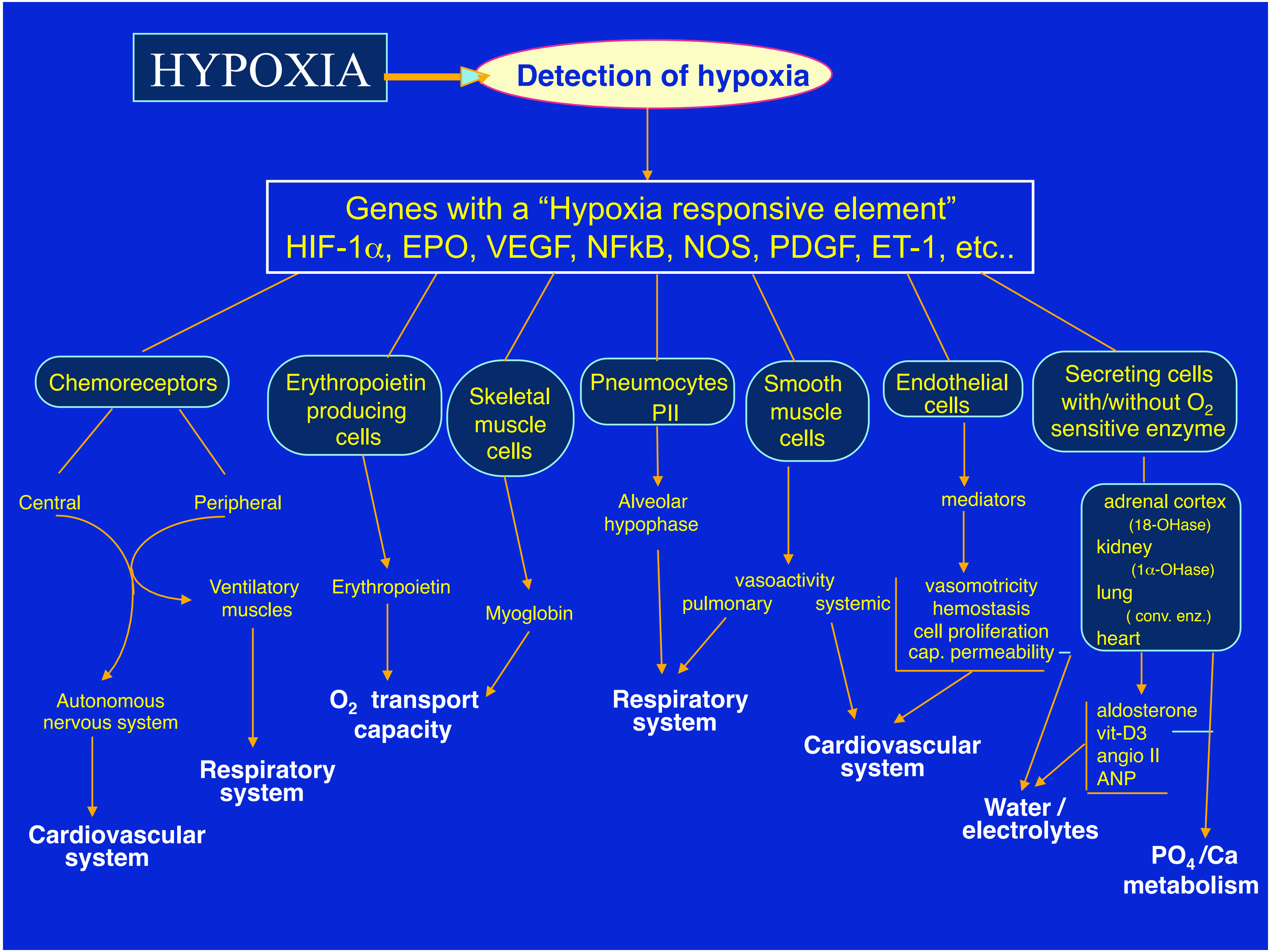




From: Lerman and Belardinelli, Circulation, 1991 and Richalet et al. 1990

Serum EPO and Red cell count during a stay at 4350 m




HYPOXIA
Detection of hypoxia
Genes with a "Hypoxia responsive element"
HIF-1α, EPO, VEGF, NFkB, NOS, PDGF, ET-1, etc..
Chemoreceptors
Erythropoietin producing cells
Skeletal muscle cells
Pneumocytes PII
Smooth muscle cells
Endothelial cells
Secreting cells with/without O_2 sensitive enzyme
Central
Peripheral
Ventilatory muscles
Autonomous nervous system
Cardiovascular system
Respiratory system
Erythropoietin
O_2 transport capacity
Myoglobin
Alveolar hypophase
Respiratory system
vasoactivity
pulmonary
systemic
Cardiovascular system
mediators
vasomotricity
hemostasis
cell proliferation
cap. permeability
adrenal cortex
(18-OHase)
kidney
(1α-OHase)
lung
(conv. enz.)
heart
aldosterone
vit-D3
angio II
ANP
Water / electrolytes
PO_4/Ca metabolism

Why training in hypoxia ?

HYPOXIA

Stimulates erythropoiesis and increases blood oxygen transport capacity

Activates the autonomous system nervous and induces vasculogenesis

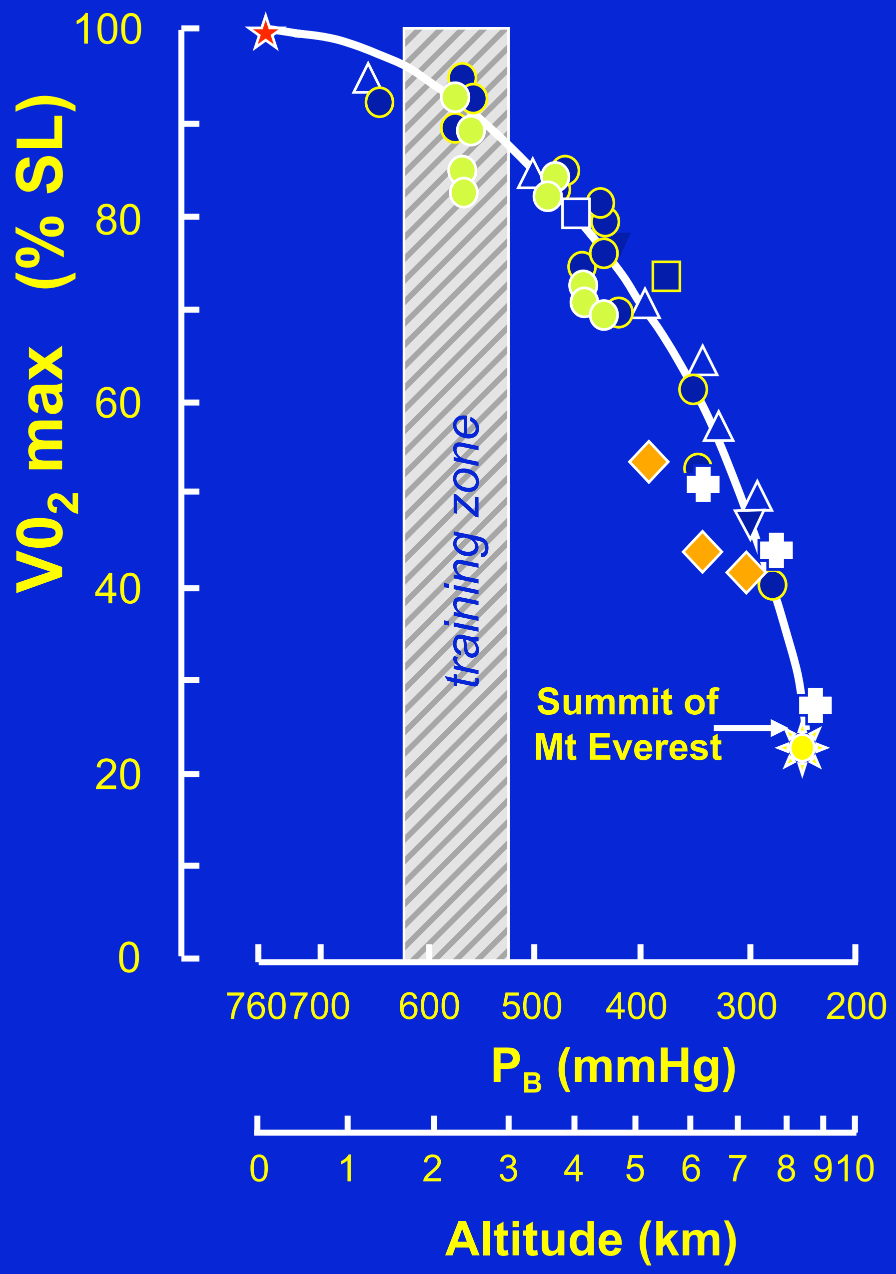
Stimulates the respiratory centres and increases pulmonary ventilation

Modifies muscular metabolism

But…

- The hypoxic environment needs an acclimatization period (because of AMS)

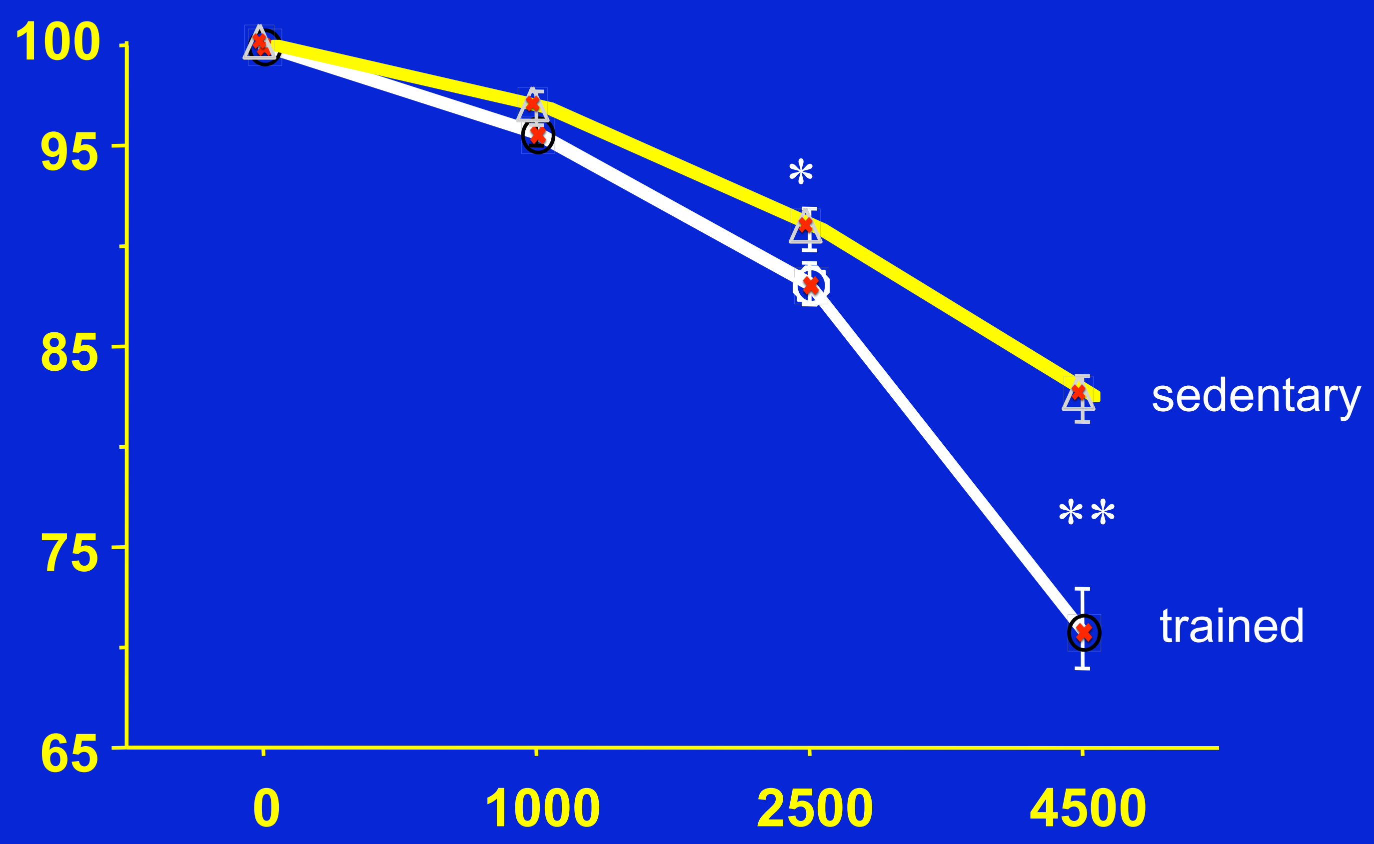
- Hypoxia induces a decrease in maximal aerobic performance :
  - 5% at 2000m
  - 15% at 3000m
  - 75% at 8848m !



Maximal aerobic power (VO_2max) decreases with altitude:

training load must be lower when training at high altitude

VO$_2$max (% of sea level)

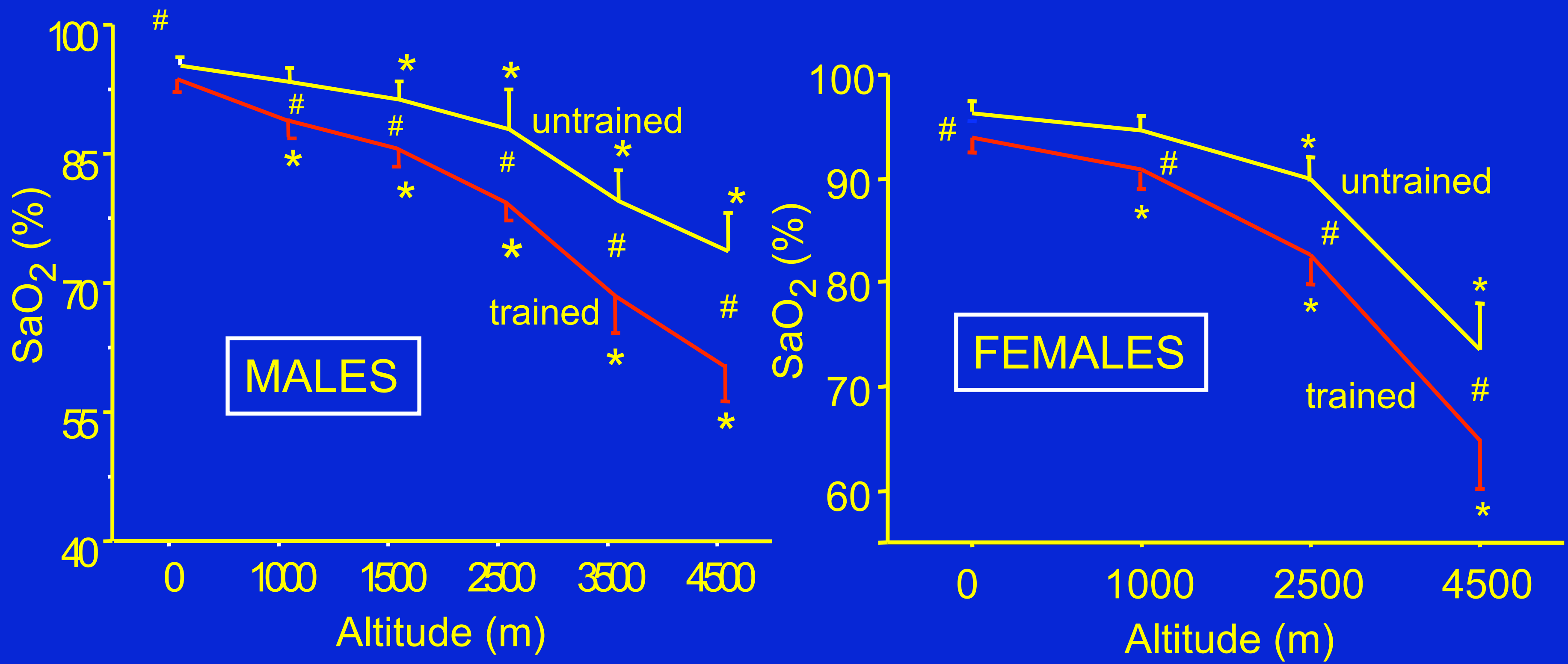


Altitude (m)

The decrease in trained subjects is greater than in sedentary subjects

From Mollard et al., 2007

SaO_2 at maximal exercise

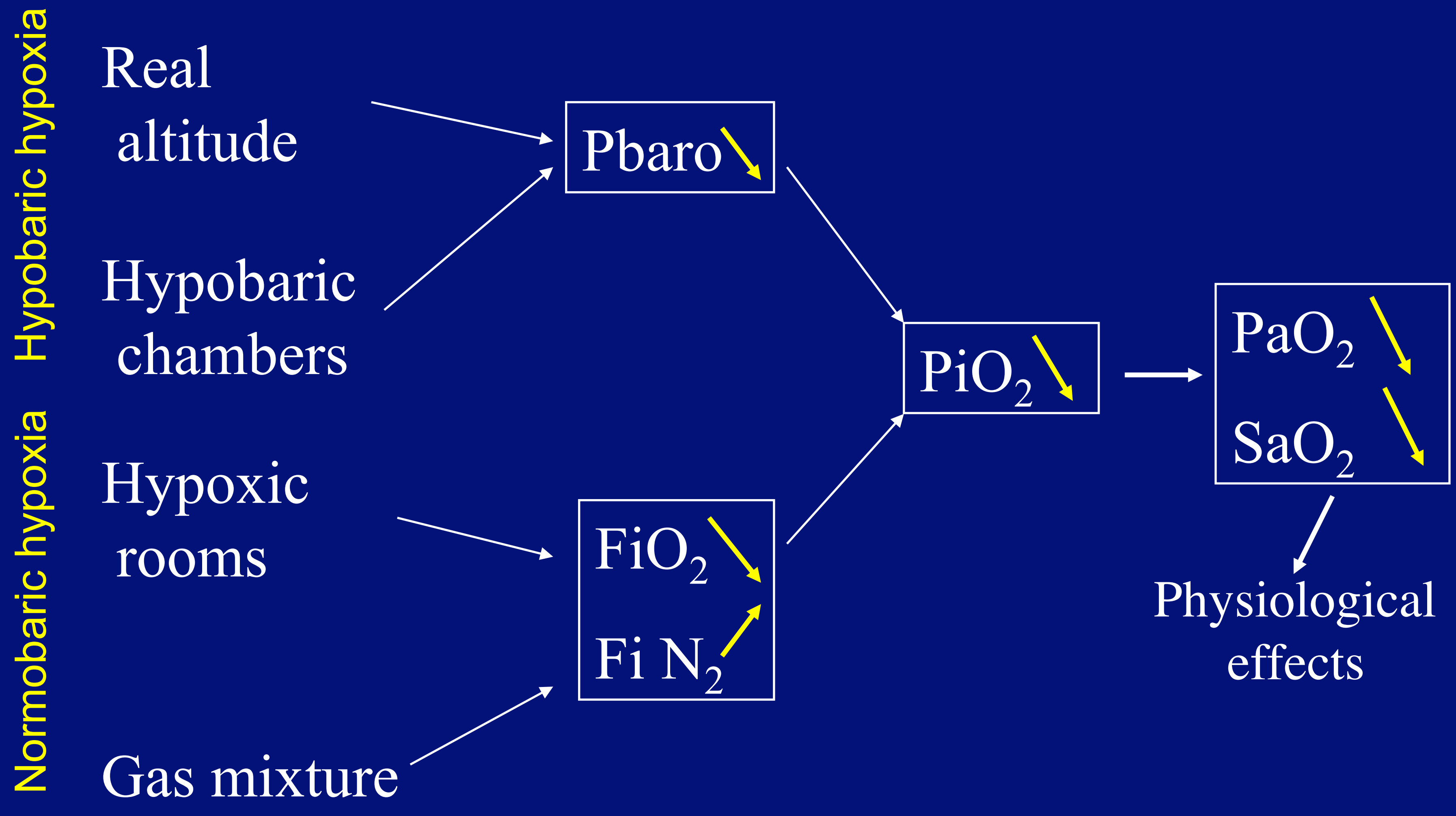


* hypoxia vs normoxia   # trained vs untrained

Trained subjects show a greater desaturation at exercise in acute hypoxia

Mollard et al., 2006; 2007; Woorons et al., 2005

How to get a hypoxic environment ?



$$PiO_2 = FiO_2 \text{ x } (\text{Pbaro} - P_{H_2O})$$

Hypoxic training procedures

I. Live high – train high (1800-2500m) :

Altitude training centers

Font-Romeu (France), Belmekem (Bulgaria),

Colorado (USA), Duoba (Qinghai, China)

Live high - train high (classical altitude training)

Controlled studies, beneficial effects found in only 2 studies, only in well trained but not elite athletes (Mellerowicz et al., 1970; Levine and Stray -Gundersen, 1997)

All other studies with elite athletes did not find a significant difference in SL performance

Beneficial effects found in uncontrolled studies…

… but a great inter-individual variability !

Hypoxic training procedures

II. Live high (sleep and recovery at 2500-3500m), train low (≤1200m)

- real altitude : mountain resort or refuge, teleferic station (Alps), mountain region (Colorado)

- simulated altitude : hypoxic rooms (USA, Europe, Australia, Qatar, Russia, China), individual hypoxic tents (Internet)

Hypoxic training procedures

III. Live low – train high

- true altitude : training in ski resort (glaciers), residence in the valley

- simulated altitude : devices producing hypoxic gas mixtures and intermittent exposure (Hypoxicator®, Altitrainer®, etc..)

Training in hypoxia in the endurance -trained athlete

Effects on performance
Individual response factors
Potential risks for health

International Olympic Committee
Ministère des Sports, France
Groupe français de recherche sur l 'entraînement en hypoxie

COMITE INTERNATIONAL OLYMPIQUE

Main objectives

- Evaluate the physiological changes induced by various modalities of training in hypoxia and their impact on performance.
  - *Hypothesis: these methods improve performance at sea-level*
- Evaluate the individual response to training
  - *Hypothesis: there are biological, physiological or psychological markers of the variability of individual response to training in hypoxia*
- Evaluate the potential risks for health
  - *Hypothesis: these methods are safe at short, medium and long term, provided a medical control of training procedures*

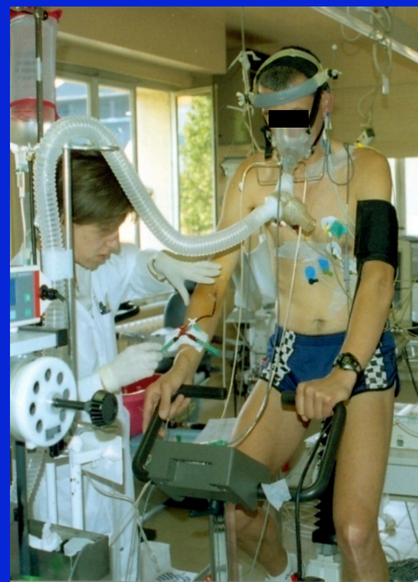
« Training in hypoxia »

1. Effects of training in hypoxia integrated into a normoxic training procedure in elite endurance athletes

(5000m, marathon, triathlon)

« train high and low, live low », using hypoxic gas mixtures

- Service Explorations Fonctionnelles, Hôpital de Strasbourg
- Institut d ’Anatomie, Université de Berne, Suisse
- Laboratoire « Sport Performance Santé », Faculté des Sciences du Sport, EA 2991, Montpellier,
- Pôle France Triathlon, CREPS de Montpellier



« Live high - train low »

2. Effects of intermittent exposure to hypoxia coupled to training at low altitude on performance in elite endurance athletes

(nordic ski, swimming, track and field, using hypoxic rooms)

- Ecole Nationale de Ski de fond, Prémanon
- Ecole Nationale de Ski et d 'Alpinisme, Chamonix
- Laboratoire « Réponses cellulaires et fonctionnelles à l 'hypoxie » EA2363, Paris 13, Bobigny
- Laboratoire national de dépistage du dopage, Chatenay-Malabry
- Laboratoire de psychologie appliquée, Reims

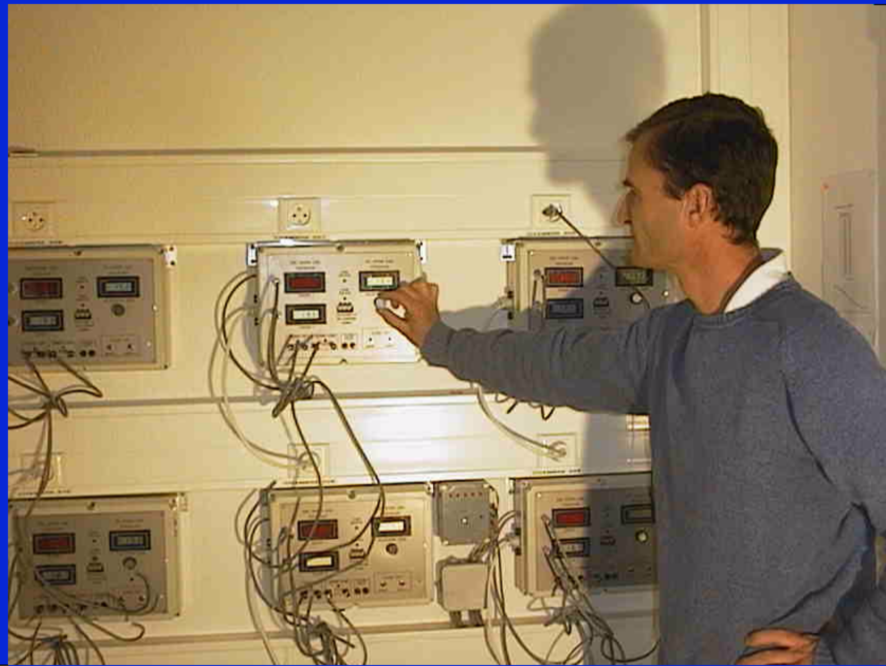
Prémanon, Jura, France

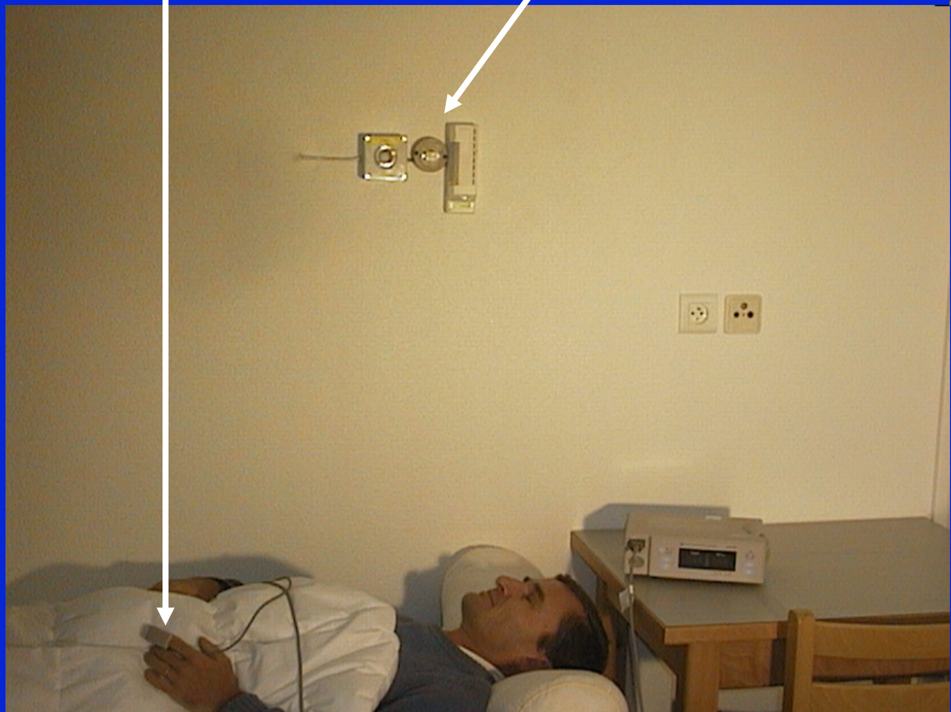

Centre National de Ski Nordique
Prémanon

HYPOXIC ROOM

Control of arterial oxygen saturation

Control of ambient O_2 and CO_2





CONTROL CENTER with a physician during the night

Prémanon, Jura, France

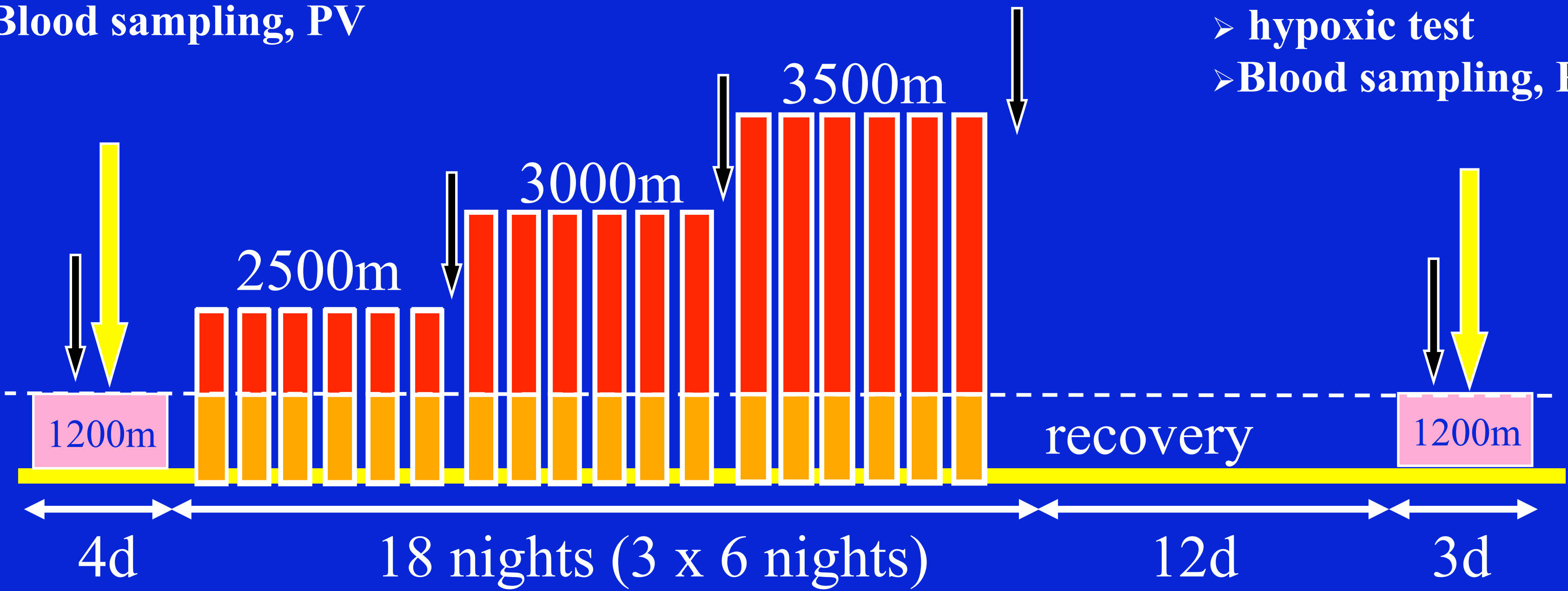
OXYGEN EXTRACTION

Nordic ski

Hypoxia
Control
Tests
Blood sampling

- VO_2max (treadmill)
- time limit at VO_2 max
- TFM, TDF
- hypoxic test
- Blood sampling, PV

- VO_2max (treadmill)
- time limit at VO_2 max
- TFM, TDF
- hypoxic test
- Blood sampling, PV



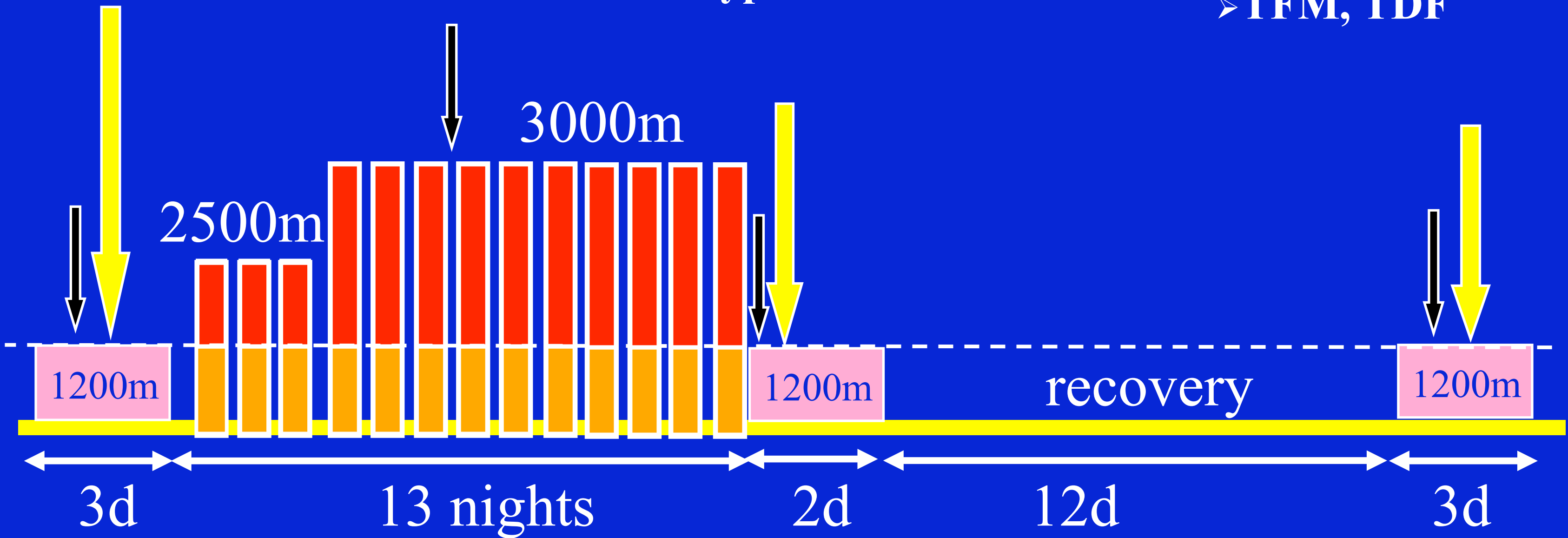
Swimming

- Hypoxia
- Control
- Tests
- Blood sampl., TDF

- VO_2max (ergocycle)
- VO_2max (swim)
- 2000m swin free style
- Blood sampl., PV
- TFM, TDF
- hypoxic test

- VO_2max (swim)
- 2000m swin free style
- Blood sampl., PV
- TFM, TDF
- hypoxic test

- VO_2max (ergocycle)
- VO_2max (swim)
- 2000m swin free style
- Blood sampl., PV
- TFM, TDF

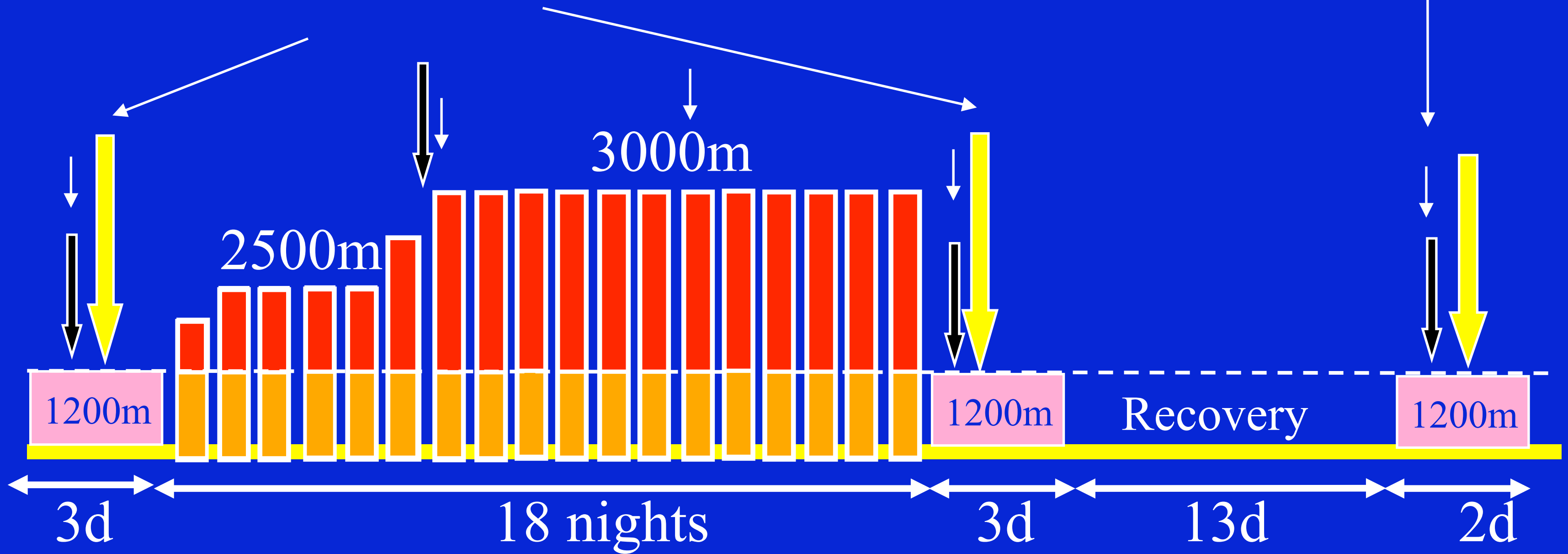


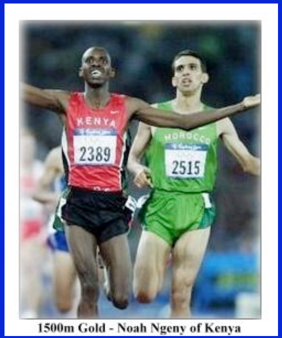

1500m Gold - Noah Ngeny of Kenya

Track and field

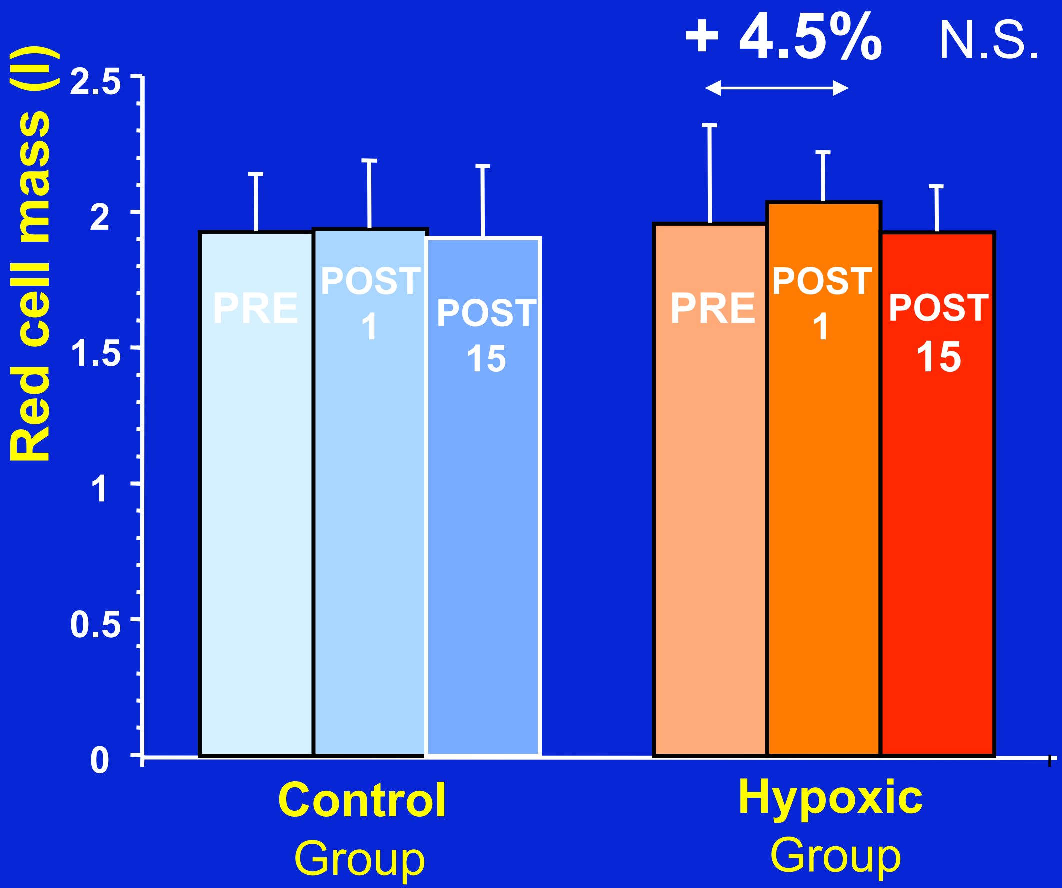
- VO_2max (treadmill)
- field test 10 min at 90% MAS.
- Blood sampl., PV
- TFM, TDF
- hypoxic test

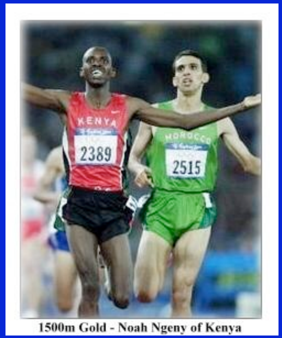
- VO_2max (treadmill)
- field test 10 min at 90% MAS
- Blood sampl., PV
- TFM, TDF
- hypoxic test



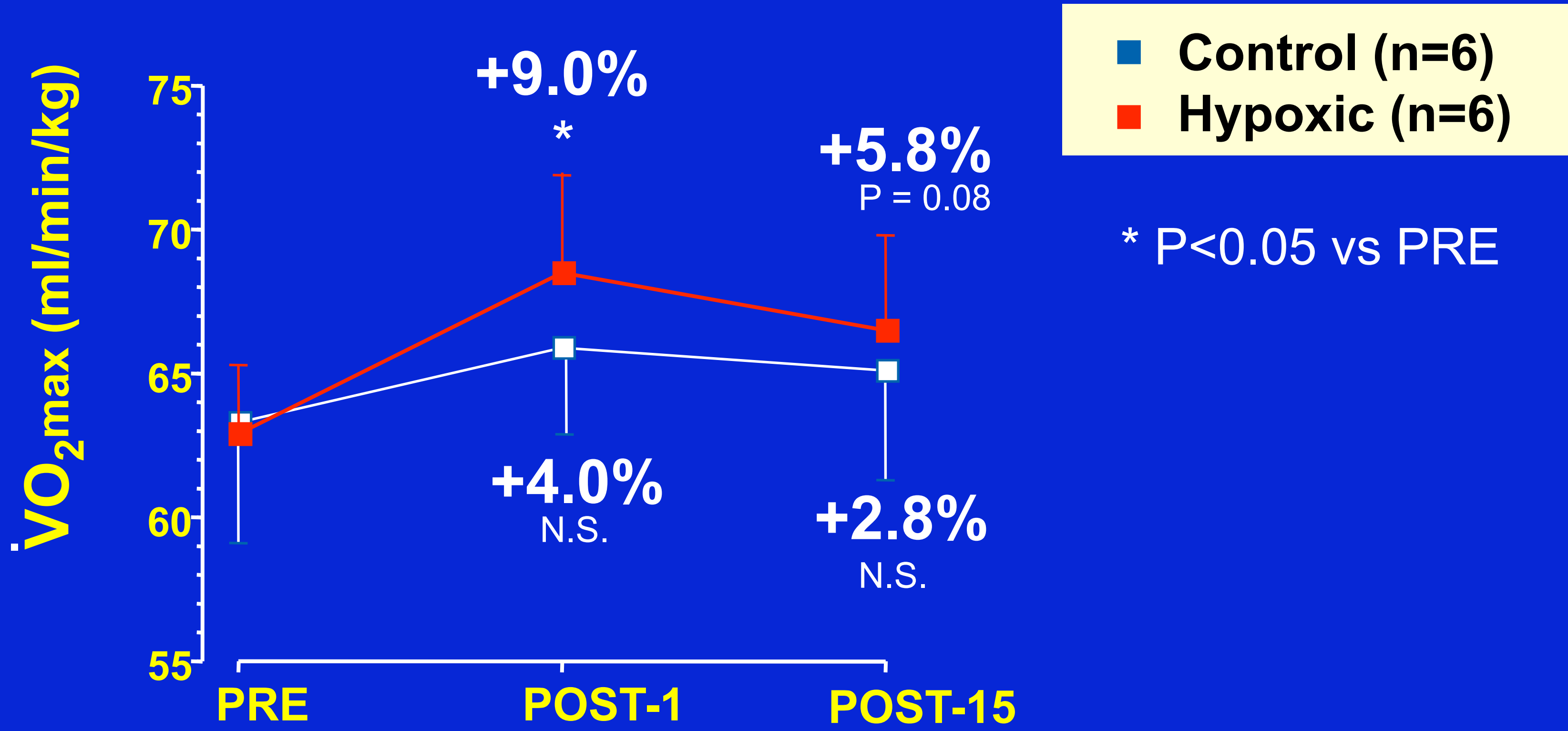


Red cell mass

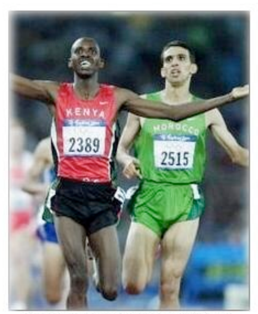




Performance ($\dot{V}O_2max$)

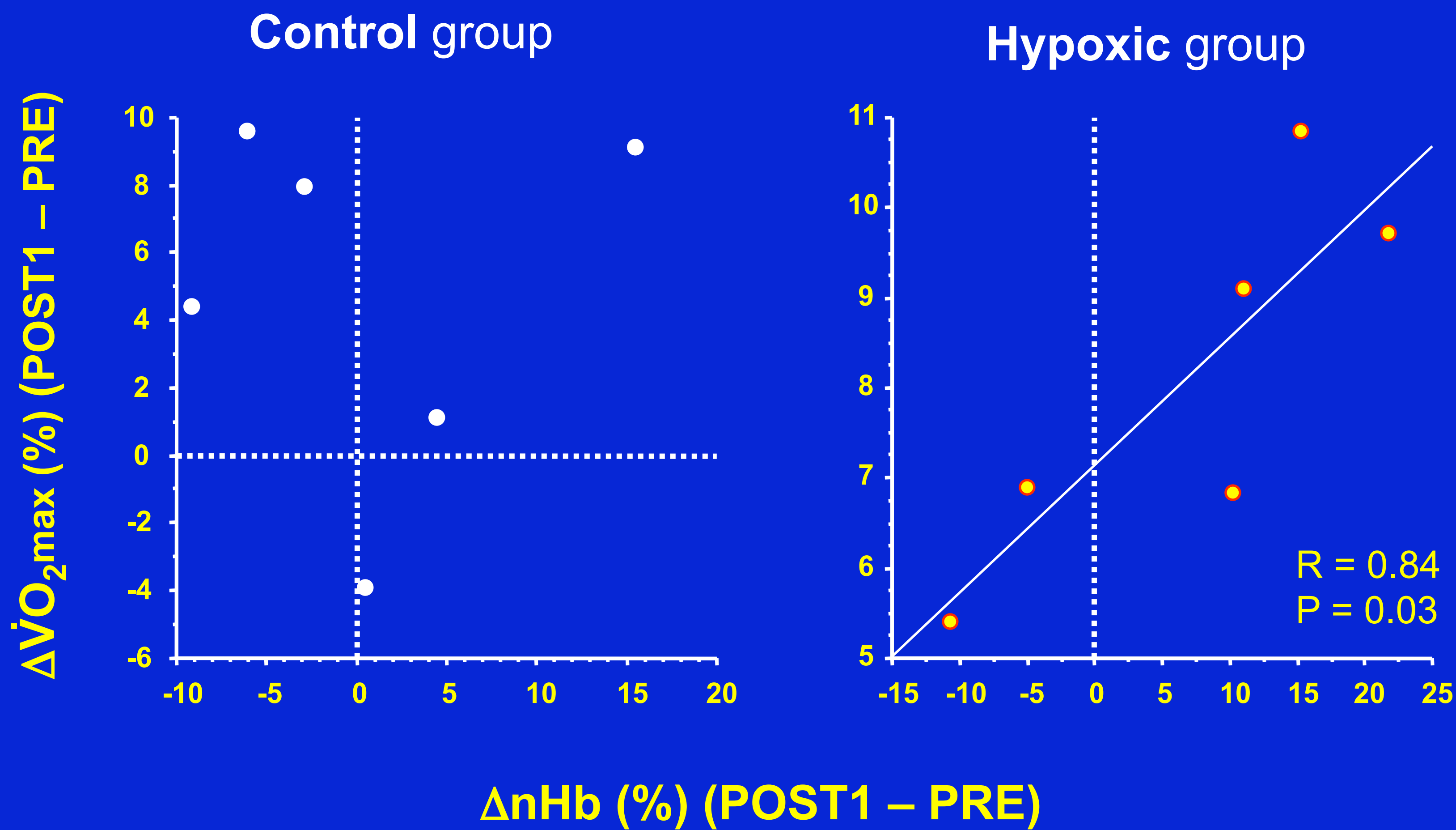


* P<0.05 vs PRE



1500m Gold - Noah Ngeny of Kenya

Variations of performance vs hemoglobin



Hb mas and VO_2max at sea level

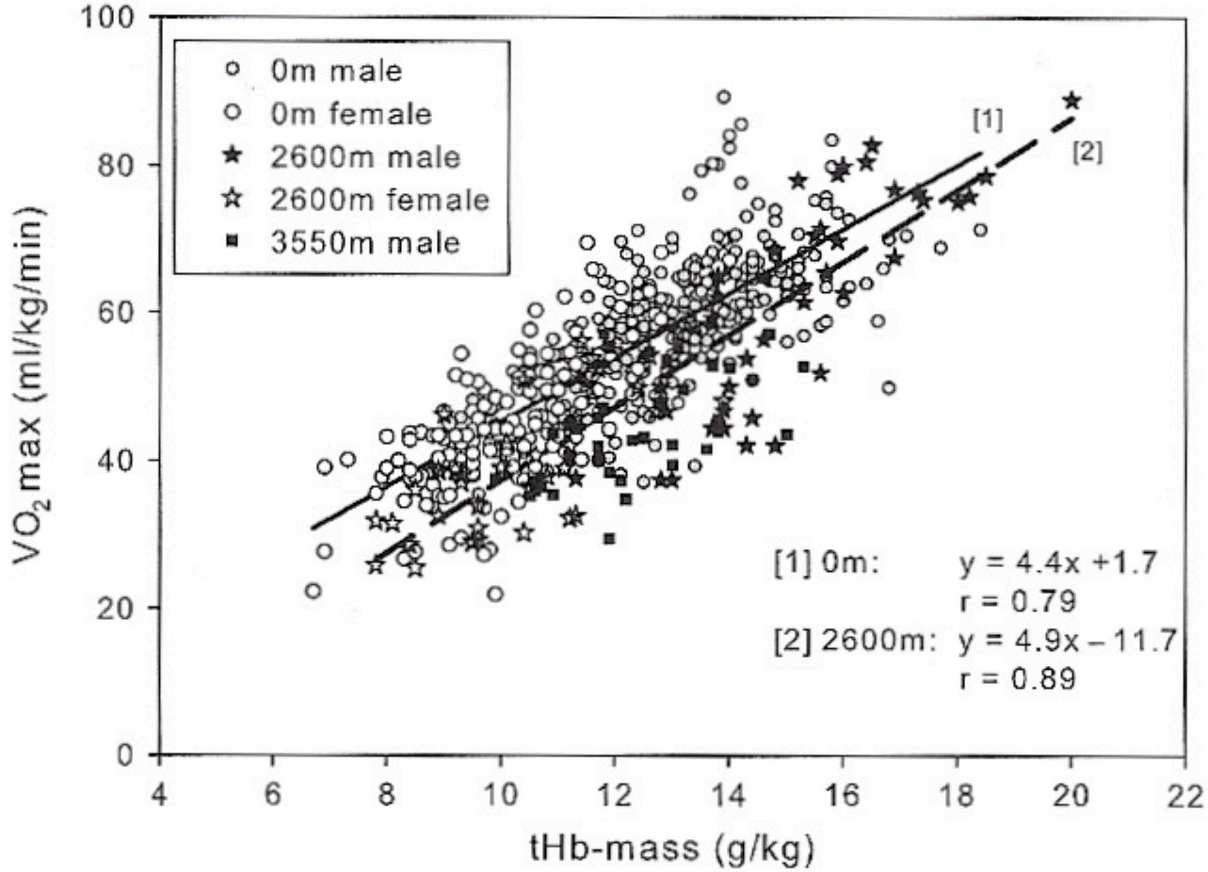


Fig. 2. Total hemoglobin mass (tHb-mass) vs VO_{2max}. Presented are relative values of 611 subjects native to lowland and to 2600 m, as well as natives and adapted subjects to 3550 m.

From: Schmidt and Prommer, *Scand J Med Sci Sports* 2008

Increase in Hb mass as a function of duration of altitude exposure

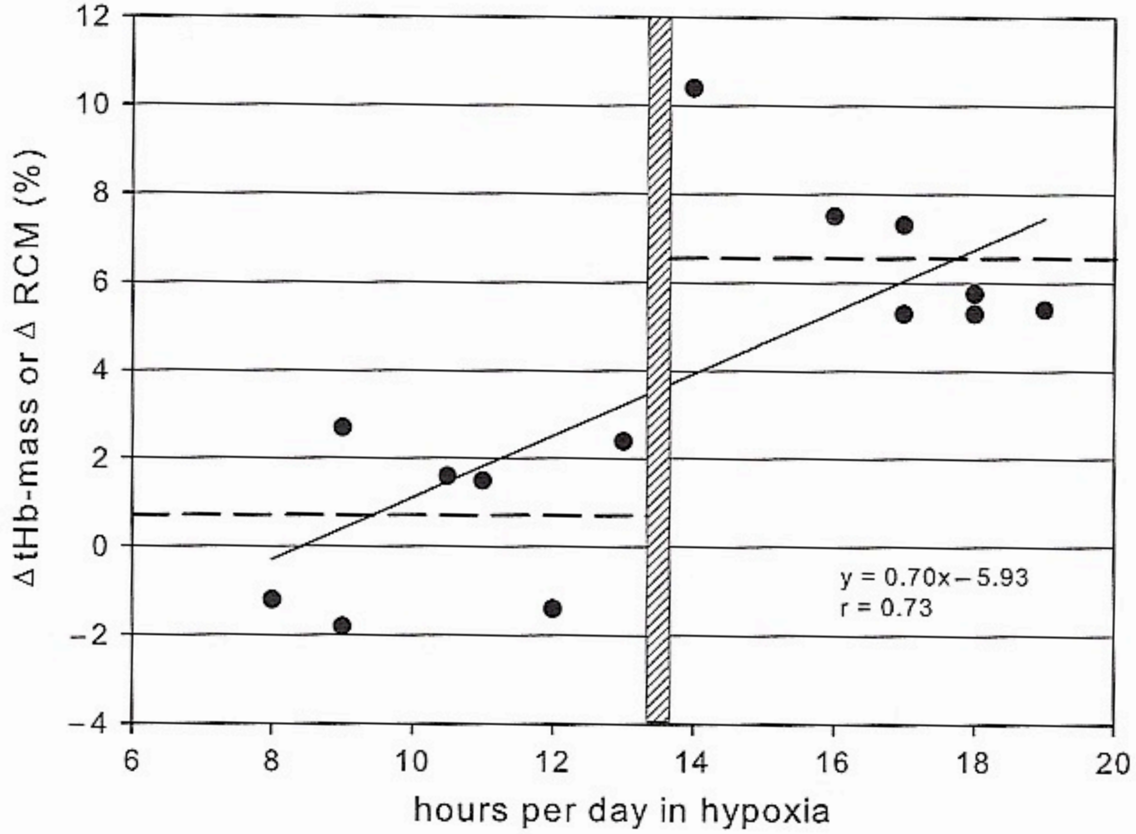
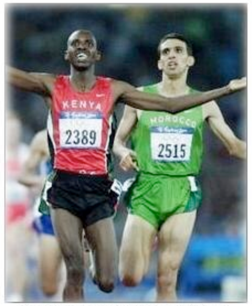


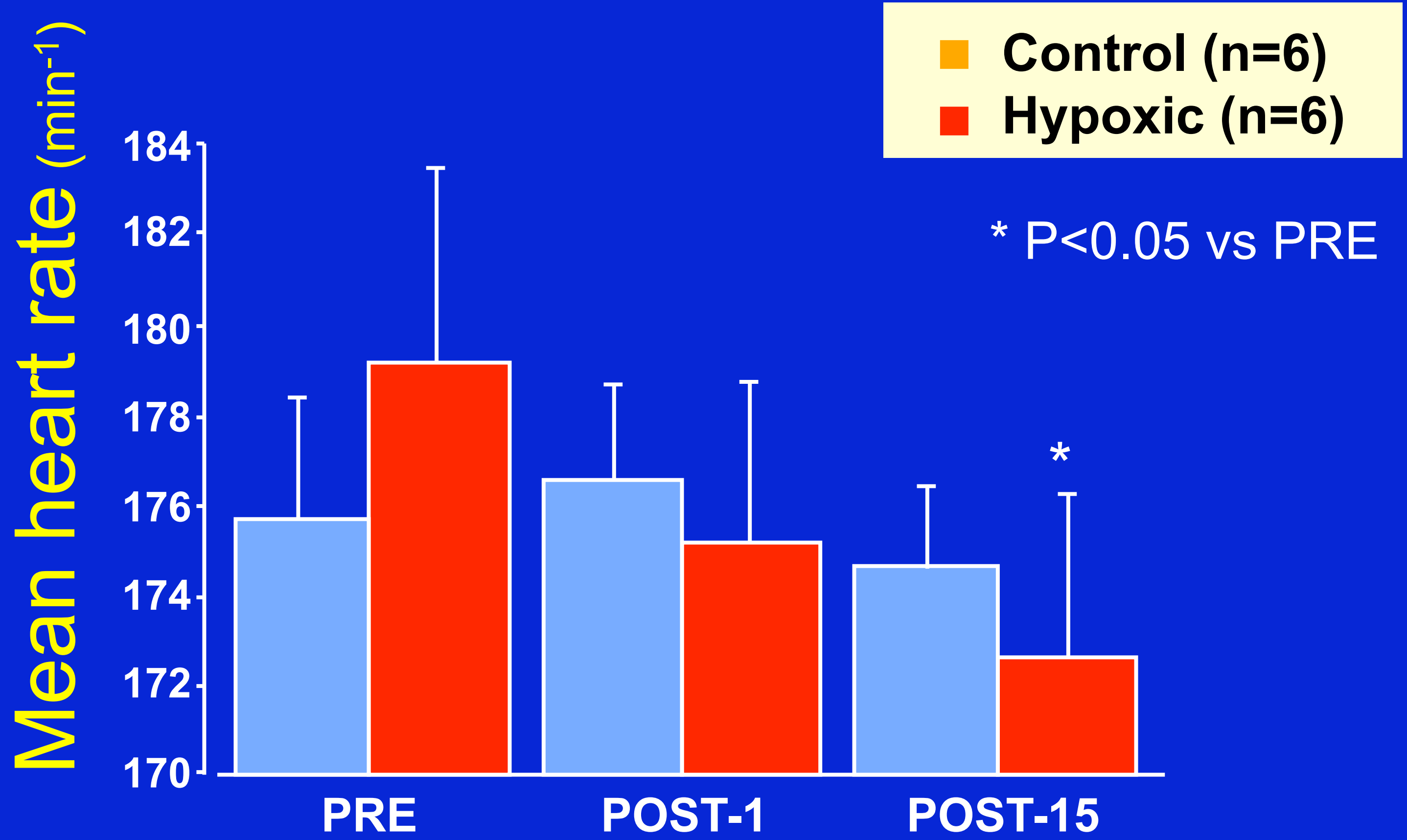
Fig. 4. Percentage changes in mean tHb-mass or red cell mass during LH–TL protocols in relation to the hours of daily exposure to hypoxia. For more information see Table 3. The horizontal lines indicate the mean change in tHb-mass for daily hypoxic exposures of less (left side) and of more (right side) than 14 h/day. tHb-mass, total hemoglobin mass; LH–TL, live high–train low.

From: Schmidt and Prommer, *Scand J Med Sci Sports* 2008

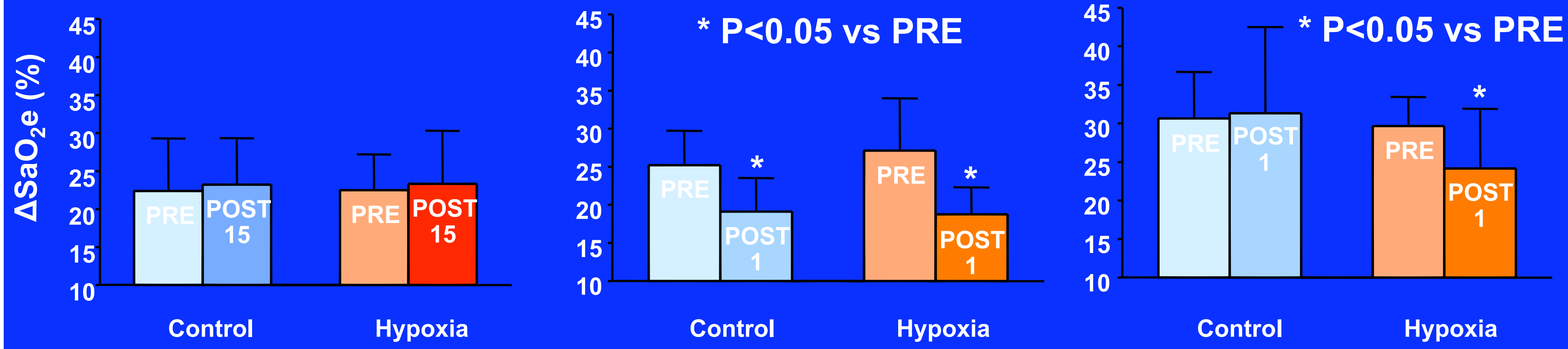
Field test: 10 min at 19.5 km/h
(≈ 90% of maximal aerobic speed)

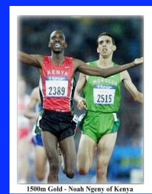


1500m Gold - Noah Ngeny of Kenya



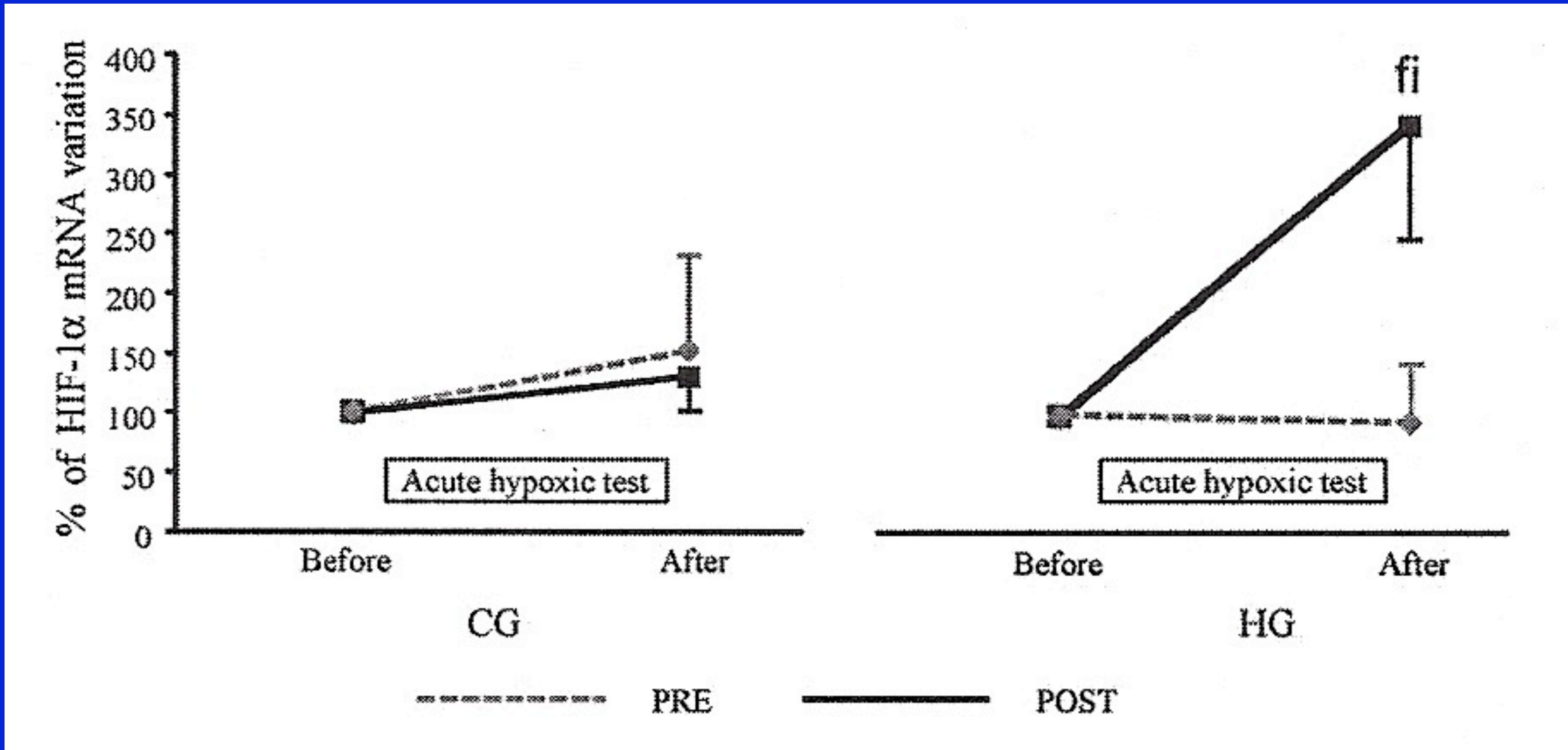
Hypoxic exercise- induced desaturation (ΔSaO_2e)





Lesser desaturation at exercise (ΔSaO_2e) at the end of the training session = sign of ventilatory acclimatization at exercice in hypoxia.

Response of HIF-1α to an acute hypoxic stress, before and after a LH-TL training session



From: Pialoux et al., *Respir Physiol Neurobiol,* 2009

Relation between changes in HVR and changes in oxidative stress during a LH-TL training session

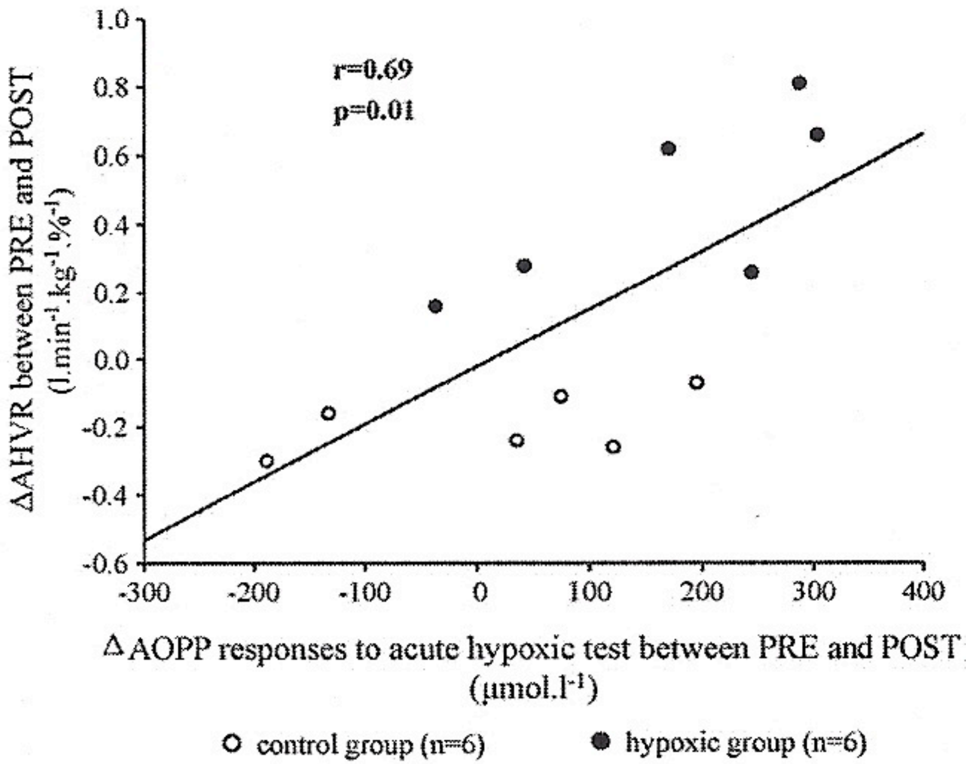
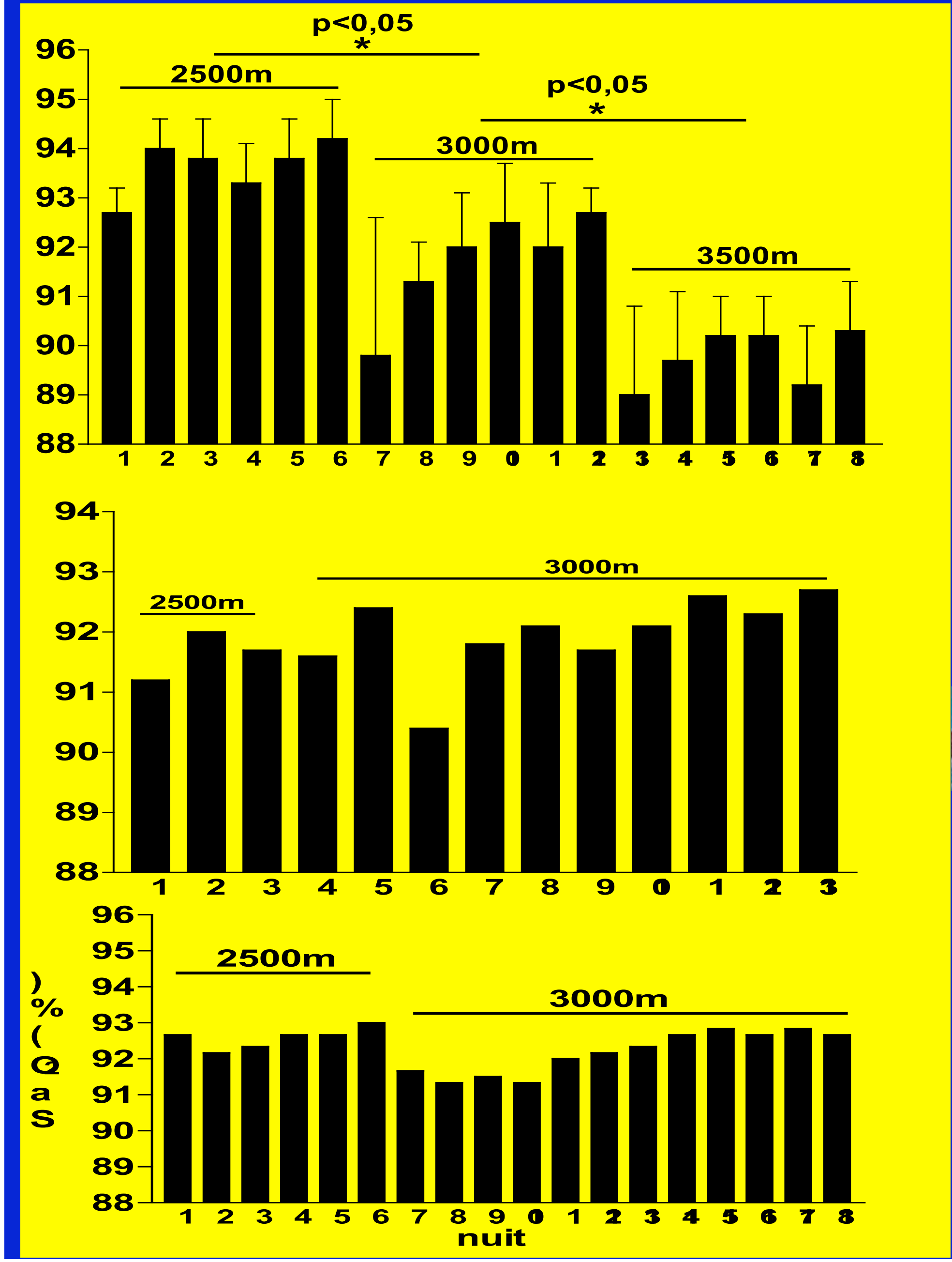


Fig. 2. Relationship between the changes in the acute hypoxic ventilatory response (ΔAHVR) during exercise and protein oxidation (ΔAOPP) during the acute hypoxic response test between before (PRE) and after (POST) 18 days of "living high–training low" ($n = 12$). The black circles represent subjects of the hypoxic group ($n = 6$) and the open circles represent subjects of the control group ($n = 6$).

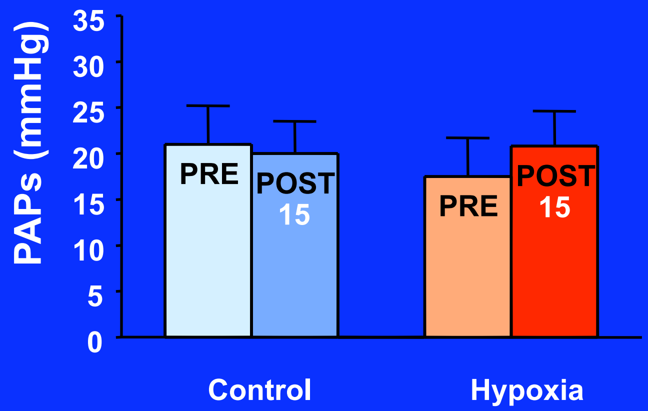
From: Pialoux et al., *Respir Physiol Neurobiol,* 2009

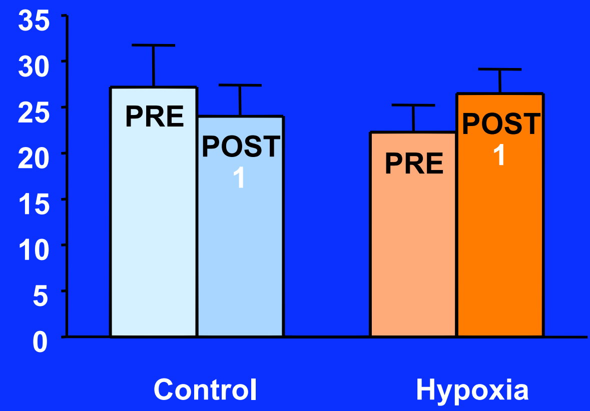
Nocturnal oxygen saturation (SaO_2)

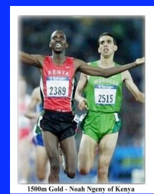


Sleep in hypoxic chambers induces episodes of desaturation, without apparent consequences on athlete's health

systolic Pulmonary artery pressure (PAPs)

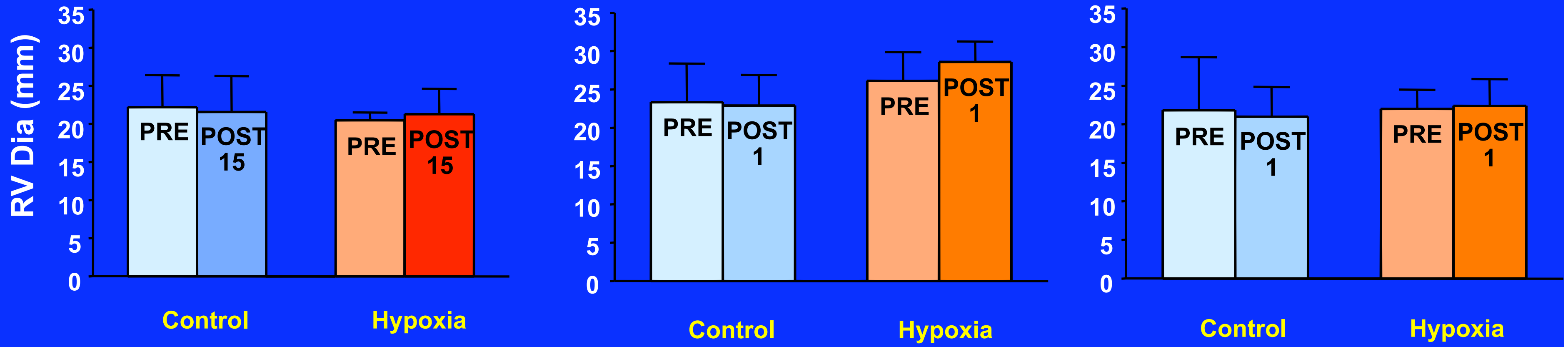


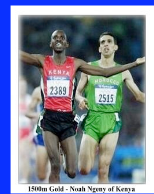




PAPs does not vary significantly:
no pulmonary hypentension

Right ventricule diameter in diastole (RVdia)





No right ventricular dilatation, classical marker of RV overload due to pulmonary hypertension

Tolerance and acclimatization

« Live high – train low » (3000/1200)

- does not induce symptoms of Acute Mounatin Sickness.
- may induce sleep perturbations and fatigue (if ≥ 3500m and training load not reduced)
- may induce sleep apneas in some subjects, without apparent clinical consequences during the day.

Tolerance and acclimatization

« Live high – train low » (3000/1200)

- is not dangerous for the health of the athlete

- induces a ventilatory acclimatization (lower desaturation at exercise in hypoxia) that fades away 15 days after the training session

The « Live high - train low » procedure in the high level endurance athlete

HYPOXIA

Naturally stimulates erythropoiesis and moderately increases the blood oxygen carrying capacity

Activates the sympathetic and inhibits the parasympathetic system, but variations are limited

Induces a ventilatory acclimatization process depending on the « dose » of the hypoxic stimulus

The « Live high - train low » procedure in the high level endurance athlete

HYPOXIA

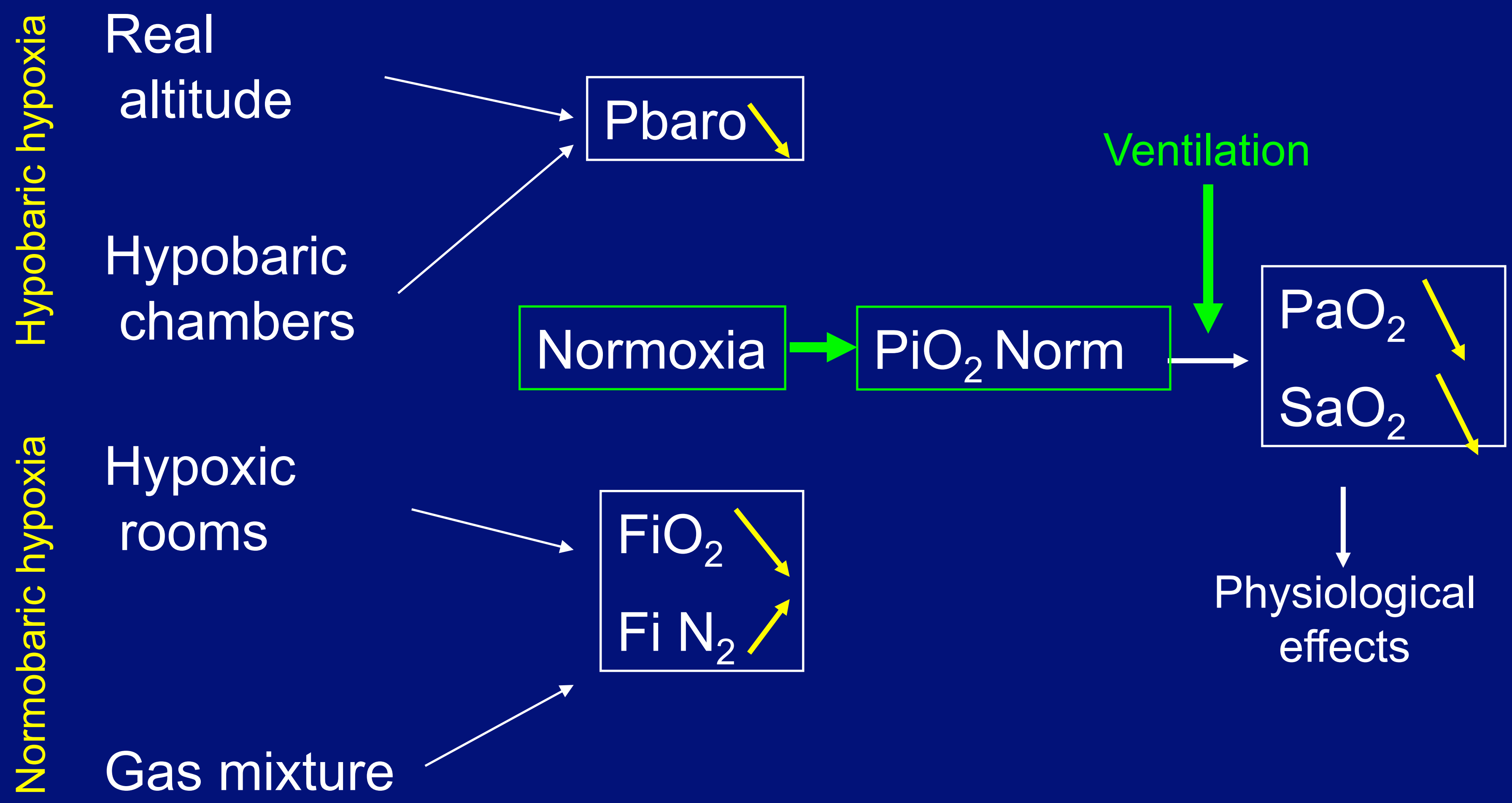
Increases aerobic performance in variable proportion depending on duration and type of exposure

Up to now, no marker of individual good response to hypoxic training is available

Induces mechanisms allowing a good tolerance to LHTL training procedure (if altitude <= 3000m)

Does not provoke negative physiological or psychological reactions at short or medium term

How to get hypoxemic with a normoxic environment ?



$$PiO_2 = FiO_2 \times (Pbaro - PH_2O)$$

Relative voluntary hypoventilation during exercise:
a new cheap technique to obtain hypoxia ?

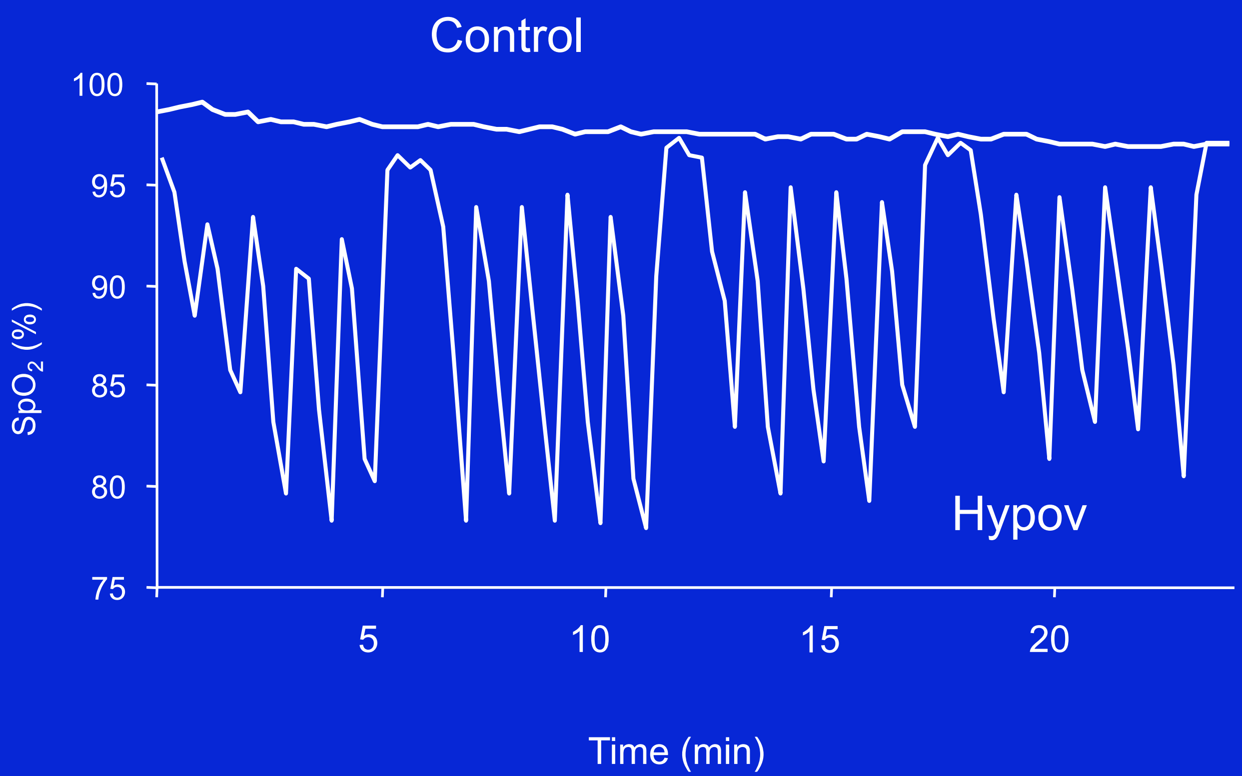
40% $\dot{V}O_{2max}$

65% $\dot{V}O_{2max}$

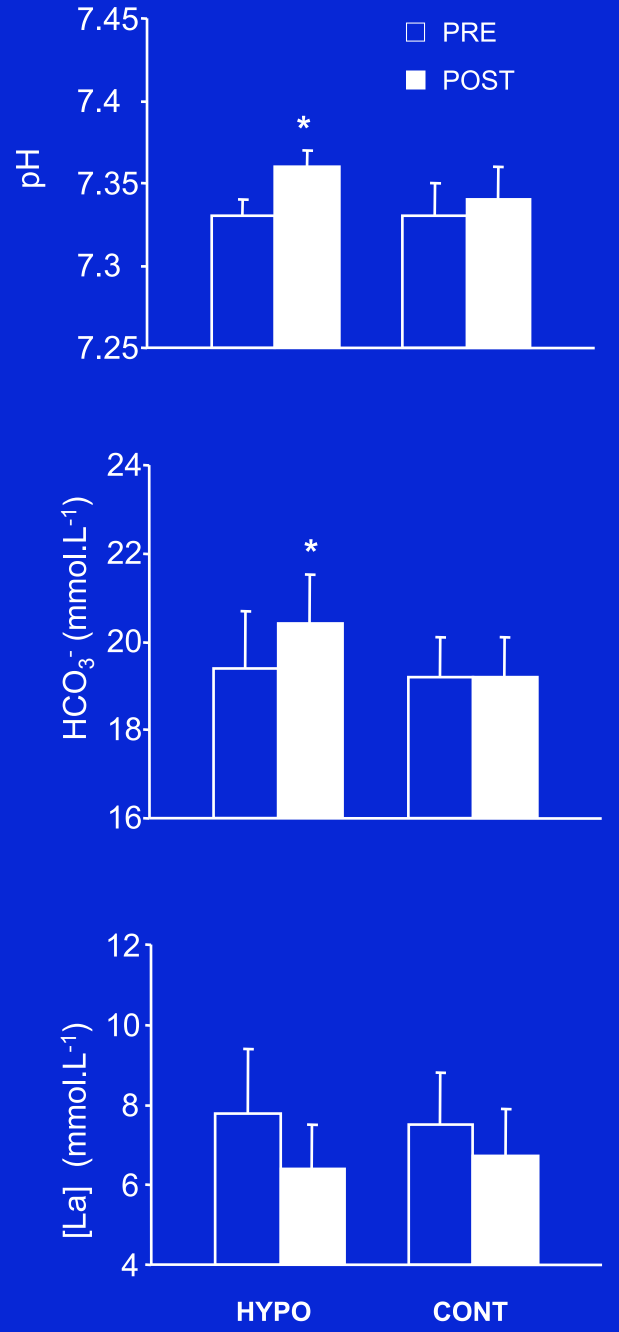
Warm up (3’)	S1 (5’)	T1 (1’)	S2	T2	S3	T3
2’ NORM – 1’ HYPOV	(15’’ NORM – 45’’ HYPOV) * 5	NORM	(15’’ NORM – 45’’ HYPOV) * 5	NORM	(15’’ NORM – 45’’ HYPOV) * 5	NORM

Woorons et al., 2009

Mean SpO2 during a training session with voluntary hypoventilation vs control normal ventilation



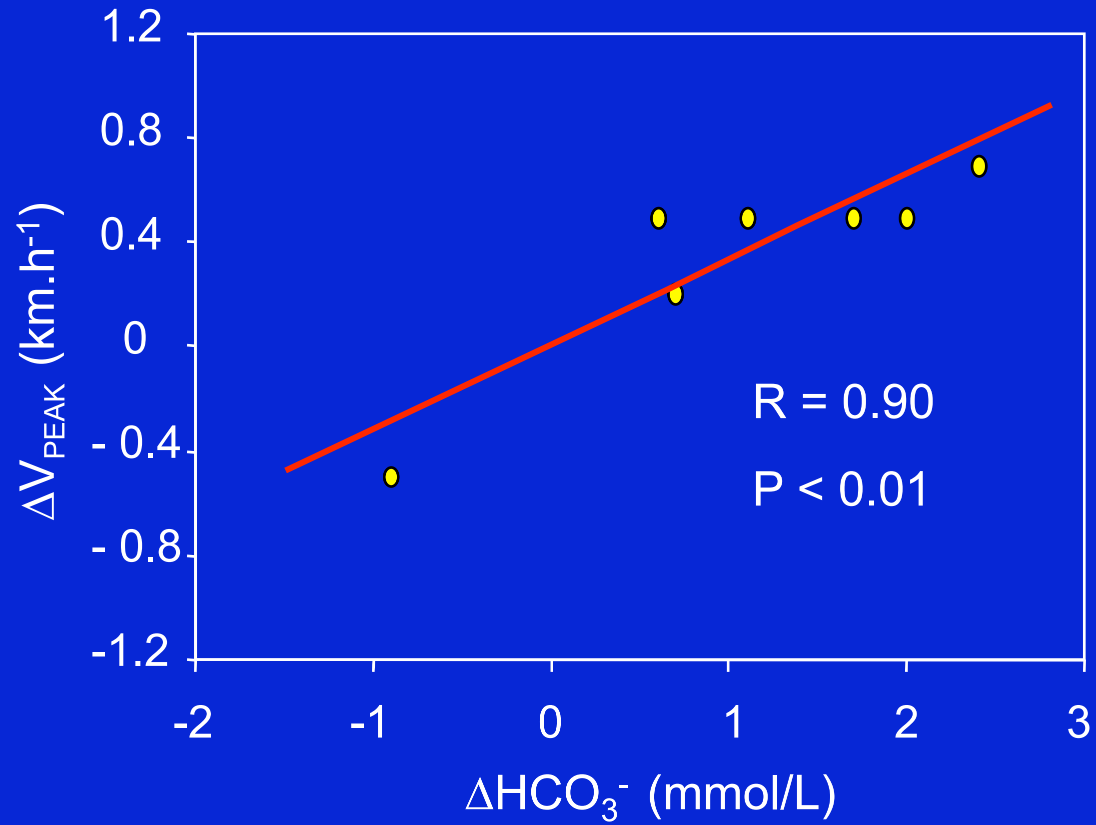
From: Woorons et al., *Respir Physiol Neurobiol*, 2008



Blood pH, bicarbonate and lactate at 90% of maxHR in voluntary hypoventilation (HYPO) and control (CONT) before (PRE) and after (POST) the training session.

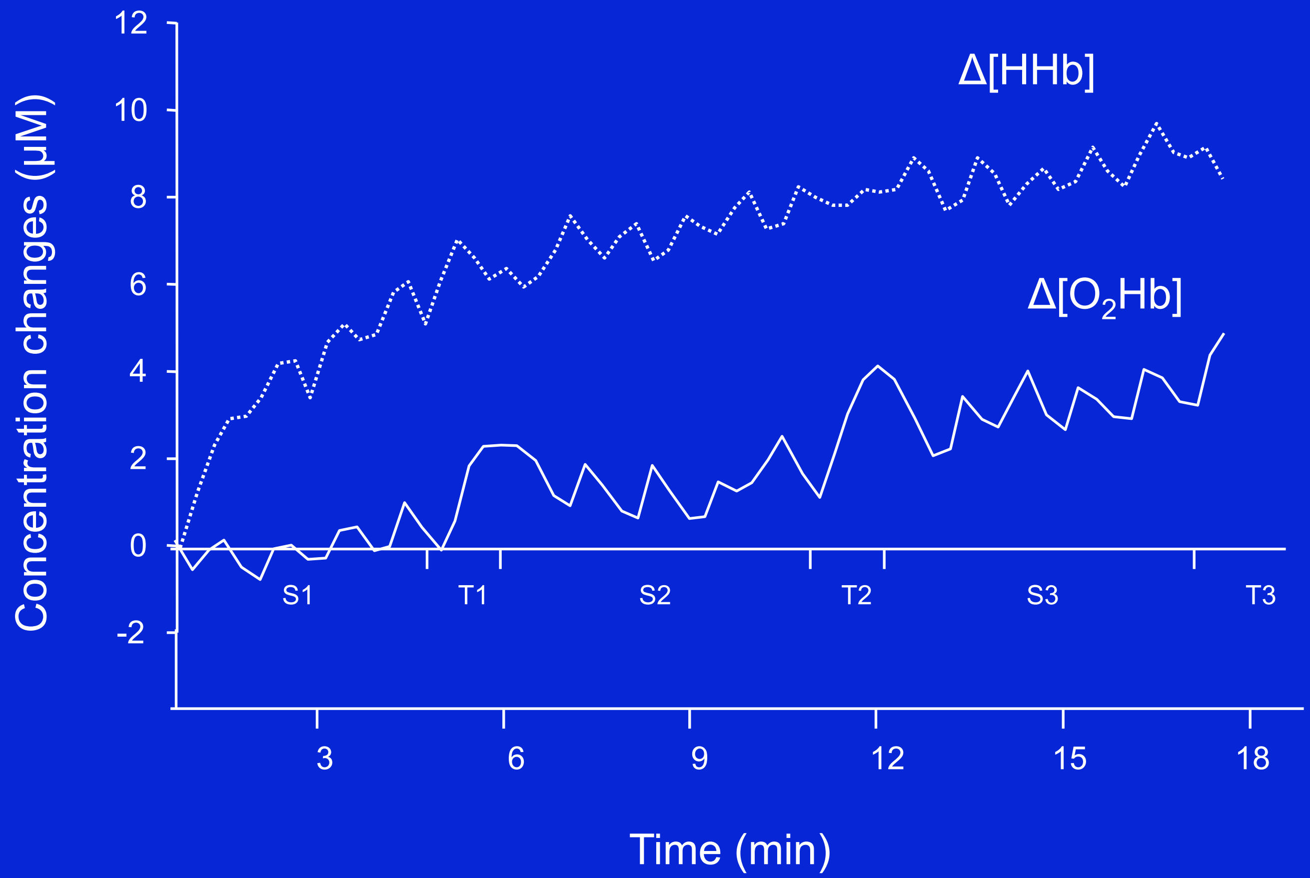
From: Woorons et al., *Respir Physiol Neurobiol*, 2008

Relationship between the change in maximal velocity reached during a VO_2max test and the change in blood bicarbonate at 90% of age predicted HRmax , with voluntary hypoventilation



From: Woorons et al., *Respir Physiol Neurobiol*, 2008

Hypoventilation during exercise: NIRS signal



Effects of prolonged exercise with voluntary hypoventilation on muscle oxygenation and blood lactate concentration

Woorons X, Bourdillon N, Vandewalle H, Lamberto C, Mollard P, Richalet JP, Pichon A

Laboratoire “Réponses cellulaires et fonctionnelles à l’hypoxie", EA2363, Université Paris 13, Bobigny, France.

Exercise with voluntary hypoventilation at low pulmonary volumes

- Severe arterial hypoxemia (SaO_2 = 87%) (Woorons et al. (2007
- Hypercapnia + respiratory acidosis (all studies)
- No increase in blood lactate concentration (all studies)
- Muscle oxygenation ?

Hypotheses

- Exercise with hypoventilation would increase muscle deoxygenation under the hypoxic and hypercapnic effects

- Blood lactate concentration would be higher if exercise with hypoventilation is prolonged and separated by recovery periods

METHODS

• **8 HEALTHY MEN** (VO_{2max} = 53.2 ± 3.7 ml/min/kg)

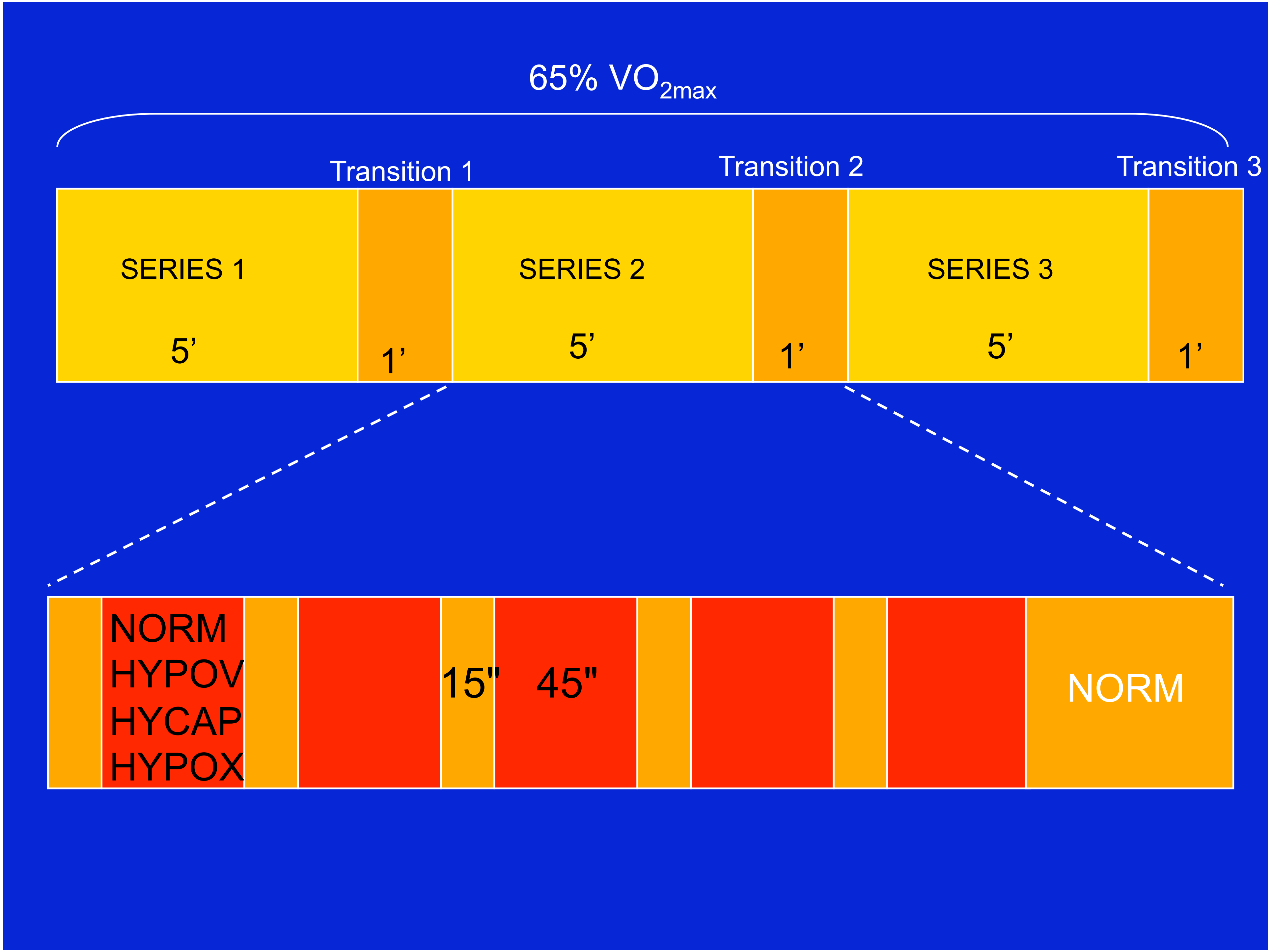
• **3 SERIES** of 5-min exercise on a cycle ergometer at 65% of normoxic VO2max

• **4 MODALITIES**

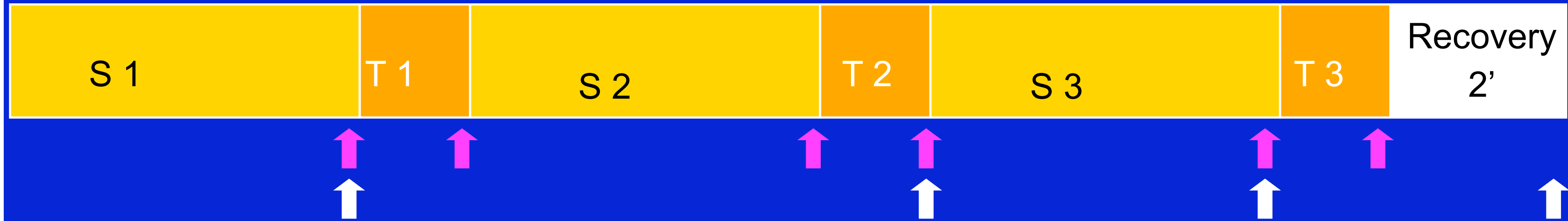
- Normal breathing in normoxia (NORM)
- Voluntary hypoventilation in normoxia (HYPOV)
- Voluntary hypoventilation in hyperoxia (FIO2 = 0.29)

→ Hypercapnia (HYCAP)

- Normal breathing in hypoxia (FIO2 = 0.15) (HYPOX)



65% VO_{2max}
Transition 1
Transition 2
Transition 3
SERIES 1
5'
1'
SERIES 2
5'
1'
SERIES 3
5'
1'
NORM
HYPOV
HYCAP
HYPOX
15"
45"
NORM



- **Muscle Oxygenation → NIRS**

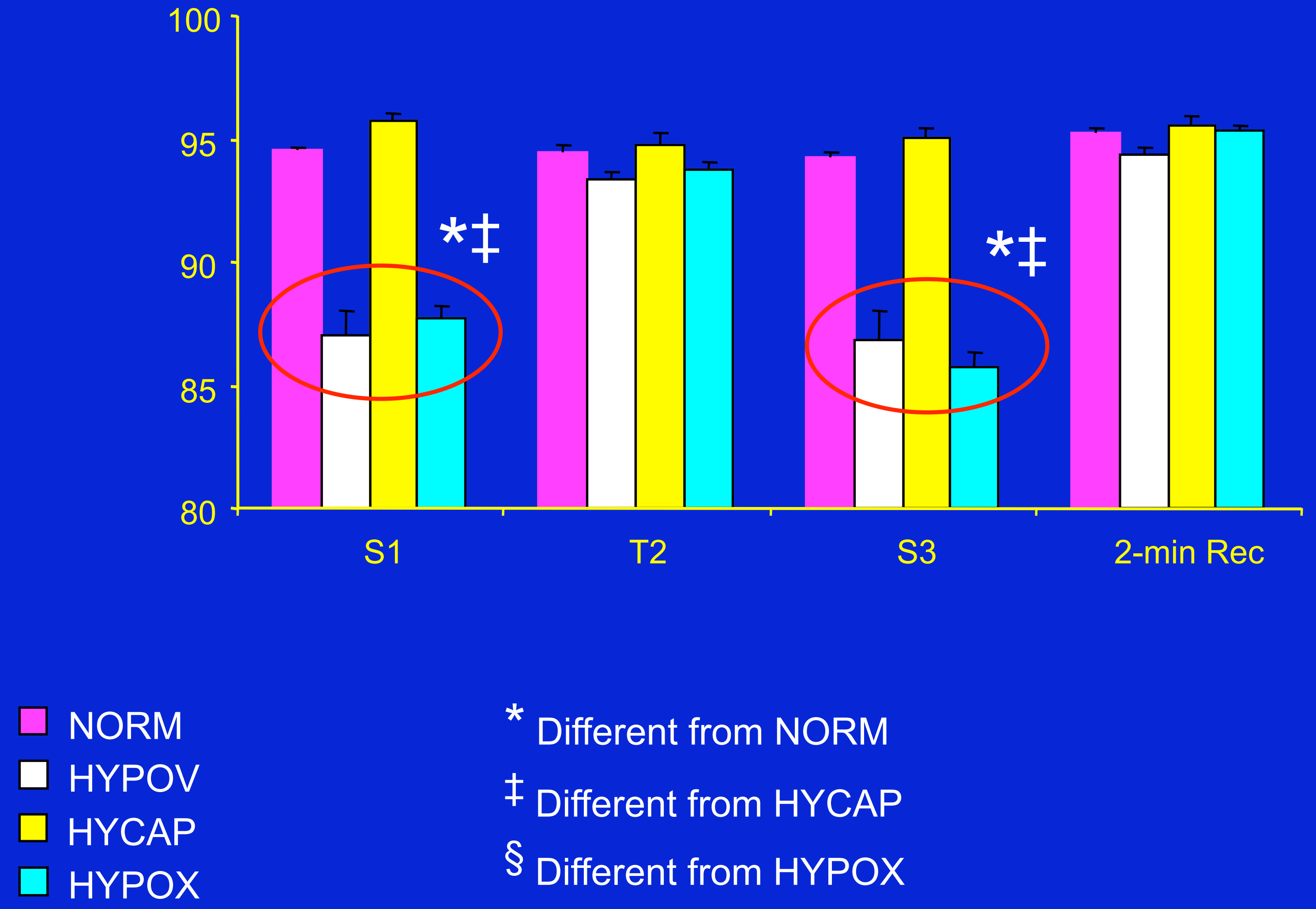
- Δ[O2Hb] = Concentration changes of oxy-hemoglobin
- Δ[HHb] = Concentration changes of deoxyhemoglobin
- Δ[O2Hb -HHb] = Oxygenation index
- Δ[THb] = Δ[O2Hb + HHb] = Change in total Hb (regional blood volume)

- **Lactate and arterialized blood gases**

- PaO2, PaCO2, pH, SaO2, Hb, La-, HCO3, P50

RESULTS

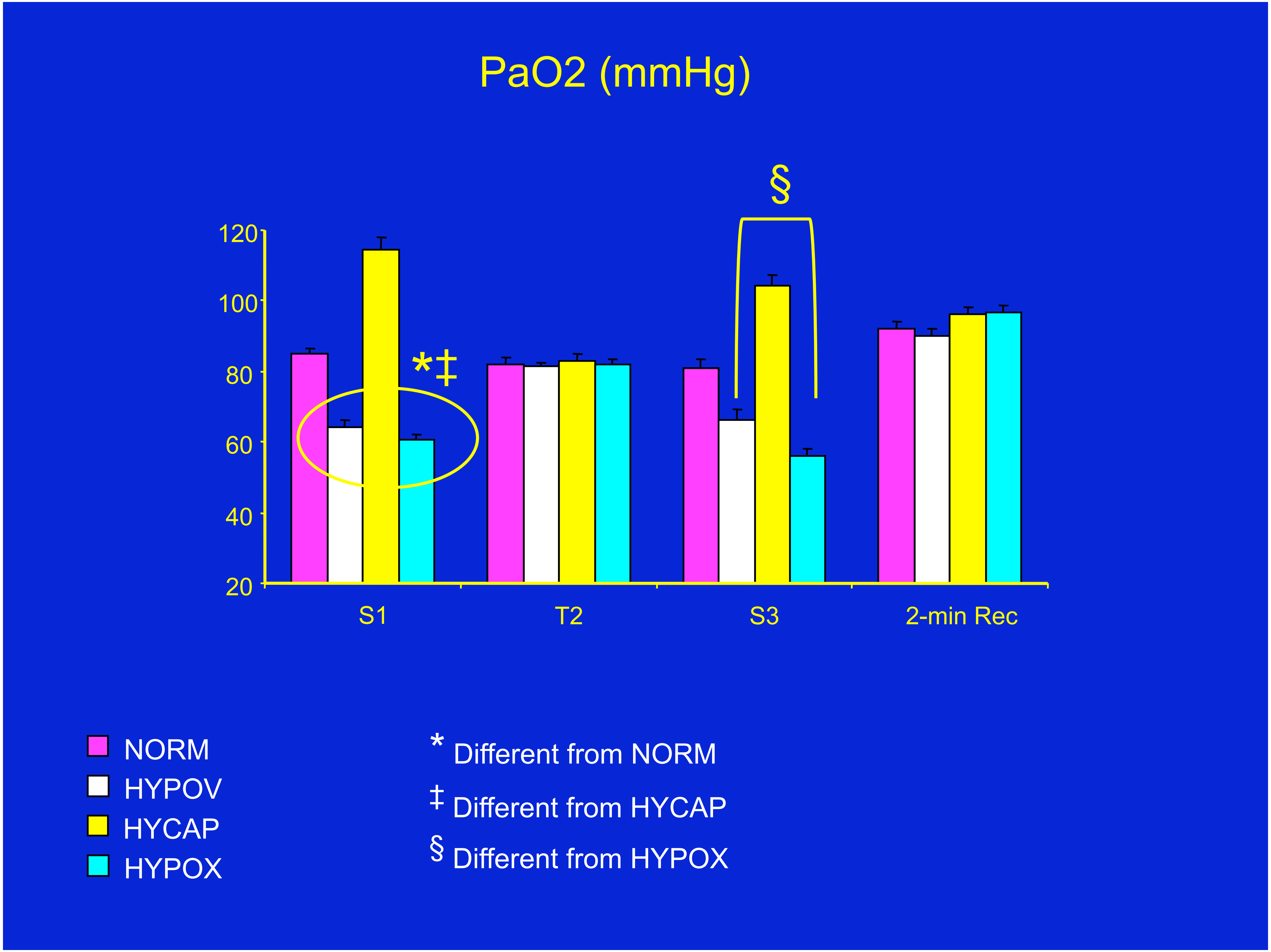
SaO2 (%)



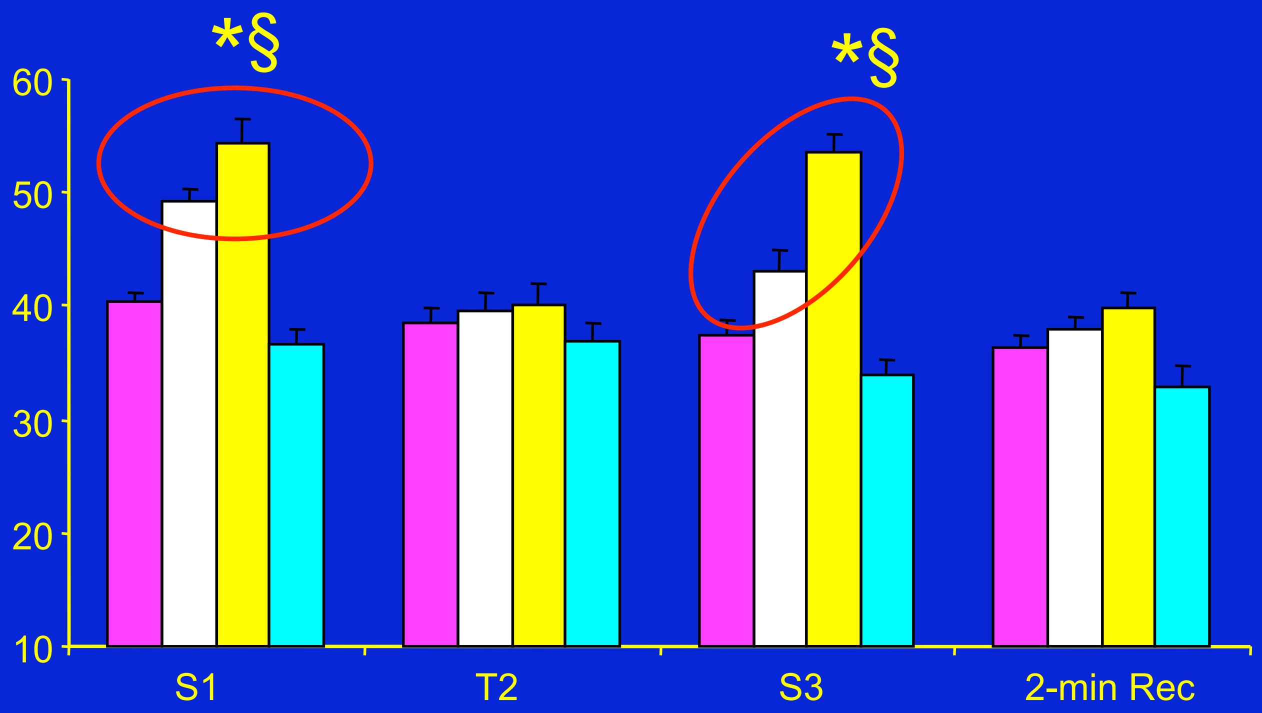
* Different from NORM

‡ Different from HYCAP

§ Different from HYPOX


PaO2 (mmHg)
§
120
100
80
60
40
20
*‡
S1
T2
S3
2-min Rec
NORM
HYPOV
HYCAP
HYPOX
* Different from NORM
‡ Different from HYCAP
§ Different from HYPOX

PaCO2 (mmHg)

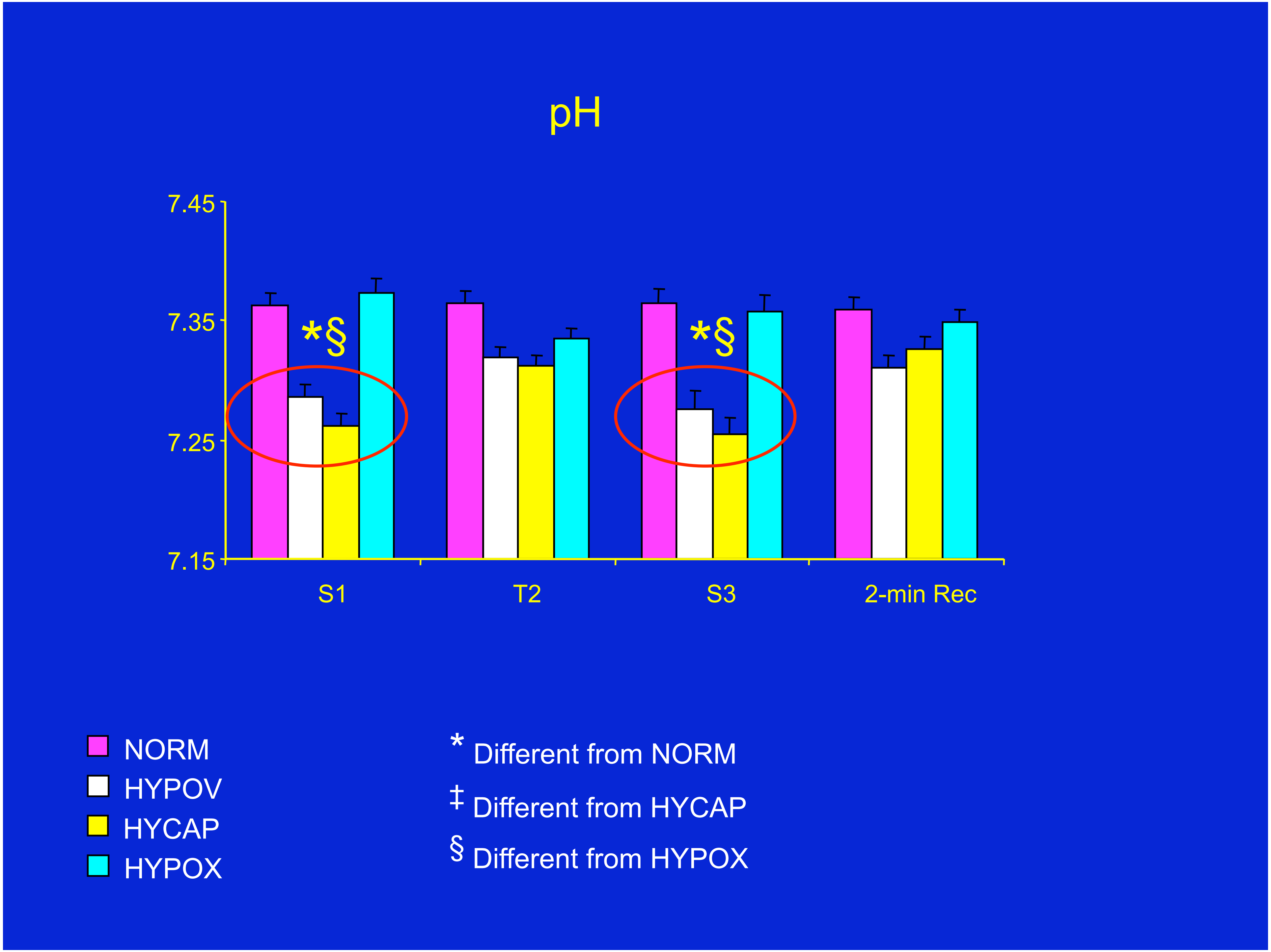


NORM
HYPOV
HYCAP
HYPOX

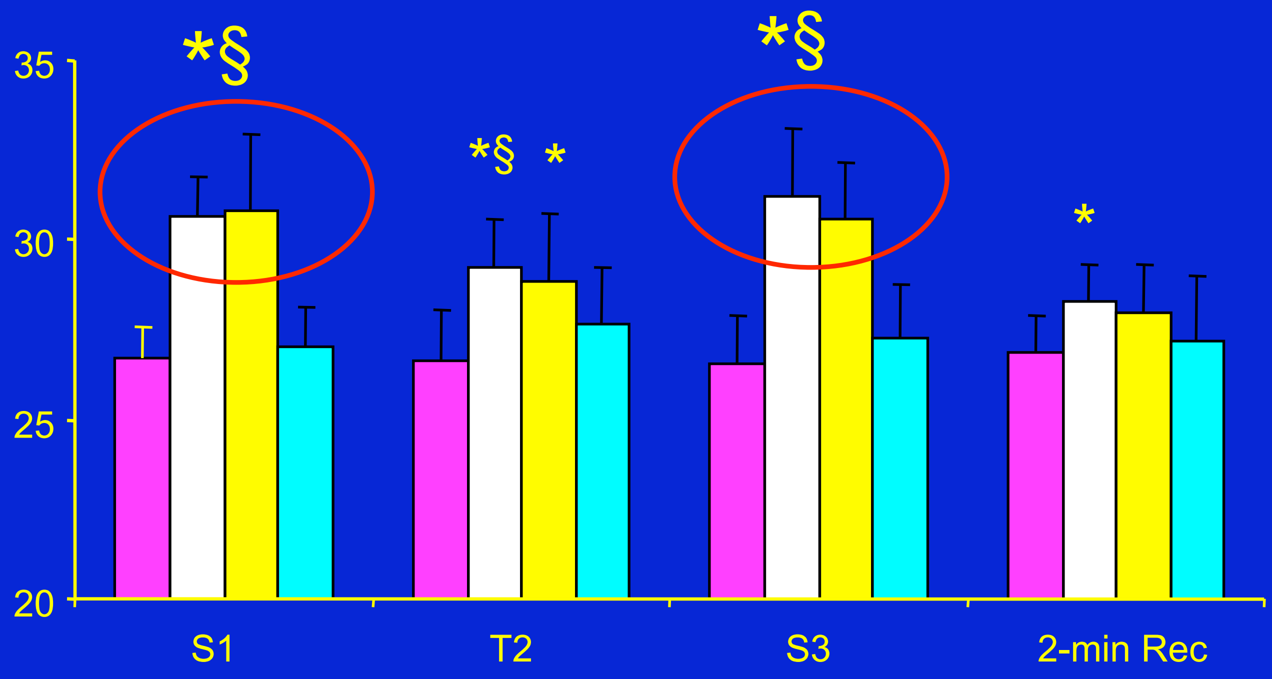
* Different from NORM

‡ Different from HYCAP

§ Different from HYPOX


pH
7.45
7.35
7.25
7.15
*§
*§
S1
T2
S3
2-min Rec
NORM
HYPOV
HYCAP
HYPOX
* Different from NORM
‡ Different from HYCAP
§ Different from HYPOX

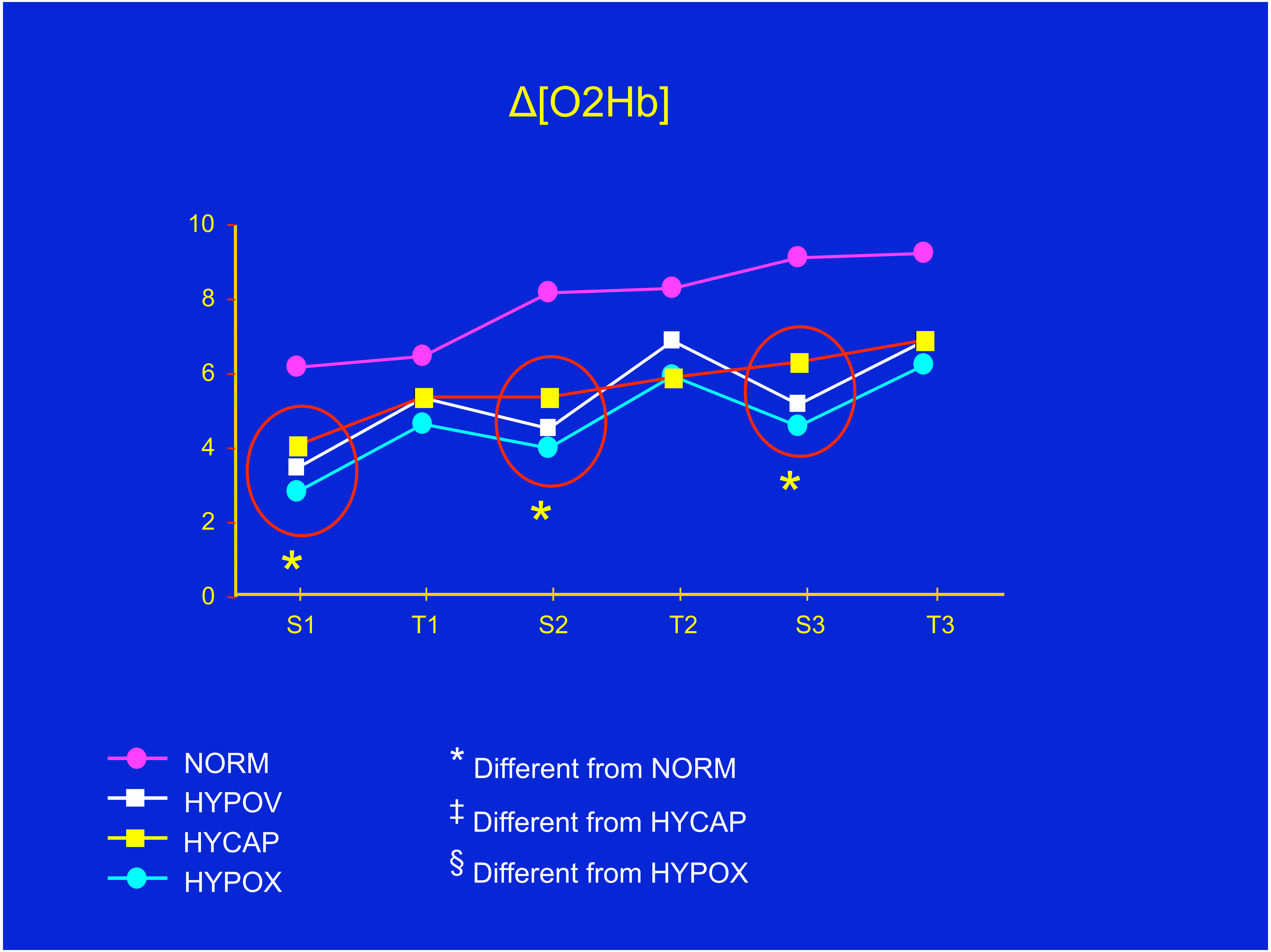
P50 (mmHg)



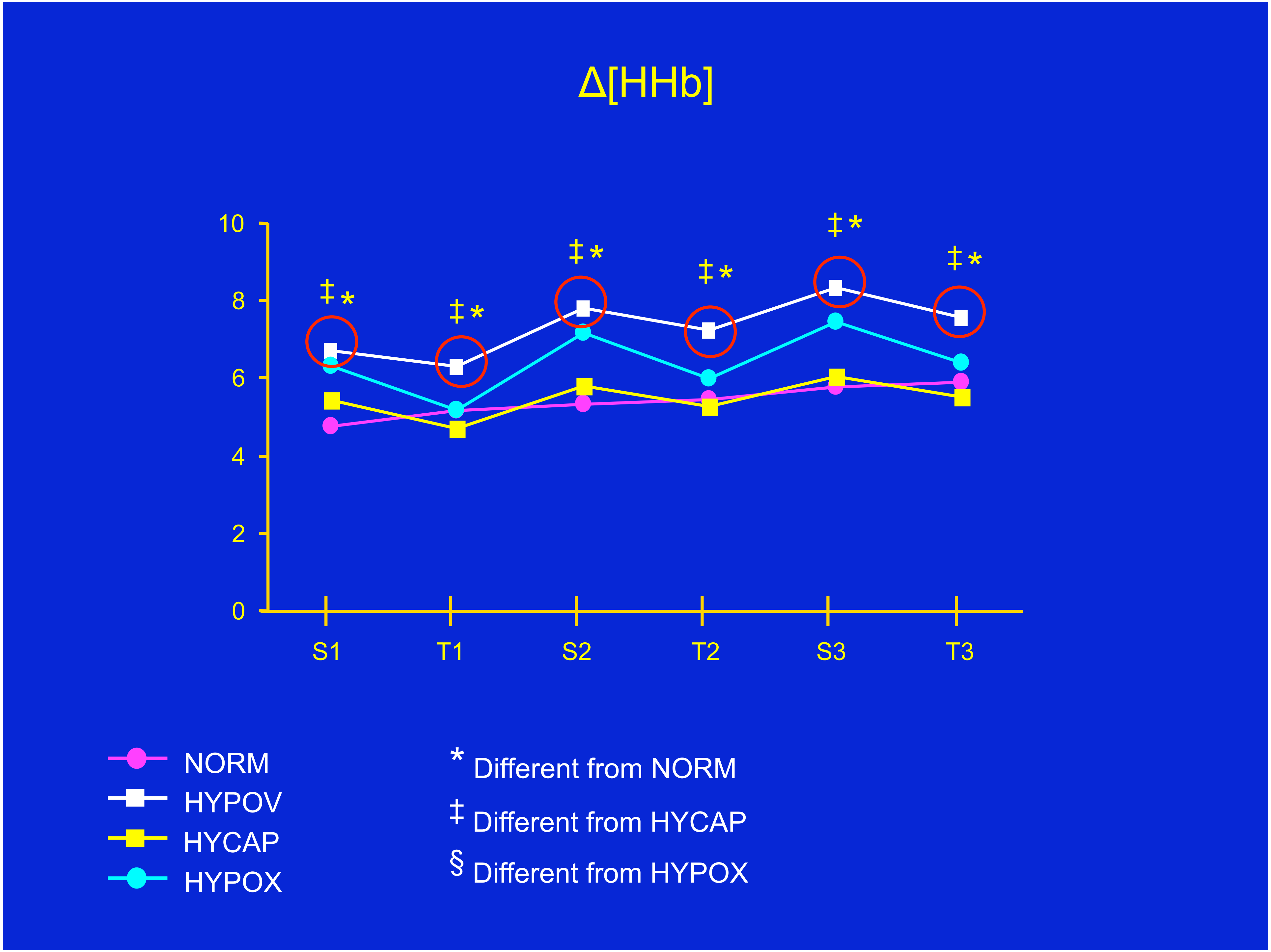
* Different from NORM

‡ Different from HYCAP

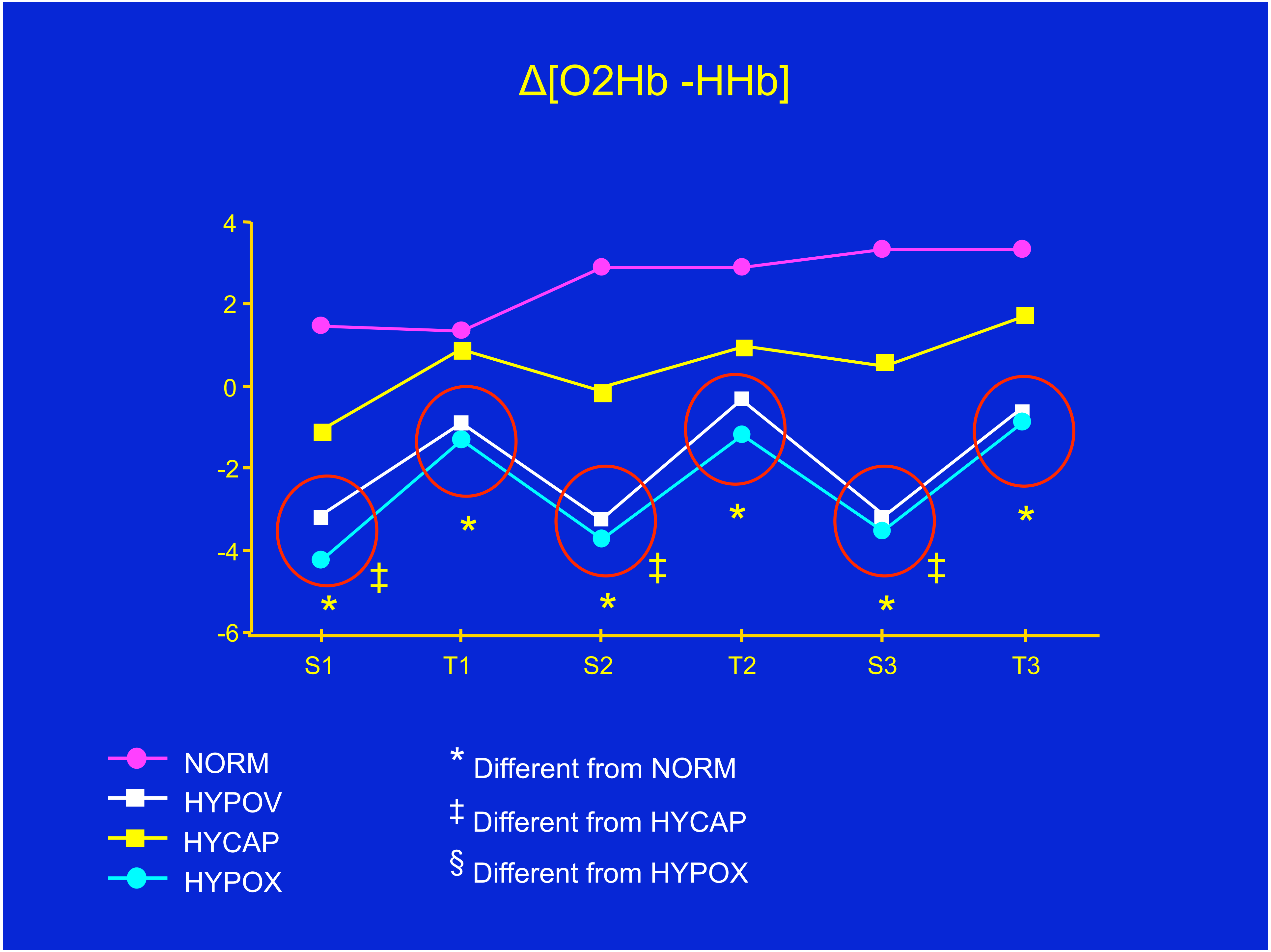
§ Different from HYPOX



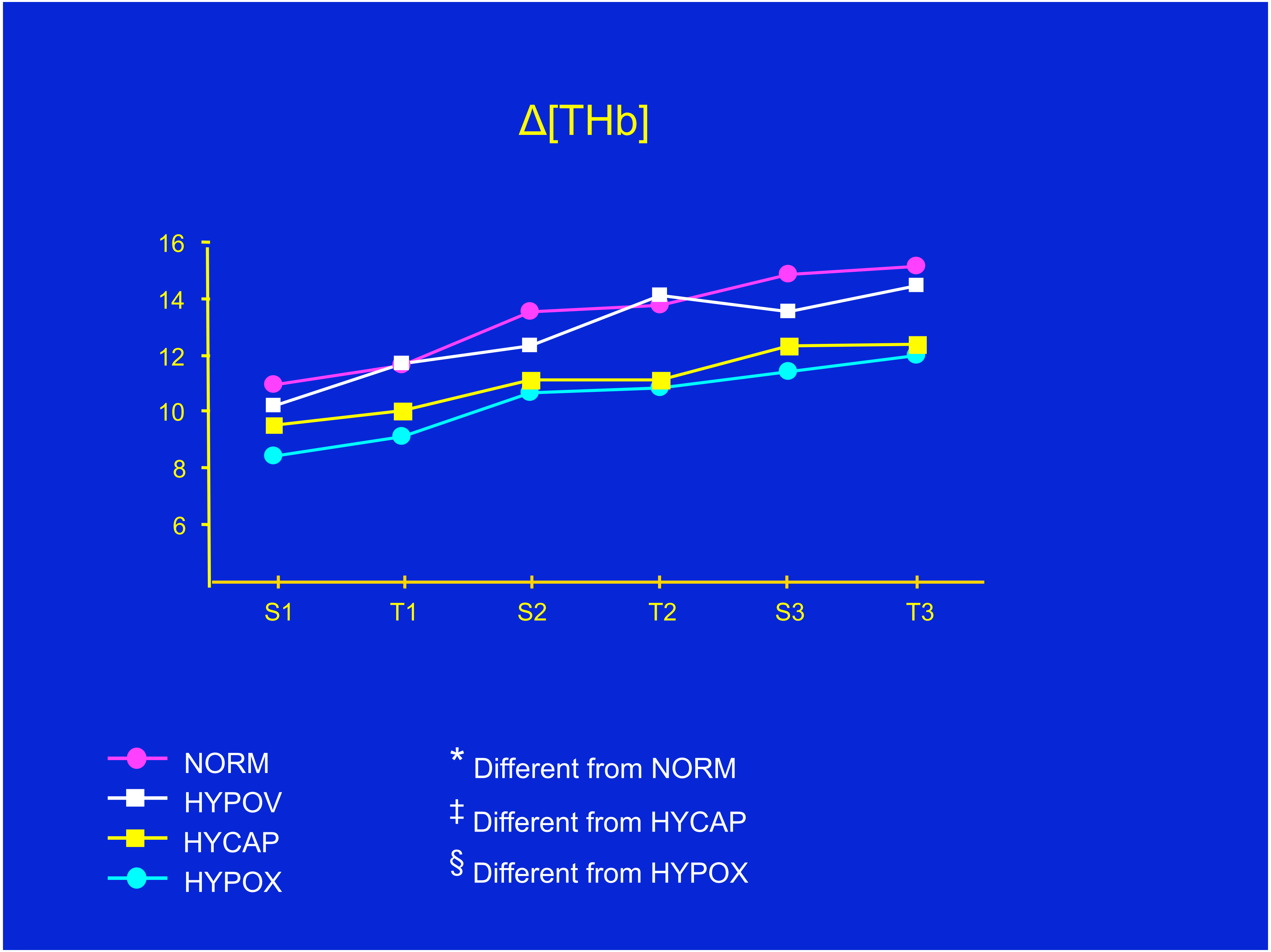
Δ[O2Hb]
10
8
6
4
2
0
S1
T1
S2
T2
S3
T3
*
*
*
NORM
HYPOV
HYCAP
HYPOX
* Different from NORM
‡ Different from HYCAP
§ Different from HYPOX



Δ[HHb]
10
8
6
4
2
0
S1
T1
S2
T2
S3
T3
‡ *
NORM
HYPOV
HYCAP
HYPOX
* Different from NORM
‡ Different from HYCAP
§ Different from HYPOX

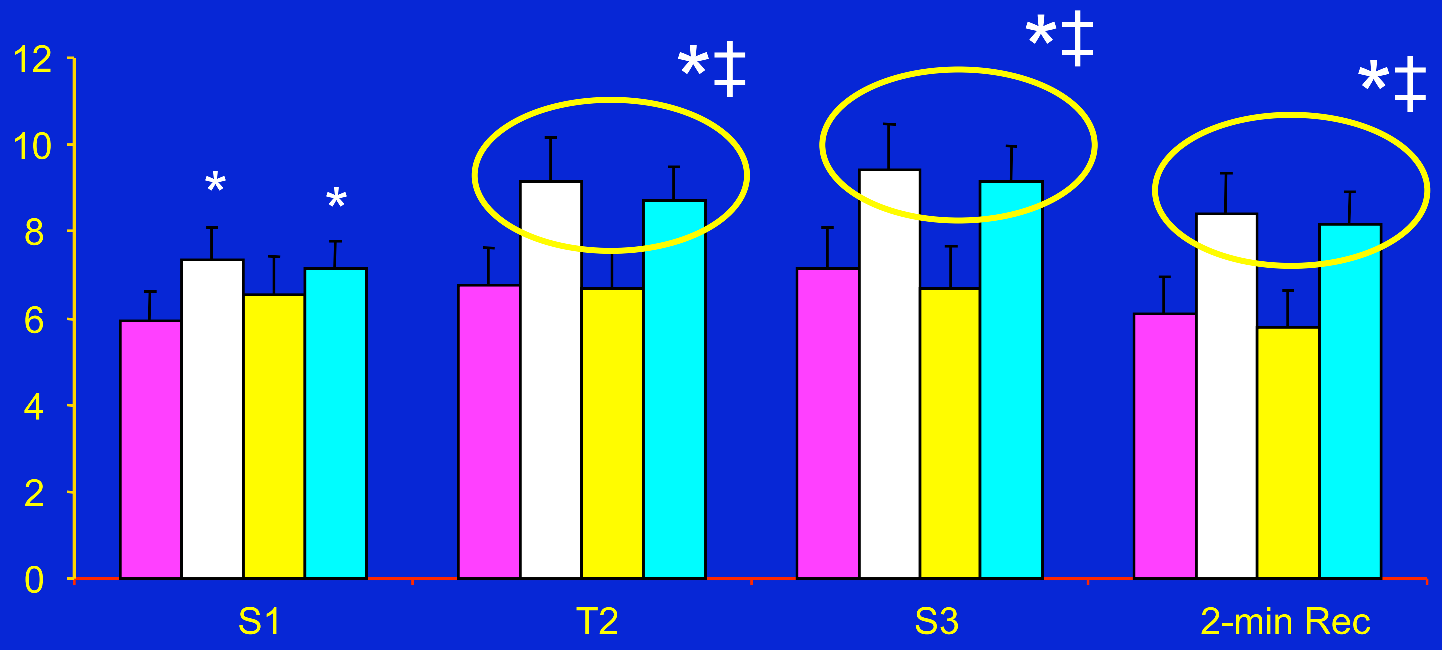


Δ[O2Hb -HHb]
4
2
0
-2
-4
-6
S1
T1
S2
T2
S3
T3
*
‡
NORM
HYPOV
HYCAP
HYPOX
* Different from NORM
‡ Different from HYCAP
§ Different from HYPOX



Δ[THb]
16
14
12
10
8
6
S1
T1
S2
T2
S3
T3
NORM
HYPOV
HYCAP
HYPOX
* Different from NORM
‡ Different from HYCAP
§ Different from HYPOX

Lactate (mmol/L)



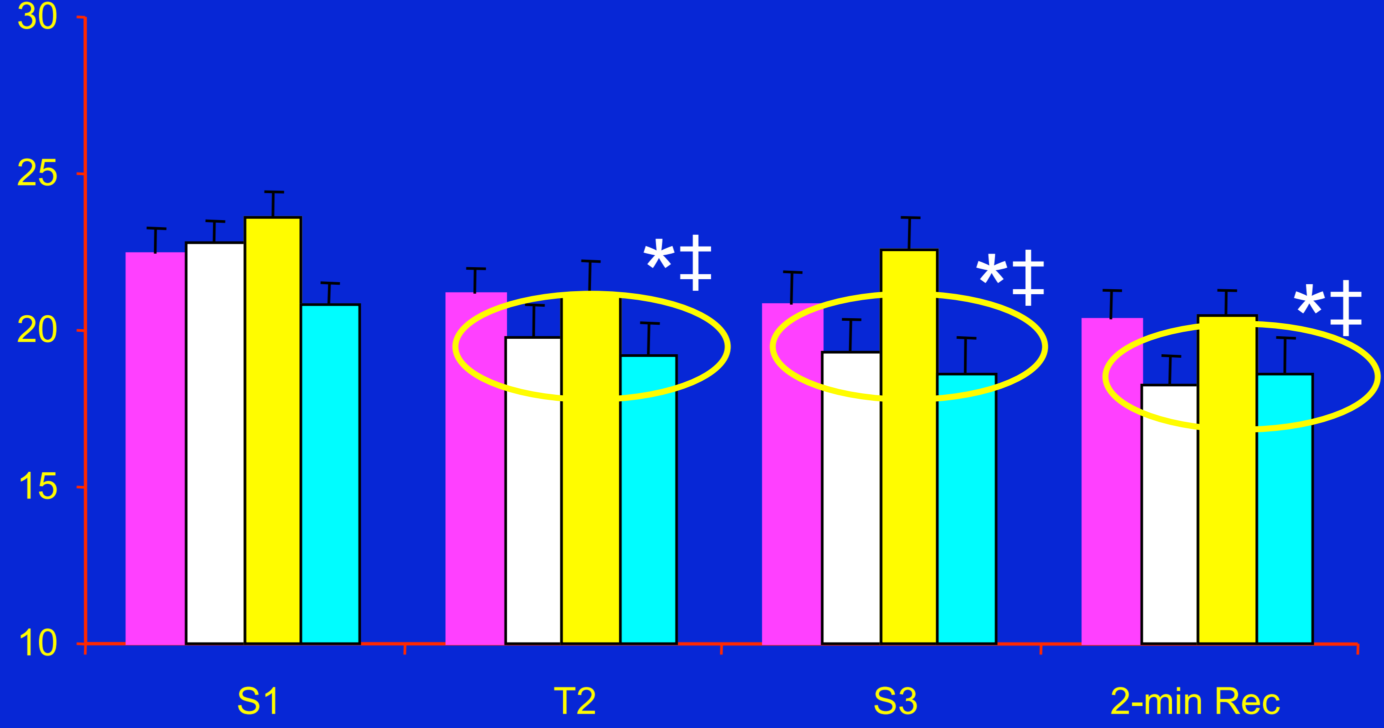
NORM
HYPOV
HYCAP
HYPOX

* Different from NORM

‡ Different from HYCAP

§ Different from HYPOX

HCO3 (mmol/L)



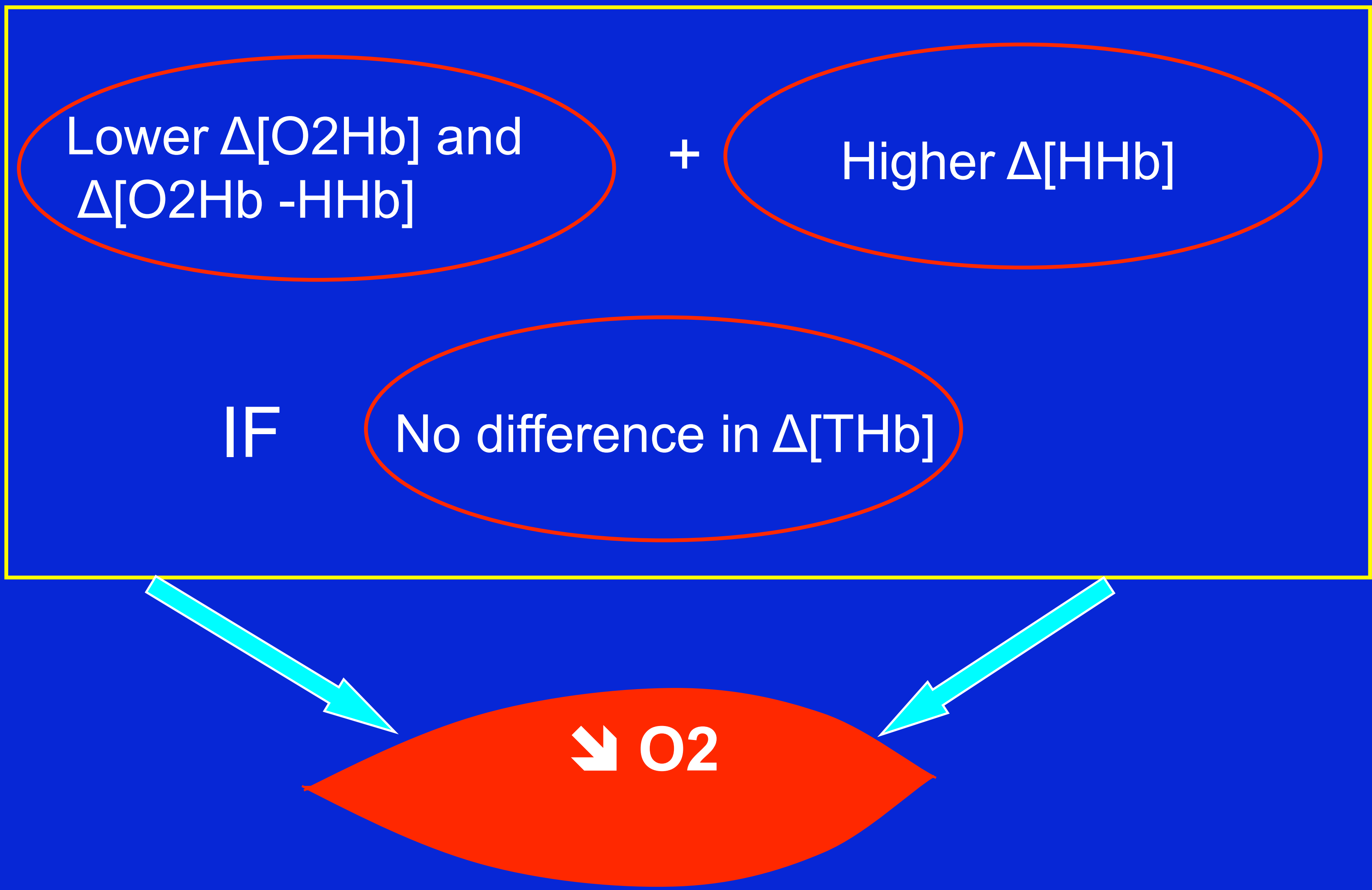
NORM
HYPOV
HYCAP
HYPOX

* Different from NORM

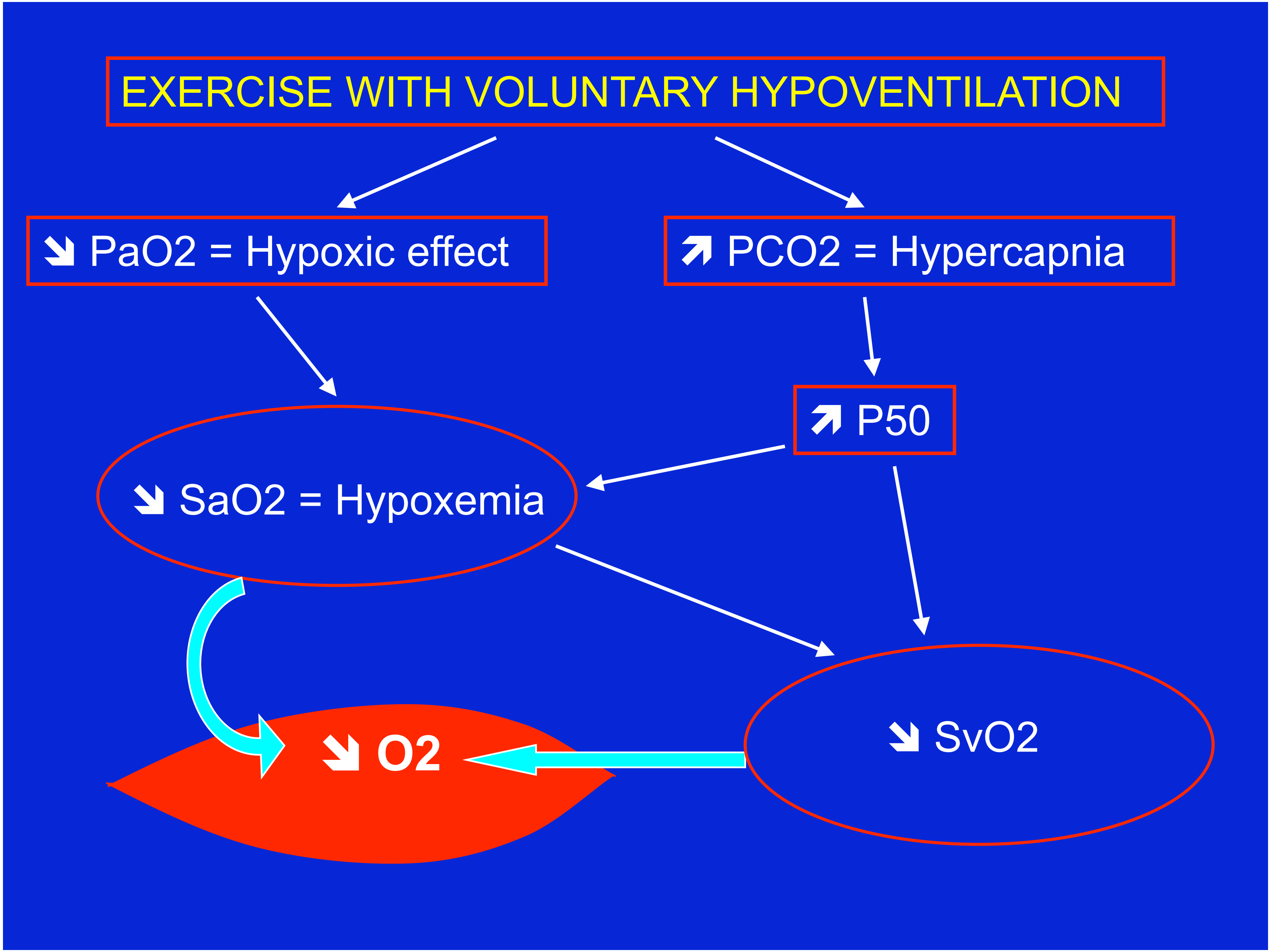
‡ Different from HYCAP

§ Different from HYPOX

First study reporting a lower muscle oxygenation during exercise with hypoventilation than with normal breathing



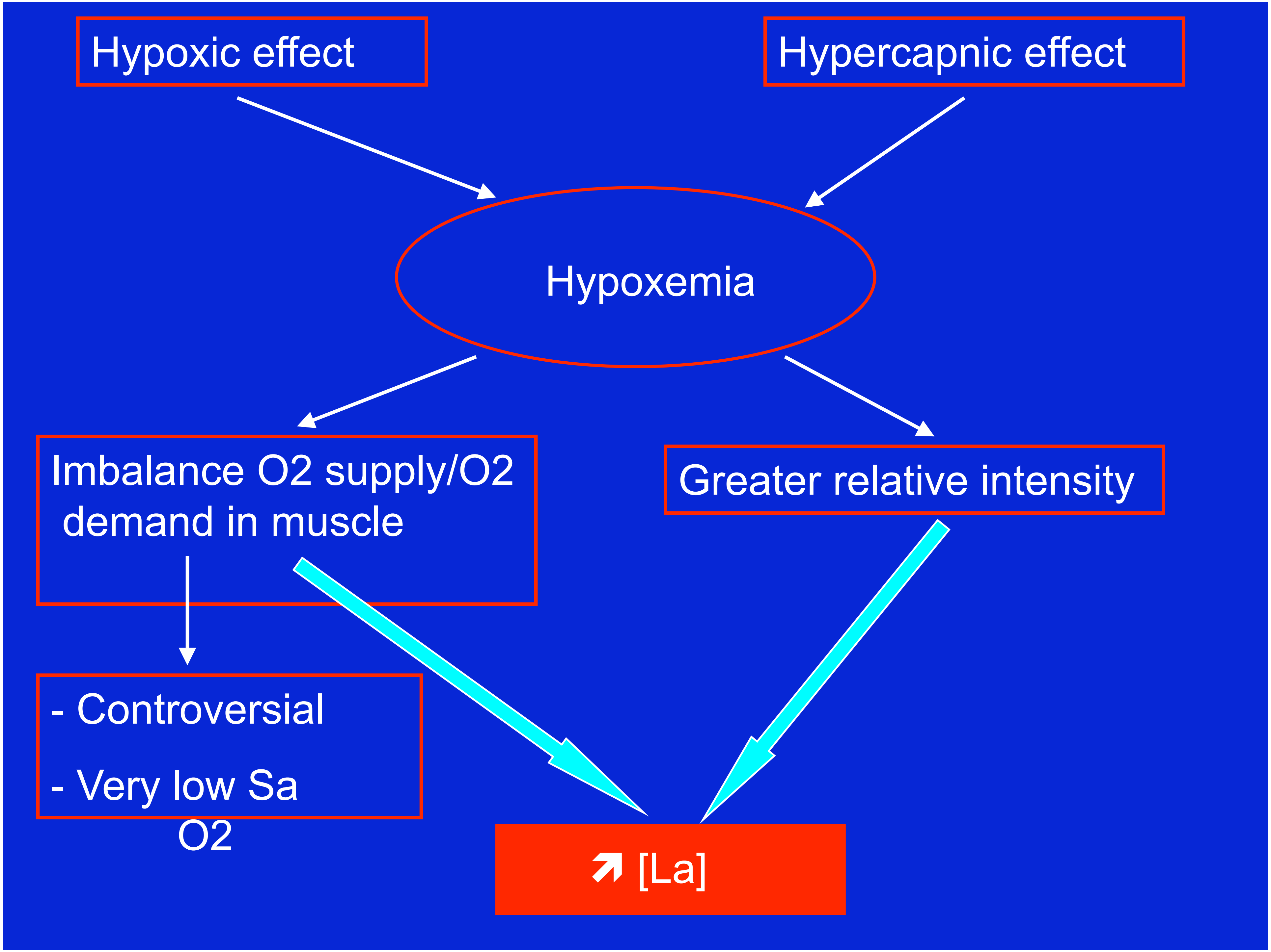
Why a lower muscle oxygenation?


EXERCISE WITH VOLUNTARY HYPOVENTILATION
↘ PaO2 = Hypoxic effect
↗ PCO2 = Hypercapnia
↗ P50
↘ SaO2 = Hypoxemia
↘ SvO2
↘ O2

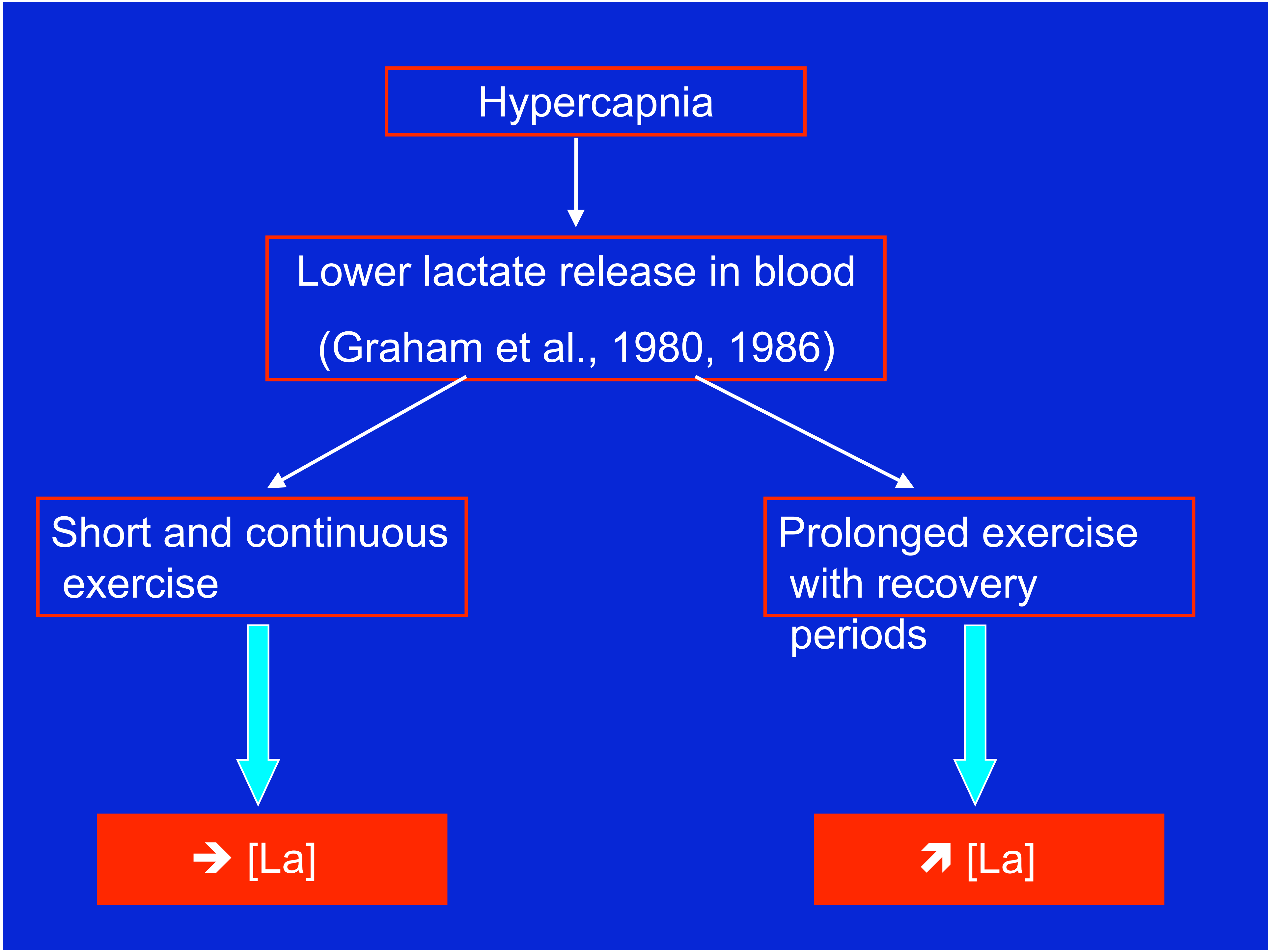
First study reporting a higher blood lactate concentration during exercise with hypoventilation than with normal breathing

⬇

Phenomenon involved ?


Hypoxic effect
Hypercapnic effect
Hypoxemia
Imbalance O2 supply/O2 demand in muscle
Greater relative intensity
- Controversial
- Very low Sa O2
↗ [La]

Why no change in [La] during short and continuous exercise?


Hypercapnia
Lower lactate release in blood
(Graham et al., 1980, 1986)
Short and continuous exercise
Prolonged exercise with recovery periods
➔ [La]
↗ [La]

CONCLUSION

- Exercise with hypoventilation increases muscle deoxygenation and blood lactate concentration
- Respective role played by hypoxic and hypercapnic effects
- For a same PaO2, lower muscle oxygenation during exercise with hypoventilation than in hypoxia

General conclusion

- Classical LH-TH method still needs some envidence to prove its efficiency
- LH-TL method has shown many evidence of efficiency for endurance sports
- Combination of techniques might be useful
- Voluntary hypoventilation could be a cheap alternative, but needs further investigations