Bull europ. Physiopath. resp.
1982, *18* (suppl. 4), 35-38

RED CELL FUNCTION ON MOUNT EVEREST

LA FONCTION DES GLOBULES ROUGES AU MONT EVEREST

R.M. Winslow *, M. Samaja **

ABSTRACT : Blood P_{50}, 2,3-DPG and acid-base status were studied in 20 Westerners during the American Medical Research Expedition to Everest. *In vivo* P_{50} was maintained on a narrow range (27.6-28.1 Torr) up to 6300 m because of the opposing effects of 2,3-DPG and respiratory alkalosis. At extreme altitude, the P_{50} decreased (18.5 Torr on the summit) due to the stronger effect of uncompensated respiratory alkalosis (pH 7.78). The net result was a preservation of arterial saturation in spite of falling Po_2.

Acid-base equilibrium ; 2,3-DPG ; hemoglobin concentration ; high altitude ; P_{50} ; red cells.

The erythropoietic system may contribute to high altitude acclimatization in at least two ways : increasing the red cell mass and shifting the blood oxygen equilibrium curve (OEC). Whereas modest increases in the red cell mass are beneficial in increasing the oxygen carrying capacity of blood, it has been recently speculated that excessive increases of hematocrit lead to exponential increases of blood viscosity, which, in turn, may lead to undesirable consequences [5]. As it regards the OEC, it is uncertain whether right or left shifts are beneficial, because only little univocal data in humans are available. Many data from previous high altitude expeditions must be rejected mainly because 2,3-DPG, which is a strong effector of the hemoglobin oxygen affinity, was not considered, and analyses were not performed on freshly drawn blood samples.

We have followed 20 westerners during the American Medical Research Expedition to Mount Everest, with the opportunity to collect hematologic data on fresh blood in the three laboratories of the Expedition : sea level, Basecamp (5 400 m) and Camp II (6 300 m). Additional data were collected on samples from the South Col of Mount Everest (8 050 m) after transfer to the expedition laboratory. We measured the hemoglobin concentration, RBC, hematocrit, the concentration of 2,3-DPG, the blood gases and the acid-base status by standard methods, and the P_{50} (the Po_2 at which hemoglobin is half-saturated with oxygen) by a new tonometry method which requires small amount of blood (up to 0.25 ml), little power consumption and is not affected by turbidity in the sample [2].

* Hemoglobinopathy Branch, Centers for Disease Control, Atlanta, Georgia 30333, USA.
** Fisiologia del Lavoro Muscolare del CNR, Milano, Italy.

Table I shows some of the results. As expected, the hemoglobin concentration, hematocrit and 2,3-DPG increased over the period of hypoxia. In particular, the increase of 2,3-DPG seems to be a rough function of pH, as previously outlined [3]. Unexpectedly, also MCHC increased, probably as a reflect of dehydration, but no apparent correlation was found between these parameters and the physical performance.

Table I. — Various measured parameters at different altitudes

	Sea level	Basecamp (5 400 m)	Camp II (6 300 m)	South Col (8 050 m)
[Hb] ($g \cdot dl^{-1}$)	14.5 ± 0.7 (18)	17.8 ± 1.0 (20)	18.8 ± 1.5 (19)	18.4 (2)
Hct	43.8 ± 2.3 (18)	50.8 ± 2.6 (20)	53.4 ± 4.0 (18)	52.0 (2)
MCHC ($g \cdot dl^{-1}$)	33.2 ± 1.1 (18)	35.1 ± 1.4 (20)	35.2 ± 1.2 (18)	35.3 (2)
[2,3-DPG]/[Hb]	0.84 ± 0.12 (18)	1.05 ± 0.90 (19)	1.04 ± 0.12 (17)	1.18 (2)
Base excess	−0.28 ± 1.22 (18)	— (0)	−8.7 ± 1.7 (15)	−5.9 (2)
Blood pH, *in vivo*	7.399 ± 0.017 (18)	— (0)	7.467 ± 0.34 (14)	7.552 (2)
P_{50} (Torr), *in vivo*	28.1 ± 1.1 (18)	— (0)	27.6 ± 2.2 (14)	24.9 (2)

Mean ± SD. Number of subjects studied in parentheses.

The value of the base excess, obtained using both the tonometry technique and the direct arterial measurement, suggests that the metabolic compensation for respiratory alkalosis is progressively less adequate as altitude increases, and probably is never complete at altitude, as it has been found in Andean natives [6]. Perhaps, as suggested by MONGE *et al.* [1], there is a new « set-point » for HCO_3^- reabsorption by renal tubules at high altitude.

Blood P_{50} values were adjusted to *in vivo* pH using the Bohr factor of −0.45, corresponding to a [2,3-DPG]/[Hb] ratio of 1.25 [3]. It is worthwhile to note that the P_{50} measured experimentally is highly comparable to that predicted by the use of the equations reported in [7], indicating that no other parameters besides H^+, CO_2 and 2,3-DPG need be postulated in the oxygen affinity regulation. The slight deviation of P_{50} (< 1 Torr) observed at Camp II can be accounted for by increased MCHC.

Since alveolar gas was measured in one climber on the summit of Mount Everest [4], if the base excess is assumed to be the same as it was 12 h after

his descent to 8 050 m, and if P_{ACO_2} is assumed to be the same as P_{aCO_2}, then the *in vivo* blood pH would have been 7.69, with a corresponding P_{50} value of 19.8 Torr.

Our data suggest that P_{50} is maintained over a very narrow range up to an altitude of 6 300 m, because of the opposing effects of increased 2,3-DPG and pH. Above 6 300 m, the OEC shifts progressively to the left due to the stronger effect of uncompensated alkalosis. Acclimatization above that altitude has not been studied and therefore it is difficult to state whether this is a reflect of poor acclimatization. However, the progressive shift to the left of the OEC suggests that the intuitive climbing strategy of acclimatizing 1-2 days below the summit, then attempting the summit in sudden pushes, may be a good one.

REFERENCES

1. MONGE (C.C.), LOZANO (R.), CARCELEN (A.). — Renal excretion of bicarbonate in high altitude natives and in natives with chronic moutain sickness. *J. clin. Invest.*, **1964**, **43**, 2303-2309.
2. SAMAJA (M.), ROVIDA (E.). — A new method to measure the hemoglobin oxygen saturation by the oxygen electrode. *J. biophys. biochem. Meth.*, 1983, in press.
3. SAMAJA (M.), WINSLOW (R.M.). — The separate effect of H^+ and 2,3-DPG on the oxygen equilibrium curve of human blood. *Brit. J. Haemat.*, 1979, **41**, 373-381.
4. WEST (J.B.), HACKETT (P.H.), MARET (K.H.), MILLEDGE (J.S.), PETERS (R.M.), PIZZO (C.J.), WINSLOW (R.M.). — Pulmonary gas exchange on the summit of Mt Everest. *J. appl. Physiol. : Resp. environm. Exercise Physiol.*, submitted.
5. WINSLOW (R.M.). — Hypoxia and polycythemia : the « optimal » hematocrit. *In* : Hypoxia : Man at altitude. J.R. Sutton *et al.* eds. Thieme-Stratton, New York, 1982, pp. 40-42.
6. WINSLOW (R.M.), MONGE (C.C.), STATHAM (N.J.), GIBSON (C.G.), CHARACHE (S.), WHITTEMBURY (J.), MORAN (O.), BERGER (R.L.). — Variability of oxygen affinity of blood : human subjects native to high altitude. *J. appl. Physiol. : Resp. environm. Exercise Physiol.*, 1981, **51**, 1411-1416.
7. WINSLOW (R.M.), SAMAJA (M.), WINSLOW (N.J.), ROSSI-BERNARDI (L.), SHRAGER (R.I.). — Simulation of the continuous blood O_2 equilibrium curve over physiological pH, DPG, and P_{CO_2} range. *J. appl. Physiol. : Resp. environm. Exercise Physiol.*, **1983**, **54**, 524-529.

RÉSUMÉ : Nous avons étudié la P_{50}, le 2,3-DPG et l'équilibre acido-basique du sang chez 20 occidentaux qui ont participé à l'expédition au mont Everest de l'American Medical Research. La P_{50} *in vivo* reste dans des limites étroites (de 27,6 à 28,1 Torr) jusqu'à 6 300 m, en raison des effets opposés du 2,3-DPG et de l'alcalose respiratoire. En altitude extrême, la P_{50} baisse (18,5 Torr au sommet), en raison de l'effet intense d'une alcalose respiratoire non compensée (pH 7,78), ce qui entraîne la préservation de la saturation artérielle malgré la chute de P_{O_2}.