

References

1. Imagawa S, Yamaguchi Y, Higuchi M, et al. Levels of vascular endothelial growth factor are elevated in patients with obstructive sleep apnea-hypopnea syndrome. *Blood*. 2001;98:1255-1257.
2. Eckardt K-U, Boutellier U, Kurtz A, Schopen M, Koller EA, Bauer C. Rate of erythropoietin formation in humans in response to acute hypobaric hypoxia. *J Appl Physiol*. 1989;66:1785-1788.
3. Guilleminault C, Connolly SJ, Winkle RA, et al. Cardiac arrhythmia and conduction disturbances during sleep in 400 patients with sleep apnea syndrome. *Am J Cardiol*. 1983;52:490-494.
4. Weitzenblum E, Krieger J, Apprill M, et al. Daytime pulmonary hypertension in patients with obstructive sleep apnea syndrome. *Am Rev Respir Dis*. 1988;138:345-349.
5. Minchenko A, Bauer T, Salceda S, Caro J. Hypoxia stimulation of vascular endothelial growth factor expression in vivo and in vitro. *Lab Invest*. 1994;71:374-379.

To the editor:

Oxygen saturation in the bone marrow of healthy volunteers

In recent years there has been a great deal of progress in understanding the homeostasis of different tissue microenvironments, and this has been particularly true of the hematopoietic system. Insights into the interaction of the microenvironmental stromal cells of the bone marrow with the blood-forming stem cells and their progeny have been substantial and have led to clinical applications. An example of this is the use of hematopoietic growth factors for the treatment of peripheral blood cytopenias and other hematologic disorders. Understanding the basic biology of the bone marrow has led to such therapies. The hemopoietics, including recombinant granulocyte colony-stimulating factor, granulocyte-macrophage colony-stimulating factor, and interleukin-11, have been cloned from the stromal cells and introduced into clinical use.¹ Recently, our laboratory and others have been examining the response of the bone marrow to hypoxia, which is an experimental model of hemorrhagic shock.² In addition, it has been demonstrated that hematopoietic growth factors signal, in part, through the formation of reactive oxygen species.³ However, to date, there have been no reports in the scientific literature regarding the partial pressure of oxygen (pO₂) and the oxygen saturation in the normal marrow microenvironment. Such information is important for interpreting the current literature.

In order to establish a baseline for comparison in experimental conditions and to design the appropriate research models, we performed gas analysis in a series of 5 healthy volunteer bone marrow donors. All subjects underwent posterior superior iliac crest bone marrow aspiration using an Illinois aspiration needle, after obtaining informed consent in accordance with a clinical protocol approved by the institutional review board of the University of Medicine and Dentistry of New Jersey. Donors were aged 24 to 46, and were all in excellent health. Specimens were obtained using aseptic technique, and marrow was aspirated into a blood gas syringe. Specimens were brought on ice to the laboratory and analyzed using a CIBA-Corning Corporation Model 278 blood gas

analyzer (East Walpole, MA). The mean pO₂ of the marrow aspirates was 54.9 mm Hg ± 0.98, with mean O₂ saturation of 87.5% ± 1.1%. Peripheral blood mean O₂ saturation was obtained by pulse oximeter at the same time, with a mean O₂ saturation of 99%. The marrow cavity is sinusoidal, and anatomically the fluid flowing through the marrow cavity is a mixture of arterial and venous blood. A search of the National Library of Medicine database (Medline) yielded a single prior report of marrow gas analysis; however, this was a study of 82 men, all with chronic pulmonary disease, and no healthy subjects were included as controls. In this group, the mean pO₂ in the marrow was 48 mm Hg (range 33.5 to 64.1 mm Hg).⁴ The pO₂ and oxygen saturation of the marrow in the normal state is a critical reference point for determining the effects of hypoxemia upon the bone marrow microenvironment both in experiment conditions and in clinical situations. We are continuing to investigate the effects of hypoxemia in experimental and clinical settings, but identifying and reporting the normal oxygen content of the marrow microenvironment here provides documentation of the baseline for comparison in future laboratory and clinical studies.

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References

1. Du X, and Williams D. Interleukin-11: review of molecular, cell biology and clinical use. *Blood*. 1997;89:3897-3908.
2. Quinlan Jr D, Rameshwar P, Qian J, et al. Effect of hypoxia on the hematopoietic and immune modulator preprotachykinin-1. *Arch Surg*. 1998;133:1328-1334.
3. Sattler M, Winkler T, Verma S, et al. Hematopoietic growth factors signal through the formation of reactive oxygen species. *Blood*. 1999;93:2928-2935.
4. Skouby A. Haematologic adaptation in patients with chronic bronchitis and pulmonary insufficiency. *Acta Med Scand*. 1976;199:185-190.