MEASUREMENT OF TISSUE OXYGEN TENSION: COMPARISON BETWEEN TWO SUBCUTANEOUS OXYGEN TONOMETERS

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ABSTRACT. We compared the 95% response time (95% RT) of two tissue oxygen tonometers under two sets of circumstances. We first evaluated the devices during normoxia, hyperoxia, and anoxia in vitro, using a transcutaneous PO2 electrode (PtcO2) as the reference. The responses to normoxia and to different grades of hyperoxia were examined in vivo in 8 healthy volunteers to assess the relationship between changes in subcutaneous PO2 and PtcO2, an estimate of arterial PO2 (PaO2). One subcutaneous method (ScA) used a technique based on a polarographic needle electrode in situ connected to an ammeter; the second method (ScB) was based on a blood gas analyzer system first described by Hunt (Lancet 164; 2:1370). ScA and PtcO2 both responded to stepwise changes in ambient oxygen concentration (21–100%) in vitro within 10 seconds; the 95% RT of ScA was 1.39 ± 0.5 to 2.39 ± 0.8 minutes and that of PtcO2 was 0.32 ± 0.1 to 0.49 ± 0.1 minutes. ScB had a lag of 3 minutes, and the 95% RT was 6.75 ± 0.5 to 8.2 ± 0.8 minutes. In contrast to the results in vitro, the response of ScA to changes in FiO2 in vivo was delayed compared with the rapid response of PtcO2, reflecting the physiologic delay of tissue PO2 in response to increased PaO2. The time lag and the long 95% RT of ScB were even more evident in vivo. ScA reacted three to four times faster than ScB, both in vitro and in vivo, to changes in the oxygen environment. The in vitro 95% RT of ScA to changes in ambient oxygen varied from 2 to 3.5 minutes. In contrast, the 95% RT of ScB was 8 to 9 minutes. PtcO2 had the fastest 95% RT, from 0.4 to 0.5 minutes. The results suggest that the subcutaneous needle electrode method (ScA) provides close to real-time assessment of tissue PO2.


Tissue O2 tonometry can be used to assess tissue perfusion [1–6] as well as the balance between oxygen delivery and demand. The oxygen tension in the extracellular tissue fluid is sensitive to small changes in local blood flow and to oxygen consumption. Tissue PO2 (PtO2) measurements have been used to evaluate oxygen transport during and after surgery [7–10], and changes in regional oxygenation in various shock models [12–19] and postoperative wound healing [1–3,6]. Most studies of PtO2 in shock and acute alterations of perfusion have applied the technique originally described by Hunt [1], which uses a silastic tube as the diffusion surface and a standard blood gas analyzer for the measurement of PtO2. The technical features of this setup potentially limit its value in evaluating dynamic changes in PtO2 and may lead to misinterpretation of PtO2 during the rapid changes of oxygen transport that occur after major surgery and in various types of shock [7–19]. This is supported by observations of slow and delayed alterations in PtO2 observed after major acute changes of oxygen supply [12–19]. We modified the original tech-
Subcutaneous \( \text{PtO}_2 \) was simultaneously measured with two techniques (Fig 1). The modified technique (ScA) consisted of a platinum needle electrode (cathode) (760; Diamond Electro-Tech, Inc, Ann Arbor, MI) inserted into one end of a silastic tube 8 cm in length (ID 0.8 mm, OD 1.3 mm; Codman and Shurtleff Inc, Randolph, MA) and an Ag-AgCl anode inserted into the other end through an infusion cannula (Viggo Venflon; Viggo Ab, Helsingborg, Sweden). The distance between the electrodes within the tube was 3 to 4 cm. The tube was filled with normal saline via the sideport of the infusion cannula. The polarization voltage was 0.67 V. The current produced by oxygen reduction, measured with an ammeter (Orange 1, Orange Medical Instruments, Costa Mesa, CA, currently available from Biomedical Sensors Ltd, High Wycomb, UK), is proportional to the surrounding \( \text{PO}_2 \). A two-point calibration was performed with an anoxic solution (Radiometer S4150 \( \text{PO}_2 \) Zero Solution, Copenhagen, Denmark) and in aerated, room temperature, normal saline before insertion. The resolution is 1 mm Hg, and the baseline drift is \( \pm 1 \) mm Hg/h. The ScB method consisted of a Silastic tube 12 cm in length similar to and implanted identically as ScA. The tube was perfused with anoxic, room temperature, normal saline at a rate of 3.0 ml/hr. The fluid was then led with a pair of 35-cm-long thin nylon tubes (ureter catheter 223606, ID 0.8 mm; Ruesch, Waiblingen, Germany) to a micro blood gas analyzer (BMS3, Mk2; Radiometer).

A two-point gas calibration of the blood gas analyzer was done according to the manufacturer's recommendations. The resolution is 0.1 mm Hg, amplifier drift is 0.1% to 0.3% of reading/\( ^\circ \)C, and 99% RT of the \( \text{O}_2 \) electrode itself at 37\( ^\circ \)C with a polypropylene membrane is 30 seconds, according to the manufacturer's specifications. Similarly, the \( \text{PtcO}_2 \) monitor (TINA, Electrode E5277; Radiometer) was calibrated with two certified gases. Baseline drift is within \( \pm 1 \) mm Hg/h. The 90% RT of the \( \text{O}_2 \) electrode is 20 seconds. The electrode placement site was the anterior chest wall, and the electrode temperature was kept at 44.5\( ^\circ \)C.

**Experimental Protocol In Vitro**

The two tonometers and the PtcO\(_2\) electrode were implanted into standard ventilator tubing (3 cm OD, 90 cm length) (Fig 1). A constant flow of gas was led into the tubing, and the fraction of oxygen (\( \text{FO}_2 \)) in the gas was confirmed with a gas analyzer (Cardiocap gas monitor; Datex, Helsinki, Finland). The \( \text{FO}_2 \) was changed stepwise from 0.21 to 0.5, 1.0, back to 0.21, and thence to 0 oxygen at 10-minute intervals. Anoxia was produced in the gas circuit by pure nitrogen gas. The measurements were repeated five times. After 15 minutes of stabilization at \( \text{FO}_2 \) 0.21, data sets were collected every 5 seconds for 1 minute and thereafter at 30-second intervals.

**Experimental Protocol In Vivo**

The in vivo experiments were carried out in 8 healthy male volunteers aged 26 to 34 years. Informed consent was obtained, and the protocol was approved by the Institutional Review Board. Subcutaneous \( \text{PtO}_2 \) was measured with two techniques (ScA and ScB; Fig 1) during normoxia and different grades of hyperoxia,