Evaluation of a combined transcutaneous carbon dioxide pressure and pulse oximetry sensor in adult sheep and dogs

Rainer Vogt, DVM; Roman Rohling, MD; Sabine Kästner, DVM

**Objective**—To evaluate a combined transcutaneous carbon dioxide pressure (tcPco2) and pulse oximetry sensor in sheep and dogs.

**Animals**—13 adult sheep and 11 adult dogs.

**Procedures**—During inhalation anesthesia, for the first 10 minutes following sensor placement, arterial blood gas was analyzed and tcPco2 was recorded every 2 minutes. Subsequently, the animals were hyper-, normo-, and hypoventilated. The simultaneously obtained tcPco2 and Paco2 values were analyzed by use of Bland-Altman statistical analysis.

**Results**—Mean ± SD overall difference between tcPco2 and Paco2 10 minutes after sensor application was 13.3 ± 8.4 mm Hg in sheep and 8.9 ± 12 mm Hg in dogs. During hyper-, normo-, and hypoventilation, mean difference (bias) and precision (limits of agreement) were 13.2 ± 10.4 mm Hg (limits of agreement, –7.1 and 33.5 mm Hg) in sheep and 10.6 ± 10.5 mm Hg (limits of agreement, –9.9 and 31.2 mm Hg) in dogs, respectively. Changes in Paco2 induced by different ventilation settings were detected by the tcPco2 sensor with a lag (response) time of 4.9 ± 3.5 minutes for sheep and 6.2 ± 3.6 minutes for dogs.

**Conclusions and Clinical Relevance**—The tcPco2 sensor overestimated Paco2 in sheep and dogs and followed changes in Paco2 with a considerable lag time. The tcPco2 sensor might be useful for noninvasive monitoring of changes but cannot be used as a surrogate measure for Paco22 (Am J Vet Res 2007;68:265–270).

**Abbreviations**

<table>
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<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>Spo2</td>
<td>Arterial oxyhemoglobin saturation by pulse oximetry</td>
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<tr>
<td>SaO2</td>
<td>Arterial oxygen saturation</td>
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<tr>
<td>tcPco2</td>
<td>Transcutaneous carbon dioxide pressure</td>
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<tr>
<td>EtcO2</td>
<td>End-tidal carbon dioxide partial pressure</td>
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Pulse oximetry used for determination of Spo2, a standard noninvasive technique used in veterinary anesthesia, provides continuous information regarding the patient’s SaO2. A disadvantage of the sole use of pulse oximetry is the lack of information regarding CO2 status. Knowledge of SaO2 and Paco2 in a patient is essential for determination of the adequacy of ventilation and oxygenation. Presently, periodic blood sampling is still required to assess Paco2, so a single noninvasive sensor, yielding continuous information on both oxygenation and ventilation, is highly desirable. The use of a combined Spo2 and tcPco2 sensor was first reported in 1999.1 The digital sensor combines the basic elements of a Severinghaus-type tcPco2 sensor and those of a reflectance pulse oximeter sensor. A built-in mixed-signal microcontroller located in the sensor head amplifies, digitizes, and analyzes the measured signals at the measurement site and sends the processed digital signal to the main monitor unit. The Pco2 is measured potentiometrically by determining the pH of an electrolyte solution. This electrolyte solution is separated from the skin by a membrane that is highly permeable to CO2. The measured changes in pH of the electrolyte solution are proportional to the logarithm of the changes in Pco2. The sensor is warmed to a surface temperature of 41°C to enhance CO2 diffusion and blood perfusion locally. The Spo2 and the pulse frequency are derived through digital signal processing of the photoplethysmogram.1

In the present study, the objective was to evaluate the digital sensor in sheep and dogs by determination of the sensor time to steady state and the ability to monitor arterial blood gas changes during different stages of mechanical ventilation.

**Materials and Methods**

**Animals and instrumentation**—Thirteen adult sheep and 11 adult dogs were used in this study. The sheep (2 rams and 11 ewes) were a variety of Swiss breeds, weighed (mean ± SD) 76 ± 9 kg, and were 3.5 ± 1.2 years old. The dogs (7 males and 4 females)
were of mixed breeds or of a variety of pure breeds, weighed 33 ± 10 kg, and were 6.2 ± 4.3 years old. All animals were client-owned and were classified as completely healthy or having mild systemic disease and underwent nonemergency orthopedic surgery (eg, repair of cruciate ligaments). The study was performed according to Swiss Federal Law concerning animal welfare. In all animals, food was withheld for 12 hours before surgery. For preanaesthetic medication, the sheep were given buprenorphine (10 µg/kg, IM) and xylazine (0.1 mg/kg, IM) and the dogs received buprenorphine (7 µg/kg, IM) and acepromazine (0.03 mg/kg, IM) half an hour prior to induction. Anesthesia was induced with ketamine (2 mg/kg, IV) and diazepam (0.1 mg/kg, IV) in sheep and propofol (4 mg/kg, IV) in dogs. Following endotracheal intubation, the animals were connected to a circle anesthetic circuit. Balanced anesthesia was maintained with isoflurane in 100% oxygen (10 mL/kg/min) and ketamine (10 µg/kg/min, IV) in sheep and fentanyl (10 µg/kg/h, IV) in dogs. Mechanical ventilation was performed with a tidal volume of 6 to 11 mL/kg and a respiration rate (6 to 22 breaths/min) according to the study protocol to achieve the desired \( \text{EtCO}_2 \) for normoventilation (35 to 50 mm Hg), hypoventilation (50 to 90 mm Hg), and hyperventilation (20 to 30 mm Hg).

For arterial blood sampling and invasive blood pressure monitoring, an arterial catheter was placed in the auricularis caudalis artery (sheep) or the metatarsalis lateralis artery (dogs). Blood gas samples were collected with a self-filling arterial sampler,\(^8\) capped, placed on ice, and analyzed within 60 minutes with a conventional blood gas analyzer.\(^9\) The analyzer was calibrated automatically and checked once daily.

The combined \( \text{tcPCO}_2 \) and \( \text{SpO}_2 \) sensor was applied to the animal’s ear. For this purpose, the inside proximal third of the ear was razor-shaved and a special sticky clip\(^9\) was mounted and additionally secured with cyanoacrylate to maintain the sensor's airtight cutaneous seal. The sensor was attached to the clip after placing a drop of sensor gel\(^9\) on the sensor membrane. The electrolyte and membrane of the sensor were replaced every 10 days. Before every use, automatic calibration was triggered by taking the sensor out and placing it back into the docking station.

Anesthesia of all sheep and dogs was routinely monitored via temperature (nasal [in sheep] and esophageal [in dogs]), ECG, invasive blood pressure monitoring, pulse oximetry, heart rate, \( \text{EtCO}_2 \), spirometry, and anesthetic gas analysis continuously with a patient monitor.\(^9\) Recordings were made every 5 minutes unless specified (ie, 2 minutes).

Nasal or esophageal temperature, mean arterial pressure, \( \text{EtCO}_2 \), heart rate derived from the ECG, \( \text{tcPCO}_2 \), \( \text{SpO}_2 \) derived from the sensor, and pulse frequency derived from the plethysmogram of the sensor were recorded at every measurement point. The analog signal outputs of the sensor docking station (\( \text{tcPCO}_2 \), \( \text{SpO}_2 \) derived from the sensor, and pulse frequency derived from the plethysmogram of the sensor) were recorded every 2 seconds with an analog digital signal-processing circuit\(^9\) and a personal computer.\(^9\)

**Experimental protocol**—After anesthetic induction, the animals were mechanically ventilated and normoventilation (\( \text{EtCO}_2 \) 35 to 45 mm Hg) was achieved by controlling the ventilator’s respiration rate. The following baseline values were recorded: \( \text{PaO}_2 \), \( \text{PaCO}_2 \), \( \text{SaO}_2 \), \( \text{EtCO}_2 \), heart rate derived from the ECG, mean arterial blood pressure, and temperature. After 13 minutes of normoventilation, a second set of baseline values was obtained. When \( \text{EtCO}_2 \) was from 35 to 45 mm Hg, \( \text{EtCO}_2 \) values varied < 4 mm Hg during the last 15 minutes, temperature was > 36°C, and mean arterial blood pressure was > 60 mm Hg, the experiment was started. In the first study cycle, the initial time following sensor placement required for the sensor to accurately estimate \( \text{PaCO}_2 \) (time to steady state) was investigated. For this measurement, the transcutaneous sensor was mounted in the previously attached clip. Sensor output (\( \text{SpO}_2 \) derived from the sensor, \( \text{tcPCO}_2 \), and pulse frequency derived from the plethysmogram of the sensor) and the physiologic variables measured with the monitor (\( \text{EtCO}_2 \), heart rate derived from the ECG, mean arterial blood pressure, and temperature) were recorded, and blood gas samples were collected at 2-minute intervals for 10 minutes.

Following the first study sequence, the animals were successively hyper-, normo-, and hypoventilated to achieve \( \text{PaCO}_2 \) from 20 to 30 mm Hg, 35 to 50 mm Hg, and 50 to 90 mm Hg, respectively. Tidal volume was kept constant, the respiratory frequency was changed to achieve the desired \( \text{EtCO}_2 \), and each ventilation setting was continued for 15 minutes. At 5-minute intervals, blood gas samples were collected and sensor output (\( \text{SpO}_2 \) derived from the sensor, \( \text{tcPCO}_2 \), and pulse frequency derived from the plethysmogram of the sensor) and the physiologic variables measured with the monitor were simultaneously obtained.

After finishing each experimental sequence, the quality of the pulse oximetry and transcutaneous signal outputs from the sensor were recorded as good or poor (on the basis of the measured plethysmographic signal amplitude) and a note was made of signal consistency or any signal disturbances.

**Statistical analysis**—Descriptive statistics were applied to analyze the biometric and clinical data.\(^1\) The time to steady state and delay (response time) of the sensor were investigated with curve-fitting calculations. Sensor time to steady state for \( \text{tcPCO}_2 \) was defined as the time until the \( \text{tcPCO}_2 \) value was approximately 90% of its steady-state plateau value after sensor application. For this purpose, all \( \text{tcPCO}_2 \) values of each animal during the first part of the study were curve-fitted with the function:

\[
f = A \cdot (1 - e^{-X/T})
\]

where \( A \) is the value of the horizontal asymptote of the function, \( X \) is time, and \( T \) is the time constant. The time constant multiplied by 2.3 is equivalent to 90% of the constant \( A \). The mean of all individual time constant values was calculated and multiplied by 2.3 to obtain the desired mean sensor time to steady state for \( \text{tcPCO}_2 \) in the sheep and dogs.\(^2\)

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Mean sensor response time (ie, the delay in detection of changes in P\textsubscript{CO}\textsubscript{2}) was calculated by measuring the time shift between the tcP\textsubscript{CO}\textsubscript{2} curve (from the data record, measured at 2-second intervals) and the Pa\textsubscript{CO}\textsubscript{2} curve (polynomial of fifth degree function through the 5-minute interval arterial blood gas values).

The P\textsubscript{CO}\textsubscript{2} bias (mean values of tcP\textsubscript{CO}\textsubscript{2} – Pa\textsubscript{CO}\textsubscript{2}) was calculated by use of measurement data from the end points of each 15-minute ventilation setting. Paired Student t tests and Bland-Altman statistics\textsuperscript{1,9} were used to compare the degree of agreement between results of arterial blood gas analyses and the noninvasive tcP\textsubscript{CO}\textsubscript{2} measurements.\textsuperscript{1,9} The Bland-Altman analysis was used for comparison between tcP\textsubscript{CO}\textsubscript{2} and Pa\textsubscript{CO}\textsubscript{2}. This analysis overcomes the limitations of traditional linear regression analysis, which reveals correlations but not interchangeability of the 2 coincident methods of measurement. Mean ± SD values are reported. For all analyses, P < 0.05 was considered significant.

Results

The sensor was evaluated in 13 sheep, although 3 sheep were excluded from the data set. One was excluded because of a sensor fault and the other 2 because of problems unrelated to the study. The sensor was evaluated in 11 dogs. Two dogs had to be excluded completely from the study because the surgeon was leaning on the sensor, which caused high sensor contact pressure and substantial signal drift. In 1 dog, the second part of the experimental protocol was not performed. For the purpose of statistical analysis, the study included 10 sheep for each experimental setting, 9 dogs for evaluation of sensor time to steady state, and 8 dogs for evaluation of sensor accuracy.

Relevant biometric data were determined (Table 1). Among all measurements, continual measurements of tcP\textsubscript{CO}\textsubscript{2} were obtained in 92% of sheep measurements and 100% of dog measurements.

After the initial sensor application, the tcP\textsubscript{CO}\textsubscript{2} value of the sensor began to increase and required some time to reach the steady-state plateau value. In both species, the sensor occasionally overestimated tcP\textsubscript{CO}\textsubscript{2}, compared with Pa\textsubscript{CO}\textsubscript{2}. Ten minutes after sensor application, the overall difference between tcP\textsubscript{CO}\textsubscript{2} and Pa\textsubscript{CO}\textsubscript{2} was 13.3 ± 8.4 mm Hg in sheep and 8.9 ± 12 mm Hg in dogs. This difference was significant (P = 0.007) in sheep and not significant (P = 0.039) in dogs.

The sensor’s tcP\textsubscript{CO}\textsubscript{2} time to steady state was calculated from the data obtained after application of the sensor until the tcP\textsubscript{CO}\textsubscript{2} measurement reached the steady-state plateau value during anesthesia with normocapnia (Et\textsubscript{CO}\textsubscript{2}, 35 to 45 mm Hg), normal temperature (>36°C), and normal blood pressure (mean arterial blood pressure > 60 mm Hg). The sensor mean tcP\textsubscript{CO}\textsubscript{2} time to steady state was 4.3 ± 2.3 minutes in sheep and 7 ± 6.5 minutes in dogs, respectively (Figure 1).

During hyperventilation in sheep, mean Pa\textsubscript{CO}\textsubscript{2} of 29.5 ± 3 mm Hg and mean tcP\textsubscript{CO}\textsubscript{2} of 47.7 ± 10 mm Hg were measured. During hyperventilation in dogs, Pa\textsubscript{CO}\textsubscript{2} of 29.3 ± 4 mm Hg and tcP\textsubscript{CO}\textsubscript{2} of 42.7 ± 9 mm Hg were measured. During normoventilation, the Pa\textsubscript{CO}\textsubscript{2} in sheep was 42.6 ± 4 mm Hg and the tcP\textsubscript{CO}\textsubscript{2} was 56.9 ± 10 mm Hg. In dogs, the Pa\textsubscript{CO}\textsubscript{2} was 41 ± 5 mm Hg and the tcP\textsubscript{CO}\textsubscript{2} was 49.8 ± 12 mm Hg. During hyperventilation, the Pa\textsubscript{CO}\textsubscript{2} in sheep was 63.3 ± 9 mm Hg in sheep and 55.3 ± 7 in dogs and the tcP\textsubscript{CO}\textsubscript{2} was 70.9 ± 11 mm Hg in sheep and 64.7 ± 10 mm Hg in dogs. During hyperventilation, Pa\textsubscript{CO}\textsubscript{2} and hypercapnia, significant (P = 0.009) differences were observed between the tcP\textsubscript{CO}\textsubscript{2} and the Pa\textsubscript{CO}\textsubscript{2} in sheep. In dogs, significant (P = 0.04) differences were detected.
between the tcPCO₂ and PaCO₂ measurement methods during normocapnia but not during hypo- and hypercapnia (P = 0.06).

Bland-Altman analysis revealed bias (mean ± SD values of tcPCO₂ – PaCO₂) and precision (bias ± 2 SD) between tcPCO₂ and PaCO₂ of 13.2 ± 10.4 mm Hg and –7.1 to 33.5 mm Hg in sheep and 10.6 ± 10.5 mm Hg and –9.9 to 31.2 mm Hg in dogs, respectively (Figures 2 and 3). The limits of agreement were fairly wide in both species. The difference between tcPCO₂ and PaCO₂ reached a maximum of 41.1 mm Hg in 1 sheep. The maximum observed difference between tcPCO₂ and PaCO₂ in dogs was 26.9 mm Hg.

Changes in PaCO₂ induced through different ventilation settings were detected by the tcPCO₂ sensor after a time lag. When the ventilation was altered from normoventilation (EtCO₂, 43.4 ± 3.5 mm Hg) to hyperventilation to induce hypocapnia (EtCO₂, 32.7 ± 8.6 mm Hg), the tcPCO₂ sensor values followed the PaCO₂ values with a 4.9 ± 3.5-minute delay in sheep, respectively, from 41.8 ± 6.8 mm Hg to 29.3 ± 4 mm Hg with a 6.2 ± 3.6-minute delay in dogs (Figure 4).

During inhalation anesthesia, calculated SaO₂ was 93.2% to 99.9% in sheep and 97.4% to 99.9% in dogs and was considered adequate in all animals. The SpO₂ sensor was capable of detecting a continual SpO₂ signal in 7 of 13 sheep and in 5 of 10 dogs. An intermittent SpO₂ signal was measured in 5 of 13 sheep and 3 of 10 dogs. No SpO₂ signal was obtained in 1 sheep and 2 dogs.

**Discussion**

The tcPCO₂ sensor overestimated PaCO₂ values but was capable of monitoring changes in PaCO₂ induced by 15-minute periods of hyper-, normo-, and hyperventilation in adult sheep and dogs. After placement of the sensor on the skin, it took 4.3 ± 2.3 minutes in sheep and 7 ± 6.5 minutes in dogs before the sensor generated a steady-state tcPCO₂ value. Immediately after this first part of the experimental protocol was completed, the second part commenced. In both species, the sensor had reached steady state before the second part of the experimental protocol started. Therefore, the sensor’s time to reach steady state value did not interfere with the accuracy of measurements in the second part of the experiment.

Ten minutes after sensor placement, with a stable plane of anesthesia and normocapnia, the divergence between tcPCO₂ and PaCO₂ exceeded 8 mm Hg in both species. During the alteration of ventilation protocol,
the tcPCO₂ values also overestimated PaCO₂ by various amounts. Bland-Altman analysis revealed a wide range between the calculated limits of agreement. We concluded, therefore, that the new method cannot be used to assess alterations in PaCO₂ in place of intermittent arterial blood gas analysis in adult sheep and dogs.

This study detected a greater difference between tcPCO₂ and PaCO₂ measurements than that in similar studies in humans. In humans, by use of identical methods, recordings obtained from the sensor evaluated here reveal excellent correlation with PaCO₂. Therefore, the discrepancy seems to be attributable to species differences. No comparative information is available regarding the anatomic and physiologic aspects of ovine, canine, and human skin. A major factor contributing to the poor correlation of tcPCO₂ with PaCO₂ may be the thick epidermis and sparse capillary perfusion of canine and ovine ear skin. A potentially vasoconstrictive agent (xylazine) was administered to the sheep. However, because xylazine has a short half-life in sheep (23 minutes) and has only peripheral vasoconstrictive effects at high plasma concentrations, we do not believe that the pharmacologic effects of xylazine affected our results.

The divergence between tcPCO₂ and PaCO₂ could be explained by the fact that tcPCO₂ values represent not only the gas pressure from the vessels but also the CO₂ generated by skin metabolism beneath the sensor. Cuveller et al. found that transcutaneous measurement in human patients did not satisfactorily assess the exact values of PaCO₂ when PaCO₂ was > 50 mm Hg. Such findings contradict our results, in which differences between tcPCO₂ and PaCO₂ decreased as PaCO₂ increased.

Hazinski and Severinghaus estimated tcPCO₂ (at 44°C) to be at least 1.37 times the simultaneously obtained PaCO₂, likely because the tcPCO₂ sensor detects CO₂ from sources other than blood vessels. Therefore, a finding of tcPCO₂ values greater than PaCO₂ values does not mean that the former were incorrectly measured. Hence, it has been postulated that it is not logical to have a correction factor because tcPCO₂ monitoring provides a new variable with its own characteristics.

To understand this new variable, one must consider that a variety of factors, both technical and physiologic, can influence tcPCO₂. Technical factors that interfere with tcPCO₂ monitoring include stabilization time of the sensor, attainment of an airtight seal between sensor and skin, the sensor's operating temperature, signal drift, fragility of the sensor membranes, and the need for recalibrations with calibration gases.

It is important to allow sufficient time for stabilization of tcPCO₂ signal to increase the precision of transcutaneous measurements, and it is important to periodically recalibrate the sensor. Secure attachment of the sensor at the skin site must be maintained. If the sensor becomes detached from the skin, the monitor will measure the PCO₂ in air, which is approximately 0 mm Hg.

It is important to note that the tcPCO₂ sensor mimics arterial blood at the capillary level at the sensor site by heating the skin to 41°C and then corrects the measured CO₂ value to 37°C rather than to the actual body temperature. In the blood gas machine, arterial blood is always analyzed at 37°C. If body temperature is > 37°C, the measured PaCO₂ will be lower (approx 2 mm Hg for each 1°C increase) than the actual PaCO₂ in the blood and vice versa for body temperature < 37°C. Accordingly, the tcPCO₂-PaCO₂ gradient is calculated on the basis of temperature-corrected PaCO₂. Therefore, the difference will be less if the body temperature is > 37°C and greater if the temperature is < 37°C.

The tcPCO₂-PaCO₂ relationship can be influenced by a number of physiologic effects, including dermal capillary blood flow, tissue CO₂ production, and CO₂ transmissibility. If the tcPCO₂ sensor is at steady state with the skin, the values obtained at the surface of the skin should be equal to tissue values. However, tissue PaCO₂ values do not have to be identical to the PaCO₂. Tissue CO₂ concentration can be increased by continued generation of CO₂ under conditions of decreased capillary blood flow, cutaneous vasoconstriction (caused by low cardiac output or vasoconstrictive agents), low cardiac output states (hypovolemic, cardiogenic, or hypotensive), local accumulation of lactic acid, and the Haldane effect (O₂ release-dependent CO₂ absorption of hemoglobin). Therefore, the tcPCO₂-PaCO₂ gradient is dependent on the ratio between the change in tissue CO₂ production and CO₂ washout.

In dogs, tcPCO₂ correlates directly with PaCO₂ when the cardiac index is > 1.5 L/min/m² and correlates inversely with cardiac index < 1.5 L/min/m² during hypovolemic shock and resuscitation. This finding was confirmed in human patients in an intensive care unit setting where a good correlation between tcPCO₂ and PaCO₂ values was observed before cardiac decompression. During cardiac decompression, cardiac arrest, and cardiopulmonary resuscitation, the tcPCO₂ values correlated inversely with cardiac index. The severity of shock could be roughly determined by comparing the tcPCO₂ values with arterial CO₂ pressures.

Heating of the skin by the sensor influences the tcPCO₂-PaCO₂ gradient because heating increases tissue CO₂ production and heating capillary blood beneath the sensor increases the PICO₂. As the heating element of the sensor increases the tissue temperature, the CO₂ dissociation curve is shifted to the right and tcPCO₂ increases. Transcutaneous PICO₂ values are therefore always higher than PaCO₂ values.

Despite the difference in absolute values, tcPCO₂ values change as PaCO₂ values change, as revealed in this study and others. For correct interpretation of the transcutaneous values, the time lag associated with this measurement method must be taken into account. The tcPCO₂ response time is dependent on the sensor's temperature; increasing the sensor's temperature decreases the sensor's response time. The response time of the sensor (at 41°C) in our study was less than that reported in other studies in which a different type of tcPCO₂ sensor (at 44°C) was used (response times, 7 to 10 minutes and 8.7 ± 0.6 minutes). This difference may be attributable to different methods of response time calculations. In addition, CO₂ resistance to flow, which depends on the permeability of the skin and the capacitive effect (solubility of gas with tissue el-
ments), determines the \( \text{tcPCO}_2 \) response time.\(^7\) Resistance to flow of \( \text{CO}_2 \) through the skin is marginal. The high solubility of \( \text{CO}_2 \) in the membrane material and in skin and the buffering of \( \text{CO}_2 \) in the tissues yields the response time.\(^7\) It should be remembered that changes in tissue \( \text{CO}_2 \) values are delayed in relation to corresponding \( \text{PCO}_2 \) blood values.\(^{10}\)

The accuracy of the pulse oximetry sensor could not be tested over a wide range of \( \text{SaO}_2 \) values. Therefore, conclusions could not be made about the accuracy, response time, or ability of the sensor to monitor changes in \( \text{SaO}_2 \). However, in both species, consistent \( \text{SpO}_2 \) readings were not detected with this sensor, even though the animals were well oxygenated.

To assess clinically important changes in \( \text{PacO}_2 \), the combined sensor cannot replace arterial blood gas measurements in adult sheep and dogs. Instead, it provides a tool for monitoring changes in \( \text{tcPCO}_2 \). The sensor is capable of detecting changes in \( \text{PacO}_2 \) during different ventilation settings but always with a time lag. Measurement of \( \text{tcPCO}_2 \) has several advantages over periodic arterial blood gas analysis. First, it represents what is occurring at the cellular level rather than at the blood level. Second, it provides continuous information. Third, it is noninvasive. Further studies are required to evaluate the accuracy of the combined sensor with different oxygen saturation concentrations in animals. It may offer particular benefits for continuous monitoring of changes in \( \text{tcPCO}_2 \).

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References

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