MONITORING OF THE INSPIRED AND END-TIDAL OXYGEN, CARBON DIOXIDE, AND NITROUS OXIDE CONCENTRATIONS: CLINICAL APPLICATIONS DURING ANESTHESIA AND RECOVERY

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ABSTRACT. Respiratory oxygen, carbon dioxide, and nitrous oxide concentrations were recorded in 20 patients breath-by-breath during general anesthesia and early recovery, using the Cardiocap multiparameter monitor. Several approved maneuvers were performed to demonstrate the usefulness of end-tidal oxygen measurement. "Oxygrams" provided by the fast paramagnetic oxygen sensor confirmed the capnometric information in the diagnosis of hypoventilation, apnea, and disconnections. In one patient, the alarm for inspiratory oxygen concentration, set at 18%, appeared to prevent alveolar hypoxia and low arterial saturation from occurring when oxygen instead of nitrous oxide was turned off. Low end-tidal oxygen levels revealed inadequate fresh gas oxygen supplementation while low flow circuits were closed. During manual hypoventilation at the end of anesthesia, the inspiratory-expiratory oxygen difference increased almost twofold while end-tidal carbon dioxide increased by only 30%. Changes in nitrous oxide concentration often complemented oxygen-related information obtained in our observations. In the recovery room, a decrease in end-tidal oxygen concentration preceded low pulse oximetry readings. Therefore, it is suggested that all three gases should be monitored continuously to prevent mishaps related to insufficient ventilation and inappropriate gas concentrations during anesthesia and immediate recovery.

KEY WORDS. Monitoring: ventilation. Oxygen: concentration. Carbon dioxide: concentration. Nitrous oxide: concentration.

A fast-responding paramagnetic oxygen sensor has recently become available for anesthesia monitoring [1]. In addition to the inhaled oxygen level, the Cardiocap (Datex Medical Instrumentation Inc, Boston, MA) measures oxygen concentration continuously and displays the oxygen waveform on its screen. In steady-state situations, the "oxygram" is roughly a mirror image of the corresponding capnogram (Fig 1). Digital displays of end-tidal carbon dioxide, oxygen, and nitrous oxide levels as well as analog outputs for recording of gas concentrations are available.

In a previous animal study [2], end-tidal oxygen, and especially the inspiratory–end-tidal oxygen concentration difference, was found to be a faster and more sensitive indicator of acute hypoventilation than end-tidal carbon dioxide concentration, which is usually used as an estimate of adequate alveolar ventilation [3–5]. This led to the assumption that the concentrations of respiratory oxygen and nitrous oxide may also be valuable in diagnosing other pathophysiologic events and mishaps in anesthetic practice. In this report, breath-by-breath recording of oxygen, carbon dioxide, and nitrous oxide levels as well as pulse oximeter oxygen saturation

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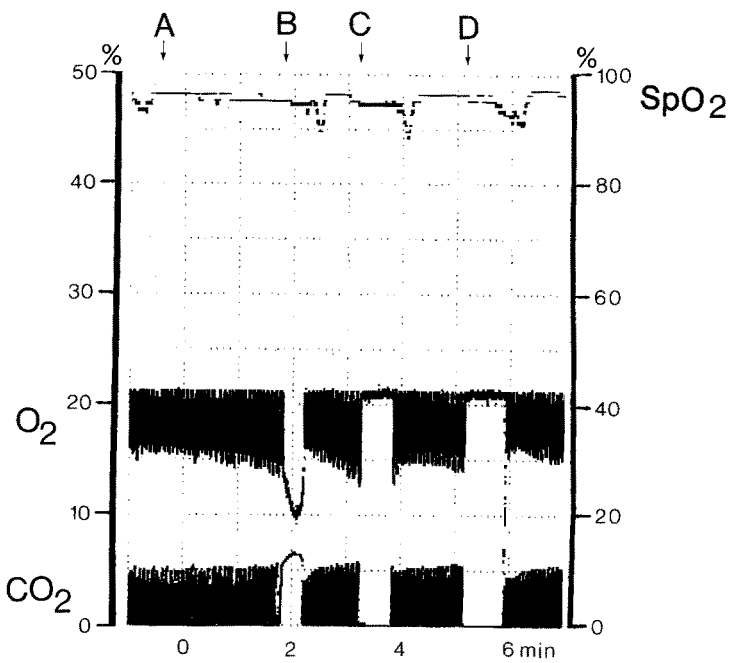

Fig 1. Pulse oximeter oxygen saturation (SpO_2) levels and end-tidal concentrations of oxygen (O_2) and carbon dioxide (CO_2) during normal breathing of room air (A). An extended, slow expiration (B) after a normal inspiration is associated with rapidly decreasing end-tidal oxygen and slowly increasing carbon dioxide concentrations while gases are continuously exchanged in the alveoli. The compensatory effect of resulting deep inspiration on end-tidal gas levels is evident. At C and D, breath was held for 30 to 40 seconds. When breathing after C is begun with a deep inspiration, end-tidal hypoxia cannot be demonstrated and gas concentrations are rapidly normalized. In contrast, apnea at D is ended with an expiration, and subsequently, abnormal gas levels (end-tidal oxygen = 8%; end-tidal carbon dioxide = 7.3%) can be measured. Note the delay in changes in SpO_2 in respect to low alveolar oxygen concentrations in each trial.

(SpO_2) measurements with pulse oximetry were performed during general anesthesia and immediate recovery.

METHODS

The Cardiocap was used in 20 consecutive patients who had no pulmonary disease (American Society of Anesthesiologists classes I to III, 16 to 82 years old) undergoing abdominal operations. We performed several maneuvers designed to demonstrate the potential usefulness of end-tidal oxygen measurement in the operating room. The monitoring continued in the recovery room. The experimental protocol was approved by the Institutional Review Board.

Anesthesia was induced with thiopental 5 mg/kg^{-1} and maintained with nitrous oxide/oxygen (2:1) and enflurane. During anesthesia, the patients were ventilated in a semiclosed circle system (Sulla 800, Dräger-werk, Lübeck, West Germany). Vecuronium was given for muscle relaxation, which was reversed with glycopyrrolate 0.4 mg and neostigmine 2.0 mg given intravenously after completion of the operation. Airway gas was sampled at the tip of the endotracheal tube. At the end of anesthesia, a nasopharyngeal airway was placed and used as the sampling site in the recovery room. The sampling tube was inserted deep enough to obtain smooth gas concentration waveforms and secured with a piece of tape. Oxycodone 3 to 4 mg intravenously was administered for pain relief when necessary. An oxygen mask with 5-L gas flow was removed after 30 minutes and replaced if the saturation values were below 90% and/or end-tidal oxygen levels were below 12% when breathing room air.

The Cardiocap samples respiratory gas at 150 ml/min. Carbon dioxide and nitrous oxide concentrations are measured conventionally with an infrared photometer (10 to 90% rise time = 250 ms, nonlinearity error <2%) [6]. The operation of the oxygen sensor is based on the relatively strong paramagnetic response of oxygen molecules as compared with other gasses [1]. The sample gas and a reference gas (air) are conducted into a strong electromagnetic field that is rapidly switched on and off. This causes an alternating pressure difference between the two gas compartments. The differential signal is converted to oxygen partial pressure and thence to vol % for display. The measurable oxygen range is 0

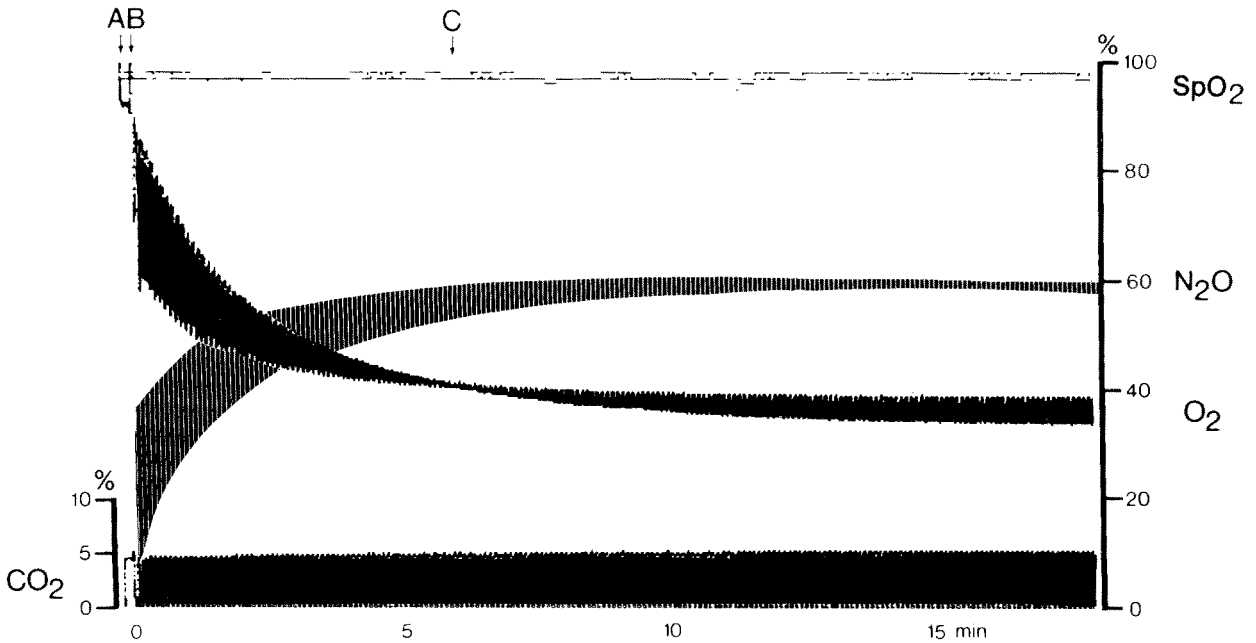

Fig 2. After anesthesia induction and before intubation, alveoli were saturated with oxygen (O_2) by means of mask ventilation with pure oxygen (A). Mechanical ventilation with a 3-L fresh gas flow in a circle system (B) caused typical concentration trends for each measured gas. End-tidal oxygen exceeded the inspired oxygen fraction during nitrous oxide (N_2O) uptake until point C, while end-tidal carbon dioxide (CO_2) stayed fairly constant. The oxygram became a mirror image of the capnogram when nitrous oxide uptake was completed. SpO_2 = pulse oximeter oxygen saturation.

to 100% with a response time (10 to 90%) of 150 ms. Gas zeroing and calibration were performed daily with a known reference gas after a warm-up time of 30 minutes.

Values for SpO_2 were measured from a finger probe with the Datex Satlite pulse oximeter. Analog outputs from the two monitors were used for trend recordings with a Hioki 8810 memory recorder (Hioki E. E. Corp, Japan).

RESULTS

The behavior of respiratory gas concentrations and SpO_2 while room air was breathed are shown first (see Fig 1). Eight typical examples were selected to demonstrate changes in gas concentrations during and after general anesthesia (Figs 2–9). Figure 2 depicts typical washin and washout curves of nitrous oxide and oxygen after anesthesia induction. The effect of disconnection on end-tidal oxygen and carbon dioxide concentrations is shown in Figure 3. The unintentional closure of oxygen flow instead of nitrous oxide set off a low inspiratory oxygen alarm that prevented more serious consequences (see Fig 4). Figures 5 to 8 summarize changes in respiratory gas concentrations and pulse oximeter saturation levels during reduction of fresh gas flow in a circle system, removal of the soda lime canister, and conclusion of anesthesia. Figure 9 shows that a low SpO_2 is preceded by low end-tidal oxygen concentrations and that carbon dioxide changes occur more slowly and are less pronounced than those of oxygen. Note that the scales for gas concentrations and time vary from figure to figure.

DISCUSSION

The fraction of inhaled oxygen is routinely monitored to avoid hypoxic gas mixtures [6]. However, this does not guarantee an adequate alveolar oxygen concentration [7]. In fact, alveolar gas space is the phase met by pulmonary perfusion, and oxygrams represent one step down the oxygen cascade from measurement of the inhaled oxygen fraction. A fast oxygen sensor enables breath-by-breath measurement of both inspiratory and end-tidal oxygen levels. The examples in this report are intended to elucidate the possible clinical usefulness of rapid oxygen analysis.

Effective buffering of carbon dioxide is responsible for the relatively blunted changes in carbon dioxide concentrations following altered alveolar ventilation [3]. Alveolar oxygen concentration depends on the dynamic balance between oxygen enrichment during inspiration

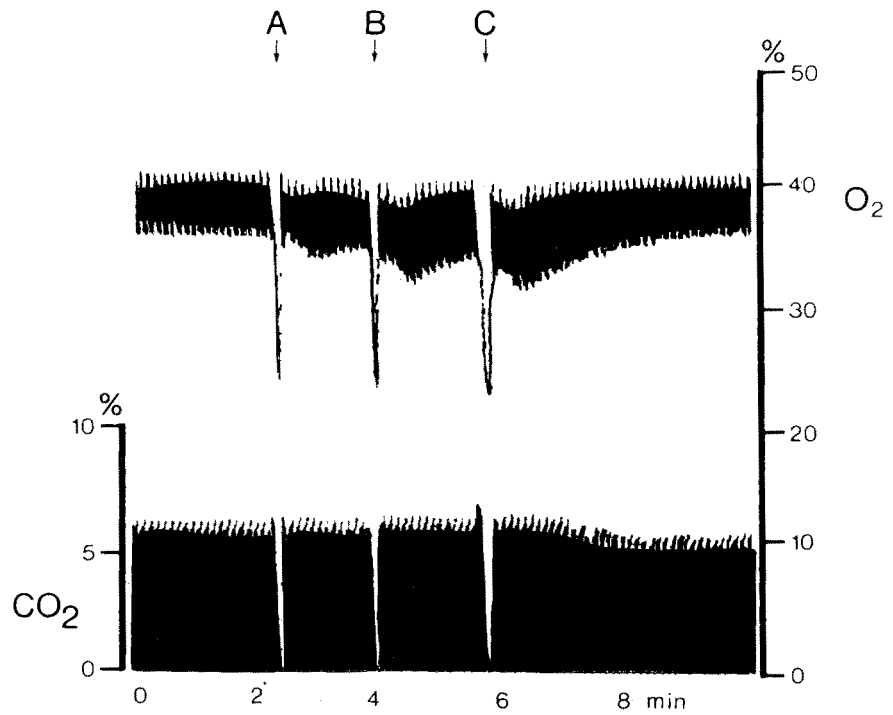

Fig 3. During intraoperative cholangiography, the patient was disconnected three times from the anesthesia circuit between the sampling connector and Y piece for 10 to 15 seconds. Apnea may be detected both from the capnogram (CO_2 = 0%) and oxygram (O_2 approaches 21%). The end-tidal carbon dioxide level did not increase significantly during apnea, but a decrease is evident in the expiratory oxygen concentrations. This transient change seems to result from a decrease in the inspiratory oxygen concentration, possibly due to air's entering the circuit during the disconnections.

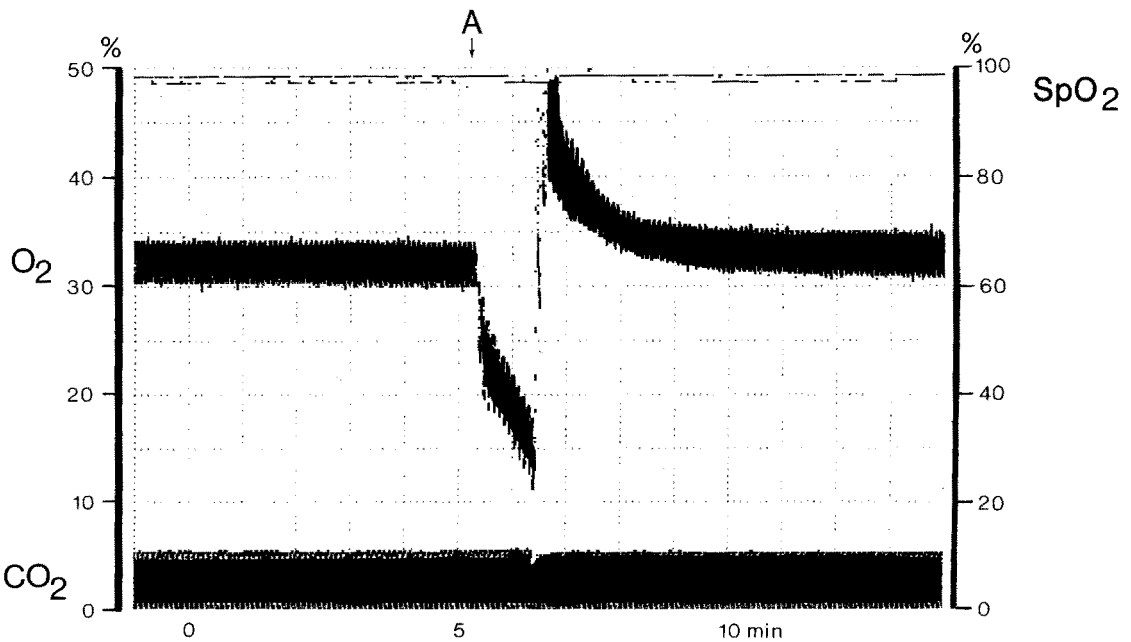

Fig 4. Oxygen (O_2) instead of nitrous oxide was unintentionally turned off at A. Inspiratory oxygen levels in the circle system rapidly decreased below 18%, which is the fixed alarm level in the Cardiocap. Note that the alveolar concentration decreased more slowly and never reached a level leading to changes in the capillary blood saturation. A "puff" of emergency oxygen and restoration of oxygen flow immediately restored safe gas levels. CO_2 = carbon dioxide; SpO_2 = pulse oximeter oxygen saturation.

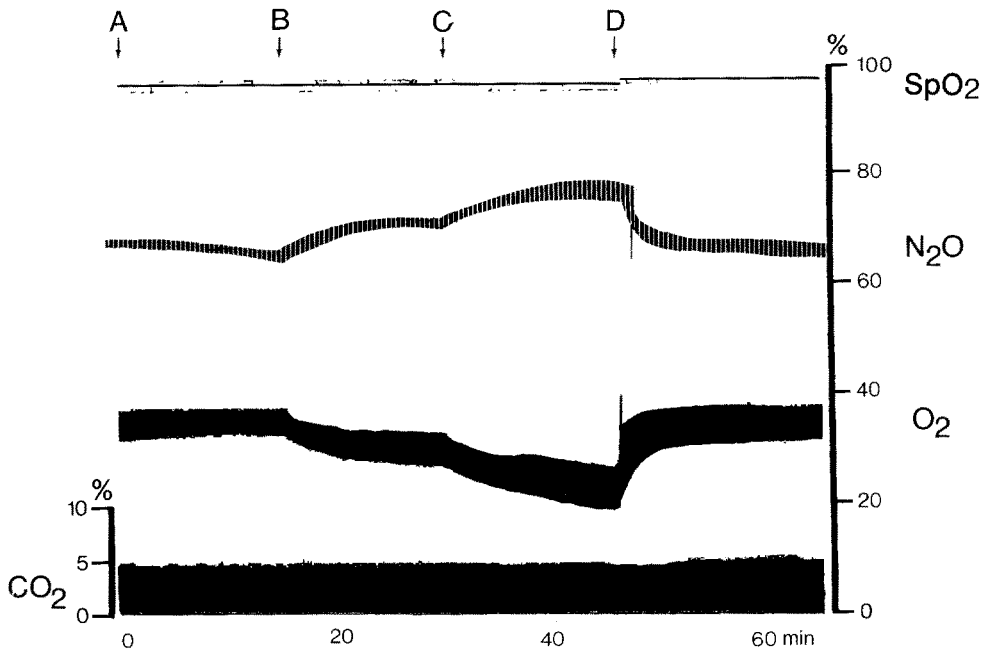

Fig 5. A reduction in the fresh gas flow (3 L/min) by half at B *(1.5 L/min) and* C *(0.75 L/min) caused a decrease in the inspired oxygen fraction in the rebreathing circuit and consequently in end-tidal oxygen* (O_2). *Note the gradual increase in the inspiratory–expiratory oxygen difference and the apparent shift of nitrous oxide* (N_2O) *into the breathing circuit. The continuing decrease of end-tidal oxygen between* C *and* D *indicates that oxygen supplementation (250 ml/min) did not meet the metabolic requirements of the patient. Restoration of the initial fresh gas flow* (D) *rapidly corrected the imminent end-tidal hypoxia. The pulse oximeter showed an essentially unaltered pulse oximeter oxygen saturation* (SpO_2) *during this period.* CO_2 = *carbon dioxide.*

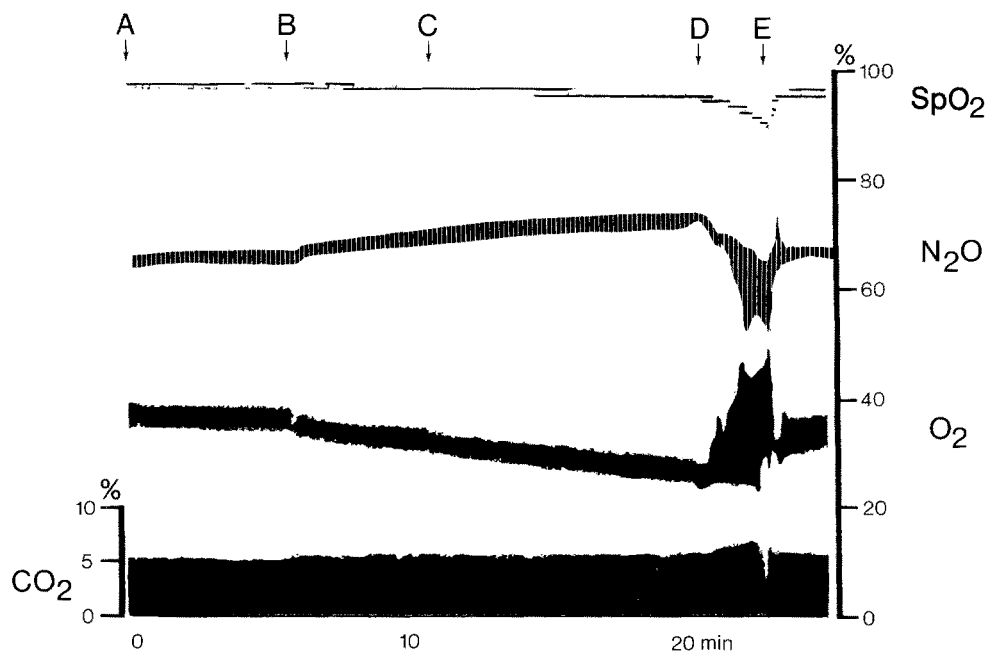

Fig 6. Gas concentration trends are shown during a similar trial reduction of fresh gas flow (B *and* C), *as in Figure 5, but where the patient's endotracheal cuff was leaking. Oxygen* (O_2) *and nitrous oxide* (N_2O) *levels changed gradually until after the last reduction* (D), *when both carbon dioxide* (CO_2) *and pulse oximetry indicated insufficient alveolar ventilation. Nitrous oxide was turned off but this was not enough to reverse the trend toward low end-tidal oxygen concentration and pulse oximeter saturation* (SpO_2). *A short "puff" of emergency oxygen* (E) *and subsequent restoration of original fresh gas flow and composition corrected all values to satisfactory levels.*

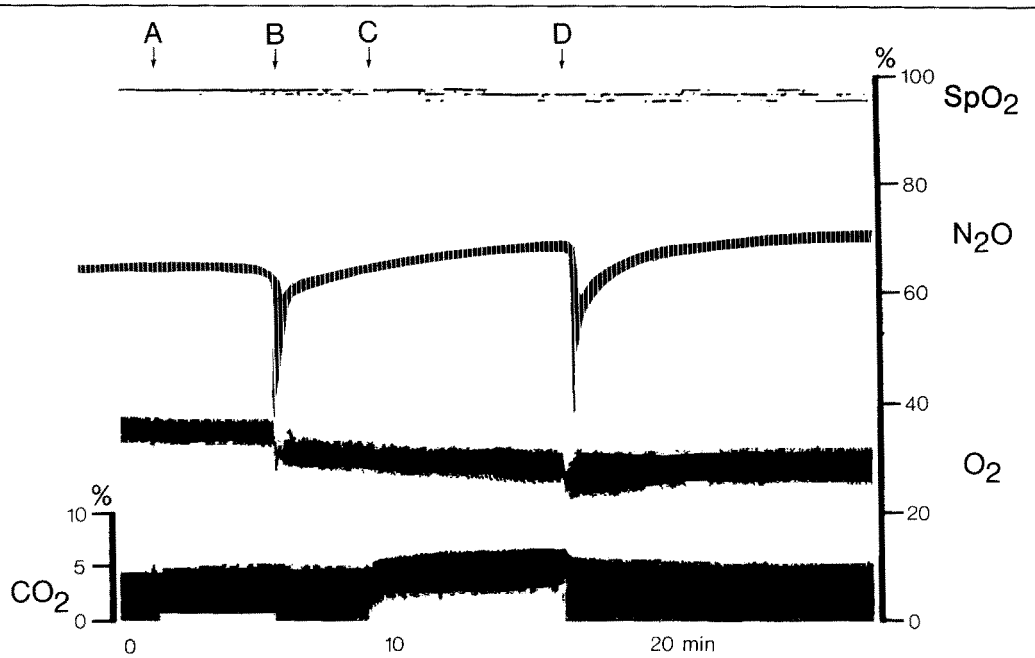

Fig 7. Removal of the soda lime canister with the original fresh gas flow of 3 L/min (A) *caused only a slight increase in both the inspired carbon dioxide fraction and end-tidal carbon dioxide* (CO_2) *and no change in oxygen* (O_2) *or nitrous oxide* (N_2O) *levels. At* B, *the carbon dioxide absorber was reattached and the fresh gas flow reduced to 1.5 L/min. After the absorber was again removed (C) with this lower fresh gas flow, considerable rebreathing of carbon dioxide caused marked carbon dioxide retention, but no stepwise changes in the steadily decreasing end-tidal oxygen level. Sharp decreases in nitrous oxide and oxygen concentrations at* B *and* D *were caused by dilution by room air that was carried into the circuit within the soda lime canister.* SpO_2 = *pulse oximeter oxygen saturation.*

and continuous oxygen extraction from the alveoli by effective pulmonary capillary perfusion [8,9], as well as in the dead space. These dissimilarities in changes of oxygen and carbon dioxide concentration during apnea are demonstrated in Figures 1, 3, and 9.

It is imperative to understand the sampling method in order for a correct interpretation of the measured values to be made [10]. In Figure 1, both the prolonged expiration and the first apnea were ended with an inspiration. The next exhaled end-tidal gas sample was enriched with atmospheric oxygen and, therefore, failed to indicate the preceding degree of hypoxia. In contrast, ending the second apnea with expiration revealed abnormal concentrations of both oxygen and carbon dioxide. The imminent hypoxemia due to decreasing alveolar oxygen is detected earlier by oxygrams than by pulse oximetry. During hypoventilation (see Fig 8), end-tidal oxygen concentration changes more rapidly than does end-tidal carbon dioxide concentration (note that the oxygen scale is only half of the corresponding carbon dioxide scale). The higher sensitivity of the inspiratory-expiratory oxygen concentration difference as an index of hypoventilation is also evident in Figure 9, where relatively low (≤12%) end-tidal oxygen levels were associated with SpO_2 levels below 90%.

In Figure 3, the rapidly decreasing oxygen concentration indicates sampling of room air from a disconnected circuit. If a disconnection occurs distal to the sampling tube, large variations between the oxygen concentrations in room air and those in fresh gas (intended for inspiration) may be seen. Simultaneous capnometry will confirm the diagnosis.

Oxygen analysis can readily detect inadvertent administration of hypoxic gas mixtures [6]. The alarm limit for low *inspiratory* oxygen in the Cardiocap is set to 18%. When this limit was reached in Figure 4, the end-tidal oxygen level was still high enough to ensure maximal SpO_2 values. The situation could be corrected more rapidly than would be possible if pulse oximetry had been the only monitoring method applied. In this context it must be pointed out that capnometry does not immediately reveal alveolar hypoxia caused by anoxic gas mixtures.

Decreases in oxygen levels, both in the rebreathing circuit and end-tidal gas, were demonstrated in Figure 5, where a new steady state was achieved after each reduction in oxygen supplementation. That oxygen flow was first increased to 1 L/min before nitrous oxide was restored is seen as a brief peak in its inspiratory concentration at point D.

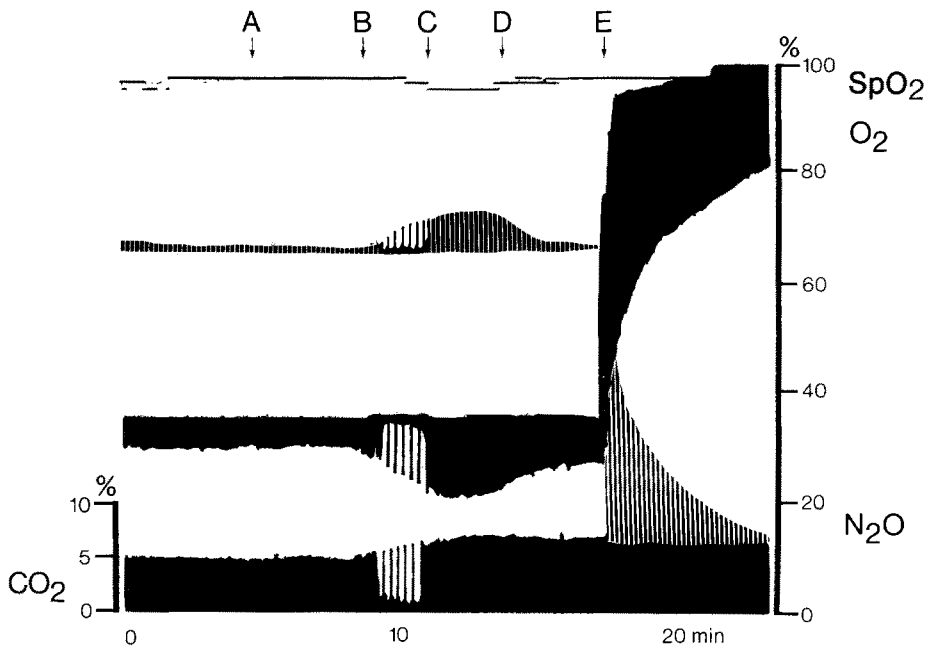

Fig 8. Behavior of pulse oximeter readings and end-tidal gas concentrations at the end of anesthesia during fresh gas flow of 3 L/min. Slow manual ventilation was started at B *to achieve a carbon dioxide* (CO_2) *level sufficient to sustain spontaneous breathing. End-tidal oxygen* (O_2) *changed more rapidly than end-tidal carbon dioxide, and the difference was compensated for with the third gas, nitrous oxide* (N_2O). *During the low end-tidal oxygen levels, a clear change in pulse oximeter oxygen saturation* (SpO_2) *could be noticed. At* C, *the patient started to breathe spontaneously. After* D, *the amount of alveolar ventilation and oxygen delivery were sufficient to meet the oxygen consumption, as reflected by decreasing inspiratory–expiratory oxygen and nitrous oxide differences, as well as increasing pulse oximeter values. At* E, *nitrous oxide was turned off, which resulted in a rapid increase in the fraction of inspired oxygen. End-tidal oxygen apparently depended on the elimination rate of nitrous oxide.*

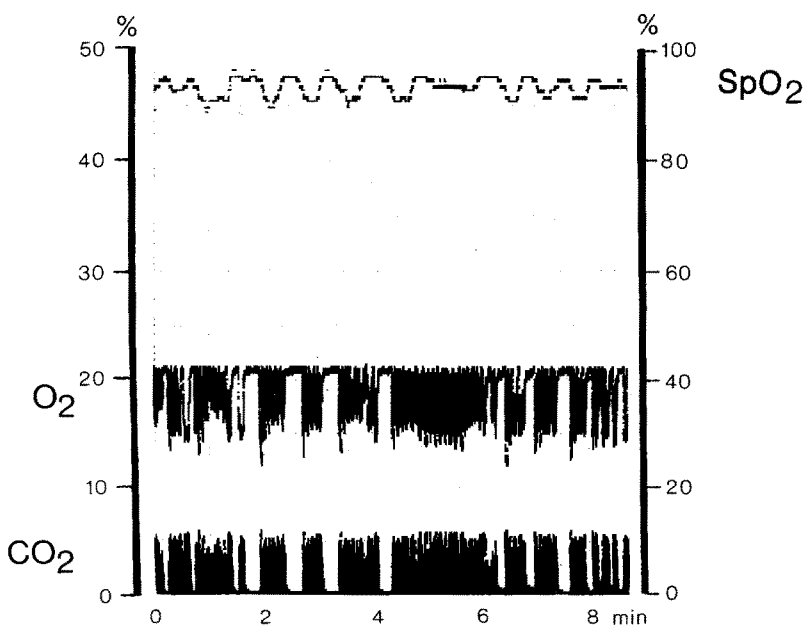

Fig 9. Pulse oximetry and respiratory oxygen (O_2) *and carbon dioxide* (CO_2) *trends were recorded from a 62-year-old patient who dozed during breathing of room air in the recovery room. Note the recurrent apneas and consequent changes in pulse oximeter oxygen saturation* (SpO_2). *Note also the larger end-tidal oxygen than carbon dioxide variations.*

The patient's oxygen consumption in Figure 6 apparently exceeded 250 ml/min and had to be compensated for by additional oxygen because of falling SpO_2 values. In the leaking circuit, however, it was necessary to restore the original 3-L fresh gas flow before all alveolar gas levels returned to satisfactory levels.

Addition of air boluses into the circuit (inside the soda-lime canister) caused instantaneous changes in nitrous oxide concentrations as shown in Figure 7. The method seems to be sensitive enough to detect even small additions of air (see Fig 3).

Low end-tidal oxygen concentrations were invariably associated with decreased SpO_2 values (see Figs 1, 8, and 9). However, due to the circulatory delay and the nature of the oxyhemoglobin dissociation curve, pulse oximetry indicated respiratory problems later than did the measurement closer to the cause itself.

In conclusion, our observations suggest that continuous monitoring of respiratory oxygen and nitrous oxide concentrations, when combined with capnometry, yields valuable information about the uptake and elimination of these gases. Measurement of end-tidal oxygen concentration appears useful during adjustment of oxygen supplementation, particularly during the closing down of circle systems. Low inspiratory and end-tidal oxygen concentrations provide an earlier warning than does pulse oximetry of inadvertently terminated or inadequate oxygen administration. Future applications of fast oxygen analysis will comprise techniques that require careful adjustment of airway oxygen concentrations (e.g., laser surgery) or exact knowledge of alveolar–arterial oxygen differences (e.g., during weaning from ventilatory support or for shunt calculations). Oxygen waveform analysis may complement the information contained in the carbon dioxide waveform [11]; the relationship of the two waveforms reflects the ventilatory gas kinetics and metabolic dynamics. Although we have concentrated on the Cardiocap, the new single-patient mass spectrometers can provide the same important information.

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