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Transcutaneous Monitoring of Oxygen Tension during Progressive Hypoxia and Sleep

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ABSTRACT

Transcutaneous O_2 monitoring ($P_{tc}O_2$) was studied in 16 adult subjects suspected of having sleep-related breathing disorders, by comparison with arterial O_2 measurements (P_aO_2). At rest the mean difference (±SD) between P_aO_2 and $P_{tc}O_2$ was 3.6 (±1.5) kPa. During progressive hypoxia 74 simultaneous measurements showed a mean decrease in $P_{tc}O_2$ from the beginning to the end of the hypoxic tests of 6.6 (± 1.4) kPa for $P_{tc}O_2$ and 7.1 (± 1.5) kPa for P_aO_2. The decrease in $P_{tc}O_2$ was slower than that in P_aO_2 during the first minute ($p<0.001$), but for the whole hypoxic period there was no difference in the rate of decrease between the two methods. Continuous $P_{tc}O_2$ monitoring has been found useful in detecting respiratory abnormalities during sleep.

INTRODUCTION

Non-invasive measurements of estimating arterial oxygen tension (P_aO_2) have been widely used in neonatal medicine for more than 10 years, but only sparsely in adults (1,3). In studies of sleep-related breathing disorders, monitoring of blood gas changes is of essential importance. As pointed out in newly published guidelines for cardiopulmonary sleep studies, measurements of transcutaneous oxygen tension ($P_{tc}O_2$) have rarely been used in sleep laboratories for adults and little data are available concerning this technique during sleep (6).

The present study had two principal aims; firstly to compare $P_{tc}O_2$ and P_aO_2 measurements both at rest and under progressive hypoxia, and secondly to analyse tracings from $P_{tc}O_2$ monitoring made during sleep.

MATERIAL and METHODS

Patients: The study comprised the same 16 consecutive patients whose characteristics are presented in Table 1 in the preceding article on S_aO_2 in this issue.

Procedure: Arterial blood samples were drawn through an indwelling cannula in the radial artery in the same way as described in the previous paper. $P_{tc}O_2$ was measured with an electrode system, E 5230/TCM 20 (Radiometer, Copenhagen, Denmark) after calibration according to the manufacturer's instructions. The electrode was attached with a plastic mounting ring to the skin of the upper anterior part of the thorax, in the midsubclavian region, after shaving and cleaning with alcohol. To produce hyperaemia of the underlying skin, the electrode was heated to a temperature of + 44 °C.

The hypoxic ventilatory test was performed by a method similar to that described by Rebuck and Campbell (7). Briefly, the patients rebreathed 8 litres of room air from a Bernstein spirometer. During the rebreathing procedure the concentration of the inspired oxygen fell as a consequence of oxygen consumption.

An arterial blood sample was taken at rest just before the hypoxic test began, and a sample was then taken every minute during and one, 2.5 and 5 minutes after the test. When approximately 50% of the arterial sample had been collected, a mark was made on the $P_{tc}O_2$ recorder (Radiometer TCM 200 recorder). All arterial sampling and $P_{tc}O_2$ markings were done by the same person.

All patients underwent whole-night polysomnographic studies with simultaneous recordings of sleep, respiratory effort and air flow (6). $P_{tc}O_2$ was also recorded simultaneously in the same way as described above, but only for 4 hours at a time because of electrode heating.

RESULTS

A total of 16 pairs of simultaneous P_aO_2 and $P_{tc}O_2$ readings were obtained at rest. Under this condition the mean P_aO_2 (± S.D.) was 11.6 (±1.4) kPa and the mean $P_{tc}O_2$ 8.0 (± 1.7) kPa. The mean difference between $P_{tc}O_2$ and P_aO_2 was 3.6 (± 1.5) kPa.

During progressive hypoxia altogether 74 simultaneous measurements of $P_{tc}O_2$ and P_aO_2 were made and after the test another 40 were performed. Their relations is shown in Fig. 1.

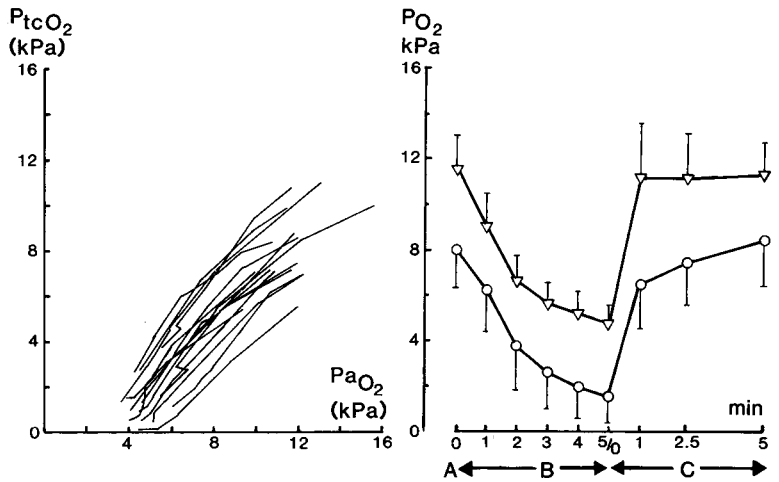

Fig. 1.
left: Relation between P_aO_2 and $P_{tc}O_2$ for each of the 16 patients before during progressive hypoxia.

right: Mean and S.D. of P_aO_2 (triangles)+ $P_{tc}O_2$ (circles) (A), during (B) and after (C) progressive hypoxia.

The mean decrease in $P_{tc}O_2$ from the beginning to the end of the hypoxic tests was 6.6 (± 1.4) kPa and that in P_aO_2 was 7.1 (± 1.5) kPa for P_aO_2 (Fig. 1b). Statistical analyses of the differences between the decreases by a paired t test revealed that the decrease in $P_{tc}O_2$ was slower than that in P_aO_2 from the start to one minute ($p<0.001$), but that for the whole hypoxic period there was no difference in the rate of decrease between the two methods. There was a delay in the increase of $P_{tc}O_2$ one minute after the test, but after 2.5 and 5 minutes no systematic differences from pre-test values were found.

$P_{tc}O_2$ monitoring during sleep showed that in subjects without respiratory abnormalities the $P_{tc}O_2$ tracings were "stable" except that when the subjects changed their body position there was a slow increase or decrease in $P_{tc}O_2$. In patients with breathing abnormalities during sleep the $P_{tc}O_2$ tracings revealed specific patterns, depending on the number and length of respiratory events. This is best illustrated by the case histories presented in Fig. 2.

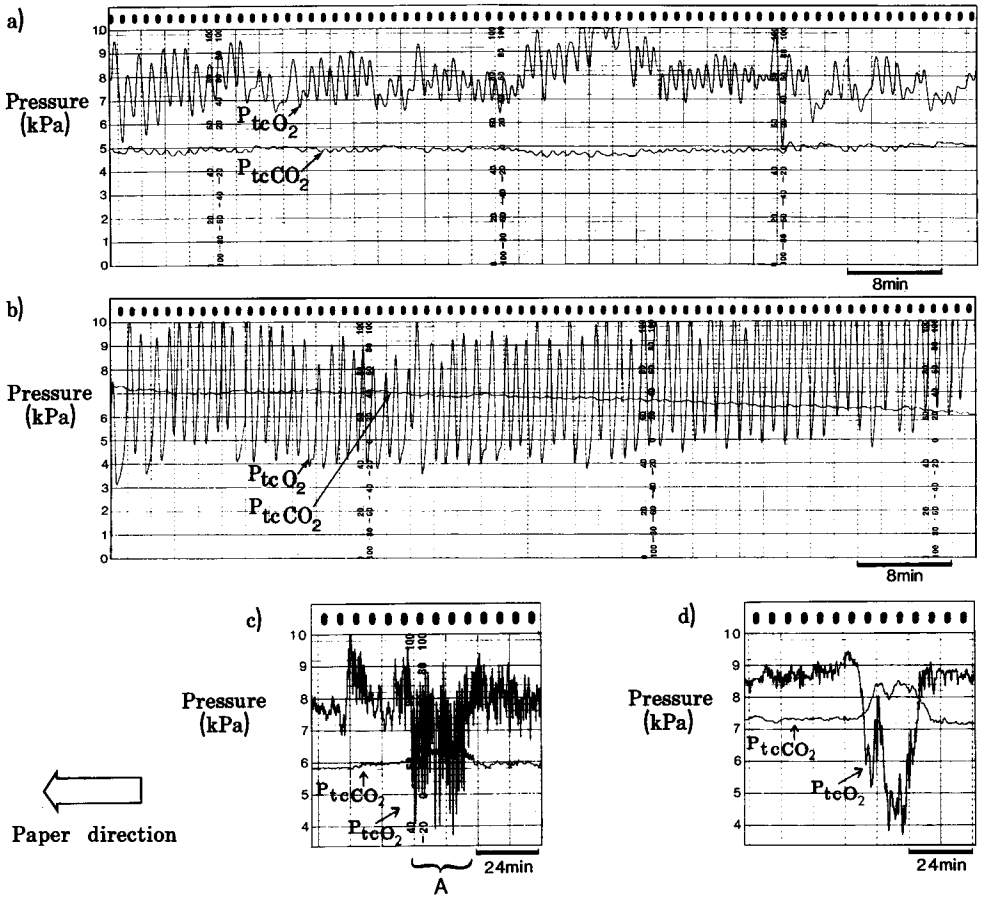

Fig. 2. Tracings from continuous $P_{tc}O_2$ (and $P_{tc}CO_2$) measurements in the following patients: (a) A 35-year-old man with 88 short (about 19 s) apnoeic episodes and a decrease in $P_{tc}O_2$ by 1-2 kPa. (b) A 50-year-old man with 435 long (about 36 s) apnoeic episodes and repeated decreases in $P_{tc}O_2$ by 3-6 kPa. (c) A man with the sleep apnoea syndrome who had twice as long apnoeas during REM sleep (marked by A) than during non-REM sleep and simultaneously showed a profound repeated decrease in $P_{tc}O_2$. (d) A woman with REM-sleep-related hypoventilation and a simultaneous long-lasting decrease in $P_{tc}O_2$.

DISCUSSION

It is important to realise that the measured $P_{tc}O_2$ reflects a balance between oxygen consumption by the skin itself and the increase in PO_2 due to the temperature effect (2, 3). $P_{tc}O_2$ is also sensitive to changes in blood pressure and flow, as capillary blood flow represents approximately 80% of the total flow and is a major determinant of $P_{tc}O_2$ (2).

Contrary to findings in neonates, our results at rest show that the mean difference and S.D. between P_aO_2 and $P_{tc}O_2$ are far too great to allow any conclusion to be drawn about the actual P_aO_2 from a short $P_{tc}O_2$ monitoring. But during progressive hypoxia $P_{tc}O_2$ follows P_aO_2 very well, as shown in Fig. 1, and reflects the actual changes in P_aO_2 adequately. Therefore, if there is a decrease in $P_{tc}O_2$ by, for example, 3 kPa, this change can be considered to reflect a corresponding change in P_aO_2 as long as other potentially effective factors (2) are unaltered. The only case in which $P_{tc}O_2$ failed to indicate changes in P_aO_2 correctly was in a subject with a very low initial $P_{tc}O_2$ value at rest and in whom $P_{tc}O_2$ had therefore reached zero while P_aO_2 was still falling. (Fig. 1).

Simultaneous measurements of $P_{tc}O_2$ and P_aO_2 during respiratory disturbances at sleep were technically impossible to perform in our study because of the rapidity of the P_aO_2 changes (Fig. 2). For such a study an intra-arterial O_2 electrode is needed. By considering the factors that are known to influence the relationship between P_aO_2 and $P_{tc}O_2$ (2) together with our findings during progressive hypoxia, it may be assumed that $P_{tc}O_2$ reflects P_aO_2 accurately at least during shorter respiratory apnoeas. With longer and repeated apnoeas, changes both in cardiac output (2) and in parasympathetic and sympathetic tone (6) might theoretically influence the final $P_{tc}O_2$. This influence is probably slight and our examples of $P_{tc}O_2$ recording during sleep (Fig. 2) clearly demonstrate that this type of monitoring is useful for continuous surveillance of PO_2 in cases where repeated arterial samples would otherwise be needed. This is in accordance with a recent report that $P_{tc}O_2$ monitoring correctly detected all instances where P_aO_2 was below 8.7 kPa among adults during fibreoptic bronchoscopy (4). If we compare the clinical usefulness of $P_{tc}O_2$ monitoring during sleep with continuous oximetry (SO_2) as described in the preceding paper, both methods are found to reflect changes in blood gases with sufficient accuracy. On the basis of the technical characteristics of the two methods and the slope of the oxyhemoglobin dissociation curve, $P_{tc}O_2$ would seem to be more sensitive to smaller changes, but its greatest disadvantage in sleep studies is the limitation of recording time due to electrode heating.

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