Europ. J. Intensive Care Medicine 2, 29–33 (1976)

European Journal of
Intensive
Care Medicine

Transcutaneous P_{O_2} Monitoring in Anaesthesia

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Abstract. In 23 patients 18 to 73 years old transcutaneous P_{O_2}, relative local perfusion and cardiorespirogram during induction and end stage of anaesthesia were monitored. This method allows continuous sufficiently exact estimation of Pa_{O_2}.

The comparison between tcP_{O_2} and corresponding blood gas analysis from arterial samples showed a good correlation of r = 0.94. Thus continuous tcP_{O_2} registration enables quick diagnosis of hypoxia and its therapy.

Key words: Transcutaneous P_{O_2}, Monitoring of anaesthesia, Induction phase, End stage of anaesthesia.

Oxygen supply during induction and end stages of intubation anaesthesia must be considered to be very critical. During some parts of these periods the body depends exclusively on its oxygen reserves, therefore on chemically bound, physically dissolved oxygen, and on the gas in the alveolar space. Until now it has not been possible to study the dynamics of oxygen pressure values as a measure of oxygen supply because all previous methods only allowed a single analysis of arterial oxygen pressure to be made, or were time limited (4) and not free of risks (4, 8).

Having shown that the transcutaneous technique of measuring oxygen enables reliable, quantitative and continuous recordings of oxygen pressure in newborn infants (6) and adults (5, 10), the changes in oxygen tension during different stages of anaesthesia were determined in a series of routine anaesthesia cases.

Material and Method

Arterial P_{O_2} was monitored continuously over the intact skin of the patient. Direct heating of the modified Clark P_{O_2}-electrode produces hyperaemia thus "arterializing" capillary blood below the electrode. This makes it possible to consider blood gas values in this region as arterial. The heating energy required for keeping the present temperature at a constant level against the cooling effect of the flowing blood can be registered and used as a relative dimension of local perfusion. The in vitro response time (95%) of the electrode covered with 12 μm cuprophane and 12 μm teflon membranes was about 6–8 sec. After a two point in-vitro calibration with water vapour saturated air and nitrogen, a procedure which takes only a few minutes, the tcP_{O_2}-electrode was fixed to the sternum of the patient by means of a self-adhesive ECG ring. Complete hyperaemia was obtained approx. 10 min. after application of heat and the P_{O_2} registration then showed a steady state under normal breathing conditions. Heart rate, transthoracic impedance and respiratory rate – a so-called cardiorespirogram – were registered using ECG electrodes and corresponding apparatus (Hellige, Freiburg, and Hewlett-Packard, Böblingen). Heart rate was recorded beat to beat. All parameters were recorded on a 6 channel multipen recorder (Rikadenki, Hellige, Freiburg) with a chart speed of 3 cm/min. Blood sampling for comparative P_{O_2} measurements and for P_{CO_2} and pH determination was from the radial artery using the method of Huch and Huch (7). Blood values of P_{O_2}, P_{CO_2} and pH were determined in a Gas-check from AVL (Bad Homburg).

The measurements were performed on 23 non-selected patients whose age ranged between 18 and 73 years. In 11 of the 23 all stages of anaesthesia were recorded. In 13 cases operations were performed abdominally, in 7 on the extremities and in the remaining 3 subtotal thyroidectomy was undertaken. Premedication was made 30–45 min. before induction of anaesthesia with Atropine and Thalamonal® (Janssen). Patients received thiopental-halothane-

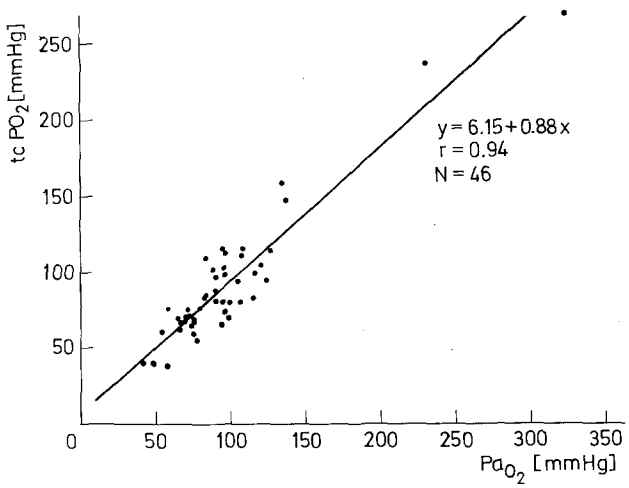

Fig. 1. Plot of 46 simultaneous measurements of transcutaneous and arterial P_{O_2} values in 17 patients. Note the correlation coefficient of 0.94

N_2O–O_2 anaesthesia or neuroleptanalgesia (NLA). N_2O–O_2 mixtures at a ratio of 2 : 1 were used together with controlled mechanical ventilation in a semi-closed circuit. The effects of non-depolarizing relaxant drugs and of Fentanyl® (Janssen, Düsseldorf) were antagonised as usual with Neostigmin and Levallorphan towards the end of anaesthesia.

Results

Fig. 1 shows the graph of correlation between the transcutaneous P_{O_2} values and those obtained from the Gascheck measurement on arterial samples. The regression curve is represented by: y = 6.15 + 0.86 x, with a coefficient of correlation of r = 0.94. This regression line deviates from the optimal 45°-line by 10%, which means that transcutaneous values in adults in this series are approximately 10% lower than the corresponding arterial samples.

Fig. 2 shows the behaviour of the following variables during the induction phase: heart rate, transcutaneous P_{O_2} (tcP_{O_2}), local perfusion, transthoracic impedance and respiratory rate. After reaching a steady state of transcutaneous P_{O_2}, 20 mg Gallamin and 250 mg Thiopental were administered.

It can be seen that heart rate and local perfusion increased. Whereas heart rate continued to increase, local perfusion and respiratory parameters decreased markedly. After about 30 sec tcP_{O_2} dropped about 17 mmHg. It increased immediately after administration of a mixture of N_2O–O_2 (6 l + 2 l). Anaesthesia was then continued with addition of halothane.

Thereafter the patient was relaxed with succinyl-bischoline (70 mg), ventilated manually and intubated with a cuffed tube. Note that tcP_{O_2} continued to increase and did not fall even during the apnoeic phase of the intubation. This was true in all of our measurements. Fig. 3 shows the behaviour of heart rate, arterial pressure, tcP_{O_2} and local perfusion during the terminal phase of a NLA.

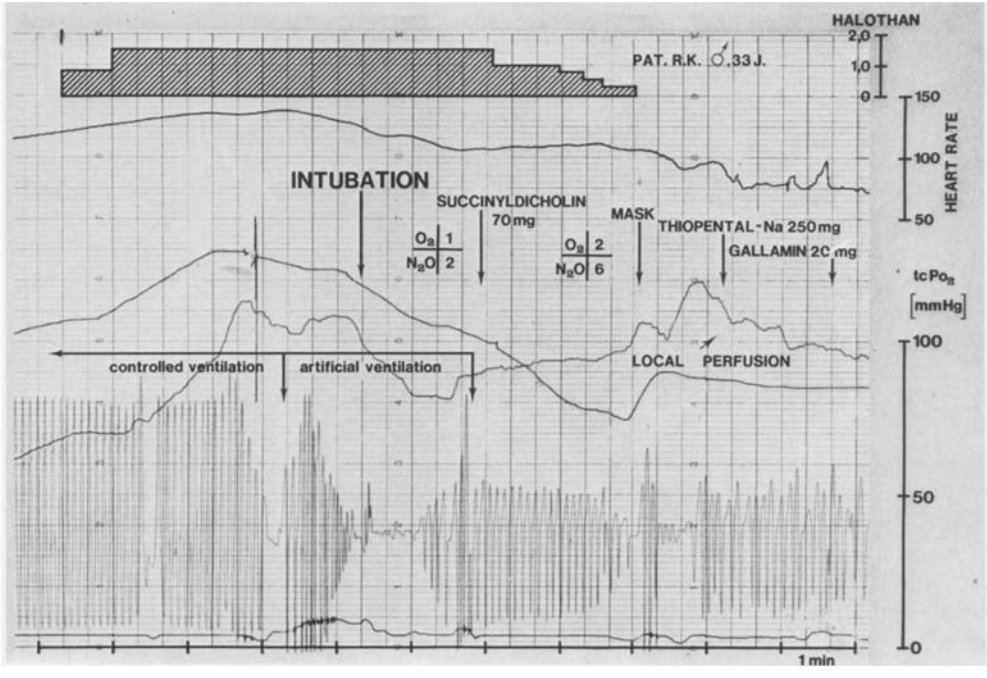

Fig. 2. Induction phase of a halothane-N_2O-O_2 anaesthesia. Time scale reads from right to left

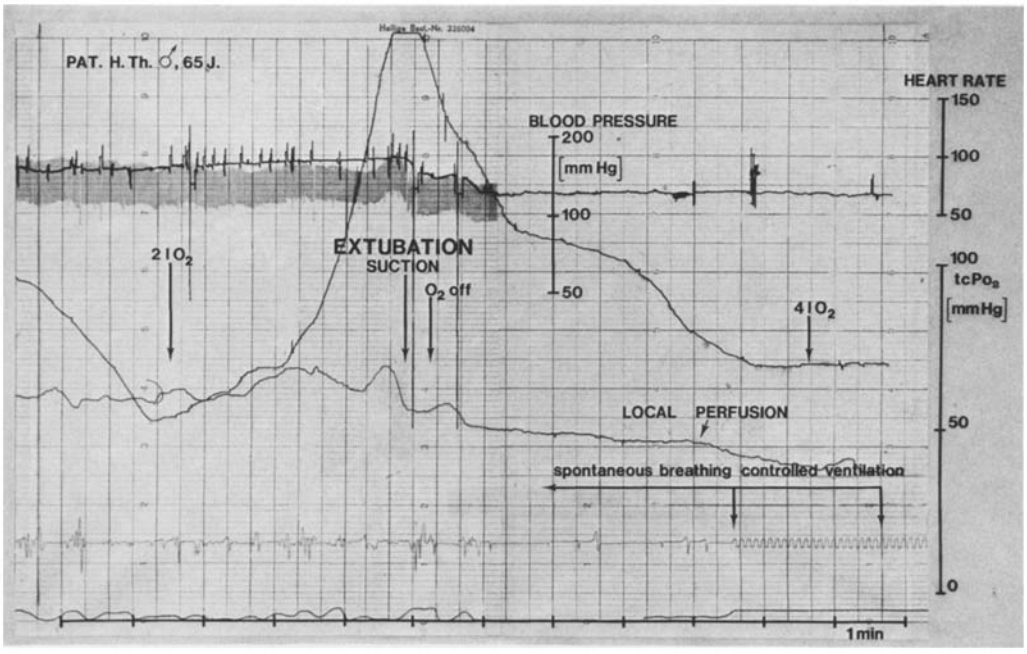

Fig. 3. Terminal phase of a NLA. Time scale reads from right to left

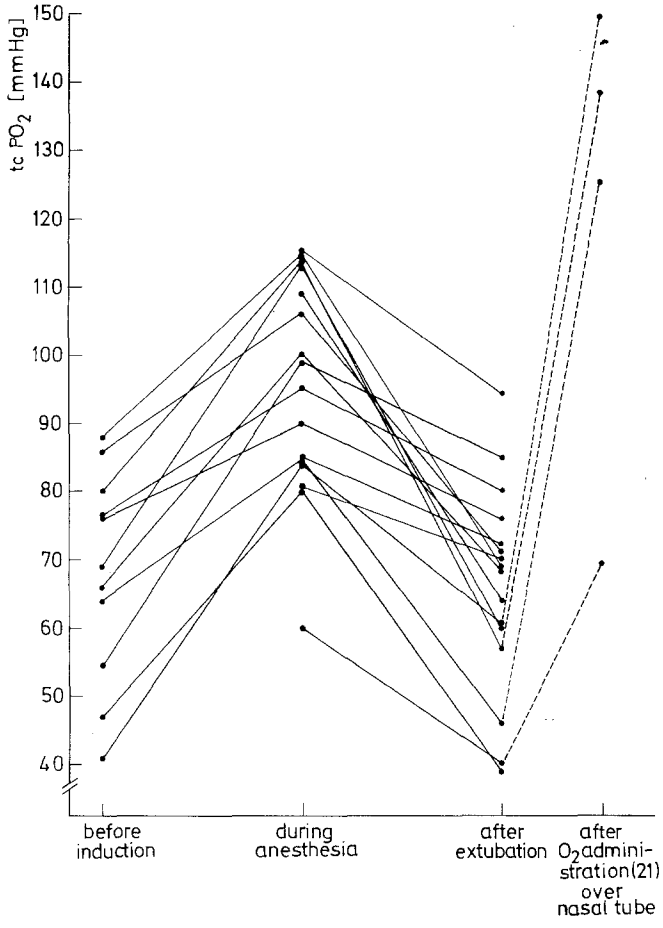

Fig. 4. Transcutaneous P_{O_2} values before induction, during anaesthesia, after extubation and after O_2 administration by a naso-pharyngeal tube

The respiratory parameters are shown on the lowest 2 curves. 40 sec after switching over to pure O_2 the tcP_{O_2} started to rise to a value higher than 200 mmHg. Shortly before extubation the patient was disconnected from the anaesthetic circuit and breathed normal air. Extubation was performed simultaneously to tracheo-bronchial suction.

During this phase blood pressure rose from 150/90 to 175/135 mm Hg. Local perfusion also increased parallel to this parameter. Spontaneous breathing of normal air produced a tcP_{O_2} fall to 55 mmHg, which increased to about 110 mm Hg immediately after administration of supplementary O_2 (21) through a naso-pharyngeal tube. The continuous measurement of peripheral flow proved to be a good supplement to routine intermittent measurement of blood pressure using the method of Riva-Rocci.

In all those cases in which simultaneous continuous blood pressure recordings were performed with a Statham element, the curves confirmed a close relationship (e. g. see Fig. 3). Furthermore, it was possible in a few cases to show that a considerable decrease of the flow indicated a disturbance in micro-circulation. This has been shown by Rooth *et al.* (in preparation) to be connected with a change in correlation coefficient.

Fig. 4 demonstrates the behaviour of tcP_{O_2} before induction, during anaesthesia and at least 30 min after extubation and after having reached a steady state during spontaneous air breathing. During controlled ventilation with a 2 : 1 N_2O–O_2 gas mixture, tcP_{O_2} rose to a level above the pre-narcotic or post-narcotic level of the same patient. The tcP_{O_2} values of 66.9 ± 13.8 (before induction) and 69.2 ± 14.0 (after extubation) were not found to be significantly different using the paired t-test at the 5% level. In those patients receiving supplementary oxygen by nasal application a prompt increase in tcP_{O_2} was noted.

Discussion

The method of transcutaneous P_{O_2} recording used in this series has certain features which make it very useful for patient monitoring during induction, course of anaesthesia, for the period immediately after anaesthesia and for high risk patients in intensive-care units. One advantage is that the electrode is non-invasive and is applied to the skin like an ECG electrode. In no way does it bother or disturb the patient and can provide continuously recordings for several hours.

In vitro response time to the electrodes used was 6–8 sec (= 95%). There was a total delay of about 20–30 sec in recording the effect on tcP_{O_2} of a sudden change in the inspiratory gas mixture. The delay time was determined by the characteristics of skin and the circulation time of the patient. Sufficiently quick recognition of hypoxaemia is therefore possible. Obviously the question arises as to how far the oxygen tension values measured on a certain skin area correspond to the true arterial levels. A judicial choice of cathode, electrode temperature and type and thickness of membranes give tcP_{O_2} values which are well correlated to arterial levels both in adults and in newborn infants (5, 6). Even our very mixed group of patients gave values which correlated well with the comparative measurements. Within this physiological range of oxygen tension the described method has proved to be reliable and gives values well correlated with the P_{O_2} levels obtained from arterial samples, shown by r = 0.94.

It should be remarked that the $P_{a_{O_2}}$ level in this series was lower during spontaneous breathing of air before and after anaesthesia than the values given in literature for normal adults. This effect could be due to drug induced respiratory depression and change of lung function of the supine patient. In contrast to Hempelmann *et al.* (4) our measurements have shown that pre-ventilation with oxygen prevents P_{O_2} falls during intubation. Even more important is the immediate post-operative phase, during which the effect of respiratory depression, hypotension, diffusion hypoxaemia (1, 3, 12) and atelectasis may occur. The results shown in Fig. 4 indicate that in all patients, irrespective of their initial P_{O_2} level, transcutaneous P_{O_2} increases under the anaesthesia administered. The lung ventilation of these patients was between 6 and 10 l/min. Arterial P_{CO_2} varied from 36 to 45 mm Hg. All patients were ventilated with intermittent positive-negative pressure. Post-operative tcP_{O_2} values of conscious patients breathing normal air were not significantly different from their pre-operative levels. Kitamura *et al.* (11), however, found a significant drop in post-operative arterial P_{O_2} as compared to the pre-operative level and after manual ventilation during anaesthesia, but no differences in P_{CO_2}. Post operative hypoxia was regulary found by Marshall *et al.* (13), especially after abdominal surgery. Rolly (15), Bergmann (12) and Norlander (14) were able to show that post-operative hypoxaemia can be promoted or prevented according to ventilation technique during anaesthesia. Our measurements displayed no trend to post-operative hypoxaemia as compared to the pre-operative level. By this continuous measurement of tcP_{O_2} the anaesthetist knows the prenarcotic state of the patient's arterial oxygen pressure. He is thus able to choose the correct ventilation and the adequate O_2 ratio. The various influences during anaesthesia – an increase of Aa Do_2 with both spontaneous breathing and controlled ventilation – can be identified and compensated.

The parallel measurement of so-called peripheral perfusion pressure (F) and of blood pressure (P) in the right radial artery demonstrates again the relation $F = \frac{1}{R} P$ (1/R = factor of heat conductivity described elsewhere (9)). Given that 1/R is practically constant during hyperthermia, changes in peripheral perfusion (F) reflect blood pressure changes. In the case of constant blood pressure a change

in F means a change in blood distribution to the organs. Without doubt this new parameter can give new perspectives to monitoring apart from the control of transcutaneous oxygen recordings, for which it was originally developed.

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