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Transcutaneous CO_2 Monitoring in Adults With Sleep-Related Breathing Disorders

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ABSTRACT

The accuracy of transcutaneous CO_2 monitoring ($P_{tc}CO_2$) was studied in 22 subjects suspected of having sleep-related breathing disorders, by comparison with arterial CO_2 measurements (P_aCO_2). At rest 40 simultaneous sets of P_aCO_2 and $P_{tc}CO_2$ were obtained. The mean P_aCO_2 ($\pm$SD) was 5.3$\pm$0.9 kPa and $P_{tc}CO_2$ was 5.7$\pm$1.0 kPa (r=0.79).

The ventilatory response to CO_2 was evaluated by a CO_2 rebreathing method, and simultaneous measurements of P_aCO_2, $P_{tc}CO_2$ and end-tidal PCO_2 ($P_{ET}CO_2$) were made every min. Both P_aCO_2 and $P_{ET}CO_2$ increased more during the first min of CO_2 rebreathing than $P_{tc}CO_2$ ($p<0.001$). Between 1 to 5 min after the start of rebreathing there were no significant differences between the three methods.

During sleep there was an increase in $P_{tc}CO_2$ (by 0.1-0.3 kPa) with each apneic event, the magnitude of the increase depending on the length and distribution of these events. With repeated long apneas there was a cumulative increase in $P_{tc}CO_2$, especially during REM sleep. Continuous $P_{tc}CO_2$ monitoring proved useful in monitoring and diagnosing sleep-related breathing disorders.

INTRODUCTION

In studies of sleep-related breathing disorders, monitoring of blood gases is of essential importance. Attempts to estimate the arterial carbon dioxide tension (P_aCO_2) noninvasively have led to the development of two types of transcutaneous measurement techniques, the pH electrode technique and the infrared absorption method (1, 2, 8, 9, 21, 24). These are based on the detection of CO_2 diffusing through the skin, usually after local heating (6, 8). They have similar accuracy in predicting P_aCO_2 but differ concerning some operational features (25).

In most countries these new noninvasive techniques are only sparsely used in adults. The majority of previous comparative studies have dealt with measurements of transcutaneous partial pressure of PCO_2 ($P_{tc}CO_2$), in neonates (3, 11, 13) and children (7, 21). Some such measurements have also been made in cri-

tically ill adults (9), patients during exercise (15), healthy volunteers (26) and persons during sleep (5, 17, 19). In recently published guidelines for cardiopulmonary sleep studies, however, the $P_{tc}CO_2$ electrodes were considered not to be useful because of limited accuracy and slow response (18).

The present prospective study was conducted in two parts. In the first part the accuracy of $P_{tc}CO_2$ measurements was estimated, by comparisons with arterial and in some cases end-tidal PCO_2 ($P_{ET}CO_2$) values, at rest and during carbon dioxide rebreathing tests. In the second part the clinical usefulness of continuous $P_{tc}CO_2$ monitoring during sleep was evaluated.

METHODS

Patients. The study comprised all patients who underwent both a sleep study and testing of the ventilatory response to CO_2 during January to June. The study had previously been approved by the Ethics Committee of the Medical Faculty of the University of Uppsala. Altogether 22 patients, 38-63 (51.3±8.5, mean±SD) years of age were investigated (Table 1). In each patient the sleep study and the CO_2 rebreathing test were performed within 24 hours. There was only one woman (case 17). The majority were snorers, some of whom complained of daytime sleepiness and morning headache and were therefore suspected of having the sleep apnea syndrome (SAS). One man (case 2) had been operated on 6 months before because of SAS and was now symptom-free. Sixteen had a body mass index exceeding 28 kg/m^2 (16), and six had signs of airway obstruction, with a forced expiratory volume in one s (FEV_1) lower than 80 % of the predicted value (Table 1).

Procedure. Arterial blood samples (6-8 ml) were drawn into heparinized glass syringes (10 ml) through an indwelling cannula in the radial artery and immediately analysed in a Corning 168 pH blood gas analyzer or placed in ice water for analysis within 30 min. All blood gas analyses were performed independently by the blood gas routine laboratory. $P_{tc}CO_2$ was measured with a PCO_2 electrode (Stow-Severinghaus type electrode system E 5230/TCM 20, Radiometer, Copenhagen, Denmark), calibrated with 5 % CO_2. A standard temperature correction for CO_2 was included (4). The electrode was attached with a plastic mounting-ring to the skin of the upper anterior 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 electrode readings of calibration gas were recorded at the end of each study to document electrode drift.

Part I

CO_2 rebreathing test. This test was performed by a modification of the tech-

Table 1.

Characteristics of the study group of 21 men and 1 woman, and the results of the CO_2 rebreathing tests.

Case No.	BMI (kg/m^2)	VC (% pred)	RV (% pred)	FEV_1 (% pred)	P_aCO_2 at rest (kPa)	$P_{tc}CO_2$ at rest (kPa)	$\Delta V/\Delta P_{tc}CO_2$ (l/min/kPa)	$\Delta V/\Delta P_aCO_2$ (l/min/kPa)	$\Delta V/\Delta P_{ET}CO_2$ (l/min/kPa)	Slope (k) (kPa/min)
1	22.9	63	89	55	4.3	4.6	3.6	3.7	4.2	2.0
2	26.5	75	60	69	6.0	6.9	6.0	6.1	7.3	1.0
3	41.9	59	177	30	7.3	7.8	2.5	2.1	2.5	0.6
4	27.1	106	93	117	4.9	5.8	15.2	19.9	19.6	2.3
5	28.7	91	119	86	4.3	5.7	15.7	11.6	20.2	1.7
6	28.4	102	71	106	4.9	5.4	30.2	27.7	26.1	1.2
7	39.0	110	154	67	5.7	6.3	6.5	7.3	9.8	2.3
8	27.8	112	65	119	5.0	5.3	39.6	31.9	30.3	1.5
9	29.0	80	100	80	4.8	4.4	38.9	28.5	30.2	1.6
10	31.0	108	57	114	5.2	5.5	9.0	13.4	13.6	2.5
11	24.5	90	83	95	5.3	5.3	32.2	28.1	29.8	1.7
12	39.1	90	127	103	4.6	6.4	11.7	24.5	19.9	1.3
13	31.4	57	92	53	5.0	5.9	8.9	10.9	11.4	1.8
14	22.8	84	122	82	5.5	6.4	14.8	16.4	15.8	0.6
15	32.5	101	89	109	4.5	5.1	24.1	26.4	32.7	2.4
16	27.8	103	119	113	5.3	6.4	23.7	13.4	10.9	1.3
17	29.0	124	111	134	4.7	5.9	23.9	33.3	36.6	2.5
18	30.6	97	95	102	5.4	5.5	23.9	29.0	30.2	3.0
19	36.3	74	113	72	4.9	5.1	20.4	21.9	21.8	2.2
20	40.9	86	158	95	6.1	5.5	23.5	17.2	18.0	1.4
21	30.4	93	103	96	4.9	5.8	12.8	20.9	21.6	2.1
22	36.0	89	86	91	3.3	3.9	16.5	13.3	16.2	1.0
Mean	31.1	91	104	90	5.1	5.7	18.4	18.5	19.5	1.7
SD	5.6	18	31	26	0.8	0.9	10.7	9.4	9.4	0.7

BMI, body mass index; VC, vital capacity; RV, residual volume; FEV_1, forced expiratory volume in 1 second; $\Delta V/\Delta PCO_2$, the increase in ventilation (ΔV) for each 1 kPa increase in PCO_2.

nique described by Read (22). All patients had a noseclip and rebreathed through a mouthpiece into a Bernstein volume spirometer with a one-turn potentiometer as a volume transducer. The volume was continuously recorded on a Model 805 ink-writer (Siemens-Elema, Stockholm, Sweden), which also recorded the carbon dioxide concentration in the mouthpiece. Gas was sampled from the mouthpiece through a tube with a flow rate of 0.5 l/min. The carbon dioxide analyzer (Beckman LB 2, Beckman Instruments AB, Stockholm, Sweden) was calibrated with 5 % CO_2. The Bernstein spirometer was filled with approximately 10 liters of hyperoxic (50 % O_2) and hypercapnic (6 % CO_2) air. The instrumental dead space was 380 ml.

Before the test the patients breathed room air in a resting state for at least 15 min, or until a stable value of $P_{tc}CO_2$ had been reached, with changes not greater than 0.1 kPa per 2 min. During phase 1, the patients became used to breathing through the mouthpiece for 2-3 min while the baseline $P_{ET}CO_2$ was being recorded. In phase 2 the patients rebreathed the hyperoxic and hypercapnic air. In phase 3, they breathed room air through the mouthpiece for 10 min. During all three phases $P_{tc}CO_2$ and $P_{ET}CO_2$ were recorded continuously. An arterial blood sample was taken at the end of phase 1, every min in phase 2, and at 1, 2, 3, 5, and 10 min in phase 3. When approximately 50 % of the arterial blood sample had been collected, a mark was made on the $P_{tc}CO_2$ recorder (Radiometer TCM 200 recorder) and $P_{ET}CO_2$ was registered. All arterial sampling and marking were done by the same person. A typical $P_{tc}CO_2$ tracing is depicted schematically in Fig 1.

Part II

Sleep studies. All patients underwent whole-night polysomnographic studies with simultaneous recording of electroencephalograms (EEG), electrooculograms (EOG) and submental electromyograms (EMG), using standard techniques (10). Respiration was monitored by abdominal and thoracic strain gauges and also by a static charge sensitive bed (Biorec O.Y., Kuusisto, Finland) and by movement sensors (Siemens 230). Air flow was detected by nose and mouth termistors and also by tracheal sound recording. Oxygen saturation was measured continuously by a Biox III pulse oximeter (Ohmeda, Boulder, Colorado, USA). All variables were recorded simultaneously on a 16-channel ink-jet recorder (Siemens Elema) with a paper speed of 1 cm/s.

$P_{tc}CO_2$ was monitored with the same system as in Part I (Rådiometer TCM 20 electrode). Arterial blood sampling and analyses were performed as described above. The first samples were collected just before "lights out" and the last ones 3.5-4 hours later, before recalibration and a change in electrode placement.

Sleep stages were scored in 30 s-epochs (23). Respiratory tracings were

evaluated for the occurrence of apnea, which was defined as complete cessation of both nasal and oral air flow for at least 10 s. Hypopnea was defined as a marked decrease in oro-nasal airflow for at least 10 s accompanied either by a reduction in oxygen saturation of at least 4 % from the baseline level, or by arousal, or both.

Statistical analyses. The strenght of the relationships between different CO_2 measurements was assessed by least-squares linear correlation analysis. Statistical probability was assessed by Student's t test on paired values. Standard deviation is abbreviated as SD.

RESULTS

At rest. Twenty-two sets of simultaneous P_aCO_2 and $P_{tc}CO_2$ recordings were obtained in phase 1, before the CO_2 rebreathing began (Table 2). The transcutaneous values were significantly higher than the arterial ones, although the correlation coefficient (r) between them was 0.72. In four patients $P_{tc}CO_2$ measurements were not performed at rest in the sleep laboratory. For the remaining eighteen patients the mean P_aCO_2 was 5.6 ± 0.9 kPa and the mean $P_{tc}CO_2$ 5.8 ± 1.1 kPa (r=0.86). The transcutaneous values were higher than the arterial ones except on five occations. The differences between transcutaneous and arterial values showed no correlation to body weight or to the results of the pulmonary function tests.

Table 2.
Mean values and standard deviations (SD) of simultaneous measurements of P_aCO_2, $P_{tc}CO_2$ and $P_{ET}CO_2$ during carbon dioxide rebreathing tests.

		P_aCO_2 (kPa)		$P_{tc}CO_2$ (kPa)		$P_{ET}CO_2$ (kPa)	
	n	mean	SD	mean	SD	mean	SD
Phase 1	22	5.09	0.78	5.68	0.85	5.09	0.61
Phase 2							
1 min	19	6.34	0.70	5.98	0.87	6.78	0.64
2	21	7.02	0.94	6.82	1.06	7.25	0.65
3	20	7.33	0.76	7.16	0.95	7.71	0.67
4	20	7.90	1.12	7.70	1.35	8.11	0.81
5	13	8.57	1.47	8.27	1.76	8.04	0.97
Phase 3							
1 min	16	5.04	0.97	6.38	1.25	4.89	0.70
2	9	4.99	1.15	6.03	1.27	4.73	0.88
3	10	5.08	0.65	5.64	0.83	4.87	0.42
5	21	5.03	0.92	5.47	0.97	4.82	0.71
10	20	5.17	1.03	5.62	1.12	4.82	0.69

Part I

CO_2 rebreathing test. In phase 2, during CO_2 rebreathing, 93 simultaneous measurements of P_aCO_2, $P_{tc}CO_2$ and $P_{ET}CO_2$ were performed (Table 2). The mean lag-

time from the start of rebreathing until $P_{tc}CO_2$ started to react was 40±14 s. There was a slower increase in $P_{tc}CO_2$ than in both P_aCO_2 and $P_{ET}CO_2$ from the start to 1 min ($p<0.001$, Table 2), but from 1 to 2 min the increase in $P_{tc}CO_2$ was somewhat faster ($p<0.05$). Subsequently there was no significant difference in the increase in PCO_2 between the three methods of measurement, as analysed for each min or for the total period from 1 to 5 min. The last arterial sample in each patient was taken just before the patient stopped breathing the CO_2 mixture, when the mean P_aCO_2 value for the group was 8.2±1.3 kPa, as against $P_{tc}CO_2$ of 8.0±1.5 kPa ($r=0.78$). After the last markings $P_{tc}CO_2$ continued to rise for a further 25±7 s. The highest $P_{tc}CO_2$ value was 8.2±1.5 kPa (correlation to P_aCO_2, $r=0.78$).

In phase 3, after the CO_2 rebreathing had been stopped, 76 triplets of PCO_2 measurements were obtained (Table 2). There was a short delay in the fall of $P_{tc}CO_2$ (Table 2), but after 3, 5 and 10 min no systematic difference from the pretest values were found.

Ventilatory response. The increase in ventilation (ΔV) for a given rise in PCO_2 (ΔPCO_2) was calculated for all three different types of PCO_2 measurements by a least squares linear correlation analysis (Table 1). When the calculated ventilatory responses based on P_aCO_2 were compared with those based on $P_{ET}CO_2$, r was 0.83, and when they were compared with those based on $P_{tc}CO_2$, r was 0.81. The descending slope (k), which represents the speed of CO_2 elimination (Fig 1), was not correlated to the ventilatory responses (Table 1).

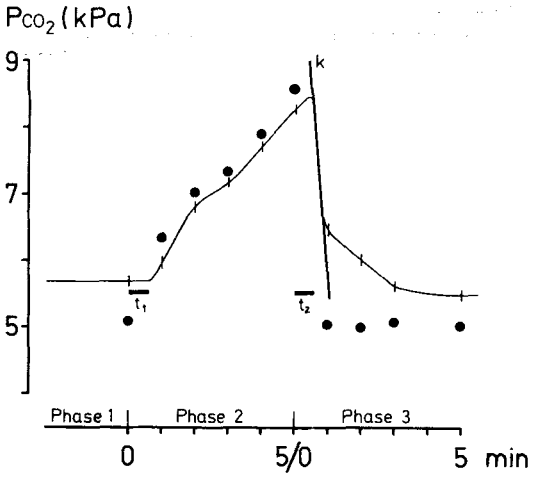


Fig 1. Schematic illustration of a typical $P_{tc}CO_2$ tracing during CO_2 rebreathing test.
● : arterial samples;
t1: the time delay until an increase in $P_{tc}CO_2$;
t2: the time delay until $P_{tc}CO_2$ starts to fall;
k: the speed of CO_2 elimination.

Part II

Sleep studies. The mean total sleep time was 312±61 min, with 79.5 % in non-REM sleep and 20.5 % in REM sleep (Table 3). Seventeen subjects had 60 or more

apneic/hypopneic events, which usually occurred during both REM and non-REM sleep; in six of these 17 subjects such events were significantly longer during REM sleep (Table 3).

Table 3. Results of sleep studies.

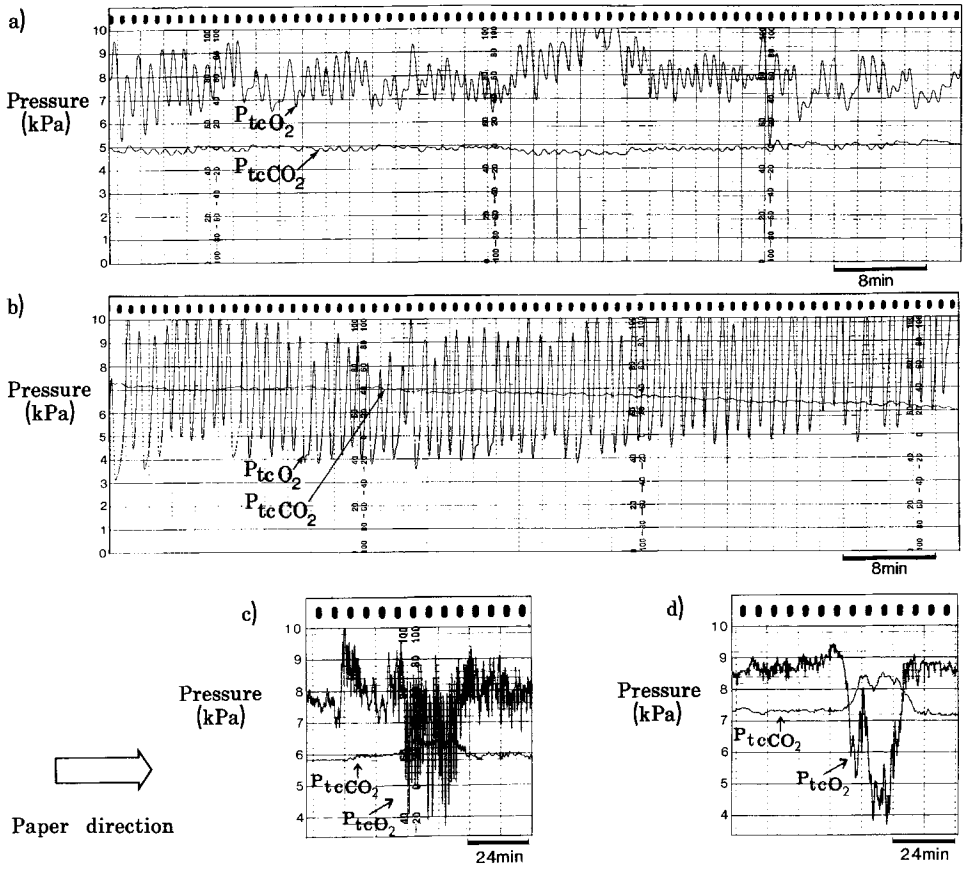
	Duration of sleep			Apneas + hypopneas				
	Total	Non-REM	REM	Number			Mean length (s)	
Case no.	(min)	%	%	Total	Non-REM	REM	Non-REM	REM
1	210	80.8	19.2	0	0	0	-	-
2	314	66.1	33.9	2	2	0	-	-
3	270	85.0	15.0	4	4	0	-	-
4	264	81.0	19.0	7	5	2	-	-
5	329	77.9	22.1	40	40	0	14	-
6	363	81.4	18.6	60	60	0	19	-
7	303	75.4	24.6	79	70	9	19	23
8	239	88.7	11.3	88	85	3	18	-
9	347	77.3	22.7	92	89	3	21	-
10	276	70.7	20.3	111	107	4	20	14
11	283	88.9	11.1	119	95	24	22	38
12	386	81.7	18.3	119	43	76	13	14
13	277	76.7	23.3	122	97	25	15	19
14	311	81.3	18.7	138	92	46	20	30
15	387	89.8	10.2	151	144	7	19	23
16	315	80.8	19.2	161	115	46	27	43
17	189	73.8	26.2	173	130	43	21	21
18	302	82.4	17.6	179	127	52	32	41
19	306	75.3	24.7	306	228	78	15	29
20	450	82.0	18.0	435	365	70	36	42
21	368	73.9	26.1	482	366	116	21	29
22	365	77.0	23.0	680	538	142	22	34
Mean	312	79.5	20.1	161	127	34	21	28
SD	61	5.8	5.5	172	135	41	6	10
n	22	22	22	22	22	22	18	14

PCO_2 during sleep. In the 22 investigated patients 42 sets of comparative recordings were made during sleep with a mean result of P_aCO_2 6.1±1.1 kPa and $P_{tc}CO_2$ 6.1±1.1 kPa (r=0.84).

The $P_{tc}CO_2$ tracings during sleep differed among the investigated patients, mainly with regard to the number, length and distribution of respiratory events. The subjects were divided into three groups according to the total numbers of respiratory events per night.

Group A. This group comprised subjects with <60 respiratory events per night (Cases 1 - 5, Table 3). They all showed a "stable" $P_{tc}CO_2$ tracing without variations, except when changing their body position. This was even true for subject 3, who suffered from chronic bronchitis and CO_2 retention. His $P_{tc}CO_2$ was stable around 8.0 kPa the whole night. Subject No. 2 also suffered from chronic bronchitis, but his SAS had been sucessfully treated 6 months earlier. He had almost no apneas, but during his REM sleep periods $P_{tc}CO_2$ increased from an average of 6.2 to 7.4 kPa, the elevated value persisting through the entire REM period.

Fig 2. Tracings from continuous $P_{tc}CO_2$ (and $P_{tc}O_2$) measurements in a) case 15 (Tables I and III) with repeated short (mean 19 s) apneic episodes but only a temporary increase in $P_{tc}CO_2$, b) case 20 with long (mean 36 s) apneic episodes and an additive increase in $P_{tc}CO_2$, c) case 19 with repeated apneic episodes and CO_2 retention especially during REM sleep and d) a women with REM sleep related hypoventilation.



Group B. This group consisted of ten patients with 60-151 respiratory events (Cases 6 - 15, Table 3). The mean length of their respiratory events was 21 s, and only in one case they were significantly longer during REM sleep (Table 3). During a typical cluster of apneas and hypopneas, each event was followed by an increase in $P_{tc}CO_2$ by 0.1 to 0.3 kPa, but there was no additive increase in this variable (Fig 2a). The $P_{tc}CO_2$ tracings were similar for all patients in this group, but subject No. 7 showed in addition an increase in $P_{tc}CO_2$ from 6.0 to 6.9 kPa during REM sleep.

Group C. This group comprised the seven remaining patients, who had the most numerous and the longest abnormal respiratory events, especially during REM

sleep (Cases 16 - 22, Table 3). As in group B, each apnea was followed by a 0.1 to 0.3 kPa increase in $P_{tc}CO_2$, and in addition there was an accumulating increase in the basic level of $P_{tc}CO_2$, as exemplified by subject No. 20, in whom the repeated apneas led to an increase in $P_{tc}CO_2$ from 6.0 to 7.3 kPa (Fig 2b). In all seven patients the increase in $P_{tc}CO_2$ was most pronounced during REM sleep (Fig 2c).

The $P_{tc}CO_2$ electrode has proved to be a valuable tool in a number of clinical settings, especially when central hypoventilation is involved. Fig 2d is a tracing from a 55 year-old scoliotic woman who had contracted poliomyelitis 30 years previously. She was admitted because of early morning headache, confusion and tiredness. Repeated sleep registrations showed REM-sleep-induced hypoventilation with hypoxia and hypercapnea.

Tolerance and electrode stability. The transcutaneous electrode was well tolerated and the only remark made by the patients was that they had mild local erythaema for 1-2 days. Changing of the electrode placement after four hours was a disadvantage, as the patients often felt disturbed, especially the "healthier" ones. Our main technical problem in the beginning was difficulty in attaching the electrode securely, so that it should not fall off during sleep. The absolute mean electrode drift between calibration and to the end of the sleep studies was 9.4 per cent and to the end of the CO_2 rebreathing tests (average 77+18 min) was 9.8 per cent. The electrode drift was similar for all patients and the results were not corrected for this drift.

DISCUSSION

The correlation between $P_{tc}CO_2$ and P_aCO_2 (r=0.79) in adults at rest is acceptable for clinical purposes, although it is somewhat lower than has been found by other authors (25), especially if compared with results in infants (11, 21). A possible explanation is that our measurements fell "statistically" within a rather narrow range (Table 1).

From the beginning of CO_2 rebreathing until $P_{tc}CO_2$ increased (Fig 1), there was a delay of 40 s (Table 1), and after the CO_2 rebreathing there was a delay of 26 s until $P_{tc}CO_2$ began to fall. This would be due both to the dead space of the instruments and to the time needed for CO_2 to diffuse through the skin (6). But with the rather high electrode temperature of 44 ^{o}C in our study, the real reactivity to fast PCO_2 fluctuations is considerably shortened (8). After the initial delay in the beginning of phase 2, all three methods for PCO_2 measurement reflected the PCO_2 changes in a similar manner (Table 2).

The results of the ventilatory response tests calculated from P_aCO_2, from $P_{ET}CO_2$ and from $P_{tc}CO_2$ measurements are acceptably correlated (Table 1). The ventilatory response to CO_2 during sleep in newborn infants has been estimated

by means of $P_{tc}CO_2$ electrodes (13). Other authors have not found $P_{tc}CO_2$ electrodes reliable for this purpose in healthy children (7), but on the basis of our results we believe that these electrodes can be used to develop more simplified ventilatory response tests in adults.

In contrast to the "standards" proposed by Martin et al. (18) the present findings indicate that $P_{tc}CO_2$ monitoring in adults during sleep adequately reflects changes in P_aCO_2, as has previously been demonstrated in infants (17). We have found this method to be a useful complement in traditional sleep studies, when investigating patients suspected of sleep-related breathing disorders. Our observations indicate that those SAS patients with the lowest ventilatory response to CO_2 also have the greatest CO_2 retention during sleep, especially during REM sleep. Hypoventilation can be technically difficult to detect in sleep studies, especially if a pneumotachograph is not used. Monitoring of $P_{tc}CO_2$ under such conditions is useful for evaluating one of the major consequenses of hypoventilation, namely CO_2 retention. This is in accordance with the results of a previously reported investigation of a 3 month-old child with apneas of suspected central origin (20) and with a report on a patient with bilateral phrenic nerve paralysis associated with an increase in $P_{tc}CO_2$ during REM sleep (11). We have also found monitoring of $P_{tc}CO_2$ useful in evaluating chest physiotherapy in patients with CO_2 retention (14), and that it is superior to intermittent arterial blood sample analyses.

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