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Gas Exchange in Two Male Age Groups in Relation to Inspiratory Oxygen Fraction, Physical Exercise and Body Posture

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ABSTRACT

Two groups of male subjects, mean age 25 and 50 years, were studied at rest supine during air breathing, 15–11 and 100 % O_2 breathing, sitting at rest and during exercise up to maximal work load, and supine during a constant submaximal work load combined with D_{LCO} measurements. At rest supine, the mean and range of P_{aO_2} was 90.2 (79–97) in the young and 78.2 (68–88) mmHg in the middle-aged. P_{A-aO_2} was 4.3 (–1–14) and 23.4 (15–33) mmHg, respectively. Corresponding S_{aO_2} values were 97.3 (95.7–99.0) and 94.5 (91.7–98.6) per cent. During hypoxia, the P_{aO_2} and P_{A-aO_2} values were lower in the young than the middle-aged. During hyperoxia the P_{aO_2} and P_{A-aO_2} were 611 (574–644) and 48(31–60) in the young and 573 (492–612) and 93 (60–108) mmHg in the middle-aged, respectively. V_D and V_D/V_T were constantly higher in the middle-aged. At max. work load, no great difference in P_{aO_2} (87.5 and 85.8 mmHg) or S_{aO_2} (95.8 and 94.2 %) was found. The mean D_{LCO} values were 39.2 (young) and 36.8 (middle-aged) ml/mmHg·min, as measured at $\dot{V}_{O_2}$ 1 490 and 1 656 ml/min, and at $\dot{V}_E$ 35.9 and 43.6 l/min, respectively.

INTRODUCTION

According to our present practice the routine evaluation of the physiological function in respiratory diseases consists of static and dynamic spirometric examinations, which give an idea of the pulmonary volumes and air-flow conditions. Information on the combined cardiopulmonary and circulatory function is often of great value, and to obtain this a graded exercise test is indicated. These different methods are well known, generally applied and based on the findings in extensive normal series from several countries including our own.

In addition to these types of examination there is reason in many cases to investigate details of the ventilation, diffusion and circulation. For example, dead space determinations, measurements of the diffusion capacity, and examination for the presence of abnormal alveolo-arterial gradients and shunts are often necessary for establishing the type and degree of severity of a disease with greater certainty. Methods for such examinations suitable for clinical use have long been available, and normal values for different factors, e.g. the pulmonary diffusion capacity at rest and during work or arterial oxygen partial pressures on inhalation of gas mixtures with different oxygen concentrations, have been reported by several authors.

Despite the fact that the normal reactions in many of these respects are thus largely known, there is a need for complementary studies. It is conceivable that population differences will give considerable variations, and technical advances in the analytical methods increase the need for repeated control studies. The present investigation served two purposes. Firstly it comprised a control of a large number of clinical examination methods and routine analyses used in our laboratory, and secondly it provided a reference material. The same examination procedures were used as were chosen previously for an extensive clinical study of patients with ventilatory insufficiency (Tammivaara-Hilty, to be published). In view of the knowledge that the unevenness between ventilation and perfusion increases with age, two age groups were studied. Consideration was also taken of the smoking habits among the subjects and the reference groups thus included both smokers and non-smokers. Since patients with ventilatory limitations are not seldom given a work load of practically maximal level in an exercise test, it was considered important that the reference subjects should also work against a load

as near to the maximum as possible. Only a few such studies appear to have been performed previously.

MATERIAL

The material consisted of two groups: 1) 6 university students, of whom 3 were moderate to heavy smokers, two previous smokers and one a non-smoker, and 2) 5 randomly selected subjects awaiting cholecystectomy with no symptoms for several months at the time of investigation: 3 of these were moderate to heavy smokers, one a previous heavy smoker and one a non-smoker. The results of a primary health examination are presented in Table I. This included pulmonary roentgenography and measurement of the heart volume, in the young supine (11) and the middle-aged sitting, with calculations according to Jonsell (34), measurement of body height and weight, total haemoglobin (THb, g) according to the alveolar CO method (59), lung volumes by a closed helium technique and ventilatory capacity by a light-weight spirometer (10, 12, 24), peak expiratory flow rate (PEF, l/min) by a Wright's peak flow meter (70) and physical working capacity (58, 60, 62, 68).

PROCEDURE

After a proposed smoking abstinence[1] from 5 p.m. on the previous day the investigation was performed with the subject in a fasting state; an exception was made for the young group, who were given a light meal before the exercise test. The oral temperature was measured at rest and immediately after exercise. Under local anaesthesia a catheter was inserted into the left brachial artery – in 4 volunteers a polyethylene quide and catheter (PP 160) were inserted by the Seldinger technique (55) using a Luer-Lock needle (40 × 1.2 mm) and in 7 volunteers an infusion cannula (70 × 1.15 mm "Infartkanyl", AB Stille, Stockholm). Both were kept patent by infusion of heparin in physiological saline (5 000 IU/1 000 ml).

The arterial blood samples for blood gas analyses were withdrawn during at least 30 sec into 5 ml glass syringes with a heparin (1 000 IU/ml) filled dead space, which were sealed immediately and kept in ice-water if not analysed immediately. The blood samples for arterial oxygen saturation, haemoglobin and haematocrit measurements were also withdrawn during at least 30 sec, into 10 ml glass syringes, with a heparin (1 000 IU/ml) filled dead space, which were sealed immediately after a drop of mercury had been added for more efficient mixing of the sample, and kept at 4°C pending analysis. The arterial blood samples for

[1] See Table I for S_{CO} % according to the alveolar CO methods in connection with THb measurements.

Table I. *Physical and physiological findings in two reference age groups*

	Group I ($n=6$)		Group II ($n=5$)	
	$\bar{X}$	Range	$\bar{X}$	Range
Age, yrs	25	21–28	50	48–52
Height, cm	179	174–184	173	169–180
Weight, kg	73	63–88	76	62–87
Heart volume, ml/m² body surface area	375	329–444	424	390–435
TLC, 1 BTPS	6.8	5.6–8.1	6.5	5.8–7.1
TLC, % of pred.	91	78–104	92	85–96
FRC, 1 BTPS	3.3	2.1–4.8	3.3	2.4–3.9
RV, 1 BTPS	2.0	1.3–3.5	2.2	1.8–2.4
VC, 1 PTPS	5.0	4.8–5.6	4.6	4.1–4.8
VC, % of pred.	87	80–95	91	84–94
$FEV_{1.0}$, 1 BTPS	4.2	3.8–4.7	3.3	2.9–3.6
$FEV_{1.0}$/VC, %	83	77–88	72	68–78
MVV_F, 1 BTPS/min	199	173–222	165	153–190
MVV_F, % of pred.	100	85–116	105	94–119
MVV_{40}, 1 BTPS/min	145	135–156	126	111–133
MVV_{40}, % of pred.	89	82–98	99	88–106
PEF, l/min	573	495–620	527	480–590
W_{max}, kpm/min (a)			1 100	900–1 200
(b)	1 220	1 115–1 350	1 050	900–1 200
W_{170}, kpm/min (a)			1 155	950–1 265
(b)	835	760–900	1 075	940–1 305
W_{150}, kpm/min (a)			900	775–1 025
(b)	655	575–770	820	740–955
HR at W_{max}, beats/min (a)			166	162–170
f at W_{max}, cycles/min (a)			33	26–36
THb, g	630	605–666	592	443–739
THb, g/kg body weight	8.7	7.1–10.0	7.8	7.0–8.5
Initial S_{CO}, %	0.86	0.56–1.10	1.35	0.58–3.12

(a) Conventional physical work test.
(b) Work test combined with measurement of ventilation by Douglas bag method and arterial blood sampling through brachial arterial catheter.

lactic acid estimations were taken into heparinized plastic tubes (Vitrum AB, Stockholm) and 0.05 ml of the sample was transferred into 4 ml of 10 % trichloroacetic acid as soon as possible.

During the investigations in the supine position the arterial blood pressure recordings were made with transducers of variable capacitance type (EMT 34 Elema-Schönander AB, Stockholm) connected to amplifier units and recorded on a direct-writing galvanometer recorder (Mingograf 81, Elema-Schönander). Mean pressures were obtained by electrical integration. As reference point for zero pressures the horizontal level 5 cm below the sternal insertion of the fourth rib was used. Calibration was performed by means of a water manometer before every investigation. During investigations in the sitting position the arterial blood pressure was measured indirectly by the conventional cuff method. During air breathing and hypoxia the expiratory gas was collected through a rubber mouthpiece (inner diameter 20 mm) inserted through a one-way low-resistance respiratory valve ("Arbetsventil", inner diameter 25 mm, Gothenburg model, Kifa AB, Stockholm) and a rubber tube (inner diameter 25 mm) into a Douglas bag, which had been flushed with expiratory gas for a few minutes. During pure oxygen breathing a one-way Lovén respiratory valve (Medihus AB, Stockholm) was used and the ventilation was measured as above.

The experiment was performed with the following sequence of simultaneous expiratory gas and arterial blood gas sampling procedures:

Procedure 1 (Resting, supine, ambient air). Expiratory gas was collected for 10 min after an adaptation period of a few minutes. Arterial blood samples were taken after direct arterial blood pressure (BP) measurement in the middle of the gas collection period. The heart rate (HR) and respiratory rate (f) were measured every second min.

Procedures 2–3 (Resting, supine, two hypoxic gas mixtures). The first hypoxic gas mixture, 14–16 % O_2 in N_2 (13.95 % in 5 of the young group and 15.62 % in the others), and the second hypoxic gas mixture, about 11 % O_2 in N_2 (10.35 % in the young group, 10.80 % in two and 11.38 % in three members of the middle-aged group), were each breathed for 10 min in direct sequence with expiratory gas collection during the last 5 min of each period. Arterial blood samples were taken in the 6th–8th min. HR, f and BP were measured every second min during, HR and BP also 1, 4 and 10 min after, breathing of hypoxic gas mixture.

Procedure 4 (Resting, supine, pure oxygen). The expiratory gas N_2 concentration was recorded according to the multibreath technique (42, 43) and the expiratory gas was collected until its N_2 content reached 2 % in order to estimate the lung clearance index (LCI with FRC from He spirometry). No correction was made for excreted tissue N_2. Arterial blood was sampled both after 2 % N_2 had been reached and after a 15-min period (including 2–3 deep inspirations) of pure oxygen breathing. The ventilation was also measured between the 12th and 17th min. HR, f and BP were measured 5, 10 and 15 min during and 10 min after oxygen breathing.

Procedure 5 (Resting, sitting on a bicycle ergometer, ambient air). Same measurements as in procedure 1, except that BP was measured indirectly in the arm contralateral to the arterial catheter.

Procedures 6–10 (On a bicycle ergometer with stepwise increasing work loads in kpm/min from 300, 600, 900 and so on to maximal work load, sitting, breathing ambient air). The expiratory air was collected during the last 3 min of each 6 min period at each work load, including the maximal work load. However, if the maximal work load was interrupted earlier, gas collection was performed during the last minute or two. Arterial blood samples were taken during gas collection, and HR, BP and ECG were measured and recorded conventionally, but f was counted repeatedly during the gas collection period. Arterial blood samples were taken for lactate determination also 2–3 min after exercise. ECG was recorded continuously for a few minutes after exercise with the subject sitting up, and 4 and 10 min in the supine posture after exercise. HR and BP were measured 10 min after exercise.

Procedure 11 (On bicycle ergometer at 600 kpm/min, supine, ambient air). The expiratory gas was collected and the arterial blood sampled as in procedures 6–9. HR and direct BP were measured continuously and f repeatedly at least once every second minute.

Procedure 12 (Continuing at 600 Kpm/min, supine, inspiring about 0.05 % CO in air). The expiratory gas for measurement of D_{LCO} was collected for 1½–2 min after the subject had breathed the CO-air gas mixture for 2 min, during which period the Douglas bag was flushed twice with expiratory gas. HR and BP were recorded co tinuously during and 1, 4 and 10 min after exercise and$f$ was measured continuously during breathing of the CO-air mixture.

The number of subjects studied by procedures 1–6, 10 and 12 was usually 6 in the young group and 5 in the middle-aged group. For procedures 7–8 it was 5 in each group, with the exception that $\dot{V}_E$, $\dot{V}_{O_2}$ and R were measured in 6 subjects in the young group. For procedure 9 (1 200 kpm/min) there were 5 subjects in the young and 2 in the middle-aged group. The lactate concentration was measured in the young group in procedures 10 (W_{max}) and 11 (600 kpm/min, supine) in only 5 subjects.

ANALYTICAL METHODS

The *expired gas volumes* were measured as described from our laboratory by Renck (49) and converted from ATPS to BTPS assuming the body temperature to be 37°C.

Quantitative gas analyses were performed of

1. O_2 and CO_2, as volume per cent in inspiratory and expiratory gas by means of the Enghoff modification (19) of the Haldane apparatus. The composition of the inspiratory gas from tubes (AGA AB, Lidingö, Sweden) was checked by a series of 5 Enghoff analyses (allowed fractional difference at most 0.05 vol%). Single analyses were made of the expiratory gas. However, the method is checked daily by duplicate analyses, the error of a single determination at the time of the present study being 0.0034 vol% (1.49%) for CO_2 and 0.0067 vol% (0.6%) for O_2.

2. The CO-fraction (F_{ICO} and F_{ECO}); this was measured in an infrared gas analyser (URAS 2, Hartman & Braun Ltd, Frankfurt/Main) by reading the analysed gas against calibration gases with a concentration of about 0.0005% CO in O_2 on a potentiometric writer (Linecomp; Hartman & Braun Ltd.), but correcting the fractions after calibration procedures with 2 gases with CO concentrations of about 0.05% CO in O_2.

The *arterial oxygen tension* (P_{aO_2}, mmHg) was measured polarographically by a Clark electrode (E5046 Radiometer, Copenhagen), but following the principles given by Hilty & Karendal (26). The electrode and humidified calibration gases were thermostated at 37°C. The calibration for measurements of P_{aO_2} during ambient air breathing and hypoxia was performed with approximately 10% O_2 and 5.5% CO_2 in N_2 and during pure oxygen breathing with approximately 6.5% CO_2 in O_2. At calibration a difference of at most 2 mmHg was allowed before and after analysis, just as between duplicate analyses. At high P_{aO_2} levels the highest single P_{aO_2} value was accepted for measurements of veno-arterial shunting if the calibration gas readings before and after analysis differed by no more than 2% (12 mmHg). The blood gas factor at all P_{aO_2} levels was assumed to be 1.045, which is an approximation of values found by several authors, including Rhodes & Moser (50) and Karendal (35). No correction for the heparin-filled syringe dead space was made. In the present study the mean difference and its S.E.M. between 85 duplicate P_{aO_2} analyses of one sample at air breathing P_{aO_2} levels was -0.33 ± 0.14 mmHg (S.D. 1.25), of 11 analyses at P_{aO_2} levels of approximately 14% O_2 breathing $+0.18 \pm 0.38$ mmHg (S.D. 1.25), of 11 analyses at P_{aO_2} levels of approximately 11% O_2 breathing $+0.18 \pm 0.35$ mmHg (S.D. 1.17) and of 19 analyses at P_{aO_2} levels of 100% oxygen breathing $+4.74 \pm 2.33$ mmHg (S.D. 10.2). The mean difference and its S.E.M. between the first analyses of 11 paired blood samples taken in sequence after 15 min 100% O_2 breathing was $+0.63 \pm 2.38$ mmHg (S.D. 7.9).

The *arterial carbon dioxide tension* (P_{aCO_2}, mmHg) was measured with a glass electrode (E5036, Radiometer) thermostated at 37°C, which is calibrated daily with two humidified gases thermostated at 37°C, about 3% CO_2 in O_2 and 6.5% CO_2 in O_2 and before and after each analysis with about 6.5% CO_2 in O_2. A difference of no more than 1 mmHg between the gas readings and 2 mmHg between the duplicate analyses was allowed. Readings were made on a digital acid base analyser (PHM 72, Radiometer). The results are given at the body temperature at the time of the procedures. The mean difference and its S.E.M. of our 124 duplicate P_{aCO_2} analyses (at levels ranging from 29.6–46.3 mmHg) was $+0.27 \pm 0.04$ mmHg (S.D. 0.50), the first measurement showing significantly higher values ($P < 0.001$).

The *arterial pH* was measured with a micro glass-calomel electrode (E 5021, G 297/K 497, Radiometer) and pH meter 27 with a gas monitor (PHA 927, Radiometer) by calibrating the apparatus with Sörensen standard precision buffers (S 1510, pH at 37°C 7.38 ± 0.05 and S 1500, pH at 37°C 6.841 ± 0.005, Radiometer) and with a buffer with pH about 7.37 before and after each analysis. The mean difference and its S.E.M. of 135 duplicate pH analyses (at levels ranging from 7.260–7.512) was -0.0023 ± 0.0006 (S.D. 0.0074) units.

The *standard bicarbonate* (HCO_3^-, mEq/l) and *base excess* (BE, mEq/l) were read by means of the blood gas calculator (BGC Type 1, Radiometer) constructed by Severinghaus (57) taking into account the haemoglobin values at the time of the procedures.

The *arterial oxygen saturation* (S_{aO_2}, %) was determined in hemolysed whole blood according to the method of Drabkin & Schmidt (17) as modified by Holmgren & Pernow (32) and described by Karendal et al. (36), using a Beckman B spectrophotometer (Beckman Instruments Inc., Fullerton, California.) The fractions of the extinctions, read at wavelengths of about 505 nm (isosbestic point) and at 475 nm, were allowed to differ at most 0.006 units in duplicate analyses. The cuvette factors were read before each analysis. The error of a single determination was 0.00024 ext. units (variation coefficient 0.19%). S_{aO_2} was also estimated by a filter spectrophotometric technique using 3 wavelengths, 548, 568 and 578 nm (Model 182 CO-oximeter; Instrumentation Laboratory Inc., Lexington, Mass.). The mean difference and its S.E.M. between Beckman and CO-oximeter S_{aO_2} from the same syringe was 0.39 ± 0.23 S_{aO_2}% (S.D. 1.71) in 58 measurements ($P = 0.05$–0.10) at air breathing levels. The corresponding mean difference and its S.E.M. between 9 paired analyses at levels of 14–16% O_2 breathing was -0.27 ± 0.39 S_{aO_2}% (S.D. 1.17), $P = 0.50$–0.60, and between 9 paired analyses at levels of about 11% O_2 breathing -1.32 ± 0.60 S_{aO_2}% (S.D. 1.81), $P = 0.05$–0.10. The equation between Beckman and CO-oximeter S_{aO_2} measurements from the same sample can be written as $y = 7.31 + 0.92 \cdot x$, $r = 0.98$ (Beckman x, CO-oximeter y).

The *haemoglobin concentration* (Hb, g/l) in arterial blood was given as the mean value of triple spectrophotometric (Hitachi Ltd., Tokyo) analyses at 540 nm by a cyanmethaemoglobin method when Hemicyanid (Kabi AB, Stockholm) solution was used as standard. Correction was made for the heparin-filled syringe dead space. The S.E.M. of 93 triple analyses was 0.13 g/l (S.D. 1.2) with a variation coefficient of 0.86%.

The *haematocrit* (Hct, %) was estimated from arterial

blood samples with a microcapillary method using heparinized capillary tubes (length 75 mm) and a microcapillary tube centrifuge (Model MB, International Equipment Co, Boston, Mass.,) without correction for trapped plasma.

The *arterial lactate* (Lact., mmol/l) was measured after deproteinization with trichloroacetic acid according to Ström's (61) modification of the method of Barker & Summerson (5). A single sample was taken from 2–3 ml of blood and duplicate analyses were performed. The coefficient of variation was 5.2%.

CALCULATIONS AND DEFINITIONS

Pulmonary minute ventilation ($\dot{V}_E$, 1 BTPS/min)

Oxygen uptake ($\dot{V}_{O_2}$, ml STPD/min) was calculated from $\dot{V}_E$ with volume corrections and differences in $F_{I_{O_2}}$ and $F_{E_{O_2}}$.

Carbon dioxide elimination ($\dot{V}_{CO_2}$, ml STPD/min) was calculated from $\dot{V}_E$ and F_{ECO_2}.

Expiratory gas exchange ratio (R).

Ventilatory equivalent for oxygen ($\dot{V}_E \cdot 100/\dot{V}_{O_2}$)

Tidal volume (V_T, ml BTPS).

Physiological dead space (V_D, ml BTPS). Calculated from Bohr's equation (14) and its modification by Enghoff (18), assuming $F_{ICO_2} = 0$ and $P_{ACO_2} = P_{aCO_2}$. The dead space of the mouthpiece and respiratory valve (anatomic dead space) of 45 ml was subtracted.

Alveolar ventilation ($\dot{V}_A$, 1 BTPS) from a modification of Bohr's equation.

Alveolar oxygen tension ($P_{A_{O_2}}$, mmHg) was estimated during ambient air breathing and hypoxia according to Asmussen & Nielsen (2), assuming $F_{ICO_2} = 0$ and $P_{ACO_2} = P_{aCO_2}$. During pure oxygen breathing $P_{A_{O_2}}$ was calculated according to the two following equations, assuming $P_{A_{O_2}} = P_{a_{CO_2}}$:

$$P_{A_{O_2}} \sim 0.99 \times (P_B - 47) - P_{aCO_2} \text{ (used in veno-arterial shunt estimations)}$$

and

$$P_{A_{O_2}} = (P_B - 47) - P_{aCO_2} - F_{E_{N_2}}(P_B - 47)$$

Alveolo-arterial oxygen tension difference ($P_{A-a_{O_2}}$, mmHg).

Veno-arterial shunting ($\dot{Q}_{sh}/\dot{Q}_{syst}$, %) was calculated at 100% O_2-breathing, as described among others by Berggren (9) and Linderholm (41), from the relation $C_{CO_2} - C_{a_{O_2}}/C_{CO_2} - C\bar{v}_{O_2}$ when C_{CO_2} is end-capillary, $C_{a_{O_2}}$ arterial and $C\bar{v}_{O_2}$ mixed venous blood oxygen content and $C_{CO_2} = C_{a_{O_2}} + (P_{A-a_{O_2}}) \times (0.023/760) \times 100$ and arteriovenous O_2 difference ($a\bar{v}_{O_2}$) was assumed to be 4 ml/100 ml blood.

Diffusing capacity for carbon monoxide (D_{LCO}, ml STPD/mmHg·min); this was calculated according to Linderholm's principles (39) as modified by Holmgren (28) in his equation for use in healthy persons, when the capillary CO pressure is not estimated from the measured blood CO content. This differs from the original Filley et al. (21) method by taking into account the capillary CO pressure.

$$D_{LCO}\ (\text{Holmgren}) = 0.97 \times D_{LCO}\ (\text{Filley}) + 4.6$$

$$= 0.97 \times \frac{\dot{V}_{CO}}{P_{ACO}} + 4.6$$

Carbon monoxide uptake ($\dot{V}_{CO}$, ml STPD/min) and alveolar carbon monoxide tension (P_{ACO}, mmHg) were calculated according to the same principles as the corresponding parameters for oxygen.

Total arterial oxygen content (C_{O_2f}, ml/l blood) includes both C_{O_2Hb} (1.39 ml O_2/g haemoglobin) and C_{O_2f} (physically dissolved oxygen) calculated according to Sendroy et al. (56).

RESULTS

Fig. 1 and Tables II, III present the results of the different procedures as mean values and ranges separately for the young and the middle-aged groups. The values obtained are included in two columns (work loads of 900 or 1 200 kpm/min and W_{max}, respectively) only if the subjects continued with the maximal work load for 6 minutes. At W_{max} the $\dot{V}_E$ was 43 (31–65) % of the MVV_F values measured at dynamic spirometry in the young and 43 (38–48)% in the middle-aged group.

D_{LCO} (ml/mmHg·min), measured about half an hour after the maximal work test, supine at 600 kpm/min, was 39.2 (29.9–48.7) in the young and 36.8 (33.4–41.1) in the middle-aged group. The simultaneously measured $\dot{V}_{O_2}$ and V_E are given as mean values and ranges for each group in Tables II and III. The corresponding oxygen pulse was 72 (62–84)% and 89 (78–96)% of the maximal oxygen pulse in the sitting posture and $\dot{V}_E$ was 43 (31–54) % and 62 (45–75) % of the maximal $\dot{V}_E$ at W_{max} in a sitting posture in the respective groups.

During hyperoxia $\dot{V}_E$ decreased at the beginning of 100% oxygen breathing, corresponding to the lung N_2-washout period, to 6.7 (4.3–11.0) in the young and to 7.7 (6.8–8.2) l/min in the middle-aged group, and at the same time LCI was 6.6 (5.3–9.2) and 7.5 (6.4–8.5) in the respective groups. At 12–17 min of oxygen breathing $\dot{V}_E$ had increased to values exceeding $\dot{V}_E$ at rest during ambient air breathing (Fig. 1). There was a mean difference in the $P_{A_{O_2}}$

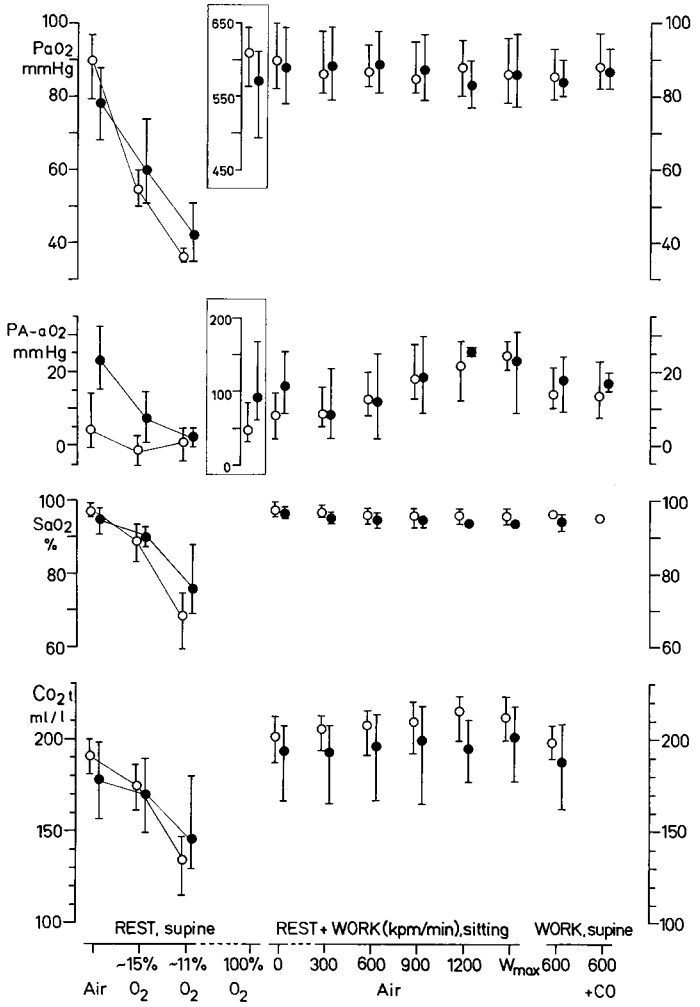

Fig. 1. Arterial oxygen tension ($P_{a_{O_2}}$, mmHg), alveolo-arterial oxygen tension difference ($P_{A-a_{O_2}}$, mmHg), arterial saturation ($S_{a_{O_2}}$, %) and total arterial oxygen content (C_{O_2t}, ml O_2/l blood) as mean values and ranges in the two groups: 6 young men (○) and 5 middle-aged men (●). Explanations: $CO = F_{ICO} \sim 0.0005$ in air; mean W_{max} and range in the young group 1 220 (1 100–1 350) and in the middle-aged group 1 050 (900–1 200) kpm/min; usually 5–6 observations, see Procedure.

values calculated in the two different ways (described under Calculations) of 3 (2–5) mmHg in the young and of 2 (–3–6) mmHg in the middle-aged group. The mean $P_{a_{O_2}}$ values obtained during 100% O_2 breathing, immediately after the expiratory gas contained less than 2% N_2, were 598 (543–628) mmHg in the young and 484 (369–575) mmHg in the middle-aged group; these values were constantly lower than values obtained after a 15-min period of pure oxygen breathing including 2–3 deep inspirations. The veno-arterial shunting ($\dot{Q}_{sh}/\dot{Q}_{syst}$) based on the highest $P_{a_{O_2}}$ value obtained during 100% O_2 breathing in the supine posture was 3.5 (2.6–6.0)% in the young and 6.5 (4.3–11.3)% in the midle-aged group, assuming the $a\bar{v}_{O_2}$ difference to be 4 ml O_2/100 ml biood in all subjects.

DISCUSSION

At rest supine, breathing ambient air, the mean $P_{a_{O_2}}$ value is higher and the mean $P_{A-a_{O_2}}$ value lower in the young than in the middle-aged group. The tendency to a decreasing $P_{a_{O_2}}$ and increasing $P_{A-a_{O_2}}$ with age is in agreement with the findings of other authors (15, 27, 45, 47, 54, 65). In our series the difference between the two groups can be explained by greater ventilatory maldistribution and veno-arterial shunting, possibly including a higher $a\bar{v}_{O_2}$ difference (not measured) in the middle-aged group, which was observed during 100% O_2 breathing in the same body posture.

In the sitting posture the mean $P_{a_{O_2}}$ in the young was the same as, and in the middle-aged group

higher than in the supine posture. The difference between the groups was therefore small. The mean $P_{A-a_{O_2}}$ increased in the young and decreased in the middle-aged group with change in posture. Several other authors have found a larger decrease in $P_{a_{O_2}}$ and increase in $P_{A-a_{O_2}}$ in the sitting posture than we did (45, 54). As the increases in $\dot{V}_E$, $\dot{V}_A$ and $P_{A_{O_2}}$ were about the same in both groups, the differences are due to $P_{a_{O_2}}$ values. In the young group the increase in ventilation failed to increase the $P_{a_{O_2}}$ values as the venous oxygen content probably decreased and lung perfusion probably became more unequal along the lung in the sitting posture, causing on account of a "shunt-like effect" a decrease in $P_{a_{O_2}}$. Bake, Bjure & Widimsky (4), using ^{133}xenon in a study on healthy subjects below the age of 40 years, found large regional differences along the lung in the sitting posture, which were shown with $^{15}CO_2$ by West who considered them to be due to hydrostatic factors (69).

The absolute level of our $P_{a_{O_2}}$ values at rest is higher than values obtained by Hofer & Scherrer (27) in a Fleisch metabograph at an approximate P_B of 710 mmHg. Though there is no great disagreement between our findings and those of other authors (6, 8, 20, 44, 65, 66), our $P_{a_{O_2}}$ values at rest in the young group in the sitting posture are lower than those reported by Raine & Bishop (47) and Mellemgaard (45) and the $P_{a_{O_2}}$ values at rest in the middle-aged group in the supine posture lower than those published by Hartley et al. (25). In resting conditions $P_{a_{O_2}}$ values seem to be easily influenced by the following factors: 1) smoking with increased ventilation/perfusion inequalities (most authors do not report the smoking in their investigations nor the lengths of smoking abstinence), 2) metabolic state (our subjects investigated fasting) as shown by Salzman & Salzano (53), who proved that the intake of carbohydrate rich food before the investigation increases the $P_{a_{O_2}}$ values significantly by increasing alveolar ventilation, 3) body posture and 4) ventilatory adaptation to breathing through a mouthpiece and ventilatory valve.

Our mean V_D and to a lesser degree also the V_D/V_T values are higher than have been found by earlier authors (1, 44, 47). In these latter studies, however, the ranges were wider than ours and, further, a different technique was used in estimations of $P_{a_{CO_2}}$. Vale (66), however, who also used a $P_{a_{CO_2}}$ electrode, reported only slightly lower V_D and V_D/V_T values than ours. Values for $P_{a_{CO_2}}$ obtained by an electrode are, on the average, 3 mmHg higher than those obtained by the Astrup technique (29). Another possible reason for our relatively high V_D and V_D/V_T values is that the real dead space of our ventilatory valve is larger than the subtracted anatomic dead space of the valve. As the precision of $P_{a_{CO_2}}$ by the electrode method is better than by the Astrup (29) technique, we consider our results to be more reliable than the earlier findings with other techniques.

During hypoxia the two age groups seem to show different types of reactions. The young have mainly a circulatory reaction with increasing HR and probably cardiac output and a slightly decreasing arterial blood pressure. Simultaneously with increased $\dot{V}_A$ in both groups the increase of V_D and V_D/V_T in the young group is smaller than in the middle-aged group, where the simultaneous HR and BP increases are slight. It has been suggested by Scherrer et al. (54) that the higher ventilatory increase in the old might be due to a higher sensitivity of the respiratory centre to hypoxia on account of cerebral arteriosclerotic changes. As our middle-aged group is considerably younger than the "old" group of Scherrer et al. (54) we consider several mechanisms to be involved, but an analysis of this question was beyond the scope of the present study.

Our finding of a decreasing $P_{A-a_{O_2}}$ during hypoxia differs from the results of Lilienthal et al. (38), who found no change in $P_{A-a_{O_2}}$ during hypoxia, and of Bartels et al. (7), who found an increased $P_{A-a_{O_2}}$ during hypoxia, but agrees better with the observations of Hofer & Scherrer (27), who showed negative $P_{A-a_{O_2}}$ differences in a few normal persons at $P_{a_{O_2}}$ levels corresponding to 15% O_2 breathing, as in some persons in our young group. The reason for the low $P_{A-a_{O_2}}$ is not known. The constant blood gas factor and a hysteresis effect in the electrode might give incorrect $P_{a_{O_2}}$ values. Further, at the actual P_{O_2} levels the Hb molecules have a great affinity to O_2 and a simultaneous hysteresis effect could result in a too low $P_{A-a_{O_2}}$. Finally the blood sampling period might not be representative for steady state conditions

During hyperoxia (100% O_2) signs of greater ventilatory inhomogeneity were seen in the older group. Assuming that the $a\bar{v}_{O_2}$ difference is the same in both groups, veno-arterial shunting is more prominent in the older group, as has also been found by other authors (15, 27, 45) but not under conditions of exercise by Scherrer &

Table II. *Circulatory, ventilatory and pulmonary gas exchange findings in the young reference group in the*

	At rest				
	Supine				Sitting
	Air	~15% O_2	~11% O_2	~100% O_2	Air
HR (beats/min)					
Mean	67	75	92	62	82
Range	52–84	61–89	77–105	44–80	63–98
f (cycles/min)					
Mean	16	16	15	19	18
Range	7–32	9–30	7–24	9–25	6–31
BP, mmHg (syst/diast and mean)					
Mean	123/70 89	115/68 83	113/65 83	118/71 88	106/70
Range	104–138 72–100	94–128 78–97	93–129 65–95	110–126 76–100	70–125
	56–78	60–77	52–74	60–79	35–90
Lactate, mmol/l					
Mean	1.1	—	—	—	1.3
Range	0.6–1.6	—	—	—	0.8–2.1
$\dot{V}_E$, l/min					
Mean	8.1	10.6	10.7	8.9	10.6
Range	5.8–10.9	7.1–17.5	8.4–12.5	6.6–13.4	6.8–13.6
$\dot{V}_{O_2}$, ml/min					
Mean	276	271	241	—	325
Range	239–336	216–294	196–294	—	292–380
R					
Mean	0.75	0.83	1.03	—	0.74
Range	0.60–0.83	0.77–0.91	0.91–1.20	—	0.67–0.78
Vent. equiv.					
Mean	29.9	39.0	45.0	—	34.7
Range	20.6–42.2	25.2–61.5	38.4–59.2	—	23.2–51.0
$\dot{V}_A$, l/min					
Mean	4.3	5.4	5.8	—	5.4
Range	3.8–4.9	3.8–7.1	5.0–6.7	—	4.5–6.5
V_D, ml					
Mean	204	293	305	—	265
Range	142–243	202–371	195–348	—	183–359
V_D/V_T					
Mean	0.37	0.44	0.39	—	0.40
Range	0.27–0.44	0.26–0.56	0.34–0.47	—	0.25–0.46
$P_{A_{O_2}}$, mmHg					
Mean	94.8	54.0	36.8	658.8	98.5
Range	79–106	47–62	32–43	646–675	93–105
$P_{A-a_{O_2}}$, mmHg					
Mean	4.6	−1.6	1.1	47.7	8.3
Range	−0.6–14.3	−5.3–2.8	−4.2–4.8	31–83	1.7–14.4

same experimental conditions as in Fig. 1

At work (work loads in kpm/min)						
Sitting					Supine	
300	600	900	1 200	W_{max}	600	600 + CO
111 87–123	143 124–152	177 170–186	196 191–202	199 192–208	140 131–151	147 140–154
18 10–24	21 14–25	27 21–34	35 25–46	36 25–46	21 14–26	26 14–34
141/69 125–150 65–80	150/70 130–160 60–90	171/71 155–190 60–80	180/72 160–200 60–90	182/75 160–200 65–90	148/78 103 126–172 90–116 65–86	144/77 98 120–166 84–108 64–83
1.6 1.1–2.2	2.4 1.9–3.2	4.4 3.8–5.9	7.3 3.1–9.1	7.9 6.6–9.1	3.0 1.8–4.5	— —
23.4 16.4–28.6	34.5 28.0–40.1	52.6 42.7–59.9	78.8 65.2–91.3	85.0 65.2–112.1	34.4 28.3–46.1	35.9 27.9–46.1
1 043 934–1 278	1 470 1 365–1 649	2 065 1 961–2 189	2 711 2 623–2 775	2 772 2 587–3 138	1 463 1 293–1 608	1 490 1 388–1 608
0.78 0.69–0.88	0.86 0.78–0.93	0.93 0.81–0.99	0.98 0.85–1.05	1.03 0.96–1.12	0.83 0.75–0.94	0.84 0.75–0.93
22.4 17.6–28.6	23.5 19.7–27.0	25.4 21.1–28.4	29.1 24.4–33.9	30.6 24.9–39.1	23.2 19.5–27.4	24.0 20.1–28.6
16.4 13.6–19.4	25.4 22.6–29.4	43.6 35.4–58.5	62.1 51.9–71.5	69.4 52.5–87.8	25.3 21.9–32.2	27.3 23.8–35.8
324 227–479	357 282–409	367 288–462	411 273–580	402 273–502	327 307–344	276 230–386
0.25 0.14–0.31	0.22 0.16–0.28	0.18 0.14–0.24	0.18 0.10–0.23	0.17 0.10–0.21	0.21 0.16–0.25	0.20 0.12–0.27
95.2 91–108	100.4 95–108	105.4 99–112	109.6 100–117	111.5 105–117	100.4 95–111	101.8 96–113
9.0 5.3–16.0	13.3 8.5–20.2	18.6 13.2–27.7	21.7 12.5–28.6	24.9 21.0–28.6	14.5 10.3–21.3	14.2 8.0–23.0

	At rest				
	Supine				Sitting
	Air	~15% O_2	~11% O_2	~100% O_2	Air
$P_{a_{CO_2}}$, mmHg					
Mean	41.6	39.5	36.7	38.1	38.8
Range	39–44	35–43	33–40	36–41	36–41
pH					
Mean	7.39	7.42	7.44	7.41	7.43
Range	7.37–7.42	7.40–7.45	7.42–7.47	7.40–7.45	7.38–7.43
HCO_3^-, mEq/l					
Mean	25	25	25	25	24
Range	24–26	23–27	24–26	24–26	24–25
BE, mEq/l					
Mean	±0	+1	+1	+1	+1
Range	−1–2	0–3	−1–2	0–2	0–2
Hb, g/l					
Mean	139	141	141	—	147
Range	135–143	135–144	136–144	—	136–154
Hct, %					
Mean	41.7	41.8	41.9	—	44.0
Range	40.5–43.0	40.5–43.0	41.0–43.0	—	42.5–45.0

CO = $F_{I_{CO}}$ ~ 0.0005 in air; W_{max} 1 220 (1 100–1 350) kpm/min; usually 5–6 observations, see Procedure.

Birchler (54). With the exception of the studies of Hofer & Scherrer and Scherrer & Birchler, which were performed at a P_B of about 710 mmHg in contrast to our 751 (737–767) mmHg in the young and 759 (752–763) mmHg in the middle-aged group, our values are lower, which might be due to the fact that our investigation was made in supine subjects, as those of Said & Banerjee (52) and of Karetzky et al. (37), whose values do not differ greatly from ours. The reason that our $P_{a_{O_2}}$ values are lower than those of Vale (67) is probably that we used a constant blood-gas factor and Vale individually extrapolated the blood/gas factor in regard to time. Another reason for our lower $P_{a_{O_2}}$ values at 100% O_2 breathing as compared for example, with the findings of Mellemgaard (45) is that we made no correction for the dilutional effect of the heparin-filled syringe dead space. At our $P_{a_{O_2}}$ levels during 100% O_2 breathing this correction would give a value about 15 mmHg higher. The $P_{a_{O_2}}$ values which we obtained in normal subjects can be used as reference values for investigations of patients, but the true veno-arterial shunting is less than was calculated from the $P_{a_{O_2}}$ values obtained, as it has been shown by Fletcher et al. (22) that an electrode connected directly to the arterial catheter gives, on the average, 23 mmHg higher $P_{a_{O_2}}$ values than the syringe sampling technique.

During exercise up to submaximal work loads there were no appreciable changes in $P_{a_{O_2}}$ or $S_{a_{O_2}}$ compared with conditions at rest in the sitting posture, but $P_{A-a_{O_2}}$ and the arterial O_2 content increased. The $P_{a_{O_2}}$ values at W_{max} were about the same as found by Holmgren & Linderholm (31) in young healthy subjects bicycling at about 1 350 kpm/min without breathing through a ventilatory valve, and as found at a corresponding work load by Asmussen & Nielsen (3). Also during exercise V_D and V_D/V_T were greater in the middle-aged group. There was no difference in $\dot{V}_E$, $\dot{V}_A$, $P_{a_{CO_2}}$, V_D or V_D/V_T at a work load of 600 kpm/min performed sitting or supine. However, in the older

At work (work loads in kpm/min)						
Sitting					Supine	
300	600	900	1 200	W_{max}	600	600 + CO
42.8	43.2	41.4	37.0	36.8	41.4	39.8
39–48	39–46	38–45	33–39	32–44	38–45	36–43
7.38	7.37	7.35	7.32	7.30	7.36	7.37
7.35–7.41	7.35–7.39	7.32–7.38	7.26–7.40	7.21–7.35	7.34–7.39	7.33–7.42
25	24	23	20	19	23	23
24–25	22–25	22–23	18–24	14–21	22–23	22–24
±0	−2	−3	−5	−7	−2	−3
−1–1	−3–0	−4–(−2)	−9–(−1)	−15–(−3)	—	−3–(−1)
150	153	155	158	157	146	144
144–154	146–157	148–160	150–163	150–162	139–152	140–151
44.6	44.5	45.2	45.4	45.9	42.1	42.5
43.0–46.0	43.0–45.0	44.0–46.0	44.0–47.0	44.6–47.5	40.0–43.0	42.0–42.5

group the O_2 uptake was greater in the supine posture at 600 kpm/min, performed about half an hour after a maximal work test, and R was lower in both groups under the same conditions. There were no appreciable differences in the parameters mentioned as compared with the results of other authors (25, 54, 65), but our subjects had lower $P_{a_{O_2}}$ values as compared with the values of Bjure et al. (13), which might be due to the fact that ours did not hyperventilate. The $P_{A-a_{O_2}}$ values during moderate exercise agree with those found by Holmgren (30) at $\dot{V}_{O_2}$ about 1 470 ml/min but are somewhat higher than the values of Bartels et al. (8). It is also possible that differences in the body temperature during exercise might cause discrepancies between the results of different authors. After maximal exercise we found, on the average, an increase of only 0.3°C in the oral temperature.

The mean D_{LCO} value in the young group is higher than in the middle-aged group, which is in agreement with other findings (16). Our groups are so small, however, that conclusions on significant differences between them cannot be drawn. Further, in general the young were not as near to their W_{max} at the time of the D_{LCO} measurement as most of the middle-aged subjects. This might decrease the difference in measured D_{LCO} between the groups. Comparing our values with those of other authors, ours are higher than the values of Filley et al. (21), about the same at the same work load or the same oxygen uptake as those of Linderholm (39, 40), Donevan et al. (16), Mostyn et al. (46), Freyschuss & Holmgren (23) and Bjure et al. (13), and lower than those of Ross et al. (51), Turino et al. (64), Mostyn et al (46) in regard to champion swimmers, and Holmgren & Åstrand (33). Some of the D_{LCO} values (64) cannot easily be compared with ours as the estimation was made in hypoxic conditions in order to measure $D_{L_{O_2}}$ simultaneously. D_{LCO} can also be influenced by lung changes caused by smoking, which is only sporadically reported in other studies. In older age groups it has been shown that smokers have lower D_{LCO} values than non-smokers and this was not due to differences in capillary CO "back

Table III. *Circulatory, ventilatory and pulmonary gas exchange findings in the middle-aged reference group in the*

	At rest				
	Supine				Sitting
	Air	~15% O_2	~11% O_2	~100% O_2	Air
HR (beats/min)					
Mean	62	64	72	55	67
Range	49–72	54–75	56–86	42–63	57–81
f (cycles/min)					
Mean	13	14	14	13	14
Range	11–17	10–20	9–20	9–18	11–18
BP, mmHg (syst/diast and mean)					
Mean	132/72 93	127/70 92	134/74 98	132/77 94	115/86
Range	110–147 80–110	108–145 80–105	119–155 90–110	115–151 86–112	100–130
	65–78	60–81	68–80	70–88	75–90
Lactate, mmol/l					
Mean	1.4	—	—	—	1.1
Range	1.0–1.7	—	—	—	0.9–1.4
$\dot{V}_E$, l/min					
Mean	8.0	11.9	15.6	10.2	10.2
Range	6.9–9.6	9.0–14.9	11.4–17.9	8.9–14.1	9.0–13.1
$\dot{V}_{O_2}$, ml/min					
Mean	237	258	240	—	287
Range	206–278	213–344	208–289	—	262–324
R					
Mean	0.80	0.86	0.98	—	0.78
Range	0.75–0.90	0.76–1.05	0.88–1.06	—	0.71–0.91
Vent. equiv.					
Mean	34.4	43.5	63.2	—	36.1
Range	24.6–43.2	37.0–58.2	54.8–76.1	—	27.8–49.6
$\dot{V}_A$, l/min					
Mean	4.1	5.0	6.0	—	5.3
Range	3.2–4.5	4.0–6.2	4.3–7.5	—	4.5–6.8
V_D, ml					
Mean	223	439	667	—	304
Range	188–252	335–574	370–896	—	226–477
V_D/V_T					
Mean	0.40	0.52	0.57	—	0.41
Range	0.30–0.45	0.45–0.62	0.50–0.63	—	0.35–0.47
$P_{A_{O_2}}$, mmHg					
Mean	101.6	67.2	44.2	665.6	104.2
Range	95–111	60–82	39–53	660–673	97–117
$P_{A-a_{O_2}}$, mmHg					
Mean	23.4	7.4	2.2	93.0	16.4
Range	15.3–32.5	0.9–15.0	−0.4–5.0	60–168	10.7–25.5

same experimental conditions as in Fig. 1

At work (work loads in kpm/min)						
Sitting					Supine	
300	600	900	1 200	W_{max}	600	600 + CO
93	121	154	174	168	123	131
91–95	108–128	137–165	164–184	162–184	103–135	109–145
16	20	26	28	29	23	27
14–18	16–24	20–33	24–31	24–33	20–28	22–37
148/81	182/80	206/84	213/73	216/85	179/89 119	176/86 116
130–160	160–215	180–245	195–230	195–230	142–210 99–140	133–205 93–138
70–90	70–90	60–100	60–85	60–100	77–102	70–100
1.7	3.0	5.1	6.7	7.0	3.6	—
1.3–2.2	1.6–4.9	2.6–6.9	6.6–6.7	6.6–8.0	1.9–4.5	—
23.2	37.2	57.5	68.0	71.8	39.0	43.6
20.4–26.2	31.1–44.0	45.6–73.0	63.7–72.3	63.7–91.1	33.4–47.4	36.3–54.2
971	1 495	2 086	2 569	2 429	1 554	1 656
858–1 076	1 382–1 583	1 935–2 177	2 402–2 735	2 026–2 803	1 418–1 740	1 513–1 792
0.78	0.88	0.95	1.01	1.01	0.82	0.84
0.72–0.83	0.84–0.93	0.89–1.02	0.96–1.06	0.96–1.08	0.80–0.87	0.77–0.90
24.1	25.8	27.5	26.7	29.7	25.1	26.3
19.3–27.0	20.3–28.5	21.2–33.5	23.3–30.1	23.3–32.5	21.6–27.2	21.7–30.2
15.4	26.5	41.8	54.0	53.6	28.0	34.0
14.4–17.5	22.8–31.1	34.5–52.7	51.6–56.3	38.9–68.4	24.7–33.7	27.3–48.3
426	461	524	446	547	447	411
359–510	323–625	393–631	441–451	441–662	330–590	332–473
0.30	0.25	0.24	0.18	0.23	0.26	0.25
0.25–0.33	0.17–0.32	0.18–0.31	0.17–0.19	0.17–0.31	0.21–0.32	0.20–0.26
97.4	101.4	105.8	109.0	109.6	101.8	104.4
89–103	96–106	98–112	103–114	103–114	97–105	98–111
9.0	12.9	19.1	24.8	23.9	17.8	17.8
2.1–21.1	2.2–25.1	9.0–30.0	23.7–25.9	9.0–30.9	9.6–24.5	14.7–20.1

	At rest				
	Supine				Sitting
	Air	~15 % O_2	~11 % O_2	~100 % O_2	Air
$P_{a_{CO_2}}$, mmHg					
Mean	40.0	37.8	34.4	38.6	36.8
Range	36–43	31–42	29–39	36–43	31–41
pH					
Mean	7.41	7.44	7.46	7.42	7.43
Range	7.37–7.42	7.41–7.48	7.43–7.51	7.36–7.47	7.40–7.47
HCO_3^-, mEq/l					
Mean	25	26	26	25	25
Range	24–27	24–27	24–27	23–27	24–27
BE, mEq/l					
Mean	+1	+1	+1	+1	0
Range	−1–3	−1–3	0–3	−2–3	−1–3
Hb, g/l					
Mean	134	134	135	—	142
Range	116–144	117–145	119–147	—	123–153
Hct, %					
Mean	40.0	40.5	40.1	—	42.0
Range	36.0–43.0	36.5–44.5	36.0–43.0	—	38.0–45.0

CO = $F_{I_{CO}}$ ~ 0.0005 in air; W_{max} 1 050 (900–1 200) kpm/min; usually 5 observations, see Procedure.

pressure" (48). In our material the average difference in $D_{L_{CO}}$ between the smokers and non-smokers was about the same as that between the two age groups. Turino et al. (63) and Ross et al. (51) have shown that the increase in ventilation, either voluntary hyperventilation at rest or during work or the normal increase during work, is more important in increasing $D_{L_{CO}}$, measured by the "steady state method", than the increase in cardiac output. The ventilatory factors should, therefore, be standardized and $D_{L_{CO}}$ measured at a work load which the subject is capable of continuing without hyperventilation during the investigation. For this reason $\dot{V}_E$ and $\dot{V}_{O_2}$ should also always be reported in connection with $D_{L_{CO}}$ measurements.

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At work (work loads in kpm/min)						
Sitting					Supine	
300	600	900	1 200	W_{max}	600	600 + CO
42.4	43.4	41.8	41.5	40.0	40.4	39.0
38–45	38–47	35–46	39–44	35–44	36–44	33–43
7.39	7.35	7.32	7.31	7.30	7.36	7.37
7.36–7.43	7.31–7.37	7.26–7.35	7.29–7.32	7.23–7.32	7.32–7.40	7.32–7.42
25	22	21	20	19	23	22
23–26	20–25	19–23	—	19–20	21–25	20–24
0	−3	−5	−6	−7	−2	−3
−2–3	−5–0	−8–(−2)	—	−8–(−6)	−5–1	−6–(−1)
144	147	151	147	153	142	—
125–157	128–162	128–164	135–158	135–164	122–154	—
42.9	44.0	44.9	44.5	45.6	42.3	—
39.0–46.5	39.5–48.0	41.0–49.0	42.0–47.0	42.0–49.0	37.0–46.5	—

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