Eur Respir J 1998; 11: 1369–1375
DOI: 10.1183/09031936.98.11061369

European Respiratory Journal
ISSN 0903 - 1936

Relationship between external resistances, lung function changes and maximal exercise capacity

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ABSTRACT: In upper airway obstruction (UAO) the relationship between the degree of obstruction, exercise limitation and lung function indices is not well established.

Therefore, we investigated in nine healthy subjects (age 36±9 yrs) the effects of two added resistances at the mouth (R_1 = added resistance with 7.8 mm diameter; R_2 = 5.7 mm) on forced expiratory volume in one second (FEV_1), peak expiratory flow (PEF), airway resistance (R_{aw}) and maximal breathing capacity (measured during 15 s = measured maximum breathing capacity (MBC_m); calculated as $FEV_1 \times 37.5$ = calculated maximum breathing capacity (MBC_c)) on the one hand, and maximum exercise capacity (W'_{max}), minute ventilation (V'_E) and CO_2 elimination (V'_{CO_2}) on the other.

We found that R_1 had almost no influence on FEV_1 but decreased PEF by ~35% and increased R_{aw} by almost 300%; it decreased W'_{max} by merely ~10% while maximal exercise ventilation (V'_{Emax}) was only 65% of control and only reached ~40% MBC_c and ~70% MBC_m; yet V'_E and V'_{CO_2} were significantly reduced at high exercise levels indicating hypoventilation. With R_2, FEV_1 was reduced by 25% and PEF by 55%, and R_{aw} was increased by 600%; W'_{max} was ~60% of control, V'_{Emax} was only 35% of control and reached ~30% MBC_c and ~60% MBC_m, V'_E was already reduced at moderate exercise levels.

We conclude that : 1) an upper airway obstruction of 6 mm diameter (but not of 8 mm) had a marked influence on maximum exercise capacity due to hypoventilation; 2) calculated maximum breathing capacity markedly overestimated measured maximum breathing capacity because the forced expiratory volume in one second is an insensitive index of upper airway obstruction and because it does not take inspiratory flow limitation into account; and 3) a 10% decrease in maximum exercise capacity was linearly related with a 7% decrease in the forced expiratory volume in one second and a 150% increase in airway resistance. A 10% decrease in maximal exercise ventilation was related to a 8.5% decrease in peak expiratory flow and 9% decrease in measured maximum breathing capacity.
Eur Respir J 1998; 11: 1369–1375.

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Keywords: Added resistance
lung function test
maximal breathing capacity
maximal exercise
upper airway obstruction

Received: January 5 1997
Accepted after revision February 27 1998

This study is supported by a grant of the NFWO Levenslijn project No. 7.0047.94

The relationship between the abnormalities in the different routine lung function tests and the degree of functional limitation during exercise is not well established in upper airway obstruction (UAO). On the contrary, in chronic obstructive pulmonary disease (COPD), for instance, a forced expiratory volume in one second (FEV_1) of 60% predicted is generally associated with a peak expiratory flow (PEF) of 60–70% pred and corresponds with a moderate physical impairment of ~20–30% [1, 2]. Yet, in UAO, FEV_1 will still be 70–80% pred when PEF is only 40–50% pred and it is not established what the clinical relevance is of each of these changes in terms of exercise limitation, although it is generally accepted that FEV_1 is an insensitive test for UAO [3–5].

In several respiratory disorders the maximal breathing capacity (MBC) is considered a useful index of the degree of ventilatory impairment because the exercise limitation shows a good relationship with the reduction in MBC [6, 7]. In instances of ventilatory limitation the maximal exercise ventilation (V'_{Emax}) will often be more than 70% MBC, which is the normal ratio in healthy individuals. In addition, it has been claimed that MBC is a sensitive index of the ventilatory limitation in UAO [3, 8–13]. It seems, however, very probable that the calculated MBC (MBC_c), derived from FEV_1 (*e.g.* $FEV_1 \times 37.5$) will overestimate the actual ventilatory reserve in UAO, because of the already mentioned underestimation of the obstruction by FEV_1. The relationship between FEV_1 or MBC_c and exercise capacity will, in addition, vary depending on the type of obstruction (*i.e.* fixed, variable extrathoracic or variable intrathoracic) because this will markedly influence the ratio of maximal expiratory over inspiratory flows [8]. Therefore, the measured MBC (MBC_m) and not MBC_c appears to be the appropriate MBC index in UAO, because it also reflects inspiratory besides expiratory flow rates, in contrast to MBC_c [14–17]. The relationship between MBC_m, MBC_c, V'_{Emax} and exercise capacity is, however, not well established in UAO. Yet LINDSTEDT *et al.* [18] have shown that added resistances with an orifice diameter of less than ~10 mm cause a decrease in maximal O_2 uptake (V'_{O_2max})

which is proportional to that in PEF and peak inspiratory flow (PIF). In addition, the relationship between the degree of exercise limitation and the anatomic obstruction in UAO is not well known. The reliability of radiology in estimating the diameter or cross-sectional area of UAO has been questioned because it is not well correlated with functional changes and because it is not able to visualize all UAOs [19].

The purpose of the present study was, therefore, to measure in UAO : 1) the degree of lung function abnormalities and exercise limitation due to added resistances (as a model for UAO); and 2) to relate the lung function abnormalities, MBC_m, MBC_c, V'_{Emax} and exercise capacity to each other in order to establish the clinical relevance of each of these changes.

Subjects and method

Subjects

Nine healthy, nonsmoking, male volunteers (age 36±9 yrs, height 176±4 cm, weight 75±9 kg) without history or clinical signs of COPD, asthma or other lung disorders were investigated. All gave informed consent prior to the investigations and the study was approved by the Ethics Committee.

Methods

Vital capacity (VC), FEV_1, forced inspiratory volume in one second (FIV_1), maximal expiratory and inspiratory flow-volume-curves (MEFV- and MIFV-curves) were recorded at the mouth with a Lilly-type pneumotachograph and integrator (Medical Graphics, St. Paul, MN, USA). Airway resistance (R_{aw}) was measured at functional residual capacity (FRC) in a constant-volume plethysmograph (Medical Graphics) as the chord slope between inspiratory and expiratory flow at 0.5 $L \cdot s^{-1}$; specific airway res-istance ($_sG_{aw}$) was calculated as $1/R_{aw} \times FRC$. All lung function values were also expressed in % control or % predicted of the reference values of the European Community for Steel and Coal (ECSC) [20]. MBC was cal-culated as $MBC_c = 37.5 \times FEV_1$ [21, 22] and was also measured as MBC_m by performing a MBC-manoeuvre during 15 s. Pulmonary function testing was carried out according to the ECSC- and American Thoracic Society (ATS)-recommendations [20, 23] in control conditions and with two added resistances (R_1 = 7.8 mm cross-sectional diameter, and R_2 = 5.7 mm). The resistances were made of 3-cm-long cylinders of metal and hard poly vinyl chloride (PVC) inserted in each other [24]. They were placed be-tween the mouth and the pneumotachograph and had a dead space of <2 mL.

Exercise testing was done on a cycle-ergometer (Part'nair 5400, version 5.0, Medisoft, Belgium and Ergometrics 900 computer, Ergoline, Germany) by 2-min increments of 30 Watt (pedalling at 60 $cycles \cdot min^{-1}$), until exhaustion or a symptom-limited maximum occurred. All exercise tests were performed under 12-lead-electrocardiographic monitoring from which heart rate (HR) was obtained. The subject breathed through a mouthpiece in a three-way valve type, adapted Otis-McKerrow (dead space 100 mL). Ex-pired gas passed through a mixing chamber at the outlet at which V'_E was obtained from a Lilly type pneumotachograph with integrator (Medisoft); and mixed expired O_2 and CO_2 levels with a paramagnetic oxygen analyser and an infra-red carbon dioxide analyser (Medisoft). From these, oxygen uptake (V'_{O_2}), CO_2 elimination (V'_{CO_2}), respiratory quotient ($RQ = V'_{CO_2}/V'_{O_2}$), V'_{O_2}/HR, V'_E/V'_{O_2} and V'_E/V'_{CO_2} were calculated. Before each exercise test, volume recordings were calibrated with a 3 L syringe along with gas analyses for precise gas mixtures. Arterial or transcutaneous blood gases were not measured. Tests were done on 3 separate days: first the control exercise (R_0) was performed, and thereafter the exercises with R_1 and R_2 in random order. The resistances (<2 mL dead space) were placed between the mouth and the three-way valve. Results were expressed in absolute values and in per cent of control [25].

Statistical analysis

All data are presented as means±1 standard deviation (SD). Analysis of variance was applied and when this was statistically significant a Duncan test was added to determine between which resistances differences were significant. The level of significance was set at $p<0.05$.

Results

Table 1 shows the lung function values for the nine subjects in control condition (R_0) and with the two added resistances (R_1 and R_2) in absolute values and in % control. While FEV_1 hardly decreases, not even with R_2, mark-ed changes occur in FIV_1, PEF, PIF, R_{aw} and $_sG_{aw}$, even with R_1. The classical ratios for UAO are clearly fulfilled with both resistances: PEF/maximal expiratory flow at 50% VC (MEF_{50}) (% pred/% pred) becomes markedly <2.1, FEV_1/PEF ($mL \cdot L^{-1} \cdot min$) becomes >8–10, and MEF_{50}/airway conductance (G_{aw}) (kPa) is >0.7 [5, 26–29]. MEF_{50}/ MIF_{50} is clearly >1 which is in agreement with a previous analysis by us in fixed UAO [24]. In addition, the MBC_m is markedly reduced with both resistances and becomes only ~50% of the MBC_c. The rather large SD for PEF, R_{aw} and $_sG_{aw}$ with R_2 are most likely due to the alinear pressure-flow characteristics of R_2 (see characteristics in [24]).

Table 2 shows the results of the maximal cycloergometric test. Standard deviations for the different values with R_1 and R_2 are small, indicating that the different subjects adapted similarly to these resistances. All values are normal in control condition (*e.g.* W'_{max} = 110±28% pred) [25]. With R_1, there is already a tendency for decreases in W'_{max}, V'_{O_2max} and maximal heart rate (HR_{max}), but these are not significant; yet the decreases in maximal V'_{CO_2} and especially in V'_{Emax} are more pronounced, and are significant. With R_2, all values are markedly and significantly reduced: HR_{max} is clearly not maximal and RQ is below 1, indicating that no cardioperipheral limit is reached and that the decreased exercise capacity has to be attributed to a ventilatory limitation, despite the fact that V'_{Emax} is only 62±17% MBC_m and only 28±6% MBC_c.

Table 1. – Lung function data in control situation (R_0) and with two added resistances (R_1 and R_2)

		R_0 Control	R_1 (Ø 7.8 mm)	R_2 (Ø 5.7 mm)	Duncan
FEV_1	L	4.6±0.6	4.5±0.6	3.4±0.8	$R_0R_1R_2$
	% control	100	98±10	74±16	R_0R_1-R_2
FIV	L	5.0±1.0	3.6±1.2	2.3±1.4	R_0-R_1-R_2-R_0
	% control	100	72±23	46±26	R_0-R_1-R_2-R_0
PEF	$L{\cdot}s^{-1}$	11.0±0.8	7.2±0.8	5.0±2.8	R_0-R_1-R_2-R_0
	% control	100	66±10	45±26	R_0-R_1-R_2-R_0
PIF	$L{\cdot}s^{-1}$	7.9±2.1	4.3±1.0	3.2±1.6	R_0-R_1R_2
	% control	100	54±13	41±20	R_0-R_1R_2
R_{aw}	$kPa{\cdot}L^{-1}{\cdot}s^{-1}$	0.13±0.04	0.35±0.07	0.78±0.36	R_0-R_1-R_2-R_0
	% control	100	275±50	605±280	R_0-R_1-R_2-R_0
$_sG_{aw}$	$kPa^{-1}{\cdot}s^{-1}$	2.16±0.65	0.69±0.13	0.54±0.75	R_0-R_1R_2
	% control	100	31±7	25±35	R_0-R_1R_2
MBC_m	$L{\cdot}min^{-1}$	153±23	94±25	63±26	R_0-R_1-R_2-R_0
	% control	100	61±13	41±17	R_0-R_1-R_2-R_0
MBC_m/FEV_1	$L{\cdot}min^{-1}$	34.1±6.5	21.3±7.1	18.2±6.5	R_0-R_1R_2
MBC_m/MBC_c	×100	91±17	57±19	48±17	R_0-R_1R_2
PEF/MEF_{50}	% pred/% pred	2.1±0.4	1.4±0.3	1.4±0.5	R_0-R_1R_2
FEV_1/PEF	$mL{\cdot}L^{-1}{\cdot}min$	6.9±0.9	10.5±1.8	12.9±3.3	R_0-R_1-R_2-R_0
MEF_{50}/G_{aw}	kPa	0.6±0.2	1.9±0.6	2.4±0.9	R_0-R_1-R_2-R_0
MEF_{50}/MIF_{50}	$L{\cdot}s^{-1}/L{\cdot}s^{-1}$	0.8±0.3	1.4±0.5	1.4±0.3	R_0-R_1R_2

Data are mean±SD; FEV_1/FIV_1: forced expiratory/inspiratory volume in one second; PEF/PIF: peak expiratory/inspiratory flow; MEF_{50}/MIF_{50}: maximal expiratory/inspiratory flow at 50% vital capacity; R_{aw}: airway resistance; $_sG_{aw}$: specific airway conductance; G_{aw}: airway conductance; MBC_m/MBC_c: measured/calculated maximal breathing capacity; groups separated by a dash in Duncan test differ significantly ($p<0.05$) *e.g.* R_0-R_1R_2 means R_0 is different from R_1 and R_2, but that R_1 and R_2 are not different from each other.

Table 2. – Maximal breathing capacity and cardioperipheral and respiratory indices during maximal exercise in control situation R_0 and with two added resistances (R_1 and R_2)

		R_0 (control)	R_1 (Ø 7.8 mm)	R_2 (Ø 5.7 mm)	Duncan
Exercise duration	min	16.3±3.3	15.1±3.3	10.4±1.8	R_0R_1-R_2
W'_{max}	watt	248±47	223±44	153±26	R_0R_1-R_2
	% control	100	91±18	62±11	R_0R_1-R_2
V'_{O_2max}	$L{\cdot}min^{-1}$	3.5±0.8	3.1±0.8	1.9±0.4	R_0R_1-R_2
	% control	100	88±23	54±11	R_0R_1-R_2
V'_{CO_2max}	$L{\cdot}min^{-1}$	4.3±0.9	3.5±1.0	1.8±0.5	R_0-R_1-R_2-R_0
	% control	100	81±23	42±12	R_0-R_1-R_2-R_0
HR	min^{-1}	179±12	170±15	143±23	R_0R_1-R_2
	% control	100	94±9	76±12	R_0R_1-R_2
RQ		1.3±0.1	1.1±0.1	0.9±0.1	R_0-R_1-R_2
V'_{Emax}	$L{\cdot}min^{-1}$	102±23	66±18	34±7	R_0-R_1-R_2-R_0
	% control	100	66±15	34±6	R_0-R_1-R_2-R_0
f_{Rmax}	min^{-1}	33±8	25±8	18±5	R_0-R_1-R_2-R_0
V'_{Emax}/MBC_c	×100	60±12	41±15	28±6	R_0-R_1-R_2-R_0
V'_{Emax}/MBC_m	×100	66±12	70±26	62±17	$R_0R_1R_2$

Data are mean±SD; W'_{max}: maximum exercise capacity; V'_{O_2max}: maximal oxygen uptake; V'_{CO_2max}: maximal carbon dioxide production; HR: heart rate; RQ: respiratory quotient; V'_{Emax}: maximal exercise ventilation; f_{Rmax}: maximal respiratory frequency; MBC_c/MBC_m: measured/calculated maximal breathing capacity; R_1 and R_2: added resistances; groups separated by a dash in Duncan test differ significantly ($p<0.05$).

Figures 1–4 show the changes in V'_E, HR, V'_{O_2} and V'_E/V'_{CO_2} at the different levels of exercise. Figure 1 demonstrates that V'_E is lower with R_1 than with R_0 and that the difference is significant at exercise levels of Š140 W. With R_2 V'_E is significantly lower than with R_0 and R_1 already at exercise levels of Š80 W; this decrease is mainly due to a decrease in breathing frequency (not shown). In addition, the increase in V'_E with increasing exercise levels tends to level off with R_2 at 170 W (suggesting a ventilatory limitation) while it increases curvilinearly at this level with R_0 and R_1. Similarly V'_{CO_2} is significantly lower with R_2 than with R_0 and R_1 from 80 Watt on (not shown) and V'_{CO_2} levels off with R_2 at 170 W (indicating hypoven-tilation) while it increases curvilinearly at this level with R_0 and R_1 (not shown). Figure 2 shows that the V'_E/V'_{CO_2} ratio also changes between R_0 and R_1 at high exercise levels of Š200 W. Figure 3 indicates that V'_{O_2} is very similar with R_0, R_1 and R_2 at the different exercise levels, except for the highest exercise level attained with R_2. The V'_{CO_2}/V'_{O_2} ratio (*i.e.* RQ) is significantly lower with R_2 at several moderately high exercise levels: at 170 W, the highest level with R_2, it is only 0.95±0.09, while

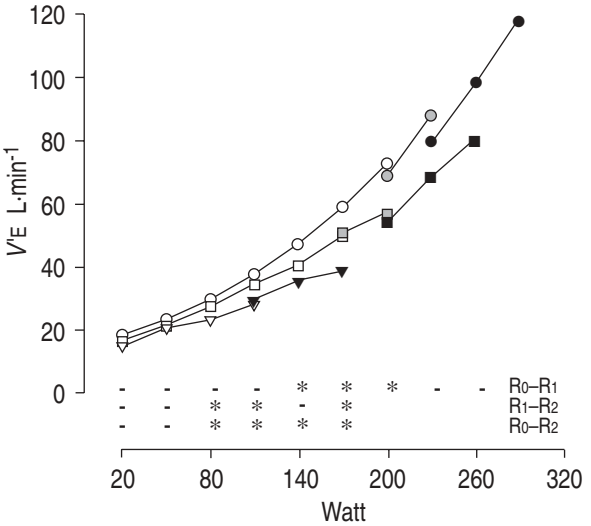

Fig. 1. – Relationship between minute ventilation (V'_E) and exercise level (Watt) in control condition (R_0, circles), with an added resistance of 7.8 mm diameter (R_1, squares) and of 5.7 mm (R_2, triangles). Shading of symbols indicate number of subjects for each condition as follows: ○=9; ○=6; ●=3; ❑=9; □=8; ■=3; ◊=9; and ▼=5. Significance ($p<0.005$) of differences R_0, R_1 and R_2 are shown by the asterisks.

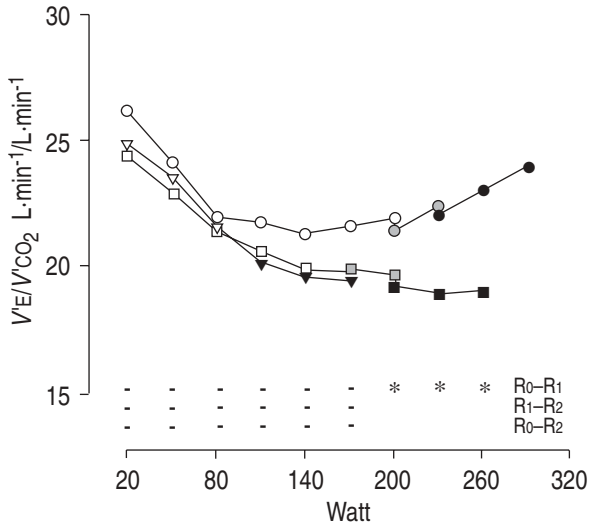

Fig. 2. – Relationship between (exercise ventilation (V'_E)/carbon dioxide production (V'_{CO_2})) and exercise level in control condition (R_0) and with added resistances R_1 and R_2. For explanation see legend to figure 1.

it is already 1.03±0.07 with R_1, and 1.09±0.08 with R_0 (not significant). Figure 4 shows that HR at each exercise level is equal with the different resistances; as expected, it is low-er for the best trained subjects who achieve the highest exercise levels. Also the V'_{O_2}/HR ratio is not significantly different between R_0, R_1 and R_2 at each exercise level but the maximal V'_{O_2}/HR ratio decreases from 19.7±4.0 L with R_0, to 18.8±5.4 L with R_1 and to 14.0±3.5 L with R_2 ($p<0.05$ for R_2 *versus* R_0 and R_1).

In figure 5, the relationship of different lung function indices (FEV_1, FIV_1, PEF, PIF, R_{aw}, sG_{aw} and MBC) with V'_{Emax} (left panels) and with W'_{max} (right panels) can be approximated. FIV_1, in particular, but also PEF and MBC_m show an almost linear relationship with V'_{Emax} and R_{aw},

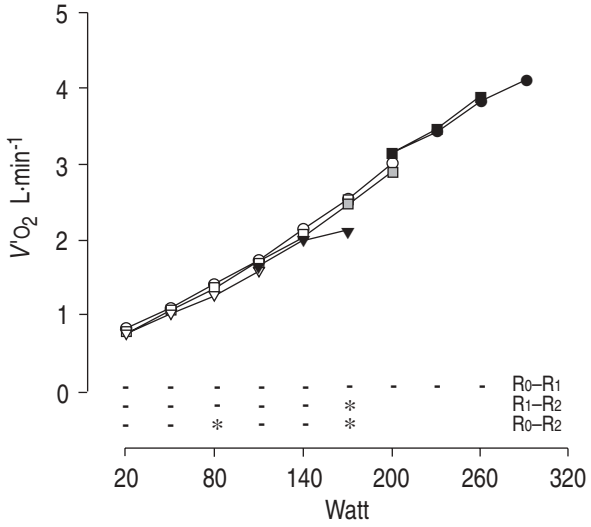

Fig. 3. – Relationship between oxygen uptake (V'_{O_2}) and exercise level in control condition (R_0) and with added resistances R_1 and R_2. For explanation see legend to figure 1.

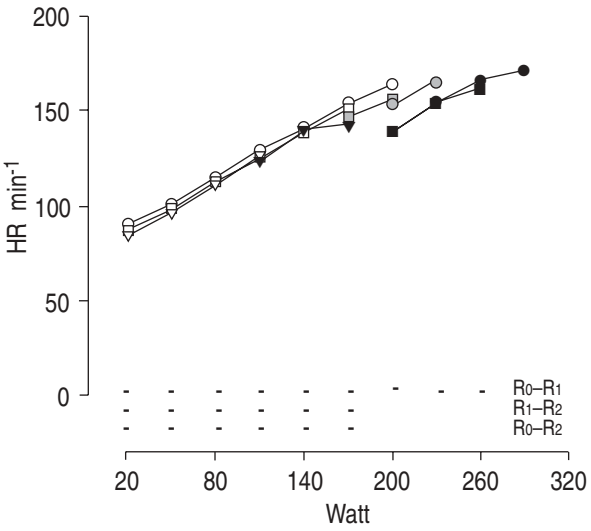

Fig. 4. – Relationship between heart rate (HR) and exercise level in control condition (R_0) and with added resistances R_1 and R_2. For explanation see legend to figure 1.

but also FEV_1 or MBC_c (which in % control is identical as FEV_1) show an almost linear relationship with W'_{max}.

Discussion

The influence of two added resistances (R_1 = 7.8 mm and R_2 = 5.7 mm) on lung function and on maximal exercise capacity were studied in nine male healthy subjects, as a model for tracheal stenosis. FEV_1 was clearly less disturbed than FIV_1, PEF, PIF, R_{aw} or sG_{aw} with both resistances which has already been demonstrated [3, 5, 26]. W'_{max} was only clearly reduced with R_2 and was rather linearly related to R_{aw}, FEV_1 (or MBC_c). Together, a decrease of W'_{max} to 60% control corresponds in UAO with a FEV_1 of 75% control, a PEF of 40% and R_{aw} of 600%. R_2 (*i.e.* a diameter of <6 mm) clearly induced a ventilatory limitation with an impaired CO_2 elimination and a RQ <1 due to hypoventilation similarly as in other studies [26]. However, V'_{Emax} was only ~60% MBC_m and only

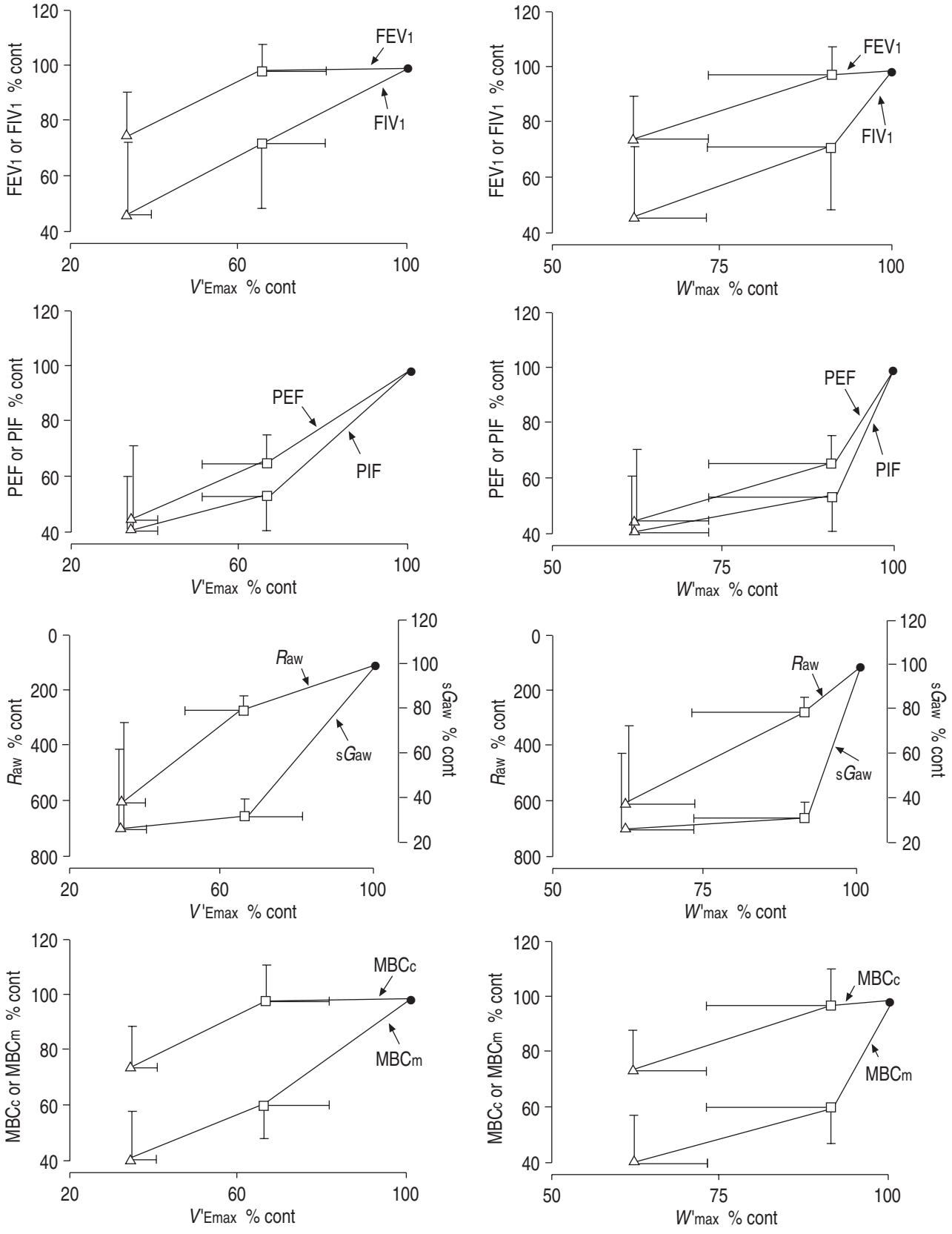

Fig. 5. – Relationship of several lung function indices with maximum exercise ventilation (V'_{Emax}) (left panels) and with maximum exercise capacity (W'_{max}) (right panels) in control condition (●) and with R_1 (❑) and R_2 (ý). All data are expressed as per cent of the control value (% cont). FEV_1 or FIV_1: forced expiratory/inspiratory volume in one second; PEF or PIF: peak expiratory/inspiratory flow; R_{aw}: airway resistance; $_sG_{aw}$: specific airway conductance; MBC_m or MBC_c: measured/calculated maximal breathing capacity.

even ~30% MBCc indicating that MBCc cannot be used as a criterion for ventilatory limitation in UAO. It should be noted that the hypoventilation and reduced CO_2 elimination with R2 were already significant at 80 W, indicating that a UAO of 6 mm impairs lung gas homeostasis during moderate exercise. Furthermore, R1 (diameter of <8 mm) had only minor effects on $\dot{W}'_{max}$ (~90% control), but re-duced $\dot{V}'_E$ and $\dot{V}'_E/\dot{V}'_{CO_2}$ at the higher exercise levels, suggesting an impairment in lung gas homeostasis during more strenuous exercise. This confirms the data of the literature which point towards the occurrence of hypoxia and hypercapnia during strenuous exercise in UAO [8, 13, 27–29].

It is well known that the relative changes in different lung function tests in UAO are quite different from those in COPD or in lung fibrosis and therefore, several ratios may have a diagnostic value for UAO *e.g.* PEF/MEF50, FEV1/PEF and FEV1/*R*aw [5, 30–34]. It is, however, much less clear which lung function test provides the most relevant information in UAO to estimate the severity of the obstruction and the degree of exercise limitation in this condition. In agreement with data of the literature we confirmed that in particular, s*G*aw and $\dot{V}'_{Emax}$ were most affected (see tables 1 and 2) and thus probably were the most sensitive indices in this type of fixed UAO, followed by MBCm, PEF, PIF and FIV1 while FEV1 was an insensitive index which tends to underestimate the degree of UAO [8, 13, 18, 26]. MBC has been considered to be a sensitive index of UAO [3, 8–13], and it could also be hypothesized that MBC may be a good indicator of exercise limitation in UAO, because UAO will cause a ventilatory type of exercise limitation. However, the classical relationship between FEV1, $\dot{V}'_{Emax}$ and MBC (MBC ~37.5 × FEV1; $\dot{V}'_{Emax}$ Š70% MBC) does not hold in UAO [3, 10, 11, 15]. MBC is often applied in COPD or lung fibrosis to estimate whether a reduced exercise capacity is due to a ventilatory limitation (*i.e.* $\dot{V}'_{Emax}$ approaches MBCc). It is obvious that this calculation of MBC cannot be applied to UAO since, even in a ventilatory limitation, $\dot{V}'_{Emax}$ will not approach MBCc: in our study $\dot{V}'_{Emax}$ is only ~30% MBCc with R2. In addition, even MBCm is only ~50% MBCc with R2. The reason is, of course, that MBCc, similarly to FEV1, only reflects expiratory limitation, and while in COPD the limitation is, indeed, only expiratory, this is not the case in UAO. The actual ratio between MBCm and FEV1 in our healthy subjects was, as expected, ~35 in control condition, but it decreased to ~20 with R1 and R2 (table 1). More important is that despite the clear ventilatory limitation during maximal exercise with R2, $\dot{V}'_{Emax}$ did not approach MBCm but remained at 60–70% of it, similar to the control situation in healthy subjects [35, 36]. This confirms the data of Demedts and Anthonisen [13] that $\dot{V}'_{Emax}$ is ~70% of the 15 s MBC independent of the level of external resistance. The reason why $\dot{V}'_{Emax}$ does not come closer to MBCm in UAO is obvious: if maximum voluntary ventilation (MVV) is sustained for several minutes it will progressively decrease due to respiratory muscle fatigue. It has, indeed, been shown even in healthy subjects that after 4 min MVV will only be ~70% MBC (with interindividual ranges from 40–90%) and this has been attributed to respiratory muscle fatigue with time [36–38]. It is less likely that the rather low $\dot{V}'_{Emax}$/MBCm is due to differences in breathing pattern between the MBCm and maximal exercise. Indeed, in contrast with COPD, where there is a tendency for faster breathing at a high lung level during the MBCm, this is not the case in UAO due to the plateau-shaped maximal flow-volume curve. In-deed, table 2 shows that with increasing resistance, breathing frequency decreases markedly at $\dot{V}'_{max}$, and it should also be noted that at the different submaximal exercise levels, breathing frequency was lower with the higher resistances.

Another aim of the study was to investigate which lung function test was best related to $\dot{V}'_{Emax}$ and to $\dot{W}'_{max}$, and thus allow the most accurate prediction of exercise limitation in UAO. From the three points on figure 2 it can be estimated that especially FIV1 and also PEF and MBCm are relatively linearly related with $\dot{V}'_{Emax}$, it can also be roughly estimated that (each) 10% decrease in $\dot{V}'_{Emax}$ was related to a 8.5% decrease in PEF or FIV1 and a 9% decrease in MBCm. However, $\dot{V}'_{Emax}$ is not linearly related with $\dot{W}'_{max}$. It should be noted that besides *R*aw, FEV1, (or MBCc) but not MBCm presents a linear relationship with $\dot{W}'_{max}$: a rough estimation shows that (each) 10% decrease in $\dot{W}'_{max}$ was related to a 7% decrease in FEV1 (or MBCc) and a 150% increase in *R*aw. It is not clear to what extent these relationships, which we deducted from added resistances (as a model for UAO), can be applied to real UAO. In real UAO it is difficult to obtain reliable *in vivo* estimations of the degree of anatomic stenosis. Indeed, there is not a good correlation between the degree of a tracheal stenosis on radiograph and computed tomography (CT)-scan [19] or magnetic resonance imaging (unpublished observation). There is also no good correlation between the radiological estimates of the stenosis and the lung function abnormalities [19]. Of course, added resistances enable one to relate accurately the degree of the obstruction (*i.e.* diameter of resistance) to functional abnormalities [3, 35]; graphs have also been constructed relating several lung function tests (*e.g.* FEV1, PEF, *etc*.) to the diameter of this added obstruction [3]. Plotting lung function data of patients with real UAO on these graphs shows good agreement with the effects of added resistances [39, 40]. This suggests that in real UAO the effective degree of obstruction can be derived from the lung function co-ordinates on these graphs. However, more elaborate studies are warranted to confirm these preliminary data.

A final important consideration is that the relationship between the lung function abnormalities at rest and the exercise limitation appears to be markedly different between UAO and COPD, and this is probably largely attributable to the fact that airflow limitation is only expiratory in COPD but inspiratory as well as expiratory in UAO. An UAO of ~8 mm allows a $\dot{W}'_{max}$ of 90% control, while FEV1 is still ~100% control, but PEF only ~65% and *R*aw ~250%. In COPD with a similar $\dot{W}'_{max}$ of 90% pred, FEV1 is clearly reduced to ~70% pred, with a PEF of 75% pred and a *R*aw of only ~150% [2]. Furthermore, an UAO of ~6 mm, causes a reduction in $\dot{W}'_{max}$ to about 60% control, while FEV1 is still 75%, but PEF is reduced to 40% and *R*aw increased to 600%. In COPD with a similar $\dot{W}'_{max}$ of 60% pred, FEV1 is markedly reduced to 55% pred with a PEF of 60% and *R*aw of only 200% [2]. Thus, as far as $\dot{W}'_{max}$ is concerned, decreases in PEF to 60–65% and increases in *R*aw to 200–250% are almost irrelevant in UAO but mod-erately detrimental in COPD, and *vice versa* for a reduction in FEV1 to 70–75%. Published tables with esti-

mates of physical impairment based on lung function abnormalities at rest in COPD, pneumoconiosis and possibly interstitial lung disease [1, 2] are therefore not applicable to UAO [40].

In conclusion, this study provides information on the relationship between the degree of airway obstruction, lung function abnormalities and the corresponding exercise capacity in artificial upper airway obstruction. To what extent this model of added resistances can also be applied to clinical upper airway obstruction requires further investigation.

References

1. American Thoracic Society. Evaluation of impairment/disability secondary to respiratory disorders. *Am Rev Respir Dis* 1986; 133: 1205–1209.
2. Werkgroep van de Belgische Vereniging voor Pneumologie. Voorstel tot herziening van de "Officiële Belgische Schaal ter bepaling van de graad van invaliditeit". Deel IV: Ademhalingsstelsel 1990.
3. Miller RD, Hyatt RE. Obstructing lesions of the larynx and trachea: clinical and physiological characteristics. *Mayo Clin Proc* 1969; 44: 145–161.
4. Miller RD, Hyatt RE. Evaluation of obstructing lesions of the trachea and larynx by flow-volume loops. *Am Rev Respir Dis* 1973; 108: 475–481.
5. Melissant CF, van Noord JA, van de Woestijne KP, Demedts M. Comparison of dynamic lung function indices during forced and quiet breathing in upper airway obstruction, asthma and emphysema. *Chest* 1990; 98: 77–83.
6. Gaensler EA, Wright GW. Evaluation of respiratory impairment. *Arch Environ Health* 1966; 2: 146–189.
7. Zamel N, Altose MD, Speiz WA. Statement on spirometry: a report of the section on respiratory pathophysiology. *Chest* 1983; 83: 547–550.
8. Al-Bazzaz F, Grillo H, Kazemi H. Response to exercise in upper airway obstruction. *Am Rev Respir Dis* 1975; 111: 631–640.
9. Sackner MA. Physiological features of upper airway obstruction. *Chest* 1972; 62: 414–417.
10. Shim C, Corro P, Park SS, Williams MH. Pulmonary function studies in patients with upper airway obstruction. *Am Rev Respir Dis* 1972; 106: 233–238.
11. Owens GR, Murphy DMF. Spirometric diagnosis of upper airway obstruction. *Arch Intern Med* 1983; 143: 1331–1334.
12. Wittenborg MH, Gyepes MT, Crocker D. Tracheal dynamics in infants with respiratory distress, stridor and collapsing trachea. *Radiology* 1967; 88: 653–662.
13. Demedts M, Anthonisen NR. Effects of increased external airway resistance during steady-state exercise. *J Appl Physiol* 1973; 35: 361–366.
14. Spivey CG, Walsh RE, Perez-Guerra F, Harleroad LE. Central airway obstruction: report of seven cases. *JAMA* 1973; 226: 1186–1189.
15. Grimby G, Söderholm B. Spirometric studies in normal subjects: static lung volumes and maximum voluntary ventilation in adults with a note on physical fitness. *Acta Med Scand* 1963; 173: 199–206.
16. Engström H, Grimby G, Söderholm B. Dynamic spirometry in patients with tracheal stenosis. *Acta Med Scand* 1964; 176: 329–334.
17. Dillard TA, Hnatiuk OW, McCumber TR. Maximum voluntary ventilation: spirometric determinants in chronic obstructive pulmonary disease patients and normal subjects. *Am Rev Respir Dis* 1993; 147: 870–875.
18. Lindstedt SL, Thomas RG, Leith DE. Does peak inspiratory flow contribute to setting V'_{O_2max}? *Respir Physiol* 1994; 95: 109–118.
19. Melissant CF, Smith SJ, Perlberger R, *et al.* Lung function, CT-scan and X-ray in upper airway obstruction due to thyroid goiter. *Eur Respir J* 1994; 7: 1782–1787.
20. Quanjer PH (ed). Standardized lung function testing: Report of working party on "Standardization of lung function tests". *Bull Eur Physiopathol Respir* 1983; Suppl. 19; 1–95.
21. Cara M. Synergies et asynergies ventilatoires: la dynamique ventilatoire. *Poumon* 1953; 9: 406–411.
22. Gandevia B, Hugh-Jones P. Terminology for measurements of ventilatory capacity. *Thorax* 1957; 12: 290–293.
23. American Thoracic Society. Standardization of spirometry 1987-update. *Am Rev Respir Dis* 1987; 136: 1285–1298.
24. Melissant CF, Lammers J-WJ, Demedts M. Rigid external resistances cause effort dependent maximal expiratory and inspiratory flows. *Am J Respir Crit Care Med* 1995; 152: 1709–1712.
25. Jones LJ, Makrides L, Hitchcock C, Chyphar T, McCartney N. Normal standards for an incremental progressive cycle ergometer test. *Am Rev Respir Dis* 1985; 131: 700–708.
26. Wassermann K, Gitt A, Weyde J, Eckel HE. Lung function changes and exercise-induced ventilatory responses to external resistive loads in normal subjects. *Respiration* 1995; 62: 177–184.
27. Ceretelli P, Sikand RS, Farhi LE. Effect of increased airway resistance on ventilation and gas exchange during exercise. *J Appl Physiol* 1969; 27; 597–600.
28. Al-Bazzaz F, Kazemi H. Mechanisms of exercise hypoxemia and dyspnea in tracheal stenosis. *Am Rev Respir Dis* 1975; 111: 1002–1003.
29. Gee JBL, Burton G, Vassallo C, Gregg J. Effects of external airway obstruction on work capacity and pulmonary gas exchange. *Am Rev Respir Dis* 1968; 98: 1003–1012.
30. Yernault JC, Englert M, Sergysels R, De Coster A. Upper airway stenosis: a physiological study. *Am Rev Respir Dis* 1973; 108: 996–1000.
31. Rotman HH, Liss HP, Weg JG. Diagnosis of upper airway obstruction by pulmonary function testing. *Chest* 1975; 68; 796–799.
32. Empey DW. Assessment of upper airways obstruction. *Br Med J* 1972; 3: 503–505.
33. Miller MR, Pincock AC, Oates GD, Wilkinson R, Skene-Smith H. Upper airway obstruction due to thyroid goiter: detection, prevalence and results of surgical management. *Q J Med* 1990; 274: 177–188.
34. Buyse B, Demedts M. Diagnostic value of Raw/FEV_1-ratio in different diseases. *Eur Respir J* 1995; 8: Suppl. 19, 475s.
35. Wassermann K, Hansen JE, Sue DY, *et al*. Principles of exercise testing and interpretation. 1st edn. Philadelphia, Lee & Febiger, 1987.
36. Weisman IM, Zeballos RJ, eds. Clinical exercise testing. *Clin Chest Med* 1994; 15: 173–451.
37. Freedman S. Sustained maximum voluntary ventilation. *Respir Physiol* 1970; 8: 230–244.
38. Babb TG, Rodarte JR. Exercise capacity and breathing mechanics in patients with airflow limitation. *Med Sci Sports Exerc* 1992; 24: 967–974.
39. Melissant C, Demedts M. Upper airway obstruction: anatomic-functional relationship. *Chest* 1991; 100: 290–291.
40. Demedts M, Melissant C, Buyse B, Verschakelen J, Feenstra L. Correlation between functional, radiological and anatomical abnormalities in upper airway obstruction (UAO) due to tracheal stenosis. *Acta Oto-rhino-laryngol Belg* 1995; 49: 331–339.