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CO transfer capacity as a determining factor of survival for severe hypoxaemic COPD patients under long-term oxygen therapy

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CO transfer capacity as a determining factor of survival for severe hypoxaemic COPD patients under long-term oxygen therapy. P.Dubois, J. Machiels, F. Smeets, J.P. Delwiche, J. Lulling.

ABSTRACT:The transfer capacity for carbon monoxide is a commonly used method in clinical practice but is rarely considered as a prognostic factor for patients under long term oxygen therapy (LTOT). LTOT was applied to 217 stable, severely hypoxaemic (arterial oxygen tension (Pao_2<7.3 kPa or 55 mmHg) chronic obstructive pulmonary disease (COPD) patients, according to the usual recommendations. The average survival rate of this series is rather poor: 46% at 24 months. There is nevertheless an important difference between patients with a normal transfer coefficient and those with a decreased one (79% survival at 2 yrs as compared to 37%). On the other hand, the degree of airflow limitation has no prognostic value in the present series of very disabled patients. We can conclude that hypoxaemic COPD patients with a severely decreased transfer coefficient have a poor prognosis, even under LTOT, compared to patients with an equivalent impairment of airflow limitation and hypoxaemia but with a normal CO transfer factor/alveolar ventilation ratio (TLCO/VA).

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Since the reference studies of the Nocturnal Oxygen Therapy Trial (NOTT-COT) group [1] and the Medical Research Council Working Party CMRC [2] were published, about ten years ago, Long Term Oxygen Therapy (LTOT) has become a usual treatment for severe hypoxaemic patients.

Selection criteria and practical recommendations are standardized [3, 4], even if there are some differences between countries, for medical and scientific reasons, but also for financial ones, as the cost of therapy is rather high. LTOT is nevertheless widely used, but there are relatively few new papers about the results of large series of patients under treatment [5–7]. The clinical experience of many centres has underlined the usefulness of a prolonged observation period to make sure that a real steady state has been obtained [8]; the patient education, the family information and the home controls of compliance and smoking cessation are also very important.

Attention has been drawn to the effects of LTOT on haemodynamics with some divergences in the results [9–11]. Prognostic factors have also been discussed in several papers [1, 2, 5–7, 11]: the severity of airflow limitation, hypoxaemia and hypercapnia, the response to exercise and O_2 therapy, and the daily duration of oxygen therapy are important for the survival rate. Strange enough, very few papers mention the CO transfer capacity and its prognostic value.

After a four year experience with LTOT, we were impressed by the high mortality rate in our series compared to previous published data. Looking for the reasons for this discrepancy, we objectivized the importance of the CO transfer coefficient for prognosis.

Materials and methods

Selection of patients, oxygen therapy and control

The Belgian Social Security regulations for reimbursement of LTOT require an arterial oxygen tension Pao_2<7.3 kPa (55 mmHg) while breathing air, under optimal therapy and in a steady state, controlled during 3 months: this was the main criterion for selection of our patients. There was no age limitation but we excluded patients who suffered from rapidly evolutive diseases (carcinoma, liver or renal failure).

For each candidate to LTOT, we performed lung function tests and blood gas analysis while breathing air and under 2 $l \cdot m^{-1}$ O_2, administered by means of nasal prongs for at least 30 min.

An oxygen concentrator was allocated to compliant patients after careful information on the therapy. The recommended daily O_2 therapy lasted at least 15 h, usually 18 h, *via* nasal prongs at an O_2 flow rate of 2 $l \cdot m^{-1}$. A few patients whose Pao_2 did not reach the target level of 8.6 kPa (65 mmHg) were equipped with a reservoir cannula [12]. The usual pharmacological treatment, including bronchodilators, diuretics and in some cases corticosteroids, was continued. Visits at home were performed by a trained nurse at least every 3 months; in case of an incorrect use of the machine, the patient was once more instructed but if the compliance did not improve, *e.g.* as controlled on the hour meter, the oxygen concentrator was removed. A recent control of the use of the oxygen concentrator has objectivated an average daily use of 16.3±4.8 hours in a group of 31 patients during the period before death and of 17.8±4.1 h in a series of 39 patients who are still under treatment (the difference is not significant).

A yearly control in the hospital is compulsory and the same criteria of hypoxaemia are required for a prolongation of LTOT. We started our LTOT programme in 1985 and we incorporated 340 patients: 254 chronic obstructive pulmonary diseases (COPD) and 86 patients who suffered from other diseases. LTOT was interrupted in 37 cases of COPD for various reasons (lack of compliance, absence of long term stability of blood gases, improvement of Pao_2 one year after beginning treatment) and were discarded; the present paper concerns accordingly 217 COPD patients in a confirmed steady state.

Lung function and blood gases

Spirometry, measurement of single breath transfer factor for carbon monoxide (TLCO) (Morgan Transfer Test or Alveolo Diffusion Test, Jaeger) and body plethysmography (Body Test, Jaeger) were carried out for each candidate; the TLCO of 48 patients who were unable to maintain a 10 s apnoea was not measurable. Predicted values are those of the European Community for Coal and Steel (ECCS) for spirometry [13], those of COTES [14] for transfer, and of PELZER *et al.* [15] for plethysmography.

Blood gases are performed and analysed in a classical way (Corning 175). In many cases, oxy-and carboxy haemoglobin were directly measured (CO-Oxymeter IL282). Haemodynamic data were obtained for about 40% of the patients, with the Swan-Ganz method.

In order to evaluate the effects of LTOT in function of the initial respiratory functional status, we divided the patients into two classes according to the severity of bronchial obstruction or to the value of the transfer coefficient. As far as the bronchial obstruction was concerned, we chose the mean value of the forced expiratory volume in one second (FEV_1) of our patients (30% of predicted; see table I) as a dividing line between two classes. Concerning TLCO/VA (alveolar volume), we chose 70% of predicted value, which is the rounded confidence limit at 95% of the mean value of normal subjects [14], as a limit between classes.

Table 1. – Baseline characteristics of our 217 COPD patients (188 males and 29 females)

	n	Mean	SD
Anthropometry			
Age yrs	217	66	8
Height cm	210	167	7
Weight kgs	197	65	13
Lung function % pred.			
FVC	211	51	14
FEV_1	211	29	12
ITGV	186	149	39
TLCO	169	40	21
TLCO/VA	169	59	31
Blood gases: on air			
Pao_2 kPa	217	6.4	0.8
$Paco_2$ kPa	217	6.4	1.2
pH	197	7.4	0.1
HbCO %	73	2.3	1.1
Haemoglobin g%	146	15.6	2.2
Blood gases: on O_2 2 $l \cdot m^{-1}$			
Pao_2 kPa	215	9.3	1.5
$Paco_2$ kPa	215	6.6	1.3

FVC: forced vital capacity; FEV_1: forced expiratory volume in one second; ITGV: intrathoracic gas volume; TLCO: transfer factor for carbon monoxide in single-breath; TLCO/VA: transfer coefficient; HbCO: measured carboxyhaemoglobin; SD: standard deviation; Pao_2, $Paco_2$: arterial oxygen and carbon dioxide tensions, respectively.

Statistical methods

Comparisons between groups are performed by t- or chi-square tests. The survival rate is evaluated by the actuarial method [16].

Results

The baseline characteristics (table I) of our 217 COPD patients (188 males and 29 females) showed that they were quite old, severely obstructed with major pulmonary hyperinflation; the mean transfer coefficient was low and 48 patients were unable to perform the test. Arterial blood analysis showed a severe hypoxaemia with some degree of polycythaemia and mild hypercapnia; under O_2 administration, we observed a mean Pao_2 improvement of 21.5 mmHg (2.87 kPa) with a slight increase of the arterial carbon dioxide tension ($Paco_2$).

The mean pulmonary artery pressure of the 80 tested patients amounted to 31.6±9.7 mmHg; there was no significant difference between the 27 patients of Group 1 (29.4±8.0 mmHg) and the 41 patients of Group 2 (31.4±8.8 mmHg) and between the latter and the 12 patients of Group 3 (37.3±14 mmHg). The difference between Group 1 and 3 proved significant ($p<0.05$).

The actuarial survival of the whole series of our COPD patients (fig. 1) objectivates a high mortality rate compared to the NOTT-COT and MRC trials.

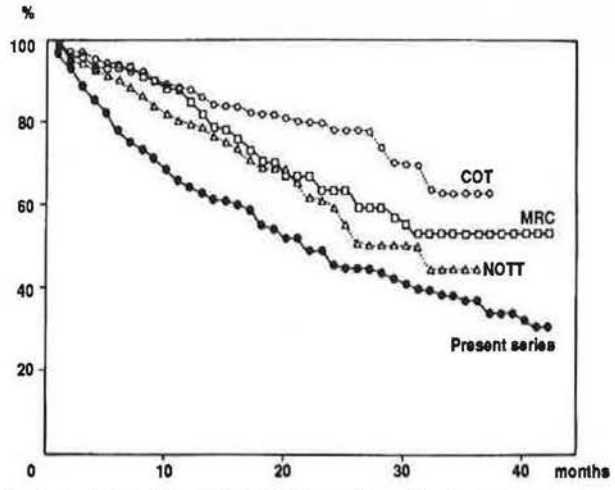

Fig. 1. – Actuarial survival of the patients in the present study, compared to data from the Nocturnal Oxygen Therapy Trial - Continuous Oxygen Therapy (NOTT-COT) and the Medical Research Council (MRC) trials.

The comparison of the lung function data of the patients who were still alive at, and those who were already dead before 12 and 24 months respectively, after LTOT was started (table 2), shows that there is no statistically significant difference in the value of FEV_1 but that TLCO/VA is highly significantly lower in the group of deceased.

Now, if we compare (table 3) the baseline characteristics of our patients with a normal transfer coefficient (Group 1) or with a decreased one (Group 2), we see that Group 2 has a highly significant lower mean body weight and a more pronounced hyperinflation of the lung; on the contrary, we find no significant difference in blood gases while breathing air; under O_2 administration, Group 2 improves the mean Pao_2 significantly more; patients in Group 1 have a higher mean haemoglobin and haematocrit. Patients who are unable to perform the single breath transfer test (Group 3) are significantly older, more hypoxaemic, hypercapnic and more obstructed than those from Group 2.

Table 2. – Comparison of mean FEV_1 and TLCO/VA (% predicted) among still alive patients and those already dead at 12 and at 24 months of treatment

	At 12 months					At 24 months				
% pred	Alive (n=60)		Dead (n=51)			Alive (n=30)		Dead (n=71)		
	Mean	SD	Mean	SD	p	Mean	SD	Mean	SD	p
FEV_1	30	10	30	13	NS	29	11	31	14	NS
TLCO/VA	74	33	49	25	***	78	26	48	24	***

Abbreviations: see Table 1; NS: non-statistically significant; *:p<0.05; **:p<0.01; ***:p<0.001.

Table 3. – Comparision of patients with normal, decreased or non-measurable transfer coefficient for CO

	Group 1 (TLCO/VA≥70% pred.) n=54			Group 2 (TLCO/VA<70% pred.) n=115			Group 3 (non-measurable) n=48	
	Mean	SD	p	Mean	SD	p	Mean	SD
Anthropometry								
Age yrs	65	9	NS	66	8	*	69	8
Height cm	168	5	NS	168	7	NS	165	8
Weight kgs	73	14	***	63	11	NS	61	14
Lung function								
FVC % pred	50	11	*	56	14	***	41	13
FEV_1	32	11	NS	30	13	***	24	11
ITGV	141	42	**	153	36	NS	148	45
TLCO/VA	96	20	***	41	16			
Blood gases: on air								
Pao_2 kPa	6.5	1.5	NS	6.5	0.7	**	6.1	0.9
$Paco_2$ kPa	6.4	0.9	NS	6.2	1.2	**	6.7	1.3
Haemoglobin g	16.5	2.1	**	15.5	2.1	NS	14.9	2.0
Blood gases: on O_2 2 $l \cdot m^{-1}$								
Pao_2 kPa	9.0	1.5	*	9.5	1.5	NS	9.4	1.5
$Paco_2$ kPa	6.7	1.0	NS	6.4	1.3	**	7.1	1.3

Abbreviations: see table 1; NS: non-statistically significant; *: p<0.05; **: p<0.01; ***: p<0.001.

Table 4. – Proportion of patients still alive at, or already dead before 12 and 24 months, according to their initial value of FEV_1 and TLCO/VA

	At 12 months		At 24 months	
	Alive (n=60)	Dead (n=61)	Alive (n=30)	Dead (n=71)
$FEV_1 \geq 30\%$ pred.	26 (23%)	22 (20%)	12 (12%)	32 (31.5%)
$FEV_1 < 30\%$	34 (31%)	29 (26%)	18 (18%)	39 (38.5%)
Chi-square	p=0.98		p=0.64	
TLCO/VA≥70% pred.	34 (31%)	8 (7%)	19 (19%)	10 (10%)
TLCO/VA<70%	26 (23%)	43 (39%)	11 (11%)	61 (60%)
Chi-square	p<0.001		p<0.001	

This table only concerns the 169 patients whose transfer coefficient has been measured. The figures show the number of patients in each sub-group (in parenthesis, expressed as a percentage). For abbreviations see table 1.

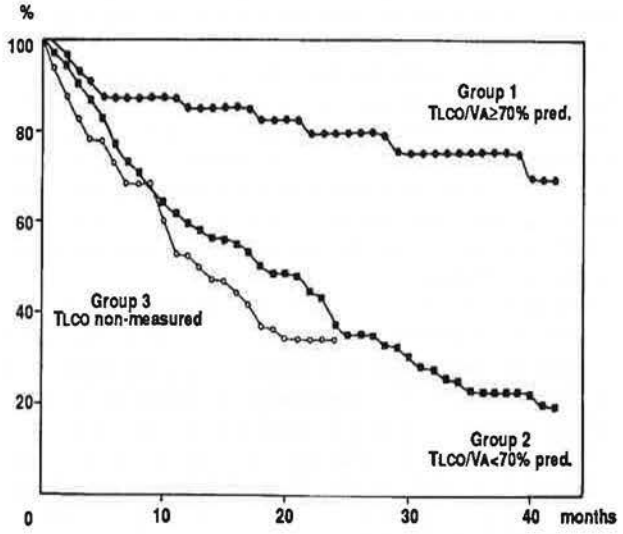

Fig. 2. – Actuarial survival of 3 groups of patients depending on their initial single-breath CO transfer coefficient corrected for alveolar ventilation (TLCO/VA). Chi-square tests between group 1 and 2 as well as between group 1 and 2+3, show that the percentage of survivors is significantly different at 12 months ($p<0.01$) and at 24 months ($p<0.0001$).

The actuarial survival (fig 2) of patients with a TLCO/VA > 70% predicted (Group 1) is better than the survival of those <70% (Group 2) and/or of those non measurable (Group 3).

Table 4 shows the proportion of patients still alive at, or already dead before, 12 and 24 months respectively, according to their initial values of FEV_1 and TLCO/VA (only for the 169 patients in whom it is measured: *i.e.* Group 1 and 2). There is no difference as far as FEV_1 is concerned but the number of patients who died is highly significantly greater is Group 2. where TLCO/VA is low (chi-square; $p<0.001$) .

Among the 114 patients who died, no correlation was found between the duration of survival and the value of FEV_1 or TLCO/VA.

Mortality between males and females is as follows: 11 (38%) of the 29 females and 103 (55%) of the 188 males are already dead. Actuarial survival curves are quite comparable.

Discussion

Prognostic factors in COPD have been extensively studied and discussed in the literature for at least three decades. The most important mortality factors are the bronchial obstruction and the annual rate of decline of FEV_1 [18-23]. All the authors seem to agree on this. Other indices of airflow limitation do not add any original information.

The degree of hypoxaemia correlates with a higher mortality rate [18] and contributes to the development of polycythaemia and pulmonary hypertension.

Many papers reported the poor prognosis of pulmonary hypertension [24] but some authors [18] didn't find any contribution of pulmonary vascular resistance regardless of FEV_1. Severe and chronic hypercapnia has a poor prognosis in COPD patients [18].

The transfer capacity of the lungs has been recognized for a long time as a contributing factor to the prognosis of COPD; BOUSHY *et al.* [18] even concluded, after multiple regression analyses, that FEV_1 and TLCO/VA are "the only tests that contributed significantly to the prediction of the duration of survival".

Prognosis of severely hypoxaemic COPD patients depends upon the same preceding factors but the classical NOTT-COT [1] and MRC [2] studies demonstrated that survival can be improved by LTOT. Some haemodynamic variables improve under O_2 therapy and can predict survival during acute [6, 11] as well as chronic trials [9]. The elegant study of WEITZENBLUM *et al.* [10] has demonstrated that LTOT can reverse the progression of pulmonary hypertension in COPD patients. Other studies [7] observe only a stabilization of pulmonary haemodynamics. On the other hand, lung function and blood gases while breathing air do not improve under LTOT.

If we accept, in accordance with data from the literature, that TLCO/VA is a contributing factor in the evaluation of the prognosis of severe COPD patients, one can be surprised that only one study [7] concerning the results of LTOT mentions the transfer factor but does not analyse its prognostic significance; the other papers, including the reference NOTT-COT and MRC studies [1, 2, 5, 6, 11] do not even mention this test and

only discuss the importance for survival of airway obstruction, initial blood gases and haemodynamics.

The high mortality rate in our series could be explained, either by a poor compliance of the patients or by an inadequate correction of their hypoxaemia. As stated in the MRC study, " compliance cannot be proven", but this is the case for every study. The control, assessed by timers, shows that the average use of O_2 in this study is quite close to the figures of the continuous O_2 therapy group from the NOTT-COT trial, *i.e.* about 17 h a day. Concerning the correction of hypoxaemia, our patients start from slightly lower mean Pao_2 values than those of the reference studies, and achieve, under O_2 administration, a mean PaO_2 (9.3 kPa or 69.8 mmHg) which approaches the result of the MRC data (9.5 kPa or 71 mmHg for females and 10 kPa or 74.9 mmHg for males).

The compliance of our patients and their response to O_2 seem to be comparable to other series and are not an explanation for their lower survival rate. Concerning the smoking habits, notwithstanding our strong recommendations for stopping, some patients continued to smoke; this was also the case in the MRC study. Nevertheless, the mean value of carboxyhaemoglobin suggested that the average tobacco consumption of our patients was not very important.

When we made out that the mortality in our series was higher than in the NOTT-COT and MRC trials, we looked at lung function parameters which could differentiate survivors and deceased, and it appeared that TLCO/VA was strikingly different (table 2), while *e.g.* the degree of airflow limitation and the severity of hypoxaemia did not make any significant difference.

The actuarial survival of patients with a "normal" TLCO/VA, as defined here (see Methods), is much better than that of patients whose transfer coefficient is low and those for whom the test was not feasible (fig 2). The proportion of patients who are still alive or already dead (table 4) is the same in the two classes of airflow limitation, while the proportion of dead is much higher in the class with an abnormally low TLCO/VA as compared to those with a normal CO transfer.

Our results show accordingly that the transfer coefficient is actually related to survival of COPD patients under LTOT. We were nevertheless unable to demonstrate any correlation between TLCO/VA (as well as FEV_1) and the duration of survival of the already dead patients; therefore, it is impossible to predict the survival of an individual by means of a mathematical equation, starting from his initial lung function parameters.

The physiopathologic significance of the transfer factor is complex [26]. Rather than a measurement of the so called "alveolar membrane diffusing capacity", TLCO is determined to a great deal by the reduction of the pulmonary capillary bed due to a variety of lung parenchyma lesions, among which is emphysema.

The classification into two classes, the first one with "normal", the second one with "abnormal" TLCO/VA values, does not mean that two different diseases are in question; according to NASH *et al.* [17, 27] there is a continuum between the A type of COPD patient (the emphysematous or "pink and puffed") and the B type (the bronchitic or "blue and bloated").

In the present series, Group 2 is characterized by a mean low body weight, a decreased transfer coefficient and an important degree of hyperinflation of the lung: Group 2 can therefore rather be considered as having more traits belonging to the A type, while Group 1 is nearer to the characteristics of the B type.

It is well known that hypoxaemia appears quite late in the course of A type COPD patients; this would mean that Group 2, selected on the basis of a Pao_2<7.3 kPa and TLCO/VA<70%, is mainly constituted of patients who are close to the A type in the far advanced stage of their disease.

As a large majority (73%) of our patients are either of the far advanced type. (Group 2) or very severely disabled (Group 3), the poor prognosis of that kind of patients is a possible explanation for the high global mortality rate that we observe.

On the other hand, the patients with a normal transfer factor show a cumulative survival proportion at 24 months (79%) that is comparable to that of the best published results: 78% in COT trial and 83% for COOPER *et al* [7]. It is nevertheless worth noticing that the mean TLCO in the last paper is quite low (3.48±1.87 $mmol \cdot min^{-1} \cdot kPa^{-1}$) but the authors didn't mention more on this interesting point.

The lack of interest for the transfer factor in the literature is surprising because it is a commonly used method in clinical practice which is a good marker of emphysema [17, 26] especially in association with airflow limitation and hyperinflation of the lungs.

With regard to the selection of patients, the Belgian regulations are quite severe (Pao_2<7.3 kPa or 55 mmHg) and this may contribute to the severity of the abnormalities that we observe.

Nevertheless, it is surprising that we do not observe the difference in survival which has been recorded by several studies between very severe and less severely obstructed patients; the effect of the severity of bronchial obstruction on mortality could have been masked by the prevalent influence of TLCO/VA.

A general impression about the survival rate in the present series is that the benefit of LTOT is much less evident than in other published papers, except for those patients who maintain a good transfer coefficient.

In conclusion, the usefulness of LTOT for patients with a very severely impaired transfer capacity should be reconsidered; this would require new prospective studies but our data already show that their prognosis is poor, even under LTOT.

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Capacité de transfert du CO, facteur déterminant de la survie dans les bronchopneumopathies obstructives chroniques hypoxémiques graves sous oxygénothérapie au long cours. P. Dubois, J. Machiels, F. Smeets, J.P. Delwiche, J. Lulling

RÉSUMÉ: La mesure de la capacité de transfert du monoxide de carbone est une technique largement répandue en clinique mais peu utilisée jusqu'ici pour le pronostic des patients sous oxygénothérapie au long cours (LTOT). Nous avons inclu dans notre programme LTOT, selon les recommandations classiques, 217 patients atteints de bronchopneumopathie obstructive chronique (BPCO) en état stable et séverèment hypoxémiques (Pao_2<7.3 kPa ou 55 mmHg). La survie moyenne de nos patients est faible: 46% à 24 mois. Il existe cependant une importante différence entre patients à coefficient de transfert normal et ceux dont T_L est réduit (survie de 79% à 2 ans pour les premiers et de 37% chez les autres). D'autre part, nous n'avons pas mis en évidence de signification pronostique du degré de la limitation au flux aérien dans cette série de patients au stade avancé de leur affection.

Nous concluons que les patients BPCO hypoxémiques dont le coefficient de transfert est très altéré ont un pronostic vital défavorable, même sous oxygénothérapie au long cours, par comparaison aux malades dont la limitation du flux aérien et l'hypoxémie sont équivalentes mais dont le rapport T_L/V_A est normal.

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